A Clinical Evaluation of the Hypothesis that Rupture of the Left Ventricle Following Mitral Valve Replacement Can Be Prevented by Preservation of the Chordae of the Mural Leaflet

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Experiences with 14 patients undergoing rupture of the left ventricle following mitral valve replacement over a period of 9 years have been described. Three different types have been recognized. Before 1978, most injuries occurred in the atrioventricular groove, apparently resulting from traction that insidiously avulsed the mitral annulus from the underlying left ventricular muscle. Several changes in operative technique, described in the text, were made to prevent this traction avulsion. Following the adoption of these principles, rupture in the atrioventricular groove virtually disappeared. A second type of injury, strut perforation, has been recognized in only one patient, a small 81-year-old female in whom the prosthesis inserted was too large for the ventricular cavity. Translucent obturators were subsequently developed not only to size the left ventricle but also to note the location of the post of the porcine prosthesis before insertion. Further problems of this type have not been seen. The most puzzling, and currently the most significant, problem is a third type of rupture, the mid-ventricular rupture, suggested as Type III by Miller[8] in 1978 and described in detail by Cobbs in 1977[5] and 1980.[6] The phenomenon seems to be a true *spontaneous rupture* of a thin left ventricle, usually occurring in small elderly women with mitral valve disease. If the friability of the left ventricle is transiently increased with potassium cardioplegia, such ventricles may spontaneously rupture following division of the chordae to the annulus of the mural leaflet. If this concept is correct, a rupture in some patients can best be prevented by preserving these chordae. It is well realized, of course, that a fortunate narrative experience of 3½ years does not have any statistical value concerning a complication that occurs in 1 to 2% of operations. The experiences are reported, however, because to our knowledge, the untethered loop hypothesis has not been previously evaluated in a large number of consecutive patients operated on. Future comparison of experiences reported by others should make it possible to determine whether or not this concept is correct.

RUPTURE of the left ventricle following mitral valve replacement is an infrequent but highly lethal complication, occurring in 0.5–2% of patients operated on.[1-3] The complication was first reported in 1967 by Roberts and Morrow,[4] who described the autopsy findings in two patients. One rupture apparently resulted from excessive excision of the base of a papillary muscle, while the other developed in the atrioventricular groove following excessive removal of a calcified annulus. Since 1967, less than 100 patients have been described in different publications, but it is almost certain that many other cases have never been reported.

Currently, there is a wide divergence of opinion about the cause of left ventricular rupture. This was clearly evident during a panel discussion chaired by the senior author at the annual meeting of the American Association for Thoracic Surgery in 1984. Some members of the panel, nationally recognized cardiac surgeons, stated that rupture of the left ventricle was simply due to excessive surgical trauma and, hence could be prevented with proper technique. Other members of the panel disagreed completely.

A traumatic injury was considered the most likely cause at New York University until the fall of 1981. Then, within a period of 2 months, four fatal ruptures occurred following operations performed by three different surgeons. This appalling experience prompted a thorough review of all previous reports, as well as detailed personal telephone conversations with colleagues among different major surgical clinics. The information obtained led to a new concept of the cause of left ventricular rupture and a basic change in operative approach.

The "untethered loop" hypothesis proposed by Cobbs in 1980,[5,6] based on autopsy findings in seven cases, seemed to be the most reasonable explanation. This con-

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TABLE 1. *Fourteen Patients with Rupture of Left Ventricle Following Mitral Valve Replacement: Clinical Data*

Case Number	Date	Age	Weight (kg)	Sex	Predominant Lesion
1	1976	52		F	MS (Calcified annulus), TR
2	1977	69	52	F	MR
3	1978	69		M	MR, AS
4	1979	71		F	MS
5	1979	81	52	F	MS
6	1979	60	60	F	MS
7	1980	75	47	F	MS (Calcified annulus), AS
8	1981	70	51	F	MS, AS
9	1981	67	55	F	MS (Calcified annulus)
10	1981	66	55	F	MS, AR
11	1981	65	60	F	MS
12	1982	65	64	F	MS
13	1984	71	50	F	MS
14	1984	73	43	F	MR (Calcified annulus)

MS = mitral stenosis; MR = mitral regurgitation; TR = tricuspid regurgitation; AS = aortic stenosis; AR = aortic regurgitation.

cept considers the supporting structures of the posterior ventricular wall to form a loop. The outer portion is composed of longitudinal muscle fibers in the ventricular wall, while the inner portion of the loop consists of the papillary muscles with the chordae attached to the annulus of the mural leaflet. Accordingly, division of mural leaflet chordae could seriously weaken the posterior ventricular wall. A similar concept was proposed by Lillehei[7] in 1964 to explain a decrease in left ventricular function following mitral valve replacement. He suggested that excision of the chordae of the mural leaflet could impair ventricular function and described a method of preservation of these chordae. Studies by others, however, could not confirm this hypothesis that excision of chordae was a significant cause of cardiac failure.

In different publications, others (Miller, 1979;[8] Cabrol, 1984[9]) have similarly expressed concern about the safety of routinely dividing the mural leaflet chordae.

To investigate the "untethered loop" hypothesis, the basic technique of mitral valve replacement was changed to preserve routinely as many mural leaflet chordae as possible, as well as portions of the base of the mural leaflet. In some patients, one or more large chordae that were divided were subsequently reattached to the annulus.

Since adopting this new technique, no further ruptures have occurred among 365 patients undergoing mitral valve replacement. Three ruptures have occurred, however, in a small group of patients operated on by two members of the faculty in whom the old technique of total excision of the mural leaflet and supporting chordae was employed.

In this report, our total experiences with 14 cases of mitral valve rupture occurring over a period of 9 years (1976–1985) are reviewed. During the first 5½ years of this experience, the diseased mitral valve was totally excised. In the last 3½ years, some mural leaflets chordae were preserved.

Methods

A review of experiences with mitral valve replacement for a period of 9 years (1976–1984) found 14 patients in whom the left ventricle ruptured following mitral valve replacement. Two of thse 14 patients survived.

The pertinent clinical data are tabulated in Tables 1–3. In Table 1, the significant clinical data are listed. Among the 14 patiens, 13 were female, one was male. The average age was 68 years, with an age range from 52 to 81. Twelve of the 14 patients were over 64 years of age. The average weight was only 54 kg (data available from only 11 patients). All patients had a rheumatic history. Mitral stenosis was the dominant lesion in 11, regurgitation in three. Severe calcification of the mitral annulus was described in only four of the 14.

Significant data concerning the valve replacement are tabulated in Table 2. Somewhat difficult technical problems were present in several patients. Extensive calcification was present in four; a concomitant aortic valve replacement was done in four; and three patients had had previous cardiac operations (mitral commissurotomy). Eleven porcine valves (2 Hancock, Hancock Laboratories, Anaheim, CA; 9 Carpentier, American Edwards Laboratories, Santa Ana, CA) and three Bjork-Shiley disc prostheses (Shiley, Inc., Irvine, CA), size ranging from 25 to 29, were inserted. In 13 patients, both mitral leaflets were excised, including the papillary muscles and all chordae. In one of the 14, the posterior leaflet was left intact but the major papillary support to this leaflet was excised.

In 11 patients, potassium cardioplegia was employed with an average cross-clamp time of 83 minutes; in three, intermittent ventricular fibrillation and cross-clamping was used. Eight of the 14 had a left ventricular apical vent inserted.

Pertinent data concerning the rupture of the left ventricle are listed in Table 3. In nine of the 14 patients, rupture occurred in the operating room after the heart began to beat, while five ruptured in the recovery room (30 minutes, 3 hours, 4 hours, 5 hours, and 48 hours after surgery). In two patients in the recovery room, significant transient hypertension occurred 15 to 30 minutes before rupture.

In four patients, the rupture was clearly in the atrioventricular groove, while in nine the rupture was located in the ventricular wall between the base of the papillary muscles and the atrioventricular groove. The exact site was uncertain in the one patient who survived following external repair.

In eight patients, the rupture was treated with a complex repair, in which the previous valve was removed, pros-

TABLE 2. *Fourteen Patients with Rupture of Left Ventricle Following Mitral Valve Replacement: Operative Data*

Case Number	Date	Operation	Valve Type and Size	Myocardial Protection Technique	Miscellaneous
1	1976	Complete valve excision, MVR, TA	#25 Bjork	Intermittent cross-clamp and fibrillation	Calcified annulus required debridement; re-op s/p mitral commissurotomy
2	1977	Complete valve excision, MVR	#29 porcine	Intermittent cross-clamp and fibrillation	Left ventricular vent
3	1978	Complete valve excision, MVR, AVR	#29 porcine MV #25 porcine AV	Intermittent cross-clamp and fibrillation	Left ventricular vent; difficult exposure required opening septum
4	1979	Complete valve excision, MVR	#29 porcine	Cardioplegia, cross-clamp—82 minutes	Re-op s/p mitral commissurotomy; left ventricular vent
5	1979	Complete valve excision, MVR	#29 porcine	Cardioplegia, cross-clamp—90 minutes	Left ventricular vent; bleeding left ventricular vent site requiring lifting to repair heart
6	1979	Complete valve excision, MVR	#29 porcine	Cardioplegia, cross-clamp—60 minutes	Left ventricular vent
7	1980	Complete valve excision, MVR, AVR	#25 Bjork MV #19 Bjork AV	Cardioplegia, cross-clamp—210 minutes	Left ventricular vent; extensively calcified annulus debrided
8	1981	Complete valve excision, MVR, AVR	#27 porcine MV #23 porcine AV	Cardioplegia, cross-clamp—140 minutes	
9	1981	Complete valve excision, MVR	#29 porcine	Cardioplegia, cross-clamp—41 minutes	Calcified annulus debrided
10	1981	Complete valve excision, MVR, AVR	#29 porcine MV #29 porcine AV	Cardioplegia, cross-clamp—82 minutes	Left ventricular vent
11	1981	Complete valve excision, MVR	#29 porcine	Cardioplegia, cross-clamp—70 minutes	
12	1982	Complete valve excision, MVR	#29 porcine	Cardioplegia; cross-clamp—58 minutes	Left ventricular vent
13	1984	Complete valve excision, MVR	#25 Bjork	Cross-clamp—72 minutes	Re-op s/p mitral commissurotomy; very small annulus required debridement
14	1984	Posterior leaflet left intact, but major papillary muscle cut	#27 porcine	Cross-clamp—73 minutes	Calcified annulus; no debridement

MVR = mitral valve replacement; TA = tricuspid annuloplasty; AVR = aortic valve replacement; MV = midventricular; AV = atrioventricular; s/p = status post.

thetic patches both inside and outside the ventricle inserted, and then a new valve inserted. Only one patient survived. In six of the 14 patients, an external repair was done with a prosthetic patch and pledgeted sutures, leaving the previous valve in place. One patient survived.

Eleven patients died in the operating room, while one died following a series of complications 2 weeks later. Two patients recovered and were well when last seen over 2 and 5 years later. One of these had a rupture repaired in the operating room with an external Dacron® patch, while the other had an atrioventricular groove tear repaired by having the valve removed and the tear repaired from both within and outside the heart.

Discussion

Significant Features in Previous Reports

The hazard of excessive removal of calcium in the atrioventricular groove was first described by Roberts and

TABLE 3. *Fourteen Patients with Rupture of Left Ventricle Following Mitral Valve Replacement: Characteristics of Rupture and Repair*

Case Number	Date	Clinical Factors prior to Rupture	Time of Rupture	Site of Rupture	Repair	Result
1	1976	Low output required IABP	OR	AV groove	Fibrillation, external patch, and pledget	Died; OR; low output
2	1977	Hematoma noted after vent removed	OR	Large hematoma at posterior strut site and AV groove without free rupture	Fibrillation, external patch	Recovered
3	1978	None	OR	AV groove	Fibrillation, external patch, and pledgets	Died; OR; Hemorrhage, Low output
4	1979	None	OR	Midventricular	Fibrillation, external patch, and pledgets	To RR; died in hospital
5	1979	Bleeding from left ventricular vent site	OR	Midventricular (strut perforation)	Cardioplegia, new valve, internal pledgets, external patch	Died; OR; hemorrhage
6	1979	Hypertension in RR	RR 5 hours postop	Midventricular	Cardioplegia, new valve, internal and external pledgets	Died; OR; hemorrhage
7	1980	None	OR	Midventricular	Cardioplegia, new valve, internal and external pledgets, external patch	Died; OR; hemorrhage
8	1981	None	OR	AV groove	Cardioplegia, new valve, internal pledgets	Died; OR; hemorrhage
9	1981	None	RR: 30 minutes postop	Midventricular	Cardioplegia, new valve, internal and external pledgets	Died; OR; hemorrhage and low output
10	1981	None	OR	Midventricular	Cardioplegia, new valve, internal pledgets	Died; OR; low output
11	1981	None	OR	Midventricular	Cardioplegia, new valve, internal and external pledgets	Died; OR; hemorrhage
12	1982	None	RR; 4 hours postop	AV groove	Cardioplegia, new valve, internal pledgets	Recovered
13	1984	Hypertension in RR	RR; 3 hours postop	Midventricular	External pledgets	Died RR
14	1984	None	RR; 48 hours	Midventricular	External pledgets	Died RR

OR = operating room; RR = recovery room; AV = atrioventricular.

Morrow[4] in their classic report in 1967. They clearly emphasized that left atrial muscle is not normally connected to left ventricular muscle. The mitral annulus is continuous with the endocardium of the left atrium. Accordingly, excessive removal of a calcified annulus can easily create a perforation in the atrioventricular groove.

Four years later (1971), McVaugh and associates[2] described six patients seen over a period of 8 years who developed a rupture in the atrioventricular groove; three of these recovered. In 1974, Zacharias[1] at the Cleveland Clinic found six patients with left ventricular rupture occurring among 1000 mitral valve replacements performed over a period of 13 years. All were apparently in the atrioventricular groove. In the same year, Treasure[10] described seven cases and classified these into two groups, based on the site of rupture. In one group, the tear was in the atrioventricular groove (Type I), while in the other it was located inferiorly in the ventricular wall (Type II). Like Roberts,[4] he thought that the ventricular wall ruptures resulted from excessive excision of a papillary muscle.

Subsequently, Katske,[11] in 1978, reported three additional patients from the Cleveland Clinic. Interestingly enough, their three ruptures were in the midportion of the ventricular wall, a location different from that of the previous five atrioventricular groove ruptures reported by Zacharias.[1]

In 1977, Bjork[3] reported experiences with eight patients, five of whom recovered, and found 18 other patients previously reported by others.[1,2,9] In 1978, Miller[8] reported two patients who died from cardiac failure following operation, one the day of operation, the other 2 weeks later. Neither had excessive hemorrhage, but in both 4–5 cm transverse incomplete midventricular ruptures *between* the unexcised papillary muscles and the mitral valve annulus were found at autopsy. He speculated that preserving the chordae of the mural leaflet could be an important consideration in preventing cardiac failure, supporting the original suggestion by Lillehei in 1964.[7]

Miller[8] referred to a 1977 report by Cobbs[5] of six cases of transverse ventricular rupture and suggested that the Type I and Type II classifications suggested by Treasure[10] should be supplemented with an additional type (Type III). These included the original atrioventricular groove rupture, the ventricular rupture from excision of a papillary muscle or strut perforation, and ventricular rupture in an intermediate zone, located between the base of the papillary muscle and the atrioventricular groove. Figure 1 in this report is reproduced from Figure 5 in the 1978 publication by Miller,[8] which clearly illustrated diagrammatically these three different types of rupture.

Subsequently, in 1980, Bjork[12] stated that their total experience now included 11 patients with ventricular rupture following 456 operations. He considered trauma the most common cause, emphasizing avoiding deep insertion of sutures that penetrated ventricular muscle rather than the fibrous annulus, and also carefully avoided lifting the heart following replacement.

In 1981, Celemin[13] in Madrid, Spain, reported seven patients with rupture occurring during a period of 966 valve replacements. The first five died of hemorrhage after unsuccessful attempts to repair the injury by external methods. Hemorrhage was controlled in the last two patients by removing the prosthesis and repairing the injury with an intraventricular patch. One patient died from renal failure, the other recovered.

In 1984, Dark and Bain in Glasgow, Scotland,[14] described experiences with 18 patients seen over a period of 6 years, during which over 1200 mitral replacements were performed, a frequency near 1.5%. Two survived. Five ruptured near the annulus, 11 in subannular areas. The authors considered the "untethered loop" hypothesis the most plausible explanation but curiously enough, did not suggest any modifications of operative technique to prevent the problem. The work of Cobbs (discussed in the next paragraph) was cited, but the original suggestion of Lillehei of preserving the papillary muscles was not.

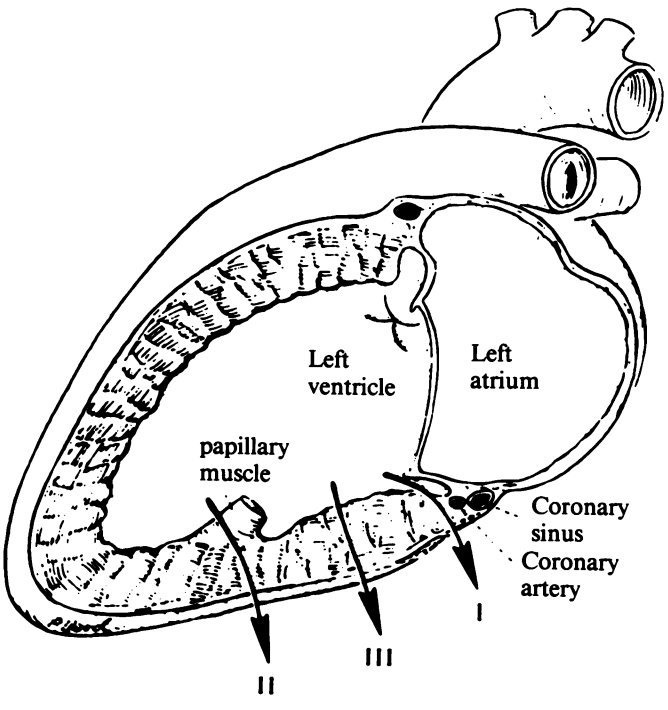

FIG. 1. Diagrammatic illustration of three sites of left ventricular rupture that may occur when the mitral valve apparatus is completely excised for mitral valve replacement. Reprinted by permission from the 1978 publication by Miller DW Jr, Johnson DD, Ivey TD. Does preservation of the posterior chordae tendineae enhance survival during mitral valve replacement? Ann Thorac Surg 1979; 28:22–27.

Perhaps the most significant reports were published by Cobbs in 1977[5] and 1980[6] from Emory University, describing in detail the pathologic findings among seven patients who died following operation. He suggested that *spontaneous* rupture of the left ventricle could occur in some patients following excision of the mural leaflet and the papillary muscles. He cited the 1970 physiological publication of Armour and Randall[15] as follows, "The papillary muscle, chordae, and leaflets form the inner arm of a longitudinally coursing loop connected at both ends with the mitral annulus. The outer arm of this loop consists of longitudinal muscle fibers and the wall of the left ventricle fixed to the mitral annulus superiorly. . . . [T]hus detaching papillary muscles from the annulus may be transecting a major vertical fiber loop." This concept is similar to the report in 1964 by Lillehei,[7] who described physiological principles attributed to Rushmer.[16,17]

Cobbs[6] suggested that rupture of the left ventricle was a "stretch" injury resulting from the confluence of multiple causes, including a small left ventricle in an elderly patient, excessive relaxation of the heart following potassium cardioplegia, and excision of the posterior papillary

muscle and chordae, creating an "untethered loop." Shortly following Cobbs' report, Ross and Streeter,[18] in a short letter to the editor, fully supported Cobbs' hypothesis, stating that this was on a sound physiological basis, consistent with a physiological theorem termed the "Clairaut Theorem."[19]

The uncertainties concerning the causes of ventricular rupture, however, are well indicated in the short statement on the last page of Cobbs'[6] report in which, referring to the concept of preserving the chordae of the mural leaflet, he stated, "our surgeons have not used this approach, suspecting that it might provide a focus for embolism."

During the 17 years since Roberts' original report[4] in 1967, several reports between 1975 and 1983 have described one to four patients. These include reports by Chi,[20] Wolpowitz,[21] Engleman,[22] Phillips,[23] Nunez,[24] Gosalbez,[25] Bortolotti,[26] and Devineni.[27] These are mostly repetitious descriptions of different types of traumatic injury, describing ventricular rupture following excessive removal of calcium, insertion of an unduly large prosthesis, strut rupture following cardiac massage, and other forms of injury.

The common theme in all of these reports, however, is that the rupture is primarily due to trauma and hence preventable with an appropriate technique. The "untethered loop" hypothesis of Cobbs,[6] however, suggests that rupture of the left ventricle may occur in a few patients following division of mural leaflet chordae regardless of the technique employed.

In 1984, Cabrol,[9] in a discussion of a paper presented by Tirone concerning the influence of mural leaflet chordae on left ventricular function, stated that he had followed the suggestion of Lillehei of routinely preserving mural leaflet chordae for nearly 20 years. In a series of approximately 2100 mitral valve replacements, he had not seen rupture of the left ventricle except when these chordae had been divided.

In a personal discussion with Carpentier in 1983 concerning left ventricular rupture, he stated that he had personally never encountered the problem during his extensive experience with thousands of mitral valve operations but always took particular care to preserve the basal chordae in the wall of the left ventricle, present in the majority of patients, feeling that these contributed significantly to the structural integrity of the ventricular wall.

New York University Experiences

Experiences at New York University over the past 9 years can be grouped into three periods. Before 1978, most experiences suggested that traumatic injuries in the atrioventricular groove were the most common cause. In no instance, however, was excessive removal of calcium recognized as the basic cause. The mechanism seemed to be a traction injury, resulting from a variety of mechanisms that insidiously avulsed the mitral annulus from the underlying left ventricular muscle. This injury only became evident after the heart began to beat and a hematoma formed and progressively enlarged in the atrioventricular groove.

To prevent this avulsion injury, particular attention was given to several different maneuvers during replacement of the mitral valve. These included the avoidance of undue traction on the valve leaflets during removal, careful insertion of sutures into the mitral annulus, but avoidance of deeper sutures that penetrated left ventricular muscle beneath the annulus. The prosthetic valve was gently inserted, tying sutures initially along the annulus of the mural leaflet. The sutures were tied with particular care, avoiding any upward traction on the mitral annulus during the tying of consecutive knots. A left ventricular vent was avoided. Lifting of the apex of the heart was also scrupulously avoided once the prosthetic valve had been inserted. Insertion of a prosthetic valve places a rigid prosthetic ring in the artrioventricular groove. The rigid ring constitutes a "hinge." Lifting of the apex of the heart following insertion of a prosthetic valve can readily result in tearing the left ventricle away from the overlying prosthetic ring.

After formulation of these guidelines in 1977–1980, traumatic atrioventricular groove ruptures virtually disappeared. For almost 2 years, no problems occurred. Then, in 1979, one patient exsanguinated in the operating room from a strut perforation, an unusual circumstance in an 81-year-old patient with a large mitral annulus but a small left ventricle. The posteriorly located strut of the Carpentier prosthesis promptly perforated the ventricle when the heart began to beat. This tragic experience led to a redesign of the obturators used for sizing the mitral valve, with translucent obturators constructed so the position of the posterior post of the prosthetic valve, and its relation to the left ventricular wall, could be observed before the prosthesis was inserted. With these precautions, no subsequent strut injuries have occurred.

Following this second basic change in technique, all seemed well in 1980 and 1981 for about 18 months, after which the previously described disastrous period occurred in the fall of 1981 when four fatal mitral valve ruptures occurred within a period of 2 months. It seemed clear that some additional cause of injury must exist. After the first of the four ruptures occurred, an unusual degree of care was taken to minimize even the slightest trauma to the posterior ventricular wall during insertion. To our consternation, despite these precautions, within a week, another rupture occurred, a massive 5 cm transverse laceration in the midportion of the left ventricle, halfway

between the mitral annulus and the base of the papillary muscle. The patient was a small 65-year-old female. It was obvious that the fatal rupture was related neither to the size of the prosthesis, nor to undue trauma during insertion of the valve. The location, however, coincided precisely with the postmortem photographs published by Cobbs in this classic analysis of seven patients.[6]

As described earlier, a careful review of published reports, combined with several detailed telephone conversations with colleagues in different large clinics in the United States, led to the conclusion that the hypothesis proposed by Cobbs was the most plausible explanation. In brief, in certain patients with an underdeveloped or atrophied left ventricular muscle, particularly women over 60 years of age weighing less than 130 pounds, the small left ventricular wall is unusually prone to rupture. If the papillary muscles or chordae to the mural leaflet are divided, creating an "untethered loop," such a ventricle is particularly vulnerable to rupture.

In the 3½ years since adopting this hypothesis, during which 365 replacements have been performed, no further problems have occurred. The chordae of the mitral valve have been preserved to part or all of the mural leaflet in virtually all cases operated on; in some instances, one or two large chordae that were divided during removal of a calcified segment of mitral valve were reattached. The residual mural leaflet has actually facilitated insertion of porcine prostheses, providing additional substance for insertion of sutures through the annulus, much as described by Lillehei in 1964.[7] With a metallic prosthesis, used infrequently, care is taken, of course, to be certain that the residual leaflet does not interfere with the motion of the poppet of the prosthetic valve.

Three ruptures (two fatal) have occurred, however, in a small group of patients in whom the old technique of total excision of the mural leaflet and supporting chordae was employed.

Treatment of Left Ventricular Rupture

Only two of the 14 patients in this series survived, one treated by removal of the prosthesis and insertion of a Dacron® patch, the other by external application of a prosthetic patch. Isolated reports of different types of repair have been published by others.[25-27] It would seem reasonable, as emphasized by Celemin,[13] that extensive tears are best treated by removal of the prosthesis, followed by insertion of an internal prosthetic patch supported with sutures traversing the ventricular wall and tied over pledgets. Although small focal perforations have been repaired in a few patients by external methods alone, the external rupture may represent only the "tip of an iceberg," giving little clue to the extent of the underlying ventricular rupture. With midventricular ruptures, the tear has often been 4 to 5 cm in length.

With the current safety of potassium cardioplegia, rearresting the heart and removing the prosthesis would seem to be a reasonable approach, though significant data are not available.

In a recent personal conversation with Mr. Stuart Lennox, a senior consultant surgeon at the Brompton Hospital in London, England, successful repair of ruptures in two patients with an external technique was described. Both of these were treated with large mattress sutures placed between a large prosthetic patch over the ventricle and another one over the atrium. The sutures were passed beneath the coronary vessels in the atrioventricular groove, compressing the ventricle below between the atrium above.

This technique of external repair is similar to the suggestion by Celemin,[7] except that the heart was not reopened. Although the technique employed will clearly vary among different patients, the basic goal in repair should probably be to place sutures through the ventricle near the papillary muscles below, using prosthetic material, and the atrium above, somewhat reconstituting the support between the ventricle and the annulus that existed before the chordae were divided.

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