Predictors of left ventricular dysfunction with right ventricular pacing: Is paced QRS duration the answer?∗

Right ventricular (RV) pacing has been demonstrated to have detrimental effects on cardiac hemodynamics and is associated with a reduction in left ventricular function [1]. It is thought that about 25% of patients receiving right ventricular pacing for sick sinus syndrome and complete heart block experience 'pacemaker syndrome' with symptoms of shortness of breath, dizziness, palpitations, abnormal pulsations or chest pain [2]. Minimizing right ventricular pacing with optimization of pacemaker settings such as rate responsiveness, atrioventricular (AV) delay management and mode switching should all be adopted [3]. Allowing spontaneous sinus rate where possible, with preservation of spontaneous AV conduction, and restricting pacing to only when absolutely necessary is paramount.

Many studies have been performed to try and identify predictors of progression of left ventricular (LV) dysfunction with RV pacing. To extrapolate conclusions from these studies there must be detailed information provided. For example, if the reported indication of pacing is complete heart block or sick sinus syndrome, knowing whether this is intermittent or permanent is important. This helps us understand the mode of pacing adopted such as single or dual chamber pacing. Knowing that RV pacing should be systematically avoided, more details would allow us understand whether long AV delays were implemented or was this not possible due to prolonged intrinsic AV conduction which may prevent this due to the development of diastolic mitral regurgitation or atrial fibrillation [4,5].

Complete heart block can be intermittent in the setting of tachycardia/bradycardia dependent heart block or initiated by a premature ventricular complex. Intrinsic AV block can result in an escape rhythm dependent on subsidiary pacemaker sites which can be unreliable and have a slow rate. The percentage of pacing should be reassessed at each pacemaker visit, or tele-monitoring transmission in order to minimize the amount of pacing if possible. Also the rate that RV pacing is set at has been associated with increased rate of adverse events with increased heart rate [6]. Knowing how patients presented, such as with syncope or congestive cardiac failure, can also be important to know why certain parameters were adopted.

Guidelines recommend that dual chamber devices be implanted in the setting of sinus node disease. In AV block, dual chamber pacing does not reduce morbidity (due to heart failure hospitalization) or mortality but is associated with less likelihood of pacemaker syndrome [2,7–9].

Alternative RV pacing sites, with His bundle, septal and right ventricular outflow tract pacing, have been studied as potential predictors of LV dysfunction for the last 2 decades. Analysis of 14 randomized controlled trials with 754 patients showed that a non-apical RV pacing position was better if there was impaired left ventricular function (<45%) but no difference with a normal baseline ejection fraction [10]. Other studies have failed to show the correlation of LV dysfunction with RV pacing site [11]. Part of the limitations of the pacing site for RV pacing is that operators may incorrectly identify a lead position as septal when it is actually free wall. Using the Right Anterior Oblique fluoroscopic image can help confirm a septal and RVOT position as can a 12 lead ECG and a lateral chest radiograph. In relation to the 12 lead ECG studies have reviewed quick algorithms to help operators identify easily if the paced rhythm is suggestive of a septal origin and a negative deflection in lead one appears to be a strong predictor [12]. A lateral chest X-ray showing the RV lead with a posterior deflection again is predictive of a septal position [13]. With all this said there are no definitive guideline recommendations on RV pacing lead position.

Paced QRS duration has also been evaluated as a predictor of congestive heart failure with RV pacing. In this issue, Sharama et al. suggest that narrow pQRSd, as a result of an RVOT pacing site, could potentially lead to less deterioration of LV function [14]. It appears it may potentially be a predictor in that with right ventricular apical pacing, the paced QRS duration which progressively prolonged from baseline to one year was associated with a progressive development in congestive heart failure [15]. Even those with initial relatively shorter paced QRS duration (pQRSd) who had a progressive

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change in \( pQRS_d \) was correlated with left ventricular dimensions and function [16]. The cut off value of paced QRS duration and correlation with left ventricular dysfunction and heart failure appears to be around 190–200 ms but these estimates are from small non randomised trials [17].

Correlation of the paced QRS duration, left ventricular function and mechanical dyssynchrony is not clear [18]. Information in studies about septal to lateral delay, left ventricular systolic and diastolic dimensions, and left ventricular ejection fraction have provided some insight.

Although in this study they report no patients experiencing new onset atrial fibrillation they do not go into details as to how they were assessing for the presence of atrial fibrillation [14]. Presumably they are referring to new onset symptomatic atrial fibrillation, and as many episodes of atrial fibrillation may be asymptomatic, they may not have fully appreciated the prevalence. Also when devices are single chamber or not details provided in dual chamber devices about mode switching it can be difficult to interpret the results. Of course the more you look asymptomatic atrial fibrillation the more it can be found [19].

Finally it is important to remember that patients with heart failure and conventional indications for pacemaker implantation may benefit from a cardiac resynchronization device from the outset. RV pacing in the presence of left ventricular dysfunction causes a progressive decline in left ventricular function and adverse remodelling which can be prevented by cardiac resynchronisation therapy [20]. It is still unclear whether they also have a better clinical outcome as there is a lack of long term data.

In the future, prospective randomized controlled trials with longer duration follow up and details about device optimisation/parameters will help us better define predictors of LV dysfunction with RV pacing. With this information, we would be able to choose the right device for our patients and hopefully prevent a deterioration in cardiac status as a result of interventions with new technologies.

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