Cooling of the atrioventricular node to unmask an accessory pathway

Per Insulander*, Hamid Bastani, and Mats Jensen-Urstad

Department of Cardiology, Karolinska Institutet, Karolinska University Hospital, S-141 86 Stockholm, Sweden

* Corresponding author. Tel: +46 8 585 80000; Fax: +46 8 585 86710. Email: per.insulander@karolinska.se

In a patient with pre-excitation and atrioventricular (AV) reentrant tachycardia due to an anteroseptal accessory pathway (AP), the ablation catheter impaired conduction through the AP making the pre-excitation disappear. Cryomapping of the AV node was performed, with slowing of AV nodal conduction and temporary recurrence of pre-excitation. The optimal site for ablation could easily be mapped and a successful ablation was performed.

Cryoablation is an alternative to radiofrequency (RF) ablation in focal substrates. We have reported the safety and efficacy of this technique with regard to the ablation of substrates in the vicinity of the atrioventricular (AV) node when treating atrioventricular nodal reentrant tachycardia as well as accessory pathways (APs) and ectopic foci in the mid-septal and the anteroseptal regions.1,2

Cryomapping may be used in substrates where an intentional cooling of the AV node might facilitate mapping and be beneficial for success.

The excellent safety profile of the cryoablation is accounted for by the reversibility of the cryothermal effect during cryomapping. This reversibility also makes it possible to terminate ablation without any permanent impairment if there is indication of injury to the electrical conduction system.

A 34-year-old male with intermittent pre-excitation and AV reentrant tachycardia was referred to us from another centre due to an anteroseptal AP where attempts using RF ablation had failed. Positioning the catheter over the AP impaired conduction through the AP and led to the disappearance of the pre-excitation after a few seconds [electrocardiogram (ECG)—Figure 1A]. An application was delivered but with immediate return of pre-excitation.

As an alternative approach, the catheter tip was withdrawn 5 mm to the presumed location of the AV node. Cryomapping was performed during 15 s, resulting in slowing of the AV nodal conduction and a temporary persistence of pre-excitation during (ECG—Figure 1B) and 20 s after cryomapping was stopped (ECG—Figure 1C). The optimal site for ablation could easily be mapped and a successful ablation was performed. Tachycardia was not induced. The patient has been arrhythmia-free during a 24-month follow-up, the pre-excitation has not reappeared, and AV nodal conduction has been normal.

Discussion

Radiofrequency ablation carries a risk of irreversible high-grade AV block in substrates adjacent to the AV node, while during cryoablation the risk is minimal. We recently published a series of more than 1300 cryoablation procedures of the substrates adjacent to the AV node,3 where we observed 158 cases of transient first-, second-, or third-degree AV block during cryomapping or cryoablation. During a 24-month follow-up no late AV block was found. Although cryoenergy probably was delivered directly to the AV node in a minority of these cases,
these findings together with the previous experimental data show that the risk for permanent damage during short cryomapping of the AV node is minimal.

The present case is an example of catheter pressure impairing the AP conduction making the mapping of ventricular pre-excitation difficult. Since transient AV block during cryomapping is a benign finding, we judged that temporarily impairing the conduction in the AV node by means of cryomapping is a safe procedure. Adenosine could have been used to block AV nodal conduction and unfold the delta wave; however, the effect of adenosine is rather short while the effect of temporary cooling of the AV node by cryomapping lasted 25 – 30 s in this case, providing with adequate time to map and find an optimal position for successful ablation. Repetitive dosages of adenosine, verapamil, vagal manoeuvres, and subthreshold His-bundle pacing could have been potential alternatives for adequate mapping of the AP.

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**References**