Left ventricular systolic torsion correlates global cardiac performance during dyssynchrony and cardiac resynchronization therapy

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Abstract:

Introduction. Left ventricular (LV) systolic torsion is a primary mechanism contributing to stroke volume. We hypothesized that change in LV torsion parallels changes in global systolic performance during dyssynchrony and cardiac resynchronization therapy (CRT).

Methods. Seven anesthetized open-chest dogs had LV pressure-volume relations. Apical, basal and mid-LV cross-sectional echocardiographic images were studied by speckle tracking analysis. Right atrial (RA) pacing served as control. Right ventricular (RV) pacing simulated left bundle branch block (LBBB). Simultaneous RV-LV free wall and RV-LV apex pacing (CRTfw and CRTa) modeled CRT. Dyssynchrony was defined as the time difference in peak strain between earliest and latest segments. Torsion was calculated as the maximum difference between apical and basal rotation.

Results. RA pacing had minimal dyssynchrony (52±36 ms). RV pacing induced dyssynchrony (189±61 ms, p<0.05). CRTa decreased dyssynchrony (46±36 ms, p<0.05 vs. RV pacing) whereas CRTfw did not (110±96 ms). Torsion during baseline RA was 6.6±3.7°. RV pacing decreased torsion (5.1±3.6°, p<0.05 vs. control), reduced stroke volume (SV), stroke work (SW), and dP/dt max compared to RA (21±5 vs. 17±5 mL, 252±61 vs. 151±64 mJ, and 2063±456 vs. 1603±424 mm Hg·s⁻¹, respectively, p<0.05). CRTa improved torsion, SV, SW and dP/dt max compared to RV pacing (7.7±4.7°, 23±3 mL, 240±50 mJ and 1947±647 mm Hg·s⁻¹, respectively, p<0.05) whereas CRTfw did not (5.1±3.6°, 18±5 mL, 175±48 mJ 1699±432 mm Hg·s⁻¹, respectively, p<0.05). LV torsion changes co-varied across conditions with SW (y=0.94x+12.27, r=0.81, p<0.0001) and stroke volume (SV) (y = 0.66 x+0.91, r = 0.81, p<0.0001). LV dyssynchrony changes did not correlate with SW or SV (r=-0.12, p=0.61 and r=0.08, p=0.73 respectively).
Conclusions. LV torsion is primarily altered by dyssynchrony and CRT that restores LV performance also restores torsion.
**Introduction**

Several recent studies have shown that left ventricular (LV) torsion contributes directly to systolic LV function (12, 22, 24, 32) although previously some authors (6) had concluded that LV function was unaffected by the twisting phenomenon. LV torsion is created by shortening of myofibrils arranged in a helical orientation and is visualized as counter clockwise apical rotation and the clockwise basal rotation relative to a stationary mid-myocardial reference point. There is a systolic twist (torsion) and an early diastolic untwist during the cardiac cycle (15, 16, 35). LV torsion has been measured non-invasively using tissue tagging magnetic resonance imaging (MRI) (3, 7, 8, 25, 26, 37, 40, 41), 2-D echocardiography (2, 23) and Doppler tissue imaging (30) in various cardiac diseases. Recently, speckle tracking echocardiography has been validated as an accurate measurement of LV rotation and torsion by comparison with sonomicrometry in dogs and MRI tagging in humans (17, 29, 38,42). LV contraction dyssynchrony is a common contraction abnormality and plays a major role in the pathophysiology of heart failure (1, 4, 5, 11, 13, 14, 19, 28, 34, 36, 43, 44, 45). Cardiac resynchronization therapy (CRT) can reverse dyssynchrony and improve global cardiac function in some but not all patients. The non-invasive measurement of LV torsion during dyssynchrony and CRT, and its relationship with global cardiac performance has not been described. Considering the central role torsion plays in global LV performance, we hypothesized that LV torsion is impaired during LV contraction dyssynchrony and restored by CRT if global LV performance also improves.

**Methods**

*Preparation:* Seven mongrel male dogs, weighing 20.6±1.5 kg were studied after an overnight
fast. The protocol was approved by the institutional animal care and use committee and conformed to the position of the American Heart Association on research animal use. All dogs were anesthetized with sodium pentobarbital (30 mg/kg induction; 1.0 mg/kg/h with intermittent boluses, as needed) and mechanically ventilated. A 6F 11-pole multi-electrode conductance catheter (Webster Laboratories, Irvine, California) and a left ventricular (LV) micromanometer catheter (MPC-500, Millar, Houston, Texas) were placed for LV pressure-volume analysis by the right internal carotid artery and the left common carotid artery, as previously described (11).

After a median sternotomy, a snare occluder was placed around the inferior vena cava to transiently alter preload. The pericardium was opened and temporary epicardial pacing wires (A & E Medical Corp. Farmingdale, NJ, USA) were placed on the right atrium (RA), right ventricular (RV) free-wall near the anterior infundibulum, LV mid-free-wall near the mid-posterior-lateral wall, and LV apex for multi-site stimulation. The pericardium was re-opposed with multiple interrupted sutures and positive end-expiratory pressure (PEEP) applied to re-expand the lungs. Afterward, 5 cm H2O PEEP was applied to maintain end-expiratory lung volume for the remainder of the experiment. Fluid resuscitation was performed prior to starting the protocol to restore apneic LV end-diastolic volume to values similar to where they were prior to sternotomy.

Hemodynamic Data Analysis: LV pressure, volume and electrocardiogram signals were digitized at 250 Hz and stored on disk for offline analysis. The following hemodynamic parameters were assessed for global LV performance: LV peak systolic pressure, stroke volume (SV) and stroke work (SW) as the integral of the LV pressure-volume loop.
**Echocardiography:** An echocardiographic system (Aplio SSA-770A, Toshiba Medical Systems Corp, Tokyo, Japan) was used to obtain images with a 3.0 MHz transducer directly applied to the heart. Digital routine grayscale 2-D and tissue Doppler cine loops from 3 consecutives beats were obtained at end-expiratory apnea from basal-LV short axis view and apical LV short axis view at depths of 8 cm using a fixed transducer position. Gray scale images were collected at frame rates of 49 Hz with a pulse repetition frequency of 4.5 kHz. Gain settings were adjusted to optimize endocardial definition. We defined the proper short axis levels as follows (30): at the basal level, the mitral valve and, at the apical level, LV cavity alone with no visible papillary muscles. Mid-LV short axis views were selected with the papillary muscle as a consistent anatomic landmark. The LV cross section was made as circular as possible. We used customized software within a personal computer workstation (ApliQ, Toshiba Corp.) for off-line analysis of speckle tracking imaging. Offline analysis of apical and basal rotation was then performed on digitally stored images (ApliQ, Toshiba Corp.). LV torsion was defined as a net-difference of LV rotation between apical and basal short axis planes. Normally, the apex rotates counter clockwise whereas the base rotates clockwise when viewed from apex. Counter clockwise LV rotation as viewed from apex was expressed as a positive value and clockwise LV rotation was expressed as a negative value (Fig. 1).

**LV rotation by Speckle Tracking Imaging:** The speckle tracking analysis was used to generate regional LV strain from routine B-mode grayscale echocardiographic images. The best quality digital 2-D cardiac cycle was selected. A circular region of interest was traced on the endocardial and epicardial border of the short axis view, using a point-and-click approach. Speckles within the region of interest were tracked in subsequent frames by the imaging
software. The location shift of these speckles from frame to frame, representing tissue movement, provided the spatial and temporal data. The workstation computes LV rotation of each short axis image. Averaged LV rotation data on the mid-myocardial contour was used for the calculation of LV torsion. The basal and apical LV rotation speckle tracking imaging data were exported to a spreadsheet program (Excel 2000, Microsoft Corp, Seattle, Washington) to calculate LV torsion. Counter clockwise rotation was represented as positive value, color-coded as blue, and clockwise rotation was represented as negative value, color-coded as red. The software divided the short-axis image into 6 equal segments. The LV torsion was calculated during RA pacing, as heart rate control, and compared to RV pacing, RA-RV-LV free wall pacing and RA-RV-LV apex pacing to reflect LV free wall CRT (CRTfw) and apical CRT (CRTa), respectively. Time-to-peak strain for each of 6 regional (9) time-strain curves for each cross-sectional LV study was determined. Dyssynchrony was defined as the time difference in peak strain between earliest and latest segments, as previously described (20).

Protocol: All measurements were made during apnea with 5 cm H\textsubscript{2}O positive end-expiration pressure. To avoid retrograde conduction for all pacing steps of the protocol, RA pacing was performed at frequencies 5-10 min\textsuperscript{-1} above the intrinsic rhythm. To control for any heart rate-specific changes in global and regional function, RA pacing was defined as normal ventricular contraction for subsequent comparisons. All succeeding ventricular pacing studies were then done with sequential pacing at an A-V delay of 20 ms. This pacing delay prevented atrial fusion beats from contaminating the ventricular pacing effects of CRT but also eliminated atrial
contraction from augmenting LV filling. High RV free-wall pacing was used to induce a LBBB-like contraction pattern (20). We then compared the impact of CRTfw and CRTa on regional and global LV performance. The order of CRTfw and CRTa was alternated among sequential animals to eliminate any sequencing effects. Pacing was maintained for > 30 seconds before measurements were made for each step so that hemodynamic equilibrium could be established. In practice, hemodynamic stability usually took < 15 seconds to occur. Between each ventricular-paced rhythm interval, the animals were returned to RA pacing and all hemodynamic variables were stabilized to baseline levels before the next step in the protocol was initiated.

Statistical Analysis: Data are expressed as mean ± SD. Analysis of variance for repeated measures was used for comparisons among different pacing modalities. One way ANOVA with repeated measures and post-hoc testing was used to evaluate the effects of different pacing modes on torsion and indexes of global LV performance. Significance was determined as p < 0.05. Linear regression analysis was used to compare changes in left ventricular torsion and changes in stroke work and stroke volume. Inter-observer variability was assessed in ten randomly selected studies for torsion and was calculated as the ratio (expressed as a percentage) of the difference between the values obtained by each observer (expressed as absolute value) divided by the mean of the two values. Intra-observer variability was calculated by a similar approach.

Results

Baseline radial strain dyssynchrony and changes in LV Dyssynchrony during different pacing modes: The maximum time difference from earliest to latest peak strain among 6 segments, as a
measure of contraction synchrony, was minimal with RA pacing (52±36 ms), but increased
during RV pacing (189±61 ms, p<0.05 vs. RA pacing) (Table 1). CRTa reduced maximal time
difference in peak strain compared to RV pacing (46 ± 36 ms, p<0.05 vs. RV pacing) whereas
CRTfw did not alter it compared to RV pacing (110±96 ms). Changes in LV dyssynchrony from
baseline did not correlate with changes in stroke work (y= - 20 x + 1175, r =-0.12, p=0.61) or
stroke volume (y= - 14 x + 1554, r =-0.08, p=0.73).

Baseline LV Torsion and changes in LV torsion during different pacing modes: Figure 2 and
Figure E1 (video) show a typical case of counter clockwise apical rotation, clockwise basal
rotation and torsion during RA pacing, RV pacing, CRTa and CRTfw.

Torsion analysis without pacing but from a lower heart rate was done and all torsion values were
similar to that of the RA baseline values (6.6±3.7 ° v. 6.6±3.7 °; sinus rhythm v. RA).
LV torsion was reduced during RV pacing compared to RA pacing: 5.1±3.6° vs. 6.6±3.7°,
p<0.05. CRTa improved LV torsion compared to RV pacing, (7.7±4.7 ° vs. 5.1±3.6 °, p<0.05),
whereas CRTfw had no effect on LV torsion compared to RV pacing (5.1±3.6 ° vs. 5.1±3.6 °)
(Table 1 and Figure 3). Changes in LV torsion from baseline correlated significantly with
changes in stroke work (r=0.81, p<0.0001) (Figure 4) and stroke volume: r=0.81, p<0.0001 and y
= 0.66 x + 0.91. Although the absolute change in torsion, in degrees, was small, with a mean
decrement in torsion from RA to RV of only -1.5°, decrements in LV SW of >20% were
universally associated with a decrease in LV torsion. Changes in LV torsion did not correlate
with changes in LV dyssynchrony (y= -0.001x -6.68, r = -0.23, p=0.30).
Effects of pacing on global LV function: RV pacing decreased LV SV and SW compared to RA. Whereas CRTa significantly improved LV SV and SW as compared to RV pacing, while CRTfw did not alter either LV SV or SW (Table 1).

Reproducibility: Intra- and inter-observer variability was analyzed in 8 randomly selected studies for torsion values by speckle tracking analysis using standard 6 segments. Intra- and inter-observer variability for torsion expressed as the mean percent error (absolute difference/mean) was 7 ± 7% and 8 ± 9%, respectively.

Discussion

Our study has two primary findings. First, LV torsion, quantified by speckle tracking, is impaired by dyssynchronous contraction and improved by CRT only if CRT also improves cardiac performance. Second, the degree of change in torsion parallels the degree of change in LV SW across all pacing modes, with the greatest sensitivity coming from decrements in LV SW of >20% always associated with a decrease in LV torsion compared to baseline. The implication of these data is that torsion, a primary cardiac contraction variable, is not only affected by dyssynchrony and CRT but can be used to tract CRT effectiveness. Changes in dyssynchrony do not parallel the degree of change in LV SW.

Left ventricular torsion has been studied using cardiac magnetic resonance (CMR) in previous studies (26,27,46). However, CMR data acquisition is cumbersome and its post acquisition data analysis laborious. Two-dimensional speckle tracking echocardiography imaging when collected at apical and basal planes enables the non-invasive measurement of apical and basal rotation and
then to calculate LV torsion. This speckle tracking technique has been validated against CMR
(17, 30, 38). These findings should not be surprising, because LV torsion is a sensitive marker of
global LV function (10, 24, 26, 27, 42). LV torsion correlates with the percentage change in LV
area (20) in humans and to stroke volume and ejection fraction in animals (10). Furthermore, we
previously showed in this canine model that LV contraction dyssynchrony can be quantified as
regional differences in radial strain wherein the principal segment displaying maximal
dyssynchrony defined most of the impaired ejection effectiveness (20).

We and others have previously shown that in the setting of dyssynchrony contraction the slope of
the end-systolic pressure volume relationship (Ees) may vary independent of stroke work (18,
20). In the present study we showed that Ees does not correlate with changes in strain (r=0.1464
and p=0.5622). Not surprisingly in the present study the relationship between Ees and torsion is
also poor (r=-0.3035 and p=0.2207). Although torsion does not correlate with Ees, it does with
both stroke work and stroke volume (r=0.81 and 0.81, p<0.0001 respectively).

Limitations: There are some limitations of our study. First we studied an intact canine model
without intrinsic conduction defects or impaired contractility in which dyssynchrony was created
by ventricular pacing. Extrapolation to clinical studies regarding site selection for optimal CRT
cannot be made. For example, we (18, 20), and others (31), have documented that LV apical
CRT is superior to LV free wall CRT in terms of global LV performance and resynchronization
in animal models of pacing-induced dyssynchrony. However, similar apical pacing superiority
has not been reported in human CRT studies. The apex may be better than free wall in otherwise
healthy animals because apical pacing can activate the His Purkinje system more centrally than
occurs with free wall pacing. Second torsion changes induced by pacing in an acute open chest
animal may not be the same as under conditions of chronic heart failure induced by arrhythmias
in humans. However we (20, 39) recently showed similar regional strain activity in an intact
chronic heart failure model as that seen in our acute open chest animal model. It is unlikely that
torsion would be qualitatively different than regional strain in chronic heart failure. Third, we
manually placed the echo probe at specific points along the long axis to get the apical and basal
rotation needed to calculate torsion. Small variations to probe distance between animals may be
responsible for the variation in absolute torsion seen across animals. Putting markers on the heart
to define apical and basal sites would have increased the consistency of measurement within
animals across pacing modes but would not address the issue of longitudinal differences in
positioning across animals. Since we used an external fixed device to hold the echo probe at the
same point and orientation across pacing modes all within animal measures were constant across
pacing modes. Longitudinal probe sensing differences may exist between animals may explain
why in one of our animal the degree of torsion was higher than in others. However, even in this
example the qualitative changes in torsion with pacing were similar across all animals.

Finally, speckle tracking echocardiography is dependent on frame rates, as well as image
resolution. Low acquisition frame rates degrade assessment of regional myocardial motion and
its subsequent strain rate analysis. In contrast, increasing frame rate reduces scan-line density,
which reduces image resolution (21, 33). Suffoletto et al. (36) found that frame rates in the range
of 30 to 90 Hz with a mean of 65 Hz suitable for speckle tracking analysis. Thus, in our study we
used a mean frame rate of 49 Hz suitable for speckle tracking analysis.
Acknowledgments

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Figures legends

Figure 1. An example of basal and apical rotations and left ventricular (LV) torsion during right atrial pacing. Counterclockwise rotation is color-coded as blue, and clockwise rotation is color-coded as red. As view from the LV apex, the LV apex rotates counterclockwise expressed as a positive value, whereas the LV base rotates clockwise expressed as a negative value. Averaged apical and basal rotation data were used for the calculation of LV torsion. LV torsion was calculated as the maximum difference between LV rotation angles obtained from basal and apical planes.

Figure 2. An example of basal rotation (green), apical rotation (blue) and torsion (red) for one animal during control (RA pacing), RV pacing and both CRTa and CRTfw. Maximum (Max.) torsion is the maximal difference between apical and basal rotation in degrees.

Figure 3. Individual left ventricular torsion across pacing modes. Individual values of left ventricular torsion using speckle tracking from RA pacing, RV pacing, CRTfw and CRTa. *p<.05 RA pacing vs. RV pacing, †p<.05 CRTa or CRTfw vs. RV

Figure 4. Linear regression plot of changes in left ventricular torsion and changes in stroke work across pacing modes demonstrating a significant correlation. Circles represent changes from RA pacing to RV pacing, triangles represent changes from RA pacing to CRTfw, solid squares represent changes from RA pacing to CRTa.

Electronic Figure 1. Video examples of basal and apical rotations and left ventricular (LV) torsion during the four separate figures: RA pacing, RV pacing, CRTfw and CRTa.
Counterclockwise rotation is color-coded as blue, and clockwise rotation is color-coded as red. As view from the LV apex, the LV apex rotates counterclockwise expressed as a positive value, whereas the LV base rotates clockwise expressed as a negative value. Averaged apical and basal rotation data were used for the calculation of LV torsion. LV torsion was calculated as the maximum difference between LV rotation angles obtained from basal and apical planes. Note that with RV pacing the torsion is minimized and restored only by CRTa.
Table 1- Left ventricular hemodynamic characteristics and LV Torsion during, right atrial (RA), RA-right ventricular (RV) pacing and simulated apical and left ventricular free wall cardiac resynchronization therapy (CRTa and CRTfw, respectively).

<table>
<thead>
<tr>
<th></th>
<th>RA Pacing</th>
<th>RV Pacing</th>
<th>CRTa</th>
<th>CRTfw</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR, beats/min</td>
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<td>139 ± 8</td>
<td>139 ± 8</td>
<td>139 ± 8</td>
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<tr>
<td>MAP, mmHg</td>
<td>96 ± 11</td>
<td>78 ± 13*</td>
<td>83 ± 11</td>
<td>83 ± 11</td>
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<tr>
<td>LV ESP, mm Hg</td>
<td>109 ± 8</td>
<td>92 ± 13*</td>
<td>94 ± 5</td>
<td>95 ± 8</td>
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<tr>
<td>LV EDP, mm Hg</td>
<td>12 ± 5</td>
<td>11 ± 5*</td>
<td>9 ± 5†</td>
<td>10 ± 5†</td>
</tr>
<tr>
<td>EDV, mL</td>
<td>40 ± 3</td>
<td>34 ± 5*</td>
<td>34 ± 5</td>
<td>34 ± 5</td>
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<td>SV, mL</td>
<td>21 ± 5</td>
<td>17 ± 5*</td>
<td>23 ± 3†</td>
<td>18 ± 5</td>
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<tr>
<td>CO, L.min⁻¹</td>
<td>2.9 ± 0.8</td>
<td>2.3 ± 0.5*</td>
<td>3.2 ± 0.5†</td>
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<tr>
<td>SW, mJ</td>
<td>252 ± 61</td>
<td>151± 64*</td>
<td>240 ± 50 †</td>
<td>175 ± 48</td>
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<tr>
<td>dP/dt max, mm Hg.sec⁻¹</td>
<td>2063 ± 456</td>
<td>1603 ± 424 *</td>
<td>1946 ± 647 †</td>
<td>1699 ± 432</td>
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<tr>
<td>dP/dt min, mm Hg.sec⁻¹</td>
<td>-2325 ± 464</td>
<td>-1684 ± 482*</td>
<td>-2061 ± 440 †</td>
<td>-1973 ± 472 †</td>
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<td>Ees, mm Hg.mL⁻¹</td>
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<td>2.9 ± 1.6‡</td>
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<td>LV Torsion, degrees</td>
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<td>5.1 ± 3.6*</td>
<td>7.7 ± 4.7‡</td>
<td>5.1 ± 3.6</td>
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<tr>
<td>Dyssynchrony, msec</td>
<td>52 ± 36</td>
<td>189 ± 61</td>
<td>46 ± 36†</td>
<td>110 ± 96</td>
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</table>

Data are mean ± SD; n = 7. RA, right atrial; RV, right ventricular; CRTa, cardiac synchronization therapy (CRT) at left ventricular (LV) apex; CRTfw, CRT at LV free wall; HR, heart rate; MAP, mean arterial pressure; LV EDP, LV end-diastolic pressure; LV ESP, LV end-systolic pressure; LV EDV, LV end-diastolic volume; SV, stroke volume; CO, cardiac output; dP/dt max, maximum
rate of change of LV pressure; dP/dt\textsubscript{min}, minimum rate of change of LV pressure; SW, LV stroke work, Ees, end-systolic elastance. * p<.05 RA vs. RV, † p<.05 vs. RV.

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RV pacing

RA pacing

Maximal Torsion 5°

Maximal Torsion 2°

Maximal Torsion 7°

Maximal Torsion 3°
$y = 0.94 x + 12.27$

$r = 0.81, p < 0.0001$

○ Change from RA pacing to RV pacing
△ Change from RA pacing to CRT fw
■ Change from RA pacing to CRT a
Apical Rotation during RA pacing

Basal Rotation during RA pacing
Apical Rotation during RARV pacing

Basal Rotation during RARV pacing
Apical Rotation during CRTa

Basal Rotation during CRTa
Apical Rotation during CRTfw

Basal Rotation during CRTfw