Non-pharmacological treatment of arrhythmias

This article reviews the role of electrophysiological study and catheter ablation for the management of supraventricular and ventricular tachycardias. Catheter ablation is playing an increasingly important role in the management of patients with cardiac arrhythmias.

Pharmacological treatment of cardiac arrhythmias has limited efficacy and may be associated with serious morbidity including proarrhythmia. Catheter ablation (CA) offers curative treatment with very low complication rates for supraventricular tachycardia (SVT) (Jackman et al, 1991, 1992; Scheinman et al, 2000) and ventricular tachycardia (VT) in patients with structurally normal hearts. Strategies for CA of atrial fibrillation (AF) and infarct-related VT (Schilling et al, 2005; Segal et al, 2005) are developing rapidly.

Electrophysiological study and catheter ablation

Electrophysiological study (EPS) and CA is a minimally invasive procedure which can be performed as a day case under light sedation. Venous access is required using multiple sheathes and multipolar electrodes are positioned at various key locations (typically right atrium, coronary sinus, His bundle and right ventricle). Next an EPS is performed using programmed stimulation and attempts are made to induce tachycardia. Using the electrical information obtained from the diagnostic multipolar electrodes and pacing manoeuvres the arrhythmia mechanism is determined. Next, a steerable mapping/ablation catheter is positioned at a critical limb of the circuit and radiofrequency energy is delivered, which heats up the tissue resulting in cell death and scar formation. This often involves destruction of just a few millimetres of cardiac tissue which acts as a 'circuit breaker' or an electrical barrier. Occasionally during energy delivery the patient may experience a warmth or discomfort in the chest.

Freezing or cryoablation is also available and can produce a reversible lesion during the cryomapping (-30°C) phase. This is used to test the effect of permanent damage which is created with cryoablation (-70°C). It is currently predominantly used for arrhythmia circuits where energy delivery could compromise atrioventricular (AV) nodal function. A repeat EPS is performed after energy delivery to confirm that the arrhythmia can no longer be induced and the re-entrant circuit has been successfully ablated.

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Complications associated with EPS are rare but mainly relate to vascular access (haematoma, 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). Complication rates vary depending on the arrhythmia being ablated and the operator's experience, but the incidence of serious complications is about 1% (Calkins et al, 1999).

Supraventricular arrhythmias

Supraventricular arrhythmias are relatively uncommon with an estimated prevalence of 2.25 per 1000. CA is the preferred therapy over pharmacological treatment. Indications for CA of SVT include recurrent symptoms, haemodynamically intolerant tachycardia and presence of structural heart disease (Blomstrom-Lundqvist et al, 2003). The most common electrophysiological mechanism responsible for SVT is re-entry. This involves repetitive circus movement of an electrical impulse around a fixed obstacle. For re-entry to occur differences in conduction properties in the two limbs of the circuit must exist (*Figure 1*). Therefore the strategy for CA involves energy delivery to one limb of the circuit rendering it electrically inactive.

SVTs can be subclassified as AV nodal re-entrant tachycardias. This is the commonest form of regular SVT, caused by different conduction properties of the slow and fast pathways which form the atrial inputs into the AV node (*Figure 1*). Initially CA targeted the fast pathway but this has been largely abandoned because of unacceptably high risks of AV block. Current therapy involves modification or abolition of the slow pathway. This can be performed as a day case procedure, effects long-term cure in 96% of cases and has a complication rate of 0–2% (Scheinman and Huang, 2000; Schilling, 2002). The major complication is AV nodal damage requiring implantation of a permanent pacemaker in <1% of patients.

Atrioventricular re-entrant tachycardias

This form of SVT typically involves an accessory pathway, an extranodal extension of myocardium connecting the atrium and ventricle across the AV annulus, as one limb of the re-entrant circuit with the AV node acting as the second limb. Accessory pathways are typically classified based on their location around the tricuspid and mitral annulus and also 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. AF can be life threatening in patients with WPW syndrome if the accessory pathway allows the rapid conduction of atrial impulses to the ventricle and subsequent development of ventricular fibrillation. However, the incidence of sudden cardiac death in patients with WPW is low, at between 0.15 and 0.3%. CA is targeted to the accessory pathway and is successful in 95% of cases (Calkins et al, 1992). CA in asymptomatic patients with pre-excitation is controversial. The current American College of Cardiology/American Heart Association/European Society of Cardiology guidelines recommend CA in patients with high-risk occupations but have not been updated following recent evidence supporting a role for electrophysiological testing in risk stratification (Pappone et al, 2003). The authors currently recommend that asymptomatic WPW only be ablated if tachycardia or AF with a fast ventricular response is initiated at EPS.

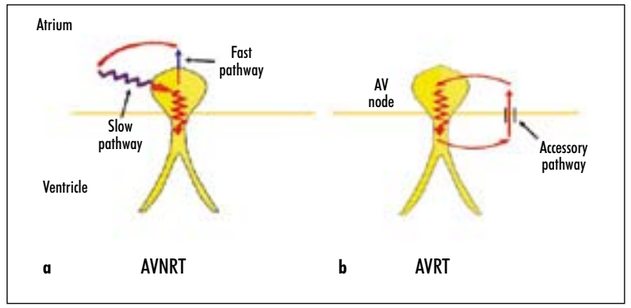


Figure 1. a. A schematic representation of the mechanism of atrioventricular nodal re-entrant tachycardia (AVNRT). Patients with AVNRT have dual atrial inputs into the atrioventricular (AV) node. The small re-entrant circuit involves slow and fast pathways and intervening atrial tissue but the ventricle is not part of the circuit. b. A schematic representation of the mechanism of atrioventricular re-entrant tachycardia (AVRT). The impulse typically travels from atrium through the AV node to the ventricle then back to the atrium via the accessory pathway (orthodromic tachycardia). The reverse can also occur (antidromic tachycardia).

Atrial flutter (macro re-entrant atrial tachycardia)

Atrial flutter and tachycardia have been reclassified to incorporate improved understanding of the responsible arrhythmia mechanisms. Atrial flutter occurs as a result of a re-entrant circuit which involves a large part of the atrium and is termed macro re-entrant atrial tachycardia (MAT). In contrast the arrhythmia previously referred to as atrial tachycardia (AT) is termed focal AT as the arrhythmia is a result of a single focus which activates the surrounding atrium by centrifugal spread. For the benefit of the non-electrophysiologist, MAT is referred to as atrial flutter.

The circuit is confined by anatomical boundaries, including a narrow region of slow conduction called the isthmus (*Figure 2*). The isthmus is the target site for CA and requires a series of ablation lesions to produce an electrically inactive line which transects the circuit and prevents further electrical spread.

The recurrence rate of atrial flutter on pharmacological therapy is up to 60%. Class Ic drugs, e.g. flecanide, can paradoxically worsen symptoms by slowing conduction in the flutter circuit allowing time for the AV node to recover and conduct 1:1 to the ventricle. A randomized study has demonstrated the superiority of CA over pharmacological therapy for typical atrial flutter (Natale et al, 2000). Atrial flutters are considered typical if the re-entrant circuit involves the isthmus between the inferior vena cava and the tricuspid annulus and ablation is successfully performed by delivering a series of lesions between these two structures to create a line of conduction block. This is successful in 95% of cases but with a later incidence of AF of up to 25% (Paydak et al, 1998). Most typical atrial flutters can be abolished by CA under local anaesthetic in a 30-minute day case procedure with a very low complication rate and thus there are very few circumstances when a patient should not be offered this treatment.

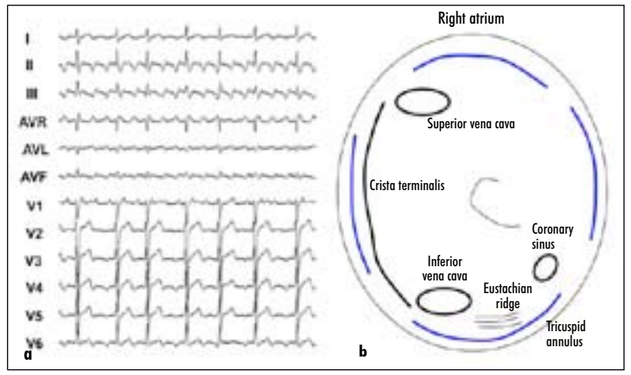


Figure 2. a. Typical 'saw tooth' pattern of atrial flutter best appreciated in leads II, III and AVF. b. A schematic representation of the typical atrial flutter circuit, looking through the tricuspid valve into the atrium. The flutter wave front passes around the tricuspid annulus on one side and a combination of the crista terminalis and vena cavae on the other. Part of this circuit involves the wave front travelling between the inferior vena cava and the tricupid annulus, which is the narrowest part of the circuit and therefore the target for catheter ablation.

Focal atrial tachycardia

Focal AT is characterized by a rapidly firing focus which activates the surrounding atrium by radial spread. Foci tend to cluster at characteristic anatomical locations, e.g. the crista terminalis (Kalman et al, 1998), tricuspid annulus and ostium of the coronary sinus in the right atrium and the pulmonary veins (PVs) and mitral annulus in the left atrium (Kistler et al, 2003). The P wave rate is usually slower (100–250 beats per minute) than in MAT although there is considerable overlap. Patients with focal AT often present for CA as medical treatment is of limited effect. CA involves a conventional approach using multipolar catheters and systematic point mapping of the atrium searching for early activation ahead of P wave onset.

These tachycardias are difficult to induce artificially and may have multiple origins. Newer tools such as electro-

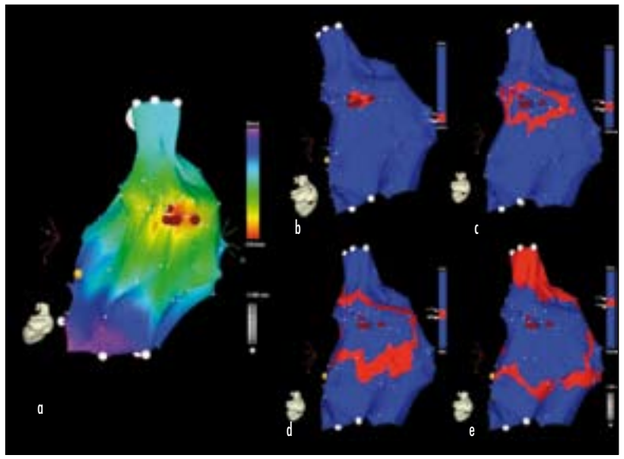
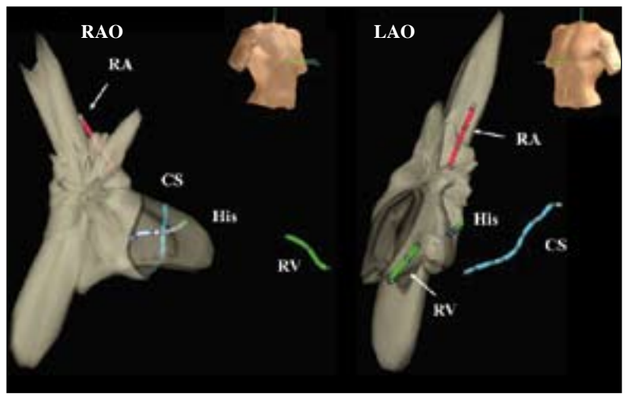


Figure 3. Electroanatomical maps from a patient with a focal atrial tachycardia arising from the crista terminalis. Point by point mapping is used to create a three-dimensional geometry of the atrium during tachycardia. The right atrium is viewed from the back. (a) demonstrates an activation map with earliest activation in red ranging to late in blue as shown on the colour bar. (b–e) represent a propagation map. In frame b the red represents initial activation and is seen to spread radially from frames c to e. The brown dots represent ablation points which successfully cured the patient.

anatomical (*Figure 3*), navigation (NAVX, *Figure 4*) and non-contact mapping systems create three-dimensional (3-D) geometries which may improve success and reduce radiation exposure. Non-contact mapping allows global, simultaneous mapping of an entire cardiac chamber. This is presented on a 3-D reconstruction of the atrium and can be helpful, particularly if the arrhythmia is infrequent or non-sustained. The success rates for CA of focal AT are 96% (Kalman et al, 1998; Kistler et al, 2003).

Figure 4. Right (RAO) and left anterior oblique (LAO) projections showing standard multipolar catheter positions for an electrophysiological study using a non-fluoroscopic navigation system (NAVX). This allows catheters to be moved around the heart without using X-ray. The grey shell represents the right atrium (RA) in regions where the mapping catheter has been manipulated. CS = coronary sinus; His = His bundle; RV = right ventricle.



Atrial fibrillation

AF is the commonest sustained arrhythmia and increases with age. Large multicentre studies examining the pharmacological management of AF demonstrated a non-superiority of rhythm control (with cardioversion) over rate control (Wyse et al, 2002). However, many patients failed to remain in sinus rhythm and most importantly anticoagulation was probably stopped prematurely so that patients who reverted to AF late after cardioversion had a high incidence of stroke. Subsequent analysis has demonstrated that the group which maintained sinus rhythm showed improved survival (Corley et al, 2004). The poor results of drug treatment for maintaining sinus rhythm contrast with the phenomenal progress with CA strategies to restore and maintain sinus rhythm.

There are two approaches to CA treatment. The first 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, it is becoming increasingly evident that pacing the heart can result in heart failure.

The second approach is based on seminal observations of Haissaguerre et al (1998) identifying the PVs as the major source of triggers which initiate AF. This has led to an evolution in technique and technology which can successfully abolish AF in 95% of patients with paroxysmal (Ouyang et al, 2004) and 76% of patients with permanent AF (Earley et al, 2005). The techniques involve delivering a series of radiofrequency lesions to encompass the PVs, electrically isolating them from the rest of the heart.

Patients considered for CA include those with significant symptoms who have failed antiarrhythmic medication. The risk of serious complication is between 1 and 2% and includes stroke, pericardial tamponade and pulmonary vein stenosis. A repeat procedure is required in about 30% of cases as a result of arrhythmia recurrence. The procedure takes 3–4 hours and requires an overnight stay. Patients require anticoagulation for at least 4 weeks before and at least 3 months after CA. A similar procedure can also be done at surgery and has outstanding results. This is particularly useful for patients undergoing other cardiac surgery and can often restore sinus rhythm even in patients with AF secondary to valvular heart disease.

Ventricular arrhythmias

Ventricular arrhythmias (VA) occur 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 strategies with high long-term success. The responsible sites include the ventricular outflow tracts (right more common than left) and the left hemifascicles of the conducting system.

The most common heart disease associated with VT is a myocardial infarct but other cardiomyopathies may also be responsible. Implantable cardioverter defibrillators (ICD) have significantly improved survival in patients with ischaemic and non-ischaemic cardiomyopathy. These devices not only have the capacity to deliver a shock but can also cardiovert some VT by rapid ventricular pacing (overdrive pacing). Patients with no other pathology that would significantly limit their life should be offered an ICD if they have structural heart disease and have suffered VT or VF with no reversible cause (secondary prevention). Survival of patients with poor left ventricular function (<35%) is also significantly improved by ICD even in the absence of previous VT or VF (Bardy et al, 2005) because these patients are at high risk of sudden arrhythmic death. It is important to remember that ICDs do not prevent VT or VF, they simply prevent most patients dying from it.

Although overdrive pacing is particularly useful in patients with slow haemodynamically stable VT, shocks can be very uncomfortable and psychological problems may be associated with ICDs particularly if the patient is getting frequent VT episodes. Therefore CA is an important therapeutic adjunct. Acute success is achieved in up to 82% of cases with long-term success in around 75%, however, 37% of patients represent with new VTs indicating that ablation is not sufficient to protect against sudden cardiac death (Segal et al, 2005). Difficulties with CA of post infarct VT include haemodynamic intolerance of the arrhythmia, multiple tachycardia circuits and identification of the arrhythmia substrate.

Conclusions

The pharmacological management of cardiac arrhythmias is palliative and may be associated with significant morbidity. CA of cardiac arrhythmias offers long-term cure in SVT and can also cure or palliate patients with VT. Patients with structural heart disease and VT also need an ICD to reduce the risk of sudden death. CA can successfully restore sinus rhythm in patients with AF and should be considered in symptomatic patients not controlled on medical therapy. BJHM

Conflict of interest: none.

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KEY POINTS

- The pharmacological management of cardiac arrhythmias is palliative and may be associated with significant morbidity.
- Electrophysiological study involves induction of the arrhythmia and determination of the tachycardia mechanism using activation characteristics on multipolar electrodes.
- Catheter ablation (CA) targets a critical portion of the re-entrant circuit or the site of focal activation.
- CA of cardiac arrhythmias offers long-term cure in >95% of patients with regular supraventricular tachycardia.
- CA 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. It may be performed as a day case with low complication rates.
- Ventricular tachycardia (VT) in patients with no structural heart disease is readily treated with CA.
- The frequency of symptoms related to VT associated with structural heart disease may be reduced by CA but implantable defibrillators are the only therapy proven to reduce risk of sudden death in these patients.