Electrocardiographic Findings in Accessory Right Precordial Leads in Adults and Seniors with Notched S Waves in Lead V₁—A Preliminary Study

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Background: Atypical right bundle branch block (RBBB) may present with an rS pattern and notched S wave in lead V₁. The notched S wave may represent slowed conduction or delayed activation of the right ventricular conduction system or ventricular myocardium.

Methods: We retrospectively analyzed the QRS patterns in accessory right precordial leads (from V₃R to V₅R) in 15 adults/senior individuals with notched S wave in lead V₁.

Results: In the right accessory precordial leads, 13 showed triphasic QRS pattern with final R' wave in their QRS complexes. This QRS pattern in association with notched S wave in lead V₁ is suggestive of the presence of RBBB (incomplete or complete).

Conclusions: A notched S wave in lead V₁ and in the right precordial accessory leads associated with a final R' wave suggests the possibility of concealed RBBB (incomplete or complete).

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notched S wave in V₁; right bundle branch block; right precordial accessory leads

INTRODUCTION

A notched S wave in lead V₁ and other leads signifies conduction delay (depolarization phenomena) and is observed in several structural heart diseases, such as myocardial infarction, arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D), and other cardiomyopathies.^{1–3}

Right bundle branch block (RBBB) may present an rS pattern with notched S wave in lead V₁ (atypical RBBB).⁴ However, RBBB is often seen in patients with acute coronary syndrome (ACS) and as a transient phenomenon in acute pulmonary embolism (APE).^{5–7} In the context of ACS, the presence of new RBBB provides

prognostic information.⁷ In the presence of massive APE, the transient appearance of incomplete (IRBBB) or complete (CRBBB) RBBB is caused by right ventricular strain and a conduction delay in the stretched myocardium or the peripheral conduction system. Right ventricular strain is diagnosed in the presence of one or more of the following electrocardiogram (ECG) findings: complete or incomplete RBBB, S1Q3T3, and negative T wave in V₁–V₄. The right ventricular strain pattern on the ECG is associated with adverse short-term outcome and adds incremental prognostic value to echocardiographic evidence of right ventricular dysfunction in patients with APE and normal blood pressure.⁸ In the general population, CRBBB is

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associated with increased cardiovascular risk and all-cause mortality.⁹ Therefore, it is important to identify RBBB from cases with notched S wave in lead V₁. Genuine IRBBB may be present in the absence of structural heart disease. A triphasic rSr' and a narrow (between 90 and 110 ms) QRS complex in the absence of true IRBBB is eventually recorded particularly in lead V₁ when a precordial electrode is placed higher than or to the right of the standard position. In these cases the duration of the final r' wave is less than 20 ms.¹⁰ This pseudo IRBBB pattern is frequent in children, adolescents, and young adults (<40 years of age), asthenic habitus, underweight body mass index, and in subject with skeletal abnormalities with a decrease in the anteroposterior diameter of the chest (pectus excavatum and straight back syndrome).

In most subjects exhibiting this pattern, the right bundle branch does not appear to be involved in any pathological processes. When the QRS duration is normal, the rSr' pattern in the right precordial leads is most often normal, and the terminal deflection represents normal terminal depolarization of the crista supraventricularis, proximal septum, and base of the heart.¹¹

We retrospectively searched for ECGs from subjects with a notched S wave in lead V₁, in whom leads V₃R to V₅R had been recorded. The aim of this study is to present the findings in leads V₃R to V₅R in these subjects.

METHODS

From January 2009 to February 2013, 15 subjects (9 men) from the Shiyuan Taihe Hospital were included in the study. Their ECG showed a notched S wave in lead V₁ and leads V₃R to V₅R were recorded.

We used the following criteria for CRBBB:¹²

- (1) QRS duration ≥ 120 ms.
- (2) Right precordial leads (V₃R, V₁, V₂) with rsr', rSR', or rsR' patterns and occasionally a wide and notched R wave.
- (3) Prolonged ventricular activation time "R peak time" in V₁ ≥ 50 ms and normal in V₅-V₆.
- (4) QR or Qr pattern in lead aVR with wide final R wave followed by a negative T wave.
- (5) Widened final S waves (≥ 40 ms) in the leads exploring the left ventricle: I, aVL, V₅, and V₆.

- (6) Ventricular repolarization (ST/T) with opposite direction to the terminal deflection of the QRS complex, consequently, a negative T wave with ST depression may be seen in V₁ and sometimes in V₂.

IRBBB is a somewhat ambiguous term due to the wide variation observed in QRS configuration and duration as well as the numerous clinical conditions. In the present manuscript we considered IRBBB criteria to be fulfilled when QRS duration was between 100 and 110 ms (<120 ms) in this adult and senior's series, because when the QRS duration is ≤ 100 ms, the IRBBB pattern is difficult to distinguish from that of normal variant. The morphological criteria were the same as for CRBBB. Additionally, we included patients with these previous characteristics and rS pattern in lead V₁, when there was a notched S wave and the QRS duration was ≥ 100 ms and <120 ms.

Exclusion Criteria

We considered as exclusion criteria all the clinical-biotypological and electrocardiographic characteristics with potential to simulate IRBBB or CRBBB.

(1) Clinical biotype

- i Children, adolescents, and young adults (<40 years of age).
- ii Asthenic habitus.
- iii Body mass index < 18.5 kg/m².
- iv Subjects with skeletal abnormalities with decrease in the anteroposterior diameter of the chest (pectus excavatum and straight back syndrome).

[2] Electrocardiographic

- i QRS duration < 100 ms;
- ii Presence of Tapia and Proudfit criteria:¹³ amplitude of initial r < 0.8 mV, amplitude of r' < 0.06 mV, R/S ratio < 1.0, because the presence of these criteria suggest normality in conduction (normal variant).

RESULTS

The ECGs of subjects 1-10 and 12-15 are presented in Figures 1 and 2, and subject 11 is

Figure 1. Standard 12 leads electrocardiograms of subjects 1–10 and 12–15 with notched S wave in lead V₁.

separately presented in Figure 3. Demographic and clinical data of the subjects included are listed in Table 1. In none of the subjects 1–10 and 12–15, criteria for CRBBB or IRBBB were fulfilled in leads V₁ or V₂. The criteria for triphasic rsr', rSR', or rsR' patterns in V₁-V₂ for CRBBB were fulfilled in leads V₃R to V₅R, but not in the standard 12 leads, in subjects 1 and 10. Our criteria for IRBBB were fulfilled in all of the V₃R to V₅R leads in subjects 4, 7, and 15, in leads V₄R to V₅R in subjects 5, 8, and 13, in lead V₃R in subject 3 (the only right chest lead recorded) and in lead V₅R in subjects 2, 12, and 14. In subjects 6 and 9, also the V₃R to V₅R leads showed notched S wave, but not CRBBB or IRBBB. In subject 1 there was accompanying left anterior

fascicular block (LAFB). Subjects 3, 4, and 5 had an S wave in lead I, a Q wave and inverted T wave in lead III as a consequence of right ventricular strain causing S1Q3T3-pattern according to the McGinn-White criteria.¹⁴

Subject 11 was a 78-year-old male with coronary artery disease, hypertension, and chronic heart failure. The ECG showed a notched S wave in lead V₁, while leads V₃R to V₅R revealed IRBBB (QRS duration 100 ms) in beats 1 and 3, while IRBBB (QRS duration 110 ms) was evident in leads V₁ and V₃R to V₅R in beats of 2 and 4 (Fig. 2A). In the same ECG recording (Fig. 2B), IRBBB (QRS duration 110 ms) was evident in lead V₁ in beats 1, 2, and 4, while beat 3 shows a wider QRS duration (130 ms)

Figure 2. Lead V₁ and accessory right precordial leads (V₃R to V₅R) of subjects 1–10 and 12–15 with notched S wave in lead V₁.

after an atrial premature beat indicating CRBBB, possibly related to aberrant conduction.

DISCUSSION

In this small series of subjects with notched S wave in lead V₁, a terminal R' wave in the

right precordial accessory leads was a frequent finding, suggesting the possibility of hidden RBBB (incomplete or complete).

Camerini and Davies¹⁰ studied 50 subjects with an age range of 10–75 years without symptoms of cardiovascular or pulmonary disease. They found secondary R waves in 84% of the subjects in one or more of the V₃R to V₅R leads. Accordingly, they considered secondary R waves as a normal finding in accessory right chest leads when the QRS duration was less than 100 ms. In our adult (and senior) series, only 3 out of 15 had no known cardiovascular disease or APE.

The left anterior superior fascicle is adjacent to the right bundle branch. LAFB and RBBB thus commonly coexist. Newly acquired RBBB with concomitant LAFB in patients with acute myocardial infarction are frequently caused by occlusion of left main coronary artery or proximal left anterior descending artery before the first septal perforator branch and may portend a poor prognosis.^{5,7,15} Sometimes LAFB may partially conceal the diagnosis of RBBB causing absence of characteristic final S waves of RBBB in left leads I, aVL, and in some cases in V₅–V₆. The absence of final S waves in I and aVL and left precordial leads is called standard and precordial masquerading RBBB, respectively.^{16,17} This phenomenon occurs in the presence of concomitant high degree of LAFB associated with RBBB, left ventricular hypertrophy and/or left ventricular free wall LV focal block caused by myocardial fibrosis or necrosis. LAFB may eventually obscure the characteristic triphasic QRS pattern of RBBB in the right precordial leads; in such cases the RBBB is totally missed.¹⁶ In cases of intermittent LAFB, the dromotropic disturbance can conceal the pattern of RBBB in the right precordial leads. LAFB can not only obscure the diagnosis of RBBB, but may also conceal high voltage associated with left ventricular hypertrophy in the left precordial leads. Additionally, the secondary T-wave changes induced by LAFB in the inferior leads and from V₃ to V₆ reverse the deep negative T waves. Our subject 1 is an example of a notched S wave in lead V₁ in CRBBB. The so-called precordial “masquerading” RBBB is usually found in the context of high-degree LAFB with concomitant severe left ventricular enlargement and/or fibrotic block in the anterolateral wall of the left ventricle, and also signifies the presence of a severe underlying heart disease.¹⁷ However, the ECG findings in our subject 10 seems to indicate

Figure 3. Electrocardiograms of case 11 with notched S wave in lead V₁.

that the precordial “masquerading” RBBB can also be found in the general population and may present as notched S wave in lead V₁.

Subjects 3 to 6 and 14 were all diagnosed with APE. Possibly IRBBB was uncovered by recording leads V_{3R} to V_{5R} in subjects 3, 4, 5, and 14, while only notched S wave was found in subject 6. These findings seem to indicate that notched S wave in lead V₁ is a common phenomenon in APE, although there may be different underlying mechanisms.⁶ IRBBB revealed by leads V_{3R} to V_{5R} in subjects 3, 4, 5, and 14 associated with APE, probably results from right ventricular strain, because they had elevated right ventricular systolic pressure (see Table 1).

The normal terminal activation of the QRS complex in lead V₁ in RBBB is directed to the right and anteriorly. The QRS complex configuration

in RBBB may be influenced by the position of the ventricular septum, the size and possible structural disease in the ventricles, the position of the recording electrode, the insertion point of the right bundle branch, the degree of RBBB, etc. If the terminal portion of the QRS complex in RBBB is directed more posteriorly, RBBB may present a notched S wave in lead V₁, while the typical bundle branch pattern is evident from leads V_{3R} to V_{5R}.

According to Schamroth et al.,⁴ a diminution of the S-wave amplitude in lead V₂ reflects the early development of RBBB. Further progression leads to slurring or notching of the upstroke of the S wave in lead V₂, followed by the development of an R' deflection. With further progression, the R' deflection becomes increasingly taller until the advent of CRBBB, which is characterized by a very tall R' deflection. In another study by

Table 1. Demographic and Clinical Data of Included Subjects

Subject	Age (years)	Sex	Diagnosis	Abnormal Parameters on Echocardiography	Leads with IRBBB or CRBBB
1	68	M	Old anterior MI, CHF	LAD 35 mm, LVEDD 59 mm, anterior septal and apical hypokinesia, RVEDD 30 mm, RVSP 35 mmHg, LVEF 48%	V ₃ R, V ₄ R, V ₅ R
2	58	F	None	Normal heart structure	V ₅ R
3	56	F	APE	RVEDD 35 mm, RVSP 38 mmHg	V ₃ R
4	42	F	APE	RVEDD 38 mm, RVSP 40 mmHg	V ₃ R, V ₄ R, V ₅ R
5	58	F	APE	RVEDD 40 mm, RVSP 45 mmHg	V ₄ R, V ₅ R
6	54	M	APE	RVEDD 42 mm, RVSP 30 mmHg	None, notched S wave
7	63	M	HTN	LAD 32 mm, LVEDD 52 mm, RVSP 32 mmHg	V ₃ R, V ₄ R, V ₅ R
8	42	M	HTN, CRF	LAD 32 mm, LVEDD 52 mm, RVSP 32 mmHg	V ₄ R, V ₅ R
9	52	M	CAD, old MI	LAD 35 mm	None, notched S wave
10	46	M	None	Normal heart structure	V ₃ R, V ₄ R, V ₅ R
11	78	M	HTN, CAD, CHF	LAD 38 mm, LVEDD 58 mm, RVEDD 34 mm, RVSP 45 mmHg	V ₃ R, V ₄ R, V ₅ R
12	59	F	HTN	LAD 33 mm	V ₅ R
13	55	M	COPD	Normal heart structure	V ₄ R, V ₅ R
14	57	F	APE	RAD 44 mm, RVEDD 34 mm, RVSP 83 mmHg	V ₅ R
15	62	M	HTN	LAD 34 mm	V ₃ R, V ₄ R, V ₅ R

IRBBB = incomplete right bundle branch block; CRBBB = complete right bundle branch block; MI = myocardial infarction; APE = acute pulmonary embolism; CRF = chronic renal failure; CAD = coronary artery disease; CHF = chronic heart failure; COPD = chronic obstructive pulmonary disease; LAD = left atrial diameter; LVEDD = left ventricular end-diastolic diameter; RVEDD = right ventricular end-diastolic diameter; RVSP = right ventricular systolic pressure; LVEF = left ventricle ejection fraction; RAD = right atrial diameter; HTN = hypertension.

Smith and Ray,¹⁸ progression in the severity of pulmonary arterial obstruction in patients with APE is closely correlated with the appearance of a notched S wave and R/S ratio regression, followed by the RSR' pattern and, finally, by CRBBB in the right precordial leads. We found three different measures of QRS duration indicating IRBBB or CRBBB in one of our subjects (#11). With the increase of QRS duration, the QRS morphology in lead V₁ changed: first a notched S wave (QRS 100 ms), second an rSR' pattern (QRS 110 ms) indicating IRBBB and finally a taller R' wave (QRS 130 ms) indicating CRBBB. Our findings indicate that a notched S wave associated with narrow QRS duration (<120 ms) in lead V₁ may be an early sign of future CRBBB.

It should be pointed out that the so-called IRBBB based on QRS duration less than 120 ms and abnormal final R' wave in the precordial accessory leads with rightward orientation is speculative,

mainly because RBBB represents pathological changes in the right bundle branch, not only morphological changes in ECG.

LIMITATIONS

The following are limitations of our study: the retrospective nature of the collection of the data, the small number of patients included in the sample, and the inexistence of electrophysiological, histopathological, and vectorcardiographic confirmation of final conduction delay in the basal right ventricular territory.

CONCLUSIONS

The presence of a notched S wave in lead V₁ associated with a final R' wave in the right precordial accessory leads suggests the possibility

of hidden RBBB (incomplete or complete). Our results suggests that a notched S wave in lead V₁ is an indication to record accessory right chest leads (V3R to V5R) as they may provide clinically important information. Further studies are needed to verify the results.

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