Premature Closure of the Mitral and Tricuspid Valves

By Donald A. Spring, M.D., John D. Folts, B.S.E.E., Ph.D.,
William P. Young, M.D., and George G. Rowe, M.D.

SUMMARY

Premature closure of an atrioventricular valve was observed in 80 of a group of 519 subjects catheterized for aortic insufficiency (AI) or mitral insufficiency (MI), or both. Sole premature mitral closure (PMC) was present in nine subjects, sole premature tricuspid closure (PTC) in 40, and combined PMC and PTC in 31. Clinically PMC was associated with a first sound that was soft or absent in 75% and an atrial contraction sound in 50% of the subjects with dominant aortic insufficiency. PMC or PTC, or both, were verified at surgery in three subjects and were produced experimentally in intact dogs by acute AI. The presence of PMC and PTC appears related to the severity and chronicity of valvular disease.

Additional Indexing Words:
Aortic insufficiency Mitral insufficiency Experimental aortic insufficiency
Pericardial restriction Bulging interventricular septum

Records from cardiac catheterization frequently reveal premature mitral valve closure in subjects with severe aortic or mitral insufficiency (fig. 1a) by demonstrating that left ventricular end-diastolic pressure exceeds left atrial pressure. A similar reversal of right atrial and ventricular end-diastolic pressures suggests premature tricuspid valve closure (fig. 1b). The incidence of these phenomena and their clinical and hemodynamic correlates was studied in human subjects and produced in dogs with acute aortic insufficiency.

Methods

Clinical and Hemodynamic Investigation

The records of 519 consecutive subjects who underwent cardiac catheterization for aortic or mitral valve disease were scrutinized for the presence of premature mitral or tricuspid valve closure as manifested by reversal of the usual end-diastolic atrioventricular pressure gradient. Two hundred sixty-two subjects had aortic insufficiency and 257 had mitral insufficiency. If insufficiency of both valves was present, the subjects were classed as having the more severe lesion.

All subjects were evaluated clinically and by right as well as retrograde and transseptal left heart catheterization. Via Statham P 23 Db strain gauges, pressures were recorded on a Waters Conley photographic recorder at a paper speed of 25 mm/sec. Simultaneous pressures were recorded by utilizing a Brockenbrough catheter in the right atrium and a no. 6 Lehman catheter in the right ventricle. We required the end-diastolic pressure to be the same during recording with both catheters in the ventricle and any observation which could not be confirmed from the reversed catheter positions was rejected. Similar technic was used for left-sided recordings. The reverse end-diastolic mitral or tricuspid gradient was accepted if it was 2 mm Hg or more. Valvular insufficiency was graded as mild, moderate, or severe, from the combined results of angiographic and indicator-dilution studies.

In three subjects during aortic valve replacement, right- and left-sided intracardiac pressure recordings were made before and after opening the pericardium.
diagnostic studies reported here. Cardiac output was obtained by indicator-dilution curves.

After control observations, aortic insufficiency was produced as described previously, and repeat observations were made. One dog was studied before and after acute aortic insufficiency by means of simultaneous right and left cineventriculography.

Results

The Clinical and Hemodynamic Investigation

Evidence for premature closure of the mitral valve (PMC), tricuspid valve (PTC), or both, was found in 80 subjects. Premature closure was associated with at least moderate, and usually severe, insufficiency of the aortic (58 cases), mitral (12 cases), or both left-sided valves (10 cases). PMC was less common (40 cases) than PTC (71 cases). Sole PMC was present in nine subjects (group 1), sole PTC in 40 (group 2), and combined PMC and PTC in 31 (group 3). The clinical and hemodynamic data are presented in table 1, and typical pressure tracings are given in figures 1 and 2.

Intracardiac pressures were recorded in one subject in each of these groups before and after opening the pericardium during aortic valve replacement. The pressure patterns were similar to those recorded at catheterization (fig. 2). In one operative study PTC was decreased (or abolished) after opening the pericardium widely, while the left ventricular end-diastolic pressure rose and premature mitral valve closure occurred.

In the 40 subjects with PMC the left ventricular diastolic flow period correlated negatively with left ventricular end-diastolic pressure \( r = -0.3691; P < 0.02 \) but not with the cardiac index. Thus a shortened filling time did not seriously limit mitral diastolic inflow. The reversed end-diastolic pressure gradient and end-diastolic pressure were directly related \( r = 0.4555; P < 0.01 \) in PMC. For the 71 subjects with PTC right ventricular end-diastolic pressure correlated negatively with right ventricular diastolic flow period \( r = -0.2486; P < 0.05 \). Left ventricular end-diastolic pressure correlated with the right ventricular end-diastolic reversed pressure gradient \( r = 0.3585; P < 0.01 \) and the

---

Experimental Animal Investigation

Twelve mongrel dogs, average weight 25 kg, were anesthetized, and cardiac catheters were positioned in the pulmonary artery and in both atria and ventricles. In six animals the pericardial space was also catheterized by a modified percutaneous technic. The manometer systems and recorder were those used in the human

---

\(^8\)Morphine sulfate, 3 mg/kg im, followed in 1 hour by 0.25 ml/kg of a 50/50 solution of veterinary pentobarbital and Dialurethane. Dialurethane contains diallylbarbituric acid, 100 mg/ml; monoethylurea, 400 mg/ml; and urethane, 400 mg/ml. Veterinary pentobarbital contains 60 mg/ml of pentobarbital.
right ventricular end-diastolic pressure \( (r = 0.7124; P < 0.001) \). There was no significant relationship between cardiac index and tricuspid diastolic flow period for those having PTC. In the group having PMC with aortic insufficiency, mild or moderate mitral insufficiency was a common finding (28 of 29 cases); tricuspid insufficiency, however, was present in only 14% of the combined group of 71 subjects with PTC.

**The Experimental Investigation**

After the induction of aortic insufficiency in the six animals in which the pericardial space had been catheterized, PMC was persistent in one animal and inconstant in two; no changes were observed in two animals, and one rapidly developed hypotension and died. PTC was observed in none of these animals; however, postmortem examination revealed that the pericardium had been torn. After the induction of aortic insufficiency in the six animals with intact pericardiums, persistent combined PMC and PTC were observed in three, inconstant PMC was present in one, no changes were observed in one animal, and one developed hypotension and died.

The hemodynamic findings in the four animals with persistent premature valve closure are summarized in table 2 and figure 3. The induction of aortic insufficiency caused the left ventricular end-diastolic pressure to rise rapidly producing typical PMC and PTC. Simultaneous biventricular angiography before and after the induction of aortic insufficiency revealed marked dilatation of the left ventricular chamber, with pronounced diastolic displacement of the left ventricular septum to the right (fig. 4).

**Discussion**

Premature mitral valve closure was postulated from results seen in simulated aortic insufficiency and after atrioventricular diastolic gradient reversal was demonstrated in subjects with severe aortic insufficiency. Subsequent reports from many laboratories have documented the occurrence of early mitral

---

**Figure 2**

Combined PMC and PTC at catheterization (panels a and b) and at operation (panels c and d) for severe aortic insufficiency. Abbreviations: RV = right ventricle; others same as in figure 1.
Table 1
Clinical Findings and Hemodynamic Summary in Subjects with Premature Atrioventricular Valve Closure

<table>
<thead>
<tr>
<th>Group</th>
<th>Sex and no.</th>
<th>Age (yr)*</th>
<th>Clinical diagnosis, etiology</th>
<th>Average duration</th>
<th>NYHA F. C.</th>
<th>Palpable enlargement</th>
<th>Decreased or absent first sound</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1:</td>
<td></td>
<td></td>
<td></td>
<td>Symptom (yr)</td>
<td>Murmur (yr)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Premature</td>
<td>9</td>
<td>40.5 ± 9.86</td>
<td>AI = 5</td>
<td>3.4</td>
<td>13.6</td>
<td>III-IV = 7</td>
<td>LV = 6</td>
</tr>
<tr>
<td>mitral valve</td>
<td>M = 8</td>
<td></td>
<td>AS &amp; AI = 2</td>
<td>(4 mo to)</td>
<td>I-II = 2</td>
<td>RV = 2</td>
<td></td>
</tr>
<tr>
<td>closure</td>
<td>F = 1</td>
<td></td>
<td>MI = 2</td>
<td>34 yr</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 2:</td>
<td>40</td>
<td>42.3 ± 13</td>
<td>AI = 12</td>
<td>3.7</td>
<td>12.5</td>
<td>III-IV = 20</td>
<td>LV = 22</td>
</tr>
<tr>
<td>Premature</td>
<td></td>
<td></td>
<td>AS &amp; AI = 10</td>
<td>(3 mo to)</td>
<td>I-II = 20</td>
<td>RV = 10</td>
<td></td>
</tr>
<tr>
<td>tricuspid</td>
<td>M = 26</td>
<td></td>
<td>MI = 14</td>
<td>50 yr</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>valve closure</td>
<td>F = 14</td>
<td></td>
<td>RHD = 34</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Group 3:</td>
<td>31</td>
<td>44.1 ± 13</td>
<td>AI = 17</td>
<td>2.4</td>
<td>16.4</td>
<td>III-IV = 16</td>
<td>LV = 27</td>
</tr>
<tr>
<td>Premature</td>
<td></td>
<td></td>
<td>AS &amp; AI = 12</td>
<td>(1 wk to)</td>
<td>I-II = 15</td>
<td>RV = 2</td>
<td></td>
</tr>
<tr>
<td>mitral and</td>
<td></td>
<td></td>
<td>MI = 2</td>
<td>38 yr</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>tricuspid</td>
<td>M = 27</td>
<td></td>
<td>RHD = 25</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>valve closure</td>
<td>F = 7</td>
<td></td>
<td>(+SBE = 2)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>ABE = 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>CMN = 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>CAVD = 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>IHSS = 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Marfan = 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*All values are mean ± standard deviation.
†Hemodynamic data for subjects with aortic insufficiency (AI) and subjects with mitral insufficiency (MI) presented separately.

Abbreviations: ABE = acute bacterial endocarditis; AI = aortic insufficiency; AF = atrial fibrillation; AS = aortic stenosis; ASHD = atherosclerotic heart disease; CAVD = congenital aortic valve disease; CMN = cystic medial necrosis (Erdheim); IHSS = idiopathic hypertrophic subaortic stenosis; LVSP = left ventricular systolic pressure; LVEDP = left ventricular end-diastolic pressure; MI = mitral insufficiency; NYHA F. C. = New York Heart Association functional capacity; RHD = rheumatic heart disease; RSR = regular sinus rhythm; RV = right ventricle; SBE = subacute bacterial endocarditis; S4 = left ventricular diastolic gallop; S1 = atrial contraction sound; TCT = torn chordae tendineae of mitral valve; TI = tricuspid insufficiency; 1° Blk = first-degree heart block; VDI = ventricular dia-stolic interval.

valve closure. Although in most cases premature mitral valve closure has been found to occur with severe aortic insufficiency, it also occurs with severe mitral insufficiency and is not a rare phenomenon. However only four examples of PMC were found from among our records of 257 subjects with primarily mitral disease, and the number cited in the literature is small.

Premature tricuspid valve closure, with or without accompanying PMC, was more common (13.6%) than PMC (7.7%) in this series. It is presumed that this phenomenon has not been described because the tricuspid valve is studied less rigorously than the aortic and mitral valves. Morphologically, the reversal of right-sided end-diastolic pressures in PTC is similar to the diastolic gradient reversal seen

Circulation, Volume XLV, March 1972
in PMC. The presence of PTC in a large number of diagnostic catheterization records (fig. 1), its persistence at open-heart surgery (fig. 2), and its reproduction in laboratory animals (fig. 3) document its authenticity. Both human and animal studies demonstrate that valve closure on the right side of the heart may be influenced by major insufficiency of the aortic or mitral valve.

Contrary to previous reports1-2 sudden, severe aortic insufficiency was found to be associated here with PMC infrequently (five of 36 subjects). The average duration of symptoms of about 2 to 4 years and known presence of a heart murmur for many years clearly indicate chronicity of the valvular lesions in most of these subjects. Thus, premature valve closure seems more closely related to the severity than to the duration of aortic or mitral insufficiency. In those subjects with dominant mitral insufficiency PTC was more common (10 subjects) than PMC (four subjects).

In 75% of our subjects with aortic insufficiency and PMC the first heart sound was soft or absent.1-2 Contrary to reports stressing its relative frequency,12,18 first-degree heart block was present in only six subjects (17%), and in five of these there was clear-cut pressure crossover during or before the atrial contraction wave. Since in these instances the atrium was contracting against a closed valve, it is not surprising that 50% of the human subjects with predominant aortic insufficiency

---

**Table:**

<table>
<thead>
<tr>
<th>S1</th>
<th>S4</th>
<th>Rhythm</th>
<th>Dominant lesion</th>
<th>Cardiac index (liters/min/m²)</th>
<th>Pressure (mm Hg)</th>
<th>Diastolic flow period (DFP) (msec)</th>
<th>DFP/VDI (% VDI)</th>
<th>End-diastolic gradient (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>5</td>
<td>RSR = 6</td>
<td>AI = 7</td>
<td>3.088 ± 0.787</td>
<td>156 ± 40</td>
<td>23 ± 8</td>
<td>6 ± 3</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1° Blk = 2</td>
<td>MI = 2</td>
<td></td>
<td></td>
<td>34 ± 13</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>AF = 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>8</td>
<td>RSR = 35</td>
<td>AI = 32†</td>
<td>3.382 ± 1.364</td>
<td>163 ± 48</td>
<td>18 ± 8</td>
<td>71 ± 11</td>
<td>5 ± 2</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(3 with</td>
<td></td>
<td></td>
<td>23 ± 11</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1° Blk = 3</td>
<td>sev. MI)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>AF = 2</td>
<td></td>
<td>3.602 ± 1.344</td>
<td>121 ± 19</td>
<td>16 ± 8</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>MI = 8</td>
<td>(mild TI =</td>
<td></td>
<td>10 ± 6</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>5/40)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>15</td>
<td>RSR = 24</td>
<td>AI = 29</td>
<td>3.425 ± 1.397</td>
<td>172 ± 43</td>
<td>20 ± 10</td>
<td>276 ± 112</td>
<td>68 ± 14</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(7 with</td>
<td></td>
<td></td>
<td>27 ± 11</td>
<td></td>
<td>(range,</td>
<td>4-94%,)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1° Blk = 6</td>
<td>sev. MI)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>AF = 1</td>
<td></td>
<td>MI = 2</td>
<td>255 ± 95</td>
<td>71 ± 14</td>
<td>5 ± 2</td>
<td>(range,</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>(mild TI =</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>40–90%).)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>5/31)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Tricuspid valve**

**Mitr valve**

_Circulation, Volume XLV, March 1972_
had a late diastolic sound (fourth heart sound). This forceable contraction is clearly reflected in the marked elevation of the left atrial “A” wave seen in figures 1 and 2.

It is interesting that the cardiac index was relatively normal in spite of rather severe valvular lesions and marked reduction in left ventricular diastolic flow period. This implies that ventricular filling from atrial systolic contraction was not important in the maintenance of an effective stroke output at these heart rates. Furthermore the modest elevations in left atrial and pulmonary artery pressures in both the human and experimental studies support the concept that PMC protects the pulmonary vascular bed from regurgitant aortic flow, and consequent elevated pressures. In addition PMC increases left ventricular end-diastolic pressure so that the force of the contraction is increased to compensate for regurgitation.8

Although direct proof of PMC has not been obtained, there is considerable deductive evidence for its occurrence: (a) the pressure curve morphology from man1–3, 9, 10, 12, 13 (figs. 1 and 2) and experimental animals (fig. 3) having severe aortic insufficiency; (b) longer diastolic cycles through prolonging aortic regurgitation produce higher left ventricular end-diastolic pressure and more marked end-diastolic atrioventricular pressure reversal; (c) a late diastolic sound that coincides with left atrioventricular pressure crossover during closure of natural2 and prosthetic14 mitral valves; and (d) implanted mitral electromagnetic flowmeters in calves with acute aortic insufficiency15 that show cessation of forward flow through the mitral valve at atrioventricular pressure crossover (premature closure). In the present investigation, the characteristic early pressure reversal of PMC was attained by the induction of aortic insufficiency in intact animals without alteration of the P-R interval. Since atrioventricular valve closure frequently took place before atrial contraction in the present data, an atriogenic mechanism of pre-closure must be uncommon. The presence of mild-to-moderate mitral insufficiency in 96% of the
Figure 3

Combined PMC and PTC resulting from the induction of severe aortic insufficiency in the intact experimental animal. (Compare panels a and b with c and d). Abbreviations same as in figures 1 and 2.

Figure 4

(a and b) Single frames taken from a cineangiogram made after contrast material was injected simultaneously into the right and left ventricles. (a) Before and (b) after induction of severe aortic insufficiency. These show a rightward shift of the interventricular septum and encroachment on the right ventricular cavity. (c) Diagrammatic representation of the changes caused by aortic insufficiency made by tracing and superimposing panels a and b. The solid lines represent panel a and the dotted lines panel b. The change in the left ventricular diastolic shape and the septal displacement to the right are shown.
subjects of this series with dominant aortic insufficiency correlates well with experimental observations and angiocardioographic demonstrations of diastolic mitral incompetence in human subjects with this condition.

While the mechanism of PMC in aortic insufficiency seems clear, the mechanism of PMC in dominant mitral insufficiency is much less clear. It is suggested that the overdistended left atrium empties so rapidly that when the ventricle reaches its capacity a rebound wave occurs and closes the mitral valve. The mechanisms causing PTC may differ from those causing PMC. The pericardium may play an important role in PTC since PTC was abolished or greatly reduced in one subject by a wide incision of the pericardium. This suggests that the pericardium distributed the pressure created by aortic regurgitation into all cardiac chambers including the right ventricle and right atrium. Since blood could flow freely out of the right atrium but not from the right ventricle, premature tricuspid closure would be expected to occur. When the pericardium no longer caused this redistribution of pressure, only PMC occurred. PTC did not occur in the series of experimental animals in which the pericardium had been torn, allowing the pericardial fluid to escape and removing or ameliorating the pericardial restrictive effect. Since PTC was seen to increase markedly during the increased aortic regurgitation of longer diastolic cycles and since biventricular angiograms during experimentally induced aortic insufficiency revealed that the septum appeared to encroach on the right ventricle in diastole (fig. 4), it appears likely that right ventricular compression is produced by left ventricular dilatation. The modest elevation in right ventricular end-diastolic pressures in these human and experimental subjects and the low incidence of right ventricular hypertrophy among human subjects with PTC support the idea that the right ventricle is only passively affected.

In conclusion, it appears that premature atrioventricular valve closure may be found on both sides of the heart. Furthermore, a variety of hemodynamic changes can alter the closure timing of both semilunar valves as well as the atrioventricular valves. It is hoped that an awareness of these possible variations in valve closure and their causative mechanisms may lead to increased understanding of the diseased heart.

References

7. MCKUSICK VA: Cardiovascular Sound in Health and Disease. Baltimore, Williams & Wilkins Co., 1958, p 278
11. KELLY ER, MORROW AG, BRAUNWALD E: Catheterization of the left side of the heart. New Eng J Med 262: 162, 1960

Circulation, Volume XLV, March 1972
21. Spring DA, Rowe GC: Backward transmission of the left atrial V wave and premature pulmonary valve closure. Amer Heart J 81: 327, 1971
Premature Closure of the Mitral and Tricuspid Valves
DONALD A. SPRING, JOHN D. FOLTS, WILLIAM P. YOUNG and GEORGE G. ROWE

Circulation. 1972;45:663-671
doi: 10.1161/01.CIR.45.3.663
Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1972 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/45/3/663

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/