

Academic dissertation: Ventricular activation patterns in conduction abnormalities and during different pacing modes

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Congestive heart failure (CHF) is a progressive disease caused by left ventricular dysfunction with high morbidity and mortality rates. Ventricular dysfunction is often linked to ventricular dilatation, which in turn may cause ventricular conduction delays and further worsening of the cardiac function. Recent decade gave rise to cardiac resynchronization therapy (CRT), a therapeutic modality based on premise that preexcitation of late activating regions by cardiac pacing may restore the left ventricular synchronous contraction.

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The goals of the publications included in the dissertation were: (1) to analyze the ventricular activation patterns in patients eligible for CRT with respect to the underlying heart disease and/or QRS morphology on the surface electrocardiogram (ECG), (2) to quantify changes in ventricular activation patterns during different pacing modes and (3) to compare the hemodynamic performance of different pacing modes during exercise.

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1. Patients with CHF and wide QRS complex represent a broad spectrum of underlying conduction disturbances with variable inter- and intraventricular delays. Those with coronary artery disease (CAD) displayed on the ECG predominantly nonspecific conduction disturbance pattern and had heterogeneous endocardial activation with predominant intraventricular conduction delay. Late activated regions were usually localized to the lateral aspect of the post-infarction scar. In contrast, patients with dilated cardiomyopathy (DCM) presented mostly with complete left bundle branch block (LBBB) or bifascicular block pattern. Those with LBBB had a more pronounced interventricular conduction delay with the latest left ventricular activation posterolaterally, the patients with bifascicular block showed a significant intraventricular delay with the latest activation located anteriorly. As those differences were not apparent from the ECG, this method is of limited value in the description of complex conduction abnormalities and thus for the selection of CRT candidates.

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2. Different pacing modes led to characteristic patterns of the left ventricular activation. Biventricular pacing shortened the left ventricular activation, minimized the interventricular delay and complexly modified the left ventricular activation pattern. Single-site left ventricular pacing was associated with similar characteristics provided by fusion of pacing wavefront with spontaneous septal activation. Right ventricular bifocal pacing resulted in a decrease in left ventricular activation time at the expenses of increase in interventricular delay. Single-site right ventricular apical pacing caused reversed left ventricular activation and led to the highest degree of inter- and intraventricular asynchrony.

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3. The comparison of hemodynamic performance of single-site left ventricular and biventricular pacing as assessed by stress test echocardiography revealed higher benefit of former pacing mode in DCM patients, which may be explained by the different nature of conduction abnormalities of these patients and CAD subjects. The DCM patients often present with predominant interventricular conduction delay, while the conduction within the left ventricle is rather preserved and homogeneous. In this case, single-site left ventricular pacing may activate the left ventricle relatively rapidly. On the other hand, CAD patients appear to have more expressed intraventricular delay within the left ventricle that reflects the presence of post-infarction scars. In this situation, it may be more important to activate the left ventricle from more sites.

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In conclusion, characteristic ventricular activation patterns can be observed during both spontaneous activation and different pacing modes. Multiple factors affect the resulting

activation pattern and this underscores the complexity of left ventricular dyssynchrony and the array of variables possible determining the success of CRT.

5 Although the degree of electrical synchronicity cannot be directly correlated to hemodynamic performance, the quantification of electrical activation patterns forms a solid background for understanding the principles and mechanisms underlying the effect of the resynchronization therapy.