New understanding of ischemic mitral regurgitation: the marionette and its masters

Please see page 326 for the article by Agricola et al. (doi:10.1016/j.euje.2004.03.001) to which this editorial pertains.

Echocardiographic observations of leaflet motion have taught us volumes about the mechanism of valvular heart disease. Identifying excessive versus restricted mitral leaflet motion, as in Carpentier’s classification, has become the foundation of repair techniques tailored to the individual patient. Ischemic mitral regurgitation (MR) has nevertheless remained a therapeutic challenge, with important failure and recurrence rates after standard annular ring reduction, and a higher peri-operative mortality than with degenerative MR. In ischemic MR as well, echocardiography has firmly established that restricted leaflet motion is the fundamental mechanism in the vast majority of patients, confirming the proposal that displacement of the papillary muscles (PMs) in the remodeling heart tethers the leaflets into the ventricular cavity. Echocardiography has provided further mechanistic clues: the anterior leaflet is deformed, with a sharp bend created near its base, at a point where intermediate (basal, secondary, or strut) chords insert; this particularly limits the ability of the distal anterior leaflet to coapt with the posterior, and has suggested the therapeutic maneuver of selective chordal cutting to relieve this tethering and reduce MR, while maintaining intact chords to the leaflet margins that prevent prolapse.

In this issue of the Journal, we learn of a new echocardiographic observation that not all ischemic MR is alike. Agricola, Alfieri and colleagues found two distinct leaflet patterns in 92 consecutive patients considered for surgery with chronic ischemic MR due to restricted leaflet motion. These patterns relate specifically to the critical zone of conjunction, where normally the distal leaflets are apposed over a surface of coaptation that ensures an effective seal. One group of patients had typical restricted leaflet closure, with both leaflets tethered apically but symmetrically into the left ventricle (LV); the other group had asymmetric coaptation, with the distal anterior leaflet, beyond the strut chordal insertion, actually less restricted, and protruding superiorly toward the left atrium relative to the adjacent, highly restricted posterior leaflet. This malcoaptation is particularly unfavorable to forming an effective coaptational surface. As in the case of MR due to excessive leaflet motion, the leaflet pathology determines jet direction: symmetric tethering produces central MR jets, whereas asymmetric tethering largely generates eccentric, posteriorly directed jets.

What is the mechanism of this asymmetry, and what are its therapeutic implications? The authors have quantitatively analyzed echocardiographic images to assess changes in both global LV function and localized geometry of tethering structures (PMs and annulus). Their findings suggest that asymmetric tethering is associated with predominant inferoposterior myocardial infarction and displacement of the posterior papillary muscles; symmetric tethering is associated with symmetric displacement of both PMs, with greater global dilatation and dysfunction, symmetrically restraining both leaflets into the ventricle. They suggest...
that asymmetric displacement of the posterior papillary muscle pulls the leaflets with a predominant posterior vector; presumably, if the leaflet tips initially meet each other, this posterior pull can cause the anterior leaflet to slide up to the level of the mid-posterior leaflet, creating this asymmetry. (Analogously, in hypertrophic cardiomyopathy, shifting the PMs anteriorly causes the posterior leaflet to meet the mid-anterior leaflet, creating a distal residual leaflet that produces SAM.24–26) Undersizing annular rings in ischemic MR,6 by shifting the annulus anteriorly, can similarly augment the posterior vector applied by the displaced papillary muscle, and can produce asymmetric coaptation of the anterior leaflet with a relatively immobilized posterior leaflet.27–29

Our group and others13,30–32 have made a similar observation, also noted in the surgical experience, that in some patients with more severe MR, the mitral valve adopts a uniquely asymmetric configuration suggesting a combination of underlying mechanisms, including both leaflet retraction and chordal imbalance. In such patients, although both leaflets are apically tethered relative to the mitral annulus, one leaflet prolapses or extends superiorly to the other, while not prolapsing relative to the annulus (leaflet overshoot), further impairing coaptation which becomes asymmetric, with a prominent eccentric regurgitant jet. Asymmetric coaptation, in our experience, is particularly evident in patients with more severe ischemic MR, consistent with malcoaptation.30,31 (Of note, moderate to severe MR was present in 88% of Agricola’s asymmetric group versus 68% of the symmetric group.) The final common arbiter of MR appears to be the leaflet coaptation surface. Levi et al. also described eccentric jets in 10% of patients with inferior wall infarction and posterior leaflet restriction.32 We have, however, observed asymmetric coaptation in patients with global LV dysfunction (although symmetric tethering is certainly more common), and symmetric coaptation with frequently central jets in patients with isolated inferior wall abnormalities; this suggests that leaflet overshoot may represent disparities of tethering not limited to asymmetric PM involvement.

To understand these observations, we can think of the mitral valve as a marionette pulled by its

Figure 1  Intra-operative transesophageal 3D echo reconstruction of the mitral valve, viewed from the left atrium, in a patient with severe ischemic MR caused by a combination of restricted leaflet closure (downward arrows) and partial posterior leaflet flail (upward arrow). Courtesy of Nadia S. Nathan, MD, with thanks to Mark D. Handschumacher, BS. Ant = anterior, Post = posterior.
masters, the papillary muscles. The counterbalancing gravitational force on the marionette is, in the case of the mitral valve, provided by the LV (intracavitary pressure and force exerted via the mitral annulus). Pulling on the leaflets symmetrically by both PMs produces uniform tautness without slack in the leaflet–chordal network. Pulling asymmetrically can induce mismatch and malcoaptation of leaflet portions for example, displacing one posterior leaflet scallop relative to another can create an intervening gap. To obtain the image of asymmetric coaptation shown by the authors, the distal anterior leaflet must actually become relatively slack. This corresponds to one tethering bar of the “marionette” being raised at one end, with the other end simultaneously lowered — for example, rotating a bar that controls both arms so that one rises, the other falls. How might this occur in the heart? Both in vitro and in vivo, we have had experimental opportunities to rotate the posterior PM around an axis perpendicular to the long-axis plane. Such rotation may occur as the underlying wall bulges outward, causing the papillary muscle to turn posteriorly. That PM is typically like a tethering bar, with anterior and posterior heads. This rotation brings the anterior head closer to the annulus, and the posterior farther away. Rotating this papillary muscle will correspondingly rotate the leaflet tips, with the anterior leaflet tip moving closer to the annulus and the posterior being restrained even more into the ventricle. Therefore, asymmetric PM positioning can induce asymmetric coaptation in both mediolateral (inter-scallop) and antero-posterior orientations. Asymmetric tethering can similarly cause tricuspid valve malcoaptation with increased regurgitation.

In summary, therefore, Agricola, Alfieri and colleagues have used echocardiographic observations to derive important new insights into the mechanism of ischemic MR. Such refinement in our understanding increases the potential to tailor effective therapy to the individual patient. Based on symmetric versus asymmetric leaflet coaptation, the authors have proposed different surgical approaches targeting the underlying problem most specifically. This will be a tantalizing hypothesis to test, both experimentally and in clinical practice.

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