Electrophysiology and ablation of arrhythmias

Major advances in diagnosis and treatment of arrhythmias have created the subspeciality of cardiac electrophysiology. This article reviews supraventricular and ventricular arrhythmias and outlines the indications and process of electrophysiological testing, arrhythmia mechanisms and their treatment by catheter ablation.

Arrhythmias are a common clinical problem and any physician will have to diagnose and manage them in routine clinical practice. Pharmacological treatment is purely palliative and suffers from problems of poor efficacy and major side effects, including pro-arrhythmia. We are now in an era of potentially curative therapies using catheter ablation which has evolved from intraoperative mapping and ablation techniques. This review summarizes the current catheter-based ablation strategies used to treat common arrhythmias. Atrial fibrillation is beyond the remit of this concise review as this is a complex arrhythmia with several different underlying mechanisms and many evolving treatment strategies, and will therefore be the subject of a further review.

A brief historical perspective of clinical electrophysiology

In the early 20th century Mines (1914) demonstrated that ring-like preparations of jellyfish tissue, under the right conditions, could sustain a continuous electrical excitation wave in a never-ending circuit: a re-entrant 'circus' movement. This mechanism accounts for common clinical arrhythmias including atrial flutter, atrioventricular nodal re-entrant tachycardia, atrioventricular re-entrant tachycardia, atrial tachycardia and ventricular tachycardia. Apart from re-entry, the two other major mechanisms of tachycardia are automaticity (relating to frequent spontaneous impulse generation from tissue), and triggered activity (relating to abnormal ion currents within cells).

Despite this understanding of arrhythmia mechanisms, it was five decades later before any curative treatment could be contemplated or performed requiring the development of programmed electrical stimulation of the heart (Wellens, 2004). By providing electrical stimuli within the heart that were critically timed, tachycardia could be reproducibly induced and terminated. Recording of activation times from catheters placed in regions of the cardiac chambers allowed localization of the tachycardia circuit.

In 1968, Cobb et al (1968) cured a patient with Wolff–Parkinson–White syndrome by surgically interrupting the accessory connection between the atria and ventricle. Other surgical procedures were used to excise abnormal ventricular myocardial sites that were shown to be driving ventricular tachycardia (Josephson et al, 1979), or to create atrial incisions to prevent atrial fibrillation (Cox et al, 1989).

Curative catheter ablation strategies soon followed these surgical approaches with the development of ablative energy that could be delivered at local myocardial sites. High-energy shocks were originally given to treat accessory pathways (Weber and Schmitz, 1983), atrial flutter (Saoudi et al, 1987) and ventricular tachycardias (Fontaine et al, 1985). However, radiofrequency energy was soon successfully developed to provide a safe cure for various arrhythmias (Jackman et al, 1991), making this the treatment of choice in most common arrhythmias. Other energy sources have since been developed, including cryo (freezing), microwave, high-intensity focussed ultrasound and laser energy, but radiofrequency energy remains the most commonly used energy source, and has largely replaced surgery as treatment of choice for most arrhythmias.

The low efficacy of antiarrhythmic drugs, together with frequent side effects including pro-arrhythmia, has led to suboptimal arrhythmia management for decades. The advent of curative catheter ablation techniques, obviating the need for further drugs in some cases, has led to improved long-term patient outcomes. As can be seen from *Table 1*, most forms of supraventricular tachycardia can be cured in 95% of patients with a single procedure making it essentially first-line therapy for these patients who would otherwise require long-term antidysrhythmics, a particular issue for young patients and women of childbearing age.

Classification of arrhythmias

Arrhythmias can be classified into ventricular or atrial, and regular or irregular. Ventricular tachycardia is typically regular, usually occuring in patients with previous myocardial infarction, and may be amenable to a curative catheter ablation approach. Ventricular fibrillation

Dr P Boon Lim *is Senior Electrophysiology Fellow,* **Mr Daniel Robb** *is Senior Cardiac Physiologist and* **Dr Pier D Lambiase** *is Senior Lecturer and Consultant Cardiologist, The Heart Hospital, University College London Hospitals NHS Trust, London W1G 8PH*

Correspondence to: Dr PD Lambiase (pier.lambiase@uclh.nhs.uk)

Table 1. Success rates of treatment of arrhythmias by ablation

Arrhythmia	Ablation success rate
Atrial flutter	88–95%
Atrioventricular nodal re-entrant tachycardia	98%
Atrioventricular re-entrant tachycardia	90–99%
Paroxysmal atrial fibrillation*	70–80%
Persistent atrial fibrillation*	50–70%
Atrial tachycardia	80–90%
Ventricular tachycardia	50–88%
Ventricular ectopy	>80%

* indicates success rates with multiple ablation procedures. From Jackman et al (1991), Calkins et al (2007), Aliot et al (2009)

or Torsades de pointes is irregular and may be associated with inherited cardiac syndromes including arrhythmogenic right ventricular cardiomyopathy, hypertrophic cardiomyopathy, long QT and the Brugada syndrome.

Regular atrial, or supraventricular arrhythmias consist of atrial flutter, atrioventricular re-entrant tachycardia, atrioventricular nodal re-entrant tachycardia and atrial tachycardia. Regular supraventricular arrhythmias are amenable to catheter ablation with very high cure rates (~95%) and low complication rates (<1%). Atrial fibrillation is an irregular atrial rhythm, with varying success rates (35–85%) from ablation depending on the type and duration of atrial fibrillation, and other factors including left atrial size and presence of structural heart disease. This article will focus only on the non-atrial fibrillation arrhythmias.

The electrophysiological study and ablation technique

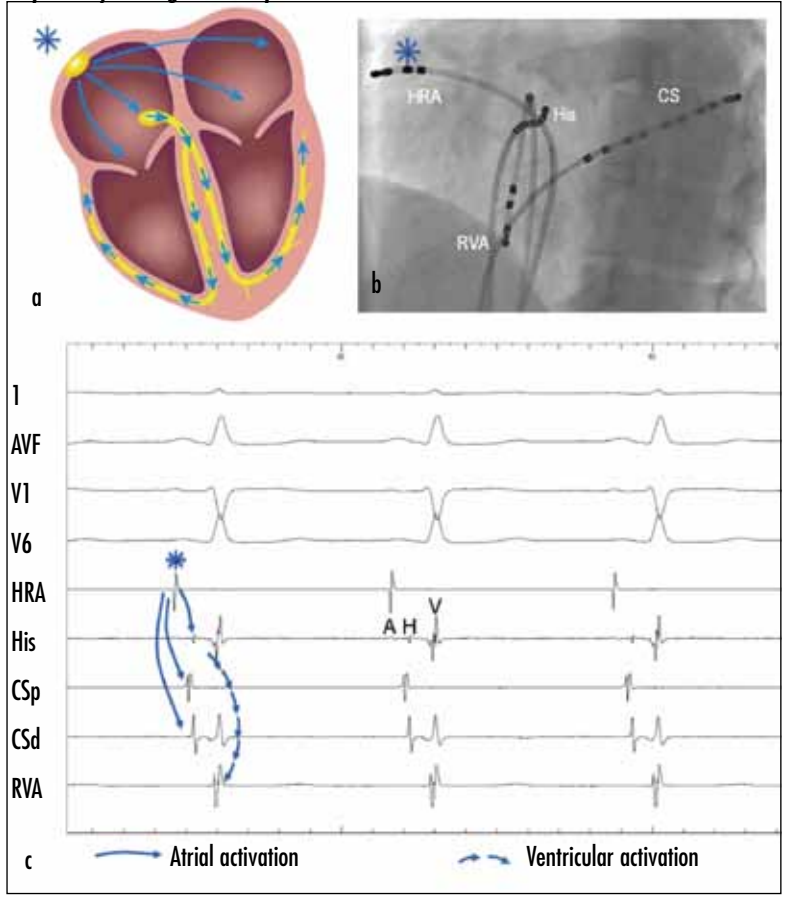
Electrophysiological studies and ablation procedures are performed in cardiac electrophysiology laboratories equipped with digital recording consoles, programmed electrical stimulators and ablators. Staff present during the case include a cardiologist, a cardiac physiologist, a radiographer and a nurse. Patients undergo the procedure either under general anaesthesia but more commonly under conscious sedation. Procedure durations vary, from 1–2 hours for treatment of supraventricular tachycardia, and between 2 and 5 hours for atrial fibrillation or ventricular tachycardia.

The electrophysiological study starts with the careful inspection of the 12-lead electrocardiogram during sinus rhythm and that of the tachycardia (very helpful if available). Up to four electrode catheters are positioned in the heart, usually through the femoral vein under fluoroscopic guidance. These catheters are routinely placed in the high right atrium, the His bundle, the coronary sinus and the right ventricular apex (*Figure 1*). Each catheter continuously records electrical activity from different regions of the heart. Programmed electrical stimulation is then performed to deliver critically-timed extra stimuli in an attempt to induce the tachycardia. The local activation times recorded in the various catheters are analysed to provide information about the mechanism of tachycardia. Pacing manoeuvres may be performed to further differentiate the mechanism of tachycardia and arrive at a diagnosis, ensuring appropriate targeting of the critical components of the circuit for ablation.

New mapping technologies

Electrophysiological mapping and ablation procedures can now also be facilitated by recently-developed three-dimensional electroanatomical mapping systems. Examples of such mapping systems include the CARTO (Biosense Webster) and Nav-X (St Jude Medical). The CARTO system comprises a miniature passive magnetic field sensor embedded in a roving ablation catheter, a reference patch,

Figure 1. Basic four-wire electrophysiological setup, with catheter electrodes placed in the high right atria (HRA), His position just below atrioventricular node (His), coronary sinus (CS) and right ventricular apex (RVA). a. Schematic drawing of the heart, with (b) a left anterior oblique projection of catheters placed within the cardiac chambers. c. Intracardiac electrograms, and surface electrocardiogram leads 1, AVF, V1 and V6. The atrial (A), His (H) and ventricular (V) inscriptions on the His catheter are shown in the second complex. The AH interval indicates conduction through the atrioventricular node, and the HV intervals represents infra-nodal conduction down the His–Purkinje system. * indicates sinus node depolarization, with atrial and ventricular activation shown respectively as long and multiple short arrows.



a low magnetic field emitter (placed underneath the patient on the catheter laboratory table) and a processing unit. The sensed changes in magnetic fields that occur during movement of the ablation catheter in the heart are tracked and displayed with a high temporal and spatial resolution, allowing accurate display of ablation catheter on the system. A three-dimensional shell of the cardiac chamber of interest can then be created, and electrogram data superimposed onto this shell to render an electroanatomical map of the chamber of interest.

In Nav-X, the principle of real-time catheter movement is similar. Three orthogonal patches that emit small electrical impulses are placed on the patient's chest, and these signals are detected by the catheters within the heart. The system then processes these signals to accurately display the catheter locations, which can be used to create three-dimensional shells of cardiac chambers containing electrical information. Typically, local electrical activity is determined in reference to a fixed electrode (e.g. in the coronary sinus), and displayed as a colour spectrum on this shell (e.g. in *Figure 2*).

Figure 2. Right atrial geometry in a right anterior oblique projection shown using a three-dimensional electroanatomical mapping system (Nav-X, St Jude Medical). Timing is displayed in the colour bar scale. a. In this example, the electrical activity is displayed as a colour map, with white indicating earliest, and purple indicating latest activation (colour bar). Black arrows indicate direction of spread of the activation wavefront. b. A focus of activity is seen to arise from the low anterolateral right atrium. In this example, ablation at this focus terminated atrial tachycardia, allowing sinus rhythm to resume. In (b), activity is seen to spread from the sinus node region in the high lateral right atrium to activate the rest of the right atrium.

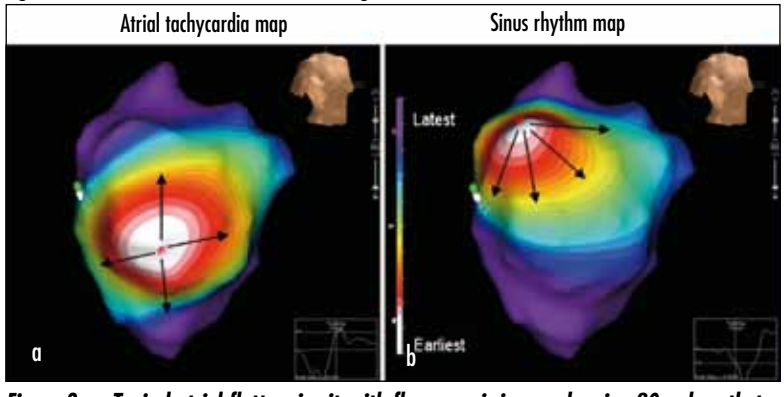
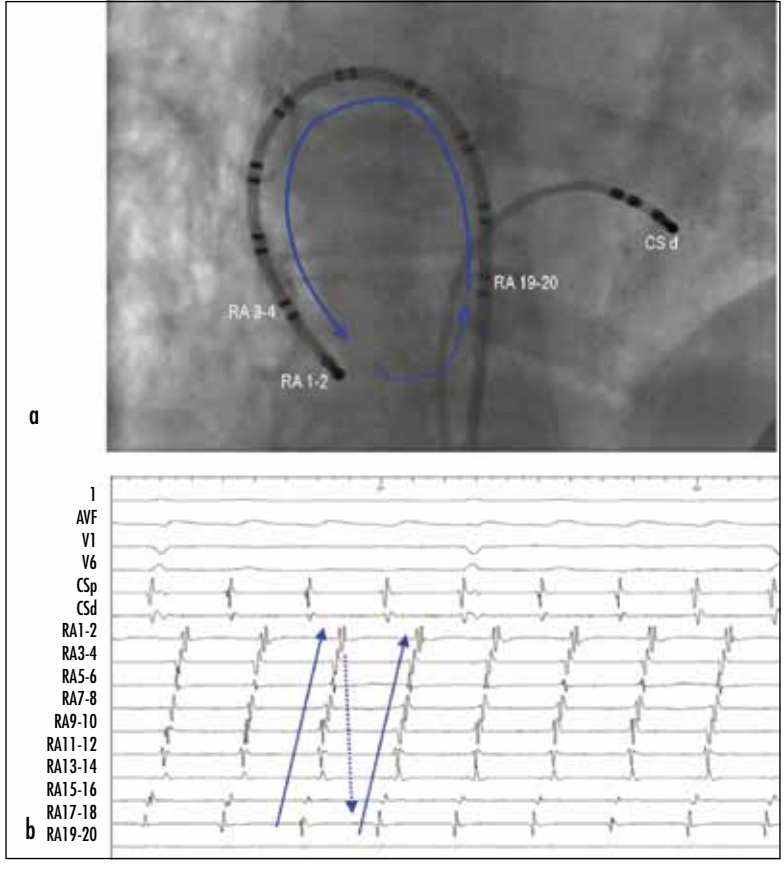


Figure 3. a. Typical atrial flutter circuit with fluoroscopic image showing 20-pole catheter placed in the right atrium (RA 1-20) and a quadripolar catheter placed within the coronary sinus (CS). The corresponding arrows shown on the both fluoroscopic image and (b) intracardiac electrograms indicate a counter-clockwise activation sequence of the flutter wavefront across the right atrium.



Ablation technique

Ablation is performed through a roving ablation catheter with up to 60 W of radiofrequency energy delivered between the tip of the catheter and a ground plate usually applied on the patient's back. The temperature and duration of energy delivery is adjusted to achieve thermal injury to the myocardial sites critical to arrhythmia perpetuation. Following energy delivery, the programmed electrical stimulation protocol (+/- pharmacological agents, e.g. isoprenaline) is repeated to confirm that tachycardia is no longer inducible.

Ablation for supraventricular arrhythmias

Atrial flutter

Typical atrial flutter, characterized by a saw-tooth baseline pattern on the electrocardiogram, is a result of a re-entrant circuit that is confined to the right atrium. *Figure 3* illustrates a typical flutter circuit with continuous activity seen in the corresponding electrograms from the multipolar catheter. Atrial flutter can be cured by transecting the circuit at the 'isthmus', which is a line in between the tricuspid annulus and the inferior vena cava. Success rates for atrial flutter are greater than 90%, with a complication rate of 1%.

Atrial tachycardia

Atrial tachycardia may have an underlying automatic, triggered or re-entrant mechanism. Inspection of the P wave on the surface electrocardiogram during atrial tachycardia can help localize the tachycardia's origin. Conventional mapping during tachycardia may reveal a focal source of earliest activation, which can then be ablated. The use of complex mapping systems allows better localization and ablation of focal atrial tachycardia, with success rates greater than 90%.

The site of earliest activation can be easily visualized and the ablation catheter can be guided to this site facilitated by the three-dimensional mapping system. *Figure 2*

gives an example of a right atrial three-dimensional geometry showing a focal atrial tachycardia from the low antero-lateral right atrium which was ablated to sinus rhythm.

An increasingly common type of atrial tachycardia is iatrogenic atrial tachycardia which can follow atrial fibrillation ablation procedures. Iatrogenic atrial tachycardias are typically created by incomplete ablation lines, creating gaps in which a scarred region of myocardium creates a zone of slow conduction, thereby setting up the substrate for re-entrant circuits. These forms of atrial tachycardia are very amenable to catheter ablation, typically guided by three-dimensional mapping systems to identify the circuit and the ideal ablation site to terminate tachycardia.

Atrioventricular re-entrant tachycardia

Atrioventricular re-entrant tachycardia, often termed Wolff–Parkinson–White syndrome, is characterized by a re-entrant circuit involving the following structures as part of the circuit: atria, atrioventricular node, ventricle and accessory pathway (*Figure 4*). Accessory pathways are located along the atrioventricular groove. Accessory pathways may be able to conduct antegradely (i.e. from atria to ventricle, which may manifest as a delta wave on the QRS complex during sinus rhythm), retrogradely (i.e. from ventricle to atria – a concealed pathway), or both. Latent pre-excitation refers to the presence of minimal evidence of the delta wave on surface electrocardiogram as the majority of conduction from the atrium to the ventricle occurs via the atrioventricular node making the delta wave difficult to see (fusion between atrioventricular nodal conduction and the accessory pathway).

The surface electrocardiogram in patients with a delta wave on the QRS complex may be used to localize the accessory pathway. A delta wave that is positive in lead V1 gives a right bundle–branch appearance (suggesting that the left ventricle activates before the right) which is consistent with a left-sided accessory pathway. Conversely, a negative delta wave in V1 suggests a right-sided accessory pathway. The appearance of the delta wave in the other electrocardiogram leads may help to further localize the accessory pathway by clarifying anterior or posterior and septal or free wall locations (Fitzpatrick et al, 1994).

An electrophysiological study is performed to localize the accessory pathway and determine its electrophysiological properties, which includes establishing pathway 'safety' before ablation is performed. Accessory pathways are associated with a small risk of sudden death, which is the result of pre-excited atrial fibrillation. In this life-threatening condition, the excitation waves (~ 500 bpm) during atrial fibrillation may conduct very rapidly to the ventricles through one or more accessory pathways with short anterograde refractory periods, causing ventricular fibrillation (Klein et al, 1979).

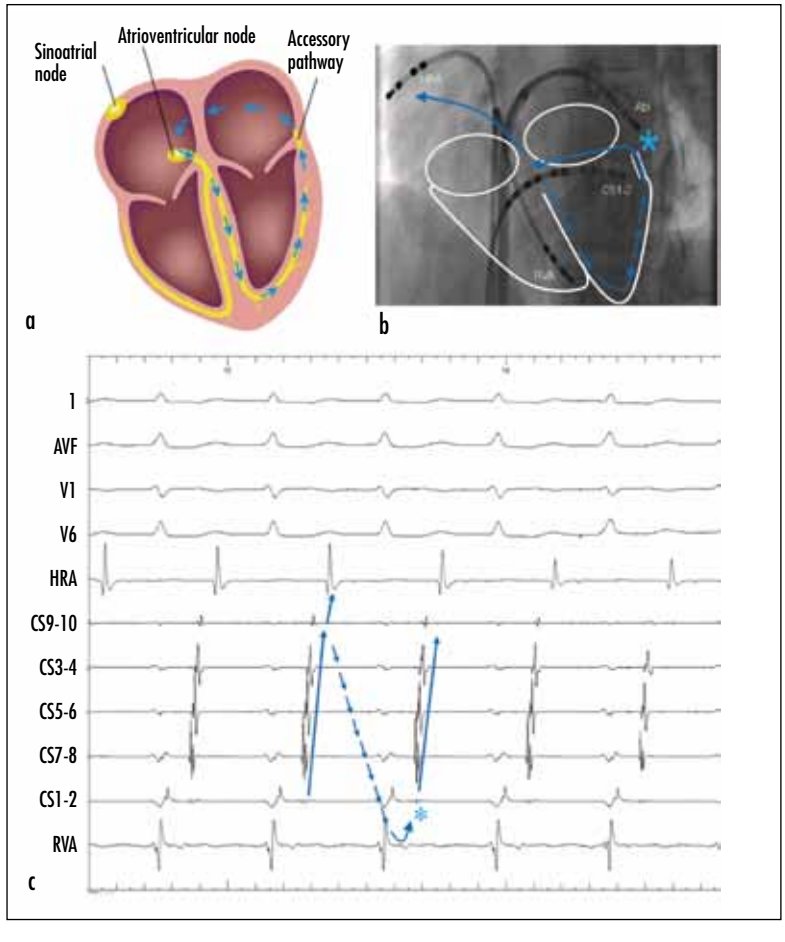
Figure 4 demonstrates how localization of the accessory pathway is performed. In this example, a catheter placed in the coronary sinus (which straddles the atrioventricular groove on the left) is used to localize an accessory pathway in the left lateral position. An ablation catheter is positioned at the site of the accessory pathway before radiofrequency energy is delivered to interrupt conduction through the pathway.

The success rates of catheter ablation for atrioventricular re-entrant tachycardia is between 90 and 99% (Jackman et al, 1991), with a complication rate of under 4%. Of note, there is a 1% rate of complete heart block with ablation of septal and antero-septal accessory pathways, because of their proximity to the atrioventricular node.

Atrioventricular nodal re-entrant tachycardia

Patients with atrioventricular nodal re-entrant tachycardia have two atrioventricular nodal pathways that are functionally distinct: a fast pathway (with a long refractory period, i.e. blocks conduction early with a prema-

Figure 4. Example of an electrophysiological study during orthodromic atrioventricular re-entrant tachycardia. a. A schematic drawing of the heart indicating a left lateral accessory pathway. b. The fluoroscopic image shows catheters positioned in the high right atrium (HRA), coronary sinus (CS), and right ventricular apex (RVA). An ablation catheter (Abl) is positioned at the site of the accessory pathway (*) to deliver radiofrequency energy which ablates the pathway. c. Intracardiac activation sequence during tachycardia showing retrograde activation of the atria in an eccentric manner, starting from the lateral wall of the left atrium (CS1–2) towards the septum and lastly the right atrium. The corresponding arrows shown on both fluoroscopic image and intracardiac electrograms indicate the direction of activation of the circuit.



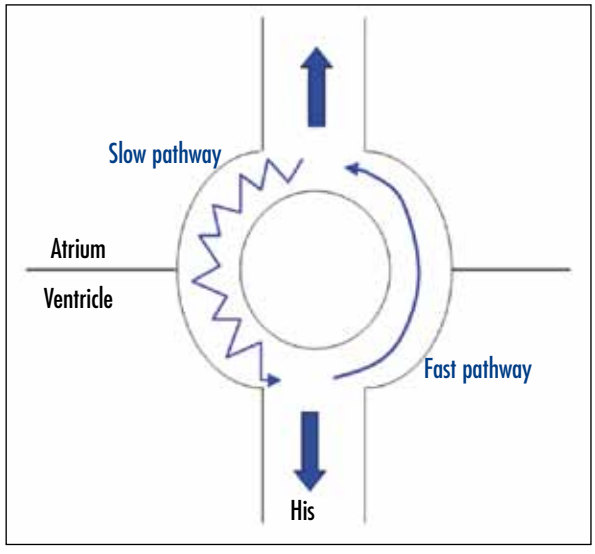
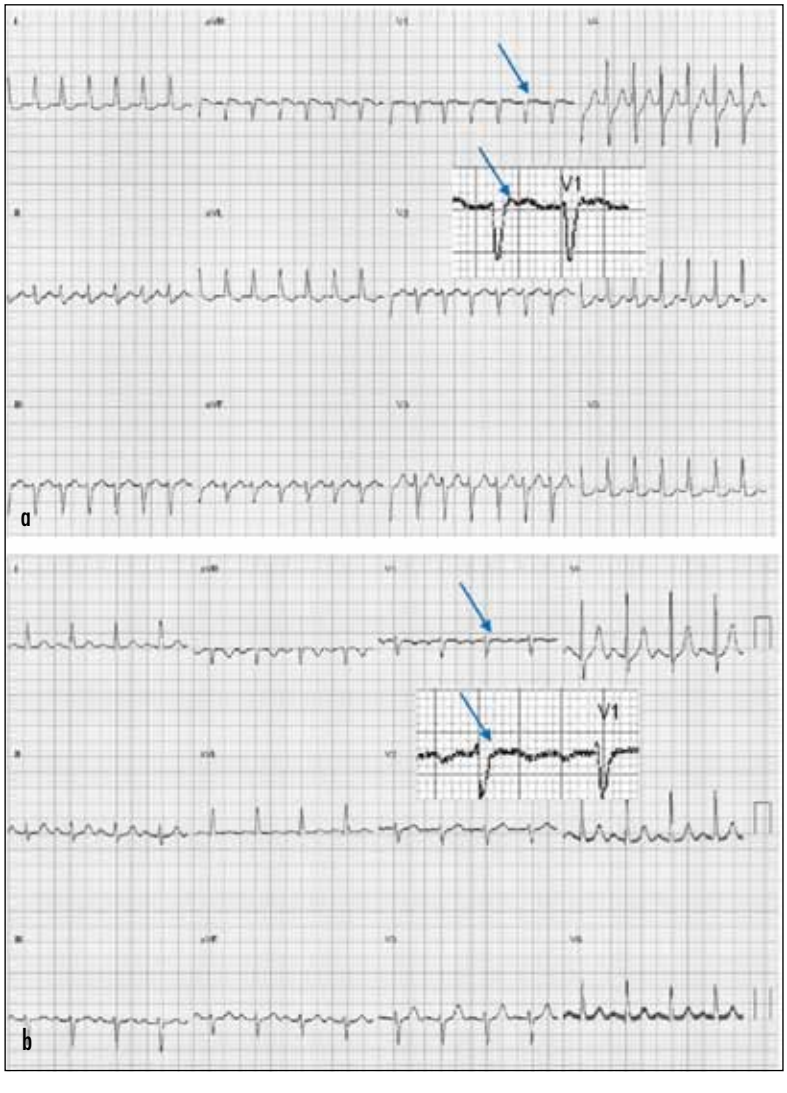


Figure 5. Schematic diagram of slow and fast pathways in the atrioventricular nodal region. The jagged arrows indicate delayed conduction down the slow pathway, which then activates the fast pathway retrogradely to complete a re-entrant circuit within the atrioventricular node.



ture beat) and slow pathway (with a short refractory period) (Wu and Zipes, 2002). Atrioventricular nodal re-entrant tachycardia is usually triggered by a premature atrial ectopic beat that encounters a refractory fast pathway, hence travels down the slow pathway and then retrogradely up the fast pathway (now recovered), simultaneously depolarizing the atria and ventricles (*Figure 5*). This simultaneous depolarization of atria and ventricles may give rise to a tachycardia electrocardiogram that shows a subtle rSr' pattern, particularly in lead V1, in which early retrograde atrial depolarization is inscribed at the tail-end of the QRS complex. Electrocardiograms taken during tachycardia are best compared with electrocardiograms during sinus rhythm to appreciate this difference (*Figure 6*).

Ablation of atrioventricular nodal re-entrant tachycardia is an anatomically-guided procedure, in which the slow pathway is ablated during sinus rhythm (Blomstrom-Lundqvist et al, 2003; Morady, 2004). Success rates of slow pathway ablation approach 98% with a negligible (<0.5%) risk of complete atrioventricular block needing pacemaker implantation (Morady, 2004). In some cases a cryoablation approach can be undertaken with no risk of complete heart block but also a slightly lower success rate (80–85%). This may be more suitable in young patients who are very anxious about the risk of permanent pacemaker implantation.

Brief overview of ablation for ventricular arrhythmias

Ventricular tachycardia

Recurrent ventricular tachycardia is most commonly found in patients who have underlying ischaemic heart disease, with an area of infarcted scar tissue serving as a substrate that allows a re-entrant circuit to develop within the peri-infarct area. It is widely accepted that most patients with poor left ventricular function and heart failure symptoms despite optimal medications should receive an implantable cardioverter defibrillator (Moss et al, 2002), but this does not prevent ventricular tachycardia from recurring. Indeed, the psychological burden of receiving repeated implantable cardioverter defibrillator shocks is extremely high. In this setting it may be appropriate to consider ablation for ventricular tachycardia.

The electrocardiogram during tachycardia gives vital clues to allow localization of the site of origin of ventricular tachycardia. Typically, a complex mapping system is used to define both the activation times during tachycardia, and voltage amplitudes of the signals in the chamber

Figure 6. Electrocardiogram appearance of atrioventricular nodal re-entrant tachycardia (a) during tachycardia and (b) during sinus rhythm. Arrows indicate the differences in the terminal portion of the QRS complex showing early retrograde atrial activation, making the diagnosis of atrioventricular nodal re-entrant tachycardia highly likely.

of interest to allow clear visualization of scar regions likely to be important in arrhythmogenesis (*Figure 7*). Advancements in imaging technology such as high-resolution magnetic resonance imaging scans have been helpful in identifying scar regions before the ablation procedure.

An ablation catheter is typically used to map the site which corresponds to the weakest link in the re-entrant circuit: the slowly-conducting peri-infarct isthmus. Programmed electrical stimulation (entrainment) is performed to confirm that this region is important in ventricular tachycardia perpetuation and radiofrequency energy is delivered to this site. When ventricular tachycardia is rapid and not well tolerated, an alternative strategy is to create transmural linear lesions from one site of scar to another, thereby transecting across the presumed re-entrant circuit. Success rates range from 50–88%, with incidence of major complications of up to 8% including cardiac tamponade, thromboembolic events or death (Aliot et al, 2009).

Ventricular ectopy

Ventricular ectopy can be highly symptomatic to patients. Ventricular ectopy that occurs frequently, either in single beats, couplets or short salvoes, can lead to impaired ventricular function and symptoms of heart failure in the long term.

The Holter monitor and 12-lead electrocardiogram allows quantification of ectopic burden and localization of the focus of ectopy. Right ventricular outflow tract ectopy is most common, and has a benign prognosis, with a very good response to ablation.

In right ventricular outflow tract ectopy, the ectopic beat appears to have a left bundle–branch appearance with positive QRS complexes in the inferior leads (II, III, AVF), indicating a high to low activation sequence that originates from the right ventricular base.

Ventricular ectopy is usually caused by an automatic or triggered focus. Therefore, mapping of this tachycardia involves localizing a site that has the earliest activation seen anywhere in the ventricles. An ablation catheter is moved around the site of interest until the earliest signal (compared to the ectopic QRS complex) is seen. Mapping can also be guided by complex navigation systems. Success rates for ablation of ventricular ectopy are generally >80% with a low complication rate of 1% (Ito et al, 2003; Vestal et al, 2003).

Ventricular fibrillation

Patients resuscitated successfully from ventricular fibrillation usually receive an implantable cardioverter defibrillator for secondary prevention. However, there are subsets of patients who have ectopic foci which occur in the vulnerable period during repolarization (i.e. causing an R on T phenomenon). In this instance, repeat ectopic foci appear to be triggers for ventricular fibrillation, and ablation of these foci may be helpful in preventing further ventricular fibrillation.

Conclusions

The understanding of arrhythmias has improved considerably since the advent of the electrophysiology study in 1967, paving the way for curative ablation techniques in the catheter laboratory. Simple arrhythmias, such as atrioventricular re-entrant tachycardia, atrioventricular nodal re-entrant tachycardia, atrial flutter and atrial tachycardia, can now be cured with an efficacy approaching 100%, and should be treatment of choice in these cases. Continued improvement in ablation and mapping technologies, as well as ongoing studies of mechanisms of arrhythmia, particularly in relation to atrial fibrillation and ventricular tachycardia, is likely to further enhance efficacy, minimize complication rates and expand the role of catheter ablation in heart rhythm management. **BJHM**

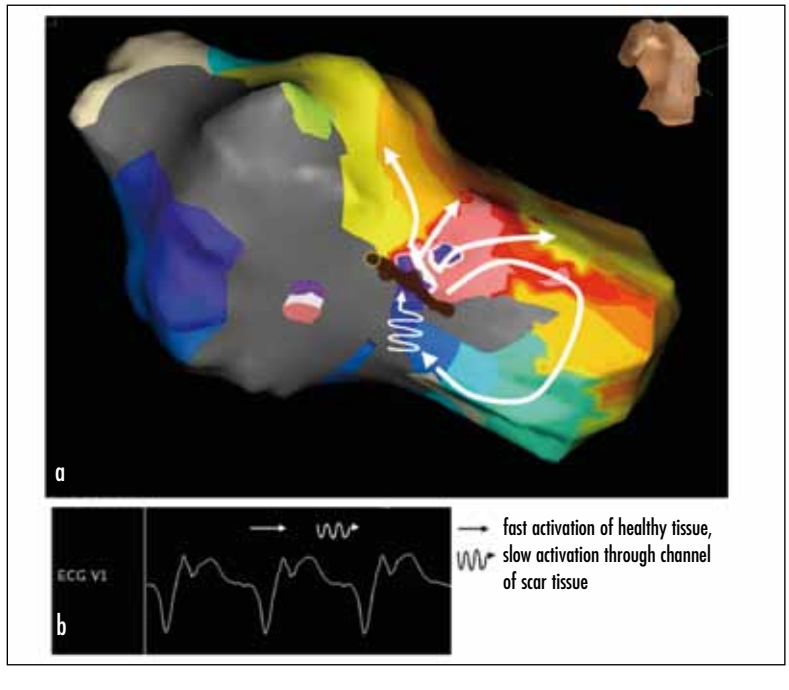
Dr PD Lambiase is supported by UCL/UCLH Biomedicine National Institute of Health Research.

Conflict of interest: none.

Aliot EM, Stevenson WG, Almendral-Garrote JM et al (2009) EHRA/HRS Expert Consensus on Catheter Ablation of Ventricular Arrhythmias: developed in a partnership with the European Heart Rhythm Association (EHRA), a Registered Branch of the European Society of Cardiology (ESC), and the Heart Rhythm Society (HRS); in collaboration with the American College of Cardiology (ACC) and the American Heart Association (AHA). *Heart Rhythm* **6**(6): 886–933

Blomstrom-Lundqvist C, Scheinman MM, Aliot EM et al (2003)

Figure 7. Left ventricular geometry seen in a right anterior oblique projection using a three-dimensional electroanatomical mapping system (Nav-X, St Jude Medical). a. The electrical activity is displayed as a colour map, with white arrows indicating the direction of the activation wavefront. Areas in grey represent low-voltage scar regions. In this example, a channel of slowly conducting scar-border tissue (indicated by the zig-zag arrow) forms the vulnerable link that perpetuates this reentrant ventricular tachycardia circuit. Linear ablation transecting this region (indicated by brown lesions) in this example terminated tachycardia. b. Lead V1 of surface electrocardiogram.



ACC/AHA/ESC guidelines for the management of patients with supraventricular arrhythmias--executive summary. a report of the American college of cardiology/American heart association task force on practice guidelines and the European society of cardiology committee for practice guidelines (writing committee to develop guidelines for the management of patients with supraventricular arrhythmias) developed in collaboration with NASPE-Heart Rhythm Society. *J Am Coll Cardiol* **42**(8): 1493–531

Calkins H, Brugada J, Packer DL et al (2007) HRS/EHRA/ECAS Expert Consensus Statement on Catheter and Surgical Ablation of Atrial Fibrillation: Recommendations for Personnel, Policy, Procedures and Follow-Up: A report of the Heart Rhythm Society (HRS) Task Force on Catheter and Surgical Ablation of Atrial Fibrillation Developed in partnership with the European Heart Rhythm Association (EHRA) and the European Cardiac Arrhythmia Society (ECAS); in collaboration with the American College of Cardiology (ACC), American Heart Association (AHA), and the Society of Thoracic Surgeons (STS). Endorsed and Approved by the governing bodies of the American College of Cardiology, the American Heart Association, the European Cardiac Arrhythmia Society, the European Heart Rhythm Association, the Society of Thoracic Surgeons, and the Heart Rhythm Society. *Europace* **9**(6): 335–79

Cobb FR, Blumenschein SD, Sealy WC, Boineau JP, Wagner GS, Wallace AG (1968) Successful surgical interruption of the bundle of Kent in a patient with Wolff-Parkinson-White syndrome. *Circulation* **38**(6): 1018–29

KEY POINTS

- A 12-lead electrocardiogram is important – 12-lead electrocardiogram recordings of tachycardia and sinus rhythm usually allows a definitive diagnosis to be made. This is vital to planning management.
- Drug therapy for supraventricular arrhythmias (atrial flutter, atrioventricular nodal re-entrant tachycardia, atrioventricular re-entrant tachycardia) generally fail to suppress symptoms adequately. Ablation for supraventricular arrhythmias has a high cure rate with a low complication rate.
- Patients with recurrent supraventricular tachycardias should be referred to a specialist arrhythmia centre for assessment for ablation.
- Patients with drug-refractory symptomatic ventricular tachycardia or ventricular ectopy should be referred for further assessment to a specialist arrhythmia centre.

Cox JL, Schuessler RB, Cain ME et al (1989) Surgery for atrial fibrillation. *Semin Thorac Cardiovasc Surg* **1**(1): 67–73

Fitzpatrick AP, Gonzales RP, Lesh MD, Modin GW, Lee RJ, Scheinman MM (1994) New algorithm for the localization of accessory atrioventricular connections using a baseline electrocardiogram. *J Am Coll Cardiol* **23**(1): 107–16

Fontaine G, Tonet JL, Frank R et al (1985) [Emergency treatment of chronic ventricular tachycardia after myocardial infarction by endocavitary fulguration]. *Arch Mal Coeur Vaiss* **78**(7): 1037–43

Ito S, Tada H, Naito S et al (2003) Development and validation of an ECG algorithm for identifying the optimal ablation site for idiopathic ventricular outflow tract tachycardia. *J Cardiovasc Electrophysiol* **14**(12): 1280–6

Jackman WM, Wang XZ, Friday KJ et al (1991) Catheter ablation of accessory atrioventricular pathways (Wolff-Parkinson-White syndrome) by radiofrequency current. *N Engl J Med* **324**(23): 1605–11

Josephson ME, Harken AH, Horowitz LN (1979) Endocardial excision: a new surgical technique for the treatment of recurrent ventricular tachycardia. *Circulation* **60**(7): 1430–39

Klein GJ, Bashore TM, Sellers TD, Pritchett EL, Smith WM, Gallagher JJ (1979) Ventricular fibrillation in the Wolff-Parkinson-White syndrome. *N Engl J Med* **301**(20): 1080–5

Mines GR (1914) On circulating excitation in heart muscles and their possible relation to tachycardia and fibrillation. *Trans Roy Soc Can* (8): 43–52

Morady F (2004) Catheter ablation of supraventricular arrhythmias: state of the art. *Pacing Clin Electrophysiol* **27**(1): 125–42

Moss AJ, Zareba W, Hall WJ et al (2002) Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. *N Engl J Med* **346**(12): 877–83

Saoudi N, Mouton-Schleiffer D, Letac B (1987) Direct catheter fulguration of atrial flutter. *Lancet* **ii**(8558): 568–9

Vestal M, Wen MS, Yeh SJ, Wang CC, Lin FC, Wu D (2003) Electrocardiographic predictors of failure and recurrence in patients with idiopathic right ventricular outflow tract tachycardia and ectopy who underwent radiofrequency catheter ablation. *J Electrocardiol* **36**(4): 327–32

Weber H, Schmitz L (1983) Catheter technique for closed-chest ablation of an accessory atrioventricular pathway. *N Engl J Med* **308**(11): 653–4

Wellens HJ (2004) Cardiac arrhythmias: the quest for a cure: a historical perspective. *J Am Coll Cardiol* **44**(6): 1155–63

Wu J, Zipes DP (2002) Mechanisms underlying atrioventricular nodal conduction and the reentrant circuit of atrioventricular nodal reentrant tachycardia using optical mapping. *J Cardiovasc Electrophysiol* **13**(8): 831–4