Atrial Tachycardia
How Important Is the Mechanism?

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Programmed electrical stimulation of the heart together with cardiac activation mapping have played an important role in the recognition and classification of the different types of supraventricular tachycardia. That knowledge is essential when cure of the arrhythmia is attempted by ablating the site of abnormal impulse formation or interrupting (as is the case in reentrant arrhythmias) a part of the tachycardia pathway.

Of the tachycardias involving supraventricular structures, either completely or partially (as the incorporation of an extra atrioventricular connection in the tachycardia circuit), arrhythmologists have been most interested in atrioventricular nodal tachycardia and tachycardias using accessory atrioventricular pathway(s). This is not surprising. The localization of tissue playing a crucial role in the occurrence of such a arrhythmia is usually not difficult for the experienced clinical electrophysiologist. Moreover, those patients usually have no cardiac abnormalities apart from their arrhythmia, and complete cure can be obtained by destroying an essential part of the tachycardia pathway.

More recently, because of a possible role for ablative therapy, interest has been growing in other types of supraventricular tachycardias such as atrial tachycardia, atrial flutter, and atrial fibrillation.

Atrial tachycardia is a relatively rare supraventricular tachycardia. Over a period of 17 years we examined 1834 patients with ECG documentation of a supraventricular tachycardia in the clinical electrophysiology laboratory in Maastricht. One hundred thirty (7%) patients suffered from atrial tachycardia. We defined atrial tachycardia as a regular tachycardia originating in the atrium, outside the sinus node or atrioventricular node. The electrographic characteristics of atrial tachycardia allowed differentiation from atrial flutter and atrial fibrillation. Atrial tachycardia may occur in all age groups and can be accompanied by structural heart disease.

It is important to know that atrial tachycardia may be of the paroxysmal or the incessant or persistent type. While the first type is characterized by a sudden onset and offset of the arrhythmia, in incessant atrial tachycardia the arrhythmia is present more than half of the day. Although already described long ago, only recently are most cardiologists becoming aware that incessant or frequently occurring supraventricular tachycardias, such as incessant atrial tachycardia, may result in a dilated cardiomyopathy, the so-called tachycardia cardiomyopathy. Of our patients with atrial tachycardia, 25% suffered from the incessant form and 40% presented with a dilated cardiomyopathy. The important point is that the left ventricular dysfunction in these patients is the consequence of the arrhythmia and is reversible after cure of the tachycardia by surgery or radiofrequency ablation.

Clinical electrophysiologists have been puzzled for many years by the mechanism of atrial tachycardia. Recently, Engelstein et al,9 and in this issue of Circulation Chen et al,10 discussed the value of pacing, pharmacological probes, and monophasic action potential (MAP) recordings to unravel the possible mechanisms of this arrhythmia. If atrial tachycardia is defined as a regular tachycardia having its origin in the atrium without involvement of the sinus or atrioventricular node in the tachycardia mechanism, what are their findings? Both authors found no relation between the site of origin in the atrium and the tachycardia mechanism. Engelstein et al suggest that adenosine may be helpful in differentiating reentrant from automatic atrial tachycardia. Although adenosine administration promptly terminated reentrant atrial tachycardia, the drug resulted only in transient slowing and suppression (for less than 20 seconds) of the arrhythmia. Pacing was used as the gold standard for making the distinction between a reentrant or automatic mechanism using reproducible initiation, termination, and entrainment of the tachycardia as arguments for a reentrant mechanism. Only two of Engelstein’s patients (one mentioned in the addendum) had an atrial tachycardia that was thought to be based on triggered activity and delayed afterdepolarizations. In those patients, the initiation of tachycardia was dependent on burst pacing and catecholamine stimulation. Termination was accomplished by administration of adenosine and acebutolol and blockade of the slow inward calcium current by verapamil.

Chen et al10 performed extensive electropharmacological studies, including recording the MAP at the presumed site of origin of the arrhythmia. In their 36 patients, these studies were followed by radiofrequency ablation of the area of abnormal impulse formation.

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The authors and their patients have to be complimented on their stamina in completing these extensive protocols! It is impressive that Chen et al were able to make stable MAP recordings in their patients that allowed the demonstration of delayed afterdepolarizations in a subgroup of patients, supporting triggered activity as their tachycardia mechanism.

Endocardial mapping was used to identify the area of earliest activation during tachycardia. In patients with reentrant atrial tachycardia, Chen et al found concealed entrainment with a long stimulus-to-P wave interval when pacing was performed at the supposed exit site of the circuit, suggesting the presence of an area of slow conduction during tachycardia. Radiofrequency energy delivered at that site eliminated atrial tachycardia.

In patients with atrial tachycardia not related to reentry, the combination of pace mapping and identification of earliest atrial activation were key in localizing the site for successful radiofrequency ablation. Apparently, bipolar atrial recordings were used for locating the earliest atrial activation site. In Maastricht, we prefer to localize focal atrial tachycardia by using unipolar recordings and looking for an initial rapid downward deflection in a negative atrial ECG.

Were there differences in electropharmacological findings between the studies of Engelstein et al and Chen et al?19 The major difference was the ability to terminate reentrant atrial tachycardia with verapamil in 19 of 20 of Chen's patients and the failure to do so in Engelstein's series. Chen et al explain the termination of reentrant atrial tachycardia by a slow channel blocker by pointing to their finding of the presence of an area of slow conduction within the reentry circuit. Verapamil therefore may not be the right drug to differentiate between reentry or triggered activity as the mechanism of atrial tachycardia.

Is the information about the mechanism of atrial tachycardia not only of scientific but also of possible therapeutic interest? In my opinion, neither article addresses the therapeutic implications satisfactorily. Engelstein et al do not tell us how the information obtained during the electrophysiological study was used in the clinical management of their patients. Chen et al treated all patients with radiofrequency ablation, independent of the effectiveness of the pharmacological agents used.

Clinically, we know that paroxysmal atrial tachycardia frequently can be treated medically by verapamil or a β-blocking agent, unlike incessant atrial tachycardia, in which drug therapy usually fails and destruction or isolation of the site of impulse formation is necessary. Therefore, decision making about the preferred mode of treatment is easy in patients with incessant atrial tachycardia, especially when a tachycardiomopathy has developed. In those patients, the site of abnormal impulse formation should be ablated. The value of the electrophysiological study in selecting the best pharmacological long-term treatment for patients with paroxysmal atrial tachycardia is not clear from the studies by Engelstein et al and Chen et al.10 It also remains unknown whether the tachycardia mechanism is an important determinant of late recurrences of atrial tachycardia after an initially successful radiofrequency ablation.

References

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