agree completely that the mitral annulus should be supported somehow, but our experience suggests that suture annuloplasty gives good results without any ring.

After our initial experience with suture annuloplasty, published 8 years ago,² we performed several hundred mitral repairs without annuloplasty rings and with good early and midterm results. There are two important factors in achieving a good result with this method. The first is the suture material. Both polypropylene (Prolene; Ethicon, Inc, Somerville, NJ) and polyester (Ethibond; Ethicon) sutures were not reliable. Prolene was too elastic, and in some cases the 3-0 Prolene thread elongated, causing redilatation of the mitral annulus. In a few cases, the Prolene suture ruptured, causing recurrent mitral regurgitation. Ethibond was difficult to use in achieving proper and equal tensions on the suture line and adequate shortening of the annulus. We had superior experiences with expanded polytetrafluoroethylene (Gore-Tex; W. L. Gore & Associates, Inc, Flagstaff, Ariz) sutures, as also reported by others.³ The other important factor is that the sutures be placed properly into the annulus. Neither sutures in the left atrium in the vicinity of the annulus nor sutures in the proximal parts of the valve cusps give good results. This should hold true even with ring annuloplasty.

Suture annuloplasty actually has advantages relative to ring annuloplasty. Transvalvular gradient is significantly lower and mitral valve area is significantly larger, without differences in mitral regurgitation.⁴ The mitral annulus has a natural flexibility, which results a 26% dilatation in diastole.⁵ The natural flexibility of the mitral annulus is conserved with suture annuloplasty. Suture annuloplasty is also a simpler and much cheaper method of achieving stability of the mitral annulus.

In our experience, if mitral repair is done, mitral annuloplasty should be performed, even if the annulus is not significantly dilated. It is not absolutely necessary to use an annuloplasty ring, as Flameng and coworkers recommend¹; suture annuloplasty can give a good result as well. Their statement that "[N]onuse of annuloplasty ring is a risk factor for recurrent mitral valve regurgitation" would be true only if the comparison included suture annuloplasty as well. We

have not compared suture annuloplasty with annuloplasty rings, but in accordance with our experience, we would recommend the following conclusion instead: "Lack of stabilization of the mitral annulus increases the risk of recurrent mitral valve regurgitation."

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Reply to the Editor:

I thank Szentkirályi, Péterffy, and Galajda for their interest in our study on the durability of mitral valve repair in Barlow disease. The point they try to make in their letter is that mitral annuloplasty in myxoid mitral valve degeneration does not necessarily have to be a ring annuloplasty. This might be correct; who knows?

The problem is that, nowadays, when you make a statement, hard data are required to convince the medical and scientific community to agree with you. In our study on the recurrence of mitral valve regurgitation after successful repair of Barlow disease

and fibroelastic degeneration, we presented and analyzed extensive preoperative, intraoperative, and postoperative data, including long-term echocardiographic follow up. In our study, patients received either a ring annuloplasty or no annuloplasty at all. We did not study any other form of annuloplasty, and therefore it would not be correct to draw such a general conclusion as, "Lack of stabilization of the mitral annulus increases the risk of recurrent mitral valve regurgitation." To advocate this statement, one should test every possible form of annuloplasty and compare them all with no annuloplasty. In their own experience, Szentkirályi, Péterffy, and Galajda mention that some materials used for suture annuloplasty, such as polypropylene and polyester sutures, cause recurrent regurgitation. Is this not why the annuloplasty ring was developed?

Before we conclude that "Lack of stabilization of the mitral annulus increases the risk of recurrent mitral valve regurgitation," I invite Szentkirályi, Péterffy, and Galajda to analyze their data on suture annuloplasty in a scientific way and compare them with results of repair procedures without annuloplasty, rather than relying on their own experience, even with several hundred patients. I am well aware of the importance of personal experiences of renowned surgeons such as our Hungarian colleagues, and I appreciate the opportunity to share these experiences. Before introducing a concept into current praxis, however, I prefer data-based, scientific proof.

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Questions on the angular (Gothic) arch configuration and its applicability after type I aortic dissection repair

To the Editor:

I enjoyed the recent article "Angular (Gothic) Aortic Arch Leads to Enhanced Systolic Wave Reflection, Central Aortic Stiffness, and Increased Left Ventricular Mass Late After Aortic Coarctation Repair: Evaluation With Magnetic Resonance Flow Mapping," by Ou and associates¹ in a recent issue of the *Journal*.¹ Why were patients

with bicuspid aortic valves excluded from the study? Did patients with Gothic arches and bicuspid valves have even greater changes? Did any of the patients with Gothic arch and a tricuspid aortic valve have aortic insufficiency or require valve replacement/repair?

Approximately 15% of patients who are status-post repair of type I aortic dissection require replacement of the proximal descending aortic owing to increasing diameter on long-term follow-up. It has also been my impression that those patients who have repair of type I aortic dissection with replacement of the ascending aorta and hemiarch repair have a greater incidence of progressive aortic insufficiency and may require aortic valve replacement or aortic root replacement. This cohort is in contrast to those patients with type I aortic dissection who simply had ascending aortic replacement and appeared to have a lesser incidence of aortic insufficiency and valve or root replacement over the long term.

Might the hemodynamic changes that were documented in the pediatric angular Gothic arch be present in this status-post aortic dissection group and predispose to aortic valve insufficiency and dilation of the proximal portion of the distal descending thoracic aorta? Might this effect be more pronounced in the hemiarch repair that predisposes to an angular Gothic arch configuration? Replacement of the ascending aorta only (without hemiarch repair) usually preserves the concave configuration of the ascending aorta and its gentle curve into the transverse arch. Ascending aorta and hemiarch replacement typically has an angulated Gothic configuration rather than the gentle curve that nature favors.

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Aprotinin and renal dysfunction: The role of exposure to angiotensin-converting enzyme inhibitors

To the Editor:

I read with great interest the recent article by Pagano and colleagues¹ detailing their favorable experience with aprotinin in a large patient series (N = 7836: 1998–2006) from a single institution. The authors demonstrated that aprotinin exposure did not significantly affect the incidence of postoperative renal dysfunction.

Careful review of this interesting paper reveals, however, that although hypertension was significantly more common in the aprotinin cohort (63.3% vs 55.1%; $P < .001$), the incidence of exposure to angiotensin-converting enzyme (ACE) inhibitors is not indicated. This is an important confounder, inasmuch as ACE inhibitors in conjunction with aprotinin have been shown to be associated with renal dysfunction after cardiac surgery.^{2,3}

As a consequence of this observation, I have the following questions:

1. What was the incidence of ACE inhibitor therapy in the aprotinin cohort?
2. Was exposure to ACE inhibitors significantly different in the aprotinin cohort?
3. What was the percentage of off-pump coronary bypass procedures, given that aprotinin and ACE inhibitors have recently been significantly associated with postoperative renal dysfunction in this patient subgroup?³
4. Could the incidence of ACE inhibitor exposure have confounded the results of the study, given the findings from the literature?^{2,3} For example, could a lack of renal toxicity from aprotinin be explained by a low incidence of exposure to ACE inhibitors in the aprotinin cohort?

I congratulate the authors again on their important contribution. I look forward to their feedback about the impact of ACE exposure and aprotinin on renal dysfunction after cardiac surgery.

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Reply to the Editor:

Dr Augoustides raises an appropriate point. It is the policy in our unit to discontinue angiotensin-converting enzyme inhibitors or equivalents 24 hours before the operation and for patients with impaired renal function, at least 48 hours before the operation. However our database does not have information on compliance to this policy. Therefore, we could not address this issue in our article.¹

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Prosthetic valve thrombosis: A regimen of treatment with low-dose and longer-course using recombinant tissue-type plasminogen activator is a promising protocol

To the Editor:

Despite the progress in anesthesia, cardiac surgery, and perioperative care, the therapeutic decision in prosthetic valve thrombosis (PVT) remains in discussion.

In recent years thrombolytic therapy has won acceptance, and for many it is the first