Determinants of Ventricular Septal Motion
Influence of Relative Right and Left Ventricular Size

ALAN S. PEARLMAN, M.D., CHESTER E. CLARK, M.D., WALTER L. HENRY, M.D.,
JOEL MORGANROTH, M.D., SAMUEL B. ITSCHCOITZ, M.D., AND STEPHEN E. EPSTEIN, M.D.

SUMMARY To test the hypothesis that the ventricular septum moves during systole toward the center of ventricular mass (so that the end-diatostic position of the septum within the heart should determine both the direction and the magnitude of septal motion during systole), echocardiograms from patients with several different hemodynamic burdens were analyzed. A linear relation was noted between the end-diastolic intracardiac position of the ventricular septum and the direction and magnitude of systolic septal motion in 1) forty-three patients with an atrial septal defect (regression coefficient \( r = 0.80 \)), 2) fourteen patients with other causes of right ventricular volume overload (\( r = 0.82 \)), 3) nineteen patients with left ventricular volume overload (\( r = 0.74 \)), 4) ten patients with right ventricular pressure overload (\( r = 0.93 \)), 5) ten patients with left ventricular pressure overload (\( r = 0.80 \)), 6) twenty-eight normal subjects (\( r = 0.82 \)). We conclude that, in the presence of normal ventricular activation and contraction, the direction and magnitude of septal motion during systole is determined by the intracardiac position of the septum at end-diastole.

RECENT STUDIES using echocardiography have indicated that the ventricular septum normally moves away from the sternum and toward the posterior left ventricular free wall during systole.\(^1\)\(^-\)\(^6\) Deviations from this pattern have been described in conditions such as left bundle branch block,\(^4\)\(^-\)\(^5\) Wolff-Parkinson-White syndrome,\(^7\) asymmetric septal hypertrophy,\(^8\)\(^-\)\(^9\) congestive cardiomyopathy,\(^8\)\(^-\)\(^10\) coronary artery disease,\(^8\)\(^-\)\(^10\) and following aortic or mitral valve replacement.\(^11\)

Abnormal septal motion also occurs commonly in patients with an atrial septal defect\(^1\)\(^-\)\(^3\)\(^-\)\(^9\) or some other cause of right ventricular volume overload.\(^5\)\(^-\)\(^8\)\(^-\)\(^10\)\(^-\)\(^13\) In these patients the ventricular septum moves anteriorly, toward the sternum, during systole. This pattern of motion has been termed "paradoxic," and has been described as one of the characteristic echocardiographic features of right ventricular volume overload.\(^1\)\(^-\)\(^3\)\(^-\)\(^13\)\(^-\)\(^16\) However, right ventricular volume overload cannot be the sole determinant of paradoxic systolic septal motion, since: 1) septal motion is clearly normal in some patients in which the right ventricle bears a large diastolic volume overload\(^12\)\(^-\)\(^15\)\(^-\)\(^18\)\(^-\)\(^21\)\(^-\)\(^22\) and 2) paradoxic septal motion has been described in some normal subjects.\(^19\)\(^-\)\(^20\)

To explain these apparently discordant observations, we examined the hypothesis that the heart contracts symmetrically toward its center of mass, so that the position of the ventricular septum within the heart at end-diastole determines both the direction and the magnitude of septal motion during systole. This hypothesis is based on several assumptions: 1) that the ventricular myocardium is activated normally and contracts during ventricular systole in a symmetric, sequential fashion,\(^24\) 2) that the ventricular muscle and ventricular blood pool have essentially uniform mass, and 3) that motion of the mid-septum (which lies exactly halfway between right and left septal surfaces) most accurately reflects motion of the septum as a whole.

Based upon these assumptions, we postulated that the total ventricular mass (including both ventricular muscle and intraventricular blood) would move symmetrically during systole toward its center of mass. This hypothesis is represented diagrammatically in figure 1, in which the septum is actually depicted as a line drawn along the mid-septum. Normally (fig. 1A), the left ventricle is ellipsoidal in cross-section\(^16\)\(^-\)\(^25\)\(^-\)\(^27\) and of greater anteroposterior diameter than the crescent-shaped right ventricle. Hence, the septum occupies a relatively anterior position within the overall ventricular volume at end-diastole. Since the densities of myocardium and blood are similar, the septum also occupies a relatively anterior position within the overall ventricular mass. According to our hypothesis, under normal conditions the mid-septum would be expected to move during systole in a posterior direction, as denoted by the arrow. However, right ventricular dilatation (fig. 1B), would displace the septum to a relatively posterior position within the ventricular mass at end-diastole. In the illustration, the mid-septum lies slightly closer to the left ventricular epicardium than to the right ventricular epicardium. Since this position would lie posterior to the center of ventricular mass, our hypothesis would predict anterior motion during systole. Moreover, the farther the mid-septum is from the center of ventricular mass at end-diastole, the more exaggerated its motion should be during systole. Thus, if the previously stated hypothesis is correct, the position of the septum at end-diastole should determine the direction and magnitude of systolic septal motion.

To test the validity of this hypothesis, echocardiograms were reviewed retrospectively from patients with an atrial septal defect, and subsequently from patients with other causes of right or left ventricular volume overload, right or left ventricular pressure overload, and from normal subjects.

Methods

Patients

Atrial septal defect. This group consisted of 43 patients with an atrial septal defect. Forty-one had left-to-right shunts, with pulmonary-to-systemic flow ratios (Qp:Qs) ranging from 1.1 to 8.3:1 (mean 3.1:1); two had Eisenmenger reactions and consequent right-to-left shunts (Qp:Qs = 0.7:1 and 0.9:1). Twenty-eight were female and 15 were male; mean age was 26 years (range 5–63 years). In all cases, diagnosis was confirmed by cardiac catheterization,
and 32 patients have subsequently had surgical repair. Thirty-four had secundum defects, and nine had primum defects; in each of the latter patients, mitral regurgitation was either absent or only mild.

**Other causes of right ventricular volume overload.** This group included fourteen patients with right ventricular volume overload occurring in the absence of an atrial septal defect. Diagnoses were confirmed by cardiac catheterization in every patient, and included tricuspid regurgitation in 11 patients (seven on a rheumatic and four on a congenital basis) and pulmonary valvular insufficiency (from previous valvotomy) in three patients. Eight were male and six were female; mean age was 35 years (range 9–56 years). Although every patient clearly had significant right ventricular volume overload, six of the patients with rheumatic disease also had some degree of left ventricular volume overload from a coexistent left-sided valvular lesion.

**Left ventricular volume overload.** This group consisted of nineteen patients with left ventricular volume overload; diagnosis was confirmed at cardiac catheterization, operation, or both, in seventeen, and was made clinically in two (both with obvious mitral regurgitation). Six patients had left-to-right shunts at the ventricular level (Qp:Qs ranged from 1.1 to 2.8:1), 11 had aortic or mitral regurgitation, and two had coronary arteriovenous fistulae (both with Qp:Qs = 1.1:1). Thirteen patients were male and six female; mean age was 34 years (range 1–59 years).

**Right ventricular pressure overload.** This group included ten patients with pressure overload of the right ventricle documented at cardiac catheterization. Six patients had primary pulmonary hypertension (pulmonary artery systolic pressure ranged from 70–140 mm Hg, and pulmonary artery wedge pressure was normal), three had valvular pulmonic stenosis, and one peripheral pulmonary stenosis (gradients ranged from 50–70 mm Hg). Seven of these patients were female and three male; mean age was 29 years (range 10–55 years).

**Left ventricular pressure overload.** This group included ten patients with left ventricular pressure overload, documented at catheterization. Four patients had valvular and two supravalvular aortic stenosis (gradients ranged from 25–160 mm Hg), and three had coarctation of the aorta. No patient had significant valvular insufficiency. One patient with bilateral fibromuscular hyperplasia of the renal arteries and persistent severe hypertension (diastolic blood pressure 110–140 mm Hg) was also included in this group. Seven patients were male and three female; mean age was 26 years (range 9–64 years).

**Normal subjects.** This group consisted of twenty-eight healthy subjects who were without clinical evidence of heart disease. Fourteen were male and fourteen female; mean age was 22 years (range 6–47 years).

Of the 124 patients studied, none had left bundle branch block, WPW syndrome, cardiomyopathy, or clinically apparent coronary artery disease. Patients with reduced left ventricular systolic function, as indicated by an obviously reduced left ventricular ejection fraction, were also excluded. Finally, echocardiograms from those patients undergoing cardiac surgery were obtained before operation, with the following exceptions. Two patients had undergone closed mitral commissurotomy many years earlier, and had residual mitral regurgitation (causing left ventricular volume overload). This operative procedure has been shown not to alter septal motion.11 Three patients had undergone previous pulmonary valvotomy, via right ventriculotomy in two, and by means of a Brock procedure in the third. Right ventriculotomy per se has been said not to alter septal motion,12 while the Brock procedure is not known to alter septal motion.

**Echocardiographic Technique**

Echocardiograms were recorded using an Ekoline 20A ultrasond unit, Honeywell 1856 Line Scan Recorder, Hewlett-Packard X-Y display, and a custom-built video amplifier. Patients were examined while partially turned into the left lateral position. With the transducer in an interspace to the left of the sternum, the T-scan technique28 was employed to locate the mitral valve, and damping and gain adjusted so as to visualize the ventricular septum and both right and left ventricular free walls at the tip of the mitral valve (at a point where both anterior and posterior mitral leaflets were identifiable). This anatomic landmark was chosen because it provided the best reproducible reference.
SEPTAL MOTION AND VENTRICULAR SIZE/Pearlman et al.

Table 1. Definition of Measurements

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Formula</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cardiac diameter (TCD&lt;sub&gt;D&lt;/sub&gt;)</td>
<td>= the distance from right ventricular epicardium to left ventricular epicardium; measured at end-diastole. This represents the maximum diastolic cardiac diameter.</td>
</tr>
<tr>
<td>Septal position ratio (RV-S)&lt;sub&gt;D&lt;/sub&gt; / TCD&lt;sub&gt;D&lt;/sub&gt;</td>
<td>= the distance from right ventricular epicardium to mid-septum, divided by total cardiac diameter to normalize for heart size; measured at end-diastole.</td>
</tr>
<tr>
<td>Septal motion (RV-S)&lt;sub&gt;D&lt;/sub&gt; - (RV-S)&lt;sub&gt;S&lt;/sub&gt; / TCD&lt;sub&gt;D&lt;/sub&gt;</td>
<td>= the distance from right ventricular epicardium to mid-septum at end-diastole, minus the distance between these points at the time of maximal systolic septal displacement, divided by total cardiac diameter. This fraction is then multiplied by one hundred to express septal motion as a percentage of total cardiac diameter; a negative sign indicates normal motion, while a positive sign denotes paradoxical motion.</td>
</tr>
<tr>
<td>Right ventricular diastolic diameter (RVDD) index</td>
<td>= the distance from the right septal surface to a point 0.5 cm posterior to chest wall echoes, divided by the body surface area; measured at end-diastole.</td>
</tr>
<tr>
<td>Left ventricular internal dimension (LVID) index</td>
<td>= the distance from the left septal surface to the endocardial surface of the left ventricular posterior free wall, divided by the body surface area; measured at end-diastole.</td>
</tr>
</tbody>
</table>

Measurements

Recordings were analyzed quantitatively only in those portions in which recognizable echoes from the right ventricular epicardium, right and left septal surfaces, and endocardial and epicardial surfaces of the left ventricular posterior wall could be identified simultaneously at both end diastole and end systole at the level of the mitral valve tip. Clear visualization of all of these structures was greatly facilitated by use of the previously mentioned strip chart recorder and switched gain circuit.

The junction at end diastole between nonmoving echoes from the inner chest wall and moving echoes from the right ventricular epicardium was identified by adjusting damping. Because the right ventricular epicardium was often difficult to visualize clearly during systole, its junction with the chest wall at end diastole was used as a fixed reference line, hereafter referred to as the right ventricular epicardium, throughout the cardiac cycle. Measurements were made as defined in table 1 and as diagrammatically depicted in figure 2. In the line diagram demonstrating these measurements (fig. 2), the distance (RV-S)<sub>D</sub> is about one-third of TCD<sub>D</sub>, and hence the septal position ratio is 0.33. A larger ratio would denote posterior displacement of the septum relative to the total cardiac diameter. The amount of septal motion, measured according to the formula, is −4.3% of the total cardiac diameter; the negative sign denotes that septal motion is posterior (normal) in this example.

Statistics

The relations between systolic septal motion and variables such as septal position ratio, right ventricular diastolic diameter index, pulmonary-to-systemic flow ratio, and right ventricular systolic pressure were analyzed by means of linear regression; coefficients of regression were calculated by the method of least squares. Mean ventricular dimension measurements from different patient groups were compared by Student’s t-test. Individual measurements were con-
considered significantly different from a population mean if they were more than two standard deviations from that mean.

Results

Atrial Septal Defect

When analyzed qualitatively, ventricular septal motion was frankly paradox in 35 (81%), flat in three (7%), and normal in five (12%) patients with an atrial septal defect. However, when septal motion was quantified as change in septal position from end-diastole to systole, and expressed as a percent of diastolic cardiac diameter, a continuum in the magnitude of septal motion was apparent, rather than a discrete separation between normal, flat, and paradoxic motion (fig. 3). In addition, measurement of the end-diastolic septal position ratio clearly separated patients with normal from those with abnormal septal motion. Thus, every patient in whom the septum was relatively anterior (as defined by a septal position ratio of 0.40 or less) had normal septal motion, while every patient in whom the septum was relatively posterior (as defined by a septal position ratio greater than 0.40) had abnormal flat or anterior motion (fig. 3). Moreover, the correlation between the end-diastolic septal position ratio and the magnitude of systolic septal motion was strong (r = 0.80, fig. 3). In contrast, the magnitude and direction of septal motion correlated weakly with RVDD index (r = 0.41), and correlated poorly with magnitude of shunt (expressed as Qp:Qs, r = 0.13) and right ventricular systolic pressure (r = 0.18).

The ASD patients with normal septal motion were generally, but not invariably, those with smaller shunts. (Qp:Qs was 1.1, 1.2, 1.4, 2.6, and 3.9:1 in these patients.) In contrast, with the exception of two patients with Eisenmenger reactions, every ASD patient with flat or paradoxic motion had Qp:Qs greater than 1.5:1. Nonetheless, four of the five patients with normal septal motion had right ventricular diastolic diameter (RVDD) index greater than 1.4, which was significantly increased from normal (mean 1.0 ± 0.2 cm/m²), and not significantly different from the ASD patients with flat or paradoxic motion. However, each of these four patients also had significant left ventricular dilatation (left ventricular internal dimension [LVID] index 3.8 cm/m² or above; LVID index for normals = 2.6 ± 0.3 cm/m²). Thus the coexistence of right and left ventricular dilatation led to normal septal position ratios in each of these ASD patients with normal septal motion (fig. 3).

Right Ventricular Volume Overload with Intact Atrial Septum

None of the fourteen patients with tricuspid or pulmonary insufficiency had any evidence of left-to-right shunt. Septal motion was paradox in 11 patients and normal in three. Despite the variation in septal motion from patient to patient, however, the relation between the end-diastolic septal position ratio and systolic septal motion was strong (r = 0.82, fig. 4). Every patient with paradoxic motion had significant right ventricular dilatation (RVDD index > 1.4 cm/m² compared to 1.0 ± 0.2 cm/m² for normal subjects). However, two of the three patients with normal motion also had right ventricular dilatation. Finally, six of the patients with rheumatic tricuspid insufficiency had coexistent left ventricular volume overload, as a consequence of associated rheumatic mitral or aortic regurgitation. The degree of associated left ventricular volume load was mild, however, in that none of these patients had left ventricular dilatation. In each patient, the direction and magnitude of systolic septal motion was determined by the septal position ratio, and not simply by the presence of right ventricular dilatation.

![Figure 3](http://circ.ahajournals.org/)

**Figure 3.** Relation between end-diastolic septal position ratio (horizontal axis) and systolic septal motion (vertical axis) in patients with an atrial septal defect. Two patients with an Eisenmenger reaction are labelled. The relation is significant (r = 0.80).

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

**Figure 4.** Relation between end-diastolic septal position ratio and systolic septal motion in patients with right ventricular volume overload as a consequence of tricuspid or pulmonary insufficiency. The relation is significant (r = 0.82).
**Left Ventricular Volume Overload**

Of nineteen patients with left ventricular volume overload, septal motion was normal in thirteen, somewhat flattened in three, and clearly paradoxic in three. The end-diastolic septal position ratio correlated strongly with systolic septal motion ($r = 0.74$, fig. 5) in this group as well, despite a discordancy between the occurrence of right ventricular dilatation and the presence of flat or paradoxic septal motion. Thus ten (53%) patients had right ventricular dilatation (RVDD index $> 1.4$ cm/m$^2$), usually resulting from either secondary right ventricular hypertension (six patients with mitral valve disease) or left-to-right shunt (two patients with ventricular septal defect and one with coronary arteriovenous fistula). Of the ten patients with right ventricular dilatation, septal motion was normal in five, flattened in two, and paradoxic in three. In contrast, of nine patients with no right ventricular dilatation, septal motion was normal in eight and somewhat flattened in one. In every patient, however, systolic septal motion correlated closely with the end-diastolic septal position ratio.

**Right Ventricular Pressure Overload**

Nine (90%) of the ten patients with right ventricular pressure overload had significant right ventricular dilatation (RVDD index $> 1.4$ cm/m$^2$). Although septal motion was normal in five and paradoxic in five, the relation between septal position ratio and septal motion was very strong ($r = 0.93$, fig. 6). Thus, those patients with relatively less right ventricular dilatation, or relatively more concomitant left ventricular dilatation, tended to have preservation of normal septal motion, as was confirmed by measurement of right and left ventricular dimensions in this group.

Of additional note was the qualitative pattern of paradoxical septal motion seen in patients with fixed pulmonary vascular obstruction. In those patients with relatively marked right ventricular dilatation (RVDD index $> 1.9$ cm/m$^2$) in association with primary pulmonary hypertension, the septum moved abruptly and rapidly anteriorly during early systole, tended to move slowly posteriorly during late systole, and sometimes nearly returned at end-systole to its original end-diastolic position. This pattern was seen in each of the four patients with paradoxical septal motion occurring in the presence of primary pulmonary hypertension and in each of the two patients with an Eisenmenger reaction associated with an atrial septal defect (fig. 7, right panel), but in none of the 50 patients with paradoxical septal motion occurring in the absence of fixed pulmonary vascular obstruction. In these latter patients, septal motion proceeded smoothly and gradually anteriorly throughout systole (fig. 7, left panel).

**Left Ventricular Pressure Overload**

Each of the ten patients with left ventricular pressure overload had increased left ventricular posterior wall thickness. Four had left ventricular dilatation (LVID index $> 3.2$ cm/m$^2$) and three had right ventricular dilatation (RVDD index $> 1.4$ cm/m$^2$). None of the patients had pulmonary hypertension to account for the observed right ventricular dilatation. Regardless of the presence or absence of right ventricular dilatation, all patients in this group with left ventricular pressure overload displayed normal systolic septal motion, and the relation between septal position ratio and septal motion was strong ($r = 0.80$, fig. 8).

**Normal Subjects**

In 28 subjects with no evidence of heart disease, systolic septal motion was normal in 25, flat in two, and slightly but clearly paradoxic in one. This variability of the pattern of

---

**Figure 5.** Relation between end-diastolic septal position ratio and systolic septal motion in patients with left ventricular volume overload. The relation is significant ($r = 0.74$).

**Figure 6.** Relation between end-diastolic septal position ratio and systolic septal motion in patients with right ventricular pressure overload. The relation is significant ($r = 0.93$).
septal motion could again be explained by the relative location of the septum within the diastolic cardiac diameter, since there was a strong correlation between septal position ratio and septal motion ($r = 0.82$, fig. 9).

**Discussion**

Previous investigators have noted that anterior (paradoxic) motion of the ventricular septum during systole is quite common in patients with an atrial septal defect and consequent right ventricular volume overload.\(^1\) Recent echocardiographic studies have indicated, however, that paradoxic septal motion may be found in patients who do not have an atrial septal defect or any other cause of right

---

**FIGURE 7.** Left panel: unretouched echocardiogram from a patient with an atrial septal defect and normal pulmonary artery pressure, demonstrating gradual anterior motion of the septum throughout systole. Right panel: unretouched echocardiogram from a patient with primary pulmonary hypertension, demonstrating distinctive pattern of paradoxic septal motion; note abrupt anterior motion of the septum in early systole.

**FIGURE 8.** Relation between end-diastolic septal position ratio and systolic septal motion in patients with left ventricular pressure overload. The relation is significant ($r = 0.80$).

**FIGURE 9.** Relation between end-diastolic septal position ratio and systolic septal motion in normal subjects. The relation is significant ($r = 0.82$).
ventricular volume overload. Furthermore, normal motion of the septum may be present in some patients who have an atrial septal defect resulting in a substantial volume overload on the right ventricle. The present study defines a mechanism that consistently explains these seemingly discordant observations. In each patient with an atrial septal defect, regardless of whether septal motion was paradoxic or normal, we found that both the direction and the magnitude of systolic septal motion was predicted by the end-diastolic septal position ratio. Furthermore, since a highly significant relation between the septal position ratio and septal motion was found in patients with right ventricular volume overload but intact atrial septum, in those with left ventricular volume overload, in those with pressure (but not volume) overload of either right or left ventricle, and in normal subjects, this relation does not appear to be peculiar to a specific anatomic or hemodynamic abnormality. Indeed, when all 124 subjects in the present study are considered together, a striking relation between end-diastolic septal position ratio and systolic septal motion ($r = 0.90$, fig. 10) is apparent.

The relation between end-diastolic septal position ratio and systolic septal motion thus seems to apply to a variety of hemodynamic abnormalities, and helps to explain the echocardiographic patterns of septal motion recorded from normal subjects as well as those with volume or pressure overloads of the right or left ventricle. The observation that the mid-septum moves posteriorly (normal) during systole when it occupies an anterior end-diastolic position, but moves anteriorly (paradoxic) during systole when it occupies a posterior end-diastolic position, is compatible with the hypothesis that geometric factors influence the direction of septal movement and that the mid-point of the septum moves during systole toward the center of ventricular mass.

Since the septal position ratio reflects the relative location of the septum within the heart, the association of paradoxic ventricular septal motion with septal position ratio $>0.40$ (denoting relative posterior displacement of the septum) indicates that the presence of paradoxic septal motion depends on two conditions: 1) the right ventricle must be dilated significantly, and 2) coexistent left ventricular dilatation must be either mild or not present. Under normal conditions, the first requirement is not met, and thus the septum moves posteriorly during systole (fig. 1, panel A). In the presence of isolated right ventricular dilatation (fig. 1, panel B), the septum is displaced to a relatively posterior position at end-diastole, and hence it moves paradoxically (in an anterior direction) during systole. However, coexistent dilatation of both right and left ventricles (fig. 1, panel C) tends to preserve the relatively anterior end-diastolic position of the septum, and hence the septum moves posteriorly during systole despite the presence of right ventricular dilatation. Indeed, in four patients with an atrial septal defect, in 17 patients with other forms of heart disease, and in one subject without evidence of heart disease, normal septal motion appeared to be preserved in spite of significant right ventricular dilatation by precisely this mechanism. Finally, in the presence of isolated left ventricular dilatation (fig. 1, panel D), the septum is displaced to a relatively more anterior end-diastolic position than normal; thus while the direction of motion is unaffected, the amplitude of systolic motion is increased, as others have also noted.

That the presence of paradoxic septal motion depends upon relative posterior displacement of the septum within the heart also helps clarify the heretofore confusing observation that paradoxic motion can occur in the apparent absence of right ventricular volume overload. Such a posterior displacement of the septum depends solely upon isolated right ventricular dilatation, whatever the cause. Thus, in the present study, of the 56 patients whose echocardiograms demonstrated right ventricular dilatation and paradoxic septal motion, eight (14%) had no apparent volume overload of the right ventricle. Although it is possible that some of these patients might have had some undetected minor degree of pulmonic or tricuspid insufficiency, it is unlikely that this occurred in every case. Other studies have reported paradoxic septal motion in patients following operative closure of an atrial septal defect. In these patients, the persistence of some degree of right ventricular dilatation also results in posterior septal displacement. The persistence of flat or frankly paradoxic septal motion in such a setting often reflects incomplete resolution of right ventricular dilatation and should not necessarily be taken as evidence for persistence of interatrial shunt.
Several other aspects of the current study deserve emphasis. First, septal motion as measured in the current study refers to motion of the mid-septum (consisting of points mid-way between right and left septal surfaces), while previous descriptions of ventricular septal motion have referred to the pattern of motion of the left septal surface. Since the septum does thicken during systole, flat motion of the mid-septum is accompanied by a slight degree of posterior movement of the left septal surface, while paradoxical motion of the mid-septum is accompanied by flattened movement of the left septal surface. Thus septal motion as defined in the present study differs from septal motion as described by previous workers. Nonetheless, it would appear reasonable to assume that motion of the mid-septum reflects motion of the septum as a whole more accurately than does motion of the left septal surface. Second, the hypothesis under investigation was based on the assumption of normal symmetric, sequential ventricular activation and contraction. Thus patients with abnormal ventricular activation (such as occurs secondary to left bundle branch block or the WPW syndrome), those with abnormal ventricular contraction patterns (such as may occur secondary to dilated cardiomyopathy, asymmetric septal hypertrophy, or coronary artery disease), and those with impaired left ventricular contractile function (reflected by an obviously reduced left ventricular ejection fraction), were specifically excluded from this analysis. It seems quite likely that in such patients the correlation between septal position and septal motion described in this study might be altered. (The presence of right bundle branch block has been shown not to alter ventricular septal motion; hence, patients with right bundle branch block were not excluded from the present analysis.) Finally, measurements of chamber diameter, septal position, and septal motion were made at the precise level of the tip of the mitral valve. This landmark was chosen specifically because we believe it is necessary to standardize the exact level of the septum under scrutiny in order to make valid comparisons between one heart and another. Our results are in no way incompatible with the observations of others that the pattern of septal motion varies depending on where along the ventricular long axis the echo beam traverses the septum, in that anterior systolic septal motion tends to be more marked at the left ventricular outflow tract and less marked toward the cardiac apex.

Although not specifically designed to compare the prevalence of paradoxical septal motion in patients with right ventricular volume as opposed to pressure overload, the present study indicates that paradoxical motion occurs more commonly in response to volume loads, as others have reported. Such a finding is not surprising, because an increased volume load tends to cause chamber dilatation, while an increased pressure load leads primarily to an increase in wall thickness. Since an increased right ventricular pressure load less commonly causes dilatation, it would be expected to cause posterior displacement of the septum, and hence paradoxical septal motion, less commonly than does an increased right ventricular volume load.

Our results also demonstrate that paradoxical septal motion appears to have a distinctive pattern when it occurs in the presence of fixed pulmonary vascular obstruction (fig. 7). In each of the four patients who demonstrated paradoxical septal motion in the presence of primary pulmonary hypertension, the septum achieved its maximum anterior position in early-to-mid systole, and then tended to move gradually posteriorly during the remainder of systole. In addition, both patients with atrial septal defect and Eisenmenger reaction (pulmonary artery systolic pressures 131 mm Hg and 90 mm Hg; Qp:Qs = 0.9:1 and 0.7:1, respectively) also demonstrated this rapid early systolic anterior septal motion. This pattern was not noted in any patient with paradoxical motion occurring in the presence of normal pulmonary arterial pressure. Furthermore, those patients with elevated pulmonary arterial pressure secondary to mitral valve disease who developed right ventricular dilatation and paradoxical septal motion demonstrated gradual anterior motion of the septum throughout systole. In these patients with reversible pulmonary hypertension, the pattern of paradoxical motion was indistinguishable from that in patients with right ventricular volume overload. Thus, it appears that the rapid early systolic anterior pattern of paradoxical septal motion may be specific for fixed pulmonary vascular occlusive disease. However, the patients who demonstrated this distinctive pattern of paradoxical motion also had the largest septal position ratios. Hence it is possible that this pattern merely reflects extreme posterior displacement of the septum, and is not etiologically specific.

In summary, the present investigation indicates that both the direction and magnitude of ventricular septal motion during systole are determined by the intracardiac position of the septum at end-diastole. This relation suggests that the mid-point of the septum moves during systole toward the center of ventricular mass. The relation between the septal position ratio and septal motion appears to be valid regardless of whether the stimulus to dilatation is volume or pressure overload. Furthermore, the magnitude of ventricular volume or pressure overload influences the nature of septal motion only insofar as it causes ventricular dilatation. Thus the echocardiographic demonstration of paradoxical anterior systolic septal motion is not a diagnostic marker for right ventricular volume overload, but merely reflects relative right ventricular dilatation of any cause.

Acknowledgment

The authors gratefully acknowledge the invaluable assistance of F. Joyce McKay, R.N., Cora Burn, R.N., and Estelle Cohen in performing echocardiographic examinations, Drs. Marian Fisher, William Blackwelder, and William Friedewald in performing statistical analyses, and Ms. Virginia King in preparing illustrations.

References

7. Ticzon AR, Damato AN, Caracta AR, Russo G, Foster JR, Lau SH: Echocardiographic evaluation of the intraventricular septal motion dur-
Echocardiographic Evaluation of the Stent Mounted Aortic Bioprosthetic Valve in the Mitral Position

In Vitro and In Vivo Studies

MICHAEL S. HOROWITZ, M.D., PAUL L. TECKLENBERG, M.D., DANIEL J. GOODMAN, M.D., DONALD C. HARRISON, M.D., AND RICHARD L. POPP, M.D.

Summary Echocardiograms were performed on 20 clinically stable patients following mitral valve replacement with glutaraldehyde-preserved porcine aortic heterografts and three patients with antibiotic sterilized aortic homografts mounted in the mitral position. Such valves were evaluated in a test chamber at varied flow rates resulting in improved understanding of movements seen with the echocardiogram in vivo. The technique for recording the valvular stent and leaflets is described and a method for measuring several parameters is demonstrated. Initial diastolic slope averaged 2.4 ± 0.5 cm/sec (range 1.9 to 3.3 cm/sec). Left ventricular outflow tract measured from the anterior portion of the stent to the interventricular septum averaged 1.5 ± 0.5 cm at end-diastole and 1.3 ± 0.6 cm at end-systole. Leaflet excursion averaged 1.5 ± 0.3 cm (with a range from 1.0 to 2.1 cm). The ratio of internal to external stent diameters averaged 0.66 ± 0.05 (with a range from 0.56 to 0.74).

Clinical and Hemodynamic Improvement has been demonstrated in most patients during the first few postoperative years after mitral valve replacement with the stent-mounted, glutaraldehyde-preserved porcine aortic heterograft. 1-4 Thromboembolism after the initial postoperative period has been low.1-8 Persistence of this favorable experience over the next several years will likely lead to more widespread use of this valve.

In recent years a large number of patients have received bioprostheses in our hospital. For this reason, we sought to use noninvasive techniques to provide a better understanding of valvular function. Ultrasonic studies were performed in a series of clinically stable patients with the stented porcine heterografts and homografts in the mitral position. An in vitro ultrasonic study suggested the origin of each echo component and improved the understanding of the movements seen on the in vivo echocardiogram. This report describes the ultrasonic representation of the normal valve.
Determinants of ventricular septal motion. Influence of relative right and left ventricular size.

A S Pearlman, C E Clark, W L Henry, J Morganroth, S B Itscoitz and S E Epstein

Circulation. 1976;54:83-91
doi: 10.1161/01.CIR.54.1.83

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1976 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:
http://circ.ahajournals.org/content/54/1/83

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/