

Letters to the Editor

IMVR since 2006.² In a cohort of patients with coronary artery disease and depressed left ventricular ejection fraction, IMVR seemed to occur mainly whenever a transmural area of delayed enhancement (necrosis) was present at cardiac MRI within the inferior myocardial segments.³

On the contrary, we did not notice any significant difference in the geometric mitral valve and papillary muscle parameters (as documented at MRI) when comparing patients with and without IMVR. Furthermore, myocardial necrosis distribution was the sole determinant for IMVR occurrence.³

This confirms that IMVR is not resulting from an abnormal valve/apparatus but from a malfunctioning ventricular muscle (in its segmental aspects), and for this reason, surgical decision making cannot be adequately performed just on the basis of a simple rest echocardiography.

We agree with Flynn and colleagues¹ that patients with excessive scar burden (at MRI) in the inferior myocardial segments (particularly those segments close to the posterior papillary muscles) will possibly have recurrent IMVR even after annuloplasty. Within this group of patients, we think some distinctions should be made. In fact, in our surgical practice we still perform a simple annuloplasty if a segmental geometric remodeling has not yet taken place and vital areas are present within the delayed enhancement (necrotic) zone. In this case, we should emphasize that standard MRI will not be able to detect hibernated myocardium that could, once the coronary target has been revascularized, prevent the geometric remodeling and the consequent IMVR recurrence.

In this context, dynamic echocardiography (with dobutamine or semi-supine physical stress) may assist in further surgical stratification and identification of “recoverable” myocardium.

In a different context, identification of large areas of MRI delayed enhancement in the inferior myocardium

should trigger further evaluation even in patients with mild IMVR. In particular, physical stress echocardiography may enhance severe IMVR even in patients with mild regurgitation at rest.⁴ This finding could support a broader application of mitral valve annuloplasty, especially when the myocardial remodeling has not yet taken place.

In conclusion, together with Flynn and colleagues,¹ we agree that IMVR is an elusive occurrence and adequate surgical stratification cannot be performed with sole rest echocardiography. IMVR is a disease of the ventricular muscle, and surgical outcomes should be reinterpreted in the light of preoperative evaluation with global morphofunctional and perfusion-vitality studies, such as those achievable with MRI. The presence of extensive necrosis in the inferior myocardial segments may suggest a high rate of postoperative IMVR recurrence. Further investigation with preoperative stress echocardiography may identify viable myocardium and trigger IMVR in those patients with mild regurgitation at rest. In both cases, myocardial surgical revascularization together with mitral valve annuloplasty may be a valid option if adequate coronary targets are present and myocardial remodeling has not yet taken place.

Giuseppe D'Ancona, MD

Michele Pilato

Department of CT Surgery

ISMETT (Mediterranean Institute for

Transplantation and Advanced

Specialized Therapies)

Palermo, Italy

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Reply to the Editor:

We thank Drs D'Ancona and Pilato for their thoughtful comments. Patients with ischemic cardiomyopathy represent a diverse population who differ in the extent of myocardial ischemia, severity of the left ventricular dysfunction, and severity of functional ischemic mitral regurgitation. A thorough understanding of the extent of ischemic changes is essential for the appropriate planning of surgical therapy. Magnetic resonance imaging has emerged as a powerful diagnostic tool for the comprehensive evaluation of patients with ischemic cardiomyopathy. In our practice, magnetic resonance imaging is routinely used in patients with ischemic cardiomyopathy for the assessment of the severity and distribution of myocardial scarring, as well as the estimation of residual myocardial viability. Severity of ischemic changes determines the choice of surgical therapy. Presence of transmural scarring in the region of the papillary muscles in patients with severe ischemic cardiomyopathy and severe functional mitral regurgitation is a poor prognostic sign indicating advanced disease. Myocardial revascularization and mitral valve repair with an undersized annuloplasty ring in these patients are often associated with the poor outcomes and high incidence of recurrent mitral regurgitation; therefore, alternative therapies, including heart transplantation, should be considered.¹ In contrast, in patients with less severe forms of ischemic cardiomyopathy and moderate functional ischemic regurgitation, substantial improvement in functional status and survival can be accomplished with isolated coronary artery bypass grafting.²

The addition of dobutamine echocardiography as a functional diagnostic method for identification of recoverable myocardium, as suggested by Drs Ancona and Pilato, could aid in better understanding this complex disease and should be considered as part of a comprehensive diagnostic workup of patients with ischemic cardiomyopathy. This diagnostic algorithm represents a welcome and long-needed shift from “valvulo-centric” to “ventriculo-centric” focus in management of patients with ischemic cardiomyopathy and functional ischemic regurgitation.³

Tomislav Mihaljevic, MD
Marc A. Gillinov, MD
Cleveland Clinic
Cleveland, Ohio

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SAFETY OF AXILLARY ARTERY CANNULATION

To the Editor:

We read the report of Takayama and colleagues¹ about the successful management of aortic root replacement with axillary artery cannulation and open distal anastomosis technique. For ascending and aortic arch repair, we use a similar cannulation site, the right brachial artery. We previously published our results of 104 arch repair cases that we performed with low-flow antegrade cerebral perfusion through the right brachial artery and with an open distal anastomosis technique during moderate

hypothermia.² We congratulate the authors for their excellent results, but we would like to remind them that neither axillary nor brachial artery cannulation is totally safe and reliable.

There are literature reports about the pitfalls of axillary artery cannulation, including arterial injury, new aortic dissection, compartment syndrome/arm ischemia, brachial plexus injury, inadequate cardiopulmonary bypass flow, and malperfusion.³ In a patient with acute type I aortic dissection, we recently experienced a complication of axillary artery cannulation that we could not have realized initially. The patient's brachial artery diameter was very small, and therefore we preferred axillary artery cannulation with a side-graft anastomosis. The axillary arterial wall looked normal, and its flow seemed to be adequate. As soon as we started cardiopulmonary bypass, the pressure in the arterial lines exceeded normal ranges, and bleeding occurred around the axillary artery cannula. We decided that our cannula was in the false lumen. We quickly switched the inflow cannula to the innominate artery. The patient did wake up without any neurological deficit. For aortic dissections extending into the axillary artery, it is possible for the surgeon not to realize this situation, and he or she might end up cannulating the false lumen. One should be very cautious about the line pressure at the initiation of cardiopulmonary bypass and should be ready for alternative techniques, if necessary.

We would like to remind the authors of another condition in which axillary artery cannulation is not safe or even contraindicated. An aberrant right subclavian artery is an anatomic variation that is more common than we think, with a prevalence of 0.4% to 2%. The aberrant right subclavian artery originates from the proximal descending aorta, and patients with this anomaly are typically asymptomatic.^{4,5} It is clear that the inflow will route to the descending aorta with right axillary artery cannulation in such a patient, and therefore alternative cannulation sites are to be

used. Direct cannulation of the right, the left, or both carotid arteries can be used in this situation. We would like to underline that the existence of an aberrant right subclavian artery is not that rare and must be kept in mind.

We conclude that in these complex surgical approaches, the ideal method of arterial access is not identical and changes individually. Perfusion, neuroprotection, and open graft replacement techniques depend on anatomic considerations and prudential judgments.

Aslihan Kucuker, MD
Erol Sener, MD
Cardiovascular Surgery Clinic
Ataturk Education and
Research Hospital
Ankara, Turkey

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Reply to the Editor:

We have read the article by Dr Küçüker and his colleagues with great interest. They should be congratulated on their excellent clinical outcome of arch repair using right brachial artery cannulation.¹ Their technique achieves the same goal as the axillary artery cannulation technique. Although the brachial artery might be easier to access, its size might occasionally prevent it from