Hypertension in Aortic Stenosis
Implications for Left Ventricular Structure and Cardiovascular Events

Åshild E. Rieck, Dana Cramariuc, Kurt Boman, Christa Gohlke-Bärwolf, Eva M. Staal, Mai Tone Lønnebakken, Anne B. Rossebø, Eva Gerds

Abstract—The impact of hypertension on left ventricular structure and outcome during progression of aortic valve stenosis has not been reported from a large prospective study. Data from 1616 patients with asymptomatic aortic stenosis randomized to placebo-controlled treatment with combined simvastatin and ezetimibe in the Simvastatin Ezetimibe in Aortic Stenosis Study were used. The primary study end point included combined cardiovascular death, aortic valve events, and ischemic cardiovascular events. Hypertension was defined as history of hypertension or elevated baseline blood pressure. Left ventricular hypertrophy was defined as left ventricular mass/height$^2.7$ ≥46.7 g/m$^2.7$ in women and ≥49.2 g/m$^2.7$ in men and concentric geometry as relative wall thickness ≥0.43. Baseline peak aortic jet velocity and aortic stenosis progression rate did not differ between hypertensive (n = 1340) and normotensive (n = 276) patients. During 4.3 years of follow-up, the prevalence of concentric left ventricular hypertrophy increased 3 times in both groups. Hypertension predicted 51% higher incidence of abnormal LV geometry at final study visit independent of other confounders (P < 0.01). In time-varying Cox regression, hypertension did not predict increased rate of the primary study end point. However, hypertension was associated with a 56% higher rate of ischemic cardiovascular events and a 2-fold increased mortality (both P < 0.01), independent of aortic stenosis severity, abnormal left ventricular geometry, in-treatment systolic blood pressure, and randomized study treatment. No impact on aortic valve replacement was found. In conclusion, among patients with initial asymptomatic mild-to-moderate aortic stenosis, hypertension was associated with more abnormal left ventricular structure and increased cardiovascular morbidity and mortality. (Hypertension. 2012;60:90-97.)

Key Words: hypertension ■ aortic valve stenosis ■ echocardiography ■ prognosis ■ antihypertensive agents

Among older patients with aortic stenosis (AS), hypertension (HT) is a common comorbidity found in up to 78% of patients.1–4 HT is a known predictor of increased cardiovascular (CV) morbidity and mortality both in general and HT populations,5,6 but it is unknown whether concomitant HT is associated with increased CV event rate in AS.

In patients with asymptomatic mild-to-moderate AS, concomitant HT has been associated with higher prevalence of left ventricular (LV) hypertrophy, particularly of eccentric type.1 In contrast, a retrospective study in patients with severe symptomatic AS reported comparable prevalence of LV hypertrophy in HT and normotensive patients with a preponderance of concentric LV geometry irrespective of the presence of HT.8 However, the impact of concomitant HT on changes in LV structure during progression of AS has not been reported previously from a large, prospective study. The aim of the present study was to assess these questions within the Simvastatin Ezetimibe in Aortic Stenosis (SEAS) Study, including initially asymptomatic patients with mild-moderate AS without diabetes mellitus or prevalent CV disease.
diastole and considered increased if
ography and American Society of Echocardiography guidelines.12,13
software. Severity of AS, LV structure, and systolic function were
(TomTec Imaging Systems GmbH, Unterschleissheim, Germany)
was performed using offline digital workstations with Image Arena
mL/min per 1.73 m²
Estimated glomerular filtration rate,
Triglycerides, mmol/L 1.4
HDL cholesterol, mmol/L 1.5
Total cholesterol, mmol/L 5.7
Diuretics, % 0 28
Angiotensin receptor blockers, % 0 12
Angiotensin-converting enzyme inhibitors, % 0 19
Calcium antagonists, % 0 18
β-blockers, % 0 33
α-blockers, % 0 2
Diuretics, % 0 28
Total cholesterol, mmol/L 5.7±1.0 5.7±1.0
LDL cholesterol, mmol/L 3.6±0.9 3.6±0.9
HDL cholesterol, mmol/L 1.5±0.4 1.5±0.4
Triglycerides, mmol/L 1.4±0.7 1.4±0.7
Estimated glomerular filtration rate,
 mL/min per 1.73 m²
‡p<0.001 vs normotensive patients.
*p<0.05 vs normotensive patients.
 did not differ in prevalence of HT, age, blood pressure, or sex
distribution.
Echocardiography
Study echocardiograms were obtained at 173 study centers in
Denmark, Finland, Germany, Ireland, Norway, Sweden, and the
United Kingdom following a standardized protocol and sent for
blinded expert interpretation at the SEAS echocardiography core
laboratory at Haukeland University Hospital. The echocardiographic
methods have been published previously.11 All of the reading
was performed using offline digital workstations with Image Arena
(TomTec Imaging Systems GmbH, Unterschleissheim, Germany)
software. Severity of AS, LV structure, and systolic function were
measured following the joined European Association of Echocardi-
ography and American Society of Echocardiography guidelines.12,13
Teichholz-derived LV volumes were used for the calculation of stroke
volume and cardiac output.14 Circumferential end-systolic stress was
estimated at midwall, and actual and predicted midwall shortening were
calculated using standard equations.12,15 LV hypertrophy was consid-
ered present when LV mass/height2.7 exceeded 46.7 g/m² in females
and 49.2 g/m² in men, respectively.5,16 Relative wall thickness was
calculated as LV posterior wall thickness/LV internal radius at end
diastole and considered increased if ≥0.43.17 LV geometry was as-

described by Briand et al20 using Teichholz-derived stroke volume indexed to height in the allometric
power of 2.04,21 Stroke work loss was calculated as Doppler-derived
mean aortic valve gradient over the sum of systolic blood pressure and
mean transaortic gradient.22,23 Systemic arterial compliance was
calculated as stroke volume indexed to body surface area/pulse
pressure ratio and total peripheral resistance as (80×mean blood
pressure/cardiac output, using brachial blood pressure. Blood pres-
sure measured at the end of the echocardiography with the patients
still in the supine position was used for the calculation of hemody-
namic variables.

Study End Points
All of the end points were adjudicated by an independent committee
blinded to study treatment. The prespecified primary end point of the
study was major CV events, a composite end point including death
from CV causes, aortic valve–related events, and ischemic CV
events.7 The secondary end points were aortic valve events (com-
bined congestive heart failure attributed to progression of AS, aortic
valve replacement, and death from CV causes) and ischemic CV
events (combined death from CV causes, nonfatal myocardial
infarction, hospitalization for unstable angina, coronary revascular-
ization, and nonhemorrhagic stroke) analyzed separately. Total
mortality was a prespecified tertiary end point.

Statistics
SPSS 18.0 (SPSS Inc, Chicago, IL) software was used for data
management and analysis. Data are expressed as mean±SD for
continuous variables and as percentages for categorical variables.
Comparisons between groups were done using the Student t test for
baseline characteristics, ANOVA for repeated measures, and χ² tests
as appropriate.

The last study echocardiogram was defined as the last performed
before a CV event in patients who experienced end points during
follow-up and as the final study echocardiogram in patients without

Table 1. Baseline Characteristics in Normotensive and Hypertensive Groups of Patients

<table>
<thead>
<tr>
<th>Variables</th>
<th>Normotensive Patients (n=276)</th>
<th>Hypertensive Patients (n=1340)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>62±11</td>
<td>68±9*</td>
</tr>
<tr>
<td>Body mass index, g/m²</td>
<td>25.5±3</td>
<td>27.1±4*</td>
</tr>
<tr>
<td>Body surface area, m²</td>
<td>1.90±0.2</td>
<td>1.89±0.2</td>
</tr>
<tr>
<td>Women, %</td>
<td>27</td>
<td>42*</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>125±9</td>
<td>152±20</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>76±7</td>
<td>84±10</td>
</tr>
<tr>
<td>Mean arterial pressure, mm Hg</td>
<td>92±6</td>
<td>107±11</td>
</tr>
<tr>
<td>Pulse pressure, mm Hg</td>
<td>50±10</td>
<td>68±18</td>
</tr>
<tr>
<td>History of hypertension, %</td>
<td>0</td>
<td>61</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>64±10</td>
<td>66±12†</td>
</tr>
<tr>
<td>Antihypertensive drug treatment, %</td>
<td>0</td>
<td>68</td>
</tr>
<tr>
<td>No. of antihypertensive drugs, n</td>
<td>0</td>
<td>1.1</td>
</tr>
<tr>
<td>Angiotensin-converting enzyme inhibitors, %</td>
<td>0</td>
<td>19</td>
</tr>
<tr>
<td>Angiotensin receptor blockers, %</td>
<td>0</td>
<td>12</td>
</tr>
<tr>
<td>Calcium antagonists, %</td>
<td>0</td>
<td>18</td>
</tr>
<tr>
<td>β-blockers, %</td>
<td>0</td>
<td>33</td>
</tr>
<tr>
<td>α-blockers, %</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Diuretics, %</td>
<td>0</td>
<td>28</td>
</tr>
<tr>
<td>Total cholesterol, mmol/L</td>
<td>5.7±1.0</td>
<td>5.7±1.0</td>
</tr>
<tr>
<td>LDL cholesterol, mmol/L</td>
<td>3.6±0.9</td>
<td>3.6±0.9</td>
</tr>
<tr>
<td>HDL cholesterol, mmol/L</td>
<td>1.5±0.4</td>
<td>1.5±0.4</td>
</tr>
<tr>
<td>Triglycerides, mmol/L</td>
<td>1.4±0.7</td>
<td>1.4±0.7</td>
</tr>
<tr>
<td>Estimated glomerular filtration rate, mL/min per 1.73 m²</td>
<td>88±16</td>
<td>85±17‡</td>
</tr>
</tbody>
</table>

LDL indicates low-density lipoprotein; HDL, high-density lipoprotein.
*P<0.001 vs normotensive patients.
†P<0.01 vs normotensive patients.
‡P<0.05 vs normotensive patients.

Aortic valve area corrected for pressure recovery in the aortic root
(energy loss index) was calculated using previously published
equations.18 Aortic regurgitation was assessed by color Doppler
using a previously described 4-point grading system.19 Valvulooa-
terial impedance, a measure of the combined valvular and arterial load
in AS, was calculated by the method of Briand et al20 using
Teichholz-derived stroke volume indexed to height in the allometric
power of 2.04.21 Stroke work loss was calculated as Doppler-derived
mean aortic valve gradient over the sum of systolic blood pressure and
mean transaortic gradient.22,23 Systemic arterial compliance was
calculated as stroke volume indexed to body surface area/pulse
pressure ratio and total peripheral resistance as (80×mean blood
pressure/cardiac output, using brachial blood pressure. Blood pres-
sure measured at the end of the echocardiography with the patients
still in the supine position was used for the calculation of hemody-
namic variables.

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Table 2. Echocardiographic Findings at Baseline and Last Study Visit in Normotensive and Hypertensive Patients

<table>
<thead>
<tr>
<th>Variables</th>
<th>Normotensive Patients (n=276)</th>
<th>Hypertensive Patients (n=1340)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Baseline Last Visit</td>
<td>Baseline Last Visit</td>
</tr>
<tr>
<td>Peak aortic jet velocity, m/s</td>
<td>3.0±0.5 3.7±0.8</td>
<td>3.0±0.5 3.7±0.8</td>
</tr>
<tr>
<td>Aortic valve area, cm</td>
<td>1.33±0.5 1.20±0.5</td>
<td>1.29±0.5 1.14±0.4</td>
</tr>
<tr>
<td>Mean aortic valve gradient, mm Hg</td>
<td>22±8 34±16</td>
<td>22±8 33±15</td>
</tr>
<tr>
<td>Energy loss index, cm/m²‡</td>
<td>0.97±0.51 0.85±0.45</td>
<td>0.92±0.43 0.78±0.37</td>
</tr>
<tr>
<td>Valvuloarterial impedance, mm Hg/mL · m⁻²</td>
<td>5.9±1.6 7.5±2.3</td>
<td>6.6±2.1 8.1±2.9</td>
</tr>
<tr>
<td>Aortic annulus diameter, cm‡</td>
<td>2.26±0.3 2.34±0.3</td>
<td>2.18±0.3 2.26±0.3</td>
</tr>
<tr>
<td>Stroke work loss, %‡</td>
<td>14 22</td>
<td>13 20</td>
</tr>
<tr>
<td>Left ventricular diameter at end diastole, cm</td>
<td>5.1±0.7 4.8±0.7</td>
<td>5.0±0.6 4.7±0.7</td>
</tr>
<tr>
<td>Left ventricular diameter at end systole, cm</td>
<td>3.2±0.6 3.1±0.7</td>
<td>3.2±0.5 3.0±0.6</td>
</tr>
<tr>
<td>Interventricular septum thickness at end diastole, cm‡</td>
<td>1.1±0.3 1.3±0.3</td>
<td>1.2±0.3 1.4±0.3</td>
</tr>
<tr>
<td>Left ventricular posterior wall thickness at end diastole, cm‡</td>
<td>0.9±0.2 1.1±0.2</td>
<td>0.9±0.2 1.1±0.2</td>
</tr>
<tr>
<td>Relative wall thickness†</td>
<td>0.35±0.08 0.45±0.09</td>
<td>0.36±0.09 0.47±0.12</td>
</tr>
<tr>
<td>Ejection fraction, %</td>
<td>66±7</td>
<td>64±6</td>
</tr>
<tr>
<td>Circumferential end-systolic stress, kdyn/cm²‡</td>
<td>102±29 85±27</td>
<td>114±32 89±25</td>
</tr>
<tr>
<td>Stress-corrected midwall shortening, %*</td>
<td>99±19 78±15</td>
<td>98±20 77±16</td>
</tr>
<tr>
<td>Left ventricular mass, g*</td>
<td>178±61 215±69</td>
<td>194±67 227±75</td>
</tr>
<tr>
<td>Left ventricular hypertrophy, %*</td>
<td>25 49</td>
<td>38 62</td>
</tr>
<tr>
<td>Systemic arterial compliance, mL/m² per mm Hg‡</td>
<td>0.87±0.36 0.71±0.25</td>
<td>0.67±0.24 0.61±0.26</td>
</tr>
<tr>
<td>Total peripheral resistance, dyne · s · cm⁻⁵‡</td>
<td>1644±472 1891±646</td>
<td>1779±611 2015±761</td>
</tr>
<tr>
<td>Aortic regurgitation, %</td>
<td>58 71</td>
<td>60 68</td>
</tr>
</tbody>
</table>

Data from baseline, y 1, y 2, and final visit are included in analysis. Only data from first and last visit are shown.
*P<0.05 vs normotensive patients using ANOVA for repeated measures.
†P<0.01 vs normotensive patients using ANOVA for repeated measures.
‡P<0.001 vs normotensive patients using ANOVA for repeated measures.

CV events. To assess the impact of HT on new-onset abnormal LV geometry, multivariate logistic regression analysis was used.

Univariate and time-varying multivariate Cox regression analyses were performed to assess the impact of HT on outcome. The primary multivariate model included systolic blood pressure, peak aortic jet velocity, and abnormal LV geometry as time-varying covariates and randomized study treatment as a fixed covariate. In further models, antihypertensive drug treatment and type of antihypertensive drug were included. A 2-tailed P<0.05 was considered statistically significant both in univariate and multivariate analyses.

Results

Patient Characteristics

Compared with normotensive patients, HT patients were older and included more women (Table 1). A total of 68.5% of HT patients used antihypertensive treatment, on average, 1.1 drugs (Table 1). Systolic blood pressure decreased during the first study years in the HT group. In contrast, a small increase in systolic blood pressure was found in the normotensive group during the first year (Figure 1). Similar changes were found for diastolic blood pressure (data not shown). AS severity did not differ between groups when measured as peak aortic jet velocity, mean transvalvular gradient, or unadjusted aortic valve area at baseline, but HT patients had lower energy loss index (Table 2). The prevalence of aortic regurgitation was similar in both groups, and the regurgitation was predominantly of a mild degree (grade 1 in >70% in both groups). Valvuloarterial impedance and stroke work loss were higher in HT patients, and HT patients also had lower systemic arterial compliance and higher peripheral arterial resistance and LV end-systolic stress (all P<0.01; Table 2). AS progression rate did not differ between HT and normotensive groups: annual decrease in aortic valve area was 0.03±0.01 cm² versus 0.04±0.02 cm² and annual increase in peak aortic jet velocity increased with 0.19±0.01 m/s versus 0.22±0.03 m/s, whereas annual increase in mean aortic gradient was 4 mm Hg per year in both groups (all P>0.17).

Change in LV Geometry During Follow-Up

HT patients had larger LV mass/unit² throughout the course of the study (Figure 2 and Table 2). Eccentric LV hypertrophy was the most common type of abnormal LV geometry at baseline. During follow-up, the prevalence of concentric LV hypertrophy increased 3 times and became the most common abnormal LV geometry in both groups (Figure 3). However, abnormal LV geometry remained more prevalent in the HT group throughout the study (Figure 3). Among patients with normal LV geometry at baseline (n=903), HT predicted a 51% higher risk of having abnormal LV geometry on the last study echocardiogram (hazard ratio, 1.51 [95% CI, 1.03–2.21]; P=0.04) even after adjustment for severity of AS, age, ejection fraction, sex, and systolic blood pressure at the last study visit. Of note, 49% of HT patients with eccentric LV hypertrophy at baseline had concentric LV hypertrophy at final study visit. In the subset of patients
undergoing aortic valve replacement (n=401), there were no significant differences in LV geometry between the normotensive and HT patients either at baseline or at the last visit before surgery.

Impact of HT on CV Events

Incidences of the primary study end point and of the secondary end point aortic valve events did not differ between normotensive patients (35% and 33%) and HT patients (34% and 32%, both \( P<0.05 \); Figure 4). Percutaneous coronary intervention was 3.5-fold more common in the HT group (1.4% in HT versus 0.4% in the normotensive group), but the difference was not statistically significant, probably because of the low incidence of this event (20 percutaneous coronary interventions) in our study population. In a multivariate Cox regression model, HT predicted a higher rate of combined ischemic CV events independent of randomized study treatment and time-varying systolic blood pressure, peak aortic jet velocity, and abnormal LV geometry (Table 3). When analyzing the individual composites of ischemic CV event end points, HT particularly predicted a 2-fold higher need for coronary artery bypass grafting at the time of planned aortic valve replacement, as well as a 2-fold higher risk of CV death in univariate analyses (Table 3).

Replacing HT with antihypertensive treatment in the model demonstrated that use of antihypertensive treatment predicted higher risk for ischemic CV independent of significant associations of systolic blood pressure (Table 4). Replacing antihypertensive treatment with type of antihypertensive drugs as individual variables revealed that treatment with \( \alpha \)-blockers predicted higher risk of ischemic CV events (hazard ratio, 2.04 [95% CI, 1.08–3.85]; \( P=0.03 \)), whereas other antihypertensive drug classes were not independently associated with outcome.

![Figure 2](image-url)  
*\( p<0.001 \) and †\( p<0.01 \) compared to normotensive patients  
**Figure 2.** Increase in left ventricular (LV) mass in hypertensive (solid line) and normotensive (dotted line) groups of patients during follow-up.

![Figure 3](image-url)  
*\( p<0.05 \) compared to normotensive patients  
**Figure 3.** Left ventricular (LV) geometry in normotensive and hypertension (HT) patient groups at baseline and at the last study visit.
Discussion

HT is a common comorbidity in patients with AS and is known to influence both assessment of AS severity and LV structure.\textsuperscript{2,3,24,25} This is the first large prospective analysis to assess the impact of HT on change in LV geometry and on patient outcome in initial asymptomatic AS. The study adds to previous knowledge by demonstrating that, during progression of AS, HT was associated with progressively more abnormal LV geometry, as well as a higher risk of death and of ischemic CV events.

Abnormal LV geometry is known to predict CV events in HT, as well as in normotensive populations.\textsuperscript{6,26,27} We have demonstrated previously that HT is the main determinant of abnormal LV geometry in asymptomatic mild-to-moderate AS and is particularly associated with eccentric LV hypertrophy, reflecting findings in patients with essential HT.\textsuperscript{1,24} Based on the postulate by Grossman et al.,\textsuperscript{28} concentric LV hypertrophy is the expected adaptation to AS, whereas HT may lead to different types of LV geometric adaptation, depending on the dominant clinical and hemodynamic factors.\textsuperscript{1,24} As proof of concept, a retrospective study by Antonini-Canterin et al.,\textsuperscript{4} including 193 patients with severe, symptomatic AS, found that concentric LV hypertrophy was the most prevalent abnormal LV geometry both in normotensive and HT patients. The present large prospective study in initially mild-to-moderate asymptomatic AS adds to this knowledge by demonstrating that progression of AS is associated with a change from predominantly normal geometry, with eccentric hypertrophy being the most frequent abnormal LV geometric pattern, to predominantly concentric LV geometry both in normotensive and HT patients. However, in spite of a 3-fold increase in the prevalence of concentric LV hypertrophy both in normotensive and HT patients during follow-up, concomitant HT was independently associated with more abnormal LV geometry throughout the study, probably reflecting the higher global valvulo-arterial load in the HT group.

The \textsuperscript{2}fold increased risk of death and of ischemic CV events associated with HT in the present study population, independent of important prognosticators like AS severity and abnormal LV geometry, is in line with findings in a meta-analysis of 61 prospective studies investigating the relationship between HT and ischemic CV mortality.\textsuperscript{29} In

Figure 4. Kaplan-Meyer plots of time to aortic valve events (A), ischemic cardiovascular (CV) events (B), CV death (C), and death from any cause (D) in normotensive (dotted line) and hypertensive (solid line) patient groups.
Table 3. Hypertension as Predictor of Ischemic Cardiovascular Events and of Total Mortality

<table>
<thead>
<tr>
<th>Variable</th>
<th>Unadjusted Hazard Ratio (95% CI)</th>
<th>Adjusted Hazard Ratio (95% CI)†</th>
</tr>
</thead>
<tbody>
<tr>
<td>Combined ischemic coronary events (n=261)</td>
<td>1.73 (1.18–2.56)*</td>
<td>1.56 (1.04–2.32)*</td>
</tr>
<tr>
<td>Coronary artery bypass grafting (n=142)</td>
<td>1.98 (1.14–3.44)*</td>
<td>1.79 (1.02–3.17)*</td>
</tr>
<tr>
<td>Myocardial infarction (n=29)</td>
<td>1.32 (0.46–3.8)</td>
<td>1.20 (0.40–3.6)</td>
</tr>
<tr>
<td>Stroke (n=57)</td>
<td>1.32 (0.62–2.75)</td>
<td>0.99 (0.45–2.15)</td>
</tr>
<tr>
<td>Percutaneous coronary intervention (n=20)</td>
<td>4.00 (0.53–29.86)</td>
<td>4.13 (0.54–31.83)</td>
</tr>
<tr>
<td>Unstable angina pectoris (n=11)</td>
<td>2.10 (0.27–16.40)</td>
<td>1.48 (0.18–12.21)</td>
</tr>
<tr>
<td>Cardiovascular death (n=72)</td>
<td>2.33 (1.01–5.38)*</td>
<td>2.23 (0.95–5.25)</td>
</tr>
<tr>
<td>Total mortality (n=143)</td>
<td>2.13 (1.20–3.76)†</td>
<td>2.16 (1.20–3.88)*</td>
</tr>
</tbody>
</table>

Results are given as hazard ratios (95% CIs).
*P<0.05 vs normotensive patients.
†P<0.01 vs normotensive patients.
Model was adjusted for randomized study treatment and time-varying systolic blood pressure, peak aortic jet velocity, and abnormal left ventricular geometry.

Table 4. Antihypertensive Treatment and Risk of Ischemic Cardiovascular Events

<table>
<thead>
<tr>
<th>Variable</th>
<th>HR (95% CI)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>1.01 (1.00–1.01)</td>
<td>0.03</td>
</tr>
<tr>
<td>Peak aortic jet velocity, m/s</td>
<td>1.20 (1.02–1.40)</td>
<td>0.03</td>
</tr>
<tr>
<td>Study treatment</td>
<td>1.20 (0.94–1.53)</td>
<td>0.14</td>
</tr>
<tr>
<td>Abnormal LV geometry</td>
<td>1.44 (1.01–2.06)</td>
<td>0.05</td>
</tr>
<tr>
<td>Any antihypertensive treatment</td>
<td>1.31 (1.02–1.69)</td>
<td>0.04</td>
</tr>
</tbody>
</table>

LV indicates left ventricular; HR, hazard ratio.

Source of Funding
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Disclosures
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### Novelty and Significance

**What Is New?**

- This is the first large prospective study to assess the influence of HT in patients with AS.
- Concomitant HT was independent of AS severity associated with more abnormal LV geometry.
- Concomitant HT was associated with a 2-fold higher mortality and a 56% increased rate of ischemic CV events.

**What Is Relevant?**

- HT is a common comorbidity in AS.

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**Summary**

In patients with AS participating in the SEAS Study, concomitant HT was associated with more abnormal LV geometry and higher CV morbidity and mortality.

Further studies are needed to test whether modern antihypertensive treatment modifies the prognostically unfavorable consequences of HT demonstrated in the SEAS Study.
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