

CARDIOLOGY UPDATE 2008

Pacing Algorithms to Minimize Right Ventricular Pacing and Sensors to Warn for Worsening Heart Failure

Dimitrios D. Manolatos, MD

*B' Cardiology Department,
Evangelismos General Hospital,
Athens*

Several clinical studies in patients with VVI or dual-chamber pacemakers have demonstrated that right ventricular apical pacing may adversely affect myocardial function and lead to an increased number of hospital admissions for congestive heart failure. Endocardial catheter mapping studies have unequivocally demonstrated that right ventricular (RV) pacing mimics the endocardial activation patterns of left bundle branch block (LBBB). Furthermore, the effect of RV pacing on left ventricular activation patterns and times are similar to that observed during LBBB. Right ventricular apical pacing results in asynchronous ventricular activation and delayed left ventricular (LV) activation time due to slow initial propagation of the electrical wavefront through ventricular myocardium rather than through the His-Purkinje system. [1] The greater the mass of the ventricular myocardium activated by muscle-to-muscle conduction prior to activation of the Purkinje system, the longer the QRS duration. It is therefore not surprising that RV pacing is pathophysiologically abnormal and results in paradoxical septal wall motion, delayed lateral wall contraction, alteration of regional adrenergic innervation, myocardial blood flow, regional perfusion defects, wall motion abnormalities, reduced LV pressure, mitral regurgitation, and reduced diastolic filling times. All of these effects contribute to reduced LV pumping function. [2] In addition, experimental studies have demonstrated that long-term RV apical pacing [RVA] induces abnormal histologic changes with myofibrillar disarray, as well as asymmetrical LV hypertrophy and thinning. [3] Therefore, intrinsic ventricular activation is generally preferred to a ventricular-paced sequence whenever possible.

Automatic intrinsic conduction search provides about 40% ventricular pacing as its best performance. [4] The impossibility to avoid unnecessary ventricular stimulation in this setting has prompted the development of algorithms to minimize ventricular pacing. In fact, the strategy of enhancing intrinsic AV conduction may lead to ventricular pacing at prolonged AV intervals, where fusion beats frequently occurs and further prolongation of the AV interval is not possible. The concept of ventricular pacing minimization is of paramount importance in patients that are not pacemaker-dependent to avoid the clinical drawbacks of RV pacing at long term, namely atrial arrhythmias and heart failure.

In a Danish study, AAI and VVI pacing were compared in patients with sick sinus syndrome. AAI pacing was associated with reduced mortality, a lower incidence of atrial fibrillation and less severe heart failure. The DAVID trial compared DDDR pacing at 70 bpm with VVI pacing at 40 bpm, in patients indicated for implantation of a defibrillator, but without an indication for antibradycardia pacing. DDDR pacing

was associated with an increased occurrence death or heart failure hospitalization.[5] These studies, along with results from the subsequent SCD-HeFT trial, which employed only single-chamber, shock-only ICDs, demonstrate potential benefits of preserving intrinsic AV conduction, when possible, to minimize wall-motion abnormalities and long-term impairment of myocardial function, and reduced the risk of congestive heart failure.

Recognition of the chronic adverse effects of RV apical pacing has stimulated interest in strategies to either eliminate or attenuate these effects. Manipulation of AV intervals in the DDD/R mode among patients with intact AV conduction may permit functional AAI/R operation and reduce unnecessary ventricular pacing, thereby preserving a normal ventricular activation sequence. This can be accomplished with sinus node dysfunction but with normal atrioventricular conduction, by establishing functional AAIR pacing with use of DDDR mode with long AV delay >250 ms. However, many a time this is not consistently feasible and it remains an inefficient way to reduce ventricular pacing in at least 17-32% of patients even when the longest fixed atrioventricular delay is programmed on.

Brian Olshansky from the university of Iowa, who presented the results of the Inhibition of Unnecessary RV Pacing with AV Search Hysteresis in ICDs [INTRINSIC RV] trial at the Heart Rhythm Society meeting, emphasized that the previous notion that DDDR poses an inherent safety risk is incorrect. In fact, in this study, by using programming to minimize RV pacing, «outcomes with dual-chamber programming were as good as, if not better than, single-chamber programming», he said.[6] The AV search hysteresis [AVSH] algorithm used in the trial is a programming feature that automatically searches for intrinsic ventricular activation, and extends beyond the programmed AV delay to allow for intrinsic conduction. Managed Ventricular Pacing [MVP] mode allows the device to provide dual-chamber pacing of the RV when needed, but it automatically switches to atrial pacing as its primary therapy when normal conduction is present, using beat-to-beat verification of the patient's natural electrical conduction system.[7] For example, this algorithm extends the AV delay with the programmed AV delay extension if the previous beat revealed spontaneous AV conduction. If no ventricular conduction event is sensed during the extended AV delay, the pacemaker returns to the normal AV delay. The extension can be programmed to 60, 80, 100 or 120 ms. This feature may prove particularly beneficial in patients who are at risk for sudden cardiac arrest and need the protection of an ICD, but whose electrical conduction may be normal and

require little or no pacing.[8]

In **conclusion**, RV apical pacing impairs LV function by inducing LBBB-like dyssynchrony and is associated with an increased risk of heart failure, atrial fibrillation, morbidity and mortality. These observations have led to the introduction of algorithms and new pacing modes to reduce RV pacing in cardiac pacemakers, and a shift of the pacing site away from the RV apex [9-11].

REFERENCES

1. Vassalo JA, Cassidy DM, Miller JM, et al. Left ventricular endocardial activation during RV pacing: effect of underlying heart disease. *J Am Coll Cardiol* 1986;7:1228-33.
2. Rosenqvist M, Isaaz K, Botvinick EH, et al. Relative importance of activation sequence compared to AV synchrony in left ventricular function. *Am J Cardiol* 1991;67: 148-156.
3. Prinzen FW, Cheriex EC, Delhaas T, et al. Asymmetric thickness of the left ventricular wall resulting from asynchronous electric activation a study in dogs with ventricular pacing and in patients with LBBB. *Am Heart J* 1995;130: 1045-1053.
4. Deering TF, Wilensky M, Tondato F, et al. Auto intrinsic conduction search algorithm: a prospective analysis. *Pacing Clin Electrophysiol* 2003;26:1080 [A].
5. Sharma AD, Rizo-Patron C, Hallstrom AP, et al. DAVID Investigators. Percent right ventricular pacing predicts outcomes in the DAVID trial. *Heart Rhythm* 2005;2:830-834.
6. Olshansky B, Day JD, Moore S, et al. Is dual chamber programming inferior to single chamber programming in an ICD? Results of the INTRINSIC RV study. Heart Rhythm Society 2006 Annual Scientific Sessions; 17-20; 2006; Boston, MA.
7. Milasinovic G, Sperzel J, Smith TW, Mead H, et al. Reduction of RV pacing by continuous optimization of the AV interval. *Pacing Clin Electrophysiol* 2006;29:406-412.
8. Sweeney M. Minimizing right ventricular pacing: a new paradigm in cardiac pacing in sinus node dysfunction. *Am Heart J* 2007;153(4 Suppl):34-43.
9. Zhang XH, Chen H, Siu CW, et al. New-onset heart failure after permanent right ventricular apical pacing in patients with acquired high-grade atrioventricular block and normal left ventricular function. *J Cardiovasc Electrophysiol* 2008;19(2):136-41.
10. Tantengco MV, Thomas RL, Karpawich PP. Left ventricular dysfunction after long-term right ventricular apical pacing in the young. *J Am Coll Cardiol* 2001 15;37:2093-100.
11. Manolis AS. The deleterious consequences of right ventricular apical pacing: time to seek alternate site pacing. *Pacing Clin Electrophysiol* 2006;29:298-315.