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Contrasting electrical effects of apical vs non-apical right ventricular pacing

The invention of artificial ventricular pacing has been a life-saving therapy for patients with asystole. Pacing was accomplished by delivering a passive electrode to the trabeculated right ventricular (RV) apex. Even now, this remains the most common pacing site, despite the availability of active fix electrodes. However, it has since become recognized that chronic RV pacing may result in LV dysfunction and thereby congestive heart failure and atrial fibrillation. The incidence of these is amplified among patients receiving higher levels of RV pacing and/or in those with preexisting LV dysfunction. Solutions include biventricular or His pacing, which require more advanced pacing skills and additional electrodes and delivery methods, prolonging procedure and adding to complications and expense. Under these circumstances, selection of alternative RV sites for deployment of the existing pacing electrode to avoid RV pacing induced cardiomyopathy is attractive. However, though extensively investigated over the last 2 decades this strategy has not yielded conclusive results. This has been disappointing. Attention to paced electrical effects may provide remedy.

RV pacing forces electrical desynchronization of ventricular activation and thereby mechanical contraction [1]. The resulting pattern is regarded as similar to LBBB, and this may be true for patients without LV dysfunction. However, pronounced differences occur in heart failure patients, in whom RV apical pacing in those with LBBB was responsible for increased mortality in the DAVID trial. Among such patients, RV apical pacing further widened the QRSd and greatly amplified the LV activation delays associated with LBBB [2]. (The paced QRS duration during RV pacing closely approximates the induced LV activation delay). Effects were less pronounced in those with normal baseline LV function, in whom the incidence of pacing induced cardiomyopathy is correspondingly lower. This difference illustrates the principle that pattern and extent ventricular desynchronization during RV pacing may be directly linked to patient outcome. What more logical than to select a RV pacing site that reduces extent of electrical desynchronization? However, this principle has not been exercised prospectively in prior studies. Rather, non-apical sites have been selected anatomically, on the presumption that septal sites will result in HPS engagement and thereby more physiological LV activation-but electrical effects were not tested.

The current study by Gupta et al. compared the QRS duration and axis resulting from various RV pacing sites. The success of septal deployment is generally overestimated but was adjudicated carefully in this study using angled fluoroscopy views. (In a separate trial, effective lead placement at the septum was maximized by using a steerable sheath system and clear guidance for implanters with bi-plane imaging at the time of implant, and yielded a success rate of only <70% [3]). The authors found that patients with shorter paced QRS durations had more often received non-apical RV pacing sites. This suggests that nonapical sites force less desynchronization and may reduce future adverse effects. However, neither acute nor chronic outcomes were assessed. Post-procedural echocardiography was undertaken but any relationship between paced QRSd and inter- or intra-ventricular dysschrony was not reported. Moreover, the authors did not randomize patients to apical vs nonapical sites-rather site of lead was selected at “operator's discretion”. Whether this was based on best paced QRSd (what was the range encountered?) from a variety of sites (how many?) tested in each individual is unclear. This practice resulted in selected groups that are unbalanced in numbers. Notably older patients more often received RV apical pacing. However, there are interesting signals: on multivariate analysis, female gender, baseline QRS duration and RVOT septal pacing were the only predictors for narrow paced QRS duration (<150 msec). Sex specific responses to pacing are increasingly appreciated [4]. Possibly, shorter baseline QRSd will lead to narrower paced QRSd-the change in QRSd might be an important metric for assessing deleterious effects of pacing.

A prior systematic review supports the current finding that the paced QRS duration was significantly shorter in patients receiving non-apical vs apical RV pacing [5]- and moreover, that subsequent LV function was better preserved. Larger differences were found when the LVEF was reduced at baseline or when the study duration was >1 year. However, data regarding exercise capacity, functional class, quality of life, and survival were limited and inconclusive. Again, RV lead deployment in all the studies assessed was based on anatomical location. Possibly, a clear benefit of septal pacing may emerge by site selection guided by electrical effect assessed by 12 lead ECG or more elaborately with e.g., electrocardiographic imaging to determine LV activation patterns [6]. The hypothesis raised by Gupta et al. in this study that individualizing nonapical pacing sites to cause less electrical desychronization is a successful strategy to avoid long term complications of traditional RV pacing merits prospective evaluation.

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