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and should be a part of a treatment strategy to reduce symptoms. While it is sometimes difficult to differentiate inappropriate sinus tachycardia from POTS, recognition of these distinct clinical syndromes is critical for treatment. Sinus node modification will be ineffective for the treatment of POTS and will possibly exacerbate the nontachycardia components of the condition. Likewise, treatment strategies aimed at increasing blood pressure will not be appropriate for inappropriate sinus tachycardia. ■ ■ FURTHER READING Mayuga KA et al: Sinus tachycardia: A multidisciplinary expert focused review. *Circ Arrhythm Electrophysiol* 15:e007960, 2022. Vernino S et al: Postural orthostatic tachycardia syndrome (POTS): State of the science and clinical care from a 2019 National Institutes of Health Expert Consensus Meeting-Part 1. *Auton Neurosci* 235:102828, 2021. William H. Sauer, Paul C. Zei

Focal Atrial Tachycardia The underlying mechanisms of focal atrial tachycardia (AT) include abnormal automaticity, triggered automaticity, or a small reentry circuit in diseased atrial tissue. The term focal is used to differentiate this form of AT from typical and atypical atrial flutter but does not define a mechanism of the arrhythmia. ATs can originate from most regions of the atria, including atrial tissue extending into a pulmonary vein, the coronary sinus, or vena cava. It can be sustained, nonsustained, paroxysmal, or incessant. Focal AT accounts for ~10% of paroxysmal supraventricular tachycardia (PSVTs) in patients referred for catheter ablation. Nonsustained focal AT is commonly observed on ambulatory electrocardiogram (ECG) recordings, and the prevalence increases with age. Asymptomatic nonsustained ATs are often labeled as “SVT” on monitor reports, prompting patients to seek advice on catheter ablation. However, treatment is not recommended for asymptomatic nonsustained AT identified on ECG monitoring. Frequent atrial ectopy and nonsustained AT are often precursors to more significant arrhythmias such as atrial fibrillation and atrial flutter. Occasionally, nonsustained, frequent atrial ectopy or short bursts of AT may be symptomatic and require therapy similar to that required for focal AT (Fig. 255-1). AT AVNRT • AV node reentry AVRT A B FIGURE 255-1 Common mechanisms underlying paroxysmal supraventricular tachycardia along with typical R-P relationships. A. Schematic showing a four-chamber view of the heart with atrioventricular (AV) node and specialized conduction tissue (His-Purkinje) in yellow. Atrial tachycardia (AT; red circuit) is confined completely to atrial tissue. Atrioventricular nodal reentry tachycardia (AVNRT; green circuit) uses AV nodal and perinodal atrial tissue. Atrioventricular reentry tachycardia (AVRT; blue circuit) uses atrial and ventricular tissue, accessory pathway between the ventricle and atrium, AV node, and His-Purkinje tissue as part of the reentry circuit. B. Typical relation of the P wave to QRS, commonly described as the R-P to P-R

relationship, for the different tachycardia mechanisms.

AT can occur in the absence of structural heart disease or may be associated with any condition that causes atrial fibrosis, including atrial tissue targeted with prior catheter ablation. Areas of fibrosis can act as a nidus for abnormal automaticity from injured but partially living cells or microreentry in zones of slow conduction within and on the border of fibrotic areas. Sympathetic stimulation is a promoting factor, and the emergence of AT can be a sign of underlying illness or drug toxicity. In particular, AT with atrioventricular (AV) block is characteristic of digitalis toxicity. Symptoms from AT are highly variable but similar to other supraventricular tachycardias (SVTs), and incessant AT can cause tachycardia-induced cardiomyopathy.

CHAPTER 255 Focal Atrial Tachycardia AT typically presents with 1:1 AV conduction or with AV block in a Wenckebach or fixed (e.g., 2:1 or 3:1) pattern. Because it is not dependent on AV nodal conduction, AT will not terminate with AV block, and the atrial rate will not be affected, which distinguishes AT from most AV nodal-dependent SVTs, such as AV nodal reentry and AV reentry using an accessory pathway (see below). A so-called warmup phase when the atrial activation rate increases after initiation or a cool-down phase when the rate slows prior to termination also favors AT rather than AV nodal-dependent SVT, as this is a common observation with enhanced automaticity. P waves are often discrete, with an intervening isoelectric segment, in contrast to atrial flutter and macroreentrant AT because atrial activation from a focal source occurs through a small portion of the tachycardia cycle (Fig. 255-2). When 1:1 conduction to the ventricles is present, the arrhythmia can resemble sinus tachycardia typically with a P-R interval shorter than the R-P interval, particularly when sympathetic tone results in rapid AV nodal conduction. It can be distinguished from sinus tachycardia by the P-wave morphology, which usually differs from sinus P waves depending on the location of the focus. Focal AT tends to originate in areas of complex atrial anatomy, such as the crista terminalis, valve annuli, atrial septum, and atrial muscle extending along cardiac thoracic veins (superior vena cava, coronary sinus, and pulmonary veins), and the location can often be approximated by the P-wave morphology. AT from the atrial septum will frequently have a narrower P-wave duration than sinus rhythm. AT from the left atrium will usually have a monophasic, positive P wave in lead V1 and negative P waves in I and aVL, indicating an activation wavefront away from the left atrial free wall. AT that originates from superior atrial locations, such as the superior vena cava or superior pulmonary veins, will be positive in the inferior limb leads II, III, and aVF, whereas AT from a more inferior location, such as the ostium of the coronary sinus, will inscribe negative P waves in these same leads. When the focus is in the superior aspect of the crista terminalis, close to the sinus node, however, the P wave will resemble that of sinus tachycardia. Abrupt onset and offset then favor AT rather than sinus tachycardia. Depending on the atrial rate, the P wave may fall on top of the T wave, or during 2:1 conduction, No P-wave visible. No P wave visible. $RP < PR$ $RP < PR$ • AV node reentry • AV reentry using an accessory pathway • Focal atrial tachycardia $RP > PR$ $RP > PR$ • AV reentry using an accessory pathway • AV node reentry uncommon form

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