of choice for left ventricular pacing in cardiac resynchronization.

References


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Reply to the Letter to the Editor

Reply to Garcia-Bengochea et al.

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Keywords: Surgically placed epicardial LV-lead; Cardiac resynchronization therapy; Doppler echocardiography; Medtronic 10626 Lead Implantation Tool

I agree with Dr Garcia-Bengochea [1] that Doppler echocardiography is important in management of patients referred to cardiac resynchronization therapy (CRT) and indeed QRS duration alone is only weakly correlated to CRT response and cardiac improvement. Optimal lead positioning is one of the most important tasks in CRT and Doppler echocardiography may help to reduce nonresponders or prevent from a worsening effect of biventricular pacing: e.g. a proarrhythmogenic effect may occur due to incorrect lead positioning and consecutive interventricular dysynchrony.

Personally, I am a fan of video-assisted or robotic approaches in CRT [2], but these techniques are sometimes time- and money-consuming. Therefore, cost-effectiveness may be an important restrictor in adoption and widespread of these techniques. Single lung ventilation with double lumen tube is mandatory; with careful monitoring it will be well tolerated even in presence of severe congestive heart failure. Nevertheless, most colleagues prefer a minithoracotomy using the Medtronic 10626 Lead Implantation Tool for the Medtronic 5071 Epicardial Pacing Lead. However, the 5071 lead faces some drawbacks: it is not steroid eluting and it is only an unipolar lead. Further advances and new prototypes for next generation of epicardial leads are already in clinical testing.

References


Letter to the Editor

Simultaneous repair of post-infarct ventricular septal defect and coronary artery bypass grafting

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Keywords: Myocardial infarction; Ventricular septal defect; Coronary artery bypass grafting; Mid-term survival

We enjoyed reading the recently published article by Jeppsson and colleagues [1], which examined the national results in Sweden for surgical repair of post-infarct ventricular septal defects (VSD). They correctly concluded that the long-term survival of these patients was limited by pre-existing coronary artery disease (CAD), post-operative renal failure, and the presence of a residual post-operative shunt. Interestingly, Jeppsson showed that the risk of death increased with the number of coronary anastomoses performed and concluded from this that this was a reflection of the extent of CAD. However, their conclusions on the potential benefits of performing concomitant coronary artery bypass grafting (CABG) were inconclusive.

In our own series of post-infarct VSD patients within the northwest of England [2], we concluded that concomitant CABG offered a significant benefit with respect to mid-term mortality. Although our series of 65 patients was smaller than the 189 patients in Jeppsson’s series [1], we found that after adjusting for other risk factors for mid-term mortality (unstable angina (class IV), current smoking, and total occlusion of the infarct related artery), patients with concomitant CABG had a substantial reduced risk of death.
with a relative risk of 0.17 (95% confidence intervals 0.04-0.74; P=0.019) [2].

In our series [2], patients with concomitant CABG were more likely to have unstable angina than patients without concomitant CABG (50 versus 30%); and unstable angina was associated with the greatest risk of death during follow-up, with such patients being 12 times more likely to die than patients with stable angina. Jeppsson admits to limitations in their data collection, and may have missed variables related to mortality [1]. We note that angina status was not included, and we feel that this variable must be considered when balancing out differences between patients with and without concomitant CABG.

Two other large studies by Cox [3] and Muehrcke [4] have reported that concomitant CABG is beneficial. Muehrcke and associates [4] found that patients who had coronary artery disease outside of the infracted region fared significantly better, long term, if they were grafted. Pretre and colleagues, in a recent publication of 54 patients who underwent patch closure of the post-infarct VSD, showed that simultaneous myocardial revascularisation controlled the added risk of coronary artery disease [5].

From our own data and other published series, we would recommend that patients who present with post-infarct VSD who have multi-vessel disease be routinely revascularised.

References


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Keywords: Post-infarction ventricular septal defect; Coronary artery disease; Survival

We thank Dr Ramnarine and Dr Grayson for their comments about our series of 189 patients surgically treated for post-infarction ventricular septal defects [1]. We share Ramnarine and Grayson’s view that concomitant CABG should be performed in patients with post-infarction ventricular septal defect and significant coronary disease. However, from a scientific point of view, the support for this approach is weak. In their letter Ramnarine and Grayson refer to their own article [2] and to the works by Cox, Muehrcke and Pretre which indicates that concomitant CABG is beneficial, but do not mention the works by Labrousse, Deja and Dalrymple-Hay [3-5] that did not find any support for CABG. Interestingly, concomitant CABG was not a predictor of survival in the two largest series [1,3].

Ramnarine and Grayson also suggest that unstable angina should be included as a factor in multivariate analyses in post-infarction-VSD patients. Ramnarine and Grayson refer again to their own material of 65 patients [2]. First, it is not clear from the article when the period of unstable angina occurred. Was it before the myocardial infarction (MI), between MI and the development of VSD or after the VSD had been diagnosed and repaired? In addition, the imbalance between number of events and included factors in the analysis in Ramnarine and Grayson’s study must be considered. In their study, 12 early and 16 late events (deaths) occurred during the follow-up period while over 30 different factors were included in the stepwise analysis. This disproportion generates a substantial risk for random significance, may imply that true predictors are missed and necessitates a critical assessment of the data before publication. Notably, recognized risk factors for death after post-infarction VSD, such as inferior infarction and repair early after diagnosis, which repeatedly has been shown to influence outcome after surgical repair were not identified in Ramnarine and Grayson’s study. In contrast, their study identified smoking and unstable angina for the first time. The importance of these new potential predictors needs to be verified in larger studies with carefully selected variables.

References


Reply to the Letter to the Editor

Reply to Ramnarine and Grayson

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