Echocardiographic Demonstration of Early Mitral Valve Closure in Severe Aortic Insufficiency
Its Clinical Implications

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SUMMARY
Severe aortic insufficiency may accelerate mitral valve closure. We noted this echocardiographic finding in several patients with the acute onset of severe aortic insufficiency. Accordingly, we examined our total echocardiographic series retrospectively for early closure of the mitral valve (ECMV) in the setting of aortic insufficiency and found it in 11 of 53 patients with confirmed aortic insufficiency. During our study ECMV was fortuitously found in two other patients without aortic insufficiency.

ECMV occurred in late diastole following the echocardiographic "A" wave, often associated with a suppressed "A" wave (classified as type "A" ECMV), or in mid-diastole in the absence of an "A" wave (type "B" ECMV). ECMV presence and subtype, along with other clinical parameters, appeared to be useful in the serial evaluation of the patient with severe aortic insufficiency. Additionally, the analysis of ECMV type helped to clarify the mechanism and significance of the Austin Flint murmur.

Analysis of 17 patients with and without ECMV, with severe aortic insufficiency judged clinically (NYHA functional class III or IV) and angiographically (3+), indicated that only ECMV patients had acute aortic insufficiency and demonstrated diminished left ventricular size following successful aortic valve replacement.

Although due primarily to aortic insufficiency, ECMV could be influenced by rhythm or conduction abnormalities, co-existent cardiac lesions, and pharmacologic interventions. Exclusive of these factors, ECMV was an excellent sign of acute, torrential aortic insufficiency, and a simple noninvasive indicator of the patient requiring immediate aortic valve replacement.

Additional Indexing Words:
Angiography
Compliance
Austin Flint murmur
Aortic valve surgery

In 1886, following the original description of the murmur which bears his name, Austin Flint first postulated the occurrence of early closure of the mitral valve (ECMV) in severe aortic insufficiency. Many years later, investigators documented this event with catheterization data by showing that left ventricular pressure exceeds left atrial pressure prior to the onset of mechanical left ventricular systole. Pridie, while analyzing echocardiographic mitral valve changes in aortic valve disease, gathered 29 patients with this abnormality, divided them into slight versus severe early closure groups, and noted a clinical difference between them. Fortuin and Craig, in evaluating the etiology of the Austin Flint murmur, noted its relation to and alteration by degrees of ECMV. However, of 15 of their patients with severe aortic insufficiency, this abnormality was present in only a single case, a patient with bacterial endocarditis, severe hemodynamic compromise and an unstable course. They postulated that ECMV should be more common among populations with "a more severe hemodynamic burden" from aortic insufficiency. However, the characteristics of a group of patients with this finding have never been fully described.

In recent months, we have been impressed with the relative infrequency of this echocardiographic finding and its relationship to acute, severe aortic insufficiency. Therefore, we reviewed our experience with ECMV on echocardiograms in an effort to document its clinical correlations and better understand its mechanisms.

Methods
At our institution, over 1500 patients have had approximately 2000 echocardiographic studies during the year.

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last two years. Of these, 53 had aortic valve disease with aortic insufficiency documented cineangographically or diagnosed clinically, and subsequently confirmed at surgery or postmortem examination. In these 53 patients, we reviewed the history, physical examination, laboratory findings, echocardiogram and, where available, the catheterization studies. We took special interest in the course of the illness and its relationship to the presence of ECMV.

Echocardiographic studies with simultaneous electrocardiogram (ECG) were performed in the usual manner using a commercially available Picker Echoview X Echocardiograph. Early in our experience, recordings were made by the Polaroid technique. Later, a Honeywell 1856 Fiberoptics strip chart recorder was used. Great care was taken to insure that both leaflets of the mitral valve were visualized so that points of mid- and/or end-diastolic coaptation could be identified accurately.

Figure 1 illustrates the mitral valve echogram from a normal patient. The valve leaflets open abruptly at the onset of diastole reaching a maximum opening excursion at the “E” point. They immediately return to a partially closed position and re-open with atrial systole inscribing the “A” point. Mitral closure, coaptation of anterior and posterior leaflets, is seen to occur just prior to or past the peak of the simultaneous ECG R wave. In over 100 consecutive subjects without aortic insufficiency or atriocentricular (A-V) block the earliest point of coaptation was observed to occur 0.02 sec following the onset of the QRS complex.

ECMV was defined as complete coaptation of the anterior and posterior mitral valve leaflets at or prior to the initial inscription of the QRS complex. For analytical purposes, this phenomenon was divided into two types (fig. 2). Type A — ECMV with mitral valve closure following the “A” wave, but preceding the Q wave. The “A” wave in these complexes was often partially suppressed and frequently there was mitral coaptation in mid-diastole preceding this aborted “A” wave. Type B — ECMV with total, nontachycardia-related suppression of the echocardiographic “A” wave. Care was taken to avoid error in “A” wave analysis and recording and confusion with absent “A” waves due to rapid heart rate alone. Other echocardiographic measurements included left ventricular end-diastolic and end-systolic minor axis diameters, diastolic septal and posterior left ventricular wall thickness, calculated ejection fraction,

\[
\left( \frac{\text{left ventricular end-diastolic} - \text{end-systolic volume}}{\text{left ventricular end-diastolic volume}} \right)
\]

the slope of early diastolic mitral valve closure (E-F’ slope), minimum diastolic E point from septum separation, and opening (D-E) mitral excursion (fig. 1).

Left ventricular angiograms were performed on 39 patients. Seven had only supravalvular injections and seven were not studied. Because of the precarious clinical state of our patients, as well as concern for vegetation dislodgement, only five of our ECMV patients were studied hemodynamically. All those undergoing left ventricular angiography had a full right and left heart catheterization. Biplane angiograms were performed using a General Electric system, and were analyzed for left ventricular end-diastolic and end-systolic volume by the method of Goerke and Carlsson. Ejection fraction was calculated. Aortic insufficiency was judged angiographically on a scale of 0 to 3+, where 0 represented no regurgitant stream (no aortic insufficiency), 1+ represented a short early diastolic regurgitant puff (mild aortic insufficiency), 2+ represented short-lived left ventricular opacification appearing within three to four beats (moderate aortic insufficiency), and 3+ represented prolonged left ventricular opacification appearing within a single beat (severe aortic insufficiency). Aortic insufficiency was classified as acute if it appeared or was significantly exacerbated within three months of the echocardiogram; chronic, if present for greater than one year prior to the study; and subacute, if present for an intermediate period.

The echocardiogram was always performed within 24 to 48 hours of the catheterization study, and within one to two hours in our unstable patients. In one patient, (J.M.), the echocardiogram was done at the time of catheterization.

Finally, two additional patients with ECMV outside the setting of severe aortic insufficiency were identified. These patients were examined for possible alternate explanations for early mitral closure.

Results

Table 1 describes the over-all characteristics of the 53 aortic insufficiency patients divided into groups I and II depending on the presence or absence of ECMV. Group I consisted of ten patients with ECMV, four with type A, six with type B. There was a heavy male predominance (9:1) with an average age of 34. The etiology of aortic insufficiency in eight of the ten was infective endocarditis, one had disruption of a
noninfected prosthetic valve, and the last had aortic insufficiency of unknown cause. The average duration of symptoms prior to study was two months. Four of those with infective endocarditis who had known prior aortic valve disease which was clinically mild. Although each patient demonstrated one predominant ECMV type, the height and at times the presence of the echocardiographic "A" wave varied, occasionally beat to beat (fig. 3). All had an enlarged left ventricle on X-ray, echocardiogram and angiogram. Of those studied angiographically (five of ten), all had severe (3+) aortic insufficiency. All ten patients demonstrated the clinical findings of wide pulse pressure, low cardiac output and pulmonary edema, and all were NYHA functional class IV. Eight of ten came to aortic valve replacement, two of whom died before surgery could be performed. Surgical indications in this group were signs of marked left ventricular failure. The time course from the performance of the echocardiogram to either surgery or death was under 48 hours in five of ten patients and under two weeks in the remaining five. The absence of evidence of other associated cardiac lesions in this group was unexpected. The ten cases demonstrated lone, acute or subacute, severe aortic insufficiency (see table 2).

Group II was comprised of the 43 remaining aortic

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**Figure 2**

Table 1

Over-all Analysis of the Patient Population, Its Clinical Characteristics and Course

<table>
<thead>
<tr>
<th></th>
<th>Group I 10 ECMV patients</th>
<th>Group IIa 16 non ECMV patients (predominant AI)</th>
<th>Group Iib 27 non ECMV patients (coincident AI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Mean 34 (22 - 55)</td>
<td>Mean 44 (17 - 65)</td>
<td>Mean 57 (29 - 79)</td>
</tr>
<tr>
<td>Sex</td>
<td>9 male/1 female</td>
<td>12 male/4 female</td>
<td>17 male/10 female</td>
</tr>
<tr>
<td>Diagnosis</td>
<td>8 - IE</td>
<td>5 - RHD</td>
<td>18 - RHD</td>
</tr>
<tr>
<td></td>
<td>5 - drug addicts</td>
<td>5 - CHD</td>
<td>8 - DHD</td>
</tr>
<tr>
<td></td>
<td>2 - RHD</td>
<td>3 - DHD</td>
<td>1 - hypertensive</td>
</tr>
<tr>
<td></td>
<td>1 - post AVR</td>
<td>3 - IE</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 - DHD</td>
<td>1 - drug addict</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 - UHD</td>
<td>1 - RHD</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>1 - UHD</td>
<td></td>
</tr>
<tr>
<td>Angiographic severity</td>
<td>all 3+</td>
<td>7 - 3+</td>
<td>all 1+</td>
</tr>
<tr>
<td>(0 - 3+)</td>
<td></td>
<td>7 - 2+</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>2 - 1+</td>
<td></td>
</tr>
<tr>
<td>Other significant lesion</td>
<td>none</td>
<td>14/16</td>
<td>all</td>
</tr>
<tr>
<td>Mean AI symptom duration</td>
<td>2</td>
<td>60</td>
<td>NA</td>
</tr>
<tr>
<td>(months)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Functional class NYHA</td>
<td>all IV</td>
<td>1 - IV</td>
<td>NA</td>
</tr>
<tr>
<td>I-IV</td>
<td></td>
<td>6 - III</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>3 - II</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>6 - I</td>
<td></td>
</tr>
<tr>
<td>AVR for AI</td>
<td>8</td>
<td>10</td>
<td>NA</td>
</tr>
<tr>
<td>Death</td>
<td>4 - 2 post AVR</td>
<td>none</td>
<td>NA</td>
</tr>
<tr>
<td></td>
<td>2 acute</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: AVR = aortic valve replacement; CHD = congenital heart disease; DHD = degenerative heart disease; IE = infective endocarditis; RHD = rheumatic heart disease; UHD = unknown heart disease; NA = not applicable.

The average age was 44 with a 3:1 male to female ratio. In this group aortic insufficiency resulted from a wide spectrum of etiologies with functional class varying from I-IV. Average symptom duration was five years. Fourteen of 16 had other significant cardiac lesions. Based on clinical criteria, ten of 16 went to surgery and none died (table 2).

In groups I and IIa (table 2), functional class correlated weakly with left ventricular end-diastolic volume ($r = 0.563$) and echocardiographic left ventricular end-diastolic dimensions ($r = 0.604$), but better with angiographic severity ($r = 0.862$) and left ventricular end-diastolic pressure ($r = 0.825$). As has

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

*Echo from Patient M.R. — variation in subtypes. Shown is the mitral valve echoogram of patient M.R., which consisted primarily of type A ECMV complexes. This strip demonstrates three complexes of the B configuration and three of the A type. Variation between types was occasionally seen, as might be expected in this precarious hemodynamic state, sensitive to minor fluctuations in transmural flow. Such fluctuations may occur during respiration and with minor changes in heart rate.*

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Table 2

The Clinical, Echocardiographic and Catheterization Data on All Patients with Predominant Aortic Insufficiency

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age</th>
<th>Sex</th>
<th>Lesion</th>
<th>Diagnosis</th>
<th>Symptom duration (months)</th>
<th>Func. class</th>
<th>ECMV (type)</th>
<th>LVEDd (cm)</th>
<th>EF (NL&gt;65%)</th>
<th>Echo data</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>D to E (mm)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

**Group I**

| H.A.    | 25  | M   | IE     | AI        | 3                          | IV           | B           | 6.1        | 73          | 20        | 130       |
| J.A.    | 55  | M   | IE     | AI        | 1                          | IV           | B           | 6.0        | 69          | 19        | 140       |
| R.B.    | 42  | M   | IE     | AI        | 4                          | IV           | B           | 6.6        | 73          | 16        | 110       |
| O.C.    | 43  | M   | UHD    | AI        | 4                          | IV           | A           | 6.4        | 74          | 13        | 110       |
| E.H.    | 31  | M   | IE     | AI        | 1                          | IV           | A           | 7.6        | 76          | 13        | 110       |
| B.J.    | 35  | M   | IE     | AI        | 1                          | IV           | A           | 6.5        | 80          | 18        | 120       |
| K.K.    | 37  | M   | DHD    | AI        | 0.25                       | IV           | B           | 6.1        | 77          | 15        | 120       |
| A.M.    | 24  | M   | IE     | AI        | 4                          | IV           | B           | 6.0        | 70          | 18        | 160       |
| J.M.    | 22  | M   | IE     | AI        | 2                          | IV           | B           | 5.9        | 71          | 24        | 180       |
| M.M.    | 32  | F   | IE     | AI        | 0.5                        | IV           | A           | 6.8        | 84          | 18        | 120       |

**Group IIa**

| W.B.    | 56  | M   | RHD    | AS, AI/MI              | 36                         | II           | —           | 7.5        | 85          | 15        | 45        |
| W.C.    | 65  | M   | ESH    | AI-MI, MS             | 36                         | III          | —           | 6.3        | 55          | 13        | 90        |
| M.C.    | 50  | F   | DHD    | AI                    | 24                         | III          | —           | 6.2        | 72          | 17        | 100       |
| R.H.    | 17  | M   | CHD    | AS, AI/*              | 3                          | I            | A           | 5.5        | 84          | 27        | 105       |
| T.J.    | 63  | M   | DHD    | AS, AI/*              | 24                         | II           | —           | 4.5        | 70          | 15        | 50        |
| C.K.    | 31  | M   | IE     | AI-MI                | 3                          | III          | —           | —          | —           | 25        | 130       |
| W.K.    | 45  | M   | IE     | AI                    | 6                          | III          | —           | 6.2        | 68          | 21        | 40        |
| A.L.    | 26  | M   | RHD    | AI-MI                | 60                         | IV           | —           | 6.5        | 75          | 16        | 120       |
| P.L.    | 48  | F   | RHD    | AS, AI/MS, MI        | 12                         | II           | —           | 5.0        | 60          | 15        | 30        |
| C.L.    | 40  | F   | CHD    | AI-MS, MI            | 240                        | II           | —           | 5.2        | 70          | 10        | 25        |
| B.M.    | 31  | M   | CHD    | AS, AI-MI            | 120                        | III          | —           | 7.6        | 23          | 19        | 150       |
| K.O.    | 30  | M   | CHD    | AS, AI/*             | 24                         | I            | —           | 6.0        | 63          | 18        | 80        |
| C.P.    | 58  | M   | DHD    | AS, AI/*             | 24                         | IV           | —           | 6.4        | 35          | 15        | 80        |
| J.T.    | 54  | F   | RHD    | AS, AI/*             | 144                        | I            | —           | 4.5        | 75          | 15        | 120       |
| P.U.    | 22  | M   | IE     | AI-MI                | 3                          | III          | —           | 5.8        | 70          | 26        | 160       |
| R.W.    | 26  | M   | CHD    | AS, AI/*             | 136                        | I            | —           | 5.3        | 74          | 15        | 120       |

Abbreviations: AI = aortic insufficiency; AS = aortic stenosis; CHD = congenital heart disease; CO = cardiac output; D-E = echocardiogram opening mitral excursion; DHD = degenerative heart disease; ECMV = early closure of the mitral valve; EF = ejection fraction; E-F slope = echocardiogram slope of mid-diastolic mitral valve closure; E-septum = echocardiogram E point to septum separation; Func. class = functional class NYHA I-IV; HR = heart rate; IE = infective endocarditis; LVEDd = left ventricular end diastolic dimension; LVEDP = left ventricular end diastolic pressure; LVEF = angiocardiographic left ventricular end diastolic volume; MI = mitral insufficiency; MS = mitral stenosis; PCWP = pulmonary capillary wedge pressure; RF = regurgitant fraction; RHD = rheumatic heart disease; UHD = unknown heart disease; Net CO = the cardiac output via the Fick method; RF = the ratio: Fick cardiac output/angiographic cardiac output.

been shown in other studies,10 left ventricular end-diastolic pressure correlated poorly with left ventricular diastolic volume (r = 0.634). Comparing findings of the ten group I and seven group IIa patients with clinically (functional class III and IV) and angiographically severe (3+) aortic insufficiency, there was no significant difference in heart rate, net cardiac output, or left ventricular end-diastolic dimension. Although different clinically, it was, in fact, impossible to distinguish ECMV patients from other patients with predominant aortic insufficiency on the basis of any hemodynamic criteria other than left ventricular end-diastolic pressure (P = 0.001). Left ventricular end-diastolic pressure was highest in the ECMV patients. This pressure was documented to exceed mean pulmonary capillary wedge pressure prior to the onset of electrical systole in two of our group I patients (fig. 4).

Group IIb, the 27 remaining aortic insufficiency patients without ECMV, had an average age of 57 and a minimal male predominance. They were primarily patients with longstanding rheumatic disease, had mild aortic insufficiency by all criteria and were brought to our attention due to a more significant cardiac lesion for which they were being evaluated.

One of our group I patients, J.M., was given amyl nitrite (fig. 5). As expected, left ventricular end-diastolic pressure fell relative to mean pulmonary capillary wedge pressure and the echocardiographic study showed a reappearance of the 'A' wave and the normalization of the timing of mitral valve closure. Also, the early diastolic Austin Flint murmur recorded...
initially disappeared. Four of our type B ECMV patients were noted to have an early diastolic Austin Flint murmur. One of our type A ECMV patients had a presystolic component as well, while another type A had no Austin Flint murmur recorded. Five of the group IIA patients had only a presystolic component. These correspond well with the findings and postulates of Craige et al.6, 12 as they related severity of aortic insufficiency to the presence and form of the Austin Flint murmur. All six patients in group I who survived surgery showed loss of ECMV and reappearence of the echocardiographic “A” wave following aortic valve replacement without significant change in heart rate (fig. 6).

Diastolic mitral vibrations were present in all 53 cases of aortic insufficiency except two with mitral stenosis and thickened mitral leaflets.11 This finding was an extremely selective and sensitive indicator of aortic insufficiency, but showed no quantitative value contrary to earlier thoughts.8 Early diastolic mitral closure rate, E-F slope, was noted to be essentially normal (mean, 120 mm/sec)13 in contrast to earlier findings.7

Another observation was the reduced mitral opening excursion and increased E-point from septum separation in most of the severe aortic insufficiency patients. We believe this finding represents the combination of a volume overload and reduced net cardiac output typical of this state.

Among the group IIA patients was one case, R.H., with apparent mild aortic insufficiency, yet demonstrating type A ECMV at a slow heart rate (fig. 7). During exercise, with acceleration of heart rate, the timing of mitral valve closure returned to normal. During our study two other patients were noted to have first degree A-V block, and also demonstrated ECMV, again type A. Clinically, these patients did not have aortic insufficiency. While patients with bradyarrhythmias showed opposition of the mitral leaflets in diastole, there was no true coaptation in the absence of other factors. Three group IIA patients with severe aortic insufficiency (functional class III and 3+...
angiographic severity) and subacute onset had normal mitral closure.

Discussion

Clinical Correlations

In 1971, Pridie made the original observation of echocardiographic ECMV in severe aortic insufficiency. This finding had been predicted, as ECMV had been referred to as an influential factor in the Austin Flint murmur for several years. Investigators of severe aortic insufficiency have reported late diastolic mitral regurgitation and simultaneous pressure measurements have confirmed a reversed pressure gradient, left ventricular pressure exceeding pulmonary capillary wedge pressure late in diastole. ECMV is the direct outcome of such a hemodynamic state and is illustrated in two of our patients in figure 3.

Although the relationship between acute severe aortic insufficiency and ECMV has been noted the clinical and hemodynamic characteristics of patients with ECMV have not been studied in depth. The outstanding feature of our ECMV group (group I) was its homogeneity representing lone, acute and severe aortic insufficiency clinically, hemodynamically and angiographically. In contrast, Pridie’s study suggested that ECMV is more common and less specific. We feel the difficulty in identifying the posterior leaflet of the mitral valve may have led to this disparity (fig. 8). In our opinion, this accounts for the disparity in the specificity of ECMV found between his study and ours.

The homogeneity seen in group I was not present in our group II patients which included seven angiographically severe (3+) aortic insufficiency patients and 36 with less severe aortic insufficiency. Although angiographic results were often a reliable guide to clinical status, some patients with angiographically severe aortic insufficiency had only mild clinical and hemodynamic findings, while others with moderate (2+) aortic insufficiency had severe symptoms in the presence of often hemodynamically mild associated lesions. All in group I required immediate surgical correction and suffered a high mortality. However, of those in group IIa, two-thirds underwent elective aortic valve replacement and none has died.

Contrary to the findings of Spring et al. ECMV clearly differentiated the acute congestive aortic insufficiency patient with or without congestive heart failure (group IIa). It has been shown by others that these lesions are different clinically and hemodynamically, and that acute severe aortic insufficiency is associated with a high mortality, if not surgically corrected immediately. Our observations confirm this belief. There were only three patients noted to have ECMV outside of this clinical setting (false positives). Only one, R.H., had mild aortic insufficiency and two others had first degree A-V block. Three patients in group IIa with moderately severe aortic insufficiency (functional class III and 3+ angiographic severity), and a history of subacute onset without ECMV could be interpreted as false positives.
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negatives. ECMV seems to be a specific and sensitive noninvasive indicator of acute or subacute torrential aortic insufficiency.

In this study, patients demonstrating ECMV in the presence of normal or accelerated heart rate and normal rhythm could be identified clinically as those with severe aortic insufficiency. However, in the individual case of severe aortic insufficiency, it was sometimes difficult to decide on the importance of immediate surgical intervention. The presence of ECMV now

Figure 5

Effect of amyl nitrite on ECMV. Simultaneous ECG, apex phonocardiogram (phono), left ventricular pressure (LVP) and pulmonary capillary wedge pressure (PCWP) of patient F.J. is shown at rest (left) and following inhalation of amyl nitrite (right). Below are the corresponding echocardiographic studies. Note that following amyl nitrite there is a fall in pulmonary capillary wedge pressure and a marked fall in left ventricular end-diastolic pressure allowing reopening of the mitral valve with atrial systole, reappearance of the "A" wave and normalisation of closure. The mid-diastolic Austin Flint murmur disappears after amyl nitrite.

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Restoration of normal mitral closure. Above is the echocardiogram of patient R.B. prior to (left) and following aortic valve replacement (right). Below are those of patient K.K. displayed in similar fashion. Note the reappearance of the "A" wave and delayed mitral closure postoperatively in both cases. The heart rate has slowed minimally in the postoperative study of both patients. This slowing might favor "A" wave reappearance, but would prejudice against normalization of valve closure. In fact, changes here are due not to heart rate alteration, but to restoration of aortic valve competence. The ECG in the bottom left study is partially obscured.

prejudices us in favor of quick intervention. Lacking sufficient history or opportunity for serial observation, the presence of ECMV assumes yet greater importance and seems to testify to the acute and urgent nature of the problem. Further, the appearance of ECMV or its progression from type A to type B might support or establish the impression of clinical deterioration. After corrective surgery its disappearance would parallel improvement (fig. 6). As the acute severe lesion was usually infective endocarditis, our diagnostic suspicions were turned in that direction when ECMV was seen. If present in a patient with a prosthetic valve, significant dehiscence was sought actively.

The characteristics of the Austin Flint murmur appear to correlate roughly with the type of ECMV (A or B). Extreme early closure (type B) was associated with only a mid-diastolic rumble, while type A was seen in the presence of a presystolic component. Others have reported that the Austin Flint murmur is
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present only in severe aortic insufficiency. Furthermore, it has been shown that the loss of the presystolic component denotes an increasingly severe lesion; likewise, type B ECMV should denote a more severe lesion. However, both types of ECMV and Austin Flint murmurs were present in the group I patients and failed to clinically categorize patients in this group. The murmur was also present in group IIa patients. Although the different characteristics of ECMV and the murmur may aid in the serial evaluation of a single patient, and in the diagnosis of a severe lesion, our data suggest that ECMV, but not the Austin Flint murmur, can differentiate acute, severe, high-risk, aortic insufficiency (group I) from chronic, severe aortic insufficiency patients (group IIa).

Factors Affecting ECMV

We have shown how heart rate might affect ECMV (fig. 7). The hemodynamic basis for this effect is illustrated in figure 9. Here, simultaneous aortic and left ventricular pressure tracings from patient J.M. are

Figure 7

ECMV without severe aortic insufficiency — false positives. Echocardiographic study of patient R.H. at rest (left) and during handgrip exercise (right). Note shift of mitral valve closure point (solid vertical lines) with increased heart rate. The effect of increased afterload on handgrip alone, disregarding the rate increase, would aggravate ECMV by virtue of increasing the regurgitant fraction in this patient with mild aortic insufficiency.

Figure 8

Technical difficulties in echocardiographic diagnosis of ECMV. Illustrated is an echocardiogram from a patient at first felt to have ECMV. Arrow A indicates a point where leaflet coaptation might appear to occur. Imaging of both leaflets, however, shows true closure to occur normally, arrow B.
shown. Left ventricular pressure is seen to equilibrate with aortic diastolic pressure late in diastole. During the pause following a premature ventricular systole, equilibration is present for a longer period and occurs earlier relative to the succeeding QRS than with normal cycles. In the same way, slowing the heart rate allows more time for left ventricular aortic diastolic pressure equilibration, a relatively higher left ventricular end-diastolic pressure and a greater possibility for ECMV. On this basis, left ventricular aortic diastolic pressure equilibration occurring more rapidly is typical of "wide open" aortic insufficiency seen commonly in the acute severe lesion and results in ECMV at normal or increased heart rates, while arrhythmias with a long diastolic pause may cause ECMV in the presence of only mild aortic insufficiency (fig. 7). Small variations in the exact timing of mitral closure as seen, even beat to beat, in patient M.R. (fig. 3), represent movement on a spectrum of ECMV, influenced by alterations in heart rate and respiration as well as aortic insufficiency.

Factors other than rate may affect ECMV. The presence of mitral stenosis, as seen in two of our group IIa patients, W.C. and P.L., would raise the left atrial pressure and delay or prevent ECMV. Hemodynamic changes as those precipitated by vasodilators such as amyl nitrite (fig. 5), will also influence the appearance of ECMV, tending to delay it. Such pharmacologic interventions would decrease regurgitant fraction and thus increase net cardiac output. Of considerable interest was the fact that amyl nitrite was used therapeutically in this manner prior to successful surgery on patient J.M. The effectiveness of such treatment was gauged clinically, as well as by its effect on ECMV and the Austin Flint murmur. This suggests a new approach to the emergency treatment of acute aortic insufficiency. Conversely, an increase in peripheral resistance would aggravate aortic insufficiency and accentuate ECMV.

The role of first degree A-V block in ECMV, noted in two of our patients without aortic insufficiency, is more difficult to explain. We hypothesize first degree A-V block may unmask atrial relaxation resulting in a reversed diastolic left ventricular-left atrial pressure gradient.

Mitral insufficiency has been reported to cause ECMV. We have seen no examples of this relationship, even in cases of acute severe mitral incompetence.

Thus, heart rate, conduction abnormalities, associated cardiac lesions, and pharmacologic interventions all influence the appearance of ECMV and testify to its dynamic nature. These factors must all be considered when evaluating the clinical significance of this finding in relation to aortic insufficiency. Of course, transducer malposition or suboptimal gain settings may result in unreliable data (fig. 7).

The exact cause for the relationship of ECMV with acute severe aortic insufficiency is problematic. Dilatation has been shown to represent an early compensation for the left ventricular volume overload of aortic insufficiency, and was seen even in our acute patients. Gault et al. have shown that patients with chronic aortic insufficiency develop increased left ventricular compliance. This increased compliance would dampen the effects of large regurgitant volumes of left ventricular end-diastolic pressure. Patients with acute aortic insufficiency probably possess left ventricles of normal compliance. ECMV is likely related to the extreme elevation of left ventricular diastolic pressure due to reflux of large volumes into a dilated, yet relatively noncompliant, chamber. Surprisingly, there was no significant difference between left ventricular size and regurgitant volumes in severe aortic insufficiency patients with or without ECMV. The
changes here are largely reversible as all our surviving ECMV patients underwent dramatic involution of left ventricular size by X-ray and echocardiogram following surgery.

ECMV is thus a sensitive and specific indicator of severe acute aortic insufficiency. Its appearance is influenced by heart rate and associated heart disease. Excluding these factors, ECMV identifies those patients who have largely reversible disease and who require immediate surgical intervention.

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