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Sudden unexpected improvement in the atrioventricular conduction. What is the mechanism?

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ABSTRACT

The 12-lead electrocardiogram (ECG) of a 79-year-old male patient with recurrent pre-syncope showed irregular sinus rhythm with constant PR interval and left bundle branch block (LBBB) with intermittently blocked P waves. The beat following the blocked P wave had a narrower QRS with a shorter PR interval. The phenomenon of bilateral bundle branch block explains the sudden improvement in the atrioventricular conduction.

1. Case

A 79-year-old male patient with no comorbidities presented with a history of recurrent pre-syncope for the last 2 weeks. The clinical examination revealed irregular pulse with intermittent pauses and the echocardiography study was normal. His 12-lead electrocardiogram (ECG) at presentation (Fig. 1) showed repeated sequences of sinus rhythm with irregular PP intervals, constant PR interval with left bundle branch block (LBBB) and blocked P waves followed by sudden shortening of PR interval with incomplete right bundle branch block (RBBB) in the subsequent beat. What are the likely mechanisms of these findings?

2. Discussion

The ECG in Fig. 1 shows irregular PP intervals likely due to sinus arrhythmia along with 6 to 5 atrioventricular (AV) conduction sequences indicating 2nd degree AV block. Although, the P wave morphology in the only available rhythm strip V1 looks identical, the possibility of ectopic atrial impulses causing irregularity in PP interval cannot be excluded. The 2nd to 5th P waves of the sequence conduct with a constant PR interval of 170 msec (digitally calculated and approximated) with LBBB QRS morphology and the 6th P wave is slightly premature and fails to conduct suggesting a diagnosis of 2nd degree Mobitz type 2 AV block. However, contrary to what one would expect in this type of AV block, the P wave following the blocked P wave (i.e., 1st P wave of the sequence) conducts with shortened PR interval of 140 msec and incomplete RBBB QRS morphology.

The LBBB pattern can occur due to either complete failure of conduction in the left bundle branch or relative delay in conduction in the left bundle compared to that in the right bundle branch. Since the P wave following the blocked P wave conducts with incomplete RBBB, both the bundle branches are likely conducting with left bundle much slower than the right bundle branch in beats 2 to 5. Had it been fixed block in the left bundle branch with Mobitz 2 block in the right bundle branch, the 1st P wave should have conducted with the same PR interval and LBBB morphology. We can think of three possibilities to explain this interesting ECG.

In the first possibility, there is LBBB with Mobitz type 2 AV block. The pause due to blocked P wave results in an escape beat arising from the left bundle branch distal to the site of delay or block in the left bundle with preserved retrograde conduction towards the right bundle branch that causes partial normalization of QRS complex

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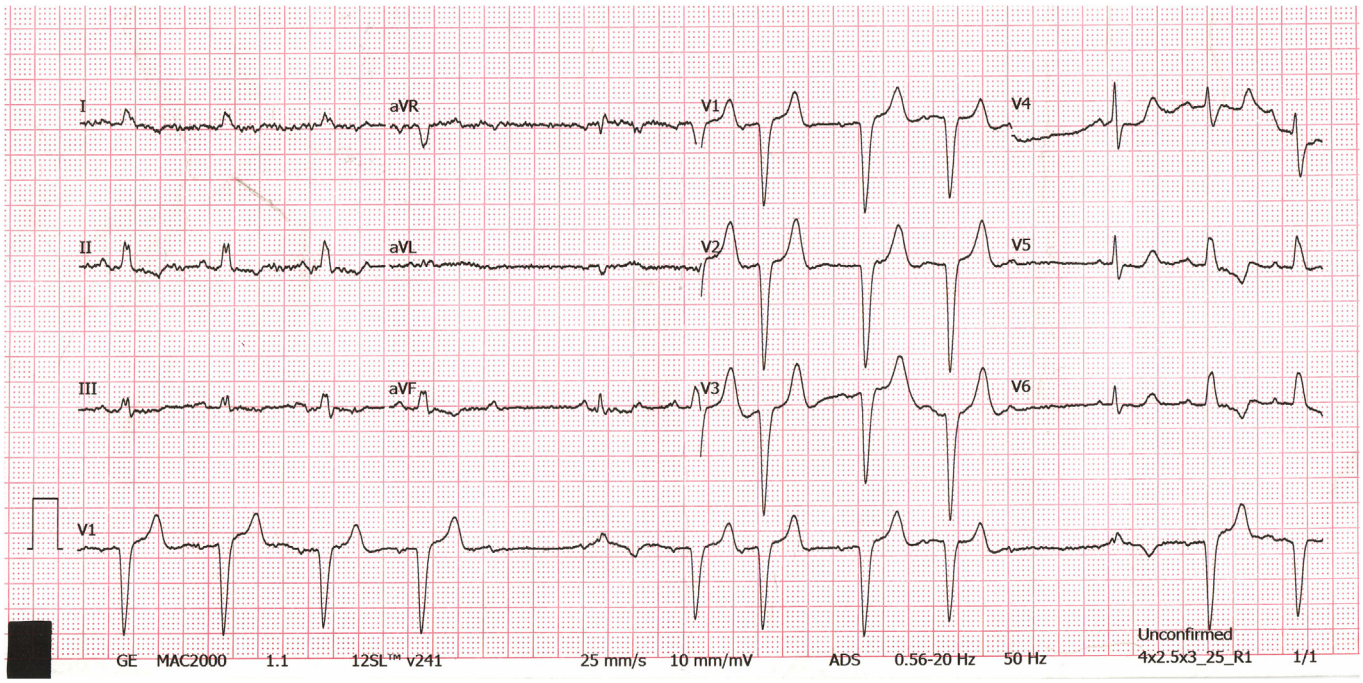

Fig. 1. Twelve-lead electrocardiogram showing 2nd degree atrioventricular block. Sinus arrhythmia, P waves conducting with left bundle branch block and a constant PR interval of 170 msec, sudden blocked P waves and first P wave of sequence conducting with PR interval of 140 msec and incomplete right bundle branch block QRS morphology.

and incomplete RBBB pattern. The escape beat occurs after the P wave but before the latter could conduct down the His-Purkinje system resulting in AV dissociation and apparently shorter PR interval (Fig. 2).

The second possibility can be explained by disease of conduction in both the bundle branches – first degree & Mobitz type 2 block in the right bundle branch and Mobitz type 1 block in the left bundle branch synchronized to the P wave so that both bundle branches block in the 6th P wave. Hence, in 2nd to 5th P waves, the right bundle branch conducts with a fixed delay but faster than the left bundle branch resulting in LBBB pattern. The delay in conduction in the left bundle branch progressively increases from the 2nd to 5th P wave and completely blocks in the 6th P wave. The right bundle branch on the other hand blocks suddenly in the 6th P wave with a resultant total failure of AV conduction. The left bundle branch conduction time is the best after the first P wave (that follows the pause) and even better than the right bundle branch conduction resulting in the RBBB pattern (Fig. 3).

A third plausible explanation is acceleration dependent or phase 3 block because the P wave associated with the block has a shorter preceding coupling interval (730 msec) and the phenomenon is seen again in the second part of the tracing [1]. However, very small degree of prematurity and RBBB in the beat following the pause makes this unlikely.

The pause due to the blocked P wave may suggest bradycardia dependent or phase 4 block in the right bundle branch and this can also explain incomplete RBBB in the next conducted P wave [1,2]. However, the postulated infra-hisian site of block in the tracing

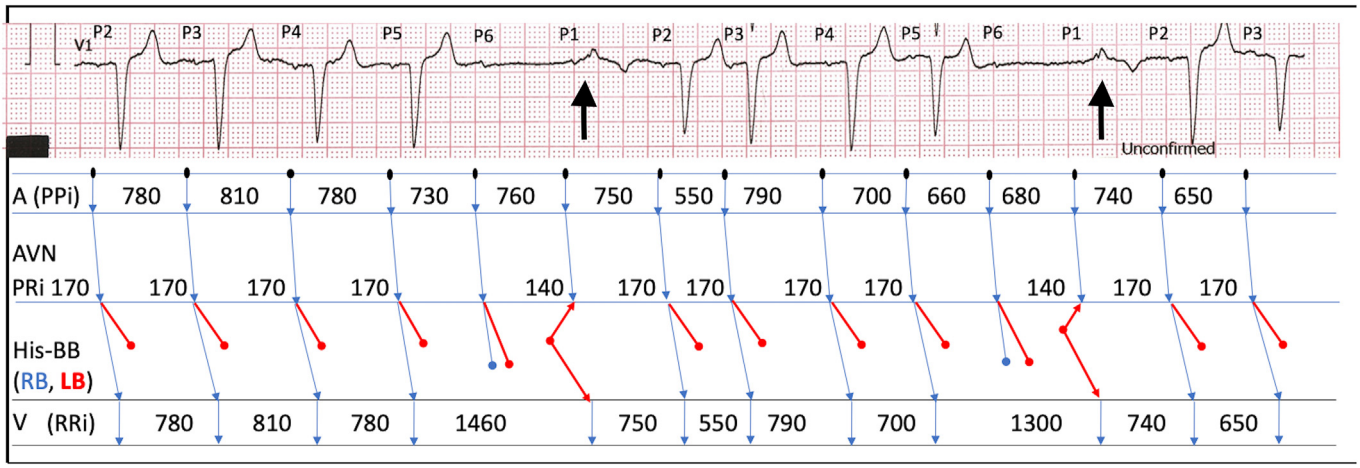

Fig. 2. Ladder diagram explaining the first possibility. The beat after the blocked P wave is an escape beat arising in the proximal left bundle branch block and is dissociated with the preceding P wave. A is atria; AVN is atrioventricular node; His-BB is His bundle and bundle branches; RB is right bundle branch (thin lines); LB is left bundle branch (thick lines) and V is ventricles. PPi (PP interval), PRi (PR interval), RRi (RR interval). Vertical arrows demonstrate beat with incomplete RBBB morphology.

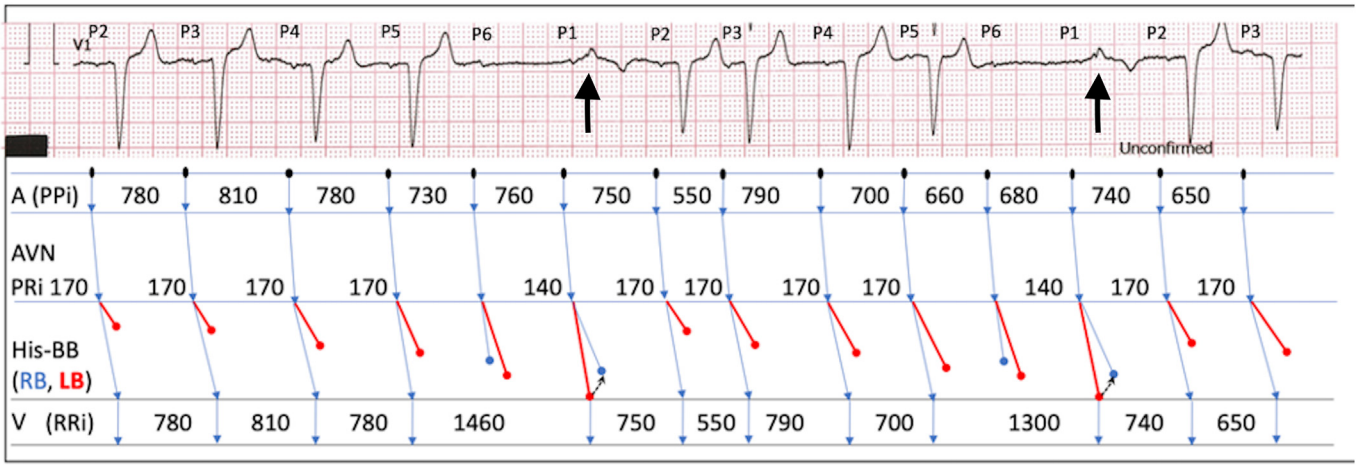

Fig. 3. Ladder diagram explaining the second possibility. Note that the left bundle branch shows a 2nd degree type 1 conduction pattern and the right bundle branch a 2nd degree type 2 conduction pattern both synchronized to P waves such that both block in the 6th P wave. Abbreviations as in Fig. 2. Vertical arrows demonstrate beat with incomplete RBBB morphology.

makes this a very unlikely possibility.

A Holter study may be of help in differentiating between the above discussed plausible explanations. If we can document RBBB morphology with a fixed PR interval instead of a variable PR in the beat following the blocked P wave at a different P wave cycle length, it will favour the second possibility. Similarly, an electrophysiological study can help in differentiating between the two by the HV interval and His bundle activation sequence (proximal to distal or distal to proximal) of the beat after the blocked P wave.

The electrocardiogram of the patient discloses the typical features of a bilateral bundle branch block (BBBB) [3]. **BBBB** was first described by Rosenbaum and Lepeschkin in the 1950s [4]. It was initially used for alternating bundle branch block with patterns of LBBB and RBBB in the same patient, which appears alternately or intermittently; and is associated with a high risk of complete heart block and syncope [3,4]. Antiperovitch P et al. discussed the importance of Lewis ladder diagrams in understanding complex electrocardiograms that is quite relevant in the present case [5]. Other explanations of a sudden improvement in the conduction such as supernormal conduction, peeling back of refractoriness, and gap phenomenon are unlikely in this case [6,7]. Acceleration dependent or phase 3 block can explain repetitive block of premature P waves. In the presence of a diseased His-Purkinje system, as in our case, the block can occur even with premature beats with a small decrease in coupling interval. The site of AV block can be known after electrophysiology study; however, despite the presence of baseline bundle branch block suggesting that block occurred in contralateral bundle branch, His recording studies performed on such patients have often demonstrated disease in the His, often with a split His [4]. Intra-Hisian block can be missed during electrophysiology study unless the His bundle recording is done carefully. Longitudinal dissociation in the His bundle can explain intrahisian site of block in patients with bundle branch block wherein the His bundle fibers destined to become left or right bundle can be diseased at the level of the His bundle. [8] This case underscores the significance of ladder diagrams in understanding difficult ECGs and complex AV conduction patterns, a method that is almost forgotten in the current era dominated by interventional electrophysiology.

Conflicts of interest

None.

Funding

None.

Declaration of competing interest

Nil.

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