Mitral Valve Closure in Atrial Flutter

MARK A. GREENBERG, M.D., L. SCOTT HERMAN, M.D., AND MICHAEL V. COHEN, M.D.

SUMMARY Twelve patients who had atrial flutter without clinical, echocardiographic or angiographic evidence of aortic insufficiency were studied with simultaneous echo- and phonocardiograms. In patients with high-grade atrioventricular (AV) block, the mitral valve opened and closed with each flutter wave. Of seven patients, two had persistent and five had intermittent early mitral valve closure before QRS inscription. In five patients (three with 2:1 AV block) the mitral valve closed on time. In one patient with a mitral valve prosthesis, echocardiography and cinefluorography demonstrated closure during mid-diastole, with reopening in late diastole after a flutter wave. Final valve closure occurred before the onset of the QRS, and each closure was associated with a click. Simultaneous phonocardiographic analysis in these patients demonstrated that the first heart sound intensity was inversely related to the degree of mitral valve preclosure. This relationship was independent of the length of the RR interval. Thus, atrial flutter independent of any other cause of abnormal hemodynamics may produce early mitral valve closure. The echocardiographic finding of premature mitral valve closure in patients with aortic insufficiency in the presence of atrial flutter, as well as other atrial arrhythmias, may not have the same diagnostic or prognostic significance previously described in patients with sinus rhythm and normal AV conduction.

ALTHOUGH MOTION of the mitral valve has been carefully studied in animals, both in vitro after valve excision and in situ, analysis of valve motion in man has, until recently, been necessarily inexact. Because cineangiography could visualize only an interface between unopacified left atrial blood and opacified left ventricular blood, and phonocardiography could record only variations in intensity of the mitral component of the first heart sound, precise timing of leaflet movement was not feasible. However, the introduction of echocardiography made possible more accurate identification of leaflet opening and closing. In 1969 Zaky and colleagues used ultrasound to study the respective roles of the left atrium and ventricle in closure of the mitral valve. They observed pre-systolic closure of the mitral valve in many patients with PR intervals > 0.17 second. Subsequently, patients with hemodynamically severe aortic regurgitation were also noted to have echocardiographically identifiable early mitral valve closure, and some authors correlated the degree of early closure with the severity of the aortic leak. Early mitral valve closure associated with either prolongation of the PR interval or aortic regurgitation has also been described in patients with normal prosthetic mitral valves were direct cinefluorographic visualization of valve motion is possible. Despite this interest in the timing of mitral closure, the effects of atrial contraction abnormalities have not been well studied. Zoneraiach's observations in two patients with atrial flutter did not include analysis of valve closure. Consequently, the present report describes the effects of atrial flutter on closure of normal and prosthetic mitral valves.

Methods

We studied 12 consecutive patients with atrial flutter seen in the noninvasive laboratory of Montefiore Hospital and Medical Center over a 3-month period. There were five males and seven females, ages 32-70 years (mean 59 years). One patient with combined rheumatic stenosis and insufficiency of the mitral and aortic valves had double valve replacement with Starr-Edwards ball-valve prostheses. One patient had previous repair of an atrial septal defect and, because of atrial flutter with a slow ventricular response, required permanent ventricular pacing. Two patients had sick sinus syndrome and one had a permanent pacemaker. Other clinical conditions included nonrheumatic mitral insufficiency in two patients, and, in one patient each, mitral valve prolapse, hyperthyroidism, and uremia with hypertension. Three additional patients had atrial flutter of undetermined etiology. One of these patients had had a normal cardiac catheterization 1 year earlier.

After clinical evaluation, echocardiographic studies were performed with either an Irex or Hoffrel Ultrasonoscope and a 2.25 MHz transducer focused at 7.5 cm. Echocardiographic information was displayed on a multichannel oscillographic recorder (Irex or Kent Cambridge Instrument Company), and permanent photographic records were made. In all patients a continuous electrocardiographic lead II was also displayed. A standard 12-lead ECG revealed absence of an isoelectric segment in lead II preceding QRS inscription. Ten patients had simultaneous phonocardiograms recorded with a piezoelectric crystal microphone positioned on the chest wall to record the first heart sound clearly. Studies were completed with the patient either supine or in the left lateral decubitus position to enable optimal visualization of mitral valve motion. Carotid sinus massage was applied to slow the ventricular response in five patients with predominantly 2:1 atrioventricular (AV) block. In two patients who had permanent demand ventricular inhibited (VVI) pacemakers inserted because of high-grade AV block and slow ventricular rates, the pace-
maker was temporarily inhibited with an external trigger. Except in the patient with double valve replacement, careful auscultation in this group failed to reveal evidence of aortic insufficiency. Because of a diastolic murmur in the patient with prosthetic heart valves, we performed aortic root angiography, which revealed a normally functioning aortic prosthesis without evidence of a leak. No patient had echocardiographic signs of aortic insufficiency.

We performed biplane cinefluorography of prosthetic valve motion in one patient with aortic and mitral valve replacement using a Phillips image intensifier and recording on 35 mm film at 50 frames/sec. A lead II ECG was displayed in one corner of each cine frame to permit accurate timing of poppet motion. The excursion of the two poppets was measured on each cine frame for one complete cardiac cycle as the distance from the sewing ring, and then expressed as a percentage of the maximum excursion. Cine as well as echocardiographic and phonocardiographic studies were repeated in this patient after conversion of the rhythm to atrial fibrillation by bursts of rapid atrial pacing and then to sinus rhythm with first-degree AV block after treatment with quinidine sulfate.

Mitral valve closure was defined as the point at which the anterior and posterior leaflets came together (C point) (fig. 1). In the one patient with a prosthetic valve, termination of the rapid posterior movement of the ball was considered the point of closure. Normal valve closure occurs no earlier than 0.05 second after the onset of the QRS.\(^\text{18}\) Closure of a normal prosthetic Starr-Edwards valve has been observed to occur 0.07 second after the beginning of QRS inscription in patients in sinus rhythm.\(^\text{9}\) Valve closure was considered premature if it occurred earlier than 0.02 second after the onset of inscription of the QRS complex.

**Results**

Three patterns of mitral valve closure could be identified in this population: persistent early closure of the mitral valve, intermittent early closure of the mitral valve in patients with variable degrees of AV block, and normal timing of mitral valve closure. Two patients had persistent early closure of the mitral valve. One of these patients had mitral valve prolapse and normal left atrial size, and the other had double valve replacement. As shown in figure 1, the C point of the mitral valve in the first patient occurred prematurely, and was recorded at the onset of inscription of the QRS complex. A representative echocardiogram of the patient (AM) who had double valve replacement is presented in figure 2. Only in beats with 2:1 AV block and, therefore, short RR intervals (beat 2) was the mitral prosthesis closed after QRS inscription. In all cardiac cycles in which there was greater than 2:1 AV block (beats 1 and 3) the mitral valve prosthesis consistently closed 180–220 msec before inscription of the QRS complex. Early diastolic closure produced an audible mitral valve closure click (MCC\(_1\)). The valve reopened later in diastole after a flutter wave and reclosed before the onset of the QRS complex. This reclosure was also associated with a closure click (MCC\(_2\)). After this second closure, we

---

**Figure 1.** Mitral valve echogram in patient with atrial flutter with 4:1 atrioventricular block and holo-systolic prolapse (Pro) of both leaflets. Mitral valve closure occurs at the onset of inscription of the QRS complex as indicated by the vertical line. AML = anterior mitral leaflet; PML = posterior mitral leaflet.
observed no further poppet motion until the valve reopened at the beginning of the next diastole. This unusual echocardiographic pattern of mitral poppet motion was confirmed by ball-valve cinefluorography. There was full excursion of the poppet within the cage and no abnormal rocking motion. The excursions of both the aortic and mitral valve poppets are plotted in figure 3 for one cardiac cycle with an RR interval of 890 msec. This figure shows that the mitral valve poppet first closed 180 msec before the onset of the QRS, reopened, and closed again 45 msec before the start of the next QRS. Because of auscultation of an early diastolic murmur and cine and echocardiographic findings of early mitral valve closure, we thought that this patient might have an aortic para-valvular leak; aortic angiography, however, revealed no aortic insufficiency.

Five patients had intermittent early closure of the mitral valve (fig. 4). Two of these patients had atrial flutter of undetermined etiology, and the other three patients had diagnoses of atrial septal defect repair, uremia with hypertensive heart disease, and sick sinus syndrome. Two patients in this group demonstrated presystolic closure only when higher degrees of AV block were produced by carotid sinus massage. No patient exhibited early closure while in atrial flutter with 2:1 AV block. During periods of variable AV block, there was a characteristic variation in the intensity of the first heart sound (S₁) (fig. 4). S₁ was markedly diminished in those beats with presystolic closure of the valve (beats 2–4). In the beats in which the mitral valve closed after the QRS, there was a loud S₁ (beats 1 and 5).

In five patients — two with non-rheumatic mitral regurgitation, one with hyperthyroidism, one with sick sinus syndrome, and one with atrial flutter of undetermined etiology — the mitral valve consistently closed after the QRS complex. Three of these patients had 2:1 AV block and in two of them, higher degrees of AV block could not be induced with carotid sinus massage. In patients in this group with higher degrees of block, the echocardiogram demonstrated opening and closing of the mitral valve with each flutter wave. However, final mitral valve closure occurred after the QRS (fig. 5).

Because the rhythm in patient AM changed from atrial flutter to atrial fibrillation and then to a sinus mechanism, additional studies were performed. The echocardiogram during atrial fibrillation is presented in figure 6. During long RR intervals there was early onset of closure of the mitral prosthesis with undulations of the ball during the remainder of diastole until final closure after the QRS. During these beats the mitral valve closure sounds were markedly attenuated (beats 2 and 3). Occasionally (beat 1), early closure of the ball-valve prosthesis would generate an early audible click (MCC₁) followed by a discrete reopening of the valve after a coarse fibrillatory wave. Final mitral valve closure after the QRS would then produce a second audible click (MCC₂). During
shorter RR intervals (beat 4), the mitral valve closed after the R wave, and distinct valve clicks were produced by sequential closing of the mitral and opening of the aortic prosthesis.

Figure 7 shows the motion of the mitral ball-valve prosthesis during sinus rhythm with first-degree heart block (PR interval 0.36 second). The prosthesis closed early after atrial systole, producing an attenuated closure sound, and remained closed during the remainder of diastole. Cinefluoroscopy confirmed these echocardiographic observations.

Discussion

The contribution of atrial systole to mitral valve closure has been known for over 50 years. The classic work by Dean1 in the isolated perfused heart demonstrated that with a normal PR interval, AV valve closure was initiated by atrial systole and completed by ventricular systole. Atrial systole adds an increment of blood to the already distended left ventricle, thus increasing left ventricular pressure.2 Final mitral valve closure occurs at the time of reversal of
the pressure gradient between the left atrium and the left ventricle. Rapid increase in left ventricular pressure during diastole related to the regurgitant jet in patients with aortic insufficiency accounts for the frequently observed premature mitral valve closure. With a normal PR interval, the increase in left ventricular pressure is initiated by atrial systole and completed during isovolumic left ventricular contraction. Prolongation of the PR interval allows completion of mitral valve closure during atrial contraction before the onset of left ventricular systole. Echocardiography has confirmed that early closure of the mitral
valve may occur in both sinus rhythm with first-degree AV block and complete AV block.6,15,16

The present study extends these observations on the role of atrial systole in mitral valve closure to patients with atrial flutter. No patient with atrial flutter and 2:1 AV block demonstrated early closure of the mitral valve. With an average RR interval of 400 msec, the abbreviated duration of diastole did not permit diastolic closure of the mitral valve to occur. Over one-half of the patients in this series had higher degrees of AV block and exhibited either persistent or intermittent early closure of the mitral valve. The mechanism of this early closure is similar to that previously proposed in patients with first-degree or complete AV block. Effective atrial contraction initiated by the flutter wave would diminish the size of the mitral annulus by one-half to two-thirds.4 Increased blood flow across the mitral valve would also generate an increased left ventricular diastolic pressure. In those patients with atrial flutter and high degrees of AV block, additional flutter waves and atrial contractions during long RR cycles would increase the chances of raising left ventricular diastolic pressure, reversing the AV pressure gradient and closing the mitral valve prematurely. Mitral valve closure would depend at least in part on the timing of the last flutter wave preceding ventricular systole. In Shah’s series of patients with complete AV block, early effective atrial contraction did not cause valve closure in the one patient with acute left ventricular failure.15

Differences in left ventricular function and the strength as well as the timing of left atrial contraction may be responsible for the variations in the mitral valve closure demonstrated in our patient population. Sarnoff and co-workers17 demonstrated in the canine heart in vivo that the ability of atrial systole to cause mitral valve closure independently was in part dependent on the strength and speed of atrial contraction and relaxation. Carotid sinus massage applied in five of our patients to increase AV block may have had a depressant effect on atrial activity. Such vagotonic maneuvers have been demonstrated to reduce left atrial contractility, prolong relaxation and interfere with atrioventricular mitral valve closure.17 Of the five patients in our series with 2:1 AV block with atrial flutter, carotid sinus massage produced higher degrees of block in three. Two of these three had only intermittent early mitral valve closure, while there was no effect on the timing of mitral closure in the third. It is possible that more consistent early closure might have occurred in this group of patients if higher degrees of AV block could have been produced without a vagotonic influence.

Previous studies have shown the role of atrial systole in producing variations in the intensity of the first heart sound during complete heart block.15,16 In those beats in which atrial contraction caused complete early closure of the mitral valve, ventricular systole was associated with either an absent or attenuated first heart sound. In beats with a PR interval < 0.20 second there was an inverse relationship between the PR interval and the amplitude of S1.

In 1950 Levine and Harvey18 noted that the intensity of the first heart sound in atrial flutter fluctuated,
and proposed that the variation of the sound was dependent on the timing of the preceding atrial contraction and independent of the length of the preceding RR interval. These astute clinical observations are now well accepted. The findings in the present study provide physiologic confirmation that the variation in the intensity of the first heart sound in atrial flutter is inversely related to the degree of early mitral valve closure and is independent of the length of the preceding diastole. Figure 4 shows a loud S₁ occurring after a long RR interval in which the mitral valve was open at the time of ventricular systole (beat 1), and a soft S₁ in shorter cycles where mitral pre-closure had occurred (beats 2-4). With further shortening of the RR interval as in beat 5, the mitral valve was again open at the onset of ventricular systole, thus accounting for the loud first heart sound.

Hamby et al.⁹ described the motion of the Starr-Edwards mitral prosthesis, and demonstrated that either atrial or ventricular contraction alone could cause mitral valve closure. In most patients with a mitral prosthesis, mitral closure and the mitral closure click occur well after the onset of the QRS complex. Hamby found an average QRS-to-closure click interval (Qcc) of 0.086 second in patients with atrial fibrillation and 0.063 second in patients with sinus rhythm. These findings are comparable to the Qcc of 0.073 second observed by Hultgren and Hubis¹⁰ in patients with normally functioning Starr-Edwards mitral valve prostheses. In two patients in Hamby’s series, as well as in patients reported by others,²⁰ ²¹ sinus rhythm with marked first-degree AV block produced mitral valve closure before the onset of ventricular systole. This premature mitral valve closure secondary to prolongation of the PR interval was also noted in patient AM (fig. 7).

Our observations of ball-valve prosthesis motion during atrial fibrillation (fig. 6) are similar to those described by others with the early onset of closure of the valve poppet which is possibly secondary to the effect of gravity on the ball.²² ²³ Although there is an inverse relationship between the length of the preceding RR cycle and the interval from QRS complex to mitral closure in atrial fibrillation, the mitral closure click actually precedes the onset of the QRS only with long RR intervals > 1 second.²⁴ In contrast, complete early closure of a mitral prosthetic valve may consistently occur during atrial flutter with shorter RR intervals of 0.84 second. A late diastolic flutter wave can reopen and close the valve prosthesis, producing an additional mitral closure click.

In three patients with mitral Starr-Edwards prostheses, Agnew and Carlisle¹¹ described premature closure which they attributed to aortic regurgitation. However, the three patients had atrial fibrillation during the study, which raises questions about the assumed association of mitral pre-closure with aortic insufficiency. Sands and co-workers¹⁰ described early closure of a Beall mitral valve prosthesis in a patient in sinus rhythm who had aortic and mitral valve replacements and a paravalvular aortic leak. Similarly, the finding of early closure of the mitral prosthesis and an early diastolic murmur suggested the possibility of an aortic paravalvular leak in patient AM; however, no leak was found, and early closure of the mitral prosthesis in this patient can be attributed to the effects of the arrhythmia.

Closure of the normal mitral valve is initiated by atrial systole and completed by the onset of left ventricular isovolumic contraction. Disturbances of AV conduction or atrial rhythm which distort the normal sequence of atrial and ventricular systole predispose to abnormal early closure of the mitral valve. Atrial flutter may cause premature closure of both the normal as well as the prosthetic mitral valve, and produce variation in the intensity of the first heart sound.

References
Hemodynamic Factors Influencing Arterial Hypoxemia in Massive Pulmonary Embolism with Circulatory Failure

FRANÇOIS JARDIN, M.D., FRANCIS GURDJIAN, M.D., PIERRE DESFONDS, M.D., JEAN-LUC FOUILLADIEU, M.D., AND ANDRÉ MARGAIRAZ, M.D.

SUMMARY Arterial hypoxemia is a common finding in acute pulmonary embolism, and its severity is generally assumed to be proportional to the extent of pulmonary artery obstruction. We studied blood gases (during room air breathing and 100% oxygen breathing) and hemodynamic data in seven patients with massive pulmonary embolism and circulatory failure. All measurements were made before and 30 minutes after medical therapy of shock. We observed that a low cardiac output state can result in a misleading improvement in arterial oxygenation during massive pulmonary embolism, and that an improved circulatory status resulting from medical therapy (including inotropic drug infusion with or without blood volume expansion) can paradoxically increase arterial hypoxemia.

We conclude that severity of arterial hypoxemia may not reflect the severity of pulmonary artery obstruction in acute pulmonary embolism if shock is present.

ARTERIAL HYPOXEMIA is a common finding in acute pulmonary embolism1–4 resulting from mismatching of ventilation and pulmonary blood flow. Many authors consider arterial hypoxemia an important diagnostic feature,5,6 and its severity is generally assumed to be proportional to the extent of reduction in pulmonary vascular bed.7

Because some researchers have found cases of massive pulmonary embolism without arterial hypoxemia,8 we studied patients suffering from massive pulmonary embolism to examine the effect of a low cardiac output on arterial oxygenation.

Patients and Methods

We studied seven patients (mean age 54 years) suffering from acute massive pulmonary embolism between October 1973 and October 1977. Pulmonary embolism was clinically diagnosed in all cases and was documented by pulmonary angiography (five cases) or autopsy (two cases); in each patient pulmonary embolism induced severe circulatory failure with metabolic acidosis. All patients were intubated and received artificial ventilation with intermittent positive pressure breathing (tidal volume 7 ml/kg, 20 breaths/min) and circulatory failure was managed with inotropic drugs in all patients (dopamine infusion 15 µg/kg/min in six cases and isoproterenol infusion 4 µg/min in one case) and vascular filling (with plasma expanders 20 ml/kg) in three patients. We made hemodynamic measurements just before management of circulatory failure, and after 30 minutes of medical therapy. All patients received urokinase therapy, but only after the second hemodynamic evaluation. Four patients survived (two with medical therapy only, one after emergency embolectomy and one after long-term extracorporeal membrane oxygenation7 with venoarterial bypass); three patients died despite emergency therapy.

Catheters were inserted to measure the radial artery, right atrial and pulmonary artery pressures; vascular pressures were measured with Statham P23Db transducers positioned at the midaxillary line, and atmospheric pressure was used as a zero reference point. Cardiac output was measured by thermodilution (right atrial injection with temperature recording in the pulmonary artery). Simultaneous sampling of
Mitral valve closure in atrial flutter.
M A Greenberg, L S Herman and M V Cohen

Circulation. 1979;59:902-909
doi: 10.1161/01.CIR.59.5.902

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1979 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/59/5/902