Editorial

Idiopathic Left Ventricular Tachycardia
Cure by Radiofrequency Ablation

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In 1979, Zipes et al\(^1\) reported on a peculiar type of ventricular tachycardia in three patients characterized by a QRS with a width of 0.12 to 0.14 second and a right bundle branch block-like shape with left-axis deviation. The patients were young and had no or minor cardiac abnormalities, and their arrhythmias could be induced by exercise, atrial pacing, ventricular pacing, and atrial and ventricular premature beats. The authors postulated that the origin of the tachycardia was probably localized to a small, relatively protected region of reentry or triggered automaticity located in the posteroinferior left ventricle. One year later, termination of a similar type of ventricular tachycardia by verapamil was reported suggesting a possible role of the slow inward calcium channel in the genesis of the arrhythmia.\(^2\) The same authors noted a concordant relation between the premature beat interval initiating ventricular tachycardia and the interval from the premature beat to the first beat of the tachycardia, supporting triggered activity as the possible underlying mechanism of the tachycardia. Observations by Belhassen et al\(^3\) in a larger series of patients proved the sensitivity of this type of ventricular tachycardia to verapamil administration. It also became clear that this arrhythmia predominantly occurs in males, is usually paroxysmal but occasionally incessant in nature, is not entirely benign, and may result in a "tachycardiomyopathy."

Although usually easily terminated by intravenous verapamil, oral administration of this drug may produce disappointing results. This may also occur with oral \(\beta\)-blockade. The young age of the patient with all the problems of side effects and compliance of antiarrhythmic drug treatment and the possibility of the development of a tachycardiomyopathy prompted a search for curative treatment. As postulated by Zipes et al,\(^1\) the morphology of the QRS during the arrhythmia pointed toward an origin of the arrhythmia in or close to the posterior fascicle of the left bundle branch. Endocardial mapping during ventricular tachycardia by German et al\(^4\) revealed different sites of earliest ventricular activation at both the intravenous apex and the mid–left ventricular septum. Interestingly, one of the patients showed a "left bundle" potential that preceded earliest ventricular activation.

In 1987, Fontaine et al\(^5\) reported cure from this type of ventricular tachycardia by the application of a high-energy DC shock (fulguration) in the inferoseptal area of the left ventricle. Subsequently, Klein et al\(^6\) described successful treatment by radiofrequency current in one patient with idiopathic left ventricular tachycardia.

In this issue of Circulation, Nakagawa et al\(^7\) report their results of the application of radiofrequency current in eight patients with idiopathic left ventricular tachycardia. They stress the necessity of a careful search for a short, sharp, high-frequency potential preceding the onset of QRS during tachycardia. Such a potential was found over an area of 2 to 3 cm\(^2\) in the posterior half of the left ventricular septum, one fourth to one third of the distance from apex to base. This area was found to be located more basally than the left ventricular area with earliest ventricular activation during tachycardia, and the potentials recorded were considered to represent activation of the Purkinje fiber network of the left posterior fascicle. Radiofrequency energy delivered in the area of Purkinje potentials resulted in cure of the tachycardia.

It is interesting to speculate on the mechanism of this type of tachycardia. Okumura et al\(^8\) showed that the tachycardia can be entrained by ventricular and atrial pacing. Entrainment by atrial pacing with apparent complete supraventricular capture suggests easy access over the specific conduction system into the reentry circuit. It therefore appears that the Purkinje network of the left posterior fascicle forms an integral part of the reentry loop with ventricular muscle as the slow component of the circuit. Differences in location of the muscle segment might explain the different areas of earliest activation (exit from the tachycardia circuit) described by German et al.\(^4\) This slow component probably is the area sensitive to verapamil, explaining the termination of tachycardia after administration of that drug. The exact location of the segment composed of ventricular muscle is unknown.

What are the consequences of these observations on the reentry circuit for the selection of the appropriate endocardial site for application of radiofrequency current? Nakagawa et al\(^7\) suggest selecting the area where a Purkinje potential precedes the QRS complex during tachycardia when looking for the earliest timing of the Purkinje potential rather than the site of earliest ventricular activation (the exit point of the circuit into ventricular muscle). They also prefer the Purkinje potential to use of pacemapping to select the ablation site.
The latter is not in agreement with our observations in 11 patients with idiopathic left ventricular tachycardia treated with radiofrequency current.9 In our hands, a “match” between 12 simultaneously recorded ECG leads during pacing and during the clinically occurring tachycardia predicted a successful ablation site. Nakagawa et al7 recorded a 12-lead ECG during pacemapping in only two patients, which might be too small a series to condemn pacemapping!

We also observed Purkinje potentials preceding the QRS during tachycardia but preferred to use pacemapping for selection of the ablation site because we were afraid that within the Purkinje network of the left posterior fascicle, pathways may become activated that are not included in the reentry circuit responsible for the tachycardia. We expected that ablation of those “blind alleys” would not result in interruption of the tachycardia circuit.

The observations reported by Nakagawa et al7 and our experience suggest that both approaches may lead to definite cure of idiopathic left ventricular tachycardia. A careful comparison between the two methods is required to select the treatment requiring the smallest number of radiofrequency energy applications and the shortest fluoroscopy time.

References


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