

## Pacing Induced Cardiomyopathy: A Clinical Problem for Further Research

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**Received:** May 13, 2019; **Published:** June 19, 2019

**Keywords:** *Right Ventricular Pacing; Cardiomyopathy; Pacing Induced Cardiomyopathy*

Since its first implantation in 1958 by Senning and Elmqvist, permanent pacemaker (PPM) become established as an effective treatment of both bradycardias and tachycardias [1].

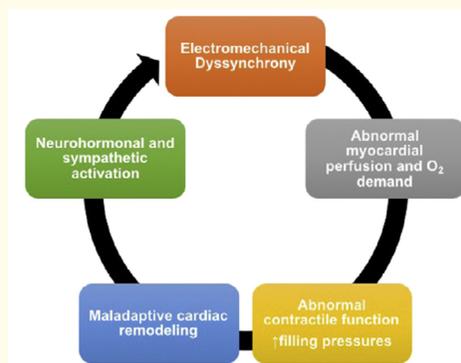
Pacing induced cardiomyopathy (PiCM) is new term used to describe the left ventricular dysfunction that complicate right ventricular pacing in patient with PPM. This may be related to the abnormal electrical and mechanical activation pattern of the ventricles (or ventricular dyssynchrony) caused by RV apical pacing [2,3]. Right ventricular apical pacing can induce both interventricular dyssynchrony (between the RV and the LV), as well as intraventricular dyssynchrony (within the LV) [3].

Pacing induced cardiomyopathy was defined as new-onset LV systolic dysfunction (LVEF < 50% on follow-up echocardiography) along with either (i) a  $\geq 10\%$  decrease in LVEF, or (ii) new-onset regional wall motion abnormality unrelated to coronary artery disease with no other identifiable causes of LV dysfunction [4,5].

The incidence of PiCM varied in previous studies, ranging from 9% to 26% depending on the definition of PiCM and follow-up duration [4].

Several predisposing risk factors of PiCM have been reported, including pre-existing LV systolic dysfunction, RV apical pacing, prolonged paced QRS duration, and higher RV pacing percentage [6-8]. Other factors include age, male sex, history of atrial fibrillation, native QRS duration, LBBB and corrected QT interval of paced beats are also considered as PiCM risk factors [4,8,9-14].

The pathophysiology of PiCM is not fully elucidated but the interplay of many factors including electrical and mechanical dyssynchrony, ventricular systolic and diastolic dysfunction and remodeling, development of mitral and tricuspid regurge, altered myocardial strain, perfusion and energetics and neurohormonal activation (Figure 1). Also, individual and genetic factors, RV pacing site, and concomitant factors with negative effect on contractile functions have added role in this situation [10,11]. The percentage of patients in whom RV apical pacing results in an LV systolic dyssynchrony detectable with echocardiographic methods has been reported to be around 50% [11].



**Figure 1:** Mechanisms of pacing induced cardiomyopathy [14].

Regarding the clinical presentation of PiCM it is variable from asymptomatic LV dysfunction to frank presentation of congestive heart failure and the diagnosis of the condition depends on the echocardiographic follow up of patients with PPM for early detection of this disorder to initiate the proper management.

Patients with permanent pacemaker who developed PiCM had poor outcome when compared to patient without this complication with more development of HF, more HF hospitalization and more all cause and cardiovascular mortality [8].

The management of PiCM involve in addition to treatment of HF the upgrade to cardiac resynchronization therapy (CRT) that proved effective in correction of ventricular dyssynchrony, cardiac functional capacity, New York heart Association NYHA class, LV hemodynamics and ventricular remodeling [11]. Also, changing of RV pacing site, use of His bundle pacing and algorithms to minimize unnecessary ventricular pacing, are another options for prevention and treatment of PiCM [12,13].

Several strategies have been proposed to prevent the development of PiCM. The first strategy was the use of high septal pacing rather than RV apical pacing however, this strategy failed to prevent PiCM in randomized trial [15]. A second strategy is to use device-based algorithms that minimize ventricular pacing but also this strategy showed no benefit in a metaanalysis of Shurrab., *et al* [16]. A third strategy is to implant a cardiac resynchronization (CRT) therapy device from the onset that show some benefit in BLOCK-HF trial [17] but with more economic and technical burden. The fourth strategy appears to be the most promising, namely, to implant these patients with a His-bundle pacemaker system that improved ventricular dyssynchrony and remodeling in patient with PiCM [14].

### Conflict of Interest

None.

### Funding

None.

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**Volume 6 Issue 7 July 2019**

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