Cardiac Resynchronization Therapy for Heart Failure

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Chronic heart failure is a common and serious disorder affecting the worldwide population. Prolonged QRS duration on surface ECG in patients with poor left ventricular (LV) ejection fraction was associated with poor outcome. Widened QRS complex represents both inter- and intra-ventricular conduction delays or electromechanical dyssynchrony, based on the various mapping and echocardiographic imaging techniques. By means of non-contact mapping, the activation sequences of the LV in patients with systolic heart failure and wide QRS complex were demonstrated to be highly heterogeneous with conduction block located in different regions of the LV. In addition, dispersion of timing to reach peak systolic contraction velocity in different LV segments have also been demonstrated by tissue Doppler imaging (TDI), further supporting the concept of asynchronous contraction between different LV segments. Such asynchronous contraction pattern contributes to mitral regurgitation, reduction in stroke volume and subsequently leading to deleterious LV remodeling. The proposed mechanism of benefit by cardiac resynchronization therapy (CRT) is to correct the LV dyssynchrony by pacing the right ventricular apex and lateral or posterolateral wall of the LV. Various randomized controlled trials have consistently confirmed the benefit of CRT in improving the symptoms, LV systolic function and reducing heart failure hospitalization. Survival benefit by CRT has also been demonstrated in COMPANION and CARE-HF trials. Based on the results from these large-scale randomized trials, the heart failure management guidelines in ACC/AHA and ESC have incorporated CRT as a class I indication for symptomatic patients with ejection fraction <35%, NYHA class III or IV and QRS duration >120ms on ECG despite optimal heart failure medication.

There are several unanswered questions about CRT. Around one-third of patients did not respond to CRT, the so-called non-responders. Identification of mechanical asynchrony by TDI and detection of scar tissue by MRI may have a role in selecting the CRT responder. Preliminary data has also suggested that CRT may be beneficial in patients with less symptomatic heart failure or at earlier stage of the disease. A substantial proportion of patients with low ejection fraction had LV dyssynchrony despite normal QRS duration. Preliminary data from our group has shown that CRT in these patients can result in clinical and echocardiographic improvement.

With advances in technology, the delivery of LV lead is much easier than those of early generation though the procedure is still not risk-free. Complications, though uncommon, related to positioning of the LV lead include coronary sinus dissection or perforation, lead dislodgement, diaphragmatic pacing and contrast nephropathy. The overall success rate for CRT implantation ranges between 90 to 95%.

In conclusion, CRT is an established non-pharmacological therapy for patients with low ejection fraction, NYHA class III or IV and wide QRS on ECG. Further studies are necessary to determine the role of the device in those with less symptomatic heart failure, moderate LV dysfunction and normal QRS complex.

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