Paradoxical and pseudoparadoxical interventricular septal motion in patients with right ventricular volume overload


ABSTRACT Cross-sectional echocardiographic measurements of normalized septal curvature (NSC), systolic anterior motion of the center of the left ventricular cavity (CAM), and the M mode ratio of left ventricular posterior wall epicardial motion (PEM) to posterior wall thickening (PWT) were made in eight normal subjects, 16 patients with right ventricular volume overload (RVVO) and five with pressure overload (RVPO). Paradoxical M mode septal motion was confined to early systole in six patients with RVVO (group I) and was sustained in 10 (group II). Similar end-diastolic septal flattening was observed in RVVO group I (NSC 0.50 ± 0.16 [SD]) and group II (0.49 ± 0.23) when compared with the normal group (0.83 ± 0.07, both p < .005). NSC increased in both RVVO groups during the first one-third of systole (p < .002) to values not significantly different from normal, but did not change significantly thereafter. CAM in RVVO group II (5.4 ± 2.2 mm) exceeded CAM in both the normal group (1.8 ± 1.9 mm, p < .001) and group I (2.1 ± 1.4 mm, p < .005). Similarly, the PEM/PWT ratios in group II (mean 2.94; range 2.13 to 8.0) exceeded those in both the normal group (mean 1.59; range 1.11 to 2.13, p < .01) and group I (mean 1.32; range 1.10 to 1.67, p < .01). In the RVPO group, CAM was insignificant, the PEM/PWT ratios were lower than normal (p < .01), and marked end-diastolic septal flattening was incompletely corrected during early systole, after which the septum was flattened further until end-systole (p < .005). Midsystolic septal flattening was also observed in those with RVVO in whom the right ventricular/left ventricular peak systolic pressure ratio exceeded 0.4. Thus, true septal paradox in patients with RVVO is an early systolic event. The apparent persistence of septal paradox beyond early systole is an artifact due to anterior left ventricular translation. These findings help explain the preservation of left ventricular systolic function in RVVO despite apparently sustained septal paradox, support the role of the transseptal pressure gradient in determining septal curvature, and indicate that regional wall motion analysis from a fixed reference point in patients with RVVO is inappropriate.


PARADOXICAL systolic motion of the interventricular septum is a characteristic feature of right ventricular volume overload (RVVO). Reasoning that truly paradoxical septal motion could not occur in the absence of an abnormal systolic increase in septal curvature, Weyman et al. qualitatively demonstrated diastolic flattening of the septum toward the left ventricle in patients with RVVO and showed that septal curvature returned toward normal (i.e., rightward) during systole. These observations were consistent with the demonstration by Pearlman et al. that the systolic motion of the septum was determined by its position at end-diastole. Furthermore, the septal curvature changes observed by Weyman et al. were maximal at the base of the heart and decreased toward the apex, a finding consistent with the observation by Hagan et al. of downward displacement of the normal “hinge point” between anterior aortic wall motion and posterior contraction of the septum in patients with RVVO. Weyman et al. concluded that the systolic correction of diastolic septal flattening constituted the major mechanism of paradoxical septal motion in patients with RVVO.

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CIRCULATION
Although quite variable patterns of septal motion may be observed in those with RVVO, the predominant previously described abnormalities have been observed to persist throughout the greater part of systole. If persistently abnormal systolic septal motion could result from diastolic septal flattening, then abnormal left ventricular systolic function might be expected in most patients with RVVO. Although left ventricular systolic function may appear to be depressed by RVVO when it is related to the left ventricular end-diastolic pressure as the index of preload, this predominantly reflects the reduced left ventricular compliance associated with leftward diastolic septal shifting. Left ventricular systolic function is found to be well preserved when it is related to left ventricular end-diastolic volume as the index of left ventricular systolic function in most patients with atrial septal defects.

We postulated that the contribution of the systolic correction of diastolic septal flattening to abnormal patterns of septal motion in RVVO should be confined to early systole, for two reasons. First, the most probable mediator of the systolic correction of septal curvature is the systolic increase in the left-to-right transseptal pressure gradient, which is maximal in early systole. Second, even if septal curvature correction continued throughout systole, its tendency to produce net anterior septal motion would be progressively attenuated by the systolic reduction in left ventricular dimensions, an effect that is maximal in midsystole.

This hypothesis implies that the septum moves toward the center of the left ventricle after early systole in patients with RVVO. If this is so, the M mode echocardiographic appearance of anterior motion, or no net motion, of the septum during mid-late systole could be explained only by anterior motion of the entire left ventricle during this period. The latter motion was, in fact, the first mechanism postulated to explain paradoxical septal motion in RVVO, but was not substantiated. If correct, our hypothesis would help explain the preservation of left ventricular systolic function in patients with RVVO. The demonstration of exaggerated anterior systolic left ventricular motion in RVVO would also have implications for the clinical assessment of regional wall motion in this condition.

Furthermore, the extent of any such motion might be related to the severity of RVVO and thus provide clinically useful information.

The aim of our study was to quantitatively evaluate the relative contributions of changes in septal curvature and motion of the entire left ventricle to abnormal patterns of septal motion in RVVO.

Methods

Subjects. Three groups of subjects were studied: 16 patients with RVVO, five patients with right ventricular pressure overload (RVPO), and eight normal subjects. There were seven male and nine female subjects, 10 to 69 (mean 39) years old, in the RVVO group. Twelve patients had an ostium secundum atrial septal defect, one had a sinus venosus defect with partial anomalous pulmonary venous return, two had Ebstein’s anomaly, and one had tricuspid regurgitation in association with rheumatic mitral valve stenosis. These diagnoses were established by cross-sectional echocardiography, including peripheral venous contrast injection, and were confirmed by cardiac catheterization or during subsequent surgical intervention.

There were two men and three women, 24 to 73 years old, in the RVPO group. Two of these patients had primary pulmonary hypertension, while pulmonary hypertension was associated with progressive systemic sclerosis in one patient and was secondary to multiple pulmonary thromboemboli in another. These diagnoses were established by cardiac catheterization (mean pulmonary arterial pressure ranged from 50 to 64 mm Hg). The fifth patient in the RVPO group had advanced chronic obstructive pulmonary disease with marked clinical and radiologic features of pulmonary hypertension. There were five male and three female subjects, 17 to 27 years old, in the control group.

Cardiac catheterization. Cardiac catheterization was performed by standard techniques via the right brachial approach. Pressures were measured with fluid-filled catheters attached to Statham P23Db transducers. In 14 patients, both left ventricular and right ventricular systolic pressures were recorded, thus permitting comparison of the peak systolic right ventricular pressure with the peak systolic left ventricular pressure.

Echocardiographic recordings. Parasternal, short-axis cross-sectional echocardiograms were recorded in all subjects at the high papillary muscle level of the left ventricle at end-expiration. M mode echocardiograms were simultaneously derived by passing a cursor through the cross-sectional images. All echocardiograms were recorded with a Diasonics V-3400R ultrasonograph equipped with a 2.25 MHz phased-array transducer. An electrocardiographic triggering system was used to automatically select end-diastolic frames at the peak of the R wave and end-systolic frames were selected as those corresponding to the M mode echocardiographic aortic valve closure point, as previously described by Shimada et al. Frames corresponding to one-third and two-thirds of systole were then selected by frame counting between the end-diastolic and end-systolic frames.

Data analysis. The selected cross-sectional frames and the M mode echocardiograms from all study groups were analyzed in a random and blind manner. The in-built microprocessor-linked light-pen planimeter system of the Diasonics V-3400R ultrasonograph was used to trace the endocardial edge of each selected left ventricular cross-sectional frame, as previously described, thus obviating the parallax error inherent in tracing images by transparent overlay techniques on the video screen. The microprocessor automatically computed the left ventricular endocardial cross-sectional area. The center of each outline was then determined by finding the point of intersection of two perpendicular lines that each, as determined by planimetry, bisected the left ventricular endocardial cross-sectional area (figure 1).

From a hard copy print of each traced endocardial outline, the radius of curvature of the interventricular septal segment was determined by the method described by Brinker et al. (figure 1).
1). Because the radius of septal curvature varies with alterations in left ventricular cross-sectional area, the measured radius was normalized for this area in the manner described by King et al.14 (figure 1).

In addition, the distance from the previously determined center of each left ventricular outline to the origin of the ultrasound "fan" was measured from the hard-copy prints. When random analysis of all the selected frames was completed, anterior systolic motion of the left ventricular center (CAM) in any given systolic frame was determined by subtracting the previously measured distance between the left ventricular center and the ultrasound origin in that frame from the independently measured distance in the corresponding end-diastolic frame. Thus, a positive CAM value indicated anterior systolic motion of the left ventricular center.

Motion of the left ventricular center is not necessarily synonymous with motion of the entire left ventricle, since alterations in left ventricular geometry, such as those that occur with alterations in septal curvature, may also alter the position of the left ventricular center. In the absence of an increase in the normalized septal curvature (NSC), however, a positive CAM value does indicate anterior left ventricular motion.

Motion of the entire left ventricle relative to the stationary ultrasound transducer was independently assessed by analysis of the motion of the posterior left ventricular wall from the simultaneously recorded M mode echocardiograms. To distinguish anterior motion of the entire left ventricle (which increases the motion but not the thickening of the posterior left ventricular wall) from enhanced posterior wall contraction (which increases both the motion and the thickening of the wall), we measured the ratio of posterior wall epicardial motion (PEM) to the systolic change in posterior wall thickness (PWT) (figure 2).

Paradoxical M mode echocardiographic septal motion was defined as anterior systolic motion of the right septal surface.7 The subjects with RVVO were divided into two groups: group I comprised six subjects with paradoxical septal motion confined to the first one-third of systole (figure 3, A), and group II comprised 10 subjects with paradoxical septal motion that persisted beyond the first one-third of systole (figure 3, B and C). Because the M mode echocardiograms were simultaneously derived from the same cross-sectional images used to determine NSC and CAM, it was possible to relate alterations in these latter parameters to the observed M mode patterns of septal motion.

Statistical analysis. Two-way analyses of variance of the data on NSC and CAM from the four study groups at all four stages of the cardiac cycle were initially performed. When significant variance was detected, sources of variance were isolated by multiple intergroup comparisons with the Bonferroni-adjusted unpaired t test and multiple intragroup comparisons were performed with the Bonferroni-adjusted paired t test.28 The PEM/PWT ratios from the four groups were initially analyzed by computing the Kruskal-Wallis test statistic.28 When an overall difference was demonstrated, multiple intergroup comparisons were performed with the Mann-Whitney rank-sum test and were appropriately adjusted for the Bonferroni inequality.28 Unless otherwise stated, results are expressed as mean ± SD.

Reproducibility of methods. Two observers independently traced the endocardial outlines of 50 left ventricular cross sections and determined NSC and the distance from the planimetrically determined left ventricular center to the ultrasound origin, as described above, for each frame. One observer repeated the tracings and computations for 20 of these left ventricular cross sections several months later. No significant interobserver differences in the NSC data were detected by one-way analysis of variance (0.56 ± 0.38 vs 0.55 ± 0.34), nor were there differences in the left ventricular center–ultrasound origin distance data (68.8 ± 8.2 vs 68.7 ± 8.3 mm) and linear regression analysis demonstrated high interobserver correlations for both the NSC (r = .94, p < .001, SEE = 0.117) and the distance measurements (r = .996, p < .001, SEE = 0.7 mm). No significant intraobserver differences were detected for either method by one-way analysis of variance and excellent intraobserver and interobserver correlations were demonstrated for both the NSC (r = .91, p < .001, SEE = 0.078) and left ventricular center–ultrasound origin measurements (r = .999, p < .001, SEE = 0.5 mm).

When two observers independently determined the PEM/PWT ratios from 50 M mode echocardiograms and one observer blindly repeated the 30 measurements, no significant interobserver differences (1.19 ± 0.45, range 0.43 to 2.33 vs 1.12 ± 0.50, range 0.43 to 2.40) or intraobserver differences were detected by Wilcoxon’s signed-rank test.28

Results.

Interventricular septal curvature. The NSC data obtained in the normal, RVVO, and RVPO groups are
summarized in table 1 and a comparison of the mean data in these groups is shown in figure 4. Mild end-diastolic septal flattening and a slight systolic increase in NSC (p < .05) were noted in the normal group. Compared with the normal group, marked end-diastolic septal flattening was observed in both RVVO groups and there was no significant difference in the degree of septal flattening between RVVO group I (NSC = 0.50 ± 0.16) and group II (0.49 ± 0.23). During the first one-third of systole, NSC increased significantly (p < .002) in both RVVO groups to values that were not significantly different from those in the normal group. During the remaining two-thirds of systole, NSC did not increase significantly in either RVVO group and

FIGURE 2. Diagram illustrating the method and rationale for measuring the M mode echocardiographic ratio of systolic left ventricular PEM/PWT. Systolic anterior motion of the entire left ventricle (bottom) increases PEM but does not alter PWT, resulting in an increased PEM/PWT ratio.

FIGURE 3. M mode echocardiograms from three subjects with RVVO, showing: A, early systolic paradoxical septal motion, but normal septal motion during mid-late systole (group I pattern); B, early systolic paradoxical septal motion and sustained paradoxical motion of the right septal surface during mid-late systole, despite flat, or slightly posterior, motion of the left septal surface (group II pattern); and C, a more exaggerated group II pattern (both sides of the septum move paradoxically throughout systole).
TABLE 1
NSC data

<table>
<thead>
<tr>
<th></th>
<th>Normal group (n = 8)</th>
<th>RVVO group I (n = 6)</th>
<th>RVVO group II (n = 10)</th>
<th>RVPO group (n = 5)</th>
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<tr>
<td>Time</td>
<td>0.83 ± 0.07</td>
<td>0.50 ± 0.16</td>
<td>0.49 ± 0.23</td>
<td>0.24 ± 0.29</td>
</tr>
<tr>
<td>⅓ S</td>
<td>0.87 ± 0.07</td>
<td>0.83 ± 0.14</td>
<td>0.88 ± 0.16</td>
<td>0.56 ± 0.22</td>
</tr>
<tr>
<td>⅔ S</td>
<td>0.91 ± 0.09</td>
<td>0.89 ± 0.16</td>
<td>0.72 ± 0.29</td>
<td>0.25 ± 0.31</td>
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<tr>
<td>ES</td>
<td>0.91 ± 0.05</td>
<td>0.86 ± 0.14</td>
<td>0.73 ± 0.30</td>
<td>0.07 ± 0.22</td>
</tr>
</tbody>
</table>

ED = end-diastole; ⅓ S = one-third systole; ⅔ S = two-thirds systole; ES = end-systole.

Statistical comparisons are with normal group: *p < .005; **p < .002; ***p < .0005; ****p < .0001 (the statistical significance of changes within each group are shown in figure 4).

The M mode echocardiograms from two RVPO patients showed early systolic anterior septal motion, which was most prominent in the only RVPO patient with a large increase in NSC during the first one-third of systole (from 0 at end-diastole to 0.63). The echocardiogram of this latter patient showed complete right bundle branch block. All patients in the RVPO group had apparently normal (i.e., posterior) mid-late systolic septal motion on their M mode echocardiograms, which was consistent with the distinctly abnormal mid-late systolic leftward septal flattening observed by simultaneous cross-sectional echocardiography.

Figure 5 shows the midsystolic change in NSC in 14 of the subjects from the RVVO and RVPO groups in whom the peak systolic right and left ventricular pressures were obtained during cardiac catheterization. Subjects with right ventricular/left ventricular peak systolic pressure ratios that were less than 0.4 showed little change in NSC during midsystole (0.09 ± 0.18), while subjects with ratios that exceeded 0.4 showed a variable midsystolic decrease in NSC (−0.31 ± 0.21). The midsystolic alterations in NSC in these two groups were significantly different (p < .01).

**Systolic left ventricular motion.** The CAM data obtained in the normal, RVVO, and RVPO groups are summarized in table 2 and a comparison of the mean data in these groups is shown in figure 6. By definition, the CAM value at end-diastole was 0 in all groups (see Methods). A positive CAM value indicates a systolic anterior shift of the left ventricular center. In the

![FIGURE 4](http://circ.ahajournals.org/)

FIGURE 4. Mean NSC data in the normal, RVVO, and RVPO groups (standard deviations have been omitted for greater clarity, but are given in table 1). Asterisks indicate the statistical significance of the change in NSC, from the value that precedes the value that follows it, within each group: **p < .005; ***p < .002 (for statistical comparisons between different groups, see table 1 and text). Time period abbreviations are as in table 1.

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normal group, a small positive CAM shift was noted only during the last one-third of systole (p < .03).

In RVVO group I, the total positive systolic CAM shift of 2.1 ± 1.4 mm did not differ significantly from that observed in the normal group (1.8 ± 1.9 mm), although CAM increased more gradually throughout systole in RVVO group I. In RVVO group II, the group with sustained M mode echocardiographic septal paradox, the total systolic positive CAM shift of 5.4 ± 2.2 mm was highly significant (p < .00001) and significantly exceeded CAM in both the normal group (p < .001) and RVVO group I (p < .005). The positive CAM shift in RVVO group II occurred mainly during mid-systole (p < .00001) in all subjects. No significant CAM shift was observed in the RVPO group.

Similarly, there was no significant difference between the M mode echocardiographic PEM/PWT ra-

<table>
<thead>
<tr>
<th>TABLE 2</th>
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<tr>
<td>CAM data (mm)</td>
</tr>
<tr>
<td>Time</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>ED</td>
</tr>
<tr>
<td>1/3 S</td>
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<td>2/3 S</td>
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<tr>
<td>ES</td>
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</table>

Abbreviations are as in table 1.
Statistical comparisons are with normal group: *p < .0001; **p < .001 (the statistical significance of changes within each group are shown in figure 6).

FIGURE 5. The midsystolic change in NSC in 14 subjects grouped according to whether the ratio of right ventricular peak systolic pressure (RVP)/left ventricular peak systolic pressure (LVP) exceeded, or was less than, 0.4. *p < .01. Time period abbreviations are as in table 1.

FIGURE 6. Mean data for CAM in the same four groups shown in figure 4 (standard deviations are given in table 2). Askew asterisks indicate the statistical significance of the change in CAM, from the value that precedes to the value that follows it, within each group: *p < .03; ****p < .00001 (for statistical comparisons between different groups, see table 2 and text). Time period abbreviations are as in table 1.
tions in RVVO group I (mean 1.32; range 1.10 to 1.67) and those in the normal group (mean 1.59; range 1.11 to 2.13), but the PEM/PWT ratios of all RVVO group II subjects exceeded 2.0 (mean 2.94; range 2.13 to 8.0) and significantly exceeded those of both the normal group and RVVO group I (both p < .001). These data suggest that anterior systolic motion of the posterior left ventricular wall in RVVO group II was disproportionate to posterior wall contraction, consistent with exaggerated anterior systolic motion of the entire left ventricle (figure 2). The PEM/PWT ratios in the RVPO group (mean 0.66; range 0.46 to 0.75) were significantly lower than those in the normal group (p < .01), consistent with the absence of a positive CAM shift in the RVPO group.

**Discussion**

In this study, significant increases in interventricular septal curvature in patients with RVVO were confined to the first one-third of systole. This finding is consistent with previous cineangiographic and radionuclide ventriculographic studies that demonstrated that rightward “bulging” of the septum in RVVO was an early systolic event. Only small positive CAM shifts were noted during the first one-third of systole in those with RVVO and these may have predominantly reflected the significant alteration in left ventricular cross-sectional geometry during this period. These findings indicate that early systolic paradoxical septal motion accompanying RVVO results predominantly from the rapid early systolic correction of diastolic septal flattening, but that paradoxical septal motion sustained beyond early systole cannot be attributed to further increases in septal curvature.

The RVVO patients with sustained M mode echocardiographically documented septal paradox (group II) were demonstrated to have exaggerated anterior systolic motion of the entire left ventricle predominantly during mid systole, as evidenced by a highly significant positive CAM shift in the absence of increasing septal curvature. This evidence was independently supported by an increased PEM/PWT ratio in the same group of patients.

Since the average normal amplitude of posterior systolic motion of the left side of the septum is only 5 mm (range 3 to 8 mm), and the normal amplitude of motion of the right septal surface is appreciably less than this, the CAM values obtained in the subjects in RVVO group II (5.4 ± 2.2 mm) were of sufficient magnitude to explain the sustained abnormalities of septal motion observed in this group. The small magnitude of anterior systolic left ventricular motion necessary to produce abnormal M mode echocardiographic patterns of septal motion may explain the inability of previous cineangiographic and radionuclide ventriculographic studies to demonstrate this phenomenon in patients with RVVO.

Thus, the typical patterns of M mode echocardiographic paradoxical septal motion that accompany RVVO appear to be a composite of true early systolic paradoxical septal motion due to a rapid increase in septal curvature and mid-late systolic pseudoparadoxical septal motion due to anterior motion of the entire left ventricle. Figure 7 illustrates this composite mechanism.

The rapid early systolic correction of diastolic septal flattening in patients with RVVO is consistent with the hypothesis that this correction is mediated by the rapid early systolic increase in the left-to-right transseptal pressure gradient, which is reduced at end-diastole in those with this condition. This hypothesis is further supported by the failure of early systolic septal curvature correction in most of the subjects with RVVO. The delayed onset of the rise of right ventricular systolic pressure caused by complete right bundle branch block might thus explain the marked early systolic increase in septal curvature observed in one subject with RVVO.

Progressive systolic septal flattening has recently been documented to be a reliable index of right ventricular systolic hypertension. Our observations were quite consistent with these previous reports. King et al. demonstrated that systolic septal flattening in children was more closely related to the right ventricular/left ventricular peak systolic pressure ratio than to the absolute right ventricular systolic pressure. We observed mid-systolic septal flattening in a predominantly adult group of subjects with right ventricular/left ventricular systolic pressure ratios that exceeded 0.4. These observations are consistent with the view that the curvature of the septum is determined by the prevailing transseptal pressure gradient, not only during diastole but also during systole.

If the systemic arterial pressure is known to be normal and right ventricular outlet obstruction has been excluded, then systolic septal flattening is also an index of pulmonary hypertension. The observation that M mode echocardiographic septal motion may not be paradoxical in patients with pulmonary hypertension complicating RVVO may be explained by the tendency of coexistent RVPO to damp the early systolic increase in septal curvature and to cause mid-late systolic septal flattening (i.e., posterior septal motion).

In this study we demonstrated exaggerated anterior
systolic left ventricular motion in patients with RVVO, but did not address the mechanism of this motion. Meyer et al.\textsuperscript{19} postulated that the left ventricle, having been displaced posteriorly in the thorax by the dilated right ventricle during diastole, would move anteriorly again during systole due to the greater stroke volume ejected by the right ventricle. The same mechanism was proposed earlier by Popp and Harrison\textsuperscript{22} to explain their observation of systolic anterior motion of the mitral valve in patients with atrial septal defects. This mechanism implies that a relationship should exist between the extent of anterior systolic left ventricular motion and the severity of RVVO. If so, the extent of this motion would provide clinically useful information. In patients with small left-to-right shunts, for example, M mode echocardiographic septal motion may appear superficially normal,\textsuperscript{31} indicating minimal pseudoparadoxical septal motion, but rapid early (i.e., true) paradoxical septal motion may still be observed in most cases.\textsuperscript{31} Similarly, the observation of normal septal motion when left ventricular volume overload coexists with RVVO\textsuperscript{34, 35} may be explicable in terms of both reduced diastolic septal flattening and a reduced discrepancy between the stroke volumes of the two ventricles.

Regardless of its mechanism, however, the demonstration of exaggerated anterior systolic motion of the left ventricle in patients with RVVO has implications for the accurate clinical assessment of regional left ventricular wall motion in those with this condition. A similar left ventricular translational artifact has been noted after cardiac surgery,\textsuperscript{20, 21} resulting in a systematic underestimation of septal motion and overestimation of posterolateral left ventricular wall motion when analyzed from a fixed frame of reference, whether by echocardiographic,\textsuperscript{36} radionuclide ventriculographic,\textsuperscript{37} or cineangiographic\textsuperscript{21} techniques. Floating-axis reference systems, which account for left ventricular translation, have been used to overcome this postoperative artifact.\textsuperscript{20, 21}

Weyman et al.\textsuperscript{4} described the essential feature of truly paradoxical septal motion: an abnormal systolic increase in septal curvature. Our study quantitatively supports, but also modifies their conclusions. In particular, the finding that truly paradoxical septal motion is confined to early systole in patients with RVVO helps explain why left ventricular contractile function is less impaired in those with this condition than the frequent M mode echocardiographic appearance of sustained systolic septal motion abnormalities might suggest.\textsuperscript{9}

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