2015 ACC/AHA/HRS Guideline for the Management of Adult Patients With Supraventricular Tachycardia: Executive Summary

A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society

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Preamble

Since 1980, the American College of Cardiology (ACC) and American Heart Association (AHA) have translated scientific evidence into clinical practice guidelines with recommendations to improve cardiovascular health. These guidelines, based on systematic methods to evaluate and classify evidence, provide a cornerstone of quality cardiovascular care.

In response to reports from the Institute of Medicine[1,2] and a mandate to evaluate new knowledge and maintain relevance at the point of care, the ACC/AHA Task Force on Clinical Practice Guidelines (Task Force) modified its methodology.[3–5] The relationships between guidelines, data standards, appropriate use criteria, and performance measures are addressed elsewhere.[4]

Intended Use

Practice guidelines provide recommendations applicable to patients with or at risk of developing cardiovascular disease. The focus is on medical practice in the United States, but guidelines developed in collaboration with other organizations may have a broader target. Although guidelines may inform regulatory or payer decisions, they are intended to improve quality of care in the interest of patients.

Evidence Review

Guideline Writing Committee (GWC) members review the literature; weigh the quality of evidence for or against particular tests, treatments, or procedures; and estimate expected health outcomes. In developing recommendations, the GWC uses evidence-based methodologies that are based on all available data.[4–6] Literature searches focus on randomized controlled trials (RCTs) but also include registries, nonrandomized comparative and descriptive studies, case series, cohort studies, systematic reviews, and expert opinion. Only selected references are cited.

The Task Force recognizes the need for objective, independent Evidence Review Committees (ERCs) that include methodologists, epidemiologists, clinicians, and biostatisticians

who systematically survey, abstract, and assess the evidence to address key clinical questions posed in the PICOTS format (P=population, I=intervention, C=comparator, O=outcome, T=timing, S=setting).[4,5] Practical considerations, including time and resource constraints, limit the ERCs to evidence that is relevant to key clinical questions and lends itself to systematic review and analysis that could affect the strength of corresponding recommendations. Recommendations developed by the GWC on the basis of the systematic review are marked "SR".

Guideline-Directed Medical Therapy

The term "guideline-directed medical therapy" refers to care defined mainly by ACC/AHA Class I recommendations. For these and all recommended drug treatment regimens, the reader should confirm dosage with product insert material and carefully evaluate for contraindications and interactions. Recommendations are limited to treatments, drugs, and devices approved for clinical use in the United States.

Class of Recommendation and Level of Evidence

The Class of Recommendation (COR; ie, the strength of the recommendation) encompasses the anticipated magnitude and certainty of benefit in proportion to risk. The Level of Evidence (LOE) rates evidence supporting the effect of the intervention on the basis of the type, quality, quantity, and consistency of data from clinical trials and other reports (Table 1).[5,7] Unless otherwise stated, recommendations are sequenced by COR and then by LOE. Where comparative data exist, preferred strategies take precedence. When >1 drug, strategy, or therapy exists within the same COR and LOE and no comparative data are available, options are listed alphabetically. Each recommendation is followed by supplemental text linked to supporting references and evidence tables.

Relationships With Industry and Other Entities

The ACC and AHA sponsor the guidelines without commercial support, and members volunteer their time. The Task Force zealously avoids actual, potential, or perceived conflicts of interest that might arise through relationships with industry or other entities (RWI). All GWC members and reviewers are required to disclose current industry relationships or personal interests from 12 months before initiation of the writing effort. Management of RWI involves selecting a balanced GWC and assuring that the chair and a majority of committee members have no relevant RWI (Appendix 1). Members are restricted with regard to writing or voting on sections to which their RWI apply. For transparency, members' comprehensive disclosure information is available online. Comprehensive disclosure information for the Task Force is also available online. The Task Force strives to avoid bias by selecting experts from a broad array of backgrounds representing different geographic regions, sexes, ethnicities, intellectual perspectives/biases, and scopes of clinical practice, and by inviting organizations and professional societies with related interests and expertise to participate as partners or collaborators.

Individualizing Care in Patients With Associated Conditions and Comorbidities

Managing patients with multiple conditions can be complex, especially when recommendations applicable to coexisting illnesses are discordant or interacting.[8] The guidelines are intended to define practices meeting the needs of patients in most, but not all, circumstances. The recommendations should not replace clinical judgment.

Clinical Implementation

Management in accordance with guideline recommendations is effective only when followed. Adherence to recommendations can be enhanced by shared decision making between clinicians and patients, with patient engagement in selecting interventions based on individual values, preferences, and associated conditions and comorbidities. Consequently, circumstances may arise in which deviations from these guidelines are appropriate.

Policy

The recommendations in this guideline represent the official policy of the ACC and AHA until superseded by published addenda, statements of clarification, focused updates, or revised full-text guidelines. To ensure that guidelines remain current, new data are reviewed biannually to determine whether recommendations should be modified. In general, full revisions are posted in 5-year cycles.[3,5]

The reader is encouraged to consult the full-text guideline[9] for additional guidance and details with regard to SVT because the executive summary contains limited information.

Jonathan L. Halperin, MD, FACC, FAHA
Chair, ACC/AHA Task Force on Clinical Practice Guidelines

1. Introduction

1.1. Methodology and Evidence Review

The recommendations listed in this guideline are, whenever possible, evidence based. An extensive evidence review was conducted in April 2014 that included literature published through September 2014. Other selected references published through May 2015 were incorporated by the GWC. Literature included was derived from research involving human subjects, published in English, and indexed in MEDLINE (through PubMed), EMBASE, the Cochrane Library, the Agency for Healthcare Research and Quality, and other selected databases relevant to this guideline. The relevant search terms and data are included in evidence tables in the Online Data Supplement. Additionally, the GWC reviewed documents related to supraventricular tachycardia (SVT) previously published by the ACC, AHA, and Heart Rhythm Society (HRS). References selected and published in this document are representative and not all-inclusive.

An independent ERC was commissioned to perform a systematic review of key clinical questions, the results of which were considered by the GWC for incorporation into this guideline. The systematic review report on the management of asymptomatic patients with Wolff-Parkinson-White (WPW) syndrome is published in conjunction with this guideline.[10]

1.2. Organization of the GWC

The GWC consisted of clinicians, cardiologists, electrophysiologists (including those specialized in pediatrics), and a nurse

Table 1. Applying Class of Recommendation and Level of Evidence to Clinical Strategies, Interventions, Treatments, or Diagnostic Testing in Patient Care*

(in the role of patient representative) and included representatives from the ACC, AHA, and HRS.

1.3. Document Review and Approval

This document was reviewed by 8 official reviewers nominated by the ACC, AHA, and HRS, and 25 individual content reviewers. Reviewers' RWI information was distributed to the GWC and is published in this document (Appendix 2).

This document was approved for publication by the governing bodies of the ACC, the AHA, and the HRS.

1.4. Scope of the Guideline

The purpose of this joint ACC/AHA/HRS document is to provide a contemporary guideline for the management of adults with all types of SVT other than atrial fibrillation (AF). Although AF is, strictly speaking, an SVT, the term SVT generally does not refer to AF. AF is addressed in the 2014 ACC/AHA/HRS Guideline for the Management of Atrial Fibrillation (2014 AF guideline).[11] The present guideline addresses other SVTs, including regular narrow–QRS complex tachycardias, as well as other, irregular SVTs (eg, atrial flutter with irregular ventricular response and

multifocal atrial tachycardia [MAT]). This guideline supersedes the "2003 ACC/AHA/ESC Guidelines for the Management of Patients With Supraventricular Arrhythmias."[12] Although this document is aimed at the adult population (≥18 years of age) and offers no specific recommendations for pediatric patients, as per the reference list, we examined literature that included pediatric patients. In some cases, the data from noninfant pediatric patients helped inform this guideline.

2. General Principles

2.1. Mechanisms and Definitions

For the purposes of this guideline, SVT is defined as per Table 2, which provides definitions and the mechanism(s) of each type of SVT. The term SVT does not generally include AF, and this document does not discuss the management of AF.

2.2. Epidemiology, Demographics, and Public Health Impact

The best available evidence indicates that the prevalence of SVT in the general population is 2.29 per 1000 persons.[13] When adjusted by age and sex in the US population, the incidence of paroxysmal supraventricular tachycardia (PSVT) is estimated to be 36 per 100 000 persons per year.[13] There are approximately 89 000 new cases per year and 570 000 persons with PSVT.[13] 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 versus 155 bpm; P=0.0006). Women have twice the risk of men of developing PSVT.[13] Individuals >65 years of age have >5 times the risk of younger persons of developing PSVT.[13]

Table 2. 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 reentry circuit location, including particular forms, such as "LA flutter" and "LA macroreentrant tachycardia" or incisional atrial reentrant tachycardia due to reentry around surgical scars.

(*Continued*)

Table 2. Continued

Arrhythmia/Term	Definition
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.
• 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 (WPW) 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.

Atrioventricular nodal reentrant tachycardia (AVNRT) is more common in persons who are middle-aged or older, whereas in adolescents the prevalence may be more balanced between atrioventricular reentrant tachycardia (AVRT) and AVNRT, or AVRT may be more prevalent.[13] The relative frequency of tachycardia mediated by an accessory pathway decreases with age. The incidence of manifest pre-excitation or WPW pattern on electrocardiogram/electrocardiographic (ECG) tracings in the general population is 0.1% to 0.3%. However, not all patients with manifest ventricular pre-excitation develop PSVT.[14–16]

2.3. Evaluation of the Patient With Suspected or Documented SVT

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

The diagnosis of SVT is often made in the emergency department, but it is common to elicit symptoms suggestive of

SVT before initial electrocardiographic documentation. SVT symptom onset often begins in adulthood; in one 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.[17] 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.[18] In comparison with AVRT, patients with AVNRT are more likely to be female, with an age of onset >30 years.[16,19–21]

SVT has an impact on quality of life, which varies according to the frequency of episodes, the duration of SVT, and whether symptoms occur not only with exercise but also at rest.[18,22] In 1 retrospective study in which the records of patients <21 years of age with WPW pattern on the ECG were reviewed, 64% of patients had symptoms at presentation, and an additional 20% developed symptoms during follow-up.[23] Modes of presentation included documented SVT in 38%, palpitations in 22%, chest pain in 5%, syncope in 4%, AF in 0.4%, and sudden cardiac death (SCD) in 0.2%.[23] A confounding factor in diagnosing SVT is the need to differentiate symptoms of SVT from symptoms of panic and anxiety disorders or any condition of heightened awareness of sinus tachycardia (such as postural orthostatic tachycardia syndrome). When AVNRT and AVRT are compared, symptoms appear to differ substantially. Patients with AVNRT more frequently describe symptoms of "shirt flapping" or "neck pounding"[19,24] that may be related to pulsatile reversed flow when the right atrium contracts against a closed tricuspid valve (cannon a-waves).

True syncope is infrequent with SVT, but complaints of light-headedness are common. In patients with WPW syndrome, syncope should be taken seriously but is not necessarily associated with increased risk of SCD.[25] The rate of AVRT is faster when AVRT is induced during exercise,[26] yet the rate alone does not explain symptoms of near-syncope. Elderly patients with AVNRT are more prone to syncope or near-syncope than are younger patients, but the tachycardia rate is generally slower in the elderly.[27,28]

In a study on the relationship of SVT with driving, 57% of patients with SVT experienced an episode while driving, and 24% of these considered it to be an obstacle to driving.[29] This sentiment was most common in patients who had experienced syncope or near-syncope. Among patients who experienced SVT while driving, 77% felt fatigue, 50% had symptoms of near-syncope, and 14% experienced syncope. Women had more symptoms in each category.

2.3.2. Evaluation of the ECG

A 12-lead ECG obtained during tachycardia and during sinus rhythm may reveal the etiology of tachycardia. For the patient who describes prior, but not current, symptoms of palpitations, the resting ECG can identify pre-excitation that should prompt a referral to a cardiac electrophysiologist.

For a patient presenting in SVT, the 12-lead ECG can potentially identify the arrhythmia mechanism (Figure 1). If the SVT is regular, this may represent AT with 1:1 conduction or an SVT that involves the atrioventricular (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) and AVRT (Section 7). 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." In unusual

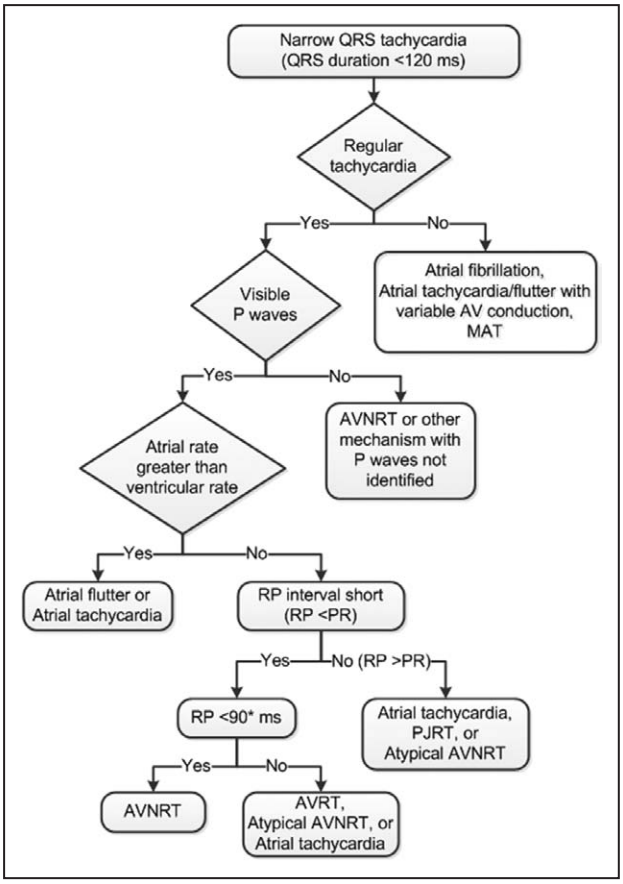

Figure 1. Differential diagnosis for adult narrow QRS tachycardia. Patients with junctional tachycardia may mimic the pattern of slow-fast AVNRT and may show AV dissociation and/or marked irregularity in the junctional rate. *RP refers to the interval from the onset of surface QRS to the onset of visible P wave (note that the 90-ms interval is defined from the surface ECG,[30] as opposed to the 70-ms ventriculoatrial interval that is used for intracardiac diagnosis).[31] 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. Modified with permission from Blomström-Lundqvist et al.[12]

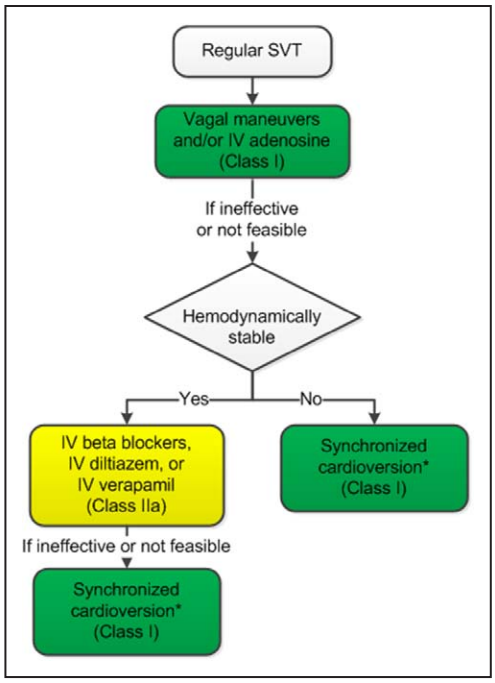

Figure 2. Acute treatment of regular SVT of unknown mechanism. Colors correspond to Class of Recommendation in Table 1; drugs listed alphabetically. *For rhythms that break or recur spontaneously, synchronized cardioversion is not appropriate. IV indicates intravenous; and SVT, supraventricular tachycardia.

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 the P wave in sinus rhythm. In sinus node re-entry 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 2 for the algorithm for acute treatment of tachycardia of unknown mechanism and Figure 3 for the algorithm for ongoing management of tachycardia of unknown mechanism. See Appendix 1 in the Online Data Supplement for a table of acute drug therapy for SVT (intravenous administration), Appendix 2 for a table of ongoing drug therapy for SVT (oral administration), and Online Data Supplements 1 to 3 for data supporting Section 2.

2.4.1. Acute Treatment: Recommendations

Because patients with SVT account for approximately 50 000 emergency department visits each year,[32] 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

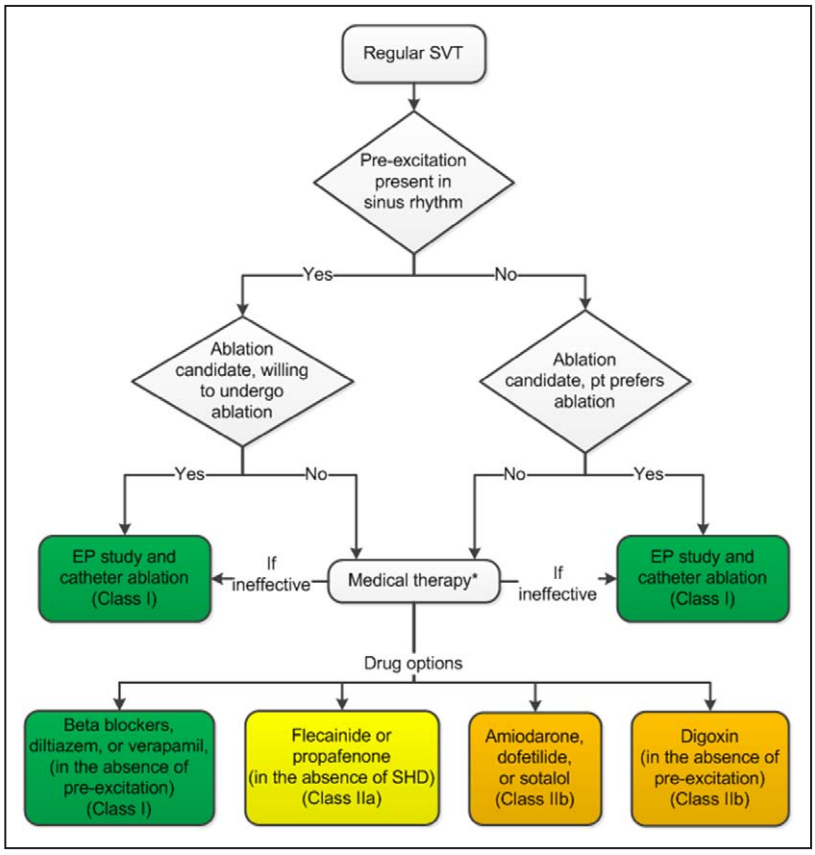

Figure 3. Ongoing management of SVT of unknown mechanism. Colors correspond to Class of Recommendation in Table 1; drugs listed alphabetically. *Clinical follow-up without treatment is also an option. EP indicates electrophysiological; pt, patient; SHD, structural heart disease (including ischemic heart disease); and SVT, supraventricular tachycardia.

component (Section 2.3.2), because treatment that targets the AV node will not reliably terminate tachycardias that are not AV node dependent.

Recommendations for Acute Treatment of SVT of Unknown Mechanism

COR	LOE	Recommendations
I	B-R	1. Vagal maneuvers are recommended for acute treatment in patients with regular SVT.[33–35]
I	B-R	2. Adenosine is recommended for acute treatment in patients with regular SVT.[34,36–43]
I	B-NR	3. Synchronized cardioversion is recommended for acute treatment in patients with hemodynamically unstable SVT when vagal maneuvers or adenosine are ineffective or not feasible.[44]
I	B-NR	4. Synchronized cardioversion is recommended for acute treatment in patients with hemodynamically stable SVT when pharmacological therapy is ineffective or contraindicated.[36,45]
IIa	B-R	1. Intravenous diltiazem or verapamil can be effective for acute treatment in patients with hemodynamically stable SVT.[36,39,42,46]
IIa	C-LD	2. Intravenous beta blockers are reasonable for acute treatment in patients with hemodynamically stable SVT.[47]

2.4.2. Ongoing Management: Recommendations

The recommendations and algorithm (Figure 3) for ongoing management, along with other recommendations and algorithms for specific SVTs that follow, are meant to include consideration of patient preferences and clinical judgment; this may include consideration of consultation with a cardiologist or clinical cardiac electrophyisiologist, as well as patient comfort with possible invasive diagnostic and therapeutic intervention. Recommendations for treatment options (including drug therapy, ablation, or observation) must be considered in the context of frequency and duration of the SVT, along with clinical manifestations, such as symptoms or adverse consequences (eg, development of cardiomyopathy).

Recommendations for Ongoing Management of SVT of Unknown Mechanism

COR	LOE	Recommendations
I	B-R	1. Oral beta blockers, diltiazem, or verapamil is useful for ongoing management in patients with symptomatic SVT who do not have ventricular pre-excitation during sinus rhythm.[48–50]
I	B-NR	2. Electrophysiological (EP) study with the option of ablation is useful for the diagnosis and potential treatment of SVT.[51–58]
I	C-LD	3. Patients with SVT should be educated on how to perform vagal maneuvers for ongoing management of SVT.[33]
IIa	B-R	1. Flecainide or propafenone is reasonable for ongoing management in patients without structural heart disease or ischemic heart disease who have symptomatic SVT and are not candidates for, or prefer not to undergo, catheter ablation.[48,59–65]
IIb	B-R	1. Sotalol may be reasonable for ongoing management in patients with symptomatic SVT who are not candidates for, or prefer not to undergo, catheter ablation.[66]
IIb	B-R	2. Dofetilide may be reasonable for ongoing management in patients with symptomatic SVT who are not candidates for, or prefer not to undergo, catheter ablation and in whom beta blockers, diltiazem, flecainide, propafenone, or verapamil are ineffective or contraindicated.[59]
IIb	C-LD	3. Oral amiodarone may be considered for ongoing management in patients with symptomatic SVT who are not candidates for, or prefer not to undergo, catheter ablation and in whom beta blockers, diltiazem, dofetilide, flecainide, propafenone, sotalol, or verapamil are ineffective or contraindicated.[67]
IIb	C-LD	4. Oral digoxin may be reasonable for ongoing management in patients with symptomatic SVT without pre-excitation who are not candidates for, or prefer not to undergo, catheter ablation.[50]

2.5. Basic Principles of Electrophysiological Study, Mapping, and Ablation

An invasive EP study permits the precise diagnosis of the underlying arrhythmia mechanism and localization of the site of origin and provides definitive treatment if coupled with catheter ablation. There are standards that define the equipment and training of personnel for optimal performance of EP study.[68] EP studies involve placement of multielectrode catheters in the heart at ≥1 sites in the atria, ventricles, or coronary sinus. Pacing and programmed electrical stimulation may be performed with or without pharmacological provocation. By using diagnostic maneuvers during the EP study, the mechanism of SVT can be defined in most cases.[31,69] Complications of diagnostic EP studies are rare but can be life threatening.[70]

A table of success and complication rates for ablation of SVT is included in the full-text guideline and in the Online Data Supplement–Appendix 3. Cardiac mapping is performed during EP studies to identify the site of origin of an arrhythmia or areas of critical conduction to allow targeting of ablation. Multiple techniques have been developed to characterize the temporal and spatial distribution of electrical activation.[71]

Several tools have been developed to facilitate arrhythmia mapping and ablation, including electroanatomic 3-dimensional mapping and magnetic navigation. Potential benefits of these technologies include more precise definition or localization of arrhythmia mechanism, spatial display of catheters and arrhythmia activation, reduction in fluoroscopy exposure for the patient and staff, and shortened procedure times, particularly for complex arrhythmias or anatomy.[72]

Fluoroscopy has historically been the primary imaging modality used for EP studies. Attention to optimal fluoroscopic technique and adoption of radiation-reducing strategies can minimize radiation dose to the patient and operator. The current standard is to use the "as low as reasonably achievable" (ALARA) principle on the assumption that there is no

threshold below which ionizing radiation is free from harmful biological effect. Alternative imaging systems, such as electroanatomic mapping and intracardiac echocardiography, have led to the ability to perform SVT ablation with no or minimal fluoroscopy, with success and complication rates similar to standard techniques.[73–77] A reduced-fluoroscopy approach is particularly important in pediatric patients and during pregnancy.[78,79]

Radiofrequency current is the most commonly used energy source for SVT ablation.[80] Cryoablation is used as an alternative to radiofrequency ablation to minimize injury to the AV node during ablation of specific arrhythmias, such as AVNRT, para-Hisian AT, and para-Hisian accessory pathways, particularly in specific patient populations, such as children and young adults. Selection of the energy source depends on operator experience, arrhythmia target location, and patient preference.

3. Sinus Tachyarrhythmias

In normal individuals, the sinus rate at rest is generally between 50 bpm and 90 bpm, reflecting vagal tone.[81–84] Sinus tachycardia refers to the circumstance in which the sinus rate exceeds 100 bpm. On the ECG, the P wave is upright in leads I, II, and aVF and is biphasic in lead V1.

3.1. Physiological Sinus Tachycardia

Physiological sinus tachycardia may result from pathological causes, including infection with fever, dehydration, anemia, heart failure, and hyperthyroidism, in addition to exogenous substances, including caffeine, drugs with a beta-agonist effect (eg, albuterol, salmeterol), and illicit stimulant drugs (eg, amphetamines, cocaine). In these cases, tachycardia is expected to resolve with correction of the underlying cause.

3.2. Inappropriate Sinus Tachycardia

Inappropriate sinus tachycardia (IST) is defined as sinus tachycardia that is unexplained by physiological demands. Crucial to this definition is the presence of associated, sometimes debilitating, symptoms that include weakness, fatigue, lightheadedness, and uncomfortable sensations, such as heart racing. Patients with IST commonly show resting heart rates >100 bpm and average rates that are >90 bpm in a 24-hour period.[81] The cause of IST is unclear, and mechanisms related to dysautonomia, neurohormonal dysregulation, and intrinsic sinus node hyperactivity have been proposed.

It is important to distinguish IST from secondary causes of tachycardia, including hyperthyroidism, anemia, dehydration, pain, and use of exogenous substances. Anxiety is also an important trigger, and patients with IST may have associated anxiety disorders.[81] IST must also be distinguished from other forms of tachycardia, including AT arising from the superior aspect of the crista terminalis and sinus node reentrant tachycardia (Section 4). It is also important to distinguish IST from postural orthostatic tachycardia syndrome, although overlap may be present within an individual. Patients with postural orthostatic tachycardia syndrome have predominant symptoms related to a change in posture, and treatment to suppress the sinus rate may lead to severe orthostatic hypotension. Thus, IST is a diagnosis of exclusion.

3.2.1. Acute Treatment

There are no specific recommendations for acute treatment of IST.

3.2.2. Ongoing Management: Recommendations

Because the prognosis of IST is generally benign, treatment is for symptom reduction and may not be necessary. Treatment of IST is difficult, and it should be recognized that lowering the heart rate may not alleviate symptoms. Therapy with beta blockers or calcium channel blockers is often ineffective or not well tolerated because of cardiovascular side effects, such as hypotension. Exercise training may be of benefit, but the benefit is unproven.

Recommendations for Ongoing Management of IST

COR	LOE	Recommendations
I	C-LD	1. Evaluation for and treatment of reversible causes are recommended in patients with suspected IST.[81,101]
IIa	B-R	1. Ivabradine is reasonable for ongoing management in patients with symptomatic IST.[85–93]
IIb	C-LD	1. Beta blockers may be considered for ongoing management in patients with symptomatic IST.[87,89]
IIb	C-LD	2. The combination of beta blockers and ivabradine may be considered for ongoing management in patients with IST.[89]

Ivabradine is an inhibitor of the "I-funny" or "I_f" channel, which is responsible for normal automaticity of the sinus node; therefore, ivabradine reduces the sinus node pacemaker

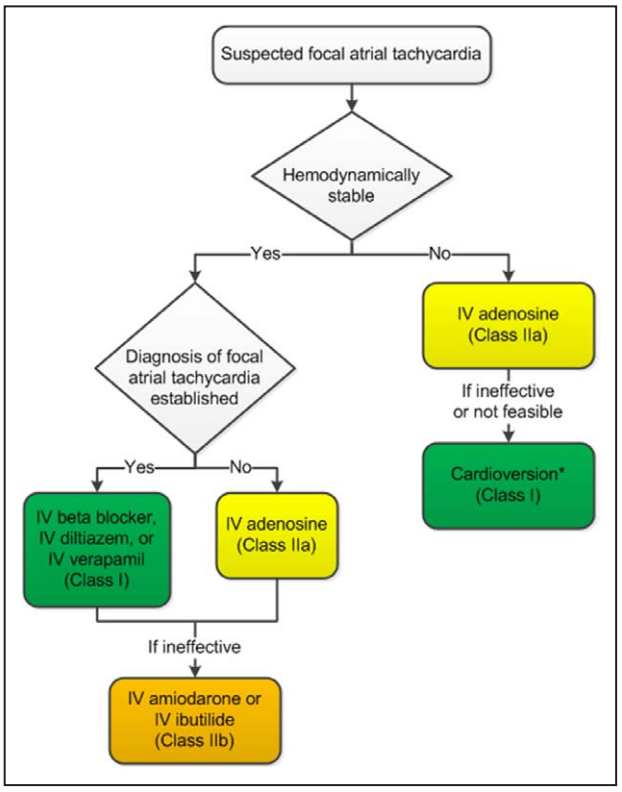

Figure 4. Acute treatment of suspected focal atrial tachycardia. Colors correspond to Class of Recommendation in Table 1; drugs listed alphabetically. *For rhythms that break or recur spontaneously, synchronized cardioversion is not appropriate. IV indicates intravenous.

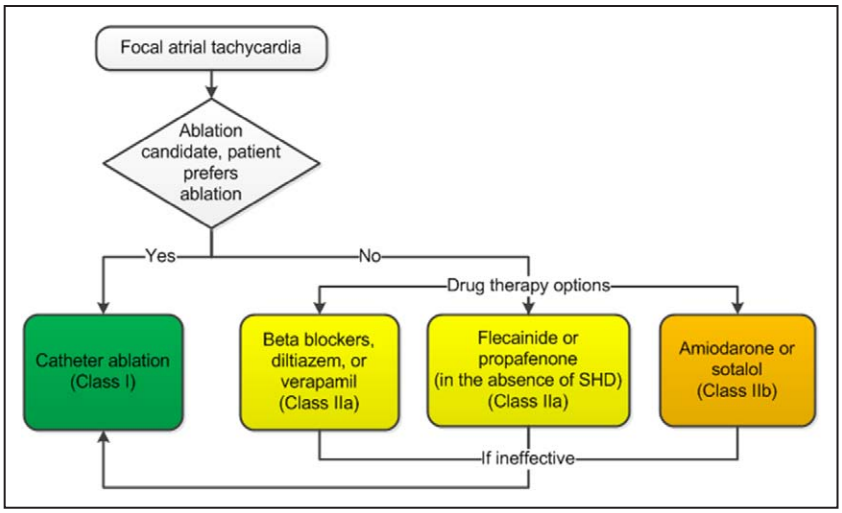

Figure 5. Ongoing management of focal atrial tachycardia. Colors correspond to Class of Recommendation in Table 1; drugs listed alphabetically. Pt indicates patient; and SHD, structural heart disease (including ischemic heart disease).

activity, which results in slowing of the heart rate. On the basis of the results of 2 large, randomized, placebo-controlled trials, this drug was recently approved by the FDA for use in patients with systolic heart failure. The drug has no other hemodynamic effects aside from lowering the heart rate. As such, it has been investigated for use to reduce the sinus rate and improve symptoms related to IST.[85–93]

Radiofrequency ablation to modify the sinus node can reduce the sinus rate, with acute procedural success rates reported in the range of 76% to 100% in nonrandomized cohorts.[94–100] Nonetheless, symptoms commonly recur after several months, with IST recurrence in up to 27% and overall symptomatic recurrence (IST or non-IST AT) in 45% of patients.[94,96,97,99] Complications can be significant. In view of the modest benefit of this procedure and its potential for significant harm, sinus node modification should be considered only for patients who are highly symptomatic and cannot be adequately treated by medication, and then only after informing the patient that the risks may outweigh the benefits of ablation.

See Online Data Supplements 4 and 5 for data supporting Section 3.

4. Nonsinus Focal Atrial Tachycardia and MAT

See Figure 4 for the algorithm for acute treatment of suspected focal atrial tachycardia (AT), Figure 5 for the algorithm for ongoing management of focal AT, and Online Data Supplements 6, 7, and 8 for additional data supporting Section 4.

4.1. Focal Atrial Tachycardia

Focal AT is defined in Table 2. Focal AT can be sustained or nonsustained. The atrial rate during focal AT is usually between 100 bpm and 250 bpm.[102] Presence and severity of symptoms during focal ATs are variable among patients. Focal AT in the adult population is usually associated with a benign prognosis, although AT-mediated cardiomyopathy has been reported in up to 10% of patients referred for ablation of incessant SVT.[103,104] Nonsustained focal AT is common and often does not require treatment.

The diagnosis of focal AT is suspected when the ECG criteria are met (Section 2). Algorithms have been developed to estimate the origin of the focal AT from the P-wave morphology recorded on a standard 12-lead ECG.[105,106] The precise location of the focal AT is ultimately confirmed by mapping during EP studies when successful ablation is achieved.[107–116] Focal AT originates more frequently from the right atrium than from the left atrium.[117,118]

Sinus node reentrant tachycardia is an uncommon type of focal AT that involves a microreentrant circuit in the region of the sinoatrial node, causing a P-wave morphology that is identical to that of sinus tachycardia (although this is not sinus tachycardia). Characteristics that distinguish sinus node reentry from sinus tachycardia are an abrupt onset and termination and often a longer RP interval than that observed during normal sinus rhythm.

4.1.1. Acute Treatment: Recommendations

RCTs of drug therapy for comparative effectiveness in patients with focal AT in the acute setting are not available. Many of the clinical outcomes are reported from small observational studies that included infants or pediatric patients.[119,120] In the clinical setting, if the diagnosis is uncertain, vagal maneuvers may be attempted to better identify the mechanism of SVT.

Recommendations for Acute Treatment of Suspected Focal Atrial Tachycardia

COR	LOE	Recommendations
I	C-LD	1. Intravenous beta blockers, diltiazem, or verapamil is useful for acute treatment in hemodynamically stable patients with focal AT.[107,119–121]
I	C-LD	2. Synchronized cardioversion is recommended for acute treatment in patients with hemodynamically unstable focal AT.[44,122]
IIa	B-NR	1. Adenosine can be useful in the acute setting to either restore sinus rhythm or diagnose the tachycardia mechanism in patients with suspected focal AT.[107,121,123]
IIb	C-LD	1. Intravenous amiodarone may be reasonable in the acute setting to either restore sinus rhythm or slow the ventricular rate in hemodynamically stable patients with focal AT.[120,124]
IIb	C-LD	2. Ibutilide may be reasonable in the acute setting to restore sinus rhythm in hemodynamically stable patients with focal AT.[120,124]

4.1.2. Ongoing Management: Recommendations

Recommendations for Ongoing Management of Suspected Focal Atrial Tachycardia		
COR	LOE	Recommendations
I	B-NR	1. Catheter ablation is recommended in patients with symptomatic focal AT as an alternative to pharmacological therapy.[104,107–112,114–116,124–126]
IIa	C-LD	1. Oral beta blockers, diltiazem, or verapamil are reasonable for ongoing management in patients with symptomatic focal AT.[107,119,120]
IIa	C-LD	2. Flecainide or propafenone can be effective for ongoing management in patients without structural heart disease or ischemic heart disease who have focal AT.[127–131]
IIb	C-LD	1. Oral sotalol or amiodarone may be reasonable for ongoing management in patients with focal AT.[104,129,132–136]

4.2. Multifocal Atrial Tachycardia

MAT is defined in Table 2. The mechanism of MAT is not well established. MAT is commonly associated with underlying conditions, including pulmonary disease, pulmonary hypertension, coronary disease, and valvular heart disease,[137] as well as hypomagnesemia and theophylline therapy.[138] The first-line treatment is management of the underlying condition. Intravenous magnesium may also be helpful in patients with normal magnesium levels.[139] Antiarrhythmic medications in general are not helpful in suppression of MAT.[140] Cardioversion is not useful in MAT.[137]

4.2.1. Acute Treatment: Recommendation

Recommendations for Acute Treatment of Multifocal Atrial Tachycardia		
COR	LOE	Recommendation
IIa	C-LD	1. Intravenous metoprolol[141] or verapamil[142,143] can be useful for acute treatment in patients with MAT.

4.2.2. Ongoing Management: Recommendations

Recommendations for Ongoing Management of Multifocal Atrial Tachycardia		
COR	LOE	Recommendations
IIa	B-NR C-LD	1. Oral verapamil (*Level of Evidence: B-NR*) or diltiazem (*Level of Evidence: C-LD*) is reasonable for ongoing management in patients with recurrent symptomatic MAT.[144,145]
IIa	C-LD	2. Metoprolol is reasonable for ongoing management in patients with recurrent symptomatic MAT.[140,141,145]

5. Atrioventricular Nodal Reentrant Tachycardia

See Figure 6 for the algorithm for acute treatment of AVNRT, Figure 7 for the algorithm for ongoing management of AVNRT, and Online Data Supplements 9 and 10 for additional data supporting Section 5.

AVNRT is the most common SVT and is defined in Table 2. It is usually seen in young adults without structural heart disease or ischemic heart disease, and >60% of cases are observed in women.[16] The ventricular rate is often 180 bpm to 200 bpm but ranges from 110 bpm to >250 bpm (and in rare cases, the rate can be <100 bpm).[19] The anatomic substrate of AVNRT is dual AV nodal physiology (Table 2).

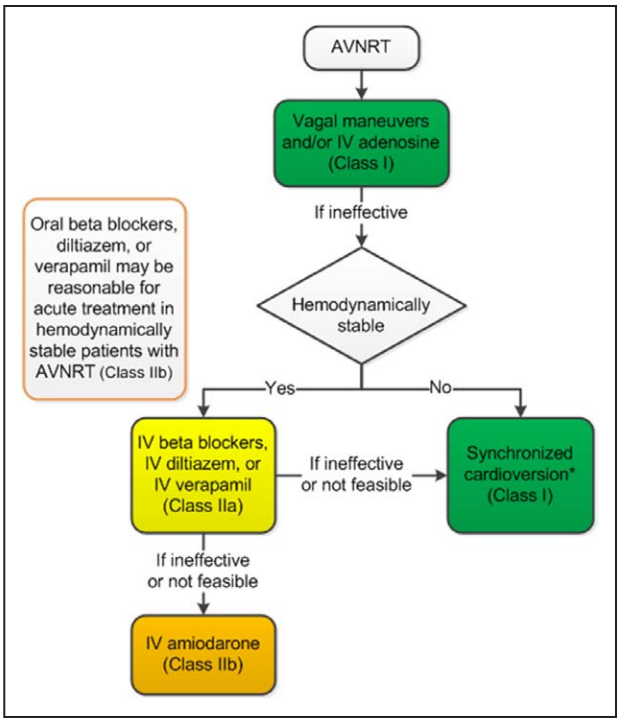

Figure 6. Acute treatment of AVNRT. Colors correspond to Class of Recommendation in Table 1; drugs listed alphabetically. *For rhythms that break or recur spontaneously, synchronized cardioversion is not appropriate. AVNRT indicates atrioventricular nodal reentrant tachycardia; and IV, intravenous.

5.1. Acute Treatment: Recommendations

Recommendations for Acute Treatment of AVNRT		
COR	LOE	Recommendations
I	B-R	1. Vagal maneuvers are recommended for acute treatment in patients with AVNRT.[33–35,146,147]
I	B-R	2. Adenosine is recommended for acute treatment in patients with AVNRT.[37,41,43,148]
I	B-NR	3. Synchronized cardioversion should be performed for acute treatment in hemodynamically unstable patients with AVNRT when adenosine and vagal maneuvers do not terminate the tachycardia or are not feasible.[44,122]
I	B-NR	4. Synchronized cardioversion is recommended for acute treatment in hemodynamically stable patients with AVNRT when pharmacological therapy does not terminate the tachycardia or is contraindicated.[36,45]
IIa	B-R	1. Intravenous beta blockers, diltiazem, or verapamil are reasonable for acute treatment in hemodynamically stable patients with AVNRT.[47,149–152]
IIb	C-LD	1. Oral beta blockers, diltiazem, or verapamil may be reasonable for acute treatment in hemodynamically stable patients with AVNRT.[153,154]
IIb	C-LD	2. Intravenous amiodarone may be considered for acute treatment in hemodynamically stable patients with AVNRT when other therapies are ineffective or contraindicated.[67]

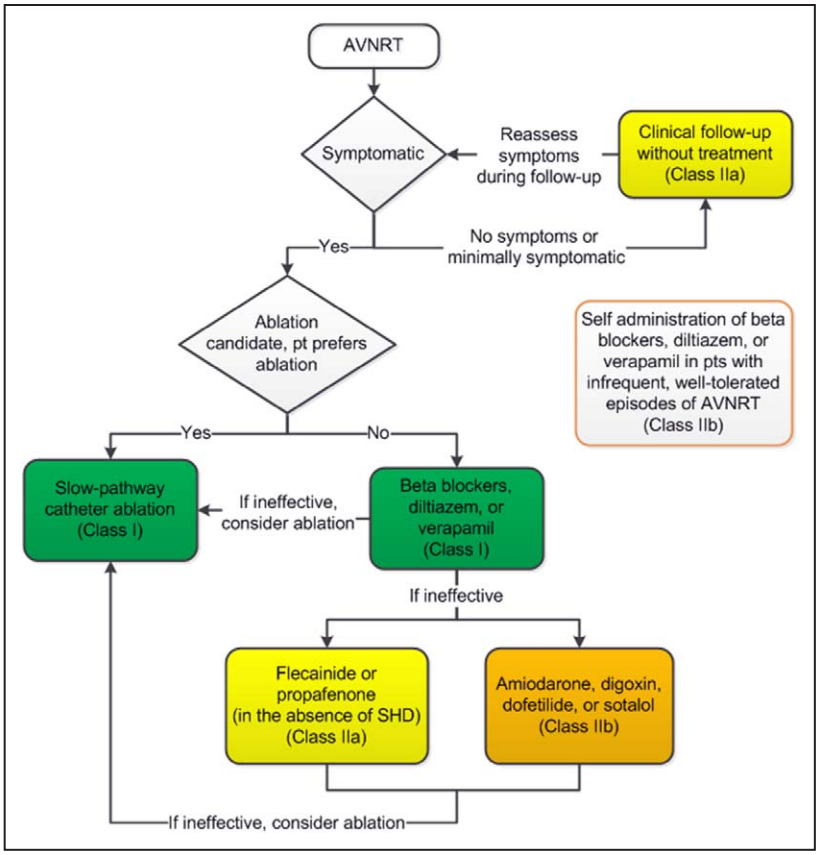

Figure 7. Ongoing management of AVNRT. Colors correspond to Class of Recommendation in Table 1; drugs listed alphabetically. AVNRT indicates atrioventricular nodal reentrant tachycardia; pt, patient; and SHD, structural heart disease (including ischemic heart disease).

5.2. Ongoing Management: Recommendations

Recommendations for Ongoing Management of AVNRT		
COR	**LOE**	**Recommendations**
I	B-R	1. Oral verapamil or diltiazem is recommended for ongoing management in patients with AVNRT who are not candidates for, or prefer not to undergo, catheter ablation.[49,50,155,156]
I	B-NR	2. Catheter ablation of the slow pathway is recommended in patients with AVNRT.[51–58,157–161]
I	B-R	3. Oral beta blockers are recommended for ongoing management in patients with AVNRT who are not candidates for, or prefer not to undergo, catheter ablation.[50]
IIa	B-R	1. Flecainide or propafenone is reasonable for ongoing management in patients without structural heart disease or ischemic heart disease who have AVNRT and are not candidates for, or prefer not to undergo, catheter ablation and in whom beta blockers, diltiazem, or verapamil are ineffective or contraindicated.[48,59–66,153,154,162,163]
IIa	B-NR	2. Clinical follow-up without pharmacological therapy or ablation is reasonable for ongoing management in minimally symptomatic patients with AVNRT.[156]
IIb	B-R	1. Oral sotalol or dofetilide may be reasonable for ongoing management in patients with AVNRT who are not candidates for, or prefer not to undergo, catheter ablation.[59,66]

Recommendations for Ongoing Management of AVNRT (Continued)		
COR	**LOE**	**Recommendations**
IIb	B-R	2. Oral digoxin or amiodarone may be reasonable for ongoing treatment of AVNRT in patients who are not candidates for, or prefer not to undergo, catheter ablation.[50,67]
IIb	C-LD	3. Self-administered ("pill-in-the-pocket") acute doses of oral beta blockers, diltiazem, or verapamil may be reasonable for ongoing management in patients with infrequent, well-tolerated episodes of AVNRT.[153,154]

6. Manifest and Concealed Accessory Pathways

Accessory pathways (defined in Table 2) can conduct in the anterograde direction, retrograde direction, or both; and can be associated with several different supraventricular arrhythmias. Some anterograde pathways may place patients at risk of SCD.

The most common tachycardia associated with an accessory pathway is orthodromic AVRT, with a circuit that uses the AV node and His Purkinje system in the anterograde direction, followed by conduction through the ventricle, retrograde conduction over the accessory pathway, and completion of the circuit by conduction through the atrium back into the AV node. Orthodromic AVRT accounts for approximately 90% to 95% of AVRT episodes in patients with a manifest accessory pathway. Pre-excited AVRT, including antidromic AVRT, accounts for 5% of the AVRT episodes in patients with a manifest pathway and involves conduction from the atrium to the ventricle

via the accessory pathway, causing a preexcited QRS complex. This is called antidromic AVRT tachycardia when the return reentrant conduction occurs retrogradely via the AV node. In rare cases of pre-excited AVRT, the return conduction occurs via a second accessory AV pathway. AF can occur in patients with accessory pathways, which may result in extremely rapid conduction to the ventricle over a manifest pathway, which increases the risk of inducing ventricular fibrillation and SCD.

Rapid anterograde accessory pathway conduction during AF can result in SCD in patients with a manifest accessory pathway, with a 10-year risk ranging from 0.15% to 0.24%.[164,165] Unfortunately, SCD may be the first presentation of patients with undiagnosed WPW. Increased risk of SCD is associated with a history of symptomatic tachycardia, multiple accessory pathways, and a shortest pre-excited R-R interval of <250 ms during AF. The risk of SCD associated with WPW appears highest in the first 2 decades of life.[165–169]

6.1. Management of Patients With Symptomatic Manifest or Concealed Accessory Pathways

See Figure 8 for the algorithm for acute treatment of orthodromic AVRT, Figure 9 for the algorithm for ongoing management of orthodromic AVRT, and Online Data Supplements 11 to 15 for additional data supporting Section 6.

6.1.1. Acute Treatment: Recommendations

Recommendations for Acute Treatment of Orthodromic AVRT		
COR	**LOE**	**Recommendations**
I	B-R	1. Vagal maneuvers are recommended for acute treatment in patients with orthodromic AVRT.[43,147,170,171]
I	B-R	2. Adenosine is beneficial for acute treatment in patients with orthodromic AVRT.[43,172,173]
I	B-NR	3. Synchronized cardioversion should be performed for acute treatment in hemodynamically unstable patients with AVRT if vagal maneuvers or adenosine are ineffective or not feasible.[170,174,175]
I	B-NR	4. Synchronized cardioversion is recommended for acute treatment in hemodynamically stable patients with AVRT when pharmacological therapy is ineffective or contraindicated.[36,45]
I	B-NR	5. Synchronized cardioversion should be performed for acute treatment in hemodynamically unstable patients with pre-excited AF.[44,170]
I	C-LD	6. Ibutilide[176] or intravenous procainamide[177] is beneficial for acute treatment in patients with pre-excited AF who are hemodynamically stable.
IIa	B-R C-LD	1. Intravenous diltiazem, verapamil[43,172,178,179] (*Level of Evidence: B-R*) or beta blockers[180] (*Level of Evidence: C-LD*) can be effective for acute treatment in patients with orthodromic AVRT who do not have pre-excitation on their resting ECG during sinus rhythm.
IIb	B-R	1. Intravenous beta blockers, diltiazem, or verapamil might be considered for acute treatment in patients with orthodromic AVRT who have pre-excitation on their resting ECG and have not responded to other therapies.[43,178,179,181]
III: Harm	C-LD	1. Intravenous digoxin, intravenous amiodarone, intravenous or oral beta blockers, diltiazem, and verapamil are potentially harmful for acute treatment in patients with pre-excited AF.[181–186]

6.1.2. Ongoing Management: Recommendations

Recommendations for Ongoing Management of Orthodromic AVRT		
COR	**LOE**	**Recommendations**
I	B-NR	1. Catheter ablation of the accessory pathway is recommended in patients with AVRT and/or pre-excited AF.[55,165,187–193]
I	C-LD	2. Oral beta blockers, diltiazem, or verapamil are indicated for ongoing management of AVRT in patients without pre-excitation on their resting ECG.[48,194]
IIa	B-R	1. Oral flecainide or propafenone is reasonable for ongoing management in patients without structural heart disease or ischemic heart disease who have AVRT and/or pre-excited AF and are not candidates for, or prefer not to undergo, catheter ablation.[60,61,64,65,195]
IIb	B-R	1. Oral dofetilide or sotalol may be reasonable for ongoing management in patients with AVRT and/or pre-excited AF who are not candidates for, or prefer not to undergo, catheter ablation.[99,106]
IIb	C-LD	2. Oral amiodarone may be considered for ongoing management in patients with AVRT and/or pre-excited AF who are not candidates for, or prefer not to undergo, catheter ablation and in whom beta blockers, diltiazem, flecainide, propafenone, and verapamil are ineffective or contraindicated.[196,197]
IIb	C-LD	3. Oral beta blockers, diltiazem, or verapamil may be reasonable for ongoing management of orthodromic AVRT in patients with pre-excitation on their resting ECG who are not candidates for, or prefer not to undergo, catheter ablation.[48,194]
IIb	C-LD	4. Oral digoxin may be reasonable for ongoing management of orthodromic AVRT in patients without pre-excitation on their resting ECG who are not candidates for, or prefer not to undergo, catheter ablation.[198]
III: Harm	C-LD	1. Oral digoxin is potentially harmful for ongoing management in patients with AVRT or AF and pre-excitation on their resting ECG.[182]

6.2. Management of Asymptomatic Pre-Excitation

6.2.1. PICOTS Critical Questions

See the ERC systematic review report, "Risk Stratification for Arrhythmic Events in Patients With Asymptomatic Pre-Excitation" for the complete evidence review on the management of asymptomatic pre-excitation,[10] and see Online Data Supplements 13, 14, and 15 for additional data on asymptomatic pre-excitation, which were reproduced directly from the ERC's systematic review. These recommendations have been designated with the notation SR to emphasize the rigor of support from the ERC's systematic review. PICOTS Question 1 did not provide adequate data for a recommendation; the other 3 PICOTS questions are addressed in the recommendations in Section 6.2.2.

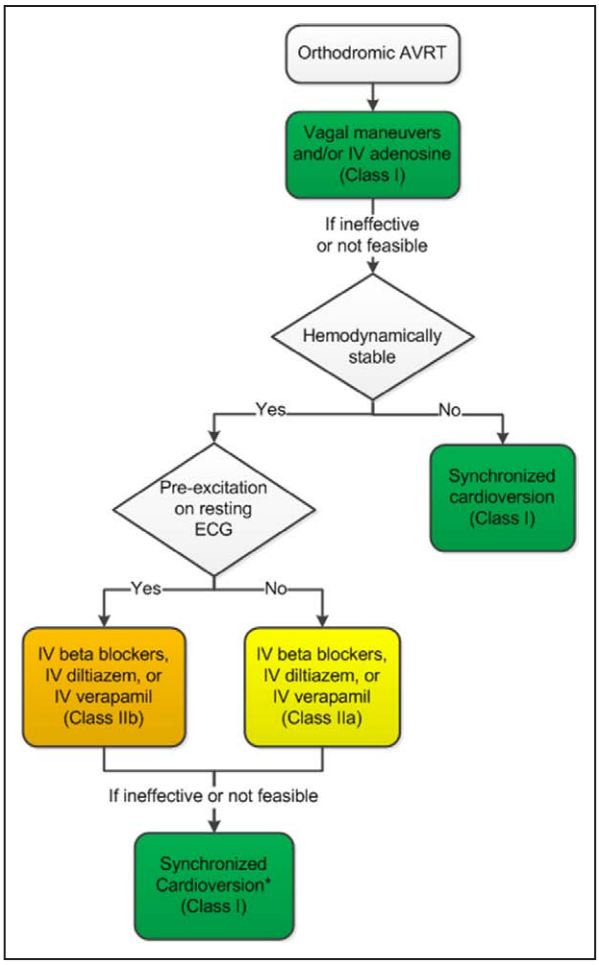

Figure 8. Acute treatment of orthodromic AVRT. Colors correspond to Class of Recommendation in Table 1; drugs listed alphabetically. *For rhythms that break or recur spontaneously, synchronized cardioversion is not appropriate. AVRT indicates atrioventricular reentrant tachycardia; ECG, electrocardiogram; and IV, intravenous.

The following 4 questions were considered by the ERC:

1. What is the comparative predictive accuracy of invasive EP study (without catheter ablation of the accessory pathway) versus noninvasive testing for predicting arrhythmic events (including SCD) in patients with asymptomatic pre-excitation?
2. What is the usefulness of invasive EP study (without catheter ablation of the accessory pathway) versus no testing for predicting arrhythmic events (including SCD) in patients with asymptomatic pre-excitation?
3. What is the usefulness of invasive EP study (without catheter ablation of the accessory pathway) or noninvasive EP study for predicting arrhythmic events (including SCD) in patients with asymptomatic pre-excitation?
4. What are the efficacy and effectiveness of invasive EP study with catheter ablation of the accessory pathway as appropriate versus noninvasive tests with treatment (including observation) or no testing/ablation as appropriate for preventing arrhythmic events (including SCD) and improving outcomes in patients with asymptomatic pre-excitation?

6.2.2. Asymptomatic Patients With Pre-Excitation: Recommendations

Recommendations for Management of Asymptomatic Patients With Pre-Excitation		
COR	**LOE**	**Recommendations**
I	B-NR [SR]	1. In asymptomatic patients with pre-excitation, the findings of abrupt loss of conduction over a manifest pathway during exercise testing in sinus rhythm[199–202] *(Level of Evidence: B-NR)* [SR] or intermittent loss of pre-excitation during ECG or ambulatory monitoring[202] *(Level of Evidence: C-LD)* [SR] are useful to identify patients at low risk of rapid conduction over the pathway.
	C-LD [SR]	
IIa	B-NR [SR]	1. An EP study is reasonable in asymptomatic patients with pre-excitation to risk-stratify for arrhythmic eve nts.[165,167,203–206]
IIa	B-NR [SR]	2. Catheter ablation of the accessory pathway is reasonable in asymptomatic patients with pre-excitation if an EP study identifies a high risk of arrhythmic events, including rapidly conducting pre-excited AF.[165,207,208]
IIa	B-NR [SR]	3. Catheter ablation of the accessory pathway is reasonable in asymptomatic patients if the presence of pre-excitation precludes specific employment (such as with pilots).[55,165,187–193,207–209]
IIa	B-NR [SR]	4. Observation, without further evaluation or treatment, is reasonable in asymptomatic patients with pre-excitation.[206,210–213]

6.3. Risk Stratification of Symptomatic Patients With Manifest Accessory Pathways: Recommendations

Recommendations for Management of Symptomatic Patients With Manifest Accessory Pathways		
COR	**LOE**	**Recommendations**
I	B-NR	1. In symptomatic patients with pre-excitation, the findings of abrupt loss of conduction over the pathway during exercise testing in sinus rhythm[199–202] *(Level of Evidence: B-NR)* or intermittent loss of pre-excitation during ECG or ambulatory monitoring[202] *(Level of Evidence: C-LD)* are useful for identifying patients at low risk of developing rapid conduction over the pathway.
	C-LD	
I	B-NR	2. An EP study is useful in symptomatic patients with pre-excitation to risk-stratify for life-threatening arrhythmic events.[165,167,203–205]

7. Atrial Flutter

See Figure 10 for the algorithm for acute treatment of atrial flutter, Figure 11 for the algorithm for ongoing management of atrial flutter, and Online Data Supplements 16 and 17 for data supporting Section 7.

7.1. Cavotricuspid Isthmus-Dependent Atrial Flutter

Cavotricuspid isthmus (CTI)–dependent atrial flutter is defined in Table 2. 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.[214]

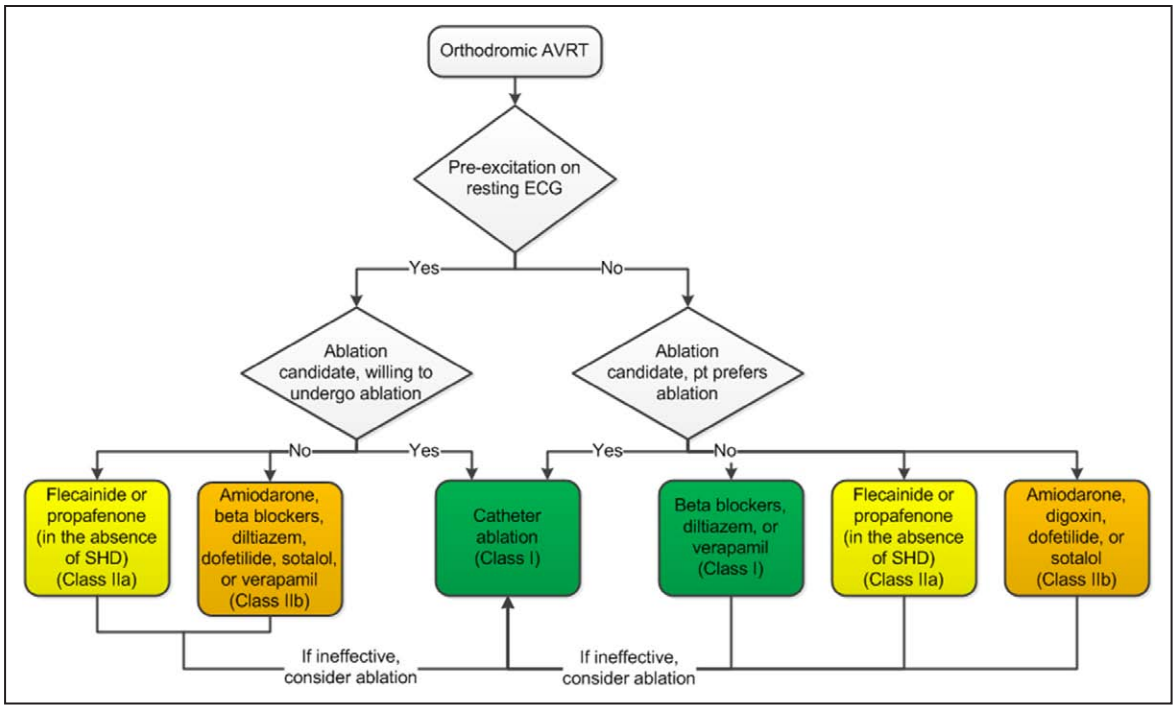

Figure 9. Ongoing management of orthodromic AVRT. Colors correspond to Class of Recommendation in Table 1; drugs listed alphabetically. AVRT indicates atrioventricular reentrant tachycardia; ECG, electrocardiogram; pt, patient; and SHD, structural heart disease (including ischemic heart disease).

Atrial flutter can occur in clinical settings similar to those associated with AF, and atrial flutter can be triggered by AT or AF.[215,216] 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.[217] 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.[216–221]

7.2. Non–Isthmus-Dependent Atrial Flutters

Non–isthmus-dependent atrial flutter or atypical flutter describes macroreentrant ATs that are not dependent on conduction through the CTI, as defined in Table 2.

Catheter ablation of non–CTI-dependent flutter requires more extensive mapping than does ablation of CTI-dependent flutter, and success rates are lower (Online Data Supplement–Appendix 3). The location of the circuit determines ablation approach and risks.

The development of a microreentrant or macroreentrant left AT after AF ablation occurs in approximately 5% of patients.[222–224] This is less frequent if ablation is limited to pulmonary vein isolation. On the other hand, these arrhythmias are more common in patients with longer-duration persistent AF or more dilated left atria or when linear ablation lesions are used.[223–228] Detailed activation and entrainment mapping of the tachycardia during a second procedure result in effective ablation in approximately 90% of patients.[225]

7.3. Acute Treatment: Recommendations

Recommendations for Acute Treatment of Atrial Flutter		
COR	**LOE**	**Recommendations**
I	A	1. Oral dofetilide or intravenous ibutilide is useful for acute pharmacological cardioversion in patients with atrial flutter.[229–236]
I	B-R	2. Intravenous or oral beta blockers, diltiazem, or verapamil are useful for acute rate control in patients with atrial flutter who are hemodynamically stable.[237–244]
I	B-NR	3. Elective synchronized cardioversion is indicated in stable patients with well-tolerated atrial flutter when a rhythm-control strategy is pursued.[245–247]
I	B-NR	4. Synchronized cardioversion is recommended for acute treatment of patients with atrial flutter who are hemodynamically unstable and do not respond to pharmacological therapies.[122,170,245,248]
I	C-LD	5. Rapid atrial pacing is useful for acute conversion of atrial flutter in patients who have pacing wires in place as part of a permanent pacemaker or implantable cardioverter-defibrillator or for temporary atrial pacing after cardiac surgery.[249–253]
I	B-NR	6. Acute antithrombotic therapy is recommended in patients with atrial flutter to align with recommended antithrombotic therapy for patients with AF.[254]
IIa	B-R	1. Intravenous amiodarone can be useful for acute control of the ventricular rate (in the absence of pre-excitation) in patients with atrial flutter and systolic heart failure, when beta blockers are contraindicated or ineffective.[240,255,256]

7.4. Ongoing Management: Recommendations

Recommendations for Ongoing Management of Atrial Flutter		
COR	LOE	Recommendations
I	B-R	1. Catheter ablation of the CTI is useful in patients with atrial flutter that is either symptomatic or refractory to pharmacological rate control.[155,257–260]
I	C-LD	2. Beta blockers, diltiazem, or verapamil are useful to control the ventricular rate in patients with hemodynamically tolerated atrial flutter.[237–239]
I	C-LD	3. Catheter ablation is useful in patients with recurrent symptomatic non–CTI-dependent flutter after failure of at least 1 antiarrhythmic agent.[261,262]
I	B-NR	4. Ongoing management with antithrombotic therapy is recommended in patients with atrial flutter to align with recommended antithrombotic therapy for patients with AF.[254]
IIa	B-R	1. The following drugs can be useful to maintain sinus rhythm in patients with symptomatic, recurrent atrial flutter, with the drug choice depending on underlying heart disease and comorbidities: a. Amiodarone[263] b. Dofetilide[236,264] c. Sotalol[265]
IIa	B-NR	2. Catheter ablation is reasonable in patients with CTI-dependent atrial flutter that occurs as the result of flecainide, propafenone, or amiodarone used for treatment of AF.[266–269]
IIa	C-LD	3. Catheter ablation of the CTI is reasonable in patients undergoing catheter ablation of AF who also have a history of documented clinical or induced CTI-dependent atrial flutter.[269,270]
IIa	C-LD	4. Catheter ablation is reasonable in patients with recurrent symptomatic non–CTI-dependent flutter as primary therapy, before therapeutic trials of antiarrhythmic drugs, after carefully weighing potential risks and benefits of treatment options.[271]
IIb	B-R	1. Flecainide or propafenone may be considered to maintain sinus rhythm in patients without structural heart disease or ischemic heart disease who have symptomatic recurrent atrial flutter.[272–274]
IIb	C-LD	2. Catheter ablation may be reasonable for asymptomatic patients with recurrent atrial flutter.[54,216,257]

8. Junctional Tachycardia

See Figure 12 for the algorithm for ongoing management of junctional tachycardia and Online Data Supplements 18 and 19 for data supporting Section 8.

Junctional tachycardia (defined in Table 2) is a rapid, occasionally irregular, narrow-complex tachycardia (with rates typically of 120 bpm to 220 bpm) that arises from the AV junction (including the His bundle). AV dissociation (often isorhythmic) may be seen, and when present, excludes the misdiagnosis of AVRT and makes AVNRT highly unlikely. If it is irregular, junctional tachycardia may be misdiagnosed as AF or MAT. The mechanism for junctional tachycardia is enhanced (abnormal) automaticity from an ectopic focus in the AV junction (including the His bundle).[275]

Junctional tachycardia is uncommon in adults[275]; it is typically seen in infants postoperatively, after cardiac surgery for congenital heart disease; this is also known as junctional ectopic tachycardia. As such, there is limited evidence with regard to diagnosis and management of junctional tachycardia in adult patients.

A related rhythm, nonparoxysmal junctional tachycardia (more commonly known as accelerated AV junctional rhythm), is far more common in adults than paroxysmal junctional tachycardia. The mechanism of nonparoxysmal junctional tachycardia is associated with automaticity or triggered activity. It occurs at a slower rate (70 bpm to 130 bpm) and is often due to digoxin toxicity[276] or myocardial infarction.[277,278] Treatment of this rhythm centers on addressing the underlying condition.

8.1. Acute Treatment: Recommendations

Recommendations for Acute Treatment of Junctional Tachycardia		
COR	LOE	Recommendations
IIa	C-LD	1. Intravenous beta blockers are reasonable for acute treatment in patients with symptomatic junctional tachycardia.[275]
IIa	C-LD	2. Intravenous diltiazem, procainamide, or verapamil is reasonable for acute treatment in patients with junctional tachycardia.[279]

8.2. Ongoing Management: Recommendations

Recommendations for Ongoing Management of Junctional Tachycardia		
COR	LOE	Recommendations
IIa	C-LD	1. Oral beta blockers are reasonable for ongoing management in patients with junctional tachycardia.[275]
IIa	C-LD	2. Oral diltiazem or verapamil is reasonable for ongoing management in patients with junctional tachycardia.[279]
IIb	C-LD	1. Flecainide or propafenone may be reasonable for ongoing management in patients without structural heart disease or ischemic heart disease who have junctional tachycardia.[280,281]
IIb	C-LD	2. Catheter ablation may be reasonable in patients with junctional tachycardia when medical therapy is not effective or contraindicated.[282–288]

9. Special Populations

9.1. Pediatrics

As discussed in the Scope (Section 1.4), the present document is aimed at the adult population (≥18 years of age) and offers no specific recommendations for pediatric patients. Nevertheless, a brief discussion of SVT in pediatric patients is included below, highlighting major considerations with regard to SVT in younger patients, including adolescent patients.

SVT in young patients varies significantly from SVT in adult patients in terms of mechanism, risk of developing heart failure or cardiac arrest, risks associated with interventional therapy, natural history, and psychosocial impact. Approximately half of pediatric SVT presents in the first 4 months of life, with age-related peaks in occurrence subsequently at 5 to 8 years and after 13 years. Accessory pathway–mediated tachycardia accounts for >70% of SVT in infants, decreasing to approximately 55% in adolescents.[21,289–291]

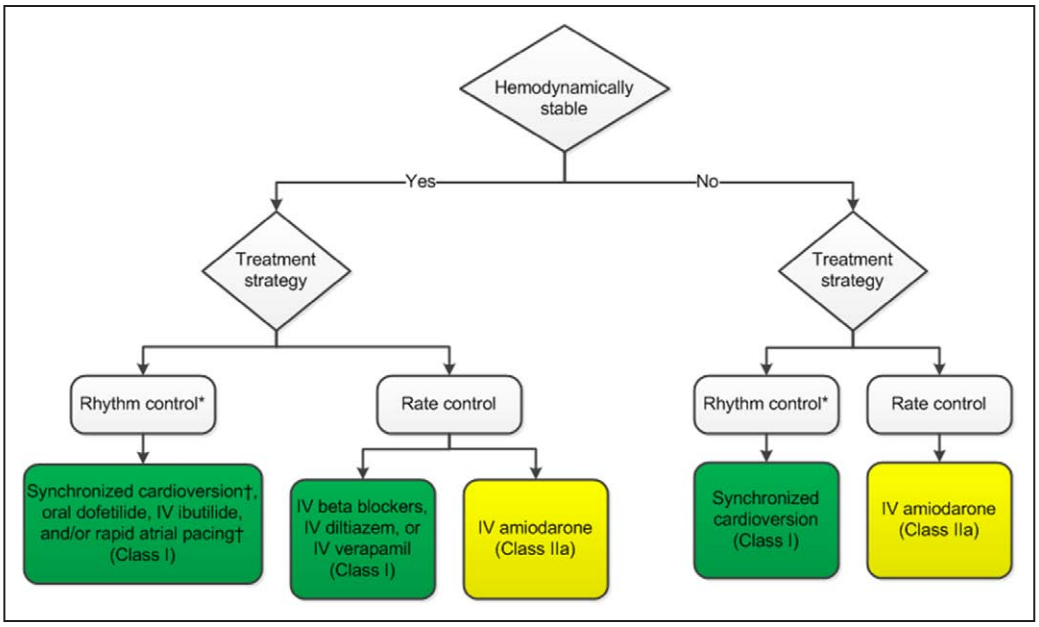

Figure 10. Acute treatment of atrial flutter. Colors correspond to Class of Recommendation in Table 1; drugs listed alphabetically. *Anticoagulation as per guideline is mandatory. †For rhythms that break or recur spontaneously, synchronized cardioversion or rapid atrial pacing is not appropriate. IV indicates intravenous.

AVNRT increases with age, from 9% to 13% of SVT in infants, to 30% to 50% of SVT in teenagers. Atrial flutter is seen in some neonates and in older children is predominantly observed after congenital heart disease. AF is uncommon in childhood, accounting for <3% of supraventricular arrhythmias, and may be a consequence of AVRT or AVNRT in adolescents or may be associated with repaired congenital heart disease. Congestive heart failure is present in up to 20% of infants and in older children with incessant tachycardia and in rare cases may necessitate mechanical cardiopulmonary support during initial therapy.[292]

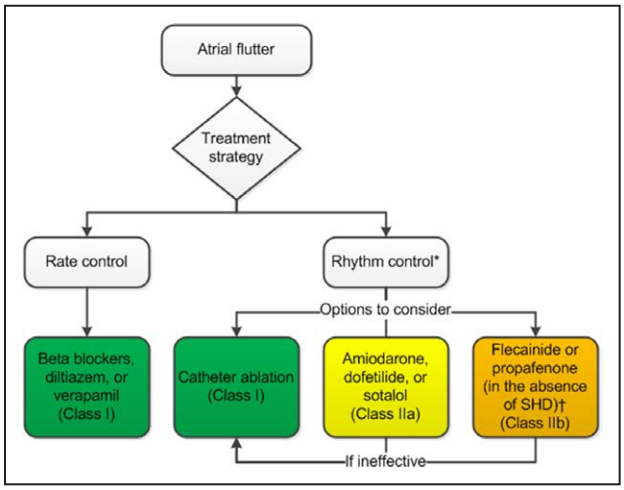

Figure 11. Ongoing management of atrial flutter. Colors correspond to Class of Recommendation in Table 1; drugs listed alphabetically. *After assuring adequate anticoagulation or excluding left atrial thrombus by transesophageal echocardiography before conversion. †Should be combined with AV nodal–blocking agents to reduce risk of 1:1 conduction during atrial flutter. AV indicates atrioventricular; SHD, structural heart disease (including ischemic heart disease).

The risk of ventricular fibrillation or SCD related to WPW in childhood is 1.3% to 1.6% and is highest in the first 2 decades of life.[23,165–168] The risk of cardiac arrest is higher in patients with AVRT precipitating AF, short accessory connection refractory periods, and posteroseptal accessory pathways.[23,165–168] Pharmacological therapy of SVT in childhood is largely based on practice patterns because RCTs of antiarrhythmic medications in children are lacking. AV nodal–blocking drugs are widely used for the most common arrhythmias, AVRT, and AVNRT. Higher initial doses of adenosine are needed in children than in adults, with children receiving from 150 mcg/kg to 250 mcg/kg.[293–295] Digoxin is avoided in the presence of pre-excitation because its use in infancy has been associated with SCD or ventricular fibrillation.[296,297] Amiodarone, sotalol, propafenone, or flecainide can be used for refractory SVT in infants. In older children presenting with SVT, beta-blocker therapy is most often the initial therapy used. Because of the rare occurrence of adverse events with flecainide, including in patients without structural heart disease, flecainide is not used as a first-line medication in children.[298]

Catheter ablation can be successfully performed in children of all ages, with acute success rates comparable to those reported in adults.[192,193,299,300] Complications were reported in 4% to 8% of the initial large series, with major complications in 0.9% to 3.2%, and complication rates were higher in patients weighing <15 kg.[192,299–301] The implications of complications, including AV block requiring pacing, perforation, and coronary artery or mitral valve injury, are profound in young patients.[302–304] In early series, death was reported in 0.12% of children with normal hearts and was associated with lower weight and increased number of ablation lesions.[305] Although most centers perform elective ablation for children weighing >12 kg to 15 kg, ablation in younger

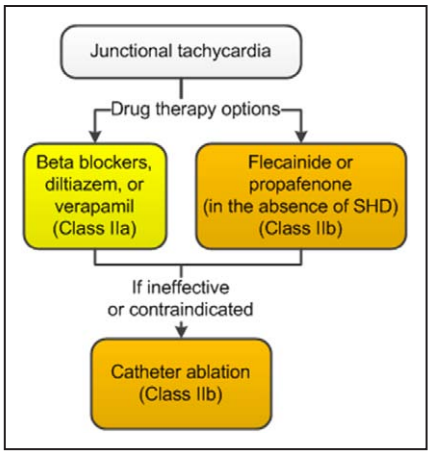

Figure 12. Ongoing management of junctional tachycardia. Colors correspond to Class of Recommendation in Table 1; drugs listed alphabetically. SHD indicates structural heart disease (including ischemic heart disease).

or smaller children is generally reserved for those with medically refractory SVT or tachycardia-induced cardiomyopathy or before surgery that may limit access for subsequent catheter-based procedures.

Junctional ectopic tachycardia occurs predominantly in very young patients either as a congenital form or, more commonly, after intracardiac repair of congenital heart disease. Nonpostoperative junctional tachycardia has been reported to respond to amiodarone or combination therapy including beta blockers, flecainide, procainamide, or propafenone.[306]

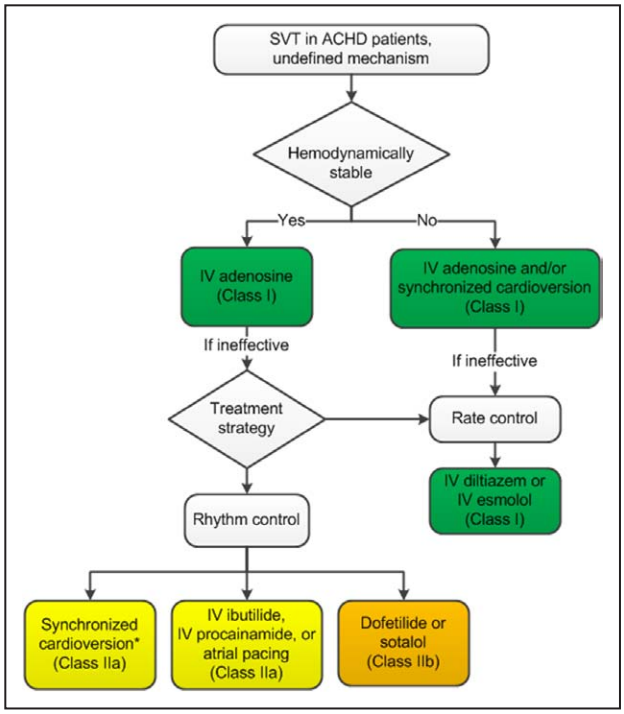

Figure 13. Acute treatment of SVT in ACHD patients. Colors correspond to Class of Recommendation in Table 1; drugs listed alphabetically. *For rhythms that break or recur spontaneously, synchronized cardioversion is not appropriate. ACHD indicates adult congenital heart disease; IV, intravenous; and SVT, supraventricular tachycardia.

Ablation for patients with refractory tachycardia or ventricular dysfunction has shown efficacy of 82% to 85%, but inadvertent AV block occurred in 18% and recurrence was seen in 14% of patients.[306] Postoperative junctional tachycardia occurs in 2% to 10% of young patients undergoing intracardiac surgery.[307,308] Treatment includes sedation with muscle relaxation, limitation of inotropic medications, reduction of core temperature to 34 to 35°C, atrial overdrive pacing, and procainamide or amiodarone infusions.[309–313] In general, postoperative junctional tachycardia resolves and does not require ongoing therapy.

Although this guideline focuses on adults, it should be noted that SVT may occur in the fetus and, if sustained, may put the fetus at risk of cardiovascular collapse manifested by hydrops. Mothers require safety monitoring by adult cardiologists during treatment. The most common mechanisms for fetal SVT are AVRT and atrial flutter.[314] Persistent SVT with hydrops carries a high mortality rate, and therefore, prompt and aggressive treatment is warranted. Maternal administration of antiarrhythmic agents has been shown to be effective through transplacental delivery.

9.2. Patients With Adult Congenital Heart Disease

See Figure 13 for the algorithm for acute treatment of non–pre-excited SVT in adult congenital heart disease (ACHD) patients; Figure 14 for the algorithm for ongoing management of non–pre-excited SVT in ACHD patients; and Online Data Supplements 20 and 21 for data supporting Section 9.

9.2.1. Clinical Features

SVT is observed in 10% to 20% of ACHD patients, and is associated with a significantly increased risk of heart failure, stroke, and SCD.[315–319] The most common mechanism of SVT in ACHD patients is macroreentrant AT (also called flutter), which accounts for at least 75% of SVT and frequently involves the CTI. Focal AT, AVNRT, and accessory pathway–mediated tachycardia each account for less than about 8% of SVT, whereas the incidence of AF is about 10% and increases with age.[320–325]

The management of SVT in ACHD patients is influenced by the underlying cardiac anatomy and surgical repair, the current hemodynamic sequelae of the anatomy and repairs, and mechanism of SVT. Management of ACHD patients should be undertaken only in collaboration with a cardiologist who has specialized training or experience in managing such patients.

Overall acute success rates of catheter ablation procedures for SVT in ACHD patients range from 70% to 85%, with recurrences in 20% to 60% of patients within 2 years.[326–331] Catheter ablation is challenged by limitations of venous access to the heart, hypertrophied atrial tissue, multiple atrial reentrant circuits, and atrial baffles partitioning the coronary sinus and CTI to the pulmonary venous atrium. The development of atrial arrhythmias in ACHD patients is often an indicator of progressive hemodynamic changes, which require in-depth functional and hemodynamic assessment. Intervention for residual hemodynamic/structural defects may need to be planned as part of chronic arrhythmia management.

9.2.2. Acute Treatment: Recommendations

Recommendations for Acute Treatment of SVT in ACHD Patients		
COR	LOE	Recommendations
I	C-LD	1. Acute antithrombotic therapy is recommended in ACHD patients who have AT or atrial flutter to align with recommended antithrombotic therapy for patients with AF.[254]
I	B-NR	2. Synchronized cardioversion is recommended for acute treatment in ACHD patients and SVT who are hemodynamically unstable.[170,332]
I	C-LD	3. Intravenous diltiazem or esmolol (with extra caution used for either agent, observing for the development of hypotension) is recommended for acute treatment in ACHD patients and SVT who are hemodynamically stable.[333,334]
I	B-NR	4. Intravenous adenosine is recommended for acute treatment in ACHD patients and SVT.[121,335–337]
IIa	B-NR	1. Intravenous ibutilide or procainamide can be effective for acute treatment in ACHD patients and atrial flutter who are hemodynamically stable.[338–340]
IIa	B-NR	2. Atrial pacing can be effective for acute treatment in ACHD patients and SVT who are hemodynamically stable and anticoagulated as per current guidelines for antithrombotic therapy in patients with AF.[338,341–344]
IIa	B-NR	3. Elective synchronized cardioversion can be useful for acute termination of AT or atrial flutter in ACHD patients when acute pharmacological therapy is ineffective or contraindicated.[332]
IIb	B-NR	1. Oral dofetilide or sotalol may be reasonable for acute treatment in ACHD patients and AT and/or atrial flutter who are hemodynamically stable.[345,346]

9.2.3. Ongoing Management: Recommendations

Recommendations for Ongoing Management of SVT in ACHD Patients		
COR	LOE	Recommendations
I	C-LD	1. Ongoing management with antithrombotic therapy is recommended in ACHD patients and AT or atrial flutter to align with recommended antithrombotic therapy for patients with AF.[254]
I	C-LD	2. Assessment of associated hemodynamic abnormalities for potential repair of structural defects is recommended in ACHD patients as part of therapy for SVT.[347,348]
IIa	B-NR	1. Preoperative catheter ablation or intraoperative surgical ablation of accessory pathways or AT is reasonable in patients with SVT who are undergoing surgical repair of Ebstein anomaly.[349–355]
IIa	B-NR	2. Oral beta blockers or sotalol therapy can be useful for prevention of recurrent AT or atrial flutter in ACHD patients.[135,323,356]
IIa	B-NR	3. Catheter ablation is reasonable for treatment of recurrent symptomatic SVT in ACHD patie nts.[222,325,326,328,331,357–361]
IIa	B-NR	4. Surgical ablation of AT or atrial flutter can be effective in ACHD patients undergoing planned surgical repair.[362–373]
IIb	B-NR	1. Atrial pacing may be reasonable to decrease recurrences of AT or atrial flutter in ACHD patients and sinus node dysfunction.[344,374,375]
IIb	B-NR	2. Oral dofetilide may be reasonable for prevention of recurrent AT or atrial flutter in ACHD patients.[323,346,376,377]
IIb	B-NR	3. Amiodarone may be reasonable for prevention of recurrent AT or atrial flutter in ACHD patients for whom other medications and catheter ablation are ineffective or contraindicated.[323]
III: Harm	B-NR	1. Flecainide should not be administered for treatment of SVT in ACHD patients and significant ventricular dysfunction.[298]

9.3. Pregnancy

Pregnancy may confer an increased susceptibility to a variety of arrhythmias, even in the absence of underlying heart disease.[378] Pregnancy is also associated with an increased risk of arrhythmia exacerbation, such as more frequent and refractory tachycardia episodes, in patients with a pre-existing arrhythmic substrate.[379] Although there is potential toxicity to the fetus with certain pharmacological and nonpharmacological therapies, safe options exist to allow for treating most cases of maternal SVT effectively.

The literature on therapeutic options for the management of arrhythmias in pregnancy is generally limited to single case reports or small series and favors the use of older antiarrhythmic agents because of more abundant reports on the safe use of these drugs. Although all medications have potential side effects to both the mother and the fetus at any stage of pregnancy, if possible, drugs should be avoided in the first trimester, when risk of congenital malformations is greatest. The lowest recommended dose should be used initially, accompanied by regular monitoring of clinical response.

9.3.1. Acute Treatment: Recommendations

Recommendations for Acute Treatment of SVT in Pregnant Patients		
COR	LOE	Recommendations
I	C-LD	1. Vagal maneuvers are recommended for acute treatment in pregnant patients with SVT.[147,380]
I	C-LD	2. Adenosine is recommended for acute treatment in pregnant patients with SVT.[380]
I	C-LD	3. Synchronized cardioversion is recommended for acute treatment in pregnant patients with hemodynamically unstable SVT when pharmacological therapy is ineffective or contraindicated.[380]
IIa	C-LD	1. Intravenous metoprolol or propranolol is reasonable for acute treatment in pregnant patients with SVT when adenosine is ineffective or contraindicated.[380]
IIb	C-LD	1. Intravenous verapamil may be reasonable for acute treatment in pregnant patients with SVT when adenosine and beta blockers are ineffective or contraindicated.[380]
IIb	C-LD	2. Intravenous procainamide may be reasonable for acute treatment in pregnant patients with SVT.[381]
IIb	C-LD	3. Intravenous amiodarone may be considered for acute treatment in pregnant patients with potentially life-threatening SVT when other therapies are ineffective or contraindicated.[382,383]

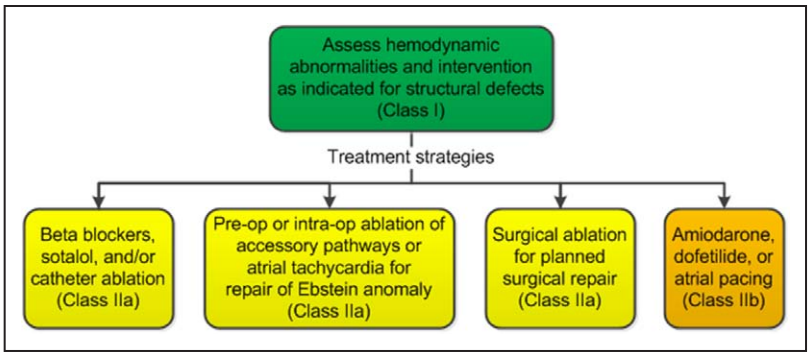

Figure 14. Ongoing management of SVT in ACHD patients. Colors correspond to Class of Recommendation in Table 1; drugs listed alphabetically. ACHD indicates adult congenital heart disease; intra-op, intraoperative; pre-op, preoperative; and SVT, supraventricular tachycardia.

9.3.2. Ongoing Management: Recommendations

Recommendations for Ongoing Management of SVT in Pregnant Patients		
COR	**LOE**	**Recommendations**
IIa	C-LD	1. The following drugs, alone or in combination, can be effective for ongoing management in pregnant patients with highly symptomatic SVT: a. Digoxin[382,384] b. Flecainide[382,384] c. Metoprolol[382,385] d. Propafenone[382] e. Propranolol[382,385] f. Sotalol[382,384] g. Verapamil[382]
IIb	C-LD	1. Catheter ablation may be reasonable in pregnant patients with highly symptomatic, recurrent, drug-refractory SVT with efforts toward minimizing radiation exposure.[386,387]
IIb	C-LD	2. Oral amiodarone may be considered for ongoing management in pregnant patients when treatment of highly symptomatic, recurrent SVT is required and other therapies are ineffective or contraindicated.[382,383]

9.4. SVT in Older Populations

9.4.1. Acute Treatment and Ongoing Management: Recommendation

The natural history of SVT is steadily changing because most patients with SVT undergo ablation at a younger age, but in general, the relative proportion of AT is higher in older populations, and AVNRT is more prevalent than AVRT among patients undergoing ablation.[16] Atypical atrial flutter and macroreentrant AT are on the rise as consequences of increasing AF ablation in this patient population, yet there are limited outcome data from RCTs for this segment of the population. Therapeutic decisions should be balanced between the overall risks and benefits of the invasive nature of ablation versus long-term commitment to pharmacological therapy.

Recommendations for Acute Treatment and Ongoing Management of SVT in Older Populations		
COR	**LOE**	**Recommendation**
I	B-NR	1. Diagnostic and therapeutic approaches to SVT should be individualized in patients more than 75 years of age to incorporate age, comorbid illness, physical and cognitive functions, patient preferences, and severity of symptoms.[27,28,388–396]

10. Quality-of-Life Considerations

Patients with SVT may experience recurring symptoms that negatively impact their quality of life. Episodes of tachycardia can cause lightheadedness and syncope, which can become an obstacle to the performance of usual activities of daily living (eg, driving).[29] However, there are minimal data on the effect of treatment on the quality of life for patients with SVT.

See Online Data Supplement 22 for data supporting Section 10.

11. Cost-Effectiveness

The small body of literature evaluating cost-effectiveness strategies in PSVT has traditionally centered on an evaluation of medical therapy versus catheter ablation. A rigorous cost-effectiveness Markov model was conducted in 2000 to compare radiofrequency ablation to medical management with generic metoprolol from the societal perspective.[57] The estimated population consisted of patients with AVNRT (approximately 65%) and AVRT. On the basis of this simulation, the authors concluded that, for symptomatic patients with monthly episodes of PSVT, radiofrequency ablation was the more effective and less expensive strategy when compared with medical therapy. An observational cohort study of patients with atrial flutter supported early ablation to significantly reduce hospital-based healthcare utilization and the risk of AF.[397]

These studies, along with other older literature, favor catheter ablation over medical therapy as the more cost-effective approach to treating PSVT and atrial flutter. However, the results of these studies were based on cost data and practice patterns that do not apply to the current environment and practice. Therefore, no recommendations are provided.

See Online Data Supplement 23 for data supporting Section 11.

12. Shared Decision Making

It is important that the patient be included in clinical decision-making processes, with consideration of his/her preferences and goals for therapy, as well as his/her unique physical, psychological, and social situation. In selected cases, personalized, self-directed interventions can be developed in partnership with the patient, such as vagal maneuvers and "pill-in-the-pocket" drug therapy.

Shared decision making is especially important for patients with SVT. As seen in this guideline, SVT treatment can be nuanced and requires expert knowledge of EP processes and treatment options. Treatment options are highly specific to the

exact type of arrhythmia and can depend on certain characteristics of a particular arrhythmia. The various choices for therapy, including drugs, cardioversion, invasive treatment, or a combination thereof, can be confusing to the patient, so a detailed explanation of the benefits and risks must be included in the conversation.

Patients are encouraged to ask questions with time allotted for caregivers to respond. Providing a relaxed atmosphere, anticipating patient concerns, and encouraging patients to keep a notebook with questions could facilitate productive conversations.

It is also important that clinicians use lay terminology to explain treatment options to their patients. It is the responsibility of the physician and healthcare team to provide the patient with the best possible understanding of all management options in terms of risks, benefits, and potential effects on quality of life.

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KEY WORDS: AHA Scientific Statements ■ tachycardia, supraventricular ■ tachycardia, atrioventricular nodal reentry ■ Wolff-Parkinson-White syndrome ■ catheter ablation ■ tachycardia, ectopic atrial ■ tachycardia, ectopic junctional ■ atrial flutter ■ anti-arrhythmia agents ■ accessory atrioventricular bundle ■ Valsalva maneuver ■ tachycardia, reciprocating ■ electric countershock ■ heart defects, congenital ■ death, sudden ■ electrophysiologic techniques, cardiac ■ sinus tachycardia

Appendix 1. Author Relationships With Industry and Other Entities (Relevant)—2015 ACC/AHA/HRS Guideline for the Management of Adult Patients With Supraventricular Tachycardia (April 2014)

Committee Member	Employment	Consultant	Speakers Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness	Voting Recusals by Section*
Richard L. Page, Chair	University of Wisconsin School of Medicine and Public Health—Chair, Department of Medicine	None	None	None	None	None	None	None
José A. Joglar, Vice Chair	University of Texas Southwestern Medical Center—Professor of Internal Medicine; Program Director, Clinical Cardiac Electrophysiology	None	None	None	None	None	None	None
Sana M. Al-Khatib	Duke Clinical Research Institute—Associate Professor of Medicine	None	None	None	None	None	None	None
Mary A. Caldwell	University of California San Francisco—Assistant Professor (Retired)	None	None	None	None	None	None	None
Hugh Calkins	Johns Hopkins Hospital—Professor of Medicine, Director of Electrophysiology	• Atricure • Boehringer Ingelheim • Daiichi-Sankyo	None	None	• St. Jude Medical†	None	None	All Sections except 2.4, 5.2, 6.1.2, 9.3.2, and 9.4.
Jamie B. Conti	University of Florida—Professor of Medicine, Chief of Cardiovascular Medicine	None	None	None	• Medtronic	• Boston Scientific‡ • Medtronic‡ • St. Jude Medical‡	None	All Sections except 2.4, 6.1.2, 9.3.2, and 9.4.
Barbara J. Deal	Feinberg School of Medicine, Northwestern University—Professor of Pediatrics; Ann & Robert H. Lurie Children's Hospital of Chicago—Division Head, Cardiology	None	None	None	None	None	None	None
N.A. Mark Estes III	Tufts University School of Medicine—Professor of Medicine	• Boston Scientific† • Medtronic • St. Jude Medical	None	None	• Boston Scientific	• Boston Scientific† • Medtronic† • St. Jude Medical†	None	All Sections except 2.4, 5.2, 6.1.2, 9.3.2, and 9.4.
Michael E. Field	University of Wisconsin School of Medicine and Public Health—Assistant Professor of Medicine, Director of Cardiac Arrhythmia Service	None	None	None	None	None	None	None
Zachary D. Goldberger	University of Washington School of Medicine—Assistant Professor of Medicine	None	None	None	None	None	None	None
Stephen C. Hammill	Mayo Clinic—Professor Emeritus of Medicine	None	None	None	None	None	None	None
Julia H. Indik	University of Arizona—Associate Professor of Medicine	None	None	None	None	None	None	None
Bruce D. Lindsay	Cleveland Clinic Foundation—Professor of Cardiology	• Biosense Webster • Boston Scientific • CardioInsight • Medtronic	None	None	None	• Boston Scientific† • Medtronic† • St. Jude Medical†	None	All Sections except 2.4, 5.2, 6.1.2, 9.3.2, and 9.4.
Brian Olshansky	University of Iowa Hospitals—Professor Emeritus of Medicine; Mercy Hospital Mason City—Electrophysiologist	• BioControl • Biotronik • Boehringer-Ingelheim • Boston Scientific-Guidant • Daiichi-Sankyo • Medtronic† • Sanofi-aventis	None	None	• Amarin (DSMB) • Boston Scientific (DSMB) • Sanofi-aventis (DSMB)	• Boston Scientific	None	All Sections except 2.4 and 9.4.
Andrea M. Russo	Cooper Medical School of Rowan University—Professor of Medicine; Cooper University Hospital—Director, Electrophysiology and Arrhythmia Services	• Biotronik • Boston Scientific • Medtronic • St. Jude Medical	None	None	• Medtronic†	• Biotronik‡ • Boston Scientific†	None	All Sections except 2.4, 5.2, 6.1.2, 9.3.2, and 9.4.

(*Continued*)

Appendix 1. Continued

Committee Member	Employment	Consultant	Speakers Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness	Voting Recusals by Section*
Win-Kuang Shen	Mayo Clinic Arizona—Professor of Medicine; Chair, Division of Cardiovascular Diseases	None	None	None	None	None	None	None
Cynthia M. Tracy	George Washington University—Professor of Medicine; Associate Director Division of Cardiology, Director of Cardiac Services	None	None	None	None	None	None	None

This table represents the relationships of committee members with industry and other entities that were determined to be relevant to this document. These relationships were reviewed and updated in conjunction with all meetings and/or conference calls of the writing committee during the document development process. The table does not necessarily reflect relationships with industry at the time of publication. A person is deemed to have a significant interest in a business if the interest represents ownership of ≥5% of the voting stock or share of the business entity, or ownership of ≥$5,000 of the fair market value of the business entity; or if funds received by the person from the business entity exceed 5% of the person's gross income for the previous year. Relationships that exist with no financial benefit are also included for the purpose of transparency. Relationships in this table are modest unless otherwise noted.

According to the ACC/AHA, a person has a relevant relationship IF: a) the *relationship* or *interest* relates to the same or similar subject matter, intellectual property or asset, topic, or issue addressed in the *document*; or b) the *company/entity* (with whom the relationship exists) makes a drug, drug class, or device addressed in the *document*, or makes a competing drug or device addressed in the *document*; or c) the *person or a member of the person's household*, has a reasonable potential for financial, professional or other personal gain or loss as a result of the issues/content addressed in the document.

*Writing committee members are required to recuse themselves from voting on sections to which their specific relationships with industry and other entities may apply.

†Significant relationship.

‡No financial benefit.

ACC indicates American College of Cardiology; AHA, American Heart Association; DSMB, data safety monitoring board; and HRS, Heart Rhythm Society.

Appendix 2. Reviewer Relationships With Industry and Other Entities (Relevant)—2015 ACC/AHA/HRS Guideline for the Management of Adult Patients With Supraventricular Tachycardia (March 2015)

Reviewer	Representation	Employment	Consultant	Speakers Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness
Eugene H. Chung	Official Reviewer—HRS	University of North Carolina School of Medicine—Associate Professor of Medicine	None	None	None	None	• Zoll Medical‡	None
Timm L. Dickfeld	Official Reviewer—HRS	University of Maryland School of Medicine—Associate Professor of Medicine; Baltimore Veterans Affairs Medical Center—Director, Electrophysiology	• Biosense Webster	None	None	• Biosense Webster* • General Electric*	None	None
Samuel S. Gidding	Official Reviewer—ACC/AHA Task Force on Clinical Practice Guidelines	Nemours Cardiac Center—Division Chief of Cardiology; Jefferson Medical College—Professor of Pediatrics	None	None	None	None	None	None
Richard J. Kovacs	Official Reviewer—ACC Board of Trustees	Krannert Institute of Cardiology—Professor of Clinical Medicine	• Biomedical Systems*	None	None	• Siemens‡	• AstraZeneca (DSMB) • MED Institute* • Eli Lilly (DSMB)* • Teva Pharmaceuticals	None
Byron K. Lee	Official Reviewer—AHA	University of California San Francisco—Professor of Medicine	• Biotronik • Boston Scientific • St. Jude Medical	None	None	• Zoll Medical*	• CarioNet*	• Defendant, Boehringer Ingelheim, 2013‡
Gregory F. Michaud	Official Reviewer—AHA	Harvard Medical School—Assistant Professor	• Boston Scientific • Medtronic • St. Jude Medical	None	None	• Biosense Webster* • Boston Scientific* • St. Jude Medical*	None	None
Simone Musco	Official Reviewer—ACC Board of Governors	The International Heart Institute of Montana Foundation—Cardiology Research Investigator	None	• Bristol-Myers Squibb • Sanofi-aventis	None	None	None	None
Mohan N. Viswanathan	Official Reviewer—AHA	University of Washington School of Medicine—Assistant Professor of Medicine	• Biosense Webster • Siemens‡ • St. Jude Medical	None	None	• Medtronic*	None	None

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Appendix 2. Continued

Reviewer	Representation	Employment	Consultant	Speakers Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness
Seshadri Balaji	Content Reviewer	Oregon Health and Science University—Professor of Pediatrics and Pediatric Cardiology, Director of Pacing and Electrophysiology	None	None	None	• Medtronic*	None	None
Nancy C. Berg	Content Reviewer—ACC Electrophysiology Section	Allina Health System	None	None	None	None	None	None
Noel G. Boyle	Content Reviewer—ACC Electrophysiology Section	University of California Los Angeles—Clinical Professor of Medicine	None	None	None	None	None	None
A. John Camm	Content Reviewer	St. George's University of London—Professor of Clinical Cardiology	• Bayer* • Biotronik • Boehringer Ingelheim • Boston Scientific • ChanRx • Daiichi-Sankyo • Medtronic • Menarini • Mitsubishi • Novartis† • Richmond Pharmacology* • Sanofi-aventis • Servier Pharmaceuticals* • St. Jude Medical • Takeda Pharmaceuticals • Xention	• Pfizer	None	None	None	None
Robert M. Campbell	Content Reviewer—ACC Adult Congenital and Pediatric Cardiology Section	Sibley Heart Center Cardiology—Director, Chief of Cardiac Services; Emory University School of Medicine—Division Director of Pediatric Cardiology, Professor of Pediatrics	None	None	None	None	None	None
Susan P. Etheridge	Content Reviewer—ACC Adult Congenital and Pediatric Cardiology Section	University of Utah—Training Program Director	None	None	None	None	None	None
Paul A. Friedman	Content Reviewer	Mayo Clinic—Professor of Medicine; Cardiovascular Implantable Device Laboratory—Director	• NeoChord	None	None	• Biotronik† • Medtronic • St. Jude Medical	• Preventice • Sorin*	None
Bulent Gorenek	Content Reviewer—ACC Electrophysiology Section	Eskisehir Osmangazi University—Professor and Vice Director, Cardiology Department	None	None	None	None	None	None
Jonathan L. Halperin	Content Reviewer—ACC/AHA Task Force on Clinical Practice Guidelines	Mt. Sinai Medical—Professor of Medicine	• AstraZeneca • Bayer Healthcare • Biotronik† • Boehringer Ingelheim† • Boston Scientific • Daiichi-Sankyo • Johnson & Johnson • Medtronic • Pfizer	None	None	None	None	None

(*Continued*)

Appendix 2. Continued

Reviewer	Representation	Employment	Consultant	Speakers Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness
Warren M. Jackman	Content Reviewer	University of Oklahoma Health Sciences Center—George Lynn Cross Research Professor Emeritus; Heart Rhythm Institute—Senior Scientific Advisor	• Biosense Webster* • Boston Scientific* • VytronUS*	• AtriCure* • Biosense Webster* • Biotronik* • Boston Scientific*	None	None	None	None
G. Neal Kay	Content Reviewer	University of Alabama—Professor Emeritus	None	None	None	None	None	None
George J. Klein	Content Reviewer	London Health Sciences Center—Chief of Cardiology	• Biotronik • Boston Scientific • Medtronic†	None	None	None	None	None
Bradley P. Knight	Content Reviewer	Northwestern University—Professor of Cardiology	• Boston Scientific • Medtronic	• Biosense Webster • Biotronik • Boston Scientific • Medtronic	None	None	None	None
John D. Kugler	Content Reviewer	University of Nebraska Medical Center—Division Chief of Pediatric Cardiology	None	None	None	None	None	None
Fred M. Kusumoto	Content Reviewer	Mayo Clinic—Professor of Medicine	None	None	None	None	None	None
Glenn N. Levine	Content Reviewer—ACC/AHA Task Force on Clinical Practice Guidelines	Baylor College of Medicine—Professor of Medicine; Director, Cardiac Care Unit	None	None	None	None	None	None
Marco A. Mercader	Content Reviewer	George Washington University—Associate Professor of Medicine	None	None	None	None	None	None
William M. Miles	Content Reviewer	University of Florida—Professor of Medicine, Silverstein Chair for Cardiovascular Education, Director of the Clinical Cardiac Electrophysiology Fellowship Program	None	None	None	None	• Medtronic (DSMB)	None
Fred Morady	Content Reviewer	University of Michigan—McKay Professor of Cardiovascular Disease	None	None	None	None	None	None
Melvin M. Scheinman	Content Reviewer	University of California San Francisco—Professor of Medicine	• Amgen • Biosense Webster • Biotronik* • Boston Scientific* • Gilead Sciences • Janssen Pharmaceuticals • Medtronic • St. Jude Medical	None	None	None	None	None
Sarah A. Spinler	Content Reviewer	University of the Sciences, Philadelphia College of Pharmacy—Professor of Clinical Pharmacy	• Portola Pharmaceuticals	None	None	None	None	None
William G. Stevenson	Content Reviewer	Brigham and Women's Hospital—Director, Clinical Cardiac Electrophysiology Program	• St. Jude Medical	None	None	None	None	None
Albert L. Waldo	Content Reviewer	University Hospitals—Associate Chief of Cardiovascular Medicine for Academic Affairs; Case Western Reserve University School of Medicine—Professor of Medicine	• AtriCure • Biosense Webster* • CardioInsight • ChanRx • Daiichi-Sankyo • Gilead Sciences • Pfizer • St. Jude Medical*	• Bristol-Myers Squibb* • Janssen Pharmaceuticals • Pfizer*	None	• Gilead Sciences*	None	None

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Appendix 2. Continued

Reviewer	Representation	Employment	Consultant	Speakers Bureau	Ownership/ Partnership/ Principal	Personal Research	Institutional, Organizational, or Other Financial Benefit	Expert Witness
Edward Walsh	Content Reviewer	Harvard Medical School—Professor of Pediatrics; Boston Children's Hospital—Chief, Division of Cardiac Electrophysiology	• Biosense Webster†	None	None	None	None	None
Richard C. Wu	Content Reviewer	University of Texas Southwestern Medical Center—Professor of Internal Medicine, Director of Cardiac Electrophysiology Lab	None	None	None	• Boehringer Ingelheim • Janssen Pharmaceutical • Medtronic	None	None

This table represents the relationships of reviewers with industry and other entities that were disclosed at the time of peer review and determined to be relevant to this document. It does not necessarily reflect relationships with industry at the time of publication. A person is deemed to have a significant interest in a business if the interest represents ownership of ≥5% of the voting stock or share of the business entity, or ownership of ≥$5000 of the fair market value of the business entity; or if funds received by the person from the business entity exceed 5% of the person's gross income for the previous year. A relationship is considered to be modest if it is less than significant under the preceding definition. Relationships that exist with no financial benefit are also included for the purpose of transparency. Relationships in this table are modest unless otherwise noted. Names are listed in alphabetical order within each category of review.

According to the ACC/AHA, a person has a *relevant* relationship IF: a) the *relationship or interest* relates to the same or similar subject matter, intellectual property or asset, topic, or issue addressed in the *document*; or b) the *company/entity* (with whom the relationship exists) makes a drug, drug class, or device addressed in the *document*, or makes a competing drug or device addressed in the *document*; or c) the *person or a member of the person's household*, has a reasonable potential for financial, professional or other personal gain or loss as a result of the issues/content addressed in the *document*.

*Significant relationship.

†No financial benefit.

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