QRS normalization during atrial pacing in a patient with complete left bundle branch block: What is your diagnosis?

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Introduction
Left bundle branch block (LBBB) is frequently observed following percutaneous aortic valve implantation. We describe an unusual case to get insight into the mechanism of the LBBB.

Case presentation
A 79-year-old patient with a medical history of hypertension, diabetes, ischemic heart disease, and severe aortic stenosis underwent transaortic valve implantation (TAVI) of an Edwards SAPIEN S3 26 mm valve. The tracing in Figure 1 was recorded during electrophysiologic study performed 5 days after TAVI owing to the occurrence of a new left bundle branch block (LBBB) with PR prolongation from 0.18 s to 0.24 s. During atrial pacing (150/min) the QRS normalized and returned to the baseline LBBB after discontinuation of pacing. What is the mechanism of the QRS normalization during atrial pacing?

Discussion
There are 4 possible explanations for this tracing. Theoretically, the “LBBB” complexes present on the first 3 beats could represent pre-excited sinus beats conducting over a right atriofascicular accessory pathway (so-called Mahaim fiber). In this case the normalized QRS complexes during atrial pacing would reflect exclusive conduction over the atrioventricular node owing to a prolonged refractory period of the accessory pathway. This speculation is easily ruled out by the finding of a prolonged HV interval preceding the LBBB complex (Figure 2).

A second possible explanation would be that the narrow QRS complexes resulted from a bilateral bundle branch block (BBB) of the same degree over the right and left
bundle branch. Such a hypothesis would require an HV interval preceding the narrow QRS greater than the one preceding the LBBB complex. This hypothesis can also be ruled out owing to the fact that the HV interval was identical in the presence of both LBBB complex and narrow QRS during atrial pacing (Figure 2).

A third possibility is supernormal conduction of the left bundle branch (LBB). A supernormal phase of conduction has been defined as a "condition under which conduction improves and occurs in relation to a supernormal phase of excitability in a sick tissue." Supernormal conduction was described in cases of intermittent BBB. The patient’s bundle recording, along with programmed atrial stimulation with a full scan of the diastolic interval, is an essential requirement for the recognition of a supernormal phase of conduction in order to exclude other confusing explanations. Normalization of conduction is preceded and followed by impaired conduction in the bundle branch, an essential requirement for the recognition of a supernormal phase of conduction in order to exclude other confusing explanations. Normalization of conduction is preceded and followed by impaired conduction in the bundle branch, indicating that the supernormal phase is immersed in the recovery curve of the affected fascicle without changes in His-Purkinje conduction times (H-V). Elizari and colleagues analyzed 20 cases of supernormal conduction in the setting of tachycardia-dependent block (phase 3 BBB). They found that the duration of the refractory period ranged between 600 and 1020 ms. The supernormal phase followed the end of the T wave by an interval ranging from 0 to 185 ms and was a rate-dependent phenomenon, being shifted to the left at fast rates and to the right at slow rates. The duration of the supernormal phase extended from 10 to 90 ms and its position in the cardiac cycle was relatively constant.

In order to test this possibility, we performed single atrial extrastimulation during sinus rhythm at cycle lengths ranging from 815 to 860 ms (Figure 3). We also assessed the effects of very late coupled atrial premature beats. Narrow QRS complexes were observed for H-H1 intervals ranging from 760 to 474 ms (ie, during a period lasting 286 ms).

However, atrial extrastimulation was associated with LBBB morphology for H-H1 < 440 ms. The long duration of H-H1 value (286 ms) associated with narrow QRS complexes is unlikely to represent the duration of the “supernormal phase of conduction,” since such period has been reported to occur only during a short time period ranging from 10 to 90 ms. We should recognize, however, that the conduction system in our patient is not in a steady state and that previous studies on supernormal activity may or may not apply to this case, making it possible that the supernormal period may be much longer.

The last possible explanation for our tracing is that the baseline LBBB itself was due to bradycardia-dependent (phase 4) BBB while the narrow QRS occurring during atrial pacing merely represented the phase of normal conduction over the LBB system. Therefore the reappearance of LBBB for the H-H1 interval of 440 ms represented the phase 3 block in the LBB conduction system. Simultaneous occurrence of phase 3 and phase 4 rate-dependent BBB after slight or moderate mechanical injury has been described in the canine conduction system. The mechanism of injury in this experimental study is reminiscent of that produced during TAVI. In all the experiments, after a period of time, normal conduction resumed depending on the magnitude of the provoked injury. This was also observed in our case. Therefore, we believe that this last working hypothesis best suits to explain the findings in our case.

Because we considered that the degree of impairment of the conduction system in our patient was minimal, we decided not to implant a pacemaker at this stage and to carefully follow the patient. At last follow-up 4 weeks after the procedure, the patient’s electrocardiogram had returned to normal and was similar to that present before the TAVI.

Figure 2  Tracing showing the same prolonged HV interval preceding the left bundle branch block complex and the normal QRS complex.
procedure, confirming that the damage caused to the LBB system was relatively minor.

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References
1. Elizari MV, Schmidberg J, Atienza A, Paredes DV, Chiale PA. Clinical and experimental evidence of supernormal excitability and conduction. Curr Cardiol Rev 2014;10:202–221.
2. Elizari MV, Nau GJ, Levi RJ, Lazzari JO, Halpern MS, Rosenbaum MB. Experimental production of rate-dependent bundle branch block in the canine heart. Circ Res 1974;34:730–742.