Letter to the Editor
Management of moderate ischemic mitral regurgitation demystified

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Hats off to Campwala et al. [1] for demystifying the management of moderate ischemic mitral regurgitation (IMR). Despite all its limitations their study can be deemed as a landmark contribution to the existing literature on management of IMR. Although they have discussed factors predicting regression of IMR after isolated coronary artery bypass grafting (CABG) in the context of 3–4 $+$ IMR yet the findings of their study can be extrapolated to define the optimal management strategy for 2 $+$ IMR which is the most controversial grade of IMR viz a viz intervention on the mitral valve at the time of CABG.

Although most surgeons would agree that severe IMR (3–4 $+$) should be corrected at the time of CABG and that trace to mild IMR (1–$+$) can probably be left alone, the optimal management of moderate IMR (2 $+$) remains controversial [2]. The pathophysiology of IMR is complex, and its presence may be related to several underlying processes that are often difficult to separate in a given patient [3]. Because of its complex pathophysiology and heterogeneous clinical presentation, the proper treatment of IMR is often debated, and the relative utility of revascularization—with and without concomitant mitral valve surgery—and the setting of moderate IMR is uncertain. Those favoring a conservative approach suggest that revascularizing ischemic areas will improve regional wall motion and correct the MR [4]. On the other hand, proponents of more liberal use of mitral annuloplasty in patients with moderate IMR at the time of CABG argue that CABG alone will not correct moderate IMR in many patients, especially those with scarring from myocardial infarction and those with anular and ventricular dilatation [2].

The study by Campwala et al. [1] has once and for all solved this controversy by providing solid predictors of regression of IMR after CABG alone. As pointed out by Campwala et al. [1] regression of IMR is related to left ventricular (LV) size reduction and improvement in LV function. Hence, a corollary of this conclusion will be that in the presence of low ejection fraction and dilated LV, moderate IMR has to be corrected. On the other hand, in the presence of viable hibernating myocardium, adequate revascularization, lack of excessive atherosclerotic burden and preoperative therapy with beta-blockers and ACE-inhibitors perhaps a more conservative approach with revascularization alone may be justified.

References


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Reply to the Letter to the Editor

Reply to Raja

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We want to thank Dr Raja for his interest in our paper [1]. The main message of our paper is that 3 or 4 $+$ MR regression in the setting of ischemic cardiomyopathy depends upon LV size reduction following CABG. This depends on presence of adequate viable myocardium and its revascularization. Medical therapy that facilitates reverse remodeling of the LV is helpful as well. Though, our study gives insights into MR regression, we are not recommending leaving 3–4 $+$ MR alone during CABG. As genesis and progression of ischemic MR depends on LV remodeling, we suggest that in addition to mitral valve repair, these patients need aggressive revascularization and therapy with beta blockers and ACEI inhibitors for the mitral valve repair to be durable.

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Coming to the important question of the handling of 2 + MR during CABG, clearly no consensus exists. However, the results of our study may be cautiously extrapolated to this group of patients. We feel that whether or not mitral valve is surgically addressed, aggressive myocardial revascularization and medical therapy with beta-blockers and ACE inhibitors may help in causing MR regression.

One other practical point we would like to highlight is that MR regression is less likely to occur in those with clinical markers of excessive diffuse atherosclerotic diseases like older age, diabetes mellitus, renal insufficiency and cerebrovascular accident. Patients on cardio-protective medications like beta-blocker and ace-inhibitors prior to CABG may be protected from peri-operative ischemia and benefit from reverse LV remodeling.

Reference


Letter to the Editor

Further discursions concerning the unique myocardial band

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In the current issue of the Journal, Corno argues that the ‘new concepts on cardiac anatomy and physiology…deserve for further and deeper investigations’ [1]. Should he not first examine existing anatomic investigations of the past 400 years showing that the heart is formed on the basis of a modified blood vessel, and not in the fashion of skeletal musculature, rather than obfuscate the issue with comments about the free-standing subpulmonary infundibulum, an important feature of anatomy that owes nothing to the presence or absence of a unique myocardial band [2]?

Castella and colleagues [3] describe microsonometric findings, which display a widely variable pattern of regional shortening in discrete areas of the ventricular walls. The authors continue to interpret the regional dephasing observed in onset and termination of shortening as being the result of delayed excitation and contraction. In reality, onset and termination of myocardial shortening are widely modulated by the amount and time-course of the action of structural afterload over the heart cycle. Delayed shortening primarily heralds the action of intrinsic resistances sustained by the inhomogeneous three-dimensional arrangement of the myocardial syncytium [4]. Onset of mechanical activation, in other words the onset of contraction, unfortunately, cannot be measured by methods designed to assess distances of shortening. If ‘the main objective of the study’, therefore, was to establish ‘the sequence of contraction within the myocardial mass’, then unfortunately the authors [3] have chosen the wrong method.

It is equally disturbing to find them using the neologism ‘nervous muscle anatomy’. Are the authors, in this regard, referring to the insulated network of musculature which is responsible for dissemination of the impulse of excitation within the ventricles, and generally called the ‘conduction system’? This, of course, is made up of specialised myocardial tissue, with any autonomic nerves present in the ventricles performing no more than a modulating function.

It is when commenting on the purported myocardial band, however, that they commit their most egregious error. They make reference to the ‘Triebwerk’, meaning Krehl’s ‘Triebwerkzeug’ [5], which consists of the circular fibres which encircle the left ventricle. Krehl believed that these fibres were the driving force for ventricular ejection. But Krehl also took into consideration the function of inner and outer myocardial layers, which he considered to be continuous in the apex and anchored at the base. Krehl assumed that systolic circular constriction and thickening of the ‘Triebwerkzeug’ is partially reversed at end-systole, or during diastole when contraction in the circular fibres ceases, by a remaining amount of tension in the embracing layers of longitudinal fibres. This concept is entirely at odds with the hypothesis of the unique myocardial band proposed by Torrent-Guasp [2]. It is an anatomic fact that dissections produced by Torrent-Guasp systematically destroy the continuum of the bordering fibres when separating the purported basal and apical loops [2]. The authors, therefore, should refrain from seeking to support the spurious notion of the unique myocardial band by misquoting classical works, even if these solecisms might have been caused by difficulties in translating the original work [5] from the German language.

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