TABLE 3 Relevant Terms and Definitions

Arrhythmia/Term	Definition
Supraventricular tachycardia (SVT)	An umbrella term used to describe tachycardias (atrial and/or ventricular rates in excess of 100 bpm at rest), the mechanism of which involves tissue from the His bundle or above. These SVTs include inappropriate sinus tachycardia, AT (including focal and multifocal AT), macroreentrant AT (including typical atrial flutter), junctional tachycardia, AVNRT, and various forms of accessory pathway-mediated reentrant tachycardias. In this guideline, the term does not include AF.
Paroxysmal supraventricular tachycardia (PSVT)	A clinical syndrome characterized by the presence of a regular and rapid tachycardia of abrupt onset and termination. These features are characteristic of AVNRT or AVRT, and, less frequently, AT. PSVT represents a subset of SVT.
Atrial fibrillation (AF)	A supraventricular arrhythmia with uncoordinated atrial activation and, consequently, ineffective atrial contraction. ECG characteristics include: 1) irregular atrial activity, 2) absence of distinct P waves, and 3) irregular R-R intervals (when atrioventricular conduction is present). AF is not addressed in this document.
Sinus tachycardia	Rhythm arising from the sinus node in which the rate of impulses exceeds 100 bpm.
• Physiologic sinus tachycardia	Appropriate increased sinus rate in response to exercise and other situations that increase sympathetic tone.
• Inappropriate sinus tachycardia	Sinus heart rate >100 bpm at rest, with a mean 24-h heart rate >90 bpm not due to appropriate physiological responses or primary causes such as hyperthyroidism or anemia.
Atrial tachycardia (AT)	
• Focal AT	An SVT arising from a localized atrial site, characterized by regular, organized atrial activity with discrete P waves and typically an isoelectric segment between P waves. At times, irregularity is seen, especially at onset ("warm-up") and termination ("warm-down"). Atrial mapping reveals a focal point of origin.
• Sinus node reentry tachycardia	A specific type of focal AT that is due to microreentry arising from the sinus node complex, characterized by abrupt onset and termination, resulting in a P-wave morphology that is indistinguishable from sinus rhythm.
• Multifocal atrial tachycardia (MAT)	An irregular SVT characterized by ≥3 distinct P-wave morphologies and/or patterns of atrial activation at different rates. The rhythm is always irregular.
Atrial flutter	
• Cavotricuspid isthmus–dependent atrial flutter: typical	Macroreentrant AT propagating around the tricuspid annulus, proceeding superiorly along the atrial septum, inferiorly along the right atrial wall, and through the cavotricuspid isthmus between the tricuspid valve annulus and the Eustachian valve and ridge. This activation sequence produces predominantly negative "sawtooth" flutter waves on the ECG in leads 2, 3, and aVF and a late positive deflection in V1. The atrial rate can be slower than the typical 300 bpm (cycle length 200 ms) in the presence of antiarrhythmic drugs or scarring. It is also known as "typical atrial flutter" or "cavotricuspid isthmus–dependent atrial flutter" or "counterclockwise atrial flutter."
• Cavotricuspid isthmus–dependent atrial flutter: reverse typical	Macroreentrant AT that propagates around in the direction reverse that of typical atrial flutter. Flutter waves typically appear positive in the inferior leads and negative in V1. This type of atrial flutter is also referred to as "reverse typical" atrial flutter or "clockwise typical atrial flutter."
• Atypical or non-cavotricuspid isthmus–dependent atrial flutter	Macroreentrant ATs that do not involve the cavotricuspid isthmus. A variety of reentrant circuits may include reentry around the mitral valve annulus or scar tissue within the left or right atrium. A variety of terms have been applied to these arrhythmias according to the re-entry circuit location, including particular forms, such as "LA flutter" and "LA macroreentrant tachycardia" or incisional atrial re-entrant tachycardia due to re-entry around surgical scars.
Junctional tachycardia	A nonreentrant SVT that arises from the AV junction (including the His bundle).
Atrioventricular nodal reentrant tachycardia (AVNRT)	A reentrant tachycardia involving 2 functionally distinct pathways, generally referred to as "fast" and "slow" pathways. Most commonly, the fast pathway is located near the apex of Koch's triangle, and the slow pathway inferoposterior to the compact AV node tissue. Variant pathways have been described, allowing for "slow-slow" AVNRT.
• Typical AVNRT	AVNRT in which a slow pathway serves as the anterograde limb of the circuit and the fast pathway serves as the retrograde limb (also called "slow-fast AVNRT").
• Atypical AVNRT	AVNRT in which the fast pathway serves as the anterograde limb of the circuit and a slow pathway serves as the retrograde limb (also called "fast-slow AV node reentry") or a slow pathway serves as the anterograde limb and a second slow pathway serves as the retrograde limb (also called "slow-slow AVNRT").
Accessory pathway	For the purpose of this guideline, an accessory pathway is defined as an extranodal AV pathway that connects the myocardium of the atrium to the ventricle across the AV groove. Accessory pathways can be classified by their location, type of conduction (decremental or nondecremental), and whether they are capable of conducting anterogradely, retrogradely, or in both directions. Of note, accessory pathways of other types (such as atriofascicular, nodo-fascicular, nodo-ventricular, and fasciculoventricular pathways) are uncommon and are discussed only briefly in this document (Section 7).
• Manifest accessory pathways	A pathway that conducts anterogradely to cause ventricular pre-excitation pattern on the ECG.
• Concealed accessory pathway	A pathway that conducts only retrogradely and does not affect the ECG pattern during sinus rhythm.

Continued on the next page

TABLE 3 Continued

Arrhythmia/Term	Definition
• Pre-excitation pattern	An ECG pattern reflecting the presence of a manifest accessory pathway connecting the atrium to the ventricle. Pre-excited ventricular activation over the accessory pathway competes with the anterograde conduction over the AV node and spreads from the accessory pathway insertion point in the ventricular myocardium. Depending on the relative contribution from ventricular activation by the normal AV nodal/His Purkinje system versus the manifest accessory pathway, a variable degree of pre-excitation, with its characteristic pattern of a short P-R interval with slurring of the initial upstroke of the QRS complex (delta wave), is observed. Pre-excitation can be intermittent or not easily appreciated for some pathways capable of anterograde conduction; this is usually associated with a low-risk pathway, but exceptions occur.
• Asymptomatic pre-excitation (isolated pre-excitation)	The abnormal pre-excitation ECG pattern in the absence of documented SVT or symptoms consistent with SVT.
• Wolff-Parkinson-White syndrome	Syndrome characterized by documented SVT or symptoms consistent with SVT in a patient with ventricular pre-excitation during sinus rhythm.
Atrioventricular reentrant tachycardia (AVRT)	A reentrant tachycardia, the electrical pathway of which requires an accessory pathway, the atrium, atrioventricular node (or second accessory pathway), and ventricle.
• Orthodromic AVRT	An AVRT in which the reentrant impulse uses the accessory pathway in the retrograde direction from the ventricle to the atrium, and the AV node in the anterograde direction. The QRS complex is generally narrow or may be wide because of pre-existing bundle-branch block or aberrant conduction.
• Antidromic AVRT	An AVRT in which the reentrant impulse uses the accessory pathway in the anterograde direction from the atrium to the ventricle, and the AV node for the retrograde direction. Occasionally, instead of the AV node, another accessory pathway can be used in the retrograde direction, which is referred to as pre-excited AVRT. The QRS complex is wide (maximally pre-excited).
Permanent form of junctional reciprocating tachycardia (PJRT)	A rare form of nearly incessant orthodromic AVRT involving a slowly conducting, concealed, usually posteroseptal accessory pathway.
Pre-excited AF	AF with ventricular pre-excitation caused by conduction over ≥1 accessory pathway(s).

AF indicates atrial fibrillation; AT, atrial tachycardia; AV, atrioventricular; AVNRT, atrioventricular nodal reentrant tachycardia; AVRT, atrioventricular reentrant tachycardia; bpm, beats per minute; ECG, electrocardiogram/ electrocardiographic; LA, left atrial; MAT, multifocal atrial tachycardia; PJRT, permanent form of junctional reciprocating tachycardia; PSVT, paroxysmal supraventricular tachycardia; SVT, supraventricular tachycardia; and WPW, Wolff-Parkinson-White.

SVT in the general population is 2.29 per 1,000 persons (32). When adjusted by age and sex in the U.S. population, the incidence of paroxysmal supraventricular tachycardia (PSVT) is estimated to be 36 per 100,000 persons per year (32). There are approximately 89,000 new cases per year and 570,000 persons with PSVT (32). Compared with patients with cardiovascular disease, those with PSVT without any cardiovascular disease are younger (37 versus 69 years; p=0.0002) and have faster PSVT (186 bpm versus 155 bpm; p=0.0006). Women have twice the risk of men of developing PSVT (32). Individuals >65 years of age have >5 times the risk of younger persons of developing PSVT (32).

Patients with PSVT who are referred to specialized centers for management with ablation are younger, have an equal sex distribution, and have a low frequency of cardiovascular disease (33,34,34–47). The frequency of atrioventricular nodal reentrant tachycardia (AVNRT) is greater in women than in men. This may be due to an actual higher incidence in women, or it may reflect referral bias. In persons who are middle-aged or older, AVNRT is more common, whereas in adolescents, the prevalence may be more balanced between atrioventricular reentrant tachycardia (AVRT) and AVNRT, or AVRT may be more prevalent (32). The relative frequency of tachycardia mediated by an accessory pathway decreases with age. The incidence of manifest pre-excitation or WPW pattern on ECG tracings in the general population is 0.1% to 0.3%. However, not all patients with manifest ventricular pre-excitation develop PSVT (47–49). The limited data on the public health impact of SVT indicate that the arrhythmia is commonly a reason for emergency department and primary care physician visits but is infrequently the primary reason for hospital admission (11,50,51).

2.3. Evaluation of the Patient With Suspected or Documented SVT

2.3.1. Clinical Presentation and Differential Diagnosis on the Basis of Symptoms

Patients seen in consultation for palpitations often describe symptoms with characteristic features suggestive of SVT that may guide physicians to appropriate testing and a definitive diagnosis. The diagnosis of SVT is often made in the emergency department, but it is common to elicit symptoms suggestive of SVT before initial electrocardiogram/electrocardiographic (ECG) documentation. SVT symptom onset often begins in adulthood; in 1 study in adults, the mean age of symptom onset was 32±18 years of age for AVNRT, versus 23±14 years of age for AVRT (52). In contrast, in a study conducted in pediatric populations, the mean ages of symptom onset of AVRT and AVNRT were 8 and 11 years, respectively (53). In comparison with AVRT, patients with AVNRT are more likely to be female, with an age of onset >30 years

TABLE 4 Differential Diagnosis of Wide-QRS Complex Tachycardia

Mechanism
Ventricular tachycardia
SVT with pre-existing bundle-branch block or intraventricular conduction defect
SVT with aberrant conduction due to tachycardia (normal QRS when in sinus rhythm)
SVT with wide QRS related to electrolyte or metabolic disorder
SVT with conduction over an accessory pathway (pre-excitation)
Paced rhythm
Artifact

SVT indicates supraventricular tachycardia.

less easily detected and can be misdiagnosed as a regular SVT (76). If the atrial rate exceeds the ventricular rate, then atrial flutter or AT (focal or multifocal) is usually present (rare cases of AVNRT with 2:1 conduction have been described (77)).

If the SVT is regular, this may represent AT with 1:1 conduction or an SVT that involves the AV node. Junctional tachycardias, which originate in the AV junction (including the His bundle), can be regular or irregular, with variable conduction to the atria. SVTs that involve the AV node as a required component of the tachycardia reentrant circuit include AVNRT (Section 6: **Figures 2 and 3**) and AVRT (Section 7: **Figures 4 and 6**). In these reentrant tachycardias, the retrogradely conducted P wave may be difficult to discern, especially if bundle-branch block is present. In typical AVNRT, atrial activation is nearly simultaneous with the QRS, so the terminal portion of the P wave is usually located at the end of the QRS complex, appearing as a narrow and negative deflection in the inferior leads (a pseudo S wave) and a slightly positive deflection at the end of the QRS complex in lead V1 (pseudo R′). In orthodromic AVRT (with anterograde conduction down the AV node), the P wave can usually be seen in the early part of the ST-T segment. In typical forms of AVNRT and AVRT, because the P wave is located closer to the prior QRS complex than the subsequent QRS complex, the tachycardias are referred to as having a "short RP." They also have a 1:1 relationship between the P wave and QRS complex, except in rare cases of AVNRT in which 2:1 AV block or various degrees of AV block can occur. In unusual cases of AVNRT (such as "fast-slow"), the P wave is closer to the subsequent QRS complex, providing a long RP. The RP is also long during an uncommon form of AVRT, referred to as the permanent form of junctional reciprocating tachycardia (PJRT), in which an unusual accessory bypass tract with "decremental" (slowly conducting) retrograde conduction during orthodromic AVRT produces delayed atrial activation and a long RP interval.

A long RP interval is typical of AT because the rhythm is driven by the atrium and conducts normally to the ventricles. In AT, the ECG will typically show a P wave with a morphology that differs from sinus that is usually seen near the end of or shortly after the T wave (**Figure 5**). In sinus node reentry tachycardia, a form of focal AT, the P-wave morphology is identical to the P wave in sinus rhythm.

2.4. Principles of Medical Therapy

See **Figure 8** for the algorithm for acute treatment of tachycardia of unknown mechanism; **Figure 9** for the algorithm for ongoing management of tachycardia of unknown mechanism; **Table 6** for acute drug therapy for SVT (intravenous administration); and **Table 7** for ongoing drug therapy for SVT (oral administration).

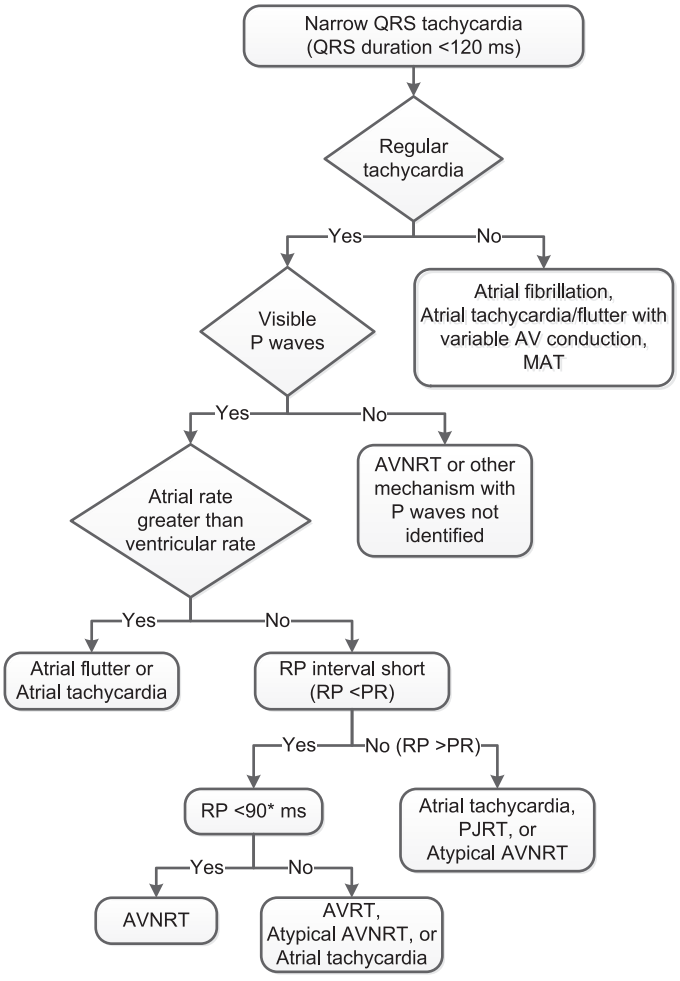
TABLE 5 ECG Criteria to Differentiate VT From SVT in Wide-Complex Tachycardia

Findings or Leads on ECG Assessed	Interpretation
QRS complex in leads V1-V6 (Brugada criteria) (73)	■ Lack of any R-S complexes implies VT ■ R-S interval (onset of R wave to nadir of S wave) >100 ms in any precordial lead implies VT
QRS complex in aVR (Vereckei algorithm) (74)	■ Presence of initial R wave implies VT ■ Initial R or Q wave >40 ms implies VT ■ Presence of a notch on the descending limb at the onset of a predominantly negative QRS implies VT
AV dissociation*	■ Presence of AV dissociation (with ventricular rate faster than atrial rate) or fusion complexes implies VT
QRS complexes in precordial leads all positive or all negative (concordant)	■ Implies VT
QRS in tachycardia that is identical to sinus rhythm (78)	■ Suggests SVT
R-wave peak time in lead II (78)	■ R-wave peak time ≥50 ms suggests VT

*AV dissociation is also a component of the Brugada criteria (73).

AV indicates atrioventricular; ECG, electrocardiogram; SVT, supraventricular tachycardia; and VT, ventricular tachycardia.

FIGURE 7 Differential Diagnosis for Adult Narrow QRS Tachycardia



Patients with junctional tachycardia may mimic the pattern of slow-fast AVNRT and may show AV dissociation and/or marked irregularity in the junctional rate.

*RP refers to the interval from the onset of surface QRS to the onset of visible P wave (note that the 90-ms interval is defined from the surface ECG [79], as opposed to the 70-ms ventriculoatrial interval that is used for intracardiac diagnosis [80]).

AV indicates atrioventricular; AVNRT, atrioventricular nodal reentrant tachycardia; AVRT, atrioventricular reentrant tachycardia; ECG, electrocardiogram; MAT, multifocal atrial tachycardia; and PJRT, permanent form of junctional reentrant tachycardia.

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2.4.1. Acute Treatment: Recommendations

Because patients with SVT account for approximately 50,000 emergency department visits each year (81), emergency physicians may be the first to evaluate patients whose tachycardia mechanism is unknown and to have the opportunity to diagnose the mechanism of arrhythmia. It is important to record a 12-lead ECG to differentiate tachycardia mechanisms according to whether the AV node is an obligate component (Section 2.3.2), because treatment that targets the AV node will not reliably terminate tachycardias that are not AV node dependent. Also, if the QRS duration is >120 ms, it is crucial to distinguish VT from SVT with aberrant conduction, pre-existing bundle-branch block, or pre-excitation (**Table 4**). In particular, the administration of verapamil or diltiazem for treatment of either VT or a pre-excited AF may lead to hemodynamic compromise or may accelerate the ventricular rate and lead to ventricular fibrillation.

FIGURE 17 Classification of Atrial Flutter/Atrial Tachycardias

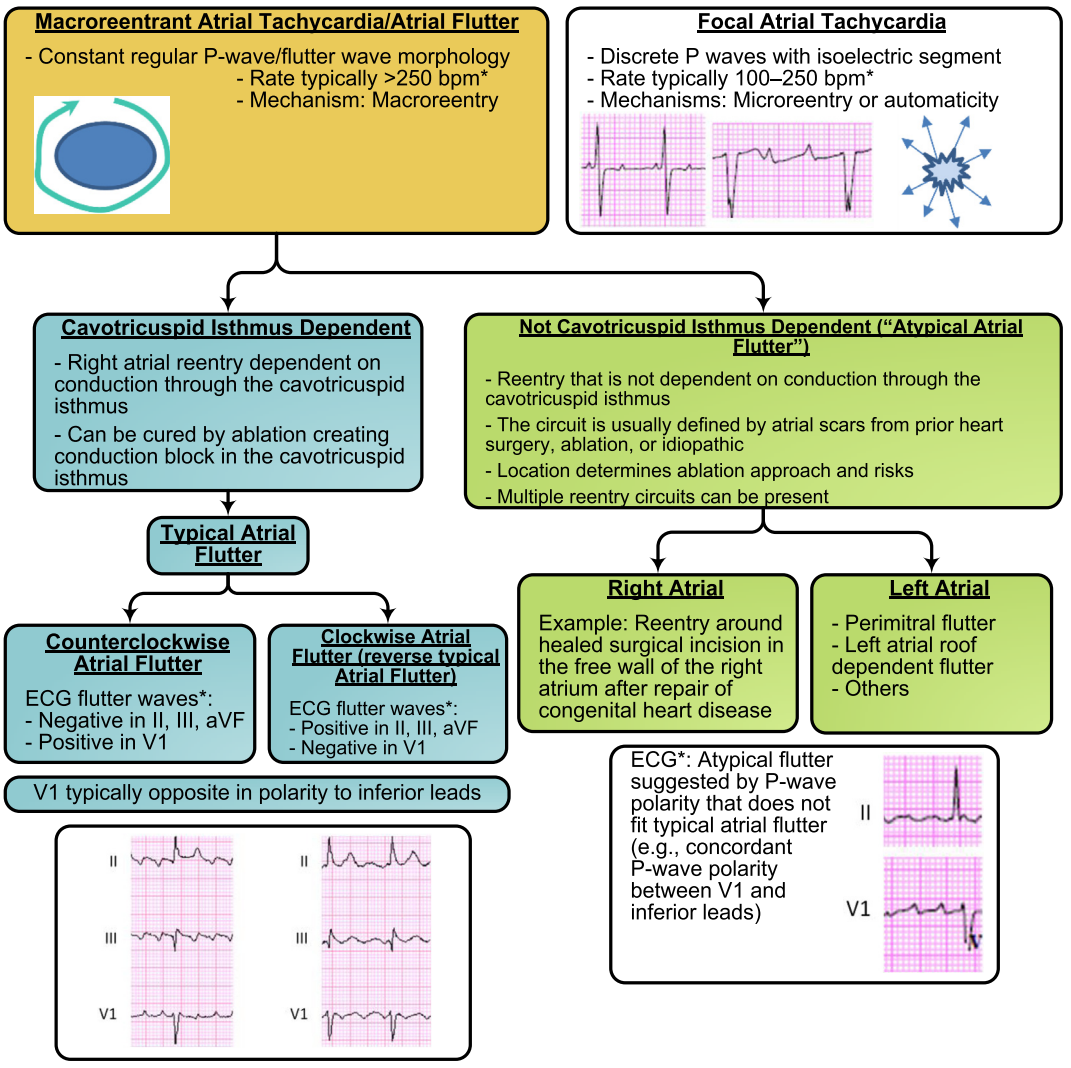

Diagram summarizing types of ATs often encountered in patients with a history of atrial fibrillation, including those seen after catheter or surgical ablation procedures. P-wave morphologies are shown for common types of atrial flutter; however, the P-wave morphology is not always a reliable guide to the re-entry circuit location or to the distinction between common atrial flutter and other macroreentrant ATs.

*Exceptions to P-wave morphology and rate are common in scarred atria.

bpm indicates beats per minute, and ECG, electrocardiogram.

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to 350 bpm (**Figure 17**). Clockwise isthmus-dependent flutter shows the opposite pattern (i.e., positive flutter waves in the inferior leads and wide, negative flutter waves in lead V1) (**Figure 17**). Although the atrial rates for flutter typically range from 250 bpm to 330 bpm, the rates may be slower in patients with severe atrial disease or in patients taking antiarrhythmic agents or after unsuccessful catheter ablation (310).

Atrial flutter can occur in clinical settings similar to those associated with AF, and atrial flutter can be triggered by AT or AF (121,311). It is common for AF and atrial flutter to coexist in the same patient. After CTI ablation, 22% to 50% of patients have been reported to develop AF after a mean follow-up of 14 to 30 months, although 1 study reported a much higher rate of AF development, with 82% of patients treated by catheter ablation for atrial flutter manifesting AF within 5 years (312). Risk factors for the manifesting AF after atrial flutter ablation include prior AF, depressed left ventricular function, structural heart disease or ischemic heart disease, inducible AF, and increased LA size (121,312–316).