SURGICAL APPROACH TO AORTIC VALVE DISEASE WITH CONCOMITANT DILATION OF THE PROXIMAL AORTA

To the Editor:

We read with interest the excellent article by Lee and colleagues,¹ in which an aggressive surgical strategy toward concomitant dilatation of the proximal aorta in patients with aortic valve disease was not recommended, even in the presence of moderately dilated aorta. We agree with their conclusions up to a point, and we therefore would like to report our personal experience in a much smaller cohort of 38 patients with slightly to moderately dilated proximal aortas (from 40 to 55 mm) and aortic valve disease who for several reasons were submitted to just isolated aortic valve replacement and were subsequently followed-up (median duration 36 months).²

Briefly, we identified 3 different behaviors of the proximal aorta during the follow-up: (1) no significant change, (2) increased growth velocity of the aortic diameter without reaching the limit of 55 mm (the commonly accepted indication to an ascending aorta replacement), and (3) increase of the proximal aorta reaching 55 mm or actual undergoing rupture or dissection. Of 36 early survivors, 3 fell into this third group (8.3%; linearized incidence of 2.3%/patient-y), because either the aortic diameter reached 55 mm or larger, precipitating elective reoperation (2 cases) or acute aortic dissection occurred (1 case).

In our regression model, a steep increase in the aortic expansion rate was correlated with a preoperative diameter of 50 mm or more ($R = 0.83$; $P = .0001$). We therefore advocated replacement (or at least a reduction plasty) of the ascending aorta at a level of approximately 48 to 50 mm, even in patients with no additional risk factors, such as the presence of a bicuspid aortic valve or Marfan syndrome.

Because the combined operation can be performed without significant additional risks while carrying the important advantage of eliminating the threat of later reoperations or acute aortic syndromes, we believe that an aggressive approach is reasonable when dealing with the common setting of concomitant aortic valve and proximal aorta disease. Patients with additional risk factors should probably be submitted to ascending aorta replacement even at lower levels of dilation (approximately 40–45 mm).

Probably a less aggressive strategy might be justified in cases of isolated aortic stenosis with mild dilatation of the ascending aorta. From our standpoint, in cases of aortic stenosis, the immediate resolution of the fluid dynamics abnormality that led to aortic expansion, obtained with the valve replacement, might be sufficient to prevent further dilatation; on the other hand, in patients with prevalent aortic regurgitation, the volume overload still persists after valve replacement, perpetuating the hemodynamic harm to the aortic wall. Actually, in our series the aortic growth velocity was -0.6 ± 1.5 mm/y in patients with prevalent stenosis and $+1.4 \pm 4.8$ mm/y in patients with prevalent regurgitation. This difference was, however, only marginally significant ($P = .10$). Indeed, in the group with isolated aortic valve replacement, the expansion rate reported by Lee and colleagues¹ is exactly the same described by us in the aortic stenosis group of our series. We therefore wonder whether they tried to identify a different behavior in the expansion rate of patients with stenosis versus those with prevalent regurgitation. Perhaps different approaches to these distinct conditions would be reasonable.

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VARIOUS SURGICAL APPROACHES TO CORONARY ARTERY ANEURYSM

To the Editor:

We read with great interest the article by Daralammouri and colleagues¹ on surgical treatment of a giant right coronary artery aneurysm. In this report, they described surgical treatment including resection of aneurysm and proximal-distal ligation with coronary artery bypass grafting in the proximal huge right coronary aneurysm. We congratulate Daralammouri and colleagues¹ on their successful treatment. We would also like to add a short comment about the choice of surgical therapy.

Surgical intervention is a reasonable approach because of the morphologic variability and enlarged size of these aneurysms. The main purpose of surgical treatment in coronary artery aneurysms is to prevent their rupture and the patient's death, as well as to avoid thrombosis and related coronary embolization.^{2,3}

There are various surgical approaches, including resection and plication of the aneurysm, graft interposition, end-to-end anastomosis technique, and ligation with distal bypass grafting.^{2,3} The surgical technique is still controversial, and there is no consensus regarding the optimal surgical strategy.

We used resection and end-to-end anastomosis techniques for a huge left anterior descending coronary artery (LAD) aneurysm. We preferred end-to-end anastomoses for the LAD rather than the use of the left internal thoracic artery because the diameter of the LAD was 4 cm, so the use of the left internal thoracic artery could have led to a size mismatch and early graft failure. In this technique, to avoid of bleeding from the suture line or occlusion caused by traction on the LAD, we mobilized the LAD sufficiently and checked for any resulting tension after the anastomoses.⁴

The resection and end-to-end anastomosis technique can be applied with confidence and is a feasible choice among all the available techniques. Furthermore, this technique is advantageous because of native-to-native coronary anastomosis. A variety of surgical techniques should be considered for treatment of huge coronary artery aneurysms.

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THE IMPORTANCE OF PULMONARY ARTERIAL PRESSURE FOR THE TREATMENT OF TRICUSPID REGURGITATION

To the Editor:

We read with great interest the report by Kim and colleagues,¹ in which they evaluated the outcomes of 51 patients who underwent tricuspid valve surgery for severe isolated tricuspid regurgitation (TR). They concluded that preoperative anemia, renal or hepatic dysfunction, right ventricular dilatation, and severe TR after tricuspid valve surgery were significant factors for determination of poor outcome.

TR is more often a secondary rather than a primary valve lesion and usually develops in association with pulmonary hypertension in patients with left-sided cardiac disease (functional TR). Pulmonary arterial pressure (PAP) increases during left-sided cardiac disease and causes elevation of right ventricular pressure. Secondary TR is a consequence of tricuspid annular dilatation and increased tricuspid leaflet tethering in relation to this right ventricular pressure overload. It is also possible to see functional TR in cases of increased PAP without left-sided cardiac disease (eg, asthma, chronic infection, smoking). In the evaluation of the study from Kim and colleagues,¹ we noted that the etiology of TR was unclear. Only 15.7% of patients reported a blunt chest trauma as the etiology. In addition, Kim and colleagues¹ also reported that none of the patients had significant left-sided cardiac disease. They did not report the value of PAP. The frequency of primary TR is thus uncertain. As we mentioned previously, PAP may be elevated for various reasons, and functional TR may occur through a mechanism other than primary valve lesion. Surgical approach to tricuspid valve repair and survival may be changed by the etiology of TR. Functional TR in response to

increasing PAP must be removed to reach a successful evaluation of valve surgery for primary TR. Kim and colleagues¹ reported preoperative right ventricular dilatation to be a criterion for poor outcome. Perhaps patients with right ventricular dilatation also had increased PAP. In addition, they reported moderate-to-severe TR in 41% of cases in a few years. This may have been occurred through the mechanism that we have discussed here.

We believe that PAP is a very valuable datum in the diagnosis and treatment of TR. Determination of the etiology of TR is essential for correct and complete treatment with good long-term results.

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IMPAIRMENT OF TRICUSPID ANNULAR PLANE SYSTOLIC EXCURSION AND TRICUSPID ANNULAR PEAK SYSTOLIC VELOCITY AFTER ARTERIAL SWITCH OPERATION

To the Editor:

We read with interest the article "Left and right ventricular performance after arterial switch operation" by Klitsie and colleagues.¹ In our opinion, this is an interesting study describing the long-term effects of heart surgery on remodeling of the right ventricle (RV) and the left ventricle in children after the arterial switch operation for