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

proposed as a first-line treatment. We agree with Botta and coworkers¹ that left subclavian artery revascularization before endograft repair should be done to prevent neurologic complications. For hemodynamically stable patients, with an aortic transection located within the proximal landing zone length necessary for adequate fixation (<20 mm), we would not propose conventional open surgical intervention with revascularization of the left subclavian artery. Rather, we believe that a prophylactic transposition of the left subclavian artery can safely be performed associated with endovascular repair of the aortic transection.

Second, a delayed treatment increases the risks of three major complications: an unforeseeable risk of delayed free rupture of an initially stable aortic tear; (2) a progressive dilation of the involved aortic segment exerting compression on the trachea and the left main bronchus; and (3) the creation of a fibrous and calcified connection within the aortic wall itself, potentially modifying aortic compliance and compromising the success of endovascular treatment. A controlled hypotension may cause cerebral hypoperfusion among patients who have often had a cerebral trauma. In a hemodynamically stable condition, a delay in treatment of a few days, as we⁴ have previously reported, seems reasonable to treat the associated traumatic lesions. In our institution, we believe that endovascular management as soon as possible offers important advantages over a deliberate wait.

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Reply to the Editor:

We thank Drs Canaud and Alric¹ for their interest and comments regarding the article "Endovascular Treatment for Acute Traumatic Transection of the Descending Aorta: Focus on Operative Timing and Left Subclavian Artery Management."² In their letter they disagree with the conclusion of our article, that "in presence of inadequate proximal landing zone, conventional open surgery still remains a favourable option as alternative to EVAR associated to surgical revascularization of the left subclavian artery."

Traumatic aortic rupture has always been a controversial topic because of its complex management and high mortality. We agree with Canaud and Alric¹ that endovascular repair should be proposed as the first-line approach in all cases of acute traumatic ruptures that need immediate treatment because of its proved lower morbidity and mortality with respect to open surgery. Nevertheless, we cannot forget some limits of this outstanding technique. The unknown lifelong durability of stent-graft materials, the long-term patency of carotid subclavian bypass, and the need of lifelong yearly x-ray monitoring are unsolved questions of endovascular techniques that cannot be underestimated, especially in young patients.

Accordingly, we adopt early endovascular repair in all traumatic injuries with clinical/imaging findings of

impending rupture or in patients who need immediate treatment of severe orthopedic or abdominal-associated lesions. Conversely, stable patients with unfavorable anatomy for endovascular repair may also have the choice of a low-risk open surgery. The concept of delayed surgery of traumatic aortic rupture started in the 1980s in the attempt to reduce one of the highest mortality rates in cardiovascular surgery; delayed surgery has been a great advance, abating the mortality rate from 30% to less than 10% in the published series. Our center has been a pioneer of this surgical strategy.³ Free rupture is reported to be less than 5%, patients can be safely monitored with computed tomography or magnetic resonance imaging, avoiding any risk of sudden aneurysm expansion and treated when clinical conditions allow a safe open surgical repair.⁴ In their letter, Canaud and Alric¹ also affirm that the success of endovascular treatment could be compromised when post-traumatic aneurysm becomes chronic: Our experience in this field started in 1997 with a patient with a chronic post-traumatic aneurysm, who is still safe and well. We did not observe any early and late treatment failure, regardless of the aneurysm age.⁵

The era of modern medicine and surgery offers multiple treatment options, each with its advantages and disadvantages, and every patient's individual clinical, anatomic, and human profile should be considered, avoiding absolute rules. In this view, the best option is, and always will be, to have more than one option.

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