Mitral Regurgitation Following Myocardial Infarction: The Syndrome of Papillary Mitral Regurgitation*

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The normal function of the mitral valve depends on the anatomic and functional integrity of the entire valvular apparatus. Mitral regurgitation results when one of its components is anatomically deficient or functionally inadequate. Mitral regurgitation is usually secondary to rheumatic involvement of the mitral cusps and its chordae tendineae. Damage to the papillary muscles (without affecting the cusps) is usually the result of inadequate blood supply to these structures or to the neighboring myocardium. In such a circumstance, mitral regurgitation may appear. To this entity, the syndrome of papillary mitral regurgitation was applied to distinguish it from the common valvular variety.

In the present communication, a study of the clinical manifestation and course of this disease will be reported in detail.

Mitral Valve Closure and Function of the Papillary Muscles

The mechanism of closure of the mitral valve is the result of interaction of several structures of the mitral valve apparatus and the atrial and ventricular musculature. The precise order of closure is still in dispute, but there is a general agreement that this occurs in two main stages. The first consists of the apposition of the mitral cusps producing closure that is firm enough to prevent regurgitation into the left atrium at low ventricular pressures. This stage is believed to follow atrial systole, rapid ventricular inflow and the taunting of the cusps by the contraction of the papillary muscles. The mechanism responsible for the movement of the mitral cusps towards each other is believed to result from the breaking of a jet, the appearance of a Venturi effect at the time of rapid ventricular inflow or the presence of eddy current behind the cusps.

In the second phase, firmer apposition of the cusps over greater area of their atrial surfaces occurs in association with narrowing of the mitral ring and tenting of the leaflets into the left atrial cavity. This phase is accomplished by the rise in the intraventricular pressure secondary to contraction of the deep sinospiral and bulbo-spiral muscles. The papillary muscles play a major role in maintaining closure of the mitral valve orifice during ventricular systole and ejection. In 1848, Parchappe was the first to note that contraction of the papillary muscle exerted tension on the chordae tendineae and prevented eversion of the leaflets into the left atrial cavity.

The papillary muscles are aligned on an axis which points obliquely upward toward the center of the atroventricular orifice. The normal function of these muscles requires: (a) healthy myocardial fibers, and (b) normal directional axis. Damage which
causes weakening of the papillary muscle results in eversion of the cusps into the left atrium and incompetence of the valve. Scarring and fibrosis cause the same functional result by producing traction of the leaflets into the left ventricular cavity. Similarly, mitral regurgitation occurs as a result of a directional displacement of the muscle when it is included in an aneurysm. During ventricular systole with expansion of the aneurysm, the outward movement of the base of the muscle pulls the mitral cusps into the left ventricular cavity.

**HISTORIC BACKGROUND**

The earliest account of a patient with a ruptured papillary muscle was that of Mérat in 1803. His patient’s illness was initiated by severe anterior chest pain and dyspnea, and lasted only three days. At necropsy, one of the papillary muscles was torn completely across and the mitral valve was partly everted. Other similar cases were reported and a few authors turned their attention to a comparable, but much more chronic clinical sequence. In 1861, Bristowe described a 54-year-old man (Case 3) with a prominent apical systolic murmur who died in congestive heart failure. Postmortem studies disclosed an enlarged heart, with thin-walled cavities, normal mitral valve, but “the musculi papillaries were small, conical and they, with the chordæ tendineæ appeared far too short to allow complete closure of the mitral valve.” “Fatty degeneration” of the papillary muscle was believed to be the cause of mitral regurgitation by several authors in the 19th century, who reported isolated cases.

### Table 1—Clinical and Laboratory Findings in 20 Patients with "Papillary Mitral Regurgitation"

| Case | Age | Sex | Past History | Present History | Duration of Illness | History of Pain | Present Rhythm | Cardiac Status | Intensity | Type | Sa | S | Rhythm | Site of M.I. | Conduction | Defect | ST-T |
|------|-----|-----|--------------|----------------|-------------------|-----------------|----------------|---------------|------------|--------|-----|-----|----------|-------------|------------|--------|-------|------|
| **Group A** | | | | | | | | | | | | | | | | | | | |
| 1. HB 61 M Gout | None | 48 mo. | 130/80 | PC 3 | II — | AF | + |
| 2. SK 69 M Hypertension | None | 6 mo. | 104/54 | PC 3 | IV + | S | PL |
| 3. VR 68 M None | + | 36 mo. | 108/70 | — | 3 | — | S | IVCD |
| 4. CT 77 F Obese, Hypert. | None | 3 da. | 150/110 | — | 2-3 | — | S | P |
| 5. DH 70 M None | + | 19 mo. | 160/110 | PC 3 | — | — | S | P |
| **Group B** | | | | | | | | | | | | | | | | | | | |
| 6. CC 68 M Hypertension | + | 23 mo. | 135/75 | C 3 | I + | S | PL |
| 7. LVP 50 M Hypertension | + | 84 mo. | 110/70 | PC 3 | II + | AF | P |
| 8. CN 71 M Hypert., Diabet. | + | 44 mo. | 150/84 | PC 3 | I — | S | P |
| 9. JN 77 M Diabetes | + | 33 mo. | 195/110 | PC 2-3 | VI — | AF | — | + |
| 10. JJ 57 M Hypertension | + | 4 mo. | 105/80 | C 2-3 | II + | S | P |
| 11. RK 45 M None | + | 6 mo. | 128/80 | C 2 | III + | S | P |
| 12. AB 36 F Hypertension | + | 7 mo. | 150/110 | C 2 | VI — | S | AL |
| 13. MB 38 F Hypertension | + | 24 mo. | 160/110 | PC 2 | VI — | S | P |
| 14. IC 56 F Hypert., Diabet. | + | 2½ mo. | 190/90 | PC 3 | II + | S | PL |
| 15. NL 55 M None | + | 54 mo. | 110/70 | C 3 | IV — | S | P |
| 16. AN 80 F Hypertension | + | 70 mo. | 160/80 | PC 4 | — | — | S,AF | P |
| 17. JL 43 F Hypert., Diabet. | + | 54 mo. | 165/115 | PC 2-3 | II + | S | PL |
| 18. EZ 55 M Gout | + | 102 mo. | 105/80 | PC 3 | — | + | S | AL |
| 19. EB 56 F Hypertension | + | 19 mo. | 200/130 | PC 3 | VI + | S | Later |
| 20. ET 69 F | 36 mo. | 160/100 | PC 3 | II + | S | LBBB |

*Type—according to Perloff-Harvey’s classification of pansystolic murmurs.

**Abbreviations:** Hypert. = systemic hypertension; Diabet. = diabetes mellitus; C = compensated cardiac state; PC = partially compensated; Site of myocardial infarction—P = posterior; PL = posterolateral; A = anterior; AL = anterolateral; IVCD = intraventricular conduction defect; LBBB = left bundle branch block; Rhythm — S = sinus; AF = atrial fibrillation.
mitral regurgitation when there is damage or fibrosis, but not rupture of, the papillary muscle seems not to have been fully appreciated. McKusick and Levy and Edwards commented on scarring of the papillary muscles as a cause of mitral regurgitation. By 1952, Hope and Askey described a systolic apical murmur and thrill in a 61-year-old woman six days after a severe attack of chest pain. The patient was a known hypertensive with diabetes mellitus and recurrent angina pectoris. Four months later, postmortem examination revealed a fibrotic posterior papillary muscle and a scar on the posterolateral surface of the left ventricle. Recently Burch and co-workers stressed the frequency of this disease.

Material

Twenty patients have been studied in the past several years (Table 1). They fall into two groups: Group A included five patients in whom the diagnosis was proved by examination of the mitral valves and papillary muscles; these patients were all diagnosed prior to either surgery (Cases 1, 2) or death (Cases 3, 4, 5). Group B was made up of 15 patients (Cases 6-20) in whom the diagnosis was made on clinical grounds. The diagnosis of papillary mitral regurgitation was based on the following clinical criteria: (1) the appearance of an apical pansystolic murmur following myocardial infarction either of the transmural or subendocardial type; (2) the absence of a previous history of rheumatic fever or cardiac murmur; (3) the demonstration of progressive enlargement of the left atrial chamber by x-ray or of the presence of an expansile left atrial chamber observed on fluoroscopy; (4) the absence of calcification in the mitral or aortic valve or in the mitral annulus.

There were eight women and 12 men. Ages ranged between 38 and 80 years averaging 60 years. In 13 patients, there was a previous history of systemic hypertension; four patients had diabetes mellitus and two had gouty arthritis. Cases 13 and 17 had hysterectomies and probable bilateral oophorectomies, six and eight years, respectively, prior to the onset of their illness. In none of these patients could a previous history of heart murmur or rheumatic fever be obtained; and 13 of these patients prior to the onset of their illness had previous medical examinations at Parkland Hospital which revealed no cardiac murmur. With the exception of Cases 1, 2, and 4, all patients experienced anginal pain prior to or at the onset of their illness.

Clinically, all patients had a pansystolic murmur which was localized to the apex and radiated to the left axilla, but an associated apical systolic thrill was present in

![Figure 1: Simultaneous phonocardiographic recordings made at the apex and at the pulmonic area showing the pansystolic character of the murmur at the apex.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=data/journals/chest/21421/)
one (Case 16). With the exception of the last patient, the intensity of the murmur varied between grades 2 and 3 on a basis of IV (Table 1). A prominent third heart sound was present in ten patients. Phonocardiographic recordings of both the apical and basal areas of the heart were made in 15 patients. The configuration of the recorded apical systolic murmurs varied. According to Perloff and Harvey's classification, the murmur was of type I in two patients, of type II in five, of type III in two, of type IV in two and of type VI in four. In the same tracing, the configuration of the murmur may change, in particular those murmurs belonging to type III. The pulmonic second sound was widely split (0.07 sec.) in one patient (Case 7) and narrowly split in the remaining patients. A prominent fourth heart sound was recorded in five patients (Cases 7, 11, 16, 18 and 20). In Case 7, diastolic vibrations followed the third heart sound and lasted 0.20 sec.

The patients were followed for a period ranging from three days to 102 months. One patient (Case 4) died on the third day of her illness, while in Case 3, the onset of the disease was probably three years prior to demise. During their follow-up, sinus rhythm was present in 17 patients; the remaining three patients had permanent auricular fibrillation. In Case 16, paroxymal episodes of atrial fibrillation were noted. The electrocardiogram revealed posterior (inferior) or posterolateral myocardial infarction in 12 patients, anterior or lateral myocardial lesion in four patients, intraventricular conduction defect and left bundle branch block in two, and marked ST depression and diphasic T waves in two patients. Case 1 had a Qr complex in V1-V3. In nine patients, ventricular premature beats occurred frequently and in several there were short runs of ventricular tachycardia.

In 13 patients, the increasing severity of congestive heart failure and the appearance of episodes of acute pulmonary edema necessitated frequent admissions to the hospital. In two patients (Cases 11, 13), digitalis therapy was discontinued a few months after the acute episode.

**Figure 2:** Cardiac series showing marked enlargement of the left atrial chamber.
Three patients (Cases 3, 17, 18) developed left ventricular aneurysm. During the course of their illness Case 3 and probably Case 1 developed bacterial endocarditis and in one patient (Case 10) an embolic occlusion of the left axillary artery occurred.

Two illustrative cases will be presented in detail.

CASE 1

The illness of this 65-year-old man started in June, 1960 with chills and fever. Prior to this date, he had been in good health. He was found to have a heart murmur and one blood culture out of eight grew gram-positive staphylococci. Treatment for bacterial endocarditis was apparently successful.

In November, 1960, he was admitted to Parkland Hospital because of recurrence of fever. A pansystolic apical murmur radiating to the axilla was present. Ten blood cultures were sterile. His temperature ranged between 99° to 103°F. rectally, but subsided in five to six days without specific antibiotic therapy. While in the hospital, he developed a swollen left knee joint. The joint fluid was sterile and the synovial biopsy was nonspecific. The serum uric acid level varied between 5 and 8 mg. per cent. Latex sheep cell agglutination was negative. C-reactive protein was 2+ and antistreptolysin titer was 50 units.

The patient did well until May, 1963 when he was readmitted to the hospital with congestive heart failure. The heart was enlarged and there was a pansystolic apical murmur (Fig. 1) radiating to the left axilla, but not associated with a thrill. The electrocardiogram revealed atrial fibrillation and a Qr wave in V1-V3. He was subsequently followed in the cardiac clinic on repeated occasions with symptoms of congestive heart failure.

The pertinent laboratory findings were: hemoglobin 17.3 gm., 100 ml., hematocrit 55 per cent, normal total leukocyte and differential counts, platelet count 115,000 per ml., blood urea nitrogen 14 mg. per 100 ml., serum uric acid 7.5 mg. per 100 ml. X-ray film of the feet was compatible with gouty arthritis.

In August, 1963, he was readmitted for cardiac evaluation and surgery. The patient had

![Figure 3: In a left anterior oblique projection, the radiopaque dye is shown to regurgitate into the left atrial chamber across the posterior mitral leaflet. In D, the left atrial cavity is completely opacified by the dye. The large dotted line outlines the diaphragm and the small dotted line the left ventricle.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21421/ on 04/01/2017)
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had two-pillow orthopnea and exertional dyspnea since May, 1963. He denied chest pain, but complained of a recurrent arthritis for the past five to six years. There was no history of heart murmur prior to June, 1960, and no definite history of rheumatic fever. The patient appeared chronically ill, the neck veins were flat at 45° angle. There were bilateral moist rales. The heart was enlarged. A pansystolic apical murmur, radiating to the axilla and back, was heard. There was no apical thrill or diastolic murmur. The pulmonic second sound was greater than the aortic. The liver edge was 4 cm. below the costal margin. There was no peripheral edema. Heberden's nodes were present.

The electrocardiogram was essentially unchanged; it revealed the presence of atrial fibrillation with a ventricular response of 80 to 90 per minute, a Qr complex in V1-V4 and nonspecific ST-T wave changes. Cardiac series revealed an enlarged left atrium (Fig. 2), biventricular enlargement and pulmonary vascular engorgement. At cardiac catheterization, the mean right atrial pressure was 11 mm.Hg, right ventricular pressure was 62/8 to 11, pulmonary arterial pressure was 62/20, left ventricular pressure was 108/13 to 15, aortic pressure was 120/65 and the mean pulmonary wedge was 25 mm.Hg. The height of the "V" wave was 44 mm. Cardiac output was 2.97 L/min. and cardiac index 1.77 L/min. m². The systemic arterial O₂ saturation was 95 per cent.

At cineangiography, dye injected into the left ventricular cavity regurgitated into the left atrial chamber across the posterior leaflet as shown in Fig. 3. Total replacement of the mitral valve was attempted, but the patient expired postoperatively. At postmortem, the volume of the left atrial chamber was approximately 400 ml and a jet intimal lesion was present. The mitral leaflets were normal grossly and microscopically (Fig. 4). The chordae tendineae were normal. Marked fibrosis of the anterolateral papillary muscle was present (Fig. 5). There was minimal fibrosis and scarring of the anterolateral myocardial wall, but no definite evidence of an old transmural myocardial infarction.

CASE 3

A 68-year-old man was admitted to the medical service of Parkland Hospital in September, 1959 because of extreme weakness, fever and irrational conduct for about two days. In 1929, he sustained a gunshot wound to the left forearm which had to be amputated. In 1939, some form of heart ailment was thought to have developed, but no details were obtained. In 1949, he experienced epigastric and right upper quadrant pain. Cholecystectomy was performed without incident. He remained in poor health after the operation, and there was strong suggestion of exertional dyspnea and several bouts of paroxysmal nocturnal dyspnea. There was no history of hypertension or rheumatic fever.

In July, 1959, he developed dull anterior chest pain, severe dyspnea, nausea and low grade fever. He was admitted to another hospital on July 30 where he was found to have a pansystolic apical murmur (grade 3), severe tachycardia and bilateral moist rales at the bases of his lungs. An electrocardiogram revealed sinus rhythm, incomplete left bundle branch block, symmetrically inverted T in limb leads II, III, aVF, changes which had appeared since April, 1957. The chest film was normal. Leukocyte count was 13,000 per ml. with 81 per cent neutrophils. He had a low grade fever for 12 days and was discharged undiagnosed but improved two weeks after admission. He did very poorly outside the hospital. He lost 30 pounds in the next two months and was admitted with shaking chills and fever in mid-September.

On September 22, physical examination showed a blood pressure of 180/70, pulse rate 120 minute and regular, temperature 101°F. rectally. There were numerous petechiae and splinter hemorrhages. The heart was slightly enlarged and a grade 3-4 pansystolic murmur was heard at

Figure 4: Cross-section of the mitral valve showing its normal structure.
the apex, obscuring both heart sounds and radiating ot the left axilla. The pulmonic second sound was accentuated. The liver and spleen were enlarged and tender.

The electrocardiogram showed a low QRS voltage, intraventricular conduction defect and flat T waves in the limb leads. Chest films showed a moderate left ventricular enlargement. There was severe anemia (7 to 9 gm. of hemoglobin) and marked neutrophilia. The urine contained numerous red and white blood cells and ++ albuminuria. The blood urea nitrogen was 62 mg./100 ml. Blood cultures grew coagulase-positive staphylococci.

Despite vigorous therapy with various antibiotics, the patient expired 20 days after admission.

At necropsy, the heart weighed 520 gm. There was a moderate out-pocketing of the posterolateral wall of the left ventricle, the wall of which was calcified. Both ventricles were dilated. The papillary muscles of the left ventricle were represented by thin fibrotic bands to which normal chordae tendineae were attached (Fig. 6). Both papillary tabs were markedly displaced upward. The anterior cusp had a friable vegetation on its atrial surface near its margin. Neither cusp was thickened or ulcerated. The left coronary artery was completely occluded just proximal to the origin of the anterior descending branch. The right coronary artery was patent. The aortic and the right heart valves were normal.

**Discussion**

The papillary muscles-chordae tendineae combination forms the supporting unit of the mitral leaflets, of which the papillary muscles are the active contractile structure. Failure of the papillary muscles to function normally usually results in mitral regurgitation.

Papillary muscle damage due to coronary insufficiency, produces two distinct clinical syndromes the first of which is therapeutically unapproachable. It occurs when, two to five days after an acute myocardial infarction, a papillary muscle rupture and acute massive mitral regurgitation supervenes. It is to this entity that the physician refers when a raucous apical pansystolic murmur appears in the immediate postinfarction period. Intractable heart failure, sudden collapse and death are the usual consequences but in rare instances, survival may be prolonged for many months.16

The second syndrome, papillary mitral regurgitation, follows a milder course and is compatible with long survival. The condition occurs when a papillary muscle is partly infarcted or when the base of the muscle is radically displaced by involvement in a ventricular aneurysm. When a ventricular aneurysm forms, mitral regurgitation may result either from a directional change of the supporting unit of the mitral leaflets which prevents them from coming into perfect apposition, or from centrifugal expansion of the wall of the ventricular aneurysm (carrying with it the base of the papillary muscle) and tending to pull the mitral leaflets downward into the left ventricular cavity.

**Figure 5:** Microscopic section of the anterolateral papillary muscle showing marked degree of fibrosis.
Infarction of the posterior papillary muscle is especially likely to result in mitral regurgitation. Its vulnerability is believed to result from its remoteness from the source of blood supply. Since it receives blood from both the right coronary and left circumflex arteries, occlusion of either vessel would damage it. Such a lesion occurs in association with either a posterior (inferior) or posterolateral myocardial infarction. Less frequently, involvement of the anterior papillary muscle in an anterior or anterolateral infarction produces papillary mitral regurgitation.

The diagnosis of papillary mitral regurgitation is based mainly on the appearance of an apical systolic murmur in the course of a developing myocardial infarction. As is apparent from clinical auscultation, and as is easily seen in the phonocardiogram (Fig. 7), the murmur is pansystolic obscuring the first and second heart sounds and tending to radiate to the left axilla. The configurations of the murmur in our cases were essentially the same as those described by Perloff and Harvey in patients with rheumatic valvular insufficiency. The types II and VI murmurs, pansystolic murmurs with even vibrations and those with late systolic accentuation, were most frequently encountered. In one patient (Case 13), a late systolic apical murmur was heard but when recorded, early systolic vibrations were observed which were missed during auscultation (Fig. 8A). This murmur appeared on the second to third day following a posterior myocardial lesion and gradually adopted an even configuration during the 24-month period of observation (Fig. 8B).

![Figure 6: Drawing of the heart showing the aneurysmal sac and the fibrotic papillary muscles. (Reprinted by permission from Arch. Int. Med., 113:318, 1964.)](image-url)

![Figure 7: Simultaneous recordings of the apical phonocardiogram and the carotid pulse tracing in Case 8. At the apex, the murmur occupied whole systole.](image-url)
Papillary mitral regurgitation has an acute onset and it usually follows a slowly progressive course. Cardiac compensation can be accomplished in some patients, but many of our patients were at best only partially compensated. In these patients, the insidious onset of congestive heart failure is principally the result of several mechanical factors acting singly or together. They are mitral regurgitation, ventricular aneurysm, and myocardial scarring. The dynamic nature of the disease process is apparent as the gradual enlargement of the left atrium further increases the size of the regurgitant jet and one can with justification say that in this syndrome, "mitral regurgitation begets mitral regurgitation." Ventricular aneurysm is a late manifestation of an early complication of myocardial infarction. Therefore, it would seem that soon after the acute episode, wastage of the left ventricular work due to the paradoxic expansion of the left ventricular wall contributes to the development of the heart failure. The combination of these mechanical factors may place an intolerable load on the left ventricle. In one patient (Case 11) after a stormy onset of the disease, the intensity of the murmur diminished with the gradual improvement in clinical status. The mechanism underlying this improvement is not certain, but may be attributable in part at least to scarring and fibrosis of the affected myocardial area.

Clinically, the frequency of this syndrome is far less than anticipated from the pathologic findings in the coronary patient. Nezlin and Shamesova found evidence of papillary muscle involvement in 20 per cent of their patients dying with myocardial infarction. Similarly, Arkhangelskii pointed to the frequent involvement of the subendocardial region in coronary artery disease. The extent of the pathologic process in the papillary muscle may in part explain the discrepancy between the frequent involvement of this muscle at necropsy, on the one hand, and the infre-

**Figure 8:** Simultaneous recordings of limb lead III and the apical phonocardiograms of Case 13 taken on the third day of her illness and 24 months later.
quency with which the clinical syndrome is recognized during life.

The syndrome of papillary mitral regurgitation should be differentiated from the other nonrheumatic clinical entities associated with a precordial pansystolic murmur and seen in the adult patient. They include: (a) acute dilatation of the left ventricle; the murmur of mitral regurgitation may appear only to subside when ventricular compensation occurs; (b) annular calcification of the mitral valve is seen mainly in the woman patient. The demonstration of calcium deposits on x-ray examination differentiate this condition; (c) rupture of the chordae tendineae results either from bacterial endocarditis or trauma. In this condition, a pseudofriction rub may be present; (d) in acquired ventricular septal defect, the result of myocardial infarction, the murmur is more intense, localized to the left sternal border and frequently associated with a thrill. The presence of pulmonic plethora would help in differentiating this condition.

As referred to above, the almost universal complication of this syndrome is congestive heart failure. In Case 2 and probably in Case 1, bacterial endocarditis appeared although the mitral leaflets were themselves not damaged. This is not surprising because in the presence of trauma such as is produced by the regurgitant jet, the likelihood of a bacterial endocarditis to develop is enhanced especially in the presence of a circulating virulent organism such as Staphylococcus aureus.

This syndrome is believed to represent a clinical spectrum. At one end of this spectrum are patients in whom heart failure can be easily controlled by medical therapy. At the other end are those patients with a gradual but progressive deterioration of their cardiac status. In these, medical therapy is unsuccessful and surgical intervention is deemed advisable.

Uncomplicated by papillary muscle involvement, surgical excision of a ventricular aneurysm can safely be performed if one or both papillary muscles are included in the aneurysmal sac, total replacement of the mitral apparatus at the time of repair of the ventricular wall must be carried out. In these cases, removal of the aneurysmal sac alone or anchoring the mitral apparatus to the left ventricular wall cannot be expected to restore normally functioning valves.

**Summary**

The clinical picture of papillary mitral regurgitation was described and two illustrative patients were reported. The diagnosis was clinically made by the appearance of a pansystolic apical murmur following the onset of myocardial infarction. In three patients, a left ventricular aneurysm was observed.

The condition is believed to result from either damage to the papillary muscle or its displacement by the aneurysmal sac or by both.

The disease is compatible with long survival. Congestive heart failure is the major complication. Two patients developed bacterial endocarditis. Total mitral valve replacement is indicated, whether or not ventricular aneurysm is present, in patients with severe form of the disease.

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**Résumé**

Discussion clinique de l'insuffisance mitrale par lésion des pliers, avec présentation de deux observations. Le diagnostic a été fait cliniquement par l'apparition, à la suite d'un infarctus du myocarde, d'un souffle holo-systolique à la pointe. Chez 3 malades, il y avait un anévrisme du ventricle gauche.

Cette insuffisance mitrale parait résulter soit d'une lésion du muscle papillaire, soit de son déplacement par le sac anévrismal, soit par l'une et l'autre processus.

La maladie est compatible avec une longue survie. L'insuffisance cardiaque congestive est la complication majeure. Deux malades ont fait une endocardite bactérienne.

Le remplacement total de la valve mitrale est indiqué, qu'il y ait ou non anévrisme ventriculaire chez des malades qui présentent une forme sévère de la maladie.

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