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Focal atrial tachycardia

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ABSTRACT
Focal atrial tachycardia is a relatively uncommon arrhythmia. Nevertheless, the management of highly symptomatic patients with focal atrial tachycardia can be problematic owing to the poor response to medical treatment. Moreover, focal atrial tachycardia can trigger other atrial arrhythmias like atrial fibrillation and flutter. Radiofrequency ablation of focal atrial tachycardia is extremely successful and this approach is becoming the preferred treatment for symptomatic patients. In this review, we describe the pathophysiology, anatomical localisation, clinical features, diagnosis and therapeutic options for the management of focal atrial tachycardia.

INTRODUCTION
Focal atrial tachycardia (AT) accounts for up to 10% of supraventricular tachycardia.1 It is generally poorly responsive to medication and may be responsible for initiating other atrial arrhythmias such as atrial fibrillation (AF) and atrial flutter. Fortunately, with the advent of radiofrequency ablation this form of tachycardia can be treated with high long-term success.2 3

In this review we will describe the pathophysiology and responsible mechanisms, anatomical localisation, clinical features, diagnosis and therapeutic options for management of focal AT.

DEFINITION OF FOCAL AT
AT may be classified as focal or macro-re-entrant according to the electrophysiological mechanism. The mechanism may be difficult to distinguish on the basis of the surface electrocardiogram (ECG). However, focal AT can generally be defined by a slower P-wave rate (<200/min) and an isoelectric interval between P waves. It is defined as atrial activation originating from a discrete focus with centrifugal spread. Typically, activation is not continuous throughout the cardiac cycle length, the hallmark of re-entry. The focus originates from an area of atrial myocardium arbitrarily defined as <2 cm in diameter.4

PATHOGENESIS AND ARRHYTHMIA MECHANISM
Focal AT may be due to abnormal automaticity, triggered activity or micro-re-entry, with the responsible mechanism largely determined at the time of electrophysiological study. AT due to abnormal automaticity occurs spontaneously or in response to isoprenaline and is not induced by programmed extrastimuli. There is a significant overlap in the electrophysiology characteristics of triggered activity and micro-re-entry as programmed stimulation may initiate and terminate both. Thus only limited insights into the mechanism of arrhythmia can be ascertained from a clinical study. The use of adenosine to differentiate among different types of focal AT has provided inconsistent results and is of limited value.5 6

A common misconception is that focal AT is a sign of underlying heart disease. While it may originate in the presence of structural heart disease, it is more often multifocal. Indeed, the majority of histological analyses conducted on myocardium from AT focuses have shown normal findings. Nevertheless, abnormal myocardium has also been seen with areas of myocardial fibrosis, myocyte hypertrophy, inflammatory infiltration and fatty substitution.7 Studies using electroanatomical mapping have demonstrated the presence of low-voltage zones within the atria of patients with focal AT, indeed regions of low amplitude and fractionated electrograms have been described at sites of successful ablation in patients with focal AT.5 Locations of slow conduction and anisotropy provide the substrate for micro-re-entry and the initiation of focal AT.

In human studies reduction in tissue voltage and conduction slowing occur with ageing.8 These observations may, in part, explain the clinical observation that micro-re-entry is a more common mechanism for focal AT in the older population.9

Anatomical localisation
The foci responsible for focal AT do not occur randomly throughout the atria but tend to cluster at characteristic anatomical locations. In the right atrium (RA) these foci tend to occur along the crista terminalis (CT),3 tricuspid annulus (TA),10 ostium of the coronary sinus (CS) and the parahisian region. In the left atrium (LA) foci occur predominantly at the ostia of the pulmonary veins (PVs)11 and, less commonly, at the mitral annulus12 LA appendage and left-sided septum.13

Right atrium
The CT is the origin of two-thirds of right-sided focal AT.3 It is the embryological junction of the smooth and trabeculated RA and an area of marked tissue anisotropy, providing the potential substrate for micro-re-entry.14 In addition, the sinus node complex extends a variable distance along the long axis of the CT. The properties of automaticity due to resident nodal tissue combined with anisotropic conduction explain the propensity for atrial arrhythmias to originate from this location. The TA is the next most common site of origin of right focal AT.10 McGuire et al demonstrated the existence of myocytes surrounding the TA which were histologically similar to atrial myocytes but possessed nodal-like electrophysiological properties.15 The CS forms the major venous drainage of the heart. The ostium is a not uncommon site of focal AT, characterised by a cuff of striated...
myocardium with an abrupt change in fibre orientation at its entry into the RA in the vicinity of the Thebesian valve. Less common sites include the atroventricular (AV) node and surrounding transitional tissue, the right atrial appendage and the superior vena cava.

Left atrium
In the LA the predominant site of origin for focal AT is the ostia of the PVs. Kalman and coworkers presented 27 patients with no prior history of AF and focal AT arising from the PVs with focal ablation associated with high long-term success. Important differences were demonstrated between AT compared with fibrillation arising from the PVs. AT is characterised by a:
1. focal process compared with PV AF, where a more diffuse process involving multiple sites within multiple veins occurs;
2. foci at the ostium compared with foci deep within the veins for PV AF;
3. cycle length significantly slower than that of PV AF;
4. focal ablation associated with high long-term success compared with venous electrical isolation required for PV AF.

The superior mitral annulus is the next most common location of focal AT. The left fibrous trigone in the vicinity of the mitral aortic continuity may hold remnant primitive AV nodal tissue which has remained following embryological development. Wit et al have observed that the anterior leaflet of the mitral valve contained muscle fibres in direct continuity over the fibrous annulus with the left atrial myocardium. These muscle fibres exhibit "nodal-like" action potentials, thereby initiating automatic impulses that could propagate into the LA.

Less common locations include the left atrial appendage, the body of the CS and the left atrial septum.

Clinical features
The onset of symptoms may occur at any age, from birth through to old age.

Patients report palpitations with substantial variation in duration and rate consistent with bursts of activity, in contrast to other forms of supraventricular tachycardia secondary to a stable re-entrant circuit. Episodes may last only a few seconds or hours within an individual patient. The atrial rate during focal AT is typically variable ranging between 130 and 250 beats/min, but may be as low as 100 beats/min or as high as 300 beats/min, especially in young patients and infants. The clinical course is usually benign, although incessant tachycardia may result in a cardiomyopathy. The cardiomyopathy usually recovers after correction of the tachycardia, with normal left

Figure 1  Panel A shows the 12-lead electrocardiogram (ECG) during a right atrial tachycardia (AT) originating from the inferolateral tricuspid annulus (TA). Note the P-wave morphology during tachycardia with a negative polarity in V1, II, III, aVF and positive in aVL. Panels B and C show the three-dimensional electroanatomical map (CARTO) and the site of successful ablation recorded by the ablation catheter.
The clinical course is highly variable with spontaneous remission common, particularly in childhood and adolescence. Diagnosis of focal AT

Focal AT is characterised by an abrupt onset and termination with bursts of activity. The duration may range from frequent atrial ectopy to sustained tachycardia. The atrial rate may vary and demonstrate dissociation from the ventricle, an important distinguishing feature on the surface ECG from other forms of supraventricular tachycardia. Focal AT may be difficult to distinguish from sinus tachycardia without the need for an electrophysiological study. In general focal AT is characterised by:

1. sudden increases and decreases in rate;
2. change in P-wave morphology, although foci arising from the superior CT may be indistinguishable from the sinus P wave;
3. dissociation between the atrium and ventricular response.

The distinction between focal AT and AV nodal re-entrant tachycardia or AV re-entrant tachycardia can be made by analysing the R–P relationship on the surface ECG. Typically, focal AT is associated with a long and variable R–P relationship.

However, focal AT can show a short R–P relationship at higher rates and increased AV node conduction. Atypical AV nodal re-entrant tachycardia and a concealed accessory pathway with slow retrograde conduction may demonstrate a long R–P interval but the R–P interval is typically constant.

Classically, the P wave for focal AT is described as distinct with an intervening isoelectric interval, in contrast to a continuous undulation typical of macro-re-entry tachycardia. However, an isoelectric interval may not be identifiable during accelerated heart rates and/or in the presence of atrial disease resulting in slowing of conduction. The joint working group of the European Society of Cardiology and North American Society of Pacing recognised the limitations of an ECG-based diagnosis and the need for a mechanism-oriented approach with the advent of electrophysiology study. Focal AT is defined as atrial activation originating from a discrete region of atrial myocardium <2 cm in diameter spreading centrifugally. In contrast, macro-re-entrant AT, which replaces the previous term atrial flutter, refers to continuous atrial activation involving a re-entrant circuit >2 cm in diameter.

Practically, the term atrial flutter remains widely used and is unlikely to be replaced.
Management of focal AT

The management of focal AT has evolved with the advent of electrophysiology study and catheter ablation. The unsatisfactory results with medical treatment and high long-term success with radiofrequency ablation have established this as accepted practice for many symptomatic patients.

The acute management of patients with focal AT involves the use of pharmacological agents. Indeed vagal manoeuvres, such as carotid massage and DC cardioversion, are seldom effective in terminating focal AT.26

Adenosine can effectively terminate focal AT related to triggered activity and micro-re-entry, whereas the response in automatic AT is generally of transient suppression.2 AV-nodal blocking agents are useful in controlling the ventricular rate. Moreover, calcium-channel blockers and β-blockers can terminate AT owing to enhanced automaticity, while β-blockers may be effective if the mechanism of the tachycardia is triggered activity.2 Class Ic drugs, in particular flecainide, have been shown to be efficacious in terminating focal AT as well as reducing recurrence.27 The class III agents sotalol and amiodarone are also effective.28 Nevertheless, given the limited efficacy of pharmacological treatment and possible side effects, radiofrequency ablation is the preferred strategy for patients with significant symptoms.

The radiofrequency ablation of focal AT relies on an accurate mapping of the site of origin of the tachycardia. The major limitation to success is insufficient ectopy or tachycardia to allow the detailed mapping required to identify the site of origin and provide an accurate end point to the procedure.

P-wave morphology

The morphology of the P-wave provides useful clues to the likely site of tachycardia origin. Studies looking at P-wave morphology have mainly involved patients with structurally normal atria. The accuracy of P-wave morphology is limited in the setting of significant atrial disease, which is currently most commonly seen after catheter ablation for AE. Importantly, an unencumbered P wave from the preceding T wave is crucial to the usefulness of the interpretation of the P wave. This may be facilitated by vagal manoeuvres, the administration of adenosine or ventricular pacing.

Leads V1, aVL and lead I have been used to distinguish between left and right atrial foci.30 Based on our findings lead V1 is more accurate than leads aVL or lead I. Although lead aVL is usually positive for right ATs, it is frequently negative for tachycardias arising from the CT. A negative P wave in lead I is highly specific for a left atrial origin; however, a positive or isoelectric P wave is less useful. A positive P wave in V1 indicates a left atrial focus with a sensitivity of 95%, specificity of 88%, positive predictive accuracy of 87%, and negative predictive accuracy of 94%.30 A negative P wave indicates an origin from the RA.

For the purpose of this review only general comments will be made regarding the P-wave morphology from typical anatomical locations (figures 1–3):

- CT: this is a common site of origin of focal AT. The majority of AT from the mid-CT and superior CT have a biphasic (positive/negative) P wave in lead V1, similar to the sinus P wave.3
- TA: the P wave is bifid negative in lead V1. Inferior annular foci have negative P waves in leads II, III, and aVF whereas superior foci are usually isoelectric or positive.10 The superior TA and right atrial appendage are in close proximity and focal AT from these sites are difficult to distinguish.
- CS ostium: the P wave is similar to that seen in typical atrial flutter. In lead V1 an initial isoelectric segment is followed by an upright component. The P wave is deeply negative in the inferior leads.
- PVs: lead V1 is upright being narrow and uniphasic for right PV foci and broad and notched for left PV foci. A positive P wave in lead I suggests a right sided PV focus in contrast to an isoelectric or shallow negative P wave for left PV foci. The P wave is deeply negative in lead I for sites arising from the left atrial appendage.1
- Mitral valve annulus: characteristic biphasic (negative/positive) P wave in lead V1 readily missed if the unencumbered P wave is not analysed. In the limb leads the P wave is low amplitude.1,20 An algorithm based on the analysis of the P-wave morphology has been developed and is useful in identifying the likely site of origin of the focal AT. In a prospective trial of 30 patients with focal AT the correct site of origin was determined in 95%.31

Electrophysiological mapping and ablation

Patients should be studied in the fasted awake state with minimal use of sedation. All antiarrhythmic drugs should be stopped a minimum of five half-lives before the procedure. In the electrophysiology laboratory the mapping of focal AT involves

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**Figure 3** From left to right, 12-lead electrocardiogram (ECG) in patients with focal atrial tachycardia (AT) originating from the right superior pulmonary vein, left inferior pulmonary vein and superior mitral annulus. In panels A and B the first sinus beat is followed by AT from the right superior and left inferior pulmonary vein. In panel C, AT from the superior mitral annulus is evident after a ventricular ectopic. LIPV, left inferior pulmonary vein; RSPV, right superior pulmonary vein; Sup MA, superior mitral annulus.
conventional activation mapping techniques including: (i) P-wave morphology; (ii) right atrial endocardial activation sequence and (iii) point mapping to locate the site of earliest endocardial activation relative to surface P-wave onset with the mapping/ablation catheter. The 3D mapping systems are useful in recreating the geometry of the chamber using activation mapping to identify the site of origin. The 3D mapping systems are of particular importance in the presence of significant atrial disease (congenital, atriotomy scars or after extensive atrial catheter or surgical ablation).

Catheter ablation is associated with high long-term success with a low complication rate. Recurrence is typically infrequent. In the early stages, tachycardia usually arises adjacent to the original site in contrast to late recurrences due to a different focus or tachycardia mechanism. Older patients, patients with structural heart disease and multiple foci are at increased risk of recurrence.

CONCLUSION
Focal AT is a localised process frequently seen in the absence of other cardiac disease. It is a relatively uncommon cause of supraventricular tachycardia but, when present, is frequently difficult to treat medically. Catheter ablation is associated with high long-term success with few complications, establishing this as a primary treatment strategy in symptomatic patients.

Competing interests None.

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