P Wave Morphology in Guiding the Ablation Strategy of Focal Atrial Tachycardias and Atrial Flutter

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Abstract: Focal atrial tachycardias arise preferentially from specific locations within the atria. Careful analysis of the P wave can provide useful information about the chamber and likely site of origin within that chamber. Macro-reentrant atrial flutter also tends to occur over a limited number of potential circuits. In this case, the ECG usually gives a guide to the chamber of origin, but unless it shows a specific morphology it is less useful in delineating the circuit involved. Nonetheless, prior knowledge of the likely chamber of origin helps to plan the ablation strategy.

Keywords: Atrial tachycardia, catheter ablation, P wave morphology.

INTRODUCTION

Assessment of the P wave morphology is the first step in the mapping and ablation of both focal atrial tachycardia (FAT) and atrial flutter (AFL). FAT is a relatively uncommon mechanism of SVT, characterized by a pattern of activation from a point source in the atrium [1]. The underlying mechanism of the focal firing can be abnormal automaticity, micro-reentry or triggered activity [2]. AFL e.g. typical right atrial flutter involves macro re-entry around one or more fixed or functional areas of block, with the circuit involving the majority or the whole of the chamber [1]. Both can be ablated with high rates of success, but an understanding of whether the tachycardia is focal or macro-reentrant is necessary for ablation to succeed.

PRACTICAL SUGGESTIONS FOR ASSESSING THE P WAVE

Both FAT and AFL by definition occur independently of the AV node and ventricle. Assessment of the P wave requires a clear view of atrial activity. This may occur fortuitously during recording of the ECG, but often the P wave is partially obscured by the QRS complex or T-wave. To obtain an ‘unencumbered’ view of atrial activity, one can give adenosine or perform carotid sinus massage. However, an effective method in the EP lab is to pace the ventricle during SVT in order to reveal the P waves in the compensatory pause that follows. In order to accurately assess the P wave morphology, one must determine the onset, and the initial positive or negative deflection may be preceded by an isoelectric interval that may vary from lead to lead. Therefore, it is best to examine P wave morphology in all 12 ECG leads simultaneously with adequately increased gains.

CONTRIBUTION TO THE P WAVE

Use of P wave morphology to localize atrial activation has been studied largely in patients with normal atria; hence in diseased or previously ablated atria it may not be as reliable. Atrial activation from a focal source in the atrium can propagate radially from myocyte to myocyte, but more rapid activation typically occurs along the direction of fiber orientation and through myocardial bundles or tracts. Connections exist between the right and left atria, the best known of which is Bachmann’s bundle superiorly. There are further connections described a) posteriorly joining the inter-caval area of the right atrium to the left atrium, b) in the region surrounding the fossa ovalis, and c) inferiorly from the left atrium running into the wall of the coronary sinus [3, 4]. As a consequence even in sinus rhythm the P wave can show considerable variation. Right P waves in the right pre-cordial leads appear to be associated with conduction, not only via Bachmann’s bundle but also via other connections in the posterior atrium, thus resulting in posterior to anterior propagation of sinus beat in both right and left atria [5]. In early intra-operative pacing studies by Waldo et al., during right atrial pacing from low endocardial sites, depending on the site paced the P wave in inferior leads could be either negative, biphasic, or positive [6]. More recently, non-contact mapping studies have confirmed variation in the preferential routes of atrial activation in vivo both during sinus rhythm and atrial tachycardia [7-9].

Due to the large circuit, AFLs are typically represented by continuous undulating atrial activity. FATs are usually represented by discrete P waves with intervening isoelectric segment. There can be exceptions to this general rule, particularly in scarred or previously ablated atria, such that AFL can have the appearance of a focal circuit, and FAT can appear ‘flutter-like’ due to slow propagation through the atrium (see Fig. 1) [10]. Variation in P wave cycle length is more suggestive of a focal mechanism. In equivocal cases, entrainment or detailed electro-anatomic activation mapping
may be required during the EP study to differentiate the two mechanisms.

FOCAL ATRIAL TACHYCARDIA

If a focal mechanism is suspected, the first step is to determine the chamber of origin. One of the first algorithms to localize the origin of a FAT was produced by Tang and colleagues from a series of 31 patients, where the most useful leads for discriminating left and right atrial origin were V1 and Avl [11]. The sensitivity and specificity of a positive or biphasic P wave in lead aVL to predict a right atrial focus were 88% and 79% respectively. The sensitivity and specificity of a positive P wave in lead V1 in predicting a left atrial focus were 93% and 88% respectively.

Kistler and colleagues developed a detailed algorithm (see Fig. 2) based on a series of 126 patients to determine the likely site of origin of FAT [12]. The algorithm was then prospectively tested in 30 patients and found to have accuracy of 93%. A negative or biphasic (positive, then negative) P-wave in lead V1 was associated with a 100% specificity and PPV for a focus from the right atrium. A positive or biphasic (negative, then positive) P-wave in lead V1 was associated with a 100% sensitivity and NPV for a focus originating in the left atrium.

A potential disadvantage of many electrophysiology diagnostic ‘algorithms’ is that if the first step of assessment of the algorithm is incorrect, this can lead the clinician down the wrong pathway. This can be exacerbated by interindividual variation of the atrial anatomic position, relative to each other and to the ECG leads. Qian and colleagues have observed that using multiple ECG leads for each algorithm decision step yields greater accuracy in locating the focus to a region of the atrium (see Fig. 2) [13]. Positive P waves in inferior leads and a negative P wave in lead aVR indicated high atrial origin with a sensitivity of 95% and a specificity of 90%. Negative P waves in inferior leads and a positive P wave in lead aVR suggested right low septal origins, with sensitivity and specificity 88% and 89%, respectively.

It is important to remember that FAT does not occur randomly throughout the atrium and tends to cluster around certain anatomic regions. Common sites of origin in the right atrium include the crista terminalis, tricuspid annulus, coronary sinus os, perinodal region, and more rarely the right atrial appendage (see Fig. 3 for examples). Within the right atrium, a negative P wave in aVR has high sensitivity and specificity for the crista terminalis [14]. FAT can arise from anywhere around the tricuspid annulus, but a P wave that includes negative polarity in V1 and is positive or isoelectric in aVL is associated with a very high specificity for a focus.
Fig. (2). P wave algorithms described by Kistler et al. and Qian et al. for comparison.

Fig. (3). P waves of common sites for focal atrial tachycardia in the right atrium. From left to right: coronary sinus os, low tricuspid annulus (between 7 and 8 o’clock), base of right atrial appendage, mid crista terminalis. Note in coronary sinus os and low tricuspid annulus the negative P waves in inferior leads and positive P waves in aVR. The coronary sinus os P wave is narrower due to a more septal location, while the tricuspid annular P wave is negative and bifid in V1. The right atrial appendage P wave by contrast is positive in the inferior leads and negative in aVR. The crista terminalis P wave is positive in V1 and across the chest leads, somewhat similar to a right pulmonary vein origin, which reflects their anatomic proximity. (The red arrow heads mark the tachycardia p waves).
at the tricuspid annulus [12]. Tricuspid FAT often has a characteristically bifid negative morphology in lead V1, and the polarity of leads II and III is deeply negative for an inferior location, and low amplitude, positive, or biphasic for a superior location. Coronary sinus os P wave morphology is deeply negative in the inferior leads, positive in aVL and biphasic negative or isoelectric to positive in lead V1, then progressively negative across the precordium [15]. FAT from the right atrial appendage has P waves that are negative in lead V1, becoming progressively positive across the precordial leads and in the inferior leads these p waves are low amplitude positive in the majority of patients [16].

Left sided FAT occurs in the pulmonary veins, mitral annulus and more rarely the left atrial appendage (see Fig. 4 for examples). Pulmonary vein FAT tends to have positive P waves across the precordium, with negative P waves in aVL and aVR. Left sided veins have a broader, notched P wave compared to right side veins, and superior veins a taller P wave in the inferior leads [17]. Yamane and colleagues reported that positive P waves in I and aVL had high specificity, but moderate sensitivity for right pulmonary vein origin. Superior pulmonary vein origin could be distinguished from inferior vein by amplitude in lead II of >100 μV [18]. Mitral annular FAT tends to cluster superiority around the aortomitral continuity with a P wave that is negative-positive biphasic in V1, low amplitude in the limb leads and negative or isoelectric in I and aVL [19]. The P wave in left atrial appendage (LAA) FAT is similar to that from a left-sided pulmonary vein, but the presence of a deeply negative P-wave in lead I suggests an origin in the LAA [12].

It is important to remember that the overall spatial resolution of the P wave may be not more than 1.7 cm [20]. Therefore locations where P wave morphology is less useful are areas of close proximity of left and right atrial structures e.g. right upper pulmonary vein and crista terminalis [21]. In this case, it can help to compare sinus and FAT P waves in V1 – right upper pulmonary vein foci show a change in configuration from biphasic in SR to upright in AT, a change not observed for right atrial tachycardias [11]. The mode of induction of tachycardia can also be helpful in distinguishing these – cristal FAT often requiring programmed stimulation, whilst pulmonary vein FAT tends to be spontaneous or requires isoprenaline for induction. The atrial septum is another potentially difficult area (see Fig. 5), where an isoelectric or low amplitude P wave in V1 points towards origin in the right septal or perinodal region, while a biphasic negative-positive P wave in V1 raises the possibility of a left septal focus [22]. Mapping and ablation of septal FAT from the aortic non coronary cusp are also recognized and in the series of Ouyang et al. this too was associated with a biphasic negative-positive P wave in V1 and V2 [23].

ATRIAL FLUTTER

Some AFLs such as typical right atrial flutter have a readily recognizable morphology that allows their identifica-

Fig. (4). P waves of common sites for focal atrial tachycardia in the left atrium. From left to right: right upper pulmonary vein, superior mitral annulus (aorto-mitral continuity), left atrial appendage. Note the pulmonary vein P wave is positive from V1 to V6 and is positive in I and negative in aVL and aVR. The mitral annular P wave is biphasic (isoelectric-positive), positive in the inferior leads and negative in aVL. The left atrial appendage P wave is positive in V1, and negative in I and aVL. (The red arrow heads mark the tachycardia p waves)
tion with confidence. However, due to the fact that AFL can arise due to scar that is of variable location, the ECG can be similarly variable. If one is trying to decide if the chamber involved is right or left, the most useful lead is V1. A broad-based upright P wave in V1 is predictive of left-sided flutter, but when V1 has an initial isoelectric (or inverted) component followed by an upright component; this is consistent with a right AFL. When V1 is deeply inverted, this strongly suggests a right-sided AFL, but when V1 is biphasic or isoelectric, it is not helpful in predicting the chamber of origin [24].

Right atrial flutters can be divided into cavo-tricuspid isthmus dependent and other independent circuits. The former can be treated by linear cavo-tricuspid ablation so predicting the circuit from the P wave morphology could be useful in selecting an ablation target. Typical atrial flutter is the commonest AFL, involving a counterclockwise circuit around the right atrium when viewed from the left anterior oblique perspective. The anatomic circuit has been well characterized with the crista terminalis, eustachian ridge, and tricuspid annulus identified as barriers to conduction [25, 26]. The P wave morphology of typical flutter is well known as having an inferior lead ‘sawtooth’ pattern, but closer inspection reveals an initial gradual downward segment followed by a sharp descent, then a sharp ascent with a low amplitude terminal positive component that merges into the next flutter wave (see Fig. 6) [27, 28]. The negative P wave in the inferior leads is because left atrial activation dominates the P wave and in typical flutter starts at the coronary sinus and proceeds in a superior direction. In the precordial leads, V1 is biphasic isoelectric then upright, and moving across the precordium the initial component becomes negative and the second component isoelectric. Lead I is low amplitude/isoelectric and aVL usually upright. This typical appearance can be modified in the presence of previous surgery or ablation in the atria, including AF ablation [29].

Reverse typical flutter involves the same circuit as typical flutter but the direction of rotation is now clockwise [30]. The ECG appearance is more variable but in some respects is the opposite of typical counterclockwise flutter (see Fig. 6). In the inferior leads, the flutter waves are usually broad and positive, with notching [31]. The positive P wave in the inferior leads is due to left atrial activation via Bachmann’s bundle that then proceeds in an inferior direction. There is usually a downward component preceding the upward notched component, but the appearance can vary from continuous undulation without a predominant upward or downward component, or it may even appear that the downward component is dominant almost resembling counterclockwise flutter. The P wave in V1 is broad negative and usually notched with transition across the precordium to an upright deflection in V6. Lead I is usually upright and aVL is low amplitude negative and notched.

Lower loop re-entry is another cavo-tricuspid dependent circuit, which can rotate counterclockwise or clockwise around the inferior vena cava with break through the conduction barrier at the crista terminalis [32]. A potential clue to the presence of lower loop re-entry or another atypical RA flutter is a cycle length that is faster than the usual approxi-
mately 200ms seen with typical flutter. P wave morphology is dependent on the caudo-cranial level at which the wavefront breakthrough occurs in the crista terminalis [33]. As the LA and septum are activated in a similar sequence to counterclockwise typical AFL, the ECG appearance is similar. However when breakthrough occurs at the low crista, an ascending wavefront collides with the counterclockwise wavefront that is heading towards the lateral wall from the interatrial septum and roof of the right atrium. Loss of the late inferiorly directed forces is seen on the ECG as attenuation in the late positive deflection of the P wave normally seen in counterclockwise typical flutter. If there are multiple breakthroughs in the crista, then the P wave morphology can vary during tachycardia.

Upper loop reentry rotates around the superior vena cava through a break in the crista terminalis. Non contact mapping studies indicate that the commonest direction of rotation is clockwise [34]. The ECG morphology is often similar to clockwise typical flutter due to similar activation pattern of the left atrium. However, it has been reported that a negative or isoelectric flutter wave with amplitude < or = 0.07 mV in lead I can differentiate upper loop reentry from clockwise typical flutter [35]. The ablation target is the gap in the crista terminalis.

Atypical flutter circuits involving the free wall of the right atrium usually arise as a result of prior surgical incisions or less commonly due to spontaneous scar. Incisional flutters will be covered in a later section. Spontaneous right atrial free wall AFL is uncommon and has been described in a couple of small series, in which the P wave morphology was quite varied [36, 37]. Due to the variable location of scar and other factors including direction of rotation and whether the typical flutter circuit is also involved in a ‘double loop’ configuration, the ECG is not of great value in defining the circuit. A feature that can be useful in indicating a right atrial free wall circuit is the presence of an inverted P wave in V1 [24]. Ablation of these circuits is targeted at the low lateral right atrium with linear ablation from the scar to the inferior vena cava or tricuspid annulus. Demonstration of bidirectional block across the linear ablation rather than just acute termination of flutter is important to ensure long-term success [38].

Left sided AFL circuits can also arise as a result of prior surgery or ablation or due to spontaneous scar – these are discussed in the context of AF ablation in a later section. Jais et al. described a series of 22 patients with left atrial AFL arising as a result of spontaneous scar [39]. Circuits observed included peri-mitral, roof dependent or around an area of scar, and in minority dual or three loops. In all patients, the surface ECG showed a regular monomorphic P wave predominantly positive in lead V1 and distinct in the limb leads from the pattern described for right atrial flutter (see Fig. 7). Linear ablation to the roof or between the mitral annulus and another anchor point were employed to interrupt the circuit. In the series by Ouyang et al. the majority of circuits were dual loop and once again linear ablation across the critical isthmus was successful [40]. In the presence of such complex circuits the P wave morphology may be of limited use apart from localizing the problem to the left atrium.

Fig. (6). Comparison of counterclockwise (CCW) and clockwise (CW) right atrial, cavotricuspid isthmus dependent, flutter. Note the inferiorly directed P waves in the inferior leads in counterclockwise flutter compared to the positively directed P waves in clockwise flutter.
HOW TO INTEGRATE THE P WAVE INTO THE ABLATION STRATEGY

The ablation target in FAT is elimination of the focal source of activity, while in AFL the aim is to create a line of block between two electrically inert atrial structures in order to interrupt the macro-reentrant circuit. In practice one would tend to use some form of activation mapping or entrainment to define the focus or circuit before ablation. Nonetheless, careful evaluation of the P wave morphology is often extremely valuable in directing where to focus efforts at mapping and in planning the ablation strategy.

CONFLICT OF INTEREST

The authors confirm that this article content has no conflict of interest.

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Fig. (7). Examples of more unusual atrial macro-reentrant circuits – on the left a posterior right atrial free wall circuit and on the right a perimital clockwise circuit. Both show positive P waves in V1.
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