THEME

Arrhythmias

Catheter ablation techniques in managing arrhythmias

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BACKGROUND

Pharmacological management of arrythmias is not curative, is sometimes difficult, and may be associated with significant morbidity.

OBJECTIVE

This article discusses the place of catheter ablation in the treatment of arrhythmias.

DISCUSSION

Supraventricular and typical atrial flutter can be cured with catheter ablation in approximately 95% of cases with very low complication rates. Catheter ablation can successfully restore and maintain sinus rhythm in patients with atrial fibrillation (AF) and should be considered in symptomatic patients not controlled on medical therapy before the initiation of amiodarone. Results for AF ablation are best in paroxysmal patients without structural heart disease. Ventricular tachycardia in patients without structural heart disease is readily treated with catheter ablation. Catheter ablation is an effective adjunct to an implantable cardioverter defibrillator in patients with ventricular tachycardia postmyocardial infarction.

The pharmacological treatment of cardiac arrhythmias is not curative and may be associated with significant morbidity including proarrhythmia. Supraventricular (SVT) and ventricular tachycardia (VT) in the structurally normal heart can be cured with catheter ablation (CA) with very low complication rates.[1,2] Outcomes for CA of atrial fibrillation (AF) and infarct related ventricular tachycardia (VT) are improving with better understanding of arrhythmia mechanisms and evolving technologies.[3,4] Electrical cardioversion and pacing/defibrillation devices are also effective tools in treating patients with cardiac arrhythmias. This article will focus on CA including expected success and complication rates.

Electrophysiologic study and catheter ablation

Electrophysiologic study (EPS) and CA is a minimally invasive procedure that can be performed under light sedation and usually requires an overnight hospital stay. Energy delivery for SVT is usually well tolerated, however ablation for typical atrial flutter or fibrillation often requires deeper sedation or general anaesthesia.

How are EPS and CA performed?

The steps involved in the EPS and CA process are:

- multiple venous sheathes are inserted and multipolar electrodes positioned at locations in the right atrium, coronary sinus, His bundle and right ventricle
- an EPS is performed that involves inducing tachycardia and determining the mechanism responsible for the arrhythmia
- a steerable mapping/ablation catheter is positioned at a critical limb of the circuit and radiofrequency energy is delivered. Radiofrequency energy provides a thermal 'burn' producing cell death and scar formation. This creates a 'circuit breaker' which prevents circumnavigation of the electrical impulse. Cryoablation or 'freezing' is useful for arrhythmias where the target for ablation is in close proximity to the atrioventricular (AV) node. This technology produces a reversible lesion during the cryomapping

Table 1. Indications for catheter ablation

	Indications	Efficacy	Complications	Comments
SVT	Class I • Recurrent symptomatic palpitations (class I) • Hemodynamic compromise (class I) • Patient preference following a single episode or infrequent episodes (class I)	Long term cure in 96% of AV node re-entrant tachycardias with recurrence in 3–5%[6,7] CA targeted to the accessory pathway in AV re-entrant tachycardias (eg. WPW syndrome is successful in 95%[8])	The incidence of serious complications is ~1%[5] The major complication in AV node re-entrant tachycardias is AV nodal damage requiring implantation of a permanent pacemaker (<1%)	AV nodal re-entrant tachycardias are the most common form of SVT The role of catheter ablation in asymptomatic patients with pre-excitation accessory pathways is controversial and usually reserved for patients in high risk occupations
Atrial flutter	Typical atrial flutter • Recurrent well tolerated palpitations (class I) • Poorly tolerated single episode (class I) • Patient preference after a single well tolerated episode (class IIA)	CA for typical atrial flutter is successful in 90% It reduces recurrent arrhythmia, improves quality of life and reduces hospitalisation over pharmacological therapy[11]	Later incidence of AF of up to 25%[12] The risk of developing AF following successful CA for atrial flutter is determined by the coexistence of AF before ablation	CA for typical atrial flutter often requires deeper sedation or general anaesthesia
	Focal AT • After failed antiarrhythmic therapy (class IIA)	Success rates for CA of focal AT are >90%[14,15]	Success rates for CA of focal AT are >90%[14,15]	In focal AT tachycardias may be difficult to induce making mapping difficult
AF	Significant symptoms in patients who have failed antiarrythmic medication	Successfully abolishes AF in 70–80% of patients[19] Best results in patients with paroxysmal AF and structurally normal hearts	Risk of serious complications 1–2 % (stroke, pericardial tamponade, pulmonary vein thrombosis)	Procedure takes 3–4 hours and requires deeper sedation or general anaesthesia and overnight stay Repeat procedure is required in about 30%
VA	• In patients with structurally normal hearts • To prevent or reduce recurrent VA in patients with ICD in situ following myocardial infarct or cardiomyopathy associated with VF or VT	Acute success in up to 82% Long term success in around 75% of patients 37% of patients represent with new VTs[3]		VA uncommon in structurally normal hearts Procedure may be difficult due to haemodynamic compromise, the presence of multiple tachycardia circuits of difficulty locating the origin of the arrhythmia circuit

Class I = conditions where there is general agreement that CA is indicated. Class IIA = situations in which the weight of evidence supports the use of CA
Adapted from: ACC/AHA/ESC guidelines, 2003

(–30°C) phase that provides an insight into the effect of permanent damage. If the location is safe then cryoablation (–70°C) creates permanent injury

- following ablation the EPS is repeated to determine the success of the procedure.

Complications

Table 1 summarises the indications, efficacy, and potential complications of CA. Complication rates vary depending on the arrhythmia being ablated and the experience of the operator, however the incidence of serious complications is approximately 1%.[5] Complications associated with EPS are unusual but include those related to:

- vascular access (hematoma, arteriovenous fistula, deep venous thrombosis, pneumothorax), and
- catheter manipulation and delivery of radiofrequency energy (cardiac perforation, damage to AV node, microemboli and coronary artery perforation or spasm).

Catheter ablation and supraventricular arrhythmias

Supraventricular arrhythmias

Catheter ablation is the preferred therapy over pharmacological treatment for the management of SVT. In general EPS and CA is best reserved for patients with documented tachycardia on electrocardiograph (ECG). Every effort should be made (ECG, continuous ambulatory monitoring) to demonstrate SVT before proceeding with EPS, although with infrequent symptoms this can be difficult.

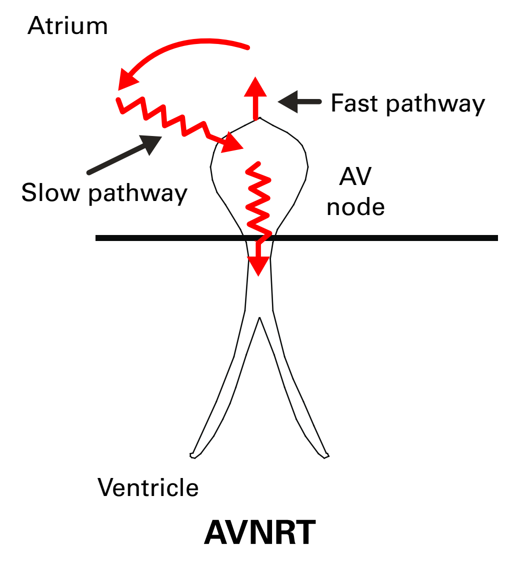

Figure 1. Mechanisms of AVNRT

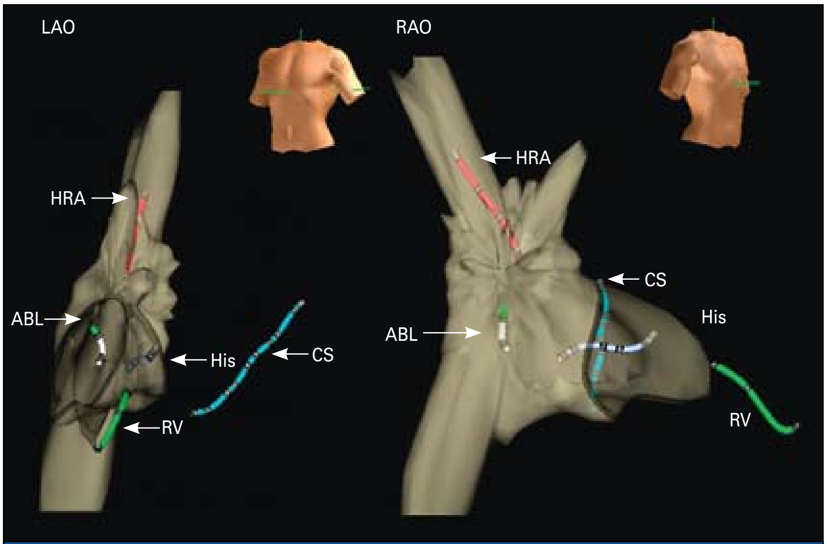

Figure 2. Right and left anterior oblique projections demonstrating standard multipolar catheter positions for an EPS using a newer nonfluoroscopic navigation system (NAVX). This allows catheters to be moved around the heart without using X-ray. The grey shell represents the right atrium
RA = right atrium, CS = coronary sinus, His = His bundle, RV = right ventricle

The most common electrophysiologic mechanism responsible for SVT is re-entry. This involves repetitive circus movement of an electrical impulse around a fixed obstacle. Differences in conduction properties in the two limbs of the circuit must exist for re-entry to occur. Therefore the strategy for CA involves modifying or blocking one limb of the circuit.

Supraventricular tachycardias can be subclassified as:

- AV nodal re-entrant tachycardias, and
- AV re-entrant tachycardias.

AV nodal re-enrant tachycardia

This is the commonest form of regular SVT and is a result of differences in the conduction properties of the slow and fast pathways that form the atrial inputs into the AV node (*Figure 1*). Current therapy involves modification or abolition of the slow pathway. Catheter ablation is associated with long term cure in 96% of patients, and recurrence in 3–5%.[6,7] The major complication is AV nodal damage requiring implantation of a permanent pacemaker in less than 1% of patients. Nonfluoroscopic mapping systems have been developed to reduce radiation exposure and provide multiple accurate views of catheter locations (*Figure 2*).

Atrioventricular re-entrant tachycardias

An accessory pathway is an extension of myocardium connecting the atrium and ventricle across the AV annulus. This contributes one limb of the re-entrant circuit with the AV node acting as the second limb (*Figure 3*). Accessory pathways are typically classified based on their location and on whether the pathway is capable of conduction in an antegrade (atrium to ventricle) or retrograde (reverse) direction. Wolff-Parkinson-White (WPW) syndrome is reserved for patients with pre-excitation and tachycardia. Atrial fibrillation can be life threatening in patients with WPW if the accessory pathway allows the rapid conduction of atrial impulses to the ventricle and the potential for ventricular fibrillation. However the incidence of sudden cardiac death in patients with WPW is 0.15–0.3%. Catheter ablation is targeted to the accessory pathway and is successful in 95% of patients.[8] Catheter ablation in asymptomatic patients with pre-excitation is controversial. Current American College of Cardiology/American Heart Association/European Society of Cardiology (ACC/AHA/ESC) guidelines recommend ablation in patients with high risk occupations, but these guidelines have not been updated following recent evidence supporting a role for electrophysiologic testing in risk stratification.[9] Referral to an electrophysiologist is recommended.

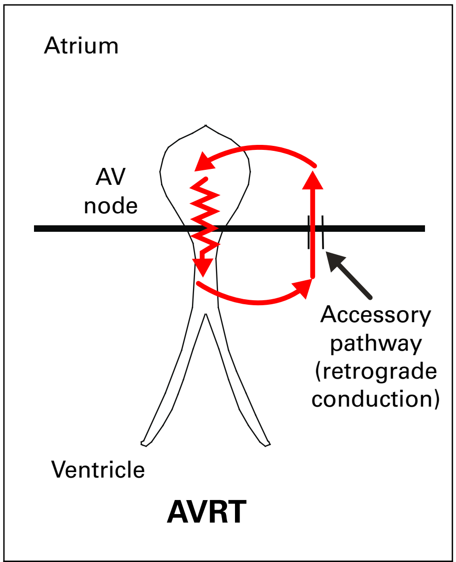

Figure 3. Mechanisms of AVRT

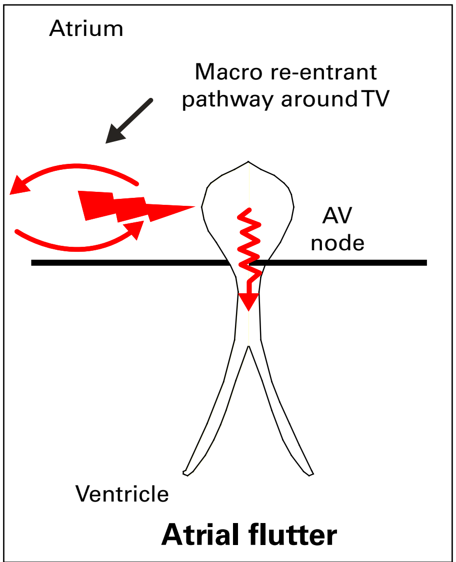

Figure 4. Mechanisms of atrial flutter

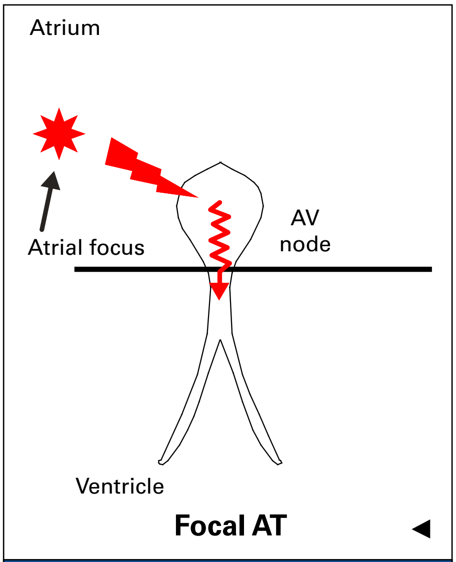

Figure 5. Mechanisms of focal AT

Atrial flutter (macro re-entrant atrial tachycardia)

Atrial flutter is due to a re-entrant circuit that involves a large part of the atrium and therefore is termed macro re-entrant AT (MAT) (*Figure 4*). The atrial flutter circuit is confined by anatomic boundaries,[10] part of which includes a narrow region of slow conduction defined as the isthmus, which is the target site for catheter ablation. The success of the procedure is not dependent upon the patient having atrial flutter at the time of the procedure.

Catheter ablation for typical atrial flutter has been shown to reduce recurrent arrhythmia, improve quality of life and reduce hospitalisation over pharmacological therapy.[11] Catheter ablation is successful in 90% of patients but with a later incidence of AF of up to 25%.[12] The risk of developing AF following successful CA for atrial flutter is determined by the coexistence of AF before ablation.

Focal atrial tachycardia

Patients with focal AT frequently present for CA as medical treatment is of limited efficacy. This group of arrhythmias is characterised by a rapidly firing focus that activates the surrounding atrium by radial spread (*Figure 5*). Foci tend to cluster at characteristic anatomic locations.[13] The P wave rate is usually slower (100–250 bpm) than in MAT although considerable overlap exists. Approaches to CA include a conventional approach using multipolar catheters and systematic point mapping of the atrium. These tachycardias may be difficult to induce making mapping difficult. Newer mapping systems (*Figure 6*) create three dimensional geometries that may improve success and reduce radiation exposure. The success rate for CA of focal AT is >90%.[14,15]

Atrial fibrillation

Atrial fibrillation has been described as an 'emerging epidemic'. Large multicentre studies examining the pharmacological management of AF demonstrated a nonsuperiority of rhythm control over rate control.[16] However these studies highlight the relative ineffectiveness of medication in maintaining sinus rhythm. Subsequent analysis has demonstrated that the group which maintained sinus rhythm showed improved survival.[17] With the advent of CA the opportunity to cure a proportion of patients with AF has become available.

Strategies for CA are based on seminal observations of Haissaguerre and coworkers in identifying the pulmonary veins (PV) as the major source of triggers responsible for the initiation of AF.[18] This has led to evolution in technique and technology which can successfully abolish AF in 70–80% of patients.[19] Results are best in patients with paroxysmal AF and structurally normal hearts. In brief, the technique involves transeptal puncture and the delivery of a series of radiofrequency lesions to electrically isolate the PV from the rest of the heart (*Figure 3*). Patients currently considered for CA include those with significant symptoms who have failed antiarrhythmic medication. The risk of serious complication is 1–2% and includes:

- stroke
- pericardial tamponade, and
- PV stenosis.

A repeat procedure is required in about 30% of patients, usually due to PV reconnection. Procedure time is 3–4 hours and requires an overnight stay in hospital. A similar procedure can also be done at surgery using a combined technique of 'cut and sew' and ablation. This is usually reserved for patients undergoing cardiac surgery for coronary revascularisation or valve replacement.

The alternative for elderly patients is to implant a permanent pacemaker and modify or abolish the AV node. This provides ventricular rate control but does not restore left atrial transport and the patient is often dependent on pacing for AV conduction. Furthermore, we are becoming increasingly aware that pacing the heart can result in heart failure.

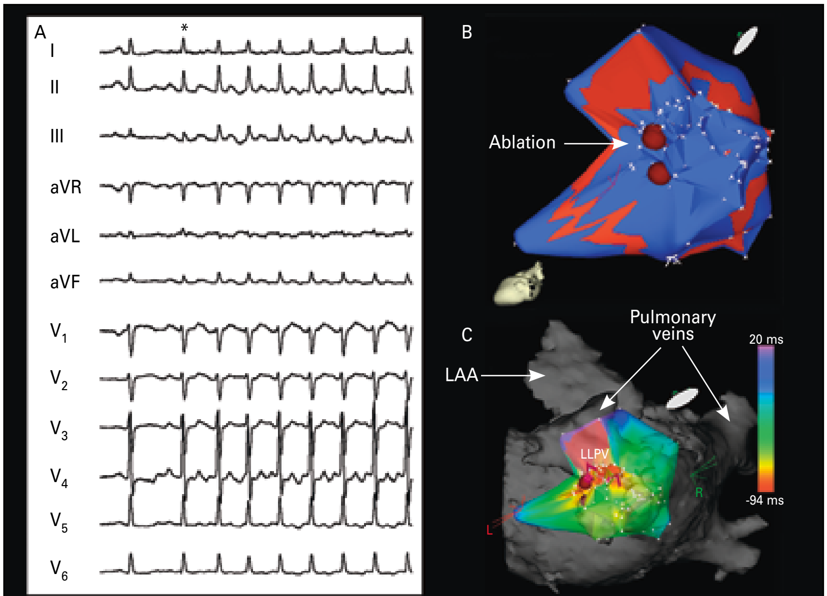

Figure 6. Panel A shows an ECG from a patient with a focal tachycardia. A distinct change in the P wave is demonstrated by the asterix. Panel B demonstrates a propagation map from the left atrium show the centrifugal spread from the region of the left pulmonary veins. Panel C shows an activation map superimposed on a CT scan of the left atrium with earliest activation in red ranging to late in blue as shown on the colour bar. The brown dots represent ablation points which successfully cured the patient

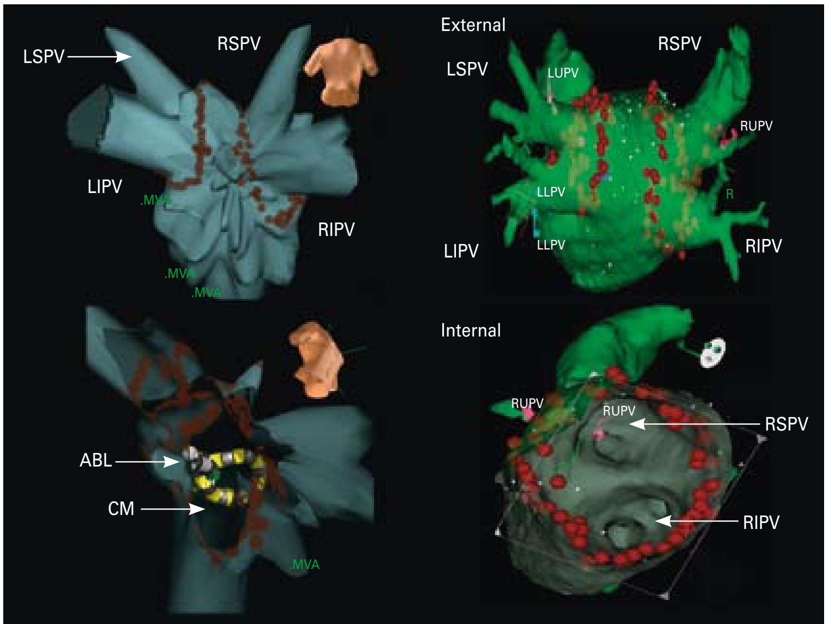

Figure 7. Catheter ablation for AF may be performed using 3D mapping systems. The left panel shows the noncontact and the right panel the contact mapping system with CT image integration Brown dots = ablation points, RSPV = right superior PV, RIPV = right inferior PV, LSPV = left superior PV, LIPV = left inferior PV

Ventricular arrhythmias

Ventricular arrhythmias can be classified as occurring in the presence or absence of structural heart disease. In the absence of structural heart disease, VA are uncommon but can be cured with focal ablation with high long term success. The responsible sites include the ventricular outflow tracts and the left hemifasicles of the conducting system.

Myocardial infarction is the most common heart disease associated with VT, although other cardiomyopathies may be responsible. Implantable cardioverter defibrillators (ICD) have significantly improved survival in patients with ischaemic and nonischaemic cardiomyopathy. These devices not only have the capacity to deliver a shock but can also cardiovert VT by rapid ventricular pacing (overdrive pacing). Patients with an otherwise reasonable life expectancy should be considered for an ICD if they have structural heart disease and have suffered VT or VF with no reversible cause. Survival of patients with poor left ventricular function (<35%) is also significantly improved by ICD even in the absence of previous VT or VF.[20] Although ICDs are effective in improving survival there is a small but significant morbidity related to implantation and therapies delivered by the device. Despite attempts at antitachycardia pacing shocks may be required to revert VA. Shocks may be very uncomfortable and psychological problems may arise, particularly if conscious patients receive multiple shocks. Catheter ablation is an important therapeutic adjunct in preventing or reducing recurrent VA. Acute success is achieved in up to 82% of patients with long term success in about 75%; however 37% of patients represent with new VTs.[3] Difficulties with CA of postinfarct VT include hemodynamic intolerance of the arrhythmia, multiple tachycardia circuits and identification of the arrhythmia substrate.

Conclusion

Catheter ablation of cardiac arrhythmias offers long term cure in SVT and should be considered as first line therapy. Catheter ablation can successfully restore sinus rhythm in patients with AF and should be considered in symptomatic patients not controlled on medical therapy.

Summary of important points

- The pharmacologic management of cardiac arrhythmias has limited efficacy and may be associated with significant morbidity.

- EPS involves induction of the arrhythmia and determination of tachycardia mechanism. Catheter ablation targets a critical portion of the re-entrant circuit or the site of focal activation.
- CA of cardiac arrhythmias offers long term cure in approximately 95% of patients with SVT with low complication rates.
- CA can successfully restore and maintain sinus rhythm in patients with AF and should be considered in symptomatic patients not controlled on medical therapy but before the initiation of amiodarone.
- VT in patients with no structural heart disease is readily treated with CA.
- CA is an effective adjunct to therapies from an ICD in patients with VT postmyocardial infarction.

Conflict of interest: none declared.

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