Serial Assessment of Left and Right Ventricular Filling in Patients With Congestive Heart Failure

Tetsuro Ohta, MD; Satoshi Nakatani, MD*; Shiro Izumi, MD; Seiki Nagata, MD*; Shintaro Beppu, MD*; Kunio Miyatake, MD*

Serial changes in the diastolic filling of both ventricles were studied using Doppler echocardiography in 19 patients with congestive heart failure from the acute to the convalescent stage. During the acute stage, left ventricular early filling velocity (E) was high (88±17 cm/s) and atrial filling velocity (A) was low (44±23 cm/s), whereas the right ventricular E was depressed (17±9 cm/s) and A was enhanced (40±9 cm/s). As the condition improved, left ventricular E decreased (43±11 cm/s, p<0.01) and A increased (59±24 cm/s, p<0.01) along with a decrease in the left ventricular and atrial dimensions. In contrast to the changes in left ventricular filling, right ventricular E increased (31±10 cm/s, p<0.01) and A decreased (32±5 cm/s, p<0.05). There are opposite directional changes in left and right ventricular filling with clinical improvement from the acute to the convalescent stage of congestive heart failure, which suggest that the changes are related to improvement of the hemodynamic conditions of both ventricles. The changes in the right ventricular filling pattern was likely to be related to changes in right ventricular afterload, ventricular interaction and external constraint rather than a change in right ventricular filling pressure. (Jpn Circ J 2001; 65: 803–807)

Key Words: Congestive heart failure; Doppler echocardiography; Left ventricular filling; Right ventricular filling

In congestive heart failure, deterioration of cardiac function and an increase in the left ventricular (LV) filling pressure result in severe pulmonary congestion, which may be fatal unless treatment is effective. Individual assessment of both the LV and right ventricular (RV) loading and function is important for appropriate treatment of heart failure because disproportion performance between the 2 ventricles without proper treatment can lead to lung congestion or low cardiac output:1–3 for example, acute myocardial infarction with or without RV infarction. A number of studies have shown abnormal LV diastolic filling in the setting of heart failure, but assessment of RV function has often been ignored, with only a few studies including RV filling in heart failure.4–8 Further, to the best of our knowledge, there has not been observation or comparison of both LV and RV filling from the acute to the convalescent stage of congestive heart failure.

In the present study, to determine the changes in both LV and RV filling and to further the understanding of hemodynamics and cardiac function in heart failure patients, we performed serial echocardiography on patients with congestive heart failure from the acute to the convalescent stage.

Methods

Clinical Characteristics of the Patients

The study group comprised 19 patients (13 males, 6 females; mean age, 56±13 years, range; 27–78 years) with severe congestive heart failure and sinus rhythm whose deteriorated conditions was clinically considered to be caused mainly by LV disease rather than RV disease (11 had idiopathic dilated cardiomyopathy, 4 had ischemic cardiomyopathy and 4 had hypertensive heart disease). The echocardiographic LV ejection fraction was 25±6% in the acute stage of congestive heart failure. All patients were symptomatic and had a history of increasing dyspnea and orthopnea. Pulmonary venous hypertension and edema were detected by plain chest roentgenogram in all patients. All patients had pretibial edema and 16 of the 19 patients had hepatomegaly. All were treated with diuretics and digitalis; 13 patients were treated with angiotensin-converting enzyme inhibitors and 7 were treated with nitrates; 15 patients required intravenous inotropes (dopamine and/or dobutamine 14 patients; amrinone 3 patients). All patients were hospitalized and studied by serial echocardiography from the acute to the convalescent stage, with a mean follow-up period of 32±27 days. Patients with complete merging of early and late filling velocities of the left ventricle during the acute stage were excluded. Informed consent was obtained from each patient.

Echocardiography

Echocardiographic examinations were performed with a commercially available ultrasound system (Toshiba SSH-140A, 160A, Toshiba Medical Co, Tokyo, Japan) with 3.75- or 2.5-MHz transducer. Left ventricular and atrial dimensions and both ventricular wall thicknesses were measured from M-mode and 2-dimensional echocardiograms; LV fractional shortening and ejection fraction were determined.9,10 Stroke volume was determined as the product of the cross-sectional area of the aortic annulus and time-velocity integral of LV ejection flow.11 The LV and RV filling velocities were recorded with
pulsed Doppler echocardiography by an apical approach together with a phonocardiogram on a strip chart at a paper speed of 100 cm/s. The sample volume was set at the level of the leaflet tips in diastole. Peak velocities of early filling (E) and atrial filling (A) and their ratio (E/A) were obtained. Deceleration time (DT) of the LV early filling wave was measured. LV isovolumic relaxation time (IRT) was measured as the time interval between the aortic valve closure on the phonocardiogram and the beginning of LV filling. The severity of mitral and tricuspid regurgitation was graded semiquantitatively by Doppler color flow mapping.12,13 The measurements were carried out at end-expiration.

In measuring the Doppler variables, interobserver variability (E 2.7±1.9%; A 1.7±1.9%; DT 5.5±3.9%; IRT 4.9±4.8%) and intraobserver variability (E 2.5±1.4%; A 1.1±0.7%; DT 3.8±3.6%; IRT 2.2±2.1%) were estimated.

Hemodynamic Measurements
Blood pressure was measured by standard sphygmomanometer just before each echocardiographic examination. Central venous pressure was measured via a central venous line inserted for drug infusion at the acute and convalescent stages. To assess RV function, the RV ejection fraction was measured by first-pass radionuclide ventriculography in 11 patients in the convalescent stage. The LV ejection fraction was assessed after the RV measurement by equilibrium-gated radionuclide ventriculography. We also estimated pulmonary artery pressure from the peak velocity of tricuspid regurgitation.

Statistics
Data were expressed as mean±SD. Comparison of measurements at the acute and the convalescent stage was done with the Student’s paired t test. Comparison of New York Heart Association (NYHA) functional class and the severity of mitral or tricuspid regurgitation were done with the Wilcoxon test. A p value less than 0.05 was considered significant.

Results

Hemodynamic Changes
In the acute stage, all patients were in NYHA class IV, but by the convalescent stage, after a mean of 32 days of treatment, the functional class had significantly improved (12 patients in NYHA class III, 7 patients in NYHA class II, p<0.01). Mean blood pressure and central venous pressure significantly decreased (95±10 vs 89±11 mmHg, p<0.01 for mean blood pressure; 9.5±2.3 vs 3.7±2.4 mmHg, p<0.01, for central venous pressure), but the heart rate did not change significantly (80±15 vs 72±8 beats/min, p=NS) in the convalescent stage. The RV ejection fraction was 46±9% and the LV ejection fraction was 26±8% as assessed by radionuclide ventriculography in the convalescent stage. We were able to estimate pulmonary artery pressure from the peak velocity of tricuspid regurgitation in 6 of 19 patients in the acute stage, and in 4 of 19 patients in the convalescent stage. The peak pulmonary artery pressure decreased significantly (from 51±18 to 21±4 mmHg, p<0.05).

Systolic Function and Chamber Size (Table 1)
In the acute stage, the left ventricle and atrium were dilated and the ejection fraction was poor. With improvement in clinical symptoms, the cardiac size reduced significantly with a significant increase in cardiac output. LV fractional shortening and the ejection fraction did not change significantly. The ventricular wall thicknesses were measured in the convalescent stage: LV anterior wall thickness decreased, and LV posterior wall thickness increased. The LV interventricular septum and RV thicknesses did not change significantly. The LV posterior wall thicknesses were measured in the convalescent stage: RV anterior wall thickness decreased, and RV posterior wall thickness increased.

Table 1 Cardiac Output and Echocardiographic Findings From the Acute to the Convalescent Stage of Congestive Heart Failure

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Acute Stage</th>
<th>Convalescent Stage</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO (L/min)</td>
<td>2.6±0.6</td>
<td>3.3±0.6</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVDd (mm)</td>
<td>71±8</td>
<td>68±9</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVDs (mm)</td>
<td>63±9</td>
<td>60±11</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVFS (%)</td>
<td>11±4</td>
<td>12±6</td>
<td>NS</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>25±6</td>
<td>27±10</td>
<td>NS</td>
</tr>
<tr>
<td>LAD (mm)</td>
<td>44±5</td>
<td>39±6</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

A, acute stage; C, convalescent stage; CO, cardiac output; LVDd, left ventricular diastolic dimension; LVDs, left ventricular systolic dimension; LVFS, left ventricular fractional shortening; LVEF, left ventricular ejection fraction; LAD, left atrial dimension.

Fig. 1. Serial ventricular inflow patterns in a 27-year-old man with dilated cardiomyopathy. (Upper) Changes in LV filling. During severe congestive heart failure, high velocity of early filling (E), low velocity of atrial filling (A) and extremely short isovolumic relaxation time and deceleration time, representing a restrictive pattern, were observed. As the patient’s clinical condition improved, E decreased, A increased and isovolumic relaxation time and deceleration time were gradually prolonged. The pattern changed from a restrictive pattern to a pseudonormal pattern and then to an abnormal relaxation pattern. (Lower) Changes in RV filling. In contrast to LV filling, E was low and A was high during severe congestive heart failure. As the clinical condition improved, E increased and A decreased gradually.
Biventricular Filling in CHF

Japanese Circulation Journal  Vol.65, September 2001

Changes in the Doppler Filling Parameters From the Acute Stage to the Convalescent Stage

Both the LV and RV filling patterns changed dramatically from the acute to the convalescent stage (Fig 1), although, interestingly, the direction of the changes was opposite. In the acute stage, LV filling showed pseudonormal or restrictive patterns, whereas RV filling showed an abnormal relaxation pattern. With improvement in the patient's clinical condition, the LV E decreased significantly (43±11 cm/s, p<0.01), A increased significantly (59±24 cm/s, p<0.01), E/A decreased significantly (0.82±0.36, p<0.01) and the IRT and DT were prolonged significantly (IRT 104±18 ms, p<0.01; DT 200±47 ms, p<0.01). Abnormal relaxation patterns were shown in the convalescent stage. In contrast, the RV filling pattern changed in the opposite direction: E increased significantly (31±10 cm/s, p<0.01); A decreased significantly (32±5 cm/s, p<0.05) and E/A increased significantly (0.97±0.28, p<0.01), approaching an almost normal pattern. These changes in LV and RV filling were consistent in all patients (Figs 2, 3).

Discussion

This study is the first to our knowledge to evaluate both LV and RV filling serially from the acute to the convalescent stage of severe congestive heart failure. LV filling, which initially showed a restrictive pattern, changed to an abnormal relaxation pattern as the clinical condition improved. In contrast, the abnormal relaxation pattern of RV filling in the acute stage changed to a normal pattern with clinical improvement.

Mechanism of Changes in the LV Diastolic Filling Pattern

In the acute stage of severe congestive heart failure, LV filling invariably showed a restrictive or pseudonormal pattern, which is usually observed when the LV filling pressure is extremely high, with severe diastolic dysfunction. After clinical improvement, the filling pattern changed to an abnormal relaxation pattern. Multiple factors are reported to affect the diastolic filling pattern, but the main factors are loading conditions and diastolic function. The decrease in the left atrial and ventricular dimensions and mitral regurgitation after improvement of congestive heart failure suggests a decrease in LV filling pressure. Therefore, the changes in the LV filling pattern could be explained mainly by a change in the LV filling pressure. An elevated filling pressure in the acute stage would mask the existence of abnormal LV relaxation. The other possible mechanism of this change would be an improvement in LV diastolic function. All patients had left heart disease leading to pulmonary congestion. With appropriate treatment, diastolic dysfunction can be improved to some extent and, moreover, a decrease in LV size may shift the pressure-volume loop to the left on the same diastolic pressure-volume relationship (along the same exponential line), causing an increase in the effective compliance, which indicates that the ventricle becomes more compliant, resulting in more normal ventricular filling.

Mechanism of Changes in the RV Diastolic Filling Pattern

The RV filling pattern initially showed an abnormal relaxation pattern. It has been reported experimentally and clinically that a high afterload impairs early filling. In the acute stage of congestive heart failure, pulmonary arterial pressure and pulmonary vascular resistance would be significantly elevated and this increase in RV afterload may be one cause of the abnormal relaxation patterns seen in the acute stage. In contrast to the change in the LV filling pattern, the depressed RV early filling increased and the enhanced atrial filling decreased with clinical improvement. The central venous pressure decreased and the severity of tricuspid regurgitation decreased after the improvement. A decrease in ventricular filling pressure should result in a decrease in the early filling wave. Thus, differing from LV filling, the changes in the RV filling pattern can not be explained by a change in filling pressure. Ventricular interaction mediated through the septum and modulated by the pericardium could be one factor because alterations in the
failing pattern in one side of the heart influence that in the other side. It has been reported that the filling of both ventricles is restrained by the pericardium over a wide range of physiological ventricular volumes, and that the right ventricle is more susceptible to the influence of the pericardium. In patients with severe heart failure, RV filling may be easily influenced by the pericardium and the septum because of the enlarged left ventricle and left atrium. The impaired relaxation of the right ventricle because of these external constraints should be alleviated with a decrease in the left-sided heart chamber size. Thus, the RV filling pattern observed in the present study can be explained by an increased afterload, ventricular interaction and pericardial constraint at the acute stage and by their alleviation in the convalescent stage.

Clinical Implications

Although Doppler indexes of ventricular diastolic filling are only indirect measures of diastolic function, they are still useful to assess global diastolic performance. In the present study, we showed dramatic changes in both the LV and RV filling in the course of recovery of congestive heart failure. The changes in filling patterns can be understood mainly by considering loading conditions. Therefore, the present results provide additional information for the estimation of a patient's hemodynamic condition and assessment of the patient's status in the course of recovery from congestive heart failure with severe LV disease.

Study Limitations

As discussed earlier, multiple factors affect ventricular filling patterns. Because of the lack of simultaneous invasive hemodynamic measurements and Doppler examinations, however, these factors could not be assessed directly in this study.

Congestive heart failure is to some extent accompanied by right-sided heart failure. Severely impaired RV systolic and diastolic function with high right atrial pressure may cause a pseudonormal or restrictive RV filling pattern, but such a pattern was not observed in this study. The ejection fraction reflects global ventricular function. The poor LV ejection fraction with preserved RV ejection fraction during the convalescent stage (RV ejection fraction by radionuclide ventriculography: 46±9%; LV ejection fraction by radionuclide ventriculography: 26±8%; LV ejection fraction by echocardiography: 27±10%) suggests that left heart disease was the main problem and that RV function was preserved in the patients in this study. Because of the preserved RV function and mildly elevated RV filling pressure, patients in the present study did not reveal a restrictive filling pattern in the right ventricle.

In this study, we investigated 19 patients who had recovered from the acute stage of severe congestive heart failure. This study is limited by the small number of patients and further investigation of larger numbers of patients will be needed to determine if changes in ventricular filling patterns can predict prognosis or if they can assess effects of treatment for congestive heart failure.

Conclusions

In the acute stage of severe congestive heart failure, the LV and RV filling velocities showed characteristic patterns. LV filling had a pseudonormal or restrictive pattern whereas RV filling had an abnormal relaxation pattern. From the acute to the convalescent stage, the LV and RV filling patterns changed in opposing directions. In the convalescent stage, LV E decreased and A increased whereas RV E increased and A decreased. These changes were accompanied by clinical improvement. Assessment of not only LV filling but also RV filling would provide a better understanding of the patient's hemodynamic condition.

References

ventricle in heart and disease: A study by pulsed Doppler technique. *Jpn Circ J* 1982; **46**: 92–102