colloid used only albumin. These subsequent infusions were provided at the discretion of the attending physicians.

Dr McKay next asks about blinding and transfusion practices. This portion of the protocol is presented in the article's section on Power Analysis. Intensivists providing postoperative care were blinded to the fluids infused, but the anesthesiologists infusing the fluids were not blinded. We followed this protocol for several reasons. Current requirements in this era of regulatory compliance call for transparent intravenous tubing. Given that albumin and hetastarch fluids have different appearances, it would have been difficult to assure a blinded process in the clinical setting of the operating room even if we had somehow covered the 500-mL containers from which the fluid was being infused. Dr McKay also complains that we do not present standardized (protocol or goal-driven) fluid administration. Presuming that Dr McKay is referring to fluid administered after the first liter (administered per protocol and described in our article's Statistical Power Analysis section), we state later in that section that the remaining standard operative regimen of crystalloids and blood products in the perioperative period were administered at the discretion of the anesthesiologist. We note in the following paragraphs on Clinical Protocol that postoperative fluid management was at the discretion of the attending intensivist according to each patient's clinical status. The fluid management policy in both periods was selected so that our study could examine the impact of the initial fluid used for volume replacement on vascular outcome in patients receiving the careful care provided following the protocols otherwise used to guide care in nonresearch environments.

Dr McKay also questions what factors (beyond infusion of the randomly assigned fluid) explain the increased blood loss and transfusion requirement in members of the hetastarch branch. She proposes that the 287.2-mL

greater infusion of colloid in the initial preoperative preparation explains the 165.0-mL greater postoperative chest tube drainage and the 0.73-unit greater transfusion of packed red blood cells. The hypothesized mechanism for this effect is the greater volume of colloid causing expanded plasma volume, increased clotting factor dilution, decreased blood viscosity, and increased venous return and cardiac output. These mechanisms might be believable if the 287.2-mL increase was from a baseline of 0 mL. Instead, it represents a 24.1% increase from the 1192.3 mL of colloid administered to those in the albumin arm (see Table 1). Combining colloid with crystalloid and cell salvage, those in the hetastarch arm were infused with 4944.9 mL while those in the albumin arm were infused with 4693.7 mL, a difference of 251.4 mL or 5.4% of the volume of fluids administered in the perioperative period. We do not think this 5.4% difference in fluid administered is clinically significant. We also note that albumin was the only fluid administered after the first liter.

We agree with Dr McKay that the question of which fluids should be considered optimal for volume replacement in CABG procedures is not settled. We are now actively involved in designing further studies using associations with the outcomes examined in our study and other outcomes to examine the effectiveness and risks of using albumin versus other fluids in this clinical context. We trust that these and other studies will provide a methodologically sound evidence base that can be used to guide future clinical decision-making in this area.

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REGIONAL WALL MOTION ABNORMALITIES AND SCARRING IN SEVERE FUNCTIONAL ISCHEMIC MITRAL REGURGITATION: A PILOT CARDIOVASCULAR MAGNETIC RESONANCE IMAGING STUDY

To the Editor:

We read with interest Flynn and colleagues' article¹ concerning preoperative evaluation with cardiac magnetic resonance imaging (MRI) in patients undergoing coronary artery bypass grafting and mitral valve annuloplasty for ischemic mitral valve regurgitation (IMVR).

In our experience, cardiac MRI has been advocated for preoperative routine evaluation of patients with

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.

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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