Left Ventricular Abnormalities in Prolapsed Mitral Leaflet Syndrome

Review of Eighty-Seven Cases

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SUMMARY
Eighty-seven patients with proven mitral leaflet prolapse were studied emphasizing cardiodynamics and left ventricular asynergy. Significant associated features were female preponderance (83%), skeletal anomalies (pectus excavatum, straight back, scoliosis, narrow antero-posterior diameter of the chest), and anomalous coronary arteries (cork-screw patterns, short left main coronary artery, anomalous origin of the coronary arteries). Prolapse of the tricuspid leaflets was found in 15 (54%) who had right ventriculography. Five types of abnormal left ventricular systolic contraction patterns were seen in 82% of the cases and these were categorized as: 1) "ballerina foot" pattern (vigorous posteromedial contraction with anterior convexity), 2) "hour glass" pattern (vigorous ring-like contraction involving the middle portion of the left ventricle), 3) inadequate long axis shortening, 4) posterior akinesis, and 5) cavity obliteration pattern. The over-all left ventricular performance was normal generally, as indicated by normal values for functional parameters including left ventricular end-diastolic pressure, cardiac index, ejection fraction, contractility index (stroke work per end-diastolic volume) and pre-ejection period/left ventricular ejection time (PEP/LVET). The myocardial component of the syndrome of prolapsed mitral (and/or tricuspid) leaflets is expressed as asynergistic patterns of ventricular motion and usually does not impair over-all cardiac dynamics.

Additional Indexing Words:
Anomalous coronary arteries Tricuspid leaflets prolapse Ventricular asynergy Skeletal anomalies Mitral regurgitation Left ventricular function

Prolapsing mitral leaflet (PML) syndrome has become a well-established clinical entity. Its clinical and electrocardiographic features have been the subject of many papers. Angiocardiographic descriptions have been focused mostly on the abnormalities of the mitral valve, demonstrating a systolic prolapse of one or both leaflets.1-10 However, additional manifestations have been described that indicate a disorder not limited to changes of the mitral apparatus. Relatively recent reports have described abnormal patterns of left ventricular contraction, indicating a myocardial element of this disorder. Grossman et al.,3 Engle,11 and Ehlers et al.8 observed an abnormal systolic contraction ring, most marked in the postero-inferior portion of the left ventricle, giving rise to a "two-lumened" appearance. Similar findings ("indentation" of the inferior border of the left ventricle) was noted by Jeresaty12 in five of 11 cases studied by left ventriculography. In addition, two patients had nonobstructive left ventricular hypertrophy with obliteration of the apical portion of the cavity. The same author13 stressed the frequency of mitral leaflet ballooning into left atrium in hypertrophic subaortic stenosis and nonobstructive left ventricular hypertrophy. Pocock and Barlow14 included two cases of idiopathic hypertrophic obstructive cardiomyopathy in their series of PML syndrome (130 cases). Feldman et al.15 described "hyperdynamic" left ventricular contractions in their 17 patients with PML. Gooch et al.16 reported a high incidence (70.8%) of left ventricular systolic and/or diastolic asynergy in 24 cases of this condition. Liedtke et al.17 studied nine patients with systolic click syndrome and found a reduced contraction of the inflow portion of the left ventricle.

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Hemodynamic data have been normal in most reported cases of PML syndrome. The only exceptions were those of Miller, Gulco, and Gulotta,18 whose data showed left ventricular dysfunction in 12 of 15 cases. The impairment was seen as elevations of left ventricular end-diastolic pressure, low cardiac output with abnormal response to exercise, and angiocardiographic evidence of reduced contractility.

This report is an extension of our previous observations18 and describes 87 proven cases of PML with detailed emphasis on hemodynamic data and modes of left ventricular contraction. Several patterns of left ventricular asynergy are categorized as well as preliminary observations on the findings of right ventriculography.

Subjects and Methods

This study involved 87 patients, 72 females and 15 males, whose ages ranged from eight to 64 years (mean, 38 years). They had been referred to Deborah Heart and Lung Center for cardiac evaluation. All had symptoms (chest pain and/or palpitations were most common) and abnormal physical findings. Systolic clicks and murmurs provided important clues to the diagnosis though they were not always conspicuous. Arrhythmias were common findings, often induced by exercise. Four cases had features of Marfan's syndrome. In all cases, a diagnosis of PML was substantiated by angiocardiographic demonstration of systolic prolapse of one or both mitral leaflets.

Seventy-eight patients underwent combined right and left heart catheterization, and nine patients had catheterization limited to the left ventricle. Thirty-eight patients with disabling chest pains also had selective coronary cineangiography by the Sones or Judkins technique.

Left ventriculography was performed in the right anterior oblique (RAO) projection at 25-30 degrees in 83 patients, in the left anterior oblique (LAO) projection in two patients, and in both projections in two patients. Angiocardiographic recordings were made on 35 mm film at 60 frames per second. Right ventriculography was carried out in the RAO projection at 15-25 degrees in 31 patients. Details concerning the technique of right ventriculography and tricuspid leaflet prolapse was reported elsewhere.19

To analyze the morphologic changes during left ventricular contractions and relaxation, a frame-to-frame analysis was made of changes of reference axes (fig. 1) during nonarrhythmic beats. The cine left ventriculogram was projected on a screen of convenient size, from which a left ventricular profile was traced. The longest measured length was designated as long axis (L), and the axis perpendicular to it at its midpoint was designated as medial axis, the anterior segment being the anteromedial axis (AM), and posterior segment, posteromedial axis (PM). Four more axes were defined such that any of the eight reference axes formed an angle of 45 degrees with the adjacent axes (fig. 1). They were anterobasal axis (AB), posterobasal axis (PB), anteroapical axis (AA), and posteroapical axis (PA). Shortening of each axis was expressed as a percentage of the end-diastolic dimension and identified segmental asynergy. The data were analyzed in reference to that obtained from a control group of 16 patients without PML and with normal ventricular motions.

Mitral leaflet prolapse was graded on an arbitrary scale of increasing severity from 1+ to 4+, 1+ denoting minimal prolapse, and 4+, the length of prolapse in excess of a quarter of the end-systolic outflow tract diameter. This judgment was made by at least two observers when viewing the left ventriculogram in late systole. Mitral regurgitation was also quantitated according to the method of Sellers et al.20 Right ventricular contraction was evaluated in terms of vigor of tricuspid valve ring excursion.

Pressure recordings were made on an Electronics For Medicine Multi-channel DB-8 Recorder. Cardiac output was measured by the Fick method. Ejection fraction (EF) was calculated with the following formula:*  

\[ EF = 1 - \frac{(As)Ld}{(Ad)Ls} \]

where Ld and Ls are the longest measured lengths on the projected left ventricular profile in end-diastole and end-systole, respectively; Ad and As are the planimetered areas of the left ventricular profile in end-diastole and end-systole, respectively.

*See appendix

Figure 1

Changes of reference axes during a cardiac cycle. Only end-diastolic and end-systolic frames traced directly from the projected cine left ventriculograms are presented here for comparison. Abbreviations: AB: anterobasal axis; AM: anteromedial axis; AA: anteroapical axis; PB: posterobasal axis; PM: posteroapical axis; PT: pteroaopical axis; L: Long axis.
Stroke work index (SWI) and contractility index (CrI) were calculated as follows:

\[
\text{SWI (gm m/m^2)} = \frac{\text{SI (ml/beat/m^2)} \times \text{LVsm (mm Hg)}}{	ext{EDV index}}
\]

\[
\text{CrI (gm m/cm^3)} = \frac{\text{EF} \times \text{LVsm} \times 0.0136}{\text{SI}}
\]

where SI is stroke index and LVsm is left ventricular mean systolic pressure.

PEP/LVET was obtained from the simultaneously recorded aortic and left ventricular pressure curves and electrocardiograms recorded at 75 mm/sec.

**Results**

**Hemodynamic Measurements and Related Parameters**

Table 1 summarizes the measurements of flow, pressures, and volumes and derived parameters. All the mean values are within normal limits. With a very few exceptions, the individual values are also within normal limits. Left ventricular end-diastolic pressure was slightly elevated (range: 13-15 mm Hg) in three patients. Two of them had a minimal increase of pulmonary arterial systolic pressure and one of them had severe mitral regurgitation. Cardiac outputs and cardiac indices were slightly decreased in six patients and borderline in five patients. The ejection fraction was below normal of a slight degree in only three cases. Their ejection fractions were 51%, 54% and 49%, respectively. The contractility index was normal in all cases. One case with a history of rheumatic heart disease showed evidence of mitral stenosis and two cases revealed atrial left to right shunt.

**Mitral Regurgitation**

Mitral regurgitation (MR) could be adequately quantitated in 85 patients. There were 26 cases (31%) with a MR exceeding 1+: 13% with 2+, 13% with 3+, and 5% with 4+ MR. The remaining 59 cases (69%) comprised 35% with no MR, 28% with minimal MR and 6% with 1+ MR. In mild to moderate MR, the onset of opacification of the left atrium was delayed until mid-systole, well beyond the first visual evidence of leaflet prolapase, while in severe MR, left atrial opacification began early in systole.

**Prolapse of the Mitral Valve Leaflet**

There were 36 patients (42%) with pronounced leaflet prolapase (3+ and 4+) of the posterior or both posterior and anterior mitral leaflets. Of these, in 29 cases (80.5%), the prolapase was limited to the posterior leaflet. Isolated prolapase of the anterior leaflet was not encountered. It should be recognized, however, that the RAO projection is not ideal for visualization of the area of anterior leaflet since in this projection it may be obscured behind the opacified left coronary sinus or it may overlap with the posterior leaflet. Prolapsed leaflets often had a scalloped appearance or exhibited clefts.

Equal degrees of prolapase of both leaflets could be recognized in 20% of those without significant MR, whereas none with significant MR was associated with equal degrees of prolapase of both leaflets. A discrepancy in the degrees of prolapase of both leaflets by at least 2+ was seen in 73% of those with significant MR and in only 36% with mild or no MR.

**Patterns of Left Ventricular Contractions**

**Systolic Phase.** Nine patients had normal left ventricular contractions without demonstrable asynergy or dyskinesia. Seventy-one patients (82%) had an abnormal mode of left ventricular contractions and were categorized in five types (table 2 and fig. 2). These 71 cases included four with Marfan's syndrome.

**Type 1.** "Ballarina foot" pattern. This most frequently seen pattern comprised 27 cases and was characterized by early vigorous contraction of the posteromedial portion of the left ventricle with anterior convexity, giving rise to the configuration resembling ballerina foot at end-systole. Anterior
Table 2

**Left Ventricular Contraction Patterns and Functional Parameters**

<table>
<thead>
<tr>
<th>Contraction patterns</th>
<th>% Axis shortening (mean ± std)</th>
<th>MR No or insig</th>
<th>MR Sig</th>
<th>Prolapse Both LFL</th>
<th>Prolapse Severe</th>
<th>Functional parameters (mean ± std)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>L</td>
<td>AB</td>
<td>AM</td>
<td>AA</td>
<td>PB</td>
<td>PM</td>
</tr>
<tr>
<td>Ballerina foot</td>
<td>21.0</td>
<td>44.5</td>
<td>31.5</td>
<td>40.8</td>
<td>23.5</td>
<td>62.7</td>
</tr>
<tr>
<td>27 cases (34%)</td>
<td>±1.4</td>
<td>±2.8</td>
<td>±2.2</td>
<td>±2.6</td>
<td>±3.9</td>
<td>±2.6</td>
</tr>
<tr>
<td>Hour glass</td>
<td>20.9</td>
<td>52.0</td>
<td>63.3</td>
<td>51.0</td>
<td>14.8</td>
<td>59.2</td>
</tr>
<tr>
<td>22 cases (27%)</td>
<td>±1.2</td>
<td>±2.7</td>
<td>±2.9</td>
<td>±3.3</td>
<td>±4.8</td>
<td>±3.2</td>
</tr>
<tr>
<td>Inadequate long axis shortening</td>
<td>14.0</td>
<td>55.6</td>
<td>56.4</td>
<td>48.7</td>
<td>27.3</td>
<td>55.5</td>
</tr>
<tr>
<td>12 cases (15%)</td>
<td>±1.4</td>
<td>±3.2</td>
<td>±2.6</td>
<td>±5.5</td>
<td>±4.8</td>
<td>±4.2</td>
</tr>
<tr>
<td>Posterior akinesis</td>
<td>16.7</td>
<td>56.5</td>
<td>59.0</td>
<td>58.8</td>
<td>26.5</td>
<td>26.8</td>
</tr>
<tr>
<td>8 cases (10%)</td>
<td>±1.6</td>
<td>±4.7</td>
<td>±4.0</td>
<td>±3.1</td>
<td>±3.8</td>
<td>±2.6</td>
</tr>
<tr>
<td>Cavity obliteration</td>
<td>33.0</td>
<td>59.0</td>
<td>94.0</td>
<td>84.0</td>
<td>38.0</td>
<td>57.5</td>
</tr>
<tr>
<td>2 cases (3%)</td>
<td>±6.0</td>
<td>±9.0</td>
<td>±4.0</td>
<td>±6.0</td>
<td>±11.0</td>
<td>±3.5</td>
</tr>
<tr>
<td>Normal</td>
<td>24.8</td>
<td>48.9</td>
<td>38.0</td>
<td>56.6</td>
<td>28.4</td>
<td>50.8</td>
</tr>
</tbody>
</table>

Abbreviations: L = long axis; AB = anterobasal axis; AM = anteromediol axis; AA = anteropapical axis; PB = posterobasal axis; PM = posteromedial axis; PA = posteropapical axis; EF = ejection fraction (%); CI = cardiac index (L/min/m²); SI = stroke index (mL/beat/m²); SWI = stroke work index (gm m/m²); CI = contractility index (gm m/cm²); LFL = leaflets; INSIG = insignificant; SIG = significant; SEM = standard error of mean; MR = mitral regurgitation.
Types of left ventricular systolic contraction patterns in right anterior oblique projection.

Type 1. Normal contraction. Bulge in early diastole often accompanied this contraction pattern.

Type 2. "Hour glass" pattern. A vigorous ring-like contraction involving the middle portions of both anterior and posterior walls (usually beginning from the posterior wall) was demonstrated in 22 cases (27%).

Type 3. Inadequate long axis shortening. The contraction pattern was otherwise normal with symmetrical shortening of all other axes. There were 12 cases (15%) in this group.

Type 4. Posterior akinesis. Eight cases (10%) exhibited vigorous anterior wall contractions with posterior wall akinesis. Ejection fractions were normal in types 3 and 4 in spite of the limited ventricular motions.

Type 5. Cavity obliteration. In two cases (3%), hyperdynamic left ventricular contractions resulted in an almost complete approximation of anterior and inferior walls. This was seen at the apical and middle portions of the left ventricle and was reminiscent of the cavity obliteration pattern seen in hypertrophic myocardiopathy.

Diastolic Phase. Abnormalities in the mode of left ventricular relaxation were noted in 48 cases (55%), of which 44 were associated with systolic asynergy. The most frequently observed asymmetry was an early bulge of the anterior and/or apical portions of
the left ventricle resulting presumably from normal relaxation in these portions and sustained contraction in other portions of the myocardium. Figure 3 contrasts a normal left ventricle with that of a patient with “ballerina foot” pattern asynergy and early diastolic bulge. An early diastolic bulge involved the anterior wall in 58%, the posterior wall in 15%, and the apical area in 9% of cases. Diastolic asynergy also involved more than one site in a few cases.

**Figure 3**

*a) Left ventriculogram at onset of diastole shows prolapsed posterior (P) mitral leaflet. Arrows indicate inferior indentation and anterior bulge.*

*b) Prolapse of both anterior (A) and posterior (P) mitral leaflets. This patient has a normal pattern of ventricular contraction.*
Relative Frequencies of Ventricular Asynergy, Systolic Clicks and Murmurs

Figure 4 is a Venn diagram depicting the relative frequencies of ventricular asynergy, systolic clicks and murmurs, singularly or in combinations. Ventricular asynergy was present in 75 cases. In the remaining 12 cases, one case had a click alone, three cases had a murmur alone, seven cases had both clicks and murmurs, and one case, neither. Left ventricular asynergy was encountered in 24 cases with neither murmur or click, indicating silent mitral prolapse. There were 48 cases with clicks and 57 cases with murmurs. Of these, 40 cases had ventricular asynergy and 43 had clicks and murmurs. Only 36 cases had all three features.

Tricuspid Leaflet Prolapse and Right Ventricular Contractions

The excursion of the tricuspid valve ring was unusually vigorous in 62% of cases. The area of the tricuspid valve ring was clearly visualized in 28 cases. In the remaining three cases, the position of the catheter was such that tricuspid regurgitation obscured the ring and leaflet movements. Tricuspid leaflet prolapse was demonstrated in 15 cases (54%). Of these, 11 cases had inferior leaflet end-systolic prolapse and in four cases there was a systolic eversion of all tricuspid leaflets. Right heart pressures were normal or low.

Clefts and slight scalloping along the closure line of the tricuspid leaflets were occasionally noted and were considered to be normal. Figure 5 shows normal configuration of a tricuspid valve in end-systole compared to a prolapsing inferior leaflet.

Coronary Cineangiographic Findings

Selective coronary cineangiography was performed in 38 patients. Only three cases showed obstructive lesions. One case had diffuse beading of coronary arteries with neither significant obstruction nor ventricular asynergy. In one case there was beading of both left anterior descending and right coronary arteries but no significant obstruction. This patient exhibited only diastolic asynergy (anteromedial bulge). The other patient who had beading of the left anterior descending coronary artery and 75% occlusion of the right coronary artery showed posterior akinesis in systole as well as early diastolic bulge of the anteromedial portion.

A vigorous systolic swing of the right coronary artery was seen in 74% of cases and this was appreciated best in the RAO view. A “cork-screw” or tortuous appearance of at least one of the major vessels was found in 46% of cases (fig. 6).

Other anomalies of the coronary arteries were common. There was a short main left coronary artery in 63% of cases. One patient had separate origins of the left anterior descending and circumflex coronary arteries. The right coronary artery had an anomalous origin from the left coronary sinus in one case.

Discussion

This study shows ventricular myocardial abnormalities to be prevalent (86%) in PML. Asynergy was observed in both systole and diastole. More varieties of abnormal contraction patterns were observed than have been described previously. We have classified these ventricular motions according to their mode of systolic contraction: 1) “ballerina foot” pattern, 2) “hour glass” pattern, 3) inadequate long axis shortening, 4) posterior akinesis, and 5) cavity obliteration pattern. Of these, the most frequently observed patterns were those associated with early vigorous contraction of the postero-inferior portion with an anterior bulge (“ballerina foot” pattern) and the “hour glass” pattern in which there is a ringlike constriction at the midportion of the left ventricle. Both types have inferior indentations, differing according to whether or not the anterior wall bulged or indented. These two types of asynergy together made up 61% of the cases studied. There were only nine cases with normal ventricular contractions as seen with RAO projection.

There appears to be no common denominator among different types of asynergy in our series. Liedtke et al.,17 however, demonstrated consistently

![Venn diagram showing relative frequencies of ventricular asynergy, systolic clicks and murmurs, singularly or in combinations. The numbers in the diagram indicate the number of cases.](http://circ.ahajournals.org/)

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reduced contraction of the left ventricular inflow tract and the inferior papillary muscle in nine cases with systolic clicks syndrome, only two of which had angiographically proven valve prolapse. Implicit in their findings is the concept of primary myocardial disorder and that impaired mitral valve ring contraction and/or lack of support of posterior mitral leaflet may facilitate leaflet prolapse during systole. Although our methodology for study of ventricular geometry is not exactly comparable, we have not found a reduction in contraction of the inflow portion consistently; nor in our judgment...
was the valve ring inadequately contracting in many cases. Grossman et al. are of the opinion that approximation of the posterior or both papillary muscles to the mitral valve causes slackening of the chordae and favors the leaflet prolapse. This may apply in the "hour glass," "ballerina foot," and cavity obliteration patterns, but provides no explanation for leaflet prolapse in other patterns. The fact that only RAO projection was employed in the latter group leaves a possibility that ventricular asynergy might have been present but was not visualized in this projection. Another factor to be considered, at least in five of our cases, is a lower insertion of the mitral valve. The available information thus far is not sufficient to establish a clear-cut cause and result relationship between ventricular asynergy and leaflet prolapse.

Significant mitral regurgitation was more frequently associated with a disparity in the degree of anterior and posterior leaflets prolapse. Equal degrees of prolapse of both leaflets, even severe, was associated with mild or no regurgitation. These observations suggest that inadequate and asymmetrical apposition of the mitral leaflets is responsible for the mitral regurgitation. No conclusions can be drawn as to which pattern of ventricular contraction renders the mitral apparatus more prone to incompetence.

The coexistence of tricuspid leaflet prolapse (TLP) occurs in approximately half the cases of PML and may be associated with right ventricular asynergy. The latter is manifested by vigorous excursions of the tricuspid ring and abnormal movements of the inferior and anteroseptal walls of the right ventricle. The right coronary artery swing observed by selective arteriography is a guide to tricuspid ring motions, because the initial course of this artery traverses the right atrioventricular groove. TLP and right ventricular asynergy would add weight to the concept of this syndrome having a generalized element of myocardial abnormality, not limited to the left ventricle and mitral apparatus.

Dyspnea and fatigue are frequently present in patients with PML or TLP. Contrary to the report of Miller et al. emphasizing abnormal left ventricular function, our studies have failed to demonstrate abnormal hemodynamic parameters in most cases. We do not know the effect of long-standing ventricular asynergy on ventricular function but suspect that it may contribute to the development of the ventricular hypertrophy. Miller et al. noted left ventricular hypertrophy in ten of their 15 cases, 14 of which had only mild MR. The constrictive pattern ("square root sign") of the ventricular pressure curves described by Jeresaty in five of his 32 cases probably represents an impairment of the ventricular compliance due most likely to ventricular hypertrophy. Perhaps these cases also represent a more advanced stage in the spectrum of this syndrome. Serial ventriculography with studies of left ventricular function, if repeated during long-term follow-up, may add more information on the evolution and pathophysiology of this condition.

We recognize that prolapsing of the mitral valve leaflets may have heterogeneous origins. Leaflet prolapse may result from a deficit in the mechanics of the mitral apparatus, involving either valve leaflets themselves (myxomatous change), valve ring (dilatation), chordae (redundancy), papillary muscle (impaired contractility), or ventricular myocardium (inadequate support to mitral apparatus). The etiological basis for these mechanisms seems not well established in substantial numbers of reported cases. Even in the large heterogeneous series (130 cases) published by Pocock and Barlow, 61% (80 cases) had unknown etiology. Of these, 23 were familial and 16 had an associated congenital heart disease. There are several aspects of this syndrome that suggest a congenital basis: 1) female preponderance (83%), 2) reported familial occurrence, 3) frequent association of skeletal anomalies (47%), i.e., pectus excavatum, scoliosis, straight back, narrow AP diameter of the chest, and 4) high incidence of anomalous coronary arteries (40%). Coronary anomalies include cork-screw
appearance of the major vessels or their branches, \textsuperscript{13} short left main coronary artery and anomalous origin of the right coronary artery.

The selection of patients in this study was based on the presence of proven mitral leaflet prolapse and did not consider variant forms. We have encountered several instances of ventricular asynergy, chest pains, and palpitations in patients without valve prolapse. These cases may be \textit{forme fruste} examples of the same condition, but with a predominance of myocardial elements. Of significance is the fact that not all of the cases in this series exhibited typical nonejection clicks and/or mid or late systolic murmurs commonly associated with mitral leaflet prolapse. The Venn diagram of figure 4 serves to illustrate relative frequencies of ventricular asynergy, clicks, and murmurs, singularly or in combinations. The clinical features of ventricular asynergy, ST-T changes, arrhythmias, and atypical chest pain, may logically be attributed to myocardial abnormalities (myocardiopathy). Unresolved problems include ascertaining the causes of abnormal myocardial motions, chest pains and arrhythmias, the relationship to valve prolapse and the therapeutic challenges of more effectively controlling chest pains and arrhythmias.

Appendix

In angiocardiographic determination of the left ventricular volume (LVV) by single plane, area-length method, the LVV is calculated by the following formula:

$$LVV = 0.848 \times \frac{A^2}{L}$$

where \( A \) is the planimetered area of the left ventricular profile, and \( L \), the longest measured length on the projected left ventricular profile.

The ejection fraction (EF) is

$$EF = \frac{SV}{EDV} = \frac{EDV - ESV}{EDV} = 1 - \frac{ESV}{EDV}$$

$$= 1 - \frac{0.848 \times \frac{(As)^2}{L_s}}{0.848 \times \frac{(Ad)^2}{L_d}} = 1 - \frac{(As)^2}{(Ad)^2} \frac{L_s}{L_d}$$

where \( d \) and \( s \) denote end-diastole and end-systole, respectively. \( SV \) = stroke volume; \( EDV \) = end diastolic volume; \( ESV \) = end systolic volume.

References


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