

Electrical and Mechanical Cardiac Resynchronisation by Novel Direct His-bundle Pacing in a Heart Failure Patient

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Efficacy of standard cardiac resynchronisation therapy (CRT) by biventricular pacing via coronary sinus depends on the target site for left ventricular (LV) pacing, which in a not insignificant number of patients is limited by anatomical constraints. Direct His-bundle pacing (DHBP) is considered an alternative method of pacing for patients requiring cardiac stimulation in order to obviate detrimental effects of right ventricular pacing on LV function. However, its role in CRT has not been investigated, with scarce number of cases recently reported. We present a case of a heart failure patient in whom CRT was considered and treated by DHBP. In addition to electrical resynchronisation and optimal clinical response, echocardiography showed successful ventricular mechanical synchrony. To our knowledge, these latter findings are for the first time described in the setting of CRT by DHBP.

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Introduction

Recognised deleterious effects of right ventricular apical pacing in certain patients have led to direct His-bundle pacing (DHBP) to be proposed as one of the alternative pacing sites for patients who require cardiac stimulation [1]. However, in the context of cardiac resynchronisation therapy (CRT), DHBP has been scarcely investigated, despite that it seems to be an ideal strategy from a physiological standpoint, preserving the intrinsic activation sequence of both ventricles.

A recent study has demonstrated that electrical synchronisation by DHBP is feasible [2]. Nevertheless, the haemodynamic effect of DHBP for CRT, as assessed by echocardiography, has not been previously described. We present a case about CRT by DHBP in which electrical and mechanical synchrony of the left ventricle (LV) was successfully achieved.

Case Report

A 62 year-old man with history of dilated cardiomyopathy and persistent NYHA function class III despite optimal

pharmacological treatment was presented with congestive heart failure. His 12-lead electrocardiogram showed sinus rhythm with left bundle branch block and QRS duration of 150 ms. Two-dimensional transthoracic echocardiography showed a dilated LV (end diastolic diameter of 65 mm) with severe systolic dysfunction (LV ejection fraction of 30%). CRT was considered according to current guidelines.

The device implantation procedure was performed in the cardiac electrophysiology laboratory. Standard CRT via coronary sinus was unsuccessful despite several attempts. His-bundle lead implantation was therefore considered under prior patient's informed consent. Implantation technique of His-bundle lead was performed as follows: a 52 cm Tendril Model 1888 TC active-fixation permanent lead (St Jude, Sylmar, CA, USA) with a retractable and active helix of 1.8 mm length was connected to a polygraph and advanced to the His area using a steering guidewire (also called preformed stylet [4]) and a diagnostic catheter as anatomical reference. The lead was initially positioned in the right ventricle outflow tract; using a steering guidewire, the lead was gently withdrawn adjusting its curve as the lead tip was positioned close to the diagnostic catheter. By rotation, the lead tip engaged septum whilst recording His potential. Once His deflection was recorded, temporary pacing was initiated. The specific pattern of ventricular activation exhibited during His-bundle pacing, with normalisation of QRS complex and repolarisation pattern, and latency between the pacing stimulus artefact and the QRS complex, demonstrated

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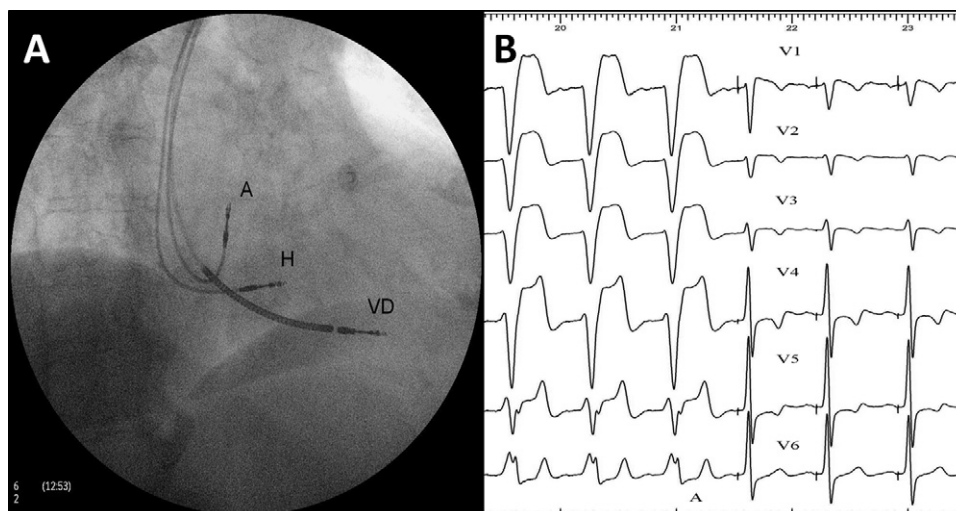


Figure 1. Implantation of resynchronisation device with lead positioned in the His area. (A) Posteroanterior projection X-ray showing a defibrillator with His-bundle pacing resynchronisation; A, lead in the right atrial appendage; H, lead positioned in the His bundle; VD, lead in the right ventricular apex. (B) The 12-lead electrocardiogram shows narrow QRS complexes following His-bundle pacing.

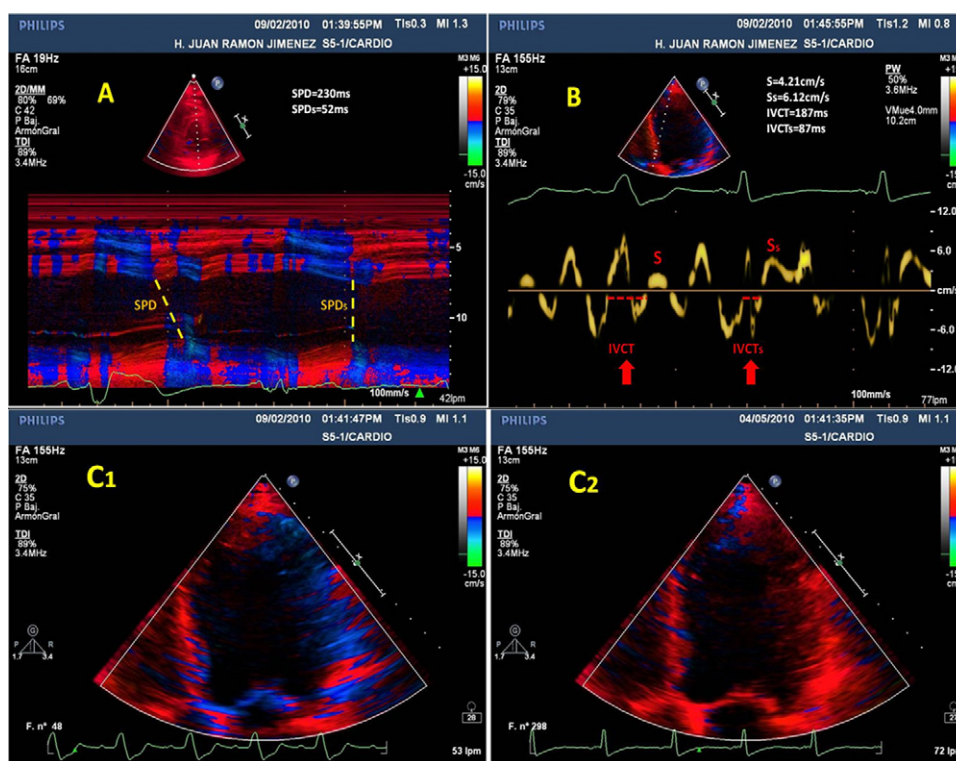


Figure 2. The transthoracic echocardiogram shows LV mechanical changes secondary to electrical resynchronisation by DHBP, demonstrating an efficient LV contraction. In (A) and (B) the first heart beat shows basal LBBB, which is followed by normalised QRS with hisian stimulation in the subsequent heart beats (sweep speed = 100 mm/s). (A) M-mode colour tissue Doppler depicts immediate abolition of septal-to-posterior wall delay (from 230 ms to 52 ms) when LBBB disappears by DHBP. (B) Pulsed-wave tissue-Doppler shows decreased isovolumic-contraction-time (from 187 to 87 ms) with increased peak systolic velocity (from 4.21 to 6.12 cm/s) simultaneously to the narrowing of QRS. (C) Synchronous ventricular contraction obtained after narrowing QRS by DHBP is represented by 2D colour tissue Doppler (depicted as homogeneously-distributed red colour within the ventricular wall at end-systole (C2) compared to previous heterogeneous contraction (C1) in the presence of LBBB). [SPWD = septum-to-posterior wall delay; IVCT = isovolumic contraction time; S = peak systolic velocity; sub-index s = stimulation].

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