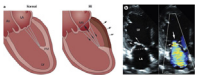

PREDICTING RECURRENT MITRAL REGURGITATION AFTER MITRAL VALVE REPAIR: A DIFFICULT ENDEAVOR AND A NECESSITY

Reply to the Editor:

Wang and Tang question our finding that basal aneurysm/dyskinesia (BAD) was the only preoperative echocardiographic characteristic that predicted recurrent mitral regurgitation (MR) after mitral valve (MV) repair. They also argued that dyskinesis should not be used as a substitute for a severe form of left ventricular (LV) ischemic remodeling, and criticized that the status of myocardial revascularization or preoperative coronary anatomy was not taken into account in our model. We wish to clarify these issues.

In terms of echocardiographic parameters, we assessed the influence of end diastolic volume, end systolic volume, ejection fraction, posterior and anterior leaflet angles, tenting height and area, as well as effective regurgitant orifice area and vena contracta. None of these parameters in univariate predictions obtained the minimum *P* value of .25 to be considered for further inclusion in the prediction model of recurrence and/or death. However, in our cohort, the presence of a basal aneurysm, which occurred frequently (52 out of 116 patients), was strongly associated with those 2 outcomes ($P < .001$). We believe BAD reflects a severe form of LV remodeling, including papillary muscle displacement, leaflet tethering, and annular dilatation, and because it is the summation of many LV structural components, it strongly predicts MR recurrence.

We disagree that the finding in our article[1] of the independent association of BAD with recurrence of ischemic mitral regurgitation (IMR) is "confounded and unreasonable." This finding is based on a fundamental mechanism for IMR. As they point out, the mechanism underlying IMR "is associated with remodeling and distortion of the ischemic left ventricle after myocardial infarction." As demonstrated in Figure 1, the development of dyskinesis or frank aneurysm from infarction of the myocardium underlying the papillary muscles sets off an abnormal remodeling cascade leading to distortion of the normal spatial relationships between LV and MV geometry necessary for normal mitral leaflet closure.[2] This distortion results in MV leaflet tethering and IMR. It is therefore not surprising that BAD was associated with recurrence of IMR because it is the primary ischemic LV remodeling feature leading to IMR. There are several reasons why direct measures of MV geometry such as tethering angles or tethering area or height were not independently associated with recurrence of IMR. There is heterogeneity in MV geometry in patients and measures of MV geometry are subject to increased variability inherent when using 2-dimensional echocardiographic reference planes. A reason that BAD was shown to be an independent predictor rather than MV geometric measures may be that the former reflects a severe form of LV ischemic remodeling that incorporates the mechanistic abnormalities of papillary muscle displacement, leaflet tethering, and annular dilatation, all of which influence IMR.

The question of whether the presence of infarcted territories can predict recurring MR, or whether grafting of

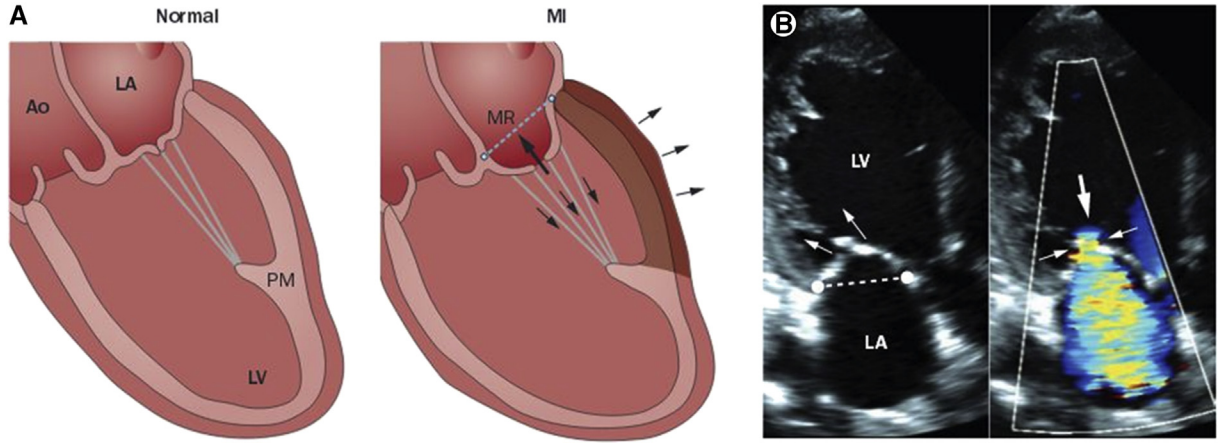

FIGURE 1. Mechanism of ischemic mitral regurgitation. A, Mechanism of ischemic mitral regurgitation caused by increased mitral leaflet tethering owing to left ventricular remodeling after myocardial infarction (*shaded wall*) with outward bulging (*arrows on the outer surface of the heart*). Leaflet closure is restricted by increased tethering forces on the leaflets exerted via the chordae (*arrows within the heart, exceeding normal on the left*). B, Echocardiogram from a patient with inferior wall infarction and tethered mitral leaflets (*arrows in left panel*) with characteristic anterior leaflet bend and concavity toward the left atrium (*LA*) indicating chordal tethering, mitral regurgitation (*MR*) orifice (*small arrows in right panel*), and MR flow (*large arrow in right panel*). *Ao*, Aorta; *LV*, left ventricle; *PM*, papillary muscle; *MI*, myocardial infarction. Reprinted by permission from Macmillan Publishers Ltd: *Nature Reviews Cardiology*,[2] copyright 2015.

Authors have nothing to disclose with regard to commercial support.

specific territories can provide segmental wall motion improvements is very valid. Myocardial viability studies would have been useful, pairing them with the revascularization strategy in hopes that a grafted viable territory, especially in a vulnerable and viable segment such as the inferior wall, could stabilize LV dimensions and the associated tethering. These exams were not part of the trial protocol, which was aimed primarily not at finding factors associated with MR recurrence following surgery, but at comparing 2 treatment strategies: MV ring annuloplasty and MV replacement with full chordae preservation. Similarly, we do not have postoperative angiograms to assess the permeability of the graft in a given territory to be able to examine its association with MR recurrence. This limitation was acknowledged in our articles.

We agree with Wang and Tang regarding the need to better predict postrepair MR recurrence to tailor the choice of surgical approach to the likelihood of recurrent IMR. The model obtained from our data based on 10 variables (ie, age, body mass index, sex, race, effective regurgitant orifice area, BAD, New York Heart Association functional class, prior percutaneous or surgical revascularization, and history of ventricular arrhythmia) is accurate in its determination of optimal candidates for MV surgical repair.

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http://dx.doi.org/10.1016/j.jtcvs.2016.09.054

CARDIAC SURGERY CENTERS ARE IDEAL PLACES TO TREAT PATIENTS UNDERGOING LIFE-THREATENING DEEP ACCIDENTAL HYPOTHERMIA USING EXTRACORPOREAL MEMBRANE OXYGENATION VENOARTERIAL THERAPY

To the Editor:

It was with great interest that we read the publication by Squiers and colleagues[1] in the July issue of the *Journal*. The authors presented an excellent review of the latest trends of extracorporeal membrane oxygenation (ECMO) therapy in adult patients. We would like to suggest the need of completing the review with an additional description of how the ECMO venoarterial (VA) therapy for patients with accidental deep hypothermia in life-threatening status was implemented.

The guidelines of the European Council of Resuscitation recommend that patients with a core body temperature less than 28°C and with hemodynamic instability (systolic arterial pressure <90 mm Hg, ventricular arrhythmia) or undergoing cardiac arrest be immediately transferred to a center experienced in extracorporeal life support therapy to implement extracorporeal perfusion and ventilation therapy preferably with an ECMO VA system.[2]

For the past 3 years, in our center, we have been using a unique system of qualifying patients with accidental deep hypothermia for ECMO VA therapy.[3] The implementation of this procedure would have not been possible without the active support of all personnel from the cardiac surgery and intensive therapy departments, as well as the mentoring supervision of our ECMO coordinator (head of our Severe Accidental Hypothermia Center).

Establishing a role of coordinator available 24 hours a day via telephone was crucial for the system's success. The tasks of the coordinator include consultation of hypothermia cases diagnosed on the territory of the voivodeship, competent aid during diagnostics and treatment in medical centers, and potential qualification for extracorporeal rewarming.

During the implementation of this system, it was necessary to inform and train everyone involved in this ''survival chain'' about the principles of ECMO therapy and the need of implementing more advanced therapy procedures, as well as to take steps to determine the immediate qualifying inclusion criteria for patients.