

Normalization of the EKG in patients with right bundle branch block by Right ventricular pacing

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Right ventricle apex (RVA) pacing bypasses the His-Purkinje system resulting in left bundle branch block like pattern on surface EKG. In preexisting right bundle branch block (RBBB), pacing from certain location of RV might change the EKG pattern. We present two cases of normalization of QRS complex upon RV septal pacing in preexisting RBBB patients. The postulated mechanisms of the phenomenon is also proposed.

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Keywords: RV pacing, RBBB, QRS normalization.

Normalisasi EKG pada Pasien dengan Blok Berkas Cabang Kanan yang Dilakukan Pemacuan Ventrikel Kanan

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Pemacuan di apeks ventrikel kanan mem-by pass sistem His-Purkinje menghasilkan gambaran EKG seperti blok cabang berkas kiri. Pada pasien dengan gambaran blok cabang berkas kanan sebelumnya, pemacuan pada lokasi tertentu di ventrikel kanan dapat merubah pola EKG yang dihasilkannya. Kami menyajikan dua kasus normalisasi kompleks QRS setelah pemacuan di septal ventrikel kanan pada pasien dengan blok cabang berkas kanan. Kemungkinan mekanisme perubahan tersebut juga kami ajukan dalam tulisan ini.

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Kata kunci: pemacuan RV, RBBB, normalisasi QRS.

The right ventricular apex (RVA) has been the elective site for placing endocardial pacing leads since 1959 when Furman described the use of the transvenous route of pacemaker implantation. This site was used because it is easily accessible, readily identified and associated with a stable position and reliable chronic pacing parameters. It was recognized, however, that pacing from the RVA did not reproduce normal ventricular conduction and contraction.^{1,2} RVA pacing appears to have potential deleterious effects on myocardial systolic and diastolic left ventricular function, especially in patients with intact AV conduction.^{3,4}

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More than 80 years ago, in 1925, Wiggers showed that RVA pacing in mammals was associated with a marked reduction in left ventricular (LV) pump function. RVA pacing bypasses the His-Purkinje system resulting in a left bundle branch block like pattern on surface on the EKG.⁵ Pacing from the RV apex produces an abnormal activation of the lateral wall of the left ventricle (LV), with ventricular remodeling resulting from neurohormonal and electrophysiological changes.⁶ This induces differential muscle strain and fibre shortening, which in turn increase myocardial work and oxygen consumption.^{7,8} The resultant changes in cardiac haemodynamics cause LV cellular abnormalities at both a gross and ultrastructural level, and ultimately lead to ventricular dilatation and impaired lusitropy.^{9,10} More importantly, LV contraction is altered and dyssynchrony may be induced. Subsequent

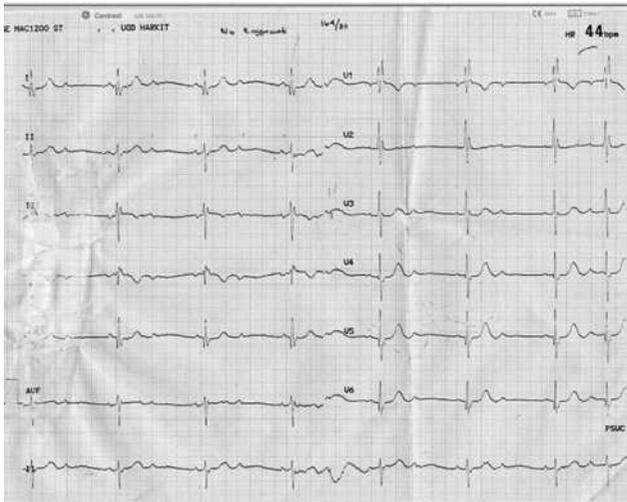


Figure 1. EKG showed sinus rhythm with QRS rate 44 x', QRS axis LAD, normal P wave (0.08"), prolonged PR interval (0.32"), QRS duration was 0.10"), persistent S in V5-V6 , complete RBBB with Mobitz type 2 (2:1)

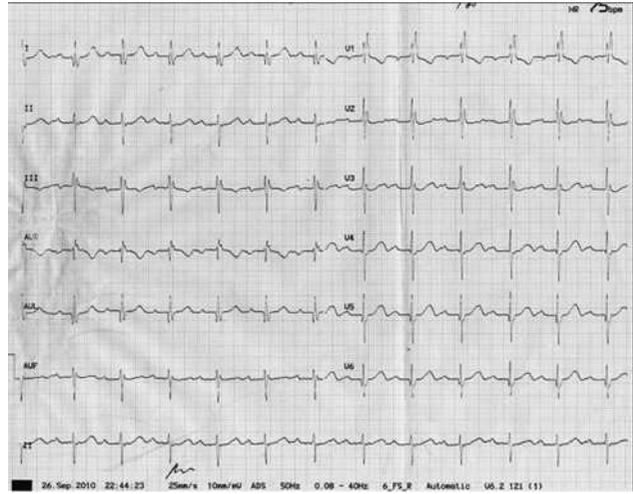


Figure 2. EKG before the procedure showed sinus rhythm, QRS rate was 75 x', LAD, with trifascicular block

studies showed that pacing results in LV remodeling with asymmetric hypertrophy and dilatation, mitral regurgitation, reduced myocardial perfusion and reduced ejection fraction.^{5,11,12}

New pacing sites in the right ventricle are being explored to overcome these detrimental effects. Alternative pacing sites in the right ventricle are the right ventricle outflow tract (RVOT) and the right ventricular septum (RVS).^{3,13}

In this case report, we demonstrate a form of ventricular fusion, namely normalization of the QRS complex in patient with pre-existing right bundle branch block by RVOT pacing.

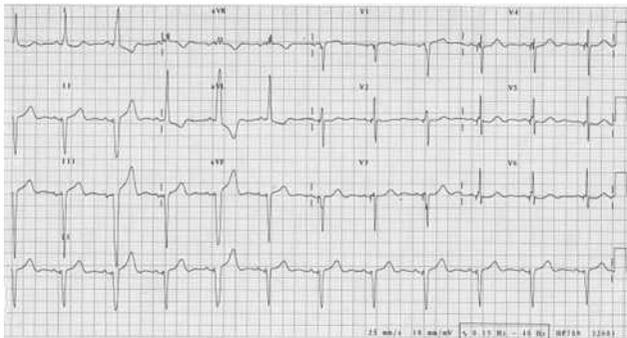


Figure 3. EKG post PPM implantation revealed sinus rhythm, QRS rate was 75 x', QRS axis was LAD, with incomplete LBBB configuration

Case report

An 81 years old woman admitted to National Cardiovascular Centre Harapan Kita (NCCHK) with palpitation and headache since one month prior to admission. One day before admission she had an episode of near syncope without any prodromal complaints. She had history of hypertension with an irregular control to the out patient clinic. The oral medicine was amlodipine (Norvask) 5 mg daily. Risk factor for coronary artery disease was hypertension, menopause and smoking.

From physical examination, her blood pressure was 210/75 mmHg, heart rate was 45 x/ minute, respiratory rate was 20 x/ minute. No anemic or icteric found, while from the chest examination pansystolic murmur grade II/6 was detected at the apex. Abdomen and both extremities were normal.

The chest X ray showed CTR was 55% with dilated aortic segment, normal pulmonary segment, positive cardiac waist and downward apex. The laboratory examination revealed Hb was 12.9 g/dl, hematocrit was 35, Leucocytes count was 7440/mm³, ureum was 35, BUN was 16, Creatinin was 0.9, blood glucose was 112 with normal electrolytes count (Na was 142, K was 4.4 , total Ca was 2.4, Chloride was 98 and Mg was 1.7).

Echocardiography examination concluded normal systolic function with ejection fraction was 57%, diastolic dysfunction ,with septal to lateral delay was 100 ms, E/A < 1 and diastolic time was 355. Increased of

left ventricular end diastolic pressure was found.

Patient was planned to undergo permanent pacemaker implantation.

PPM implantation was performed with DDDR mode, using atrial lead (Medtronic 4592-53 cm) inserted into RA appendage which showed that output threshold was 0.5 v; current 1 mA; P wave 5.2 mV, and resistance was 422 ohm. Ventricle lead (Medtronic 5076-58 cm) was inserted into RVOT which showed output threshold was 0.8 V; current was 0.9 mA; resistance was 444 ohm; and R wave 8 mV. After fixation of leads to surrounding muscle and tissue, an appropriate size of pocket was stitched subcutaneously and Unasyn 1.5 g was doused. The generator was connected to the leads and the EKG showed atrial sensing and ventricle pacing. The incised wound was stitched and PPM was set with DDDR 80 bpm, AV delay was 150 ms. No complication occurred during the procedure. Patient was clinically stable. During the follow up of the patient, the EKG showed a different pattern than previously before PPM procedure in which normalization of RBBB occurred post cardiac RVOT pacing.

Echocardiography post PPM implantation revealed that septal to lateral delay was 70 ms with E/A <1 and diastolic time was 466.

Case 2

A 46 years old man with chronic heart failure NYHA functional class III due to severe Mitral Stenosis, Pulmonal hypertension, LA thrombus and Tricuspid regurgitation caused by Rheumatic Heart Disease underwent Mitral Valve Replacement, Tricuspid anuloplasty and thrombus evacuation. Pre operative examination showed that his 12 leads surface ECG was atrial fibrillation and RBBB with QRS duration 0.14 msec. Unfortunately there was bradyarrhythmia complication post operation, ECG showed Idioventricular escape rhythm and sometimes junctional escape rhythm with long pause. The 4th day after surgery the rhythm was still the same so TPM was performed. Then he was implanted PPM as bradyarrhythmia did not resolved after 12 days. VVIR type was implanted, with active lead inserted through right axillaries artery to RV low septal. Beyond expectation, ECG post PPM showed pacing rhythm with normal QRS duration (0,10 msec). In fact, there was also better myocardial contraction proved by echocardiography with normalization septal movement and increasing ejection fraction from 45% to 56% after 6 weeks.



Figure 4. ECG preoperative: Atrial fibrilasi, Right QRS axis, RBBB complete, QRS duration 0,14 sec



Figure 5. ECG on Temporary Pace Maker : Pacing rhythm, Right QRS axis, QRS duration 0.22 sec, LBBB configuration

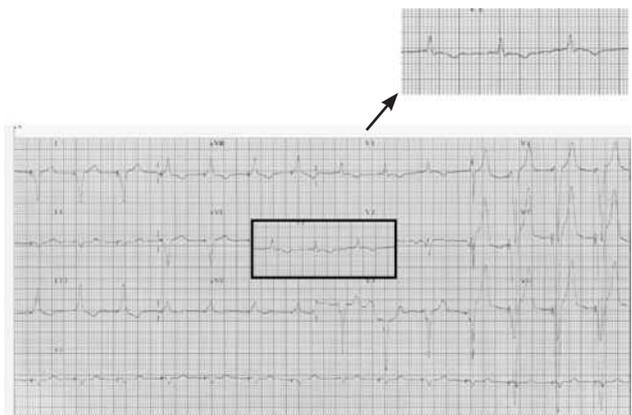


Figure 6. ECG on Permanent Pace Maker VVIR (lead at RV low septal), Right QRS axis, QRS duration 0.10 sec, RBBB configuration

Discussion

Four goals have been defined to optimize pacemaker therapy, particularly in patients with sinus node disease:⁵

1. Prevent symptomatic bradycardia
2. Provide chronotropic competence when necessary
3. Maintain AV synchrony
4. Maintain ventricular activation

To prevent the deleterious effects of long-term right ventricular pacing, alternative Pacing sites have been studied.¹ The RVOT and right ventricular septum (RVS) are the most frequently described alternative right ventricular pacing sites. Numerous acute haemodynamic studies have shown an improvement in cardiac output with pacing leads placed in these sites and this led to limited work in improving haemodynamic in impaired hearts.² These sites are theoretically associated with a more physiological ventricular activation. Despite the perceived advantages of septal pacing, results to date are not confirmatory. On review of early work of Durrer et al in 1970 the septal regions of the RVOT and mid RV are the first zones of the ventricle to depolarize, suggesting that pacing from these areas on the right side of the septum would achieve as normal contraction pattern as possible. In contrast, the free wall of the RV is the last zone to be depolarized.¹³

The safety and efficacy of active fixation leads in RVA has been well validated, however, positioning the leads in an RVS or RVOT position may be associated with an increased risk of dislodgement or even perforation, particularly with the thinner tissue of the RVOT region.^{2,14}

RVOT septal pacing is associated with shorter QRS durations than anywhere else in the RV and, in particular, the RVOT free wall. This suggests that pacing from the septal RVOT, although not as good as intrinsic conduction, may be the most desirable site for chronic RV pacing, as narrow QRS duration is associated with an improved LV dynamic.¹⁴ However RVS and RVOT pacing both produce an LBBB and wide QRS complex during pacing, but a reduction in QRS duration has been observed in comparison with the QRS duration of pacing from the RVA.³ De Cock et al. compared the haemodynamic effects of RVOT and RVA pacing and found that RVOT pacing is superior to RVA pacing. Direct His-Bundle pacing (DHBP) produces a normal QRS complex in patients with intra- and infra-Hissian conduction. In

terms of haemodynamics and ventricular function, DHBP is superior to RVA, RVS and RVOT pacing, since DHBP results in sequential synchronous right and left ventricular activation. Unfortunately, after mapping and localizing the His-bundle tissue the procedure was successful in only 60% of the patients. Besides, a serious limitation of DHBP is that it can only be deployed in patients with conduction abnormalities located proximally to the pacing site.^{3,15}

These patients had RBBB meant QRS already wide before pacing so due to some studies that RV septal pacing will give shorter QRS duration, so lead was inserted to low septal RV.¹⁶ Septal pacing produced typically a negative or isoelectric vector in lead I. Indeed, this feature has a 90% positive predictive value for septal placement. RV apical pacing may give prolong QRS duration, left BBB morphology but still right axis deviation due to RBBB. Pacing at RV apex start from inferior part of the heart goes superiorly but Low septal RV pacing will travel superior and also inferiorly. Electrical activation of the left ventricle differs when comparing sinus rhythm and RV apical pacing, the main difference is only a single break-out site on the LV endocardium with apical pacing. On Sinus rhythm there are two break-out locations on LV endocardium: Inferior border of the mid-septum and superior basal aspect of free wall.¹⁷

Our postulated mechanism of QRS narrowing in this RBBB patient is due to time period of depolarization from right side which stimulated by pacing at low septal RV and from the left bundle branch was nearly the same.

Conclusion

Patients with RVS pacing and pre-existing RBBB demonstrated completely normal QRS complex, which was the result of ventricular fusion by pacing distally from anatomical position of the right bundle branch block and spontaneous ventricular activation over the left bundle of the His-Purkinje system. This mechanism was due to the time period of depolarization from the right side and from the left bundle branch was nearly the same. Follow up of these patients were necessary to establish how stable this pattern of normalization remains. Further investigation was required to observe the normalization pattern of pre-existing RBBB with RVOT and RVS pacing.

References

1. Albouaini K, Alkarni A, Mudawi T, Gamage MD, Wright DJ. Selective site right ventricular pacing. *Heart*. 2009;95:2030-2039
2. Harris ZI, Gammage MD. Alternating right ventricular pacing sites – where are we going? *Europace*. 2000;2:93-98.
3. Scheffer M, Tanis GJ, van Mechlen R. Normalization of the ECG in a patient with right bundle branch block by cardiac pacing. *Neth Heart J*. 2005;13:151-153.
4. Riedlbauchova L, Kautzner J, Hatala R, Buckingham TA. Is right ventricular outflow tract pacing an alternative to left ventricular/ biventricular pacing? *Pacing Clin Electrophysiol*. 2004;27:871-877.
5. de Cock CC. Fifty years of cardiac pacing : the dark side of the moon? *Neth Heart J* 2008;16:S12-S14.
6. Wyman BT, Hunter WC, Prinzen FW. Mapping propagation of mechanical activation in the paced heart with MRI tagging. *Am J Physiol*. 1999;276:881-891.
7. Prinzen FW, Hunter WC, Wyman BT. Mapping of regional myocardial strain and work during ventricular pacing: experimental study using magnetic resonance imaging tagging. *J Am Coll Cardiol*. 1999;33:1735-1742.
8. Delhaas T, Arts T. Regional fibre stress-fibre strain area as an estimate of regional blood flow and oxygen demand in the canine heart. *J Physiol* 1994;477:481-496.
9. Giudici MC, Thornburg GA, Buck D. Comparison of right ventricle outflow tract and apical lead permanent pacing on cardiac output. *Am J Cardiol*. 1997;79:209-212.
10. Tse HF, Lau CP. Long term effect of the right ventricular pacing on myocardial perfusion and function. *J Am Coll Cardiol* .1997;29:744-749.
11. Tantengco MV, Thomas RL, Karpawich PP. Left ventricular dysfunction after long term right ventricular apical pacing in the young. *J Am Coll Cardiol*. 2001;37:2093-2100.
12. Johnson F, Jayesh B, Ashiskumar M, Faizal A, Mond H. Right ventricular septal pacing: Has it come of age? *Indian Pacing Electrophysiol J*. 2010;10:69-72.
13. Tim JF, Cate T, Mike GS, George RS, Fred JV, Nobert MVH. Right ventricular outflow and apical pacing comparably worsen the echocardiographic normal left ventricle. *Eur J Echo*. 2007;9:672-677.
14. Richard JH, Harry GM. Pacing the right ventricular outflow tract septum – Are we there yet? *Asia Pacific cardiol*. 2007;1:57-59.
15. De Cock CC, Giudici MC, Twisk JW. Comparison of the haemodynamic effects of the right ventricular outflow-tract pacing with right ventricular apex pacing: a quantitative review. *Europace*. 2003;5:275-8.
16. Mond HG, Gammage MD. Selective site pacing: The future of cardiac pacing? *Pacing Clin Electrophysiol*. 2004;27:835-836.
17. Cassidy DM, Vassallo JA, Marchlinski FE, Buxton AE, Untereker WJ, Josphson ME. Endocardial mapping in humans in sinus rhythm with normal left ventricles : activation patterns and characteristics of electrograms. *Circulation*. 1984;70:37-42.