The Mechanism of Closure of the Mitral Valve: A Continuing Controversy

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MITRAL VALVE CLOSURE has intrigued investigators since William Harvey compared the structure to the valves of a mechanical force pump.1 Chauveau and Fivre2 extended this analogy in 1856 by postulating that mitral closure was produced by the systolic increase in ventricular pressure. This classic "ventriculogenic" explanation of closure was challenged, and by the beginning of this century several other mechanisms had been proposed. Despite advances in investigative techniques, these same explanations are still widely advocated, and a unanimous decision on the mechanism of mitral valve closure is not available.3 In this brief review I highlight the state of uncertainty and suggest the need for further study.

Atrigenic vs Venticulogenic Closure

Baumgarten4 in 1843 and later Henderson and Johnson5, 6 demonstrated in isolated perfused animal hearts that abruptly ending forward flow through an open atrioventricular (AV) valve, as might occur at the end of atrial contraction, produced a zone of negative pressure in the area of the valve cusps. Influx of fluid into this zone from the sides of the ventricle caused the valve leaflets to roll closed before the onset of ventricular contraction. They suggested that this type of closure, later included under the general title "atriogenic," was accompanied by minimal or no regurgitation. In contrast, if closure resulted from ventricular action without presystolic positioning, the valve cusps would swing shut like "barn doors in a windstorm" and closure would be accompanied with considerable regurgitation.7 While the concept of a "breaking jet" at the end of atrial systole has been abandoned as not being consistent with the peristaltic properties of atrial contraction,8 the association of non-regurgitant closure with an atrigenic mechanism and a large backflow with ventriculogenic closure has, until recently, remained unverified experimentally.

A second atrigenic mechanism for closure of the AV valves was added in 1889 when Krehel9 postulated that eddy currents are set up behind the open leaflets during atrial ejection and that these swing the valve closed at the end of atrial systole. Recent mathematical models of mitral valve function10 support this concept and suggest that an asymmetric ring vortex forms in the ventricle during diastole. This current has been implicated in the early diastolic movement of the mitral valve toward closure in individuals with reduced venous return and a slow heart rate. When combined with the deceleration of flow at the end of atrial systole, it apparently may also be sufficient to again move the valve cusps toward closure.

Direct study of the movements of the AV valves in the beating heart was undertaken in 1916 when Dean11 attached a hair to a mitral valve cusp of an exposed dog heart and recorded its movement by optical means. Unfortunately, the dynamic effect of atrial systole was significantly reduced by the connection of a large open reservoir to the left atrium.12 Nevertheless, Dean showed that the valve cusps move toward closure near the end of atrial systole, and that reopening before the onset of ventricular contraction did not occur if the interval between atrial and ventricular systole (AV valve interval) was short. However, the valve cusps began to reopen before being finally closed by ventricular contraction if the AV valve interval was greater than 0.147 second.

In 1951, Little13 confirmed Porter's earlier suggestion13 that the normal AV pressure gradient may reverse after atrial systole. He also showed that this presystolic reversal was sufficient, at least in the presence of first-degree AV block, to close the tricuspid valve of an anesthetized dog before the onset of ventricular contraction. Similar atrigenic closure of the mitral valve was subsequently reported in animal studies by Siecke and Essex,14 Sarnoff et al.,15

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and Brockman, while Grant et al. and Zaky et al. demonstrated atriogenic closure in human subjects. The classic ventriculogenic view of mitral closure was extended to include contraction of intraventricular, annular and papillary muscles at the onset of ventricular systole by Lian and Erlanger and more recently by Sonnenblick et al., Cooper et al., Priola et al. and Tsakiris et al. While the presence of muscle fibers in the AV valve leaflets had been known since 1840, their contractile activity was not observed until early in this century. In recent years these muscles have been shown to exhibit length-tension and force-velocity relationships and electrical activity similar to ventricular muscle, although they appear to have pharmacological responses that are more typical of atrial muscle. The intraventricular muscle fibers are activated just before or simultaneously with ventricular depolarization, and contraction causes the atrial surface of the leaflets to become concave. This change in shape may assist in leaflet apposition at the time of ventricular contraction, and development of tension within the leaflets themselves may serve to oppose bulging of the valve cusps into the atrium during ventricular systole.

The role of the annular muscle fibers in mitral closure has recently been studied. Perloff and Roberts point out that the mitral ring resists dilatation, but is sufficiently pliable to permit sphenicter-like contraction of the annulus during systole. Tsakiris et al. demonstrated by radiographic techniques in dogs that such eccentric narrowing of the mitral valve ring occurred during both atrial and ventricular contraction. This finding indicated that the significant reduction in the area of the annular orifice at the onset of ventricular systole is responsible for the final leakproof closure of the valve.

The role of the papillary muscles in AV valve closure remains somewhat speculative. Rushmer et al. suggested that papillary muscle contraction would pull the valve leaflets together because of their chordal attachment to each cusp. However, Karas and Elkins presented radiographic evidence that the left ventricular papillary muscle does not shorten during contraction, but only maintains tension on the chordae tendinae. This is consistent with the observation that papillary muscle dysfunction leads to mitral valve insufficiency as a result of erosion of the leaflets into the atrium during ventricular systole and not from improper closure. Moreover, Tsakiris et al. demonstrated that surgical damage to the papillary muscles of the anesthetized dog does not itself affect AV valve closure.

Mitral Closure and Regurgitant Flow

Fundamental to the study of AV valve closure has been the concept that atriogenic closure is accomplished with minimal or no backflow. Data are now available on this point. Using indicator dilution techniques, Williams et al. and Rutishauser et al. have shown that mitral closure produced by isolated atrial contractions is accompanied by considerable regurgitation. An atrial contraction followed by a properly placed ventricular systole has, however, been shown in both animal studies and in man to result in efficient closure of the mitral valve with minimal or no regurgitation. An exception is the recent animal study reported by Laniado in which closing regurgitant volumes equal to 14% of the total mitral flow were reported with normal sinus rhythms. In these studies, regurgitant flow increased moderately with atrial fibrillation, provided the ventricular rate remained less than 140 beats/min. However, at higher ventricular rates or when ventricular function was increased by extrasystolic potentiation or through the use of pharmacological agents, efficient valve closure occurred without detectable regurgitation.

The suggestion that ventriculogenic closure of the AV valves is accompanied by considerable backflow is supported by the observation in both animals and in man that ectopic ventricular contractions frequently produce atrial pressure pulse contours typical of AV valve insufficiency. In addition, regurgitation has been demonstrated by injecting a variety of markers into the ventricle and detecting their presence in the atrium during ectopic ventricular beats without a properly timed atrial contraction or during atrial fibrillation. A number of other studies have, however, reported that ventriculogenic closure is effective and does not lead to significant regurgitation. For example, failed to demonstrate meaningful reflux during angiocardiographic studies in a large series of patients with absence or inappropriate timing of atrial contraction. Vanderberg et al. reported that ectopic ventricular contractions were sometimes associated with minor regurgitation, but only when they occurred in midcycle. However, when such extra beats occurred early in diastole, the more forceful ventricular contraction, produced as a result of extrasystolic potentiation, prevented significant reflux during mitral closure.

Mitral Closure and Production of S1

The timing and intensity of the first heart sound (S1) can reveal information regarding closure of the AV valves. For example, in a series of normal subjects, Little et al. showed the Q-S1 interval is significantly increased during ectopic ventricular contractions compared with normal beats. This suggested that in the absence of atrioventricular presystolic valve movement, ventriculogenic closure started from an open position and required a longer time for the leaflets to complete their movements. In other studies, the intensity of S1 has been shown to have a bimodal relationship to the P-R interval. Zaky et al. and Burggof and Craig report that the loud S1 with P-R intervals less than 200 msec is associated with a large amplitude excursion of the valve cusps as shown by echocardiography. This suggested that insufficient time was available after atrial contraction for prepositioning of the valve cusps and full closure occurred as a result of
ventricular action. The soft or absent S₁ with P-R intervals of 200–500 msec was associated with atriogenic pre-systolic closure, while very long P-R intervals permitted the valve to reopen before final ventriculogenic closure and production of a loud S₁.

Recent Studies of Valve Closure

Movement of the mitral leaflets have been extensively studied during the last few years by cineangiocardiography in animals with implanted radiopaque markers on the valve edge and in animals and man by M-mode echocardiographic techniques. These procedures have, however, provided only a restricted view of valve closure due to the non-uniform movement of the valve leaflets and either the distance between markers or the narrow diameter of the sonic beam. Therefore, the final leaflet coaptation probably cannot be adequately categorized from such fragmentary information. In addition, valve closure cannot be predicted from intracardiac pressure measurements, as recent studies have shown that closure may occur as late as 30 msec after AV pressure crossover. In spite of these difficulties recent studies of mitral valve movement in normal hearts have confirmed Dean's observation that the valve opens widely during atrial systole and begins to close before the onset of ventricular contraction. The timing and mechanism of final closure is controversial. Impressive recent evidence supports each of the following conflicting views of mitral closure: 1) closure is completed after the onset of ventricular contraction as a result of ventricular action; 2) coaptation occurs simultaneously with ventricular systole; or 3) closure occurs before the onset of ventricular contraction as a consequence of atrial systole, particularly if the A₃-V₃ interval is 200–500 msec. Thus the mechanism and timing of final closure remain enigmatic. Some of these conflicting observations may be more apparent than real. The mechanism of mitral closure may, for example, vary from time to time in animals and man, depending on the condition of the myocardium, level of autonomic nervous system input, size of the heart or other variables. Perhaps new techniques, such as short-axis cross-sectional echocardiographic scanning with slow motion visualization of mitral valve motion, along with a systematic study of the effect of cardiac function on valve closure, will resolve this matter.

Summary and Conclusions

Conflicting information regarding the mechanism of closure of the mitral valve makes a simple explanation for this dynamic event unlikely. Normal closure of the mitral valve probably results from a combination of atriogenic and ventriculogenic events. However, the relations and importance of each factor are still not known. The complexity of these interactions requires that this facet of cardiac physiology should be studied further.

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