Influence of Atrioventricular Interaction on Mitral Valve Closure and Left Ventricular Isovolumic Contraction Measured by Tissue Doppler Imaging

Annelies Decloedt, DVM, PhD; Tinne Verheyen, DVM, PhD; Stanislas Sys, MD, PhD; Dominique De Clercq, DVM, PhD; Bart Bijnens, PhD; Gunther van Loon, DVM, PhD

Background—The influence of atrioventricular (AV) interaction on mitral valve closure (MVC) and left ventricular (LV) isovolumic contraction is not fully clarified. We investigated the relationship among AV delay, MVC, and LV isovolumic contraction using a horse model because of the low heart rate and physiologically long AV delay.

Methods and Results—Six horses were evaluated during sinus rhythm, right ventricular pacing without preceding atrial contraction, and dual-chamber pacing at AV delays of 150 to 350 ms, programmed at a constant rate. Right parasternal 4-chamber views were recorded for simultaneous measurements of MVC from anatomic M-mode and radial tissue Doppler-based LV pre-ejection velocity and isovolumic acceleration. During sinus rhythm and long AV delays (≥300 ms), 2 positive pre-ejection velocity peaks were present. The first peak was identified as LV recoil during atrial relaxation and consistently preceded MVC by 33±17 ms. The second peak was related to LV isovolumic contraction, occurring after MVC. This suggests that MVC was caused by atrial relaxation and followed by true isovolumic contraction. During short AV delays (<300 ms) and right ventricular pacing, MVC occurred significantly later. Only 1 pre-ejection peak was present, of which the end coincided with MVC with a mean difference of -1.5±10 ms. This suggests that LV contraction caused MVC. Peak velocity and isovolumic acceleration were significantly higher (P<0.001) because the mitral valve was open at the onset of LV contraction.

Conclusions—Depending on the AV delay, MVC can be atrio- or ventriculogenic, resulting in significant alterations of the LV peak pre-ejection velocity and isovolumic acceleration. (Circ Cardiovasc Imaging, 2013;6:109-116.)

Key Words: contractility  ■ echocardiography  ■ mitral valve  ■ pacing

Left ventricular (LV) peak velocity and acceleration measured by tissue Doppler imaging (TDI) during isovolumic contraction (IVC) have been proposed as load-independent measurements of myocardial contractility. However, the myocardial mechanics during IVC have not yet been completely elucidated. Both longitudinal and transverse motion have been described, which resulted in a more spherical external LV shape. By TDI, a biphasic wall motion can be detected. This has been attributed to asynchronous deformation of the subendocardial and subepicardial myocardial layers, with subendocardial shortening in the right-handed helical direction accompanied by transient lengthening in the cross-fiber left-handed helical direction. The biphasic myocardial velocity spike might also be explained by interaction with mitral valve closure (MVC). Good agreement has been demonstrated between the timing of MVC and the interruption of pre-ejection shortening. In patients with severe mitral valve regurgitation, the peak pre-ejection velocity spike was significantly higher before valve surgery.

The exact mechanism of MVC is still debated. Convention holds that MVC is caused by the reversed flow after atrioventricular (AV) pressure crossover as a result of the LV pressure rise at the initiation of LV contraction. However, the left atrium (LA) might also play an important role. Reduced atrial contraction during occlusion of the proximal left circumflex coronary artery in sheep resulted in delayed MVC. This might be caused by the absence of inward flow during atrial contraction. The cessation of this forward flow has been described to cause negative pressures at the mitral valve leaflets. Furthermore, the presence of late diastolic vortices along the ventricular surfaces of the leaflets might facilitate leaflet appositioning. Recently, electromechanical coupling of the LA, mitral annulus, and mitral valve leaflets has been described. At the onset of IVC, annular area reduction and valve stiffening through contraction of myocytes in the basal portion of the anterior leaflet occur after atrial activation, by an electric connection through the AV-node.

Clinical Perspective on p 116

The aim of this study was to investigate the influence of the AV delay on MVC and its relationship with IVC as measured by TDI. The horse was used as an animal model because of its low heart rate at rest (25–45 bpm) and physiologically long...
AV delay, so that a wide range of AV delays could be studied using pacing. Measurements were performed during spontaneous sinus rhythm (SR), AV pacing with different AV delays, and right ventricular pacing (RVP) without preceding atrial contraction. Pre-ejection LV wall motion could be measured simultaneously with mitral valve motion using a color TDI 4-chamber view. We hypothesized that the AV delay would influence the time of MVC, and that this would affect the LV myocardial velocity and acceleration during IVC.

Methods

Experimental Preparation

This study included 6 horses (4 female, 2 male) with a mean body-weight of 568±63 kg. The experiment was approved by the ethical committee of the Faculty of Veterinary Medicine of Ghent University (2011/015). Animal handling and care were performed following those guidelines.

The study was performed on the standing horses at rest without sedatives. Two horses had a permanent implanted dual-chamber pacemaker (Thera DR 7960 and Kappa KDR 901, Medtronic, Minneapolis, MN). Four horses were instrumented with temporary pacing leads. Two introducer sheaths (Baxter Intro-Flex 8.5F, 1350BF85, Edwards Life Sciences, Irvine, CA) were placed in the proximal third of the right jugular vein after local anesthesia with procaine (Procaine hydrochloride 4%, VMD, Arendonk, Belgium). Two bipolar pacing catheters (Bipolar Intracardiac Electrode, USC Division, C.R. Bard Inc, Billerica, MA) were inserted and positioned in the right ventricular apex and the right atrium. Catheter placement was guided by analysis of the intracardiac ECG and echocardiography, and stable positioning of the catheters was checked repeatedly throughout the study. Both catheters were connected to an external pacing device (Programmer 9790, Medtronic, Minneapolis, MN). Pacing was performed at twice the diastolic threshold amplitude.

Pacing Protocol

Pacing was performed at a constant rate in excess of sinus rate. A stabilization period of 1 minute was allowed for each pacing modality before measurements were started. Measurements were performed during spontaneous SR, dual-chamber pacing at different AV delays, and RVP without preceding atrial contraction, programmed in a random sequence. The AV delay during dual-chamber pacing ranged from 150 to 350 ms (150–200–250–300–320–350 ms). For RVP, care was taken to obtain ventricular contractions that were not preceded by spontaneous atrial depolarization.

Echocardiography

Images were acquired using a Vivid 7 Dimension ultrasound machine equipped with a 35 Phased Array transducer (GE Healthcare, Horten, Norway). A base-apex surface ECG was recorded simultaneously. Color TDI loops of 4 consecutive cardiac cycles were acquired at a frequency of 1.7/3.4 MHz from a right parasternal 4-chamber view. The image depth was 28 cm, with a single focus positioned at 22 cm. The gray scale sector width was decreased to 45° and the velocity area of 12×4 mm was placed in the LV free wall at chordal level. The cine compound function was used to obtain an average of 3 consecutive cycles, and a 30 ms temporal smoothing filter was applied. Peak myocardial velocities were measured during late diastole and the pre-ejection period. Isovolumic acceleration (IVA) was calculated as the mean slope of the pre-ejection wave (v′<sub>max</sub>−v′<sub>max</sub>/acceleration time; unit m/s²), where v′<sub>max</sub> represents the maximal LV velocity during IVC and v′ the velocity at the onset of the pre-ejection wave. If 2 pre-ejection velocity spikes were present, IVA was measured from the second peak. If biphasic pre-ejection motion was present, the negative velocity peak occurring before ejection was called negative velocity peak. All timings were measured relative to onset QRS. Onset and end of the velocity peaks were measured where the curve crossed the 0 line or where a clear deviation occurred. The time between onset QRS and end of IVC was measured. Duration of the late diastolic peak was calculated as end diastole–onset diastole.

The time of MVC was measured as the time of leaflet coaptation in an anatomic color-coded TDI M-mode image through the mitral valve on the same loop as the TDI measurements. Similarly, an anatomic M-mode at chordal level was used for measuring fractional shortening, which was calculated from the LV internal diameter (LVID) measured at end-diastole (d) and end-systole (s): Fractional shortening = ([LVIDd–LVIDs]/LVIDd)×100. The time of AVO and aortic valve closure was measured from a long-axis LV outflow tract M-mode image. The pre-ejection period was defined as the time between the onset of QRS and AVO; the ejection time was defined as the time between AVO and aortic valve closure. From these measurements, the ratio of LV pre-ejection period and ejection time was calculated. The IVC time was calculated as the time between MVC and AVO. To minimize measurement variability, all recordings were performed and analyzed by 1 experienced echocardiographer (A.D.).

Statistical Analysis

Statistical analyses were performed using dedicated computer software (SPSS Statistics 19.0, Chicago, IL). Data are reported as raw means±SD. Pre-ejection peak velocity and IVA, TDI time measurements and timing of valve events during SR, RVP and pacing at different AV delays were compared by a linear mixed model with type of stimulation as a fixed categorical effect and with repeated measures on the horses as random subjects with compound symmetry variance structure. A global significance level of 0.05 was used; for all possible multiple comparisons the Bonferroni adjustment was applied. In addition, to compare either SR and long AV delays (≥300 ms) versus RVP and short AV delays (<300 ms) or long AV delays (≥300 ms) versus short AV delays (<300 ms), custom hypotheses were formulated and tested as linear contrasts within the linear mixed model. To compare timing of pre-ejection velocity peaks and timing of MVC, similar linear mixed models were adapted to the difference in timings.

Results

The resting heart rate in SR was 36±4 bpm with a mean PQ interval of 392±62 ms. Pacing was performed at 40 bpm in 4 horses and at 45 bpm in 2 horses. Good quality TDI images could be acquired at a frame rate of 105 fps in all horses. Radial late diastolic and pre-ejection LV velocity peaks could be easily identified. For each pacing mode, the PQ interval, TDI measurements, and M-mode measurements were tabulated in the Table. To compare timing of pre-ejection velocity peaks and timing of MVC, similar linear mixed models were adapted to the difference in timings.

Peak myocardial velocity during atrial contraction did not differ significantly between pacing modes (overall P=0.60). However, duration of atrial contraction was significantly shorter at AV delay 150 ms compared with longer AV delays and SR (P≤0.002), as atrial contraction was interrupted by MVC. During SR and long AV delays, 2 positive pre-ejection velocity peaks were present (Figure 1A). The first peak was identified as recoil of the LV during atrial relaxation. The second peak
coincided with LV IVC. During RVP and short AV delays, only 1 pre-ejection peak was present, which was called IVC as this peak occurred after the onset of QRS (Figure 1B). Peak IVC velocity was remarkably higher when only 1 peak was present during RVP and short AV delays (P<0.001, Figure 2A). IV A was significantly higher as well (P<0.001) vs *SR; †AV 350 ms; ‡AV 150 ms; and ¶RVP. For clarity, significant differences vs AV 200 ms to AV 320 ms have been omitted.

During short AV delays and RVP, MVC occurred after onset QRS (Figure 1B) and coincided with end IVC (Figure 3) with a mean difference of 1.5±10 ms, which was independent of the pacing mode (P=0.25). MVC occurred significantly earlier during long AV delays and SR (P<0.001) and was consistently preceded by recoil of the LV during atrial relaxation by 3±17 ms, independent of the pacing mode (P=0.63, Figure 3). Minor valve reopening during pre-ejection often occurred at long AV delays, mostly at 350 ms and during SR (Figure 1A). This resulted in double valve closure. The second closure was called MVC2 and coincided with end IVC with a mean difference of –3±12 ms, independent of the pacing mode (P=0.32).

A VO occurred latest during RVP and earliest during SR, with a trend of earlier AVO at longer AV delays. However, because of the significantly earlier timing of MVC at long AV delays and SR, the true isovolumic period (A VO–MVC) was significantly longer compared with short AV delays and RVP (P<0.001, Figure 4A). However, if MVC2 was considered for calculation of the true isovolumic period, no significant difference was present (Figure 4B). The ratio of LV pre-ejection period and ejection time as measured by M-mode was not altered by the AV delay during AV pacing but was significantly higher during RVP (P<0.001) and lower during SR (P=0.02). Fractional shortening was lower during long AV delays and SR compared with short AV delays and RVP (P=0.001).

**Discussion**

This study demonstrates the influence of AV interaction on the following:

1. The morphology of the pre-ejection velocity spikes;
2. The peak pre-ejection LV velocity and acceleration measured by TDI;
3. The time of MVC.

During SR and long AV delays, the TDI curves showed 2 pre-ejection peaks in the LV free wall. MVC was induced by atrial relaxation, later followed by a true isovolumic velocity peak. During RVP and short AV delays, MVC occurred significantly later and coincided with the end of IVC, suggesting the role of LV pressure development for MVC. The TDI curves showed 1 pre-ejection peak with a significantly higher velocity and acceleration.

### Table. ECG, TDI, and M-Mode Measurements

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Pacing Mode</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RVP</td>
</tr>
<tr>
<td>ECG</td>
<td></td>
</tr>
<tr>
<td>PQ interval</td>
<td>NA</td>
</tr>
<tr>
<td>TDI</td>
<td></td>
</tr>
<tr>
<td>A, cm/s</td>
<td>NA</td>
</tr>
<tr>
<td>durA, ms</td>
<td>NA</td>
</tr>
<tr>
<td>rA, cm/s</td>
<td>NA</td>
</tr>
<tr>
<td>nIVC (cm/s)</td>
<td>NA</td>
</tr>
<tr>
<td>MVC, ms</td>
<td>133±15†</td>
</tr>
<tr>
<td>MVC2, ms</td>
<td>NA</td>
</tr>
<tr>
<td>MVC2§</td>
<td>4</td>
</tr>
<tr>
<td>AVO, ms</td>
<td>199±28†‡</td>
</tr>
<tr>
<td>LVPEP/ET</td>
<td>0.57±0.11†</td>
</tr>
<tr>
<td>FS, %</td>
<td>38.1±1.4*</td>
</tr>
</tbody>
</table>

A indicates atrial contraction; AV, atrioventricular pacing; AVO, aortic valve opening; durA, duration of atrial contraction; end IVC, time between onset QRS and end IVC; FS, fractional shortening; IVC, isovolumic acceleration; MVC, mitral valve closure; MVC2, mitral valve closure after valve reopening; NA, not available; nIVC, negative velocity peak following IVC; rA, recoil of left ventricle during atrial relaxation; RVP, right ventricular pacing; SR, sinus rhythm; and TDI, tissue Doppler imaging.

§Number of horses in which the peak was present (on a total of 6); significant difference (P<0.05) vs *SR; †AV 350 ms; ‡AV 150 ms; and ¶RVP.
Morphology of the Pre-ejection Velocity Spikes
Radial myocardial velocity curves in normal adult humans usually show 1 positive pre-ejection peak. Similarly, 1 peak was present during pacing at short AV delays in this study. However, 2 peaks were observed during long AV delays. Similarly, pacing at a long AV delay (300 ms) in patients with third degree AV block resulted in 2 pre-ejection velocity waves, whereas pacing at a short (50 ms) and nominal (130 ms) delay revealed only 1 peak. The first pre-ejection peak can be explained by atrial relaxation or by passive recoil of the LV following expansion after atrial contraction. The second peak was identified as LV isovolumic contraction (IVC). Minor mitral valve reopening occurred during pre-ejection, resulting in double valve closure. The second closure was called mitral valve closure after valve reopening (MVC2) and coincided with end IVC. Biphasic pre-ejection motion was present, with IVC followed by a small negative velocity peak (nIVC). The time intervals of end IVC and MVC to onset QRS are indicated on top of the image. During dual-chamber pacing at an atrioventricular delay of 150 ms, MVC occurred significantly later. Only 1 positive pre-ejection velocity peak was present (IVC), of which the end coincided with MVC. Biphasic pre-ejection motion was present, with IVC followed by a large nIVC. The time intervals of end IVC and MVC to onset QRS are indicated on top of the image. AV indicates atrioventricular pacing.

Peak IVC Velocity and IVA Measured by TDI
Peak IVC velocity and acceleration were significantly influenced by AV interaction. Both measurements are often used as parameters of LV function. Peak IVC velocity is a strong marker of myocardial dysfunction in severely ischemic and dyskinetic myocardium. IVA has been proposed as a load-independent index of LV contractility. Changes in IVA during dobutamine and esmolol infusion were correlated to invasively measured systolic elastance, and IVA was unaffected by preload reduction within a physiological range. In patients, IVA remained unchanged during inferior vena cava occlusion or after closure of an atrial septal defect, whereas myocardial velocities were significantly altered. In contrast, other investigators revealed that IVA is not load-independent and can be altered by experimental volume loading and caval constriction. Similarly, this study demonstrated the influence of AV interaction on IVC velocities and acceleration, with higher peak IVC velocity and IVA during short AV delays and RVP. We hypothesize that this can be explained by the delayed MVC. As a consequence, the mitral valve is still open at the onset of contraction of the LV, which sees an initially lower afterload resulting in higher LV velocities.

The concept of a true IVC phase has been debated before. Goetz et al demonstrated that MVC does not occur until after 3-quarters of the pre-ejection period. Remme et al hypothesized that the myocardial pre-ejection velocity spikes might be caused by a simple mechanism. At the onset of systole, LV wall shortening closes the valve leaflets and moves them toward the LA. When leaflet motion is stopped by the chordae tendinae, wall shortening is interrupted which results in a biphasic spike on the TDI velocity curve. Our findings support this hypothesis. During short AV delays and RVP, MVC coincided with the end of

Figure 1. Example of a radial tissue Doppler imaging (TDI) velocity curve in the left ventricular free wall and an anatomic M-mode through the mitral valve, derived from the same right parasternal modified 4-chamber view. The x-axis shows time (1 second of the cardiac cycle). In the TDI velocity curve, A indicates the peak myocardial velocity during atrial contraction. Duration of A (durA) was measured as end-onset A. During dual-chamber pacing at an atrioventricular delay of 350 ms, 2 positive pre-ejection velocity peaks were present. The first peak was identified as left ventricular (LV) recoil during atrial relaxation (rA) and consistently preceded mitral valve closure (MVC). The second peak was identified as LV isovolumic contraction (IVC). Minor mitral valve reopening occurred during pre-ejection, resulting in double valve closure. The second closure was called mitral valve closure after valve reopening (MVC2) and coincided with end IVC. Biphasic pre-ejection motion was present, with IVC followed by a small negative velocity peak (nIVC). The time intervals of end IVC and MVC to onset QRS are indicated on top of the image. During dual-chamber pacing at an atrioventricular delay of 150 ms, MVC occurred significantly later. Only 1 positive pre-ejection velocity peak was present (IVC), of which the end coincided with MVC. Biphasic pre-ejection motion was present, with IVC followed by a large nIVC. The time intervals of end IVC and MVC to onset QRS are indicated on top of the image. AV indicates atrioventricular pacing.
IVC and was followed by a highly negative velocity peak. During long AV delays and SR, MVC occurred before IVC and negative velocity peak was often absent. However, other mechanisms have been proposed to explain the radial biphasic wall motion during IVC, such as layer-dependent deformation. Subendocardial fiber shortening accompanied by subepicardial fiber stretch results in wall thickening within the isovolumic constraint.\(^{18}\) The subepicardial pre-ejection stretch might be important to adjust the cardiac myosin power to variations in load.\(^{4}\)

**Mitrail Valve Closure**

MVC is initiated by LA/LV pressure crossover, but this can be caused both by increased LV pressure after LV contraction or by decreased LA pressure after LA relaxation. In patients paced at AV delays of 50 to 250 ms, MVC was correlated to onset of ventricular systole at short AV delays but this correlation was lost at long AV delays.\(^{19}\) Similarly, during short AV delays and RVP in our study, MVC was associated with LV isovolumic contraction. However, during long AV delays and SR, MVC was consistently associated with atrial relaxation and sometimes occurred before onset QRS. This indicates that MVC can be caused by atrial relaxation alone during long AV delays. Atrial relaxation results in a decreased LA pressure, deceleration of flow until flow reversal, and simultaneous movement of the valve leaflets toward closure.\(^{20}\)

In addition, presystolic mitral annular contraction facilitates valve closure by approximating the mitral leaflets. This annular reduction is functionally coupled to left atrial depolarization, and 89% of this reduction occurs before ventricular systole.\(^{21,22}\) Valve closure is also aided by anterior mitral valve leaflet stiffening caused by myocytes in the basal portion of the leaflets that are activated after atrial depolarization.\(^{11}\) However, leaflet stiffening and annular reduction seem to be induced by activation through the AV-node, suggesting that it plays a
role in the MVC associated with IVC rather than with atrial relaxation itself.

During long AV delays and SR, minor mitral valve reopening occurred after atriogenic MVC, followed by a second ventriculogenic MVC. Similarly, both atrial relaxation and ventricular contraction could close the valve at long AV delays in a patient with a prosthetic mitral valve and complete heart block, resulting in double closure. It is possible that atrial relaxation does not induce full closure of the mitral valve leaflets and that they are ultimately sealed by the rise in ventricular pressure. However, sustained MVC after atrial systole or diastolic mitral valve locking has also been described during pacing. Whether or not complete closure occurs after atrial relaxation could be investigated by evaluating diastolic mitral regurgitation (MR). Although late diastolic MR during reopening of the mitral valve has been described at prolonged AV delays in patients with implanted dual-chamber pacemakers, in patients with AV block, mitral and tricuspid diastolic regurgitation occurred about 240 to 330 ms after onset P, after the mitral valve reached near closure after atrial relaxation. It has been suggested that MR can also occur when ventricular contraction interrupts leaflet motion toward the ventricle during atrial contraction. This might have contributed to the increased fractional shortening values during RVP and pacing at short AV delays in our study. In sheep, it was shown that ventricular pacing resulted in a greater end-diastolic leaflet opening angle, delayed MVC, and had a higher regurgitant fraction compared with atrial pacing.

This might be particularly important in patients with atrial fibrillation (AF). During AF, both normal LA contraction and electric activation are absent. The absence of electric activation causes a loss of the presystolic mitral annular reduction. The altered mechanism of MVC during AF might be important in the pathogenesis of atrial function MR, a secondary, normal leaflet motion MR, which is often present in patients with AF, and an enlarged mitral annulus. In addition, LV peak IVC velocity and IVA might be different in AF compared with SR.

Finally, AV delay optimization is increasingly important in cardiac resynchronization therapy. Both patients with a short AV delay and blunting of the atrial contraction wave and patients with a prolonged AV delay and diastolic MR have a high likelihood of being a clinical cardiac resynchronization therapy responder. AV delay optimization results in changes in stroke volume and diastolic filling, which can be assessed by pulsed or continuous wave Doppler of the aortic or mitral valve. Peak IVC velocity or IVA might be additional parameters to consider as surrogates for assessing mitral valve dynamics (rather than contractility).

Limitations

Ventricular pacing possibly influenced the LV activation pattern and pre-ejection motion. Although this might complicate the comparison with SR, the pacing catheter position was stable throughout the study and did not interfere with the influence of the AV delay during dual-chamber pacing. Furthermore, no indications for severe dyssynchrony, such as a wide QRS complex or septal flash, were present. The sequence of pacing modes might also affect results. Therefore, pacing was performed in a random sequence, and each recording period was preceded by a 1 minute stabilization period. Invasive studies have demonstrated a stabilization period of 5 to 20 seconds sufficient to achieve hemodynamic equilibrium.
Apical images are impossible to obtain in adult horses because of anatomic restrictions. As a consequence, radial velocity measurements were performed from parasternal images, and accurate transmural flow measurements could not be achieved. Further investigation is needed to confirm whether these findings can be reproduced in a small mammal model using longitudinal velocity measurements. Invasive LA and LV pressure measurements would have provided additional information but are difficult to obtain in horses without sedation or anesthesia, which might in turn influence LV contractility.

Conclusions
AV interaction significantly influences the timing of MVC and pre-ejection LV wall motion. Depending on the AV delay, MVC can be atrio- or ventriculogenic, resulting in a significantly higher pre-ejection LV peak velocity and IVA, if the mitral valve is still open at the onset of LV contraction.

Sources of Funding
Annelies Decloedt was a PhD fellow of the Research Foundation Flanders (FWO-Vlaanderen).

Disclosures
None.

References
ventricular dyssynchrony is only one of multiple mechanisms. *Eur Heart J.* 2009;30:940–949.


**CLINICAL PERSPECTIVE**

Left ventricular peak isovolumic contraction velocity and acceleration measured by tissue Doppler imaging (TDI) have been proposed as load-independent measurements of myocardial contractility. However, it has been suggested that the biphasic wall motion measured by TDI is associated with mitral valve closure (MVC). We investigated the influence of the atrioventricular (AV) delay on MVC and left ventricular isovolumic contraction. The horse was used as an animal model because of its low resting heart rate (25–45 bpm) and physiologically long AV delay. During sinus rhythm and pacing at long AV delays, the TDI curves showed 2 pre-ejection peaks. MVC was associated with atrial relaxation, later followed by a true isovolumic velocity peak. Minor valve reopening during pre-ejection sometimes occurred at long AV delays, resulting in double valve closure. During right ventricular pacing and pacing at short AV delays, the TDI curves showed only 1 pre-ejection peak with a significantly higher velocity and acceleration. MVC occurred significantly later and coincided with the end of this peak, suggesting the role of left ventricular pressure development for MVC. The influence of AV interaction should be taken into account when assessing left ventricular pre-ejection velocity and acceleration and MVC. Peak isovolumic velocity and acceleration measured by TDI are not load-independent parameters because they are significantly influenced by the AV delay. In patients with atrial fibrillation, the absence of normal left atrial electric activation and contraction might result in an altered mechanism of MVC and different peak isovolumic velocity and acceleration compared with sinus rhythm.
Influence of Atrioventricular Interaction on Mitral Valve Closure and Left Ventricular Isovolumic Contraction Measured by Tissue Doppler Imaging
Annelies Decloedt, Tinne Verheyen, Stanislas Sys, Dominique De Clercq, Bart Bijnens and Gunther van Loon

Circ Cardiovasc Imaging. 2013;6:109-116; originally published online November 28, 2012; doi: 10.1161/CIRCIMAGING.112.978692
Circulation: Cardiovascular Imaging is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2012 American Heart Association, Inc. All rights reserved.
Print ISSN: 1941-9651. Online ISSN: 1942-0080

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circimaging.ahajournals.org/content/6/1/109

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation: Cardiovascular Imaging can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation: Cardiovascular Imaging is online at:
http://circimaging.ahajournals.org/subscriptions/