LETTERS TO THE EDITOR

doi:10.1093/europace/eus017
Published online 14 February 2012

A new method for induction of atrioventricular nodal reentrant tachycardia: do we really need it?

We have read with interest the article by Sharif et al.1 The authors tested the new method for induction of atrioventricular nodal reentrant tachycardia (AVNRT). The study group consisted of 10 patients with recurrent episodes of paroxysmal supraventricular tachycardia documented by electrocardiography, in whom tachycardia was not inducible during electrophysiologically testing even after isoproterenol infusion and atropine injection. However, dual atrioventricular (AV) node physiology was observed in all cases. The authors attempted to induce tachycardia by applying low-watt (15–20 W), low-temperature (40–45°C) radiofrequency (RF) currents into the slow pathway (SP) area for 40 s. The most likely pathophysiological background is that the warming of slow pathway increases conduction velocity facilitating the propagation of junctional ectopic beats or premature beats and induction of AVNRT. By applying this method, AVNRT was inducible in five patients (50%). Several issues arise while reading the article. Unfortunately, the authors did not provide more details about the actual induction pattern in five inducible cases. What were the exact stimulation protocols and were they used during or immediately after RF application? Also, the position of the ablation catheter during induction is not provided. Was it guided anatomically or was the operator looking for SP potentials? It is possible that the tachycardia would be inducible in five ‘non-inducible’ cases at different locations of the ablation catheter during diagnostic heating. It seems that there was only one therapeutic RF pulse applied in all cases without moving the ablation catheter from the area of low-temperature, low-energy induction. Unfortunately, the authors did not provide any information about the endpoint of ablation. Was it the elimination of anterograde SP conduction or perhaps non-inducibility of AVNRT by diagnostic RF pulse in five originally inducible cases? We would be grateful if the authors can provide this very important information. It is also worth mentioning that, according to current guidelines,2 the presence of dual AV node physiology in symptomatic patients without other identified causes of arrhythmia constitutes the indication for SP ablation (class I recommendation). Actually, the authors performed SP ablation in all cases regardless of the inducibility of AVNRT by their new method. Therefore, we believe that the clinical application of the induction method described would be limited and at least controversial.

Conflict of interest: none declared.

References

Jacek Pawel Majewski* and Jacek Lelakowski
Department of Electrocardiology, Jagiellonian University, Collegium Medicum, Institute of Cardiology, John Paul II Hospital, Kraków, Poland
*Corresponding author. Department of Electrocardiology, ul Pradnicka 80, 31-202 Kraków, Poland. Tel: +48 126142381; fax: +48 126335299; Email: jmajewski@interia.pl

Response to a letter from Dr Jacek Pawel Majewski and Dr Jacek Lelakowski

I thank Dr Jacek Pawel Majewski and Dr Jacek Lelakowski for their precise attention to our manuscript, entitled: ‘A new method for induction of atrioventricular nodal reentrant tachycardia in non-inducible cases’.1 In their letter to the editor, Dr Majewski and Dr Lelakowski state, ‘The presence of dual AV node physiology in symptomatic patients without other identified cause of arrhythmia constitutes the indication for slow pathway ablation (class I recommendation), therefore we believe that the clinical application of induction method described would be limited and at least controversial’.2

Based on Dr Majewski and Dr Lelakowski’s comment, there would be no need for further measures and stimulations to induce atrioventricular nodal reentrant tachycardia (AVNRT) (e.g. Isuprel infusion, Atropine injection, and other measures were explained in our paper), if we observed dual atrioventricular (AV) node physiology in a patient with clinical paroxysmal supraventricular tachycardia in the electrophysiology lab and only in the case of dual AV node physiology would we be allowed to perform slow pathway ablation. It seems that all electrophysiologists have general agreement on using all the necessary stimulations to induce AVNRT and slow pathway ablation only should be performed in non-inducible cases in the presence of dual AV node physiology as the last resort.

Consequently, the method which we introduced in our paper is intended to supplement the methods already available in the armamentarium for a safer slow pathway ablation.

We performed induction in all our 10 patients in the following way:

(1) Introduction of atrial or ventricular premature depolarization beats and double atrial stimuli.
(2) Continuous burst pacing or rapid incremental atrial pacing in the right atrium.
(3) If there was no success with the above pacing protocol, we utilized pharmacological agents (isoproterenol infusion and atropine injection) and repeated the aforementioned stimulation protocol.

In three cases, AVNRT was inducible during radio frequency (RF) currents application and the presence of junctional beats; and in two cases, no arrhythmia was inducible during low-watt, low-temperature RF current application but inducible after the above-mentioned stimulation protocol. The position of the ablation catheter during the induction of arrhythmia was at the slow pathway area (zone 5-6-7-8) anatomically with small A potential and large V potential.

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