Overview of the acute management of tachyarrhythmias

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Literature review current through: **Jan 2024.**
This topic last updated: **Aug 31, 2023.**

INTRODUCTION

Tachyarrhythmias, defined as abnormal heart rhythms with a ventricular rate of 100 or more beats per minute, are frequently symptomatic and often result in patients seeking care at their provider's office or the emergency department. Signs and symptoms related to the tachyarrhythmia may include shock, hypotension, heart failure, shortness of breath, chest pain, acute myocardial infarction, palpitations, and/or decreased level of consciousness. An overview of the management of these various arrhythmias will be presented here. More complete reviews of the individual arrhythmias are discussed separately.

INITIAL DIAGNOSTIC AND TREATMENT DECISIONS

In patients who present with a symptomatic tachyarrhythmia, a 12-lead electrocardiogram (ECG) should be obtained while a brief initial assessment of the patient's overall clinical assessment is performed. If the patient is hemodynamically unstable, it may be preferable to obtain only a rhythm strip prior to urgent cardioversion and not wait for a 12-lead ECG. The information acquired from these initial assessments is crucial for subsequent management of the patient.

Is the patient clinically (or hemodynamically) unstable? — The most important clinical determination in a patient presenting with a tachyarrhythmia is whether or not the patient is

experiencing signs and symptoms related to the rapid heart rate. These can include hypotension, shortness of breath, chest pain suggestive of coronary ischemia, shock, and/or decreased level of consciousness.

Determining whether a patient's symptoms are related to the tachycardia depends upon several factors, including age and the presence of underlying cardiac disease.

- **Hemodynamically unstable and not sinus rhythm** – If a patient has clinically significant hemodynamic instability potentially due to the tachyarrhythmia, an attempt should be made as quickly as possible to determine whether the rhythm is sinus tachycardia (algorithm 1). If the rhythm is not sinus tachycardia, or if there is any doubt that the rhythm is sinus tachycardia, **urgent conversion** to sinus rhythm is recommended. (See "Narrow QRS complex tachycardias: Clinical manifestations, diagnosis, and evaluation", section on 'Similar to sinus rhythm' and "Basic principles and technique of external electrical cardioversion and defibrillation" and "Wide QRS complex tachycardias: Approach to the diagnosis", section on 'Assessment of hemodynamic stability'.)

- **Hemodynamically stable** – If the patient is not experiencing hemodynamic instability, a nonemergent approach to the diagnosis of the patient's rhythm can be undertaken [1-3]. A close examination of the 12-lead ECG should permit the correct identification of the arrhythmia in 80 percent of cases [4]. (See 'Is the QRS complex narrow or wide? Regular or irregular?' below and "Narrow QRS complex tachycardias: Clinical manifestations, diagnosis, and evaluation", section on 'Evaluation' and "Wide QRS complex tachycardias: Approach to the diagnosis", section on 'Evaluation of the electrocardiogram'.)

Is the QRS complex narrow or wide? Regular or irregular? — Treatment of any tachyarrhythmia depends on a variety of clinical factors. However, most treatment decisions are made based on the width, morphology, and regularity of the QRS complex (algorithm 2). In most patients, the differentiation between narrow and wide QRS complex tachyarrhythmias requires only a surface ECG.

- Narrow QRS complex tachyarrhythmias have a QRS complex <120 milliseconds in duration
- Wide QRS complex tachyarrhythmias have a QRS complex ≥120 milliseconds in duration

The various types of narrow and wide QRS complex tachyarrhythmias are discussed below. (See 'Narrow QRS complex tachyarrhythmias' below and 'Wide QRS complex tachyarrhythmias' below.)

NARROW QRS COMPLEX TACHYARRHYTHMIAS

Discussion of the treatment of narrow QRS complex tachycardias will be divided into those with a regular ventricular response and those with an irregular ventricular response (algorithm 3 and algorithm 1).

Regular narrow QRS complex tachyarrhythmias — The regular narrow QRS complex tachycardias include (algorithm 2) [3]:

- Sinus tachycardia (see "Sinus tachycardia: Evaluation and management")
- Inappropriate sinus tachycardia (see "Sinus tachycardia: Evaluation and management", section on 'Inappropriate sinus tachycardia')
- Sinoatrial nodal reentrant tachycardia (SANRT) (see "Sinoatrial nodal reentrant tachycardia (SANRT)")
- Atrioventricular nodal reentrant tachycardia (AVNRT) (see "Atrioventricular nodal reentrant tachycardia")
- Atrioventricular reentrant (or reciprocating) tachycardia (AVRT) (see "Atrioventricular reentrant tachycardia (AVRT) associated with an accessory pathway")
- Atrial tachycardia (AT) (see "Focal atrial tachycardia")
- Atrial flutter (see "Overview of atrial flutter")
- Intraatrial reentrant tachycardia (IART) (see "Intraatrial reentrant tachycardia")
- Junctional ectopic tachycardia
- Nonparoxysmal junctional tachycardia

Because the vast majority of regular narrow QRS complex tachycardias are due to sinus tachycardia, AVNRT, AVRT, AT, and atrial flutter, these conditions will be presented here. Discussions regarding the treatment of the other less common types of regular narrow QRS complex tachycardias are presented separately. (See "Sinus tachycardia: Evaluation and management", section on 'Inappropriate sinus tachycardia' and "Sinoatrial nodal reentrant tachycardia (SANRT)", section on 'Treatment' and "Intraatrial reentrant tachycardia", section on 'Treatment' and "Treatment of arrhythmias associated with the Wolff-Parkinson-White syndrome", section on 'Permanent junctional reciprocating tachycardia'.)

Sinus tachycardia — The most common tachycardia is sinus tachycardia. If it is certain that the patient's rhythm is sinus tachycardia and clinically significant cardiac symptoms are present, management should be focused on the underlying disorder and on treating any contributing cause of the rapid heart rate (eg, coronary ischemia, pulmonary embolism, respiratory or cardiac failure, hypovolemia, anemia, hyperthyroidism, fever, pain, or anxiety). This may include volume replacement or diuresis, antibiotics, anti-pyretics, oxygen, pain control, or other treatments as appropriate. In patients with sinus tachycardia and certain forms of heart disease, such as coronary disease or aortic stenosis, treatment may need to be directed at the

heart rate itself. In such cases, cautious use of an intravenous beta blocker is appropriate. (See "Sinus tachycardia: Evaluation and management" and "Acute myocardial infarction: Role of beta blocker therapy" and "Medical management of symptomatic aortic stenosis".)

Atrioventricular nodal reentrant tachycardia (AVNRT) — Patients with AVNRT associated with hemodynamic compromise or severe symptoms due to the tachycardia (eg, angina, hypotension, or heart failure) require rapid termination of the arrhythmia. (See "Atrioventricular nodal reentrant tachycardia", section on 'Initial management'.)

- For patients with AVNRT who are hemodynamically unstable related to their arrhythmia, we recommend immediate DC cardioversion. Consideration for using vagal maneuvers (Valsalva maneuver or carotid sinus massage) is also reasonable if it does not delay cardioversion. (See "Vagal maneuvers".)

- For patients with AVNRT associated with severe symptoms due to the tachycardia (eg, angina, hypotension, heart failure, or mental status changes) in whom intravenous access is available, we suggest an initial attempt at termination with adenosine (algorithm 4) rather than cardioversion. If adenosine cannot be administered or is ineffective, patients should undergo immediate DC cardioversion.

- For patients with AVNRT that is not associated with severe symptoms or hemodynamic collapse, including patients without symptoms, we suggest the following sequential approach to acute termination:

  - Vagal maneuvers (see "Vagal maneuvers")
  - IV adenosine (algorithm 4)
  - IV non-dihydropyridine calcium channel blocker or an IV beta blocker

Atrioventricular reentrant tachycardia (AVRT) — Patients with arrhythmias that may be caused or complicated by the presence of an accessory pathway (ie, orthodromic AVRT, antidromic AVRT, preexcited atrial fibrillation/flutter) should have a prompt initial assessment of hemodynamic status. The management of AVRT depends on the stability of the patient and whether the mechanism of tachycardia is known or unknown. The approach to management of orthodromic AVRT, which most commonly presents with narrow QRS complexes, is discussed separately. (See "Treatment of arrhythmias associated with the Wolff-Parkinson-White syndrome", section on 'Orthodromic AVRT'.)

Atrial tachycardia — Focal atrial tachycardias (AT), usually paroxysmal and self-limited, arise from a single site or area of microreentry or enhanced automaticity outside of the sinus node. (See "Focal atrial tachycardia", section on 'Acute treatment'.)

- For patients with AT who are felt to be hemodynamically unstable related to their arrhythmia, we recommend immediate DC cardioversion.

- For a hemodynamically stable patient with symptomatic AT, we suggest acute treatment with an oral or intravenous beta blocker or non-dihydropyridine calcium channel blocker (ie, diltiazem or verapamil). Such treatment may slow the ventricular response and/or terminate the arrhythmia. Intravenous amiodarone is an acceptable alternative that may be preferred in a patient with borderline hypotension as amiodarone may slow the rate or convert the rhythm back to normal sinus.

Atrial flutter — Atrial flutter usually presents as a regular narrow complex tachycardia, though it occasionally may have an irregular ventricular response. Atrial flutter should always be considered high on the differential diagnosis when a patient presents with a regular narrow complex tachycardia with a ventricular response of approximately 150 beats per minute. As with atrial fibrillation, the early steps in the management of a patient with new onset atrial flutter involve an assessment of the need for cardioversion, ventricular rate slowing therapy, and antithrombotic therapy. Our initial approach to the management of patients with atrial flutter is the same as our approach for atrial fibrillation. (See 'Atrial fibrillation' below.)

Irregular narrow QRS complex tachyarrhythmias — The irregular narrow QRS complex tachycardias include (algorithm 2):

- Atrial fibrillation (AF) (see "Atrial fibrillation: Overview and management of new-onset atrial fibrillation")
- Atrial flutter with variable conduction (see "Overview of atrial flutter")
- Focal atrial tachycardia with variable conduction (see "Focal atrial tachycardia")
- Multifocal atrial tachycardia (MAT) (see "Multifocal atrial tachycardia")

Atrial fibrillation — Most patients with new onset (ie, first detected or diagnosed) AF with a rapid rate present with symptoms related to the arrhythmia. Except for embolization, the symptoms associated with new onset AF are primarily due to a rapid and/or irregular ventricular response. The early steps in the management of a patient with new onset rapid AF involve an assessment of the need for cardioversion, ventricular rate slowing therapy, and antithrombotic therapy. (See "Atrial fibrillation: Overview and management of new-onset atrial fibrillation".)

- Urgent or emergent cardioversion should be considered for patients with active ischemia, significant hypotension, severe heart failure, or the presence of a preexcitation syndrome associated with rapid conduction using the accessory pathway. (See 'Atrioventricular reentrant tachycardia (AVRT)' above.)

- For all patients who do not require urgent or emergent cardioversion, we recommend rate control to improve symptoms and to reduce the risk of tachycardia-mediated cardiomyopathy. We believe a goal of less than 110 beats per minute is reasonable for an asymptomatic patient with a normal ejection fraction. Beta blockers and non-dihydropyridine calcium channel blockers are preferred as first-line agents in most patients, and digoxin should only rarely be used. Intravenous preparations are preferred to oral preparations when rapid control of rate is necessary.

- For patients with AF less than 48 hours in duration in whom cardioversion is planned, the use of antithrombotic therapy pre-cardioversion to reduce the risk of embolization can be considered.

- For patients with AF longer than 48 hours in duration (or of unknown duration), we recommend four weeks of therapeutic oral anticoagulation prior to cardioversion, as opposed to immediate cardioversion. Transesophageal echocardiography-based (TEE) screening for the presence of atrial thrombi is recommended if cardioversion is desired earlier than four weeks. Anticoagulation must be continued for a minimum of four weeks after cardioversion. Whether long-term anticoagulation is indicated depends on assessment of the patient's thromboembolic risk profile. (See "Atrial fibrillation in adults: Selection of candidates for anticoagulation".)

Atrial flutter — As with atrial fibrillation, the early steps in the management of a patient with new onset atrial flutter involve an assessment of the need for cardioversion, ventricular rate slowing therapy, and antithrombotic therapy. Our initial approach to the management of patients with atrial flutter is the same as our approach for atrial fibrillation. (See 'Atrial fibrillation' above.)

Multifocal atrial tachycardia — Multifocal atrial tachycardia (MAT) is an arrhythmia with organized atrial activity yielding P waves with three or more different morphologies. MAT is commonly associated with significant underlying pulmonary or cardiac illness. (See "Multifocal atrial tachycardia", section on 'Treatment'.)

- Most episodes of MAT do not precipitate hemodynamic compromise or limiting symptoms. Thus, therapy in patients with MAT should be aimed at the inciting underlying disease.

- Patients with MAT and associated hypokalemia or hypomagnesemia should undergo electrolyte repletion prior to the initiation of additional medical therapy for MAT.

- Medical therapy for MAT is indicated only if MAT causes a sustained rapid ventricular response that causes or worsens myocardial ischemia, heart failure, peripheral perfusion,

or oxygenation. Options for medical therapy for patients with symptomatic MAT requiring ventricular rate control include non-dihydropyridine calcium channel blockers and beta blockers. For patients without heart failure or bronchospasm, we suggest initial therapy with a beta blocker, usually metoprolol, before calcium channel blockers. Conversely, for patients with severe bronchospasm, we suggest initial therapy with a non-dihydropyridine calcium channel blocker, usually verapamil, rather than a beta blocker. Beta blockers may be used cautiously in patients with stable heart failure. Rate control therapy is typically unsuccessful, however, without treating the underlying disorder.

WIDE QRS COMPLEX TACHYARRHYTHMIAS

Discussion of the treatment of wide QRS complex tachycardias, similar to narrow QRS complex tachycardias, can be divided into those with a regular or irregular ventricular rate.

Regular wide QRS complex tachyarrhythmias — The regular wide QRS complex tachycardias include (algorithm 2):

- Monomorphic ventricular tachycardia (VT). (See "Sustained monomorphic ventricular tachycardia in patients with structural heart disease: Treatment and prognosis" and "Ventricular tachycardia in the absence of apparent structural heart disease".)
- Supraventricular tachycardia with aberrant conduction, underlying conduction delay, conduction over an accessory pathway (eg, AVNRT with right bundle branch block), or a paced ventricular response.
- Supraventricular tachycardia in a patient on certain antiarrhythmic medications or with significant electrolyte abnormalities.
- Antidromic AVRT.

The most concerning potential cause of a wide QRS complex tachycardia is VT, and, in the majority of patients, the arrhythmia should be assumed to be VT until proven otherwise.

Immediate assessment of patient stability takes precedence over any further diagnostic evaluation. (See "Wide QRS complex tachycardias: Approach to the diagnosis", section on 'Summary and recommendations'.)

- A patient who is unresponsive or pulseless should be treated according to standard advance cardiac life support (ACLS) algorithms (algorithm 5).

- In a patient who is unstable but conscious, we recommend immediate synchronized cardioversion with appropriate sedation when possible.

- In a stable patient, a focused diagnostic evaluation may proceed to determine the etiology of the arrhythmia and guide specific therapy.

Ventricular tachycardia — In stable patients with known or presumed VT, we recommend the following approach (see "Wide QRS complex tachycardias: Approach to the diagnosis", section on 'Summary and recommendations'):

- We recommend synchronized external cardioversion, following appropriate sedation, as the initial therapy for most patients with stable VT. If the patient has an implantable cardioverter-defibrillator, it may be possible to terminate the arrhythmia by antitachycardia pacing prior to an attempted cardioversion.

- In patients with refractory or recurrent wide complex tachycardia (WCT), we suggest an intravenous class I or III antiarrhythmic drug (table 1), such as amiodarone, lidocaine, or procainamide (algorithm 6).

- In selected patients known to have one of the syndromes of VT in the setting of a structurally normal heart, we suggest calcium channel blockers or beta blockers be used for arrhythmia termination or suppression. However, the decision to use these drugs in this setting should be made in consultation with a cardiologist experienced in arrhythmia management.

Supraventricular tachycardia with aberrant conduction — The narrow complex supraventricular tachycardia (SVT) rhythms may present with a wide complex in the setting of aberrant conduction or conduction over an accessory pathway (not including AVRT).

- In stable patients with a WCT that is **known** to be an SVT, initial management is similar to that of an SVT with a narrow QRS complex. A continuous rhythm strip should be obtained during any intervention that is intended to slow or terminate the arrhythmia. (See 'Regular narrow QRS complex tachyarrhythmias' above.)

- For AVNRT or AVRT, or an SVT in which the specific arrhythmia is **unknown**, we suggest the following sequence of interventions in order to terminate the arrhythmia or to slow ventricular response and facilitate diagnosis in stable patients:

  - Vagotonic maneuvers (eg, Valsalva or carotid sinus pressure)
  - Intravenous adenosine (algorithm 4)
  - Intravenous calcium channel blockers or beta blockers

- Cardioversion in selected persistent cases, or if the patient is unstable

Supraventricular tachycardia with a pacemaker — Regular wide QRS complex tachycardias in patients with a pacemaker may be due to tracking of one of the typical supraventricular tachycardias (eg, sinus tachycardia, atrial flutter, etc) or may be due to pacemaker-mediated tachycardia (PMT, also referred to endless loop tachycardia [ELT]). (See "Unexpected rhythms with normally functioning dual-chamber pacing systems", section on 'Pacemaker-mediated tachycardia'.)

- In patients with tracking of a native supraventricular tachyarrhythmia, the pacemaker usually should automatically mode switch to a non-tracking mode. If it does not, placing a magnet on the pacemaker will lead to asynchronous pacing at a fixed and lower rate, and the pacemaker settings can be adjusted to prevent rapid pacing.

- If the rhythm is due to PMT, retrograde conduction from the ventricle to the atrium is sensed by the pacemaker and serves as a trigger to pace the ventricle, which again conducts back to the atrium and perpetuates the tachycardia. Placing a magnet on the pacemaker leads to asynchronous pacing and will stop the tachycardia. Most pacemakers have algorithms to prevent or treat PMT, but pacemaker settings can usually be reprogrammed if they are ineffective.

Antidromic AVRT — For patients with acute symptomatic antidromic AVRT (regular and wide QRS complexes) who are hemodynamically stable, the approach to acute management is discussed separately. (See "Treatment of arrhythmias associated with the Wolff-Parkinson-White syndrome", section on 'Antidromic AVRT'.)

Irregular wide QRS complex tachyarrhythmias — The irregular wide QRS complex tachycardias include (algorithm 2):

- Polymorphic VT, including torsades de pointes. (See "Acquired long QT syndrome: Definitions, pathophysiology, and causes".)

- Irregular narrow complex tachycardias with aberrant conduction, antegrade conduction over an accessory pathway (eg, preexcited AF), or underlying conduction delay (eg, AF with right bundle branch block).

- Ventricular fibrillation.

Polymorphic ventricular tachycardia — Polymorphic ventricular tachycardia (VT) is defined as an unstable rhythm with a continuously varying QRS complex morphology in any recorded electrocardiographic (ECG) lead. Polymorphic VT is generally a rapid and hemodynamically

unstable rhythm, and urgent defibrillation is usually necessary. In addition to immediate defibrillation, further therapy is intended to treat underlying disorders and to prevent recurrences. The specific approach depends upon whether or not the QT interval on the baseline ECG is prolonged. Polymorphic VT that occurs in the setting of QT prolongation in sinus rhythm is considered as a distinct arrhythmia, called torsades de pointes. (See "Acquired long QT syndrome: Definitions, pathophysiology, and causes".)

- Prompt defibrillation is indicated in patients with hemodynamically unstable torsades de pointes.
- In the conscious patient with recurrent episodes of torsades de pointes:
  - Intravenous magnesium sulfate (initial dose of 1 to 2 grams IV over 15 minutes, may be followed by an infusion) is first-line therapy, as it is highly effective for both treatment and prevention of recurrence of long QT-related ventricular ectopic beats that trigger torsades de pointes. The benefit is seen even in patients with normal serum magnesium concentrations at baseline.
  - Temporary transvenous overdrive pacing (atrial or ventricular) at about 100 beats per minute is generally reserved for patients who do not respond to intravenous magnesium.
  - In those with congenital long QT syndrome, beta blockers may be used to reduce the frequency of premature ventricular contractions and shorten the QT interval.
  - For patients with polymorphic VT triggered by pauses or bradycardia, isoproterenol (initial dose 0.05 to 0.1 mcg/kg per minute in children and 2 mcg/minute in adults, then titrated to achieve a heart rate of 100 beats per minute) can be used as a temporizing measure to achieve a heart rate of 100 beats per minute prior to pacing.
- For patients with polymorphic VT and a normal baseline QT interval, the most likely cause is myocardial ischemia. Treatments may include:
  - Prompt defibrillation in the hemodynamically unstable patient.
  - Beta-blockers if blood pressure tolerates. Metoprolol 5 mg intravenously every five minutes, to a total of 15 mg, may be given.
  - IV amiodarone may prevent a recurrent episode.
  - Urgent coronary angiography and possible revascularization.

- Short-term mechanical circulatory support.
- Magnesium is less likely to be effective for polymorphic VT if the baseline QT interval is normal.

- If the polymorphic VT is due to catecholaminergic polymorphic ventricular tachycardia (CPVT), beta blockers should be used. If it is due to Brugada syndrome, isoproterenol should be initiated. (See "Catecholaminergic polymorphic ventricular tachycardia", section on 'Acute management' and "Brugada syndrome or pattern: Management and approach to screening of relatives".)

Preexcited atrial fibrillation — Patients with preexcited atrial fibrillation are typically treated with agents that minimize the risk of rapid conduction of atrial fibrillation or atrial flutter directly to the ventricle. The approach to patients with an accessory pathway and atrial fibrillation or flutter is discussed separately. (See "Treatment of arrhythmias associated with the Wolff-Parkinson-White syndrome", section on 'Atrial fibrillation with preexcitation'.)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Atrial fibrillation" and "Society guideline links: Arrhythmias in adults" and "Society guideline links: Ventricular arrhythmias" and "Society guideline links: Basic and advanced cardiac life support in adults" and "Society guideline links: Supraventricular arrhythmias".)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a

variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see "Patient education: Tachycardia (The Basics)" and "Patient education: Ventricular tachycardia (The Basics)" and "Patient education: Supraventricular tachycardia (SVT) (The Basics)")

SUMMARY AND RECOMMENDATIONS

- **Initial diagnostic and treatment decisions** – In patients who present with a symptomatic tachyarrhythmia, a 12-lead ECG should be obtained while a brief initial assessment of the patient's overall clinical assessment is performed. If the patient is hemodynamically unstable, it may be preferable to obtain only a rhythm strip prior to urgent cardioversion and not wait for a 12-lead ECG. The information acquired from these initial assessments is crucial for subsequent management of the patient (see 'Initial diagnostic and treatment decisions' above):
  - **Narrow QRS complex tachycardias** – If the QRS complex is narrow (algorithm 2), the approach to acute management is based on whether the patient is stable (algorithm 3) or unstable (algorithm 1). (See 'Narrow QRS complex tachyarrhythmias' above.)
  - **Wide QRS complex tachycardias** – If the QRS complex is wide (algorithm 2), the approach to acute management is based on whether the patient is stable or unstable; pulseless patients should be treated according to the adult cardiac life support algorithm (algorithm 5). (See 'Wide QRS complex tachyarrhythmias' above.)

ACKNOWLEDGMENTS

The UpToDate editorial staff acknowledges Philip Podrid, MD, FACC, and Leonard Ganz, MD, FHRS, FACC, who contributed to an earlier version of this topic review.

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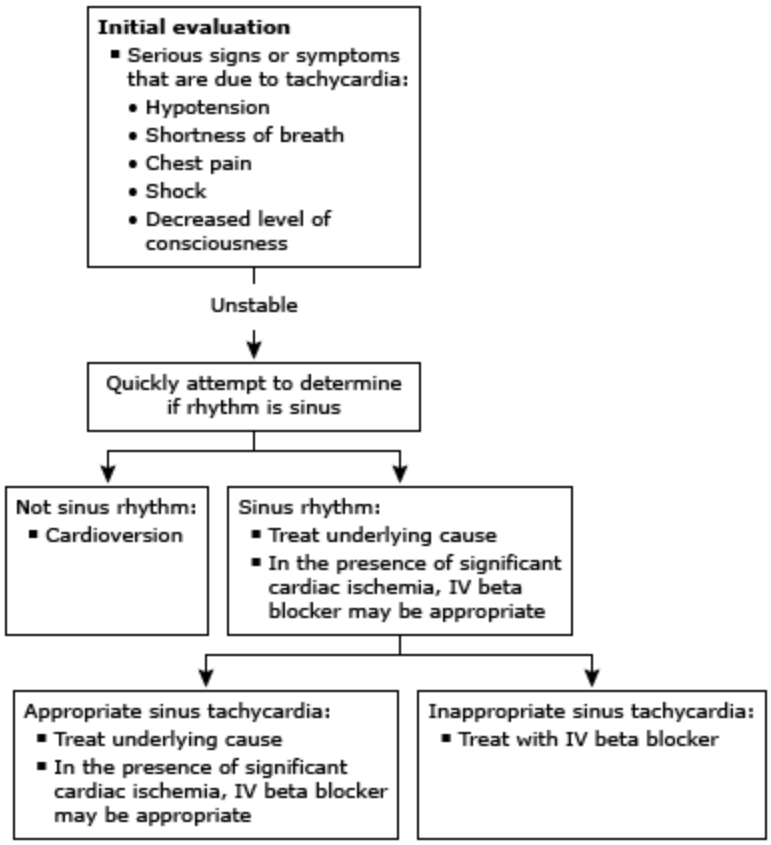
Topic 936 Version 45.0

GRAPHICS

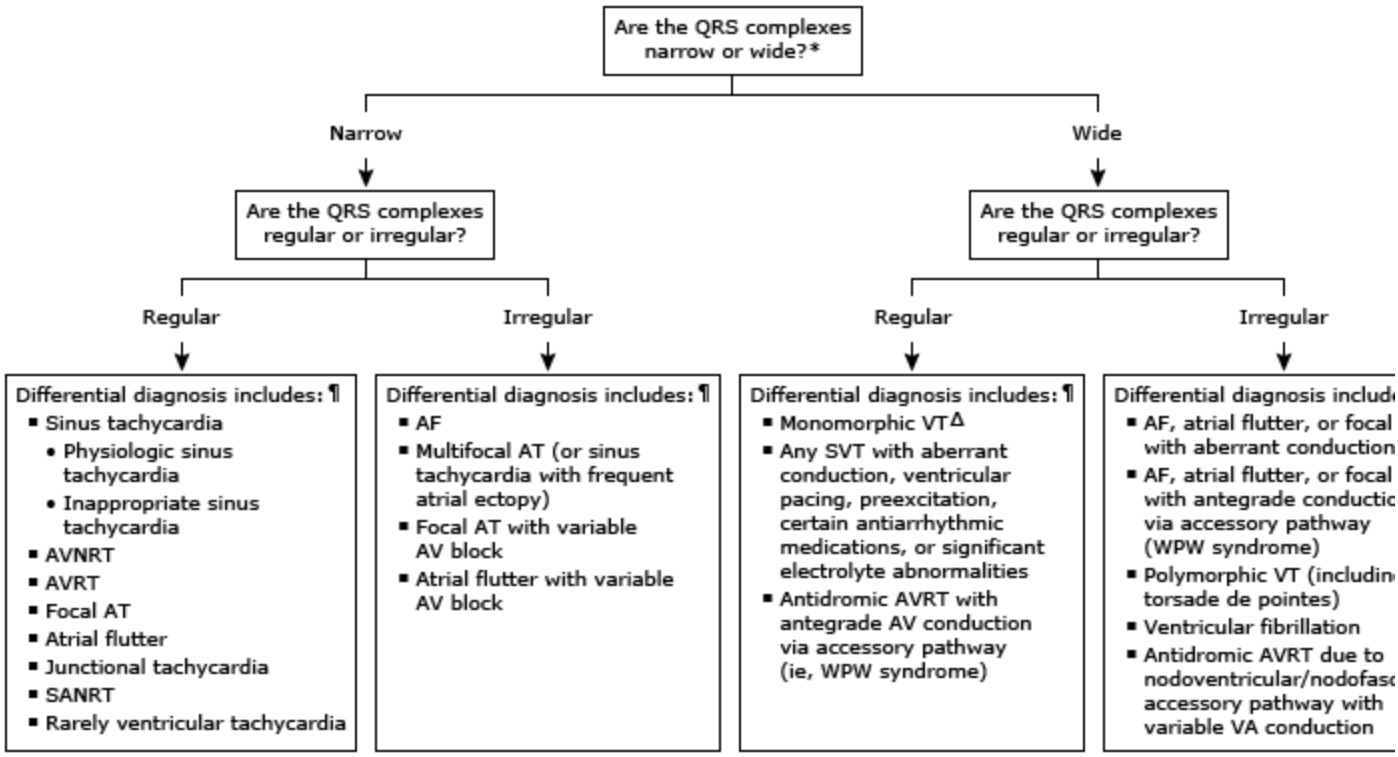
Algorithm for the evaluation of narrow QRS complex tachycardias in unstable patients



IV: intravenous.

Graphic 109811 Version 2.0

Algorithm for the initial ECG review and differential diagnosis of tachycardia



ECG: electrocardiogram; AVNRT: atrioventricular nodal reentrant tachycardia; AVRT: atrioventricular reciprocating (bypass-tract mediated) tachycardia; AT: atrial tachycardia; SANRT: sinoatrial nodal reentrant tachycardia; AF: atrial fibrillation; AV: atrioventricular; VT: ventricular tachycardia; SVT: supraventricular tachycardia; WPW: Wolff-Parkinson-White.

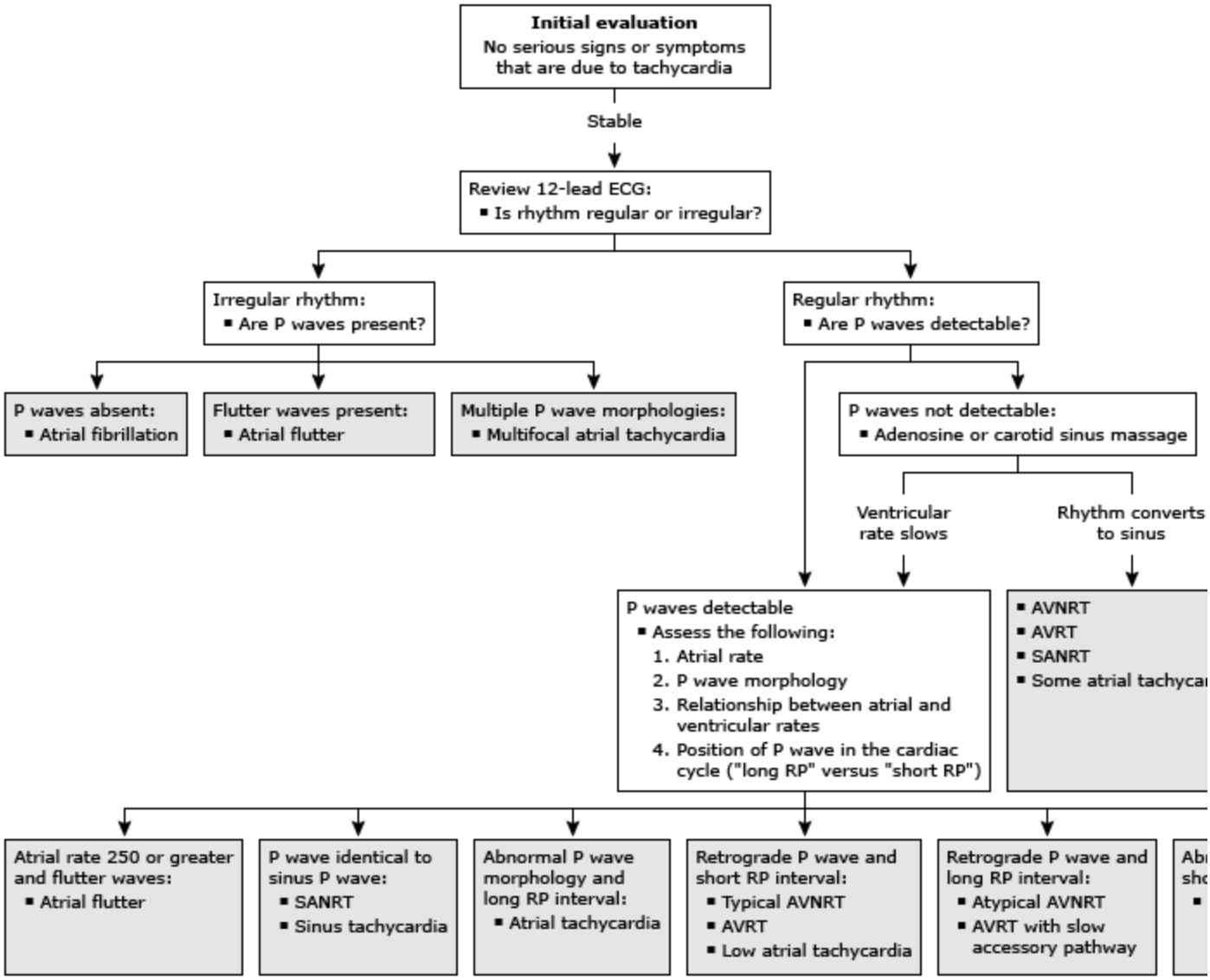
* A narrow QRS complex is <120 milliseconds in duration, whereas a wide QRS complex is ≥120 milliseconds in duration.

¶ Refer to UpToDate topic reviews for additional details on specific ECG findings and management of individual arrhythmias.

Δ Monomorphic VT accounts for 80% of wide QRS complex tachycardias; refer to UpToDate topic on diagnosis of wide QRS complex tachycardias for additional information on discriminating VT from SVT.

Graphic 117571 Version 3.0

Algorithm for the evaluation of narrow QRS complex tachycardias in stable patients



ECG: electrocardiogram; AVNRT: atrioventricular nodal reentrant tachycardia; AVRT: atrioventricular reentrant tachycardia (due to an accessory pathway); SANRT: sinoatrial nodal reentrant tachycardia.

Graphic 76920 Version 4.0

Approach to adenosine dosing in stable adults with suspected supraventricular tachyarrhythmia

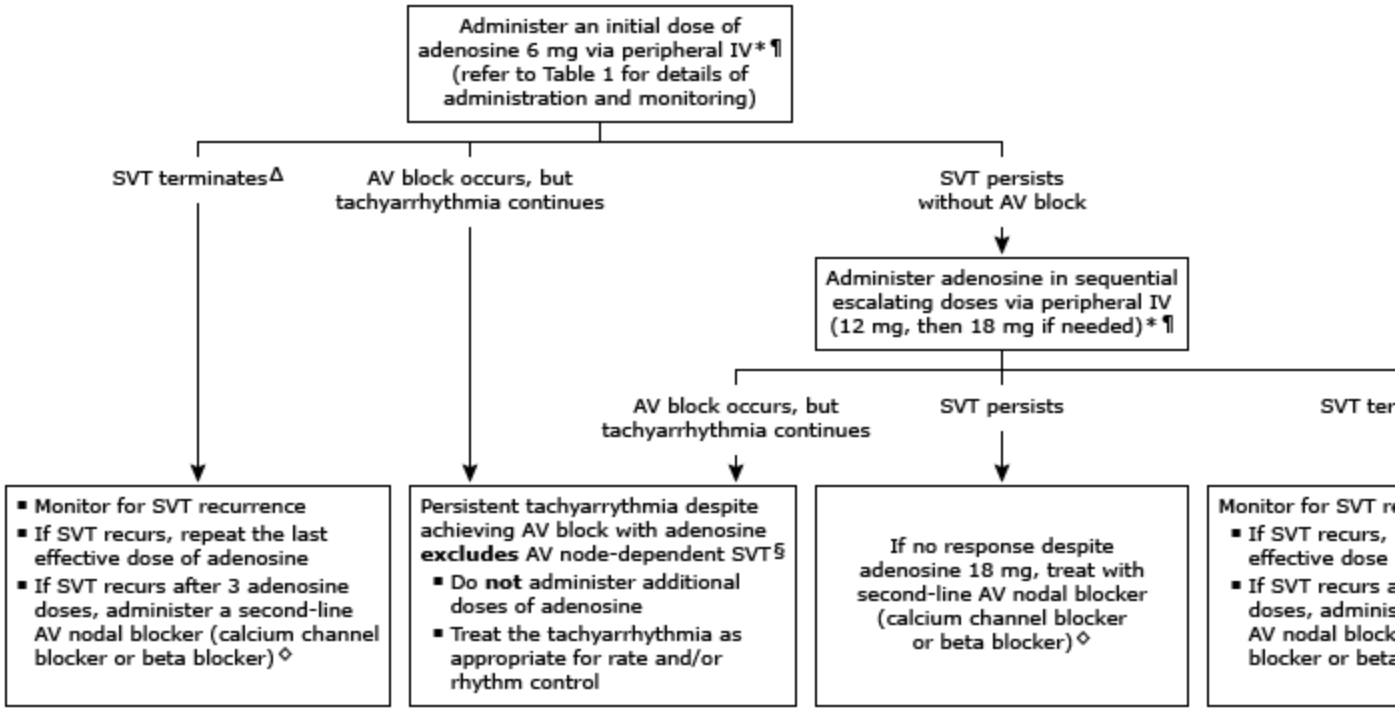


Table 1

Best practices for adenosine administration:

- The patient should be supine during adenosine administration and subsequent monitoring.
- Monitor blood pressure and record a 12-lead ECG during and for 1 to 2 minutes after administration to assess the response to adenosine. If a 12-lead ECG is impractical, record telemetry.
- Administer adenosine in the most proximal available peripheral IV site (eg, antecubutal vein); administration via a hand IV site is generally avoided. If central venous administration of adenosine is performed, lower doses are used than for peripheral administration.¶
- Administer adenosine via a 3-way stopcock, with 1 port leading to the patient, 1 port for the adenosine dose, and 1 port for normal saline flush (10 to 20 mL). Both the adenosine and normal saline syringes should be attached before adenosine is given.
- The IV adenosine dose is pushed over 1 to 2 seconds, immediately followed by turning the stopcock for normal saline flush over 1 to 2 seconds.

This figure summarizes the initial dosing, administration, and need for repeat dosing of adenosine in a hemodynamically stable adult with suspected AV node-dependent SVT. Adenosine can be used as a diagnostic aid to identify the underlying rhythm or as a therapy to terminate SVTs that rely on AV node conduction. AV node-dependent SVTs include AV nodal reentrant tachycardia (AVNRT) and AV reentrant (or reciprocating) tachycardia (AVRT). The management of suspected AV node-dependent SVT in hemodynamically stable adults commonly starts with a trial of 1 or more vagal maneuvers prior to proceeding to adenosine therapy. If the patient has hemodynamically unstable SVT, cardioversion is indicated. Refer to UpToDate content for details on management of SVTs.

AV: atrioventricular; SVT: supraventricular tachycardia; IV: intravenous; ACC: American College of Cardiology; AHA: American Heart Association; HRS: Heart Rhythm Society; ECG: electrocardiogram.

* Lower adenosine doses are indicated in certain clinical settings. If central venous administration is performed, the initial adenosine dose is 3 mg; if needed (SVT persists and there is no AV block), the dose may be increased to 6 mg and then 9 mg for subsequent doses. For heart transplant recipients, the initial adenosine dose is 1 mg; if needed (SVT persists and there is no AV block), the dose may be increased to 2 mg and then 3 mg for subsequent doses.

¶ An 18 mg dose is more likely to be required to produce AV block in patients with a body weight >70 kg, particularly in those weighing >110 kg.[1] When a third dose is indicated, an alternative approach is to administer 12 mg (instead of 18 mg) as described in the 2015 ACC/AHA/HRS Guideline for the Management of Adult Patients with Supraventricular Tachycardia.[2]

Δ SVT termination in response to adenosine includes SVT termination closely followed by SVT recurrence.

◇ Refer to UpToDate content on treatment of SVT for dosing of second-line AV nodal blockers. If the SVT persists despite second-line AV nodal blocker therapy, electrical cardioversion is performed, as discussed in UpToDate content on treatment of SVT.

§ SVTs that are **not** AV node dependent include atrial flutter, atrial fibrillation, and atrial tachycardia. When AV nodal block is induced by adenosine, the ECG may reveal atrial activity diagnostic of these rhythms.

References:

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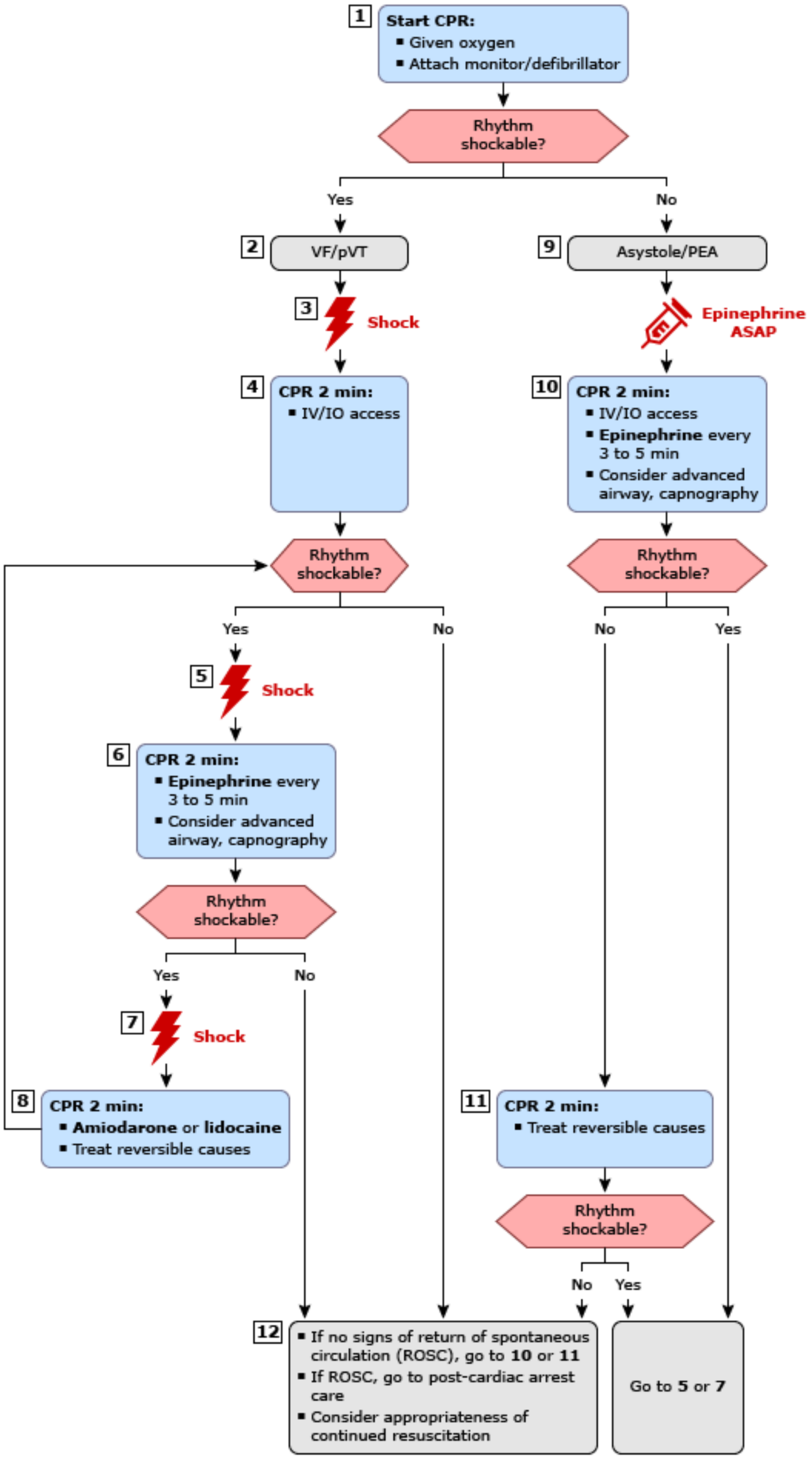
Graphic 141417 Version 3.0

Adult cardiac arrest algorithm



CPR quali

- Push hard (at least 2 inch
fast (100 to 120/min) and
chest recoil.
- Minimize interruptions in
- Avoid excessive ventilatio
- Change compressor every
sooner if fatigued.
- If no advanced airway, 30
ventilation ratio.
- Quantitative waveform ca
  - If $PETCO_2$ is low or decr
CPR quality.

Shock energy for d

- **Biphasic:** Manufacturer r
(eg, initial dose of 120 to
unknown, use maximum
and subsequent doses sh
and higher doses may be
- **Monophasic:** 360 J.

Drug thera

- **Epinephrine IV/IO dos**
1 mg every 3 to 5 minute
- **Amiodarone IV/IO dos**
First dose: 300 mg bolus.
Second dose: 150 mg.
or
- **Lidocaine IV/IO dose:**
First dose: 1 to 1.5 mg/kg
Second dose: 0.5 to 0.75

Advanced ai

- Endotracheal intubation o
advanced airway.
- Waveform capnography o
confirm and monitor ET t
- Once advanced airway in
breath every 6 seconds (1
with continuous chest com

Return of spontaneous c

- Pulse and blood pressure.
- Abrupt sustained increase
(typically ≥40 mmHg).
- Spontaneous arterial pres
intra-arterial monitoring.

Reversible c

- **H**ypovolemia
- **H**ypoxia
- **H**ydrogen ion (acidosis)
- **H**ypo-/hyperkalemia
- **H**ypothermia
- **T**ension pneumothorax
- **T**amponade, cardiac
- **T**oxins
- **T**hrombosis, pulmonary
- **T**hrombosis, coronary

Graphic 129983 Version 10.0

Revised (2018) Vaughan Williams classification of antiarrhythmic drugs abridged table

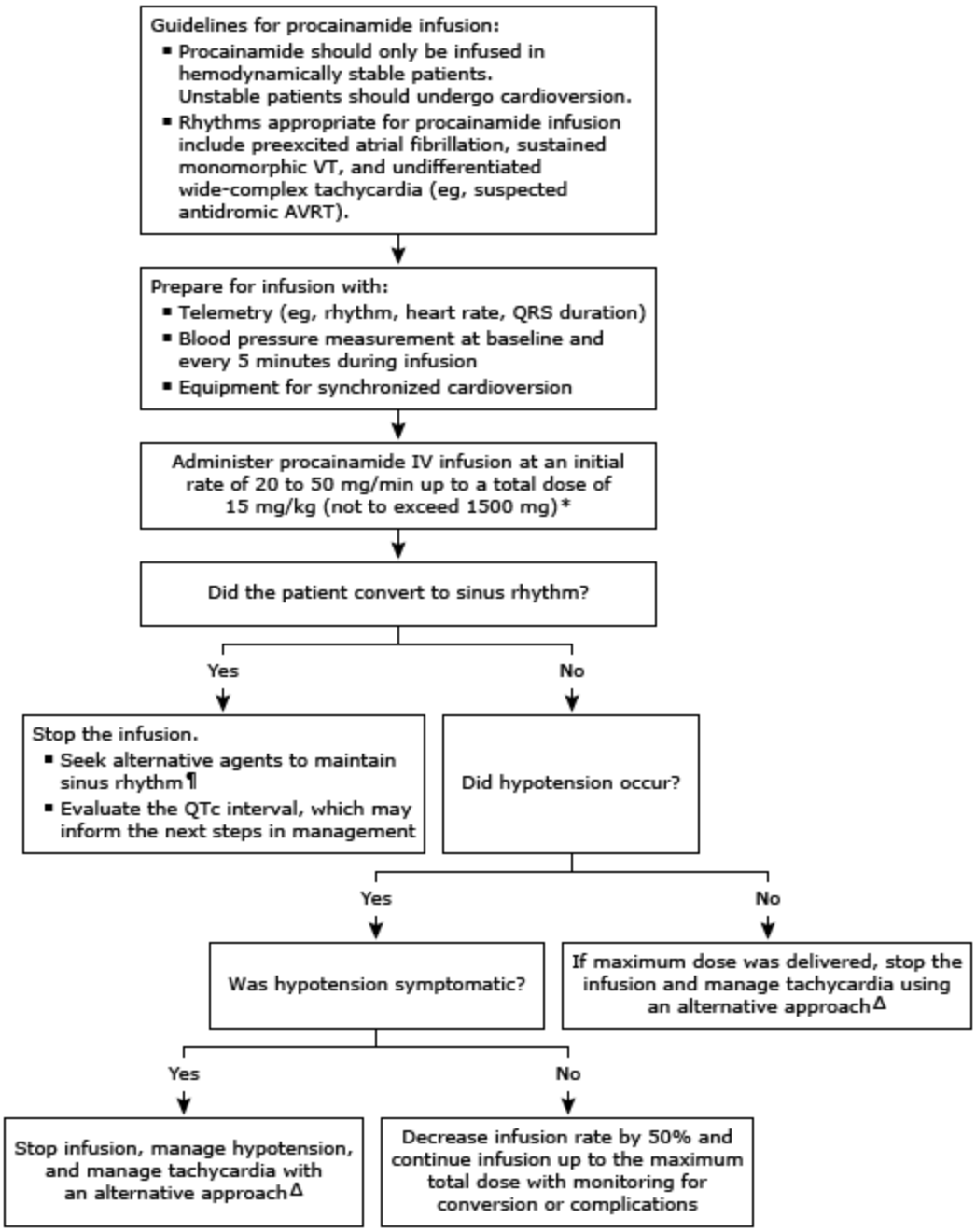
Class 0 (HCN channel blockers)
Ivabradine
Class I (voltage-gated Na+ channel blockers)
Class Ia (intermediate dissociation):
▪ Quinidine, ajmaline, disopyramide, procainamide
Class Ib (rapid dissociation):
▪ Lidocaine, mexilitine
Class Ic (slow dissociation):
▪ Propafenone, flecainide
Class Id (late current):
▪ Ranolazine
Class II (autonomic inhibitors and activators)
Class IIa (beta blockers):
▪ Nonselective: carvedilol, propranolol, nadolol
▪ Selective: atenolol, bisoprolol, betaxolol, celiprolol, esmolol, metoprolol
Class IIb (nonselective beta agonists):
▪ Isoproterenol
Class IIc (muscarinic M2 receptor inhibitors):
▪ Atropine, anisodamine, hyoscine, scopolamine
Class IId (muscarinic M2 receptor activators):
▪ Carbachol, pilocarpine, methacholine, digoxin
Class IIe (adenosine A1 receptor activators):
▪ Adenosine
Class III (K+ channel blockers and openers)
Class IIIa (voltage dependent K+ channel blockers):
▪ Ambasilide, amiodarone, dronedarone, dofetilide, ibutilide, sotalol, vernakalant

Class IIIb (metabolically dependent K+ channel openers):
▪ Nicorandil, pinacidil
Class IV (Ca++ handling modulators)
Class IVa (surface membrane Ca++ channel blockers):
▪ Bepridil, diltiazem, verapamil
Class IVb (intracellular Ca++ channel blockers):
▪ Flecainide, propafenone
Class V (mechanosensitive channel blockers):
No approved medications
Class VI (gap junction channel blockers)
No approved medications
Class VII (upstream target modulators)
Angiotensin converting enzyme inhibitors
Angiotensin receptor blockers
Omega-3 fatty acids
Statins

HCN: hyperpolarization-activated cyclic nucleotide-gated; Na: sodium; K: potassium; Ca: calcium.

Graphic 120433 Version 3.0

Approach to procainamide infusion in adults



VT: ventricular tachycardia; AVRT: atrioventricular reentrant tachycardia; IV: intravenous; QTc interval: corrected QT interval; NAPA: N-acetylprocainamide.

* Some sources describe a maximum dose of 17 mg/kg[1]. Higher infusion rates are more likely to cause hypotension. Refer to Lexicomp for specific dose adjustments for older patients and those with kidney or liver dysfunction.

¶ Maintenance infusion of procainamide is associated with toxicity (eg, QRS and QTc interval prolongation, torsades de pointes in susceptible patients); seek alternative agents for maintenance of sinus rhythm. If procainamide is selected for ongoing treatment, may administer at 1 to 4 mg/min. Some experts use higher infusion rates (eg, 6 to 10 mg/min). If infusion of procainamide extends beyond 12 to 24 hours, measure QRS duration, QTc interval, and procainamide levels twice daily. Signs of toxicity

include QRS prolongation of more than 50% compared with QRS in sinus rhythm at the onset of therapy or a QTc interval greater than normal sex-specific values. Procainamide toxicity may begin at levels >9 mcg/mL but varies considerably between patients. Some experts also measure NAPA levels, but toxic and therapeutic levels are not well-defined.

Δ Alternative agents and approaches available for management depend on the specific tachycardia. For details, refer to UpToDate content on specific arrhythmias (eg, AVRT, preexcited atrial fibrillation, wide-complex tachycardia).

Reference:

1. *Neumar RW, Otto CW, Link MS, et al. Part 8: adult advanced cardiovascular life support: 2010 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. Circulation 2010; 122:S729.*

Graphic 141920 Version 4.0