Abstract: P1168

AV delay optimisation in patients with congenitally corrected transposition of the great arteries

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Topic(s):
Cardiac Resynchronization Therapy

Citation:
Background: Adults with congenitally corrected transposition of the great arteries (ccTGA) are at risk of systemic right ventricular (RV) failure and cardiac arrhythmias requiring the implantation of pacemakers, ICDs and CRT devices. The importance of atrioventricular delay optimisation in patients with ccTGA has not been investigated. This study assessed the acute response to AV delay optimisation and difference between the empirically programmed AV delay and optimal AV delay in patients with ccTGA.

Methods: Records of eight patients with ccTGA and a dual chamber ICD or CRT device who had undergone echocardiographic AV delay optimisation were reviewed (86% men, mean age 49±8 years, mean systemic right ventricular fractional area change 28±3%, 100% ventricular or biventricular pacing). The optimal AV delay was compared to that programmed empirically prior to optimisation. Diastolic filling time and tricuspid valve regurgitation time data were reviewed at every tested AV delay. For comparison between subjects the optimal AV delay during sequential atrial and ventricular pacing was included in the analysis as 6 out of 8 patients were atrial pacing during optimisation.

Results: The median optimal paced AV delay assessed by echocardiography was 95ms (min AV delay 60ms, max AV delay 140ms). This was significantly shorter than the empirically programmed AV delay (median empirical AV delay 176ms: min 150ms, max 220ms) in all patients (mean difference: -80±41ms; P<0.03). A =40ms increase from the optimal AV delay decreased diastolic systemic right ventricle filling time by 40±16ms (p<0.05) and increased diastolic tricuspid valve regurgitation time by 38±25ms (p<0.05).

Conclusions: Atrioventricular dyssynchrony may be present in the paced ccTGA population and optimisation of the AV delay may be necessary to correct suboptimal empirical settings. The optimal AV delay in patients with ccTGA may be shorter than in patients with structurally normal hearts. The mechanisms underpinning a short optimal AV delay in patients with ccTGA could be related to the abnormal electromechanical properties of the systemic right ventricle. Future studies should assess whether AV delay optimisation has a benefit on clinical outcomes in patients with ccTGA.