

## Biventricular Pacing during Cardiac Operations

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### ABSTRACT

**Background:** Biventricular pacing (resynchronization therapy) improves the duration and quality of life in a subset of patients with congestive heart failure, but this technique has received little attention in the cardiac surgery literature. This report presents some preliminary ideas about its rationale and technique, and some likely indications for this procedure during the performance of cardiac operations.

**Methods:** We briefly summarize the theory and the results of the randomized clinical trials of resynchronization therapy that led us to consider biventricular pacing for high-risk cardiac surgery patients. We present some techniques for using temporary and permanent biventricular pacing in the operating room. We review the hospital records and present early results of the first 25 patients in whom we implanted permanent left ventricular free wall pacing electrodes with the intent of implanting biventricular pacing devices.

**Conclusions:** Biventricular pacing has great potential to simplify the management and improve the outcomes of some cardiac surgical patients.

### INTRODUCTION

#### *The Basic Electrophysiological Concept*

Biventricular pacing exploits the relationship between the duration of electrical systole (the width of the QRS complex) and the synchronicity of mechanical systole. Normally it takes 80 to 90 milliseconds for an electrical signal to traverse the His-Purkinje system and activate ventricular muscle. This rapid depolarization produces a synchronous ventricular contraction, that is, one in which all the ventricular walls are simultaneously moving toward each other. Abnormal conduction states such as unipolar pacing, bipolar pacing, and left bundle branch block (LBBB) prolong electrical activation (>130 milliseconds) because the depolarizing wave cannot travel through the fast-conducting His-Purkinje tissue and must travel through the slower-conducting muscle tissue. The resulting prolonged electrical activation delays mechanical systole; ie, some ventricular walls are contracting before others are activated. This less synchronous contraction

reduces cardiac output, and in the worst cases, blood pressure as well. Of course not all ventricles experience clinically important reduction in cardiac output in paced rhythms, LBBB, and other wide QRS rhythms. In general, dilated, hypocontractile ventricles are more susceptible to clinically important declines in output when depolarization is longer than 130 milliseconds. Biventricular pacing can improve function in such ventricles by initiating a faster electrical depolarization and thereby causing a more synchronous mechanical contraction [Kerwin 2000]. This effect can be accomplished simply by using two simultaneously activated circuits instead of one to pace the ventricles. When these circuits are placed far apart and simultaneously activated, QRS width is reduced and contraction improved. Synchronizing the contractions of the septum and lateral wall allows the force vectors to converge at the center of the chamber simultaneously, which mimics the natural mechanics of the contraction process.

### CLINICAL TRIALS

Four randomized clinical trials have shown the value of biventricular pacing. None of these trials examined patients who underwent cardiac operations but instead focused on patients with chronic congestive heart failure associated with a prolonged QRS complex. The Multicenter InSync Randomized Clinical Evaluation (MIRACLE) [Abraham 2002] and Multisite Stimulation in Cardiomyopathy (MUSTIC) [Cazeau 2001] trials demonstrated significantly improved quality of life, exercise capacity, and peak oxygen consumption in such patients. The Dual Chamber and VVI Implantable Defibrillator (DAVID) [Wilcock 2002] trial examined a subset of congestive heart failure patients who had indications for an implantable cardioverter defibrillator (ICD). One group received an ICD with ventricular back-up pacing at a rate of 40 bpm. The other group received an ICD with dual-chamber (DDD) pacing set at 70 bpm. One might expect that DDD pacing would provide atrioventricular (AV) synchrony and improve outcomes, but at 1 year, the mortality rate was significantly higher in this group, and mortality trended higher in those who were paced most frequently. Traditional pacing increased mortality in this group presumably because it caused ventricular desynchronization. The Comparison of Medical Therapy, Pacing, and Defibrillation in Chronic Heart Failure (COMPANION) [Saluhke 2003] trial studied 1600 patients with congestive heart failure and QRS duration greater than 120 milliseconds. All patients were treated with maximal medical therapy, which was the only therapy in the control group. One study group also

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Table 1. Patient Demographic Characteristics (N = 25)\*

Factor	No. of Patients
Age, mean (range), y	74.6 ± 7.8 (60-88)
NYHA 3+	20
Previous myocardial infarction	10
Previous cardiac surgery	8
Diabetes	8
Renal insufficiency	5
Cerebrovascular disease	5
Peripheral vascular disease	5

\*NYHA indicates New York Heart Association classification.

received biventricular pacers and the other received biventricular ICDs. Biventricular pacing reduced mortality by 20% compared to controls, and biventricular ICDs reduced mortality by 40%. These studies show that desynchronization contributes to mortality in congestive heart failure patients and that biventricular pacing is effective treatment.

Ventricular desynchronization reduces cardiac function by predictable mechanisms. For example, it causes wall motion abnormalities, reduces dP/dT, reduces diastolic filling time, and most intriguingly, prolongs mitral regurgitation time [Saxon 1998, Kass 1999, LeRest 1999, Porciani 2000, Walker 2000]. The latter is interesting to surgeons because it may explain some of the failures of mitral annuloplasty for ischemic regurgitation.

## MATERIALS AND METHODS

### Surgical Implications and Techniques

Presently, most biventricular pacing devices are being implanted by cardiologists for elective treatment of congestive heart failure. In addition to the standard electrodes positioned in the right ventricle and right atrium, biventricular pacing requires a dedicated lead in the left ventricle. Placement of this lead is accomplished by maneuvering a lead through the coronary sinus into the lateral cardiac vein. This procedure is not an easy task and can be consuming of both operator time and total fluoroscopy time.

There are 3 important cardiac surgical implications of resynchronization therapy. First and most simply, cardiologists have occasional difficulty passing the left ventricular (LV) free wall electrode transvenously. Surgeons can easily place an LV lateral-wall electrode through a 4-cm left lateral thoracotomy using double-lumen endotracheal anesthesia and a screw-on or sutured device. The lead connector is passed to the left infraclavicular incision where the pacer will reside. This procedure requires no special equipment and takes 30 to 40 minutes of operating time.

Second, we have found that temporary biventricular pacing after open-heart operations improves cardiac performance in patients with large, hypocontractile hearts in complete heart block, wide QRS rhythms, or chronic pacing [Foster 1995, Weisse 2002]. This procedure is easy to do. We place standard right atrial and right ventricular (RV) free wall temporary

electrodes, and in addition, a midwall posterolateral LV electrode. The key point to understand is how to attach the ventricular electrodes to a standard temporary external pacing unit to permit it to act as a biventricular pacer. We exteriorize both ventricular electrodes to a left subcostal site and place a skin ground at the same site. We then attach both ventricular electrodes to the negative pole of the pacing harness. The skin ground goes to the positive pole. This positioning creates two unipolar pacing circuits. When electrons flow, RV to ground and LV to ground simultaneously activate the heart from two sites that are roughly 180 degrees apart on its circumference. Along with the atrial electrodes, we can institute biventricular DDD pacing and optimize both PR interval and electrical systole. We have used this technique without difficulty in all high-risk cases for the past 2 years.

Third, we have begun to identify those surgical patients who might benefit from a permanent LV electrode placed prospectively at the time of cardiac surgery, with the connector buried in the left infraclavicular space. This procedure greatly simplifies the placement of a biventricular pacer or ICD in the postoperative period because it obviates the tedious task of percutaneous lateral LV electrode placement. For patients whose hearts are already chronically paced, we can upgrade to a biventricular device easily at the time of cardiac surgery. The remainder of this paper describes 25 high-risk cardiac surgery patients in whom we used this approach.

## RESULTS

We began placing permanent electrodes for biventricular pacing shortly after the US Food and Drug Administration approved a biventricular DDD device on August 18, 2001. Table 1 summarizes the demographics of this group, and Figure 1 shows the distribution of LV sizes and ejection fractions. The operations performed are summarized in Table 2. Only 7 of the 25 patients had one procedure. Figure 2 shows QRS widths compared to LV size. Those patients who had indwelling pacemakers preoperatively are distinguished with triangles. As you can see, most of these 25 patients had large, hypocontractile hearts and underwent relatively complicated cardiac operations.

Table 3 summarizes the results. One patient required intraaortic balloon counterpulsation, 1 died in the hospital, and 2 suffered late deaths. One patient had a stroke 9 days after surgery. Five patients did not require chronic biventricular pacers; 2 of these had right bundle branch block (RBBB)

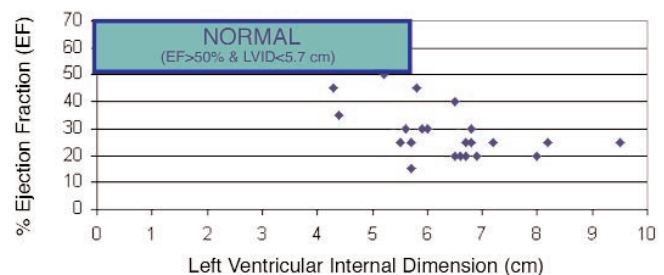


Figure 1. Patient ejection fraction versus left ventricular size.

Table 2. Procedures Performed in 25 Patients

Procedure	No. of Patients
Coronary artery bypass	14
Mitral valve procedure	15
Aortic valve replacement	12
Maze	4
Left ventricular aneurysmectomy	2
Aortic root reconstruction	4
Ascending aortic reconstruction	3
Tricuspid valve repair	2
Total no. of procedures	56

and 3 had LV end-diastolic diameter <6.0 cm. There were no complications related to pacing

### DISCUSSION

Five potentially important ideas about resynchronization therapy in the operating room deserve further study. First, we are still learning when to place permanent electrodes in high-risk patients and how to diagnose dyssynchrony. For instance, we now realize that RBBB has less effect on ventricular synchrony than LBBB, so QRS width alone is not enough information. As a result we are now more careful to review preoperative electrocardiogram diagnoses before deciding on therapy. We also need to improve our capacity to diagnose dyssynchrony by echocardiography in the operating room. Second, as mentioned above, dyssynchrony contributes to “ischemic” mitral regurgitation [Breithardt 2003] and must now be considered a correctable part of the syndrome that neither ring placement nor mitral valve replacement addresses. Third, LV aneurysmectomy to physically remodel the heart, as performed in two of our cases, can no longer be complete unless we “electrically remodel” the heart as well. Fourth, the maze operation has the potential to improve cardiac performance by restoring AV synchrony, but as an extension of the DAVID trial, we cannot expect improved ventricular function in high-risk patients unless we restore both AV synchrony and ventricular synchrony in those who require pacing after a maze. Fifth, chronically paced, large, hypocontractile hearts are easy to upgrade to a biventricular system during an open-heart operation, and this procedure appears likely to be beneficial.

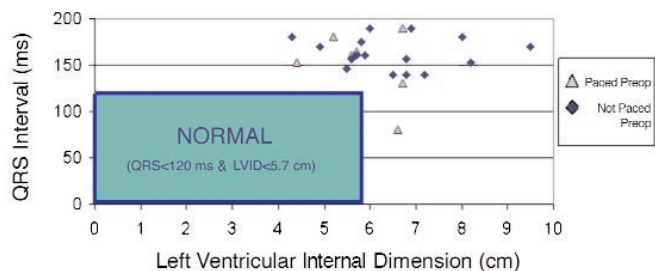


Figure 2. Patient QRS interval versus left ventricular size.

Table 3. Early and Late Outcomes

Outcome	No. of Patients
Operative mortality	1
Late mortality	2
Stroke	1
Intraoperative intraaortic balloon pump	1
Ventricular tachycardia ablation	1

In summary, we recommend temporary biventricular DDD pacing in all high-risk cardiac surgery patients. We are continuing to explore the role of placing a posterolateral LV free wall electrode in those who may benefit.

### REFERENCES

Abraham WT, Fisher WG, Smith AL, et al. 2002. Cardiac resynchronization in chronic heart failure. *N Engl J Med* 346:1845-53.

Breithardt OA, Sinha AM, Schammenthal E, et al. 2003. Acute effects of cardiac resynchronization therapy on functional mitral regurgitation in advanced systolic heart failure. *J Am Coll Cardiol* 41:765-70.

Cazeau S, Leclercq C, Lavergne T, et al. 2001. Effects of multisite biventricular pacing in patients with heart failure and intraventricular conduction delay. *N Engl J Med* 344:873-80.

Foster AH, Gold MR, McLaughlin JS. 1995. Acute hemodynamic effects of atrio-biventricular pacing in humans. *Ann Thorac Surg* 59:294-300.

Kass D, Chen CH, Curry C, et al. 1999. Improved left ventricular mechanics from acute VDD pacing in patients with dilated cardiomyopathy and ventricular conduction delay. *Circulation* 99:1567-73.

Kerwin WF, Botvinick EH, O’Connell JW, et al. 2000. Ventricular contraction abnormalities in dilated cardiomyopathy: effect of biventricular pacing to correct interventricular dyssynchrony. *JACC* 35:1221-7.

Le Rest C, Couturier O, Turzo A, et al. 1999. Use of left ventricular pacing in heart failure: evaluation by gated blood pool imaging. *J Nucl Cardiol* 6:651-6.

Porciani MC, Puglisi A, Colella A, et al. 2000. Echocardiographic evaluation of the effect of biventricular pacing: the InSync Italian Registry. *Eur Heart J* 2(suppl J):J23-J30.

Saluhke TV, Francis DP, Sutton R. 2003. Comparison of medical therapy, pacing and defibrillation in heart failure (COMPANION) trial terminated early, combined biventricular pacemaker-defibrillators reduce all-cause mortality and hospitalization. *Int J Cardiol* 87:119-20.

Saxon LA, Kerwin WF, Calahan MK, et al. 1998. Acute effects of intraoperative multisite ventricular pacing on left ventricular function and activation/contraction sequence in patients with depressed ventricular function. *J Cardiovasc Electrophysiol* 9:13-21.

Walker S, Levy T, Rex S, et al. 2000. Left ventricular remodeling with chronic biventricular pacing. *Europace* 1(suppl D):abstract 212/5.

Weisse U, Isagro F, Werling C, et al. 2002. Impact of atrio-biventricular pacing to poor left-ventricular function after CABG. *Thorac Cardiovasc Surg* 41:131-5.

Wilkoff BL, Cook JR, Epstein AE, et al. 2002. Dual-chamber pacing or ventricular backup pacing in patients with an implantable defibrillator: the Dual Chamber and VVI Implantable Defibrillator (DAVID) Trial. *JAMA* 288:3115-23.