ATRIAL TACHYCARDIA

Atrial tachycardia is a rare form of supraventricular tachycardia, accounting for about 10-15% of patients presenting to experienced arrhythmia centres for radiofrequency catheter ablation. Atrial tachycardias include a heterogeneous group of tachyarrhythmias such as focal atrial tachycardia, typical atrial flutter and atypical atrial flutter. Focal atrial tachycardias arise from automatic, triggered or micro-reentrant mechanisms, while typical and atypical atrial flutters are macroreentrant arrhythmias. Typical flutter is maintained by a reentrant circuit (cavotricuspid isthmus) whereas atypical flutter includes lesional and de novo macroreentrant circuits in the right and left atria. Electrocardiographic criteria to distinguish tachycardia mechanisms are not specific while adenosine administration may be helpful in establishing the diagnosis when incessant tachycardia is present, tachycardiomyopathy may develop. Management of focal atrial tachycardias and macroreentry is based on rate control, antiarrhythmic drug therapy, catheter ablation and oral anticoagulation. Catheter ablation is highly effective (> 90%), particularly for typical atrial flutter and much higher than antiarrhythmic therapy for most atrial tachycardias.

Focal Atrial Tachycardia

The efficacy of chronic antiarrhythmic drug therapy for management of atrial tachycardia is not well defined. Class IC or class I drugs may be administered in re-entrant atrial tachycardia while verapamil, beta-blockers or class IC drugs in focal atrial tachycardias. If antiarrhythmic therapy is ineffective, amiodarone is used as an alternative. Experience with radiofrequency catheter ablation to eliminate atrial tachycardia is limited, but preliminary results are encouraging with success rates ranging from 80% to 95% with a low recurrence and complication rate. Thus, it is conceivable that RF catheter ablation will become the therapy of choice for atrial tachycardia when this arrhythmia is not controlled by drug therapy. Over the last decade, the lack of efficacy of antiarrhythmic drug therapy and the increasing experience of radiofrequency ablation have changed our strategy for the treatment of focal atrial tachycardia. The diagnosis of focal AT may be made from a standard electrocardiogram (ECG); however, sometimes differentiation from other forms of supraventricular tachycardia may be difficult. Focal AT may be due to several different mechanisms, including abnormal automaticity, triggered activity, and microreentry. Focal AT does not occur randomly throughout the atria but has a characteristic anatomic distribution. Focal ATs are characterized by regular atrial activation from atrial areas with centrifugal spread not extending through the entire atrial cycle and usually manifest by atrial rates between 100 to 250 bpm and rarely at 300 bpm. Focal ATs account for 10 to 23% of SVTs in children with normal hearts and a much higher rate in those with congenital heart disease. The outcome of focal AT is usually benign with the exception of incessant forms, which in the absence of treatment may lead to tachycardia-induced cardiomyopathy. The diagnosis of multifocal atrial tachycardia is based on an irregular tachycardia characterized by three or more different P-wave morphologies at different rates. The rhythm is always irregular resembling an atrial fibrillation, but, unlike atrial fibrillation, its rate is usually not excessively rapid. This arrhythmia is most commonly associated with pulmonary disease or metabolic or electrolyte derangements and rarely is due to digitalis toxicity.
Macro-reentrant Atrial Tachycardia

Macro-re-entrant atrial arrhythmias can be considered in terms of those that are dependent on the cavo-tricuspid isthmus and those that are not. Cavo-tricuspid isthmus-dependent (CTI) arrhythmias include typical counterclockwise AFL in addition to ‘typical’ clockwise flutter and lower loop re-entry. CTI-dependent arrhythmias can be effectively treated by catheter ablation with success rates > 95% and very low procedural risk. No-CTI-dependent atrial macro-re-entry may develop in both the right and left atrium (LA). Presence of surgical atrial scars (Atrial septal defect, Fontan, Tetralogy of Fallot, mitral valve surgery etc.) may also lead to macroreentrant atrial tachyarrhythmias (lesion or incisional re-entry). In addition, in patients with non-CTI ‘flutter’, particularly in the presence of prior atrial surgery or significant SHD, multiple circuits may develop simultaneously (e.g. dual-loop re-entry).

The substrate underlying typical AFL (either counterclockwise or clockwise) has been characterized and can be safely eliminated by catheter ablation. In atypical AFL, the location of the circuit is variable and may involve dual-loop re-entry or complex re-entrant mechanisms. Predicting a non-isthmus-dependent AFL before catheter ablation is useful to better define the procedural time, success rate and potential risks of the procedure. The clinical context as well as the flutter wave morphology and axis analysis on the surface ECG may support the underlying macro-re-entrant substrate easily amenable for catheter ablation.

Typical AFL involves stereotyped rotation (either counterclockwise or clockwise) of the tachycardia circuit around the tricuspid valve annulus, and flutter wave appearance in these subtypes is generally consistent and predictive of the underlying mechanism. However, the correlation between flutter wave appearance and the underlying re-entrant circuit may be limited, as some “atypical” non-isthmus-dependent flutters may manifest typical surface ECG patterns while some “typical” flutters do not conform to the classic ECG appearance. Typical atrial flutter is a common atrial arrhythmia that may cause significant symptoms and serious adverse effects including embolic stroke, myocardial ischemia, and rarely a tachycardia-induced cardiomyopathy as a result of rapid atrioventricular conduction. Due to drug failure, currently catheter ablation is considered as a safe and effective alternative or first-line treatment. Although the appearance of flutter is suggestive of macro-re-entry, it may also be seen in focal arrhythmias under certain conditions. Although atrial flutter is characterized by a ‘sawtooth’ pattern in leads II, III, and AVF, a closer examination of the flutter wave yields much additional information. The inferior leads demonstrate an initial gradual downsloping segment followed by a sharp steep descent, then a sharp ascent with a low amplitude terminal positive component, which continues into the gradual descent of the subsequent flutter wave. Lead V1 typically shows an initial isoelectric component followed by an upright component. With progression across the precordium, the initial component rapidly becomes inverted and the second component isoelectric, usually by V2 to V3. This produces the overall impression of an upright flutter wave in V1 which becomes inverted by V6. Lead I is low amplitude/iseoelectric and AVL usually upright. Although this classic appearance rarely shows much variation, occasionally unusual morphologies occur or a left AFL may mimic a counterclockwise flutter appearance.

Lower loop re-entry (counterclockwise) is ‘cavo-tricuspid isthmus-dependent’. The flutter wave appearance on the surface ECG in lower loop re-entry is variable and depends upon the site of breakthrough of the wavefront at the crista terminalis. When breakthrough occurs at the low lateral RA, the resulting clockwise
ascending wavefront collides with the counterclockwise wavefront propagating from the interatrial septum and roof of RA, thus abolishing the late descending wavefront on the lateral RA wall seen in counterclockwise typical AFL. Abolishing these late inferiorly directed forces is reflected by attenuation in the late positive deflection of the flutter wave compared with that of counterclockwise typical AFL. As the LA and septum are activated in a similar sequence to counterclockwise typical AFL, the flutter waves are otherwise comparable. This arrhythmia will also be successfully ablated in the cavo-tricuspid isthmus.

Clockwise or reverse typical atrial flutter
The re-entrant anatomical circuit is identical as in typical AFL, with a clockwise direction of rotation around the tricuspid annulus. A minority of patients with typical counterclockwise AFL (10%) also has clockwise AFL but in a higher percentage this pattern may be induced in the electrophysiology laboratory. The surface ECG appearance is more variable than that of typical counterclockwise AFL. In the inferior leads, the flutter waves are usually broadly positive, with characteristic notching. However, there is an inverted component preceding the upright notched component. Depending on the amplitude of this component, the appearance can be of continuous undulation without an obviously predominant upright or inverted component. On other occasions, it may appear that the inverted component is dominant, thus superficially mimicking counterclockwise flutter. V1 is characterized by a broad negative and usually notched deflection. There is transition across the precordium to an upright deflection in V6. Lead I is usually upright and AVL is low amplitude negative and notched.

Right atrial free wall atypical atrial flutter
Atrial macro-re-entry in the right free wall is the commonest form of right atrial atypical flutter. Such macro-re-entrant circuits may propagate around low voltage areas or scar in the lateral or postero-lateral right atrial wall. Usually, scarring is due to previous atrial surgery. Electroanatomical mapping has identified both single-loop re-entry and dual-loop re-entrant circuits utilizing neighboring anatomical structures. This arrhythmia may be frequently associated with typical and/or reverse typical AFL. The ECG appearance of free wall AFL is highly variable; depending on factors including anatomic location (superior or inferior), direction of rotation, conduction block in the atrium, and concomitant presence of a peri-tricuspid circuit. For example, the flutter morphology of a free wall circuit will be markedly altered by the presence of pre-existing cavo-tricuspid isthmus block. If there is a hallmark for a right atrial free wall flutter, it shows the presence of an inverted flutter wave in V1. Depending on the predominant direction of septal activation, right atrial free wall flutter can mimic either clockwise or counterclockwise flutter.

Upper loop re-entry atypical atrial Flutter
Upper loop re-entry (ULR) defines an atypical AFL involving the upper portion of the RA and is not cavo-tricuspid isthmus-dependent. Although this macro-re-entrant circuit may exist in isolation, episodes often arise after transition from typical clockwise AFL or atrial fibrillation. Three-dimensional mapping has documented the re-entrant circuit around the superior vena cava and the upper crista terminalis. Usually, this macroreentrant right atrial tachycardia closely resembles the appearance of clockwise or reverse typical AFL on the surface ECG, with positive P-waves in the
inferior leads, as in the majority of cases, activation of the septum and LA occurs via inferiorly directed forces. The cycle length of this circuit is shorter in comparison with CTI-dependent flutters, because of the shorter circuit length. An algorithm to help distinguish ULR from reverse typical AFL has been proposed, based on the polarity and amplitude of the P-wave in lead I. Negative or isoelectric/flat P waves in lead I were associated with ULR, and when P waves were positive in lead 1, UL was probable when P wave amplitude was ≤0.07 mV, and reverse typical AFL was probable when P wave amplitude was >0.07 mV. Distinguishing UL from reverse typical AFL from the ECG prior to electrophysiological study is potentially valuable, as the two arrhythmias require different mapping and ablation techniques. However, definitive diagnosis of UL requires detailed intracardiac mapping.

Other unusual forms of atypical right AFL have been described. Circuits involving the septum have been demonstrated, particularly after prior surgery involving this area but are relatively uncommon. They are usually characterized by a biphasic or isoelectric flutter morphology in V1.

Left atrial flutter

Left AFLs are less common than typical AFL and typically are associated with hypertension, mitral valve disease, left atrial dilation, and cardiac failure. Anatomical macroreentrant circuits are highly variable and usually develop around areas of spontaneous scarring often in the posterior LA. Activation propagate around the mitral valve annulus, around regions of scarring, and the ostia of the pulmonary veins or infrequently around the fossa ovalis in the septum. Left AFL circuits are less well-studied than typical and atypical right AFLs; however, the most common form involves a perimitral circuit. The surface ECG findings are frequently similar despite different underlying substrates. The flutter wave typically shows a prominent positive deflection in lead V1 and uncommonly is flat or isoelectric. The flutter waves in leads II, III, and aVF may be upright but are frequently of low amplitude. However, in a minority of patients, the morphology resembles typical flutter. The two most commonly observed patterns in the left AFL would include: a broad upright flutter wave in V1 with upright waves in inferior leads; or broad upright flutter wave in V1 with low amplitude; or isoelectric waves in all other leads. Owing to a high prevalence of generalized atrial disease and slower conduction, longer cycle lengths with a greater isoelectric interval between flutter waves have been observed. As a result, left AFL may mimic a focal atrial tachycardia.

ECG wave pattern analysis to distinguish right-sided vs. left-sided macro-re-entrant circuits

The most useful lead to evaluate right from left AFL is V1. A broad-based upright V1 is highly predictive of a left-sided flutter. However, when V1 has an initial isoelectric (or inverted) component (followed by an upright component), this is consistent with a right AFL. Conversely, when V1 is deeply inverted, this is highly suggestive of a right-sided flutter. However, when V1 is biphasic or isoelectric, it is not helpful in predicting the chamber of origin.

Macro-re-entrant atrial tachycardia vs. focal atrial tachycardia

The pattern and behavior of the atrial tachycardia may suggest the underlying substrate. Focal atrial tachycardias typically present alterations in cycle length with speeding (‘warm up’) and slowing (‘cool down’) at the onset and termination of tachycardia. The AT cycle length is accurately assessed using
coronary sinus electrograms during AT. The mean cycle length and the range between the longest and shortest cycle lengths is measured over a 1-minute period. The TCL variability is then calculated by dividing the range by the mean TCL. A dynamic and intermittent arrhythmia pattern with frequent interruptions followed by reinitiating is indicative of a true focal mechanism. Focal atrial tachycardias rarely are incessant but often are recorded as bursts of tachycardia with spontaneous onset and termination and frequently accelerate after sympathetic activation. The differentiation between focal and macroreentrant atrial tachycardia is crucial since identification of earliest region of activation is crucial for focal AT while for macroreentrant AT the wavefront is continuously propagating around the circuit. The tachycardia cycle length not always may be utilized in differentiating between focal and macro-re-entrant mechanisms. Although the cycle length is usually ≥250 ms in focal atrial tachycardia, shorter cycle lengths have been reported. In this situation, particularly in the presence of intra-atrial conduction delay, there may be no observable isoelectric interval between P-waves, and an undulating baseline resembling AFL may be seen. Conversely, macro-re-entrant circuits may have long cycle length in the presence of SHD and anti-arrhythmic agents. Furthermore, in the presence of significant atrial scarring, there may be a long isoelectric interval between flutter waves, incorrectly suggesting a focal mechanism. This is particularly observed for left AFL in the presence of large areas of electrical silence. Entrainment remains the cornerstone to confirming the diagnosis of reentry. A post-pacing interval not exceeding the TCL by more than 20 ms in three different areas of the circuit typically confirms the diagnosis of macroreentry. Atrial overdrive pacing is useful in localizing focal AT. The aim of overdrive pacing in the context of truly focal arrhythmia is to reset the tachycardia. In some cases, focal versus macroreentrant circuits cannot be established because of widespread abnormal electrograms or scars facilitating slow conduction or block with more complex arrhythmias, particularly when a focal arrhythmia is near to complete ablation line, simulating macroreentry. In challenging cases entrainment can be useful to discriminate between macroreentrant and focal AT.

3D Electroanatomical Navigation Systems in atrial tachycardia

Analysis of the flutter wave is difficult when conduction is 1:1 or 2:1, as the flutter wave is totally or partially concealed within the QRS complex or T-wave. This is compounded at times when the flutter wave amplitude is low. Frequently, macro-re-entry develops in patients with structural diseases which may alter non-uniformly the direction of wavefront propagation in the atrium making the P-wave morphology analysis not useful in predicting the anatomic location. This limitation applies particularly to patients with atrial tachycardias and prior atrial surgery or extensive atrial ablation. It is well known that in these patients there is an abnormal anatomy due to congenital abnormalities as well as to prior atrial surgery. In these cases, surface ECG is of limited value for establishing a correct diagnosis of atrial tachycardia localization. For these reasons, macroreentrant circuits require 3D endocardial mapping for a detailed and accurate anatomic reconstruction. Three-dimensional navigation systems are used worldwide to improve the efficacy of the ablation. Electroanatomical systems provide an accurate reconstruction of the atrial and PV anatomy greatly reducing fluoroscopic time and radiation exposure for both patients and operators. Most importantly, 3D electroanatomic systems with activation mapping are useful in
evaluating the location of focal or macroreentrant AT, particularly in patients with postablation AT facilitating their elimination by an accurate ablation strategy. Unfortunately, these electroanatomic mapping systems are not routinely used in all centers.