HIT who have required valve replacement. Bivalirudin's half-life of 25 minutes, the ability to monitor activated clotting time, and the lack of cross-reactivity with heparin antibodies were advantages that convinced my group to use it for these patients with HIT. The patients did well, requiring an average of 2 units of blood per patient.

My suspicion is that off-pump revascularization with bivalirudin rather than danaparoid would result in lower use of blood products. It is likely that even on-pump coronary artery bypass grafting with bivalirudin as the anticoagulant will result in lower blood product use in the HIT cohort.

Frank A. Baciewicz, Jr, MD
Cardiothoracic Surgery
Wayne State University
School of Medicine
Detroit, MI 48201

References

1. Carrier M, Robitaille D, Perrault LP, Pellerin M, Page P, Cartier R, et al. Heparin versus danaparoid in off-pump coronary bypass grafting: results of a prospective randomized clinical trial. *J Thorac Cardiovasc Surg*. 2003;125:325-9.
2. Vasquez JC, Vichiendilokkul A, Mahmood S, Baciewicz FA. Anticoagulation with bivalirudin during cardiopulmonary bypass in cardiac surgery. *Ann Thorac Surg*. 2002;74:2177-9. doi:10.1016/S0022-5223(03)01309-6

Reply to the Editor:

I thank Dr Baciewicz for his comments regarding our article and thank you for the opportunity to respond to him. We chose to compare low dose of the anti-Xa inhibitor danaparoid with standard heparin in off-pump coronary artery bypass grafting because of our significant clinical experience with the former drug. Although it is not superior to standard heparin, low-dose danaparoid offers a safe alternative for patients undergoing off-pump coronary artery bypass grafting when heparin is contraindicated.

My group and I have read with interest the reports on bivalirudin, but we remain concerned by the limited clinical data available. In fact, we found only two reports of cardiac surgical patients in the literature, with one of the patients showing a large blood drainage through the chest tubes.[1,2]

Our current options for patients with heparin-induced thrombocytopenia are to use low-dose danaparoid and off-pump coronary artery bypass grafting or to wait for disappearance of the antiplatelet antibodies and use a standard protocol of heparin.

Michel Carrier, MD
Department of Surgery
Montreal Heart Institute
Research Center
Montreal, Quebec, Canada

References

1. Vasquez JC, Vichiendilokkul A, Mahmood S, Baciewicz FA. Anticoagulation with bivalirudin during cardiopulmonary bypass in cardiac surgery. *Ann Thorac Surg*. 2002;74:2177-9.
2. Davis Z, Anderson R, Short D, Garber D, Valgiusti A. Favorable outcome with bivalirudin anticoagulation during cardiopulmonary bypass. *Ann Thorac Surg*. 2003; 75:264-5. doi:10.1016/S0022-5223(03)01311-4

Apical versus basal partial ventriculectomy

To the Editor:

With regard to the recent study of Koyama and colleagues published in the *Journal of Thoracic and Cardiovascular Surgery*,[1] your readers should be aware that the authors have done no more than confirm the previous observations of Savage and colleagues,[2] namely, that reducing volume by constricting or resecting the apical half of the ventricular cone has no significant effect on ventricular pump function. Their findings also confirm extensive clinical studies[3-6] showing that reduction of the radius of the ventricle in its upper two thirds improves pump function, provided that the ventricle had been markedly dilated prior to such an intervention. Thus, simply by using a figure-of-eight symmetrical technique of resection, Konertz and colleagues[5] have achieved results that surpass the current success of cardiac transplantation.

Your readers should also note that the technique used by the Japanese group to induce cardiac failure[1] produced minimal alterations in left ventricular function. Thus, having reduced the radius of the left ventricle, the diameter in their experimental study was smaller than under control conditions. As is well established, however, any persistent therapeutic effect of reducing ventricular radius in the clinical situation is dependent in the degree of preexisting ventricular dilation.[3-6]

Furthermore, due to the beta-blockade used as part of the experimental setup, the ensuing bradycardia will have prevented their hearts from compensating adequately for the confined stroke volume, as would have occurred under physiological conditions simply due to an increase in heart rate. It is irrelevant, therefore, to measure cardiac output under these experimental conditions. At all events, stroke volume increased by one quarter when the radius was reduced along the basal two thirds of the heart.

There are then several other problems with the description and interpretation of the Japanese group[1] that need to be drawn to the attention of your readers. It is incorrect to state that Batista advocated the apical region of the left ventricle as a primary area for resection. His primary intention was simply to reduce the radius of the dilated left ventricle. Furthermore, contrary to the conclusion drawn by the authors,[1] the experimental results show clearly that plication of the apical segment produces no positive therapeutic effect. Nor, contrary to the assertions made by Torrent-Guasp and his colleagues,[7,8] does such plication have any major detrimental impact on global ventricular pump function. When considering the differences observed in left ventricular function after apical or subbasal reduction of radius in this study, we need to remember that it is the extensive circular muscular layer enclosing the upper two thirds of the left ventricular cone that is largely responsible for left ventricular ejection. By reducing its radius, working conditions for the left ventricle are improved, thus ameliorating its pump function. This positive effect, however, is mitigated by plication of the interpapillary segment as performed by the Japanese investigators, as this procedure plicates also the marginal arteries. As has been shown,[9] the resulting ischemic damage extends well beyond the plicated segment. Indeed, such collateral damage may well have been more significant in those hearts that were plicated up to the base in the Japanese study as compared with those plicated only along the apical half. The positive effect of reducing left ventricular radius probably would have been more pronounced had a less traumatic

technique been used with the aim of preserving the marginal arteries.

In summary, therefore, the authors have done no more than confirm that resection or constriction at the apex has a strictly cosmetic effect on ejection fraction and a negligible effect on global left ventricular function. There is no need to invoke any pivotal impact of the spiraling muscles presumed to be involved in ventricular filling so as to explain the lack of therapeutic benefit. Indeed, the authors have provided no evidence of a major impairment of the dynamics of the ventricular filling apart from a moderate increase in the filling pressure of the left ventricle and a decrease by one tenth in stroke volume. In our opinion, this latter finding is more likely due to the overcorrection of the volume of the ventricle by reducing its long axis. They should remember that clinical results have now provided ample evidence that when the left ventricle is dilated, resection of any part of its walls, including the septum, is tolerated without impairing left ventricular function.[3-6] Indeed, a marked improvement in left ventricular function is achieved whenever the radius of the upper two thirds of the left ventricle is significantly reduced. The conclusions drawn by the authors, namely, that surgical intervention on the left ventricular apex is markedly detrimental, are not, in our opinion, supported by their experimental evidence.

P. P. Lunkenheimer[a]
R. H. Anderson[b]
Klinik und Poliklinik für Thorax-, Herz- und Gefäßchirurgie[a]
University Münster
Münster, Germany
Cardiac Unit, Institute of Child Health[b]
University College
London, United Kingdom

References

1. Koyama T, Nishimura K, Soga Y, Unimonh O, Ueyama K, Komeda M. Importance of preserving the apex and plication of the base in left ventricular volume reduction surgery. *J Thoracic Cardiovasc Surg*. 2003;125:669-77.
2. Savage EB, Downing SW, Ratcliffe MB, et al. Repair of left ventricular aneurysm. Changes in ventricular mechanics, hemodynamics, and oxygen consumption. *J Thorac Cardiovasc Surg*. 1992;104:752-62.
3. Batista RJV, Santos JLV, Takeshita N, et al. Partial left ventriculectomy to improve left ventricular function in end-stage heart disease. *J Card Surg*. 1996;11:96-7.
4. Dowling RD, Koenig SC, Ewert DL, Laureano MA, Gray LA. Acute cardiovascular changes of partial left ventriculectomy without mitral valve repair. *Ann Thorac Surg*. 1999;67:1470-2.
5. Konertz W, Khoynezhad A, Sidiropoulos A, Borak V, Baumann G. Early and intermediate results of left ventricular reduction. *Eur J Cardiothorac Surg*. 1999;15(Suppl 1):S26-30; discussion S39-43.
6. Suma H, the RESTORE GROUP. Left ventriculoplasty for nonischemic dilated cardiomyopathy. *Semin Thorac Cardiovasc Surg*. 2001;13:514-21.
7. Torrent-Guasp F, Ballester M, Buckberg GD, et al. Spatial orientation of the ventricular muscle band: physiologic contribution and surgical implication. *J Thorac Cardiovasc Surg*. 2001;122:389-302.
8. Buckberg GD, Clemente C, Cox JL, et al. The structure and function of the helical heart and its buttress wrapping. Concepts of dynamic function from the normal macroscopic helical structure. *Semin Thorac Cardiovasc Surg*. 2001;13:342-57.
9. Lunkenheimer PP, Redmann K, Cryer CW, et al. Late ventricular structure following partial left ventriculectomy: a case report. *Ann Thorac Surg*. 2000;69:1257-9.

doi:10.1016/S0022-5223(03)01330-8

Reply to the Editor:

Savage and colleagues' study[1] demonstrated that volume reduction surgery (VRS) in a model of left ventricular (LV) aneurysm without global LV dysfunction plicated only LV apex. On the other hand, apex-sacrificing VRS in our series reduced the diameter of not only LV apex but also LV mid-portion in a model of global LV dysfunction. The effects of volume reduction in akinetic or dyskinetic area are different from hypokinetic area.[2] Drs Lunkenheimer and Anderson support reduction of the radius of the ventricle in its upper two thirds (ie, basal). We agree with them on that point, and reduction of the upper two thirds of left ventricle is the same as our apex-sparing VRS. In clinical series, however, no one has described or advocated reduction of the radius of the ventricle in its upper two thirds. The letter by Drs Lunkenheimer and Anderson focused on the issue in LV aneurysm or ischemic cardiomyopathy (raised by Savage and colleagues[1]), but the disease that we studied via this animal model was dilated cardiomyopathy (ie, nonischemic). From this viewpoint, their criticism is interesting and important but misdirected.

Konertz and coworkers[3] reported excellent results of VRS but they also did not described reduction of the upper two thirds of left ventricle, and a majority of their cases are ischemic cardiomyopathy. Again, our study was focused on the dilated cardiomyopathy (nonischemic cardiomyopathy, a different disease). It is difficult to adopt their results to our study.

Theoretically the therapeutic effect of reducing ventricular radius is at least in part dependent on the degree of preexisting ventricular dilation. In the clinical setting, however, operative mortality is high and LV redilatation and dysfunction are not rare after VRS.[4-10] This suggests that the theoretical concept is not necessarily reproducible in the clinical situation and that the way to excise and repair the LV is also important. Although there are some limitations due to the model of acute heart failure induced by β-blocker, the heart rate before and after VRS was statistically no different between the 2 groups. LV contractility in apex-sparing VRS was shown to be superior to apex-sacrificing VRS, and LV enddiastolic pressure was lower in apex-sparing VRS. Those differences between the 2 groups were significant.

Batista's group described an incision made at the apex of the left ventricle. In our article,[2] we never stated that Batista and colleagues advocated the apex resection, but they did not try to preserve the apex as much as possible. In fact, Lunkenheimer and colleagues[11] use the term "oyster-shaped excision" in their case report, following the scheme of Batista's operation by widely resecting the LV apex. Throughout our article we tried to show the potential pitfall of the great operation described by Batista and colleagues and to improve it. There would be some ischemic damage extending beyond the plicated segment in VRS, and we have described plication effects in the adjacent area in our study limitation. It is unknown, however, whether the ischemic damage follows according to the position of resection or plication area. However, this potential concern of compromising the marginal arteries is equally applicable to conventional Batista procedure. Further investigation would be warranted.

We have already demonstrated these 2 types of VRS in a model of chronic dilated cardiomyopathy.[12] Elevation of LV enddiastolic pressure and LV redilatation were