Transition From Chronic Compensated to Acute Decompensated Heart Failure
Pathophysiological Insights Obtained From Continuous Monitoring of Intracardiac Pressures

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Background—Approximately half of all patients with chronic heart failure (HF) have a decreased ejection fraction (EF) (systolic HF [SHF]); the other half have HF with a normal EF (diastolic HF [DHF]). However, the underlying pathophysiological differences between DHF and SHF patients are incompletely defined. The purpose of this study was to use echocardiographic and implantable hemodynamic monitor data to examine the pathophysiology of chronic compensated and acute decompensated HF in SHF versus DHF patients.

Methods and Results—Patients were divided into 2 subgroups: 204 had EF < 50% (SHF) and 70 had EF ≥ 50% (DHF). DHF patients had EF of 58 ± 8%, end-diastolic dimension of 50 ± 10 mm, estimated resting pulmonary artery diastolic pressure (ePAD) of 16 ± 9 mm Hg, and diastolic distensibility index (ratio of ePAD to end-diastolic volume) of 0.11 ± 0.06 mm Hg/mL. In contrast, SHF patients had EF of 24 ± 10%, end-diastolic dimension of 68 ± 11 mm, ePAD of 18 ± 7 mm Hg, and diastolic distensibility index of 0.06 ± 0.04 mm Hg/mL (P < 0.05 versus DHF for all variables except ePAD). In SHF and DHF patients who developed acute decompensated HF, these events were associated with a significant increase in ePAD, from 17 ± 7 to 22 ± 7 mm Hg (P < 0.05) in DHF and from 21 ± 9 to 24 ± 8 mm Hg (P < 0.05) in SHF. As a group, patients who did not have acute decompensated HF events had no significant changes in ePAD.

Conclusions—Significant structural and functional differences were found between patients with SHF and those with DHF; however, elevated diastolic pressures play a pivotal role in the underlying pathophysiology of chronic compensated and acute decompensated HF in both SHF and DHF. (Circulation. 2008;118:000-000.)

Key Words: diastole • heart failure • hemodynamics • physiology • systole

The pathophysiology of both chronic compensated and acute decompensated heart failure (HF) remains incompletely understood, especially with respect to the differences between patients with HF and a decreased ejection fraction (EF) (systolic HF [SHF]) and those with HF and a normal ejection fraction (diastolic HF [DHF]). Recent studies have suggested that patients with SHF have structural and functional changes consistent with eccentric remodeling and dominant abnormalities in systolic function; in contrast, patients with DHF have structural and functional changes consistent with concentric remodeling and dominant abnormalities in diastolic function.¹–⁶ Functional measurements made in these studies used noninvasive Doppler and tissue Doppler estimates of LV diastolic pressure in patients with DHF.⁵–⁷ However, these noninvasive studies did not directly compare SHF and DHF patients. In addition, each study made measurements at only 1 point in time in chronic compensated patients. Likewise, studies using noninvasive and invasive methods have been able to measure diastolic pressures at the time of acute symptomatic decompensation; however, the time period leading up to decompensation has not been examined.¹⁰ In fact, no previous study has been able to continuously measure diastolic pressures over an extended time period in compensated chronic HF or acute decompensated HF in patients with either SHF or DHF. Having these measurements would help to more clearly define the pathophys-
iology of chronic compensated and acute decompensated HF in both SHF and DHF patients. Accordingly, the purpose of the present study was to use the hemodynamic, structural, and functional data obtained with an investigational implantable hemodynamic monitor and echocardiographic studies to examine and compare the pathophysiology of chronic compensated and acute decompensated HF in patients with DHF versus SHF.

Methods

Patients

The present study is a substudy of the Chronicle Offers Management to Patients with Advanced Signs and Symptoms of Heart Failure (COMPASS-HF) study. The inclusion and exclusion criteria for the COMPASS-HF study were described previously. Briefly, patients were eligible for the study if they were ≥18 years old, had New York Heart Association class III or IV HF, were managed in an HF program with optimized medical therapy for at least 3 months before enrollment, and had at least 1 HF-related hospitalization or emergency department visit necessitating intravenous treatment within the previous 6 months. Patients were excluded from the study if they had severe chronic obstructive or restrictive pulmonary disease; had primary pulmonary hypertension; had a major cardiovascular event within 3 months before enrollment; had known atrial or ventricular septal defects, tricuspid or pulmonic stenosis, or mechanical right heart valves; had a severe, noncardiac condition limiting 6-month survival; had serum creatinine ≥3.5 mg/dl or chronic renal dialysis; were likely to undergo cardiac transplantation within 6 months of enrollment; were receiving continuous positive inotropic therapy; were presently implanted with an incompatible pacemaker or implantable cardioverter-defibrillator; were receiving cardiac resynchronization therapy that had not achieved optimal programming for 3 months; or were of childbearing age and were not using reliable contraceptive measures. The investigational review board of each participating institution approved the study protocol, and all patients provided written informed consent.

Study Design

One prespecified and planned subgroup analysis in COMPASS-HF was based on left ventricular (LV) ejection fraction (EF) <50% and ≥50%. Two hundred seventy-four New York Heart Association class III or IV patients were enrolled; 201 patients had an EF <50%, and 70 patients had an EF ≥50%. Furthermore, the 70 patients with HF and a normal EF actually fulfilled the diagnostic criteria for DHF. Patients transmitted implantable hemodynamic monitor (IHM) data at least weekly via a home-based remote monitor. At enrollment, each patient underwent a Minnesota Living With Heart Failure Questionnaire and a 6-minute walk test.

An HF-related event (HFRE) was defined as an unexpected hospitalization, emergency department visit, or urgent clinic visit requiring intravenous therapy. During the 6-month study period, all but 16 of the 197 reported HFREs were associated with hypervolemia. Sixteen hospitalizations for cardiovascular hypovolemia, hypotension, or pre-eclampsia occurred in 15 patients during the 6-month study period. In the present study, the IHM data obtained from patients who experienced hypervolemic HFREs were examined separately from the IHM data obtained from patients who experienced hypovolemic HFREs. An independent Clinical Events Review Committee, blinded to the randomization assignment, adjudicated all HFREs during the study period.

Echocardiographic Substudy

Each of the 274 patients underwent a screening echocardiogram analyzed at the study site to provide LVEF and LV end-diastolic dimension (EDD). In addition, a subset of patients (n=146) underwent echocardiographic and Doppler studies analyzed by the core laboratory (M.S.J.S.). Forty patients with DHF and 106 patients with SHF were enrolled in this echocardiographic substudy. In the core laboratory analysis, 15 cardiac cycles were recorded and analyzed for each echocardiographic view. Echocardiographic and Doppler measurements and calculations of LV volume and systolic and diastolic function were made using the methods and the recommendations of the American Society of Echocardiography. These data were compared with normal values for this core laboratory.

Device Description and Measurements

The IHM used in this study continuously measured and stored right ventricular (RV) systolic pressure, RV diastolic pressure, an estimate of pulmonary artery diastolic pressure (ePAD), maximum positive and negative changes in pressure over time (dP/dt and −dP/dt, respectively), heart rate, and activity. The system components, implantation procedure, monitoring process, storage and retrieval methods, and pressure analysis methods have previously been described. The ePAD is defined as the RV pressure at the time of pulmonary artery valve opening, which occurs at the time of maximum dP/dt. A strong correlation has been shown to exist between the ePAD and actual pulmonary artery diastolic pressures measured under a variety of physiological conditions. In the absence of significant pulmonary vascular disease (an exclusion criteria for COMPASS-HF), ePAD provides a clinical estimate of pulmonary capillary wedge pressure and LV diastolic pressure.

Two sets of measurements were made using the IHM in this study: the minimum nighttime values (used as resting values) and the 24-hour daily median (used as ambulatory values). Nighttime data were collected between the hours of midnight and 4 AM when no activity was detected. Values at enrollment represent the average of the resting values from day 8 to 14 after placement of the IHM. These times were chosen to allow complete recovery from the implantation procedure. Minimum nighttime and 24-hour daily median data in the patients who had an HFRE were reported at day 49 before the event (baseline), 1 day before the event (peak), and day 5 after the event (post).

Statistical Analysis

Comparison of demographic, hemodynamic, structural, and functional data between SHF and DHF patients was made by use of an unpaired t-test or Wilcoxon rank-sum test. Changes in IHM-derived pressures in the patients who had an HFRE were made using a linear mixed model. For each HFRE, the change in pressure parameters and body weight comparing baseline values with values at the time of the HFRE (peak) and recovery from HFRE (post) were calculated. These changes were compared using a linear mixed model accounting for repeated events within patients by including the patient as a random effect. Fixed effects in this model included baseline hemodynamic values and an indicator variable specifying peak or post values. An interaction term also was included in the model to test whether the changes from baseline were different between DHF and SHF patients. Data presented in the tables are mean±SD. Data presented in the figures are mean±SE. Values of P<0.05 were considered statistically significant. Body weight was measured daily. However, for the purposes of presentation clarity, all available values of body weight were presented at 7, 4, and 2 weeks and 1 day before HFRE and 5 days after HFRE. In addition, the percent change in body weight from baseline to time of the HFRE (peak) and recovery from HFRE (post) for all available paired samples were also presented. A method based on a standard statistical process control technique known as a cumulative sum chart was used to determine the number of days of advanced notice from a detection of a change in pressure to the development of an HFRE in DHF versus SHF patients. Data management and statistical analyses were conducted by the sponsor of the study, Medtronic, Inc.

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.

Results

Patient Characteristics at Enrollment

Significant differences in demographics were found between the patients with SHF and those with DHF enrolled in COMPASS-HF (Table 1). In the DHF group, a higher percentage of patients were women, patients were older, their systolic
Table 1. Patient Characteristics at Enrollment

<table>
<thead>
<tr>
<th>Variable</th>
<th>DHF (n=70)</th>
<th>SHF (n=204)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gender, M/F, %</td>
<td>47/53</td>
<td>72/28</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Age, y</td>
<td>62±12</td>
<td>57±14</td>
<td>0.005</td>
</tr>
<tr>
<td>Heart rate, bpm</td>
<td>76±14</td>
<td>77±14</td>
<td>0.48</td>
</tr>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>117±19</td>
<td>111±18</td>
<td>0.01</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>67±10</td>
<td>68±11</td>
<td>0.40</td>
</tr>
<tr>
<td>NYHA class III/IV, %</td>
<td>90/10</td>
<td>84/16</td>
<td>0.24</td>
</tr>
<tr>
<td>6-min walk, m</td>
<td>211±115</td>
<td>245±122</td>
<td>0.04</td>
</tr>
<tr>
<td>MLWHF score</td>
<td>69±22</td>
<td>63±25</td>
<td>0.07</td>
</tr>
<tr>
<td>History of coronary artery disease, %</td>
<td>57</td>
<td>53</td>
<td>0.68</td>
</tr>
<tr>
<td>History of hypertension, %</td>
<td>86</td>
<td>70</td>
<td>&lt;0.008</td>
</tr>
<tr>
<td>Years with heart failure</td>
<td>4.5±4.7</td>
<td>5.9±5.2</td>
<td>0.037</td>
</tr>
</tbody>
</table>

NYHA indicates New York Heart Association; MLWHF, Minnesota Living With Heart Failure questionnaire. Data are mean±SD.

Cardiac Structure and Function in DHF Versus SHF Patients: Echocardiographic Data

Significant differences in echocardiographic measurements were found between the SHF and DHF patients enrolled in COMPASS-HF. Data from the echocardiographic substudy analyzed by the core laboratory are presented in Table 3. Core laboratory normal control values for the echocardiographic and Doppler measurements include LVEDD of 50±5 mm, LV end-systolic dimension of 34±5 mm, LVEDV of 67±12 mL/m², and left atrial area of 19±5 mm². Both LV dimensions and volumes were larger in SHF patients than DHF patients and the core laboratory normal control subjects. LV dimensions and volumes in DHF patients were not significantly different from those of core laboratory normal control subjects. Left atrial area was increased in both SHF and DHF patients compared with core laboratory normal control subjects. Measurements of Doppler filling velocities and isovolumic relaxation time were comparable between SHF and DHF patients.

In addition to the echocardiographic analysis performed in the core laboratory on 40 of the 70 DHF patients and 106 of the 204 SHF patients, individual site investigators were asked to analyze screening echocardiograms and submit just LVEF and LVEDD data on all 274 patients; other echocardiographic and Doppler measurements were not provided by the site investigators. LVEF

Table 2. IHM-Derived Data at Enrollment

<table>
<thead>
<tr>
<th>Variable</th>
<th>24 Hours</th>
<th>Resting</th>
<th>24 Hours</th>
<th>Resting</th>
<th>24 Hours</th>
<th>Resting</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>RV systolic pressure, mm Hg</td>
<td>46±15</td>
<td>32±14</td>
<td>50±16</td>
<td>37±14</td>
<td>0.09</td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>RV diastolic pressure, mm Hg</td>
<td>14±6</td>
<td>7±8</td>
<td>14±6</td>
<td>8±7</td>
<td>0.84</td>
<td>0.27</td>
<td></td>
</tr>
<tr>
<td>ePAD, mm Hg</td>
<td>27±8</td>
<td>16±9</td>
<td>28±8</td>
<td>18±7</td>
<td>0.35</td>
<td>0.11</td>
<td></td>
</tr>
<tr>
<td>Peak RV dP/dt, mm Hg/s</td>
<td>439±163</td>
<td>280±118</td>
<td>392±134</td>
<td>264±95</td>
<td>0.03</td>
<td>0.33</td>
<td></td>
</tr>
<tr>
<td>Peak RV dP/dt, mm Hg/s</td>
<td>−373±143</td>
<td>−268±115</td>
<td>−394±139</td>
<td>−296±131</td>
<td>0.29</td>
<td>0.10</td>
<td></td>
</tr>
</tbody>
</table>

Resting values are the minimum nighttime values; 24-hour values are the 24-hour daily median. Data are mean±SD. P values represent statistical differences between DHF and SHF.
CIIMH data for the DHF group were derived from a total of 41 HFREs in 22 patients and for the SHF group from a total of 122 HFREs in 68 patients. Overall, the SHF group had slightly higher pressures at baseline, at the peak event, and after treatment of the HFREs than DHF patients. In patients who had an HFRE, CIIMH pressures increased significantly before the event in both the DHF and SHF patients (P<0.001). However, as a percentage of change from baseline pressures, no significant differences were found in peak and postevent pressures between DHF and SHF patients (Figure 4A). CIIMH pressures decreased significantly in both DHF and SHF groups after treatment. In the SHF patients, the postevent pressures were slightly lower than baseline pressures; in the DHF patients, postevent pressures were the same as baseline pressures. This pattern of changes in CIIMH pressures before and after an HFRE also was present when only HF hospitalizations were examined (Figure 4B).

Table 4. CIIMH Pressure Changes Associated With Hypervolemic HFREs

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>Peak</th>
<th>Post Event</th>
<th>Baseline</th>
<th>Peak</th>
<th>Post Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>RV SP, mm Hg</td>
<td>50±11</td>
<td>54±10*</td>
<td>49±10†</td>
<td>55±16</td>
<td>60±14*</td>
<td>53±13†</td>
</tr>
<tr>
<td>RV DP, mm Hg</td>
<td>16±4</td>
<td>19±6*</td>
<td>16±5†</td>
<td>17±8</td>
<td>21±8*</td>
<td>17±8†</td>
</tr>
<tr>
<td>ePAD, mm Hg</td>
<td>28±6</td>
<td>32±6*</td>
<td>28±6†</td>
<td>31±9</td>
<td>35±9*</td>
<td>30±8†</td>
</tr>
<tr>
<td>RV dP/dt</td>
<td>399±113</td>
<td>410±115*</td>
<td>396±110†</td>
<td>414±125</td>
<td>406±139*</td>
<td>397±150</td>
</tr>
<tr>
<td>RV −dP/dt</td>
<td>−383±102</td>
<td>−415±117*</td>
<td>−381±89†</td>
<td>−424±135</td>
<td>−432±135*</td>
<td>−410±142†</td>
</tr>
</tbody>
</table>

Baseline indicates 24-hour median value at day 49 before the event; Peak, 24-hour median value 1 day before the event; Post, 24-hour median value on day 5 after the event; SP, systolic pressure; and DP, diastolic pressure. Data are mean±SD. *P<0.001 for the change between baseline and peak for DHF and SHF patients; †P<0.001 for the change between peak and post for DHF and SHF patients. These changes in pressure parameters were similar in the DHF and SHF patients; the interaction term was not significant in any of the parameters measured (P>0.1 for all parameters).
Separate linear mixed models were used to analyze the change in RV diastolic pressure, RV systolic pressure, ePAD, \(\frac{dP}{dt}\), and body weight from baseline to peak and post of an HFRE. Analysis using these models for RV diastolic pressure, RV systolic pressure, ePAD, and \(\frac{dP}{dt}\) indicated statistically significant changes from baseline to the peak of an HFRE (\(P<0.001\) for all) and statistically significant changes from peak to post after an HFRE (\(P<0.001\) for all). These changes in pressure parameters were similar in the DHF and SHF patients; the interaction term was not significant in any of the models (\(P=0.48\) for RV diastolic pressure; \(P=0.60\) for RV systolic pressure; \(P=0.79\) for ePAD; and \(P=0.65\) for \(-\frac{dP}{dt}\)). For \(\frac{dP}{dt}\), no significant changes were found from baseline to peak or from peak to post.

The rate of increase in IHM pressures before the HFRE was somewhat more rapid in the DHF patients and somewhat slower in the SHF patients (Figures 2 and 3). A statistical analysis using a linear mixed model was performed to determine whether the pattern of change in IHM pressures during the period before an HFRE was different in the DHF and SHF patients. The observed slopes of ePAD values for the 3 weeks (day \(-21\) to \(-1\)), 2 weeks (day \(-14\) to \(-1\)), and 1 week (day \(-7\) to \(-1\)) period before an event were \(0.17\), \(0.16\), and \(0.29\) mm Hg/d for DHF and \(0.15\), \(0.18\), and \(0.28\) mm Hg/d for SHF. No statistically significant differences were found between DHF and SHF patients in the slope of the ePAD pressures before the event (\(P=0.10\)). The ePAD data were used in the cumulative sum analysis to determine the number of days of advanced notice from a detection of a change in pressure to the development of an HFRE; the number of days of advanced notice was significantly fewer in the DHF (19.3\(\pm\)17.3) than in the SHF (29.1\(\pm\)22.3) patients (\(P<0.05\)).

In addition, diastolic pressure versus time was examined in DHF and SHF patients who did not experience an HFRE and is plotted in Figure 5. We examined 169 patients with no HFREs during the 6-month randomized follow-up period. Time segments spanning 75 days were selected randomly beginning at day 0 and taking 75 days after day 0. No significant changes in IHM pressures were observed during this 75-day period. These data demonstrate that during time periods when no HFREs were present, IHM pressures did not significantly change.

Body weight was measured in DHF and SHF patients at 7 weeks, 4 weeks, 2 weeks, and 1 day before and 5 days after an HFRE.
No statistically significant changes in average body weight were detected during this examination period. When examined as percent change in paired samples, body weight increased by 1.1 ± 3.4% in DHF and 1.0 ± 3.0% in SHF at the time of HFRE (peak) compared with body weight at baseline. Body weight at time of recovery from HFRE (post) was decreased by 1.9 ± 1.9% in DHF and 1.5 ± 2.2% in SHF compared with peak. These changes in body weight were similar in the DHF and SHF patients; the interaction term was not significant.

IHM-Derived Pressure Changes Associated With Hypovolemic HFREs

Changes in IHM-derived hemodynamic data before, during, and after hypovolemic HFREs also were examined. Sixteen hypovolemic HFREs occurred in 15 patients: 3 with DHF and 13 with SHF. Daily median ePAD pressures were 26 ± 5 mm Hg at baseline, 23 ± 7 mm Hg at peak, and 25 ± 5 mm Hg after hypovolemic HFRE (post). Too few hypovolemic HFREs were found for meaningful statistical analysis or for division of data between DHF and SHF patients; however, data were sufficient to suggest that hypovolemia was associated with a fall in IHM pressures, and treatment of hypovolemia was associated with a return to baseline of IHM pressures.

Discussion

The purpose of the present study was to use the hemodynamic, structural, and functional data obtained with IHM and echocardiographic studies to examine and compare the pathophysiology of chronic compensated and acute decompensated HF in patients with DHF and those with SHF. Data presented in this study support the following conclusions. First, patients with DHF had normal LV volume and EF, whereas patients with SHF had increased LV volume and decreased EF. Second, patients with DHF or SHF had increased diastolic pressures (both RV diastolic pressure and ePAD), but DHF patients had decreased diastolic distensibility and SHF patients had increased diastolic distensibility. Third, acute decompensated HF in both DHF and SHF patients was preceded by a significant increase in diastolic pressures. Finally, DHF and SHF patients who did not develop acute decompensated HF had no significant changes in diastolic pressure during the study period. Therefore, although significant structural and func-
tional differences were found between patients with SHF and those with DHF, elevated diastolic pressures play a pivotal role in the underlying pathophysiology of chronic compensated and acute decompensated HF in both SHF and DHF.

Pathophysiology of Chronic Compensated DHF Versus SHF

Previous studies have suggested that significant structural and functional differences exist between patients with clinical evidence of HF and a decreased LVEF (SHF) and patients with clinical evidence of heart failure, a normal LVEF, and abnormal diastolic function (DHF).1–9 Although the nomenclature applied to these 2 groups of HF patients remains a matter of discussion, the structural and functional characteristics used to distinguish these 2 groups are becoming more clearly understood.1–9 Data from the present study are concordant with previous studies showing that patients with SHF have significant and progressive LV dilation; in contrast, patients with DHF have no significant change in LV end-diastolic volume compared with normal control subjects.1–9 Together with previous studies, the measurements of LV volume in the present study support the conclusion that patients with DHF frequently have concentric remodeling and patients with SHF commonly have eccentric remodeling.

Previous studies have shown that LV structural remodeling in patients with DHF was associated with changes in LV diastolic function, particularly increased diastolic pressure and increased LV stiffness.1–9 Data from the present study showed that patients with DHF had a decreased index of instantaneous diastolic distensibility and thus are concordant with previous studies. However, it should be noted that these 70 patients represent the largest cohort of DHF patients in whom diastolic pressures were directly measured. In addition, data from the present study showed that although diastolic pressures also were increased in SHF patients, the index of instantaneous diastolic distensibility was increased in SHF patients. This represents the first study to compare directly measured diastolic pressures and distensibility in SHF and DHF patients. From published data and data from the echocardiography core laboratory, indexes of instantaneous diastolic distensibility based on the ratios of ePAD to EDD and ePAD to EDV for normal control subjects were 0.30 mm Hg/mm and 0.08 mm Hg/mL, respectively, values that are lower than DHF but higher than SHF patients. These differences highlight the pivotal role that decreased diastolic distensibility plays in the pathophysiology of chronic compensated DHF.

Pathophysiology of Acute Decompensated DHF Versus SHF

The present study is the first to document the ambulatory hemodynamic changes that precede the development of acute decompens-
ated HF in patients with DHF or SHF. When patients with DHF or SHF developed an HFRE associated with hypervolemia, it was preceded by a significant increase in diastolic pressures. In contrast, when patients with DHF or SHF developed HFRE associated with hypovolemia, it was preceded by a decrease in diastolic pressures. During periods when no significant changes in IHM pressures were found, neither hypervolemic nor hypovolemic HFREs occurred. Therefore, changes in diastolic pressures appear to play a pivotal role in the underlying pathophysiology of acute decompensated HF in both SHF and DHF. It is possible that the ability to monitor these ambulatory hemodynamic data would allow clinicians to tailor therapy and to prevent the development of HFREs.

Although not statistically significant, a trend was found for diastolic pressures to rise more rapidly during the development of hypervolemic HFREs in patients with DHF. This finding has clinical relevance because it correlates with the finding that patients with DHF also have decreased LV diastolic distensibility. In patients with DHF, only a small increase in diastolic volume can result in a marked increase in diastolic pressure. This finding may be important in the development of clinical applications of ambulatory hemodynamic monitoring using IHM because it suggests that it may be necessary to monitor pressures in patients with DHF more frequently.

Successful treatment of acutely decompensated HF patients with both SHF and DHF was associated with a decrease in diastolic pressures to values equivalent to or below those present at baseline. The ability to continuously monitor pressures during treatment may allow clinicians to tailor treatment more accurately and avoid excessive or insufficient diuresis. Ambulatory IHM monitoring might therefore provide hemodynamic information that would help avoid discharging patients from the hospital before pressures have decreased sufficiently to return to a chronic compensated state; alternatively, it may help avoid overdiureting patients and significant impairment of renal function.

Within the context of this study, these results suggest that IHM data appear to correlate with the development of an HFRE but measurement of daily body weight did not. However, it is important to recognize that the relative value of measuring body weight instead of intracardiac pressures was not selectively or directly tested in the present study. Home monitoring of daily body weights remains an important component in the management of patients with HF. The results of the present study suggest that IHM data can be used with body weight data to improve management results in patients with CHF.

Conclusion
Although significant structural and functional differences were found between patients with SHF and those with DHF, elevated diastolic pressures play a pivotal role in the underlying pathophysiology of chronic compensated and acute decompensated heart failure in both SHF and DHF.

Disclosures
Dr Zile, Dr Adamson, Dr Aaron, Dr Abraham, Dr Stevenson, and Dr Bourge have received research grants from Medtronic Inc. Dr Zile, Dr St. John Sutton, Dr Adamson, Dr Aaron, Dr Aranda, Dr Abraham, Dr Stevenson, and Dr Bourge have been consultants for and on the advisory board of Medtronic Inc. Dr Bennett, Dr Cho, and Dr Kueffer have ownership interests in Medtronic Inc. Dr Bennett, Dr Cho, and Dr Kueffer are employees of Medtronic Inc. Dr Adamson, Dr Aaron, Dr Aranda, Dr Abraham, Dr Stevenson, and Dr Bourge have received honoraria from Medtronic Inc. Dr Adamson has received a research grant and honoraria from and been a consultant for and on the advisory board of Sigma Tau. Dr Adamson has been a consultant for and on the advisory board of Cardiomes and Glaxo-Smith-Kline. Dr Aaron has been on the speakers’ bureau for Glaxo-Smith-Kline. Dr Smart has been on the speakers’ bureau for Scios and BioSite and has received honoraria from Scios, BioSite, and Thoratec.

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CLINICAL PERSPECTIVE

The purpose of this study was to examine the pathophysiology of chronic compensated and acute decompensated heart failure (HF) in patients with systolic HF (SHF) versus patients with diastolic HF (DFH). In this study, continuous measurements of intracardiac pressures were made during periods of compensation, during the development of acute decompensation, and after effective treatment returned patients to a compensated state. This study showed that significant structural and functional differences exist between patients with SHF and those with DFH. However, elevated diastolic filling pressures play a pivotal role in the underlying pathophysiology of chronic compensated HF in both SHF and DFH. Acute decompensation in both DFH and SHF patients was preceded by a significant increase in diastolic pressures, which occurred >2 weeks before hospitalization for HF. In contrast, the patients who did not develop acute decompensated HF had no significant changes in diastolic filling pressures. In addition, effective treatment of acute decompensated HF resulted in improved diastolic filling pressures. Intracardiac hemodynamic monitoring may prove to be helpful in preventing and treating acute decompensated HF. Therefore, an increased understanding of the underlying pathophysiology of chronic compensated and acute decompensated HF in both SHF and DFH should allow us to develop more effective treatment and prevention strategies for all HF patients.
Transition From Chronic Compensated to Acute Decompensated Heart Failure. Pathophysiological Insights Obtained From Continuous Monitoring of Intracardiac Pressures

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_Circulation_. published online September 15, 2008;

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/early/2008/09/15/CIRCULATIONAHA.108.783910.citation

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