pISSN 1975-4612/ eISSN 2005-9655
Copyright © 2011 Korean Society of Echocardiography
www.kse-jcu.org

DOI: 10.4250/jcu.2011.19.1.13

EDITORIAL COMMENT J Cardiovasc Ultrasound 2011;19(1):13-14

Pacing-Induced Alterations in Left Ventricular Mechanical Properties: Effect of Pacing Sites

Chi Young Shim, MD, PhD

Division of Cardiology, Severance Cardiovascular Hospital, Yonsei University College of Medicine, Seoul, Korea

REFER TO THE PAGE 15-19

The normal electrical cardiac activation sequence is required for optimal left ventricular (LV) contraction and efficient pump function. Pacing that is conducted at virtually any ventricular site disturbs the natural activation pattern and ventricular contraction because the applied impulse travels slowly through the myocardium rather than through the rapidly conducting His-Purkinje system.[1)2)] This results in prolonged QRS durations that resemble left bundle branch block and lead to ventricular dyssynchrony. Unfavorable effects of right ventricular (RV) pacing include ventricular remodeling, dilation, elevated diastolic filling pressure, increased functional mitral regurgitation, myocardial perfusion defects, and reduced LV ejection fraction. Of all ventricular sites, the RV apex seems to be the most hemodynamically unfavorable, but there is limited information regarding the effects of alternative pacing sites on LV mechanical properties.[2)3)]

In this issue of the Journal, Cho et al.[4)] report the results of a prospective, multicenter study designed to assess the impacts of different RV pacing sites on LV dyssynchrony and adverse changes in global longitudinal LV contraction. They failed to demonstrate any differences in mechanical dyssynchrony parameters among the groups of different pacing sites, although the electrical dyssynchrony parameter was significanly different. However, patients with RV septal pacing showed better LV longitudinal systolic movement than those with RV apical pacing.

Prinzen et al.[5)] described nonuniformity of myocardial fiber strain during RV pacing using sonomicrometry in a dog model. Interestingly, in early-activated LV areas the amount of shortening was lower than in remote areas, resulting in decreases in net LV strain pattern during RV pacing.[5)] Nonphysiologic pacing sites such as the RV apex have been shown to decrease the contractile state of normal myocardium due to abnormal electrical propagation. Previous studies demonstrated histopathologic abnormalities in paced patients. Karpawich et al.[6)] demonstrated increases in histopathological alterations in patient biopsy samples following pacing, including myofiber variation, fibrosis, fat deposition, sclerosis, and mitochondrial morphological changes. These findings indicate that chronic RV apical pacing may adversely alter myocellular growth at the cellular and subcellular levels. Therefore, the observations of LV global longitudinal contraction by Cho et al.[4)] add to the growing body of evidence that RV apical pacing increases the risk of LV dysfunction and heart failure.

Although several studies have investigated the effects of pacing from alternative RV sites other than the apex, the results of studies examining eletromechanical dyssynchrony are conflicting. Flevari et al.[7)] and Cano et al.[8)] reported that RV septal pacing was associated with a more synchronous contraction pattern than was RV apical pacing, but Ng et al.[9)] found that RV septal pacing was associated with worse LV dyssynchrony than RV apical pacing. Interestingly, Cho et al.[4)] detected no significant differences in LV dyssynchrony according to pacing sites. However, none of these previous studies examined large series. Currently, three randomized prospective multicenter clinical trials, Optimize RV Selective Site Pacing Clinical Trial (Optimize RV), Right Ventricular Apical and High Septal Pacing to Preserve Left Ventricular Function (Protect Pace), and Right Ventricular Apical versus Septal Pacing, are in progress with the goals of obtaining definitive evidence of the importance of RV pacing sites to long-term alterations of LV mechanical properties and preservation of LV function.[10)] These studies, surveying a total of almost 800 patients, will help to identify optimal RV pacing sites.

There are some important issues that limit the interpretation of Cho et al.'s study. First, the study was relatively small in scope. It is difficult to assess whether the lack of difference in LV mechanical dyssynchrony that was detected is real, or is

• Editorials published in the Journal of Cardiovascular Ultrasound do not necessarily represent the views of JCU or the Korean Society of Echocardiography.
• Address for Correspondence: Chi Young Shim, Division of Cardiology, Severance Cardiovascular Hospital, Yonsei University College of Medicine, 250 Seongsan-ro, Seodaemun-gu, Seoul 120-752, Korea Tel: +82-2-2228-8453, Fax: +82-2-2227-7732, E-mail: cysprs@yuhs.ac
• This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (http://creativecommons.org/licenses/by-nc/3.0) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

merely due to a limited power to defect differences caused by low sample sizes. Second, information regarding the baseline LV mechanical properties were not available at the time of insertion of permanent pacemakers. Patients who received different pacing sites differed in terms of age and sex, and pre-existing impairment in global longitudinal LV contraction might also affect results.

References

1. Lindsay BD. *Deleterious effects of right ventricular pacing. N Engl J Med 2009;361:2183-5.*
2. Sweeney MO, Prinzen FW. *A new paradigm for physiologic ventricular pacing. J Am Coll Cardiol 2006;47:282-8.*
3. Thambo JB, Bordachar P, Garrigue S, Lafitte S, Sanders P, Reuter S, Girardot R, Crepin D, Reant P, Roudaut R, Jais P, Haissaguerre M, Clementy J, Jimenez M. *Detrimental ventricular remodeling in patients with congenital complete heart block and chronic right ventricular apical pacing. Circulation 2004;110:3766-72.*
4. Cho GY, Kim MJ, Park JH, Kim HS, Youn HJ, Kim KH, Song JK. *Comparison of ventricular dyssynchrony according to the position of right ventricular pacing electrode: A multi-center prospective echocardiographic study. J Cardiovasc Ultrasound 2011;19:15-19.*
5. Prinzen FW, Augustijn CH, Arts T, Allessie MA, Reneman RS. *Redistribution of myocardial fiber strain and blood flow by asynchronous activation. Am J Physiol 1990;259:H300-8.*
6. Karpawich PP, Rabah R, Haas JE. *Altered cardiac histology following apical right ventricular pacing in patients with congenital atrioventricular block. Pacing Clin Electrophysiol 1999;22:1372-7.*
7. Flevari P, Leftheriotis D, Fountoulaki K, Panou F, Rigopoulos AG, Paraskevaidis I, Kremastinos DT. *Long-term nonoutflow septal versus apical right ventricular pacing: relation to left ventricular dyssynchrony. Pacing Clin Electrophysiol 2009;32:354-62.*
8. Cano O, Osca J, Sancho-Tello MJ, Sanchez JM, Ortiz V, Castro JE, Salvador A, Olague J. *Comparison of effectiveness of right ventricular septal pacing versus right ventricular apical pacing. Am J Cardiol 2010;105:1426-32.*
9. Ng AC, Allman C, Vidaic J, Tie H, Hopkins AP, Leung DY. *Long-term impact of right ventricular septal versus apical pacing on left ventricular synchrony and function in patients with second- or third-degree heart block. Am J Cardiol 2009;103:1096-101.*
10. Kaye G, Stambler BS, Yee R. *Search for the optimal right ventricular pacing site: design and implementation of three randomized multicenter clinical trials. Pacing Clin Electrophysiol 2009;32:426-33.*