Biventricular diastolic behaviour in patients with hypertrophic and hereditary hemochromatosis cardiomyopathies

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Received 27 May 2003; received in revised form 24 December 2003; accepted 5 January 2004

Abstract
Aim To define biventricular diastolic behaviour in patients with cardiomyopathies with predominant diastolic left ventricular (LV) dysfunction.

Methods and results Doppler tissue echocardiography and both mitral and tricuspid Doppler inflow profiles were investigated in hypertrophic (n = 17), hereditary hemochromatosis (n = 12) cardiomyopathies and age-matched normals (n = 31). The cardiomyopathy group had both lower early diastolic mitral lateral annular (E\textsubscript{l}), cm/s (13 \pm 9 vs. 16 \pm 6.5) and medial (E\textsubscript{m}) (10.0 \pm 4.5) velocities compared with normals (19.5 \pm 5.5, 15.9 \pm 3.4, p < 0.01, respectively). In the cardiomyopathy group, late isovolumic relaxation myocardial velocity gradient (IVR-MVG) (s\textsuperscript{-1}) was positive compared with negative in normals (1.3 \pm 1.3 vs. –0.7 \pm 1.4, p < 0.01, respectively). In both the cardiomyopathy group and in normals the onset of the tricuspid E-wave preceded the onset of the mitral E-wave. However, the onset of early diastolic tricuspid annular (E\textsubscript{t}) motion preceded the onset of E\textsubscript{l} (ms) only in normals, but not in the cardiomyopathies (43 \pm 26 vs. –8 \pm 44, p < 0.01, respectively). In the cardiomyopathy group there was a positive correlation between the onset of E\textsubscript{t} and abnormally positive late IVR-MVG (r = 0.51, p = 0.002).

Conclusions Biventricular early diastolic behaviour is abnormal in the selected group of cardiomyopathy patients. The delay in the E\textsubscript{t} (early diastolic longitudinal right ventricular relaxation) may have a negative effect on LV diastolic function.

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Introduction

Recent studies have shown that cardiac resynchronization therapy is a promising new therapeutic approach in patients with severe heart failure due to left ventricular (LV) systolic dysfunction and intraventricular conduction delay. Either simultaneous or sequential with individualized interventricular delay programming biventricular stimulation improved both patients’ symptoms and LV systolic performance. In addition, previous studies have shown the importance of atrioventricular adjustment to optimize hemodynamic response in patients with heart failure. However, little data exist on diastolic biventricular mechanical behaviour and it is unknown whether ‘similar’ diastolic resynchronization therapy may bring any benefit in patients with symptoms of predominant diastolic heart failure. It has been suggested that in some patients with predominant systolic LV dysfunction the right ventricle (RV) is dilated such that LV filling is impeded by the surrounding RV and pericardium. Diastolic biventricular interaction may thereby contribute to increased symptoms due to raised intracavitary filling pressures (secondary to increased external force) and impaired ability to augment stroke volume. A similar effect of RV volume related ventricular interaction on LV diastolic behaviour can also be observed in patients with constrictive pericarditis and in patients with pulmonary embolism. However, potential mechanical asynchrony between RV relaxation and LV diastolic behaviour in patients with diastolic dysfunction is unknown. Therefore, the aim of this study was (i) to determine whether biventricular diastolic mechanical behaviour is abnormal in a selected group of cardiomyopathies with predominant diastolic dysfunction (hypertrophic—HCM, and hereditary hemochromatosis—HHC), and (ii) whether there are links between potentially abnormal RV relaxation and LV diastolic function. For the purpose of this study, combined information derived from conventional echocardiographic studies of mitral and tricuspid Doppler inflow profiles and Doppler tissue echocardiography of both longitudinal mitral and tricuspid annular velocities and circumferential LV velocities were studied.

Methods

Our study group consisted of 60 subjects; including patients with HCM (n = 17), patients with HHC (n = 12), and 31 age-matched normal subjects. Patients with impaired LV systolic function (LV ejection fraction ≤50%), left or right bundle branch blocks, resting LV outflow tract obstruction (≥10 mmHg), atrial fibrillation, systemic or pulmonary hypertension, clinical evidence of coronary artery disease, and/or significant (grade ≥2) valvular disease were not included into the study. The study group was formed prospectively between 1999 and 2001 at the Prince Charles Hospital, Brisbane, Australia. The study was approved by The Prince Charles Hospital Research Ethics committee and prior to the examination written consent was obtained from all subjects. The diagnosis of HCM was based on the presence of unexplained LV hypertrophy. In all HHC patients the initial diagnosis was based on an elevated transferrin saturation and serum ferritin level. Ten HHC patients were Cys282Tyr homozygotes, while 2 were compound heterozygotes (Cys282Tyr/His63Asp). Age-matched healthy normals were selected from the local community. Each subject underwent a standard echocardiographic assessment (M-mode, 2-dimensional, Doppler blood-flow measurements) and Doppler tissue echocardiographic study using an Acuson ultrasound scanner (Sequoia, California, USA) with a variable frequency transducer (2.5–4.0 MHz). All echocardiographic measurements were averaged over 3 cardiac cycles. Two-dimensional, 4-chamber apical view was used to record lateral mitral, septal (medial) and lateral tricuspid annular velocities. Peak annular velocities were measured in both early diastole (mitral lateral—Eₐ, mitral medial—Eₘ, tricuspid—Eₙ) and in late diastole. Using parasternal color M-mode Doppler tissue echocardiography, LV posterior wall myocardial velocity gradient (MVG) was analyzed. MVG was measured in early diastole during IVR, in rapid ventricular filling (RVF), and in late diastole during atrial contraction. IVR period was divided into first half (early IVR) and second half.
(late IVR). Additionally, timings between the onset of electrocardiographic Q-wave and (1) mitral and tricuspid inflow velocity (the onset and the peak of E-wave and A-wave), (2) annulus velocities (the onset and the peak of the $E_1$, $E_m$, $E_t$, and late diastolic-lateral mitral, sepal [medial] and lateral tricuspid velocities, and (3) MVG (the peak during early IVR, late IVR, RVF and atrial contraction) were measured (Fig. 1). To present and to illustrate our complex data, consecutive diastolic events observed on either left or right side of the heart were added together to form one cycle. The events separated by less than 15 ms apart were combined and constituted the first diastolic sequence. Next diastolic sequences (from 2 to 9) were created in a similar fashion starting from the following consecutive diastolic event when the time was longer than 15 ms between two consecutive events.

**Statistical analysis**

All data are expressed as mean value ± SD for continuous variables and as frequency number (%) for dichotomous variables. ANOVA with Scheffe’s $F$ adjustment for multiple comparisons was used to assess the differences between HCM, HHC, and age-matched normals. Unpaired and paired $t$-tests were used when appropriate. Univariate and multivariate logistic regressions were used to assess potential relationships of LV hypertrophy, LV dimensions, LV volumes and other echocardiographic measurements including Doppler tissue echocardiographic indexes. In addition, possible relationships between echocardiographic measurements derived from RV and LV were examined. A value of $p < 0.05$ was considered significant.

**Results**

The cardiomyopathy group had lower $E_t$ and $E_m$ velocities compared with normal subjects (Table 2). Both $E_t$ and $E_m$ velocities were similarly low in the HCM and HHC subgroups. Although $E_t$ velocities were lower in the cardiomyopathy group compared with normal subjects, only in the HHC
subgroup $E_t$ velocities were significantly reduced. All late diastolic annulus velocities were similar in the cardiomyopathy group compared with normal subjects. In the cardiomyopathy group as well as in both subgroups (HCM, HHC) late IVR-MVG (s$^{-1}$) was positive ($1.3 \pm 1.3$, $1.0 \pm 1.6$, $1.6 \pm 0.9$, respectively) compared with negative in normal subjects ($-0.7 \pm 1.4$), $p<0.01$. MVGs measured during both RVF and atrial contractions were lower only in HCM but not in HHC subgroup compared with normal subjects. In both cardiomyopathy subgroups and in normal subjects 9 consecutive sequences were defined (Table 3). The differences between cardiomyopathies and normal subjects in annular motion and MVG peak values were present only in early diastolic sequences (1–5), but not in late diastolic sequences (6–9). In the cardiomyopathy group, the onsets of $E_t$, $E_m$, and $E_l$ were all noted later (sequence 3) than in normal subject (sequences 1 and 2). Also, in the cardiomyopathy group, the peak of $E_t$, and $E_l$ were found in sequences 5 and 4 compared with 4 and 5 in the normal subjects group. As a consequence, the peak of tricuspid E-wave was delayed in the cardiomyopathy group and noted in sequence 5 compared with sequence 4 in normal subjects group. Both subgroups HCM and HHC had similar sequences and their timing events (Table 4). In both the cardiomyopathy group and in normal subjects the onset of the tricuspid E-wave preceded the onset of the mitral E-wave (ms) by 40 $\pm$ 33, and 54 $\pm$ 29, respectively ($p=NS$). Similarly, in both cardiomyopathy subgroups (HCM, HHC) the onset of the tricuspid E-wave preceded the onset of the mitral E-wave (ms) by 41 $\pm$ 37, and 40 $\pm$ 28, respectively ($p=NS$). However, the onset of $E_t$ preceded the onset of $E_l$ (ms) only in normal subjects ($43 \pm 26$).

![Figure 1](image)

**Table 2** Doppler tissue echocardiographic data of study subjects

<table>
<thead>
<tr>
<th></th>
<th>All cardiomyopathies</th>
<th>HCM</th>
<th>HHC</th>
<th>Normals</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Annulus velocity (cm/s)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$E_l$</td>
<td>13.9 $\pm$ 6.5*</td>
<td>12.6 $\pm$ 4.8*</td>
<td>15.8 $\pm$ 8.2*</td>
<td>19.5 $\pm$ 5.5</td>
</tr>
<tr>
<td>$E_m$</td>
<td>10.0 $\pm$ 4.5*</td>
<td>9.6 $\pm$ 4.9*</td>
<td>10.6 $\pm$ 4.1*</td>
<td>15.9 $\pm$ 3.4</td>
</tr>
<tr>
<td>$E_t$</td>
<td>15.9 $\pm$ 5.5#</td>
<td>15.9 $\pm$ 6.3*</td>
<td>16.0 $\pm$ 4.3*</td>
<td>18.4 $\pm$ 2.8</td>
</tr>
<tr>
<td>Late diastolic mitral</td>
<td>15.7 $\pm$ 6.5</td>
<td>15.0 $\pm$ 7.0</td>
<td>16.8 $\pm$ 6.2*</td>
<td>14.8 $\pm$ 5.5</td>
</tr>
<tr>
<td>Late diastolic medial</td>
<td>10.9 $\pm$ 5.5</td>
<td>10.2 $\pm$ 6.9</td>
<td>12.0 $\pm$ 2.7</td>
<td>13.3 $\pm$ 4.5</td>
</tr>
<tr>
<td>Late diastolic tricuspid</td>
<td>16.0 $\pm$ 7.1</td>
<td>15.0 $\pm$ 7.9</td>
<td>17.4 $\pm$ 6.0</td>
<td>15.5 $\pm$ 6.1</td>
</tr>
<tr>
<td><strong>Myocardial velocity gradient (s$^{-1}$)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Early IVR</td>
<td>$-0.8 \pm 0.7$</td>
<td>$-0.6 \pm 0.7$</td>
<td>$-1.1 \pm 0.6$</td>
<td>$-1.0 \pm 0.7$</td>
</tr>
<tr>
<td>Late IVR</td>
<td>1.3 $\pm$ 1.3#</td>
<td>1.0 $\pm$ 1.6*</td>
<td>1.6 $\pm$ 0.9*</td>
<td>$-0.7 \pm 1.4$</td>
</tr>
<tr>
<td>Rapid ventricular filling (RVF)</td>
<td>5.1 $\pm$ 2.9#</td>
<td>4.1 $\pm$ 1.9*</td>
<td>6.4 $\pm$ 3.6</td>
<td>7.5 $\pm$ 3.8</td>
</tr>
<tr>
<td>Atrial contraction</td>
<td>1.8 $\pm$ 1.7#</td>
<td>1.4 $\pm$ 1.5*</td>
<td>2.3 $\pm$ 1.8</td>
<td>3.1 $\pm$ 2.4</td>
</tr>
</tbody>
</table>

For abbreviations see Table 1. $E_l$, $E_m$, $E_t$, early diastolic annulus velocity (mitral, medial, tricuspid, respectively).

*p<0.01 compared to normals; #p<0.05 compared to normals.
but not in the cardiomyopathy group and both subgroups HHC and HHC (−8 ± 44, −12 ± 53, −3 ± 28, respectively, \( p < 0.01 \) compared with normal subjects). In parallel, the peak \( E_t \) and the peak of the tricuspid E-wave preceded the peak \( E_t \) and the peak of the mitral E-wave only in normal subjects. In normal subjects, the difference in time (ms) between the peak \( E_t \) and the peak \( E_l \) was 20 ± 22, and the timing difference between the peak of the tricuspid E-wave and the peak of the mitral E-wave was 30 ± 23. In the cardiomyopathy group, and both subgroups HCM and HHC, the situation was opposite. The timing difference (ms) between the peak \( E_t \) and the peak \( E_t \) was −19 ± 44 for the whole group, −21 ± 52 for HCM, and −15 ± 31 for HHC. The differences between peak tricuspid E-wave and peak mitral E-wave were −12 ± 26 (whole cardiomyopathy group), −13 ± 31 (only HCM), and −11 ± 17 (only HHC), \( p < 0.01 \) compared with normal subjects. The timing differences in late diastolic tricuspid and mitral annulus velocities or tricuspid and mitral A-wave velocities were.

### Table 3: Diastolic timing sequences of blood inflow velocities (mitral and tricuspid), annulus velocities (lateral, medial and tricuspid) and LV myocardial velocity gradient (MVG) in patients with cardiomyopathies and in normal subjects

<table>
<thead>
<tr>
<th>Sequence Number</th>
<th>Sequence Time (mean ± SD)</th>
<th>Events</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Early IVR-MVG</td>
<td>420 ± 42</td>
</tr>
<tr>
<td>2</td>
<td>Late IVR-MVG</td>
<td>457 ± 40</td>
</tr>
<tr>
<td>3</td>
<td>Onset of mitral E-wave</td>
<td>473 ± 39</td>
</tr>
<tr>
<td>4</td>
<td>RVF-MVG</td>
<td>509 ± 46</td>
</tr>
<tr>
<td>5</td>
<td>Peak ( E_m )</td>
<td>539 ± 42</td>
</tr>
<tr>
<td>6</td>
<td>Onset late diastolic</td>
<td>834 ± 154</td>
</tr>
<tr>
<td>7</td>
<td>Onset of tricuspid</td>
<td>850 ± 170</td>
</tr>
<tr>
<td>8</td>
<td>Peak late diastolic</td>
<td>888 ± 162</td>
</tr>
<tr>
<td>9</td>
<td>Peak of mitral A-wave</td>
<td>902 ± 162</td>
</tr>
</tbody>
</table>

**For abbreviations see Tables 1 and 2.**

There was no significant difference between the timings (ms) of the sequence number 1 in the cardiomyopathy group compared with normal subjects (420 ± 42 vs. 410 ± 30, \( p = NS \)). The timing differences (ms) between two consecutive sequences (1 vs. 2; 2 vs. 3; 3 vs. 4; 4 vs. 5; 5 vs. 6; 6 vs. 7; 7 vs. 8; 8 vs. 9) were as follows: for the cardiomyopathy group: 35 ± 26, 18 ± 24, 37 ± 32, 30 ± 39, 294 ± 127, 16 ± 53, 37 ± 39, 14 ± 62; for normal subjects: 39 ± 19, 15 ± 20, 25 ± 20, 23 ± 16, 269 ± 119, 33 ± 52, 15 ± 34, 36 ± 43 (\( p < 0.05 \) for all).
were similar for the cardiomyopathy group and normal subjects. Multivariate logistic regression analysis showed that, apart from the peak $E_m$ velocity in HCM patients, other Doppler tissue echocardiographic indices (peak values and timings) were independent of the degree of LV hypertrophy or LV dimension/volume. Among the echo-derived measurements obtained either from RV and LV, a significant relationship was present only between the time of the onset of $E_t$ and late IVR-MVG. In the cardiomyopathy group there was a positive correlation between the onset of $E_t$ and abnormally positive late IVR-MVG ($r = 0.51, p = 0.002$). In contrast, in normal subjects this correlation was weaker but negative ($r = -0.36, p = 0.025$, Fig. 2). Fig. 3 shows schematically the differences in consecutive early diastolic sequences (from 1 to 5) and the differences in Doppler echocardiographic indices in the cardiomyopathy and in normal subject groups.

**Table 4** Diastolic timing sequences of blood inflow velocities (mitral and tricuspid), annulus velocities (lateral, medial, and tricuspid) and LV myocardial velocity gradient (MVG) in patients with HCM and HHC

<table>
<thead>
<tr>
<th>Sequence</th>
<th>Number</th>
<th>Time (mean ± SD)</th>
<th>HCM (Q–Q interval 926 ± 213 ms)</th>
<th>Events</th>
<th>HHC (Q–Q interval 935 ± 150 ms)</th>
<th>Time (mean ± SD)</th>
<th>Events</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Early IVR-MVG</td>
<td>415 ± 48</td>
<td>1</td>
<td>Early IVR-MVG</td>
<td>426 ± 30</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Late IVR-MVG</td>
<td>452 ± 43</td>
<td>2</td>
<td>Late IVR-MVG</td>
<td>465 ± 36</td>
<td>2</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Onset of mitral E-wave</td>
<td>471 ± 46</td>
<td>3</td>
<td>Onset of mitral E-wave</td>
<td>475 ± 29</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>RVF-MVG</td>
<td>513 ± 55</td>
<td>4</td>
<td>RVF-MVG</td>
<td>504 ± 30</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Peak $E_l$</td>
<td>537 ± 50</td>
<td>5</td>
<td>Peak $E_l$</td>
<td>542 ± 31</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Peak $E_m$</td>
<td>834 ± 154</td>
<td>6</td>
<td>Peak $E_m$</td>
<td>834 ± 160</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Peak of mitral E-wave</td>
<td>847 ± 183</td>
<td>7</td>
<td>Peak of mitral E-wave</td>
<td>854 ± 160</td>
<td>7</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Peak of tricuspid E-wave</td>
<td>894 ± 171</td>
<td>8</td>
<td>Peak of tricuspid E-wave</td>
<td>879 ± 154</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Peak of mitral A-wave</td>
<td>890 ± 160</td>
<td>9</td>
<td>Peak of mitral A-wave</td>
<td>919 ± 171</td>
<td>9</td>
<td></td>
</tr>
</tbody>
</table>

For abbreviations see Tables 1 and 2.

**Discussion**

In HCM, due to the presence of myocardial fibers disarray and fibrosis LV diastolic dysfunction is a common feature even at an early stage of disease progression. Also, in HHC iron-catalyzed myocardial injury leads to diastolic dysfunction affecting predominantly LV subepicardial layers. It has been shown that in patients with severe LV systolic dysfunction Doppler tissue echocardiography can be used to select optimum interventricular delay during cardiac systolic resynchronization during biventricular pacing. In order to understand whether cardiac diastolic resynchronization therapy may also be of help in the treatment of diastolic dysfunction, consecutive sequences of diastolic events relating to both RV and LV diastole and mechanical biventricular diastolic asynchrony in the selected group of cardiomyopathies with
predominantly diastolic dysfunction (HCM and HHC patients) have been defined.

**Findings in the current study**

In agreement with previous studies by others in HCM, we found that in HHC, similar to HCM, LV early diastolic abnormalities can be demonstrated as shown by a reduction in longitudinal $E_l$ and $E_m$ velocities.\textsuperscript{17,28,30} The most likely explanation is that in the studied group of cardiomyopathies active longitudinal LV relaxation is impaired. In addition, we have found that the onset of $E_t$, $E_m$ and $E_l$, and the peak of $E_t$ were delayed in time. In normal subjects, the mitral lateral and medial annular motion ($E_l$ and $E_m$ velocities) commenced before the opening of the mitral valve in late IVR and their peak motion was observed before the peak of the mitral E-wave (difference 19 ± 17 ms, $p<0.01$). In contrast, in the cardiomyopathy group the onset

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**Figure 2** Relation between the onset of $E_t$ and IVR-MVG (a) cardiomyopathies, (b) normal subjects. Filled circles, HCM; empty circles, HHC. Thin solid lines indicate regression line and thin dotted lines indicate the 95% predictive interval.
of $E_t$ and $E_m$ were simultaneous with the onset of mitral early diastolic velocity (E-wave) and also the peak of $E_m$ was simultaneous with the peak of E-wave. In addition, only in normal subjects, did the onset and the peak of $E_t$ and the peak of tricuspid E-wave precede parallel diastolic events in the LV (the onset and the peak of $E_t$ and the peak of mitral E-wave). In HCM and HHC patients, these events were parallel except for the peak of $E_t$, which appeared after the peak of $E_t$. In addition in cardiomyopathy group, but not in normal subjects, the onset of tricuspid E-wave preceded $E_t$, which emphasized abnormal RV relaxation and suggests presence of passive RV filling. Both, HCM and HHC had abnormally positive circumferential LV late IVR-MVG. In our previous work, we postulated
that this parameter may be used as a new marker of abnormal diastolic performance.\textsuperscript{21,22,30} The presence of positive late IVR-MVG indicates a decrease or even a lack of LV wall circumferential subepicardial motion during the late IVR phase. During IVR phase, the mitral and aortic valves are closed. Therefore the abnormally positive IVR-MVG is directly related to active LV circumferential wall thinning rather than physiological thickening. In contrast to HHC patients, MVG was significantly reduced during RVF and in late diastole during an atrial contraction in the HCM patients. These abnormalities may be explained by the presence of both abnormal myocardial relaxation\textsuperscript{26} and an increase in passive LV stiffness in HCM patients.\textsuperscript{6} As previously discussed, the LV damage begins from the subepicardial layers in HHC. However, as LV subepicardial layers extend and wrap around the RV, this may explain why HHC patients have reduced RV longitudinal motion as reflected in this study by significantly lower $E_t$. This is further illustrated by the presence of a positive correlation between abnormally positive late IVR-MVG and the delay in $E_t$. Conceptually this means that the pathological circumferential LV wall thinning due to subendocardial motion (rather than physiological thickening due to subepicardial motion) during late IVR may have a negative impact on early diastolic longitudinal RV relaxation (Fig. 4). Until now, a possible link between LV changes during IVR (late IVR-MVG) and RV active relaxation was unknown. We speculate that the described abnormalities in RV relaxation, as a rebound effect, may have an independent effect on LV early diastolic behaviour and contribute to LV diastolic dysfunction.

Limitations

Pre-load dependent changes may potentially have affected Doppler tissue echocardiographic

\begin{figure}
\centering
\includegraphics[width=\textwidth]{figure4}
\caption{Schematic diagrams show anatomical (dots) and functional link between subepicardial left ventricular (LV) myocardial fibers and right ventricular (RV) myocardial fibers in early diastole during LV isovolumic relaxation (IVR) period in healthy normals (a) and in patients with cardiomyopathy (b). Horizontal arrows indicate direction of subepicardial circumferential LV velocities (positive late IVR myocardial velocity gradient (MVG)); cross horizontal arrows indicate lack of these velocities. Vertical arrow indicates direction of longitudinal RV velocities (early diastolic tricuspid annulus velocities ($E_t$)); cross vertical arrow indicates lack of these velocities during IVR period. The onset of $E_t$ was “shift” from IVR period to LV filling phase. RA, right atrium; LA, left atrium.}
\end{figure}
measurements. However, this potential influence will be less pronounced in patients with abnormal myocardial relaxation as compared to healthy myocardium. Both blood pressure and heart rate can potentially influence echo/Doppler measurements. However, in our study there were no significant differences in these measurements. Previous reports showed that Doppler tissue echocardiographic indices are age-related, and therefore the group of normal subjects was carefully age-matched. We did not use hemodynamic data to analyze LV/RV diastolic parameters. However, previous study by others showed a relatively flat dynamic LV diastolic pressure/volume relationship in HCM patients. They concluded that not only right-heart loading but also other maneuvers to reduce biventricular diastolic interaction might hold the promise of substantial benefit to this group of patients. Because electrical asynchrony may affect mechanical biventricular diastolic behaviour, patients with conduction abnormalities were excluded from the current study. Prior attempts to assess biventricular behaviour have met with limited success, thwarted by the low temporal resolution of angiographic, nuclear, or magnetic resonance imaging systems. In order to minimize potential errors in timing measurements, we have tried to obtain as high as possible temporal resolution of Doppler echocardiographic images (between 2 and 8 ms). Although our study group of cardiomyopathies was carefully selected (e.g. patients with conduction abnormalities or pulmonary hypertension were excluded), we did not examine other cardiomyopathies or hypertensive patients.

References


