Electrocardiographic Characteristics of Focal Atrial Tachycardias

Haris M. Haqqani, MBBS(Hons), PhD[a,b,1],
Gwilym M. Morris, BmBCh, PhD[c,d,1],
Peter M. Kistler, MBBS, PhD[e],
Jonathan M. Kalman, MBBS, PhD[c,e,*]

KEYWORDS

• Ventricular tachycardia • Electrocardiograph • Myocardial infarction

KEY POINTS

- Focal atrial tachycardia (AT) is an uncommon form of supraventricular tachycardia that most often occurs in structurally normal hearts.
- Focal AT is characterized by centrifugal activation of the atria from a point source.
- The sites of origin of AT are not randomly distributed throughout the atria but instead cluster around stereotypical sites of anatomic and electrophysiologic heterogeneity.
- The P-wave morphology on the surface ECG in focal AT is generally a reliable guide to the site of origin in the absence of significant structural heart disease or previous ablation.

INTRODUCTION AND DEFINITION

Focal atrial tachycardia (AT) is an uncommon form of supraventricular tachycardia (SVT) defined by its characteristic centrifugal pattern of atrial activation from a focal site of origin. It accounts for 5% to 15% of adults being referred for evaluation of SVT.[1,2] Unlike other forms of SVT, AT requires activation of neither AV nodal or ventricular tissue for tachycardia continuation. Patients may present with palpitations, dyspnea, or, rarely, syncope. Incessant AT is a well-recognized phenomenon that may lead to the development of tachycardia-mediated cardiomyopathy.[3]

PATHOPHYSIOLOGY

Multiple electrophysiologic mechanisms may be responsible for focal AT. These include abnormal automaticity, triggered activity, and microreentry. The absence of a definitive gold standard for in vivo diagnosis of each of these mechanisms means that each is inferred from a combination of observations. Triggered activity and microreentry may be induced by programed stimulation, although triggered activity can also be induced with burst pacing. Automatic focal AT frequently has spontaneous onsets and terminations. In the absence of spontaneous activity it is likely to only

The authors have nothing to disclose.

[a] School of Medicine, University of Queensland, Queensland, Australia; [b] Department of Cardiology, The Prince Charles Hospital, Rode Road, Brisbane, QLD 4032, Australia; [c] Department of Cardiology, The Royal Melbourne Hospital, Melbourne 3050, Australia; [d] Institute of Cardiovascular Sciences, University of Manchester, Manchester, UK; [e] Department of Medicine, University of Melbourne, Royal Parade, Parkville, Victoria 3052, Australia

[1] Dr H.M. Haqqani and Dr G.M. Morris contributed equally to the drafting of this article.

* Corresponding author. Department of Cardiology, The Royal Melbourne Hospital, Melbourne 3050, Australia.

E-mail address: jon.kalman@mh.org.au

Card Electrophysiol Clin 6 (2014) 459–468
http://dx.doi.org/10.1016/j.ccep.2014.05.001
1877-9182/14/$ – see front matter

be initiated with the use of isoproterenol and only transiently suppressed with adenosine. Abrupt termination of AT with adenosine suggests triggered activity.[4] Adenosine unresponsiveness may indicate microreentry as the mechanism, particularly if long-duration fractionated electrograms are found at the site of origin.[5]

Focal ATs do not occur randomly throughout the right and left atrial chambers. Instead, they arise from stereotypical sites of anatomic and electrophysiologic heterogeneity.[6] In the right atrium, the crista terminalis is most frequent, followed by the tricuspid annulus, coronary sinus (CS) ostium, right atrial appendage, and perinodal region.[7–10] The pulmonary veins, mitral annulus (usually the aortomitral continuity), left atrial appendage, CS body, and left septal region are the most common sites of origin in the left atrium.[11–15] The septal, perinodal region is particularly complex because several structures are located in close proximity, including the AV annuli, fossa ovalis, and the noncoronary aortic sinus of Valsalva. Some focal ATs arise from or may be ablated via the latter structure.[16] Multiple focal ATs may be seen in the same patient.[17]

ECG CHARACTERISTICS OF FOCAL AT

The P wave on the surface ECG during focal AT commences when a sufficient mass of atrial myocardium has been depolarized and continues until both atria are fully activated. Unlike macroreentrant ATs (atrial flutters), there is a diastolic period of quiescent atrial activity that accounts for most of the tachycardia cycle length. The vector, duration, and sequence of atrial depolarization determine the P-wave morphology (PWM) on the ECG. These are, in turn, determined by the location of the focus of origin and by the centrifugal wavefront propagation characteristics away from that focus. Additional factors that can influence the PWM include ECG electrode positioning, antiarrhythmic drugs, surgical or spontaneous atrial scars, and translational, rotational, or attitudinal variation in the normal cardio-thoracic anatomic relationship. In the absence of structural heart disease, the PWM represents a reliable guide to the general region of AT origin.[18]

GENERAL PRINCIPLES SURROUNDING THE ECG IN FOCAL AT

- Focal AT presents as a narrow complex tachycardia on the surface ECG.
- Spontaneous or induced AV block excludes orthodromic reciprocating tachycardia (ORT) mediated by a bypass tract and makes the diagnosis of AV nodal reentrant tachycardia (AVNRT) less likely.
- The sinus rhythm ECG is likely to be normal with no preexcitation.
- The PWM in tachycardia is described as positive, negative, or isoelectric, and is monophasic or multiphasic. Notching is also described.
- The initial P-wave vector is a vital component of the overall PWM and every effort should be made to induce an isoelectric interval following the preceding T wave by the use of carotid sinus massage, intravenous adenosine, or transient ventricular burst pacing before analyzing the P wave (Fig. 1).

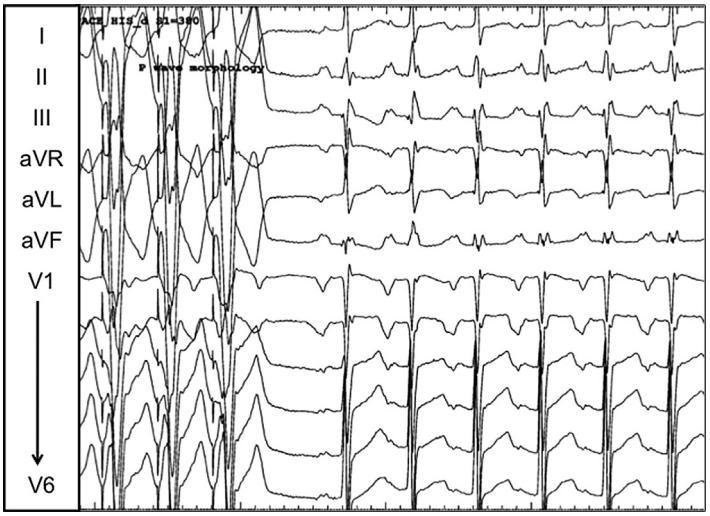

Fig. 1. Ventricular burst pacing causing retrograde AV nodal concealment and increased AV block during AT. This maneuver can be used to ensure an adequate preceding isoelectric interval that does not distort the initial P-wave vector.