septal pacing on left ventricular dyssynchrony and function

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ABSTRACT

Right ventricular apical versus septal pacing impact on left ventricular synchrony and function

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Background: Right ventricular (RV) pacing alters left ventricular (LV) mechanical activation, resulting in adverse impacts on LV function. Alternative RV septal pacing results in narrower QRS duration and may be more physiologic than RV apical pacing. This study was aimed to investigate the effect of RV apical (RVA) and septal pacing (RVS) on LV dyssynchrony and function.

Patients and methods: 40 patients clinically indicated for dual chamber pacing were included, subjected to conventional M-mode and 2-D echocardiography with following parameters looked for: left ventricular end diastolic diameter (LVEDD), left ventricular end systolic diameter (LVESD), ejection fraction (EF%), fractional shortening (FS%), cardiac output (CO L/m) and tissue Doppler imaging to assess LV dyssynchrony baseline study on temporary RV apical pacing. Then patients were divided randomly into two groups:Group1: 20 patients underwent permanent RV apical pacing.Group11: 20 patients underwent permanent RV apical pacing.Group11: 20 patients underwent permanent RV septal pacing. QRS duration, Electrical parameters including RV stimulation threshold, R wave, and ventricular lead impedance together with fluoroscopic time were measured in every patient. Both groups were followed up within one week and at least 6 months after implantation by echocardiography, and tissue Doppler imaging.

Results: QRS duration was significantly narrower in pts with septal pacing compared to RV apical pacing $(148.85\pm6.89 \text{ vs} 162.1\pm5.98, P < 0.001)$. Electrical parameters at implant were satisfactory for all patients and no patients required lead repositioning. There were no significant differences in the RV mean stimulation threshold, R-wave sensing, lead impedance and fluoroscopic time between the RV apical and RV septal lead positioning. Within one week following implantation there was no significant difference in LVEDD, LVESD, LVEF, CO and LV mechanical delay. On follow up, in RV septal paced patients compared to RV apical paced patients LVEDD(cm) was lower $(4.73\pm0.59 \text{ Vs} 4.94\pm0.61, P \text{ value} = 0.27)$, LVESD(cm) was significantly lower $(3.02\pm0.37 \text{ Vs} 3.42\pm0.45, P \text{ value} = 0.004)$, LVEF(%) was significantly higher $(69\pm8Vs 62\pm7, P \text{ value} = 0.006)$, CO (L/min) was significantly higher $(4.88\pm0.29 \text{ Vs} 4.5\pm0.62, P \text{ value} = 0.019)$,LV lateral to septal delay was significantly lower $(72\pm5 \text{ Vs} 83\pm6, P \text{ value} < 0.001)$.

Conclusion: Long term RV septal pacing is feasible, reliable and efficient associated with less adverse effects on LV dyssynchrony and function compared to long term RV apical pacing.

Key words: RV septal pacing, LV dyssynchrony.

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List of Abbreviations

СО	:	Cardiac Output
LAO	:	Left anterior oblique
Lat to sept. delay	:	Lateral to septal delay
LO	:	Lateral oblique
LVEDD		Left ventricular end diastolic diameter
LVEF		Left ventricular ejection fraction
LVESD	:	Left ventricular end systolic diameter
No.	:	Number
РА	:	Postro-anterior
Pts	:	Patients
RAO	:	Right anterior oblique
RV	:	Right ventricular
RVA	:	Right ventricular apical
RVOT	:	Right ventricular out flow tract
RVS	:	Right ventricular septal

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Introduction

Emerging evidence suggests that prolonged pacing from the right ventricular (RV) apex may lead to progressive left ventricle (LV) dysfunction, exacerbation of heart failure, atrial fibrillation (AF), and increased mortality.⁽¹⁻²⁾ The mechanism behind the negative hemodynamic effects of RV apical pacing appears to be related to abnormal ventricular activation and contraction, ultimately resulting in LV remodeling. ⁽³⁻⁴⁾ This deleterious effect has led to a growing interest in alternate ventricular pacing sites with a more favorable hemodynamic profile.

Among the possible RV pacing sites, the septum, particularly at the mid right ventricle and outflow tract (RVOT), appears to be particularly attractive.

The hypothesis is that RV septal pacing allows a more physiological activation of the ventricles. During normal atrio-ventricular conduction, the intraventricular septum and the lateral wall of the LV are almost simultaneously activated. ⁽⁵⁾ The activation wavefront then advances from the base to the apical region producing a narrow QRS complex on the surface electrocardiogram (ECG). Of interest, the free wall of the RV is almost the last region to be depolarized. ⁽⁵⁾

In contrast during RV apical pacing, the impulse advances toward the base area through the slow conducting myocardium with the

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ventricular free wall being the last to be activated. The results a broad QRS complex on the surface ECG. ⁽⁶⁻⁷⁾

With RV septal pacing, a more physiological pattern of ventricular activation should be achieved, thus avoiding the deleterious effects of RV apical pacing.⁽⁸⁻⁹⁾ It is important to recognize that nonseptal sites such as the RV free wall be avoided as this is theoretically the worst area to pace.

Aim of The Work

This study was initiated prospectively to evaluate:

- The feasibility and efficacy of RV septal pacing.
- The effect of RV apical versus RV septal pacing on LV systolic function and intra ventricular delay.