the jaws of the bipolar clamp toward the coronary-free spot of the mitral annulus. This can be found toward P2 in right coronary dominance, toward P3 in codominance, and near the crux cordis in left coronary dominance. These facts are clearly shown in mitral annulus dissections, where the right coronary or circumflex artery circulates on the atrial side within the fat of the atrioventricular groove. Our study emphasizes Benussi and colleagues' concept of tailoring the ablation pattern to the coronary anatomy, but we profoundly disagree that the actual technology used in bipolar radiofrequency can reach the posterior mitral annulus. We have tested this with the 2 most referred bipolar clamps in the literature, Cardioblade (Medtronic, Minneapolis, MN) and AtriCure (AtriCure, Inc, Cincinnati, OH), in 8 explanted human hearts. Neither system reached the mitral annulus in any heart. Rigor mortis or contracture was not present in any heart at the time of testing because these hearts were studied soon after explantation and preservation had been at 4°C. I personally performed the studies in all of the 8 explanted hearts, and there was no rigor or rigidity that limited our clamp positioning. The hearts felt like they were cardioplegically arrested. Our findings are corroborated with the postoperative necropsy of a heart in which a bipolar radiofrequency ablation was performed under cardioplegic arrest. Macroscopic and microscopic studies confirmed that the ablation line did not reach the mitral annulus.

The difficulty of reaching the mitral annulus at the level of the posterior leaflet with a clamp can be described by 3 anatomic facts. First, there is an important discrepancy in muscle thickness between the atrial and the ventricular walls. Second, the muscle of the atrial wall is followed by the posterior mitral leaflet in the endocardial side of the thick ventricular wall. Third, at the epicardial level, fat covers the difference in thickness between both walls at the atrioventricular groove, mainly covering the atrial wall and hosting the coronary ar-

tery and sinus. Clamping the atrial tissue including the annulus would entail clamping the first millimeters of the ventricular wall, the last one being 9 to 11 mm thick and impossible to clamp with actual bipolar technology. This is why even in small left atria, and regardless of the length of the radiofrequency clamp, it is impossible to reach the mitral annulus at the posterior leaflet.

The results obtained by Benussi and colleagues³ using only bipolar radiofrequency technology are encouraging, but we doubt all patients in their report have the mitral line complete. Therefore, it is possible that this line is not as important as previously thought or that leaving a 4- to 8-mm gap in the line does not influence the results.

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CHANGES IN NONINFARCTED MYOCARDIUM EXPLAIN BENEFITS OF ON-PUMP BEATING-HEART CORONARY ARTERY BYPASS GRAFTING FOR ACUTE MYOCARDIAL INFARCTION

To the Editor:

We read with great interest the article by Miyahara and associates¹ comparing outcomes in patients undergoing emergency coronary artery bypass grafting (CABG) for acute myocardial infarction (MI), using on-pump beating-heart

(OPBH) surgery, with outcomes of conventional CABG using aortic cross-clamping and cardioplegia. This study is one of four published over the past 3 years reporting improved outcomes with this new technique.

These studies claim the avoidance of reperfusion injury after crossclamping of the aorta as the reason for the benefits seen. However, we believe an important concept has been overlooked here, namely, the effects of global ischemia on the noninfarcted myocardium. There is extensive evidence, especially from the work of Gudbjarnason and associates,² that the "normal," noninfarcted myocardium, remote from the infarct, undergoes profound physiologic and biochemical changes as a result of the infarct. The noninfarcted myocardium is far from normal owing to depletion of adenosine triphosphate and creatine phosphate, increased oxygen consumption, and a reduction in glycogen levels^{2,3} (Figure 1). These changes indicate a state of stimulated metabolism in response to the increased workload performed by the noninfarcted myocardium to compensate for the nonfunctioning, akinetic infarct. As a result, hypertrophy occurs in the noninfarcted portion in an attempt to maintain normal cardiac function.⁴ These metabolic and structural changes render the noninfarcted myocardium highly susceptible to the effects of ischemia, such as those induced by crossclamping the aorta.³

Many years ago, in our laboratory, we studied the effects of cardioplegic arrest on the recently infarcted heart in greyhound dogs.³ After cardioplegic arrest, we demonstrated a dramatic decrease in recovery of function in the recently infarcted hearts compared with recovery in noninfarcted hearts (18% recovery of prearrest function in infarcted hearts compared with 56% in noninfarcted hearts; $P < .001$). The implication from this work is that the avoidance of global ischemia is the major reason for the success of OPBH surgery after MI.

We have developed a novel technique of myocardial preconditioning

FIGURE 1. Myocardium after coronary artery occlusion. The infarcted myocardium is necrotic and akinetic. The larger noninfarcted myocardium undergoes physiologic and biochemical changes. ATP, Adenosine triphosphate; CP, creatine phosphate; MVO₂, myocardial oxygen consumption.

by 2 to 4 days of preoperative metabolic therapy with oral orotic acid as a means of protecting the recently infarcted heart during emergency cardiac surgery. Orotic acid is a pyrimidine precursor that can augment the rate of synthesis of RNA, proteins, and glycogen. Pretreatment with orotic acid, in animal models, improves the functional recovery of the infarcted heart after cardioplegic arrest (43% recovery of prearrest function in orotic acid-treated groups compared with 18% in untreated groups; $P < .001$).³

In conclusion, the results of Miyahara and associates are promising and provide practical evidence for the benefits of avoiding crossclamping of the aorta. These benefits can best be explained by an improved understanding of the changes that occur in the remote, noninfarcted myocardium. We have shown that these changes can be attenuated by preoperative metabolic therapy with orotic acid. We propose that the use of the OPBH technique, combined with metabolic strategies, could result in better recovery in patients undergoing cardiac surgery after MI than with either technique alone and could allow cardiac surgery after recent MI to be performed even more safely and effectively.

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Reply to the Editor:

We appreciate the comments of Varatharajah and Rosenfeldt on our article, "On-Pump Beating-Heart Coronary Artery Bypass Grafting After Acute Myocardial Infarction Has Lower Mortality and Morbidity."¹ The comments indicate the importance of preserving the function of noninfarcted myocardium, which is highly susceptible to the effects of ischemia, and represent theoretical grounds for the benefits of avoiding reperfusion injury after crossclamping the aorta during coronary artery bypass grafting (CABG) in acute myocardial infarction (AMI).

We are impressed by the fact that the metabolic² and structural³ changes in the myocardium after infarction were investigated many years ago during the early stages of cardiac surgery.

We have treated another 45 patients with AMI since our report and have applied this technique consistently. The results have been excellent, with an overall hospital mortality of 2.2%. The only patient who died had a left main trunk infarction and required percutaneous cardiopulmonary support preoperatively. We confirmed the reliability of this technique. Technological advances in devices such as a suction stabilizer, apical suction cardiac positioning device, intravascular shunt, and temporary occlusion device enable us to perform on-pump beating-heart surgery reliably and safely.

To save these critically ill patients, in addition to avoiding cardioplegic arrest, we would like to emphasize the need for cardiopulmonary bypass to compensate the residual myocardial function and visceral organ failure.

We know little about orotic acid. On the basis of experimental findings in dogs,⁴ it is attractive and may help to improve the results. In our series of patients with AMI, the mean time interval from the onset of AMI to CABG was 18 hours. Therefore, orotic acid can be administered only during the few hours that are available between admission and surgery, allowing one or two doses at most. Further study is required to determine whether preoperative metabolic therapy with oral orotic acid is effective in this clinical setting.

In summary, on-pump beating-heart CABG is the best form of surgical treatment for patients with AMI.

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