Anterior Chordal Transection Impairs Not Only Regional Left Ventricular Function But Also Regional Right Ventricular Function in Mitral Regurgitation

Thierry Le Tourneau, MD; Daniel Grandmougin, MD; Claude Foucher, MD; Eugene P. McFadden, MRCP; Pascal de Groote, MD; Alain Prat, MD; Henri Warembourg, MD; Ghislaine Deklunder, MD, PhD

Background—Preservation of annuloventricular continuity through the chordae tendinae aims to maintain left ventricular (LV) function and thus improve postoperative prognosis. This study was designed to prospectively investigate the effect of anterior chordal transection on global and regional LV and right ventricular (RV) function in mitral regurgitation (MR).

Methods and Results—Sixty-five patients with severe MR underwent radionuclide angiography before and after either mitral valve (MV) repair (42 patients) or replacement with anterior chordal transection (23 patients). LV and RV ejection fractions (EF) were determined at rest. Both ventricles were divided into 9 regions to analyze regional EF and the effect of anteromedial translation related to surgery. After surgery there was a significant decrease in LVEF (P=0.038) and an increase in RVEF (P=0.036). However, LVEF did not change after MV repair (63.8±9.9% to 62.6±10.3%), whereas RVEF improved (40.7±10.1% to 44.5±8.1%, P=0.027). In contrast, LVEF decreased after MV replacement (61.7±10.1% to 57.2±9.9%, P=0.03), and RVEF was unchanged (40.9±10.9% to 41.3±9.1%). LVEF 4 and 5, in the area of anterior papillary muscle insertion, were impaired after MV replacement compared with MV repair (region 4, 77.6±16.7% versus 87.7±10.8%, P=0.005, and region 5, 73.9±19.3% versus 89.9±13.1%, P<0.001). Moreover, anterior chordal transection led to a significant impairment in the apicoseptal region of the RV (RVEF 4, 50.3±15.6% versus 59.3±13.8%, P=0.02).

Conclusions—Anterior chordal transection during MV replacement for MR impair not only regional LV function but also regional RV function. (Circulation. 2001;104[suppl I]:I-41-I-46.)

Key Words: mitral valve ▪ surgery ▪ ventricles ▪ myocardial contraction

In patients with mitral regurgitation (MR), mitral valve (MV) replacement with transection of the entire subvalvular apparatus usually results in a change in left ventricular (LV) geometry and in depressed LV systolic performance with regional myocardial contractile abnormalities.1–5 Preservation of the MV apparatus during MV surgery for MR, particularly with MV repair, improves postoperative LV function.3,6–10 Although several studies demonstrated better results in MV replacement with subvalvular preservation,3,4,6,7,10 these techniques are not in widespread routine use and are not always feasible. Whereas the effect of chordal transection on LV function is well documented, little is known concerning right ventricular (RV) function in this setting. Mitral valve repair and MV replacement with anterior chordal transection but posterior chordal preservation are techniques currently used in our institution, providing the opportunity to study the effect of anterior chordal transection on LV and RV function.

Therefore this prospective, nonrandomized study was designed to evaluate the effect of anterior chordal transection on regional and global LV and RV function in non ischemic MR, with the use of preoperative and postoperative radionuclide angiography.

Methods

Study Patients

After informed consent was obtained, we prospectively enrolled (between 1994 and 1997) 80 consecutive patients with moderately severe or severe pure nonischemic MR before MV surgery. Patients with a history of myocardial infarction or angina, significant coronary disease on preoperative coronary angiography, previous thoracic surgery, or other significant valve disease apart from tricuspid regurgitation were not included in this study. Ten patients were withdrawn after the operation because they refused the postoperative study, 4 patients died before this study, and 1 patient with recurrent MR was excluded from the postoperative study. Thus 65 patients completed the study. The study protocol was approved by the Research Ethics Committee of the University.

From the Service d’Explorations Fonctionnelles Cardio-vasculaires (T.L.T., E.P.M., G.D.) and the Services de Chirurgie Cardio-vasculaire (D.G., A.P., H.W.), Hôpital Cardiologique, the Service de Médecine Nucléaire (C.F.), Hôpital Roger Salengro, and the Service de Cardiologie C (P.d.G.), Hôpital Cardiologique, Centre Hospitalier Régional et Universitaire, Lille, France.

Correspondence to Dr Thierry Le Tourneau, Service d’Explorations Cardio-Vasculaires, Hôpital Cardiologique, CHRU, Boulevard du Pr. J. Leclerc, 59037 Lille Cedex, France. E-mail thletourneau@yahoo.fr

© 2001 American Heart Association, Inc.

Circulation is available at http://www.circulationaha.org
Surgery
The decision regarding the type of corrective surgery was made by the cardiovascular surgeon on the basis of preoperative data and after assessment of the anatomic status of the MV during surgery. Twenty-three patients (35%) underwent MV replacement with posterior chordal preservation and anterior chordal resection (resection of chordae inserting on the anterior leaflet). St Jude prosthetic valves were used in 17 patients, ATS medical prosthetic valve in 1 patient, Sorin Bicarbon prosthetic valve in 1 patient, and Carpentier-Edwards bioprostheses in the remaining 4 patients. Forty-two patients (65%) underwent MV repair using the technique described by Carpentier11 with placement of a rigid ring (Carpentier’s ring) on the mitral annulus. No patient had significant residual MR after surgery. Associated procedures included 2 tricuspid annulus implantations and 1 carotid endarterectomy in the replacement group and one Maze operation in each group.

Radionuclide Angiography
All patients underwent radionuclide angiography before and 10 months (mean, 301 days) after surgery. Radionuclide angiography was performed at rest in the supine position using red blood cells labeled in vivo with 20 mCi of technetium-99m. Data were acquired in a 45° and a 70° left anterior oblique view by means of a gamma camera (DST camera, Sopha Medical, data processing Sophy, SMV International). All studies were formatted at 16 frames per cardiac cycle. R-R intervals and heart rate (beats/min) were recorded. Cardiac cycles with R-R intervals that were not within 20% of the average value were discarded. LV and RV ejection fractions (EF) were determined by means of the equilibrium technique by automated detection of end-diastolic and end-systolic contours, with manual correction if necessary. Both the LV and the RV were divided into 9 regions to analyze regional EF in the 45° left anterior oblique view (Figure 1). The LV was divided into 9 regions: regions 1 and 9 corresponded to the base of heart; regions 2 and 3 to the posterolateral region; regions 4, 5, and 6, respectively, to the lateral, apical, and anteroapical region; and regions 7 and 8 to the anteroseptal and septal regions. The RV was also divided into 9 regions: regions 1 and 2 corresponded to the base of heart; region 3 to the septal region; region 4 to the apicoventricular region; regions 5 and 6 to the anteroapical region; and regions 7, 8, and 9 to the free wall of the RV. All the radionuclide angiograms were interpreted by the same experienced investigator, who was not aware of the type of surgery performed.

Doppler Echocardiography
All patients underwent preoperative and postoperative echocardiography on the same day as radionuclide angiogram. All 2D echocardiographic and Doppler examinations were performed by experienced investigators. Systolic pulmonary artery pressure was estimated from the maximal tricuspid regurgitation velocity.

Follow-Up
Follow-up information was obtained by phone contact with patients, their family physician, or cardiologist in March 2001.

Statistical Analysis
Results are expressed as mean±SD. Comparisons between groups were performed with χ² tests or with paired or unpaired Student’s t tests, as appropriate. Event-free actuarial survival rates were calculated by the Kaplan-Meier method. The log rank test was used to compare actuarial events. A value of P<0.05 was considered statistically significant.

Results
Clinical Data
Eleven patients were in New York Heart Association functional class I (17%), 30 in NYHA functional class II (46%), 23 in NYHA functional class III (35%), and 1 (2%) in NYHA functional class IV before surgery. Patients were more symptomatic before (mean NYHA functional class, 2.22±0.74) than after operation (1.34±0.51, P<0.001). Sixteen patients were in atrial fibrillation before surgery and 9 patients after surgery.

Table 1 gives the baseline characteristics of both groups. Patients who underwent valve replacement were older than those who underwent valve repair and were more symptom-

<table>
<thead>
<tr>
<th>Table 1. Preoperative Clinical Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mitral Valve Replacement</td>
</tr>
<tr>
<td>-------------------------------</td>
</tr>
<tr>
<td>n</td>
</tr>
<tr>
<td>Age, y</td>
</tr>
<tr>
<td>Sex, M/F</td>
</tr>
<tr>
<td>Rhythm</td>
</tr>
<tr>
<td>Sinus</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
</tr>
<tr>
<td>NYHA functional class</td>
</tr>
<tr>
<td>I/IV</td>
</tr>
<tr>
<td>III/V</td>
</tr>
<tr>
<td>Cause</td>
</tr>
<tr>
<td>Degenerative</td>
</tr>
<tr>
<td>Rheumatic fever</td>
</tr>
<tr>
<td>Endocarditis</td>
</tr>
</tbody>
</table>

Data presented are mean±SD or number of patients.
Effect of Anterior Chordal Transection on Regional Function

Before surgery, there were no differences in LV or RV regional EF between the 2 groups. However, regional LVEF significantly decreased after surgery (repair, 61.7±10.1% to 63.8±9.9%; P<0.036). MV repair was associated with a significant decrease in LVEF 5 to 9 (anteroapical and septal area), related to anteromedial cardiac translation. On the contrary, analysis of regional RVEF revealed a significant increase in regional EF 1, 2, 3, and 4 (septal and apicoseptal areas) and a significant decrease in regional EF 5, 6, and 8 (anterolateral area).

Effect of Anterior Chordal Transection on Regional Function

After surgery, there was a significant increase in regional LVEF 2 and 3 (posteriorateral area) and a significant decrease in regional LVEF 5 to 9 (anteroapical and septal area), related to anteromedial cardiac translation. On the contrary, analysis of regional RVEF revealed a significant increase in regional EF 1, 2, 3, and 4 (septal and apicoseptal areas) and a significant decrease in regional EF 5, 6, and 8 (anterolateral area).

Table 2. Results of Preoperative and Postoperative Radionuclide Angiography

<table>
<thead>
<tr>
<th></th>
<th>All Patients</th>
<th>Replacement</th>
<th>Repair</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preoperative LVEF, %</td>
<td>63.1±9.9</td>
<td>61.7±10.1</td>
<td>63.8±9.9</td>
</tr>
<tr>
<td>Postoperative LVEF, %</td>
<td>60.6±10.5*</td>
<td>57.2±9.9†</td>
<td>62.6±10.3†</td>
</tr>
<tr>
<td>Preoperative RVEF, %</td>
<td>40.8±10.3</td>
<td>40.9±10.9</td>
<td>40.7±10.1</td>
</tr>
<tr>
<td>Postoperative RVEF, %</td>
<td>43.2±8.6*</td>
<td>41.3±9.1</td>
<td>44.5±8.1*</td>
</tr>
</tbody>
</table>

Values are mean±SD. *P<0.05 postoperative vs preoperative; †P<0.05 replacement vs repair.

Cardiac Translation

After MV surgery, there was a significant increase in mean NYHA class after operation (repair, 1.31±0.47 versus replacement 1.41±0.59, P=0.59). Degenerative disease was the most frequent cause of MR in the repair group, whereas rheumatic disease was more frequent in the replacement group. Aortic cross-clamp time and total cardiopulmonary bypass time were 96±32 minutes and 116±35 minutes, respectively, in the repair group compared with 72±41 minutes (P=0.01) and 80±43 minutes (P<0.01), respectively, in the replacement group.

Systolic and mean arterial pressure were measured in 40 of 65 patients before and after operation. There were no significant differences in mean and systolic arterial pressures before surgery, but these differences were not significant. There was no significant difference in mean NYHA class after operation (repair, 1.31±0.47 versus replacement 1.41±0.59, P=0.59). Degenerative disease was the most frequent cause of MR in the repair group, whereas rheumatic disease was more frequent in the replacement group. Aortic cross-clamp time and total cardiopulmonary bypass time were 96±32 minutes and 116±35 minutes, respectively, in the repair group compared with 72±41 minutes (P=0.01) and 80±43 minutes (P<0.01), respectively, in the replacement group.

Systolic and mean arterial pressure were measured in 40 of 65 patients before and after operation. There were no significant differences in mean and systolic arterial pressures before (repair, 100±11 and 129±19 mm Hg, versus replacement, 102±10 and 133±21 mm Hg) or after the operation (repair, 101±13 and 131±22 mm Hg, versus replacement, 103±15 and 135±21 mm Hg) between the 2 groups.

Radionuclide Angiography

Global LV and RV Function

Baseline RV and LV function were similar in the two patient groups (Table 2). Ten months after surgery, LVEF decreased significantly from 63.1±9.9% to 60.6±10.5% (P=0.038). Patients who underwent MV replacement had a significant decrease in LVEF (61.7±10.1% to 57.2±9.9%, P=0.027), whereas LVEF did not change significantly (63.8±9.9% to 62.6±10.3%) after MV repair. Moreover, LVEF differed significantly between the two groups after surgery (replacement, 57.2±9.9% versus repair, 62.6±10.3%, P=0.045).

For all patients, RVEF increased from 40.8±10.3% to 43.2±8.6% (P=0.036). MV repair was associated with a significant increase in RVEF (40.7±10.1% to 44.5±8.1%, P=0.027). In contrast, RVEF was unchanged after MV replacement (40.9±10.9% to 41.3±9.1%).

Doppler Echocardiography

The major results of the echocardiographic and Doppler examinations are summarized in Table 3. No patient had significant postoperative MR. Fifteen patients had grade 1/
residual mitral regurgitation after MV repair and 3 patients grade 2/4. Left atrial diameter was slightly higher in the replacement group before surgery. Mitral valve surgery was associated with a significant decrease in left atrial and LV end-diastolic diameters and in systolic pulmonary artery pressure, whereas LV end-systolic diameter was unchanged. Systolic pulmonary artery pressure remained modestly increased after MV replacement.

Follow-Up
For the 65 patients who completed the study, mean follow-up was 4.7 ± 1.8 years after operation, and total follow-up was 307 patient-years; 2 patients were lost to follow-up. The overall death rate was 14% (6 of 42 patients) in the MV repair group and 22% (5 of 23 patients, P = 0.5) in the MV replacement group at the time of follow-up. Overall, for 80 patients who were studied before operation, the actuarial survival rate including early death (4%) was 79.3% (repair) and 70.6% (replacement, P = 0.36) after 6 years.

Discussion
The aim of this prospective study was to assess the effect of anterior chordal transection on global and principally on regional LV and RV function during MV surgery for severe nonischemic MR. The effect of cardiac translation on the LV and the RV was analyzed. In agreement with previous studies,3,5,7–10 MV repair led to better preservation of cardiac function than did MV replacement with anterior chordal transection. Anterior chordal transection resulted in a significant impairment of global and regional LVEF in the area of papillary muscle insertion (apicolateral area). However, the most important finding of this study was the deleterious effect of anterior chordal transection on global and regional RV function, with a significant decrease in RV septoapical function, remote from the insertion of the anterior papillary muscle.

Cardiac Translation
Abnormal septal motion occurs in 42% to 91% of patients after cardiopulmonary bypass.12 Cardiopulmonary bypass is known to induce anteromedial translation of the heart, resulting in apparent LV septal hypokinesis and apparent postero-lateral hyperkinesis.13,14 Although cardiopulmonary bypass appears to be an obligatory step in the genesis of cardiac translation, this phenomenon may be due to cardiopulmonary bypass itself, to myocardial preservation techniques, or to surgical manipulations that occur during this time period.13,15 The effect of anteromedial translation on the RV has not previously been reported. We demonstrated in this study an apparent RV septoapical hyperkinesis associated with an apparent anterolateral hypokinesis occurring after MV surgery.

Left Ventricular Ejection Fraction
In MR, postoperative LV function is principally determined by both the preoperative ventricular function and the postoperative increase in afterload. Chronic MR results in a progressive deterioration in LV contractile function,16,17 although the LVEF is maintained over a relatively long period. After surgery, myocardial contractility recovers in most cases within 1 year after surgery.17 Afterload or systolic wall stress is increased in MR because of LV dilation. Adaptation of the LV to the abrupt postoperative increase in afterload depends on the integrity of the mitral subvalvular apparatus.9,10 In our study, mean LVEF decreased after surgical correction of MR in the entire cohort of patients. However, separate analysis of both groups demonstrated that LVEF did not significantly decrease after MV repair, whereas it did decrease after MV replacement with anterior chordal transection. This result is consistent with previous studies, which indicates that depression of LVEF after MV surgery is related to chordal transection3–7,10 rather than to alterations in work load or to myocardial systolic dysfunction. Partial or total subvalvular apparatus resection induces significant changes in the geometry of the LV,9,10,18 an increase in end-systolic wall stress,9,10 an impairment of diastolic and systolic LV function,3,9,10 regional abnormal motion,1–5,8 and perturbation of regional systolic torsional deformation.19 On the other hand, Okita et al20 showed that chordal preservation, by maintaining ventricular shape and decreasing LV volume, was associated with a postoperative decrease in systolic wall stress. Moreover, preservation of the MV apparatus during mitral valve surgery for MR maintains LV function and thus improves survival.20

Effect of Anterior Chordal Transection on LV Regional Function
The heart is a complex muscle structure with a continuous change in myocardial fiber orientation from a left-handed helix in the subepicardium to a right-handed helix in the subendocardium. Basal epicardial fibers descend vertically to

---

**TABLE 3. Results of Preoperative and Postoperative Echocardiography and Doppler Examinations**

<table>
<thead>
<tr>
<th></th>
<th>Replacement</th>
<th>Repair</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>LAD, mm</td>
<td>51 ± 5†</td>
<td>43 ± 8*</td>
</tr>
<tr>
<td>LVEDD, mm</td>
<td>57 ± 7</td>
<td>51 ± 5†</td>
</tr>
<tr>
<td>LVEDS, mm</td>
<td>36 ± 6</td>
<td>36 ± 6</td>
</tr>
<tr>
<td>Syst PAP, mm Hg</td>
<td>49 ± 13</td>
<td>35 ± 6‡</td>
</tr>
</tbody>
</table>

Values are mean ± SD. LAD indicates left atrial diameter; LVEDD, left ventricular end-diastolic diameter; LVEDS, left ventricular end-systolic diameter; Syst PAP, systolic pulmonary artery pressure.

*P < 0.05 after vs before surgery; †P < 0.005 after vs before surgery; ‡P < 0.05 repair vs replacement.
the apex and penetrate inward to form the papillary fibers. Papillary muscles contract during systole, contributing to ejection by drawing the mitral ring toward the apex, thereby causing shortening of the long axis and sphericity of the chamber. After chordal resection, papillary muscles can no longer assist contraction at their insertion sites or in contiguous areas. Moreover, resection of the chordae tendinae may disrupt the muscle bundle alignment and induce contractile abnormalities remote from the area of papillary muscle insertion. In a canine model, anterior chordal transection had a considerably more deleterious effect on LV systolic function than section of the chordae to the posterior leaflet. This fact may be explained by the concept of regional afterload distribution. The anterior leaflet is larger, and development of tension in the chordae to this leaflet should be greater at a given LV pressure. Indeed, despite preservation of the posterior chordae tendinae, anterior chordal transection during MV replacement in our study was associated with a significant impairment of LV regional function in the area of anterior papillary muscle insertion (apicolateral area). This phenomenon appears to be related to anterior chordal transection, as concomitant ischemic heart disease was excluded on coronary angiography before enrollment of patients. Although several studies have demonstrated the deleterious effect of the resection of the subvalvular apparatus on global LV function, regional abnormal motion, particularly in the area of ventricular chordal insertion, has been reported in a few human studies. Goldman et al demonstrated intraoperative echocardiography that myocardial contractile abnormalities in the anterior and posterior septum develop immediately after removal of the papillary muscles. Corin et al found regional dysfunction in the area of posterior papillary muscle insertion after MV replacement with resection of the subvalvular apparatus. In their study, assessment of LV function was performed in the right oblique anterior view, which did not allow the authors to evaluate the area of anterior papillary muscle insertion. On the opposite, we evaluate LV function only in the 45° anterior oblique view, so it is possible that changes in other regional EF as the inferior wall were not detected.

**Right Ventricular Function**

Right ventricular function has been poorly studied in the setting of MR. Right ventricular EF is depressed before surgery in severe MR. The impairment of RV performance is significantly influenced by pulmonary artery pressure, and it has been found that RVEF was inversely related to pulmonary artery pressure in MR. Although Borer et al reported a rapid improvement of RVEF after valve surgery, RV function improved after MV repair but remained unchanged after MV replacement in our study. Echocardiographic and Doppler examination demonstrated higher postoperative levels of systolic pulmonary artery pressure in the replacement group compared with the repair group (35±6 versus 31±6 mm Hg, P<0.05). Although systolic pulmonary artery pressure is only slightly increased after MV replacement, this difference may explain in part the lack of improvement in RVEF. However, the main finding of this study was that anterior chordal transection induces significant impairment of regional function in the apicoseptal area of the RV. These postoperative regional contractile abnormalities may participate in the observed impairment of RVEF after MV replacement. Explanation of regional RV function impairment is not easy. After anterior chordal transection, chamber shape is modified with dilation of the LV. This geometric alteration might have directly influenced regional RV function in the septoapical area. Another explanation lies in the complex architectural structure of the heart, with a continuity between mitral annulus, myocardial fibers, papillary fibers, chordae tendinae, and mitral leaflet. Section of the mitral subvalvular apparatus is followed by a decrease in LV contractility, an increase in regional afterload, and a decrease in the preload of fibers along the circumference of the heart. These important modifications may induce a RV myocardial fiber desequilibrium predominant in the apicoseptal area of the RV.

**Study Limitations**

The lack of randomization between the two types of surgery is the main limitation of this study. The decision regarding the choice of corrective surgery was made by the cardiovascular surgeon on the basis of preoperative data and after assessment of the anatomic status of the MV during surgery. However, because it is well established that MV repair is the operation of choice for MV disease, a randomized, controlled trial may no longer be considered ethical. Moreover, the patients in the two groups were similar with respect to preoperative clinical characteristics, and radionuclide angiographic and echocardiographic Doppler variables. There were only slight differences between the two groups concerning age, cause of MR, and functional capacity assessed with the NYHA classification. Moreover, reanalysis of data after exclusion of younger patients (<45 years old) in the repair (7 patients) and the replacement (1 patient) group was performed to exclude the possibility that age differences might account for the observed results. Another limitation of our study is the lack of assessment of global and regional systolic stress, which is increased after chordal transection. However, LV afterload approximated by systolic or mean arterial pressure was not different between the 2 groups before or after surgery, whereas systolic pulmonary artery pressure was only slightly higher after MV replacement compared with repair. Differences in systolic stress might explain in part the effect of chordal transection on ventricular function. Finally, it is possible that changes in regional LV function apart from the anteroapical area were not detected, as LV regional EF was analyzed only in the 45° left anterior oblique view.

**Conclusions**

Preservation of the annulolventricular continuity through the posterior chordae tendinae with anterior chordal transection was associated with a significant impairment of regional and global LV function. Moreover, anterior chordal transection was associated with significant impairment of the apicoseptal region of the RV, which may have contributed in part to the lack of improvement in RV function after MV replacement. Although MV repair is not always feasible, particular effort...
should be made to preserve anterior and posterior chordae tendinae attachments when MV replacement is necessary.

References


Anterior Chordal Transection Impairs Not Only Regional Left Ventricular Function But Also Regional Right Ventricular Function in Mitral Regurgitation
Thierry Le Tourneau, Daniel Grandmougin, Claude Foucher, Eugene P. McFadden, Pascal de Groote, Alain Prat, Henri Warembourg and Ghislaine Deklunder

_Circulation_. 2001;104:I-41-I-46
doi: 10.1161/hc37t1.094602
_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2001 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:

http://circ.ahajournals.org/content/104/suppl_1/I-41