Relation between reduction in ischaemic mitral regurgitation and improvement in regional left ventricular contractility during low dose dobutamine stress echocardiography

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The indication of adjunctive mitral valve surgery in patients with ischaemic mitral regurgitation (MR) undergoing coronary artery bypass grafting remains controversial. Furthermore, no approach currently predicts in which patients ischaemic MR will resolve with revascularisation alone. It has been recently suggested that a reduction in ischaemic MR caused by an improvement in regional left ventricular (LV) contractility during low dose dobutamine stress echocardiography (LDSE) might predict a beneficial effect of revascularisation on ischaemic MR. This study tested the hypothesis that LDSE induced changes in ischaemic MR are associated with changes in regional LV contractility.

METHODS
Fifteen consecutive patients with stable coronary artery disease, ischaemic LV dysfunction, ejection fraction (EF) < 50%, and at least moderate MR (regurgitant fraction (RF) ≥ 30%) were selected for enrolment from all patients that had a resting echocardiogram. Exclusion criteria included the following: technically inadequate echocardiogram, structurally abnormal mitral valve, more than trace aortic regurgitation, and non-sinus rhythm. The mean (SD) age of the 15 patients was 68 (8) years; 10 were male and 11 had a prior myocardial infarction. The site of the infarction was anterior in five patients, lateral in one patient, inferoposterior in one patient, both anterior and lateral in one patient, and both anterior and inferoposterior in three patients. Two patients had right bundle branch block and two patients had left bundle branch block. The protocol was approved by the hospital ethics committee and all patients gave written informed consent.

Each patient underwent LDSE (a peak dobutamine dose of 10 μg/kg/min). The apical long axis, four chamber, and two chamber views were used for all evaluations at baseline and during LDSE. The degree of MR was quantified as RF by the Doppler method using mitral and aortic stroke volumes. The LV end diastolic volume, end systolic volume, and EF, were calculated by the modified biplane Simpson method. The mid systolic mitral annular area, mitral valvar tenting area, and coaptation height were measured to estimate mitral deformation in the same manner as previously described. The anterior and posterior papillary muscle (PM) leaflet tethering lengths (TL) were measured to estimate displacement of the PM in the same manner as previously described. Regional LV wall motion at the PM attachment was evaluated with a four grade scale, eight segment (four basal and four mid ventricular) model, allowing calculation of the wall motion score index (WMSI). Out of these eight segments, four anterolateral segments and four inferoposterior segments were considered as those at the anterior PM and those at the posterior PM, respectively. Myocardial contractile reserve was defined as improvement in wall motion of ≥ 1 grade.

RESULTS
Out of the 15 patients, LDSE decreased the RF in 13 (87%) and the degree of MR became mild (RF < 30%) in six patients (40%). The mean RF decreased from 44 (12)% (range 30–74%) at baseline to 35 (13)% during stress (range 14–62%; p < 0.01). As shown in Table 1, LDSE induced changes in coaptation height, posterior TL, the sum of bilateral TL, and inferoposterior WMSI were significantly associated with changes in RF, while changes in global LV volume and function, anterolateral WMSI, and global WMSI, were not.

In the 13 patients with some dysynergy in the inferoposterior segments, five patients who had at least one dysynergic inferoposterior segment with contractile reserve showed a significantly larger decrease in RF during stress than the eight others (17 (7)% v 4 (8)% p < 0.05). In the 14 patients with some dysynergy in anterolateral segments, there was no difference in stress induced reduction in RF between the five patients who had at least one dysynergic anterolateral segment with contractile reserve and the nine others (9 (12)% v 10 (10)%).

DISCUSSION
A previous study has shown that the important predictors of changes in ischaemic MR during exercise were changes in regional remodelling in the posterior LV and mitral deformation. Our results using LDSE confirmed this finding. LDSE induced changes in posterior wall motion, PM displacement, and mitral deformation, were significantly associated with changes in MR, but changes in anterior wall motion or global LV volume and function were not. Improvement in wall motion in segments supporting the posterior PM may reduce the degree of MR by decreasing PM leaflet tethering forces that cause incomplete mitral leaflet closure.

A previous study showed that a mild degree of ischaemic MR (effective regurgitant orifice area < 0.2 cm²) carried a better prognosis than a more severe degree of ischaemic MR. The present study suggests that contractile reserve in the dysynergic posterior LV, identified with LDSE, may serve as a predictor of reduced MR in response to revascularisation.
LDSE can predict the decrease of MR to a mild degree in patients after revascularisation alone, then adjunctive mitral valve surgery may not be required in such patients. Several limitations should be acknowledged. Firstly, the number of patients studied was small so we could not assess the role of clinical characteristics such as the infarct site or the presence of bundle branch block in modulating the severity of ischaemic MR. Secondly, we could not assess changes in the ventricular force required to close the leaflets as represented by the systolic pressure gradient between the LV and atrium because of the technical difficulty of accurately measuring the velocity of MR, especially in mild MR, during LDSE. Thus, we were unable to assess changes in effective regurgitant orifice area calculated using the velocity of MR. Prospective studies involving these factors and larger numbers of patients are required.

In conclusion, the severity of ischaemic MR may decrease during LDSE and this reduction is associated with an improvement in regional wall motion of the posterior LV, but not with changes in anterior wall motion or global LV volume and function.

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