Mitral Regurgitation in Coronary Artery Disease*

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Over a period of 5.25 years, 1,530 patients with coronary artery disease (CAD) underwent catheterization; 104 had associated mitral regurgitation (MR), and 60 had no complications. Twelve patients underwent coronary artery bypass graft surgery (CABG), with both pre- and postoperative angiograms. Nine of the 12 patients (75 percent) were in functional class 3 or 4. Left ventricular ejection fraction ranged from 34 to 75. The MR was considered severe (3+) in three, moderate (2+) in six, and trivial (1+) in three patients. Following CABG, all except two patients were in class 1. Of the 43 patients medically treated, 31 patients (72 percent) were in functional class 3 or 4. Angiographic results showed that five patients had 3+ MR, 14 had 2+ MR, and 24 had 1+ MR. The EF was <30 in 23 patients and ≥30 in 20 patients, and left ventricular filling pressure was elevated. Twenty patients died, with a mean follow-up period of 11 months. Our study demonstrates that the surgically treated patients showed angiographic improvement in MR, improved functional status, and relief of symptoms compared with medically treated patients. We believe that a subset of patients with MR and CAD would benefit with CABG.

Mitral regurgitation (MR) in patients with coronary artery disease (CAD) is relatively uncommon. Papillary muscle dysfunction, papillary muscle rupture, chordal rupture, scarring of the papillary muscle, or left ventricular aneurysm all may be associated with MR in CAD. Mitral regurgitation secondary to papillary muscle rupture or chordal rupture occurs in acute myocardial infarction and is usually fatal unless surgically treated. The aim of this study was to find out the effect of revascularization on MR that was due only to ischemic heart disease. In addition, we wanted to assess the prognosis of patients who were not considered good candidates for bypass surgery.

**MATERIAL AND METHODS**

Catheterization reports on 2,138 cases from the Buffalo Veterans Administration Medical Center, dating from February 1975 through May 1980, were reviewed. CAD was documented in 1,530 cases by cardiac catheterization, and 104 (6.8 percent) also had MR proved by ventriculogram. Forty-four patients were assigned different groups according to the cause of their MR (Table 1).

Sixty of the 1,530 patients (4.0 percent) had MR that could not be assigned to any cause, leaving ischemic heart disease as the possible cause. They were further subdivided into medical and surgical groups. Forty-three patients were treated medically. Seventeen patients had coronary artery bypass graft surgery (CABG). Three of these patients refused coronary angiograms, and two patients died of non-cardiac causes, leaving 12 patients with postoperative angiograms to form our surgical study group.

The following parameters were observed during cardiac catheterization: ejection fraction (EF); left ventricular end-diastolic pressure (LVEDP); aortic pressure; wall movements; and number of coronary arteries involved. The severity of MR was estimated. In addition, graft patency and progression of disease in the native vessels were studied. When all grafts that supplied all the graftable vessels were patent, it was considered to be a complete revascularization. Four patients had right heart catheterization done preoperatively, and eight patients had right heart catheterization performed postoperatively.

The MR was considered to be severe (3+) when the ventriculogram showed a distinct systolic filling of the left atrium with every ventricular contraction and re-

<table>
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<tr>
<th>Table 1—Distribution of Mitral Regurgitation (MR) in 44 Patients</th>
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<tr>
<td>Prolapse mitral valve (PMV)</td>
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<tr>
<td>Idiopathic hypertrophic subaortic stenosis</td>
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<tr>
<td>Infective endocarditis</td>
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<tr>
<td>Cardiomyopathy (diffuse)</td>
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<tr>
<td>Rheumatic heart disease</td>
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<tr>
<td>Mitral valve replacement*</td>
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<tr>
<td>PVC-induced MR</td>
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<td>Catheter-induced MR</td>
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*Two had PMV; three had CAD.
mained opacified for a few beats. The pulmonic veins were usually also seen. When a trivial regurgitant stream of dye was seen during ventricular systole that rapidly disappeared, this was labeled as 1+ MR. Anything between 1+ and 3+ was considered to be 2+ MR. The EF was calculated using the method described by Dodge et al. LV end-diastolic pressure measurements were analyzed from the reports both before and after dye injection. Right heart pressures were analyzed when available, including right atrial, pulmonary arterial, and pulmonary arterial wedge pressures. Pulmonary arterial wedge pressures were analyzed for prominence of V waves.

The length of survival was calculated from the time of cardiac catheterization to death or to September 1980. The relationship of the severity of MR, LV dysfunction, and CAD to mortality was analyzed. The functional classification of these patients was according to the New York Heart Association classification.

RESULTS

Surgical Group

All 12 patients in our surgical study group were men, with an age range of 50 to 66 years (mean age, of 56.5 years). Preoperatively, among these 12 patients, four had angina, six had angina and shortness of breath (SOB), and two had SOB only. Postoperatively, none of the patients had angina, two had SOB. These two patients had only angina preoperatively, and their postoperative EF did not change. Hence, the reason for the onset of SOB was unexplained. Preoperatively, nine of 12 patients (75 percent) were in NYHA functional classification 3 or 4, and postoperatively only one patient (8.3 percent) remained in class 3.

Electrocardiographic analysis revealed inferior wall myocardial infarction (MI) in five patients and posterior wall MI in two patients. Two patients had anterior MI in combination with an inferior MI. The remaining three patients had no ECG evidence of infarction. No patient in our study group had isolated anterior wall MI.

Four of the 12 patients were receiving long-acting nitrates preoperatively, and one patient was receiving hydralazine (Apresoline) preoperatively.

The interval between preoperative and postoperative catheterization ranged from three to 30 months, with a mean of 15.1 months. There was no significant change in the EF between the preoperative and postoperative studies.

Nine patients preoperatively (75 percent) had 3+ or 2+ MR, while only five patients (42 percent) had similar regurgitation postoperatively.

There was no significant change in systolic or diastolic pressure prior to the left ventriculogram between the preoperative and postoperative studies (P>0.1). Mean systolic pressure preoperatively was 140 mm Hg (SD=18); mean systolic pressure postoperatively was 132 mm Hg (SD=14); mean diastolic pressure preoperatively was 76 mm Hg (SD=13.3), and mean diastolic pressure postoperatively was 76 mm Hg (SD=11.2).

Preoperative left ventriculograms showed that five patients had inferior hypokinesia, three had inferior-posterior hypokinesia, and one had anterior-inferior hypokinesia. The remaining three patients had no segmental wall motion abnormalities. All but two of the nine patients with localized hypokinesia retained the abnormal wall motion postoperatively. The one patient with anterior-inferior hypokinesia had an improvement in the anterior wall motion, leaving him with only inferior hypokinesia, but his level of MR stayed 2+. One patient with inferior hypokinesia dramatically regained his wall movements and showed a complete disappearance of his MR (Fig 1).

Eight of the 12 patients had triple vessel and four had double vessel involvement. None had single vessel disease. Preoperative and postoperative right heart catheterization data was available in four patients. In two of the four patients, the V wave and the level of MR stayed the same. In the

![Figure 1A (upper) and B (lower). Preoperative ventriculogram in diastole and systole. Note mitral regurgitation and inferior hypokinesia.](http://www.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21288/)
third patient, the V wave increased from 8 to 20, but the MR remained the same. In the fourth patient, the V wave remained the same, but the MR was slightly worse.

In general, the patients had a high left ventricular end-diastolic pressure. There was no significant change in the resting (preventriculogram) LVEDP between the preoperative (mean 17 mm Hg) and postoperative (mean, 15 mm Hg) study. The mean postventriculogram LVEDP decreased from 29 to 25 mm Hg, but this change was not significant (P > .01).

There was a total of 34 grafts done in 12 patients, with a mean of 2.8 grafts per patient. All grafts were patent on repeated angiogram. These grafts were considered to be complete for total revascularization. Native coronary arteries showed progression of disease invariably in all patients.

Medical Group

The medical group consisted of 43 male patients. The age range was from 43 to 74 years, with a mean of 56.5 years. There were ten patients in functional class 4, 21 in functional class 3, eight in functional class 2, and four in functional class 1. The majority of patients (72 percent) belonged to functional classes 3 and 4. Electrocardiographic studies revealed anterior infarction in 11 patients, inferior infarction in 15 patients, and lateral in one patient. Sixteen patients had no ECG evidence of infarction. Most of the patients were receiving digoxin, diuretics, nitropaste, or long-acting nitrates, and none of the patients was in clinical heart failure at the time of catheterization.

Twenty-four patients had 1+ MR, 14 had 2+ MR, and five had 3+ MR. Six patients had two-vessel disease, and 37 had three-vessel disease. Distribution of the ejection fraction is shown in Table 2.

The majority of patients had a marked increase in filling pressure. The mean filling pressure before the ventriculogram was 21 mm Hg, and after ventriculogram, it was 30 mm Hg. Ventriculogram showed local hypokinesia in 14 patients, local dyskinesia in six patients, global hypokinesia in 19 patients, and four patients had no wall motion abnormalities.

Twenty of the 43 patients (47 percent) died. In three patients, the exact date and cause of death was not available. The mean follow-up on the remaining 17 patients was 10.6 months, with a range of three days to 32 months. Table 3 shows the distribution of mortality according to the ejection fraction and severity of mitral regurgitation. All of these patients died of cardiac causes.

Nineteen of the 37 patients with triple-vessel disease (51 percent) were dead on follow-up. In this group, four had 3+ MR, eight had 2+ MR, and seven had 1+ MR. Of the six patients with double-vessel disease, one patient died (17 percent). This patient had an LVEF of 14 percent.

The surgical and medical patients were followed up until September 1980. The surgical group had 17 patients, including the three patients who refused repeated cardiac catheterization and two patients who died. The follow-up period ranged from 4.5 to 84.7 months, with a mean of 25.2 months. The medical group consisted of 43 patients. Twenty patients died, but follow-up data was available in only 17 patients. Our graph indicates the follow-up of 40 patients where complete information was available (Fig 2). The follow-up range was from three days to 58 months, with a mean of 18.5 months. The survival in this group was 57.5 percent.

### Table 2—Distribution of Ejection Fraction (EF) and Mortality in the Medical Group

<table>
<thead>
<tr>
<th>EF</th>
<th>No. of Patients (%</th>
<th>Mortality (%)</th>
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<tbody>
<tr>
<td>&lt;20</td>
<td>13 (30)</td>
<td>9/13 (69)</td>
</tr>
<tr>
<td>20-40</td>
<td>14 (33)</td>
<td>7/14 (50)</td>
</tr>
<tr>
<td>&gt;40</td>
<td>16 (37)</td>
<td>4/16 (25)</td>
</tr>
<tr>
<td>Total</td>
<td>43 (100)</td>
<td>20/43 (47)</td>
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### Table 3—Distribution of Mortality According to Ejection Fraction and the Severity of Mitral Regurgitation (MR)

| Ejection Fraction | 1+MR | 2+MR | 3+MR | Total Mortality, No. (%)
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<tbody>
<tr>
<td></td>
<td>No. of Patients</td>
<td>No. Dead (%)</td>
<td>No. of Patients</td>
<td>No. Dead (%)</td>
</tr>
<tr>
<td>&lt;20</td>
<td>5 (1)*</td>
<td>4 (80)</td>
<td>6</td>
<td>3 (50)</td>
</tr>
<tr>
<td>20-40</td>
<td>8 (1)</td>
<td>3 (37.5)</td>
<td>4</td>
<td>3 (75)</td>
</tr>
<tr>
<td>&gt;40</td>
<td>11 (4)</td>
<td>1 (9)</td>
<td>4</td>
<td>2 (50)</td>
</tr>
<tr>
<td>Total</td>
<td>24</td>
<td>8 (33)</td>
<td>14</td>
<td>8 (52)</td>
</tr>
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</table>

*In MR 1+ patients, numbers in parentheses indicate the number of patients with double-vessel disease. All other patients had triple-vessel disease. Only one patient with two-vessel disease died on follow-up. He had an ejection fraction of 14%."
Mitral regurgitation in CAD can occur acutely during a myocardial infarction or develop chronically due to ischemia-induced papillary muscle dysfunction (PMD). The latter seems to be the most common cause of MR in CAD. PMD has been noted in the acute MI and is usually a benign disorder. On the other hand, papillary muscle rupture with infarction usually leads to pulmonary edema and death unless recognized early and treated effectively, both medically and surgically.

The recent use of afterload and preload therapy, combined with intra-aortic balloon pumping, has been helpful in stabilizing some of these conditions before corrective surgery. The surgical correction has usually been replacement of the MV with and without CABG. When MR occurs in ischemic heart disease, the benefit of revascularization has not been adequately evaluated. Our study was aimed at finding out whether revascularization helps the MR in patients with angiographic evidence of the same. The major finding was that the incidence of MR itself is indeed very low in CAD. Among the 1,530 patients with CAD, only 104 (6.8 percent) had MR. Of these, 60 (4 percent) had ischemia as the most plausible explanation of their MR.

Gahl et al. have reported a 31 percent incidence of MR in their patients who were referred primarily for intractable angina pectoris or incapacitating congestive heart failure. Their incidence is high because they selected severely ill patients for their group. Our group, unlike theirs, includes all patients catheterized for CAD, and hence the incidence of MR was much lower. It is indeed important to rule out the many different causes of MR when observed in association with CAD.

In our series, we had 17 patients with a prolapsed MV, two with idiopathic hypertrophic subaortic stenosis, one patient with infective endocarditis, one patient with diffuse cardiomyopathy, five patients with rheumatic heart disease, 13 patients with MR associated with premature ventricular contractions or catheter-induced MR, and five patients with severe MR requiring MV replacement. This left us with 60 patients who had MR secondary to CAD. In this group, 17 patients were treated surgically (28 percent), while the remaining 43 patients (72 percent) were treated medically. Five of the 17 patients treated surgically were unavailable for postoperative catheterization, leaving 12 patients in the surgical group.

The mean time interval between pre- and postoperative angiogram in our surgical group was 15.1 months (range, 3 to 30 months). There was good clinical improvement shown by complete relief of angina in all of the 12 patients following CABG. This was accompanied by a dramatic improvement in their functional classification, as assessed subjectively. There were nine of 12 patients in class 3 and 4 preoperatively, with only one patient in class 3 and none in class 4 following surgery. Ten of the 12 patients were in class 1 following surgery, and seven of these ten were in class 3 or 4 preoperatively. This shows the dramatic improvement in the functional classification following surgery. It is possible that the ischemia-induced left ventricular dysfunction and the MR related to papillary muscle ischemia were responsible for the poor functional status preoperatively, and revascularization considerably benefited these patients.

It is not surprising that a large number of surgical patients (75 percent) had inferior infarction on their ECGs. Since the posteromedial papillary muscle is supplied by either the right coronary artery or the left circumflex branches, ischemia or infarction that can follow right coronary or circumflex disease can lead to PMD. This is in contrast to the anterolateral papillary muscle, which usually gets a dual blood supply and, hence, is less prone to injury during infarctions. Unlike patients in the medical group, the surgical patients had good left ventricular function. It appears that inferior infarction causes mitral regurgitation in patients with relatively good left ventricular function. On the other hand, there may be other reasons for MR when left ventricular function is severely impaired. Also, any disease that alters the spatial relationships between the various elements of the mitral valve apparatus can cause mitral insufficiency.

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**Figure 2.** The number of patients followed up for a period of time is plotted on the X axis and the Y survival on the Y axis. The difference in survival for the medical group is according to the EF (n1 ≥ 30, n2 < 30).

**Discussion**

Mitral regurgitation in CAD can occur acutely during a myocardial infarction or develop chronically due to ischemia-induced papillary muscle dysfunction (PMD). The latter seems to be the most common cause of MR in CAD. PMD has been noted in the acute MI and is usually a benign disorder. On the other hand, papillary muscle rupture with infarction usually leads to pulmonary edema and death unless recognized early and treated effectively, both medically and surgically.

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tient showed isolated anterior infarction in the surg-
ical group, which again probably is due to the dis-
tribution of blood supply to the papillary muscles.

The angiographic data revealed that many of our
surgical patients (92 percent) had an LVEF of
greater than 40 percent preoperatively. This is prob-
ably due to the selection bias before surgery. The
EF did not change significantly after surgery; how-
ever, we do not have the exercise radioisotope EFs
in these patients before and after surgery,
which might have revealed a difference. Eight of
our patients had triple-vessel involvement, four
double, and none single-vessel involvement. This
agrees with results of the study by Gahl et al.\(^5\) that
when MR occurs due to CAD, it is indeed a re-
flection of severe CAD.

The changes noted in the severity of the MR are
interesting in that 75 percent of the patients had
2+ or 3+ MR preoperatively, while only 42 percent
had 2+ or 3+ MR postoperatively. Only one pa-
tient had 3+ MR postoperatively, while three had
3+ MR preoperatively. One patient with 3+ MR
and inferior hypokinesia dramatically improved fol-
lowing CABG. He totally lost his MR and his in-
ferior hypokinesia became completely normal (Fig
3A,B). Another patient with anterior-inferior hypo-
kinesia lost his anterior hypokinesia, although his
level of MR remained at 2+.

Left ventricular pressure data in the surgical
group showed no change in the resting LVEDP.
The subtle changes in the LVEDP following ven-
triculogram were not significant.

The grafts were all patent in the 12 patients, and
hence any influence of graft closure on the severity
of regurgitation is not possible. In addition, we
analyzed other factors that might affect MR, such
as medications, the preload and afterload, and pro-
gression of disease in the native vessels distal to
grafting. Four of the 12 patients were receiving
long-acting nitrates (Nitropaste and isosorbide dini-
trate) preoperatively. One patient received Apre-
solone preoperatively. It might well be that the
preoperative filling pressures were deceptively nor-
mal or comparable to the postoperative values be-
cause of these medications. The intra-arterial blood
pressures prior to angiogram were virtually the same
preoperatively and postoperatively in each of these
patients, thereby eliminating the effect of afterload
on the severity of the MR. All patients had signif-
ican progression of disease in the native coronary
arteries proximal to the graft site, but there was no
distal disease noted.

It is possible that the MR in PMD can vary from
time to time. It is not uncommon for severe MR to
develop only during angina with significant regur-
tigation and V wave in wedge pressure tracing, which
disappears with the administration of nitrates. In
our patients, unfortunately, right heart pressure
measurements were not available in all of the pa-
tients, and in those available, no correlation be-
tween the V wave and severity of regurgitation
could be made when the preoperative and post-
operative data were compared. Provocation of
angina under proper circumstances might cause
transient significant MR with a prominent V wave.
The dynamic nature of papillary muscle function
has been well described.\(^6\)

The best way to diagnose MR due to CAD is
by left ventricular angiography. It allows one to
grade the severity of regurgitation in addition to
showing abnormal ventricular contraction. On the
other hand, cardiac catheterization is unreliable to
estimate chronic mitral insufficiency with left atrial
enlargement. Also, the form and magnitude of the
V wave appears to be a complex result of several
factors. Hence, the use of the ventriculogram is
probably the best means of assessing MR,\(^3\) as in
our group. Pre- and postoperative right heart cathe-
terization data, available for four patients, were in-

![Figure 3A (upper) and B (lower). Postoperative ventriculo-
gram following coronary artery bypass graft surgery. Note
mitral regurgitation completely disappeared, with good in-
ferior wall movement.](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/21288/ on 04/02/2017)
conclusive.

It is remarkable that many patients who were treated medically were dead on follow-up. It is conceivable that this is a reflection of the fact that they were not good surgical candidates. However, on analysis, it turns out that a good number of those who were dead had had severe triple-vessel disease (95 percent). Four of five patients (80 percent) who had 3+ MR died, and the EF showed a good correlation with mortality. Forty-five percent of the patients who died had EF less than 20 percent. The mean LVEDPs were consistently elevated in this group. The mean duration of survival between catheterization and death was 10.6 months (range, three days to 32 months). Thus, our results suggest that MR seen with CAD is usually associated with severe CAD and poor left ventricular function. The mortality in these patients is high, particularly when they are not suitable candidates for surgery.

The mortality in the medically treated group as a whole was exceedingly high (47 percent). Most of those patients who died had poor left ventricular function or severe mitral regurgitation. All but one had severe triple-vessel disease. The prognosis seems to be related to the above factors. Fourteen of the 20 patients who died had diffuse hypokinesia, four segmental hypokinesia, and two ventricular aneurysms. Thus, marked global hypokinesia seems to indicate a poor prognosis.

In conclusion, we believe that mitral regurgitation secondary to CAD is relatively rare and is usually seen with severe CAD and poor ejection fraction. Only a small proportion of patients probably have operable coronary arteries and moderate left ventricular function. Our experience in this small, select group suggests that adequate revascularization produces a dramatic improvement of symptoms and functional status. There seems to be a trend toward a decrease in the severity of the MR with CABG. One patient dramatically illustrated this point. The improvement in MR would be dramatic if indeed the MR were due to ischemia alone. If permanent structural alterations of the MV apparatus, such as scarring, rupture of the chordal structures, or global left ventricular dysfunction are responsible for MR, it is conceivable that the MR may not improve with CABG. Future studies are indicated for isolating the factors responsible in a given patient. This will make a significant contribution to management of such cases.

REFERENCES
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