Left ventricular vortex formation is unaffected by diastolic impairment

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Left ventricular vortex formation is unaffected by diastolic impairment. Am J Physiol Heart Circ Physiol 303: H1255–H1262, 2012. First published September 7, 2012; doi:10.1152/ajpheart.00093.2012.—Normal left ventricular (LV) filling occurs rapidly early in diastole caused by a progressive pressure gradient within the ventricle and with a low left atrial pressure. This normal diastolic function is altered in patients with heart failure. Such impairment of diastolic filling is manifested as an abrupt deceleration of the early filling wave velocity. Although variations within the early filling wave have been observed previously, the underlying hydrodynamic mechanisms are not well understood. Previously, it was proposed that the mitral annulus vortex ring formation time was the total duration of early diastolic filling and provided a measure of the efficiency of diastolic filling. However, we found that the favorable LV pressure difference driving early diastolic filling becomes zero simultaneously with the deceleration of the early filling wave propagation velocity and pinch-off of the LV vortex ring. Thus we calculated the vortex ring formation time using the duration of the early diastolic filling wave propagation velocity and pinch-off of the LV vortex ring. We hypothesized that the LV vortex ring formation is unaffected by diastolic impairment.

IN ORDER FOR THE HEART to normally function, the left ventricle (LV) must not only eject blood during systole but rapidly fill during diastole without requiring an elevated left atrial pressure both at rest and when the cardiac output increases during exercise. In a healthy heart this is accomplished by the LV functioning as a suction pump. Early in diastole there is a progressive pressure difference that pulls blood from the left atrium to the LV apex (8, 42). In LV diastolic dysfunction (LVDD), the ability of the heart to relax and expand decreases, negatively impacting the left heart’s capacity to fill and generate a favorable pressure difference from the left atrium to the LV during diastole. In patients with LVDD, maintenance of LV filling requires an elevated left atrial pressure (16, 25), which becomes especially apparent during exercise (6, 33). Almost all patients with heart failure have abnormalities of the LV filling dynamics, and over 50% of patients with heart failure have preserved ejection fraction (17). Therefore, improved understanding of LVDD fluid dynamics may contribute to the diagnosis and treatment across all heart failure conditions (30).

During early diastolic filling as blood flows across the mitral valve from the left atrium into the LV, the inflow jet produces a vortex ring. This vortical structure formed within the LV has been studied both in vivo and in vitro (1, 10, 18, 19, 22, 23). The strength of the vortex ring continues to increase until the vortex ring is pinched off; this instant is defined as the vortex ring formation time (FT). The strength of the vortex ring at the time of pinch-off, when it has reached its maximum value, is either achieved because the inflow jet is terminated or the vortex ring reaches its energetic limit (13). At this point, the primary vortex ring detaches from the inflow jet and pinches off. After the vortex ring is pinched off, it is impossible for additional energy to strengthen the vortex ring. Vortex ring formation within the LV inflow tract is predicted to improve LV filling efficiency and has been investigated as a possible metric of cardiac function (12, 23, 31). Gharib et al. (13) hypothesized that a universal timescale may be present for the FT of optimal LV filling by a vortex ring within the ventricle.

For a vortex ring generated by a piston-cylinder arrangement within a large volume compared with the cylinder diameter, Gharib et al. (13) proposed that a dimensionless FT of ~4 corresponds to the vortex ring pinch-off when the maximum vortex ring propulsive efficiency is attained. Later, Gharib et al. calculated the dimensionless FT associated with LV diastolic filling as follows:

\[
\text{Formation time} = \frac{U_{\text{mean}} t_{E\text{-wave}}}{D},
\]

where \(U_{\text{mean}}\) is the mean filling velocity, \(t_{E\text{-wave}}\) is the E-wave duration, and \(D\) is the maximum mitral valve diameter (12). The dimensionless FT was compared among patients, as opposed to the time from the E-wave initiation in seconds, to account for individual patient variations. Gharib et al. proposed that the FT can be used as a specific indicator of cardiac function. However, the analysis was based on the assumption that the vortex ring FT coincides with the completion of the E-wave filling (when the transmitral velocity falls to zero), though this assumption has not been verified.

We hypothesized that the LV vortex ring formation is governed by the decrease of the favorable LV pressure difference that is driving early diastolic filling, and the vortex ring pinch-off occurs simultaneously with the time when the LV filling pressure difference becomes zero in at least one location along the length of the LV. Thus the vortex ring FT should be calculated using the interval from the initiation of the early diastolic filling wave to the onset of filling propagation velocity deceleration, instead of the total E-wave duration. We used color M-mode (CMM) echocardiography that provides a spatiotemporal map of blood velocities during diastole across a scan line from the LV apex to the mitral valve to investigate the velocities and pressures within the LV throughout early diastolic filling in patients with varying degrees of diastolic dysfunction. These data were used to integrate the Euler equation, providing a measurement of the intraventricular pres-
sure difference (IVPD) (40, 44). We examined the early diastolic filling wave deceleration and its relationship to the pinch-off of the vortex ring formed beyond the mitral annulus inlet. Additionally, we investigated whether the vortex ring FT is altered with the development of diastolic impairment.

MATERIALS AND METHODS

Echocardiography acquisition. Echo-Doppler examinations were completed using an iE33 ultrasound imaging system with a multiple frequency transducer (Philips Medical Systems, Andover, MA). Standard two-dimensional images were obtained in the parasternal long and short axes and in the apical four- and two-chamber views. Pulsed-wave Doppler tracings of mitral valve inflow were recorded at the leaflet tips. CMM ultrasound was obtained with a sweep speed of 100 mm/s with a scale that optimized visualization of the isovelocity color contour as judged by the recording sonographer. Recordings of the mitral annular velocity were also obtained on the septal and lateral walls from an apical four-chamber view. LV volumes and Doppler tracings were analyzed using a digital echocardiography workstation as previously described (11, 43).

Statistical analysis. JMP Statistical Discovery Software (SAS Institute, Cary, NC) was used for all statistical analysis. All calculated properties were averaged for each category and are expressed as means ± 1SD. We analyzed statistical significance among groups using a one-way analysis of variance and the Tukey-Kramer honestly significant difference test.

Description of the automated CMM analysis algorithm. An automated algorithm was developed to analyze CMM data and to calculate the pressure distributions within the LV. Details on the CMM analysis algorithm can be found in the methodology section and supplementary material in previous work by Stewart et al. (39).

Patient population. A total of 128 patients were used in this study and were selected from routine comprehensive echocardiography and Doppler evaluations at Wake Forest University Baptist Medical Center. Propagation velocity ($V_p$) was calculated for each patient during regular clinical treatment according to the American Society of Echocardiography guidelines (26). This study was conducted according to protocols approved by the Virginia Tech and Wake Forest University Internal Review Boards. Patient data were collected from clinically indicated comprehensive echocardiography, MRI, and Doppler evaluations at the Wake Forest University Baptist Medical Center; therefore, informed consent was not required.

The patient population was selected using the inclusion criteria of no preexisting cardiac conditions and good quality echocardiography scans. Previous work including these patients have been published by Brucks et al. (5) and Stewart et al. (39).

Phase-contrast MRI. Phase-contrast MRI (pcMRI) measurements were performed on an Avanto 1.5T scanner from Siemens Medical Solutions, located at the Wake Forest University Baptist Medical Center in Winston-Salem, NC. Two-dimensional pcMRI scans were acquired of two patients, one with normal filling and one with LVDD, in accordance with Institutional Review Board guidelines preestablished for the study. Velocity encoding for each scan was 100–130 cm/s, with a repetition time of about 20 ms and 40, 45, or 50 reconstructed phases (depending on patient heart rate). Echo time was 3.3, and there was one view per segment. Flip angle was 20°, and the resolution was 320 × 256 at 1.25 mm/pixel in-plane with a 5-mm slice thickness. Retrospective ECG gating was used for acquisition. Each line of k-space was read three times and then averaged for each scan.

In addition, a separate high signal-to-noise ratio imaging scan was acquired immediately following each pcMRI over the same field of view and used to perform image segmentation on the velocity portraits, as noise in the real part of the pcMRI images often made boundary detection difficult. These images were registered to the pcMRI scans via common anatomical landmarks, and the boundaries of the LV, left atrium, aortic outflow tract, right ventricle, and descending aorta were mapped and transferred to the pcMRI images.

RESULTS

A cohort of 128 patients with varying diastolic health was studied, including a subset of 46 patients with normal filling classified based on a ratio of peak transmitral inflow velocity to mitral annulus velocity ($E/E'$) value $< 8$ (27). Previous work including these patients have been published by Brucks et al. (5) and Stewart et al. (39). Table 1 displays the characteristics of the patient cohort classified by clinically diagnosed diastolic dysfunction stage. Analysis of patient CMM data provided quantitative information for three characteristic events during diastole and the respective times at which they occurred. First, the $V_p$ deceleration point time is the time of the most statistically significant change (deceleration) of the early filling wave $V_p$, as shown in Fig. 1A by a change in the slope of the $V_p$ (green line) (39). Second, the onset of zero pressure difference corresponds to the time at which the interventricular pressure difference (IVPD) along the scan line first falls to zero in at least one location along the length of the LV from the mitral valve to the apex (at this time, the pressure difference is not zero along the entire scan line). At the location where the pressure difference is zero, there is no driving force for flow. The first onset of a zero pressure difference time is indicated in Fig. 1A and C, as the vertical white solid line and in B as the vertical black bold solid line. The IVPD is the pressure within the LV reference to the mitral annulus pressure and does not take into account the left atrial pressure difference. Therefore at the onset of zero pressure difference, the relative pressure at a specific depth.

### Table 1. Clinical characteristics of the cohort

<table>
<thead>
<tr>
<th>Diastolic Dysfunction Stage</th>
<th>0-Normal</th>
<th>1-Delayed Relaxation</th>
<th>2-Pseudonormal</th>
<th>3-Restrictive</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients, n</td>
<td>60</td>
<td>29</td>
<td>23</td>
<td>16</td>
</tr>
<tr>
<td>Age, yr</td>
<td>39 ± 13</td>
<td>66 ± 13</td>
<td>68 ± 12</td>
<td>60 ± 15</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>0.6 ± 0.1</td>
<td>0.6 ± 0.1</td>
<td>0.4 ± 0.1</td>
<td>0.3 ± 0.1</td>
</tr>
<tr>
<td>E/E'†</td>
<td>7.2 ± 1.6</td>
<td>11.9 ± 4.1</td>
<td>15.5 ± 5.1</td>
<td>16.5 ± 5.0</td>
</tr>
<tr>
<td>E, cm/s</td>
<td>89 ± 19</td>
<td>71 ± 16</td>
<td>97 ± 18</td>
<td>99 ± 19</td>
</tr>
<tr>
<td>A, cm/s</td>
<td>57 ± 14</td>
<td>92 ± 22</td>
<td>67 ± 19</td>
<td>39 ± 14</td>
</tr>
<tr>
<td>E/A*</td>
<td>1.6 ± 0.6</td>
<td>0.8 ± 0.3</td>
<td>1.5 ± 0.4</td>
<td>2.8 ± 0.9</td>
</tr>
<tr>
<td>IVPD, mmHg‡</td>
<td>3.1 ± 1.1</td>
<td>2.5 ± 1.0</td>
<td>2.9 ± 1.5</td>
<td>2.3 ± 1.3</td>
</tr>
<tr>
<td>LV length, cm</td>
<td>7.8 ± 0.9</td>
<td>7.8 ± 1.0</td>
<td>8.3 ± 1.3</td>
<td>8.7 ± 0.9</td>
</tr>
<tr>
<td>LVEDV, ml</td>
<td>88 ± 27</td>
<td>100 ± 40</td>
<td>123 ± 60</td>
<td>144 ± 59</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>68 ± 18</td>
<td>57 ± 13</td>
<td>66 ± 13</td>
<td>76 ± 12</td>
</tr>
</tbody>
</table>

Values are means ± 1SD (range). Patients are classified based on clinically diagnosed diastolic dysfunction stage. *E wave-to-A wave transmitral velocity ratio (E/A); †E-wave transmitral-to-E-wave mitral annulus velocity ratio (E/E'); ‡Interventricular pressure difference (IVPD). LVEDV, left ventricular (LV) end-diastolic volume.
within the LV equals the pressure at the mitral inlet. Third, the peak IVPD time occurs when the pressure difference within the LV reaches a maximum value. This time is indicated in Fig. 1, A and C, as the vertical dashed white lines and in B as the vertical dashed black line. The onset of a zero pressure difference and peak IVPD times are displayed with reference to the mitral inflow velocity waveform in Fig. 1B, which indicates the E-wave and A-wave inflow peaks. The \( V_p \) deceleration point time, the time at which \( V_p \) decreases, occurs before the time of the peak mitral inflow velocity while the transmitral velocity is still increasing, shown in Fig. 1B. From Fig. 1, A–C, it can be seen that the onset of a zero pressure difference occurs after the peak IVPD time (15, 24, 40, 44).

Figure 1D displays pressure difference versus position within the LV contours for two time instants. The variation in IVPD as a function of position represents the progressive pressure gradient within the ventricle, which has been demonstrated in previous studies (8, 28, 29, 44). The dashed black line represents the spatial pressure difference within the LV from the mitral annulus to the apex at the peak IVPD time, and the bold black line represents the spatial pressure difference within the LV at the first onset of a zero pressure difference time. Figure 1C illustrates the succession of these events.

The nondimensional times at which the \( V_p \) deceleration point and the onset of a zero pressure difference occurs were calculated from the CMM data as shown in Eq. 2.

\[
\text{Nondimensional time} = \frac{U_{\text{mean}(t-t_0)}(t-t_0)}{D}
\]  

where \( t \) is the time of the zero pressure difference or the deceleration point time for their corresponding nondimensional times, \( t_0 \) is the time of the start of the early filling wave, and

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mitral valve diameters and mean inflow velocities and account for variations among patients with different mitral valve diameters and mean inflow velocities. Figure 2 displays the relationship between our two nondimensional times of interest. The nondimensional time of the onset of zero pressure difference is positively correlated with the nondimensional \( V_p \) deceleration point time (slope = 0.96, \( r^2 = 0.28, P < 0.0001 \)). The solid line represents the linear regression, whereas the shaded area represents the 95% confidence interval of the data points. The two nondimensional times are nearly equivalent as indicated by a slope of 0.96 when the intercept is constrained to zero, implying that the two times are associated with the same physical event.

Similar to the \( V_p \) deceleration point in this work, Baccani et al. (2) and Verdonck and Vierendeels (41) found changes in the early filling wave \( V_p \) within computational fluid dynamics studies of filling for normal and dilated cardiomyopathy conditions. By performing a CMM type of analysis on the numerical data, Baccani et al. (2) found two distinct propagation velocities and reported a decrease in the \( V_p \) after the vortex ring detached from the mitral annulus. A biphasic early filling wave has been reported in other in vivo and numerical studies where phase one represents the initial columnar high velocity filling into the ventricle. The second phase represents the propagation

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**Figure 2.** Nondimensional time of the first onset of zero pressure difference calculated from the CMM versus the nondimensional \( V_p \) deceleration point time. The centerline displays the linear fit and the shaded region represents the 95% confidence interval.

**Figure 3.** Phase-contrast MRI data for a normal and abnormal filling patient. A: vorticity fields for a time instant before and after the \( V_p \) deceleration point time. C: corresponding simulated CMM echocardiogram data generated from extracted one-dimensional slices from the phase-contrast MRI data for a normal filling patient. B and D: similar data for an abnormal filling patient. The intersection of the magenta and green lines indicates the \( V_p \) deceleration point.
of the maximum velocity region into the ventricle, which has been associated to the vortex ring propagation (28, 36). Additionally, a decrease in the LV centerline velocity (7, 34) has been shown in numerical models after a vortex ring has been formed and begins to propagate toward the apex. In our work, an example of the relationship between the \( V_p \) deceleration point and vortex ring pinch-off is displayed using pcMRI data for a normal and an abnormal filling patient. CMM data were simulated by extracting a one-dimensional slice from the left atrium through the mitral annulus and the LV apex for each pcMRI image in time. The recreated CMM images for an example normal and abnormal filling case are displayed in Fig. 3, C and D. The \( V_p \) deceleration point for the early diastolic filling wave was calculated and is indicated by the intersection of the magenta and green lines. Vorticity fields of the pcMRI data at two time steps, one preceding and one following the calculated \( V_p \) deceleration point time are displayed above the CMM images. In the pcMRI data images in Fig. 3A (normal filling case) and B (abnormal filling case), the vortex ring is still forming and has not pinched off from the mitral annulus before the \( V_p \) deceleration point time, shown by the attachment of the isovorticity contour to the mitral annulus in the first image. The second pcMRI image occurs after the \( V_p \) deceleration point when the isovorticity contours have detached from the mitral annulus indicating vortex ring pinch-off. Because of the low temporal resolution of the pcMRI data, the simulated CMM images were interpolated to smooth the data and simulate standard CMM data. Therefore, there is corresponding uncertainty in the time of the \( V_p \) deceleration point. However, this analysis does support the presence of vortex ring pinch-off before the end of early diastolic filling. The present study in combination with the work by Stewart et al. (39) provides support and validation, using clinical data, to Baccani et al. (2) and Verdonck and Vierendeels (41). However, the previous works did not quantify the precise \( V_p \) deceleration point time.

Synthesizing the results in Figs. 2 and 3 with the work by Baccani et al. (2) and Verdonck and Vierendeels (41) reveals that the vortex ring pinch-off within the LV occurs at the time of the diastolic filling wave \( V_p \) deceleration point and that both events correspond to the loss of a favorable pressure difference within the LV. These observations further suggest that pinch-off occurs substantially before the completion of early diastole (E wave). An overview of this process is demonstrated in Fig. 4. During the initial stage of diastole, a continuous jet issues through the mitral valve that generates a rapid initial \( V_p \). At the time of the vortex ring pinch-off, the supply of momentum to the wave front (led by the vortex ring) in the flow is abruptly reduced, thus resulting in a sudden deceleration of the propagation velocity wave as shown by the reduced terminal \( V_p \) slope.

We calculated two FTs for the 46 patients with normal filling as indicated by \( E/E' < 8 \). First, based on our results indicating that the vortex ring, pinch-off occurs at the \( V_p \) deceleration point time; a FT was calculated using the time between the initiation of the early filling wave and the deceleration point time as well as the mean velocity over this time period and will be referred to as \( FT_{dec} \). Second, \( FT_{E-wave} \) was calculated using the total early filling wave time following the assumption of the work of Gharib et al. (12). In Fig. 5A, we plot both \( FT_{dec} \) and \( FT_{E-wave} \) against age (\( FT_{dec} \) slope = \(-0.0062/\text{year}\), \( r^2 = 0.053, P < 0.12 \); \( FT_{E-wave} \) slope = \(-0.037/\text{year}\), \( r^2 = 0.30, P < 0.0001 \)). \( FT_{E-wave} \) displays a very similar inverse relationship to that presented by Gharib et al. (12), with the lower and upper quartile values of the FT distribution at 3.9 and 5.4. The slope of the \( FT_{E-wave} \) versus age relationship for our normal patient cohort was \(-0.037/\text{year} \) versus a slope of approximately \(-0.031/\text{year} \) for the patient cohort of Gharib et al. that was primarily composed of normal filling patients. As such, our results corroborate their measurements. However, \( FT_{dec} \) (diamonds), which we showed better represents the vortex ring pinch-off time, as opposed to \( FT_{E-wave} \) calculated using the duration of early diastole, shows that the vortex ring FT does not vary with age (slope = \(-0.0062/\text{year}\), \( r^2 = 0.053, P = 0.012 \), and has a mean value of 1.61 for healthy patients.

Early filling peak IVPD provides an independent measure of the strength of early diastolic suction and allows delineation between healthy and diseased patients as well as stage of diastolic dysfunction (44). In Fig. 5B, we show the same FT
related to variations in LV stiffness, viscoelasticity, and load. This clearly implies that the LV and its operation are adapting with the progression of diastolic impairment. However, FT\textsubscript{dec} remains nearly invariant as a function of early diastolic peak IVPD, and the \( V_p \) deceleration point occurs at an approximate nondimensional time of 1.63 regardless of the severity of diastolic impairment. This value is essentially unchanged compared with the healthy patients shown in Fig. 5A. The nondi-

## DISCUSSION

We found that the favorable LV pressure difference driving early diastolic filling first falls to zero simultaneously with the \( V_p \) deceleration of the early filling wave and pinch-off of the LV vortex ring (Fig. 2). These findings, in combination with the results by Baccani et al. (2) and Verdonck and Vierendeels (41) displaying the relationship between a decrease in the filling wave velocity with the vortex ring pinch-off, reveal that the vortex pinch-off is responsible for the \( V_p \) deceleration point calculated within early diastolic filling. Moreover, both events occur simultaneously with the first onset of a zero pressure difference within the LV. The favorable pressure gradient driving early diastolic filling has been shown to fall to zero at approximately the time of the peak E-wave velocity just after the completion of the ventricular suction (8, 20). We found that the early diastolic vortex ring FT occurs substantially before the end of the early diastolic filling wave approximately just before the peak E-wave inflow velocity, at the time of the completion of early diastolic suction. At this time the vortex ring has reached its maximum circulation strength and pinches off resulting in a decreased centerline inflow velocity.

Although we believe that the vortex ring pinch-off results in a decreased \( V_p \), it is also possible that the decelerating flow results in the vortex ring pinch-off caused by a decrease in the vorticity flux/supply. Because the transmitral inflow velocity is still accelerating at the time of the \( V_p \) deceleration point (shown in Fig. 1B), the vorticity flux/supply into the vortex ring remains positive. Therefore, it is physically more probable that the vortex ring pinch-off occurs first and yields a reduction in the vortex ring \( V_p \).

The vortex ring formed within the confined LV does not behave as the classically explored vortex ring in a large volume. At the \( V_p \) deceleration point when the vortex ring pinches off, the remaining fluid entering the ventricle is within the trailing jet. Based on the vortex ring-to-ventricular diameter ratio, the dynamics of the generated vortex rings will be altered, which may affect the \( V_p \) toward the apex and the vortex circulation strength over time (4, 28, 35, 37). The FT of the vortex ring pinch-off may also be altered by many factors of the LV filling, including the inflow velocity profile, the orifice geometry, downstream confinement domain, and LV wall motion. Work by Dabiri and Gharib (9) has shown that FT can be altered by counterflow (flow in the opposite direction of the vortex ring formation jet), and work by Stewart and Vlachos (38) has displayed that the FT can be altered in severely confined downstream domain conditions.

We found that the nondimensional early filling duration time (\( \text{FT}_{E-wave} \)), calculated using the total duration of the early
diastolic filling, varied with age and peak IVPD, specifically, the E-wave FT decreased as the heart was compromised by age or disease. These results are in agreement with previous LV vortex FT work (12, 21, 32). However, this FT calculation based on the early diastolic filling wave duration assumes that the vortex ring does not pinch off until the end of the E wave. In contrast, we found that the pinch-off of the vortex ring occurs before the completion of early diastole. The \( V_p \) deceleration point time indicating the time of vortex ring pinch-off was calculated using the \( V_p \), however, the patients’ diastolic function or the nondimensional FT was not validated versus the \( V_p \). Consistent with this, we calculated FT\(_{dec}\), and we discovered that it remains unaffected by age or diastolic impairment, with a mean of \( \sim 1.63 \) within our data cohort. This value is much lower than the previously reported FT, of \( \sim 4 \), described for vortex rings formed using a piston cylinder configuration in semi-infinite domains. Changes in the inflow velocity profile, orifice geometry, and downstream domain over time will affect the vortex ring generation and therefore alter the FT, in this case decreasing its value.

Clinical relevance. The objective of this work is not to develop a diagnostic parameter for LVDD conditions but to reveal the physics of the filling process in normal and abnormal filling cases. This work identifies that the vortex ring pinch-off occurs before the end of the early diastolic filling wave, and this pinch-off is the physical reason for the abrupt deceleration of the \( V_p \) within the early filling wave. The LV vortex FT, accounting for the time of vortex ring pinch-off as calculated by the \( V_p \) deceleration point time, was shown to be invariant to pathophysiological changes associated with diastolic dysfunction and age within the current data cohort. Although the FT\(_{dec}\) was shown to be relatively unchanged, the mean inflow velocity, duration of the E wave from initiation to the \( V_p \) deceleration point, and the mitral valve diameter did vary among patients. This work displays that vortex ring FT is not an indication of cardiac function as previously suggested (12).

Limitations. Our study should be interpreted in light of its limitations. The patients used in this study include clinically diagnosed patients with normal filling and varying stages of LVDD. However, patients with other abnormal filling conditions were not included. This study does not account for variations in ventricular geometry, such as dilation of the ventricle which will affect vortex ring formation (28). The calculation of the vortex formation parameters assumes normal mitral valve function with no mitral regurgitation and that the CMM scan line was aligned through the center of the mitral inflow jet from the mitral valve to the apex (26). Acquisition of the CMM data off axis could result in errors throughout the calculated variables. Additionally, the vortex formation parameters calculated use the mean mitral inflow velocity as a simplification to the running mean velocity \( \bar{U}_p = \int f_{in,p} dt/\int f_{in,p} dt \) used in previous experimental vortex ring generation work (13). The invariance of the nondimensional \( V_p \) deceleration point time is shown versus age and the calculated peak IVPD values from the CMM data; previous studies have validated this method by comparison to direct measurements with micromanometers (15, 44). The number of pcMRI patients evaluated was limited, and they were used only as a qualitative visual example of the vortex ring pinch-off and the \( V_p \) deceleration point.

Conclusions. We found that vortex ring pinch-off is governed by the decrease of the IVPD and occurs before the completion of early diastole, simultaneously with the time the LV filling wave pressure becomes zero and the end of the classically defined diastolic suction. The calculation of the vortex FT was adjusted to reflect this finding, and the results showed that the FT remains invariant to changes of diastolic function. Hence, our study supports the existence of a vortex formation timescale for the optimum LV filling that is independent of diastolic function. Thus, the vortex ring FT is not an indication of cardiac function as previously suggested.

REFERENCES
LV VORTEX FORMATION IS UNAFFECTED BY DIASTOLIC IMPAIRMENT


