International Journal of Cardiology Research
www.cardiologyjournal.in
E-ISSN: 2663-4112, P-ISSN: 2663-4104
Received Date: 03-11-2018 Accepted Date: 05-12-2018; Published: 05-01-2019
Volume 1; Issue 3; 2019; Page No. 07-09

Right ventricular apical pacing induced left ventricular systolic dysfunction

Naresh Gaur[1*], Prakash Chand Negi[2], Rajeev Bhardwaj[3], Arvind Kandoria[4], Sanjeev Asotra[5], Neeraj Ganju[6], Rajeev Merwaha[7], Rajesh Sharma[8], Kunal Mahajan[9]
[1*] MD, Senior Resident, Department of Cardiology Indira Gandhi Medical College, Shimla, Himachal Pradesh, India
[2-4] MD, DM, Professor, Department of Cardiology, Indira Gandhi Medical College, Shimla, Himachal Pradesh, India
[5-7] MD, DM, Associate Professor, Department of Cardiology, Indira Gandhi Medical College, Shimla, Himachal Pradesh, India
[8, 9] MD, DM, Assistant Professor, Department of Cardiology, Indira Gandhi Medical College, Shimla, Himachal Pradesh, India

DOI: https://doi.org/10.33545/26634104.2019.v1.i1a.4

Abstract
Right ventricular apical pacing remained the conventional pacing site for the cardiologists from beginning. But studies later on confirmed that long term right ventricular pacing is a cause of left ventricular dysfunction even in patients with pre pacemaker implantation normal LV ejection fraction. Some observational studies and small RCTs also analyzed the predictors of LV dysfunction. In current scenario main focus is on alternate more physiological pacing site, like RVOT pacing and to enable some special algorhythms that decrease the total percentage of the pacing. Thus it is Important to understand the mechanism of post RV apical pacing LV dysfunction, other predictors of LV dysfunction and how to treat/prevent the LV dysfunction.

Keywords: RV apical pacing, RVOT pacing, LV dysfunction

Introduction
Right Ventricular Apical (RVA) pacing remained the only ventricular pacing site from beginning. Previous studies showed benefit as compare to atrial pacing in view of less frequent atrial fibrillation, heart failure and almost equal incidence of stroke, thromboembolism, and all-cause mortality. Dual chamber pacemaker was found to be superior to single ventricular chamber pacing but long term follow up of the studies showed that even dual chamber pacemaker patients developed heart failure and then many retrospective and prospective studies confirmed that RVA pacing leads to LV dysfunction.

Mechanism of LV dysfunction
RVA pacing leads to activation of left ventricular myocardium in the way that mimics LBBB. There is dyssynchronus electrical and consequently mechanical activation with a potential induction for heart failure. In study by Haiyan Xu *et al.* [1] the mRNA levels of OPA1 and SERCA2a were significantly lower in the RVA pacing group at 1 month's follow-up (both $p < 0\ 001$). Early changes in the expression of selected genes OPA1 and SERCA2a were associated with deterioration in global longitudinal strain (GLS) that became apparent months later.

Predictors of LV Dysfunction
Several studies were done to assess the predictors of LV dysfunction. In a retrospective study by Shaan Khurshid BA *et al.* [2] Pacemaker Induced Cardiomyopathy (PICM) was defined as ≥10% decrease in LVEF, resulting in LVEF <50%. Pre pacemaker implantation LV function and qrs duration >115ms were associated with post pacemaker implantation LV dysfunction. Gierula *et al.* [3] retrospectively analyzed the patients admitted for PGR and found that% RV pacing was an independent variable associate dwith poor outcome. Those with LVSD and high amounts of RVP are at higher risk of hospitalization or death. A meta-analysis of 15 South Korean studies [4] also confirmed that in patients with complete AV block Pre pacemaker implantaion LV function & paced qrs (pQRS) duration significantly correlated with LV dysfunction. A post pacemaker QRS duration with a cut-off value of above 140 ms had a sensitivity of 95% while a QRS duration with a cut-off value of above 167 ms had a specificity of 90% in this study. As per various studies conducted to assess the predictors for LV dysfunction pre- pacemaker implantation LVEF and% of RV pacing are likely to have strong association in predicting LV dysfunction. Other parameters that can have association are pre pacemaker QRS duration, Qtc, male gender, LBBB.

Prevention/Treatment of LV Dysfunction
RVA pacing induced LV dysfunction can be prevented or treated by the attempts that mainly focused on minimizing ventricular pacing in sinus node disease, optimizing pacemaker settings, and seeking better pacing sites. Other pacing sites include the RV mid or low septum, the RV inflow tract, the RV outflow tract, and the His bundle region. A meta-analysis of 14 RCTs by Shimony *et al.* [5] demonstrated deleterious effects of RVA pacing as compared to RV non apical (RVNA) pacing as better LVEF in follow up of patients with RVNA pacing group. RVA-pacing was associated with greater interventricular mechanical dyssynchrony and intra-LV dyssynchrony than RVOT-pacing.

Biventricular Pacing

Biventricular pacing is more physiological; maintaining interventricular electrical as well as mechanical synchrony and consequently no risk of LV dysfunction. In BLOCK-HF trial[6] AV block patients with biventricular pacing group had less primary outcome in the form of death from any cause and urgent care visit for heart failure. Although once thought to be only beneficial in patients with LV dysfunction; PACE trial [7] showed long term benefit even with normal LVEF patients in view of better LVEF in biventricular pacing group as compare to RVA pacing.

For reduction in percentage RVA pacing several specific pacing algorithms are there; broadly divided into two groups

a. AV hysteresis: Algorithms which work to prolong AV interval periodically to search for, and if present permit, intrinsic AV conduction
b. Algorithms that operate in a default atrial pacing mode (AAI), with mode switch to ventricular pacing mode (DDD) in case of AV block detection.

The most studied algorithm is the Managed Ventricular Pacing (MVP) which operates in primary atrial-based pacing mode labeled as "AAI(R)+" with switch to secondary DDD(R) mode if AV block occurs in two out of four consecutive atrial–atrial intervals. (Figure-1).

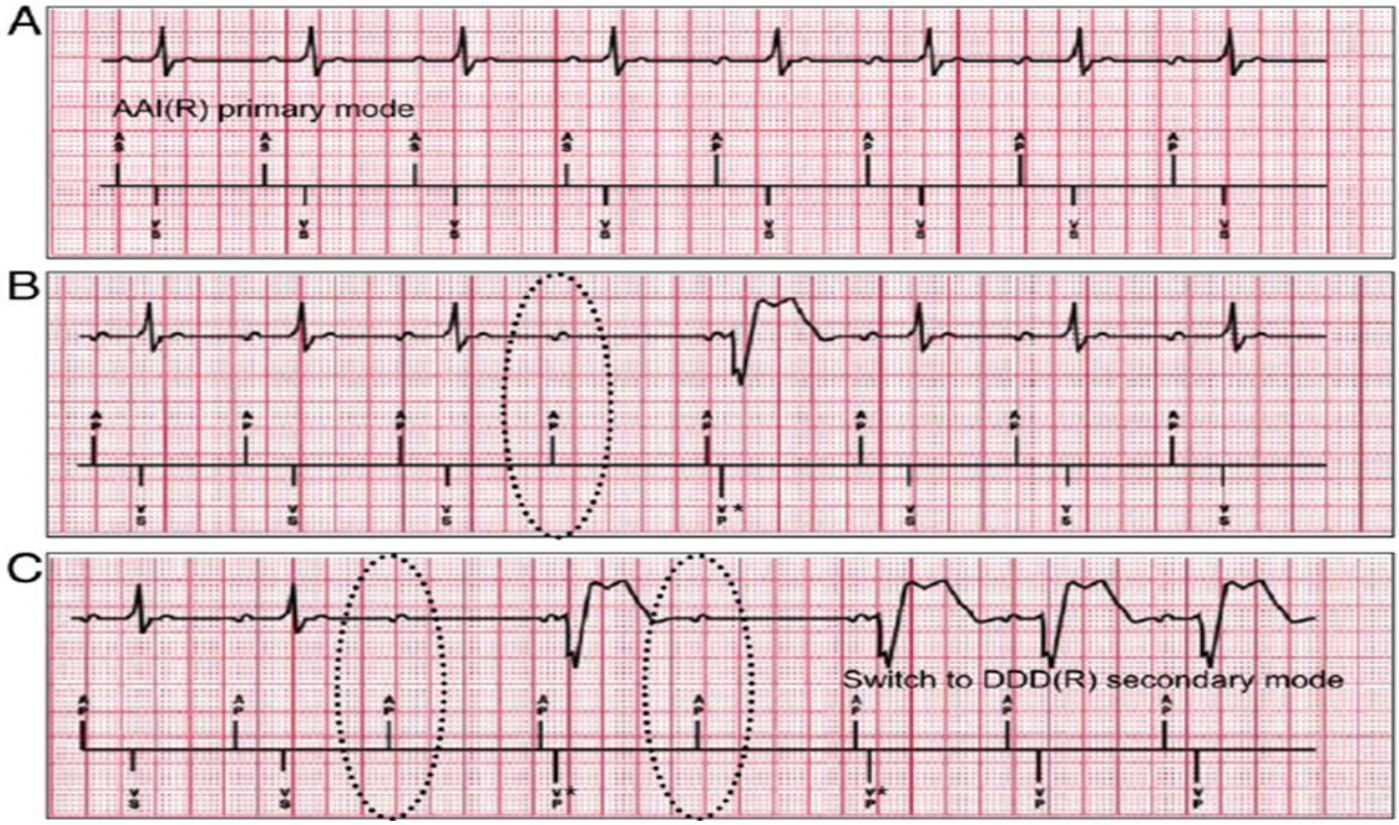

Fig 1: The Managed Ventricular Pacing™ (MVP) (Medtronic) pacemaker algorithm, which operates in primary AAI (R) mode at baseline. B: Atrioventricular (AV) block (indicated with a discontinuous circle) is defined as the absence of a ventricular sensed event between two atrial events. Backup ventricular pacing (indicated with *) is delivered at 80 ms following AV block. C: AV block in 2 of 4 consecutive A-A intervals triggers switch to secondary DDD(R) mode.

A pilot cross-over study [8] for MVP algorithm showed significant reduction in% VP as compared with DDD(R) mode and reduced VP from 80.6% to 3.8% in patients with mainly SSS. The same algorithm was also found to be significantly more effective in reducing the amount of RVA pacing when compared to AV hysteresis algorithm in patients with SSS and various degrees of AV block (66.1% vs. 54.3% had ‹40% of RV pacing).

De novo cardiac resynchronization therapy (CRT) should be considered in HF patients, reduced EF and expected high percentage of ventricular pacing in order to decrease the risk of worsening HF (class I- B). The ESC guidelines also indicate upgrade to a CRT system in conventional pacemaker patients who have developed LV dysfunction (LVEF ≤ 35%) and HF symptoms (NYHA III–IV) (Class IIa-B).

Conclusion

It is widely accepted now that long term RVA pacing has detrimental effects on LV function, especially patients with baseline LV dysfunction and high amount of ventricular pacing are at increased risk. In patients with normal baseline LV function appropriate pacemaker programming and use of appropriate pacemaker algorithms, are the important aspects that should be considered. Further studies for predictors of RVA pacing induced LV dysfunction and their control are needed.

References

1. Haiyan Xu, Xiongwei Xie, Jiangjin Li, *et al*. Early Right Ventricular Apical Pacing-Induced Gene Expression Alterations Are Associated with Deterioration of Left

Ventricular Systolic Function. Disease Markers, Hindawi, 2017. Article ID 8405196.
2. Shaan Khurshid, Andrew Epstein E, Ralph Verdino J, *et al*. Incidence and Predictors of Right Ventricular Pacing-Induced Cardiomyopathy. Heart Rhythm. 2014; 11(9):1619-1625.
3. Gierula J, Cubbon RM, Jamil HA, *et al*. Patients with long-term permanent pacemakers have a high prevalence of left ventricular dysfunction. J Cardiovasc Med (Hagerstown). 2015; 16(11):743-50.
4. Jun Hyung Kim, Ki-Woon Kang, Jung Yeon Chin, *et al*. Major determinant of the occurrence of pacing-induced cardiomyopathy in complete atrioventricular block: a multicentre, retrospective analysis over a 15-year period in South Korea. BMJ open, 2017-019048.
5. Shimony A, Eisenberg MJ, Filion KB, Amit G *et al*. Beneficial effects of right ventricular non-apical vs. apical pacing: a systematic review and meta-analysis of randomized-controlled trials. Europace. 2012; 14(1):81-91.
6. Anne Curtis B, Seth Worley J, Philip Adamson B, *et al*. Biventricular Pacing for Atrioventricular Block and Systolic Dysfunction. April 25, 2013. N Engl J Med. 2013; 368:1585-1593.
7. Fung JW, Chan JY, Omar R, *et al*. The Pacing to Avoid Cardiac Enlargement (PACE) trial: clinical background, rationale, design, and implementation. J Cardiovasc Electrophysiol. 2007; 18(7):735-9.
8. Sweeney MO, Shea JB, Fox V, *et al*. Randomized pilot study of a new atrial-based minimal ventricular pacing mode in dual-chamber implantable cardioverter defibrillators. Heart Rhythm. 2004; 1:160-7.