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 Ambroise Paré (1510-1590):  
 The Gentle Surgeon  
 (Refer to page 112-113)

# Cardiac Resynchronisation Therapy – New Treatment for Heart Failure

**K S Ng**

Congestive heart failure remains an important challenge to our health care system. The National Healthcare Group alone reported 2,574 inpatient admissions (48% of the national figures) in year 2000. The DRG code (252) for heart failure is also one of the top indicators for hospital readmission. The existing armamentarium of therapy ranges from anti-failure drug treatment to cardiac transplantation.

Teo et al's report<sup>(1)</sup> underscores the emergence of cardiac resynchronisation therapy (CRT) for the treatment of congestive heart failure (CHF) as yet another new therapeutic option for a certain subset of patients. In addition to symptom palliation and improvement of quality of life, CRT has also been shown to reverse ventricular remodelling. This finding is particularly appealing in light of the fact that, short of a heart transplant, treatment options are otherwise extremely limited for these candidates. To some extent, the significantly high mortality rates associated with CHF may be due to the limited availability of heart transplantation, and the advent of CRT for such individuals is therefore likely to circumvent the need for transplantation, particularly if the patient is treated at an earlier stage of the disease.

The subject of cardiac pacing for patients with heart failure is not new. It was discussed as early back as 1990 when Hochleitner<sup>(2)</sup> reported on the benefits of conventional dual chamber pacing in patients with dilated cardiomyopathy. It was postulated that its benefit was derived mainly through resynchronisation of the abnormal AV delay. But his results and optimism were not shared by others<sup>(3,4)</sup>, as a result of which interest in the subject waned for a while. Interest was resurrected again thanks mainly to the current attention on patients with dilated cardiomyopathy and left bundle branch to block (LBBB). Studies have shown LBBB to be an independent marker for increased mortality, with one demonstrating a proportional increase in mortality with increasing QRS duration<sup>(5)</sup>. It would hence appear to stand to reason that correction of the interventricular asynchrony using biventricular pacing may symptomatically improve patients with medical refractory ventricular failure. Cazeau<sup>(6)</sup> and Bakker<sup>(7)</sup> published the first case reports in 1994. In a subsequent paper, Cazeau showed that biventricular pacing resulted in an increased cardiac index, decreased mean V wave and decreased pulmonary wedge pressure<sup>(8)</sup>. Other subsequent studies have shown attributes of an improved contraction pattern – viz. reduced paradoxical septal wall motion, improved left ventricular wall motion, lower end systolic volume and an improved left ventricular dP/dt<sup>(9,10)</sup>.

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Currently the kind of patients who are perceived to derive the best benefit are patients with Class III heart failure, an ejection fraction <35%, preferably idiopathic in origin, QRS duration >120 milliseconds, left bundle branch block with PR interval >150 milliseconds. The benefit of cardiac pacing is now believed to come in two forms. By optimising AV delay it is said to improve blood transfer from the atrium to the ventricle, lengthen the diastolic filling period, and decrease diastolic mitral regurgitation. The second benefit comes in the form of coordinated ventricular activation; this results in a correction of left ventricular desynchrony leading to a more efficient ejection fraction.

As with any new technology, for biventricular pacing to be accepted as a treatment modality, it needs to attain three distinct objectives. First it needs to be shown that the implantation technique is safe and feasible. Next, importantly, it needs to show a positive impact on the functional status, and finally the positive impact must translate to an improvement survival and functional class. Teo's paper comes at a time when the first two objectives have been attained. His results are in keeping with the trends observed, viz. a high implant success rate, with little complication, and clinical end-points of both subjective and objective improvement. Whether biventricular pacing conclusively improves survival or not will be seen from three randomised controlled trials specifically designed to study this. In November last year, Guidant happily announced the termination of their COMPANION trial, declaring that they have successfully achieved the primary endpoint. We should be hearing about their data very soon. CARE-HF and PERFECT are still ongoing.

While biventricular pacing addresses the problem of heart failure, it does not take away the problem of sudden cardiac death. The pacemaker industry has models available which incorporate a cardioverter-defibrillator function. Considering that heart failure patients are inherently at risk for sudden cardiac death, it remains unclear who in terms of primary prevention deserves a biventricular pacemaker-cum-cardioverter-defibrillator. An unspoken drawback here is the associated cost, especially in an Asian society, where insurance coverage for such devices is virtually non-existent.

Under the recently updated ACC/AHA/NASPE guidelines for implantation of cardiac pacemakers and antiarrhythmia devices, biventricular pacing has now become a class IIA indication for patients with dilated cardiomyopathy and left bundle branch block<sup>(1)</sup>. It will be interesting to follow the developments in the years to come and watch if biventricular pacing will eventually be accepted by the heart-failure-centric physician community and be upgraded to a Class I indication. **SMD**

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