Effect of increased preload on the synthesized aortic blood pressure waveform

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The Vascular Function Study Group, Division of Cardiology, and Molecular Cardiology Research Institute, Tufts Medical Center, Boston, Massachusetts; Menzies Research Institute, University of Tasmania, Hobart, Australia; and The Health and Integrative Physiology Laboratory, Department of Sports Informatics, University of Seoul, Seoul, Korea

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Heffernan KS, Sharman JE, Yoon ES, Kim EJ, Jung SJ, Jae SY. Effect of increased preload on the synthesized aortic blood pressure waveform. J Appl Physiol 109: 484–490, 2010. First published June 17, 2010; doi:10.1152/japplphysiol.00196.2010.—In the present study, we examined the influence of preload augmentation via passive leg elevation (PLE) on synthesized aortic blood pressure, aortic augmentation index (AIx), and aortic capacitance (a reflection of aortic reservoir function). Central and peripheral hemodynamics were measured via tonometry with a generalized transfer function in 14 young, healthy men (age = 24 ± 1 yr). Aortic blood flow was calculated from the left ventricular outflow tract (LVOT) velocity-time integral (VTI) using standard two-dimensional echocardiographic-Doppler techniques. Measures were made in the supine position at rest (Pre), during PLE, and during recovery (Post). There was a significant increase in LVOT-VTI, synthesized aortic systolic blood pressure (BP) and AIx from Pre to PLE, with values returning to baseline Post (P < 0.05). There was a reduction in aortic capacitance from Pre to PLE, with values returning to baseline Post (P < 0.05). There was no change in heart rate, systemic arterial compliance, aortic elastance, aortic wave travel timing, or vascular resistance (P > 0.05). Change in AIx from Pre to PLE was associated with change in LVOT-VTI (r = 0.66, P < 0.05) and inversely associated with change in aortic capacitance (r = −0.73, P < 0.05). These data suggest that in a setting of isolated augmented preload with minimal changes in other potential confounders, the morphology of the synthesized aortic BP waveform and AIx may be related to changes in aortic reservoir function.

THE CONTOUR of the central blood pressure (BP) waveform has been traditionally ascribed to the contribution of overlapping incident and reflected pressure waves (31). This interaction is often quantified via a single composite measure, the augmentation index (AIx) (30). Central BP and AIx are each associated with numerous cardiovascular (CV) morbidities and predict future CV events and mortality in select patient populations (37, 44). As such, central BP and AIx may be useful therapeutic targets and secondary endpoints (1, 50).

It has been posited that wave reflections make the largest contributions to the shape of the central BP waveform and AIx (31). However, this has recently been challenged (2). Indeed, new data suggest that AIx may be predominantly associated with the capacitance function of the aorta (7). According to windkessel theory, as the left ventricle ejects blood into a compliant aorta, the vessel expands during systole and then recoils during diastole, converting intermittent pulsatile flow to a more laminar smooth flow. A reduction in this capacitance or “reservoir” function concomitant with an increase in aortic volume may be expected to increase aortic pressure because when proximal aortic inflow exceeds outflow capacity, this volume distends the aorta and generates a rise in pressure (8, 43).

Given the clinical significance of aortic BP and AIx as measures of CV risk, it is important to fully understand the physiological underpinnings of these parameters. The purpose of this study was to investigate the effect of preload augmentation on synthesized aortic BP, aortic AIx, and aortic reservoir function. Passive leg elevation (PLE) was used to nonpharmacologically increase preload without significantly altering heart rate, arterial stiffness, or vascular resistance (known correlates of AIx and potential confounders). We hypothesized that an increase in preload with PLE would increase synthesized aortic BP and AIx while reducing aortic reservoir function.

METHODS

Fourteen healthy young men volunteered to participate in this study (age = 21 ± 1 yr; body mass index = 24.4 ± 0.6 kg/m²; body fat = 16.1 ± 1.1%). Exclusion criteria included self-reported history of hypertension, diabetes mellitus, hyperlipidemia, renal disease, pulmonatory disease, and habitual smoking. One participant was excluded due to a resting systolic BP (SBP)/diastolic BP (DBP) > 140/80 mmHg on serial measures: 154/85 mmHg. All remaining men (n = 13) were free from any overt disease, were nonsmokers, and were not taking medications of any kind. This study was approved by the University Institutional Review Board, and all subjects provided written informed consent before study initiation. All subjects refrained from caffeine and exercise for 24 h before testing.

Study design. Body composition was obtained using bioelectric impedance analysis (Biospace, Inbody 3.0). Echocardiographic, peripheral, and central hemodynamic measurements were obtained with subjects in the supine position following 10 min of quiet rest (Pre). The participant’s legs were then passively elevated using a hinged examination plinth until their femoral ischial joint was at a 60° angle (PLE). This PLE technique has been shown to increase venous return and thus augment preload via classical Frank-Starling mechanisms (16). Each participant was maintained in the PLE position for 5 min with echocardiographic and hemodynamic examinations started at 2 min after initiation of PLE. Following this, both legs were returned to the starting supine position and, after 5 min, all measures were once again repeated (Post).

A control trial was completed in a separate group of 13 young healthy men (age = 22 ± 1 yr; body mass index = 23 ± 1 kg/m²) to examine the effect of vascular path angle manipulation per se on synthesized aortic BP and AIx. For this trial, radial pressure waveforms and brachial BP were obtained with subjects in the supine position (Pre) and with the participant’s upper torso elevated to an angle of 60° (upper torso elevation, UTE). Each participant was maintained in the supported UTE position for 5 min with hemodynamic examination starting 2 min after initiation of UTE.
Echocardiography. Stroke volume was calculated from the left ventricular outflow tract (LVOT) diameter and pulse wave Doppler flow profiles (velocity-time integral, VTI) using standard two-dimensional echocardiographic techniques (ACUSON X 300 Ultrasound Imaging System, Siemens, Mountain View, CA).

Peripheral hemodynamics. Brachial BP was measured in the supine position (at Pre, PLE, and Post) using standard sphygmomanometry and auscultation. All brachial BP measurements were made in duplicate, following established guidelines (16). If these values deviated by more than 5 mmHg, a third measurement was conducted. The average of the two closest values was recorded and used for subsequent analysis.

Central hemodynamics. Radial artery pressure waveforms were attained in the supine position from a 10-s epoch using application tonometry and a high-fidelity strain-gauge transducer (Millar Instruments, Houston, TX). Using a generalized validated transfer function (33), a central aortic pressure waveform was reconstructed from the aforementioned radial artery pressure waveform (SphygmoCor, AtCor Medical, Sydney, Australia) (6). Mean pressure was derived from integration of the area under the central BP waveform. Left ventricular (LV) systolic ejection duration was taken as the time from the foot of the pressure wave upstroke to the incisura of the dicrotic notch (also the point at which end-systolic pressure was calculated). Diastolic time was calculated as total pulse period – LV ejection duration. Augmented pressure (AP) was defined as the difference between central SBP and the pressure at the forward/primary wave peak (P1). P1 was defined as the pressure at the first inflection point – central/aortic DBP. Previous studies have suggested P1 as an index of peak ventricular ejection velocity, assuming no change in aortic root diameter. Conversely, forward wave pressure was defined as P1 + DBP. Aortic AIx was calculated as the ratio of amplitude of the pressure wave above its systolic shoulder (i.e., the difference between the early- and late-systolic peaks of the arterial waveform) to the total pulse pressure (PP) expressed as a percentage [(P1 – P2/PP) × 100]. Peripheral AIx from radial waveforms was calculated as P2/P1.

Aortic AIx in this laboratory is 0.97. Aortic pulse timing (Tr) was determined from the time from the initial upstroke of the pressure wave to the foot of the reflected wave. PP amplification was calculated as the ratio of brachial PP to central PP (10, 14). PP amplification devoid of the influence of wave reflection (nonaugmented PP amplification) was calculated as the ratio of peripheral PP to nonaugmented pressure (i.e., aortic P1 – aortic DBP). The systolic pressure-time integral (the area under the systolic portion of the aortic pressure wave) and the diastolic pressure time index (the area under the diastolic portion of the aortic pressure wave) were quantified to examine systolic-to-diastolic shifts in the proportion of pressure comprising the overall pressure waveform.

Aortic capacitance was calculated as previously described (13, 21, 40). Diastolic runoff (DR), the portion of stroke volume that is stored in the aorta during systole and then flows into peripheral arteries during diastole by means of the cushioning properties of the vessel, was calculated as DR = SAC × (ESP – DP), where SAC is systemic arterial compliance, ESP is end-systolic pressure (obtained from the synthesized aortic pressure wave), and DP is synthesized aortic diastolic pressure. This value was then expressed as a percentage relative to total stroke volume (SV) and has previously been used as a reflection of the reservoir function of the aorta (aortic capacitance = DR/SV × 100) (13, 21, 40). SAC was calculated as τ/PVR, where τ is the diastolic decay time constant and PVR is peripheral vascular resistance [calculated as mean arterial pressure (MAP)/cardiac output (Q)]. τ was calculated (21) as diastolic time interval/(lnESP – lnDP).

Aortic compliance was estimated from the ratio of stroke volume to aortic PP (SV/PP ratio) (4, 35). Effective aortic elastance, a measure of vascular load related to characteristic impedance, was calculated as aortic ESP (synthesized from the aortic pressure waveform)/SV (17). The potential energy of blood pressure, defined as the energy imparted to stretch the walls of the large arteries, was calculated as the product of cardiac output (ml/min) and mean pressure (dyn/cm²). The kinetic energy of blood flow, defined as the energy imparted to accelerate blood through the aortic valve, was calculated as \( E = \frac{m}{2}v^2 \), where \( m \) is blood mass (g/min) and \( v \) is blood velocity (cm/s).

Overall, several vascular methods were used and/or derived in order to provide a comprehensive appraisal of the vascular determinants of synthesized aortic BP. Windkessel theory holds that the cardiovascular system as a closed hydraulic chamber with capacitive (arterial compliance), resistive (vascular resistance), and local inertia (characteristic impedance/elastance) elements. However, this three-element model does not take into account the well-established phenomena of wave reflections. Conversely, wave-only theory (i.e., the aortic BP waveform is comprised of overlapping incident and reflected pressure waves) neglects the capacitance function of the large arteries. Therefore, aforementioned methods included those that make use of windkessel theory (infinite pulse wave velocity) and wave-only theory (finite pulse wave velocity) in an attempt to provide a balanced examination of central hemodynamics and peripheral/central vascular correlates.

Statistical analysis. Statistical analysis was carried out only in normotensive participants (n = 13). All variables were compared over time using an ANOVA with repeated measures. If a significant main effect was detected, post hoc comparisons were made by t-test. A Bonferroni adjustment was made for multiple comparisons. Pearson and Spearman correlation coefficients were used to assess relationships between variables of interest. All data are reported as means ± standard error of the mean (SE). Significance was set a priori as \( P < 0.05 \). All statistical analyses were made using statistical package for the social sciences (SPSS v. 16.0).

RESULTS

There was no significant change in brachial SBP, DBP, or PP in response to PLE. Similarly, there was no change in LVOT diameter from Pre (22.3 ± 0.4 mm) to PLE (22.5 ± 0.4 mm) to Post (22.3 ± 0.3 mm, \( P = 0.813 \)). There was also a significant increase in synthesized aortic SBP and mean pressure from Pre to PLE with values returning to baseline Post (Table 1, \( P < 0.05 \)). There was a significant increase in LVOT-VTI, stroke volume, cardiac output, end-systolic pressure, kinetic energy of flow, systolic pressure-time integral, radial AIx, aortic AIx, and aortic augmentation index rate correct to a heart rate of 75 beats/min (AIx75) from Pre to PLE, with values returning to baseline Post (Fig. 1, Table 1, \( P < 0.05 \)). There was a trend for an increase in nonaugmented pressure (\( P = 0.054 \)) and diastolic run-off (\( P = 0.062 \)) from Pre to PLE, but this did not attain significance (Table 1). There was a reduction in aortic capacitance from Pre to PLE, with values returning to baseline Post (Fig. 1, \( P < 0.05 \)). There was no change in heart rate, SAC, aortic elastance, SV/PP ratio, Tr, or PVR (Tables 1 and 2, \( P > 0.05 \)).

Change in synthesized aortic AIx from Pre to PLE was associated with change in LVOT-VTI (\( r = 0.66, P < 0.05 \)) and change in potential energy of pressure (\( r = 0.52, P < 0.05 \)) and inversely associated with change in aortic capacitance (Fig. 2; \( r = -0.73, P < 0.05 \)). Change in LVOT-VTI from Pre to PLE was associated with change in nonaugmented pressure (\( r = 0.46, P < 0.05 \)), change in \( P_1 (r = 0.67, P < 0.05) \), and change in diastolic run-off (\( r = 0.67, P < 0.05 \)) and inversely associated with change in aortic capacitance (\( r = -0.58, P < 0.05 \)). Change in radial AIx was associated with change in synthesized aortic Alx (\( r = 0.88, P < 0.05 \)). The association
between change in peripheral AIx and change in aortic capacitance ($r = -0.40$, $P = 0.097$) and change in LVOT-VTI ($r = 0.41$, $P = 0.09$) did not reach significance. Change in radial AIx was not associated with change in any other hemodynamic/vascular parameter.

With UTE, there was no significant change in peripheral SBP (Pre: $122 \pm 3$ mmHg vs. UTE: $120 \pm 3$ mmHg), peripheral DBP (Pre: $62 \pm 2$ mmHg vs. UTE: $63 \pm 2$ mmHg), synthesized aortic SBP (Pre: $98 \pm 2$ mmHg vs. UTE: $98 \pm 2$ mmHg), aortic DBP (Pre: $63 \pm 2$ mmHg vs. UTE: $62 \pm 2$ mmHg), or aortic AIx (Pre: $-5 \pm 2\%$ vs. UTE: $-7 \pm 2\%$) (all $P > 0.05$).

Intraclass correlation coefficients (ICC) were calculated from Pre and Post measures to determine test-retest reproducibility for select vascular and echo-based parameters. Results are as follows for ICC: LVOT-VTI = 0.84; LVOT diameter = 0.87; stroke volume = 0.91; synthesized aortic AIx = 0.86; synthesized aortic SBP = 0.91 ($P < 0.05$ for all).

**DISCUSSION**

There were several novel observations to this study. First, there was a significant increase in synthesized aortic SBP and aortic AIx with PLE, with no change in brachial SBP. Second, the change in aortic AIx with PLE was related to change in LVOT-VTI and inversely related to change in aortic capacitance. These data suggest that in a setting of an isolated increase in preload with minimal changes in other potential confounders such as heart rate and peripheral vascular resistance and vascular compliance, the morphology of the synthesized aortic BP waveform and AIx is related to forward-traveling wave properties and the reservoir function of the aorta.

The aortic pressure waveform is a composite of ventricular-vascular load and the pump function of the heart, and there is ample evidence to suggest that some of this interaction may be load dependent. Results from invasive ventricular-vascular coupling studies support this contention and note that an isolated reduction in preload via inferior vena cava constriction reduces central SBP while an increase in preload with dextran/saline infusion increases central SBP (5, 12). Cross-sectional studies note an association between LV function and AIx (3, 45, 46). Patients with heart failure have reduced systolic pressure wave generation, and this may be due to reduced stroke volume (27). This in turn may reduce central BP and AIx in these patients (45). Parenthetically, AIx can be increased with implantation of a LV assist device (38). Enhanced external counterpulsation, a technique that increases preload, also acutely increases aortic pressure (32). In accordance with our findings, Lydakis et al. (25) found that lower-body negative pressure (a technique which reduces preload) causes reductions in AIx. Similarly, head-up tilt reduces central but not peripheral BP (18). However, lower-body negative pressure and head-up tilt also cause reflexive sympathetically mediated increases in heart rate, which may confound the association between preload and AIx. Indeed, Sharman et al. noted an association between LV contractility and AIx with infusion of dobutamine; however, with profound and inseparable changes in heart rate, the association of LV function with AIx is abolished (39). Thus it was concluded that chronotropic factors trump inotropic factors in modulating AIx, and this can be independent of changes in wave reflection kinetics/dynamics (39).

PLE has been used as a means of increasing preload (via classic Frank-Starling mechanisms) in numerous settings. In the present study, PLE caused significant increases in LVOT-VTI and stroke volume. We also noted an increase in AIx with PLE. There was no change in heart rate with PLE in accordance with previous findings (15, 19), removing a potentially important confounding factor on the association of preload augmentation with AIx (48, 49). Change in LVOT-VTI with PLE was associated with forward wave pressure suggesting that increases in synthesized central BP and AIx may be a function of incident wave characteristics in this setting (Fig. 3). Indeed changes in the central pressure waveform with aging may be a consequence of greater forward wave pressure rather than reflected wave pressure (7, 28, 29). Thus factors governing genesis of the incident pressure wave should not be

### Table 1. Central hemodynamics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre</th>
<th>PLE</th>
<th>Post</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brachial SBP, mmHg</td>
<td>122 ± 2</td>
<td>124 ± 3</td>
<td>120 ± 3</td>
<td>0.130</td>
</tr>
<tr>
<td>Brachial DBP, mmHg</td>
<td>72 ± 3</td>
<td>74 ± 2</td>
<td>73 ± 2</td>
<td>0.236</td>
</tr>
<tr>
<td>Brachial PP, mmHg</td>
<td>49 ± 2</td>
<td>50 ± 3</td>
<td>47 ± 3</td>
<td>0.494</td>
</tr>
<tr>
<td>Mean pressure, mmHg</td>
<td>86 ± 2</td>
<td>89 ± 2</td>
<td>86 ± 2</td>
<td>0.043</td>
</tr>
<tr>
<td>Aortic SBP, mmHg</td>
<td>102 ± 2</td>
<td>106 ± 2</td>
<td>101 ± 2</td>
<td>0.021</td>
</tr>
<tr>
<td>Aortic DBP, mmHg</td>
<td>73 ± 3</td>
<td>75 ± 2</td>
<td>74 ± 2</td>
<td>0.150</td>
</tr>
<tr>
<td>Aortic PP, mmHg</td>
<td>29 ± 1</td>
<td>31 ± 2</td>
<td>27 ± 1</td>
<td>0.276</td>
</tr>
<tr>
<td>PP amplification, %</td>
<td>1.71 ± 0.03</td>
<td>1.64 ± 0.04</td>
<td>1.72 ± 0.02</td>
<td>0.210</td>
</tr>
<tr>
<td>Augmented pressure, mmHg</td>
<td>-3 ± 1</td>
<td>-1 ± 1</td>
<td>-3 ± 1</td>
<td>0.002</td>
</tr>
<tr>
<td>Nonaugmented pressure, mmHg</td>
<td>102 ± 2</td>
<td>105 ± 2</td>
<td>101 ± 2</td>
<td>0.054</td>
</tr>
<tr>
<td>Nonaugmented PP amplification, %</td>
<td>1.70 ± 0.02</td>
<td>1.70 ± 0.02</td>
<td>1.72 ± 0.02</td>
<td>0.584</td>
</tr>
<tr>
<td>P1 height, mmHg</td>
<td>28 ± 1</td>
<td>30 ± 2</td>
<td>27 ± 1</td>
<td>0.412</td>
</tr>
<tr>
<td>Aortic AIx, %</td>
<td>-14 ± 2</td>
<td>-9 ± 2</td>
<td>-14 ± 2</td>
<td>0.003</td>
</tr>
<tr>
<td>End systolic pressure, mmHg</td>
<td>89 ± 3</td>
<td>93 ± 2</td>
<td>90 ± 2</td>
<td>0.026</td>
</tr>
<tr>
<td>Systolic PTL, mmHg/s per min</td>
<td>1978 ± 86</td>
<td>2080 ± 77</td>
<td>1877 ± 87</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diastolic PTL, mmHg/s per min</td>
<td>3193 ± 97</td>
<td>3285 ± 69</td>
<td>3302 ± 83</td>
<td>0.132</td>
</tr>
<tr>
<td>Tr, ms</td>
<td>163 ± 6</td>
<td>176 ± 6</td>
<td>166 ± 7</td>
<td>0.223</td>
</tr>
<tr>
<td>Radial AIx, %</td>
<td>35 ± 3</td>
<td>41 ± 3</td>
<td>37 ± 2</td>
<td>0.018</td>
</tr>
</tbody>
</table>

Data are means ± SE. Pre, supine position at rest; PLE, passive leg elevation; Post, recovery; SBP, systolic blood pressure; DBP, diastolic blood pressure; PP, pulse pressure; Tr, pressure wave travel time; P1, pressure at the first inflection point/systolic peak; PTL, pressure-time integral; AIx, augmentation index; AIx50, augmentation index corrected to a heart rate of 75 beats/min.
neglected when appraising central hemodynamics and its derivatives, namely AIx.

Each cardiac cycle generates an outgoing pressure wave that traverses the aorta. These pressure waves arrive at areas of impedance mismatch in the periphery and are reflected back to the left ventricle summating with the forward pressure wave.

There are numerous potential major and diffuse reflection sites that give rise to a single effective reflection coefficient (−0.85 at rest) (37). One of the more prominent reflection sites occurs just distal to the abdominal aorta at the level of the aortic bifurcation/aortoiliac junction/iliac bifurcation (20, 30, 34, 42). Thus it is plausible that PLE may physically alter the geometry of the regional vascular bed, altering a significant wave reflection site. Indeed, bilateral femoral occlusion increases distal wave reflection (20). We included a control trial, consisting of UTE, to address this issue. Aortic geometry at the level of the aortic bifurcation/aortoiliac junction/iliac bifurcation was manipulated in a similar fashion as during PLE using upper torso elevation (maintained at an angle of 60°), theoretically creating a similar physical geometric alteration to the regional vasculature. With this manipulation, we noticed no change in synthesized AIx or aortic BP. Thus the possibility that changes in AIx were due to PLE physically altering potential reflection sites seems unlikely.

Another potential modulator of AIx is vascular resistance. A reduction in PVR owing to peripheral vasodilation may alter reflection sites, reducing the reflection coefficient and altering timing/magnitude of pressure from wave reflections. During PLE, there may be baroreflex/cardiac mechanoreflex-mediated modulation of brachial artery tone (i.e., increases in brachial artery diameter) (23). In normotensive individuals, these...
In the present study, there was a significant increase in synthesized aortic AIx witnessed in the present study in young men may have been influenced by baseline vessel compliance.

Clinical implications. While there was no change in brachial SBP with PLE, there was a significant increase in synthesized aortic SBP. PLE is a commonly used method to increase blood pressure for the initial treatment of hypovolemic shock (19). However, PLE has no benefit in normovolemic patients with a stable cardiocirculatory status (11, 36). It is likely that the lack of significant effect noted in these studies was related to measurement of peripheral and not central BP. The discrepancy between the central and peripheral BP response to PLE highlights that noninvasive assessment of central BP may have clinical utility for the monitoring of fluid responsiveness and aid in diagnosis of central hypovolemia. Moreover, the findings of this study indicate that elevated LV preload and inappropriate ventricular-vascular interaction contribute to increases in AIx. Thus strategies to improve this relationship may represent an important target for intervention.

Limitations to this study should be noted. Measures of aortic BP and AIx were noninvasively and indirectly synthesized from peripheral pressure waveforms using a generalized transfer function (GTF). The validity of this method has been shown to be robust at rest and with hemodynamic perturbations such as Valsalva maneuver, abdominal compression, nitroglycerin administration, and vena caval obstruction (6, 33, 41). Individualized transfer functions do not substantially improve aortic waveform derivation under resting conditions (47). Although invasive measures would produce more accurate measures of aortic pressure, AIx is a relative measure that does not change if the waveform is calibrated using different pressures. We did not consider these limitations in our analysis.

Table 1: Changes in aortic pressure and AIx variables with PLE

<table>
<thead>
<tr>
<th>Variable</th>
<th>Rest</th>
<th>PLE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ao SBP</td>
<td>107 mmHg</td>
<td>99 mmHg</td>
</tr>
<tr>
<td>Ao AIx</td>
<td>-11 %</td>
<td>-18 %</td>
</tr>
<tr>
<td>Ao PP</td>
<td>27 mmHg</td>
<td>24 mmHg</td>
</tr>
<tr>
<td>Ao P1</td>
<td>27 mmHg</td>
<td>24 mmHg</td>
</tr>
<tr>
<td>Ao AP</td>
<td>-3 mmHg</td>
<td>-4 mmHg</td>
</tr>
<tr>
<td>PT1_systolic</td>
<td>1573 mmHg/s per min</td>
<td>1332 mmHg/s per min</td>
</tr>
<tr>
<td>PT1_diastolic</td>
<td>3863 mmHg/s per min</td>
<td>3764 mmHg/s per min</td>
</tr>
</tbody>
</table>

Fig. 3. Sample synthesized aortic pressure waveform at rest (Pre) and during PLE. All participants had type C waveforms (i.e., negative AIx due to the inflection point occurring after peak pressure during waveform downstroke, with a subsequent fall in pressure after the shoulder). Note the increase in aortic systolic pressure and AIx due primarily to an increase in pulse pressure/forward wave pressure, a greater shift in pressure to the systolic portion of the aortic waveform [increased pressure-time integral (PTI) during systole], and no significant change in augmented pressure (AP). Ao, aortic; SBP, systolic blood pressure; PP, pulse pressure; P1, pressure at the forward/primary wave peak; PP2, pressure at the inflection point.
not calculate aortic reservoir pressure but rather capacitance, a reflection of aortic reservoir flow (diastolic run-off relative to total stroke volume expressed as a percentage). Aortic reservoir pressure is the pressure generated from aortic distension from blood entering the aorta faster than it can leave (7). It is proportional to the volume of blood stored in the aorta (7). Our examination of aortic capacitance/reservoir function represents the converse of this principle, i.e., the discharge of stored blood volume during diastolic recoil.

In conclusion, an isolated increase in LV preload is associated with an increase in synthesized aortic AIx and aortic SBP, but not brachial SBP. The change in AIx in relation to aortic capacitance and forward wave pressure. These findings provide evidence that the morphology of the synthesized aortic pressure waveform (and in turn AIx) may be affected by factors other than wave reflection and recapitulate previous contentions. The change in AIx is related to aortic volume during diastolic recoil.

DISCLOSURES
No conflicts of interest, financial or otherwise, are declared by the author(s).

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