EP CASE REPORT

Mechanical tachycardia of the slow pathway induced by a percutaneous tricuspid valve device

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A 47-year-old woman with Ebstein's anomaly was referred to our department for management of narrow QRS tachycardia with short RP' intervals (*Figure 1A*, left). Her medical history included tricuspid valve plasty followed 10 years later by transcatheter tricuspid valve replacement. One year before presentation, she underwent percutaneous closure of a septal periprosthetic leak by an Amplatzer™ Duct Occluder™. She experienced palpitations a few weeks following implantation and the symptoms progressive until incessant tachycardia despite antiarrhythmic drug therapy. During electrophysiological study, no evidence of dual atrioventricular node (AVN) physiology was found and tachycardia initiation did not depend on AH-interval prolongation. A 1:1 atrioventricular relationship was found with concomitant atrial and ventricular activations (*Figure 1A*, right). Right atrial activation mapping showed a centrifugal activation pattern with an earliest inferoseptal activation site (*Figure 1B*, left, the yellow ball indicates the location of the His bundle), concordant with a scar area corresponding to the device (*Figure 1B*, middle, the blue tag indicates the position of the device). Fluoroscopic views showed the position of the Amplatzer™ in the septal right atrium (*Figure 1B*, right, black arrow). A SmartTouch™ ablation catheter was positioned at the site of earliest atrial activation, which coincided with the position of the atrial tip of the device. Radiofrequency ablation at this site successfully terminated the tachycardia, with further non-inducibility (Supplementary material online, *Video S1*).

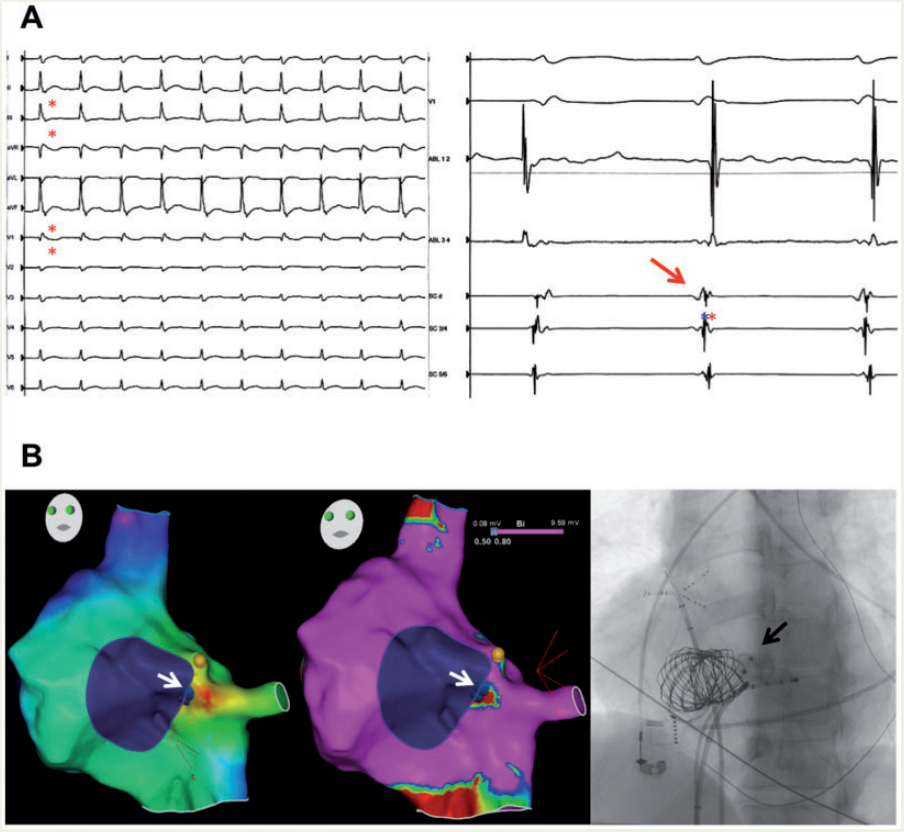

Figure 1 Focal junctional tachycardia arising from the atrioventricular node slow pathway region, corresponding to the position of the septal transtricuspid device.

This is an original report of a focal junctional arrhythmia caused by a mechanical irritation of the AVN slow pathway by a trans-tricuspid device. Conceptually, this junctional automaticity could be compared with the classical accelerated junctional rhythm provoked by radiofrequency applications at the slow pathway during ablation of AVN re-entrant tachycardia. In this setting, the mechanism of generating

junctional rhythm is unclear, but it has been suggested that heating the AVN region may result in electrical uncoupling of the different cellular subtypes and automatic firing.[1] Similarly, the structural and electrophysiological heterogeneity of the AVN region could explain its sensitivity to mechanical irritation.

One could assume that, with the development of transcatheter tricuspid valve replacement, this complication may not remain exceptional.

Supplementary material

Supplementary material is available at *Europace* online.

Conflict of interest: none declared.

Reference

1. Boyle NG, Anselme F, Monahan K, Papageorgiou P, Zardini M, Zebede J *et al*. Origin of Junctional Rhythm During Radiofrequency Ablation of Atrioventricular Nodal Reentrant Tachycardia in Patients Without Structural Heart Disease. *Am J Cardiol* 1997;**80**:575–80.