Catheter Ablation of Ventricular Tachycardia Related to Coronary Heart Disease
Defining the Target
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Over the past decade, radiofrequency catheter ablation has become the preferred curative treatment for the most common forms of supraventricular tachycardias.1-3 These tachyarrhythmias are produced by different reentrant mechanisms. In the case of accessory atrioventricular pathways,4-5 all the components of the reentrant circuit had already been functionally and anatomically well identified before catheter ablation was introduced. Radiofrequency catheter ablation uses the accessory pathway as a target to disrupt this reentrant substrate with an excellent success rate reaching 89–99% in the most experienced centers,1-3 but the technique necessitates a laborious and time-consuming mapping procedure because of the small size of the lesion. In the case of atrioventricular nodal reentrant tachycardia, the reentrant substrate was less well defined anatomically before the advent of radiofrequency catheter ablation. The existence of two functional pathways (slow and fast) has been well demonstrated with programmed stimulation,6 but the anatomic location of these pathways, whether both intranodal or not, was undetermined.7 Due to the small extent of the lesions caused by radiofrequency energy, radiofrequency catheter ablation allowed the anatomic substrate underlying this tachycardia to be better defined. It is now established that radiofrequency energy applied in the atrium just caudal to the area where the His bundle potential is recorded is associated with disappearance of the functional fast pathway. In contrast, radiofrequency energy applied remote from the node itself, at the base of the triangle of Koch in an area comprised between the ostium of the coronary sinus, the tricuspid ring, and the atrioventricular node, is associated with the disappearance of the slow pathway. Radiofrequency catheter ablation at either site was not only associated with a high rate of cure8,9 but also provided electrophysiologists with new insights into the mechanism and anatomic substratum of this arrhythmia.

Sustained monomorphic ventricular tachycardia related to coronary heart disease is also believed to be due to a reentrant mechanism as suggested by its behavior during programmed ventricular stimulation10 and by intraoperative activation mapping.11 However, there are no precise anatomic landmarks to guide catheter ablation,12 even though it is admitted that reentry takes place in a structure composed of mixed normal and abnormal tissue with different electrophysiological properties found in the vicinity of a scar due to a previous myocardial infarction.13,14 Previous reports of catheter ablation of ventricular tachycardia related to coronary heart disease and a remote myocardial infarction using DC shock described highly variable success rates that ranged from 20% to 80%.15-18 Unfortunately, comparison among these studies is difficult because of differences in patient selection, definition of success, mapping technique, and energy delivery. Overall, DC shock ablation appears to be less effective than surgery.19

In this issue of Circulation, Morady et al20 describe their experience with radiofrequency catheter ablation of ventricular tachycardia in 15 patients with coronary heart disease and a history of myocardial infarction. There were 20 monomorphic ventricular tachycardias approached for radiofrequency catheter ablation in these 15 patients. Four different methods of localizing the ablation site were used: endocardial activation mapping, pace mapping, identification of isolated mid-diastolic potentials, or concealed entrainment. The first two methods identify the site of emergence, and the latter two identify the area of slow conduction. Morady et al achieved a positive therapeutic goal with the clinical control of recurrent ventricular tachycardia in 15 patients (73%) during a 7-month follow-up period and the noninducibility of sixteen of the 20 morphologies of ventricular tachycardia (80%). As stated by the authors, the studied population is highly selected and represents only 10% of all the patients referred to their center for management of ventricular tachycardia or cardiac arrest related to coronary heart disease and a remote myocardial infarction. Moreover, the authors do not claim a definitive curative role for radiofrequency catheter ablation and point out that it should be considered an adjunct therapy since only two patients of the 11 successfully ablated patients were discharged from the hospital without antiarrhythmic drugs. Consequently, the clinical implications of this preliminary but nonetheless provocative study remain rather limited at the present time. However, two important questions

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come to mind. First, what is the extent of the lesion that is sufficient for successful ablation? Second, how can the optimal ablation site be determined? The success rate (80%) obtained by Morady et al is comparable to the best published results reported by Fontaine et al (81%) who used DC shock ablation, and is better than the 60% success rate previously reported by the same authors using DC shock and a similar mapping procedure. Therefore, the small size of the lesions caused by radiofrequency energy compared with the larger lesions produced by DC shock does not seem to be critical for the successful ablation of VT.

The main problem seems to be the localization of the optimal site of ablation. Rerentry in ventricular tachycardia related to chronic myocardial infarction was initially believed, on the basis of catheter recordings and intraoperative mapping using single-site recordings, to be confined to the subendocardial regions bordering the scar tissue. Further studies using computed mapping or experimental models identified functional and anatomic components of the rerentry circuit such as a zone of slow conduction connecting two zones of normal conduction. By using simultaneously recorded epicardial and endocardial mapping data obtained during ventricular tachycardia surgery with a computed mapping system, our group recently demonstrated that the substrate of ventricular tachycardia has, in a significant number of cases, a three-dimensional structure involving the subendocardium, the myocardium itself, and the epicardium. The subepicardial location or the three-dimensional nature of the ventricular tachycardia substrate can explain failures of catheter ablation in a certain number of cases because critical zones of the rerentry circuit can be situated out of reach of the energy delivery site.

Nevertheless, Morady et al were able to achieve a respectable success rate considering the limited extent of the lesions caused by radiofrequency currents. By using different mapping methods and considering the very localized nature of the radiofrequency lesions, they nicely demonstrated that both the site of emergence and the site of slow conduction on the endocardium might be critical for the maintenance of rerentry. Identifying the components of the anatomic and functional substructures of rerentry may help to better define the target for catheter ablation, as has been the case with atrioventricular nodal rerentrant tachycardia. The small number of highly selected patients and the nonsystematic mapping used in this study does not define the optimal approach to radiofrequency catheter ablation of ventricular tachycardia related to chronic myocardial infarction, but it opens the way to further studies where different mapping techniques will have to be studied in a more controlled fashion. The electrical nature of the scar tissue involved in the rerentry substratum that can occasionally act like an insulating sheath and the limited size of the lesions caused by radiofrequency current also suggest that other types of energy delivery systems such as microwaves should be investigated to address the three-dimensional nature of the substrate.

References


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