Amplitude and Timing of Central Aortic Pressure Wave Reflections in Heart Transplant Recipients


**Background:** Hypertension (HTN) assessed by sphygmomanometer is a common finding in heart transplant recipients (HTR); however, little is known about the contribution of arterial wave reflection to central aortic pressure in these patients. The aim of this study was to measure the central aortic pressure wave in HTR on antihypertensive therapy and determine the effects of amplitude and timing of wave reflection on the various components of the wave.

**Methods:** A total of 53 stable adult HTR on antihypertensive medication underwent brachial artery blood pressure (BP; by sphygmomanometry) and central aortic pressure (by noninvasive radial artery applanation tonometry and use of a generalized transfer function) measurements at rest. Central aortic augmentation index (Ala), an indicator of arterial stiffness, was calculated from the aortic pressure waveform. Patients were divided into three groups (A, B, and C) based on the amplitude of Ala.

**Results:** Mean brachial BP was 136 ± 15/84 ± 9.4 mm Hg. Group A patients (n = 25) had a higher Ala (average 21% ± 7.6%) than group B (n = 18, Ala = 6.5% ± 3.0%, P < .001) or group C (n = 10, Ala = −8.7% ± 8.1%, P < .001) patients. The amplitude of Ala was inversely related to the travel time (Δt/2) of the reflected pressure wave from the periphery to the heart (r = −0.78, P < .001). Despite this clear stratification of patients by aortic pulse wave analysis, standard cuff pressure was similar among the groups.

**Conclusions:** Noninvasive analysis of the central aortic pressure wave identified a subgroup of hypertensive HTR with increased arterial stiffness, increased propagation of the reflected wave, and augmented aortic systolic and pulse pressure not identified with the sphygmomanometer. Am J Hypertens 2002;15:809–815 © 2002 American Journal of Hypertension, Ltd.

**Key Words:** Augmentation index, wave reflection, arterial stiffness, heart transplant, hypertension.

Heart transplant recipients (HTR) are a unique population of cardiac patients who are at increased risk for adverse cardiovascular events. These patients are frequently hypertensive, diabetic, hyperlipidemic, overweight, and have a high incidence of insulin resistance and elevated plasma levels of homocysteine.1–4 Given the significant cardiovascular risk profile of patients after cardiac transplantation, cardiologists are faced with the need to aggressively approach the recognized adverse cardiac risks and to correct them whenever possible.

Post-transplantation hypertension (HTN) is perhaps the most well recognized complication of cardiac transplantation; it has been linked to increased aortic elastance or stiffness2 and to chronic use of cyclosporine and corticosteroids.6 Many patients require three or more antihypertensive agents to optimally control their chronic post-transplant HTN. Traditionally, the brachial sphygmomanometer cuff technique has been used for clinical determination of arterial blood pressure (BP) in HTR, and it is the benchmark for clinical decision-making. However, recent studies have shown that this technique offers distorted or incomplete information about left ventricular (LV) afterload and function,7 especially during pharmacologic interventions.8 Consideration of the aortic pressure wave and alterations in the amplitude and timing of wave reflection provide additional information about arterial function and should better define the degree of arterial...
stiffness in HTR. New techniques have recently become available to measure central aortic BP and augmentation index noninvasively, which could add greater insight into the mechanism of hypertension and BP reduction with antihypertensive medication. The aims of this study, therefore, were 1) to compare central aortic systolic BP with antihypertensive medication. The study included 53 patients (46 men and seven women), with an average age of 55 ± 10 years (Table 1). They were 178 ± 6.7 cm tall, weighed 89 ± 15 kg, and had a body mass index (BMI) of 27 ± 3.9 kg/m². Each patient was taking one or more antihypertensive drugs in addition to standard antirejection medication, and the average number of antihypertensive drugs used per patient was 2.3 ± 1.1. Loop diuretics were required by a large number (45%) of patients to manage postoperative volume retention; for the purposes of our analysis loop diuretics were classified as antihypertensive agents. Three patients were taking a β-blocker, 13 an angiotensin converting enzyme (ACE) inhibitor, 14 an angiotensin receptor blocker (ARB), 24 a calcium channel blocker, and 20 an α-blocking. A total of 14 patients were on monotherapy for HTN. A substantial proportion (81%) of patients were on a 3-hydroxy-3-methylglutaryl coenzyme A (HMG CoA) reductase inhibitor for management of hyperlipidemia and approximately half (49%) of the patients were under active treatment for.

Methods
A total of 53 stable adult HTR (age range 21 to 70 years) were recruited for participation in the study. All patients were taking antirejection and antihypertensive medication at the time of the study, and control of HTN was believed to be adequate based on measurements of brachial cuff BP. The protocol was reviewed and approved by the local Institutional Review Board, and all patients gave informed consent to participate in the research project. Study patients were at least 10 days postoperative from their transplantation procedure and were recruited at the time of presentation for routine post-transplantation follow-up. Patients with known active cardiac rejection, infection, or uncontrolled hypertension were excluded. Patients were in the fasting state at the time of evaluation.

After a supine resting period of at least 15 min, patients underwent duplicate measurements of brachial arterial cuff BP by oscillometric sphygmomanometry and calibrated noninvasive radial artery pulse waveform using application tonometry and a high-fidelity strain gauge transducer (Millar Instruments, Houston, TX). A central aortic BP waveform was synthesized from the measured radial artery pressure waveform using a generalized transfer function (SphygmoCor, Sydney, Australia), and the amplitude and timing of pulse wave reflections were calculated (Fig. 1). Left ventricular ejection time (LVET) was measured from the foot of the pressure wave upstroke (or forward traveling wave), Pd, to the trough of the incisura. Diastolic time was measured from the trough of the incisura to Pd. The travel time (Δt) of the forward pressure wave from the heart to the major reflection site and back was measured from Pd to the upstroke of the reflected wave, P1. The augmentation index (AIa), an indicator of arterial stiffness and wave reflection amplitude, was obtained from the synthesized aortic pressure waveform (Fig. 1). According to the method of Murgo et al.12 patients were stratified into three groups based upon the amplitude of AIa, with an AIa >12% denoting a type A waveform associated with elevated aortic stiffness and wave reflection intensity, an AIa of between 0% and 12% denoting a type B waveform associated with moderate aortic stiffness and wave reflection intensity, and an AIa < 0% denoting a type C waveform, which is considered normal.

Statistical analysis used the statistical package supplied with Microsoft Excel 2000 (Microsoft, Redmond, WA). Analysis of variance and Student t test were used to compare the group differences, and data were expressed as means and standard deviations. A value of P < .05 was considered significant. Multiple regression analysis was performed to establish the relation between the various cardiovascular variables.

Results
Subject Characteristics
The study included 53 patients (46 men and seven women), with an average age of 55 ± 10 years (Table 1). They were 178 ± 6.7 cm tall, weighed 89 ± 15 kg, and had a body mass index (BMI) of 27 ± 3.9 kg/m². Each patient was taking one or more antihypertensive drugs in addition to standard antirejection medication, and the average number of antihypertensive drugs used per patient was 2.3 ± 1.1. Loop diuretics were required by a large number (45%) of patients to manage postoperative volume retention; for the purposes of our analysis loop diuretics were classified as antihypertensive agents. Three patients were taking a β-blocker, 13 an angiotensin converting enzyme (ACE) inhibitor, 14 an angiotensin receptor blocker (ARB), 24 a calcium channel blocker, and 20 an α-blocker. A total of 14 patients were on monotherapy for HTN. A substantial proportion (81%) of patients were on a 3-hydroxy-3-methylglutaryl coenzyme A (HMG CoA) reductase inhibitor for management of hyperlipidemia and approximately half (49%) of the patients were under active treatment for.
diabetes, of whom 17% were insulin dependent and 83% were non-insulin dependent.

Brachial artery cuff systolic BP was 136 ± 15 mm Hg, diastolic BP was 84 ± 9.4 mm Hg, mean BP was 102 ± 9.6 mm Hg, and pulse pressure was 52 ± 14 mm Hg. Central aortic BP components were 120 ± 13, 86 ± 9.4, 103 ± 11, and 34 ± 11 mm Hg, respectively. Heart rate was 88 ± 13 beats/min and was inversely related to both aortic (r = −0.61, P < .001) and brachial artery (r = −0.42, P < .001) pulse pressure. The LVET was 279 ± 24 msec (41% ± 4.4% of the cardiac cycle) and diastolic time was 411 ± 93 msec (59% ± 4.5% of the cardiac cycle). Both LVET and diastolic time were directly related to aortic pulse pressure (r = 0.56 and r = 0.47, respectively, both P < .001) and were inversely related to heart rate (r = −0.76 and r = −0.89 respectively, both P < .001).

When the patients were separated according to the type of waveform identified from the synthesized central aortic pressure wave, 25 had type A waveforms (AIa = 21% ± 7.6%; group A), 18 had type B waveforms (AIa = 6.5% ± 3.0%; group B), and 10 had type C waveforms (AIa = −8.7% ± 8.1%; group C). An example from each group of the measured radial artery pressure wave and synthesized ascending aortic pressure wave is shown in Fig. 2.

There were no statistically significant differences in the average age, height, weight, BMI, number or type of antihypertensive drugs, or number of months post-transplantation among the three patient groups (Table 1). Likewise, there was no difference in brachial systolic, diastolic, or mean BP between the groups despite their segregation into three distinct groups based upon the central aortic pressure waveform. Patients in each group were taking fairly similar proportions of each class of antihypertensive agent, with the highest use of ACE inhibitors, ARB drugs, and calcium channel blockers in the patients with the highest augmentation index (group A). Among the group A patients, 65% were taking either an ACE inhibitor or an ARB drug, 56% were taking a calcium channel blocker, 34% an α-blocker, 8% a β-blocker, and 47% a loop diuretic. Among the group B patients, 38% were on ACE inhibitors or ARB drugs, 31% were on calcium channel blockers, 38% α-blockers, 6% β-blockers, and 44% loop diuretics. In the group C patients, 55% were on ACE inhibitors or ARB drugs, 54% calcium channel blockers, 55% α-blockers, none were on β-blockers, and 45% were taking a loop diuretic.

### Table 1. Patient characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Type A (n = 25)</th>
<th>Type B (n = 18)</th>
<th>Type C (n = 10)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>57 ± 9.0</td>
<td>57 ± 7.8</td>
<td>49 ± 13</td>
<td>NS</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>179 ± 7.0</td>
<td>177 ± 7.6</td>
<td>176 ± 7.6</td>
<td>NS</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>89 ± 14</td>
<td>85 ± 13</td>
<td>90 ± 17</td>
<td>NS</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>28 ± 3.25</td>
<td>27 ± 2.9</td>
<td>29 ± 5.5</td>
<td>NS</td>
</tr>
<tr>
<td>Antihypertensive medications</td>
<td>2.4 ± 1.3</td>
<td>1.7 ± 0.9</td>
<td>2.2 ± 0.9</td>
<td>NS</td>
</tr>
<tr>
<td>Post-transplantation (months)</td>
<td>32 ± 26</td>
<td>35 ± 43</td>
<td>33 ± 37</td>
<td>NS</td>
</tr>
</tbody>
</table>

BMI = body mass index; NS = not significant.

Amplitude of the Various Pressure Components

Brachial artery pulse pressure was higher in group A than in group C patients (57 ± 14 vs 45 ± 9.2 mm Hg, P < .008), and systolic BP tended to be higher (140 ± 16 vs 131 ± 10, P = .058) but did not reach statistical significance (Table 2). Synthesized ascending aortic pulse pressure was higher in group A patients than either group B (41 ± 11 vs 30 ± 8.6 mm Hg, P < .001) or group C patients (41 ± 11 vs 24 ± 5.5 mm Hg, P < .001). Also, patients in group B had a higher aortic pulse pressure than group C patients (30 ± 8.6 vs 24 ± 5.5 mm Hg, P < .04). Aortic systolic BP was higher in group A (126 ± 14, P < .001) and group B (115 ± 12, P < .01) patients than in group C patients (113 ± 7.1 mm Hg), and nonaugmented pressure, Pd − Ps, was higher (32 ± 8.1 mm Hg) in group A patients than in group C patients (24 ± 5.4 mm Hg, P < .002).

The reflected pressure wave from the lower body arrived in the ascending aorta during early systole in group A and group B patients; and its amplitude (Ps − Pa), was greater (9.0 ± 4.3 mm Hg and 2.0 ± 1.2 mm Hg, respectively, both P < .001) than reflected wave amplitude in group C (0 mm Hg, P < .001) patients. Compared with brachial systolic BP, central aortic systolic BP was less in each of the three groups. As a result, the difference between central aortic and brachial systolic BP was significantly less (P < .001) in groups A and B than in group C patients. Augmentation index, AIa, was positive in group A and B patients and was negative in group C patients (see above). When referenced to a normal heart rate of 75 beats/min, AIa became positive in all three groups and remained higher in group A (24% ± 6.7%) than in group B (14% ± 3.1%) or group C (5.0% ± 5.3%) patients. For the entire group, AIa was 10% ± 14% and was inversely related to heart rate (r = −0.68, P < .001).

Timing of the Various Pressure Components

Heart rate was greater in group C patients (100 ± 10 beats/min) than in group B (91 ± 6.2 beats/min, P < .001)
or group A patients (81 ± 11 beats/min, \(P < .001\)) (Table 3). Heart rate was also significantly greater in group B compared with group A patients (\(P < .001\)). In group A patients, the pressure wave traveled faster to and from the major reflection site and arrived at the LV early so that \(\Delta t_p\) (113 ± 5.8 msec) was reduced in group A patients compared with group B (131 ± 8.6 msec, \(P < .001\)) and group C (146 ± 5.7 msec, \(P < .001\)) patients. The \(\Delta t_p\) was also less in group B compared with group C patients (\(P < .001\)). Travel time of the reflected wave, \(\Delta t_p/2\), from the periphery to the heart for the entire group of patients was inversely related to AIa (\(r = -0.78, P < .001\)). The LVET was greater in group A (293 ± 22 msec) than in either group B (264 ± 17 msec, \(P < .001\)) or group C (267 ± 15 msec, \(P < .001\)) patients. Also, group A patients (457 ± 102 msec) had a greater diastolic duration than either group B (385 ± 43, \(P < .003\)) or group C (342 ± 75 msec, \(P < .001\)) patients.

### Discussion

The present study demonstrates that hypertensive HTR can be stratified into groups of high, medium, and low degrees of arterial stiffness based on noninvasive analysis of the aortic pressure wave, and that these stratifications are not possible by means of standard brachial cuff BP measurements alone. In the group A patients (and, to a lesser extent, the group B patients), a subgroup of patients was therefore identified with highly abnormal arterial stiffness and elevated augmented systolic BP despite relatively
controlled brachial BP. These patients, already at high risk for cardiovascular events by virtue of their cardiac transplantation procedure, are therefore not optimized on their cardiovascular drugs and may represent targets for more aggressive risk factor intervention or pharmacotherapy. Therefore, the goal of antihypertensive drug therapy in these patients, in addition to reducing mean arterial pressure, should also be to reduce the amplitude of the reflected wave by delaying its return to the central aorta so that it occurs after peak pressure or during diastole.

Results of cardiac catheterization have consistently shown that HTR have significant abnormal elevations in heart rate and aortic systolic, diastolic, mean, and pulse BP. The observed systemic arterial hypertension is likely due to hypervolemia and increased systemic vascular resistance secondary to cyclosporine-induced sodium retention and persistence of heart failure-related increase in elastic artery stiffness. Increased arterial stiffness increases pulse wave velocity and causes the reflected wave to arrive early, increasing aortic pressure in late systole. These elevated LV afterload components lead to a decrease in stroke volume and cardiac output, an increase in myocardial mass, and a decrease in diastolic BP. The present study identified an increased AIa, a reduced \( \Delta t_p/2 \) and increased pulse pressure in the group A patients, all of which are markers of increased LV afterload and cardiovascular risk.

The system used to obtain the central aortic pressure waveform from the radial artery pressure waveform uses a generalized mathematical transfer function to synthesize the ascending aortic pressure waveform. The use of this transfer function has been previously validated in human subjects. Comparison of the noninvasive synthesized ascending aortic pressure waveform with simultaneous direct measurement of the intra-arterial ascending aortic waveform has shown excellent agreement at baseline and during physiologic and pharmacologic interventions. Also, the reproducibility of the method has been found to be excellent, with the standard deviation of the difference between repeated measurements being about 4.0%. Several recently published articles have successfully used the SphygmoCor system to study cardiovascular responses to a variety of diseases and vasoactive drug interventions.

Care must be taken when using AIa as an index of arterial stiffness in subjects of different heights and heart rates. Both of these variables are inversely related to AIa. In the present study, height, weight, and BMI were similar in the three groups, but heart rate was lowest in the group with the highest AIa (group A).

### Table 2. Amplitudes of the various pressure components

<table>
<thead>
<tr>
<th>Pressure component</th>
<th>Type A ((n = 25))</th>
<th>Type B ((n = 18))</th>
<th>Type C ((n = 10))</th>
<th>(A \times B) ((P))</th>
<th>(A \times C) ((P))</th>
<th>(B \times C) ((P))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brachial systolic BP (mm Hg)</td>
<td>140 ± 16</td>
<td>134 ± 15</td>
<td>131 ± 10</td>
<td>NS</td>
<td>&lt;.059</td>
<td>NS</td>
</tr>
<tr>
<td>Brachial diastolic BP (mm Hg)</td>
<td>83 ± 10</td>
<td>85 ± 10</td>
<td>86 ± 5.6</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Brachial mean BP (mm Hg)</td>
<td>102 ± 12</td>
<td>101 ± 11</td>
<td>101 ± 8.4</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>Brachial pulse pressure (mm Hg)</td>
<td>57 ± 14</td>
<td>49 ± 14</td>
<td>45 ± 9.2</td>
<td>NS</td>
<td>&lt;.008</td>
<td>NS</td>
</tr>
<tr>
<td>Aortic systolic BP (mm Hg)</td>
<td>126 ± 14</td>
<td>115 ± 12</td>
<td>113 ± 7.1</td>
<td>&lt;.01</td>
<td>&lt;.001</td>
<td>&lt;.04</td>
</tr>
<tr>
<td>Aortic pulse pressure (mm Hg)</td>
<td>41 ± 11</td>
<td>30 ± 8.6</td>
<td>24 ± 5.5</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>((P_0 – P_s)) (mm Hg)</td>
<td>9.0 ± 4.3</td>
<td>2.0 ± 1.2</td>
<td>0</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>((P_0 – P_d)) (mm Hg)</td>
<td>32 ± 8.1</td>
<td>28 ± 7.9</td>
<td>24 ± 5.4</td>
<td>NS</td>
<td>&lt;.002</td>
<td>NS</td>
</tr>
<tr>
<td>Brachial systolic BP – aortic systolic pressure (mm Hg)</td>
<td>14 ± 5.0</td>
<td>18 ± 5.3</td>
<td>19 ± 4.6</td>
<td>&lt;.02</td>
<td>&lt;.01</td>
<td>NS</td>
</tr>
<tr>
<td>AIa (%)</td>
<td>21 ± 7.6</td>
<td>6.5 ± 3.0</td>
<td>-8.7 ± 8.1</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.002</td>
</tr>
<tr>
<td>AIa (%) at heart rate of 75 beats/min</td>
<td>24 ± 6.7</td>
<td>14 ± 3.1</td>
<td>5.0 ± 5.3</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

\(BP\) – blood pressure; \(AIa\) – aortic augmentation index; \((P_0 – P_s)\) – amplitude of the reflected wave; \((P_0 – P_d)\) – amplitude of the forward or incident wave; other abbreviation as in Tables 1 and 2.

### Table 3. Timing of the various pressure components

<table>
<thead>
<tr>
<th>Pressure Component</th>
<th>Type A ((n = 25))</th>
<th>Type B ((n = 18))</th>
<th>Type C ((n = 10))</th>
<th>(A \times B) ((P))</th>
<th>(A \times C) ((P))</th>
<th>(B \times C) ((P))</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart rate (beats/min)</td>
<td>81 ± 11</td>
<td>91 ± 6.2</td>
<td>100 ± 10</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.03</td>
</tr>
<tr>
<td>Ejection time (msec)</td>
<td>293 ± 22</td>
<td>264 ± 17</td>
<td>267 ± 15</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>NS</td>
</tr>
<tr>
<td>% of Cardiac cycle</td>
<td>40 ± 4.6</td>
<td>41 ± 3.1</td>
<td>44 ± 3.4</td>
<td>NS</td>
<td>&lt;.01</td>
<td>&lt;.02</td>
</tr>
<tr>
<td>Diastolic duration (msec)</td>
<td>457 ± 102</td>
<td>385 ± 43</td>
<td>342 ± 75</td>
<td>&lt;.003</td>
<td>&lt;.001</td>
<td>NS</td>
</tr>
<tr>
<td>% of Cardiac cycle</td>
<td>60 ± 4.6</td>
<td>59 ± 3.1</td>
<td>57 ± 5.5</td>
<td>NS</td>
<td>NS</td>
<td>NS</td>
</tr>
<tr>
<td>(\Delta t_p) (msec)</td>
<td>113 ± 5.8</td>
<td>131 ± 8.6</td>
<td>146 ± 5.7</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>(\Delta t_f/2) (msec)</td>
<td>57 ± 5.9</td>
<td>66 ± 4.3</td>
<td>73 ± 2.8</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

\(\Delta t_p\) – travel time of the pressure wave to and from the periphery; \(\Delta t_f/2\) – travel time of the reflected wave from the periphery to the heart; other abbreviation as in Tables 1 and 2.
Ala was adjusted to a normal heart rate of 75 beats/min in the three groups, however, a similar pattern was observed, with Ala still significantly elevated in the group A patients and moderately elevated in the group B patients. This observation suggests a progressive increase in arterial stiffness from the group C to the group A patients, even though these patients were taking antihypertensive medication and had controlled BP as assessed by cuff BP measurements.

Elevated heart rate is common after heart transplantation as a result of cardiac denervation and loss of vagal influence. Although elevated heart rate has been shown to be a significant risk factor for cardiovascular death in individuals with hypertension, its significance as a cardiovascular risk factor in HTR is unclear. In the present study, heart rate was abnormally high in both group B and group C patients. As heart rate decreased from group C to group A patients, LVET increased and was directly related to the observed increase in pulse pressure, which could be associated with an increase in stroke volume or aortic stiffness. Studies in which heart rate was increased by atrial pacing found an inverse relation between heart rate, LVET, pulse pressure, and stroke volume. In these studies, aortic stiffness did not change. However, in our study in HTR, it is more likely that the increase in pulse pressure is related to aortic stiffness. Although increased systolic and pulse pressure is known to occur with aging, there were no differences in age among our three groups of patients that could explain the differences among group A, B, and C patients. Likewise, BMI and patient height, factors that are known to affect wave reflection into the central aorta, were equivalent among the groups.

The late aortic systolic BP augmentation (P Ao – P a) noted in the group A and B patients is due to increased pulse wave velocity and early return of reflected pressure waves into the central aorta from the lower body, and indicates an increase in left ventricular afterload and a mismatch in ventricular/vascular coupling. This is further borne out by the reduced ΔPp noted in the group A and B patients, given that pulse wave velocity is known to be inversely related to ΔPp. As noted in the present study, critical alterations in central arterial pressure waves are most often not detectable with standard brachial artery pressure measurement, and force the clinician to make assumptions about the adequacy of vasodilator or antihypertensive therapy that may not be correct. Increased central arterial pulse pressure is known to be a powerful predictor of cardiovascular events in nontransplantation patients, and is likely to have similar (if not more pronounced) effects in HTR, who have, in general, a worse cardiac risk profile than the average population of cardiac patients and are often markedly hypertensive. In a similarly high-risk group of 180 patients with end-stage renal failure, London et al found that an increase in the central (carotid) Ala was independently associated with both all-cause and cardiovascular mortality. Although the present study was not designed to assess cardiac event rates in our patients, it would seem intuitive that lowering elevated aortic pulse pressure and reducing left ventricular afterload would have favorable short- and long-term effects on the cardiovascular system in these patients. Our speculation would be that improving aortic pulse pressure and augmentation index would be beneficial in HTR, and could conceivably translate into a reduction in cardiac events over time.

Radial arteryplanation tonometry as applied in the present study is a noninvasive, reproducible, and portable measuring tool for estimating central aortic pressure and wave reflection. This technique can be a valuable adjunct to traditional BP measurement in that it describes the state of central arterial function and compliance (and, therefore, left ventricular afterload) more completely. It can be applied without risk to the same individual many times, providing a continuum of data over time as antihypertensive therapy is altered, giving a measure of the relative efficacy of therapy independent of the findings of cuff BP. More widespread use of this technique as a supplement to conventional BP measurement might allow more optimal risk factor reduction in hypertensive patients who are at high risk for cardiovascular events.

References


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