anastomosis of a coronary graft to the aorta. The results presented by Emmert and colleagues[1] for the HEARTSTRING device are equivalent to their aortic no-touch technique. Although we believe that anaortic OPCAB with total arterial revascularization should be the criterion standard, we also appreciate that some surgeons prefer not to perform a T or Y graft anastomosis to the left internal thoracic artery. In this case, the use of a device for proximal anastomosis (rather than use of a side clamp) should be considered for anastomosis of the graft to the aorta.

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Reply to the Editor:

We thank Edelman and colleagues for their comments and additions to our article.

The ROOBY trial, as the largest available prospective, randomized trial has been criticized for its limitations with regard to its significant selection bias, for mainly including low-risk patients.[1] The low off-pump coronary artery bypass grafting (OPCAB) case load of the involved surgeons was reflected by a conversion rate as much as 10-fold higher, less complete revascularization, and lower patency rates than the data from high-volume OPCAB centers. Another randomized trial focusing on graft patency after OPCAB and on-pump coronary artery bypass grafting confirmed that OPCAB was associated with a significantly lower overall graft patency rate and did not rule out a potential benefit for OPCAB with regard to major adverse cardiac and cerebrovascular events and neuropsychologic functioning.[2]

As pointed out by Edelman and colleagues, however, and as also presented in most of the available studies, the ROOBY trial importantly failed to use aortic no-touch, total arterial grafting strategies. The significant value of combining OPCAB and aortic no-touch, total arterial revascularization techniques has been repeatedly highlighted in several studies, which have reported significantly decreased rates for stroke, ranging from 0% to 1%.[3] In addition, in their recent meta-analysis, Edelman and associates[4] have demonstrated the potential benefit of aortic no-touch techniques relative to both conventional on-pump coronary artery bypass grafting and to partial clamping techniques during off-pump coronary artery bypass grafting.

We agree that the use of in situ grafts or composites should be the criterion standard whenever possible. The use of free grafts requiring a proximal anastomosis is preferable in some patients, however, and may be the preferable revascularization strategy for some surgeons as well. In this situation, the proximal anastomosis can be efficiently carried out by using a clampless anastomotic technique to avoid any aortic manipulation, yielding similar results for stroke as achieved with a no-touch, all arterial grafting approach.[5]

The risk of neurologic complications cannot be completely eliminated, even if a no-touch, total arterial off-pump approach is used. This risk also applies to interventional revascularization strategies, such as percutaneous coronary intervention, and seems to be associated with the underlying, individual risk profile defined by the general health condition and comorbidities of the patient. A no-touch, total arterial off-pump approach yields similar neurologic outcomes to those reported with percutaneous coronary intervention, and the often used argument of inferior neurologic outcomes after surgery can therefore be invalidated.

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IS ANNULOPLASTY ENOUGH FOR FUNCTIONAL TRICUSPID REGURGITATION?

To the Editor:

Congratulations to the authors for this study.[1] We benefited from this report, but we want to emphasize some

points that should be taken into consideration by the authors.

What are the body surface areas of the patients in both the treated and untreated groups? Did any tricuspid stenosis develop after the operation in the treated group? Is there any residual gradient or regurgitation in mitral valve after repair? We think the mortality rate would decrease with continuous blood cardioplegia or perfusion.

In addition, there was residual tricuspid regurgitation detected in 40% of cases, although an annular diameter of 22 mm was reached postoperatively in the treated group. Was persistence of pulmonary artery pressure (PAP) at 32 mm Hg due to residual mitral regurgitation or to mitral pressure gradient secondary to replacement? Was this a reason for residual tricuspid regurgitation as well? Could we answer these questions by dividing the patients into 2 groups, that is, mitral valve replacement and mitral valve repair?

Table 4 shows that mild tricuspid regurgitation could also be achieved with a PAP of 32 mm Hg. Do the authors think that an additional tricuspid annuloplasty technique might be necessary to obtain coaptation even at a PAP of 32 mm Hg?

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Reply to the Editor:

We thank Dr Kestelli and colleagues for their comments and am glad they appreciate our study.

The 2 groups of patients were comparable in terms of body surface area (1.76 ± 0.2 m^2 in the untreated group vs 1.78 ± 0.2 m^2 in the treated group), and no patient showed echocardiographic or clinical signs of tricuspid stenosis (mean gradient, 2.4 ± 0.6). We follow what Frater proposed[1] only because our results are satisfying. If we had been aware of an increased gradient across the tricuspid valve, Frater's concept (tricuspid annulus to be reduced to fit a number 25 size) would have been abandoned.

We did not include patients with any problem in the left side that could cause, by itself, persistence of functional tricuspid regurgitation (FTR). ''During follow-up, those patients who showed any sign of repair failure, prosthetic malfunction, or severe impairment of left ventricular function, events that could justify the development of additional functional tricuspid regurgitation (FTR), were excluded from the present study (n =15).'' This sentence is at the end of the paragraph ''Population'' (p 308).[2]

Dr Kestelli and colleagues are concerned about the value of systolic pulmonary pressure in group T (32 mm Hg), which seems close to normal to me. Pulmonary pressure does not normalize in most patients, and we do not need to advocate residual mitral regurgitation or high transmitral gradients to justify such a mean value. As stated before, patients who showed one of these patterns were excluded.

Tricuspid regurgitation (TR) depends on many factors that act at different levels (annulus, leaflets, ventricle), but we work mainly on the annulus. Ventricular factors, such as papillary muscle displacement with increased chordal tethering, are not addressed in tricuspid repair for FTR. For this reason, we have to accept results that are sometimes not perfect (do you really think that residual 1+ TR is a suboptimal result?). Furthermore, we have to remember that medical treatment influences FTR grade.

Excessively forcing the coaptation of the tricuspid leaflets will not ensure better results. There is always a compromise between the reduction of the annular size and the increased chordal tethering related to the movement of the anterior and posterior leaflets toward the septal leaflet. On the other hand, the association between annular size and FTR is not clear. We know that normal tricuspid annuli do not avoid the presence of FTR, even with a non-severe grade.[3,4] Sadeghi and colleagues[5] reported 27 patients with pulmonary thromboembolism, severe pulmonary hypertension, and severe TR who underwent surgery without tricuspid valve annuloplasty. In 19 patients (70%), the pulmonary pressure decreased by a mean of 49 mm Hg and TR was reduced to mild. In the remaining 30% of patients, TR remained severe and pulmonary pressure was reduced to 32 mm Hg, less than in the other group. In both groups, the annular size remained unchanged (4-mm reduction between pre- and postoperative echocardiograms) and similar (41 mm in the first group and 42 mm in the second group in the postoperative assessment). We can deduct that, even if the annulus remains dilated, this does not prevent TR reduction, but the mechanisms related to right ventricular reverse remodeling, after pulmonary artery pressure reduction of different degrees, are more important than the pure annular size.

In regard to the myocardial protection strategy, we thank Dr Kestelli and colleagues for their advice, but, as far as we know, there is no randomized trial that shows any significant benefit in using continuous blood cardioplegia (warm? cold? antegrade? retrograde?). Early mortality depends more on the quality of patients undergoing operation (eg, comorbidities, urgency or emergency, left or right ventricular function) rather than on the pure cardioplegic technique.