ELECTROPHYSIOLOGY

Radiofrequency catheter ablation of ventricular tachycardia

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Management of patients with ventricular tachycardia (VT) is often difficult. Drug treatment is often ineffective. Implantable defibrillators terminate episodes but do not prevent them. Radiofrequency (RF) catheter ablation offers potential arrhythmia control without the adverse effects of antiarrhythmic treatment. However, the procedure is often challenging and efficacy is less than for ablation of supraventricular tachycardias. The efficacy and safety depend on the particular type of tachycardia and its likely origin. These factors can be predicted from the underlying heart disease and the electrocardiographic characteristics of the tachycardia

VTs are either polymorphic or monomorphic. Polymorphic tachycardias have a continuously changing QRS morphology, indicating a variable sequence of ventricular activation and no single site of origin. The cause is often ischaemia or drug induced QT prolongation; ablation is not an option.

Monomorphic VT has a constant QRS morphology from beat to beat, indicating repetitive ventricular depolarisation in the same sequence. An arrhythmia focus or structural substrate is present that can be targeted for ablation. The QRS morphology often indicates the likely arrhythmogenic region. A left bundle branch block-like configuration in lead V1 indicates an origin in the right ventricle or the interventricular septum. A frontal plane axis that is directed inferiorly (dominant R waves in leads II, III, AVF) indicates an origin in the superior aspect of the ventricle, either the anterior wall of the left ventricle or the right ventricular outflow tract. A frontal plane axis directed superiorly indicates initial depolarisation of the inferior wall of the left or right ventricle. Dominant R waves in leads V3–V4 favour a location nearer the base of the heart than the apex. Dominant S waves in these leads favour a more apical location. The QRS morphology is an excellent guide to the arrhythmia origin when the ventricles are structurally normal, but less reliable when VT is caused by infarction or ventricular scar.

The underlying heart disease provides further important information. VT in patients without identifiable structural heart disease is referred to as "idiopathic". These tachycardias usually occur in specific locations and have specific QRS morphologies. Tachycardias associated with scar, such as prior myocardial infarction, have a QRS morphology that tends to indicate the location of the scar. Patients with non-ischaemic cardiomyopathies, including valvar heart disease, have an increased incidence of bundle branch re-entry tachycardia (see below), although other mechanisms are frequent in these patients as well.

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Idiopathic VT

VT in patients without structural heart disease is uncommon.[1–3] Most originate from a small focus, making them susceptible to ablation. The prognosis is good; sudden death rarely if ever occurs unless some other form of heart disease is present, but tachycardia can be sufficiently rapid to cause syncope or severe symptoms. Rarely, VT is incessant and causes heart failure with depressed ventricular function that resolves with control of the arrhythmia. Although the focus can be anywhere in the ventricles, the vast majority originate from one of two locations.

Idiopathic right ventricular outflow tract tachycardia

The most common idiopathic VT originates from a focus in the outflow tract of the right ventricle (fig 1).[1 2] The mechanism is most likely triggered automaticity.[4] VT has a left bundle branch block configuration in ECG lead V1 with a frontal plane axis that is directed inferiorly or inferiorly and to the right. Premature ventricular contractions with an identical morphology are often present during sinus rhythm. Tachycardia may occur in repetitive bursts (referred to as repetitive monomorphic VT). In some patients non-sustained VT and frequent premature beats are severely symptomatic and warrant treatment. Although echocardiogram, ECG, and angiography are generally normal, cardiac magnetic resonance imaging may identify areas of focal thinning, hypokinesis or fatty infiltration. The major diagnostic consideration is that of arrhythmogenic right ventricular dysplasia (see below).

In contrast to scar related re-entry (see below) the automaticity that causes these tachycardias is often provoked by adrenergic stimulation and appears to be sensitive to increases in intracellular calcium. Treatment with calcium channel blockers (verapamil and diltiazem), which is contraindicated in most other types of VT, often suppresses the arrhythmia. β Adrenergic blockers are also often effective, particularly if the arrhythmias are provoked by exercise. Catheter ablation is a reasonable consideration when pharmacologic treatment is not effective or tolerated. It can be considered for patients with sustained VT, non-sustained bursts of VT, or frequent symptomatic ventricular premature beats. The focus is located by finding the earliest site of activation during tachycardia (activation sequence mapping) (fig 1), or by finding the site where pacing exactly reproduces the QRS

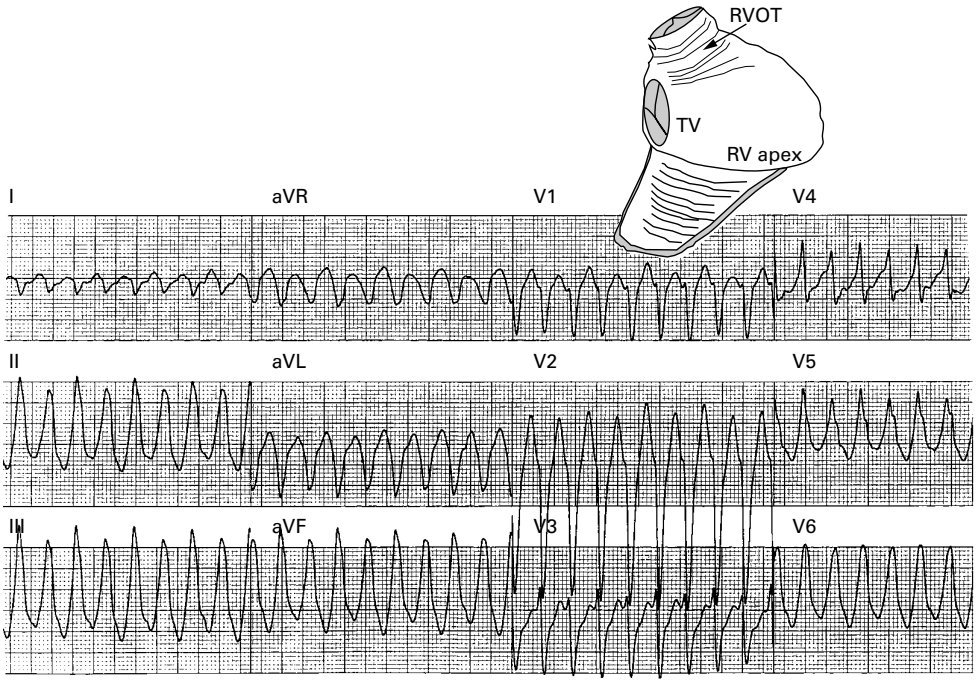

Figure 1. Idiopathic right ventricular outflow tract tachycardia. The 12 lead ECG shows tachycardia with a left bundle branch block, configuration and frontal plane axis directed inferiorly. The schematic at the upper right shows the right ventricle viewed from the right anterior oblique position with the free wall of the ventricle folded down. The location of the tachycardia in the right ventricular outflow tract (RVOT) is indicated with an arrow. TV, tricuspid valve; RV, right ventricle.

morphology of the tachycardia (pace mapping). Ablation is successful in approximately 85% of patients.[1] Failures are caused either by an inability to induce the arrhythmia in the laboratory, preventing adequate mapping, or by the location of the focus deep within the septum or in the epicardium over the septum, beyond the reach of endocardial RF ablation lesions. Occasionally ablation from the left side of the interventricular septum is required. Complications are infrequent, but cardiac perforation and coronary artery occlusion during ablation in the left ventricular outflow tract have occurred.[2]

Idiopathic left ventricular, verapamil sensitive tachycardia

The most common idiopathic left VT has a right bundle branch block configuration with a frontal plane axis that is directed superiorly, or rarely inferiorly and to the right.[1 3] Administration of intravenous verapamil terminates tachycardia suggesting that slow calcium channel dependent tissue is involved. The mechanism appears to be re-entry involving the distal fascicles of the left bundle branch. Re-entry involving Purkinje tissue in or adjacent to a left ventricular false tendon, which is present in more than 90% of patients, has also been suggested.

When treatment with β adrenergic blockers and/or calcium channel blockers is ineffective or not tolerated catheter ablation is a reasonable alternative. Mapping for ablation seeks sites where a discrete Purkinje potential precedes the QRS complex during tachycardia.[3] Ablation is successful in approximately 90% of patients. Failures are sometimes caused by catheter induced trauma to the arrhythmia focus (or possibly the false tendon) which then prevents initiation, precluding mapping. Complications are infrequent but damage to the aortic or mitral valve apparatus from catheter manipulation can occur.

VT related to regions of scar

The majority of sustained monomorphic VTs are caused by re-entry involving a region of ventricular scar. The scar is most commonly caused by an old myocardial infarction, but arrhythmogenic right ventricular dysplasia, sarcoidosis, Chagas' disease, other non-ischaemic cardiomyopathies and surgical ventricular incisions for repair of tetralogy of Fallot, other congenital heart diseases, or ventricular volume reduction surgery (Batista procedure) can also cause scar related re-entry. Dense fibrotic scar creates areas of anatomic conduction block. Secondly, fibrosis between surviving myocyte bundles decreases cell to cell coupling, and distorts the path of propagation causing slow conduction, which promotes re-entry (fig 2).[5] These re-entry circuits often contain a narrow isthmus of abnormal conduction. Depolarisation of the small mass of tissue in the isthmus is not detectable in the body surface ECG. The QRS complex is caused by propagation of the wavefront from the exit of the circuit to the surrounding myocardium (fig 2). After leaving the exit of the isthmus, the circulating re-entry wavefront may propagate through a broad path along the border of the scar (loop), back to the entrance of the isthmus. A variety of different circuit configurations are

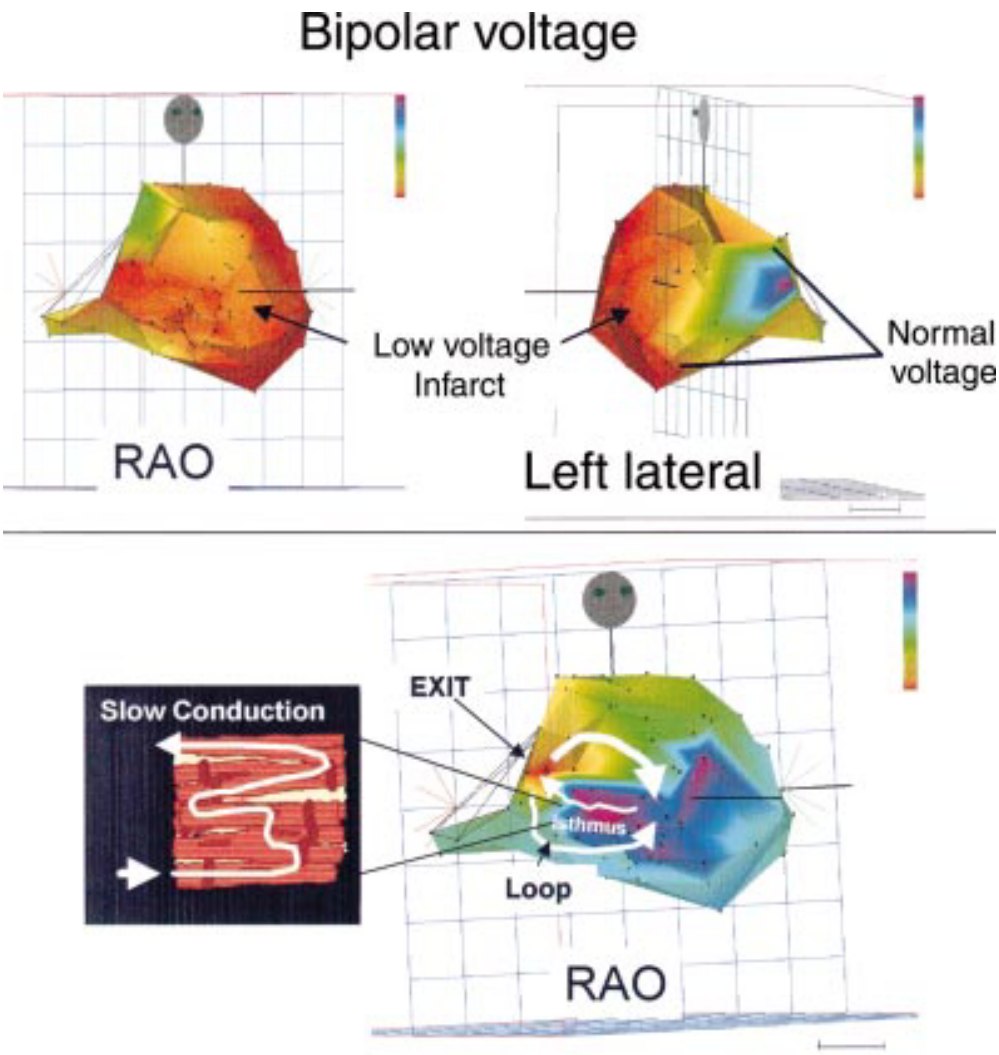

Figure 2. The mapping data are from a patient with VT late after anterior wall myocardial infarction. Mapping was performed using a system that plots the precise catheter position along with colour coded electrophysiologic information (CARTO Biosense Webster, Diamond Bar, California, USA). The top two panels show the left ventricle in right anterior oblique (RAO) and left lateral views. In this case, colours indicate the electrogram voltage, rather than timing. The lowest voltage regions are shown in red, progressing to greater voltage regions of yellow, green, blue, and purple. A large anteroapical infarction is indicated by the extensive low voltage, red region. The lower right panel shows the map of VT in the same patient. The ventricle is again shown in a right anterior oblique projection with the apex at the right and the base at the left hand side of the image. The colours indicate the activation sequence and arrows have been drawn to clarify the activation sequence of the circuit. The re-entry circuit is located in the septum. The wavefront starts at the red area (exit) near the base of the septum and splits into two loops that circle around the superior and inferior aspect of the septum toward the apex, re-entering an isthmus in the circuit that is proximal to the exit region. RF ablation in the isthmus abolished tachycardia. The mechanism of slow conduction through the infarct region that has been observed in previous histopathologic studies is illustrated schematically in the inset at lower left. Surviving myocyte bundles are separated by fibrous tissue that forces the wavefront to take a circuitous path through the region.

possible. Ablation lesions produced with standard RF ablation catheters are usually less than 8 mm in diameter, relatively small in relation to the entire re-entry circuit, and can be smaller than the width of the re-entry path at different points in the circuit. Successful ablation of a large circuit is achieved either by targeting an isthmus where the circuit can be interrupted with one or a small number of RF lesions, or by creating a line of RF lesions through a region containing the re-entry circuit.

Identification of critical isthmuses is often challenging. The abnormal area of scarring, where the isthmus is located, is often large and contains "false isthmuses" (bystanders) that confuse mapping. In most cases a portion of an isthmus is located in the subendocardium where it can be ablated. However, in some cases the isthmuses or even the entire circuits are deep to the endocardium or even in the epicardium and cannot be identified or ablated from the endocardium.

The situation is further complicated by the frequent presence of multiple potential re-entry circuits, giving rise to multiple different monomorphic VTs in a single patient. Ablation in one area may abolish more than one VT, or leave VT circuits in other locations intact. The frequent presence of multiple VTs also complicates interpretation of outcomes. VTs that have been documented to occur spontaneously are referred to as "clinical tachycardias". Those

that are induced in the electrophysiology laboratory, but have not been previously observed, are sometimes referred to as "non-clinical tachycardias". However, a "non-clinical VT" may occur later, after ablation of the "clinical VT". In addition the ECG of spontaneous VTs terminated by an implanted defibrillator or emergency medical technicians is often not available. Thus the distinction between "clinical" and "non-clinical" is often uncertain.

When VT is slow and haemodynamically tolerated a re-entry circuit isthmus can usually be found during catheter mapping (fig 2). Extensive mapping during VT is not possible when VT causes haemodynamic instability or the re-entry circuit is not stable, but repeatedly changes causing multiple different morphologies of monomorphic VT.

Ablation of VT after myocardial infarction

Most reported series included patients who had at least one mappable VT. Gonska and colleagues selected 72 patients who had a single clinical VT. RF ablation abolished the clinical VT in 74% of patients; 60% of the total group remained free of spontaneous VT recurrences during follow up.[6] Stevenson,[7] Rothman,[8] and Strickberger[9] and associates targeted multiple VTs for ablation in 108 patients with recurrent VT. An average of 3.6–4.7 different VTs were inducible per patient. All inducible monomorphic VTs were abolished in 33% of patients; in 22% of patients ablation had no effect. In the remaining 45% of patients the re-entry substrate was "modified"; the VTs targeted for ablation were rendered non-inducible, but other VTs remained. During mean follow ups ranging from 12–18 months, 66% of patients remained free of recurrent VT and 24% suffered recurrences. The incidence of sudden death was 2.8%, but most patients had an implanted defibrillator; the sudden death risk may be higher if ablation is used as sole treatment.

Saline irrigation of the ablation electrode (cooled RF ablation) may create larger lesions to reach deep portions of re-entry circuits by allowing current delivery without excessive heating at the surface of the tissue, which can cause formation of coagulum that prevents further energy application. A recent multicentre trial evaluated a saline irrigated RF ablation catheter (Cardiac Pathways Corp, Sunnyvale, California, USA) in 146 patients (prior myocardial infarction in 82%; average (SD) left ventricular ejection fraction 31 (13)%) who had an average of 25 (31) episodes of VT in the two months before ablation despite antiarrhythmic drug treatment.[10] All mappable VTs were eliminated in 75% of patients. During a follow up of 243 days 54% of patients remained free of spontaneous VT; 81% experienced a more than 75% reduction in the number of VT episodes in the two months after ablation, as compared to before ablation.

Patients with VT caused by prior infarction have depressed ventricular function and concomitant illnesses. Ablation is often a late attempt in controlling refractory arrhythmias, sometimes after significant haemodynamic compromise has developed. Significant complications of stroke, transient ischaemic attack, myocardial infarction, cardiac perforation requiring treatment, or heart block occur in approximately 5–8% of patients. Procedure related mortality is 1% in pooled data and 2.8% in the one reported multicentre trial of cooled RF ablation discussed above.

During follow up the largest source of mortality is death from heart failure, with an incidence of approximately 10% over the following 12–18 months.[6–10] This risk of death is not unexpected in this population. However, ablation injury to contracting myocardium outside the infarct or injury to the aortic or mitral valves during left ventricular catheter manipulation are procedural complications that could exacerbate heart failure. Restricting ablation lesions to areas of infarction, as identified from low amplitude electrograms in regions observed to have little contractility on echocardiogram or ventriculogram, is prudent.

Arrhythmogenic right ventricular dysplasia

Arrhythmogenic right ventricular dysplasia is associated with fibrous and fatty scar tissue in the right and often the left ventricles. VT typically has a left bundle branch block-like configuration in V1, consistent with a right ventricular origin. When right ventricular involvement is extensive, the success of ablation is variable.[11] Individual VTs can be ablated, but others may develop later possibly related to progression of the disease process. Ablation is reserved as a palliative treatment for frequent episodes. Although the right ventricle can be quite thinned, the risk of perforation during mapping does not seem to be substantially increased.

VT caused by non-ischaemic cardiomyopathy

The mechanisms of sustained monomorphic VT in non-ischaemic cardiomyopathies (including idiopathic cardiomyopathy and valvar heart disease) are diverse. In a series of 26 patients with monomorphic VT the causes were scar related re-entry circuits in 62% of patients, an ectopic focus in 27%, and bundle branch re-entry in 19%.[12] Ablation was successful for 60% of the scar related VTs and 86% of the VTs caused by focal automaticity. The difficulties in ablation of scar related VT are similar to those encountered in patients with prior myocardial infarction; multiple tachycardias are not uncommon, but reduction in the number of episodes and termination of incessant tachycardia can often be achieved. Successful ablation of scar related VTs in patients with sarcoidosis, scleroderma, Chagas' disease,[13] and late after repair of tetralogy of Fallot[14] have also been reported, although experience is limited.

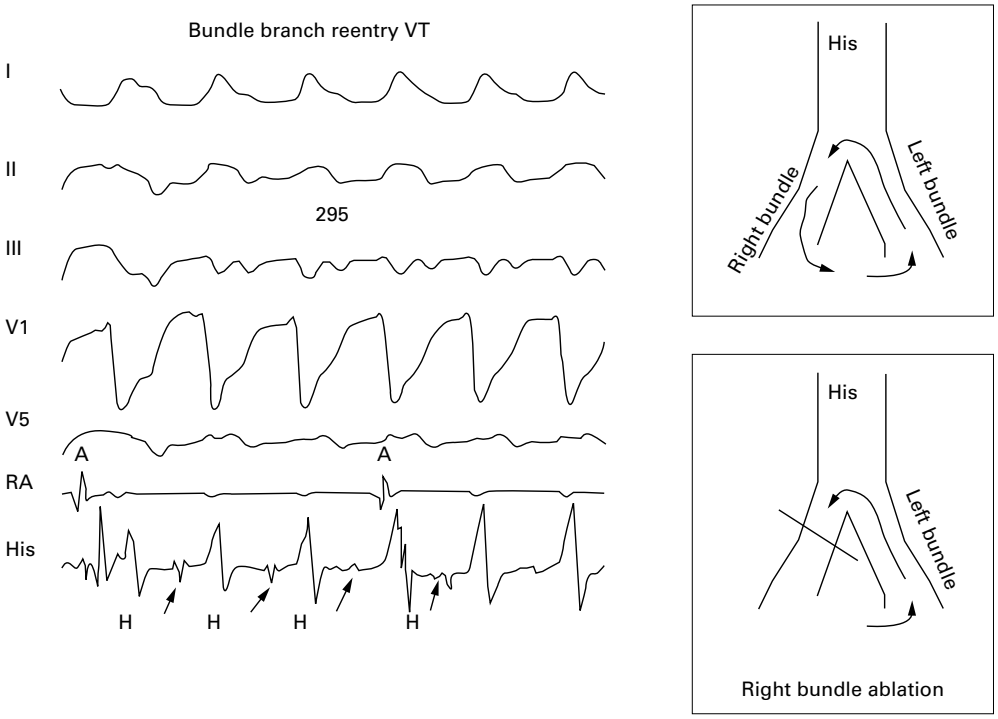

Figure 3. Bundle branch re-entry tachycardia. The left hand panel shows bundle branch re-entry tachycardia initiated in the electrophysiology laboratory. From the top are surface ECG leads and intracardiac recordings from the right atrium (RA) and His bundle position (His). VT has a left bundle branch block configuration and cycle length of 295 ms. Atrioventricular dissociation is evident in the right atrial recording (RA). A His bundle deflection (arrows) precedes each QRS indicating that the His-Purkinje system is closely linked to the tachycardia. The schematic in the right hand panels illustrates the mechanism. The wavefront circulates down the right bundle, through the interventricular septum, and up the left bundle (top panel). Ablation of the right bundle branch interrupts the circuit (bottom panel).

Problems and emerging solutions for ablation of scar related tachycardias

Intramural and epicardial circuits

Mapping arrhythmia foci or circuits that are deep within the myocardium or in the epicardium is being attempted in one of two ways. Small, 2 French electrode catheters can be introduced into the coronary sinus and advanced out into the cardiac veins. Epicardial circuits can sometimes be identified, but only when the vessel cannulated happens to be in the region of the circuit. Ablation through the vein may carry the risk of injury to adjacent coronary artery.

Sosa and colleagues have developed an epicardial approach inserting an introducer into the pericardial space in the manner used for pericardiocentesis.[13] Epicardial foci have been identified and ablated using this approach. The risk of damage to adjacent lung and epicardial vessels requires further evaluation.

The size of standard RF ablation lesions is limited by formation of a high resistance barrier of coagulated proteins on the ablation electrode when its temperature reaches 100°C. To increase current delivery without coagulum formation, the electrode can be cooled by irrigation with saline, or by using a larger tip electrode, which increases the surface area available for cooling by the circulating blood.[10] Ablation methods that increase lesion size could increase the risk of myocardial damage that could further depress ventricular function. Careful assessment of risks are required with each advance.

Unstable monomorphic VT

Two approaches are being evaluated for ablation of scar related VT that is difficult to map with a roving catheter because of haemodynamic instability or instability of the re-entry circuit. One approach involves defining the area of scar from its low amplitude sinus rhythm electrograms (fig 2, top panels); then selecting portions of the scar likely to contain a part of the re-entry circuit based on the VT QRS morphology or pace mapping; and then placing a series of anatomically guided ablation lesions through the abnormal region.[15 16] Ellison and colleagues targeted the likely re-entry exit region in five patients with frequent unmappable VT. All three patients with prior myocardial infarction were free of recurrent VT during follow ups of 14–22 months. The procedure was not successful in the two with non-ischaemic cardiomyopathy.[15] Marchlinski and colleagues applied a more extensive series of RF ablation lines through regions of scar in 16 patients with recurrent unmappable VT (prior myocardial infarction in nine patients).[16] During a median follow up of eight months 75% remained free of VT recurrences. One patient suffered a stroke, emphasising the potential risk of placing extensive lesions in the left ventricle.

Table 1 Ventricular tachycardia mechanisms and ablation considerations

	Mechanism	*Ablation efficacy*	*Complication risk*
Idiopathic VT			
RV outflow tract	Automaticity	80–90%	Low, but rare fatalities
LV verapamil sensitive	Re-entry	90%	Low
Post-MI "mappable" VT	Re-entry		
Reduction in VT episodes		70–80%	5–10%
Prevention of all VT		50–67%	5–10%
Post-MI "unmappable"		?	?
Other scar related VTs	Re-entry		
RV dysplasia + RV dilation		Palliative	?
Non-ischaemic cardiomyopathy		~60%	Low
Bundle branch re-entry VT	Re-entry through bundle branches	100%	AV block

AV, atrioventricular; LV, left ventricular; RV, right ventricular; MI, myocardial infarction; VT, ventricular tachycardia.

VT that is unmappable with a single roving catheter may be mapped with a system that simultaneously records electrograms throughout the ventricle during one or a few beats of the unstable VT, following which the VT can be terminated to allow ablation during stable sinus rhythm. Multielectrode basket catheters have been successfully deployed through a long sheath into the ventricle, but have somewhat limited sampling.[17] An alternative system (Endocardial Solutions, St Paul, Minnesota, USA) records electrical potentials from an electrode grid array within the cavity of the ventricle. Electrical potentials at the endocardial surface some distance away are calculated. Sites of early endocardial activity, which are likely adjacent to re-entry circuit exits, are usually identifiable; in some cases, isthmuses have been identified.[18 19] Schilling and colleagues used this system to guide ablation in 24 patients (20 with prior infarction) and recurrent VT. During a mean follow up of 18 months, 64% were free of recurrent VT. In 15 patients Strickberger and associates achieved ablation of 15 of 19 (78%) VTs that were selected for ablation in 15 patients with prior infarction; 10 were free of recurrent VT during a short one month follow up. Major complications of stroke, perforation, and death from pump failure occurred in three patients. Further evaluation with regards to safety and efficacy are warranted.

Bundle branch re-entry VT

Bundle branch re-entry causes only 5% of all sustained monomorphic VTs in patients referred for electrophysiologic study, but is important to recognise because it is easily curable.[20] In its usual form the excitation wavefront circulates up the left bundle branch, down the right bundle branch, and then through the interventricular septum to re-enter the left bundle (fig 3), causing VT with a left bundle branch block configuration. Less commonly, the circuit revolves in the opposite direction. This VT occurs in patients who slowed conduction through the His Purkinje system and is usually associated with severe left ventricular dysfunction. The sinus rhythm ECG usually displays incomplete left bundle branch block. The VT is often rapid, commonly causing syncope or cardiac arrest. Ablation of the right bundle branch is relatively easy and effective. AV conduction is further impaired by ablation, necessitating implantation of a pacemaker or defibrillator with bradycardia pacing in 15–30% of patients. Bundle branch re-entry VT coexists with scar related VTs in some patients; implantation of a defibrillator is usually considered.

Current clinical application

Catheter ablation is a useful treatment for selected patients with VT. It should be considered for patients with recurrent, symptomatic idiopathic VT and is the first line treatment for bundle branch re-entry VT.

Catheter ablation offers improved arrhythmia control in two thirds of patients who have a mappable scar related VT (table 1). It can be lifesaving for patients with incessant VT, and can decrease frequent episodes of VT causing therapies from an implanted defibrillator. Before considering ablation possible aggravating factors should be addressed. Although myocardial ischaemia by itself does not generally cause recurrent monomorphic VT, it can be a trigger in patients with scar related re-entry circuits. Furthermore severe ischaemia during induced VT increases the risk of mapping and ablation procedures. An assessment of the potential for ischaemia is generally warranted in patients with coronary artery disease who are being considered for catheter ablation. Patients with left ventricular dysfunction should also have an echocardiogram to assess the possible presence of left ventricular thrombus that could be dislodged and embolise during catheter manipulation in the left ventricle.

The difficulty of the procedure increases when unmappable VTs are present. Many laboratories restrict ablation attempts to patients with mappable VTs. Current studies focusing on methods of ablation of unmappable VTs and epicardial and intramural arrhythmia foci are likely to increase efficacy and applicability. Scar related VTs are often associated with poor ventricular function and multiple inducible VTs. Most patients will remain

candidates for an implanted defibrillator, with ablation used for control of symptoms caused by frequent arrhythmia recurrences.

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