Acute Mitral Regurgitation in Man

Hemodynamic Evidence and Observations Indicating an Early Role for the Pericardium

By Stuart H. Bartle, M.D., and Hector J. Hermann, M.D.

SUMMARY

Four patients are presented who developed acute mitral regurgitation 5 days, 2, 3, and 18 months, respectively, before cardiac catheterization. The hemodynamic findings in the three early cases resemble those found in the presence of a constricting pericardium: left and right ventricular, as well as left and right atrial pressures, equilibrated during diastole. As expected, giant left atrial v waves accompanied markedly elevated pulmonary arterial pressures and low cardiac indices. Biplane angiocardiography showed only slight left atrial and moderate left ventricular enlargement, but showed a large regurgitant fraction and dilated pulmonary veins. Orthopnea was transient or absent in two of the three early cases.

The pericardium may play a role in the early phase of acute mitral regurgitation in mimicking signs of right heart failure and preventing pulmonary edema. Development of increased pulmonary venous capacitance may be another major early mechanism of compensation since the pericardium may be preventing left atrial enlargement. Later in the course, after 6 months, the pericardium does not appear to be a factor in the hemodynamics, as noted in our patient and in patients reported by others.

Additional Indexing Words:
Ruptured papillary muscle Mitral valve surgery Ruptured chordae tendineae
Pulmonary venous capacitance Left ventricular volume Orthopnea
Left atrial volume Biplane angiocardiography Cardiac catheterization

Much attention has been given recently to a syndrome of acute mitral regurgitation resulting from rupture of either a papillary muscle or chordae tendineae. Clinical observations include the sudden onset of an apical systolic murmur often extending to the anterior chest or neck, the rapid onset of congestive heart failure concomitant with the appearance of the murmur, and the discrepancy between the severe degree of regurgitation and the modest cardiac, especially left atrial, enlargement. Noted originally by Corvisart, the relatively small left atrium is not seen in chronic cases, especially those with atrial fibrillation. This is attributed to poor compliance of the left atrial wall. Only a few of the cases reported, however, have been studied less than 6 months after the onset of mitral regurgitation.

This report is of three cases studied at 5 days, 2 months, and 3 months following the onset of acute mitral regurgitation. The hemodynamic findings resembled those observed in the presence of a constricting pericardium. Our attention was thus called to the possible role of the pericardium in this particular syndrome in which a volume load is suddenly imposed on the heart. It seemed likely to modify the clinical course as well as the hemodynamic and angiocardiographic findings. One additional patient with late mitral
incompetence (more than 6 months after the acute onset) is presented by way of contrast.

Methods

Patients underwent right- and left-sided cardiac catheterization by conventional methods. Biplane angiography was performed; pictures were taken at the rate of 4 to 6 films per second following the injection of 60 ml of contrast material into the left ventricle. Left atrial volume was measured according to the method of Arvidsson,\(^9\) left ventricular volume by a slight modification\(^10\) of the method of Dodge and associates,\(^11\) and left ventricular mass by the method of Rackley and associates.\(^12\) Regurgitant fraction was estimated by subtracting the net forward flow (obtained from the Fick or dye-dilution method) per beat from 0.85 x the angiographic stroke volume, 0.85 being a constant which corrects for the slightly increased stroke volumes measured using the angiographic method.\(^13\) Pressures were recorded on an oscilloscopic recorder* with Statham P23Db gauges which were calibrated before each procedure and adjusted to equal sensitivity at the gains used. Gauges were balanced before each run and referenced to zero level 5 cm below Louis's angle with the patient supine. Cardiac output was measured in duplicate by the Fick method (using the Van Slyke manometric method for blood oxygen content and the Scholander technique for lung gases) and the dye-dilution technique (indocyanine green being the indicator measured in a Gilford cuvette densitometer).

Report of Cases

Case 1

J. C., a 60-year-old white man without history of cardiovascular disease, was in good health according to routine annual medical examinations. Three months prior to admission he suffered chest pain. He then admitted to another hospital where a diagnosis of myocardial infarction was based on clinical findings and increased serum glutamic oxalacetic transaminase (SGOT) and lactic dehydrogenase (LDH). No diagnostic changes were observed in the electrocardiogram. Seven days later a harsh, holosystolic murmur was heard at the apex for the first time. The patient went rapidly into congestive heart failure with severe dyspnea at rest, and peripheral edema. Orthopnea was transiently present. He responded poorly to digitalization, diuretics, and prolonged rest in bed, and he was admitted to the University of Virginia Hospital for possible cardiac surgery.

Physical examination on admission showed a white male with mild dyspnea who was comfortable while lying flat in bed. Pulse was 90 and regular. Blood pressure was 120/70 mm Hg. Neck veins were distended with prominent pulsations when the patient assumed a 45° angle. The chest was clear to percussion and auscultation. Point of maximal impulse (PMI) was palpable 2 cm to the left of the midclavicular line. No thrill was present. A right ventricular thrust was palpable. A grade III/VI holosystolic high-pitched murmur was heard at the apex and radiated well into the axilla. The pulmonary second sound was accentuated. Hepatomegaly was present. There was no edema.

The electrocardiogram revealed normal sinus rhythm with an RSR' pattern in the right precordium. T waves were inverted in V1 to V3, and the S-T segments were slightly depressed in V2 through V6.

X-rays of the chest showed congestion of the pulmonary vasculature. The heart was enlarged primarily in the region of the left ventricle. There was no evidence of left atrial enlargement.

Cardiac catheterization was performed (tables 1 and 2) and the existence of severe mitral regurgitation was established. The regurgitant fraction was estimated at 64% of the stroke volume, although the mean left atrial volume was only 70 ml/m², slightly enlarged. Right ventricular end-diastolic and left ventricular end-diastolic pressures were 13 and 18, respectively, early in the procedure and before the angiograms; following the injection of contrast material they were equal (fig. 1). The angiograms are shown in figure 2.

At the time of surgery the mitral valve was examined. The anterior papillary muscle, supplying chordae to the aortic leaflet, was completely torn and the remains of the papillary muscle were atrophic. Repair was not possible. Accordingly, mitral valve replacement with a no. 4 Starr-Edwards prosthesis was performed. The tissue along the anulus of the mitral valve at the point of its attachment to the aortic leaflet was extremely friable. The pathological report of the chordae and attached papillary muscle, excised during surgery, showed focal degeneration of the heart muscle, with hemorrhage and mild leukocytic infiltration, secondary to occlusion of a coronary artery. The patient sustained cardiac arrest on the evening of the operation and died with bronchopneumonia and extensive cerebral damage. The Starr valve had become partially dislodged on the septal side. The left circumflex artery supplied the area of the anterior papill-
Table 1

Hemodynamic Data in Acute Mitral Regurgitation

<table>
<thead>
<tr>
<th></th>
<th>Recent J.C.</th>
<th>E.T.</th>
<th>J.K.</th>
<th>Late C.S.</th>
</tr>
</thead>
<tbody>
<tr>
<td>BSA (m²)</td>
<td>1.82</td>
<td>1.57</td>
<td>1.62</td>
<td>2.15</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>76</td>
<td>96</td>
<td>136</td>
<td>88</td>
</tr>
<tr>
<td>Pressure (mm Hg)</td>
<td>22</td>
<td>a:25</td>
<td>22</td>
<td>20</td>
</tr>
<tr>
<td>Right atrium</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right ventricle*</td>
<td>90/22</td>
<td>70/25</td>
<td>56/23</td>
<td>70/13</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>83/42,58</td>
<td>72/45,58</td>
<td>—</td>
<td>67/30,40</td>
</tr>
<tr>
<td>Central aorta</td>
<td>100/68,77</td>
<td>115/65</td>
<td>150/70</td>
<td>108/67</td>
</tr>
<tr>
<td>Left ventricle*</td>
<td>104/23</td>
<td>119/25</td>
<td>150/25</td>
<td>104/24</td>
</tr>
<tr>
<td>Left atrium</td>
<td>v:72,47</td>
<td>—</td>
<td>a:29</td>
<td>—</td>
</tr>
<tr>
<td>Pulmonary wedge</td>
<td>v:40,28</td>
<td>—</td>
<td>v:68,28</td>
<td>—</td>
</tr>
<tr>
<td>Dp/dt (mm Hg/sec)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiac index (L/min/m²)</td>
<td>2.0</td>
<td>1.5</td>
<td>1.8</td>
<td>1.9</td>
</tr>
<tr>
<td>Stroke index (ml/beat/m²)</td>
<td>26.2</td>
<td>15.4</td>
<td>13.2</td>
<td>21.6</td>
</tr>
<tr>
<td>PAR (dynes cm⁻²)</td>
<td>240</td>
<td>1050</td>
<td>—</td>
<td>235</td>
</tr>
</tbody>
</table>

*Ventricular pressures are systolic/end-diastolic. BSA = body surface area; dp/dt = maximum rate of rise of ventricular pressure; PAR = pulmonary arteriolar resistance.

Table 2

Volume Calculations in Acute Mitral Regurgitation

<table>
<thead>
<tr>
<th></th>
<th>Normal</th>
<th>Recent J.C.</th>
<th>E.T.</th>
<th>J.K.</th>
<th>Late C.S.</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVEDV (ml/m²)</td>
<td>&lt; 90</td>
<td>169</td>
<td>102</td>
<td>86</td>
<td>124</td>
</tr>
<tr>
<td>LVESV (ml/m²)</td>
<td>&lt; 39</td>
<td>63</td>
<td>45</td>
<td>39</td>
<td>56</td>
</tr>
<tr>
<td>Stroke volume (ml/m²)</td>
<td></td>
<td>106</td>
<td>57</td>
<td>47</td>
<td>68</td>
</tr>
<tr>
<td>Ejected fraction</td>
<td>0.61 ± 0.08</td>
<td>0.61</td>
<td>0.56</td>
<td>0.54</td>
<td>0.54</td>
</tr>
<tr>
<td>Regurgitant fraction</td>
<td></td>
<td>0.60</td>
<td>0.69</td>
<td>0.68</td>
<td>0.63</td>
</tr>
<tr>
<td>Regurgitant volume</td>
<td></td>
<td>64</td>
<td>35</td>
<td>27</td>
<td>37</td>
</tr>
<tr>
<td>LA max (ml/m²)</td>
<td></td>
<td>83</td>
<td>108</td>
<td>60</td>
<td>81</td>
</tr>
<tr>
<td>LA min (ml/m²)</td>
<td></td>
<td>56</td>
<td>82</td>
<td>37</td>
<td>60</td>
</tr>
<tr>
<td>LA Δ V (ml/m²)</td>
<td>42% of LV stroke volume*</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LV wall thickness (mm)</td>
<td>11.9 ± 1.6†</td>
<td>7.0</td>
<td>7.0</td>
<td>13.0</td>
<td>9.0</td>
</tr>
<tr>
<td>LV mass (g/m²)</td>
<td>99 ± 13†</td>
<td>147</td>
<td>85</td>
<td>205</td>
<td>195</td>
</tr>
</tbody>
</table>

LVEDV = left ventricular end-diastolic volume; LVESV = left ventricular end-systolic volume; stroke volume = end-diastolic minus end-systolic volume; ejected fraction = stroke volume/end-diastolic volume; regurgitant fraction, see text; regurgitant volume, see text; LA max = left atrial volume at (ventricular) end-systolic; LV min = left atrial volume at (ventricular) end-diastolic; LA Δ V = LA max − LA min; LV wall thickness, see text; LV mass, see text.

*Reference 16.
†Reference 17.
had rheumatic fever at the age of 7 years. He was admitted to the University of Virginia Hospital because of increasing dyspnea on exertion during the previous 3 years. He reported occasional episodes of paroxysmal nocturnal dyspnea, had been a heavy smoker for the past 25 years, and had a chronic nonproductive cough. His blood pressure was 110/80 and his pulse was 84 per minute and regular. Examination of the chest revealed distant breath sounds but was otherwise unremarkable. Auscultation of the heart was consistent with tight mitral stenosis. There were no signs of congestive failure.

Chest x-rays revealed a heart with normal transverse diameter but with evidence of hypertrophy of the right ventricle. The pulmonary arteries were prominent, and there was evidence of left atrial enlargement. Kerley A- and B-lines were present. On fluoroscopy no intracardiac calcifications were seen and left atrial enlargement was again noticed.

The electrocardiogram showed normal sinus rhythm and was essentially within normal limits.

A mitral commissurotomy was performed with the aid of the Tubbs dilator in December 1964. On the third postoperative day a holosystolic murmur at the apex radiating to the axilla was heard for the first time. Three days later the patient had an episode of severe respiratory distress with orthopnea. Crepitant rales were heard over both lung fields, and the liver was palpable 5 fingerbreadths below the right costal margin. He
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Figure 3

Simultaneous right ventricular and left ventricular pressures in patient E. T. (case 2) 2 months after appearance of mitral valve incompetence. Note equalization of pressures during diastole.

responded well to the usual treatment of pulmonary edema and remained relatively asymptomatic for 1 week; then symptoms and signs of acute left ventricular failure reappeared. He responded rapidly in the hospital to small doses of morphine but continued to complain of markedly decreased exercise tolerance. Two months later the patient was again admitted with severe shortness of breath, anorexia, and sharp transitory left-sided chest pain. Rales were present in both lung fields. Neck veins were distended. There was a grade IV/VI rough systolic murmur at the apex which radiated well toward the axilla and precordium. The pulmonary second sound was accentuated. There were no friction rubs or gallops. Hepatomegaly and ankle edema were present.

Cardiac catheterization was performed (tables 1 and 2). The patient was able to lie flat throughout the procedure. A markedly elevated pulmonary artery pressure and a giant left atrial v wave were found. Left and right ventricular end-diastolic pressures were markedly elevated and essentially equal to each other throughout the catheterization (fig. 3). There was a 10-mm Hg fall in the systolic pressure with inspiration. Left ventricular end-diastolic volume was only minimally increased, although regurgitation through the mitral valve was approximately 69% of the gross stroke volume. Calculated left atrial volume was approximately two times the normal size, or in the average range for a patient with long-standing mitral stenosis (fig. 4).

The patient continued to deteriorate rapidly and emergency replacement of the mitral valve using a no. 3 Starr-Edwards prosthesis was performed a few days later. The pericardium was dense and firmly adherent to the heart. The aortic leaflet of the mitral valve was torn in its midportion almost to the annulus and the papillary muscle attached laterally to this leaflet had been ruptured. All chordae were much shortened and both mitral valve leaflets were deformed. The patient failed to survive the procedure. Postmortem examination confirmed the operative findings.

Case 3

J. K. was an 18-year-old male with easy fatigability and dyspnea on exertion which had been present since early childhood. During the year prior to admission he had noticed progressive, decreased exercise tolerance and suprasternal chest pain associated with dyspnea.

Physical examination revealed a well-nourished, well-developed young man with blood pressure of 90/70. Pulse rate was 72 and regular. Abnormal physical findings were limited to his heart. PMI was in the sixth intercostal space 2 cm to the left of the midclavicular line. There was a palpable systolic thrill at the base of the heart, supra- sternal notch, and over both carotid arteries. A grade IV/VI harsh ejection systolic murmur was heard well over the left sternal border, and it radiated over the entire precordium and into both lung fields. Systolic bruits were also heard over all major peripheral vessels. There was a definite grade II diastolic decrescendo murmur on the left sternal border. P2 was slightly accentuated. The rest of the physical examination was entirely within normal limits.

Chest x-rays revealed left ventricular enlargement without other definite chamber enlargement. Lung fields were unremarkable.

Electrocardiogram showed sinus rhythm and left ventricular hypertrophy and strain.

Cardiac catheterization was performed, and a gradient of 148 mm Hg was found between the left ventricle and ascending aorta. Angiograms revealed a slightly enlarged left ventricular chamber with a thick wall and good systolic contraction. Cineangiograms obtained with injection of contrast medium into the root of the aorta demonstrated minimal aortic insufficiency, a fibrous subvalvular ring, and a predominant left coronary artery, although the fibrous ring was noted only in retrospect.

Open heart surgery was performed 1 month later and a normal tricuspid aortic valve was found. A ringlike fibrous band lay just below the base of the aortic valve. This fibrous obstruction was
Figure 4

Biplane angiograms in patient E. T. (case 2) showing increased left atrial volume during left ventricular systole (lower frames) and regurgitation of contrast media to the pulmonary veins.

carefully divided with a scalpel at five separate points. An aortic valve dilator was inserted and the ring was dilated to approximately 3 cm. This appeared to relieve the obstruction adequately. A rather ragged edge of fibrous ring remained, however, and this was grasped with an angle pituitary rongeur and incised circumferentially. The aortic root now admitted the dilator for a circumference of approximately 3 cm. The patient tolerated the procedure well, but over the next 3 days he began to have progressive difficulties manifested by shortness of breath. Orthopnea was not noted. A harsh holosystolic murmur in the precordium radiating toward the axilla appeared. On the third postoperative day cardiac catheterization was again performed (tables 1 and 2) and severe mitral regurgitation was seen through the anterior leaflet of the mitral valve. There was no residual obstruction at the fibrous ring site. It was of interest that pullback systolic
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gradients were now consistently obtained between the apex and the outflow tracts of the left ventricle. Left ventricular end-diastolic pressure was approximately equal to right ventricular end-diastolic pressure (fig. 5). The regurgitant fraction was estimated at 68% of the gross stroke volume. A variation of 14 mm Hg in peak aortic systolic pressure with respiration was noted. Angiocardiograms are shown in figure 6.

Since the patient continued to deteriorate rapidly, open heart surgery was performed on the fourth postoperative day. It was then discovered that the removal of the fibrous band, which lay in the subaortic region, and of the left coronary cusp had removed the fibrous support for the aortic leaflet of the mitral valve. Subsequently, the leaflet had torn away from its supporting fibrous structures and this had led to severe mitral insufficiency. Valvuloplasty was then performed which was thought to have cured the mitral insufficiency. The patient expired, however, 3 days after the second operation, primarily of pulmonary complications.

Late Mitral Insufficiency
Case 4

C. S., a 63-year-old white man, had suffered progressive dyspnea, pallor, and weakness for

Figure 5
Pressure patterns of patient J. K. (case 3) with incompetence of the anterior leaflet of the mitral valve of 5 days' duration. Note virtual equalization of pressures at end diastole.

Figure 6
Patient J. K. (case 3). Lateral plane angiograms following left ventricular injection. Note the normal-sized left atrium.
The precordium was not remarkably active, but there was a suggestion of a diffuse right ventricular lift. A grade IV holosystolic high-pitched murmur was heard at the apex radiating to the axilla, precordium, and both lung fields. The mitral first sound was markedly diminished in intensity. The pulmonary second sound was accentuated. Diastole was clear. The liver was palpable 1 fingerbreadth below the right costal margin and was not pulsatile. Peripheral pulses were full. There was no edema or cyanosis.

The electrocardiogram showed sinus tachycardia with first degree A-V block, as well as S-T changes of ischemia or divitalis effect. There were Q waves in leads I, aV_L, and V_2 through V_6 consistent with old anterolateral myocardial infarction. There was no evidence of left ventricular hypertrophy.

Chest x-rays showed slight cardiomegaly, apparently affecting both ventricles, marked congestion in both lung fields, and bilateral basilar pleural effusion. There was no evidence of left atrial enlargement. Vigorous systolic pulsations were seen on fluoroscopy.

Laboratory data, except for an iron deficiency anemia, were unremarkable. Several blood cultures were negative.

In May 1965, cardiac catheterization was performed (tables 1 and 2). Figure 7 shows simultaneous left and right ventricular pressure curves. End-diastolic pressure was clearly higher on the left side. Cineangiograms and biplane angiograms revealed a significant amount of mitral regurgitation estimated at 63%, but the end-diastolic volume was only 30% greater than normal, and the ejected fraction (stroke volume/end-diastolic volume) was at the lower limit of normal. The left atrium was not particularly enlarged.

In July 1965, mitral valve replacement was performed using a no. 3M Starr-Edwards prosthesis. At surgery the left atrium was thought to be small for a patient with this degree of mitral insufficiency. When the mitral valve was exposed, severe mitral insufficiency was noted. The mural leaflet of the mitral valve was completely flail. Chordae tendineae were torn at the junction with the papillary muscle, and the posterior papillary muscle was thin and friable. These changes were considered secondary to vascular occlusion.

The patient survived and was doing well when last seen in March 1967.

**Discussion**

All four patients suffered an acute onset of mitral regurgitation, though from three different causes. They resemble other reported cases in the rapid onset of congestive failure shortly after the appearance of the murmur.

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**Figure 7**

*Late mitral insufficiency. Left and right ventricular pressure curves in patient C. S. (case 4) with onset of mitral insufficiency 18 months previously. Note elevation of both end-diastolic pressures, clearly greater in left ventricle. Compare with figures 1, 3, and 5. ECG redrawn.*
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common membrane, the pericardium, limits their diastolic expansion as in constrictive pericarditis or pericardial tamponade.\textsuperscript{18, 19}

An alternative explanation is that right ventricular filling pressure increased coincidentally to the same level as that of the left ventricle. This is a most unusual occurrence, however, in chronic heart disease involving primarily the left side of the heart. There is no evidence other than the elevated filling pressure of impaired contractility of the right ventricle; the rate of rise of the right ventricular pressure is in the normal range\textsuperscript{20} and no evidence of tricuspid insufficiency is present. The theoretical possibility also exists that circulatory

![Figure 8](http://circ.ahajournals.org/)

*Simultaneous left and right ventricular pressure curves in patient with proven pericardial constriction. Note similarity of curves during diastole to curves of figures 1, 3, and 5.*

of mitral insufficiency, symptoms out of proportion to the degree of cardiomegaly, giant left atrial v waves, markedly elevated pulmonary arterial systolic pressures, low net cardiac output, and relatively small left atrial volumes. Of interest to us, however, is that the mitral insufficiency was of very recent origin in three of the cases, that orthpnea was inconspicuous in J. C. and J. K., and that the right and left ventricular pressures in diastole resembled those seen in restrictive pericardium\textsuperscript{8} (fig. 8), as do the right and left atrial pressures (fig. 9). A paradoxical pulse was not noted in J. C. or E. T. J. K. had a fall in peak systolic pressure of 14 mm Hg with inspiration.

**Role of the Pericardium in Ventricular Volume and Function**

Of the reports of other cases of less than 6 months' duration only that of Sanders and associates\textsuperscript{5} mentions RV pressures in two patients; left and right ventricular end-diastolic pressures were recorded in two patients at 18 versus 12, and 8 versus 5. No mention is made of simultaneity. The nearly identical pressure curves in the two ventricles, as well as in the atria, in our cases are strong evidence that a

![Figure 9](http://circ.ahajournals.org/)

*Simultaneous left and right atrial pressures in patient J. C. (case 1) Note equalization during diastole, but not during ventricular systole.*

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abnormalities secondary to acute mitral insufficiency in some way impair right ventricular function. There is, however, little experimental evidence for this.

If the pericardium in man has any hemodynamic function, it should prevent harmful dilatation of the heart during situations of acute volume or pressure load.† Dilatation could be harmful by resulting in (1) irreversible sarcomere stretch,21 (2) a deleterious increase in myocardial tension—for a thin-walled sphere, in the Laplace relation, T (tension) = P (pressure) × r (radius of curvature)—leading to excessive myocardial oxygen consumption22; or (3) significant mitral insufficiency. No one has shown conclusively that the pericardium restrains dilatation in man, although in the plethoric dog Holt and associates23 have shown that left and right ventricular end-diastolic pressures, right atrial pressure, and the pericardial pressures all rise linearly. Kenner and Wood, however, noted no systematic rise in pericardial pressure with aortic or pulmonary artery balloon occlusion.24 Observations in our laboratory, on the other hand, have suggested that the pericardium limits left ventricular end-diastolic volume in the acutely hypervolemic dog.25

Role of the Pericardium, Left Atrial Wall, and Pulmonary Veins in Limiting Left Atrial Volume

The development of acute mitral insufficiency stimulates the need for greater capacitance in the system between the pulmonary capillary bed and the left ventricle. The response of the system, whether in the left atrium or pulmonary venous bed, is not known.

Our patients confirm previous angiocardio- graphic measurements26, 27 and the clinical observations of others5, 6 in that the measured left atrial volumes are small in comparison to those of patients with valvular disease. Normal figures adjusted for weight or body surface area are not yet available in the literature. The upper limit of normal in our laboratory is less than 50 ml/m². Measured left atrial volumes were normal in J. K. 5 days after onset of mitral regurgitation in J. C. with 2 months of mitral regurgitation, and even in C. S. with 18 months of mitral regurgitation, although the pressure tracings show large v waves and elevated mean left atrial pressures. The twice normal left atrial volume of E. T. is attributable to his long-standing mitral stenosis. The close relationship of left atrial size to the duration of mitral insufficiency has been recognized in rheumatic heart disease,9, 26–28 and in patients with ruptured chordae tendineae.5, 6 The largest left atrial volumes have been measured in patients with long-standing mitral incompetence associated with atrial fibrillation.9, 29 There is no correlation between left atrial size and pressure.9, 26

The distensibility of the left atrial wall has been stressed as the chief factor in limiting left atrial size.5, 6, 28 Plasticity would be a more precise word in defining the chronic enlargement. Any consideration of the role of distensibility or plasticity however must also include the role of the pulmonary veins, which in figures 2 and 4 are shown to absorb a great deal of the actual contrast material, and of course the accompanying or preceding distending pressure. Volume absorption by the pulmonary veins26 explains the relatively small cyclic changes in left atrial volume (27, 27, and 23 ml/m², respectively) in the presence of large regurgitant volumes (64, 35, and 27 ml/m²). Though regurgitation to the pulmonary veins occurs in patients with equivalent amounts of regurgitation but for a longer time,29 the cyclic changes in left atrial size are considerably larger—84 ± 42 ml (not adjusted for body surface area).25 Burton26 has pointed out that small-radius vessels with very thin walls can withstand large transmural pressures because of the Laplace relation (see above). Thus the walls of the four pulmonary veins have an advantage over the left atrial wall and the pericardium in withstanding increases in pressure.

The pulmonary veins are distended through the entire cardiac cycle however. In contrast, apparently only during systole is there a large transmural pressure gradient across the left atrial wall, and only during diastole is the pericardium distended. During systole the left atrium is always larger than it is in diastole,16, 26, 27 but at this time the giant left
atrial \( v \) waves are not transmitted to the right atrium (fig. 9) as would be expected if there were a common, distended membrane; therefore transmural pressure in the atrium must fall somewhat as it partially empties, but if the pericardium is being distended at this time, transmural left atrial pressure will be actually quite low. The chief factor limiting left atrial enlargement may well be the development of greater capacitance by the pulmonary veins. Greater capacitance of the veins also lets the left atrium function more as a conduit during diastole, rather than as a reservoir,\(^9\) thus decreasing the atrial stroke volume. It should be noted here however that at least in one reported case,\(^6\) a thickening of the intimal lining of the pulmonary veins occurred implying reduced capacitance. This was not seen in our microscopic sections.

**Possible Role of the Pericardium in Altering Symptomatology**

If the ability of the left ventricle to increase its end-diastolic volume and thus its stroke volume lies in the fibrous pericardium rather than in the left ventricular wall, then one would expect this distended pericardium to transmit the elevated pressures during diastole to the right-sided chambers and limit the venous inflow. Thus an increase in pulmonary blood volume and severe, acute elevation of pulmonary capillary pressure might be modified somewhat. This is analogous to the studies in dogs by Berglund and associates\(^31\) in which the aorta was constricted with the pericardium intact. When the pericardium was constricted, right atrial pressure rose and left atrial pressure fell, as did both left and right ventricular stroke work. Thus the edemogenic influence of elevated pulmonary pressures was mitigated at the expense of a higher right atrial filling pressure. In severe left heart dilatation then one might expect to see early manifestations of elevated right-sided filling pressures.

Orthopnea results from increased venous return to the right heart and lungs due to a change in hydrostatic pressure relationships in the supine position. Orthopnea is a classic symptom of an elevated pulmonary capillary pressure, nearly great enough to exceed plasma oncotic pressure\(^32\) with the patient in the erect or sitting position. If right atrial pressure is already maximal, as in pericardial constriction, little further venous influx can occur acutely when the supine position is assumed. Thus lack of orthopnea in the presence of signs of elevated left-sided filling pressures suggests that the pericardium is limiting inflow. Wood\(^23\) reported on a patient with severe hypertension and pericardial effusion who was able to lie flat comfortably, and even able to tolerate his head being tilted down, without distress. He developed severe orthopnea and paroxysmal dyspnea only after pericardiocentesis. Nocturnal dyspnea, though related to posture, appears to be the result of a more gradual derangement of pulmonary vascular volume relationships. It is of interest that in none of Roberts and associates' 10 cases\(^6\) is orthopnea mentioned, though all the patients had easy fatigability, exertional, and nocturnal dyspnea and nine of the 10 had pedal edema. In only one of Menges and associates'\(^4\) six cases is it mentioned, and in two of Osmundson and associates' five\(^6\); in all three it occurred only after many months of symptoms. Orthpnea also occurred late in three of Bailey and Hickam's six cases.\(^34\) It appears to be only rarely present near the onset of mitral regurgitation\(^35\); if present it seems not to persist even in the presence of other symptoms of severe heart failure.

Whether the pericardium prevents mortality is even more speculative. One notes however that contrary to the speculation of others,\(^6\) death within a few days of onset of mitral regurgitation secondary to ruptured chordae tendineae alone (the purest form of acute mitral regurgitation) is rarely, if ever, seen. This is in contradistinction to the ruptured papillary muscle syndrome, of which a myocardial infarction is almost always a part and may be an important cause of congestive failure and death\(^36\) in addition to the organic mitral regurgitation. All deaths occurring from a few days to several months of onset of mitral regurgitation have been in patients with papillary muscle rupture.\(^1, 37, 38\)
Of the deaths from ruptured chordae tendineae without other major cardiac pathology in nonoperative cases,3, 36, 38 the earliest deaths recorded are at 8 months35 and 1 year (`case 19 of Osmundson and associates.3) One wonders why it takes so long to die with a lesion which probably has as high a regurgitant fraction at its onset as after compensation. Dilatation of the left ventricle and left atrium may increase the regurgitation somewhat, but the compensatory enlargement should mitigate these symptoms. Additional chordae may rupture, but pathological changes at postmortem examination have not established this,8, 34 It seems possible that the pericardium alleviates an otherwise overwhelming pulmonary congestion in the early months following the onset of acute mitral regurgitation.

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STUART H. BARTLE and HECTOR J. HERMANN

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