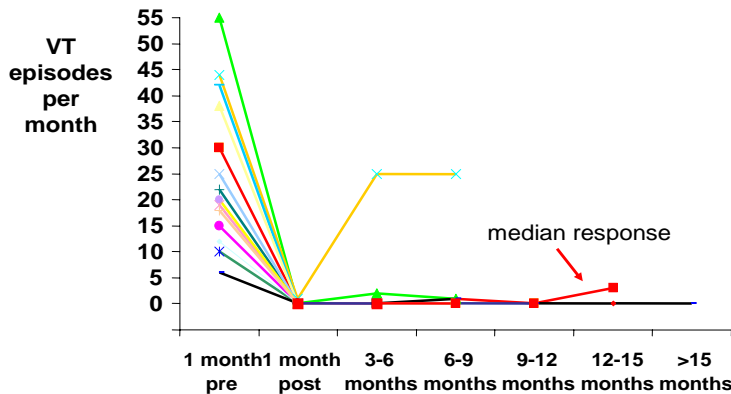


VT Ablation

Catheter Ablation of Ventricular Tachycardia and Ventricular Fibrillation at the University of Colorado at Denver and Health Science Center

The Ventricular Tachycardia Ablation Program at UCDHSC strives to achieve the highest level of efficacy while maintaining the safest approaches that have been thoroughly evaluated by experienced high-volume academic centers. Each patient with ventricular tachycardia (VT) or ventricular fibrillation (VF) is evaluated and treated individually and therefore there is no specific approach that we employ for everyone who undergoes the procedure. Most patients seek medical attention for their ventricular arrhythmias because they experience symptoms of palpitations, syncope, or painful shocks from an ICD. VT ablation has not been shown to improve survival but it has proven to be an effective treatment for controlling symptoms and preventing ICD shocks (Figure 1).

Figure 1. Reduction in ICD therapies following VT Ablation



The subtypes of VT include outflow tract tachycardias arising from the regions of the aortic and pulmonic valves, idiopathic VT in structurally normal hearts (usually arising from fascicular tissue), and VT utilizing circuits defined within or on the borders of myocardial scar in patients with ischemic or non-ischemic cardiomyopathy. The last type of VT can sometimes present as incessant VT requiring an urgent evaluation and ablation.

In addition, there are some patients who experience shocks from an implanted cardioverter-defibrillator (ICD) for VF but actually have VT or premature ventricular contractions (PVCs) that trigger VF (Figures 2 and 3).

Figure 2. Initiation of VF from PVCs after myocardial infarction

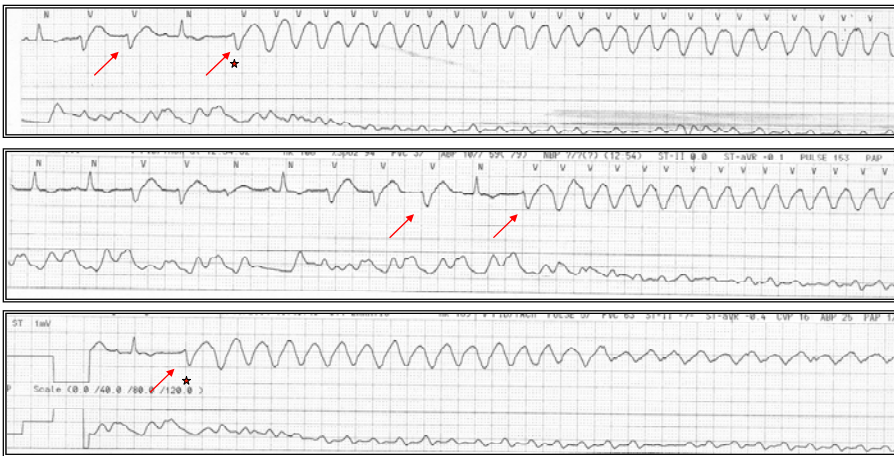
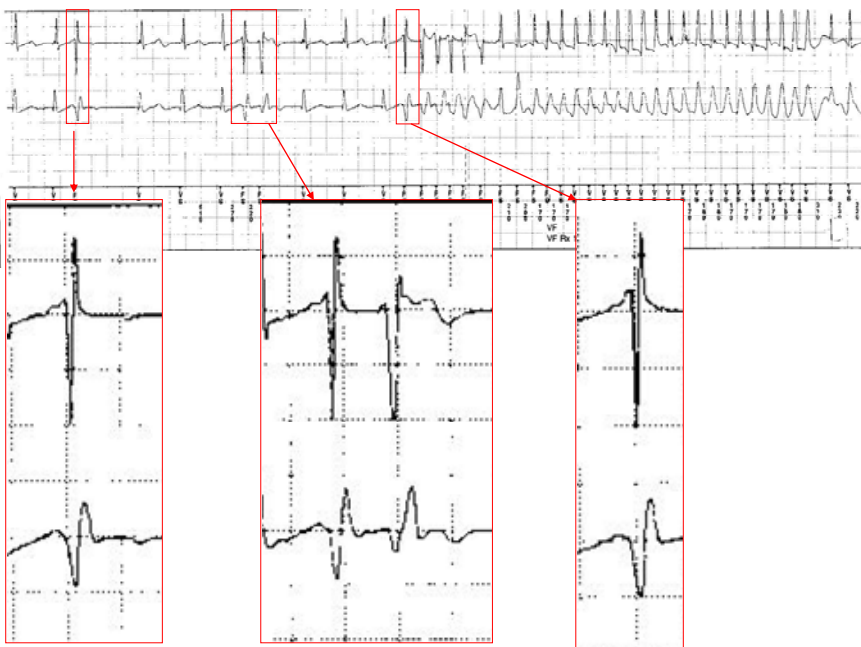
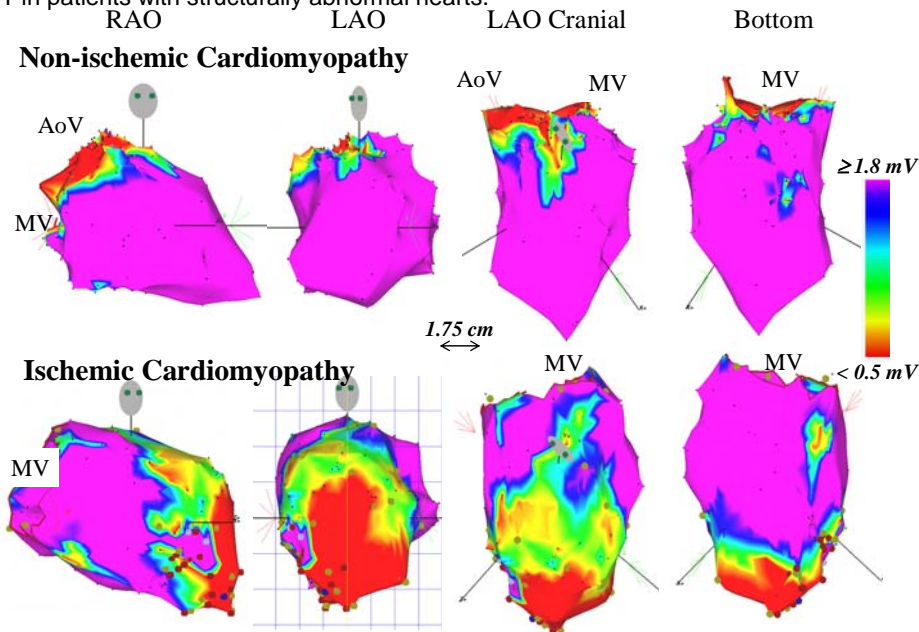


Figure 3. Stored ICD intracardiac electrograms of PVCs causing VF.



If a 12-lead electrocardiogram of the VT is available, the approximate region and subtype of VT can usually be determined ahead of time. Therefore the ventricular chamber and ablation strategy to be employed can often be discussed with the patient ahead of time. A patient who is considered a candidate for a VT ablation will undergo an electrophysiological study (EPS) and a 3-dimensional map will be created of the ventricle in order to identify the anatomy and regions of scar or abnormal tissue. This map assists in localizing the origin of the induced ventricular tachycardias (patients with scar-related VT typically have multiple VT circuits), as well as mapping where ablation lesions have been placed. The map is typically color-coded in a way to indicate regions of normal and abnormal myocardium (Figure 4).

Figure 4. Regions of low voltage or scar, identified by red color, are a frequent source of VT in patients with structurally abnormal hearts.



During the EPS, the arrhythmia is typically induced with programmed stimulation, and then mapping and pacing maneuvers are used to localize critical areas of a circuit or arrhythmia focus that can be targeted for ablation. If the induced VT is not tolerated, or if VF is induced, electrical cardioversion will immediately be used to terminate the rhythm. Even in this situation, important mapping information can be obtained during the brief episode of VT that allows for ablation of the VT origin during sinus rhythm. The target end-point to the procedure is always lack of inducible VT.

The risks of the procedure depend on the chamber involved in the tachycardia, the VT subtype targeted for ablation, and patient comorbidities, and include cardiac perforation and tamponade, stroke, myocardial infarction, valvular injury, and vascular trauma. Overall, the risk for a major complication is low but cannot be predicted.

The success rates for VT ablation vary depending on an individual's substrate for the arrhythmia. A patient with an automatic VT arising from the right ventricular outflow tract in a structurally normal heart will be quoted a much higher success rate when compared to a patient with a severely reduced ejection fraction from a large anterior wall myocardial infarction with multiple VT morphologies.

For more information on ablation of VT and VF, we recommend the following articles available online and through our office:

Stevenson W, Friedman P, Kocovic D, Sager P, Saxon L, Pavri B. Radiofrequency catheter ablation of ventricular tachycardia after myocardial infarction. *Circulation*. 1998;98:308–314.

Rothman S, Hsia H, Cossú S, Chmielewski I, Buxton A, Miller J. Radiofrequency catheter ablation of postinfarction ventricular tachycardia (long-term success and the significance of inducible nonclinical arrhythmias). *Circulation*. 1997;96:3499–3508

Marchlinski F, Callans D, Gottlieb C, Zado E. Linear ablation lesions for control of unmappable ventricular tachycardia in patients with ischemic and nonischemic cardiomyopathy. *Circulation*. 2000;101:1288–1296.

Marchlinski F, Garcia F, Siadatan A, Sauer W, Beldner S, Zado E, Hsia H, Lin D, Cooper J, Verdino R, Gerstenfeld E, Dixit S, Russo A, Callans D. Ventricular Tachycardia and Ventricular Fibrillation Ablation in the Setting of Ischemic Heart Disease. *Journal of Cardiovascular Electrophysiology* 2005; 16 S59-S70

Hsia H, Callans D, Marchlinski F. Characterization of the electrophysiologic substrate in patients with non-ischemic cardiomyopathy and monomorphic ventricular tachycardia. *Circulation*. 2003;108:704–710.

Hsia HH, Lin D, Sauer WH, Callans DJ, Marchlinski FE. Anatomical Characterization of Endocardial Substrate for Hemodynamically Stable Reentrant Ventricular Tachycardia: Identification of Endocardial Conducting Channels. *Heart Rhythm* 2006; 3: 503-512