Mechanics of the Single Left Ventricle
A Study in Ventricular-Ventricular Interaction II

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Background—Left ventricular (LV) effects on right ventricular (RV) function are well known. Less is understood about the effect of the RV on systemic LV mechanics. To determine this interaction, we compared systemic LVs with and without an RV mechanically coupled to them.

Methods and Results—MR myocardial tagging was used to examine 18 subjects with systemic LVs: 10 with functional single LVs (SLV) and 8 normal subjects (NL). Tracking the systolic motion of the intersecting stripes were used to determine regional twist and radial motion. Finite strain analysis was applied to derive principal strains at the atrioventricular valve (AVV) and apical short-axis levels and in 4 anatomic wall regions. Similar $E_1$ (circumferential shortening) strain and heterogeneity of strain were noted between SLV and NL except in the septal wall. At the septal wall, NL displayed greater absolute strain (AVV $= -0.16 \pm 0.02$, apex $= -0.17 \pm 0.02$) and less heterogeneity of strain than SLV (AVV $= -0.12 \pm 0.02$, apex $= -0.13 \pm 0.02$). Similar $E_2$ (wall thickening) strain and heterogeneity of strain were also noted between SLV and NL except again at the septal wall. At the septal wall, SLV displayed greater absolute $E_2$ strain (AVV $= 0.17 \pm 0.08$, apex $= 0.19 \pm 0.09$) and less heterogeneity of strain than NL (AVV $= 0.07 \pm 0.07$, apex $= 0.05 \pm 0.05$). SLV twisted significantly less counterclockwise than NL in 6 of 8 wall regions and actually twisted clockwise at the AVV lateral wall. Although there was no significant difference between groups in radial wall motion, the septal and inferior walls of SLV demonstrated significantly less radial motion compared with other SLV walls.

Conclusions—A major influence of the RV on systemic LV strain and radial motion occurs in the septal wall, whereas absence of the RV causes marked differences in LV twist. These findings may yield clues to the long-term functioning of the SLV and be useful in determining strategies for RV augmentation of LV function. (Circulation. 1998;98:330-338.)

Key Words: contraction ■ ventricles ■ Fontan procedure ■ magnetic resonance imaging

Normally, left ventricles (LVs) and right ventricles (RVs) affect each other’s mechanics by augmenting the function of the other ventricle. LV effects on RV pressure generation have been demonstrated in the human and animal heart as well as RV effects on LV function. We have also recently described the effects of a pulmonary LV on systemic RV mechanics. The mechanism of this ventricular-ventricular interaction is thought to be the mechanical coupling of the ventricles through the interventricular septum. Because it is known that the myocardium constituting the ventricles forms an anatomic continuum around these chambers, others have suggested that the free walls might also affect the contralateral ventricle independent of septal effects.

Understanding that ventricular-ventricular interaction is an integral part of cardiac mechanics, it follows that if a ventricle is absent and therefore no ventricular-ventricular interaction is occurring, cardiac mechanics may be affected. Patients with functionally single LVs (such as tricuspid atresia) who presently undergo staged surgical reconstruction culminating in the Fontan procedure afford a unique opportunity to study how the RV changes LV mechanics. Comparing the mechanics of the systemic LV coupled and uncoupled to an RV tests the hypothesis that the absence of ventricular-ventricular interaction changes LV strain and wall motion. This comparison also may give some insight into the causes of heart failure (eg, poor systolic shortening, clinical congestive heart failure, significant atrioventricular valve regurgitation, high end-diastolic pressures, and so forth), which occurs in some single LVs. We chose patients with tricuspid atresia without transposition of the great arteries {S, D, S} to compare with normal subjects because their LVs have similar anatomic and electrical morphology.
We have previously used a magnetic resonance tagging technique called SPAtial Modulation of Magnetization (SPAMM) \textsuperscript{14-16} to evaluate regional strain and wall motion in congenital heart disease \textsuperscript{6,17,18} and, specifically, ventricular-ventricular interaction. \textsuperscript{6} Strain and its distribution across the myocardial wall were demonstrated to be altered in the systemic RV when a pulmonary LV was not coupled to it. \textsuperscript{6} In addition, systemic RV twist (in an LV, twist is counterclockwise in the short axis \textsuperscript{18-20} and radial wall motion (in an LV, radial wall motion is generally uniform across the short axis \textsuperscript{18-20}) were also shown to be altered when the LV was not present. \textsuperscript{6} The present study was undertaken to determine if this is the case for the systemic, morphologic LV as well and to determine what changes in particular occur. Normal human LVs studied in our laboratory were used as reference. \textsuperscript{18}

**Methods**

**Patients**

Eighteen subjects with systemic LVs were prospectively studied. Ten subjects had a functional single LV (all had tricuspid atresia (S, D, S), Edward’s classification IA or IB). All had completed the Fontan procedure \textsuperscript{7-10} with an atrial lateral wall tunnel type total cavopulmonary connection and were followed at The Children’s Hospital of Philadelphia between December 30, 1993, and September 30, 1995. All patients were clinically well from a cardiovascular standpoint. All underwent echocardiography within 8 months of MRI and had qualitatively good ventricular shortening. All had either trace or no semilunar valve regurgitation. All values are mean±SD. Single LV patients \textsuperscript{1} ages ranged from 3 to 26 years (10.6±7.7 years, median=8 years), heart rate was 88±21 bpm, and time operation was 5.8±4.5 years (median=5 years). Patients had to be stable enough to undergo a 1 hour of MRI scan under sedation. All patients were in normal sinus rhythm and had no evidence on surface ECG of altered electrical activation.

A retrospective review of the cardiac catheterization data on single LV subjects was undertaken, of which 7 were available for review. Hemodynamic data were acquired within 2.3±1.1 years from the date of MRI. Cardiac index, ventricular end-diastolic pressure, inferior vena cava oxygen saturation, pulmonary and systemic pressures, and vascular resistance were all acquired.

**Control Population**

The LVs of 8 healthy adult volunteers, used in a previous investigation, \textsuperscript{15} were studied with magnetic resonance tagging as a control group. Ages ranged from 21 to 35 years (26.2±4.3 years), and heart rate was 70±15 bpm.

Informed consent was obtained from all subjects. The human investigations committee approved the study protocol on February 4, 1992. No patient had any arrhythmias that precluded study in the scanner. All patients fasted 4 hours before MRI.

**Magnetic Resonance Imaging**

Studies were performed on a Siemens 1.5-T Magnetom SP-63. All patients were monitored with pulse oxymetry, ECG, and by direct visualization by video monitor. Our scanning protocol has been previously described in great detail. \textsuperscript{6,17,18} Briefly, after localizers were performed, T1-weighted transverse images were acquired throughout the thorax to evaluate cardiovascular anatomy. Standardization and localization of the short axis of the LV was performed by using the transverse images. This was performed by choosing the long axis of the LV as passing between the center of the mitral valve in the anteroposterior plane (at the left edge of the aortic root) and the LV apex. The short axis is perpendicular to this. Finally, myocardial tagging with image acquisition was done. A high temporal resolution (20 ms) cine sequence was performed through the LV outflow tract to determine the timing of end-systole, which was defined as aortic valve closure. Two short-axis levels (one was one third of the way from the atrioventricular valve to the apex [designated as “atrioventricular valve”] and one was two thirds of the way from the atrioventricular valve to the apex [designated as “apex”]) were chosen for SPAMM imaging.

**Myocardial Tagging**

The SPAMM sequence has been previously described. \textsuperscript{15-16} Briefly, SPAMM imaging uses a prepulse sequence, applied immediately after the R wave, that saturates 2 series of parallel stripes perpendicu lar to each other. Each set of parallel stripes is generated by 5 radiofrequency pulses separated by field gradients. The tissue is thus divided into “cubes of magnetization,” as we have chosen to call them \textsuperscript{17,18} (because each image has thickness) (Figure 1). In-plane cardiac motion moves and distorts these “cubes of magnetization.” Tracking this movement and distortion enables measurement of strain and wall motion. We acquired 6 to 8 gradient-echo images throughout systole starting immediately after the SPAMM prepulse with the following parameters: the repetition time (TR) was the RR interval (range 650 to 1100 ms), flip angle=30 degrees, thickness=6 to 10 mm, inversion time (TI)=16 ms, number of excitations=3 and matrix size=128×256 interpolated to 256×256, and the field of view ranged from 200 to 400 mm. End-diastole was determined by the R wave on ECG. The separation of the grid lines was selected to allow 3 to 4 lines between endocardial and epicardial surfaces (ie, 3 to 4 rows of cubes). Black band (tag) width ranged from 1.5 to 2 mm.

**Image and Data Analysis**

Images were transferred to a Sun SPARC 10 workstation (Sun Microsystems). The VIDA (Volumetric Image Display and Analysis) \textsuperscript{21} software package was used to manipulate the images and perform all measurements as described previously. \textsuperscript{6,17,18} Evaluation of wall motion and strain has also been previously described. \textsuperscript{6,17,18} The appendix in our previous publication details the mathematics of strain calculations. \textsuperscript{17} In brief, the first step was to track the magnetically tagged grid intersections through systole on a computer-based image by a video cursor and mouse with image processing software. \textsuperscript{21} Delaunay triangulation \textsuperscript{22,23} was used to create a triangular grid automatically from the intersections that provided uniform, nonoverlapping triangles. To compute regional wall motion using wall motion software on the computer, the centroid of each triangle was...
used. The regional deformations of the myocardium were then characterized by using homogeneous finite strain analysis on the deforming triangles6,17-20 with the strain software module of VIDA.21 This methodology has been validated in a phantom22 and used in vivo by Young et al.23 These studies demonstrated that homogeneous strain analysis produced unbiased estimates of the principal strains, principal angles, and orientations of the principal axes.

Wall Motion
As described previously,6,17,18 after computing the centroid of all triangles during each systolic phase, the (X, Y) coordinates of the ventricular cavity centroid were obtained on the basis of the endocardial border (Figure 1). Twist and radial shortening were then measured by using the relative motion of the centroid of the triangles relative to the ventricular cavity centroid. How far the muscle marked by the triangle was displaced away from the ventricular cavity centroid (radial motion) from phase n to phase n+1 is described by Equation 1:

\[
|P_{n+1} - P_n| = \sqrt{(X_{n+1} - X_n)^2 + (Y_{n+1} - Y_n)^2}
\]

where (Xn , Yn), and (Xn+1, Yn+1) were the coordinates of the centroid of the triangle at phase n and n+1, respectively (Xc, Yc), were coordinates of the ventricular cavity centroid, and \(|P_n|\) and \(|P_{n+1}|\) were lengths of the vectors from the ventricular cavity centroid to the centroid of triangle at phase n and n+1, respectively. Radial motion was taken as the net inward motion of the centroid of each triangle toward the ventricular cavity centroid relative to the end-diastolic distance (measured in pixels of distance moved divided by initial radial length to normalize for heart size). By convention, radial motion inward was positive, and outward was negative.

Twist was computed by finding the angle \(\Theta\) made by 2 vectors drawn from the ventricular cavity centroid to the centroid of the triangle at phases n and n+1 as described in Equation 2:

\[
\Theta = \cos^{-1}\left(\frac{(P_1 \cdot P_{n+1})}{(|P_1||P_{n+1}|)}\right)
\]

where \(P_1\) and \(P_{n+1}\) were vectors \(|P_1|\) and \(|P_{n+1}|\), respectively, and \(P_1 \cdot P_{n+1}\) was the length of those vectors) from the ventricular cavity centroid to the centroid of triangle at phase n and n+1, respectively, and \(P_1 \cdot P_{n+1}\) was the vector dot product. Net twisting (twist measured from end-diastole to end-systole, sampling at 7 time points) was quantified for magnitude and direction. By convention, clockwise motion was negative and counterclockwise was positive, viewing the heart from apex to base.

Wall motion data was displayed graphically as in Figure 1. “Dots” are the location of the centroid of the triangle at end-diastole and “tails” represent the subsequent motion. The myocardial wall was divided into standard anatomic regions (septal, inferior, lateral, and anterior walls) by using the papillary muscles and other anatomic landmarks from the transverse images as reference to perform the analysis.

Homogeneous Finite Strain Analysis
Details of the mathematics are outlined in the appendix in our previous investigation.17 Briefly, Delaunay triangulation of the tagged intersections was used to compute uniform triangles across the wall.12 Continuum mechanics mathematics, which characterized the complex deformation patterns of the myocardium,19 has been described and used by us17,18 and others23 by using homogeneous finite strains to characterize 2-dimensional shape changes of the magnetically tagged grids. As we have noted, this approach assumed that deformations within each triangle relative to end-diastole were locally homogeneous (Figure 1). After a Lagrangian (Green’s) strain tensor, \(E\) was computed for each triangle; the strain tensors were diagonalized so that they were independent of any coordinate system. The local deformations were described by 2 principal strains, \(E_{\text{P}}\), and the orientation of the principal axes relative to the original coordinate system. The first principal strain, \(E_1\), was defined as the most negative strain (also called thickening, stretch, or maximal principal strain). Strains reported in this study were obtained by averaging the strain of all the triangles within the region. Strains are reported as mean ± SD. Our research focused on the maximum average systolic strain in each region, which occurred at end-systole. The data were quantified and displayed for qualitative analysis in color-coded or gray scale form superimposed onto the anatomic images (Figure 1).

Statistics
Comparisons between 2 means or a mean with a hypothesized value were made by use of the unpaired, 2-way or 1-way (where appropriate) Student’s t test and the Wilcoxon rank sum test. Differences between groups compared by use of a personal computer with JMP version 3.1.4 (SAS Institute).

To obtain the homogeneity of strain within the region (ie, the dispersion of strain within an entire wall region), the standard deviation of all the strains within a given region was used, indexed to the average, meaning the coefficient of variation:

\[
CV = \frac{SD}{\bar{X}}
\]

In each category (strain and wall motion), data are grouped according to short-axis level (atrioventricular valve and apical) and of course, subject group.

Cardiac Catheterization
Cardiac index was 2.7 ± 0.6 L · min⁻¹ · m⁻², LV end-diastolic pressure was 9.4 ± 3.3 mm Hg, aortic pressures were 115 ± 14.1/70 ± 10.6 mm Hg, systemic vascular resistance was 30.5 ± 11.4 Wood units/m², and superior vena cava oxygen saturation was 62.2 ± 6.1%. Mean pulmonary artery pressure was 12.2 ± 2.6 mm Hg, and pulmonary vascular resistance was 1.8 ± 0.8 Wood units/m².

\(E_1\) Strain
Figure 2A displays strain data in gray scale form superimposed on the anatomic image. Figure 2B and 2C display principal compressive strain (\(E_1\) multiplied by −1) for both single LV and normal subjects in the 4 anatomic quadrants (anterior, inferior, posterior, and superior walls) evaluated in 2 short-axis planes (1 near the atrioventricular valve and 1 near the apex). Coefficient of variation for strain measurements was 5.3 ± 2.1%. At both the atrioventricular valve (Figure 2B) and apical levels (Figure 2C) single LVs demonstrated significantly less strain than the control group only at the septal wall (\(P < 0.05\)). Furthermore, in comparing strains within groups at both levels, single LVs again demonstrated significantly less strain in the septal wall when compared with other wall regions (\(P < 0.05\)), whereas there was
no significant difference among wall regions in the control group.

Distribution of $E_1$ Strain

Heterogeneity of Strain Within a Given Anatomic Region

Similar to the absolute measures of strain above, at both the atrioventricular valve (Figure 3A) and apical (Figure 3B) levels, it was only the septal wall of single LVs that differed significantly from controls, displaying greater heterogeneity of strain ($P<0.05$). Similarly, when comparing wall regions within each group, it is solely the septal wall of single LV subjects that demonstrated a greater heterogeneity of strain than other wall regions ($P<0.05$), whereas no significant difference was noted in normal subjects.

Atrioventricular Valve to Apical Plane Strain Ratio

(Distribution of Strain Along the Long Axis of the Ventricle)

No significant differences were noted between single LV and normal groups or when comparing wall regions within each group.

$E_2$ Strain

Figure 4A displays $E_2$ strain data in gray scale form superimposed on the anatomic image and Figure 4B and 4C display the principal $E_2$ strain in graphical form. As with $E_1$ strain, at both the atrioventricular valve (Figure 4B) and apical levels (Figure 4C), single LVs demonstrated altered strain at only the septal wall when compared with control. Single LVs displayed significantly more $E_2$ strain (myocardial wall thickening) than the control ($P<0.05$). However, when comparing wall regions within groups, no significant differences were noted.
Figure 4D and 4E display the heterogeneity of $E_2$ strain within a given anatomic region. As with the absolute measures of $E_2$ strain, at both the atrioventricular valve (Figure 4E) and apical levels (Figure 4F), single LV subjects demonstrated altered strain at only the septal wall when compared with control. Single LV subjects displayed significantly less heterogeneity of $E_2$ strain (myocardial wall thickening) than the control group ($P < 0.05$). However, when comparing wall regions within groups, no significant differences were noted.

An alternate strain notation for $E_1$ is subtracting the absolute value of the strains presented in this study from 1. Similarly, the alternate strain notation for $E_2$ is adding the strains presented in this study to 1. If conservation of mass is preserved, the product of $E_1$ and $E_2$ should equal 1. The Table displays the product of $E_1$ and $E_2$ for all anatomic wall regions in each patient group. Strain products of all wall regions except the septal walls of the single LV are $<1$. The strain products of the septal walls of the single LV were only 3% greater than unity, and considering the error in the measurement could well be below 1. A 1-way t test demonstrated that the strain products of the septal walls of the single LV were not significantly different from 1, although they were signif-

**Product of $E_1$ and $E_2$ for All Anatomic Wall Regions in Single and Normal LVs**

<table>
<thead>
<tr>
<th>Region</th>
<th>Single LV</th>
<th>Normal LV</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Septal Wall</td>
<td>Inferior Wall</td>
</tr>
<tr>
<td>AVV</td>
<td>1.03±0.08</td>
<td>0.94±0.06</td>
</tr>
<tr>
<td>Apex</td>
<td>1.03±0.08</td>
<td>0.98±0.07</td>
</tr>
<tr>
<td>AVV</td>
<td>0.91±0.08</td>
<td>0.92±0.05</td>
</tr>
<tr>
<td>Apex</td>
<td>0.88±0.08</td>
<td>0.95±0.12</td>
</tr>
</tbody>
</table>

AVV indicates atrioventricular valve; LV, left ventricle.

All calculations are mean ± SD. Each value of the anatomic wall region for both groups of patients is the average of the product of $E_1$ and $E_2$ for all patients within each group.
significantly different from the septal walls of the normal LV (1.03 versus 0.91, respectively, for the AVV, \( P = 0.016 \) and 1.03 versus 0.88, respectively, for the apex, \( P = 0.001 \)).

**Wall Motion**

**Twist**

Figure 5A depicts the twisting motion in the short axis of single LVs and controls in graphical format; Figure 5B and 5C display the data quantitatively. Intraobserver variability = 6.7 ± 2.1%. At the atrioventricular valve level (Figure 5B), single LV subjects twisted significantly less counterclockwise in 3 of 4 wall regions (except for the inferior wall) when compared with controls and at the lateral wall, twisted counterclockwise. When comparing wall regions within groups, the lateral wall of single LVs (twisted clockwise) differed significantly from the other wall regions that twisted counterclockwise (\( P < 0.05 \)). No significant differences between wall regions were noted in the control group. At the apical level (Figure 5C), similar to the atrioventricular valve level, single LVs twisted significantly less counterclockwise in 3 of 4 wall regions (with the exception at this level, of the anterior wall) when compared with controls. When comparing wall regions within groups, no significant differences were noted between wall regions in both subject groups.
Notice the uniformity of counterclockwise motion of the normal LV across both short-axis planes. Also note the extent of the twist, and contrast this with the motion of the single LV (Figure 5A). Observe the clockwise motion of the lateral wall of the single LV at the atrioventricular valve plane.

**Radial Motion**

Figure 5A also depicts the radial motion in the short axis of single LVs and control LVs in graphical format; Figure 5D and 5E display the data quantitatively. Intraobserver variability was $6.4 \pm 2.2\%$. At both the atrioventricular valve (Figure 5D) and apical levels (Figure 5E), no significant differences were noted between single LVs and normal control LVs. When comparing wall regions within groups, at both short-axis levels, the septal and inferior walls of single LVs demonstrated significantly less radial motion when compared with the lateral and anterior walls ($P<0.05$). In control LVs, no significant differences were noted among the wall regions.

**Discussion**

Some authors have suggested that ventricular-ventricular interaction is caused by the shared septal wall, while others have suggested that the free walls affect the contralateral ventricle independently of the septum. The mechanism of free wall contribution is the fact that the myocardium is a “syncytium of muscle fibers tethered within a collagen network” with muscle fibers in continuity between RV and LV. Santamore et al showed in rabbits that the free wall of the RV as an anchor for the LV to pump blood.

Additional, this inverse relation between $E_1$ and $E_2$ strains between single LVs and normal LVs may represent a fundamental change in septal systolic mechanics from mostly circumferential shortening to predominantly wall thickening. The septum does not have RV pressure to buttress itself against during contraction and therefore may bulge radially and do less circumferential shortening. A difference in septal orientation among the 2 groups may also explain this. Finally, septal remodeling may have occurred, changing the $E_1$ and $E_2$ relation, and may represent the most energy-efficient way for a single LV to pump blood.

Because the single LV septum does not have an attached RV, it is a “free wall” as well. It could be argued that its mechanics should mimic free wall mechanics. Our data demonstrates that this does not occur. The LV septal wall is morphologically different from the free walls and this fact probably plays a role in explaining the behavior of the septum.

It is interesting that $E_1$ and $E_2$ strains are inversely related to each other in the septal wall in the single LV when compared with normal control LVs ($E_1$ less in control LVs and $E_2$ greater in control LVs). We refer to the way $E_1$ and $E_2$ change relative to each other between types of hearts (single LV versus normal LV). It might be expected, however, that if the single LV $E_1$ is less in control LVs, then $E_2$ might also be less in control LVs. One explanation may be that our measurements are in 2 dimensions, and the difference is in the third dimension (base to apex dimension). Alternatively, this may represent a difference in the material properties of the myocardium (eg, the level of anisotropy or the elastic modulus—the amount of deformation relative to the stress) between single LVs and normal LVs. Relative amounts of septal wall coronary blood flow between single LVs and normal LVs may also play a role.

Strain

This study demonstrated that the single LV septal wall had a significantly lower “circumferential shortening” strain ($E_2$) and a significantly higher “stretch” $E_1$ strain than in normal subjects. This was the only wall region that differed from the control group. Therefore, at least from absolute strain measures, it is the septal wall that is affected most by the RV. This may either be a direct effect of RV pressure on the septal wall, or it may simply be a “passive” effect: the absence of the RV as an anchor for the LV to pump blood.

Wall Motion

As we and others have noted, LV systolic twist plays a role by distributing stress, strain, and energy requirements.
across the ventricle. It enhances diastolic filling by storage of potential energy and diastolic elastic recoil.\textsuperscript{6,17,18,33,34} This wall motion is thought to be a result of complex fiber architecture\textsuperscript{31,32} and electrical activation. Altered twist may effect all parts of the cardiac cycle with detrimental effects on function.

Single LVs twisted less counterclockwise at both short-axis levels than did normal LVs. This decrease in twist is consistent with the decreased cardiac index and wall hypokinesis we\textsuperscript{19} and others\textsuperscript{36–39} have observed after the Fontan procedure in both single RVs and LVs. The second ventricle may therefore augment the twist of the systemic ventricle as well. Interestingly, single LV lateral wall twisted clockwise, consistent with the abnormal wall motion that Akagi et al.\textsuperscript{59} found in their single LVs. This can be explained by anatomic myocardial fiber orientation, which is a left-handed helix in the epicardium and a right-handed helix in the endocardium.\textsuperscript{31}

No significant differences were noted in radial wall motion between subjects with single LV and control subjects. However, comparisons within the single LV group, at both short-axis levels, demonstrated that septal and inferior walls had significantly less radial motion when compared with other walls. Again, this would be consistent with altered fiber orientation as noted above and may be the most energy-efficient way for a single ventricle to pump.

We have previously discussed our twist findings in the normal LV when compared with other studies in the literature.\textsuperscript{6,17,18} Briefly, there are conflicting reports on the direction of twist at the LV short axis. We\textsuperscript{19,20} and others\textsuperscript{19,40} believe that a counterclockwise twist is present when viewed apex to base and this twist is uniform throughout the short axis.

**Limitations**

We have previously discussed\textsuperscript{6,17,18} in detail the limitation of acquiring data in fixed planes as 2-dimensional MRI does, with possible artifactual deformations caused by motion into and out of this plane.\textsuperscript{41} We have discussed\textsuperscript{6,17,18} in detail the study of Moore et al.\textsuperscript{19} and noted that we studied both single LV and control subjects by the same protocol. One must remember that because our study deals with large thickness slices relative to the amount of through plane motion, it is not thought that this would have an appreciable effect on the findings.

There are other limitations to this imaging approach. First, as we have previously discussed,\textsuperscript{18} 2-dimensional strain analysis excludes the remaining components of the 3-dimensional strain tensor.\textsuperscript{42} Solutions to 3-dimensional strain analysis, however, require a priori knowledge of the deformation to interpolate the data that, by its very nature, is dependent on assumptions. This may introduce error in the calculations.

Second, we note that homogeneous strain analysis assumes that deformation is constant within a given triangular element. Strains, however, have been known to have some transmural variation. This therefore represents the limit of resolution on the strain data.

Use of the single LV as a model of ventricular-ventricular interaction has its limitations in that these hearts have undergone cardiopulmonary bypass. We would anticipate, however, that the altered strain and wall motion abnormalities noted would be more diffuse if cardiopulmonary bypass played a role. Ideally, the control group would have been normal hearts in patients who have undergone thoracotomy, pericardial stripping, and a cardiopulmonary bypass run.

Ages for subjects with single LV and control subjects are significantly different. Although quantitative changes may occur in subjects with single LV as they grow, it is unlikely that the fundamental differences in regional strain, twist, and radial motion we have observed will change with time. Further, if age-dependent differences do exist, it is likely to affect all walls, not just the septal wall as in our study.

Finally, many of the steps in the data collection and analysis were performed by the same person. This adds a bias to the data; however, we do not believe this would have significantly changed our results.

**Conclusions**

It appears that the RV affects systemic LV mechanics at the septal wall only by decreasing the absolute circumferential shortening strain, increasing the wall thickening strain, and increasing general strain dispersion within a region. However, a decrease in the relative endocardial strain with respect to epicardial strain was noted in the lateral wall in the absence of an RV. Furthermore, a significant decrease in LV twist relative to normal subjects was noted in patients with single LV, with reversal of the normal counterclockwise rotation at the lateral wall. Ventricular-ventricular interaction, which has been known to occur in the normal human heart and the systemic RV, is evident in vivo by looking at the effects of the absence of an RV on LV wall mechanics. This study adds to the body of knowledge on ventricular-ventricular interaction and single ventricle mechanics and may lead to a better understanding of LV function. The knowledge may contribute to designing better medical and surgical treatments for the patient with single ventricle.

**Acknowledgments**

Dr Fogel was funded through a fellowship grant of the Southeastern Pennsylvania affiliate of the American Heart Association. Dr Hoffman is an established investigator of the American Heart Association.

**References**

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Circulation. 1998;98:330-338
doi: 10.1161/01.CIR.98.4.330
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1998 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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