Effective Closure of the Mitral Valve without Atrial Systole

By Eugene Braunwald, M.D., S. David Rockoff, M.D., H. Newland Oldham, Jr., M.D., and John Ross, Jr., M.D.

The precise mechanisms responsible for closure of the atroventricular valves under various physiologic and pathologic circumstances have not yet been completely defined. The factors considered to be of importance have been summarized by McKusick and include (1) elevation of pressure in the ventricle to a level greater than that in the atrium, (2) contraction of the myocardium surrounding the atroventricular valve, resulting in a reduction of the cross-sectional area of the valve ring, (3) contraction of the papillary muscles, thus drawing the valve cusps together, and (4) approximation of the valve cusps in the wake of the jet produced by atrial systole. During recent years there has been particular interest in the role played by atrial contraction, an interest that stems largely from the recent reappraisals of atrial function in experimental animals and man. The availability of relatively simple techniques for abolishing atrial fibrillation and other atrial arrhythmias, and of cardiac pacemakers that permit maintenance of the normal sequence of atrial and ventricular contraction, may provide additional impetus for defining the role of an effective, appropriately timed, atrial contraction in mitral and tricuspid valve closure.

In the course of diagnostic studies in which selective angiograms with left ventricular injection were performed, we became impressed that the presence, or the development of various cardiac arrhythmias was not necessarily associated with perceptible mitral regurgitation. This observation, which was similar to that of Bjork and co-workers, is not compatible with the view that atrial systole is always necessary for mitral valve closure. In order to determine the frequency of this occurrence, all of our angiograms with left ventricular opacification were reviewed.

Methods and Results

A review of 500 selective angiograms in which the left ventricle was opacified yielded 106 studies in which the mitral valve was competent. In 43 of these studies the valve was competent in the absence of an appropriately timed atrial systole. Twenty-nine of these 43 studies were biphase serial angiograms, made with Schonander film changers, at rates of 4 to 12 films per second. The other 14 were cineangiographic studies at film speeds of 30 frames per second, with either 16 or 35-mm. films.

The diagnoses of the patients studied are listed in Table 1. The contrast material (Renovist) was injected into the left ventricle in 26 patients;

Table 1

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>No. of patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>A. Patients with sinus rhythm</td>
<td></td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
<td>11</td>
</tr>
<tr>
<td>Aortic valve disease</td>
<td>8</td>
</tr>
<tr>
<td>Ventricular septal defect</td>
<td>5</td>
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<tr>
<td>Atrial septal defect</td>
<td>5</td>
</tr>
<tr>
<td>Hypertrophic subaortic stenosis</td>
<td>6</td>
</tr>
<tr>
<td>Transposition of great arteries</td>
<td>2</td>
</tr>
<tr>
<td>Mitral stenosis</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>38</td>
</tr>
<tr>
<td>B. Patients with atrial fibrillation</td>
<td></td>
</tr>
<tr>
<td>Mitral stenosis</td>
<td>3*</td>
</tr>
<tr>
<td>Postoperative atrial septal defect</td>
<td>1</td>
</tr>
<tr>
<td>Cardiomyopathy</td>
<td>1</td>
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<tr>
<td></td>
<td>5</td>
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*Two of these patients had associated aortic valve disease.

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in 19 of them the catheter was introduced retrograde through the aortic valve, while in the other seven patients transseptal left ventricular catheterization was carried out.14 In 14 patients the injection was made into the right ventricle, the left ventricle being well opacified through a large ventricular septal defect. In three patients with severe aortic regurgitation the left ventricle was opacified following injection into the ascending aorta.

In each instance the angiogram was of excellent quality, the left ventricle was densely opacified and no detectable opacification of the left atrium occurred (figs. 1-3). Special attention was directed to the area of the left atrium and mitral valve to ensure that the projection and exposure factors would have allowed recognition of even small amounts of regurgitated contrast material; angiograms of less than excellent quality were not included in the analysis. Standard lead II of the electrocardiogram was recorded on a multichannel recorder before, during, and after the injection of contrast material. When biplane serial radiographic studies were performed, the exposure of each film was recorded together with the electrocardiogram. With the cineangiographic studies precise synchronization of the electrocardiogram with each cine frame was made possible by a mechanical marker in the radiologic field whose movement was triggered by the electrocardiogram. In some studies, the Cinetrace,* a device which permits recording of the electrocardiogram and other oscilloscopic data directly on the cine film,15 was used.

Normal sinus rhythm was present prior to the injection in 38 patients; 27 of them developed multiple (three to five) ventricular extrasystoles, while the other 11 exhibited brief bouts of ventricular tachycardia during the exposure of the angiogram (figs. 1 and 2). Atrial fibrillation was present in the other five patients prior to the injection of the contrast material. No ventricular extrasystoles occurred in two of them (fig. 3); one patient developed multiple premature ventricular contractions during the exposure of the angiogram, while the other two developed ventricular tachycardia (six or more consecutive ventricular extrasystoles).

Discussion

The results of many experiments in the anesthetized dog with an open chest, and usually with an open pericardium, support the view that atrial contraction can result in complete closure of the atrioventricular valves.2,5-8 Using indicator-dilution methods, Daley et al.7 demonstrated in dogs that mitral incompetence develops with the onset of atrial fibrillation, Friedman and co-workers showed that extrasystoles frequently induced mitral regurgitation,16 while Skinner et al. demonstrated in dogs with heart block that mitral regurgitation was present when atrial systole was improperly timed8 or when atrial fibrillation was present.17 In addition, Grant et al.18 showed in patients with complete atrioventricular block that a small reversed pressure gradient (ventricular pressure exceeding that in the atrium) existed across the tricuspid valve following a prolonged diastole and suggested that the tricuspid valve had been closed as a result of atrial activity. These workers postulated that atrial relaxation reverses the atrioventricular pressure gradient and is important in closing the tricuspid valve. Alterations in the atrial pressure pulse, which indicate that tricuspid regurgitation occurs commonly in patients with atrial arrhythmias,7,19,20 have also been reported. As a result of these experimental and clinical studies, the view that a properly timed atrial contraction is essential for complete closure of the atrioventricular valves in early systole has become widely accepted.

On the other hand, a number of investigators, using indicator-dilution methods with left ventricular injection and left atrial sampling, have shown that atrial fibrillation or multiple ventricular premature contractions that alter the temporal relationship between atrial and ventricular contraction do not induce mitral regurgitant flow in the dog.21-24 Similarly, Paul et al., utilizing selective angiography with left ventricular injection, found no evidence for mitral regurgitation in dogs with atrial fibrillation,25 while Vanden-berg, Sturm, and Wood, working with a videodensitometer to detect reflux of contrast material, found that isolated ventricular extrasystoles were not associated with significant regurgitation.26 Both Conn and co-workers21 and Burchell9 have observed patients with atrial fibrillation in whom indicator in-
Figure 1
Lateral roentgenograms from a biplane serialographic left ventricular (L.V.) angiogram in a 27-year-old patient with valvular aortic stenosis. Panel A shows film with transseptal cath-
CLOSURE OF MITRAL VALVE

Frames from a cineangiogram performed in the left posterior oblique projection through a retrograde left ventricular (L.V.) catheter in a 45-year-old patient with pure mitral stenosis, during an arrhythmia superimposed upon atrial fibrillation (see fig. 2B). Note the absence of regurgitation of contrast material into the left atrium (L.A.). The mechanical marker (C.M.) was activated by the R wave of the electrocardiogram. Ao. = aorta.

The present study was undertaken because of the above controversy and the lack of information concerning the role played by atrial contraction in closure of atrioventricular valves in man. Forty-three patients with various forms of congenital and acquired heart disease were found to have no detectable mitral regurgitation in the presence of cardiac arrhythmias in which the normal sequence of atrial and ventricular contraction was disturbed. The arrhythmias in which the mitral valve appeared to be competent included atrial fibrillation alone (fig. 3), atrial fibrillation associated with frequent ventricular extrasystoles, sinus rhythm with multiple extrasystoles, and ventricular tachycardia (figs. 1 and 2). The angiocardiographic technic is a sensitive one, and when technically excellent films are available even trivial degrees of regurgitation can be detected. From these observations, it is concluded that a properly timed atrial contraction is not always essential for mitral valve closure in man. In addition, the finding that mitral regurgitation was

Circulation, Volume XXXIII, March 1966
not present in seven patients in whom the catheter was introduced into the left ventricle through the mitral valve, indicates that trans-septal left ventricular catheterization does not necessarily interfere with complete closure of the mitral valve.

From these observations we do not intend to suggest that atrial systole never plays a role in mitral valve closure, or that atrioventricular dissociation, such as occurs with ventricular premature contractions, cannot result in valvular regurgitation. Indeed, we have observed a large number of patients whose angiocardiograms exhibited mitral regurgitation in the presence of atrial arrhythmias or atrioventricular dissociation, and the possibility cannot be excluded that the absence of an appropriately timed atrial systole played a role in the regurgitation that was present. Since all of these patients had organic heart disease, it is also possible that an abnormality of the mitral valve was wholly or partly responsible for the observed regurgitation. Thus, analysis of the left ventricular angiocardiograms in which mitral regurgitation occurred provided no definitive information concerning the importance of atrial systole in mitral valve closure, and consequently conclusions cannot be drawn concerning the relative incidence of mitral regurgitation during various arrhythmias. The demonstration in 43 patients, however, that mitral regurgitation did not occur in the absence of an appropriately timed atrial contraction does allow the conclusion that such an atrial contraction need not occur for effective closure of the mitral valve to take place.

Summary

There has been considerable dispute concerning the role of atrial contraction in the closure of the atrioventricular valves. Selective angiocardiograms with left ventricular
opacification in 43 patients having a variety of arrhythmias that resulted in the absence of atrial systole, or in an abnormal temporal relationship between atrial and ventricular contractions, revealed no evidence of mitral regurgitation. From this observation it is concluded that a properly timed atrial contraction is not always essential for effective closure of the mitral valve in man.

References
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Circulation. 1966;33:404-409
doi: 10.1161/01.CIR.33.3.404

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