

## Editorial Comment

# Activation Mapping in Ventricular Tachycardia: Role of The Epicardium\*

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Recurrent sustained ventricular tachycardia remains a significant clinical problem with more than its share of disappointing therapeutic alternatives. As properly defined by electrophysiologic testing, antiarrhythmic drug therapy, including treatment with amiodarone, has no more than a 30% to 50% success rate and is in addition limited by allergy, intolerance and proarrhythmic potential (1,2). Although the automatic implantable defibrillator has had an important impact on sudden death, the individual patient remains prone to clinical recurrences of arrhythmia, a fact that is at least intellectually dissatisfying (3). These two factors have led to a persistent search for surgical cures of ventricular tachycardia, and indeed there is no happier or more grateful patient than one who has been relieved of the burden of frequent drug dosing or 30 J shocks, or both.

**Role of surgery.** Because ventricular tachycardia has been known for many years to be primarily a disease of patients with a left ventricular aneurysm, the earliest operations were directed toward aneurysm resection with or without revascularization (4). These operations had an extremely high failure rate, presumably because the rim of scar left behind for closure also leaves behind the ventricular tachycardia circuits, which tend to arise from the aneurysmal edges or border zones (5). More modern approaches, particularly those utilizing activation mapping and endocardial excision, have markedly improved the success rate of surgery, lowering operative mortality in one series from 42% to 7% and the incidence of recurrent ventricular tachycardia from 79% to 10% (6). The failure rate, however, remains substantial, particularly in patients with multiple configurations of tachycardia, akinetic areas rather than discrete aneurysms and inferoposterior scars (7). The current report by Svenson et al. (8) in this issue of the Journal may provide some important insights into why these failures occur.

\*Editorials published in *Journal of the American College of Cardiology* reflect the views of the authors and do not necessarily represent the views of JACC or the American College of Cardiology.

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**Activation mapping.** The earliest attempts at activation mapping for ventricular tachycardia in humans were based on the assumption that the tachycardia circuits were macroreentrant, as in Wolff-Parkinson-White syndrome, possibly involving even the bundle branches (9). Mapping was mostly confined to the epicardium. Surgically, a transmural ventriculotomy 4 to 5 cm in length was performed at sites that were slow conducting during sinus rhythm and early during ventricular tachycardia. These operations met with mixed results, particularly in patients with coronary artery disease. As the techniques of programmed ventricular stimulation and endocardial mapping were refined, it became clear that ventricular tachycardia in humans was, in fact, microreentrant, involving only a small and relatively protected segment of the ventricle, thus implying that ventricular tachycardia could be mapped as a relative point source (10). In subsequent studies (11) in animals with ventricular tachycardia resulting from acute left anterior descending or anteroseptal artery occlusion, a geographic mismatch was demonstrated between the endocardial sites of origin and epicardial sites of breakthrough, such that surgical procedures directed at the epicardium might easily miss the tachycardia sites of origin on the endocardial surface.

Catheter endocardial mapping studies, performed at approximately the same time, showed low amplitude fragmented presystolic potentials and even holodiastolic activity on the endocardium during ventricular tachycardia, particularly around left ventricular aneurysmal edges; this strongly suggested that ventricular tachycardia in humans arose from the endocardium as well and that a portion of the reentrant circuit itself was being recorded (12). Thus, multiple lines of evidence supporting the primacy of the endocardium ultimately led investigators back to the operating room where combined epicardial and endocardial mapping studies during ventricular tachycardia confirmed the mismatch between endocardial origin and epicardial breakthrough, and endocardial excision proved highly effective for the cure of ventricular tachycardia (5). Other successful endocardially directed approaches were thereafter developed (13-15).

**Determining which factors were important in allowing reentrant ventricular tachycardia to originate on the endocardium became the focus of subsequent animal and human investigation.** Indeed several studies (16) using catheter endocardial mapping showed localized fragmented electrical activity on the endocardium extending beyond the QRS complex in normal sinus rhythm in patients with an aneurysm and ventricular tachycardia. It was postulated that these electrograms represented regions of slow conduction, a clear-cut prerequisite for reentry. Wiener et al. (17) performed endocardial and epicardial sinus rhythm maps in such patients in the operating room and compared them with

similar maps in patients with an aneurysm but without ventricular tachycardia. They found extensive fragmentation at the endocardial border in patients with as compared with patients without ventricular tachycardia, and no fragmentation on the epicardium or at the epicardial border in either group. In endocardial preparations resected from the arrhythmogenic area demonstrating fragmentation, both in patients and in animals with ventricular tachycardia, combined morphologic and electrophysiologic studies (18,19) showed that fractionated electrograms occur in regions where surviving muscle fibers are separated and distorted by connective tissue with a resultant decrease in normal intracellular connections. Despite maintenance of normal resting potentials and action potential upstroke velocities, the decrease in intracellular connections appears to increase the resistance to current flow and results in slow conduction in all directions (20). When coupled with the anisotropic properties of the muscle, reentrant rhythms may presumably be readily initiated in such tissue.

**The present study.** Despite the early success of endocardial excision, however, some failures have simply not been accounted for. As Svenson et al. (8) point out, these have primarily been tachycardias in patients with inferoposterior scars, multiple tachycardia configurations or akinetic areas without a true aneurysm, or both. It has usually been presumed that such failures represent problems of access and anatomy (e.g., the inability to resect the papillary muscle) as well as the extended operating room time necessary to resect or ablate posterior sites and the inability to induce and map all tachycardia configurations completely. Svenson et al. suggest otherwise, demonstrating apparent epicardial or intramural origin, or both, of some of the multiple configurations of ventricular tachycardia mapped. They postulate, therefore, that patients without aneurysm and those with an inferoposterior scar may have an infarct with anatomic and electrophysiologic characteristics sufficiently different from those of patients with a typical aneurysm to allow occurrence of reentry that is not dependent on the endocardium. Although detailed anatomic analysis could not be done, the infarct was visibly "patchy" and histologically may have had a more "mottled" appearance than a routine aneurysm leading to multiple inhomogeneous "border zone" areas stretching from endocardium to epicardium. Such an electrophysiologic milieu could certainly support epicardial or intramural reentry, or both. Indeed, the tachycardia characteristics found support such a view, including pandiastolic activity on the epicardium during ventricular tachycardia and, most particularly, the elimination of ventricular tachycardia with epicardial laser application.

Support for the concepts of intramural or epicardial reentry, or both, can be found in the elegant animal and human experiments of Kramer et al. (21) and Hoyt et al. (22). They used extensive computer-assisted three-dimensional epicardial, endocardial and intramural beat to beat mapping

at the initiation and maintenance of ventricular tachycardia and demonstrated "macro"-reentrant circuits up to 14 cm<sup>2</sup> in length in humans and both epicardial "micro"-reentrant and transmural "macro"-reentrant circuits in dogs. The new finding of the current study (8) is an apparent demonstration that some human ventricular tachycardias can be eliminated *only* by therapy applied to intramural or epicardial sites, or both, implying a noncritical role for the endocardium in such tachycardias.

Whether inferoposterior infarcts and akinetic areas are truly histologically different from anterior infarcts and aneurysms in patients with ventricular tachycardia remains to be determined and could not have been addressed in this study (8). Moreover, the study does not separate out whether epicardial or intramural reentry alone or combined epicardial and intramural reentry is critical in the tachycardias cured from the epicardial side because the laser injury penetrates as much as 6 mm in depth. More sophisticated multisite intramural mapping would be needed to make these determinations; nevertheless, the authors (8) have provided important new clues about the origins of ventricular tachycardia in humans and have identified possible reasons for surgical failure in certain patient subsets.

**Laser therapy for ventricular tachycardia.** Although it is not the main focus of the current report (8), a word or two about laser therapy seems appropriate. The application of laser to the surgical therapy of ventricular tachycardia provides an important advance over routine surgical excision (23). First, the therapy can be delivered while the heart is warm and in a sustained arrhythmia, providing immediate verification of therapeutic efficacy and ensuring that second and third arrhythmia configurations can be subsequently induced and treated. Second, access to difficult areas is improved, especially those around the mitral apparatus and under the aortic valve. Third, as with cryoablation, the treated tissues are left intact, but the laser is faster and has its best depth of penetration in the warm heart whereas cryoablation is enhanced in the cold cardioplegia-treated heart. These factors allow more difficult cases to be undertaken and, coupled with the insights into tachycardia origins provided by Svenson et al. (8), they expand the possibilities for surgical cure of ventricular tachycardia while opening new vistas for more basic investigation.

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