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PROCEDURE TECHNIQUE AND A RARE INTRAPROCEDURAL COMPLICATION DURING PERMANENT HIS BUNDLE PACING

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A case of successful endocardial lead implantation into the His bundle position is presented. Procedure technique and transient atrioventricular block during implantation are described.

Key words: cardiac pacing; atrial fibrillation; heart failure; atrioventricular block; His bundle pacing; complication

Conflict of Interests: nothing to declare

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His-bundle pacing (HBP) is a new method for brady-arrhythmia management. Data in favor of using HPB with pacemaker [1] and cardiac resynchronization device [2, 3] is continuously rising. Planned AV nodal ablation for rate control in atrial tachyarrhythmias with fast AV conduction

is considered as an indication for HBP since 2019 [4-6]. The risk of intraprocedural and postprocedural complications (lead dislodgement, pacing threshold rise, exit block) is an important factor precluding many electrophysiologists from the wide use of permanent HBP.

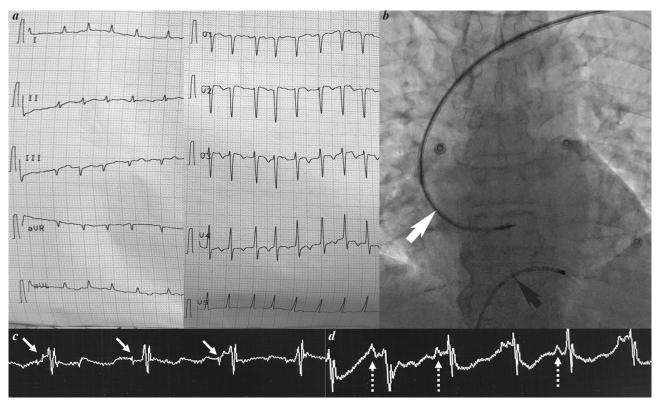


Fig. 1. a. ECG at the admission, 25 mm/s. Atrial fibrillation with the fast ventricular response, HR 168 bpm, QRS 80 ms. b. Delivery tool (white arrow) is positioned in the upper part of the triangle of Koch to register the His bundle electrogram and subsequent lead fixation (The pacing lead is inside the sheath). A steerable diagnostic catheter (grey arrow) is positioned in the right ventricle for the safety pacing in case of AV conduction disturbances during the His bundle lead fixation. c, d: Local electrogram from the His bundle pacing lead. C. His bundle EGM before the lead fixation - low amplitude fibrillatory atrial activity, irregular ventricular spikes with preceding narrow His bundle spikes (white arrows) D. His bundle EGM after the lead fixation. His bundle spikes (dotted arrows) morphology changed, current of injury is visible.

We present a rare case of transitional AV conduction impairment during HBP lead fixation in a patient with atrial fibrillation and tachy-induced cardiomyopathy.

A fifty-four-year-old male patient was hospitalized due to chronic heart failure. Atrial fibrillation of unknown duration and high ventricular rate of 168 bpm was diagnosed for the first time (Fig. 1, A). Oral anticoagulation (rivaroxaban 20 mg daily), diuretic (furosemide) and rate-control (digoxin 250 µkg daily, metoprolol 50 mg daily) therapy was prescribed. Echocardiography revealed diffuse left ventricular (LV) contractility reduction with LV ejection fraction (EF) of 20%. The NT-proBNP level was increased to 3085 ng/ml.

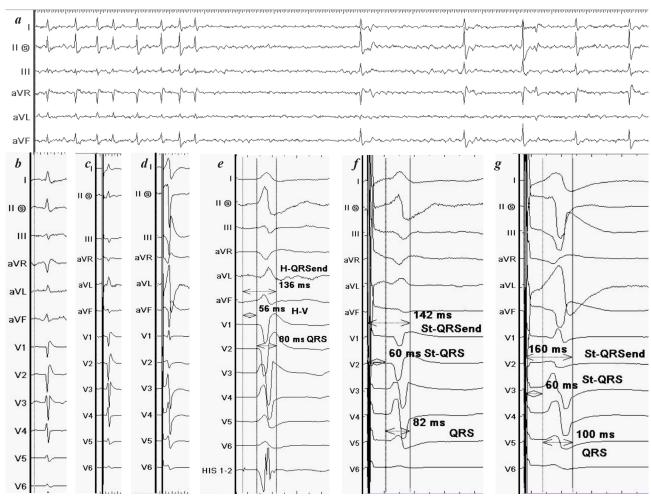


Fig. 2. a. ECG during the His bundle pacing lead fixation, 25 mm/s. Atrial fibrillation with HR 120-160 bpm is followed by 4,7 s pause and then restoration of AV conduction with HR 40-50 bpm. b-d: 12-lead ECG (25 mm/s) demonstrates similar QRS morphologies (RBBB) during spontaneous rhythm (b) and selective HBP (c). During the non-selective HBP (after the lead fixation) the RBBB is resolved but the axis becomes vertical (see the text). e-g: 12-lead ECG (100 mm/s) demonstrates similar duration of intervals H-V (56 mc)/H-QRSend (136 mc) during spontaneous conduction (e) u St-QRS (60 mc)/St-QRSend (142 mc) during selective HBP (f) before the lead fixation. QRS width (80 ms) is also similar. With nonselective HBP after the lead fixation (f), there is some change in the QRS morphology (incomplete RBBB resolution, left axis deviation) and QRS width (100 ms) and St-QRSend interval (160 ms) rise.

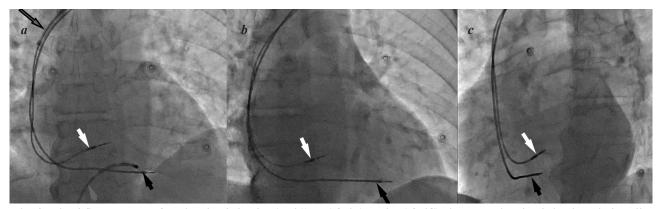


Fig. 3. Final fluoroscopy of pacing leads in the AP (A), RAO (B) and LAO (C) views. Pacing leads in the His bundle (white arrow) and the interventricular septum (black arrow), decapolar diagnostic catheter (grey arrow) and delivery sheath (figured arrow) are marked.

The attempts to lower the heart rate were ineffective due to the hypotension which became more pronounced with metoprolol doses higher than 50 mg daily. The rhythm-control strategy was not applicable due to the presence of mobile LV thrombus revealed by transthoracic echocardiography. Severe heart failure status and several prothrombotic factors (low LV EF, uncontrolled high ventricular rate) did not allow us to wait for the spontaneous lysis of the thrombus on the anticoagulant therapy. Due to the inability to effectively control heart rate or rhythm, pacemaker implantation and subsequent AV nodal ablation were decided to be a single option. As the right ventricle pacing could lead to the additional LV EF reduction and cardiac resynchronization therapy was not technically applicable, we decided to perform permanent His-bundle pacing.

Procedural data

Two endocardial leads (active-fixation CaptureFix Novus 5076 58 cm (Medtronic, USA) and passive-fixation ELBI-211 58 cm (ElestimKardio, Russia) were positioned through the left axillary vein to the right atrium. ELBI-211 lead was introduced to the right ventricle and fixed at the apical trabeculated part of the interventricular septum. The right femoral vein was canulated by the 8F hemostatic introducer and decapolar steerable diagnostic catheter CS EZSteer D-F (Biosense Webster, USA) used to map interatrial septum, Koch's triangle, and His bundle area. Then CaptureFix Novus lead was positioned in the upper part of the Koch's triangle using AcuityPro CS-EH-STR-curve (Boston Scientific, USA) (Fig. 1, B). His bundle electrogram was registered (Fig. 1, C) and selective HBP was performed with a pacing threshold of 1,7V@0,4ms (Fig. 2, C and F). His bundle injury-current was revealed during lead fixation (Fig. 1, D) [1,7] and the pacing threshold was not changed after the fixation. Before the splitting-out of the delivery sheath, all entire His pacing lead was screwed in clockwise for more durable fixation. During this manipulation, the intermittent AV block with 5-second pause and subsequent ventricular rate deceleration to 50-80 bpm was documented (Fig. 2, A). This lower heart rate stayed until the end of the procedure despite the bolus injection of 1 mg of atropine and 8 mg of dexamethasone. Effective non-selective HBP

was performed after the delivery sheath removal

(Fig. 2, D and G). Right ventricular myocardial and His bundle pacing thresholds did not differ and therefore the capture was lost simultaneously during threshold testing without any change in QRS morphology while the pacing amplitude was decreased. Interestingly, the St-QRSend interval duration during non-selective HBP was slightly longer than H-QRSend during the spontaneous conduction (Fig. 2, E-G). In our opinion, this phenomenon could be explained by the partial capture of His bundle fascicles responsible for the conduction to the right and left posterior bundle branches, causing the morphology of RBBB and left axis deviation. Both leads were fixed and connected to the 460 DR (ElestimKardio, Russia) dual-chamber pacemaker. The HBP lead was connected to the atrial channel. The device was programmed to DVI mode with a 60 bpm lower rate. Leads' position in the AP, LAO, and RAO fluoroscopy views is presented in Fig.3.

Postprocedural follow-up. Repeat Holter monitoring after the procedure revealed the decrease in mean daily heart rate to 80 bpm (from 40 to 100 bpm) despite the withdrawn rate-decelerating medications. Echocardiography before the discharge revealed the increase in LV EF to 25%. LV thrombosis had been also resolved probably due to the anticoagulant therapy. Repeat hospitalization was planned to perform electrical cardioversion but ambulatory ECG revealed a sinus rhythm of 72 bpm and normal AV conduction (PQ 150 ms). Taking into account the absence of implanted atrial lead, we decide not to ablate the AV node. Leads' parameters are presented in Table 1. Unfortunately, the patient was lost for follow up and catheter ablation to prevent AF recurrence was not performed.

Table 1. His-pacing lead parameters, measured during and 1 months after the implantation.

Parameters		Intraprocedural	1 months after
His bundle lead	R-wave	3.8 mV	5.6 mV
	Impedance	425 Ohm	520 Ohm
	Threshold	1 V @ 0.4 ms	1.5 V @ 0.4 ms
Right ventricular lead	R-wave	12 mV	9,6 mV
	Impedance	550 Ohm	600 Ohm
	Threshold	0,3 V @ 0.4 ms	0,5 V @ 0.4 ms

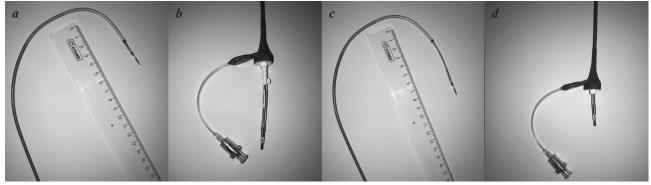


Fig. 4. The lengths of the non-steerable delivery sheath and standard active fixation endocardial pacing lead CaptureFix Novus 5076 58 cm (Medtronic). A and B. If the pacing lead is introduced with fixing cuff, its distal tip is out of the delivery tool by 3 cm. It makes pacing lead dislodgement the intracardiac manipulations and delivery sheath cutting out more probable. C and D. If the pacing lead is introduced without fixing the cuff, its distal tip is out of the delivery tool by 6 cm. It makes pacing lead dislodgement the intracardiac manipulations and delivery sheath cutting out less probable.

DISCUSSION

This case is interesting from several points of view. Firstly, this case is one of the first description conduction system pacing with standard endocardial active-fixation lead and unspecialized delivery tool. Using the lead with retractable screw and non-steerable delivery sheath, which is routinely used for CS catheterization, is not widely used. There are two techniques to deploy the pacing lead to the His bundle: through the delivery sheath and over the stylet. The last one has demonstrated relatively low effectiveness [8] but has low costs. The most widely used variant of the first approach is "classical" 3830 lead implantation using non-steerable C315 (Medtronic, USA) or steerable C304 (Medtronic, USA) delivery tools. Nonetheless, the use of unspecified delivery tools and standard endocardial leads is also possible [9]. In our case, we used endocardial active-fixation lead CapsureFix Novus 5076 58 cm (Medtronic, USA) and non-steerable delivery sheath AcuityPro CS-EH-STR-curve (Boston Scientific, USA). Lead and delivery tool lengths should be considered during the preimplantation setup. Pacing leads of 52 cm length are too short to be used. Removing the fixing cuff is useful before implantation of 58 cm leads allows more deep lead insertion into the delivery sheath and slack formation in the right atrium before slitting out the sheath (Fig. 4).

The second interesting moment is the AV nodal conduction impairment during the procedure. In our case, AV nodal conduction deficit persisted until the discharge and caused a withdrawal of beta-blockers and digoxin. The AV block could be caused by the AV nodal artery damage, or by the mechanical injury of the His bundle (or AV nodal) tissue, which is diagnosed in 1,1% of conduction system pacing cases [10,11].

Heart wall deformation in the Koch's triangle during the additional lead rotation could also be the reason for AV block. In such cases AV conduction impairment should be intermittent and resolve within several days after the injury. We consider this mode of AV conduction impairment the most probable.

Of complications, which are traditionally mentioned after HBP, the most prevalent are lead dislodgement (0,6%) and threshold rise (2,1%) [8]. Infection, exit block, and detection problems are rarer. It is known from private communication with the opinion leader in HBP that AV conduction impairment during the lead fixation is sometimes happening (with no exact prevalence), but is usually intermittent, resolves by the end of the procedure, and is not affect the final complication statistics. Nonetheless, it is considered to use the additional temporary lead in the RV during the first procedures of HBP [1,12].

LIMITATIONS

The alternative mechanism of His bundle electrogram changes after the lead fixation is the far-field potential registration. We could not differentiate those two options (injury-current and far-field electrogram) using the data we had and mentioned the most probable in our opinion.

CONCLUSION

His bundle pacing is a perspective technique to treat patients with AF and uncontrolled ventricular rate before the AV nodal ablation. HBP allows physiological synchronous excitation of ventricular myocardium over the conduction system. Intermittent AV nodal conduction impairment is a rare but possible intraprocedural complication of lead implantation into to the His bundle. It doesn't need any additional interventions but follow up.

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