Mechanisms of Preejection and Postejection Velocity Spikes in Left Ventricular Myocardium

Interaction Between Wall Deformation and Valve Events

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Background—Normal left ventricular myocardium demonstrates distinct spikes in the velocity trace before and after left ventricular ejection. We tested the hypothesis that the preejection and postejection velocity spikes reflect early systolic shortening and late systolic lengthening that are interrupted by mitral and aortic valve closure, respectively.

Methods and Results—In 11 anesthetized dogs, timing of valve closure was determined by pressure variables; left ventricular dimensions were determined by sonomicrometry. Myocardial shortening started 20±10 ms (mean±SD; P<0.001) before mitral valve closure and was interrupted at the time of mitral valve closure (time difference, 4±7 ms). Similarly, myocardial lengthening started 31±15 ms (P<0.001) before aortic valve closure and was interrupted at the time of aortic valve closure (time difference, 0±3 ms). Prevention of mitral (n=4) and aortic (n=4) valve closure by stenting the valves abolished the preejection and postejection velocity spikes, respectively. Echocardiographic measurements of patients (n=15) with severe mitral regurgitation showed that the preejection velocity spike was reduced after prosthetic valve replacement (43±25 versus 32±15 mm/s; P=0.036), indicating that preejection shortening was larger with a leaking valve. Similarly, late systolic lengthening was reduced in patients (n=15) with severe aortic regurgitation after prosthetic valve replacement; minimum postejection velocity spike was increased from −32±11 to −17±11 mm/s; P=0.0003). Asynchronous onset of contraction/relaxation and atrioventricular interaction were investigated as alternative mechanisms of the velocity spikes in dogs and patient groups but were found implausible.

Conclusions—This study supports the hypothesis that normal left ventricular preejection and postejection velocity spikes are attributed to valve closures that interrupt early systolic shortening and late systolic lengthening, respectively.

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Key Words: echocardiography • mechanics • myocardium • valves • velocity

The typical myocardial velocity trace seen in left ventricular (LV) long-axis views by tissue Doppler echocardiography consists of a positive systolic shortening wave during LV ejection (S wave) and negative myocardial lengthening waves during early filling (E’ wave) and atrium-induced filling (A’ wave). In addition, 2 spikes or rapid oscillations can be seen: one just before the ejection wave and one right after the ejection wave. These spikes are distinct features of the velocity trace (Figure 1). However, the cause of the preejection and postejection velocity spikes has not been determined.

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Garcia et al1 speculated that the spikes reflect geometric changes caused by asynchronous contraction and ventricular interdependence. Sengupta et al2 proposed that the biphasic waves are associated with asynchronous deformation of subendocardial and subepicardial LV wall layers.

We propose a simple mechanism behind the myocardial preejection and postejection velocity spikes: At the onset of systole, LV wall shortening starts before mitral valve closure (MVC). The initial wall shortening pushes blood toward the valve, closes the valve leaflets, and moves them toward the left atrium (LA). We suggest that this mechanism accounts for the positive preejection velocity, which represents the upstroke of the preejection velocity spike. During this phase, LV cavity pressure and hence myofiber stresses are low, and high shortening velocities may be generated. The closing motion of the leaflets stops when the chordae tendineae and papillary muscles tense and prevent further leaflet motion.
This restrains further wall shortening and accounts for the downstroke of the preejection velocity spike. Therefore, only minor wall movement occurs between MVC and aortic valve opening (AVO) until shortening resumes with AVO and onset of LV ejection. At the end of systole, we propose a similar mechanism, with myocardial lengthening starting before aortic valve closure (AVC) and causing a negative myocardial velocity, which represents the downstroke of the post-ejection velocity spike.

To investigate the hypothesis that the preejection and post-ejection velocity spikes were due to valve closures, we investigated the relationships between the timing of valve closures and the velocity spikes in a dog model and in healthy humans. Furthermore, we predicted that removal of mitral and aortic valve function would abolish the preejection and post-ejection velocity spikes, respectively. Therefore, in the dog model, measurements were done before and after removal of valve function by the insertion of stents into the mitral and aortic valve orifices, which accounts for the upstroke of the post-ejection velocity spike.

Finally, we investigated whether the preejection velocity spike may be caused by atrioventricular (AV) interactions and, in particular, whether the preejection velocity may reflect passive recoil of LV myocardium, which has been stretched by atrial contraction. To this end, we included patients with atrial fibrillation to determine whether the preejection velocity spike was abolished or reduced relative to individuals in sinus rhythm. Furthermore, we studied patients during AV sequential pacing and different AV delays, which allowed complete separation of timing of atrial relaxation and LV contraction. If atrial relaxation causes a velocity resulting from passive recoil of LV myocardium, this should be identified during long AV delays as an LV shortening velocity before LV systole.

Methods

Experimental Study

Eleven adult mongrel dogs (7 female; average weight, 31±4 kg) were used in this study. The laboratory animals were supplied by the Center for Comparative Medicine, Rikshospitalet University Hospital, Oslo, Norway. The study was approved by the Norwegian National Animal Experimentation Board. The animals were anesthetized, ventilated, and surgically prepared as previously described.3

Micromanometer-tipped 5F catheters (model MPC-500, Millar Instruments, Houston, Tex) were positioned in the LV, in the LA, and in the ascending aorta close to the aortic valve. Calibration, data sampling, and analysis were done as previously described.3

Sonomicroscopy

The LV dimensions were measured by ultrasonic crystals (Sonometrics, London, Ontario, Canada) implanted in the subendocardium. One crystal was placed at the apex, and 4 crystals were placed in the basal half of the LV 65% to 75% of the distance from apex to base in the anterior, lateral, posterior, and septal walls, respectively. This enabled measurements of 4 long-axis diameters and 2 short-axis diameters from the septal to lateral wall and the anterior to posterior wall. The sampling rate was set at >200 Hz.

Analysis of Baseline Recordings

The timing of valve closure and opening was defined as follows: MVC, defined as the peak of the c wave in the LA pressure trace;4–6 AVO, defined as the beginning of the upstroke of the aortic pressure trace; AVC, defined as dP/dtmin of LV pressure; and mitral valve opening (MVO), defined as the first diastolic LA-LV pressure crossover.

Figure 2 illustrates how these timing criteria were applied. The figure also includes associated diameter, shortening velocity, and shortening acceleration traces demonstrating typical deformation pattern in relation to valve closures and openings. According to our hypothesis, MVC interrupts shortening and should therefore correspond to a peak in deceleration of shortening (downstroke of the preejection velocity spike). Similarly, AVC interrupts lengthening and should correspond to a positive peak in the shortening acceleration trace (upstroke of the post-ejection velocity spike). For each of the 4 long-axis and 2 short-axis traces, the relationship between the velocity spikes and valve events was investigated by a comparison of the time of MVC and peak early systolic deceleration and of AVC and peak late systolic acceleration.

LV volume (LVV) was calculated as follows: LVV=(π/6)×LAX×SAXap×SAXsl, where LAX is the average of the 4 long-axis diameters, and SAXap and SAXsl are the anteroposterior and septal to lateral wall short-axis diameters, respectively. The reduction in LVV before MVC and the increase in LVV before AVC were calculated.

Stenting of the Mitral and Aortic Valves

To test the hypothesis that MVC causes the preejection velocity spike, a stent (Wallstent uni, Boston Scientific, Boston, Mass) was
inflated in the mitral orifice over the mitral valve leaflets to prevent the valve from closing. The stenting, performed at the end of the experiment, was successful in 4 of 5 animals. The stent allowed blood to flow freely from the LV into the LA during the entire systole. The presence and magnitude of the preejection velocity spike were investigated before and after stenting.

In a separate set of animals, the aortic valve was prevented from closing by the inflation of a stent (Memotherm Aortic stent, Bard, Billerica, Mass) in the valve orifice over the aortic valve cusps. The implantation was successful in 4 of 6 animals. The aortic valve stent allowed blood to flow freely from the aorta into the LV after the ejection phase. The presence and magnitude of the postejection velocity spike were investigated before and after stenting.

**Synchronicity of Contraction and Relaxation**

To test the alternative hypothesis that the preejection and postejection velocity spikes, we investigated the timing and velocities of the preejection and postejection velocity spikes in the 4 long-axis diameters (apex to the anterior, posterior, septal, and lateral walls) during baseline conditions.

**Clinical Studies**

The velocity spikes were investigated in 10 healthy volunteers (2 women; mean age, 59±6 years), 15 patients (8 women; mean age, 67±12 years) with severe mitral regurgitation before and after implantation of a mechanical mitral valve prosthesis, 15 patients (7 women; mean age, 48±16 years) with severe aortic regurgitation before and after implantation of a mechanical aortic valve prosthesis, 10 patients (5 women; mean age, 75±17 years) with chronic atrial fibrillation, and 10 patients (5 women; mean age, 76±9 years) with third-degree AV block and a dual-chamber pacemaker.

The studies were approved by the National Committee for Medical Research Ethics of Norway. All participants gave informed consent. Echocardiographic images were acquired with a Vivid 7 Ultrasound scanner (GE Vingmed Ultrasound, Horten, Norway). Basal septal velocities were investigated by tissue Doppler imaging in an apical 4-chamber view with frame rates of 161±45 frames per second. We confirmed in the animal study, using sonomicrometry as the reference method, that these frame rates resulted in errors <1±1 mm/s. MVC and AVC were defined as the time the valve leaflet stopped moving in the closing direction.

**Study of Patients Before and After Prosthetic Mitral or Aortic Valve Implantation**

To investigate how valvular regurgitation modified the preejection and postejection velocity spikes, we investigated the velocity spikes in patients with severe mitral or aortic valve regurgitation before and after prosthetic valve implantation (14 ON-X valves [On-X Life Technologies, Inc, Austin, Tex] and 16 CarboMedics valves [CarboMedics, Austin, Tex]).

**Synchronicity of Contraction and Relaxation**

To test the alternative hypothesis that the preejection and postejection velocity spikes could be attributed to normal asynchrony, we compared velocities in basal septal and lateral wall segments in the healthy subjects.

**Study of AV Interaction**

To test the hypothesis that the preejection velocity spike is caused by AV interaction, the presence and amplitude of the preejection velocity spike were investigated in the patient group with atrial fibrillation to determine whether the velocity spike was lost. Furthermore, in the patients with third-degree AV block and a dual-chamber pacemaker, we performed pacing with AV delay at 3 different levels: (1) at a short delay of 50 ms, when the atrial myocardium was still in a stiff, contracted state during activation of the ventricular myocardium; (2) at the nominal delay of 130 ms, when atrial relaxation coincided with the time of ventricular activation; and (3) at a long delay of 300 ms, when the atrium had relaxed before ventricular activation.

**Statistical Analysis**

Continuous measurements are summarized as mean±SD. The average value from 3 consecutive heart cycles was calculated and used for analysis. Student’s paired t test was used to compare the timing of onset of shortening and lengthening relative to valve closures, the magnitude of the velocity spikes before and after stenting of valves, the velocities before and after implantation of prosthetic valves, and the timing and velocities in the septal and lateral walls in healthy subjects. One-way ANOVA with Bonferroni correction was used to test for statistical differences between timing and velocities in the 4 long-axis diameters in the animal study. A value of P<0.05 was considered significant.

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**

**Experimental Study**

Figure 3 displays LV pressure-volume relations, with the timings of valve openings and closures indicated. As demonstrated in the pressure-volume loop, the LV contracts during early systole, but the reduction in LVV ceases at the time of MVC and remains unchanged until AVO. The loop also demonstrates that the LV starts expanding before AVC, but the increase in LVV ceases at the time of AVC and remains constant until approximately MVO. The average LVV reduc-
tion before MVC was 4.7±2.2%, and the volume increase before AVC was 1.4±0.6%, calculated relative to end-diastolic volume.

Changes were found in both long- and short-axis diameters that corresponded to the described LVV changes (Table 1 and 2). Shortening of the LV diameters started 20±10 ms ($P<0.001$) before MVC and was interrupted at the time of MVC. The agreement between the timing of MVC and interruption of shortening was very good, with a mean time difference of 4±7 ms. Similarly, lengthening started 31±15 ms ($P<0.001$) before AVC and was interrupted at the time of AVC. Excellent agreement was found between the timing of AVC and the interruption of lengthening; the mean time difference was 0±3 ms.

Figure 2 displays the relationship between traces of LV diameter, velocity, acceleration, and pressure variables, together with timing of valve events. The figure demonstrates that early systolic shortening stops at the time of MVC and resumes at the time of AVC, as seen by the knee points in the diameter trace at these 2 times. It further shows that late systolic lengthening is interrupted, as seen by the knee point at the time of AVC. The observed LV shortening after AVC represents postsystolic shortening, which may be seen in hearts after surgical instrumentation.

**Stenting of the Mitral Valve**

Figure 4 shows an example of a diameter trace before and after stenting of the mitral valve. Before stenting, early systolic shortening was interrupted at the time of MVC. This resulted in a preejection velocity spike that had an average magnitude of 63±29 mm/s. After stenting, shortening continued without interruption as the stent prevented valve closure and allowed the LV to eject blood back into the LA.

This resulted in a smooth transition from lengthening to shortening; therefore, the pre-ejection velocity spike was essentially removed. However, because of the nature of differentiation, only small curvature changes in the diameter trace resulted in a wave in the velocity trace. Thus, the spike was not totally removed in all traces, and the average poststent spike was 10±14 mm/s, which still was markedly smaller than before stenting ($P<0.001$).

**Stenting of the Aortic Valve**

Representative recordings from stenting of the aortic valve are shown in Figure 5. Before stenting, the traces displayed the typical lengthening pattern just before AVC, followed by a knee point at AVC and a shortening or plateau right after AVC. The derivative of these traces exhibited the distinct post-ejection velocity spike. After stenting, lengthening was allowed to continue uninterrupted as the stent prevented valve closure and allowed blood to flow back from the aorta into the LV. The smooth transition from shortening to lengthening essentially removed the post-ejection velocity spike. The average magnitude of the velocity spike was reduced from 22±11 mm/s before stenting to 2±3 mm/s after stenting ($P<0.001$).

**Synchronicity of Contraction and Relaxation**

As demonstrated by the representative recordings in Figure 6 (top) and summarized in Table 3, the preejection and post-ejection velocity spikes were synchronous throughout the LV. The maximum time difference of the 4 measured preejection velocity spikes (pre,) was 11±11 ms; it was 12±8 ms for the post-ejection velocity spikes (post,).

**Clinical Study**

Representative velocity recordings from the different clinical substudies are shown in Figure 7.

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**Table 1. Experimental Study: Timing of LV Wall Deformation in Relation to Valve Closure**

<table>
<thead>
<tr>
<th>Time Interval</th>
<th>From Onset of Shortening to MVC</th>
<th>From MVC to Peak Early Systolic Deceleration</th>
<th>From Onset of Lengthening to AVC</th>
<th>From AVC to Peak Late Systolic Acceleration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Long-axis diameter, ms</td>
<td>$17\pm1$</td>
<td>$9\pm6$</td>
<td>$44\pm12$</td>
<td>$0\pm4$</td>
</tr>
<tr>
<td>Short-axis diameter, ms</td>
<td>$23\pm12$</td>
<td>$0\pm5^*$</td>
<td>$20\pm2^*$</td>
<td>$-1\pm3$</td>
</tr>
<tr>
<td>Average, ms</td>
<td>$20\pm10$</td>
<td>$4\pm7$</td>
<td>$31\pm15$</td>
<td>$0\pm3$</td>
</tr>
</tbody>
</table>

|$P<0.001$, $^*P<0.001$, long- vs short-axis diameter.
Mitral and Aortic Regurgitation

In patients with mitral valve regurgitation, the peak pre-ejection velocity spike (prea in Figure 1) was 43±12 mm/s before valve surgery and 32±15 mm/s afterward (P=0.036). Minimum velocity of the downstroke of the spike (preb) changed from 6±15 to −11±13 mm/s (P=0.002). These changes indicate a trend toward less shortening before MVC and more complete interruption of shortening by the valve prosthesis. In patients with aortic regurgitation, the peak post-ejection velocity spike (postb) was virtually unchanged after valve surgery: 14±12 mm/s before and 19±12 mm/s after (P=0.56). However, minimum velocity at the bottom of the upstroke of the post-ejection velocity spike (posta) changed from −32±11 to −17±11 mm/s (P=0.0003), indicating that LV wall lengthening occurring before AVC was reduced after valve surgery.

Synchronicity of Contraction and Relaxation

In the normal subjects, the pre-ejection and post-ejection velocity spikes occurred simultaneously in the septal and lateral walls (Figure 6, bottom). Mean data are presented in Table 4. In each patient, MVC occurred during downstroke of the pre-ejection velocity spike (posta) was present. The average peak velocity (prea) was 31±12 mm/s, and the minimum velocity of the downstroke (preb) was −8±16 mm/s.

AV Interaction

In patients with atrial fibrillation, a distinct pre-ejection velocity spike clearly was present. The average peak velocity (prea) was 31±12 mm/s, and the minimum velocity of the downstroke (preb) was −8±16 mm/s.

In patients with third-degree AV block, pacing with a short and nominal AV delay did not alter the pre-ejection velocity spike (37±20 and 39±11 mm/s, respectively). In contrast, pacing with a 300-ms-long AV delay altered the morphology of the velocity trace, and 2 pre-ejection velocity spikes were present (Figure 7, bottom right). After atrial contraction, a small positive spike (16±8 mm/s) appeared during atrial relaxation (indicated by an arrow in Figure 7 on the bottom right). This spike occurred before activation of the LV and is consistent with elastic recoiling of the LV during atrial relaxation. The second pre-ejection velocity spike was 37±9 mm/s.

Discussion

This study demonstrates that the pre-ejection velocity spike in normal LV myocardium is due to early systolic shortening, which is interrupted by MVC, and the post-ejection velocity spike is due to late systolic lengthening, which is interrupted by AVC. These mechanisms were supported by observations in healthy human hearts and by animal experiments that included implantation of stents over the mitral and aortic valves to prevent valve closure. When MVC was prevented, the pre-ejection velocity spike was abolished, and when AVC was prevented, the post-ejection velocity spike was abolished, confirming the role of valve closures in generating the velocity patterns.

Valve stenting represents extreme cases of valvular regurgitation. As demonstrated in this study, even patients with severe mitral and aortic regurgitation have relatively preserved pre-ejection and post-ejection velocity spikes. This is
consistent with the notion that even regurgitant valves represent a restriction to transvalvular backflow. An analogy may be sails on a boat: When a gust of wind comes along and folds out the sail, the boat will be pushed along even if the sail has big holes. Only if all ropes and rigging are cut will no force be transferred from the sail to the boat.

**Role of Synchronicity of Contraction and Relaxation**

We could not attribute the velocity spikes to asynchronous contraction and relaxation in the normal heart because our investigation showed that the preejection and postejection velocity spikes were synchronous throughout the LV. All long- and short-axis diameters were included in the formula for calculation of LVV. The volume reduction before MVC and volume increase before AVC are consistent with the notion that most of the myocardial segments contract and lengthen simultaneously during these 2 periods, respectively.

**Role of AV Interaction**

The maintained preejection velocity spike in patients with atrial fibrillation and in patients with short AV delay in whom atrial relaxation occurred after the spike indicates velocity spikes were synchronous throughout the LV. All long- and short-axis diameters were included in the formula for calculation of LVV. The volume reduction before MVC and volume increase before AVC are consistent with the notion that most of the myocardial segments contract and lengthen simultaneously during these 2 periods, respectively.

**Table 3. Experimental Study: Velocities and Timing of Long-Axis Preejection and Postejection Spike Velocities in Different Walls**

<table>
<thead>
<tr>
<th></th>
<th>Anterior</th>
<th>Posterior</th>
<th>Lateral</th>
<th>Septal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Velocities, mm/s</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pre_a</td>
<td>45±24</td>
<td>37±25</td>
<td>42±26</td>
<td>31±16</td>
</tr>
<tr>
<td>pre_b</td>
<td>-21±19</td>
<td>-13±10</td>
<td>-7±14*</td>
<td>-25±11</td>
</tr>
<tr>
<td>post_a</td>
<td>-16±6</td>
<td>-13±8</td>
<td>-13±5</td>
<td>-14±8</td>
</tr>
<tr>
<td>post_b</td>
<td>12±9</td>
<td>11±10</td>
<td>14±5</td>
<td>13±7</td>
</tr>
<tr>
<td>Time from ECG R peak, ms</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>pre_a</td>
<td>30±11</td>
<td>35±6</td>
<td>34±5</td>
<td>28±13</td>
</tr>
<tr>
<td>pre_b</td>
<td>56±4</td>
<td>52±8</td>
<td>56±7</td>
<td>50±11</td>
</tr>
<tr>
<td>post_a</td>
<td>244±19</td>
<td>245±19</td>
<td>239±20</td>
<td>239±20</td>
</tr>
<tr>
<td>post_b</td>
<td>284±20</td>
<td>282±25</td>
<td>281±24</td>
<td>285±33</td>
</tr>
</tbody>
</table>

See Figure 1 for an explanation of labels.

*P=0.048 lateral vs septal wall.

**Table 4. Clinical Study (Healthy Subjects): Preejection and Postejection Velocity Spikes in the Septal and Lateral Walls**

<table>
<thead>
<tr>
<th></th>
<th>Septal</th>
<th>Lateral</th>
</tr>
</thead>
<tbody>
<tr>
<td>Velocities, mm/s</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pre_b</td>
<td>27±8</td>
<td>25±16</td>
</tr>
<tr>
<td>pre_b</td>
<td>4±10</td>
<td>-8±21</td>
</tr>
<tr>
<td>post_b</td>
<td>-19±11</td>
<td>-27±17</td>
</tr>
<tr>
<td>post_b</td>
<td>-5±10</td>
<td>-17±12*</td>
</tr>
<tr>
<td>Time difference between septal and lateral walls, ms</td>
<td></td>
<td></td>
</tr>
<tr>
<td>pre_b</td>
<td>7±7</td>
<td></td>
</tr>
<tr>
<td>pre_b</td>
<td>8±9</td>
<td></td>
</tr>
<tr>
<td>post_b</td>
<td>7±6</td>
<td></td>
</tr>
<tr>
<td>post_b</td>
<td>15±19</td>
<td></td>
</tr>
</tbody>
</table>

See Figure 1 for an explanation of labels.

*P=0.034, septal vs lateral wall.
that the atrial contraction/relaxation sequence is not the mechanism of the spike. However, during long AV delays, a small positive LV myocardial velocity was observed during atrial relaxation. This velocity may be attributed to passive LV shortening resulting from elastic recoiling of the LV wall during atrial relaxation. This mechanism may contribute to the LV preejection velocity, but its magnitude is small, and it cannot be the principal mechanism. This is supported by the finding of preejection velocities in the normal range in patients with atrial fibrillation.

**Comments on Methodology**

In the present study, MVC was defined as the time when the mitral valve stopped moving in the closing direction rather than as the time of leaflet coaptation. The latter definition is appropriate in assessments of isovolumic contraction time, which is the time between the end of mitral inflow and the onset of LV outflow. In the normal heart, LV preejection shortening precedes the isovolumic phase, causing a rise in LV pressure, which decelerates mitral inflow and causes the leaflets to coapt. LV preejection shortening continues during the isovolumic phase by displacing blood toward the mitral region, resulting in bulging of the mitral leaflets into the LA. When the mitral valve reaches its final closing position, preejection shortening is suddenly arrested as the chordae tendineae and papillary muscles tense and stop further leaflet motion.\(^4\)\(^-\)\(^6\) Therefore, preejection shortening occurs before and during the isovolumic contraction. The c wave in the LA pressure curve indicates the time when the leaflets, which are pushed toward the LA, reach their final closing configuration.\(^4\)\(^-\)\(^6\)

**Volume Shifts During Valve Closure**

The LV cavity volume showed a small volume reduction at the beginning of systole before MVC and a small volume increase at the end of systole before AVC (Figure 3, bottom). The volume estimate did not encompass the movement of the valve surfaces or the most basal region of the LV wall. During early systolic contraction, some blood volume may be shifted from the region included in our volume calculation into the more basal LV region. Most likely, the reduced volume at the beginning of systole is enclosed within the mitral leaflets that have moved toward the LA. This interpretation is in accordance with previous reports that have shown that significant contraction may occur before final valve closure.\(^4\)\(^-\)\(^10\)

Although the total cavity volume is isovolumic from the instant the valve leaflets coapt, wall shortening is not restrained until the motion of the valve leaflets is stopped and consequently restrains further volume shift and shortening. Figure 8 (left) shows a schematic of the volume shift within the LV during MVC. Similarly, the closing motion of the aortic cusps into the LV may be responsible for part of the measured volume increase. The latter is supported by Rutley et al,\(^11\) who found an LVV increase during protodiastole. Figure 8 (right) illustrates the volume shift within the LV during AVC. The volume increase at the end of systole is smaller than the volume decrease at the beginning of systole, consistent with the smaller cusps of the aortic valve compared with the mitral valve leaflets.
consistent with the notion that moderately ischemic fibers shorten less and perform less work during early systole but are still capable of causing a rapid velocity change when the fiber stress is low at the onset of systole.

Study Implications
Myocardial velocity spikes are widely used clinically to evaluate LV function, and it is essential that we understand how they are modified by physiological events and disease processes. The present study indicates that valve events are the determinants of normal prejection and postejection velocity spikes and that velocity spikes could represent a means to automatically detect timing of valve closure and opening.

Conclusions
The present study supports our hypothesis that the LV myocardial prejection velocity spike arises from early systolic shortening that is temporarily interrupted by MVC and resumes at the time of AVO and similarly that the postejection velocity spike is a result of late systolic lengthening that is interrupted by AVC and resumes at the time of MVO. These insights may be important when velocities and deformation imaging are used to diagnose myocardial dysfunction.

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Disclosures
None.

References

CLINICAL PERSPECTIVE
Tissue Doppler echocardiography is used clinically to assess left ventricular systolic and diastolic function in terms of myocardial velocities during ejection and filling, respectively. In addition, brief velocity spikes occur before and after ejection, but the cause and clinical importance of these velocity spikes have been debated. This combined experimental and clinical study indicates that the normal prejection velocity spike is due to myocardial shortening, which displaces blood toward the mitral region and causes bulging of the mitral leaflets into the left atrium; when the valve reaches its final closing position, prejection shortening is suddenly arrested. Hence, the initial shortening is reflected by the upstroke of the spike, whereas the downstroke reflects the interrupted shortening. The postejection velocity spike appears to be caused by a similar mechanism; reverse blood flow closes the aortic valve and slightly expands the ventricle until valve closure interrupts the expansion. The expansion is seen as a negative velocity at end systole, and the interruption of lengthening is reflected by the upstroke of the postejection velocity spike. In the normal heart, onset of shortening occurs simultaneously throughout the left ventricular wall as indicated by synchronous prejection velocity spikes in different left ventricular wall regions. Thus, investigation of prejection wall deformation may allow assessment of dysynchronous contraction and the effects of resynchronization therapy. Furthermore, because prejection and postejection velocity spikes reflect valve events, they may represent a means to detect timing of valve closure and opening.
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