Pacemakers and Defibrillators for Congestive Heart Failure

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Introduction
Coronary intervention has improved survival in selected patients with left ventricular dysfunction from acute ischemic episodes. This has led to increased recognition of congestive heart failure (CHF). A new diagnosis of CHF is made in 400,000 Americans every year. These patients have a mortality rate of 25% over a 2.5-year follow-up period. Although medical therapy is the primary therapy for patients with heart failure, use of pacemakers to improve cardiac hemodynamics is under investigation. Manipulation of heart rate, atrioventricular (AV) delay, site of right ventricular pacing, and use of biventricular pacing has been explored to attempt to optimize cardiac hemodynamics. Further, it has been shown that sudden cardiac death accounts for 50% of deaths in patients with CHF. Use of amiodarone and an implantable cardioverter-defibrillator (ICD) has been shown to reduce arrhythmic mortality. CHF patients represent a single identifiable population that can be targeted for primary prevention of sudden cardiac death. Thus, the use of pacemakers and ICDs might offer nonpharmacologic means of significantly improving morbidity and mortality in these patients.

Use of Pacemakers for Hemodynamic Improvement
The following parameters can be used to define the role of permanent pacemakers in patients with congestive heart failure.

Heart rate
Patients with severe myocardial dysfunction often have involvement of a specialized conduction system. In addition, medications such as amiodarone, β-blockers, and digoxin may cause chronotropic incompetence, making prophylactic permanent pacemaker insertion necessary. Currently, standard indications for permanent pacing are used for prescribing pacemakers to patients with heart failure, although there are protocols investigating whether heart failure patients with a heart rate of less than 60 bpm should receive a prophylactic pacemaker before administration of a β-blocker.

Cardiac output may be enhanced by an increase in stroke volume or by an increase in venous return by increasing heart rate [1]. Patients with heart failure are operating at the flat part of the Frank-Starling curve, so stroke volume cannot be significantly increased. The Treppe phenomenon supposes that an increase in heart rate is accompanied by an increase in contractility. However, an increase in heart rate may also have deleterious effects. The reduction in diastolic filling time that accompanies an increase in heart rate may compromise cardiac output, especially if venous return does not increase proportionately. β-Blocks have been demonstrated to cause an improvement in survival in patients with CHF without decreasing the heart rate [2]. Although the true benefit of heart rate is not completely understood, currently patients who already have pacemakers are often paced at higher rates to attempt to improve cardiac output in times of acute clinical decomposition.

Atrioventricular synchrony
Atrial contraction adds an estimated 15% to 30% to total cardiac output in patients with left ventricular dysfunction.
[3], Atrial contraction assists in diastolic filling of the ventricle. Conversely, the absence of AV synchrony may produce systolic mitral and tricuspid valve insufficiency owing to delayed valve closure, pulmonary and systemic venous congestion, and an inappropriate decrease in peripheral resistance. All of these factors affect cardiac output. It is clear that in patients with normal sinus rhythm and CHF who already have pacemakers, the optimal mode of pacing is DDD or AAI, if the patient has a clinically acceptable AV delay. It is also well established that those patients with CHF who need a pacemaker for a known indication should have an AV sequential device unless they are in chronic atrial fibrillation.

**Atrioventricular delay**

Many patients with structural heart disease have abnormalities of the conduction system as well. AV nodal and His-Purkinje disease, although not causing heart block, can contribute to a long delay in AV contraction [3]. The AV delay is affected by many variables: intra-atrial conduction time, AV conduction time, the level of hydration, and the presence of intraventricular conduction time defects.

Several initial studies have examined optimal AV delay in patients with right ventricular apical leads and heart failure. The conclusion is that a shorter AV delay, as compared with optimal AV delays in patients with normal left ventricular function, may be beneficial in patients with cardiomyopathy. Hochleitner [4] demonstrated, in a controlled study of 16 patients with dilated cardiomyopathy, an improvement in functional capacity with dual chamber pacing and an AV delay of 100 ms. Long-term follow-up continued to demonstrate an improvement for up to 5 years. However, many investigators believe that this improvement is only observed in patients with long AV delay at baseline and may not affect individuals without first-degree AV block. Furthermore, there exists problematic reproducibility of these data. Also there is significant variability in measuring hemodynamic function whether using a fluid-filled catheter or an echocardiographic probe.

Follow-up studies conducted in larger series failed to demonstrate any significant hemodynamic benefit from right ventricular (RV) pacing at any AV delay programmed. Gold et al. [5] failed to demonstrate hemodynamic benefit in 12 class III CHF patients with VDD pacing at an AV delay of 100 ms compared with VVI pacing within the same patient. All of these patients had baseline first-degree AV block. Therefore, despite promising initial results, controlled studies have not verified the benefit of application of VDD or DDD pacing to a nonselected population of severely symptomatic CHF patients.

**Site of right ventricular pacing**

When pacing techniques were originally designed, right ventricular apical pacing was chosen for its ease of location and good pacing characteristics (lead stability). However, there are many reasons for why right ventricular pacing and specifically apex pacing may worsen cardiac hemodynamics, especially in patients with heart failure. Some small studies have demonstrated a higher cardiac output with AAI pacing than with DDD pacing. The main problems include 1) paradoxical septal motion, 2) interference with mitral/tricuspid apparatus, 3) altered diastolic function, 4) alteration in ventricular activation pattern, and 5) increased serum catecholamine concentration.

Because of these findings, studies to optimize the location of pacing in the right ventricle were conducted. Initial animal data demonstrated an improvement in cardiac hemodynamics with pacing from the right ventricular outflow tract rather than the right ventricular apex. Clinical studies by Gold et al. [5], Victor et al. [6], and Brockman et al. [7], have failed to demonstrate a clinical benefit with right ventricular septal and outflow tract pacing, as compared with right ventricular apex pacing. All of these studies programmed the pacemaker at an optimal AV delay during implant. Interestingly, those patients who did benefit from the right ventricular pacing techniques in these studies all had left bundle branch block.

In a follow-up study, Schwaab et al. [8] investigated 14 patients with sinus rhythm and complete AV block. All patients were paced at the right ventricular apex and the right ventricular septum. AV delay was optimized. Their conclusion was that the right ventricular site, which narrowed the QRS duration acutely, might improve cardiac hemodynamics. However, this site could not be predicted in all patients. In summary, despite early promising results from uncontrolled studies, the location of pacing in the right ventricle does not appear to be an important determinant of cardiac performance. The routine usage of septal pacing in CHF patients undergoing pacemaker implantation cannot be recommended at this stage.

**Left ventricular stimulation**

Because CHF is mostly a result of left ventricular dysfunction, and prior attempts at optimizing right ventricular pacing have failed, there is increasing interest in pacing the left side of the heart or simultaneously pacing the right and left ventricles. This should theoretically be particularly useful in patients with intraventricular delay (especially of the left bundle branch morphology) and long PR intervals, which may cause an inefficient pattern of contraction. Such delays may lead to an increase in mitral regurgitation and the prolonged contraction time, which will diminish diastolic filling time. A long PR interval results in a very early contraction of the atria, which means that at the end of diastole there is mitral regurgitation in the diastolic period. Although this may be a trivial volume, it does decrease the amount of ventricular filling, particularly in sicker patients. Also, in patients who apparently have very long left bundle branch block delay in their QRS, there is very discordant contraction, with the base contracting first and the left ventricular wall contracting later. This results in paradoxical septal motion, bulging of the lateral wall in