Mitral Incompetence Complicating Acute Myocardial Infarction

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Mitral incompetence appearing in connexion with acute coronary heart disease without papillary muscle rupture has not received much attention until recently. Castex (1933) presented eight patients with electrocardiographic findings of inferior myocardial infarction and with an apical murmur of mitral incompetence as a distinct clinical syndrome. Nezlin and Shamesova (1951) paid attention to the occurrence of mitral incompetence in association with extensive myocardial infarction involving papillary muscles. Froment *et al.* (1955) observed mitral incompetence in 48 of 189 patients with myocardial infarction, and pointed out that it was usually associated with an inferior wall lesion and that it could occur without an increase in heart size. Their series, however, was not presented in detail.

During the past few years mitral incompetence developing in patients with acute myocardial infarction has been a subject of increasing interest. In some small series of patients with this complication the occurrence of mitral incompetence without papillary muscle rupture has been ascribed to "papillary muscle dysfunction" caused by infarction or ischaemia (Burch, De Pasquale, and Phillips, 1963; Phillips, Burch, and De Pasquale, 1963; Orlando *et al.*, 1964; Bashour, 1965). The murmur arising from this kind of mitral incompetence has been described by some of these investigators as pansystolic (Orlando *et al.*, 1964; Bashour, 1965), and by others as ejection in type (Burch *et al.*, 1963; Phillips *et al.*, 1963; Segal and Likoff, 1964). In a few cases the presence of mitral incompetence has been confirmed by angiocardiographic studies (Mazzitello, 1964; Holloway, Whalen, and McIntosh, 1965; Tavel, Campbell, and Zimmer, 1965).

When experimental coronary artery ligation or papillary muscle damage has been produced in dogs,

significant mitral regurgitation has been found to occur in about half the animals (Bailas, 1965; Hider, Taylor, and Wade, 1965).

In a necropsy study the papillary muscles were involved in over 50 per cent of patients with myocardial infarction (Arkhangelsky, 1959). Thus, a high frequency of mitral incompetence might be expected in patients with myocardial infarction. So far no prospective studies have been presented on the occurrence, clinical characteristics, and subsequent course of mitral incompetence developing in connexion with acute myocardial infarction. This is a preliminary report of such a study in 195 patients.

SUBJECTS AND METHODS

The study compared 195 consecutive patients, 164 men and 31 women, who were admitted to hospital for acute myocardial infarction within 3 days from the estimated onset of attack. Their ages ranged from 33 to 86 years (mean 57.2 years). The diagnosis was based on clinical features, electrocardiographic evidence, and laboratory data. During the first ten days every patient was carefully examined by the author for the presence of abnormal cardiac pulsation and signs of congestive heart failure and was meticulously auscultated for murmurs and gallop rhythm. The murmurs were graded from 1 to 6 (Levine and Harvey, 1959). Thereafter, the patient was examined every other day until he was discharged: the average time of hospital treatment was four weeks. When murmurs were found, they were recorded by phonocardiography. Radiological examination of the heart was done 2 to 4 weeks after admission.

In all, 31 patients died, and in 28 the heart was examined at necropsy. The grade of anatomical occlusion in the major branches of the coronary arteries was estimated. The heart valves were carefully examined. Then the heart was cut perpendicular to the long axis of the left ventricle into 5 or 6 slices to reveal the site of the infarct. The papillary muscles were examined macroscopically and microscopically at three or four different levels for evidence of necrosis.

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TABLE I

RELATION OF MURMUR OF MITRAL INCOMPETENCE TO ELECTROCARDIOGRAPHIC LOCATION AND EXTENT OF MYOCARDIAL INFARCTION

Group	Total No. of patients	Location of infarction		Extent of infarction		Reinfarction
		Anterior	Infero-posterior	Transmural	Non-transmural	
Acute mitral incompetence	107	57	50	78	29	22
No murmur	63	48	15	45	18	8
Mitral incompetence on admission	15	6	9	6	9	7
Murmur of aortic sclerosis or stenosis	10	8	2	4	6	2
Total	195	119	76	133	62	39

TABLE II

TIME OF ONSET OF MITRAL SYSTOLIC MURMUR IN 107 PATIENTS

Day	1	2	3	4	5	6	7	8	9	10	11-15	16-20	21-25	26-30
No. of patients	22	9	12	19	13	3	3	5	6	2	6	3	1	3

RESULTS

An apical systolic murmur consistent with mitral incompetence developed during their hospital stay in 107 patients. In 10, the murmur was transient and disappeared before the patient left hospital. Pericardial friction was observed in 17 patients.

Relation of Murmur to Electrocardiographic Site and Extent of Infarction. There was no difference between the occurrence of anterior (antero-septal, anterior, antero-lateral) and infero-posterior (inferior, infero-lateral, strictly posterior) infarction in patients developing a mitral systolic

murmur (Table I). On the other hand, anterior myocardial infarction was more common in patients without a murmur (75%).

Characteristics of Mitral Systolic Murmur Developing in Connexion with Myocardial Infarction. The time of onset of the murmur is presented in Table II. In only 37 per cent of the patients was the murmur of grade 3 or more (Fig. 1 and 2). Consequently, in the majority of patients (63%) the murmur was faint, easily escaping detection in the usual noise in the ward, if not particularly listened for (Fig. 3). The murmur was almost

FIG. 1.—Phonocardiogram from a patient with inferior myocardial infarction complicated by acute severe mitral incompetence. On auscultation the murmur was loud (grade 4), pansystolic, high frequency at the apex, with transmission to the axilla. Note the 3rd heart sound. The nominal frequency of the phonocardiograph filter in cycles per second (Hz) is shown on each phonocardiographic tracing.

FIG. 2.—Phonocardiogram recorded simultaneously with kinetocardiogram (KCG) in a patient with antero-lateral myocardial infarction complicated by acute mitral incompetence. An externally fixed photo-cell pick-up of the KCG records an absolute displacement of the præcordial chest wall. On auscultation the murmur was grade 3 pansystolic; crescendo at apex. A prominent paradoxical cardiac pulsation was felt medially from the apex beat. This is well evident in the KCG curve as a sustained pansystolic outward "bulge" instead of the normal retraction during the ejection period.

The filters used are indicated on the tracings as in Fig. 1.

always apical, and it was not transmitted from the primary location in 46 per cent of patients. Transmission to the left sternal border was found in 37 per cent and to the axilla in 27 per cent. The murmur had a high-frequency pitch in 86 per cent. A constant pansystolic plateau, crescendo, or decrescendo murmur was heard in 81 per cent of patients. Only in a few patients was the murmur persistently of ejection type. In 20 per cent a pansystolic murmur changed to a harsher ejection type murmur and was then loudest at the left sternal

border instead of at the apex. Sometimes an ejection type murmur changed to a clear-cut high-frequency murmur of pansystolic type during severe anginal pain. If the murmur later disappeared, a pansystolic type usually changed to an ejection type before completely disappearing. In 16 per cent of patients the murmur disappeared for some days only and then reappeared.

Necropsy Findings. Of the 28 patients examined at necropsy, 18 (64%) had had a murmur of mitral incompetence. Death occurred between the 3rd and the 51st day in hospital in this group, and the murmur appeared on the 1st to the 17th day. There were 6 (33%) anterior and 12 (67%) inferior and/or posterior infarcts. In 15 patients (83%) a large macroscopical necrosis was present in the papillary muscles; in 3 it was present in the anterior, in 8 in the posterior, and in 4 in both papillary muscles. In 3 patients no necrosis was seen in the papillary muscles. One of the latter 3 patients died 6 hours after progression of a recent inferior infarction to the infero-septal region, and in another patient a healthy anterior papillary muscle was attached to the site of a moderate-sized aneurysm due to a recent anterior infarct. The third patient had only a transient murmur.

There was no significant macroscopical necrosis in the papillary muscles in the hearts of the 10 patients without a murmur. All hearts were involved by an anterior infarct. In one, there was necrosis of the last few millimetres of the tip of one papillary muscle. In another, a small longitudinal segment ending in 2 chordæ tendineæ was necrosed, but the remaining 7 chordæ were attached to the healthy main mass of the same papillary muscle. The mitral annulus was not significantly enlarged in either group, the circumference in the group with a murmur being 95–128 mm. (mean 112 mm.), and in the group without a murmur 98–128 mm. (mean 112 mm.).

DISCUSSION

Clinically detectable mitral incompetence complicating acute myocardial infarction appears to be a common finding if carefully looked for. This is understandable and is to be anticipated when the pathophysiology of acute coronary heart disease is considered. Acute coronary occlusion leads to profound hæmodynamic alterations in the function of the left ventricle. Perfusion pressure and blood flow must decrease in the vessels distal to the occluded artery. Increased intramyocardial pressure caused by the raised left ventricular end-diastolic pressure further impairs the coronary flow. Ob-

FIG. 3.—Phonocardiogram showing a faint (grade 1) barely audible high-frequency pansystolic crescendo murmur of mitral incompetence in a patient with a large infero-postero-lateral myocardial infarction (see Fig. 4). The filters used are indicated on the tracings as in Fig. 1.

FIG. 4.—A section in a transverse plane from the heart of a man of 70 years with a recent infero-postero-lateral infarct (pale area indicated by white dotted line) with involvement of the whole posterior papillary muscle (P.P.). RV—right ventricle, LV—left ventricle, A.P.—anterior papillary muscle.

viously these mechanisms can readily cause ischaemia in the sub-endocardial parts of the myocardium, including the papillary muscles.

An ischaemic papillary muscle generates tension in the isovolumetric phase of cardiac contraction, preventing mitral regurgitation during this time, but fails to contract during the ejection phase, and then

causes regurgitation and an ejection type murmur (Burch *et al.*, 1963). Completely necrosed papillary muscle would allow bulging of the corresponding mitral cusp into the left atrium immediately at the onset of systole, and cause a pansystolic murmur. This type of murmur probably appears also when the papillary muscle is attached to a dynamic or

FIG. 5A.—Low-power view of a histological specimen from the posterior papillary muscle reveals an extensive necrosis of the papillary muscle myocardium. The pale areas are mainly necrotic myocardium and the dark areas are caused by nuclear debris. (van Gieson. $\times 9.5$.)

FIG. 5B.—Microphotography of the same specimen at greater magnification shows pale staining necrotic muscle fibres. (van Gieson. $\times 100$.)

FIG. 6.—A section in a transverse plane from the heart of a man of 64 years showing a marked thinning of the myocardial wall by an extensive anterior infarct. The location of the infarct is indicated by white dotted line. Note that the infarct does not reach to the anterior papillary muscle (A.P.). For abbreviations see Fig. 4.

anatomical left ventricular aneurysm, or when acute general left ventricular dilatation pulls the papillary muscles down and to a more obtuse angle (Levy and Edwards, 1962). In the present study paradoxical cardiac pulsation (Fig. 2) pointing to an abnormal cardiac contraction occurred in 65 per cent of the patients with a mitral systolic murmur, and in 38 per cent of those with no murmur. Temporary ischaemic dysfunction of papillary muscles and transient acute dilatation of the left ventricle may explain the occurrence of the transient murmurs. On the other hand, total necrosis and eventual fibrous shortening of papillary muscles apparently may lead to permanent regurgitation.

The lower incidence of mitral incompetence in anterior myocardial infarction is understandable when the topographical position of the papillary muscles in the left ventricle is reviewed in relation to the siting of the major muscle damage, which does not always reach the papillary muscles (Fig. 4–7). Furthermore, at least anatomically, it seems that the anterior papillary muscle has better collateral circulation than the posterior muscle (James, 1965).

In some cases the apical systolic murmur that develops may be explained by the fact that a low output during the initial clinical phase of the infarction may obscure the presence of a murmur of

aortic sclerosis or dilatation. This, however, applies only to the faint ejection or decrescendo type murmurs of low or medium frequency.

Hæmodynamically, mitral regurgitation is apparently usually somewhat insignificant, though the whole spectrum from mild regurgitation to stormy irreversible left ventricular failure and death is seen. The appearance of a mitral systolic murmur is not directly related to the extent of myocardial damage, but may be due to a small lesion or ischaemic dysfunction in a part of the heart muscle which is strategically important for mitral valve competence. It may be of interest that the development of a murmur of mitral incompetence was observed in 9 patients with coronary insufficiency but without demonstrable infarction. Further analysis of the present series is in progress. The relation of the heart size and other clinical factors to the development of mitral incompetence is being analysed. A follow-up study of the patients has been undertaken in order to estimate the frequency of permanent mitral incompetence. So far the follow-up after about six months has covered 46 patients in whom mitral incompetence had occurred after myocardial infarction; in 6 of these patients the murmur had disappeared. Some of them with permanent mitral incompetence are being studied by cardiac catheterization.

FIG. 7A.—Low-power view of the macroscopically healthy anterior papillary muscle from the same heart. (van Gieson. $\times 9.5$.)

FIG. 7B.—Microphotography at greater magnification from the anterior papillary muscle verifies that the papillary muscle myocardium is undamaged. (van Gieson. $\times 93$.)

SUMMARY

The occurrence and clinical characteristics of mitral incompetence developing in connexion with acute myocardial infarction were studied in 195 patients consecutively admitted to hospital.

Apical systolic murmurs consistent with mitral incompetence developed during the hospital stay of 107 patients. The murmur usually appeared within five days of the estimated onset of the infarction. In two-thirds of the patients the murmur was faint (grade 1-2), easily escaping detection if not particularly searched for. The character of the murmur was most often high frequency and pansystolic, but many times it changed later to a harsher ejection type of murmur.

In this study in patients developing the acute mitral systolic murmur, no difference was found between the incidence of anterior and inferoposterior infarction as judged by electrocardiography. Necropsy revealed a large macroscopical necrosis in one or both papillary muscles in 15 of the 18 patients with clinically observed mitral incompetence. In the hearts of 10 patients without a murmur there was no significant macroscopical necrosis in the papillary muscles; all hearts were involved by an anterior infarction in this group. The common occurrence and characteristics of the systolic murmur of mitral incompetence in acute myocardial infarction are discussed with reference to the pathophysiology of acute ischaemic heart disease.

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