long must anticoagulants be maintained? Derumeaux et al. conclude that one of the predictive factors for a thromboembolic episode is the time since transplantation. What is their explanation for this finding?

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References

A reply

We read with interest the comments of Fernandez Gonzalez and Herreros concerning our article.[1] This letter is to answer their questions:

Question 1: We did not find any relationship between recipient atrial rhythm and the presence of atrial thrombi. At the time of the study, only seven patients demonstrated atrial fibrillation in the recipient atrium as proven by electrophysiological evaluation. Among these patients, only two had a left atrial thrombus.

Question 2: Thrombi were diagnosed 35 months after surgery at the time of the TEE study but several arguments support the fact that they occurred earlier. One of these arguments is the time at which thromboembolic episodes occurred. We recently reported on a larger series of transesophageal examinations comparing standard transplantation by the Lower and Shumway technique with total transplantation by bicaval anastomosis.[2] We demonstrated that thromboembolic events occurred only in patients with standard transplantation and that five of the 11 thromboembolic events occurred within the first 6 months, suggesting therefore a very early formation of atrial thrombi. In this paper, we also demonstrated that bicaval orthotopic heart transplantation was highly effective in avoiding thrombi formation and in restoring normal atrial function. We therefore disagree with the conclusions of El Gamel.[3]

Question 3: Regarding treatment, we used fluindione (international normalized ratio 2-3 to 3) and we replaced fluindione by ticlopidine only if the thrombus had completely disappeared at the 6-month TEE control.

Question 4: In this series of 64 patients, statistical analysis was the only predictive factor for a thromboembolic episode; however, the time elapsed since transplantation was significantly higher in cases with embolism. One explanation for this observation is the fact that with time, the retained atria have rigid and fibrotic walls which are less compliant, therefore increasing blood stasis and promoting both spontaneous echo contrast and clotting in the left atrium. However, the statistical results depend on the series of patients. In a larger series (n = 75)[2] there was no predictive risk factor for thromboembolic events as regards time from transplantation, left atrial diameter, left ventricular ejection fraction and haemodynamic data.

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References

ST segment variations in lateral limb leads

I read with great interest the article by Hasdai et al.[1] and editorial by Tamura[2], correlating the ST segment variations in lateral limb leads with the site of coronary obstruction in inferior wall acute myocardial infarction.

As Dr Tamura points out, the right precordial leads were not analysed. The value of lead V4R in acute MI should be mentioned when identifying the occluded coronary artery, the presence or absence of right ventricular infarction and the patients at risk of AV block. In lead V4R, the ST segment is elevated when the proximal right coronary artery is occluded. It becomes a positive T wave when the distal right coronary artery is occluded; and when the circumflex artery is occluded, the T wave is inverted.

Thus I commend Hasdai and colleagues for an interesting article, but if the analysis of both precordial and lateral leads had been combined this would have been of greater interest as regards the culprit artery and site of obstruction.

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References

Implantable defibrillatory shocks

D'Avila et al.[1] report that changes in autonomic tone in the early morning play a role in the circadian variations of sudden death. The neurobiological manifestations are suggested by diurnal variations in serotonergic-mediated inhibition of dopamine lateralized to the right hemisphere. This hypothesis is supported by optimal response organization in