Chordal Cutting
A New Therapeutic Approach for Ischemic Mitral Regurgitation

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Background—Mitral regurgitation (MR) conveys adverse prognosis in ischemic heart disease. Because such MR is related to increased leaflet tethering by displaced attachments to the papillary muscles (PMs), it is incompletely treated by annular reduction. We therefore addressed the hypothesis that such MR can be reduced by cutting a limited number of critically positioned chordae to the leaflet base that most restrict closure but are not required to prevent prolapse. This was tested in 8 mitral valves: a porcine in vitro pilot with PM displacement and 7 sheep with acute inferobasal infarcts studied in vivo with three-dimensional (3D) echo to quantify MR in relation to 3D valve geometry.

Methods and Results—In all 8 valves, PM displacement restricted leaflet closure, with anterior leaflet angulation at the basal chord insertion, and mild-to-moderate MR. Cutting the 2 central basal chordae reversed this without prolapse. In vivo, MR increased from 0.8±0.2 to 7.1±0.5 mL/beat after infarction and then decreased to 0.9±0.1 mL/beat with chordal cutting (P<0.0001); this paralleled changes in the 3D leaflet area required to cover the orifice as dictated by chordal tethering (r²=0.76).

Conclusions—Cutting a minimum number of basal chordae can improve coaptation and reduce ischemic MR. Such an approach also suggests the potential for future minimally invasive implementation. (Circulation. 2001;104:1958-1963.)

Key Words: mitral valve □ regurgitation □ remodeling □ myocardial infarction □ echocardiography

Mitral regurgitation (MR) is a common complication of ischemic heart disease that conveys adverse prognosis after both myocardial infarction and coronary revascularization, more than doubling the risk of late death.1-4 Extensive work has confirmed the relation of ischemic MR to remodeling and distortion of the ischemic left ventricle (LV) and the papillary muscles (PMs) to which the mitral leaflets are attached.5-11 Displacement of the PMs away from the mitral annulus tethers the leaflets into the LV and restricts their ability to close effectively at the level of the annulus. This problem is compounded by LV contractile dysfunction, which decreases the force available to close the leaflets in opposition to the increased tethering.12,13

Present therapies directed at reducing annular size alone often leave the patient with important MR because of persistent leaflet tethering by chordae to the displaced PMs and ventricular walls.14 One solution is to reshape the ventricle by infarct excision or plication with PM shortening or reimplantation, potentially combined with annuloplasty.11,15-17 Such techniques, however, involve extensive surgical manipulation. We therefore proposed a simpler approach to reduce tethering by cutting a limited number of critically positioned chordae tendineae that restrict leaflet closure to the greatest extent but are not required to prevent prolapse. Such an approach also opens the door to potential minimally invasive transcatheter implementation.

How to achieve this is suggested by clinical observations of mitral valve shape in ischemic MR5,6 and basic valve anatomy18 (Figure 1). The mitral leaflets are supported by 2 major sets of chordae: finer marginal chordae that position the leaflet tips and prevent prolapse, and thicker basal or strut chordae that exert force on the body of the anterior leaflet near its base. Increased tethering attributable to ventricular remodeling most noticeably distorts the basal portion of the anterior mitral leaflet near the annulus (Figure 1, center). This leaflet portion is held nearly rigid and tented toward the LV apex by basal chordae inserting closest to the annulus. The more distal leaflet pivots around this “knee,” but only its tip can then meet the posterior leaflet, decreasing the coaptational surface needed to ensure an effective seal.

We therefore proposed the hypothesis that cutting a limited number of these critically positioned basal chordae can improve coaptation and reduce ischemic MR. Eliminating the bend in the anterior leaflet can allow the leaflets to assume a
more normal and less taut configuration, with more effective coaptation at their tips (Figure 1, right). The intact marginal chordae to the leaflet edges should, at the same time, prevent prolapse. As an initial approach to alter the minimum number of structures, we planned to cut the 2 basal chordae attached to the center of the anterior leaflet, which are put under the greatest tension by PM displacement away from the central axis of the LV (Figure 2). These chordae would be cut at their valvular insertions. After an in vitro pilot study, this approach was tested in vivo in a model of ischemic MR using three-dimensional (3D) and Doppler echocardiography to quantify MR and relate it to 3D changes in valve configuration.

**Methods**

**In Vitro Pilot Study**

Chordal cutting was first explored using an excised porcine mitral valve in a physiological pulsatile flow left heart simulator. PM displacement in this model has been shown to generate functional MR as seen in ischemic heart disease. The intact mitral valve and PMs were slightly fixed (1% glutaraldehyde, 1 minute) to prolong tissue integrity but limit stiffening and avoid hemodynamic changes. The annulus was sutured to a physiologically D-shaped ring (6.4 cm², based on 25-mm intertrigonal distance) and mounted in a model designed to simulate normal LV dimensions at the onset of systole. Pulsatile mitral inflow and aortic outflow with normal patterns were generated by a computer-driven pump connected to the model apex. The Dacron-wrapped PMs were attached via plastic rings and swivel joints to rods that permit PM displacement. Regurgitant flow was measured with an electromagnetic cannular flow probe (EP680, Caro-

**In Vivo Model**

After positive in vitro findings, chordal cutting was tested in an in vivo model of ischemic MR, modifying that of Llaneras et al. Seven Dorsett hybrid sheep were anesthetized with thiopental (0.5 mL/kg), intubated, and ventilated at 15 mL/kg with 2% isoflurane and oxygen and given glycopyrrolate (0.4 mg IV), with procan-

**3D Echocardiography**

Thirty rotated LV apical views were acquired (5 MHz epicardial Agilent Sonos 5500) with suspended respiration, as previously described and validated against sonomicrometry. LV volumes were obtained using endocardial borders from 9 views. MR stroke volume was calculated as LV ejection volume minus aortic outflow volume directly measured by flowmeter, and MR orifice area was calculated from regurgitant stroke volume and LV-LA pressure gradient (Yellin-modified Gorlin equation). Regurgitant fraction was calculated as (MR stroke volume)/(Forward aortic+MR stroke volumes).

The least-squares plane of the mitral annular hinge points (confirmed by cineloop review) was established as reference frame ;
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where the leaflets coapt) was reconstructed from intersecting leaflet atrial and ventricular cavities (that is, not including any surfaces versus chordal cutting), with significance at $P<0.0001$.

This decreases the surface area available for coaptation to prevent MR.

fig:3: The leaflet covering area, defined as the leaflet surface area needed to cover and occlude the annular orifice based on mitral valve shape by 3D echo analysis (A), is increased when tethering forms a tented leaflet geometry (B); this decreases the surface area available for coaptation to prevent MR.

In Vitro

PM displacement tented the mitral leaflets away from the annulus, with angulation of the anterior leaflet with the basal chordae insert (Figure 4); MR increased by 18.1±0.5 mL/beat relative to the normal early-systolic closing volume ($P=0.004$). Basal chordal cutting eliminated the angulation in the anterior leaflet, which assumed a more relaxed (untethered) configuration closer to the annulus, with its distal margin bending to coapt with the posterior leaflet. No prolapse or flail was observed, and MR decreased to 2.2±0.5 mL/beat above normal closing volume ($P<0.0001$ versus tethered stage).

In Vivo

In all 7 sheep, inferior ischemia produced mild bulging of the affected wall and displaced the PM tip away from the annulus, with apical tenting of the mitral leaflets and mild to moderate MR (Figure 5, Table; regurgitant fraction of $28\pm3\%$). The anterior mitral leaflet developed a discrete angulated bend between its basal portion and the rest of the leaflet, becoming convex toward the LV (Figure 5, middle, single arrow). Chordal cutting alleviated this angulated tenting and MR (Figure 5, right) despite persistent inferior wall bulging (lower right). No prolapse or flail was observed in any view. The changes in MR volume, orifice area, and regurgitant fraction paralleled those in the leaflet covering area required to occlude the orifice as dictated by mitral leaflet geometry (Table); there were also parallel changes in both IMLC leaflet tenting volume and LA pressure, which returned toward baseline with chordal cutting after nearly doubling with infarction and MR. There were no significant changes before versus after chordal cutting in LV pressure, LV end-systolic volume, or mitral leaflet closing force. Mitral annular area increased with inferior ischemia and the development of MR and a higher LA pressure and decreased with chordal cutting and relief of MR.

Univariate predictors of MR stroke volume were leaflet covering area, IMLC volume, and mitral annular area, but not LV ejection fraction, $dP/dt$, or closing force. Multiple stepwise regression identified leaflet closing area as the strongest independent determinant of MR stroke volume ($r^2=0.71$). MR stroke volume rose steeply with leaflet covering area above 8 cm$^2$ (>one third increase from typical baseline of 6 cm$^2$, $r^2=0.76$) (Figure 6).

Discussion

Ischemic heart disease often initiates a self-reinforcing cycle of LV remodeling and MR, which limits functional capacity and prognosis. Recent studies have confirmed that in the absence of PM rupture, ischemic MR relates to an imbalance of forces applied to the mitral leaflets, with increased tethering forces caused by displacement of the mitral leaflet attachments overcoming the closing force exerted by the
This study shows that the resulting malcoaptation can be relieved in the situations studied by cutting a minimum number of chordae that attach to the anterior mitral leaflet near its base as opposed to its free margin. These chordae usually buttress the anterior leaflet body, but with PM displacement, they exert a dominant and maladaptive role in distorting the leaflet configuration to limit the effectiveness of coaptation, as shown by echocardiography. Cutting these chordae in a relatively simple manner restores the anterior leaflet toward its normal configuration without a sharp angulation near its base. The leaflet body can then approach the annulus more closely and become concave toward the LV cavity, so that the leaflet tip bends into the cavity and meets the posterior leaflet more effectively. With less tethering, less leaflet surface area is required to prevent MR, so that the normal surplus of leaflet area becomes available again to form a coaptational seal. The steep rise in MR only at relatively large leaflet covering areas (Figure 6) is also what we would expect if increased tethering exhausts the normal surplus leaflet area to cause MR. At the same time, the intact marginal chordae continue to prevent leaflet prolapse or flail and can in principle continue to maintain LV function through chordal continuity as a benefit of valve repair as opposed to replacement.

Practically, such an intervention aims to overcome the variable, often frustrating results of annuloplasty techniques that only incompletely address tethering by modifying the annulus but not the chordal-ventricular leaflet attachments. Other approaches to this problem include infarct plication or resection and PM shortening or reimplantation. Chordal cutting, however, seems to be simpler, with less extensive and invasive manipulation.

Limitations and Future Directions
The clinical spectrum of ischemic MR includes widely varying location and chronicity of ischemia, PM tip geometry, and potentially leaflet length. The purpose of this study, however, was specifically to demonstrate that cutting a limited number of basal chordae can, in fact, reduce ischemic MR without producing prolapse in a model of inferior ischemia resembling the pattern seen in many patients with such MR. A similar tethered and angulated mitral leaflet configuration is also observed in patients with MR secondary to more chronic or diffuse ischemia, in those with posterolateral infarctions or anterior infarctions with global LV dilatation displacing the PMs, or in those with dilated cardiomyopathy, in whom eliminating MR has been
shown to improve ventricular function, symptomatic status, and survival. It would therefore be reasonable to pursue future experimental studies of chordal cutting in models of more global as well as more chronic LV dysfunction, of anterior and posterolateral infarction, and of more severe MR, recognizing that additional basal chordae might need to be cut with more severe LV distortion or a combined approach addressing both chordae and annulus used to overcome the limitations of annular ring reduction alone in chronic infarction.

Survival studies are also indicated to demonstrate continued stability of the marginal chordae, although the large number of remaining chordae suggests that individual chordal tension will not measurably increase and may even decrease as the leaflets assume a more normal, less-taut configuration and, over time, diminished MR stabilizes or reduces LV volume. Several additional lines of evidence suggest the safety of this procedure. First, basal chordae to the anterior leaflet have been disconnected in routine surgical therapy of rheumatic and myxomatous mitral valve disease without adverse effect. Second, in experimental studies of such disconnection without infarction, severing the 2 most central basal chordae in sheep does not cause prolapse or alter the 3D shape of the mitral valve or the timing of its motion. In isolated perfused hearts, even severing all the basal chordae (as opposed to only 2 in this study), although slightly decreasing a single measure of segmental shortening, does not cause prolapse. LV size and function, in fact, were completely unchanged when 2 basal chordae were cut in 8 beating sheep hearts studied in situ. Finally, in preliminary work to date, we have imaged 2 sheep at 3.5 months after ligation of left circumflex obtuse marginals 2 and 3 with chordal cutting at the same time; neither have MR, in contrast to sheep with such ligation and intact chordae, who develop moderate MR over 8 weeks as the LV remodels, and neither showed declines in LV function after the initial infarct.

**Summary**

Ischemic MR typically relates to ventricular distortion with increased tethering of the mitral leaflets and conveys adverse prognosis. Cutting a minimum number of critically positioned chordae, on the basis of insights from chordal anatomy and ultrasound imaging, can improve mitral leaflet coaptation and reduce or eliminate MR without the need for ventricular incision or reshaping.
Acknowledgments
Supported in part by grants HL38176, HL53702, and K24 HL67434 from National Institutes of Health, Bethesda, Maryland, and by a donation from Bernard L. Adams, Holyoke, Mass. Dr Messas was supported in part by fellowships of the Georges Lurcy Charitable Trust, the French Foreign Ministry Lavoisier Grant, and the Harvard Club of France. We thank Shirley Sims for her expert assistance with the manuscript.

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doi: 10.1161/hc4201.097135

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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