Recurrent Stenosis of the Mitral Valve

By Jose R. de Jesus, Jr., M.D., Gerald M. Breneman, M.D., and John W. Keyes, M.D.

REPEAT mitral commissurotomy has become necessary when there is evidence of refusion of the mitral valve. Patients presenting with evidence of recurrent mitral stenosis have been classified by Belcher into two groups, i.e., "true" and "false" re-stenosis. In the true type, stenosis occurs after one or both commissures have been completely opened; false restenosis is that which occurs when neither commissure has been divided beyond the area of insertion of the papillary muscles. Whether or not a difference exists in the pathogenetic mechanism of these two types is not clear. Reactivation or a continued smoldering of the rheumatic process may cause restenosis of the diseased valve. Since a rigid valve is more likely to give trouble, division of both commissures, if at all possible beyond Brock’s critical area of tendon insertion, should restore the valve to maximum mobility and reduce the chances of restenosis.

Incidence

At the Henry Ford Hospital Symposium on Cardiovascular Surgery in 1955, Brock reported four cases of restenosis in a series of 350; Harken reported 10 in 800 patients, and Keyes and Lam reported one in 180 commissurotomies. Bailey et al. in 1957, reported 22 patients with restenosis; Patterson and Marshall in 1959 reported seven cases, and Belcher in 1960 reported 46 patients with restenosis. Single cases had been reported previously.

Likoff and Uricchio in a study including 200 patients who are living 5 or more years after commissurotomy estimated the rate of restenosis as about 5 per cent. Recurrence of stenosis at Brompton Hospital in England is 2 per cent per annum (Wood).10

Material for Study

Since the first operation for mitral stenosis in this hospital in March 1950, 672 mitral commissurotomies have been done up to January 1960. Of this group, 35 patients plus eight patients who had their primary operation done in another hospital comprise the subjects of this report. The entire series of 43 patients was reoperated upon for suspected restenosis of the mitral valve. In 40 cases, restenosis was proved at operation or autopsy. There was no demonstrable restenosis at operation in three cases.

Clinical Data

The ages of the patients at the time of reoperation for restenosis ranged from 23 to 52 years (table 1). Seventy-two per cent of the series were in the fourth and fifth decades. There were 29 females and 14 males. Of the females, five experienced incapacitating symptoms for the second time when they became pregnant following the initial operation. The average interval between operations in all patients was 4 years, with a range of 1 to 8 years. One patient reoperated upon 3 months following primary commissurotomy was found to have no restenosis.

A definite past history of rheumatic fever was obtained from 28 (65 per cent) cases; two (5 per cent) cases had scarlet fever; and 13 (30 per cent) could not recall any history of either. Recurrence of active rheumatic fever is difficult to detect among these patients after their first operation. Postcommisurotomy syndrome occurred in five cases (Nos. 2, 30, 31, 39, and 43) after the first operation. One case developed subacute bacterial endocarditis a year after the primary commissurotomy.

All patients on readmission for restenosis had exertional dyspnea and decreased effort tolerance, and were unable to carry on their usual activities. Recurrent symptoms were present several months to 2 years prior to the repeat commissurotomy. Patients were classified as to functional capacity as follows: class II, 11 cases (26 per cent); class III, 27 cases (63 per cent); class IV, 5 cases (11 per cent); the latter were improved by intensive medical care before operation.

As in primary stenosis, refusion of the mitral
Clinical Data at Second Operation

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RVH, right ventricular hypertrophy; AF, atrial fibrillation.
valve is characterized by the usual physical findings. In some cases, however, signs of advanced mitral valvular disease were evident that were not present at the original operation, such as the murmurs of pulmonary valvular insufficiency (Graham Steell), tricuspid insufficiency, increased cardiac size, and evidence of right-sided congestive heart failure.

The presence of an apical systolic murmur, particularly if high-pitched, blowing in character, holosystolic, and well referred to the left axilla, in addition to the diastolic murmur of mitral stenosis, usually suggests a combined mitral lesion and, hence, requires further evaluation. There were 17 cases in this series that presented such a problem, the intensity of the murmur being graded as II to III, on a grade I to VI basis.

Significant apical systolic murmurs are usually heard in patients with a subvalvular component to the stenosis. Involvement and shortening of the chordae tendineae produce a funnel-shaped configuration of the mitral cusps to account for regurgitation. There were 11 patients in this study with such valvular deformity at the time of surgery.

Normal sinus rhythm was present in 19 patients; paroxysmal atrial fibrillation in eight, and established chronic atrial fibrillation in the remaining 16. The electrocardiogram showed a pattern consistent with right ventricular hypertrophy in 13 cases or 30 per cent. One patient had electrocardiographic evidence of biventricular hypertrophy.

Cardiac fluoroscopy and four views of the chest with barium in the esophagus showed right ventricular hypertrophy in 68 per cent of the cases. In all instances, left atrial enlargement and prominence of the pulmonary artery were noted. The majority showed Kerley's lines and prominence of the upper lobe veins. Mitral valvular calcification was present in 47 per cent of the cases. In several patients, serial chest x-rays showed a progressive increase in heart size prior to the second operation.

Left heart catheterization was carried out in 17 patients; in 14 to assess the degree of mitral insufficiency because of an apical systolic murmur. Of these 14, nine had a 1 plus mitral insufficiency effect by Fisher's classification and two were graded as 2 plus, while the tracing of the remaining patient was inadequate for the assessment of the presence or absence of mitral insufficiency. All these patients had a diastolic gradient indicating a significant degree of mitral stenosis. Case 42 suffered an acute coronary occlusion with posterolateral myocardial infarction 17 months after the initial operation, and although 6 months later she developed the symptomatology of seeming progression of mitral impendence, it was neces-
in width. There was extreme sclerotic thick-
euning with irregular calcification of the cusps.
The other (case 8) did well for 1½ years, when
she became pregnant; had a recurrence of con-
gestive heart failure, and died in another
hospital in the seventh month of pregnancy.

The case with which the valve restenoses
is for the most part due to inadequate open-
ing at the time of the first operation. How
good an opening is obtained in the initial
commissurotomy is not altogether a matter
of technic of the surgeon but for the most
part stems from the factors encountered dur-
ing the operation, such as deformity and
calcification. These are the cases referred
to as false restenosis.

The clinician must be alert to the possi-
bility of recurrent attacks of rheumatic
activity. Its occurrence after primary com-
missurotomy is one of the probable causes of
true stenosis.

Mortality

There were 13 deaths in this series of 43
patients; 10 occurred in the immediate post-
operative period, an operative mortality of
23 per cent. There were three late deaths,
two due to recurrent congestive heart failure
following periods of marked improvement of
11 and 20 months, respectively, and the third
(case 32) reported above.

Atrial fibrillation appears to have played
a significant role in mortality. Ten of the 13
patients had chronic atrial fibrillation. One
death occurred in a patient who fibrillated
paroxysmally. Two of the chronic fibrillators
died of cerebral embolism with pulmonary
edema.

Two cases died of pulmonary edema alone,
one of which (case 23) was the only instance
in which the pre-existing mitral regurgita-
tion was aggravated and contributed to the
patient’s demise. Death from arrhythmia
occurred in four cases; one of them from
ventricular fibrillation and the other three
from cardiac arrest at the time of surgery.
Another patient died of excessive blood loss
during operation. One patient died of mas-
sive pulmonary embolism on the fourth post-
operative day.

Discussion

It has been our feeling that when there
are obvious signs of mitral restenosis, a pa-
tient should undergo a second operation. In
doubtful cases of restenosis, left atrial cath-
terization is indicated. The three patients in
this series who failed to show restenosis
at the time of the second operation had not
had prior cardiac catheterization. Catheter
studies are especially indicated in certain
cases when myocardial rather than valvular
factors are considered as possible causes of
the patient’s symptoms. Catheterization is
not deemed necessary when the clinical fea-
tures are apparently typical.

The probable causes of the true type of
restenosis, where there has been adequate
and complete opening of one or both com-
missures at the original operation, may be
the following: spontaneous refusion of the
valve edges opened by commissurotomy, en-
couraged by less than ideal physiologic
excursion without attendant specific inflam-
matory response; recurrent acute attacks of
rheumatic fever after the primary operation;
presence of a smoldering rheumatic process
during or after the first operation, and the
questionable role of postcommissurotomy syn-
drome as an entity related to rheumatic
reactivation.

In general, operation for patients below
20 years of age is rarely indicated because
congestive heart failure in these young pa-
tients is frequently due to active rheumatic
carditis rather than to mitral obstruction.
For this reason, the probability of restenosis
may be greater among patients who during
primary commissurotomy have a smoldering
rheumatic process.

As in primary stenosis of the mitral valve,
the incidence of true restenosis can be low-
ered by instituting proper prophylaxis,
prompt recognition and treatment of acute
rheumatic fever and subacute bacterial
endocarditis.

It will be noted that the subject of case
30 suffered not only from reactivation of rheumatic fever but also from postcommisurotomy syndrome. The relationship of postcommisurotomy syndrome to rheumatic fever remains controversial despite voluminous literature on the subject. Mention is made of the fact that five cases (nos. 2, 30, 31, 39, and 43) in this series developed this syndrome after their first operation. These patients presented with pleuritic chest pains, fever without chills, general malaise, and diminution in effort tolerance. The syndrome occurred at an interval of from 11 days to 20 months following the initial operation and antedated the recurrence of symptoms due to restenosis by an interval of 6 to 24 months. In three of them, prompt response to the use of steroids was observed. In the other two cases symptoms subsided with conservative management.

In contrast to the true restenosis, the occurrence of false restenosis can be predicted with relative certainty. This is based on inherent valvular factors such as calcification and deformity and the adequacy of the opening established at the original operation. Phonocardiography may be valuable in demonstrating objectively the adequacy of the surgical intervention. It is hoped that the incidence of false restenosis will be much less with improved surgical techniques and the use of open-heart surgery in selected cases.

Inadequately mobilized leaflets with limited excursion obviously are more predisposed to restenose. Moreover, if subvalvular changes are present with fused chordae, poor function of the valve rather than real stenosis may account for the symptoms. There were seven cases that had recurrent stenosis with a subvalvular component. Patients who survived the second operation, experienced subjective improvement despite incomplete commissural splitting. Apparently, the most trivial increase in valve size is often sufficient to ameliorate the severe pre-existing pathologic hemodynamics. This observation also holds for primary commisurotomy. Usually, most operations are limited to splitting of the anterolateral commissure. In a great many instances, the knife cannot be placed safely to cut the medial commissure because of the risk of producing mitral regurgitation. Open-heart surgery may significantly improve the results in those individuals who require operation for restenosis, particularly those having a severely deformed or calcified valve and those with a history of thromboembolism. This is true also of cases complicated by a significant degree of mitral incompetency. A greater number of these cases are now being considered by us for open-heart valvuloplasty. Only four cases in this series were subjected to open-heart surgery.

The histopathology of the amputated atrial appendages from primary operation of five cases showed no evidence of inflammatory changes, except in one (case 3) in which there was mild pericarditis and myocarditis. The material is insufficient to make definite conclusions regarding relationship of Aschoff nodules in amputated appendages to restenosis of the mitral valve.

Summary

Forty-three patients with suspected restenosis of the mitral valve are reported. Forty of these occurred in a series of 672 mitral commissurotomies. A significant degree of recurrent stenosis was found in 40 of the 43 cases at the time of the second operation. The clinical features and findings in these patients are discussed, as well as the probable etiologic factors. Left heart catheterization is indicated in many of these cases to define precisely the hemodynamic status, particularly in those individuals with suspected concomitant mitral incompetency or myocardial disease. Open-heart surgery should significantly improve the results of a repeat mitral commissurotomy.

References

2. Brock, R. C.: The surgical and pathological

The Circulation of Water

There are a number of causes which bring about ocean currents. In the tropics high temperature causes a far greater evaporation of water than can be offset by rainfall and the flow of rivers; near the poles this relation is reversed. Hence water must steadily flow from high to low latitudes, there to evaporate and complete the cycle in the atmosphere and on the land. In polar regions the cold water sinks and penetrates along the bottom of the sea in great deep currents to the tropics. The surface currents of the ocean have a different origin, for they depend upon winds, especially trade winds, etc. Beside the rotation of the earth, the eccentricity of its orbit, and the inclination of its axis, the thermal properties of water enter as fundamentally important factors.

The magnitude and the extent of the movements which result from such influences are very considerable. Of all ocean currents, the Gulf Stream, a branch of the northern equatorial current, has been most carefully studied. Its maximum velocity is 220 kilometers per day, greater therefore than that of the Rhine at Coblenz; the mean about 134 kilometers a day. In the Straits of Yucatan the Gulf Stream carries 0.2 cubic kilometer (200,000,000 tons) per second. If all this water were to be cooled to the temperature of the polar ocean this would be equivalent to the transport of about 5,000,000,000,000,000,000 gram calories per second. In this manner vast quantities of water, carrying enormous stores of heat, are constantly in motion all over the globe.—Lawrence J. Henderson. The Fitness of the Environment. New York, The Macmillan Co., 1924, p. 180.
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