in sinus rhythm. Evidence of ventricular dyssynchrony is required on 12-lead ECG (QRS duration > 120 ms) or echocardiographic evidence of mechanical dyssynchrony if the QRS duration is 120–149 ms (UK guidance only). The North American and European guidelines also recommend that there is evidence of LV enlargement (LV end diastolic diameter > 55 mm) [11, 12].

In these patients, bi-ventricular pacing significantly reduces mortality from progressive heart failure as well as all-cause mortality. It also reduces the number of hospitalisations due to heart failure, improves functional status (NYHA class, VO2 max and exercise tolerance) and improves quality of life.

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References


eComment: Does cardiac resynchronization therapy improve survival and quality of life in patients with end-stage heart failure?

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The beneficial impact of cardiac resynchronization therapy (CRT) on Heart Failure (HF)-related morbidity and further mortality [1] is attributed to the
improvement of AV, inter- and intra-ventricular conduction delays which occur in advance heart failure and desynchronize the mechanical activity of the ventricles, thus affecting their pump performance. It is notable that three major types of myocardial asynergy can occur in heart failure patients. One is a progressive loss of integrity of the myocardial collagen matrix, typical of the familial cardiomyopathies but common to all dilated cardiomyopathies. Disruption of the collagen network by altering theordinate cellular architecture impairs both the intra-ventricular conduction of the electrical impulses and the coordinated mechanical response of the ventricles. The consequences are both prolongation of the QRS and loss of mechanical efficiency. Another type of ventricular asynergy is intra-ventricular conduction delay, generated by bundle branch blocks which most frequently impair conduction through the left bundle branch. A further type of ventricular asynergy is that of regional wall motion abnormalities typical of ischaemic heart disease. Uncoordinated ventricular contraction alters regional workload and stress. The region of early activation contracts against minimal load, rapid early systolic shortening does not translate into pressure because the rest of the myocardium is still inactive; late-activated regions have to face considerable systolic pre-stretch and are subjected to disproprotionate load and stress. Much of the ventricular myocardial work is wasted in powerless activity and in transferring ejection from one portion of the chamber to another. Therefore, this pathology results in a prolongation of the ventricular pre-ejection time, a shortening of the ejection and relaxation times, a reduction of ejection fraction, and an increase in mitral regurgitation [2]. Consequently, CRT attempts to resynchronize the desynchronized ventricular activity by modifying their activation sequence. Of course this cannot entirely compensate for intraventricular desynchronization but it can, at least in some patients, improve the ventricular mechanical efficiency. Thus, dysynchrony seems to represent a patho-physiological process that directly depresses ventricular function, causes LV remodelling and CHF, and as a consequence independently predicts a higher risk of morbidity and mortality. Therefore, there is no doubt that cardiac remodelling constitutes an important target in the treatment of CHF.

A positive relationship between reverse ventricular remodelling and outcome has been demonstrated with drugs such as angiotensin-converting enzyme-inhibitors, angiotensin-receptor blockers, and beta-adrenergic blockers, with a parallel improvement in ventricular geometry and function and reduction in morbidity and mortality [3]. On the other side, several non-controlled studies have demonstrated that CRT reverses LV remodelling, decreases LV end-systolic and end-diastolic volumes, and increases LVEF. These benefits were attributed to CRT, since discontinuation of pacing resulted in loss of improvement in cardiac function [4]. Especially, in CARE-HF study, the mean reduction in LV end-systolic volume increased from 18.2% after 3 months to 26% after 18 months of CRT. Similarly, mean LVEF increased from 3.7% at 3 months to 6.9% at 18 months. These observations provide consistent evidence of a large, progressive, and sustained reverse remodelling effect conferred by CRT [5].

References