described previously in 1988 and 2003, is not a folding on the ventricular septum but rather a plication of the "ventricular" wall itself (Figure 1).

However, it is true that a large plication of the tricuspid annulus could induce a kinking of the right coronary artery in the groove. We experienced this situation, which was revealed by an electric pattern of posterior myocardial ischemia. Removal of the stitches restored normal electrocardiographic results.

Regarding the spatial relationship of the coronary artery with the ventricular wall, I would have been interested to know whether the posterior descending coronary artery could be located in the myocardium itself.

At present, our indication for plication of the atrialized right ventricle is the presence of a dyskinetic wall. We pay extreme attention to the plication suture to avoid direct injury or kinking of the coronary arteries.

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Reply to the Editor:

We thank Dr Chauvaud for his interest in our recent article¹ regarding the coronary blood supply of the right ventricular inferior wall in patients with Ebstein malformation. In the heart specimens we examined, the posterior descending coronary artery was in the epicardial fat of the interventricular

groove, as shown in Figure 2 in our article.¹ As Dr Chauvaud commented, the presence of an intramyocardial coronary artery such as that encountered occasionally in the left anterior descending artery of normally structured hearts² would be of interest. Other than a case reporting myocardial bridging in the left anterior descending and obtuse marginal branches,³ we are not aware of any specific association between Ebstein anomaly and myocardial bridging in the posterior descending coronary artery. This is certainly worthy of investigating in a large series. We agree with Dr Chauvaud that purely endocardial sutures will be safest from epicardially sited coronary arteries.

Regarding Dr Chauvaud's second comment, we concur that the original technique reported plicates the free wall and when made carefully, as advocated by the authors,^{4,5} can avoid harming the posterior descending coronary artery in the interventricular groove. Our study¹ draws attention to patients with rightward deviation, which occurs in 59% of heart specimens examined, who could be at risk of deep sutures but could be more vulnerable when the plication is folded onto the septum.

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ENDOVASCULAR TREATMENT FOR ACUTE TRANSECTION OF THE DESCENDING THORACIC AORTA

To the Editor:

We read with great interest the article by Botta and coworkers.¹ They concluded that in the presence of an inadequate proximal landing, conventional open surgical intervention still is a favorable option as an alternative to endovascular repair associated with surgical revascularization of the left subclavian artery. The authors report that this decision is strengthened by the excellent results in terms of morbidity and mortality of their group in open repair.² However, we disagree with their statement for several reasons.

First, the good results in terms of morbidity and mortality reported by their group were only in case of delayed open repair. In case of immediate surgical repair, they reported a rate of paraplegia or of paraparesis of 19.1% and a mortality rate of 19%. A recent meta-analysis³ of endovascular versus open repair for traumatic rupture of the descending thoracic aorta concluded that endovascular treatment of trauma to the descending thoracic aorta is an alternative to open repair and is associated with lower postoperative mortality and ischemic spinal cord complication rates. Midterm results of endovascular treatment for traumatic rupture of the descending thoracic aorta suggest that endovascular repair should be