Sustained monomorphic ventricular tachycardia in patients with structural heart disease: Treatment and prognosis

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INTRODUCTION

Sustained monomorphic ventricular tachycardia (SMVT) is defined by the following characteristics:

- A regular wide QRS complex (≥120 milliseconds) tachycardia at a rate greater than 100 beats per minute
- The consecutive beats have a uniform and stable QRS morphology
- The arrhythmia lasts ≥30 seconds or causes hemodynamic collapse in <30 seconds

In patients with significant coronary heart disease (CHD) or other structural heart disease, a wide QRS complex tachycardia should be considered to be VT until proven otherwise [1]. (See "Wide QRS complex tachycardias: Approach to the diagnosis".)

This topic will focus on the treatment of SMVT in patients with structural heart disease. The diagnostic approach to SMVT and the treatment of SMVT during an acute myocardial infarction, as well as the management of nonsustained VT, are discussed separately. The approach to patients with idiopathic VT is also discussed separately. (See "Sustained monomorphic ventricular tachycardia: Clinical manifestations, diagnosis, and evaluation" and "Ventricular

arrhythmias during acute myocardial infarction: Incidence, mechanisms, and clinical features" and "Nonsustained ventricular tachycardia: Clinical manifestations, evaluation, and management" and "Ventricular tachycardia in the absence of apparent structural heart disease".)

TREATMENT

The initial management of a patient with SMVT depends on the hemodynamic stability of the patient (algorithm 1). Emergency management is required in unstable patients, while additional time may be spent determining the etiology and treating any underlying precipitating factors in patients who are hemodynamically stable (although treatment for such patients should usually be promptly administered). Following initial management and stabilization of the patient, subsequent management of the patient will be guided by the initial presentation (ie, hemodynamically stable or unstable) and the initial approach to treatment.

Initial management — All patients with SMVT should have a brief immediate assessment of the symptoms, vital signs, and level of consciousness to determine if they are hemodynamic stable or unstable (algorithm 1). While the assessment of hemodynamic status is being performed by a clinician, other members of the health care team should:

- Attach the patient to a continuous cardiac monitor
- Establish intravenous access
- Obtain a 12-lead electrocardiogram (ECG)
- Administer supplemental oxygen
- Send blood for appropriate initial studies (see "Wide QRS complex tachycardias: Approach to the diagnosis", section on 'Ancillary testing')

Differentiation between a hemodynamically stable versus unstable patient is as follows; hemodynamic stability does not necessarily imply SVT with aberrancy, and hemodynamic instability does not always imply VT (see "Wide QRS complex tachycardias: Approach to the diagnosis", section on 'Assessment of hemodynamic stability'):

- An unstable patient will have evidence of hemodynamic compromise (eg, hypotension, altered mental status, chest pain, or heart failure [HF]) and may remain awake with a discernible pulse, but may also be unresponsive and pulseless. Patients who become unresponsive or pulseless are considered to have a cardiac arrest and are treated according to standard resuscitation algorithms (algorithm 2). (See 'Unstable patients' below.)

- A stable patient shows no evidence of hemodynamic compromise despite a sustained rapid heart rate, but should have continuous monitoring and frequent reevaluations due to the potential for rapid deterioration as long as the SMVT persists. (See 'Stable patients' below.)

Patients with SMVT who are initially stable may rapidly become unstable, particularly in the setting of extremely rapid heart rates (greater than 200 beats per minute) or significant underlying cardiac comorbidities.

Unstable patients — Patients with SMVT who are felt to be hemodynamically unstable, severely symptomatic, or become pulseless require prompt treatment with electrical cardioversion/defibrillation (algorithm 1). Initial treatment with antiarrhythmic medications is not indicated for hemodynamically unstable or pulseless patients.

- Patients with SMVT who are hemodynamically unstable and pulseless, or who become pulseless during the course of evaluation and treatment, should be managed according to standard advance cardiac life support (ACLS) resuscitation algorithms, with immediate high-energy countershock and cardiopulmonary resuscitation (CPR) (algorithm 2). Patients should initially be treated with a synchronized shock at maximal energy (ie, 200 joule shock from a biphasic defibrillator or a 360 joule shock from a monophasic defibrillator) [2]. Subsequent shocks, if required, should be at the highest output available on the defibrillator. (See "Advanced cardiac life support (ACLS) in adults", section on 'Pulseless ventricular tachycardia and ventricular fibrillation' and "Overview of the acute management of tachyarrhythmias", section on 'Wide QRS complex tachyarrhythmias'.)

- For patients with SMVT who are **hemodynamically unstable but still responsive with a discernible blood pressure and pulse**, we recommend urgent cardioversion (following administration of sedation). Patients should initially be treated with a synchronized 200 joule shock with subsequent shocks that use escalating energy levels. (See "Cardioversion for specific arrhythmias", section on 'Ventricular tachycardia'.)

Subsequent antiarrhythmic therapy (intravenous amiodarone, procainamide, lidocaine, or oral agents such as sotalol or amiodarone) is generally indicated only if SMVT recurs. For repetitive episodes of SMVT, consideration should be given to catheter ablation. (See 'Stable patients' below and 'Radiofrequency catheter ablation' below.)

Stable patients — Patients with SMVT who are **hemodynamically stable on presentation** may remain stable or may become unstable rapidly and without warning. As such, therapy should be promptly provided to most patients. The choice of initial treatments for hemodynamically stable SMVT includes electrical or pharmacologic cardioversion

(algorithm 1). We generally prefer to begin with an intravenous antiarrhythmic agent and reserve electrical cardioversion for refractory patients or for those who become unstable. If SMVT is not terminated with the initial antiarrhythmic drug, external cardioversion may be performed or additional antiarrhythmic therapy may be administered (eg, second bolus of amiodarone, adding lidocaine to amiodarone, etc). Rarely, electrical cardioversion may be chosen as the initial therapy for patients with SMVT who are hemodynamically stable, but only if adequate procedural sedation can be administered prior to delivery of the shock.

Pharmacologic cardioversion — For pharmacologic cardioversion, we administer one of the following antiarrhythmic drugs:

- Intravenous amiodarone (150 mg IV over 10 minutes, followed by 1 mg/minute for the next six hours).
- Intravenous lidocaine (1 to 1.5 mg/kg [typically 75 to 100 mg] at a rate of 25 to 50 mg/minute; lower doses of 0.5 to 0.75 mg/kg can be repeated every 5 to 10 minutes as needed), which may be more effective in the setting of acute myocardial ischemia or infarction.
- Intravenous procainamide (20 to 50 mg/minute until arrhythmia terminates or a maximum dose of 15 to 17 mg/kg is administered) (algorithm 3).

There is no consensus on the choice of initial antiarrhythmic medication for patients with stable monomorphic VT. Some of our experts give lidocaine first, given its ease of rapid administration and because it does not cause hypotension. Other experts choose to give amiodarone or procainamide as the first antiarrhythmic drug for stable monomorphic VT, even though the time to administer is longer than lidocaine. Lidocaine does not usually cause hypotension, while amiodarone and procainamide often reduce the blood pressure, which may hasten the need for electrical cardioversion.

Intravenous pharmacologic therapy with amiodarone, procainamide, lidocaine, or sotalol (not available in all countries) may be attempted prior to electrical cardioversion [2-8]. For patients in whom VT terminates during drug infusion, the intravenous drug can usually be discontinued at that point, unless the patient has been experiencing recurrent episodes. For patients with frequent recurrent episodes, the infusion should be continued, and consideration should be given to initiating oral antiarrhythmic drug therapy or referring for catheter ablation. Regardless of the choice of antiarrhythmic drug, the patient should be reevaluated daily (or more frequently if unstable) to determine the optimal approach to antiarrhythmic therapy.

Intravenous amiodarone is slower in action than procainamide or lidocaine, but it improves the reversion rate of refractory SMVT and decreases its recurrence after reversion [3,6-8]. Procainamide has the advantage of slowing VT even when it fails to terminate, usually resulting in greater hemodynamic stability, whereas lidocaine usually does not slow SMVT. Procainamide terminates over 50 percent of episodes of SMVT, while lidocaine usually terminates only 10 to 20 percent.

Electrical cardioversion — If electrical cardioversion with appropriate procedural sedation is the chosen approach, intravenous analgesics or sedatives should be cautiously administered if the blood pressure will tolerate their use. If the QRS complex and T wave can be distinguished, an attempt at synchronized cardioversion can be performed with a synchronized shock of at least 100 joules using either a biphasic or monophasic defibrillator. If the QRS complex and T wave cannot be distinguished accurately, and a synchronized shock is not possible, we administer an unsynchronized 120 to 200 joule shock from a biphasic defibrillator or a 360 joule shock from a monophasic defibrillator. If the initial shock is unsuccessful, subsequent shocks should be delivered at escalating energy levels.

Initial dosing of antiarrhythmic drugs — When an antiarrhythmic drug is prescribed, the following regimens can be used:

- Lidocaine – Intravenous lidocaine can be given in an initial dose of 1 to 1.5 mg/kg (typically 75 to 100 mg at a rate of 25 to 50 mg/minute); lower doses of 0.5 to 0.75 mg/kg can be repeated every 5 to 10 minutes as needed. If VT terminates, then we usually do not begin a continuous infusion. If VT recurs (ie, becomes incessant), a continuous intravenous infusion of 1 to 4 mg/minute may be begun. The maximum total dose is 3 mg/kg (300 mg) over one hour. It is rarely necessary to continue the infusion for more than 24 hours, and the incidence of neurotoxicity increases greatly after 24 hours of infusion.

- Procainamide – Intravenous procainamide can be administered in a number of ways. The standard method is an infusion of 20 to 50 mg/minute while monitoring the blood pressure closely every 5 to 10 minutes until the arrhythmia terminates or hypotension ensues, or a total of 15 to 17 mg/kg has been given (algorithm 3). Once VT terminates, it is usually not necessary to continue a maintenance infusion, although procainamide can be resumed or continued if VT recurs.

- Amiodarone – Intravenous amiodarone is administered with a 150 mg bolus over 10 minutes, followed by a continuous intravenous infusion of 1 mg/minute for six hours and 0.5 mg/minute, generally for an additional 18 hours or longer. Repeated boluses can be given over 10 minutes every 10 to 15 minutes to a maximum total dose of 2.2 g in 24

hours. The blood pressure must be carefully monitored because the diluent can cause hypotension when the administration of amiodarone occurs too rapidly. (See "Amiodarone: Clinical uses" and "Amiodarone: Clinical uses", section on 'Side effects with IV administration'.)

Commonly, oral amiodarone in doses up to 400 mg orally every eight hours is initiated overlapping with intravenous amiodarone for 24 to 48 hours. The high-dose oral amiodarone loading can be continued up to 7 to 10 days before decreasing to maintenance dosing of 200 mg daily. The duration of intravenous and oral amiodarone loading is dependent on the clinical response and the tolerance of the drug.

- Sotalol – In countries in which intravenous sotalol is available, the dose is 1 to 1.5 mg/kg (or 100 mg) at a rate of 10 to 20 mg/minute, watching for bradycardia, hypotension, or the development of other arrhythmias that can be caused by the proarrhythmic effect of sotalol, such as torsades de pointes. This dose may be repeated after six hours if necessary. (See "Clinical uses of sotalol".)

Treatment of associated conditions — Treatment of underlying conditions associated with VT, such as myocardial ischemia, electrolyte disturbances, drug proarrhythmia, and HF, as well as decreasing the sympathetic facilitation of SMVT, are important components of the acute management of VT. However, most episodes of SMVT do not have identifiable precipitating factors, and SMVT is rarely precipitated by acute myocardial ischemia or electrolyte imbalance, although it most commonly occurs in patients with prior myocardial infarction (MI). The presence of hypokalemia or hypomagnesemia should prompt correction of these electrolyte disturbances, but electrolyte abnormalities alone should not be accepted as the cause for SMVT. Some of the standard pharmacologic therapies for cardiomyopathy/HF have been shown to improve survival, in some cases by reducing the incidence of SCD, but have not necessarily been proven to prevent recurrent VT in patients previously manifesting ventricular arrhythmias. (See "Overview of the management of heart failure with reduced ejection fraction in adults".)

- Adjunctive use of beta blockers, in combination with antiarrhythmic agents other than sotalol, is an effective approach for reducing sympathetic facilitation of SMVT, especially in patients with frequent recurrences of VT within a short time period (eg, VT "storm") [9,10]. (See "Electrical storm and incessant ventricular tachycardia", section on 'Initial management'.)

- Electrolyte disturbances, particularly hypokalemia and hypomagnesemia, should be promptly treated when present. (See "Clinical manifestations and treatment of hypokalemia in adults" and "Hypomagnesemia: Evaluation and treatment".)

- Drug-induced proarrhythmia most commonly results in polymorphic VT in association with marked QT prolongation (QTc ≥500 milliseconds) and rarely results in SMVT, but should be considered in patients taking potentially proarrhythmic medications. A less common manifestation of drug-related proarrhythmia is incessant slower (usually <150 beats per minute) monomorphic VT. This can occur when high doses of amiodarone or any other drug capable of slowing intraventricular conduction is given. (See "Acquired long QT syndrome: Definitions, pathophysiology, and causes".)

SMVT in patients with ICDs — If a patient with an ICD presents with SMVT, one of several possibilities has occurred:

- The device is malfunctioning and is not effectively detecting VT.
- The device has been ineffective in terminating VT, and all programmed therapies have been exhausted.
- The device has effectively terminated the VT, but SMVT has recurred repeatedly and therapies have been exhausted.
- The rate of the VT is below the programmed VT detection rate of the device.

For any of these possibilities, the initial response is the same as in patients without implanted ICDs. If the patient is hemodynamically stable and a programmer is immediately available, the implanted device can be used to attempt termination of SMVT by antitachycardia pacing. An external defibrillator should be immediately available, because there is a possibility that SMVT can be accelerated to VF.

Recurrent or refractory SMVT — If SMVT recurs following initial therapies, including in patients who already have an implantable cardioverter-defibrillator (ICD), suppression of the arrhythmia by pharmacologic means should be attempted, and further evaluation should focus upon the presence of arrhythmia triggers (eg, ischemia, electrolyte abnormalities, and drug toxicity). Patients who have multiple episodes of SMVT within a relatively short period of time (generally three or more episodes of SMVT within 24 hours or SMVT recurring soon after [ie, within five minutes] termination of another SMVT episode) are considered to have electrical (VT) storm.

- Patients with hemodynamically unstable VT storm should initially undergo electrical cardioversion/defibrillation according to advanced cardiac life support protocol, while antiarrhythmic drug therapy is generally indicated for treatment of VT storm in patients who are hemodynamically stable.

- Among antiarrhythmic medications, amiodarone is the most effective for preventing recurrent SMVT, although sotalol and dofetilide are also efficacious for reducing recurrent SMVT [2]. We prefer empiric therapy with amiodarone for patients with recurrent SMVT as well as for those who have refused (or are not candidates for) ablation or ICD placement [11]. Following stabilization of the patient, if there are concerns about potential toxicity related to amiodarone, particularly for anticipated long-term use, sotalol or dofetilide may be considered. (See "Amiodarone: Adverse effects, potential toxicities, and approach to monitoring".)

- If active myocardial ischemia is felt to be a contributing factor, urgent coronary revascularization should be pursued. However, it should not be expected that myocardial revascularization will "cure" SMVT.

- Ibutilide has been reported as an effective option in patients with VT/VF refractory to both amiodarone and lidocaine in the setting of incomplete revascularization requiring mechanical support [12]. (See "Electrical storm and incessant ventricular tachycardia".)

A full discussion of the management of VT storm is presented separately. (See "Electrical storm and incessant ventricular tachycardia".)

Chronic therapy — Chronic therapy of patients with SMVT usually requires utilization of multiple therapeutic modalities, including the ICD, antiarrhythmic drugs, catheter ablation, and/or arrhythmia surgery. In the absence of a clearly identifiable and reversible cause for SMVT occurring in the setting of cardiomyopathy (ischemic or nonischemic), nearly all patients with a history of SMVT will be candidates for ICD insertion to treat recurrent VT, as well as to reduce the risk of sudden cardiac death (SCD), unless the patient refuses or the risks of ICD insertion are felt to outweigh the potential benefits. Patients with multiple recurrent episodes of SMVT resulting in painful ICD shocks should be evaluated for antiarrhythmic drugs or an ablation strategy (either via catheter-based or surgical approaches) to reduce or eliminate the likelihood of additional shocks.

Beta blockers — Nearly all patients who experience SMVT have an indication for therapy with a beta blocker, including patients with a prior MI, patients with HF and reduced LV systolic function, etc. Beta blockers provide some level of protection against recurrent SMVT, probably by blocking sympathetic input to the heart. The role of beta blocker therapy in patients with HF and/or a prior MI is discussed in detail separately. (See "Acute myocardial infarction: Role of beta blocker therapy", section on 'Long-term therapy' and "Primary pharmacologic therapy for heart failure with reduced ejection fraction", section on 'Beta blocker'.)

ICD therapy — Patients who survive an episode of SMVT in the setting of structural heart disease (post-MI or nonischemic cardiomyopathy) are typically candidates for implantation of an ICD to treat recurrent VT as well as to reduce the risk of SCD. Although some patients are treated with other therapies, such as antiarrhythmic drugs, radiofrequency ablation (RFA), or surgery, an ICD is the most common initial treatment for SMVT, although survival benefit and superiority of the ICD over other therapies have not been established for any particular therapy since these patients were excluded from ICD trials. These other therapies are used either as an adjunct to an ICD or as an alternative in patients who are not candidates for or who refuse ICD therapy.

Three large, prospective randomized clinical trials (AVID, CASH, and CIDS) and several subsequent meta-analyses have evaluated ICD therapy compared with amiodarone or other antiarrhythmic drugs in survivors of cardiac arrest or life-threatening ventricular tachyarrhythmias, including hemodynamically unstable SMVT [13-18]. Although the AVID trial was the only one to demonstrate a statistically significant survival benefit, the meta-analyses have all shown a significant improvement in overall mortality with ICD therapy. (See "Secondary prevention of sudden cardiac death in heart failure and cardiomyopathy".)

There remains some debate regarding the optimal management of sustained, hemodynamically stable VT in persons with a prior MI but normal or near normal left ventricular (LV) systolic function, in whom there is increasing consensus that VT ablation should be considered for primary or initial therapy [19].

Antiarrhythmic drugs — No antiarrhythmic medication has been demonstrated to reduce the mortality of patients with SMVT, except beta-adrenergic blocking agents. With that in mind, the use of antiarrhythmic drugs in patients with SMVT is typically limited to two settings:

- As an adjunct to an ICD in patients with frequent arrhythmia recurrences and ICD shocks. In a 2016 systematic review that included 2268 patients from eight trials, patients taking an antiarrhythmic drug had significantly lower likelihood of appropriate ICD interventions (odds ratio [OR] 0.66, 95% CI 0.44-0.97) [20].

- As primary therapy or as adjunctive therapy to catheter ablation in patients who do not want or are not candidates for an ICD.

Antiarrhythmic drugs may also be used to improve quality of life in patients with frequent SMVT leading to ICD shocks, or in those patients who are not candidates for or who decline ICD implantation. In the presence of HF and/or structural heart disease, antiarrhythmic drug therapy is limited to a small number of choices (primarily amiodarone, sotalol, and mexiletine) [1,21]. Mexiletine is rarely helpful unless combined with other antiarrhythmic agents, usually

amiodarone. Several clinical trials and systematic reviews have evaluated the efficacy of antiarrhythmic drugs as adjuvant therapy in ICD patients [22-27]. There were significant differences in trial methodologies, which limit direct comparisons. Amiodarone has generally been the most effective antiarrhythmic drug for preventing ventricular arrhythmias (and associated ICD shocks). This is based upon a greater efficacy and a lower risk of proarrhythmia compared with other antiarrhythmic drugs [28-33]. (See "Secondary prevention of sudden cardiac death in heart failure and cardiomyopathy", section on 'Antiarrhythmic drugs' and "Pharmacologic therapy in survivors of sudden cardiac arrest", section on 'Antiarrhythmic drugs'.)

The long-term efficacy of other class III drugs, such as dofetilide and dronedarone (neither of which is approved in the United States or many other countries for the treatment of VT), has not been adequately evaluated, although a comparative study in 135 patients found that dofetilide was as effective as sotalol for preventing the induction of SMVT with electrophysiologic testing (36 versus 34 percent) and was better tolerated and more effective during long-term therapy [34,35]. Combination therapy using different classes of antiarrhythmic drugs is rarely used [36]. (See "Clinical use of dofetilide", section on 'Ventricular tachyarrhythmias'.)

The class IC drugs are contraindicated in patients with coronary artery disease, while the class IA drugs are used rarely because of concerns about proarrhythmia. (See "Ventricular arrhythmias during acute myocardial infarction: Prevention and treatment".)

Dosing — The dosing of amiodarone and sotalol for chronic maintenance therapy in patients with recurrent SMVT is as follows:

Amiodarone — The initial dosing of amiodarone will vary depending on the route (intravenous [IV] or oral) as well as the clinical situation (table 1). Most patients are started on IV amiodarone and transitioned to oral dosing once clinically stable.

- For patients who have been on IV therapy for one week or less, or were never given IV amiodarone, we usually start with a full oral amiodarone loading dose of 400 to 1200 mg/day (typically in two or three divided doses and usually with meals to minimize associated GI side effects). This should be continued until a total loading dose of up to 10 grams has been received, and then the dose should be reduced to the usual maintenance dose of 200 mg/day after three to four days.

- For patients who have been on IV therapy for one to two weeks, we start an intermediate maintenance oral amiodarone dose of 400 to 800 mg/day. This should be continued until a total loading dose of 10 grams has been received, and then the dose should be reduced to

the usual maintenance dose of 200 mg/day. As oral amiodarone is only approximately 50 percent bioavailable, a total of 20 to 30 grams needs to be administered.

- For patients who have been on IV therapy for more than two weeks, we start maintenance oral amiodarone at a dose of 200 mg/day. (See "Amiodarone: Clinical uses", section on 'Amiodarone for ventricular arrhythmias'.)

- Patients receiving amiodarone chronically for SMVT (and other indications) should have baseline evaluations and regular follow-up of lung, liver, thyroid, skin and eyes. While historically 400 mg per day was considered an appropriate dose for VT and 200 mg per day an appropriate dose for atrial fibrillation, we strive to find the lowest effective dose to reduce the risk of adverse effects and toxicity. In contemporary practice, amiodarone is rarely used at a dose of 400 mg/day. Patients stable on 400 mg/day initially are generally reduced to 200 mg/day. If a patient is stable on 200 mg daily, we generally propose 200 mg alternating with 100 mg daily, and then eventually 100 mg daily. The risk of recurrent VT on a lower dose, with the possibility of an ICD shock, must be balanced against the desire to reduce the risk of adverse effects in follow-up, which often require discontinuation of the drug. (See "Amiodarone: Adverse effects, potential toxicities, and approach to monitoring".)

Sotalol — In contrast to amiodarone, sotalol is not universally available in intravenous form. Bradycardic and proarrhythmic events can occur after the initiation of sotalol therapy and with each upward dosing adjustment. As a result, sotalol should be initiated and doses increased in a hospital with facilities for cardiac rhythm monitoring and assessment.

- We start sotalol at a dose of 80 mg twice daily, with dose adjustments at three-day intervals once steady-state plasma concentrations have been achieved and the QT interval has been reviewed on a surface ECG. Patients with renal insufficiency require a modification of the dosing interval. (See "Clinical uses of sotalol", section on 'Dosing'.)

ICD management with chronic antiarrhythmic drug therapy — In ICD patients treated with oral antiarrhythmic drugs, several issues require consideration. If SMVT recurs in the setting of antiarrhythmic drug therapy, the rate of the tachycardia is generally slower. Detection parameters should therefore be modified. Antiarrhythmic drug therapy may also render SMVT more susceptible to pace termination; increasing the number of ATP sequences may therefore be reasonable. Finally, some antiarrhythmic drugs, particularly amiodarone, can increase the defibrillation energy requirement. It may therefore be reasonable in some patients to verify the defibrillation safety margin after amiodarone is added.

Radiofrequency catheter ablation — For patients with recurrent SMVT resulting in ICD shocks despite treatment with an antiarrhythmic drug, we suggest catheter-based RFA rather than the addition of a second antiarrhythmic agent. RFA is also an alternative to antiarrhythmic drugs as the initial therapy for SMVT [21,37,38]. In addition, RFA, with or without antiarrhythmic drug therapy, is an option for patients with SMVT who are not candidates for or who refuse ICD implantation.

SMVT in patients with a prior MI is usually due to reentry in a circuit created by the heterogeneous electrical properties of residual myocardium in the region of the scar from the infarct. However, other mechanisms such as abnormal automaticity or triggered activity may occasionally occur. RFA can alter or eliminate such circuits or foci and prevent VT recurrence in selected patients. (See "Reentry and the development of cardiac arrhythmias".)

Our suggested approach to utilizing RFA in the treatment of ventricular arrhythmias is generally consistent with the recommendations of professional society guidelines [1,19,37]:

- For patients with structural heart disease (such as prior MI, dilated cardiomyopathy, or arrhythmogenic right ventricular cardiomyopathy), catheter ablation **is recommended** for any of the following conditions:
  - Symptomatic SMVT, including VT terminated by an ICD, that recurs despite antiarrhythmic drug therapy or when antiarrhythmic drugs are not tolerated or not desired
  - Incessant SMVT or VT storm not due to a transient reversible cause
  - Recurrent SMVT and ventricular fibrillation (VF) that is refractory to antiarrhythmic therapy when there is a suspected trigger that can be targeted for ablation
- For patients with structural heart disease, catheter ablation **should be considered** for patients with any of the following:
  - One or more episodes of SMVT despite therapy with one or more class I or III antiarrhythmic drugs
  - Recurrent SMVT due to prior MI and expectation for at least one year of survival as an acceptable alternative to amiodarone therapy
  - Hemodynamically tolerated SMVT due to prior MI who have reasonably preserved left ventricular ejection fraction (LVEF >35 percent) even if they have not failed antiarrhythmic drug therapy

Catheter ablation techniques — A variety of VT ablation techniques have evolved over the years, including ablation focused on abolition of inducible VT based upon the results of activation and entrainment mapping (waveform 1) versus more extensive ablation of myocardium displaying abnormal electrogram characteristics ("substrate-based ablation"). The technical aspects of catheter ablation for VT are presented separately. (See "Overview of catheter ablation of cardiac arrhythmias".)

While no technique has proven to be universally effective or clearly superior, smaller nonrandomized studies have suggested lower rates of VT recurrence following more extensive ablation [39-43]. Subsequently, in a multicenter trial of 118 patients with hemodynamically tolerated VT and ischemic cardiomyopathy, in which patients were randomized to clinical ablation of inducible VT (60 patients) or substrate-based ablation targeting all "abnormal" electrograms seen on mapping (58 patients) and followed for 12 months, patients in the substrate-based ablation group had significantly fewer VT recurrences (16 versus 48 percent), less need for antiarrhythmic drugs (12 versus 58 percent), and fewer hospitalizations (12 versus 32 percent) compared with patients in the clinical VT ablation group [44]. There was no difference in periprocedural complications between the two approaches; however, 22 patients in the substrate-based ablation group also underwent EP study and induction of VT, raising some concerns that this may have improved the efficacy of the ablation procedure in the substrate-based group. While these data are encouraging, additional data in larger numbers of patients and additional populations are required prior to recommending widespread implementation of the substrate-based ablation technique, even in patients with hemodynamically-tolerated VT. Additionally, a systematic review and meta-analysis of non-randomized trials suggests significantly lower rates of recurrent VT following a combined endocardial/epicardial ablation compared with endocardial ablation alone, in particular among patients with ischemic cardiomyopathy [45]. (See 'Long-term efficacy' below.)

Ablation performed via an epicardial approach is another option for select patients with ventricular tachyarrhythmias. Among a cohort of 444 consecutive patients with VT and prior MI who were referred for catheter ablation, 27 patients (6 percent) had successful epicardial ablation of at least one VT after endocardial ablation failed [46]. Some advocate epicardial in addition to endocardial ablation when performing substrate-based ablation.

Cardiac magnetic resonance (CMR) imaging with contrast enhancement is also being investigated as a means of identifying critical sites for reentrant ventricular tachyarrhythmias [47]. In one study of 20 patients (mixed nonischemic and ischemic cardiomyopathy) with prior failed endocardial catheter ablation, CMR with late gadolinium enhancement (LGE) imaging was performed, and subsequently ablation strategies were modified based on LGE findings (nine

patients underwent epicardial ablation, while 11 underwent repeat endocardial ablation) [48]. At mean follow-up of 17 months, 18 of 22 patients remained free of recurrent VT.

Acute procedural success — Depending upon a number of factors, acute success rates (defined as lack of inducible VT or termination of incessant VT) for RFA of VT vary from 70 to 90 percent, with a procedure-related mortality of 0.5 percent [19].

Catheter ablation of VT has been applied most often in patients with frequent episodes. Two-thirds of these patients have experienced at least a 75 percent reduction in frequency of VT [19].

Long-term efficacy — Catheter ablation has been evaluated as a therapy to prevent recurrent SMVT with good results in several individual trials [19,49-54].

Depending on the clinical presentation of VT (cardiac arrest versus relative hemodynamic stability), ICDs are usually considered in conjunction with ablation, except in patients with fairly normal LV function who present with stable VT.

In the VANISH trial, VT ablation was shown to lower VT storm and appropriate ICD shocks. In this a multicenter, non-blinded study, 259 patients with prior MI and a previously implanted ICD who had at least one episode of VT within the preceding six months while on antiarrhythmic drug therapy were randomly assigned to catheter ablation (132 patients) or escalated antiarrhythmic therapy. Antiarrhythmic therapy was defined as initiating or increasing the dose of amiodarone if the prior dose was less than 300 mg daily, or addition of mexiletine to amiodarone if the prior dose was 300 mg daily or greater (127 patients) [52]. Over a mean follow-up of 28 months, patients in the ablation group had a significantly lower rate of the composite primary outcome of death, VT storm, or appropriate ICD shock (59 versus 69 percent; hazard ratio [HR] 0.72, 95% CI 0.53-0.98). The difference in outcomes was driven by reductions in VT storm and ICD shocks in the ablation group, as there was no significant difference in total mortality between the two groups.

There is continuing concern over evidence that ICD shocks, both "appropriate" and "inappropriate," have adverse effects on survival [55]. As a result, earlier use of VT ablation has been proposed as a way to potentially reduce shocks and thereby improve survival. Two trials have suggested that early VT ablation among patients with cardiomyopathy may reduce cardiovascular events, including VT recurrence and ICD shocks [56,57]. One of these trials evaluated VT ablation at the time of ICD placement [56] and the other evaluated VT ablation shortly after a first ICD shock [57].

- **PAUSE-SCD** – Among patients with cardiomyopathy (ischemic, nonischemic, or arrythmia-related), early catheter ablation performed at the time of ICD implantation significantly

reduced the composite primary outcome of VT recurrence, cardiovascular hospitalization, or death [56]. This trial included 121 patients with cardiomyopathy, SMVT, and an indication for ICD implantation. Participants were randomly assigned to either early VT ablation plus ICD, or medical therapy plus ICD, and were followed for a composite of VT recurrence, cardiovascular hospitalization, or death. Participants assigned to early VT ablation were observed to have fewer primary adverse events compared with the control group (49.3 versus 65.5 percent, HR 0.58 95% CI 0.35-0.96). The observed difference was driven by a reduction in VT recurrence (31.7 versus 50.8 percent), ICD shocks (10 versus 24.6 percent), and antitachycardia pacing (16.2 versus 32.8 percent) in the VT ablation compared with control group. However, no differences in cardiovascular hospitalization (32 versus. 33.7) or mortality (8.9 versus 8.8 percent) were observed. Complications occurred in 8.3 percent of patients.

- **PARTITA** – This small trial provides support for performing VT ablation in patients with either ischemic or nonischemic cardiomyopathy after their first ICD shock [57]. The study randomly assigned 47 such patients to either a VT ablation prior to ICD or standard therapy and followed them for the development of death or heart failure (HF) hospitalization. Over a median follow-up of 2.4 years, patients assigned to ablation were less likely to experience death or HF hospitalization (4 versus 42 percent, HR 0.11, 95% CI 0.01-0.85) compared with the control group. The trial was stopped early because it showed early benefit. The ablation group was observed to have fewer deaths (0 versus 33 percent), fewer ICD shocks (9 versus 42 percent), and statistically similar but numerically less HF hospitalizations (4 versus 17 percent).

These findings were consistent with two earlier trials of ablation after an initial episode of VT (SMASH-VT and VTACH) [49,53] but differed from the BERLIN VT trial, which did not find benefit for an early VT ablation strategy among patients with ischemic cardiomyopathy [38]. In this study, patients with ischemic cardiomyopathy (left ventricular ejection fraction from 30 to 50 percent) and documented VT were randomly assigned ICD and preventive ablation (76 patients) at the time of enrollment or deferred ablation (83 patients) after three or more appropriate ICD shocks [38]. After a mean follow-up of 13 months, there was no significant difference in the composite primary outcome of death or unplanned hospitalization for VT or HF (33 percent in the early ablation group versus 28 percent in the deferred ablation group), at which point the study was also terminated early for futility. Fewer patients in the early ablation group received appropriate ICD therapy (34 versus 47 percent) or experienced sustained VT (40 versus 48 percent).

BERLIN-VT may have had null findings because this study included only patients with ischemic cardiomyopathy (whereas PAUSE-SCD [56]and PARTITA [57] included patients with nonischemic cardiomyopathy). However, because PARTITA was stopped early for benefit, the observed HR may overestimate the efficacy.

Consistent with the findings of most individual trials, earlier systematic reviews and meta-analyses have shown that, compared with medical therapy alone, VT ablation generally does not improve mortality but leads to significantly fewer episodes of recurrent VT and VT storm, as well as lower likelihood of an appropriate ICD intervention [20,45,58,59]. As an example, in a 2020 meta-analysis of five randomized trials (635 patients) comparing ablation with medical therapy alone, patients treated with ablation had the following outcomes:

- Lower likelihood of appropriate ICD therapies (odds ratio [OR] 0.49; 95% CI 0.28-0.87) and appropriate ICD shocks (OR 0.52; 95% CI 0.28-0.96)
- Lower likelihood of future VT storm (OR 0.64; 95% CI 0.43-0.95)
- Lower likelihood of cardiac hospitalization (OR 0.67; 95% CI 0.46-0.97)
- No significant improvement in all-cause mortality (OR 0.89; 95% CI 0.60-1.34)

Complications — The potential risks and benefits of the ablation procedure include those risks associated with any invasive electrophysiology (EP) study alone (table 2), although the overall incidence of complications is higher with ablation, which is a lengthier procedure typically performed in higher-risk patients. In a nationwide sample of 4653 patients who underwent ablation in the United States between 2002 and 2011 for post-MI VT, the overall in-hospital complication rate was 11 percent with in-hospital mortality of 1.6 percent [60]. Generally, risks are lower in experienced operators and centers that perform larger numbers of procedures. These risks are discussed in greater detail separately. (See "Invasive diagnostic cardiac electrophysiology studies", section on 'Complications of invasive cardiac electrophysiology studies'.)

As experience with catheter-based RFA accumulates among patients with VT, rates of early mortality and procedural complications appear to be declining. In a registry of 2061 patients with structural heart disease (mean age 62 years, mean LVEF 34 percent; 53 percent ischemic cardiomyopathy) who underwent RFA for VT (including 35 percent with VT storm), procedure-related complications occurred in 127 patients (7 percent), although in-hospital procedure-related mortality occurred in only 12 patients (0.6 percent) [61]. Early all-cause mortality at 31 days occurred in 100 patients (5 percent), with a greater likelihood of early mortality associated with lower LVEF, worse renal function, and pre-ablation VT storm. Among patients for whom data were available on mode of death, the cause was more frequently deemed advancing HF rather than to worsening arrhythmias (39 versus 22 percent). In a different study of 1251

patients who underwent RFA for VT between 2002 and 2013 and were enrolled in the international VT registry, LVEF <30 percent, previous ablation attempt, and presentation with electrical storm were the factors most associated with higher mortality [62].

Patients undergoing catheter ablation are also at risk of hemodynamic decompensation during or after the procedure. Among a single-center cohort of 193 consecutive patients undergoing catheter ablation for scar-related VT, acute hemodynamic decompensation (defined as persistent hypotension in spite of vasopressors requiring mechanical support or discontinuation of the procedure) occurred in 22 patients (11 percent) [63]. Patients with acute hemodynamic decompensation were more likely to be older, have diabetes or chronic obstructive pulmonary disease, have more severe HF, and more often received general anesthesia. In a separate multicenter cohort of 1365 patients (mean age 64 years, mean LVEF 30 percent) with HF undergoing VT ablation, including 111 patients with NYHA class 4 symptoms, there was no significant difference in the rate of acute complications between patients with NYHA class 2/3 symptoms (7 percent) and those with NYHA class 4 symptoms (10 percent), suggesting that VT ablation is safe for patients with the most severe HF symptoms [64]. In select cases, percutaneous hemodynamic support can be used during catheter ablation for patients at high risk of complications [65].

Other therapies — Not all patients will have their VT controlled with antiarrhythmic medications or RFA. In such cases, surgical therapy for VT remains effective in some patients, while energy sources other than radiofrequency are being investigated.

Surgical therapy — For most patients, because of the proven efficacy of catheter-based RFA and the development of ICDs, surgical treatment of ventricular arrhythmias is infrequently performed. However, surgical treatment of ventricular arrhythmias may be considered in certain subsets of patients with VT/VF:

- Patients with VT/VF that is refractory to antiarrhythmic therapy, resulting in frequent ICD shocks
- Patients with VT/VF that is refractory to antiarrhythmic therapy who have failed prior ablation of VT/VF
- Patients who are unable to tolerate antiarrhythmic therapy
- Patients requiring surgery for myocardial revascularization
- Patients with refractory HF and LV aneurysms that may benefit from aneurysm resection and LV reconstruction

Although surgical therapies for SMVT are rarely performed today, they do remain a viable therapeutic option for selected patients with SMVT. Arrhythmia surgery is particularly useful

when surgical revascularization (coronary artery bypass grafting [CABG]) is also planned, since it offers added hemodynamic benefits, including improvement in LVEF and alleviation of symptoms of HF, especially those requiring CABG and/or LV aneurysmectomy.

Surgical ablation using endocardial resection was the mainstay of therapy for recurrent SMVT following MI unresponsive to pharmacologic therapy until the early 1990s, when RF catheter ablation for VT became feasible. A variety of surgical approaches with inconsistent efficacy have been performed, including LV aneurysmectomy, bilateral sympathectomy, encircling endocardial ventriculotomy, and subendocardial resection. Using LV aneurysmectomy as an example, the association of VT with LV aneurysms has been noted since the early 1900s, but aneurysmectomy alone has shown to have limited success in curing VT, presumably due to the fact that VTs may arise from the border of the aneurysm which is rarely excised during conventional aneurysmectomy [66]. By contrast, long-term control of VT/VF has been excellent when LV aneurysmectomy is combined with LV reconstruction and subendocardial resection guided by VT mapping, with or without cryoablation [67,68]. Cardiac sympathetic denervation (ie, sympathectomy) can be effective as a temporizing strategy in patients with refractory VT and prior failed catheter ablation [69,70].

Stereotactic radiation therapy — Stereotactic radiation therapy, in which high doses of radiation are precisely targeted, has been effective and well-tolerated as part of the treatment of some malignancies and has been investigated for VT in some preclinical studies and case reports [71-74].

- In a single-center case series of five patients with structural heart disease (three with nonischemic cardiomyopathy, two with ischemic cardiomyopathy) with failed prior radiofrequency ablation (or who were not candidates for ablation) and ongoing VT in spite of two or more antiarrhythmic drugs, episodes of VT decreased following stereotactic radiation therapy [71]. The procedure was generally well-tolerated, with no apparent deterioration of systolic function, HF exacerbations, or other toxicities.

- Subsequently, in a prospective nonrandomized single arm study of 19 patients (17 for VT, two for premature ventricular complex/contraction [PVC; also referred to a premature ventricular beats or premature ventricular depolarizations]) who underwent stereotactic radiation therapy, median VT (or PVC) burden was markedly reduced, and the procedure was generally safe and well-tolerated (two patients developed radiation pneumonitis, six developed pericardial effusions [five of which were asymptomatic]) [72].

- In another series of 10 patients who underwent stereotactic radiation therapy after failed catheter ablation and were followed for a median of 28 months, 8 of 10 patients

experienced recurrent VT during follow-up, although the overall VT burden was reduced by 88 percent [73]. Of note, two patients in this series experienced an increased frequency of VT after radiation therapy.

Prior to offering this therapy to patients outside of a clinical trial, stereotactic radiation therapy requires additional efficacy and safety evaluation in larger studies.

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" and "Society guideline links: Cardiac implantable electronic devices" and "Society guideline links: Catheter ablation of 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

- Sustained monomorphic ventricular tachycardia (SMVT) is defined as a regular, wide (≥120 milliseconds) QRS complex tachycardia with uniform and stable QRS morphology at a rate of more than 100 beats per minute that lasts for 30 seconds or longer or causes hemodynamic collapse within 30 seconds. (See 'Introduction' above.)

- All patients with SMVT should have a brief immediate assessment of the symptoms, vital signs, and level of consciousness to determine if they are hemodynamic stable or unstable. Differentiation between a hemodynamically unstable versus stable patient depends upon hemodynamic compromise, such as hypotension, altered mental status, chest pain, or heart failure (HF). (See 'Initial management' above.)

- Patients with SMVT who are **hemodynamically unstable and pulseless**, or who become pulseless during the course of evaluation and treatment, should be managed according to standard advance cardiac life support (ACLS) resuscitation algorithms, with immediate high-energy countershock and cardiopulmonary resuscitation (CPR) (algorithm 2). Patients should initially be treated with a synchronized 120 to 200 joule shock from a biphasic defibrillator or a 360 joule shock from a monophasic defibrillator. (See 'Unstable patients' above.)

- For patients with wide complex tachycardia (WCT) who are hemodynamically unstable, but still responsive with a discernible blood pressure and pulse, we recommend urgent cardioversion (following administration of sedation) (**Grade 1B**). (See 'Unstable patients' above.)

- For patients with SMVT who are **hemodynamically stable on presentation**, after recording a 12-lead ECG we generally prefer to begin with an intravenous antiarrhythmic agent and reserve electrical cardioversion for refractory patients or for those who become unstable (algorithm 1). (See 'Stable patients' above.)

  - If pharmacologic cardioversion is the chosen approach, we administer intravenous amiodarone, procainamide (algorithm 3), or lidocaine.

  - If electrical cardioversion with appropriate procedural sedation is the chosen approach, intravenous analgesics or sedatives should be cautiously administered if the blood pressure will tolerate their use. If the QRS complex and T wave can be distinguished, an attempt at synchronized cardioversion can be performed with a synchronized shock of 100 joules using either a biphasic or monophasic defibrillator.

- Treatment of underlying conditions associated with VT, such as myocardial ischemia, electrolyte disturbances, drug proarrhythmia, and HF, as well as decreasing the sympathetic facilitation of SMVT, are important components of the acute management of VT. (See 'Treatment of associated conditions' above.)

- Chronic therapy of patients with SMVT usually requires utilization of multiple therapeutic modalities, including the implantable cardioverter-defibrillator (ICD), antiarrhythmic

drugs, radiofrequency catheter ablation, and/or arrhythmia surgery.

- In the absence of a clearly identifiable and reversible cause for SMVT, nearly all patients with a history of SMVT will be candidates for ICD insertion for secondary prevention of sudden cardiac death, unless the patient refuses or the risks of ICD insertion are felt to outweigh the potential benefits. (See 'ICD therapy' above.)

- Nearly all patients who experience SMVT have an indication for therapy with a beta blocker, including patients with a prior myocardial infarction, patients with HF and reduced LV systolic function, etc. Beta blockers provide some level of protection against recurrent SMVT, primarily by reducing myocardial oxygen demand and blocking sympathetic input to the heart. (See 'Beta blockers' above.)

- Antiarrhythmic drugs may also be used to improve quality of life in patients with frequent SMVT leading to ICD shocks, or in those patients who are not candidates for, or who decline, ICD implantation. Amiodarone has generally been the most effective antiarrhythmic drug for preventing ventricular arrhythmias (and associated ICD shocks). (See 'Antiarrhythmic drugs' above.)

- For patients with recurrent SMVT resulting in ICD shocks despite treatment with an antiarrhythmic drug, we suggest radiofrequency ablation (RFA) rather than the addition of a second antiarrhythmic agent (**Grade 2C**). RFA is also an alternative to antiarrhythmic drugs as the initial therapy for SMVT. In addition, RFA, with or without antiarrhythmic drug therapy, is an option for patients with SMVT who are not candidates for or who refuse ICD implantation. (See 'Radiofrequency catheter ablation' above.)

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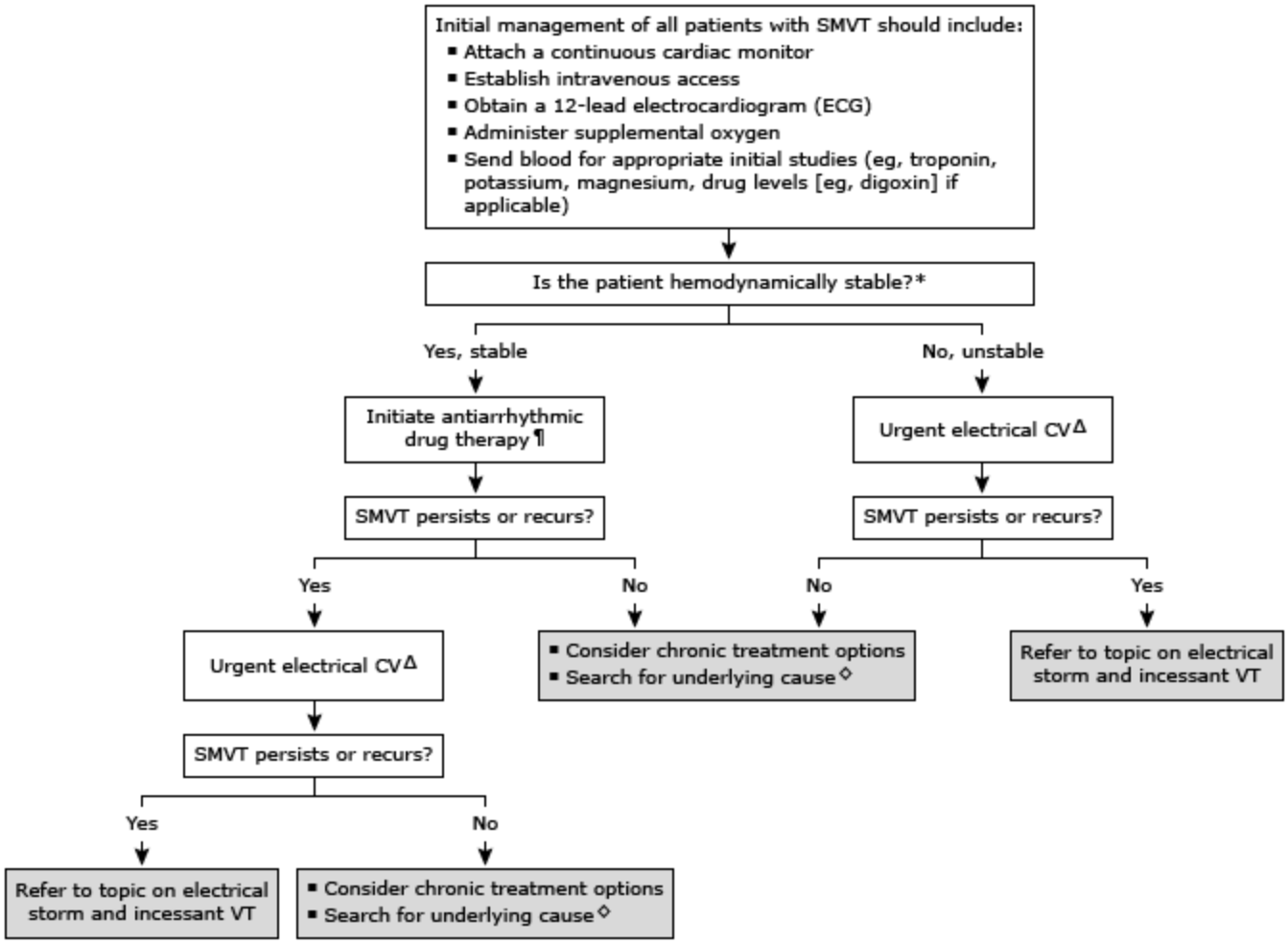
Topic 1040 Version 56.0

GRAPHICS

Algorithm for initial treatment of SMVT in responsive patients with a pulse



SMVT: sustained monomorphic ventricular tachycardia; CV: cardioversion.

* Hemodynamically unstable patients have evidence of hemodynamic compromise, such as hypotension, altered mental status, chest pain, or heart failure. Hemodynamically stable patients should have none of these findings.

¶ Initial choice of pharmacologic agents includes:

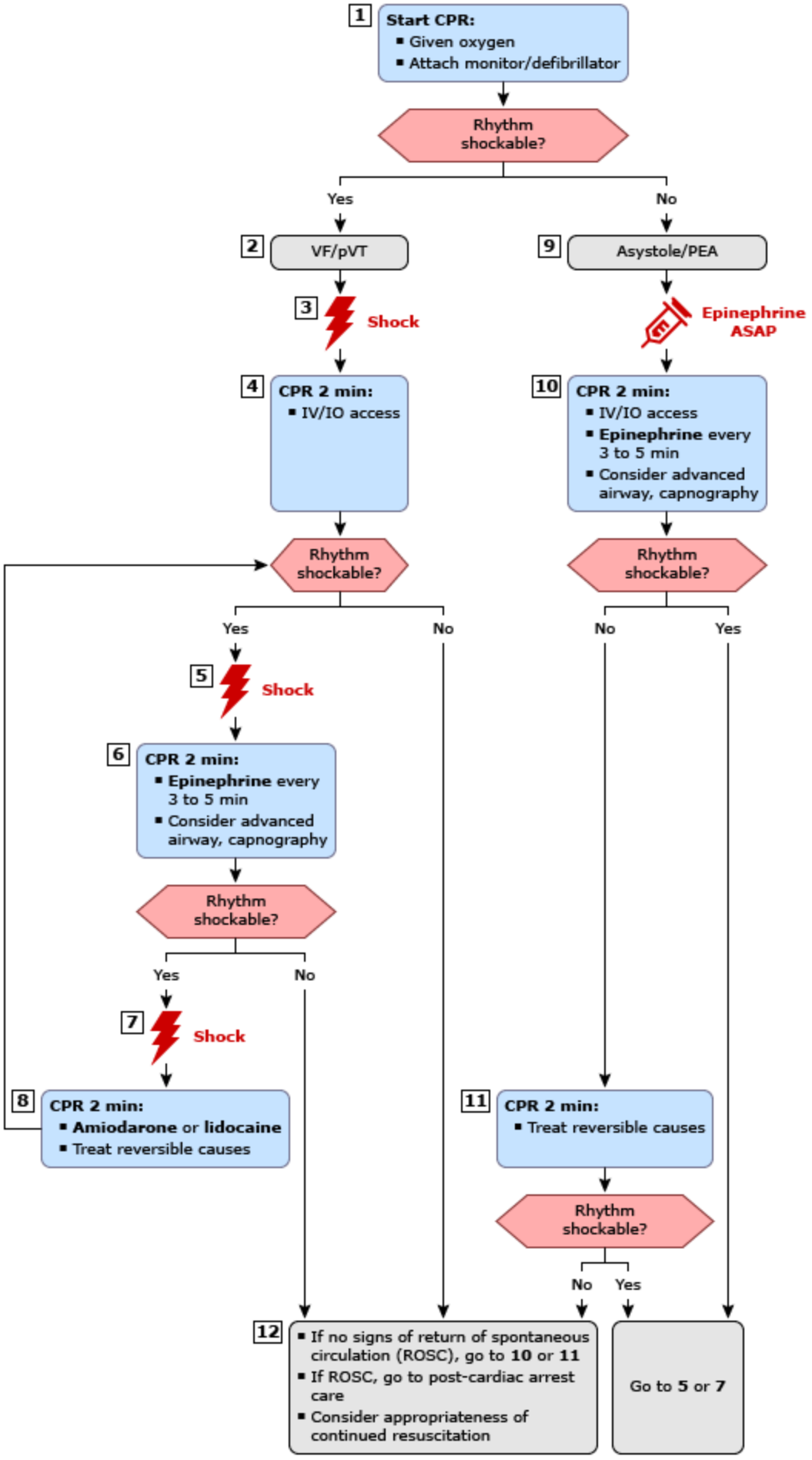
- Intravenous lidocaine (1 to 1.5 mg/kg [typically 75 to 100 mg] at a rate of 25 to 50 mg/minute; lower doses of 0.5 to 0.75 mg/kg can be repeated every 5 to 10 minutes as needed), which may be more effective in the setting of acute myocardial ischemia or infarction
- Intravenous procainamide (20 to 50 mg/minute until arrhythmia terminates or a maximum dose of 17 mg/kg is administered)
- Intravenous amiodarone (150 mg IV over 10 minutes, followed by 1 mg/minute for the next six hours; bolus can be repeated if VT recurs)

Δ Electrical cardioversion should be synchronized if possible, using 100-joule biphasic shock or 200-joule monophasic shock. If first shock is unsuccessful, energy level should be escalated on subsequent shocks.

◇ Conditions associated with SMVT include myocardial ischemia, electrolyte disturbances (eg, hypokalemia, hypomagnesemia), drug-related proarrhythmia, and heart failure.

Graphic 108831 Version 1.0

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

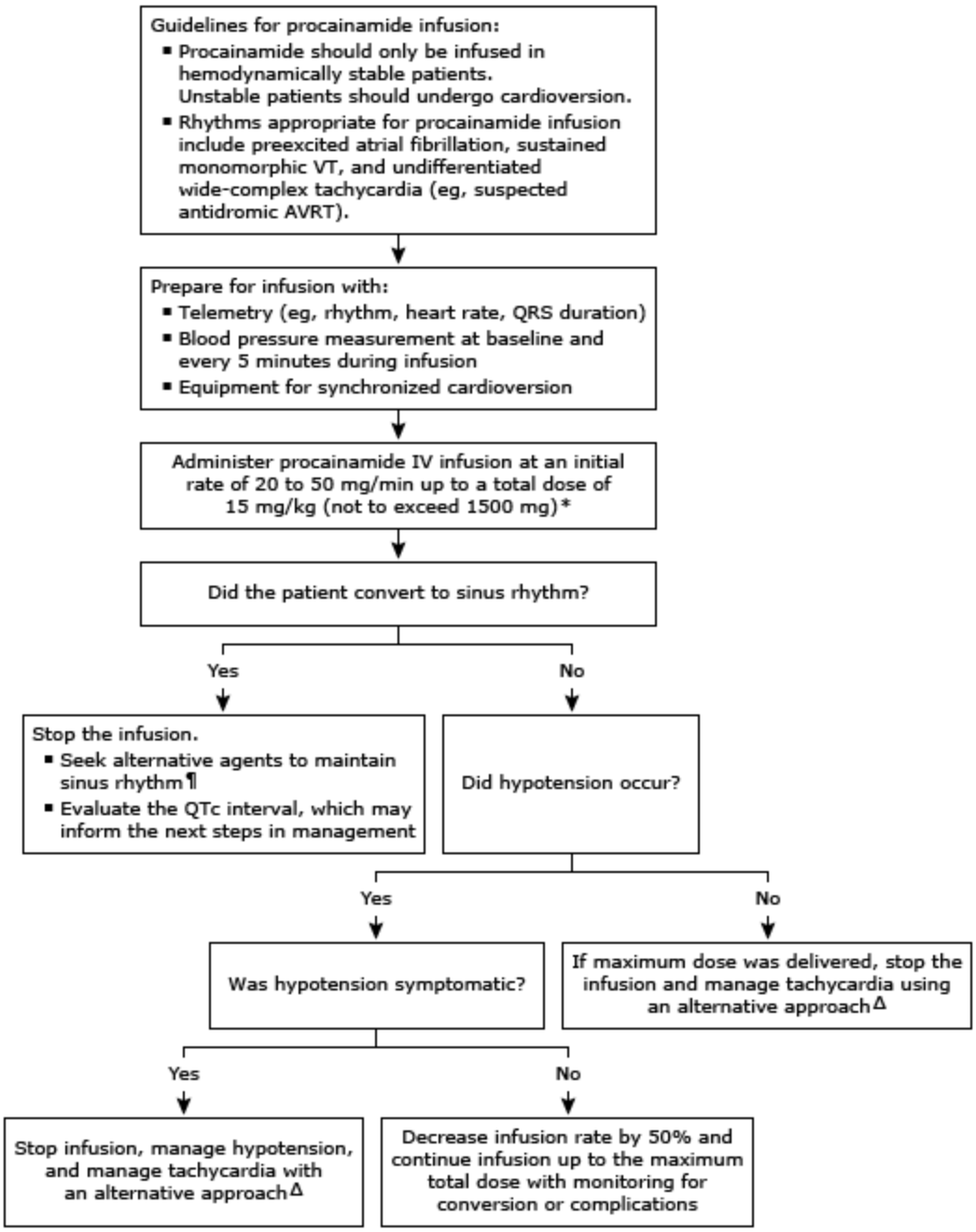
- Pulse and blood pressure.
- Abrupt sustained increase
(typically ≥40 mmHg).
- Spontaneous arterial pres
intra-arterial monitoring.

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

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

Amiodarone dosing in adults by indication

Indications	Loading dose	Maintenance dose
Atrial arrhythmias		
▪ Prevention of recurrent PAF ▪ Pharmacologic cardioversion of PAF	▪ Total loading dose: 6 to 10 grams ▪ Outpatient: Given as 400 to 600 mg orally per day in divided doses with meals ▪ Inpatient: Given as 400 to 1200 mg orally per day in divided doses with meals	▪ Lowest effective dose, usually 100 to 200 mg orally once per day
▪ Pretreatment before elective cardioversion or catheter ablation of AF	▪ Total loading dose: 6 to 10 grams orally over 2 to 6 weeks ▪ Given as 400 to 1200 mg orally per day in divided doses	▪ Lowest effective dose, usually 100 to 200 mg orally once per day
▪ Restoration and maintenance of NSR in critically ill patients with AF ▪ Ventricular rate control in critically ill patients with AF and rapid ventricular response	▪ Total IV loading dose: 1050 mg ▪ Given as 150 mg IV bolus over 10 to 30 minutes, followed by continuous IV infusion at 1 mg per minute for 6 hours, then 0.5 mg per minute for 18 hours* ▪ IV infusion (0.5 mg per minute) may need to be extended past 24 hours if unable to transition to oral therapy ▪ If amiodarone will be used chronically: Following IV infusion, give 400 to 1200 mg orally per day in divided doses to complete a total (IV plus oral) loading dose of 10 grams; consider overlapping IV and oral amiodarone for 24 to 48 hours	
Ventricular arrhythmias		

▪ Primary and secondary prevention of SCD in patients with LV dysfunction who are not candidates for or refuse ICD implantation	▪ Total oral loading dose: 6 to 10 grams ▪ Outpatient: 400 to 600 mg orally per day in divided doses with meal ▪ Inpatient: 400 to 1200 mg orally per day in divided doses with meals for 1 to 2 weeks	▪ Lowest effective dose
▪ Prevention of ventricular arrhythmias in patients with ICDs to decrease risk of shocks	▪ Total loading dose: 6 to 10 grams ▪ Outpatient: Given as 400 to 600 mg orally per day in divided doses with meals ▪ Inpatient: Given as 400 to 1200 mg orally per day in divided doses with meals until desired dose is achieved	▪ Lowest effective dose
▪ Cardiac arrest associated with VF or pulseless VT	▪ 300 mg IV or IO rapid bolus with a repeat dose of 150 mg as indicated ▪ Upon return of spontaneous circulation follow with an infusion of 1 mg per minute for 6 hours and then 0.5 mg per minute for 18 hours*	
▪ Electrical (VT) storm and incessant VT in hemodynamically stable patients	▪ Total IV loading dose: 1050 mg ▪ 150 mg IV bolus over 10 minutes, followed by continuous IV infusion at 1 mg per minute for 6 hours, then 0.5 mg per minute for 18 hours ▪ IV infusion (0.5 mg per minute) may need to be extended past 24 hours if unable to transition to oral therapy¶ ▪ Additional 150 mg boluses may be given if VT storm recurs ▪ If amiodarone will be used chronically: Following IV	▪ If amiodarone is used chronically: Lowest effective dose

	infusion 400 to 1200 mg orally per day in divided doses to complete a total (IV plus oral) loading dose of 10 grams. Consider overlapping IV and oral amiodarone for 24-48 hours	

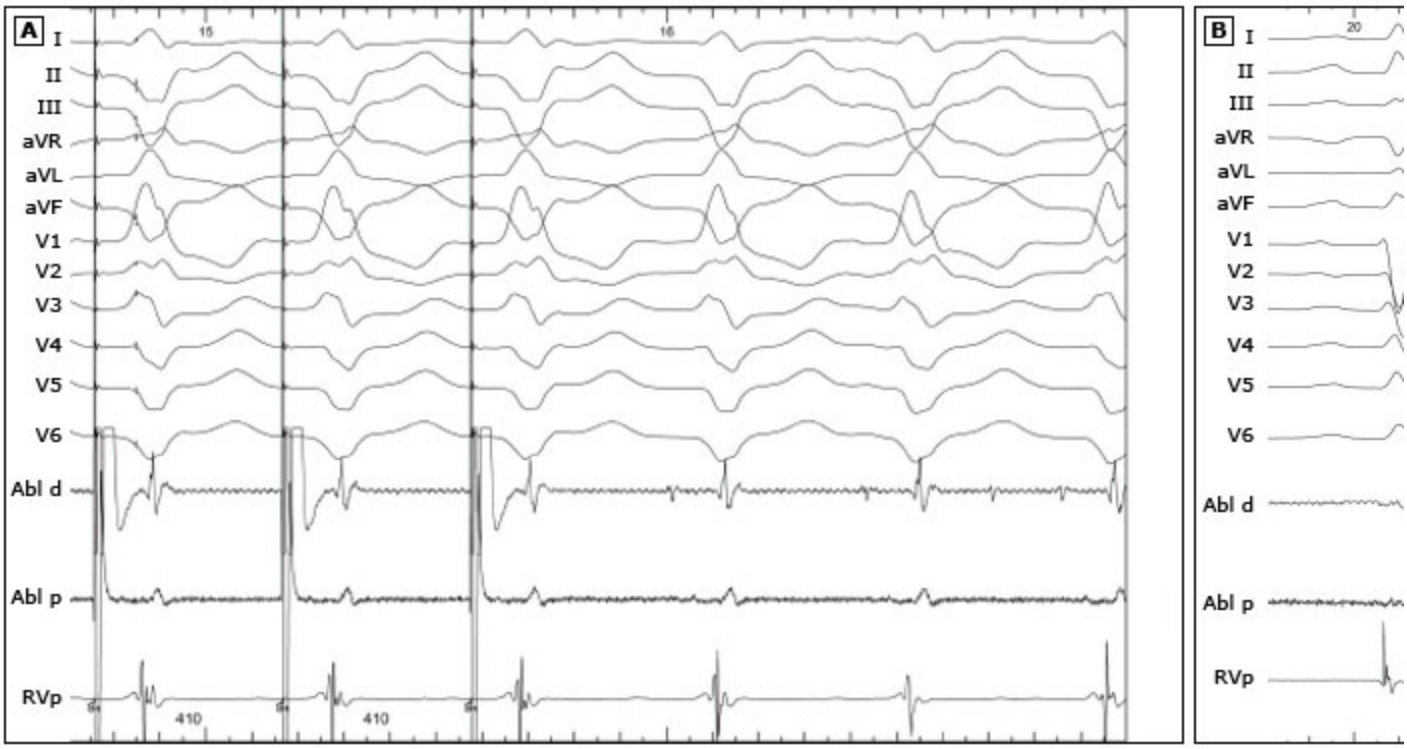
PAF: paroxysmal atrial fibrillation; AF: atrial fibrillation; NSR: normal sinus rhythm; IV: intravenous; SCD: sudden cardiac death; LV: left ventricular; ICD: implantable cardioverter-defibrillator; VF: ventricular fibrillation; VT: ventricular tachycardia; IO: intraosseous.

* When administered to critically ill patients with atrial fibrillation and rapid ventricular response, repeated 150 mg boluses can be given over 10 to 30 minutes if needed, but no more than six to eight additional boluses should be administered in any 24-hour period.

¶ Typically, patients are given 1 or 2 doses of oral amiodarone prior to discontinuation of the IV infusion.

Graphic 117524 Version 6.0

Electrophysiology study (EPS) tracings showing concealed entrainment



(A) Pacing from the distal electrode pair of the mapping/ablation catheter during mapping of sustained ventricular tachycardia. On the left side of the figure are the last three stimuli during a train of pacing at a cycle length of 410 milliseconds. Following pacing, the tachycardia (cycle length 430 milliseconds) resumes. The interval from the last paced beat to resumption of tachycardia, measured in the distal ablation catheter (Abl d), is just 10 milliseconds greater than the tachycardia cycle length. Note that the interval from the pacing stimulus to onset of the QRS complex is the same as the interval from the local electrogram to the QRS onset when VT resumes after pacing. Note also that the paced VT morphology replicates the morphology of the spontaneous tachycardia. This is an example of "concealed entrainment" and implies the catheter is within a critical part of a reentrant circuit.

(B) Same site on the ablation catheter (Abl d) during sinus rhythm as shown during VT in figure A. During sinus rhythm, a late potential is present. The vertical line denotes the end of the QRS complex. Delivery of radiofrequency energy at this site resulted in termination of VT. The tachycardia was no longer inducible after ablation. Sites such as this displaying late potentials may be targeted during "substrate-guided ablation."

VT: ventricular tachycardia.

Graphic 107237 Version 2.0

Complications of invasive cardiac electrophysiology studies

Associated with percutaneous catheterization of veins and arteries
Pain
Adverse drug reaction
Infection/abscess at the catheterization site, sepsis
Excessive bleeding, hematoma formation
Thrombophlebitis
Pulmonary thromboembolism
Arterial damage, aortic dissection
Systemic thromboembolism
Transient ischemic attack/stroke
Associated with intracardiac catheters and programmed cardiac stimulation
Cardiac chamber or coronary sinus perforation
Hemopericardium, cardiac tamponade
Atrial fibrillation
Ventricular tachycardia/ventricular fibrillation
Myocardial infarction
Right or left bundle branch block
Associated with transcatheter ablation
Complete heart block
Thromboembolism
Vascular access problems (bleeding, infection, hematoma, vascular injury)
Cardiac trauma (myocardial perforation, tamponade, valvular damage)
Coronary artery thrombosis/myocardial infarction
Cardiac arrhythmias
Pericarditis
Pulmonary vein stenosis
Phrenic nerve paralysis
Radiation skin burns
Possible late malignancy

Atrioesophageal fistula
Death resulting from one of the above complications

Graphic 63157 Version 4.0