Supraventricular and ventricular arrhythmias: interventional management

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Abstract

Cardiac electrophysiology is the subspecialty of cardiology dealing with heart rhythm disorders, particularly the investigation and management of bradyarrhythmias and tachyarrhythmias, syncope and the prevention of sudden cardiac death. Recent technological developments include ever smaller and more complex implantable pacing devices, as well as the ability to map an arrhythmia circuit in three dimensions, potentially in one beat, and to safely destroy small areas of myocardium known to be critical to a particular arrhythmia. These have led to a dramatic expansion in ability to treat heart rhythm problems. This article focuses on some of the treatment strategies.

Keywords Ablation; arrhythmia; ECG; electrophysiological; Holter; MRCP; palpitation

Key points

- Ablation involves the localized destruction of a small amount of tissue to render it non-functional; radiofrequency energy is the most frequent energy source
- Almost all arrhythmias are now understood in terms of their electrophysiological mechanism and can be ablated successfully
- Ablation can be performed as a day-case procedure
- Structural heart ventricular tachycardia remains a complex arrhythmia; ablation forms part of the treatment strategy, as do implantable cardioverter-defibrillators and antiarrhythmic medications

Supraventricular tachycardia (SVT)[1]

An SVT is a rhythm that requires at least a part of the atrium for its mechanism. Regular SVTs comprise atrial flutter, atrial tachycardias and atrioventricular (AV) re-entry tachycardias (AVRTs). Although some SVTs relate to spontaneous activity in a group of atrial cells (focal automaticity or triggered activity), the mechanism underlying most SVTs is re-entry. In its simplest form, re-entry is the circulation of an electrical impulse around an obstacle in which the rotation time around the circuit should be longer than the recovery period of all segments of the circuit. In most circumstances, there is one 'pathway' that conducts rapidly and one that conducts more slowly, and it is this difference that creates the milieu for re-entry. This re-entry is initiated by ectopic beats. The specific anatomical re-entry circuit defines the optimal treatment.

SVT mechanisms (Figure 1)

The simplest form of SVT, mechanistically, is atrial flutter.[2] This involves a circuit where activation spreads counterclockwise around the tricuspid valve annulus. The slowly conducting 'pathway' is the narrow rim of tissue, the cavo-tricuspid isthmus, between the tricuspid valve annulus and inferior vena cava. Slowing of conduction in this area because of disease (e.g. ischaemia) or atrial enlargement (e.g. hypertension, atrial septal defect) increases the time the wave takes to travel and increases the likelihood of re-entry. On the electrocardiogram (ECG), the flutter waves appear as continuous negative saw-tooth waves in the inferior leads, and positive deflections in lead V1. The circuit spreads in the opposite direction in some patients, but the critical part of the circuit remains the same – the isthmus. Another simple re-entry SVT is AVRT. In this there are two conduction pathways connecting the atria to the ventricles: the fast conducting accessory pathway and the more slowly conducting AV node.

Atrioventricular nodal re-entry tachycardia (AVNRT) is the most common SVT in young people. The AV node is usually activated by two conduction pathways with differing properties – the fast and slow pathways. The fast pathway conducts quickly but recovers excitability slowly. The slow pathway conducts slowly but recovers more quickly. In a few people, these differences are sufficient to set up a re-entrant circuit around the AV nodal area.

Atrial tachycardias are more unusual; they can occur either as a result of re-entry around natural or iatrogenic scarring[3] or from focal activity caused by abnormal automaticity. The latter often arise from sites where atrial muscle joins to other tissue, for instance the pulmonary veins or vena cavae, perhaps because of fibre disarray.

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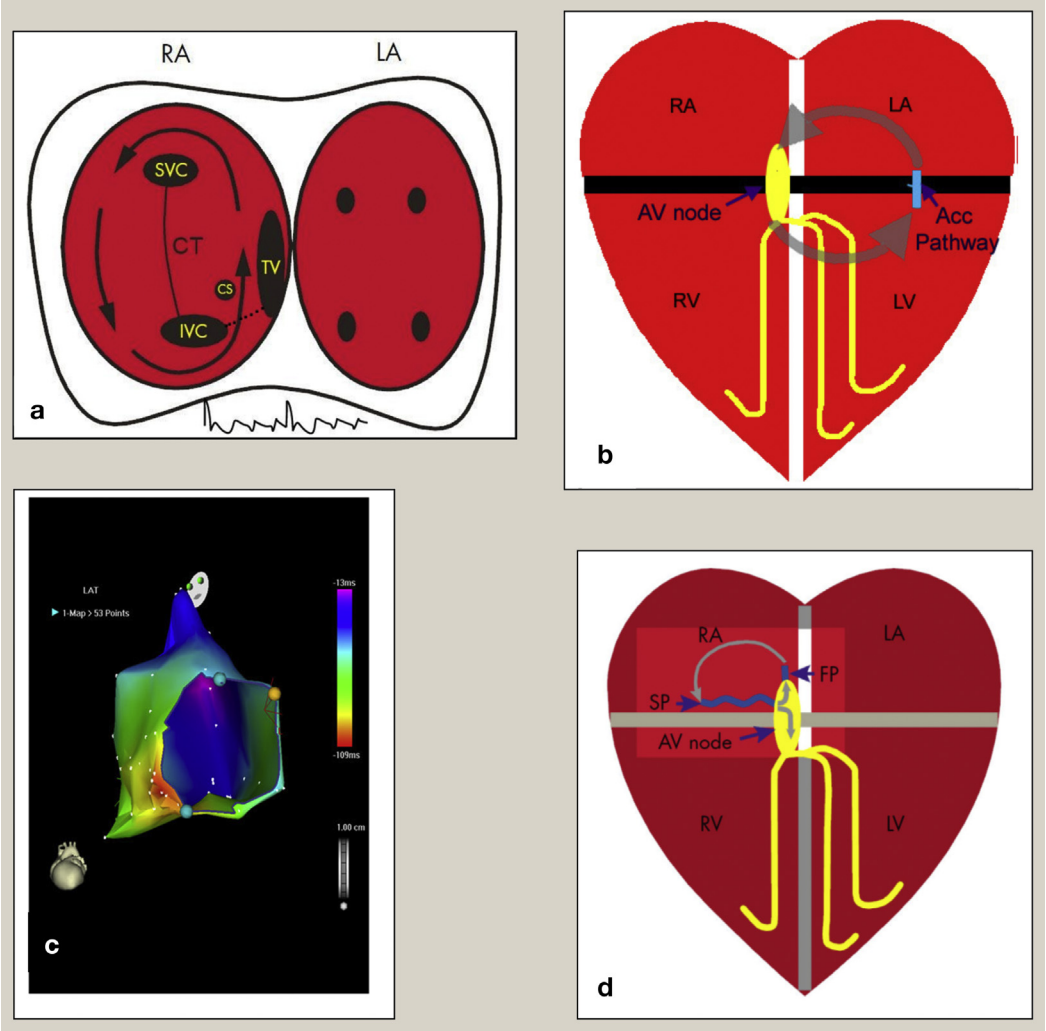

Figure 1 SVTs. (**a**) A schematic of classical atrial flutter circulating around the tricuspid valve annulus. The critical isthmus between the annulus and inferior vena cava (IVC) is the critical area for ablation (dotted line joining the tricuspid valve (TV) and IVC). An ECG showing the typical saw-tooth waves in the inferior leads is also shown. (**b**) A schematic of AVRT showing activation of the ventricle via the AV node producing a narrow QRS pattern and circulation back to the atrium via the accessory pathway. The fixed circuit length produces the regular tachycardia. (**c**) A three-dimensional electro-anatomical map of a focal atrial tachycardia from the low lateral TV annulus. Earliest activity is shown in red, with activation spreading out centrifugally indicated by the changing colours. (**d**) A schematic of AVNRT showing the circuit of slow pathway (SP) to fast pathway (FP) in the region of the AV node. Acc, accessory; CS, Coronary sinus; CT, Crista terminalis; LA, left atrium; LV, left ventricle; RA, right atrium; RV, right ventricle; SVC, superior vena cava.

SVT treatment

It is generally accepted that, for most regular SVTs, catheter ablation is the treatment of choice and can be offered as first-line therapy. It is more efficacious than antiarrhythmic drugs and also more cost-effective. Although untested in randomized trials, the safety of catheter ablation for regular SVT is probably similar to that of long-term antiarrhythmic drug therapy.

Catheter ablation for SVT ablation involves cauterizing the arrhythmia's source. Initially the use of catheter-delivered DC shocks to the AV node in patients with medically refractory SVTs were described. The use of radiofrequency energy and other technological developments have since led to the safe and effective therapy we have today. Ablation follows confirmation of a diagnosis during an intracardiac electrophysiology study, an invasive study in which catheters to measure electrical activity inside the heart are positioned in certain critical areas. Various forms of pacing manoeuvres are undertaken to assess the conducting system and to induce arrhythmias so that the mechanism can be studied. The critical part of the arrhythmia, either the focal source or the narrowest part of the circuit, is targeted so that selective damage will lead to a cure but will be unlikely to affect important local structures. Thus, for atrial flutter the isthmus is ablated, in AVNRT it is the slow pathway and in AVRT the accessory pathway. Success and complication rates are listed in Table 1.

As with electrophysiology studies, most procedures are performed as day cases. Antiarrhythmic medication is usually stopped at the procedure. Aspirin can be given for 4–6 weeks after the procedure to prevent thromboembolic complication in procedures where ablation has been undertaken on the left side of the heart; some operators also prescribe this for right-sided

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