**RE: INTERPAPILLARY
MUSCLE DISTANCE
INDEPENDENTLY AFFECTS
SEVERITY OF FUNCTIONAL
MITRAL REGURGITATION IN
PATIENTS WITH SYSTOLIC
LEFT VENTRICULAR
DYSFUNCTION
To the Editor:**

The recent article by Kim and coauthors¹ adds to the steadily growing body of evidence that identifies papillary separation as a dominant factor in the mitral insufficiency that is caused by left ventricular dysfunction. It does not, however, explain why this is so. I performed an old fashioned gross anatomic study, which revealed the mechanism.²

The normal movements of the mitral valve are produced by changing fluid forces interacting with the leaflets, which are subject to the control of the chordae. In a normal heart, the changes in the annulus, ventricular wall, and papillary muscles are synchronized with the fluid forces. Leaflet flexibility, such that the leaflet tissue bends under its own weight, is essential to this behavior.

The critical gross anatomy that governs the behavior of the leaflets in the presence of papillary displacement is the fact that the distance from the origin of a second-order, load-bearing, main chorda to its insertion into the junction of the

meeting of rough and smooth zones of the anterior leaflet is less than the combined lengths of the origin of a first-order, aligning chorda to its free-edge insertion and the length of the rough zone from free edge to the insertion of the main chorda.³

In their study, Kim and coauthors¹ discuss the classic tenting appearance of the closed mitral valve in the presence of papillary displacement. They emphasize that papillary separation has a greater effect on mitral insufficiency than either apical or posterior papillary displacement.

These consequences of papillary separation are explained by in vitro studies of the effects of papillary position on mitral valves with a rigid tight reduction of the mural annulus.²

With apical displacement of papillary chordal origins, the pull of a main chordal insertion is transmitted to the leaflet tissue between the chordal insertion and the annulus. This stiffens and restricts the mobility of the anterior leaflet from the annulus to the rough zone. This stiffened part of the anterior leaflet and a less obvious similar stiffened portion of the posterior central scallop together produce the appearance of a tent while the still flexible rough zone of the mitral leaflets is capable of at least partial closure. When the papillary muscles are separated, however, this also tightens the central first-order chordae. With these chordae tight, the rough zone between the free edge and the insertion of the main chordae also becomes stiff. The whole central portion of the anterior leaflet and the central scallop from base to free edge have at this point lost the flexibility essential to normal opening and closing. Further narrowing of the annuloplasty increases the tightness of the chordae and the stiffness of the leaflets.

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**WHY DOES PAPILLARY
SEPARATION PLAY A MAJOR
ROLE IN FUNCTIONAL
MITRAL REGURGITATION
CAUSED BY LEFT
VENTRICULAR FUNCTION?**

Reply to the Editor:

Functional mitral regurgitation (FMR) is a common complication in patients with ischemic heart disease, and it occurs as a consequence of left ventricular dysfunction. Previous studies have shown that leaflet tethering by displaced papillary muscles is an essential pathway of FMR.¹ In addition, the tethering distance of papillary muscles between papillary muscle tips and the anterior annulus is a dominant factor in the severity of FMR.² Reducing the distance between the anterior and posterior papillary muscles, the interpapillary muscle distance (IPMD), with the papillary muscle approximation technique is effective for restoring leaflet coaptation.³ Papillary separation is therefore considered a dominant factor in FMR caused by left ventricular dysfunction. The reason that papillary separation plays an important role in

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