Impact of Mild Hypertension on Left Atrial Size and Function

Suzanne Eshoo, MD; David L. Ross, MD; Liza Thomas, MD, PhD

Background—Left atrial (LA) enlargement has been documented to occur in moderate and severe hypertension.

Methods and Results—One hundred twelve mild hypertension patients were prospectively recruited and compared with 198 healthy volunteers. All recruits had a transthoracic echocardiogram. Maximum LA biplane volume, minimum LA biplane volume, and pre ’p’-LA biplane volume were measured, and left atrial passive, active emptying, and conduit volumes were calculated at baseline and in a subgroup of patients after 12 months. After adjusting for age, gender, and body mass index, maximum LA biplane volume, pre ’p’-LA biplane volume, and their indexed volumes were increased in the hypertension group. Active emptying volume and fraction were significantly increased in the hypertension group, with no change in conduit and passive volumes. Subgroup analysis comparing hypertensives with normal/mildly increased left ventricular mass (group 1) with those with moderate/severely increased left ventricular mass (group 2) at baseline demonstrated that maximum LA biplane volume (62.8±17.9 mL versus 45.4±13.7 mL; P<0.001) was significantly increased in group 2. Active emptying volume was also increased.

Conclusion—Even mild hypertension seems to be associated with a reduction in early diastolic filling. This results in augmented late ventricular diastolic filling due to active atrial contraction and may be the mechanism for the increase in left atrial size. (Circ Cardiovasc Imaging. 2009;2:93-99.)

Key Words: hypertension ■ left atrium ■ phasic volumes ■ diastole ■ echocardiography

Left atrial (LA) enlargement occurs in patients with moderate and severe hypertension (HT).\(^1\)\(^,\)\(^2\) HT results in left ventricular (LV) hypertrophy and reduced LV diastolic function. It has been established that LA volume is a sensitive marker for the severity of diastolic dysfunction.\(^3\)

Clinical Perspective see p 99

Data from the Framingham study suggest that LA enlargement is among the strongest predictors for subsequent development of atrial fibrillation (AF).\(^4\) The risk of developing AF is increased 1½ times in hypertensive patients.\(^5\)\(^,\)\(^6\) The occurrence of paroxysmal AF in hypertensive subjects is associated with LA enlargement and reduction of atrial contractile function.\(^5\)

Few studies have addressed in detail the changes in total LA volume and its phases (passive emptying, conduit volume, and active emptying) in mild HT. We sought to evaluate (1) whether maximal LA volume (Vol\(_{max}\)) increases significantly in mild HT, (2) if active atrial contraction increases to compensate for decreased diastolic function consequent to HT, and (3) the effect of increased LV mass (LVM) on atrial size in mild HT.

Methods

Study approval was obtained from the Human Research Ethics Committee at Westmead Hospital. The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

The study population consisted of 112 patients in sinus rhythm with mild HT (51 males) and 198 normal controls (89 males). The patients were recruited from the Cardiology and Renal departments and also from the community based on a history of mild HT (documented blood pressure [BP] of >140/90 mm Hg but <160/100 mm Hg) by the treating physician before and during antihypertensive therapy. A detailed history was obtained on duration and documented BP. These details were corroborated from patient hospital records with their treating physician. Patients who had previously documented BP levels higher than 160/100 mm Hg, diabetes mellitus or coronary artery disease, significant valvular disease (more than mild valvular regurgitation or stenosis), a history of atrial or ventricular arrhythmias, an ejection fraction of <50%, previous cardiac surgery, or implanted devices were excluded. Of the total of 154 patients recruited, 42 patients were excluded based on the above exclusion criteria. The final HT population analyzed consisted of 112 patients. BP was checked lying, standing, and seated (2 recordings made 10 minutes after arrival) at the time of the echocardiographic examination.

The normal population consisted of volunteers recruited from hospital staff and the community. There was no history of HT (BP <135/85 mm Hg), ischemic heart disease, significant valvular disease, peripheral vascular disease, cerebrovascular disease, or diabetes. None of the normal population was on antihypertensive or other cardiac medications. BP for the normal population was recorded twice in the seated position 10 minutes after arrival.

Received May 21, 2008; accepted January 2, 2009.

From the Westmead Hospital (S.E., D.L.R.), University of Sydney, Sydney, Australia; and Liverpool Hospital (L.T.), University of New South Wales, New South Wales, Australia.

The online-only Data Supplement is available at http://circimaging.ahajournals.org/cgi/content/full/10.1161/CIRCIMAGING.108.793190/DC1.

Correspondence to Suzanne Eshoo, MD, Department of Cardiology, Westmead Hospital, Darcy Road, Westmead, 2145, New South Wales, Australia.

E-mail suzannee@westgate.wh.usyd.edu.au

© 2009 American Heart Association, Inc.

Circ Cardiovasc Imaging is available at http://circimaging.ahajournals.org

DOI: 10.1161/CIRCIMAGING.108.793190

93
Standard Transthoracic Echocardiogram

Echocardiograms were performed according to established clinical practice using 3 commercially available instruments (General Electric Vivid 5 and 7, Horten, Norway, and Philips IE 33, Eindhoven, The Netherlands), using harmonic 3.5-MHz variable frequency phased-array transducers. LA diameter by M-mode was measured in the parasternal long-axis view. LV wall thickness was measured by M-mode using American Society of Echocardiography (ASE) criteria. Relative wall thickness was measured as 2×posterior wall thickness in diastole divided by LV diastolic dimension. LV end-diastolic and end-systolic diameters were determined from the apical 4- and 2-chamber views using the biplane method of discs.

LA Volumes and Mechanical Function

LA volumes were measured at 3 points: (1) Volmax, in ventricular systole just before mitral valve opening; (2) volume before active atrial contraction volume (Volp) at P-wave onset on ECG; and (3) minimal LA volume (Volmin) after mitral valve closure. All volumes were calculated from the apical 4- and 2-chamber zoomed views using the biplane method of discs. The following LA emptying parameters were derived.

- Passive emptying volume = Volmax - Volp
- Passive emptying fraction = LA passive emptying volume/Volmax
- Active emptying volume = Volp - Volmin
- Active emptying fraction = LA active emptying volume/Volp
- Conduit volume = LV stroke volume = -(Volmax - Volmin)
- LA total emptying fraction = LA total emptying volume/Volmax

Atrial volumes were indexed to allometric height, as the average body mass index (BMI) was too high to allow normalization to body surface area.

Mitrail Inflow and LV Diastolic Function

Pulsed Doppler mitral inflow was obtained at a sweep speed of 100 mm/s from the apical 4-chamber view, placing the sample volume at the mitral leaflet tips. Peak A-wave velocity, its velocity time integral and atrial fraction (A-wave velocity time integral/total velocity time integral) were measured as an average of 3 beats. LA ejection force was calculated as previously described. LV diastolic function was determined using standard echocardiographic parameters including peak E velocity, peak A velocity, E/A ratio, and the deceleration time. The early diastolic E' velocity and late diastolic A' velocity were estimated by Doppler tissue imaging, by placing the sample volume at the septal annulus, recording at a sweep speed of 100 mm/s and measured as an average of 3 beats. Diastolic filling patterns were categorized as normal, impaired relaxation, pseudonormal, and restrictive based on previously published criteria using a combination of transmitral and Doppler tissue imaging parameters.

Left Ventricular Mass

The ASE-recommended area-length method was used to determine LVM. LVM was indexed to allometric height. Patients in the HT group were divided into 2 groups based on LVM; group 1 consisted of normal/mildly increased LVM and group 2 consisted of moderately/severely increased LVM using the reference values from the Strong Heart Study. Normal LVM in women was classified as <44 g/m² and in men was <49 g/m², mildly increased LVM in women was 45 to 50 g/m² and in men 50 to 56 g/m², moderately increased LVM in women was 51 to 56 g/m² and in men 57 to 63 g/m², and severely increased LVM in women was ≥57 g/m² and in men ≥64 g/m².

Follow-Up Echocardiogram

Patients who had increased LVM on their baseline study were invited to return at 12 months for a repeat echo. Physician advice on lifestyle modification was given with regard to optimal BP control, and though no medications were initiated by the study doctors, patients were encouraged to see their treating physicians to optimize the BP control and aim for target BP <140/90 mm Hg. Forty-four of 50 patients with increased LVM returned for a repeat echocardiogram 12 months later. Of the 6 patients who did not return for follow-up, 1 patient died from noncardiac causes, 2 patients had pacemakers implanted, 2 patients developed chronic AF, and 1 patient refused repeat echocardiogram.

Observer Agreement

In 10 randomly selected studies from each group, 2 readers independently measured the LA Volmax, Volmin, and Volp. One observer remeasured the same 20 studies at a separate time to determine intraobserver agreement from the baseline studies. The same individual acquired further LA images 1 hour later to assess interstudy reproducibility.

Analysis

All values are expressed as mean ± SD. Linear regression was used to examine the difference between the normal and HT groups. All values were adjusted for age, gender, and BMI; raw values were indexed allometric height. Differences within patients at baseline and follow-up at 12 months were analyzed using a paired Student t test. The correlation between 2 variables was assessed by Pearson rank correlation coefficient. Univariate and multivariate regression analysis was used to examine independent predictors of increased LA size. Bland Altman analysis was performed to analyze intraobserver variability, interobserver variability, and interstudy reproducibility. Data were analyzed using SPSS version 15 (SPSS Inc, Chicago, Ill).

Results

Baseline Study

Clinical characteristics of the HT group at baseline are given in Table 1. Eighty percent of the population was on ≥2 antihypertensive medications and most were asymptomatic. The mean values for clinical and echocardiographic variables for the HT and normal groups are listed in Table 2. Standing and lying systolic BPs were significantly lower than seated

<table>
<thead>
<tr>
<th>Clinical Characteristics of the Mild HT Population (n=112)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medication</td>
</tr>
<tr>
<td>β-blockers, %</td>
</tr>
<tr>
<td>ACE inhibitors, %</td>
</tr>
<tr>
<td>All inhibitors, %</td>
</tr>
<tr>
<td>Calcium antagonists, %</td>
</tr>
<tr>
<td>Diuretics, %</td>
</tr>
<tr>
<td>Nitrites, %</td>
</tr>
<tr>
<td>Other, %</td>
</tr>
</tbody>
</table>

HT duration, months 101 ± 116

Increased LVM on echocardiogram, % 47

Asymptomatic, % 92

Shortness of breath on exertion, % 5

Pallitations, % 3

Chest tightness, % 0

History of CAD, % 0

Diabetes mellitus, % 0

Cholesterol >5 mmol/L or medicated, % 74

Current smoker, % 12

Normal serum creatinine <110 mmol/L, % 95

ACE indicates angiotensin-converting enzyme; All, angiotensin two receptor inhibitors; LVM, left ventricular mass; CAD, coronary artery disease.
systolic BP ($P<0.02$ and 0.001, respectively). Lying DBP was significantly lower than seated diastolic BP ($P<0.001$).

**LA Volumes**

LA Vol$_{max}$, Vol$_{p}$, and indexed values were significantly increased in the HT group (Table 3). The active emptying volume and fraction were increased in the HT group. The conduit volume and the passive emptying volume and fraction were not significantly different between groups (Table 3). Defining LA enlargement as the mean+2SD as calculated from the LA Vol$_{max}$ indexed to height$^2.7$ for the normal group in this study, LA enlargement was found in $22\%$ of the hypertensive population.

**LA Volumes and Degree of LV Hypertrophy**

Hypertensive subjects were further divided into 2 groups on the basis of LVM previously described. LA Vol$_{max}$, Vol$_{min}$, Vol$_{p}$, and indexed values were all significantly increased in group 2 compared with group 1. The active emptying volume remained significantly higher in group 2, but the active emptying fraction failed to reach significance (Table 4).

**LV Diastolic Function**

In the HT group, none had restrictive diastolic physiology, $9\%$ had pseudonormalized filling pattern, and $16\%$ had impaired relaxation. The remainder ($75\%$) had normal diastolic physiology. The peak E and A velocities were significantly higher in the HT group and the deceleration time was prolonged (Table 2). Septal E’ was significantly lower in the HT group, whereas E/E’ showed a significant increase in the HT group. LA ejection force was significantly higher in the HT group. In the subgroup analysis of patients with HT based on LVM, peak E, peak A, deceleration time, E’, E/E’, and A’ were not significantly different in the 2 groups (data not shown).

**Factors That Influence LA Enlargement**

Patients with SBP values $\geq 150$ mm Hg at enrollment had higher LVM when compared with those patients with a BP
<150 mm Hg (P=0.024 for indexed LVM). There was no statistically significant difference in height, weight, BMI, or body surface area between the 2 subgroups.

To determine the independent correlates of LA Volmax, univariate and multivariate regression analyses were performed. Input variables included the duration of HT, antihypertensive drugs, seated systolic and diastolic BP, BMI, LVM, and parameters of diastolic function (peak E and E' velocity). By univariate analysis, a positive correlation was noted between LA Volmax and seated systolic BP (r=0.34), BMI (r=0.37), and LVM (r=0.57) (P=0.001 in all). Neither the duration of HT nor the use of antihypertensive medications influenced LA geometry, phasic volumes or LA function.

When the univariate predictors were entered into a multivariate analysis, the only independent correlates of increased LA Volmax included LVM (P=0.001) and peak E (P=0.02). Using a best-fit model, LA Volmax = 6.8 + 0.1LVM + 25.6 peak E (adjusted r²=0.30).

We additionally performed correlations between atrial volumes and indexed LVM and BMI. A significant correlation was present between indexed LVM and Volmax (r=0.47), Volmin (r=0.34), and Volp (r=0.44) (P=0.001 in all). Similarly, a

Data adjusted for age, gender, and BMI.

### Table 4. LA Volumes in Normal/Mildly Increased LV Mass (Group 1) Versus Moderate/Severely Increased LV Mass (Group 2)

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (n=78)</th>
<th>Group 2 (n=33)</th>
<th>95% CI of the Difference</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic BP, mm Hg</td>
<td>141±17</td>
<td>152±19</td>
<td>-18.3 to -3.8</td>
<td>0.003</td>
</tr>
<tr>
<td>Diastolic BP, mm Hg</td>
<td>83±10</td>
<td>90±11</td>
<td>-10.7 to -2.2</td>
<td>0.003</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>28.1±4.3</td>
<td>32.2±4.5</td>
<td>-5.8 to -2.3</td>
<td>0.001</td>
</tr>
<tr>
<td>Heart rate, beats/minute</td>
<td>73±13</td>
<td>70±13</td>
<td>-2.3 to 8.3</td>
<td>NS</td>
</tr>
<tr>
<td>LA Volmax, mL</td>
<td>45.4±13.7</td>
<td>62.8±17.9</td>
<td>-23.8 to -10.9</td>
<td>0.001</td>
</tr>
<tr>
<td>LA Volmax indexed to height, mL/m²</td>
<td>11.4±2.9</td>
<td>16.6±4.0</td>
<td>-6.6 to -3.8</td>
<td>0.001</td>
</tr>
<tr>
<td>LA Volmin, mL</td>
<td>18.3±7.1</td>
<td>27.5±11.5</td>
<td>-12.9 to -5.6</td>
<td>0.001</td>
</tr>
<tr>
<td>LA Volmin indexed to height, mL/m²</td>
<td>4.6±1.6</td>
<td>7.3±2.8</td>
<td>-3.8 to -1.6</td>
<td>0.001</td>
</tr>
<tr>
<td>LA Volp, mL</td>
<td>30.6±10.2</td>
<td>47.0±16.4</td>
<td>-2.8 to -9.8</td>
<td>0.001</td>
</tr>
<tr>
<td>LA Volp, indexed to height, mL/m²</td>
<td>7.8±2.4</td>
<td>12.4±3.8</td>
<td>-6.1 to -3.1</td>
<td>0.001</td>
</tr>
<tr>
<td>LA passive emptying volume, mL</td>
<td>14.6±7.3</td>
<td>15.8±8.4</td>
<td>-4.5 to 2.0</td>
<td>NS</td>
</tr>
<tr>
<td>Indexed passive emptying volume, mL/m²</td>
<td>3.6±1.5</td>
<td>4.2±2.1</td>
<td>-1.4 to 0.3</td>
<td>NS</td>
</tr>
<tr>
<td>LA conduit volume, mL</td>
<td>26.6±13.7</td>
<td>31.1±16.5</td>
<td>-10.8 to 1.8</td>
<td>NS</td>
</tr>
<tr>
<td>Indexed conduit volume, mL/m²</td>
<td>6.7±3.3</td>
<td>8.1±4.2</td>
<td>-2.9 to 0.2</td>
<td>NS</td>
</tr>
<tr>
<td>LA active emptying volume, mL</td>
<td>26.6±13.7</td>
<td>31.1±16.5</td>
<td>-10.8 to 1.8</td>
<td>NS</td>
</tr>
<tr>
<td>Indexed active emptying volume, mL/m²</td>
<td>12.6±5.3</td>
<td>19.4±8.1</td>
<td>-10.1 to -3.6</td>
<td>0.001</td>
</tr>
<tr>
<td>LA total emptying fraction, %</td>
<td>58.9±9.6</td>
<td>56.8±9.7</td>
<td>-2.1 to 6.2</td>
<td>NS</td>
</tr>
<tr>
<td>LA passive emptying fraction, %</td>
<td>31.8±11.1</td>
<td>25.5±11.6</td>
<td>1.4 to 11.0</td>
<td>0.007</td>
</tr>
<tr>
<td>LA active emptying fraction, %</td>
<td>40.7±11.5</td>
<td>41.6±11.1</td>
<td>-5.9 to 3.9</td>
<td>NS</td>
</tr>
</tbody>
</table>

Data adjusted for BMI.
modest correlation was noted between BMI and Volmax (r=0.41), Volmin (r=0.30), and Volp (r=0.37) (P=0.001 in all).

Twelve-Month Follow-Up Study
Repeat echocardiograms were performed in 44 hypertensive patients after a mean follow-up of 12±3.7 months. The change in antihypertensive therapy is shown in Figure 1. Mean systolic BP, diastolic BP, and MAP were significantly reduced after 12 months. Raw and indexed LVM were significantly reduced at follow-up (Table 5). No difference was present in conduit, passive, or active indexed emptying volumes, although indexed active emptying volume approached significance (P=0.07; Figure 2A and 2B).

Table 5. Clinical and Echocardiographic Characteristics in the 44 Hypertensive Patients Who Had Echocardiograms at Baseline and Follow-Up

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Baseline (n=44)</th>
<th>12-Month Follow-Up (n=44)</th>
<th>95% CI of the Difference</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height, m</td>
<td>167±10</td>
<td>166±10</td>
<td>0.4 to 1.9</td>
<td>0.004</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>83.4±16.8</td>
<td>82.5±16.9</td>
<td>−0.6 to 2.3</td>
<td>NS</td>
</tr>
<tr>
<td>Age, years</td>
<td>59±13</td>
<td>60±13</td>
<td>−1.2 to −0.8</td>
<td>0.001</td>
</tr>
<tr>
<td>Gender, male/female</td>
<td>24/20</td>
<td>24/20</td>
<td>...</td>
<td>NS</td>
</tr>
<tr>
<td>BMI, (kg/m²)</td>
<td>29.8±4.5</td>
<td>29.9±4.4</td>
<td>−0.7 to 0.6</td>
<td>NS</td>
</tr>
<tr>
<td>Systolic BP seated, mm Hg</td>
<td>152±16</td>
<td>145±20</td>
<td>0.9 to 12.6</td>
<td>0.024</td>
</tr>
<tr>
<td>Diastolic BP seated, mm Hg</td>
<td>86±11</td>
<td>81±11</td>
<td>1.0 to 8.7</td>
<td>0.015</td>
</tr>
<tr>
<td>Mean arterial pressure seated, mm Hg</td>
<td>108±10</td>
<td>102±11</td>
<td>1.5 to 9.6</td>
<td>0.009</td>
</tr>
<tr>
<td>Heart rate, beats/minute</td>
<td>69±13</td>
<td>66±12</td>
<td>0.4 to 8.8</td>
<td>0.034</td>
</tr>
<tr>
<td>Ventricular septal thickness, mm</td>
<td>12.3±1.5</td>
<td>11.5±1.6</td>
<td>0.3 to 1.3</td>
<td>0.001</td>
</tr>
<tr>
<td>Posterior wall thickness, mm</td>
<td>12.1±1.4</td>
<td>11.5±1.5</td>
<td>0.2 to 1.1</td>
<td>0.001</td>
</tr>
<tr>
<td>Left ventricular mass, g</td>
<td>219±48</td>
<td>199±52</td>
<td>12 to 29</td>
<td>0.001</td>
</tr>
<tr>
<td>Left ventricular mass index to height, g/m²</td>
<td>48.5±10.0</td>
<td>44.2±11.1</td>
<td>2.3 to 6.3</td>
<td>0.001</td>
</tr>
<tr>
<td>Relative wall thickness, mm</td>
<td>0.49±0.1</td>
<td>0.47±0.1</td>
<td>−0.01 to 0.05</td>
<td>NS</td>
</tr>
<tr>
<td>Peak E, m/s</td>
<td>0.73±0.14</td>
<td>0.71±0.17</td>
<td>−0.02 to 0.07</td>
<td>NS</td>
</tr>
<tr>
<td>Peak A, m/s</td>
<td>0.78±0.22</td>
<td>0.74±0.20</td>
<td>−0.002 to 0.09</td>
<td>0.061</td>
</tr>
<tr>
<td>E/A</td>
<td>1.0±0.3</td>
<td>1.0±0.3</td>
<td>−0.07 to 0.06</td>
<td>NS</td>
</tr>
<tr>
<td>Deceleration time, ms</td>
<td>232±55</td>
<td>249±56</td>
<td>−34.3 to 2.0</td>
<td>NS</td>
</tr>
<tr>
<td>Septal E’ m/s</td>
<td>6.5±2.0</td>
<td>6.5±2.1</td>
<td>−0.6 to 0.6</td>
<td>NS</td>
</tr>
<tr>
<td>LA Volmax mL</td>
<td>61.9±18.9</td>
<td>55.8±17.6</td>
<td>1.8 to 10.3</td>
<td>0.006</td>
</tr>
</tbody>
</table>

Table 5. Clinical and Echocardiographic Characteristics in the 44 Hypertensive Patients Who Had Echocardiograms at Baseline and Follow-Up

Interobserver/Intraobserver Variability and Interstudy Reproducibility
LA Volmax, Volmin, and Volp were evaluated in 20 studies. Bland-Altman analysis for LA Volmax demonstrated an intrasubject mean difference of −0.004 mL (95% CI, −1.3 to 1.3 mL). The interobserver variability showed a mean difference of 0.73 mL (95% CI, −1.7 to 3.1 mL). Interstudy reproducibility was performed at 1 hour under the same clinical conditions. The mean difference between sequential studies was 1.9±4.2 mL (r=0.95) for Volmax, −1.1±1.8 mL (r=0.98) for Volmin, and 0.87±2.2 mL (r=0.97) for Volp.

Discussion
The results of our study demonstrate that LA volume is increased even in mild HT, with an associated increase in active volume. Conversely, there was no change in LA passive emptying or conduit volume. We have demonstrated that peak E velocity and LVM were independent predictors of LA volume on multivariate analysis. After adjusting for age, gender, and BMI, LA size and active emptying volume were increased in those with a higher LVM within the HT group. Thus, as LV diastolic function progressively worsens, LA enlargement occurs to augment active LA emptying.

Normal Aging and Diastolic Properties
Age-related changes in LV diastolic properties are well recognized with a decrease in early diastolic filling and
compensatory increase in late filling. In addition, the Doppler tissue imaging derived $E'$ velocity decreases with an increase in $A'$ velocity. We previously demonstrated that normal aging does not result in a significant increase in LA size. However, passive emptying and conduit volumes were decreased consequent to age-related LV diastolic dysfunction, with a compensatory increase in active atrial emptying. In contrast, even mild HT results in an increase in overall LA size due to further reduction in LV diastolic function due to HT over and above normal age-related changes. The LA ejection force was increased in the HT group and represents the compensatory increase in atrial contribution by recruitment of starling forces due to the reduction in early diastolic relaxation.

**Effect of HT on LA Size**

Although there has been recent interest in LA size in HT, many of the reported studies have evaluated only maximal LA volume. Population-based studies have found that LA enlargement occurs in 22% of patients with HT when using LA diameter indexed to body surface area and 27% when LA volume is indexed to body surface area. Similarly, 22% of our patient population was found to have LA enlargement when LA volume was indexed to height.

Atrial phasic changes in untreated severe hypertensives demonstrated a decrease in conduit and passive volumes with an increase in active emptying fraction. In this study of mild hypertensives, no change was noted in passive emptying or conduit volumes with an increase in active emptying volume and an overall increase in LA volume compared with normals. Thus, we could postulate that worsening HT would compromise conduit and passive emptying volumes as previously demonstrated.

The subgroup with systolic BP >150 mm Hg had a larger LA, and although the peak systolic and mean arterial pressure correlated with LA volume, the duration of HT did not influence LA size. Further compromise of LV diastolic function due to increased LVM (group 2) in the population with HT results in further increases in total LA volume as well as active atrial emptying similar to the report by Cioffi et al. Intensive treatment of more severe grades of HT has previously shown a regression of LV hypertrophy and reduction in LA volume. In the subgroup with an increased LVM followed up for 12 months, improved BP control resulted in reduction in LVM and in LA size. Thus, careful monitoring of LA size and its changes may have a role as a surrogate marker to monitor the efficacy of HT therapy.

**Impact of Increased LA Size in HT**

Increased LA size is associated with the increased occurrence of AF and hypertensives carry an increased risk of AF. Thus, although treatment strategies in AF are directed toward rhythm control, careful evaluation of associated uncontrolled HT and its effective management is important in the management of patients with AF. This needs to be studied more carefully in a population with AF with associated HT. The finding of an enlarged LA in a subject who is apparently “normotensive” may warrant more careful monitoring of BP including ambulatory BP monitoring to rule out hitherto undiagnosed HT.

**Limitations**

Most of the hypertensive patients were on antihypertensive treatment before recruitment into this study. Because of the increased awareness of benefits of early treatment, we found it impossible to recruit large numbers of untreated patients with mild HT. A detailed history and review of medical records of previously recorded BP was taken to ensure that documented BP was <160/100 mm Hg.

Patients did not have ambulatory or nocturnal BP checked. Although these would likely have provided additional information, these were considered beyond the scope of the present study. Medications were not stopped before the echocardiographic examination and this may have altered diastolic filling parameters.

**Conclusion**

This study has systematically evaluated LA volumes and function in a large group of patients with mild HT. Even mild HT seems to be associated with a reduction in early diastolic filling. This results in augmented late LV diastolic filling due to active atrial contraction and may be the mechanism for the increase in LA size. With increasing LVM within the HT population, LA size enlarges with an increase in active atrial
emptying. Patients with mild HT should be evaluated for LA enlargement as this may be a useful surrogate marker for monitoring the effectiveness of medical therapy.

Sources of Funding
Dr Eshoo is a postgraduate medical scholar supported by a University of Sydney postgraduate award.

Disclosures
None.

References
8. Lang RM, Bierig M, Devereux RB, Flachskampf FA, Foster E, Pellikka PA, Picard MH, Roman MJ, Seward J, Shanewise JS, Solomon SD, Spencer KT, Sutton MS, Stewart WJ. Recommendations for chamber quantification: a report from the American Society of Echocardiography’s Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr. 2005;18:1440–1463.

CLINICAL PERSPECTIVE
Several reports have demonstrated that left atrial size can be used as a marker for the severity of diastolic dysfunction and as a prognostic marker of future cardiovascular events. In addition, an enlarged left atrium predisposes to the development of atrial fibrillation with its associated morbidity and mortality. This prospective study demonstrates that the diastolic dysfunction consequent to mild hypertension (blood pressure <160/100 mm Hg) is associated with preserved atrial passive emptying and conduit volumes, with an increase in active atrial emptying volume. These data suggest that the increased “left ventricular stiffness” consequent to mild hypertension may play a role in the recruitment of starving forces and thus may be the mechanism responsible for left atrial dilatation. Our data additionally indicate that echocardiographically determined left atrial size may be a useful method to monitor the efficacy of antihypertensive therapy and the adequacy of blood pressure control.
Impact of Mild Hypertension on Left Atrial Size and Function
Suzanne Eshoo, David L. Ross and Liza Thomas

Circ Cardiovasc Imaging. 2009;2:93-99; originally published online January 26, 2009;
doi: 10.1161/CIRCIMAGING.108.793190
Circulation: Cardiovascular Imaging is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2009 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-9651. Online ISSN: 1942-0080

The online version of this article, along with updated information and services, is located on the
World Wide Web at:
http://circimaging.ahajournals.org/content/2/2/93

Data Supplement (unedited) at:
http://circimaging.ahajournals.org/content/suppl/2009/03/19/CIRCIMAGING.108.793190.DC1

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation: Cardiovascular Imaging can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation: Cardiovascular Imaging is online at:
http://circimaging.ahajournals.org//subscriptions/
Figure Legend

Supplementary Figure 1: LA volumes in Normal/Mildly increased LV mass (Group 1) vs. Moderate/Severely increased LV mass (Group 2)
Group 1 n = 78

Group 2 n = 33

* = p < 0.005