Mechanism of mitral regurgitation in patients with myocardial infarction: a study using real-time two-dimensional Doppler flow imaging and echocardiography

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ABSTRACT  The aim of the present study was to elucidate the mechanisms of mitral regurgitation accompanying myocardial infarction. Severity and site of mitral regurgitation was evaluated by the real-time two-dimensional Doppler flow imaging technique in 81 patients with old myocardial infarction. The incidence of mitral regurgitation did not depend on the region of infarction. There was, however, a close relationship between the site of regurgitation and the region of infarction. In patients with mitral regurgitation spurring from the posteromedial area of the valve, the inferior wall was involved in infarction without exception and in some of these patients, the posteromedial papillary muscle was also found to be affected by myocardial infarction; in those with regurgitation spurring from the anterolateral area, the anterior wall showed asynergy. On the other hand in patients with mitral regurgitation spurring from the central area, the region of infarction varied. In these patients, however, the larger the diameter of the mitral anulus, the more severe the grade of regurgitation. The extent of asynergy was another factor related to the severity of mitral regurgitation. Both longitudinally and transversely, broad infarction leads to the enlargement of the mitral anulus. However, even if the mitral anulus is not so dilated, severe involvement of either commissural area results in severe mitral regurgitation from the same commissural side. Thus, there are two major causative factors of mitral regurgitation: (1) asynergy of the papillary muscle or the ventricle that results in mitral regurgitation located in the commissural area of the same side as asynergy, and (2) enlargement of mitral anulus, which results in regurgitation from the central area of the orifice. The mechanisms of mitral regurgitation unveiled in the present study will contribute much to the clarification of the concept of so-called “papillary muscle dysfunction.”


MITRAL REGURGITATION is frequently observed in patients with myocardial infarction. Since it develops in the absence of any lesions in the mitral valve leaflet, its pathogenesis has been explained by the concept of “papillary muscle dysfunction” proposed by Burch et al. At present, papillary muscle dysfunction is thought to be a sequence of unsuccessful coordination of the whole mitral apparatus (which is composed of the anulus, leaflets, chordae tendineae, papillary muscles, and the left ventricular wall), rather than a mere disorder of the papillary muscle. However, the concept of papillary muscle dysfunction is a rather theoretical one and its existence has not been proven from the point of view of functional anatomy.

The purpose of this present study was to elucidate the pathogenesis of mitral regurgitation observed in patients with old myocardial infarction by assessment of the topographic features of regurgitation with a real-time two-dimensional Doppler flow imaging technique as well as two-dimensional echocardiography.

Materials and methods

The subjects were consecutive 81 patients with myocardial infarction, 76 men and five women, who were hospitalized in the National Cardiovascular Center during the period from July 1983 to July 1984. Their ages ranged from 24 to 77 years, with
an average of 56 years. Myocardial infarction was diagnosed based on medical history, echocardiography, electrocardiography, myocardial enzyme content in the serum, and other laboratory tests. Seventy of the 81 patients underwent coronary angiography and left ventriculography.

Asynergy of the ventricular wall was demonstrated in all 81 patients by two-dimensional echocardiography. The patients were classified into three groups according to the region of asynergy as follows: (1) anterior infarction (n = 29), (2) inferior infarction (n = 26), (3) anterior and inferior infarction (n = 26). Echocardiography demonstrated abnormal Q waves in 21 patients with anterior infarction, in 15 patients with inferior infarction, and in 21 patients with anterior and inferior infarction. Methods of identifying regions of asynergy are described below.

Methods. The equipment used included a real-time two-dimensional Doppler flow imaging system (Aloka XA-54 prototype with a 2.5 MHz transducer) that has been described in a previous report and a commercially available real-time two-dimensional echocardiograph (Toshiba SSH-40A, 2.5 MHz transducer). The former system enabled one to visualize topography of the intracardiac flow, to literally obtain a reconstructed image of intracardiac flow based on flow velocity on a color television screen, where the flow velocity components toward and away from the transducer were displayed in red and blue colors, respectively. The flow image was superimposed on the two-dimensional echocardiogram, which was displayed on the same screen. The magnitude of the flow velocity component was represented by the brightness of the color. Green tone was added to each color in proportion to the turbulence of the flow. The latter system was used for evaluation of morphology and dynamic features and for measurement of the geometry of the heart.

In the present study, Doppler and echocardiographic examinations were performed 1 month after the onset of infarction.

With the real-time two-dimensional Doppler flow imaging system, not only the presence of mitral regurgitation, but also its direction, site, and extent could be diagnosed, being characterized by anatomy of the heart. Whether the regurgitant jet was toward the anterior or the posterior atrial wall was determined in the long-axis view of the left side of the heart and whether the direction was toward the right or the left was determined in the short-axis view at the mitral commissural level. The spurring site of the mitral regurgitant jet was determined to be the anterolateral, central, or posteromedial area of the mitral orifice from the short-axis view. The maximum intrusion distance of the regurgitant jet signal from the mitral orifice into the left atrial cavity was used to determine the severity of regurgitation: a distance of less than 1.5 cm was designated as grade I, that of 1.5 cm or more but less than 3.0 cm as grade II, that of 3.0 cm or more but less than 4.5 cm as grade III, and that of 4.5 cm or more as grade IV. Although the general principle behind determination of the severity of mitral regurgitation by the color flow imaging has already been reported elsewhere, we examined whether this method could also be used in patients with myocardial infarction. Therefore, we compared severity of mitral regurgitation determined by the Doppler flow imaging technique with that evaluated by the Sellers’ classification using the left ventriculograms from the patients who underwent cardiac catheterization.

The following factors related to the site and severity of mitral regurgitation were studied: the presence and site of mitral valve prolapse, the region and extent of asynergy, the diameter of the mitral anulus, the size of the left ventricle, and the location and severity of the coronary artery lesions.

Mitral valve prolapse was determined by two-dimensional echocardiography. To determine the site of the prolapse, three different parasternal long-axis views of the mitral valve were examined, that is, cross sections through the medial, central, and lateral parts of the valve, respectively. The degree of mitral valve prolapse was evaluated by measurement of the difference between the levels of the anterior and posterior mitral leaflets at the coaptation area, which was graded as follows: a difference up to 5 mm, degree I; a difference greater than 5 mm up to 10 mm, degree II; a difference greater than 10 mm, degree III. Left ventricular asynergy was diagnosed by two-dimensional echocardiography. In the present study, dyskinesis, akinesis, and also hypokinesis were taken into consideration. To analyze wall motion, the left ventricular wall was divided into segments in the short-axis view at each of three different levels (basal, midventricular, and apical) for a total of nine segments, according to Heger et al. The anterior and lateral segments thus categorized were then designated as anterior wall segments, and the medial and posterior segments were designated inferior wall segments. The extent of asynergy was sized semiquantitatively by two modes, namely the longitudinal and transverse extents of asynergy. The extent of the area of asynergy was sized semiquantitatively in the short-axis view by the following method. The transverse extent was determined by counting up segments exhibiting asynergy in the short-axis view and the longitudinal extent by shifting the view in the three different levels. Special attention was paid to asynergy of the attaching wall of the papillary muscles. The echocardiographic evaluation of asynergy was done by two observers with no other clinical information. There were interobserver discrepancies for data from eight patients, although differences were limited with respect to hypokinesis. When discrepancies existed, the echocardiograms were reviewed by the two observers and a consensus was reached.

The echocardiographic diagnosis of the severity of asynergy was compared with the ventriculographic one in the 70 patients who underwent left ventriculography. There was agreement in 65 of the 70 patients (93%). Discrepancy was limited with respect to diagnosis of hypokinesis. Because these results were considered acceptable, the following study was done, depending on the echocardiographic diagnosis.

The end-diastolic diameter of the mitral anulus was measured as the distance between the proximal ends of the anterior and posterior leaflets in the parasternal long-axis view through the central part of the valve by two-dimensional echocardiography.

Left ventricular end-diastolic volume was measured from the right anterior oblique view of the left ventriculogram by the area-length method in the 70 patients who underwent left ventriculography.

The location and severity of coronary lesions were ascertained by coronary angiography.

Spearmann’s rank correlation method was used for the comparison of the results on the severity of regurgitation obtained by Doppler flow imaging and those by left ventriculography. The Fisher exact probability test was used for the evaluation of relationships between incidence, site, and severity of mitral regurgitation, and the region and extent of myocardial infarction. Analysis of variance was applied in the evaluation of the relationship between the severity of mitral regurgitation and the diameter of the mitral anulus. A p value less than .05 was considered indicative of statistical significance.

Results

Severity of mitral regurgitation determined by the Doppler flow imaging technique in comparison with that by left ventriculography. In the 70 patients who underwent left ventriculography, the severity of mitral regurgitation was graded by this method as well as by the Doppler
technique. The results obtained by the latter were the same as those by the former in 52 of 70 patients and differed by one grade in the remaining 18. Mitral regurgitation was imaged by the Doppler method in eight patients of the 38 in whom it was not observed on the left ventriculogram, and it was detected on the ventriculogram in eight patients of 38 in whom Doppler results were negative (figure 1). However, regurgitation was mild in these patients. Thus, since the correlation between results was satisfactory, the Doppler technique was shown to be noninvasively applicable for grading the severity of mitral regurgitation in patients with myocardial infarction and was used for the duration of the study.

Incidence and severity of mitral regurgitation referred to the region of infarction. Regurgitation was detected in 43 of the 81 patients (53\%), i.e., 11 of 28 (39\%) in the anterior infarction group, 14 of 25 (56\%) in the inferior infarction group, and 18 of 28 (64\%) in the anterior and inferior infarction group (figure 2). Of the patients with regurgitation, 21 patients were assessed as having grade I, 13 as grade II, four as grade III, and five as grade IV mitral regurgitation. The incidence and the severity of regurgitation appeared to be rather high in the anterior and inferior infarction group, but the difference was not statistically significant.

Site of mitral regurgitation. The spurring site of mitral regurgitation was clearly demonstrated in the mitral orifice by the real-time Doppler flow imaging technique, while it was not by the left ventriculography. In each patient, it was limited to one of the following three areas: the anterolateral commissural area, the central area, or the posteromedial commissural area (figure 3). It was anterolateral in four patients, central in 29 patients, and posteromedial in 10 patients.

The positional relationship between the site of regurgitation and the region of infarction was examined (table 1). It is noteworthy that the four anterolateral cases included two patients with anterior infarction, but none with inferior infarction, while the 10 postero medial cases included eight patients with inferior infarction, but none with an anterior one. Thus, a close relationship was noted between commissural areas of regurgitation and the region of infarction (p < .05).

Mitral valve prolapse was demonstrated at the site of regurgitation in one of the four patients with regurgitation from the anterolateral area, in three of the 29 patients with regurgitation from the central area, and in four of the 10 patients with regurgitation from the posteromedial area.

In three of the four patients with prolapse in the posteromedial area, prolapse was of degree III and was noted in the posterior leaflet, being accompanied by mitral regurgitation of grade III or IV at the same site as that of prolapse. The posteromedial papillary muscle was thin and finger-like in configuration by enhanced echocardiography, being accompanied by asynergy of its attaching wall (figure 4). Direct observation was made at surgery or autopsy in two of the three patients, confirming infarction involving the papillary muscle.

FIGURE 1. Severity of mitral regurgitation determined by Doppler flow imaging technique in comparison with that determined by left ventriculography. There was satisfactory correspondence between results with the two methods. The correlation coefficient was .99 (Spearman's rank correlation method). Numbers from I to IV are gradings of severity of mitral regurgitation. This classification is based on 1.5 cm step increases in distance. LVG = left ventriculography; Doppler = Doppler flow imaging; (-) = no mitral regurgitation.

FIGURE 2. Relationship between mitral regurgitation and region of myocardial infarction. The incidence and the severity of regurgitation appeared to rather high in the anterior and inferior infarction group, but the difference was not statistically significant. Numbers of patients are indicated. MR (+) = with mitral regurgitation; ANT = anterior infarction; INF = inferior infarction; ANT + INF = anterior and inferior infarction; MR = III, IV = severe mitral regurgitation (not less than grade III); MR = II = mitral regurgitation of grade II; MR = I = mitral regurgitation of grade I.
FIGURE 3. Sites of mitral regurgitation in the mitral orifice demonstrated by Doppler flow imaging technique. Short-axis view at the level of the mitral commissures. A. Mitral regurgitant jet spurs from the anterolateral area of the valve into the left atrial cavity. B. Mitral regurgitant jet from the central area of the valve. C. Mitral regurgitant jet from the posteromedial area of the valve. LV = left ventricle; LA = left atrium; R = right; L = left.

TABLE 1

<table>
<thead>
<tr>
<th>Region of infarction</th>
<th>Anterior</th>
<th>Anterior and inferior</th>
<th>Inferior</th>
<th>Total</th>
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<tr>
<td>Site and severity of mitral regurg.</td>
<td></td>
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<tr>
<td>Anterolateral</td>
<td></td>
<td></td>
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<tr>
<td>Slight</td>
<td>2 (1)</td>
<td>2 (2)</td>
<td>0 (1)</td>
<td>4 (1)</td>
</tr>
<tr>
<td>Severe</td>
<td>2 (1 ant)</td>
<td>0 (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Slight</td>
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<td>14 (1 ant)</td>
<td>6 (1 ant)</td>
<td>29 (3)</td>
</tr>
<tr>
<td>Central</td>
<td>14 (1 ant)</td>
<td>0 (1)</td>
<td>2 (1 ant)</td>
<td>10 (4)</td>
</tr>
<tr>
<td>Severe</td>
<td>0</td>
<td>2 (2)</td>
<td>8 (3 post)</td>
<td></td>
</tr>
<tr>
<td>Slight</td>
<td>2 (1)</td>
<td>0 (1)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Posteromedial</td>
<td>0</td>
<td>0 (1)</td>
<td>4 (3 post)</td>
<td></td>
</tr>
</tbody>
</table>

The columns show the region of asynergy and the rows show the site of mitral regurgitation. Values are numbers of patients. Values in parentheses indicate the numbers of cases of mitral valve prolapse and prolapsed leaflet. ant = anterior leaflet; post = posterior leaflet; slight = mitral regurgitation of grade I or II; severe = mitral regurgitation of grade III or IV.
In the other five patients with mitral valve prolapse, prolapse was noted in the anterior leaflet, being of high grade with severe regurgitation in two and of low grade with slight regurgitation in the remaining three. In these five patients, no specific findings were noted regarding the papillary muscle.

**Extent of asynergy and severity of mitral regurgitation.**

The longitudinal extent of asynergy was classified as follows: In the presence of anterior infarction, left ventricular asynergy limited to the apical region under the papillary muscle level was designated as narrow infarction, and asynergy extending to the basal level as broad; in patients with inferior infarction, asynergy limited to the basal level was designated as narrow infarction, and asynergy extending to the attaching wall of the papillary muscle as broad; and in the presence of anterior and inferior infarction, when either anterior and/or inferior walls had broad infarction, the extent of asynergy was designated as broad. The results indicated broad infarction in 54 patients and narrow infarction in 27 (figure 5).

Among the 54 patients with longitudinal broad infarction, nine exhibited mitral regurgitation of grade III or IV, 25 that of grade I or II, and 20 had no regurgitation. Of the 27 patients with narrow infarction, nine exhibited mitral regurgitation of grade I or II and 18 had no regurgitation. All nine patients with mitral regurgitation of grade III or IV had longitudinally broad infarction, and no patient with narrow infarction had severe mitral regurgitation (figure 5). Thus, there was significant relationship of the longitudinal extent to the severity of mitral regurgitation (p < .05).

The transverse extent of asynergy was classified as follows: Myocardial infarction accompanied by asynergy in six or more segments was designated as broad infarction, and that accompanied by less than six segments was designated narrow infarction. The results indicated broad infarction in 10 patients and narrow infarction in 71 (figure 6).

Of the 10 patients with transversely broad infarction, four exhibited mitral regurgitation of grade III or IV, four that of grade I or II, and two had no regurgitation. Of the 71 patients with transversely narrow infarction, five exhibited mitral regurgitation of grade III or IV, 30 that of grade I or II, and 36 had no regurgitation (figure 6). It is worthy of note that mitral regurgitation in seven of eight patients with transversely broad infarction spurted from the central area.

Of the nine patients with mitral regurgitation of grade III or IV, infarction was longitudinally and transversely broad in four, and was longitudinally broad but transversely narrow in five. It should be emphasized that the site of regurgitation was central in the former four

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**FIGURE 4.** Mitral valve prolapse in a patient with inferior infarction. Long-axis view from the parasternal approach in systole. Severe prolapse of the posterior mitral leaflet was observed, as indicated by the small arrow. As shown by the large arrow, the posteromedial papillary muscle was unusually thin, like a finger in configuration, and its echo intensity appeared to be enhanced, suggesting a cicatricial change due to papillary muscle infarction. LV = left ventricle; LA = left atrium; AO = aorta.

**FIGURE 5.** Relationship between the longitudinal extent of asynergy and the severity of mitral regurgitation. There was a correlation between the longitudinal extent of asynergy and the severity of mitral regurgitation. All patients with severe mitral regurgitation were included in the longitudinally broad infarction group (BROAD). There were no patients with severe mitral regurgitation and longitudinal narrow infarction (NARROW). Numbers of patients are indicated: MR(III,IV) = severe mitral regurgitation (not less than grade III); MR(II) = mitral regurgitation of grade II; MR(I) = mitral regurgitation of grade I; MR(-) = no mitral regurgitation.
patients, while it was posteromedial in four of the latter five, including the three patients with papillary muscle infarction (table 1). A statistically significant relationship was noted between the site of regurgitation and the mode of extent of infarction (p < .01).

Diameter of the mitral anulus. The diameter of the mitral anulus in the patients with mitral regurgitation spurring from the central area correlated with the end-diastolic left ventricular volume (n = 25, r = .58, p < .005). This relationship also held in the patients without mitral regurgitation (figure 7). In the patients with regurgitation spurring from the central area, the diameter of the mitral anulus correlated well with the severity of regurgitation. However, it is noteworthy that such correlation was absent in the patients with regurgitation spurring from the commissural areas (figure 8).

Mitral regurgitation and coronary lesions. Stenosis of the coronary artery more severe than 75% of diameter was designated as a significant stenosis. All five patients with mitral regurgitation from the posteromedial or anterolateral area were included in the transversely narrow infarction group (NARROW). Numbers of patients are indicated. MR(III,IV) = severe mitral regurgitation (not less than grade III); MR(II) = mitral regurgitation of grade II; MR(I) = mitral regurgitation of grade I; MR(−) = no mitral regurgitation.

Discussion

An important finding of the present study was that myocardial infarction is frequently accompanied by mitral regurgitation, and that mitral regurgitation accompanying old myocardial infarction is often localized within a certain area in the coaptation line of the mitral orifice. Use of the real-time Doppler flow imaging technique was advantageous for demonstration of such a characteristic feature of mitral regurgitation in myocardial infarction. Although such a feature might be observed by the conventional pulsed Doppler method,3 its use under these circumstances would be very troublesome in comparison with our technique. The continuous-wave Doppler technique may easily detect mitral regurgitation, but is difficult to judge its site and severity by this method. The Doppler flow imaging technique was indispensable for the purpose of the present study.

![Figure 6](image6.png)

**FIGURE 6.** Relationship between the transverse extent of asynergy and the severity of mitral regurgitation. All patients with severe mitral regurgitation from the central area were included in the transversely broad infarction group (BROAD), while all patients with severe mitral regurgitation from the posteromedial or anterolateral area were included in the transversely narrow infarction group (NARROW). Numbers of patients are indicated. MR(III,IV) = severe mitral regurgitation (not less than grade III); MR(II) = mitral regurgitation of grade II; MR(I) = mitral regurgitation of grade I; MR(−) = no mitral regurgitation.

![Figure 7](image7.png)

**FIGURE 7.** Relationship between left ventricular end-diastolic volume (LVEDV) and the diameter of the mitral anulus in the patients with mitral regurgitation from the central area and without regurgitation. The end-diastolic diameter of the mitral anulus correlated with LVEDV (n = 61, r = .58, p < .005). Closed and open circles represent patients with and without mitral regurgitation, respectively.
The results regurgitation.

FIGURE 8. Diameter of the mitral anulus in reference to the severity of mitral regurgitation. Left, Patients with regurgitation spurring from the central area of the mitral valve. Right, Patients with regurgitation spurring from the posteromedial area or anterolateral area of the mitral valve. The severity of regurgitation is graded based on the two-dimensional image. In patients with mitral regurgitation spurring from the central area of the mitral valve, the wider the mitral anulus, the more severe the mitral regurgitation. This characteristic was rarely noted in patients with regurgitation spurring from the commissural areas. Data are presented as the mean ± SD.

We examined, in a preliminary manner whether Doppler flow imaging could be used instead of left ventriculography for evaluating the severity of mitral regurgitation. The results showed that the correlation between results with the two methods was generally significant, although some differences were noted in patients with mild cases of mitral regurgitation. Thus, for the duration of the study, the Doppler flow imaging technique was used for grading mitral regurgitation. It was also possible to easily determine the site and direction of mitral regurgitant jet. The Doppler flow imaging technique provides a new tool for understanding the pathologic physiology of mitral regurgitation in myocardial infarction.

In the past, involvement of the papillary muscle had been considered to be a cause of mitral regurgitation after myocardial infarction and the concept of “papillary muscle dysfunction” was proposed. This concept was later stretched to explain the pathogenesis of mitral regurgitation not only in the presence of myocardial infarction, but also in other conditions, such as dilated cardiomyopathy and myocarditis. However, the theory has not yet been definitely established from the anatomic and pathologic points of view although some investigators have tried to demonstrate using echocardiography that some intracardiac events are related to papillary muscle dysfunction.

In the present study, a close relationship was noted between the site of mitral regurgitation and the region of infarction in patients with regurgitation spurring from the posteromedial and anterolateral commissural areas. Irrespective of the direct involvement of the papillary muscle, asynergy in the neighboring area of the papillary muscle may influence the orientation of the papillary muscles on contraction, resulting in insufficient coaptation of the mitral leaflet, although this is not necessarily detected by echocardiography. Godley et al. reported that, in cases of mitral regurgitation accompanying myocardial infarction, dyskinesis was regionally noted in the left ventricular wall at the base of the papillary muscles. Also, it was reported that systolic shortening of the papillary muscle is more impaired than that of the circumferential free wall in acute experimental myocardial ischemia. Generally, the region of asynergy usually appears to be nearer to the posteromedial papillary muscles in the presence of inferior infarction than it is to the anterolateral papillary muscle in the presence of anterior infarction. This may be the reason that the incidence of commissural mitral regurgitation in patients with anterior infarction was less than that in patients with inferior infarction.

All of the patients with papillary muscle infarction had mitral valve prolapse and some without papillary muscle infarction did also, resulting in mitral regurgitation. The number of patients with mitral valve prolapse was small in the present study. However, the incidence of mitral valve prolapse was plainly higher than that in the general population, although it is uncertain whether these prolapses resulted from infarction in all the patients concerned. Thus, it would appear that myocardial infarction is likely to result in dysfunction of the mitral valve apparatus on the same commissural side irrespective of the direct involvement of the papillary muscles.

At this time, the anatomic conditions of the papillary muscles should be discussed. While the blood supply to anterolateral papillary muscles and the surrounding area usually comes from both the diagonal branch and left circumflex artery, that to the posteromedial papillary muscles mainly comes from the right coronary artery only, so that the latter is likely to be more vulnerable to ischemia than the former. The shape of the papillary muscle is another factor relating to vulnerability. The shape of the papillary muscle has been anatomically classified into three types: finger-like, tethered, or mixed. While the papillary muscles of the mixed and those of the tethered types have subendocardial anastomosis in their attachment area, those of the finger-like type are exclusively perfused only by
their own central artery, so that the latter is considered to be more vulnerable to ischemia than the former. The anatomic conditions described seem to explain the fact that the infarcted papillary muscle was exclusively in the posteromedial area in the present study. In all three cases of papillary muscle infarction, the papillary muscles were finger-like.

In the patients with regurgitation from the center of the orifice, the enlargement of the mitral anulus is assumed to be mainly responsible for mitral regurgitation, which is supported by the result that worsens with the enlargement of the mitral anulus diameter. In general, it has been accepted that enlargement of the mitral anulus results in mitral regurgitation.\textsuperscript{17} The mitral anulus is enlarged by the increase in the left ventricular volume, so that the coaptation area of the mitral valve leaflets may consequently become narrow, resulting in mitral regurgitation. The results obtained in the present study show that this is also the case in mitral regurgitation from the center of the mitral orifice in patients with old myocardial infarction. This resembles the mechanism of functional mitral regurgitation in dilated cardiomyopathy.\textsuperscript{18, 19}

The extent of asynergy was also another factor related to the severity of mitral regurgitation. Of the nine patients with severe mitral regurgitation, the extent of asynergy was broad longitudinally in five and broad both longitudinally and transversely in four. It should be emphasized that while mitral regurgitation spurted from the commissural areas in the former five patients, it spurted from the central area of the mitral orifice in the latter four. Here, it is assumed that very broad infarction such as that in the latter four patients leads to the dilatation of the left ventricle, followed by the enlargement of the mitral anulus, so that mitral regurgitation spurted from the central area of the mitral orifice. Thus, relationships between the diameter of the mitral anulus, the extent of asynergy, and the severity and site of mitral regurgitation are systematically understandable. However, even if the extent of asynergy was broad, mitral regurgitation was not necessarily severe. For mitral regurgitation from the central area, the diameter of the anulus appears to be a more directly determining factor than the extent of asynergy (figures 7 and 8).

The circumstances were different in the latter five patients, in whom the extent of asynergy was broad longitudinally, but not transversely. In these patients, mitral regurgitation spurted from the commissural area. In the present study, the severity of mitral regurgitation did not necessarily depend on the diameter of the mitral anulus in the patients with regurgitation from the commissural areas (figure 8). All three patients with papillary muscle infarction were included in the above-mentioned five patients with severe mitral regurgitation. These data show that the severe involvement of either commissural area results in severe mitral regurgitation from the same side, even if the left ventricle is not dilated to a great extent.

Thus, the major determining factors of the severity of mitral regurgitation can be related to the extent of dilatation of the left ventricle, since this determines the enlargement of the mitral anulus, and to the extent of the involvement of either commissural area. However, these two major factors are not completely independent from each other; they must be related to the extent of infarction. Mitral regurgitation may secondarily result in the dilatation of the left ventricle. These factors may cooperate with each other to various extents in individual patients to cause mitral regurgitation.

In conclusion, it appears from our results that there are two major causative factors of mitral regurgitation accompanying myocardial infarction: (1) asynergy of the papillary muscles or the left ventricular wall, which results in mitral regurgitation located in the commissural area of the same side as asynergy, and (2) enlargement of the mitral anulus, which results in regurgitation from the central area of the orifice. It is anticipated that the results of the present study will result in greater understanding of the concept of so-called papillary muscle dysfunction.

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