Right Ventricular Performance in Essential Hypertension

JACK FERLINZ, M.D.

SUMMARY Effects of systemic hypertension on right ventricular (RV) performance have not been previously investigated. In this study, 10 normal patients were compared to 20 patients with uncomplicated, asymptomatic essential hypertension (defined as cuff diastolic blood pressure ≥ 100 mm Hg) after a complete hemodynamic and RV cineangiographic evaluation. The mean intra-arterial pressure in the normal group was 93 ± 11 vs 120 ± 13 mm Hg in the essential hypertension group (p < 0.01). All right-sided pressures were substantially higher in the hypertensive than in the normal patients (mean right atrial pressure: 2 ± 1 mm Hg for normals, 5 ± 3 mm Hg for hypertensives, p < 0.01; RV end-diastolic pressure: 3 ± 2 mm Hg for normals, 5 ± 2 mm Hg for hypertensives, p < 0.05; mean pulmonary artery pressure: 12 ± 3 mm Hg for normals, 17 ± 5 mm Hg for hypertensives, p < 0.01; mean pulmonary capillary wedge pressure: 6 ± 1 mm Hg for normals, 9 ± 3 mm Hg for hypertensives, p < 0.01). Cardiac index in normal and hypertensive patients was nearly identical (2.93 ± 0.80 l/min/m² in normals, 3.11 ± 0.67 l/min/m² in hypertensives, NS). In contrast, a markedly lower cineangiographic RV ejection fraction was present in the hypertensive compared with the normal patients (68 ± 6% vs 59 ± 7%, p < 0.01). The lower RV ejection fraction was due to the proportionately greater increase in the RV end-systolic volume index (22 ± 5 ml/m² for normals, 34 ± 8 ml/m² for hypertensives, p < 0.01) than in the end-diastolic volume index (69 ± 13 ml/m² for normals, 82 ± 16 ml/m² for hypertensives, p < 0.04) in the hypertensive patients. It is concluded that 1) hypertensive patients have higher right-sided pressures than normals, and 2) RV performance in essential hypertension may be characterized by a lower RV ejection fraction than in normal subjects.

RIGHT VENTRICULAR (RV) performance in adult heart disease has received only scant attention compared with the efforts designed to study the left ventricular function. The most extensively investigated area of potential RV dysfunction is coronary artery disease, where RV ejection fraction has been measured in patients with and without the right coronary artery occlusion.1,2 Furthermore, patterns of RV asynergy have been evaluated in acute3 and chronic4 ischemic coronary disease. Attempts have been also made to determine the extent of RV dysfunction in chronic obstructive pulmonary disease5 and adult congenital heart disease with RV pressure and volume overload,6 and to compare these findings with the results in normal adult population.7 Very little is known, however, about the nature of RV performance when the left ventricle is exposed to a chronic pressure overload that takes place in essential hypertension. Because the distortions of the normal geometry of the two ventricles influence each other’s performance,8-16 this study was designed to investigate the impact of chronic left ventricular pressure overload in patients with essential hypertension on RV function and to compare these performance characteristics with the RV function seen in the concomitantly studied normal subjects.

Materials and Methods

Thirty patients were selected for this study. All gave informed consent and were divided into two groups. Ten patients had the chest pain syndrome and were evaluated with a complete hemodynamic assessment, selective left ventriculography and coronary arteriography. When cardiac catheterization failed to reveal any organic cardiac pathology, these patients were allocated to the normal group (NI), and served as control subjects. The other 20 patients had documented essential hypertension for at least 3 months. Their sphygmomanometer-determined diastolic blood pressure was consistently ≥ 100 mm Hg, and they were allocated to the essential hypertension group (EH). None of the EH patients had a history of cerebrovascular disease, pulmonary disorders, congestive heart failure or significant valvular or congenital lesions. While coronary anatomy was not studied in the hypertensive subjects, and latent coronary artery disease could therefore have been present in some of them, none had a history or symptoms indicative of coronary disease or electrocardiographic evidence of a prior myocardial infarction. All subjects were in normal sinus rhythm and had no conduction abnormalities. Funduscopic examination was normal in all patients, and none of the subjects had elevated serum creatinine. A clinical profile for the entire patient population is presented in table 1. Some of the EH patients were receiving antihypertensive medications; these were stopped at least 2 weeks before the study. These patients were closely monitored after the antihypertensive medications were discontinued in order to detect immediately a potentially dangerous acceleration of hypertension. This did not occur in any of the subjects, and they did not develop new symptoms or complications. Therefore,
none of the patients had to be excluded because the withdrawal of medication was poorly tolerated.

All patients in both groups were studied with a combined hemodynamic-angiographic approach. Circulatory measurements and angiograms were performed in the fasting state, in the supine position, and without premedication. Cardiac output (CO) and pressure data were collected before the right ventriculogram. CO was determined with the Lyons indocyanine green indicator-dilution computer by averaging four sequential measurements. All pressures — aortic (AP) and pulmonary artery (PAP) systolic, diastolic, and mean; RV systolic and end-diastolic; mean right atrial (RAP); and mean pulmonary capillary wedge pressure (PCWP) — were measured in each case with Statham model P23Db strain gauges and recorded with an Electronics for Medicine DR-12 Simultrace recorder over three full respiratory cycles. Heart rate (HR) was monitored throughout the study. In addition, systemic vascular resistance (SVR = [mean AP − mean RAP] / CO) and pulmonary vascular resistance (PVR = [mean PAP − mean PCWP] / CO) were calculated for each patient.

Single-plane, cine right ventriculograms in the 30° right anterior oblique (RAO) projection were performed in all 30 patients. Fifty-five to 65 ml of 76% meglumine sodium diatrizoate (Renografin 76) were injected over a 3-second interval into the RV chamber through a #8 Eppendorf or NIH catheter in each case, and right ventriculograms recorded at 64 frames/sec on 35-mm Ilford Cinegram F film with Philips 9-inch image intensifier. Only the first six beats after complete opacification were analyzed by two independent observers. Whenever different values for the RV ejection fraction were obtained, the two values were averaged. In general, there was only a negligible variance in the calculated values; the largest discrepancy was only 4%, and the correlation coefficient for the two observers was 0.96. If a premature ventricular contraction occurred, at least two successive normal sinus beats (after the premature beat) had to occur before a frame was selected for volumetric analysis. A typical high-resolution frame (EH patient 16) is presented in figure 1.

RV volumes were determined with the method for single-plane RV volume measurements previously developed in our laboratory. This method is quite accurate (r = 0.93) compared with the very precise biplane RV volumetric analysis. End-diastolic volume index (EDVI), end-systolic volume index (ESVI), stroke volume index (SVI) and ejection fraction (EF = SVI/EDVI) were calculated for each patient.

All data were analyzed with the t test for nonpaired variables.

**Results**

The complete mean hemodynamic and volumetric data for both groups are given in table 2, and a summary of key cardiovascular findings for each NI and EH patient in tables 3 and 4.

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**TABLE 1. Clinical Profile of Patient Population**

<table>
<thead>
<tr>
<th>Group</th>
<th>No. pts</th>
<th>Age (years) (mean ± sp)</th>
<th>Average of six hypertensive BP readings (± sp) during 2 weeks before study</th>
<th>Electrocardiogram</th>
<th>Cardiomegaly (CT ratio ≥ 0.50) on chest x-ray</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>10</td>
<td>46 ± 9</td>
<td>None</td>
<td>None</td>
<td>All</td>
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<tr>
<td>Hypertensive</td>
<td>20</td>
<td>51 ± 8</td>
<td>169 ± 15</td>
<td>110 = 8</td>
<td>13 (65%)</td>
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<tr>
<td></td>
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<td>7 (35%)</td>
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</tbody>
</table>

Abbreviations: BP = blood pressure; CT = cardiothoracic; LVH = left ventricular hypertrophy; WNL = within normal limits.

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**FIGURE 1. A representative right ventricular cineangiogram in end-systole (left) and end-diastole (right).**
The EH patients are characterized by a substantially higher (p < 0.01) systolic, diastolic, and mean systemic intraarterial pressures than their normal counterparts. The diastolic pressure for the EH group was > 90 mm Hg — a reading that has been associated with a greater incidence of cardiovascular complications than in control subjects.  

SVR in the hypertensive group was partially lowered by the higher CO and RAP than was present in the control group. All right-heart pressures, including the right and left ventricular filling pressures (mean RAP and PCWP, respectively) were significantly higher in the EH patients. The relationship between the systemic and the right-sided pressures is depicted in figure 2. PVR, however, was only slightly greater in hypertensive than in the normal subjects, probably again reflecting higher CO and PCWP in the EH group.

There was no appreciable difference in HR between the two groups. Cardiac index was higher in the EH patients, but this increase was not statistically significant. In contrast, RV volumetric analysis revealed that the RV EDVI and ESVI are substantially greater in the EH patients (p < 0.04 and < 0.01, respectively), while the RV SVI is almost the same in both groups. Because the increase in the RV ESVI is proportionally much larger than the increase in the RV EDVI (ESVI increased from 22 to 34 ml/m², representing a rise of 55%; EDVI rose from 69 to 82 ml/m², representing an increase of only 19%), RV EF is significantly lower (p < 0.01) in the EH patients than in the controls. The range of RV EF for normal subjects was 56–78% (only one patient had a RV EF less than 60%), while in the EH subjects the RV EF varied from 51–71% (with 10, or 50%, of all patients having an RV EF that was less than 60%). The relationship between the RV EDVI vs RVEDP, and the RV ESVI, SVI and EF vs RV filling pressure (that is, mean RAP), is presented in figure 3.

In a recent report, 22 Olivari et al. suggested that the RV function in systemic hypertension can be predicted on the basis of a standard ECG. If the ECG of the EH patients shows evidence of left ventricular hypertrophy without strain, the RV function is likely to be supernormal. If left ventricular hypertrophy with strain is seen, however, RV performance purportedly deteriorates below the normal levels. This differentiation between the normal and abnormal RV function based on ECG could not be confirmed in the present study. The patient (EH patient 6) with the most severely abnormal ECG (SVI + RV = 46 mm with strain) had one of the highest RV EFs in the EH group (70%). However, a better correlation between the RV performance and ECG might emerge if more of our EH patients had abnormal ECG tracings.

**Discussion**

This study demonstrates that right-sided circulation is not immune to the effects of systemic hypertension. The increase in the right-heart pressures among the EH patients is significantly different from the values in the normotensive controls in this study and in the classic study of normal subjects by Fowler et al. 23 Earlier reports have suggested that pulmonary pressures remain normal in essential hypertension. 24–27 More recently, relative elevation of right-sided pressures in hypertensive patients has been reported. 28, 29

Some investigators have postulated that when
TABLE 3. Summary of Key Cardiovascular Findings for Each Normal Patient

<table>
<thead>
<tr>
<th>Pt no.</th>
<th>Mean AP (mm Hg)</th>
<th>Mean RAP (mm Hg)</th>
<th>RVEDP (mm Hg)</th>
<th>Mean PAP (mm Hg)</th>
<th>Mean PCWP (mm Hg)</th>
<th>CI (l/min/m²)</th>
<th>RV EDVI (ml/m²)</th>
<th>RV ESVI (ml/m²)</th>
<th>RV EF (%)</th>
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<td>69 ± 13</td>
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<td>68 ± 6</td>
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Abbreviations: See table 2.

Pulmonary pressures rise in patients with essential hypertension, this increase reflects the increasing diastolic pressures in the failing left ventricle. None of the EH patients in this study had clinical, chest x-ray or hemodynamic findings suggestive of even borderline left ventricular decompensation. Left ventricular failure, therefore, cannot be considered the major mechanism responsible for the elevated right-sided pressures of the EH patients. However, because an impaired cardiac pump function can be detected during exercise even in patients with early, uncomplicated essential hypertension, a latent left ventricular failure in the hypertensive group cannot be completely excluded, and this incipient cardiac in-

TABLE 4. Summary of Key Cardiovascular Findings for Each Hypertensive Patient

<table>
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<tr>
<th>Pt no.</th>
<th>Mean AP (mm Hg)</th>
<th>Mean RAP (mm Hg)</th>
<th>RVEDP (mm Hg)</th>
<th>Mean PAP (mm Hg)</th>
<th>Mean PCWP (mm Hg)</th>
<th>CI (l/min/m²)</th>
<th>RV EDVI (ml/m²)</th>
<th>RV ESVI (ml/m²)</th>
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<td>3.11 ± 0.67</td>
<td>82 ± 16</td>
<td>34 ± 8</td>
<td>59 ± 7</td>
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Abbreviations: See table 2.
sufficiency may contribute to the rise in right-sided pressures.

However, if it is accepted that left ventricular function is virtually intact in the EH patients, and it therefore plays only a minor (or no) role in increasing pulmonary pressures, the cause for elevated right-sided pressures in hypertensive subjects becomes obscure. Decades ago it was postulated that systemic hypertension may cause hypertrophy of the left ventricular septum, which encroaches on the right ventricular chamber to such an extent that there is a functional obstruction within the RV cavity (the Bernheim's syndrome). As a result, peripheral venous and right atrial pressures increase, resulting in eventual right-heart failure. The very existence of this syndrome, however, is still in doubt. Furthermore, even if it is real, it is probably not a factor in the EH patients in this study because in the Bernheim's syndrome pulmonary circulation should remain unaffected and RV volumes should decrease; yet our EH subjects have higher pulmonary arterial pressures and RV volumes than the normal controls. Potentially much more important than the Bernheim's syndrome is the effect of various humoral mechanisms on right-sided circulation. Patients with essential hypertension have enhanced sympathetic activity and have resting plasma catecholamine concentrations that are substantially increased. Furthermore, the rate of angiotensin generation is clearly elevated in most of these subjects. Because pulmonary circulation...
responds readily to norepinephrine and angiotensin with pulmonary vasoconstriction and elevated pulmonary pressures.\(^49\)\(^-\)\(^44\) increased right-heart pressures in essential hypertension may reflect enhanced activity of catecholamines, angiotensin, or some other humoral substance that is capable of producing pulmonary vasoconstriction.\(^45\) Whether these humoral agents are also responsible for the recent observation that the decreased vascular compliance in essential hypertension is not limited to the arterial system, but is also present in the venous circulation,\(^46\) is still unresolved.

CO has been reported to be elevated in uncomplicated essential hypertension,\(^20\)\(^,\)\(^47\)\(^,\)\(^48\) as has the resting HR.\(^35\)\(^,\)\(^48\)\(^,\)\(^49\)\(^-\)\(^51\) Most investigators believe that the high resting CO is a direct result of the increase in HR.\(^51\) Only one study reports a normal HR and a marked increase in cardiac index.\(^47\) No significant difference in CO and HR between the NI and EH subjects was noted in this study. Therefore, it must be concluded that CO and HR frequently remain normal in patients with essential hypertension — a finding consistent with reports from many other investigators.\(^46\)\(^,\)\(^52\)\(^-\)\(^54\)

Ventricular EF is one of the most sensitive indices of cardiac function.\(^55\)\(^-\)\(^57\) The volumetric analysis of RV performance in NI and EH patients is, therefore, the cornerstone of this study. Compared with the normal subjects, the RV EF is significantly lower in the hypertensive patients, indicating that RV emptying may become impaired in essential hypertension.

Several mechanisms may account for this finding. It has been demonstrated that the end-systolic dimensions increase, and ventricular contractility decreases with increases in afterload.\(^58\) Because of the increased pulmonary pressures in the EH patients (fig. 2), the RV afterload becomes augmented, and the RV responds with larger ESVI and lower EF (fig. 3). This study, therefore, does not confirm prior findings in experimental animals that systemic hypertension generates a stimulus in response to the increased functional requirements of myocardium, which, until the left ventricle fails, causes the RV to function in an augmented state;\(^59\)\(^,\)\(^60\) or that RV performance can be increased by the right-sided Anrep effect.\(^61\)

Other mechanisms that may depress the RV performance in systemic hypertension may occur because of the interdependence of the two ventricles.\(^5\)\(^,\)\(^6\)\(^-\)\(^16\)\(^,\)\(^62\) Thus, a stressed left ventricle may adversely affect RV performance and bring about changes in the RV volume-pressure characteristics. This influence may be exerted not only by the interventricular septum, which is comprised of a thin right and a relatively thick left muscle mass,\(^63\)\(^-\)\(^66\) but also by the circular and spiral bundles of muscle fibers that encircle both ventricles.\(^66\)\(^,\)\(^67\)

In conclusion, the concept that the right ventricle is immune from the effects of uncomplicated systemic hypertension is no longer tenable. RV pressures, volumes, and EF change significantly even with early abnormal increases in the arterial pressure. Systemic hypertension therefore exerts its influence on the whole heart, strengthening the concept that the right and left ventricles physiologically represent a single functional unit. The causes for right-heart changes seen in essential hypertension are complex. To evaluate these mechanisms further, simultaneous right and left ventricular volume-pressure relationships must still be investigated.

Acknowledgment

The expert secretarial assistance of Wilma Mottin is greatly appreciated.

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