

## Cryoablation-Induced Ventricular Preexcitation

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A 14-year-old girl was referred to our institution for a third attempt at catheter ablation of an anteroseptal accessory pathway. A concealed accessory pathway was confirmed, with inducible orthodromic atrioventricular (AV) reciprocating tachycardia. Earliest retrograde atrial activation was identified by contact mapping (EnSite NavX, Endocardial solutions, St. Paul, MN, USA) to guide cryoablation (Freezor<sup>®</sup> Xtra, Medtronic CryoCath LP, Montreal, Canada). Cryomapping was initially performed in sinus rhythm to monitor antegrade AV nodal conduction. Once the temperature reached  $-30^{\circ}\text{C}$ , ventricular preexcitation appeared (Fig. 1A). Preexcitation abated upon rewarming (Fig. 1B) and was reproducibly provoked by subsequent cryo-applications. There was no evidence of AV nodal damage (Fig. 2).

**Figure 1.** Cryoablation-induced ventricular preexcitation. Shown are surface ECG leads I, II, and V1 and intracardiac electrograms recorded from the distal cryoablation catheter (MAP<sub>d</sub>). In Panel A, cryomapping is performed during sinus rhythm. Electrical noise during ice ball formation at the distal tip of the cryoablation catheter corresponds with the onset of ventricular preexcitation. In Panel B, preexcitation disappears upon interruption of cryoablation, with passive rewarming.



**Figure 2.** Atrial pacing during cryoablation. Shown are surface ECG leads I, II, and V1 and intracardiac electrograms recorded from the distal cryoablation catheter (MAP<sub>d</sub>), distal (HIS<sub>d</sub>) and proximal (HIS<sub>p</sub>) His catheter, and proximal coronary sinus catheter (CS<sub>p</sub>). During pacing from the proximal coronary sinus, the stimulus to H interval remained constant (172 ms) for all narrow QRS complexes preceding and following preexcited beats. The His signal appears buried in the ventricular electrogram of preexcited beats. Noise on the cryoablation catheter was induced by ice ball formation.

Potential mechanisms underlying this paradoxical cryoablation-induced preexcitation include concealed conduction, a “gap” phenomenon, and electrotonic interactions. The former may postulate that baseline antegrade accessory pathway conduction block resulted from concealed retrograde conduction. However, the short PR interval is inconsistent with cryoablation-induced “release” of concealment from an impulse arising from the very same AV node-conducted beat. Collision between antegrade accessory pathway and retrograde conduction from the preceding AV node-conducted beat would imply a long seemingly non-physiological retrograde conduction time, rendering this scenario unlikely. A “gap” mechanism may contend that proximal reversible cryothermal effects produced crit-

ical antegrade conduction delay permitting recovery of a more distal portion of the accessory pathway. At variance with this hypothesis, site of preexcitation induction was more ventricular than successful ablation. An electrotonic effect mechanism, perhaps the most likely culprit, may conjecture that antegrade accessory pathway conduction was initially dispersed among broad or multiple atrial inputs, with subthreshold depolarization of a portion essential for preexcitation. Loss of the electrotonic downstream effect during cryoablation of a non-crucial atrial input unmasked preexcitation. Electrotonic modulation resumed upon rewarming, thwarting action potential propagation across the excitable gap required for preexcitation.