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Society Position Statement

2020 Canadian Cardiovascular Society/Canadian Heart Rhythm Society Position Statement on the Management of Ventricular Tachycardia and Fibrillation in Patients With Structural Heart Disease

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ABSTRACT

This Canadian Cardiovascular Society position statement is focused on the management of sustained ventricular tachycardia (VT) and ventricular fibrillation (VF) that occurs in patients with structural heart disease (SHD), including previous myocardial infarction, dilated cardiomyopathy, and other forms of nonischemic cardiomyopathy. This patient population is rapidly increasing because of advances in care and improved overall survival of patients with all forms of SHD. In this position statement, the acute and long-term management of VT/VF are outlined, and the many unique aspects of care in this population

RÉSUMÉ

Le présent énoncé de position de la Société canadienne de cardiologie est axé sur la prise en charge de la tachycardie ventriculaire (TV) et de la fibrillation ventriculaire (FV) soutenues qui surviennent chez les patients présentant une cardiopathie structurelle, par exemple des antécédents d'infarctus du myocarde, une cardiomyopathie dilatée et d'autres formes de cardiomyopathie non ischémique. Cette population de patients augmente rapidement, en raison des avancées réalisées en matière de soins et de l'amélioration de la survie globale des patients présentant une cardiopathie structurelle sous une forme ou

1. Scope of the Position Statement

This Canadian Cardiovascular Society position statement is focused on the acute and long-term management of sustained ventricular tachycardia (VT) and ventricular fibrillation (VF) in patients with structural heart disease (SHD), defined by the presence of abnormal myocardium and scar. SHD includes conditions such as myocardial infarction (MI), dilated cardiomyopathies, hypertrophic cardiomyopathy, infiltrative cardiomyopathies (eg, sarcoidosis), and arrhythmogenic right ventricular cardiomyopathy

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The disclosure information of the authors and reviewers is available from the CCS on their guidelines library at www.ccs.ca.

This statement was developed following a thorough consideration of medical literature and the best available evidence and clinical experience. It represents the consensus of a Canadian panel comprised of multidisciplinary experts on this topic with a mandate to formulate disease-specific recommendations. These recommendations are aimed to provide a reasonable and practical approach to care for specialists and allied health professionals obliged with the duty of bestowing optimal care to patients and families, and can be subject to change as scientific knowledge and technology advance and as practice patterns evolve. The statement is not intended to be a substitute for physicians using their individual judgement in managing clinical care in consultation with the patient, with appropriate regard to all the individual circumstances of the patient, diagnostic and treatment options available and available resources. Adherence to these recommendations will not necessarily produce successful outcomes in every case.

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are emphasized. The initial evaluation, acute therapy, indications for chronic suppressive therapy, choices of chronic suppressive therapy, implantable cardioverter-defibrillator programming, alternative therapies, and psychosocial care are reviewed and recommendations for optimal care are provided. The target audience for this statement includes all health professionals involved in the continuum of care of patients with SHD and VT/VF.

une autre. L'énoncé de position présente les grandes lignes de la prise en charge aiguë et à long terme de la TV et de la FV, et fait ressortir les nombreux aspects propres aux soins dont cette population a besoin. L'évaluation initiale, le traitement aigu, les indications commandant un traitement suppresseur prolongé, les options de traitement suppresseur prolongé, la programmation des défibrillateurs cardioverteurs implantables, les thérapies parallèles et les soins psychosociaux sont examinés, et des recommandations concernant les soins optimaux sont formulées. Ce document est destiné à tous les professionnels de la santé qui interviennent dans le continuum des soins aux patients présentant une cardiopathie structurelle et une TV/FV.

(ARVC). Almost all such patients have an indication for an implantable cardioverter-defibrillator (ICD).[1] Although many of the recommendations in this document can apply to patients with complex congenital heart disease, this population is not specifically addressed. The reader is referred to other published resources on the management of VT/VF in that population.[2,3]

This statement is not intended to replace the guidelines for cardiopulmonary resuscitation,[4] but provides guidance for the ongoing care of patients with VT/VF after resuscitation. This statement is targeted at health professionals involved in the continuum of care of patients with SHD and VT/VF, including emergency medicine, critical care, internal medicine, and cardiology. The management of premature ventricular complexes and nonsustained VT in patients with SHD, and the management of VT/VF in patients with structurally normal hearts (including inherited arrhythmia syndromes) are beyond the scope of this statement.

2. Position Statement Development

This position statement was written by a multidisciplinary panel of experts who care for patients with VT/VF (see Supplemental Table S1). The recommendations were developed using the Grading of Recommendations, Assessment, Development, and Evaluation standards with strength of recommendations classified as "strong" or "conditional." The recommendations are presented, along with their background and rationale.

3. Definitions

Sustained VT is defined as an episode of VT lasting > 30 seconds or requiring intervention before 30 seconds.[5] Hemodynamically stable VT is defined as VT without signs or symptoms of organ hypoperfusion. Electrical storm (also known as VT/VF storm) is defined as the occurrence of 3 or more distinct episodes of VT/VF within 24 hours. Monomorphic VT has similar QRS complexes on the electrocardiogram (ECG), whereas polymorphic VT has changing beat-to-beat morphology. Torsade de pointes is polymorphic VT occurring in the setting of QT prolongation. VF is chaotic and does not have distinct QRS complexes on the ECG. Monomorphic VT might degenerate to polymorphic VT or VF. A summary of classification and mechanisms of VT and VF in patients with SHD is presented in Supplemental Table S2.

4. Incidence and Prognosis of Sustained VT/VF in Patients With SHD

Ventricular arrhythmias can cause sudden cardiac death (SCD) or arrest, most frequently in those with previous MI.[6] The past decade saw a decreasing incidence of post-MI VT/VF causing SCD, yet no change in the incidence of SCD in patients without ischemic heart disease.[7] The development of VT/VF in patients with SHD is associated with a higher risk of future episodes of VT/VF, of electrical storm, and of death.[8]

5. Initial Evaluation and Management of SHD Patients With Sustained VT/VF

Patients who present with VT/VF often have a preexisting diagnosis of SHD. However, VT/VF might be the initial indication of the presence of SHD for some patients. The initial evaluation must rapidly assess the need for acute intervention and identify potential etiologies or precipitating factors. When the patient is stabilized, the evaluation should include a complete history and examination, comprehensive laboratory testing, and assessment of cardiac function (Fig. 1).

RECOMMENDATION

1. We recommend that all patients presenting with VT/VF undergo a comprehensive initial evaluation including a detailed history, physical examination, laboratory investigations, ECG, ICD interrogation (if present) and transthoracic echocardiography (Strong Recommendation, Low-Quality Evidence).

Values and preferences. A comprehensive initial evaluation can be performed quickly and at a reasonable cost. This evaluation is critical for guiding subsequent treatment.

Practical tip. If not previously diagnosed, most forms of SHD will be apparent after this initial evaluation. Unless a reversible cause is identified (such as acute MI), all patients with VT/VF and SHD should be referred for consideration of an ICD, if not already present, for the prevention of sudden death.

A 12-lead ECG recorded during VT is important to localize VT and give insight into pathophysiology. An ECG performed after restoration of the underlying rhythm can be used to evaluate for reversible causes, such as ischemia and QT prolongation. Laboratory tests might identify precipitating

Initial Evaluation of Sustained VT/VF

In All Patients

History	Clinical Evaluation	ECG

• Precipitating factors
• Symptoms during and preceding VT/VF
• Symptoms of heart failure
• Nature of structural heart disease (if known)
• Medications (prescribed and other)
• Drugs, alcohol, stimulants
• Family history
• Other pertinent medical history
	• Hemodynamic assessment
	• Heart failure assessment
	Laboratory Investigations
	• CBC
	• Electrolytes, renal function, liver function
	• TSH
	• Troponin
	• BNP/NTproBNP
	• Toxicology, drug levels (if suspected)
		• ***During VT*** (if stable)
		• After restoration of underlying rhythm
		  • Ischemia/infarction changes
		  • Conduction disease
		  • QT interval
		Echocardiography
		• Transthoracic*
		ICD Interrogation
		• If present

In Selected Patients

Cardiac Magnetic Resonance Imaging (CMR)	Coronary Evaluation	Positron Emission Tomography (PET)
• In patients with suboptimal images or equivocal findings on echocardiography or other imaging modalities • For further evaluation of inflammation or scar/fibrosis	• Coronary angiography in patients with VT/VF and acute STEMI (emergent) • In patients with polymorphic VT/VF and suspicion of CAD • In selected patients with monomorphic VT in whom CAD is suspected	• In patients with suspected inflammatory processes (particularly sarcoidosis), to guide decisions regarding anti-inflammatory/ immunosuppression

Figure 1. Initial evaluation of patients with sustained VT/VF. * Even if recently performed, a repeat transthoracic echocardiogram should be acquired to rule out acute changes. BNP, brain natriuretic peptide; CAD, coronary artery disease; CBC, complete blood count; NT, N-terminal; STEMI, ST-elevation myocardial infarction; TSH, thyroid stimulating hormone; VF, ventricular fibrillation; VT, ventricular tachycardia.

factors, prognostic factors, and contraindications to specific antiarrhythmic drug (AAD) therapies (such as renal or hepatic dysfunction). Cardiac imaging is useful to determine the presence of underlying SHD and aid prognostication. Transthoracic echocardiography remains the first-line diagnostic imaging tool for assessment of ventricular function, wall motion abnormalities, and valvular disease. Nevertheless, it has limitations in identifying scar. Cardiac magnetic resonance (CMR) imaging, with gadolinium contrast, can support the diagnosis, particularly with nonischemic and infiltrative cardiomyopathies.[9,10]

RECOMMENDATION

2. We recommend that CMR imaging be performed in patients who present with VT/VF when the initial evaluation has failed to establish the etiology of the underlying heart disease (Strong Recommendation, Moderate-Quality Evidence).

Values and preferences. CMR imaging provides enhanced assessment of the presence, location, and quantity of myocardial scar in patients with SHD and can identify inflammatory conditions, such as myocarditis.

Practical tip. In patients for whom the initial workup is not definitive, early use of CMR imaging has a high diagnostic and prognostic yield.

Emergent angiography should be performed for VT/VF in the setting of ST-elevation MI. Urgent angiography should also be considered in those who present with polymorphic VT/VF potentially due to acute ischemia. Other coronary imaging modalities can be considered if there is a lower index of suspicion of coronary disease. Monomorphic VT is rarely caused by acute ischemia, but coronary imaging might be warranted to establish the presence and severity of coronary artery disease.

Positron emission tomography can be useful in identifying patients with active inflammatory states, such as sarcoidosis, when other imaging modalities yield negative or equivocal findings.[11] Invasive electrophysiological testing of patients with wide complex tachycardia might be useful to distinguish VT from SVT or to diagnose bundle-branch reentry VT.[12]

5.1. Management of hemodynamically unstable VT/VF

Figure 2 illustrates the acute management of patients with VT/VF. In patients with unstable VT electrical cardioversion/defibrillation is indicated and advanced cardiac life support algorithms should be followed (Fig. 2A).[13,14] In

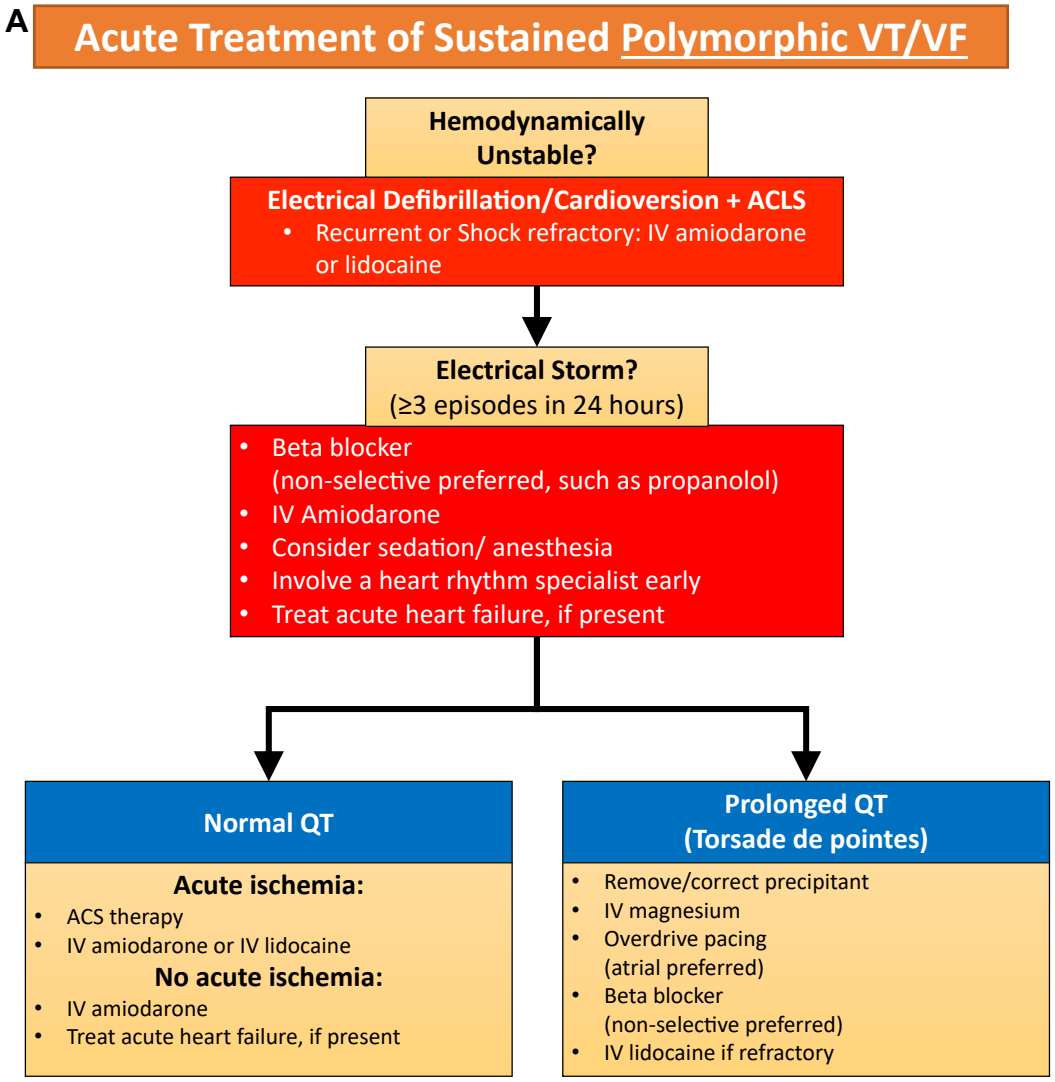

Figure 2. Initial management of sustained VT/VF. The initial management of VF and polymorphic VT is outlined in (**A**), whereas the initial management of monomorphic VT is outlined in (**B**). For unstable patients with VT/VF that recurs despite defibrillation/cardioversion or VT/VF that persists despite 1-2 attempts at defibrillation/cardioversion, intravenous (I.V.) amiodarone or lidocaine should be administered. For unstable VT/VF that recurs, the treatment should follow the electrical storm recommendations. See the accompanying text for drug dosing. ACLS, Advanced Cardiac Life Support; ACS, acute coronary syndrome; VF, ventricular fibrillation; VT, ventricular tachycardia.

patients with shock-refractory VF or VT, bolus intravenous (I.V.) amiodarone or lidocaine should be used.[15,16] Intravenous AAD dosing is summarized in Supplemental Table S3.

RECOMMENDATION

3. We recommend the administration of I.V. amiodarone or lidocaine for acute treatment of patients with shock-refractory VT/VF (failure of at least 1 attempt at defibrillation) or patients with recurrent polymorphic VT/VF, unless there is a strong suspicion of torsade de pointes (Strong Recommendation, Moderate-Quality Evidence).

Values and preferences. The outcome of unstable patients with VT/VF arrest is poor, particularly in out-of-hospital settings. There is likely a small benefit to these drugs, which outweigh their risks.

Practical tip. Simplified, rather than weight-based dosing is easier in the setting of unstable VT/VF. Amiodarone can be given as 300 mg I.V. push (150 mg I.V. push for those < 45 kg) with a subsequent dose of 150 mg I.V. push in the event of failure of another shock. Lidocaine can be given as 100 mg I.V. push (50 mg I.V. push if < 45 kg) with a subsequent dose of 50 mg I.V. push in the event of failure of another shock.

5.2. Electrical storm

Initial management of electrical storm often includes a combination of sedation, β-blockade, and AAD therapy. Short-acting β-blockers, (eg, esmolol) might be considered to assess tolerability. In a recent randomized trial, non-selective

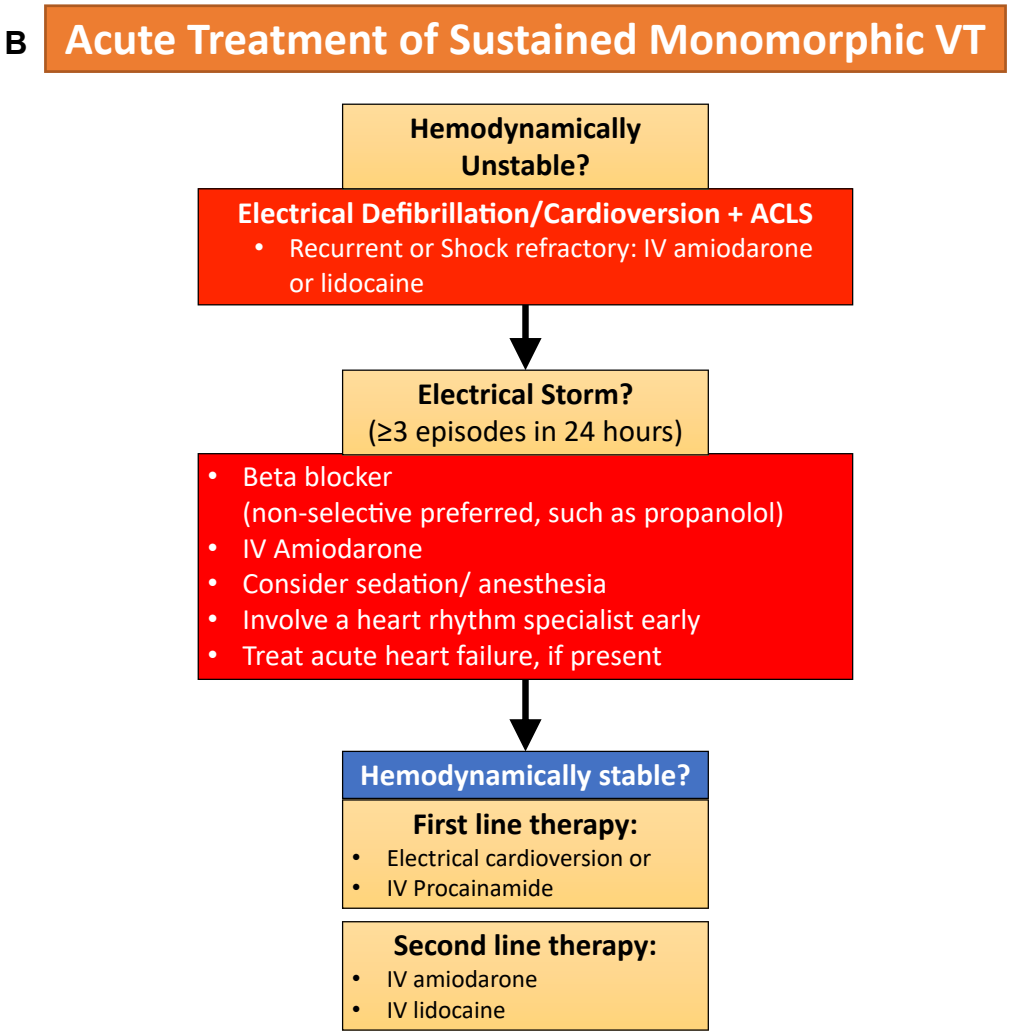

Figure 2. (*continued*).

β-blockade with propranolol, given immediately and continued during the hospitalization, was superior to selective β1 blockade (metoprolol) for suppressing VT, in conjunction with usual care including I.V. amiodarone.[17] If initial management strategies fail, general anaesthesia can be considered. Dexmedetomidine and propofol are preferred sedating agents because of their sedative and sympatholytic effects.[18,19] Patients with an ICD and electrical storm should undergo early device interrogation; ICD programming can be adjusted (such as optimizing antitachycardia pacing [ATP]) or ICD therapy can be temporarily disabled, if appropriate.

Amiodarone is useful for electrical storm in patients with SHD, and a target loading dose of 1000-2000 mg I.V. over the first 24 hours is recommended (Supplemental Table S3).[20,21] Combinations of AADs with distinct mechanisms of action (eg, amiodarone and lidocaine) might prove effective for acute arrhythmia suppression in selected patients. Ablation should be considered in selected patients with drug-refractory electrical storm due to monomorphic VT.[22,23] For recurrent polymorphic VT or VF in the absence of acute ischemia, administration of I.V. magnesium can be considered. Treatment directed at acute heart failure should also be considered, because this can precipitate VT/VF.

Refractory patients might benefit from thoracic epidural anaesthesia or percutaneous cardiac sympathetic denervation. For recurrent VT/VF with prolonged hemodynamic instability, mechanical circulatory support might be considered (the readers are referred to the reference list of a review for temporary mechanical support in critical cardiac care[24]).

RECOMMENDATION

4. We recommend the use of β-blockade, preferably nonselective β-blockade, and I.V. amiodarone in patients with electrical storm in the setting of underlying SHD (Strong Recommendation, Moderate-Quality Evidence).

Values and preferences. The outcomes of patients with electrical storm are poor. As such, aggressive intervention is often required.

Practical tip. In patients with stable blood pressure, oral, short-acting, nonselective β-blockers should be used preferentially (such as propranolol 40 mg orally every 6 hours).

5.3. Stable monomorphic VT

Electrical cardioversion or I.V. AADs can acutely treat stable monomorphic VT in patients with SHD (Fig. 2B). Procainamide is superior to amiodarone for conversion of hemodynamically tolerated monomorphic VT, with higher efficacy and fewer adverse effects.[25] Procainamide is also superior to lidocaine for acute termination of stable monomorphic VT.[26]

RECOMMENDATION

5. We recommend electrical cardioversion or I.V. procainamide for the acute treatment of stable monomorphic VT in patients with SHD (Strong Recommendation, Moderate-Quality Evidence).

Values and preferences. Procainamide therapy can frequently terminate monomorphic VT without the need for cardioversion, but might interact with chronic AADs (such as amiodarone or sotalol). Cardioversion, although effective at terminating monomorphic VT, requires sedation and does not prevent recurrent VT. Selection of the preferred first-line approach should take into consideration local resources and expertise, as well as patient preference.

Practical tip. There are a wide variety of reported doses of I.V. procainamide used in the literature but a rapid infusion of 10 mg/kg over 20 minutes has good efficacy and a low rate of hypotension (Supplemental Table S3). The infusion can be slowed if hypotension is encountered. This can be followed by an ongoing infusion of 1-2 mg/min. Amiodarone I.V. (150 mg I.V. over 10 minutes, followed by an infusion of 1 mg/min for 6 hours, followed by 0.5 mg/min for 18 hours) or lidocaine (1 mg/kg I.V. push, followed by 1-2 mg/min infusion), can be given as second-line alternatives to procainamide.

5.4. Polymorphic VT

Most patients with sustained polymorphic VT are unstable and should be acutely treated as per VF (Fig. 2) with aggressive early investigation and treatment of underlying causes, such as acute ischemia (most commonly), decompensated heart failure or other potential contributors (Supplemental Table S2). Polymorphic VT due to QT prolongation (torsade de pointes) can be related to drugs, electrolyte disturbance (hypokalemia/hypomagnesemia), or (rarely) concomitant long QT syndrome. Magnesium can be useful in patients with torsade de pointes regardless of measured serum magnesium levels,[27] but not for ischemia-related ventricular arrhythmias.[28] Although no evidence-based dosing or targets exist for VT, it would be reasonable to target a serum potassium $\geq$ 4.0 mmol/L and serum magnesium $\geq$ 0.9-1.0 mmol/L.

6. Initiation of Long-Term Suppressive Therapy for Sustained VT/VF

6.1. Goals for initiating long-term suppressive therapy for VT/VF

Although appropriate ICD shocks are often acutely life-saving, they are associated with a subsequent increased mortality risk, even compared with VT/VF terminated with ATP therapy. Furthermore, ICD shocks can result in significant psychosocial morbidity (see section *11. Psychosocial Care of Patients With VT/VF*). Treating VT/VF should aim to reduce VT/VF burden and ICD shocks. Optimization of ICD programming (see section *9. ICD Programming in Patients With Sustained VT/VF in the Setting of SHD*) should be performed in all patients with VT/VF to minimize shocks. Suppression of VT/VF might be accomplished by use of AADs and/or catheter ablation, the latter predominantly indicated for monomorphic VT (Fig. 3).

6.2. First episode of sustained VT/VF

The risk of recurrent VT/VF after a first event, without suppressive therapy is approximately 22% after 1 year and approximately 53% after 2 years.[29-31] Catheter ablation after the first episode of monomorphic VT reduces recurrences of VT (hazard ratios 0.54-0.61 vs controls for recurrent VT at 1-2 years),[31-33] but these studies did not routinely use active controls with AAD therapy (AAD use was only 32%-35%). In the most recent randomized trial, VT ablation did not reduce the composite outcome of death, VT hospitalization, or worsening heart failure, compared with ICD therapy alone.[33] No randomized trial has specifically evaluated the effect of AAD therapy after a first VT/VF episode, although these patients were represented in larger trials that assessed drug efficacy. Although usually unnecessary, suppressive therapy beyond β-blockade after a first VT/VF episode might be warranted despite the potential risks of therapy, particularly if the patient experienced significant morbidity with the index episode (Fig. 3). Importantly, ICD implantation should be performed with optimization of programming to minimize future shocks (see section *9. ICD Programming in Patients With Sustained VT/VF in the Setting of SHD*).

6.3. Recurrent sustained VT/VF

Recurrent episodes of VT/VF despite optimal medical therapy are associated with increased risks of electrical storm and mortality[29,31,34] and therefore treatment with AADs or catheter ablation is generally recommended (see Fig. 3 and sections *7. AAD Therapy for Long-term Management of Sustained VT/VF* and *8. Catheter Ablation for the Treatment of Sustained VT in Patients With SHD*). In addition to optimizing β-blockade and ICD programming (see section *9. ICD Programming in Patients With Sustained VT/VF in the Setting of SHD*), all patients should have other cardiac therapy optimized. β-Blockers should be uptitrated to evidence-based doses for heart failure, when heart rate, blood pressure, and side effects allow.[35] Ideally, β-blocker doses should target a resting heart rate of 50-65 beats per minute.

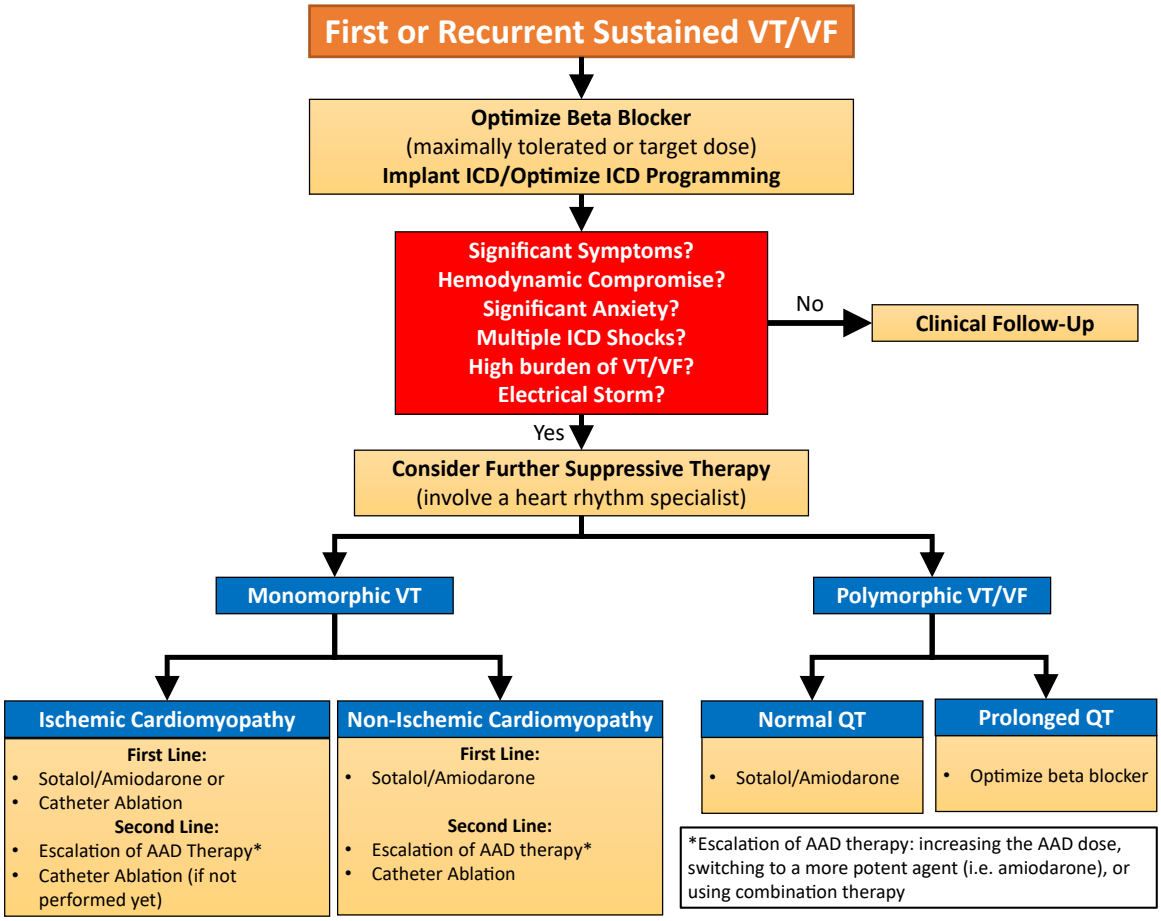

Figure 3. When to initiate chronic suppressive therapy for sustained VT/VF. * Escalation of AAD therapy: increasing the AAD dose, switching to a more potent agent (ie, amiodarone), or using combination therapy. AAD, antiarrhythmic drug; ICD, implantable cardioverter-defibrillator; VF, ventricular fibrillation; VT, ventricular tachycardia.

RECOMMENDATION

6. In patients with SHD and new or recurrent VT/VF, we recommend: (1) optimizing β-blocker dose in all patients; (2) optimizing ICD programming; and (3) consideration of initiation of additional suppressive therapy (either class III AAD therapy or catheter ablation), particularly in patients with VT/VF resulting in ICD shock(s), in those with a high burden of VT/VF, and in those with severe symptoms/hemodynamic compromise or psychosocial distress (Strong Recommendation, Low-Quality Evidence).

Values and preferences. The risk of recurrent VT/VF is high, especially after multiple events. The negative effect of recurrent events, particularly those associated with severe symptoms or ICD shocks, warrants therapy.

Practical tip. For patients receiving no or a low-dose β-blocker, consider the introduction/titration of a β-blocker alone. However, in patients already receiving reasonable doses of β-blocker, additional suppressive therapy should be used.

6.4. Electrical storm

Electrical storm has a 2-year recurrence rate of 45%-60%.[36,37] Chronic suppressive AAD therapy is almost always indicated. When AAD therapy is unsuccessful, catheter ablation for monomorphic VT can reduce VT recurrences.[22,23]

RECOMMENDATION

7. We recommend optimizing β-blocker dose and using additional suppressive therapy (amiodarone or catheter ablation), in patients with SHD who present with electrical storm (Strong Recommendation, Moderate-Quality Evidence).

Values and preferences. The poor prognosis and high recurrence rate of VT/VF in patients with electrical storm warrants long-term suppressive therapy to reduce the risk of recurrence.

Practical tip. After the acute treatment of electrical storm, in addition to β-blocker therapy, chronic suppressive therapy should be initiated/undertaken while in hospital. Amiodarone is the preferred AAD for patients with electrical storm.

Table 1. First-line class III antiarrhythmic drugs for suppression of VT/VF in structural heart disease

Drug	Starting dose	Target dose	Caution	Monitoring	Common and severe effects
Sotalol	40-80 mg BID	120-160 mg BID	Baseline prolonged QT • > 450 msec with normal QRS • > 480 msec with bundle branch block Renal failure: • GFR 30-60: reduce dose and consider once-daily dosing • GFR < 30: contraindicated Advanced heart failure (NYHA ≥ 3 and/or LVEF < 20%) • Use with caution Body weight ≤ 60 kg	Laboratory • Renal function: GFR baseline and every 6 months ECG: (QT monitoring) • Baseline, 5-7 days after starting and every 6 months • Reduce dose or stop if QTc ≥ 500 msec	Fatigue Bradycardia (sinus bradycardia or atrial fibrillation with slow ventricular response) Dizziness/hypotension Proarrhythmia (prolonged QT with torsade de pointes)
Amiodarone	Initial dose: 400 mg BID for 14 days (consider 400 mg TID for 8 days in inpatients) Maintenance dose: 200 mg daily	200-400 mg daily	Concomitant digoxin administration (amiodarone increases serum digoxin concentration) Concomitant warfarin • Amiodarone potentiates warfarin • Reduce warfarin dose by 20%-30% when starting amiodarone and monitor INR frequently	Laboratory: • Liver enzymes and thyroid function (TSH) baseline and every 6 months ECG: • Baseline Chest x-ray: • Baseline if preexisting lung disease and with symptoms Pulmonary function testing • Baseline for those with preexisting lung disease Ophthalmologic evaluation: • Baseline if existing visual impairment and with symptoms	Skin (photosensitivity) Ataxia/tremor Visual changes Nausea or diarrhea (particularly with loading dose) Constipation Bradycardia (sinus bradycardia or atrial fibrillation with slow ventricular response) Hyperthyroidism Hypothyroidism * Increased creatinine Pulmonary toxicity (pneumonitis or fibrosis—typically with long-term use) Liver toxicity

BID, twice per day; ECG, electrocardiogram; GFR, glomerular filtration rate; INR, international normalized ratio; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association; TID, 3 times per day; TSH, thyroid stimulating hormone; VF, ventricular fibrillation; VT, ventricular tachycardia.

*Amiodarone blocks the tubular secretion of creatinine by p-glycoprotein. This reduces the total clearance of creatinine resulting in a 5%-15% increase in serum creatinine concentrations in patients who start amiodarone therapy.

7. AAD Therapy for Long-Term Management of Sustained VT/VF

AADs (with the exception of β-blockers) are not known to improve survival in patients with SHD[38,39] and cannot replace ICD therapy, which has consistently resulted in survival benefit compared with AAD therapy for VT/VF. Nevertheless, in patients who are not candidates for ICD implantation, amiodarone might be useful for treatment of VT/VF.[40]

7.1. β-Blockers

β-Blocker therapy is recommended in patients with SHD for the prevention of VT/VF and sudden death, in conjunction with an ICD.[41,42] Clinical data support a modest AAD effect of β-blockers compared with placebo.[43,44]

RECOMMENDATION

8. We recommend β-blocker therapy, titrated to a maximally tolerated dose (optimized dose), in patients with SHD with VT/VF (Strong Recommendation, Moderate-Quality Evidence).

Values and preferences. β-Blockers are almost always indicated in patients with SHD, particularly those with VT/VF. The risks of β-blocker therapy are outweighed by the many benefits.

Practical tip. Most patients with SHD will also have nonarrhythmic indications for β-blocker therapy, such as for treatment of coronary artery disease with ischemia or heart failure with reduced ejection fraction.

7.2. Sotalol

Sotalol has better efficacy in reducing VT compared with placebo or β-blocker monotherapy but is less effective than amiodarone.[29,30] Practical considerations for the use of sotalol are outlined in Table 1. Sotalol has a side effect profile similar to other β-blockers with the added risk of QT prolongation. However, rates of sotalol discontinuation were higher than other β-blockers in an open-label randomized trial (23.5% vs 5.3% at 1 year).[29] Sotalol proarrhythmic risk might be higher in patients with advanced heart failure,[45] but its use might be appropriate in those with less severe heart failure or as an adjunct to an ICD.[29,30] Sotalol should be avoided in patients with a prolonged QT and should be used with caution in those with severe heart failure (left ventricular ejection fraction < 20% and/or New York Heart Association classification ≥ 3) or renal failure. Although sotalol was most commonly tested in randomized trials without concomitant use of other β-blocker therapy,[29,30] consideration can be given to using sotalol in conjunction with an evidence-based β-blocker (carvedilol, bisoprolol,

metoprolol succinate, nebivolol)[35] for patients with heart failure and reduced ejection fraction.

7.3. Amiodarone

Amiodarone is the most effective AAD for reducing VT/VF recurrence in patients with SHD and an ICD.[29] It requires approximately 10 g (I.V.) to 20 g (oral) dose to achieve steady state and full antiarrhythmic effect (Table 1). Its main limitation is the risk of long-term adverse effects. The discontinuation rate for amiodarone at 1 year is approximately 10%-20%, and increases with time.[29,34]

RECOMMENDATION

9. If AAD therapy is chosen for suppressive therapy, we recommend that either sotalol or amiodarone be used as first-line AAD therapy for suppression of VT/VF in patients with SHD (Strong Recommendation, High-Quality Evidence).

Values and preferences. Although amiodarone is more effective in suppressing VT/VF compared with sotalol, it is associated with long-term toxicities, hence both agents are reasonable as first-line therapy. Each appears to be relatively safe in patients with SHD.

Practical tip. Sotalol, given its more favourable long-term toxicity profile, should be preferentially used. Sotalol can be used alone or in addition to preexisting β-blocker therapy. However, amiodarone should be used for the treatment of electrical storm, because of its superior efficacy. See Supplemental Table S3 for guidance on drug selection and dosing.

8. Catheter Ablation for the Treatment of Sustained VT in Patients With SHD

8.1. Patient selection

Catheter ablation is effective for patients with monomorphic VT and SHD. Procedural success is generally highest for patients with hemodynamically tolerated monomorphic VT and those with higher left ventricular ejection fraction,[31,46] although substrate ablation techniques have improved ablation effectiveness in patients with worse ventricular systolic function.[47,48] There is a wide spectrum of SHD that leads to scar-related VT. Distribution, location, and heterogeneity of the underlying scar tissue are important determinants of procedural outcomes.

8.1.1. Ischemic cardiomyopathy. A randomized study in patients who have refractory monomorphic VT despite AAD therapy showed that catheter ablation is more effective than escalation of AAD therapy, particularly after failure of amiodarone treatment.[34] Catheter ablation may be considered for first-line suppressive therapy,[31,49] to reduce recurrent subsequent ICD therapies, although randomized comparisons with AADs in this setting are still under way.

RECOMMENDATION

10. We suggest that catheter ablation can be considered, in selected patients, as first-line suppressive therapy, in addition to β-blocker therapy, for patients with ischemic cardiomyopathy (previous MI) and monomorphic VT (Conditional Recommendation, Low-Quality Evidence).

Values and preferences. Catheter ablation, using an endocardial approach, has a low risk of complications and good efficacy for monomorphic VT suppression in ischemic cardiomyopathy. Furthermore, it avoids the side effects and long-term risks of AAD therapy.

Practical tip. First-line catheter ablation should be performed by skilled teams at centres that routinely perform VT ablation.

RECOMMENDATION

11. We recommend catheter ablation of monomorphic VT in patients with ischemic cardiomyopathy (previous MI) in whom treatment with sotalol or amiodarone has been ineffective (Strong Recommendation, High-Quality Evidence).

Values and preferences. Catheter ablation has good efficacy at suppressing monomorphic VT in this population, and has a low risk of procedural complications.

Practical tip. After failure of amiodarone treatment, catheter ablation appears to be much more effective than escalation of AAD therapy (increasing the AAD dose, switching from sotalol to amiodarone, or combination AAD therapy).

8.1.2. Nonischemic cardiomyopathy. Nonischemic cardiomyopathies include a heterogeneous group of cardiac diseases with heterogeneous myocardial scar distribution. There are limited data regarding the relative efficacy of catheter ablation or AAD therapy in such patients.[50] Long-term success rates for ablation of monomorphic VT appear more modest compared with those in patients with ischemic cardiomyopathy (Supplemental Table S4), with a frequent need for epicardial ablation.[51,52] Accordingly, there is a higher threshold for the use of catheter ablation in this population.

RECOMMENDATION

12. We recommend catheter ablation of monomorphic VT in patients with nonischemic cardiomyopathy in whom treatment with sotalol or amiodarone has been ineffective (Strong Recommendation, Low-Quality Evidence).

Values and preferences. The reduced efficacy of catheter ablation, and the increased complexity of the procedure in patients with nonischemic cardiomyopathies led to the recommendation that catheter ablation should be considered second-line in this population.

Practical tip. Catheter ablation in patients with nonischemic cardiomyopathy should be performed by skilled teams at centres that routinely perform VT ablation, including experience with epicardial ablation.

8.1.3. Epicardial mapping and ablation. Percutaneous epicardial access facilitates mapping and ablation of epicardial myocardial substrate, but is associated with added risks. Epicardial mapping should be considered in patients in whom previous endocardial catheter ablation has failed and an epicardial substrate is suspected.[53-56] Epicardial mapping during the first ablation procedure should be reserved for patients with ARVC or dilated cardiomyopathy, who commonly have an epicardial VT substrate.[57-61]

8.2. Complications

Catheter ablation of VT in patients with SHD is a complex procedure performed in a vulnerable patient. The procedure carries significant risks, which can be minimized with experience, technique, and patient optimization. In contemporary trials, reported procedural complication rates have been between 3% and 6%.[31,34,47,49,62] One analysis of administrative data suggested an acute complication rate of 9.9% and in-hospital mortality of 1.8%.[63]

9. ICD Programming in Patients With Sustained VT/VF in the Setting of SHD

The goals of ICD programming are to ensure appropriate therapy for VT/VF, to minimize inappropriate shocks, to minimize symptoms from VT/VF, and to prevent mortality (Table 2). Secondary goals include avoidance of arrhythmia induction and of nonessential therapies.[64,65] ICD shocks are associated with increased mortality, hospitalization, and health care costs compared with ATP therapy alone.[64,66]

In patients with VT/VF, prolonged detection times reduce inappropriate therapy and nonessential appropriate therapy, with no increase in mortality or arrhythmic syncope.[65,67,68] Numerous trials support the use of ATP programming for fast VT (188-250 beats per minute).[69-71]

10. Suppression of VT/VF When Initial Therapy Is Ineffective (Second- and Third-Line Therapy)

Initial suppressive therapies for VT/VF in patients with SHD are sotalol, amiodarone, or catheter ablation. After failure of one of these therapies, a trial of an alternate first-line therapy should be undertaken. Amiodarone and catheter ablation have similar efficacy after failure of sotalol treatment.[34] A significant proportion of patients will have recurrent VT/VF despite first-line therapy, and alternate strategies must be considered.

10.1. Second- and third-line antiarrhythmic therapy

10.1.1. Mexiletine. Mexiletine, a class I AAD with properties similar to I.V. lidocaine, has been reported in small cohort studies to have some efficacy in combination with amiodarone.[72,73] Nevertheless, the additional use of mexiletine with high-dose amiodarone (> 300 mg daily) is inferior to catheter ablation.[74] A pre-ICD era randomized trial of mexiletine showed increased mortality when used in patients with heart failure.[75] Thus mexiletine should be used with caution in patients with SHD and heart failure.

10.1.2. Dofetilide (not currently available in Canada). Dofetilide prolongs repolarization and has a risk of QT prolongation and proarrhythmia. However, it as been shown

Table 2. Suggested ICD programming for patients with sustained VT/VF

Zone	Rate cutoff	Detection intervals/time	Therapy
VF	> 250 BPM (240 msec)	Medtronic: 30/40 intervals Abbott: 30 intervals Boston Scientific: 2.5 s Biotronik: 30/40 intervals MicroPort CRM: 20 cycles, 6/8 majority	Shocks with ATP during charge
Fast VT	188-250 BPM (320-240 msec)	Medtronic: 30/40 intervals Abbott: 30 intervals Boston Scientific: 12 s Biotronik: 30 intervals MicroPort CRM: 20 cycles, 6/8 majority	1-4 ATP bursts followed by shocks
Slow VT	10-20 BPM slower than slowest documented VT*	Medtronic: 32-36 intervals Abbott: 30 intervals Boston Scientific: 60 s Biotronik: 30 intervals MicroPort CRM: 30 cycles, 6/8 majority	ATP predominant Shocks are optional and may be omitted for slow VT

ATP, antitachycardia pacing; BPM, beats per minute; CRM, cardiac rhythm management; ICD, implantable cardioverter-defibrillator; VF, ventricular fibrillation; VT, ventricular tachycardia.

*Twenty BPM slower favoured if VT rate ≥ 150 BPM or if amiodarone initiated for the treatment of VT.

to reduce VT/VF and ICD shocks in cohort studies.[76,77] In a cross-over study, dofetilide had efficacy similar to sotalol.[78] However, many patients who responded to one drug did not respond to the other.

10.1.3. Class 1C agents. Class 1C agents might lead to increased mortality risk in patients with ventricular scar/dysfunction (in the absence of an ICD).[79] However, case series have reported successful combination therapy using sotalol and flecainide in patients with ARVC who had preserved left ventricular ejection fraction and refractory VT.[80,81]

RECOMMENDATION

13. We suggest that mexiletine (given in addition to amiodarone) or dofetilide can be used in patients with SHD and refractory VT/VF who are not candidates or in whom therapy with sotalol, amiodarone, or catheter ablation has failed (Conditional Recommendation, Low-Quality Evidence).

Values and preferences. Despite the lack of evidence supporting the use of mexiletine and dofetilide, there are few other therapeutic alternatives in this setting.

Practical tip. Mexiletine has limited efficacy as monotherapy and should be given in addition to amiodarone. Dofetilide can be given as monotherapy.

10.2. Emerging and alternate ablation modalities

Several newer ablation approaches have been used for treatment-refractory monomorphic VT, including needle ablation, bipolar ablation, transvascular ethanol, and stereotactic radiotherapy. Each permits ablation of deep substrate, not easily reached from the endocardium.[82-85]

10.3. Cardiac sympathectomy

There is increasing evidence for the use of bilateral cardiac sympathetic denervation for the acute and long-term management of refractory VT/VF in patients with SHD.[86] This procedure is carried out via thoracoscopic surgery in a single or staged procedure.

RECOMMENDATION

14. We suggest that bipolar radiofrequency ablation, extendable/retractable radiofrequency needle ablation, stereotactic ablative radiotherapy, and sympathectomy may be considered for treatment of VT/VF after failure of one or more standard ablation procedures and after failure of amiodarone therapy (Conditional Recommendation, Low-Quality Evidence).

Values and preferences. Most of these techniques are emerging therapies, with sympathectomy having the most supporting evidence. Despite the limited data supporting these techniques, they might be beneficial in patients with refractory VT/VF.

Practical tip. These techniques should be used, by experienced operators, and preferably in the context of a research protocol.

11. Psychosocial Care of Patients With VT/VF

Studies of the roles of psychosocial factors in the genesis and management of VT/VF are appearing with increasing frequency. Individuals experiencing VT/VF might be susceptible to poor mental health and reduced quality of life as they manage the symptoms of the VT/VF and those of its treatment.

11.1. Preimplantation and stable postimplantation patients

Anxiety and depression are prevalent among patients with an ICD and particularly those who have experienced VT/VF.[87] Preimplantation factors that contribute to this psychological distress include: premorbid psychological distress, ICD concerns, reduced perceived control, and type D (distressed) personality.[88] These factors place the patient at increased risk of complications/difficulties post implantation and at increased risk of mortality. Unfortunately, these symptoms often go undetected and untreated.

Optimal care pathways should include screening and treatment for psychological distress among patients with VT/VF and an ICD to safeguard health status. Managing psychological distress while living with an ICD is essential to improve outcomes.

In patients with VT/VF and SHD, special attention should be directed to reducing ICD shocks, because they are a significant cause of anxiety due to anticipation of shocks and associated pain.[89] The fear of shocks can have as much or more psychological effect on a patient as an actual shock.[90]

RECOMMENDATION

15. We recommend frequent systematic assessment of psychological status in all patients with SHD and VT/VF, and recommend referral for treatment of such distress when identified (Strong Recommendation, Low-Quality Evidence).

Values and preferences. The psychological effect of VT/VF and its therapies is substantial and can affect all facets of a patient's life. This effect might not be fully appreciated in routine care discussions and needs to be explicitly evaluated.

Practical tip. Simple screening tools, such as the **P**atient **H**ealth **Q**uestionnaire-**2** (PHQ-2) and the **G**eneralized **A**nxiety **D**isorder-**2** (GAD-2) questionnaire are an effective way to screen psychological status (see the *Psychological Status Screening Tools* section of the Supplementary Material). If either of these are positive (score $\geq$ 3), a more in-depth assessment of psychological status is warranted.

11.2. Special populations; ICD generator change in frail/elderly patients

Patients might develop new or worsening illness subsequent to their initial ICD implantation and their goals of care might

change to favour comfort over longevity. A significant proportion of these patients might be unaware of the option of deactivation of tachycardia therapy. Furthermore, they might overestimate the potential benefits of the ICD near the end of life, particularly as the risk of nonarrhythmic death increases (such as in advanced heart failure).[90]

11.3. End of life

The management of VT/VF at the end of life can be associated with significant patient and family distress. Clinicians need to ensure that patients are aware that deactivation of ICD tachyarrhythmia therapies is an option at any time, and is not akin to euthanasia,[91] and will not lead to immediate death.[92] These conversations are best carried out before ICD implantation, continued on a regular basis as part of routine device care, and then revisited at times of significant clinical decline.[93]

ICD deactivation near the end of life can potentially minimize physical and psychological distress. Although patients might choose to maintain ICD therapies near the time of death, it is important to ensure that such patients have made well informed decisions in line with their goals of care.[94]

RECOMMENDATION

16. In patients with VT/VF, we recommend ongoing incorporation of patient values and preferences in goals of care discussions, including ICD tachycardia therapy deactivation or ICD replacement with a pacemaker, particularly at times of ICD generator replacement or changes in clinical status (Strong Recommendation, Low-Quality Evidence).

Values and preferences. Despite the lack of systematic evidence supporting goals of care discussions, the importance of these discussions is paramount for patient-centred care.

Practical tip. Incorporating discussion of goals of care and ICD deactivation into routine device clinic standard of care promotes enhanced patient understanding of end of life options and facilitates patient decision-making.

12. Conclusion/Future Directions/Knowledge Gaps

Implantable defibrillators have dramatically modified the prognosis and the management of ventricular arrhythmias in the presence of SHD. Further study is needed to minimize sudden death risk in the population, to understand when and which arrhythmia-suppressive therapy is best, and to understand the short-and long-term clinical outcomes of available therapies, as well as their effects on mortality, cost-effectiveness, and quality of life.

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Please see Supplemental Table S1 for a complete list of disclosures.

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Supplementary Material

To access the supplementary material accompanying this article, visit the online version of the *Canadian Journal of Cardiology* at http://www.onlinecjc.ca and at https://doi.org/10.1016/j.cjca.2020.04.004.