Ablation or modification of slow pathway: the final word has still to be uttered

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Atrioventricular nodal reentrant tachycardia is the most frequent of paroxysmal supraventricular tachycardias. Several studies have reported the efficacy of radiofrequency catheter ablation in the treatment of this arrhythmia and have encouraged its use in clinical practice[1-3]. Understanding the mechanisms responsible for a disease and the development of techniques for its
control may result in optimal treatment, but may also raise new questions related to safety and appropriateness of indications[2]

Two large prospective studies[4,5] have shown that radiofrequency catheter ablation for atrioventricular nodal conduction can be performed with a high degree of success in patients with atrioventricular nodal re-entrant tachycardia. This arrhythmia, which may result in poorly tolerated symptoms and be either sporadic or incessant, has been treated in the past with a variety of drugs ranging from beta-blockers to antiarrhythmics such as flecainide or amiodarone. The knowledge that the circuit for this reentrant arrhythmia may be the anterior and posterior inputs to the atrioventricular node has guided antiarrhythmic therapy and been used to target fast or slow pathways for radiofrequency catheter ablation. Due to the greater risk of complete atrioventricular block[3,4] and the relatively smaller success rate associated with fast pathway ablation, the slow pathway approach is now the preferred procedure. As a result of the very high success rate (>90%) and the lower arrhythmia recurrence (<8%) observed in experienced centres, growing use of this procedure in less specialized centres has already started, with an increasing risk of complications[2]. Among them, complete atrioventricular block and myocardial damage in connection with the application of radiofrequency energy are of clinical importance.

In the present issue, Clague and co-workers report[9] the result of a study in which they compared immediate and short-term efficacy of slow pathway ablation and modification for atrioventricular nodal re-entrant tachycardia. The initial success rate and the incidence of complete heart block were, respectively, 97.0% and 0.8%, with a recurrence rate of 6.9%; these figures reflect the extensive experience of the authors. The retrospective nature of the study, however, prevents definitive conclusions to be drawn on the differences and advantages of the two approaches aimed at producing modification or ablation of slow pathways.

To avoid or to limit complete atrioventricular block, identification of the appropriate ablation site remains a critical issue[1,3,4], but the electrophysiological significance of slow pathway potentials arising from transitional atrioventricular node cells remains to be established. Nevertheless, in about 75% of patients, target sites for slow pathway ablation of atrioventricular nodal re-entrant tachycardia can be consistently found along the tricuspid annulus at the level of the coronary sinus ostium. Junctional ectopy during radiofrequency energy application is considered a sensitive but not specific marker for a successful ablation site. Moreover, monitoring of junctional ectopy for immediate detection of loss of ventriculo-atrial conduction and interruption of energy application is important to reduce the incidence of complete atrioventricular block[3].

In this regard, the possibility of maintaining some conduction along the slow pathway, as indicated by the presence of single atrioventricular nodal reentrant beats or of an A-H jump in patients in whom atrioventricular nodal re-entrant tachycardia was no longer inducible, opens new potential perspectives.

It must be recalled that in most of the published studies[4,5] the incidence of complete atrioventricular block requiring pacemaker therapy could be underestimated as a result of the relatively young age of the patients (average age, mean 40 years), the short duration of follow-up (a few years) and the physiological reduction in atrioventricular node function related to ageing. For all these reasons, modification rather than ablation of the slow pathway, if not associated with a greater incidence of recurrences[6-8], would be preferable by preserving, to some extent, a more physiological atrioventricular node function. Unfortunately, the retrospective nature of this study[9] does not allow a comparison of the inducibility and clinical outcome of patients in whom modification, instead of ablation, was performed. Moreover, it is unclear whether modification was an intermediate step toward ablation in patients with an unfavourable atrioventricular node anatomy, and more difficult mapping of this targeting was selected by the operator at the beginning of the ablation procedure. As a result, no information about a possible reduction in fluoroscopy time and number of radiofrequency pulses is available.

Two additional features of the study must be taken into consideration. First, there was a significant difference in mean total fluoroscopy time between the initial 100 and the subsequent 279 cases. Moreover, not only age but also the number of pulses and fluoroscopy time were found to be positively associated with both primary failure and atrioventricular nodal re-entrant tachycardia recurrence. There was, therefore, a learning curve effect that has to be taken into consideration, particularly in view of the fact that radiofrequency catheter ablation of atrioventricular nodal re-entrant tachycardia is a procedure that nowadays is considered relatively simple and is performed in less experienced centres where the number of patients referred for this arrhythmia is likely to be small.

A second point that must be considered is the reported incidence of recurrence of arrhythmias based on either documentation of tachycardia during Holter monitoring or presence of symptoms. Indeed,
the observed clinical recurrence in Clague’s study[8] was 12.4%, whereas 11.3% of patients were still on antiarrhythmic medication. The reason for maintaining antiarrhythmic therapy in some patients is unclear and deserves further evaluation. Another point that remains to be established is the possibility that for patients with rare episodes of atrioventricular nodal re-entrant tachycardia and good clinical tolerance a single dose of an antiarrhythmic drug might be the best treatment for determining rapid tachycardia termination and avoiding risk and costs related to radiofrequency catheter ablation.

In conclusion, this study provides additional support to the evidence that radiofrequency catheter ablation of atrioventricular nodal re-entrant tachycardia is an effective and safe procedure. Moreover, modification of the slow pathway seems to be associated with a similar success and complication rate as ablation. Additional evidence is, however, necessary in order to support the concept that preservation of some atrioventricular node function not only does not limit the efficacy of treatment but also reduces short- and long-term complications related to application of radiofrequency energy to the atrioventricular node.

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References


Smoking, nicotine and thrombotic risk — a role for platelet dependent thrombin generation?

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Smoking accelerates atherosclerosis, and is a well-known risk factor for acute cardiovascular complications, such as myocardial infarction. There are several haemostatic risk factors for cardiovascular disease, increased platelet activity being one of them[1], and the beneficial effects of antiplatelet therapy[2] support the notion that platelets are important in the genesis of myocardial infarction. Thrombin is a key enzyme in the coagulation cascade, and the generation of thrombin is a complex process which involves multiple positive and negative feed-back mechanisms. Thrombin is also a potent platelet agonist, and activated platelets may amplify thrombin generation. Thus, full-blown thrombin generation may be triggered by minute amounts of thrombin if flow is low (so that the thrombin formed is not washed away) or in a platelet aggregate. Activated platelets may release factor V, provide a procoagulant surface (via the ‘flip-flop’ reaction which exposes certain phospholipids on the platelet membrane), and form procoagulant platelet microparticles[3]. Thus, platelet-dependent thrombin generation may be an important component of the initial triggering of thrombus formation.

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