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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Reference

1. Calafiore AM, Iaco AL, Romeo A, Scandura S, Medduri R, Varone E, et al. Echocardiographic-based treatment of functional tricuspid regurgitation. *J Thorac Cardiovasc Surg*. 2011;142:308-13.

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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 ($1.76 \pm 0.2 \text{ m}^2$ in the untreated group vs $1.78 \pm 0.2 \text{ m}^2$ 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 proposed¹ 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 colleagues⁵ 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.

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Notice of Correction

Re: Chu LM, Osipov RM, Robich MP, Feng J, Oyamada S, Bianchi C, Sellke FW. Is hyperglycemia bad for the heart during acute ischemia? *J Thorac Cardiovasc Surg.* 2010;140:1345-52.

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