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

Decloedt et al: AV Delay Influences MVC and Isovolumic Contraction

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Abstract

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 between 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 (SR), right ventricular pacing without preceding atrial contraction (RVP) and dual-chamber pacing at AV delays of 150-350 ms, programmed at a constant rate. Right parasternal four-chamber views were recorded for simultaneous measurements of MVC from anatomical M-mode and radial tissue Doppler-based LV pre-ejection velocity and isovolumic acceleration (IVA). During SR and long AV delays (≥300 ms), two 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 RVP, MVC occurred significantly later. Only one 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 IVA were significantly higher (P<0.001) as 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 IVA.

Key Words: echocardiography, pacing, mitral valve, contractility
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.\textsuperscript{1, 2} 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.\textsuperscript{3} 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.\textsuperscript{4} 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.\textsuperscript{5} 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 following atrioventricular pressure cross-over due to the LV pressure rise at the initiation of LV contraction.\textsuperscript{6} 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.\textsuperscript{7} 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.\textsuperscript{8} Furthermore, the presence of late diastolic vortices along the ventricular surfaces of the leaflets might facilitate leaflet appositioning.\textsuperscript{9} 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 electrical connection through the AV-node.\textsuperscript{10, 11}
The aim of this study was to investigate the influence of the atrioventricular (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 ventricular pacing without preceding atrial contraction (RVP). Pre-ejection LV wall motion could be measured simultaneously with mitral valve motion using a color TDI four-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 six horses (four female, two male) with a mean bodyweight 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 their guidelines.

The study was carried out on the standing horses at rest without sedatives. Two horses had a permanent implanted dual-chamber pacemaker (Thera DR 7960i and Kappa KDR 901, Medtronic, Minneapolis, MN). Four horses were instrumented with temporary pacing leads. Two introducer sheaths (Baxter Intro-Flex 8.5Fr, I350BF85, 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, USCI 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 one minute was allowed for each pacing modality before measurements were started. Measurements were performed during spontaneous sinus rhythm (SR), dual-chamber pacing at different AV delays and right ventricular pacing without preceding atrial contraction (RVP), 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 3S Phased Array transducer (GE Healthcare, Horten, Norway). A base-apex surface ECG was recorded simultaneously. Color TDI loops of four consecutive cardiac cycles were acquired at a frequency of 1.7/3.4 MHz from a right-parasternal four-chamber view. The image depth was 28 cm, with a single focus positioned at 22 cm. The greyscale sector width was decreased to 45° and the velocity scale was set from -16 to +16 cm/s in order to obtain frame rates exceeding 100 frames per second (fps). As a result, the LV apex and LA dorsal wall were not imaged, however, the mitral valve was visualised throughout the entire cardiac cycle. Care was taken to ensure optimal alignment of the ultrasound beam with radial motion of the LV free wall at chordal level. For each pacing modality, additional M-mode recordings of aortic valve opening and closure were obtained from the right parasternal LV outflow tract long-axis view.

Off-line analysis

All measurements were performed off-line using commercially available software (EchoPAC Software Version 108.1.5, GE Healthcare, Horten, Norway). For radial TDI velocity measurements, a sample area of 12x4 mm was placed in the LV free wall at chordal level. The cine compound function was used to obtain an average of three consecutive cycles and a 30 ms temporal smoothing filter was applied. Peak myocardial velocities were measured during late diastole (A) and the pre-ejection period. Isovolumic acceleration (IVA) was calculated as the mean slope of the pre-ejection wave \(\frac{v_{\text{max}} - v_0}{\text{acceleration time}}; \text{unit m/s}^2\), where \(v_{\text{max}}\) represents the maximal LV velocity during IVC and \(v_0\) the velocity at the onset of the pre-ejection wave. If two 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 nIVC. All timings were measured relative to onset QRS. Onset and end of the velocity peaks were measured where the curve crossed the zero line or where a clear deviation occurred. The time between onset QRS and end of IVC was measured. Duration of the late diastolic peak (durA) was calculated as end A – onset A. The time of mitral valve closure (MVC) was measured as the time of leaflet coaptation in an anatomical color-coded TDI M-mode image through the mitral valve on the same loop as the TDI measurements. Similarly, an anatomical M-mode at chordal level was used for measuring fractional shortening (FS), which was calculated from the LV internal diameter (LVID) measured at end-diastole (d) and end-systole (s): FS = ((LVIDd − LVIDs) / LVIDd) * 100. The time of aortic valve opening (AVO) and closure (AVC) 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 AVC. From these measurements, the ratio of LV pre-ejection period and ejection time (LVPEP/ET) was calculated. The isovolumic contraction time (IVCT) was calculated as the time between MVC and AVO. To minimize measurement variability, all recordings were performed and analysed by one experienced echocardiographer (AD).

**Statistical analysis**

Statistical analyses were performed using dedicated computer software (SPSS Statistics 19.0, Chicago, IL). Data are reported as raw mean ± standard deviation (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 (LMM) 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 LMM. In order to compare timing of pre-ejection velocity peaks and timing of mitral valve closure, similar LMM’s were adapted to the difference in timings.
Results

The resting heart rate in sinus rhythm was 36±4 bpm with a mean PQ interval of 392±62 ms. Pacing was performed at 40 bpm in four horses and at 45 bpm in two 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 are tabulated in the Table.

Peak myocardial velocity during atrial contraction (A) did not differ significantly between pacing modes (overall P=0.60). However, duration of A was significantly shorter at AV delay 150 ms compared to longer AV delays and SR (P<0.002), as atrial contraction was interrupted by MVC. During SR and long AV delays, two positive pre-ejection velocity peaks were present (Figure 1A). The first peak was identified as recoil of the LV during atrial relaxation (rA). The second peak coincided with LV isovolumic contraction (IVC). During RVP and short AV delays, only one 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 one peak was present during RVP and short AV delays (P<0.001, Figure 2A). IVA was significantly higher as well (P<0.001, Figure 2B). The time of end IVC did not differ significantly between short and long AV delays (overall P=0.51). Biphasic pre-ejection motion was present in most horses during short AV delays and RVP, with IVC followed by a negative velocity peak (nIVC). During long AV delays and SR, this peak was often absent and significantly less negative if present (P=0.001, Figure 2C).

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 (rA) by 33±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 -0.3±12 ms, independent of the pacing mode (P=0.32).

AVO occurred latest during RVP and earliest during SR, with a trend of earlier AVO at longer AV delays. However, due to the significantly earlier timing of MVC at long AV delays and SR, the true isovolumic period (AVO-MVC) was significantly longer compared to 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).

LVPEP/ET 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). FS was lower during long AV delays and SR compared to short AV delays and RVP (P=0.001).
Discussion

This study demonstrates the influence of AV interaction on:

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 two 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 one pre-ejection peak with a significantly higher velocity and acceleration.

Morphology of the pre-ejection velocity spikes

Radial myocardial velocity curves in normal adult humans usually show one positive pre-ejection peak. Similarly, one peak was present during pacing at short AV delays in this study. However, two 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 two pre-ejection velocity waves, while pacing at a short (50 ms) and nominal (130 ms) delay revealed only one 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 true isovolumic contraction.

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 while myocardial velocities were significantly altered.\textsuperscript{15, 16} In contrast, other investigators revealed that IVA is not load-independent and can be altered by experimental volume loading and caval constriction.\textsuperscript{17} 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 isovolumic contraction phase has been debated before. Goetz et al.\textsuperscript{3} demonstrated that MVC does not occur until after three-quarters of the pre-ejection period. Remme et al.\textsuperscript{5} 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 left atrium. 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 IVC and was followed by a highly negative velocity peak (nIVC). During long AV delays and SR, MVC occurred before IVC and nIVC 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.\textsuperscript{18} The subepicardial pre-ejection stretch might be important to adjust the cardiac myosin power to variations in load.\textsuperscript{4}
Mitral valve closure

MVC is initiated by LA/LV pressure cross-over, but this can be caused both by increased LV pressure following LV contraction or by decreased LA pressure following 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.24 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 re-opening occurred after atrioventricular 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.23 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.24 Whether or not
complete closure occurs after atrial relaxation could be investigated by evaluating diastolic mitral regurgitation (MR). Although late diastolic MR was visualised by color flow Doppler in some horses, this regurgitation could not be quantified because accurate pulsed wave Doppler transmitral flow measurements could not be achieved from parasternal images.

**Clinical implications**

The influence of AV interaction should be taken into account when assessing MVC timing. At long AV delays, double closure of the mitral valve led to different measurements of IVCT depending on whether the first or second MVC was used for calculation. This can be clinically important when calculating the Tei index in patients with long AV delays, for example due to AV block. Late diastolic MR during re-opening of the mitral valve has been described at prolonged AV delays in patients with implanted DDD 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 following 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 FS 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 a higher regurgitant fraction compared to atrial pacing. This might be particularly important in patients with atrial fibrillation (AF). During AF, both normal LA contraction and electrical activation are absent. The absence of electrical 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 to sinus rhythm.
Finally, AV delay optimization is increasingly important in cardiac resynchronization therapy (CRT). Both patients with a short AV delay and blunting of the A wave and patients with a prolonged AV delay and diastolic MR have a high likelihood of being a clinical CRT responder.31 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.32 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 sinus rhythm, the pacing catheter position was stable throughout the study and this could thus 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.31 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 5–20 seconds sufficient to achieve hemodynamic equilibrium.33

Apical images are impossible to obtain in adult horses due to anatomical restrictions. As a consequence, radial velocity measurements were performed from parasternal images, and accurate transmitral 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.

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Disclosures
None.

References
Table. ECG, tissue Doppler (TDI) and M-mode measurements

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# number of horses in which the peak was present (on a total of 6); A, atrial contraction; AV, atrioventricular pacing; AVO, aortic valve opening; durA, duration of atrial contraction; end IVC, time between onset QRS and end IVC; IVA, isovolumic acceleration; IVC, isovolumic contraction; FS, fractional shortening; LVPEP/ET, pre-ejection period/ejection time; 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; SR, sinus rhythm; TDI, tissue Doppler; RVP, ventricular pacing.

Superscript indicates significant difference (P<0.05) versus \(^\text{a}\), SR; \(^\text{b}\), AV 350 ms; \(^\text{c}\), AV 150 ms; \(^\text{d}\), RVP. For clarity, significant differences versus AV 200 ms to AV 320 ms have been omitted.
Figure Legends

Figure 1. Example of a radial TDI velocity curve in the left ventricular free wall and an anatomical M-mode through the mitral valve, derived from the same right parasternal modified four-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. Panel A: During dual-chamber pacing at an atrioventricular delay of 350 ms, two positive pre-ejection velocity peaks were present. The first peak was identified as LV recoil during atrial relaxation (rA) and consistently preceded 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 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. Panel B: During dual-chamber pacing at an atrioventricular delay of 150 ms, MVC occurred significantly later. Only one 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 negative velocity peak (nIVC). The time intervals of end IVC and MVC to onset QRS are indicated on top of the image.

Figure 2. Graphical illustration of radial TDI velocity measurements of pre-ejection left ventricular wall motion. For each pacing mode, the median and spread of the peak value is indicated by a boxplot, with the box span indicating the middle half of the observations, the line in the box marking the median, and the whiskers indicating the range of observations within 1.5 times the interquartile ranges. Outliers are plotted individually at the end of the whiskers as open circles, extreme outliers are plotted as asterisks. (A) Peak velocity during
isovolumic contraction (IVC); (B) Peak acceleration of IVC (IVA); (C) Peak negative velocity following IVC (nIVC).

AV, atrioventricular; RVP, right ventricular pacing without preceding atrial contraction; SR, sinus rhythm

**Figure 3.** Timing of mitral valve closure (MVC), second mitral valve closure after minor valve reopening (MVC2), end of isovolumic contraction (end IVC) and recoil of the left ventricle during atrial relaxation (rA), relative to the onset of QRS measured on the ECG.

AV, atrioventricular; RVP, right ventricular pacing without preceding atrial contraction; SD, standard deviation; SR, sinus rhythm

**Figure 4.** Isovolumic contraction time (IVCT) calculated as time of aortic valve opening (AVO) – time of mitral valve closure (MVC), as measured by M-mode: (A) IVCT calculated as AVO – MVC; (B) IVCT calculated as AVO – MVC or as AVO – MVC2 if a second closure of the mitral valve (MVC2) was present after minor valve reopening during long atrioventricular delays.

AV, atrioventricular; RVP, right ventricular pacing without preceding atrial contraction; SD, standard deviation; SR, sinus rhythm
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