Overdrive Suppression of Antegrade Conduction over the Accessory Pathway

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SUMMARY

In a patient with Wolff-Parkinson-White syndrome whose accessory pathway was primarily capable of bidirectional conduction, antegrade conduction over the accessory pathway was transiently inhibited after rapid atrial or ventricular pacing or after spontaneous termination of atrioventricular reentrant tachycardia. Pacing rate and duration of tachycardia were related to the duration of the suppression of preexcitation, while the coupling interval of the first sinus beat to the last driven or tachycardia beat was irrelevant to the phenomenon. Thus, overdrive suppression of conduction may be the most likely mechanism of this phenomenon. (Jpn Heart J 2000; 41: 767-772)

Key words: Overdrive suppression of conduction, Wolff-Parkinson-White syndrome, Accessory pathway

The mechanism of intermittent antegrade conduction over the accessory pathway is a long-standing question. Although there are a few plausible interpretations such as linking and the gap phenomenon, none of them can singly explain all aspects of the phenomenon. Post-pacing conduction block has been reported in the atrioventricular node, bundle branch, and His-Purkinje system, and has been called a fatigue phenomenon or overdrive suppression of conduction.1-5) Similar findings were reported in accessory pathways as well.6-8) However, in earlier studies, overdrive suppression of preexcitation was observed in the accessory pathway lacking retrograde conduction6) or that affected by pharmacological treatment.7,8) In this article, we report a case of transient post-pacing or post-tachycardia disappearance of antegrade conduction over the accessory pathway which was primarily capable of bidirectional conduction. Overdrive suppression of conduction over the accessory pathway occurred in the absence of pharmacological treatment in our case, and may offer an insight into a peculiar electrophys-
CASE REPORT

A 33-year-old man was referred for radiofrequency catheter ablation of medically intractable paroxysmal supraventricular tachycardia. In the electrophysiology laboratory, preexcitation was constantly observed in the baseline state. Programmed stimulation induced orthodromic atrioventricular reentrant tachycardia (AVRT), with the accessory pathway located on the right lateral side being the retrograde limb of AVRT. Tachycardia cycle length was about 350 ms. The antegrade and retrograde effective refractory periods (ERP) of the accessory pathway were 280 ms and 240 ms at a basic cycle length (BCL) of 600 ms, respectively. Conduction of the accessory pathway was observed consistently during right atrial or right ventricular overdrive pacing at cycle lengths (CLs) of 300, 333, 400, 500 and 600 ms. Premature extra stimulations did not eliminate preexcitation until ERPs either (atrial BCLs: 300, 400, 600 and 700 ms, ventricular BCL: 600 ms).

When the atrial or ventricular overdrive pacing ended, however, preexcitation transiently disappeared (Figure 1A, B). Only atrial overdrive pacing at a BCL of 600 ms failed to show this phenomenon (Figure 1B). Preexcitation was also temporarily abolished after spontaneous termination of AVRT (Figure 1C). Although preexcited QRS sometimes interposed between non-preexcited QRS waves, the first post-pacing or post-tachycardia beat invariably showed non-preexcited QRS. Moreover the same phenomena were observed at atrial and ventricular premature extra stimulations. To assess whether the pacing or tachycardia cycle length is relevant to the subsequent disappearance of preexcitation, we measured the period from the last QRS of the overdrive pacing or tachycardia to the last non-preexcited QRS as the duration of post-pacing or post-tachycardia modulation of preexcitation. As shown in Figure 2A, the pacing cycle length was inversely related to the duration of the pacing-induced suppression of preexcitation, suggesting the phenomenon is rate-dependent. Also, suppression of preexcitation by right ventricular pacing was more prolonged than that by right atrial pacing, if consideration is given to the pacing cycle length, suggesting the phenomenon is direction-dependent. In Figure 2B, the period of tachycardia-induced suppression of preexcitation is plotted against the duration of the preceding AVRT. There was an apparent relationship between the two variables, and time-dependency of the phenomenon was indicated. We also compared the post-pacing or post-
Figure 1. Disappearance of preexcitation. Surface ECG leads I and V1 and the high right atrial (HRA) are shown. A: Preexcitation alternately disappeared after the end of 15-second overdrive pacing from the right ventricular apex at a BCL of 428 ms. B: After 30-second atrial pacing at a BCL of 600 ms, preexcitation remained visible (upper panel), while atrial pacing at a BCL of 500 ms or shorter caused post-pacing disappearance of preexcitation (lower panel). C: Preexcitation disappeared after spontaneous termination of AVRT. AVRT lasted for 38 seconds. QRS waves with preexcitation interposed between non-preexcited beats.
Figure 2. A: Relationship between the cycle length and the duration of disappearance of preexcitation. HRA = High right atrial overdrive pacing; RV = Right ventricular overdrive pacing. B: Relationship between the duration of AVRT and the duration of disappearance of preexcitation.

Figure 3. The post-pacing or post-tachycardia coupling intervals between preexcited and non-preexcited QRS beats indicate that the phenomenon was unrelated to these intervals.
tachycardia coupling intervals between preexcited and non-preexcited QRS beats, finding only that the phenomenon was entirely independent of these intervals (coupling intervals of preexcited QRS: 260-1200 ms, non-preexcited QRS: 270-1380 ms. Figure 3).

**DISCUSSION**

In earlier reports, overdrive suppression of conduction over the accessory pathway was associated with treatment by disopyramide or amiodarone. Otherwise, this phenomenon was seen in the accessory pathway capable of solely antegrade conduction. Accordingly, it is believed that pharmacological or pathologic modification of the conduction property is essential for the occurrence of post-pacing or post-tachycardia disappearance of preexcitation. In our case, however, transient inhibition of conduction over the accessory pathway was found in the absence of pharmacological treatment. To our knowledge, this is the first report of "overdrive suppression" of antegrade conduction over the accessory pathway primarily capable of bidirectional conduction.

Besides the mechanism of overdrive suppression of conduction, there are a few other explanations for the present phenomenon, such as phase-dependent block (phase three and phase four block), linking, and the gap phenomenon. However, in our case, preexcitation disappeared without preference for a certain range of the coupling interval. Also, the atrial interval prior to linking should be considerably shorter (70-290 ms) than observed in our case. These findings appear to support the mechanism of overdrive suppression, and are rather inconsistent with other explanations.

The details of the electrophysiologic basis for overdrive suppression of conduction are not known. This phenomenon was rate, time and direction dependent. The same dependencies were demonstrated in the atrioventricular node, bundle branch and His-Purkinje system. Faster pacing rates or longer durations of pacing in conduction properties increases cytosolic sodium and calcium concentrations as well as the extracellular potassium level, and subsequently mitigates cellular excitability. The mechanism responsible for direction-dependency is not clear but is probably related to the geometry of the conduction properties and the antegrade versus the retrograde activation sequence. Because neither tachycardia nor rapid pacing generally yield this phenomenon, some unknown concomitant factor must have participated in creating a particular electrophysiologic property of the accessory pathway in the present case. If such a factor functions at a slower heart rate, the mechanism of overdrive sup-
pression of conduction may explain intermittent preexcitation in some patients.

REFERENCES

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