Nonsustained ventricular tachycardia: Clinical manifestations, evaluation, and management

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INTRODUCTION

Nonsustained ventricular tachycardia (NSVT) is a common but poorly understood arrhythmia. It is usually asymptomatic and most often diagnosed during cardiac monitoring (eg, continuous ambulatory electrocardiography or inpatient telemetry) or on an exercise test performed for other reasons.

The presence of NSVT has long been recognized as a potential marker for the development of sustained ventricular arrhythmias and sudden death. However, while NSVT predicts overall mortality, it doesn't specifically predict sudden cardiac death (SCD). Unfortunately, our understanding of which patients with NSVT are at greatest risk for lethal arrhythmias or how the NSVT relates to the lethal arrhythmias is still quite rudimentary. One clearly established premise is that NSVT in the presence of structural heart disease carries a more serious prognosis than NSVT in the absence of a cardiac abnormality. Since NSVT doesn't specifically predict SCD, the nature of the underlying structural heart disease is the primary determinant of mortality. (See "Nonsustained VT in the absence of apparent structural heart disease".)

There are two general goals in the management of NSVT:

- Identification of patients at risk for malignant, sustained arrhythmias and SCD
- Treatment to suppress symptoms caused by NSVT, when present and clinically significant

This topic will review the diagnosis and management of NSVT. Detailed discussions of risk stratification for arrhythmic death after a myocardial infarction, and the roles of implantable cardioverter-defibrillators and antiarrhythmic drugs in such patients, are presented separately. (See "Incidence of and risk stratification for sudden cardiac death after myocardial infarction" and "Ventricular arrhythmias during acute myocardial infarction: Prevention and treatment".)

DEFINITION OF NSVT

A variety of definitions of NSVT have been published, but the most commonly used definition is [1]:

- Three or more consecutive ventricular beats
- Rate of >100 beats per minute
- Duration of less than 30 seconds

The variable published definitions of NSVT have included:

- Rates ranging from as low as 100 beats per minute to as high as 140 beats per minute
- Between three and five consecutive ventricular complexes
- Durations as short as 15 seconds to as high as one minute
- Beat limits as low as 15 beats or as high as 99 beats, beyond which the arrhythmia is considered sustained

The approach to distinguishing VT from other causes of wide QRS complex tachycardias (ie, supraventricular tachycardia [SVT] with aberrant conduction, SVT with preexcitation, pacemaker-associated tachycardia, or artifact) is discussed separately. (See 'Differential diagnosis' below and "Sustained monomorphic ventricular tachycardia: Clinical manifestations, diagnosis, and evaluation", section on 'Differential diagnosis'.)

CLINICAL MANIFESTATIONS

The history, physical examination, and 12-lead electrocardiogram (ECG) can all provide information helping to confirm the diagnosis of NSVT.

History and associated symptoms — Patients with NSVT are usually asymptomatic, although some patients may notice symptoms associated with episodes of NSVT. Most patients with NSVT will have a history of underlying structural heart disease (eg, coronary heart disease, heart failure, hypertrophic cardiomyopathy, congenital heart disease, etc), although NSVT can also be

seen in patients without known structural heart disease. (See "Nonsustained VT in the absence of apparent structural heart disease".)

The type and intensity of symptoms, if present, will vary depending upon the rate and duration of the NSVT along with the presence or absence of significant comorbid conditions. Patients with NSVT who notice symptoms typically present with one or more of the following symptoms:

- Palpitations
- Chest pain
- Shortness of breath
- Syncope or presyncope

Most commonly, symptomatic patients will report palpitations that may or may not be associated with chest pain and/or shortness of breath. If the duration of the episode approaches 20 to 30 seconds with an associated rate of NSVT that is rapid enough to result in hemodynamic compromise, patients may experience presyncope or even syncope.

Physical examination — Few physical examination findings in patients with NSVT are unique and specific. By definition, patients will have a pulse exceeding 100 beats per minute during the episode. In addition, if the physical examination coincides with an episode of NSVT, this can reveal evidence of atrioventricular (AV) dissociation, which is present in up to 75 percent of patients with VT, although it is not always easy to detect [2-4]. During AV dissociation, the normal coordination of atrial and ventricular contraction is lost, which may produce characteristic physical examination findings including (see "Wide QRS complex tachycardias: Approach to the diagnosis", section on 'AV dissociation'):

- Marked fluctuations in the blood pressure because of the variability in the degree of left atrial contribution to left ventricular filling, stroke volume, and cardiac output.

- Variability in the occurrence and intensity of heart sounds (especially S1) ("cacophony of heart sounds"), which is heard more frequently when the rate of the tachycardia is slower.

- Cannon "A" waves – Cannon A waves are intermittent and irregular jugular venous pulsations of greater amplitude than normal waves. They reflect simultaneous atrial and ventricular activation, resulting in contraction of the right atrium against a closed tricuspid valve. Prominent A waves can also be seen during some SVTs. Such prominent waves result from simultaneous atrial and ventricular contraction occurring with every beat. (See "Examination of the jugular venous pulse".)

Electrocardiogram — All patients with suspected NSVT should have a 12-lead electrocardiogram, although NSVT is frequently identified on continuous telemetric monitoring, in which case only one or two leads may be available for review. As with the interpretation of any ECG, the standard initial approach to diagnosis of NSVT includes an assessment of rate, regularity, axis, QRS duration, and QRS morphology. NSVT typically generates a wide QRS complex, usually with a QRS width >0.12 seconds. A full discussion of the ECG features of VT is presented separately. (See "Sustained monomorphic ventricular tachycardia: Clinical manifestations, diagnosis, and evaluation", section on 'Electrocardiogram' and "Wide QRS complex tachycardias: Approach to the diagnosis", section on 'Evaluation of the electrocardiogram'.)

Diagnostic evaluation — Once NSVT has been identified, reversible causes of arrhythmia should be sought, including electrolyte imbalances, myocardial ischemia, hypoxia, adverse drug effects, anemia, hypotension, and heart failure. For patients who have only a single asymptomatic episode of NSVT, often no further investigation is required. However, for patients with multiple episodes or for those with symptoms felt to be related to NSVT, a thorough diagnostic evaluation to exclude structural heart disease is warranted, including cardiac imaging and ambulatory ECG monitoring for most patients and invasive electrophysiology studies (EPS) only on rare occasions.

- For patients with recurrent episodes or those who are highly symptomatic, even young, otherwise healthy patients need a thorough cardiac imaging evaluation to exclude entities such as undiagnosed dilated cardiomyopathy, hypertrophic cardiomyopathy, or arrhythmogenic right ventricular cardiomyopathy. Typically, the evaluation for structural heart disease includes an imaging study of the heart, most commonly echocardiography, although cardiac magnetic resonance (CMR) imaging is also reasonable (if locally available) as it provides the most detailed structural information.

- Continuous ambulatory ECG monitoring is indicated in many patients with NSVT with vague symptoms to establish a correlation between symptoms and arrhythmia and to quantify the frequency of the arrhythmia. Ambulatory monitoring is also useful to exclude the presence of sustained VT.

- Exercise treadmill testing is indicated in patients with exercise-related symptoms or NSVT and in patients with suspected coronary ischemia.

- In patients with suspected arrhythmogenic right ventricular cardiomyopathy (ARVC), a signal-averaged ECG can be useful.

- Invasive EPS are rarely required in the initial evaluation of NSVT. In patients with syncope, near-syncope, or sustained palpitations, EPS should be considered to evaluate for the presence of sustained VT. In addition, in patients with ischemic cardiomyopathy, ejection fraction 35 to 40 percent, and NSVT, invasive EPS may be used to risk stratify patients for implantable cardioverter-defibrillator implantation [5,6].

An in-depth discussion of the diagnostic evaluation of patients with VT is presented separately. (See "Sustained monomorphic ventricular tachycardia: Clinical manifestations, diagnosis, and evaluation", section on 'Additional diagnostic evaluation'.)

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for a wide QRS complex tachycardia (WCT) includes NSVT, supraventricular tachycardia with aberrant conduction (either preexistent or rate-related), supraventricular tachycardia with preexcitation, supraventricular tachycardia in a pacemaker-dependent patient, and electrocardiogram (ECG) artifact. Differentiating VT from other causes of WCT may be difficult, particularly if a high-quality 12-lead ECG is not available during the time of the arrhythmia. In general, however, a WCT, particularly when poorly tolerated, should be considered to be VT until proven otherwise.

The approach to the differential diagnosis of VT from other causes of WCT is discussed separately. (See "Sustained monomorphic ventricular tachycardia: Clinical manifestations, diagnosis, and evaluation", section on 'Differential diagnosis'.)

TREATMENT

Symptomatic patients — Patients with symptomatic NSVT should usually be treated with beta blockers as the initial therapy. Many patients with NSVT will have coexisting cardiac conditions in which beta blockers are also indicated (eg, coronary heart disease, heart failure), in which case the patient may derive multiple benefits from the use of beta blockers. For patients with NSVT who remain symptomatic in spite of beta blockers, or who are unable to tolerate beta blockers due to side effects, nondihydropyridine calcium channel blockers (ie, verapamil and diltiazem) can be added to the medical regimen, although these agents should only be used in patients with structurally normal hearts and should not be used in patients with uncontrolled heart failure. Antiarrhythmic medications are generally reserved for patients with severely symptomatic NSVT despite therapy with beta blockers and nondihydropyridine calcium channel blockers who are not candidates for catheter ablation of the VT.

NSVT is most often asymptomatic, but some patients experience palpitations, chest pain, shortness of breath, presyncope, or syncope. Because many of the symptoms that may be attributed to NSVT are vague and nonspecific, it is important to try to correlate symptoms to episodes of NSVT before initiating therapy specifically to treat NSVT.

Beta blockers — For the initial treatment of patients with symptomatic NSVT, we suggest beta blockers. This preference is based on significant indirect evidence of the efficacy of beta blockers for reducing ventricular ectopy and tachyarrhythmias in other cardiac conditions, as well as coadministration of beta blockers for other cardiac conditions and the fact that beta blockers are generally safe and well tolerated [5]. Metoprolol (usual effective dose 50 to 200 mg daily) and carvedilol (usual effective dose 12.5 to 50 mg daily) are the most commonly prescribed beta blockers for the suppression of NSVT.

Nondihydropyridine calcium channel blockers — For patients with NSVT who remain symptomatic in spite of beta blockers, or who are unable to tolerate beta blockers due to side effects, we suggest adding a nondihydropyridine calcium channel blockers (ie, verapamil [usual effective dose 360 to 480 mg daily] or diltiazem [usual effective dose 240 to 360 mg daily]) rather than an antiarrhythmic medication. For most patients, nondihydropyridine calcium channel blockers are better tolerated and associated with less toxicity than antiarrhythmic drugs. However, nondihydropyridine calcium channel blockers should not be used in patients with structural heart disease or uncontrolled heart failure. (See "Calcium channel blockers in the treatment of cardiac arrhythmias", section on 'Ventricular arrhythmia'.)

Antiarrhythmic drugs — For some patients who have frequent, highly symptomatic NSVT not adequately suppressed by beta blockers or calcium channel blockers, the addition of antiarrhythmic medications (table 1) may be helpful. We suggest amiodarone as the initial choice, rather than other antiarrhythmic drugs, based on its efficacy. However, due to potential toxicities of antiarrhythmic drugs, we use them sparingly and only in the most symptomatic patients who have failed other medical therapy and who are not candidates, decline, or fail ablation therapy.

Amiodarone may be given at a dose of 200 mg three times daily for two weeks, then 200 mg two times daily for two weeks, and then 200 mg daily. The dose can be further reduced to 100 mg daily if there is concern over toxicity, and follow-up monitoring should be performed.

Mexiletine is usually given as 150 to 200 mg every eight hours. These two drugs require careful monitoring, particularly in patients with structural heart disease. In selected cases, other agents such as sotalol or procainamide can be used for suppression of NSVT, but these medications are rarely used and must be used with caution (algorithm 1).

Amiodarone — Amiodarone is the most effective antiarrhythmic agent for suppressing VT, an effect that has clearly been demonstrated in multiple trials of amiodarone in post-myocardial infarction (MI) and congestive heart failure patients in whom baseline and follow-up 24-hour ambulatory ECGs were performed [7-9].

- In the Canadian Amiodarone Myocardial Infarction Arrhythmia Trial (CAMIAT) pilot study, which compared amiodarone with placebo in patients with frequent or repetitive asymptomatic ventricular premature beats (VPBs), patients receiving amiodarone had significantly fewer VPBs and episodes of NSVT [7].

- In the CHF-STAT trial, which compared amiodarone with placebo in patients with heart failure, left ventricular ejection fraction (LVEF) of 40 percent or less, and frequent ventricular premature beats (more than 10 per hour), significantly fewer patients on amiodarone had ventricular tachycardia on Holter monitor (33 versus 76 percent) after two weeks of therapy [10].

Similar results have been seen in patients treated with both amiodarone and a beta blocker [11,12]. In an analysis of eight trials that included over 5000 patients with a prior MI and frequent VPBs (median frequency 18 per hour), amiodarone therapy was associated with a significant 35 percent reduction in arrhythmic/sudden death, and a nonsignificant 8 percent reduction in total mortality [12].

While amiodarone has never been shown to reduce overall mortality, its use is not associated with an increase in mortality due to proarrhythmia (as is the case with class IC antiarrhythmic drugs). However, its well-described, potentially toxic side effects need to be considered before prescribing, and routine monitoring of liver, thyroid, and lung function should be performed in patients on amiodarone. (See "Amiodarone: Clinical uses" and "Amiodarone: Adverse effects, potential toxicities, and approach to monitoring".)

Class I agents — In selected cases, Class IA drugs (eg, procainamide) and class IB drugs (eg, mexiletine) can be used for suppression of NSVT, but these medications must be used with caution. Class IC drugs are generally not used in patients with structural heart disease because of safety concerns. (See "Nonsustained VT in the absence of apparent structural heart disease".)

The Cardiac Arrhythmia Suppression Trial (CAST) evaluated the efficacy of flecainide, encainide, and moricizine in suppressing ventricular ectopy in almost 1500 post-MI patients, many of whom had depressed LV function [13,14]. Additionally, only 20 percent of patients had NSVT and only 10 percent had more than one run in 24 hours. The findings were dramatic, showing an increase in arrhythmic sudden death and total cardiovascular mortality in the treated

patients even though the original ventricular ectopy was suppressed (figure 1). Thus, class IC agents are not used in the treatment of NSVT in coronary heart disease.

Radiofrequency catheter ablation — In patients who have very frequent, symptomatic monomorphic NSVT not controlled by medications or who are unable or unwilling to take medications, catheter ablation can be effective for reducing or eliminating NSVT and associated symptoms [1]. It would be appropriate to consider catheter ablation as a primary alternative to antiarrhythmic drugs. This strategy is most commonly used in patients with idiopathic, triggered arrhythmias, which often originate in the outflow tracts, septum, or papillary muscles. In such patients, catheter ablation can be a highly successful procedure to eliminate the symptoms of arrhythmia [15]. (See "Ventricular tachycardia in the absence of apparent structural heart disease".)

Implantable cardioverter-defibrillators — Implantable cardioverter-defibrillators (ICDs) are not indicated for the treatment of NSVT as NSVT is self-limited and self-terminating. However, some patients with NSVT who are found to have a cardiomyopathy may be a candidate for ICD placement for primary prevention of sudden cardiac death related to sustained ventricular tachyarrhythmias. The use of an ICD for primary prevention is discussed in detail separately. (See "Primary prevention of sudden cardiac death in patients with cardiomyopathy and heart failure with reduced LVEF".)

Asymptomatic patients — Patients with NSVT and no identified symptoms do not require any specific therapy directed toward the NSVT. However, patients with NSVT and associated underlying cardiac comorbidities (eg, coronary heart disease, heart failure) should be treated with optimal medical therapy as indicated for the relevant associated condition. (See "Chronic coronary syndrome: Overview of care".)

The Multicenter Unsustained Tachycardia Trial (MUSTT trial), which was not primarily designed as a randomized ICD clinical trial but rather to study the management of high-risk patients using the results of electrophysiology study (EPS), enrolled 704 patients with a prior MI (less than one month to more than three years previously), asymptomatic NSVT, an LVEF ≤40 percent, and inducible sustained ventricular tachycardia [6,16]. Patients were randomly assigned to no therapy or EP-guided antiarrhythmic therapy, which included either an antiarrhythmic agent (class IA with or without mexiletine, propafenone, sotalol, or amiodarone) or an ICD if at least one antiarrhythmic agent was ineffective; the primary end point was arrhythmic death or resuscitated cardiac arrest, with a secondary endpoint of total mortality. The reduction in the primary and secondary end points in the electrophysiologically guided group was largely attributable to ICD therapy; at five years the primary end point occurred in 9 percent of those receiving an ICD, compared with 37 percent of those receiving an antiarrhythmic drug, and the

secondary end point occurred in 24 and 55 percent, respectively. There was no difference in outcome between patients receiving no therapy and those treated with an antiarrhythmic drug (figure 2) [17].

Patients with asymptomatic NSVT may also be considered for an ICD in the presence of structural heart disease with LVEF <40 percent. Many patients will meet other criteria for primary prevention with an ICD. For post-MI patients with moderate LV dysfunction (ie, LVEF 35 to 40 percent), who do not otherwise meet current criteria for ICD implantation, NSVT remains an indication for EPS and possible ICD implantation. A full discussion of the role of ICDs in primary and secondary prevention of sudden cardiac death is presented separately. (See "Primary prevention of sudden cardiac death in patients with cardiomyopathy and heart failure with reduced LVEF" and "Secondary prevention of sudden cardiac death in heart failure and cardiomyopathy".)

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: Ventricular 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: Ventricular tachycardia (The Basics)")

SUMMARY AND RECOMMENDATIONS

- **Definition** – The most common definition is three or more consecutive ventricular beats, a heart rate of >100 beats per minute, and a duration of arrhythmia of less than 30 seconds. (See 'Definition of NSVT' above.)

- **Symptoms** – Patients with nonsustained ventricular tachycardia (NSVT) are usually asymptomatic, although some patients may notice symptoms associated with episodes of NSVT. Symptoms may include palpitations, chest pain, shortness of breath, syncope, or presyncope. Symptoms may vary depending upon the rate and duration of the NSVT along with the presence or absence of significant comorbid conditions. (See 'History and associated symptoms' above.)

- **Physical examination** – By definition, the pulse rate is >100 beats per minute.

  Few physical examination findings are unique and specific for NSVT.

  If the physical examination coincides with an episode of NSVT, this can reveal evidence of atrioventricular (AV) dissociation, including marked fluctuations in blood pressure, variability in the occurrence and intensity of heart sounds (especially S1), and cannon A waves. (See 'Physical examination' above.)

- **Evaluation** – All patients with suspected NSVT should have a 12-lead electrocardiogram (ECG), although NSVT is frequently identified on continuous telemetry monitoring, in which case only one or two leads may be available for review. (See 'Electrocardiogram' above.)

  - Reversible causes – Once identified, reversible causes of NSVT should be sought, including electrolyte imbalances, myocardial ischemia, hypoxia, adverse drug effects, anemia, hypotension, and heart failure.

  - Single asymptomatic episode – Often, for these patients, no further investigation is required.

  - Multiple or symptomatic episodes – For patients with multiple episodes or with symptoms felt to be related to NSVT, a thorough diagnostic evaluation to exclude structural heart disease is warranted, including cardiac imaging and ambulatory ECG monitoring for most patients and invasive electrophysiology studies (EPS) only on rare occasions. (See 'Diagnostic evaluation' above.)

- **Treatment**

  - **Asymptomatic patients** – In general, asymptomatic patients do not require any specific therapy directed toward the NSVT.

    However, some asymptomatic patients with NSVT who are found to have infarct-related cardiomyopathy with significantly reduced left ventricular systolic function may be evaluated for implantable cardioverter-defibrillator placement for primary prevention of sudden cardiac death related to sustained ventricular tachyarrhythmias. (See 'Asymptomatic patients' above and "Primary prevention of sudden cardiac death in patients with cardiomyopathy and heart failure with reduced LVEF".)

  - **Symptomatic patients**

    - **Initial therapy** – For the initial treatment of patients with symptomatic NSVT, we suggest beta blockers rather than calcium channel blockers or antiarrhythmic medications (**Grade 2C**). (See 'Beta blockers' above.)

      For patients with NSVT who remain symptomatic in spite of beta blockers, or who are unable to tolerate beta blockers due to side effects, we suggest adding a nondihydropyridine calcium channel blocker (ie, verapamil or diltiazem) rather than an antiarrhythmic medication (**Grade 2C**). (See 'Nondihydropyridine calcium channel blockers' above.)

    - **Alternative therapy** – For some patients who have frequent, highly symptomatic NSVT not adequately suppressed by beta blockers or calcium channel blockers, the addition of antiarrhythmic medications (table 1) may be helpful. We suggest amiodarone as the initial choice, rather than other antiarrhythmic drugs, based on its efficacy (**Grade 2C**). (See 'Antiarrhythmic drugs' above.)

      In patients with very frequent symptomatic monomorphic NSVT not controlled by medications or who are unable or unwilling to take medications, catheter ablation can be effective for reducing or eliminating NSVT and associated symptoms. (See 'Radiofrequency catheter ablation' above.)

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Topic 917 Version 36.0

GRAPHICS

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

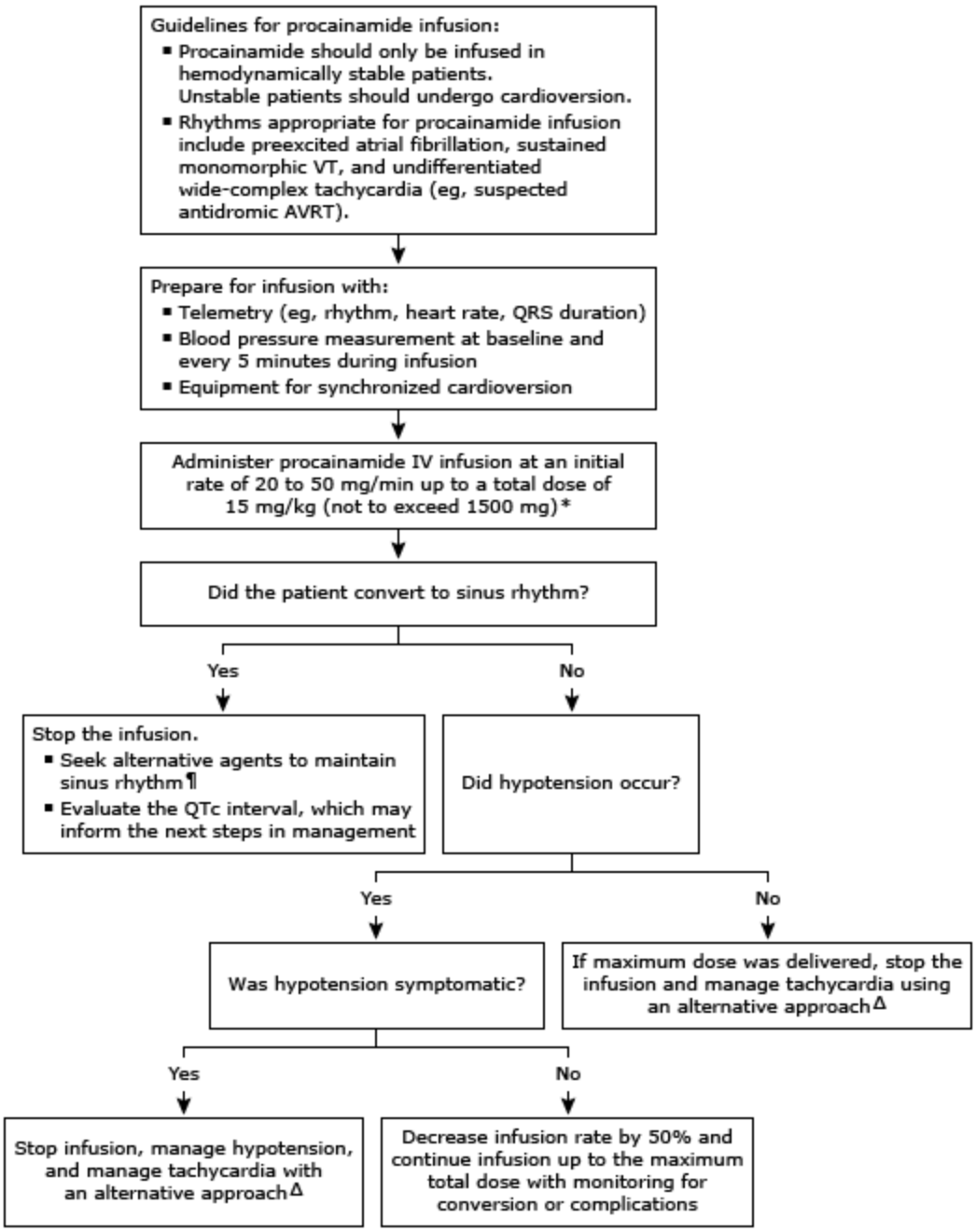
Class 0 (HCN channel blockers)
Ivabradine
Class I (voltage-gated Na+ channel blockers)
Class Ia (intermediate dissociation):
▪ Quinidine, ajmaline, disopyramide, procainamide
Class Ib (rapid dissociation):
▪ Lidocaine, mexilitine
Class Ic (slow dissociation):
▪ Propafenone, flecainide
Class Id (late 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).

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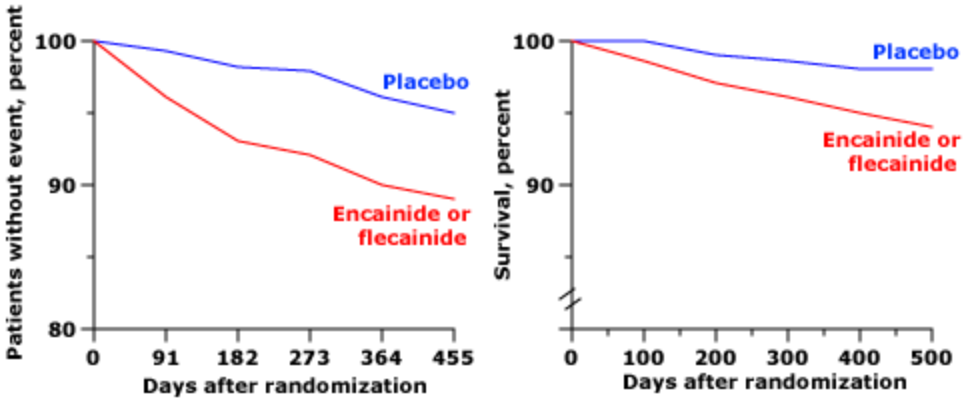
Graphic 141920 Version 4.0

Encainide and flecainide increase cardiac mortality

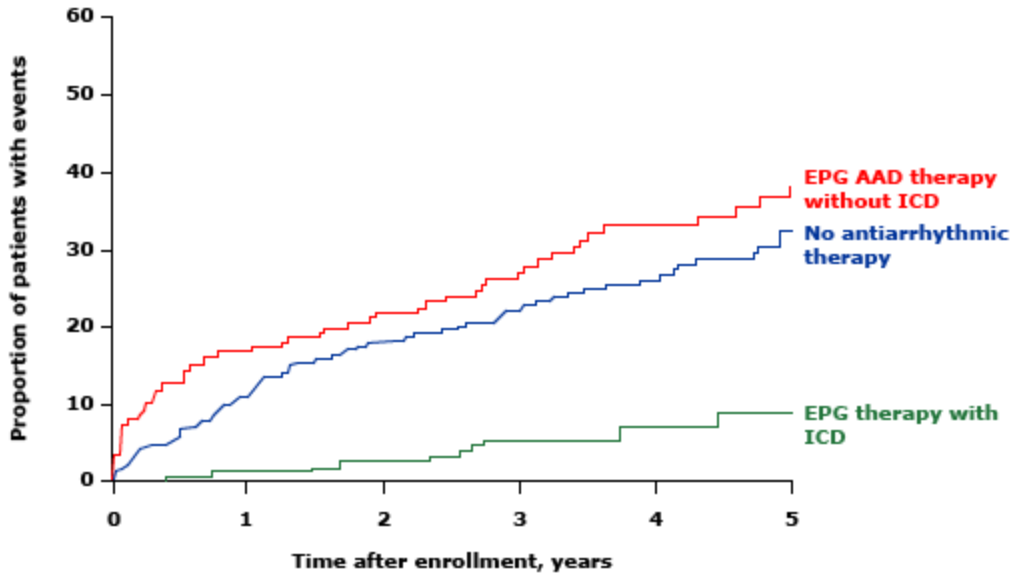


Results of the Cardiac Arrhythmia Suppression Trial (CAST) in patients with ventricular premature beats after myocardial infarction. Patients receiving encainide or flecainide had, when compared with those receiving placebo, a significantly lower rate of avoiding a cardiac event (death or resuscitated cardiac arrest) (left panel, p = 0.001) and a lower overall survival (right panel, p = 0.0006). The cause of death was arrhythmia or cardiac arrest.

Data from Echt DS, Liebson PR, Mitchell B, et al. N Engl J Med 1991; 324:781.

Graphic 59975 Version 5.0

ICD reduces sudden death in MUSTT



The MUSTT trial enrolled 704 patients with coronary artery disease, nonsustained ventricular tachycardia (VT), and a left ventricular ejection fraction ≤40 percent who had sustained VT induced during electrophysiologic (EP) study. Kaplan-Meier estimates show that the incidence of cardiac arrest or death from arrhythmia is significantly lower in those receiving an implantable cardioverter-defibrillator (ICD) compared with those receiving no therapy or those with EP-guided (EPG) antiarrhythmic drug (AAD) therapy.

Data from: Buxton AE, Lee KL, Fisher JD, et al. N Engl J Med 1999; 341:1882.

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