An Unusual Cause of Intermittent Broad QRS Complexes

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Case Presentation
A 23-year-old asymptomatic male patient was admitted for invasive evaluation of pre-excitation on the surface ECG persisting during exercise testing. The electrophysiological study, performed under sedation with midazolam, revealed a midseptal accessory pathway (AP) with retrograde conduction and a 1:1 conduction during atrial pacing up to a cycle length of 240 ms. No tachycardia could be induced during pacing of the atrium and ventricle. We opted for ablation of the AP due to the malignant anterograde characteristics. Two seconds after the delivery of the first application of radiofrequency energy, the delta wave disappeared and normal atrioventricular conduction was restored. Energy application was continued for an additional 58 seconds. One day after the procedure, a control ECG revealed recurrence of the pre-excitation pattern. A second invasive evaluation was, therefore, scheduled 1 week after the initial electrophysiological study. Surprisingly, the surface ECG was normalized at that time, with AH and HV intervals within normal limits during sinus rhythm. A decapolar catheter was placed in the high right atrium in order to avoid any mechanical block of the AP. During atrial pacing at a pacing cycle length of 500 ms, no pre-excitation was noticed. Second, atrial stimulation was performed with the decapolar catheter in the cavotricuspidalis isthmus to minimize the distance between the stimulation site and the atrial insertion of the previously ablated AP. Figure 1 shows that during pacing with a cycle length of 400 ms a regular irregular pattern appeared with clusters of 5 QRS complexes. The first complex shows an incomplete RBBB; the third and fourth complexes have a complete RBBB pattern. The fifth QRS complex is different resembling the QRS during sinus rhythm before the ablation attempt. The second complex has an intermediate morphology between QRS complexes 3 and 5. The RBBB configuration was rate-dependent, aberrancy being the mechanism. But how to explain the morphology of QRS 2 and 5? As shown in Figure 2, the regular irregular pattern and the QRS changes seen in Figure 1 can be explained.

Commentary
First, the decapolar catheter was placed in the high right atrium in order to avoid any mechanical block of the AP. During atrial pacing at a pacing cycle length of 500 ms, no pre-excitation was noticed. Second, atrial stimulation was performed with the decapolar catheter in the cavotricuspidalis isthmus to minimize the distance between the stimulation site and the atrial insertion of the previously ablated AP. Figure 1 shows that during pacing with a cycle length of 400 ms a regular irregular pattern appeared with clusters of 5 QRS complexes. The first complex shows an incomplete RBBB; the third and fourth complexes have a complete RBBB pattern. The fifth QRS complex is different resembling the QRS during sinus rhythm before the ablation attempt. The second complex has an intermediate morphology between QRS complexes 3 and 5. The RBBB configuration was rate-dependent, aberrancy being the mechanism. But how to explain the morphology of QRS 2 and 5? As shown in Figure 2, the regular irregular pattern and the QRS changes seen in Figure 1 can be explained.

Figure 1. Twelve-lead ECG showing a regular irregular pattern of different broad QRS complexes.
by an interplay of decremental conduction over both the atrioventricular (AV) node and the AP. The first paced atrial complex is conducted through the normal AV conduction system with incomplete RBBB. Complete RBBB is present following the third and the fourth atrial complexes. The second atrial complex is showing prolonged conduction through both the AV node and the AP. From the first to the fourth complex, there is a prolongation of the AV conduction time. The fifth QRS complex is fully pre-excited after a long AV interval and shows a negative HV interval (−10 ms). The sixth atrial complex is blocked both in the AV node and in the AP. Apparently, both the normal AV conduction system and the AP showed decremental properties after radiofrequency ablation. One could argue that there is a 3:1 conduction pattern via the AP, but the AV interval of beat 5 is shorter than the AV interval of beat 2 (Figure 2), suggesting decremental properties. This pattern is also seen in Figure 1. The phenomenon of temporarily decremental conduction in the AP has been described previously, interestingly in septally located APs. Possibly, edema after ablation could be the explanation. The close location of the AP to the normal conduction system may have been the reason for decremental conduction over the normal AV conduction system during atrial pacing with a cycle length of 400 ms. There was no evidence of dual AV nodal pathways before the first ablation because the refractory period of the AP was shorter than the refractory period of the AV node.

Disclosures
None.

References

KEY WORDS: ablation • accessory pathway • Wolff–Parkinson–White syndrome • midseptal accessory pathway
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