Effects of modulation of left ventricular contractile state and loading conditions on tissue Doppler myocardial performance index

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Cannesson, Maxime, Didier Jacques, Michael R. Pinsky, and John Gorcsan III. Effects of modulation of left ventricular contractile state and loading conditions on tissue Doppler myocardial performance index. Am J Physiol Heart Circ Physiol 290: H1952–H1959, 2006.—The myocardial performance index (MPI), first described by Tei et al. (34) in 1995, has been shown to be a predictor of global left ventricular (LV) (35) and right ventricular (1, 4, 11) function in various clinical settings. MPI combines elements of systolic and diastolic function and has been reported to be relatively independent of heart rate (16, 27), ventricular geometry (5), and blood pressure in steady states (34). An important limitation of MPI is that it is derived from LV inflow and outflow Doppler velocities from two separate imaging planes that cannot be recorded simultaneously (Fig. 1). Accordingly, MPI may be affected by changes in cardiac cycle length that may occur from data acquisition in one view to the other. Furthermore, rapid changes in contractility cannot be readily assessed. A new approach using tissue Doppler (TD) of the mitral annulus can rapidly calculate the MPI from a single beat; however, its ability to quantify contractility and the effects of acute changes in loading have not been determined. Our aim was to test the hypothesis that TD MPI can quantify contractile state but is affected by acute alterations in loading, using LV pressure-volume relations in an animal model. Eight dogs were studied using transmural annular TD, high-fidelity pressure, and conductance catheters. TD MPI was calculated as \( a’ - b’/b’ \), where \( a’ \) was the duration of mitral annular velocity during diastole and \( b’ \) was the duration of the systolic wave. End-systolic elastance (\( E_{es} \), the time constant of isovolumic relaxation (\( r \)), and peak positive and negative first derivative of pressure (\( dP/dt_{max} \) and \( dP/dt_{min} \), respectively) were used as measures of LV function. Data were obtained at baseline, at dobutamine and esmolol infusion to alter contractile state, and at inferior vena cava and aortic occlusion to alter preload and afterload. TD MPI decreased from 0.83 (SD 0.19) to 0.62 (SD 0.20) with dobutamine and increased to 1.19 (SD 0.26) with esmolol. TD MPI significantly correlated with \( dP/dt_{max} \) \((r = -0.76) \), \( E_{es} \) \((r = -0.68) \), \( dP/dt_{min} \) \((r = 0.82) \), and \( r \) \((r = 0.78) \); however, it was affected by acute decreases in preload [from 0.83 (SD 0.19) to 1.09 (SD 0.36) and acute increases in afterload [to 1.23 (SD 0.17)]. All the above increases and decreases in \( r \) values were significant \((P < 0.05 \) vs. baseline). In conclusion, TD MPI can rapidly quantify alterations in LV contractile state but is affected by acute alterations in preload and afterload.

THE MYOCARDIAL PERFORMANCE INDEX (MPI), first described by Tei et al. (34) in 1995, has been shown to be a predictor of global left ventricular (LV) (35) and right ventricular (1, 4, 11) function in various clinical settings. MPI combines elements of systolic and diastolic function and has been reported to be relatively independent of heart rate (16, 27), ventricular geometry (5), and blood pressure in steady states (34). An important limitation of MPI is that it is derived from LV inflow and outflow Doppler velocities from two separate imaging planes that cannot be recorded simultaneously (Fig. 1). Accordingly, MPI may be affected by changes in cardiac cycle length that may occur from data acquisition in one view to the other. Furthermore, rapid changes in contractility cannot be readily assessed. A new approach using tissue Doppler (TD) of the mitral annulus can rapidly calculate the MPI from a single beat; however, its ability to quantify contractility and the effects of acute changes in loading have not been determined. Our aim was to test the hypothesis that TD MPI can quantify contractile state but is affected by acute alterations in loading, using LV pressure-volume relations in an animal model. Eight dogs were studied using transmural annular TD, high-fidelity pressure, and conductance catheters. TD MPI was calculated as \( a’ - b’/b’ \), where \( a’ \) was the duration of mitral annular velocity during diastole and \( b’ \) was the duration of the systolic wave. End-systolic elastance (\( E_{es} \), the time constant of isovolumic relaxation (\( r \)), and peak positive and negative first derivative of pressure (\( dP/dt_{max} \) and \( dP/dt_{min} \), respectively) were used as measures of LV function. Data were obtained at baseline, at dobutamine and esmolol infusion to alter contractile state, and at inferior vena cava and aortic occlusion to alter preload and afterload. TD MPI decreased from 0.83 (SD 0.19) to 0.62 (SD 0.20) with dobutamine and increased to 1.19 (SD 0.26) with esmolol. TD MPI significantly correlated with \( dP/dt_{max} \) \((r = -0.76) \), \( E_{es} \) \((r = -0.68) \), \( dP/dt_{min} \) \((r = 0.82) \), and \( r \) \((r = 0.78) \); however, it was affected by acute decreases in preload [from 0.83 (SD 0.19) to 1.09 (SD 0.36) and acute increases in afterload [to 1.23 (SD 0.17)]. All the above increases and decreases in \( r \) values were significant \((P < 0.05 \) vs. baseline). In conclusion, TD MPI can rapidly quantify alterations in LV contractile state but is affected by acute alterations in preload and afterload.

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phy and hemodynamic data. For the conductance catheter, a 20-kHz constant-amplitude current of 0.03-mA root mean square between proximal distal electrode pairs was used with a data processor as described previously (Sigma 5DF; Leycom; Leyden, Netherlands) (7, 13). Changes in volume were sensed as a change in resistance in the cross-sectional area of each electrode pair, with the sum of all segments reflecting total volume. Parallel conductance was calculated by the hypertonic saline solution method and subtracted to measure left ventricular volume. All physiological signals were digitized at 150 Hz (WINDAQ Software; DATAQ Instruments; Akron, OH) and recorded on a computer.

Protocol. Hemodynamic and echocardiographic data were obtained simultaneously during end-expiratory apnea. The measurements were obtained during the following sequences: 1) baseline (control period), 2) transient IVC occlusion to decrease preload to minimum LV volume, 3) transient aortic occlusion to increase afterload by a 30- to 40-mmHg pressure elevation, 4) dobutamine infusion (5 µg·kg⁻¹·min⁻¹) to increase contractility, and 5) esmolol infusion (bolus of 500 µg and continuous infusion of 100 µg·kg⁻¹·min⁻¹) to decrease contractility. During each sequence, mitral annular velocities were recorded. The time for physiological data to return to baseline was observed between each manipulation.

Data analysis. Pressure-volume data were analyzed off-line. The maximal slope (left upper shoulder) of the end-systolic pressure-volume relation, maximal elastance (Es) during caval occlusion, LV end-diastolic volume, and peak positive and negative first derivative of LV pressure (dP/dtmax and dP/dtmin, respectively) were calculated. The time constant of isovolumic relaxation (τ) was calculated by the Weiss method (38). TD MPI was calculated as previously described (9). Accordingly, the TD MPI was calculated as (a' − b')/b', where

\[ \text{TD MPI} = \frac{a' - b'}{b'} \]

\[ \text{ICT, isovolumic contraction time; IRT, isovolumic relaxation time.} \]
RESULTS

Validation of ET for TD MPI. Simultaneous TD mitral annular velocity data were compared with aortic pressure data to confirm the correct timing and interpretation of the mitral annular systolic wave, because the TD mitral annular velocity profile in our open-chest animal model had a slightly different morphology with a more prominent isovolumic relaxation velocity than that usually observed in humans. The duration of the mitral annular systolic wave was compared with the time interval between the onset of the aortic pressure and the dicrotic notch on the aortic pressure tracing (Fig. 3) and correlated favorably \([r = 0.92, P < 0.001]\), Bland-Altman analysis bias = 16 ms (SD 15).

Effects of inotropic modulation. Dobutamine infusion induced a significant increase in both \(dP/dt_{max}\) \([1,892 (SD 504)\) to 4,713 mmHg/s (SD 1,008); \(P < 0.001\)] and \(E_{es}\) \([4.1 (SD 1.4)\) to 7.0 mmHg/ml (SD 1.8); \(P = 0.01\)], consistent with an increased contractile state (Fig. 4 and Table 1). Heart rate was also increased \([108 (SD 14)\) to 135 beats/min (SD 21); \(P = 0.01\)]. We also observed a decrease in \(dP/dt_{min}\) and \(\tau \approx -1,846\) (SD 500) to \(-2,970\) mmHg/s (SD 684), \(P = 0.004\); and 40 (SD 6) to 30 ms (SD 10), \(P = 0.003\), respectively, indicative of improved diastolic relaxation consistent with the lusitropic properties of dobutamine. TD MPI demonstrated a significant decrease from 0.83 (SD 0.19) to 0.62 (SD 0.2) \((P = 0.004)\), indicative of increased contractile state, associated with a significant decrease in total isovolumic time [from 164 (SD 33) to 114 ms (SD 44); \(P = 0.005\)] and total isovolumic time/(R-R interval)\(^{1/2}\) [from 219 (SD 37) to 166 ms (SD 51); \(P = 0.01\)]. No change in either ET [from 198 (SD 23) to 182 ms (SD 27); \(P = 0.14\)] or ET/(R-R interval)\(^{1/2}\) [from 265 (SD 25) to 269 ms (SD 30); \(P = 0.53\)] was observed (Fig. 5). Esmolol infusion resulted in a significant decrease in both \(dP/dt_{max}\) and \(E_{es}\) \([1,892 (SD 504)\) to 1,155 mmHg/s (SD 413), \(P = 0.01\); and 4.1 (SD 1.4) to 2.6 mmHg/ml (SD 1.1), \(P = 0.038\), respectively], consistent with a decrease in contractile state and associated with a significant decrease in heart rate from 108 (SD 14) to 96 beats/min (SD 9) \((P = 0.03)\). Similarly, both \(\tau\) and \(dP/dt_{min}\) were increased \([40 (SD 6)\) to 70 ms (SD 18), \(P = 0.001\); and \(-1,846\) (SD 500) to \(-1,206\) mmHg/s (SD 433), \(P = 0.01\), respectively]. TD MPI was significantly increased from 0.83 (SD 0.19) to 1.14 (SD 0.19) \((P = 0.004)\), consistent with decreased contractile state, with a significant increase in total isovolumic time [from 164 (SD 33) to 258 ms (SD 48); \(P < 0.001\)] and total isovolumic time/(R-R interval)\(^{1/2}\) [from 219 (SD 37) to 324 ms (SD 50); \(P < 0.001\)]. No change in either ET [from 198 (SD 23) to 219 ms (SD 30); \(P = 0.21\)] or ET/(R-R interval)\(^{1/2}\) [from 265 (SD 25) to 277 ms (SD 35); \(P = 0.51\)] was observed (Fig. 5). TD MPI correlated significantly with \(dP/dt_{max}\) \((r = -0.76; P < 0.001)\) and \(E_{es}\) \((r = -0.68; P = 0.001)\) (Fig. 6), as well as \(-dP/dt_{min}\) \((r = -0.82; P < 0.001)\) and \(\tau \approx 0.78; P < 0.01\) (Fig. 7).

Effects of acute alterations in preload and afterload. Acute decreases in preload induced by IVC occlusion were associated with a significant decrease in LV end-diastolic volume [123 (SD 33) to 111 mmHg (SD 33); \(P < 0.001\)] and mean arterial...
Fig. 4. Sequential pressure-volume loops (A–C, left) and corresponding mitral annular velocity recordings (A–C, right) at baseline (A), during dobutamine infusion (B), and during esmolol infusion (C) in 1 illustrative dog. TD MPI was inversely correlated to end-systolic elastance ($E_{es}$).

### Table 1. Changes in left ventricular tissue Doppler MPI and hemodynamic indexes during inotropic modulation

<table>
<thead>
<tr>
<th></th>
<th>Baseline</th>
<th>Dobutamine</th>
<th>Esmolol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tissue Doppler-MPI</td>
<td>0.83 (SD 0.19)</td>
<td>0.62 (SD 0.20)*</td>
<td>1.14 (SD 0.19)*</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>108 (SD 14)</td>
<td>135 (SD 21)*</td>
<td>96 (SD 9)*</td>
</tr>
<tr>
<td>Total isovolumic time, ms</td>
<td>164 (SD 33)</td>
<td>114 (SD 44)*</td>
<td>258 (SD 48)*</td>
</tr>
<tr>
<td>Total isovolumic time/R-R1/2, ms</td>
<td>219 (SD 37)</td>
<td>166 (SD 51)*</td>
<td>324 (SD 50)*</td>
</tr>
<tr>
<td>Ejection time, ms</td>
<td>198 (SD 23)</td>
<td>182 (SD 27)</td>
<td>219 (SD 30)</td>
</tr>
<tr>
<td>Ejection time/R-R1/2, ms</td>
<td>265 (SD 25)</td>
<td>269 (SD 30)</td>
<td>277 (SD 35)</td>
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<tr>
<td>Left ventricular dP/dt max, mmHg/s</td>
<td>1,892 (SD 504)</td>
<td>4,713 (SD 1,008)*</td>
<td>1,155 (SD 413)*</td>
</tr>
<tr>
<td>End-systolic elastance, mmHg/ml</td>
<td>5.8 (SD 2.3)</td>
<td>8.0 (SD 1.9)*</td>
<td>2.5 (SD 1.1)*</td>
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<td>Left ventricular dP/dt min, mmHg/s</td>
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</tr>
<tr>
<td>Time constant of relaxation, ms</td>
<td>40 (SD 6)</td>
<td>30 (SD 10)*</td>
<td>70 (SD 18)*</td>
</tr>
</tbody>
</table>

Values are means (SD). MPI, myocardial performance index; R-R, R-R time interval; dP/dt max and dP/dt min, peak positive and negative first derivative of pressure. *$P < 0.05$ compared with baseline.
pressure [133 (SD 22) to 97 mmHg (SD 18); P < 0.001], as expected, with no change in heart rate [108 (SD 14) to 109 beats/min (SD 15); P = 0.67] and a significant decrease in dP/dt max [1,892 (SD 504) to 1,326 mmHg/s (SD 345); P < 0.01] (Table 2). During IVC occlusion, TD MPI significantly increased from 0.83 (SD 0.19) to 1.09 (SD 0.36) (P < 0.05). This increase was related to a significant decrease in ET [from 198 (SD 23) to 175 ms (SD 15); P = 0.004] and ET/(R-R interval)1/2 [from 265 (SD 25) to 234 ms (SD 28); P = 0.004] with no change in either total isovolumic time [from 164 (SD 33) to 188 ms (SD 53); P = 0.11] or total isovolumic time/(R-R interval)1/2 [from 219 (SD 37) to 248 ms (SD 54); P = 0.08].

Acute increases in afterload induced by partial aortic occlusion significantly increased LV systolic pressure [138 (SD 28) to 176 mmHg (SD 43); P = 0.001] and mean arterial pressure [133 (SD 22) to 154 (SD 17) mmHg; P = 0.03], as expected, with no significant change in heart rate [108 (SD 14) vs. 97 beats/min (SD 12); P = 0.10] and no significant change in dP/dt max [1,892 (SD 504) to 1,791 mmHg/s (SD 431); P = 0.76]. Increased afterload increased TD MPI from 0.83 (SD 0.19) to 1.23 (SD 0.17) (P = 0.001), which was associated with a significant increase in total isovolumic time [164 (SD 33) to 241 ms (SD 16); P = 0.003] and total isovolumic time/(R-R interval)1/2 [from 219 (SD 37) to 305 ms (SD 53); P = 0.01] or ET/(R-R interval)1/2 [from 265 (SD 25) to 251 ms (SD 31); P = 0.33].

**Intraobserver and interobserver variability.** We found a 1.0% (SD 7.7) intraobserver and a 6.2% (SD 7.4) interobserver variability of measurement of TD MPI.

**DISCUSSION**

This study demonstrates that TD MPI can reliably detect alterations in LV function induced by inotropic modulation. TD MPI correlated favorably with invasive hemodynamic pressure-volume loop measurements of LV contractile state and diastolic relaxation in an experimental animal model. In addition, calculation of TD MPI was rapidly acquired and reproducible. We also observed that TD MPI was directly affected by acute changes in preload and afterload induced by IVC occlusion and partial aortic occlusion. Accordingly, TD MPI may have limitations in clinical scenarios associated with rapidly changing hemodynamic conditions.

The conventional MPI, first described by Tei et al. (34), is based on the recording of both mitral and aortic flows using pulsed Doppler. This index has been shown to be a useful clinical index of LV and right ventricular function (4, 33, 35). It is a unitless ratio of total isovolumic time (isovolumic contraction time + ET + isovolumic relaxation time) to ET, which can be recorded and measured by using routine conventional pulsed Doppler. Conventional MPI has gained clinical acceptance in assessing congenital cardiac disease (1, 22, 28, 29, 31, 39) and acquired cardiac disease, in particular as a useful predictor of clinical outcome in patients with cardiac
Amyloidosis (33), myocardial infarction (20), and dilated cardiomyopathy (26). The main advantages of this index are that it appears to be independent from ventricular geometry (5) and heart rate (16, 27, 34). Furthermore, it is a unique index of both systolic and diastolic (global) ventricular function (34). The MPI combines elements of LV systolic and diastolic function by reflecting systolic dysfunction in a shortened ET and a lengthened isovolumic contraction time and diastolic dysfunction in a lengthened isovolumic relaxation time (30). Consequently, an increased MPI indicates worsened LV performance, whereas a decreased MPI will indicate improved performance. An important feature of these previous investigations of MPI is that patients were studied during clinical stability with chronic disease states and steady-state hemodynamic conditions, unlike the acute loading alterations induced in our present animal study. One of the main limitations of the conventional MPI is that it cannot be calculated over a single cardiac cycle because the interval between the end and the onset of mitral inflow and the ET are measured sequentially. Accordingly, single beat analysis cannot be done. Cheung et al. (3) have recently shown that LV MPI was unable to detect acute change in LV contractile function induced by dobutamine or esmolol infusion in a porcine model (3). Because simultaneous LV inflow and outflow data cannot be recorded simultaneously, this limitation may account for these findings, although the precise reason is unknown.

TD recordings of the mitral annulus can be recorded on-line and provide the timing elements necessary to calculate TD MPI on a beat-to-beat basis (9, 10, 36). TD of the mitral annulus to describe LV function was first described by our group in 1996 (8) and is now widely available on most echocardiography devices. Mitral annular TD has subsequently been extended to the determination of diastolic function in a variety of cardiac diseases and also to estimated LV filling pressures, when expressed as the mitral inflow-to-annular velocity ratio (12, 23, 24, 32). The load dependence of the indexes derived from the mitral annulus velocities has long been questioned. Our group (12) and others (6) have shown that diastolic indexes derived from these velocities are load dependent. In addition, TD recordings of the mitral annulus velocities allow the determination of isovolumic relaxation time, ET, and isovolumic relaxation time over a single cardiac cycle in normal conditions in animals (30) and in humans (14, 17). TD MPI significantly correlates with conventional LV MPI, and a few clinical studies have already focused on this new index (25, 37, 40). Our results show that there should be a favorable relationship between these invasive indicators and TD MPI, suggesting that this index is a reliable indicator of LV global function. On the other hand, our results are consistent with most previously published studies (3, 15–18, 21) showing that MPI is affected by acute changes in loading conditions, such that reduced preload or increased afterload results in an increased TD MPI.

The cardiac hydraulic phenomena (mitral inflow and aortic outflow) are the consequences of the mechanical cardiac events...
(LV relaxation and contraction), clearly relating the routine MPI to the TD MPI. However, cardiac dysfunction may affect the coupling of the time intervals. For example, the difference in time to onset of early diastolic velocity of the mitral annulus and time to onset of mitral inflow is increased in the case of abnormal relaxation (30), suggesting that the assessment of isovolumic relaxation time using TD may be more sensitive of abnormal relaxation than conventional pulsed Doppler. Our data reported (see results) support a strong relationship and close agreement between time duration of the mitral annular systolic wave and ET assessed with the use of aortic pressure recordings at baseline and during dobutamine and esmolol infusion. TD MPI, however, appears to be similarly affected by changes in loading as does conventional MPI.

Study limitations. The animals in this study did not maintain a constant heart rate, and significant changes occurred during dobutamine and esmolol infusion. However, previous investigators (16, 27) have shown that MPI is relatively unaffected by heart rate. Furthermore, we attempted to correct ET and total isovolumic time values for R-R intervals to minimize confounding effects. In addition, potential effects of heart rate on total isovolumic time and ET are likely to be mathematically canceled when expressed as a ratio:

\[
\frac{\text{total isovolumic time}}{(R-R \text{ interval})^{1/2}} = \frac{\text{total isovolumic time}}{ET/(R-R \text{ interval})^{1/2}}
\]

Another limitation is that a direct comparison of routine MPI with TD MPI was not performed, although previous investigations have established this relationship. Another possible limitation of our study is that the values for TD MPI were higher than those reported in humans. We postulate that this was due, in part, to the myocardial depressing action of the anesthetic drugs in our open-chest preparation. Mancini et al. (19) observed similar elevated indexes at baseline with long isovolumic relaxation times in a canine model. Thus we can postulate that MPI and TD-MPI values may possibly be higher in dogs than in humans, although this is unknown. Because we examined changes in TD MPI from baseline using each animal as its own control, potential interspecies variations in the absolute value do not appear to affect our conclusions. Finally, we focused only on acute changes in loading, and the potential effects of chronic adaptation to loading on TD MPI remain unknown.

In conclusion, TD MPI is a simple and rapidly calculated index that can quantify alterations in LV contractile state. It has an advantage over the traditional MPI because it may be performed on a single beat from a single echocardiographic view. However, TD MPI is directly influenced by acute decreases in preload and acute increases in afterload, and both maneuvers are associated with increases in TD MPI. Accordingly, TD MPI may have limitations in comparing contractile states in patients with different loading conditions or in clinical scenarios associated with rapidly changing hemodynamic conditions, such as critically ill or hemodynamically unstable patients. In view of the significant preload and afterload sensitivity, the utility of this index for clinically assessing contractile state may potentially be limited. Accordingly, these data suggest that the TD MPI may be best suited as a measure of cardiac function in patients in steady-state hemodynamic conditions. Further analysis in humans is needed to show that the range of afterloads and preloads encountered clinically is within a narrow enough range that the impact of these factors is negligible compared with the impact of changes in contractility.

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GRANTS

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REFERENCES


Table 2. Changes in left ventricular tissue Doppler MPI and hemodynamic indexes during changes in loading conditions

<table>
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<td>164 (SD 33)</td>
<td>188 (SD 53)</td>
<td>241 (SD 16)*</td>
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<td>Total isovolumic time/(R-R interval)^{1/2}, ms</td>
<td>219 (SD 37)</td>
<td>248 (SD 54)</td>
<td>305 (SD 20)*</td>
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Values are means (SD). *P < 0.05 compared with baseline.


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