Sustained Ventricular Tachycardia: A Review of Treatment and Prognosis.

Abstract
Sustained ventricular tachycardia is a ventricular rhythm greater than 100 bpm usually lasting more than 30 seconds. It manifests with a broad QRS tachyarrhythmia which has a similar QRS configuration. This happens from one beat to another, showing a similar chain of ventricular depolarization for every beat. Ventricular tachyarrhythmia has its origin from a stable focus. However, in conditions like structural cardiac disease, the substrate is the place that has patchy replacement fibrosis because of infarction which may originate functional reentry or anatomical pathways. Symptoms of VT rely on the underlying heart function, and rate of arrhythmia. The prognosis depends on the existing heart disease and the first treatment always follows advanced cardiac life support.

Keywords: Coronary artery disease, tachycardia, hypotension, presyncope, syncope, cardiac arrest, amiodarone, radiofrequency catheter ablation.

Introduction
Over time, much literature has explored the various treatment modalities used in sustained ventricular tachycardia in different demographics and their long-term effects on health. Different studies conducted have explored the overall expected development of the disease including improvement and deterioration in the signs and symptoms with respect to time, expectations of quality of life, and the possibility of complications and related health problems [1,3-9]. Certain factors including route of administration, cost of medication and health service, and patient medication adherence have greatly affected the overall outcome of certain treatment options. Sustained ventricular tachycardia refers to a ventricular rhythm faster than 100 beats per minute usually lasting a minimum of 30 seconds or requiring intervention earlier due to hemodynamic instability. It often results in presyncope, syncope, hypotension, and cardiac arrest [2]. Treatment is an effort to address a health issue, usually in response to a diagnosis. It can range from medical treatment involving the use of enteral or parenteral medication to surgical interventions. Several studies have done extensive research on the medical treatment of sustained ventricular tachycardia which revealed high efficacy levels in class III potassium-channel blockers like amiodarone in the medical treatment of recurrent sustained ventricular tachycardia [3]. However, amiodarone a class 3 antiarrhythmic drug normally very effective in the prevention of life-threatening arrhythmias of sustained ventricular tachycardia was found to be very efficacious in the treatment of recurrent sustained ventricular tachycardia but its use was complicated by frequent recurrence and various side effects [4-6]. Radiofrequency catheter ablation is highly effective in the treatment of sustained ventricular tachycardia associated with coronary artery disease with a subsequent complete remedy of recurrent ventricular tachycardia reported in over half of the test subjects, although prospective longitudinal data concerning the long-term effects of ablation related to ventricular tachycardia control and related mortality is insufficient [7-9].

Aim of study
This study aimed to analyze the hemodynamics, ECG features, underlying disease, mode of termination, and outcome of patients presenting with VT.

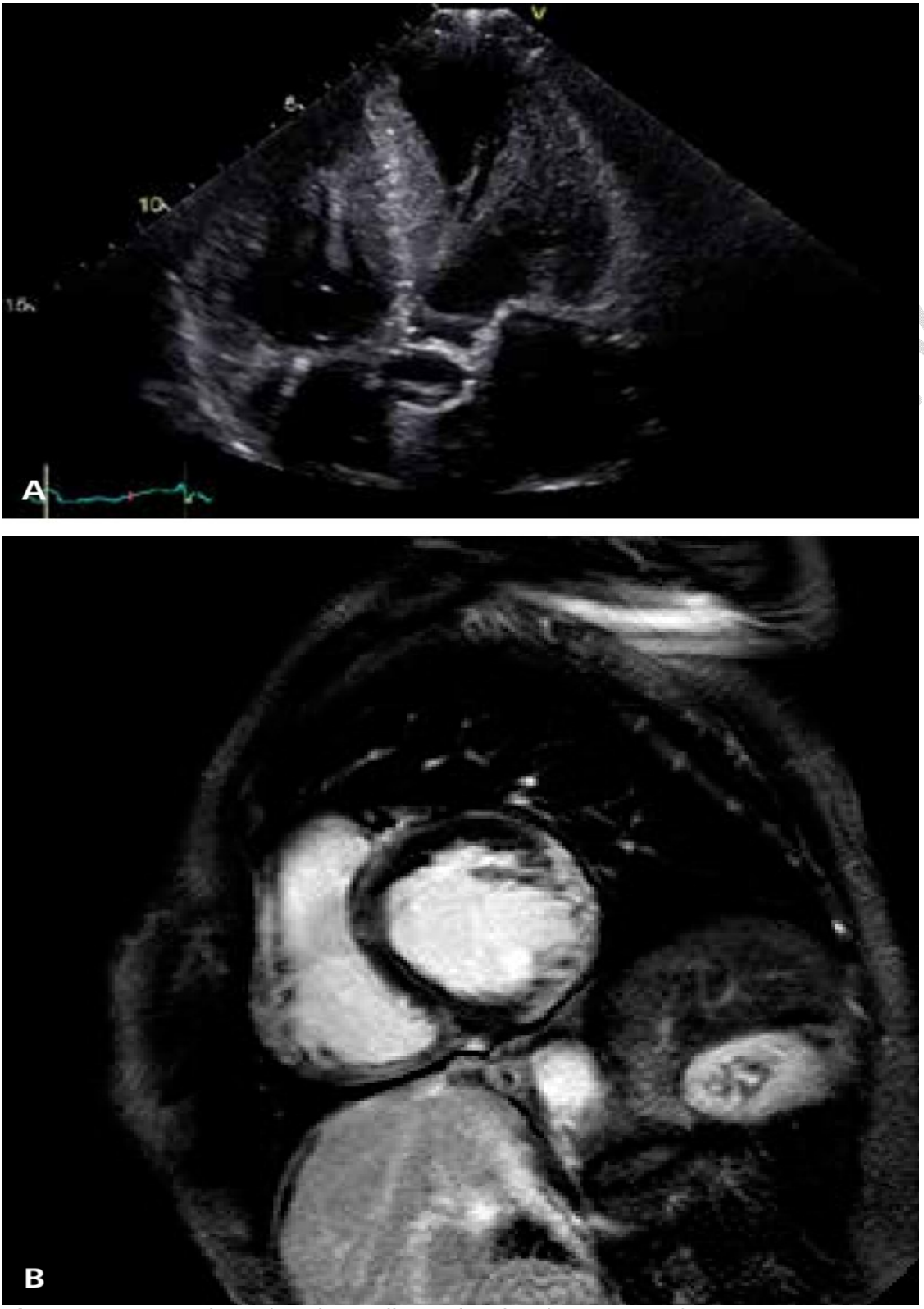


Figure 1. A. Transthoracic echocardiography showing severe eft ventricular hypertrophy. **B.** Late gadolinium enhanced magnetic resonance imaging showing widespread septal enhancement

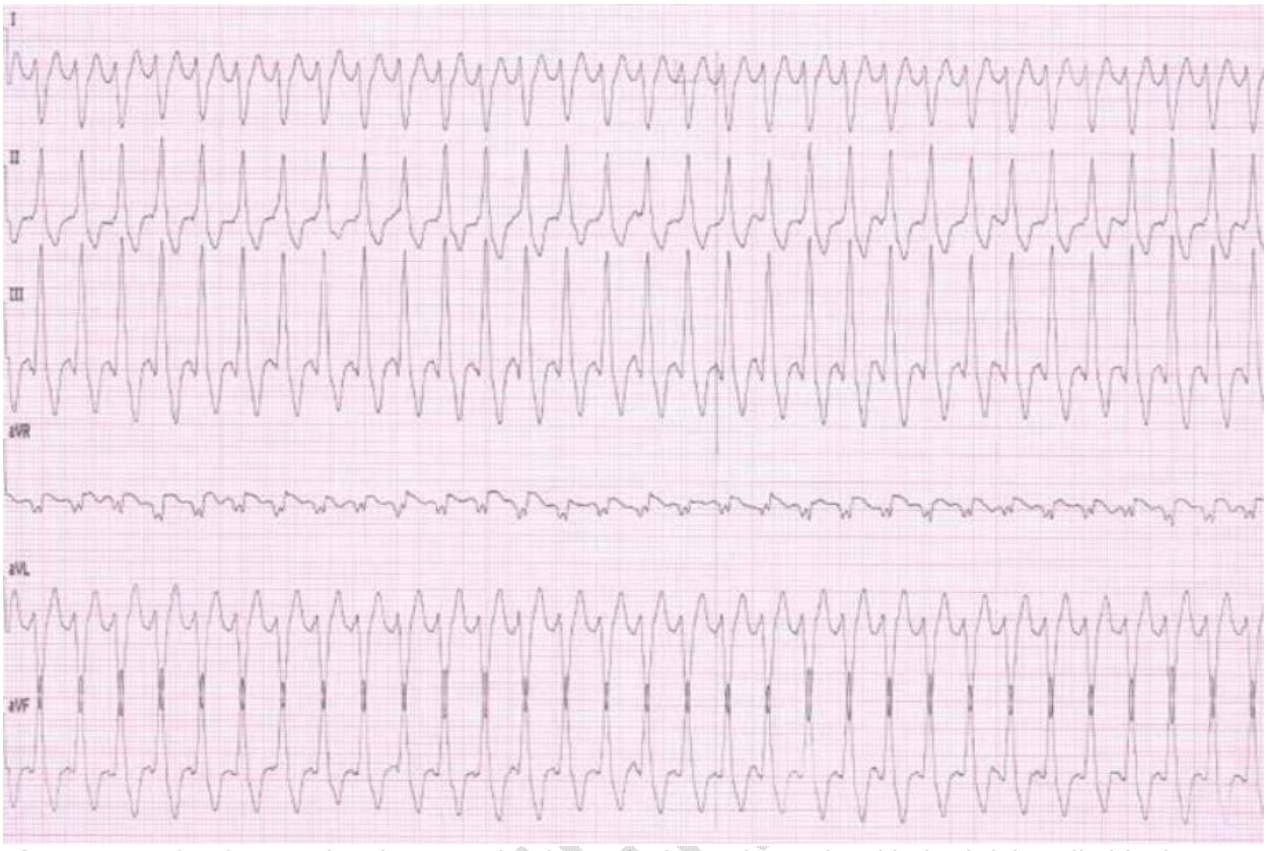


Figure 2. 12-lead ECG showing sustained ventricular tachycarda with the left bundle block morphology

Review

A wide complex tachyarrhythmia with a heart rate greater than 100 beats per minute is referred to as ventricular tachycardia. It is classified by duration as non-sustained or sustained. Non-sustained ventricular tachycardia is described as more than 3 ventricular-originated beats lasting less than 30 seconds at a rate greater than 100 beats per minute [10]. It is referred to as sustained ventricular tachycardia if the rhythm persists for more than 30 seconds or if hemodynamic instability occurs in less than 30 seconds [10]. The predominant cause of death continues to be cardiovascular disease [10,11]. Most sustained ventricular arrhythmias are to blame for the abrupt nature of around half of all cardiovascular fatalities [12,13]. The most frequent underlying heart illness from which sustained monomorphic ventricular tachycardia (SMVT) and ventricular fibrillation (VF) occur is coronary artery disease (CAD), mainly a history of myocardial infarction (MI). Patients with more severe MI and lower left ventricular ejection fraction are more likely to develop sustained, monomorphic VT [14]. People who have other cardiac diseases, like nonischemic idiopathic dilated cardiomyopathy, Chagas disease, sarcoidosis, arrhythmogenic cardiomyopathies, or corrected congenital heart disease, can also have sustained, monomorphic VT [14-16]. In the absence of structural heart disease, SMVT can also occur (e.g., idiopathic left ventricular [LV] tachycardia, outflow tract ventricular arrhythmias), but seldom results in death. Patients without underlying structural cardiac disease seldom develop sustained VT, which can cause syncope or sudden death. A basic structural cardiac abnormality, such as a history of MI, cardiomyopathy, valve disease with fibrosis (scarring), or ventricular enlargement, is present in the majority of individuals with this form of high-grade ventricular ectopy (i.e., lengthy runs of ventricular tachycardia). Coronary artery disease with a prior MI is the most typical cause of sustained, recurrent monomorphic VT in American adults

[17]. However, due to significant improvements in MI therapy, which have led to smaller infarct scars, it has been estimated that the overall incidence of prolonged VT following MI has decreased to 1% from the formerly accepted range of 3% to 5% [18]. Certain patients are still at significant risk of experiencing life-threatening relapses of persistent VT or VF, despite receiving medication. An implantable cardioverter defibrillator (ICD) has been created specifically for these patients to give an internal electric shock to the heart during a life-threatening tachycardia [19]. Most important components of sustained VT are discussed in this essay. The primary management follows advanced cardiac life support. However, if there is a decrease in blood pressure, altered state of consciousness, or pulmonary edema, synchronized cardioversion has to be done, preferably when the patient is conscious [20]. Adenosine is beneficial in stable tachycardia because it may clarify supraventricular tachycardia with aberrancy, it is the first drug to be considered if cardiovascular disease is present [18-20]. Clinical assessments and hospitalization to decide underlying heart pathology are necessary, following the re-establishment of sinus rhythm. Through evaluation of cardiac enzymes for proof of myocardial infarction, however, acute myocardial infarction hardly leads to SVT, and an increase in CK-MB or troponin is more likely to indicate myocardial damage which is secondary to low blood pressure and ischemia from ventricular tachycardia [19-21]. Subsequent management relies on the underlying cardiac disease and frequency of ventricular tachycardia. Administration of antiarrhythmic drugs or catheter ablation is required to restore stability if ventricular tachycardia occurs frequently [19,20]. Usually, sustained monomorphic ventricular tachycardia occurs as an isolated episode, but with a risk of recurrence. Implantable cardiovascular defibrillators are normally warranted for sustained ventricular tachycardia associated with structural heart disease [21]. The prognosis of ventricular tachycardia relies on the etiology and cardiac status. Patients who develop ventricular tachycardia can suffer from hemodynamic failure and the mortality can exceed 30% if no treatment is provided [18]. In the setting of percutaneous coronary intervention, ventricular tachycardia occurring before revascularization is associated with very high mortality [21].

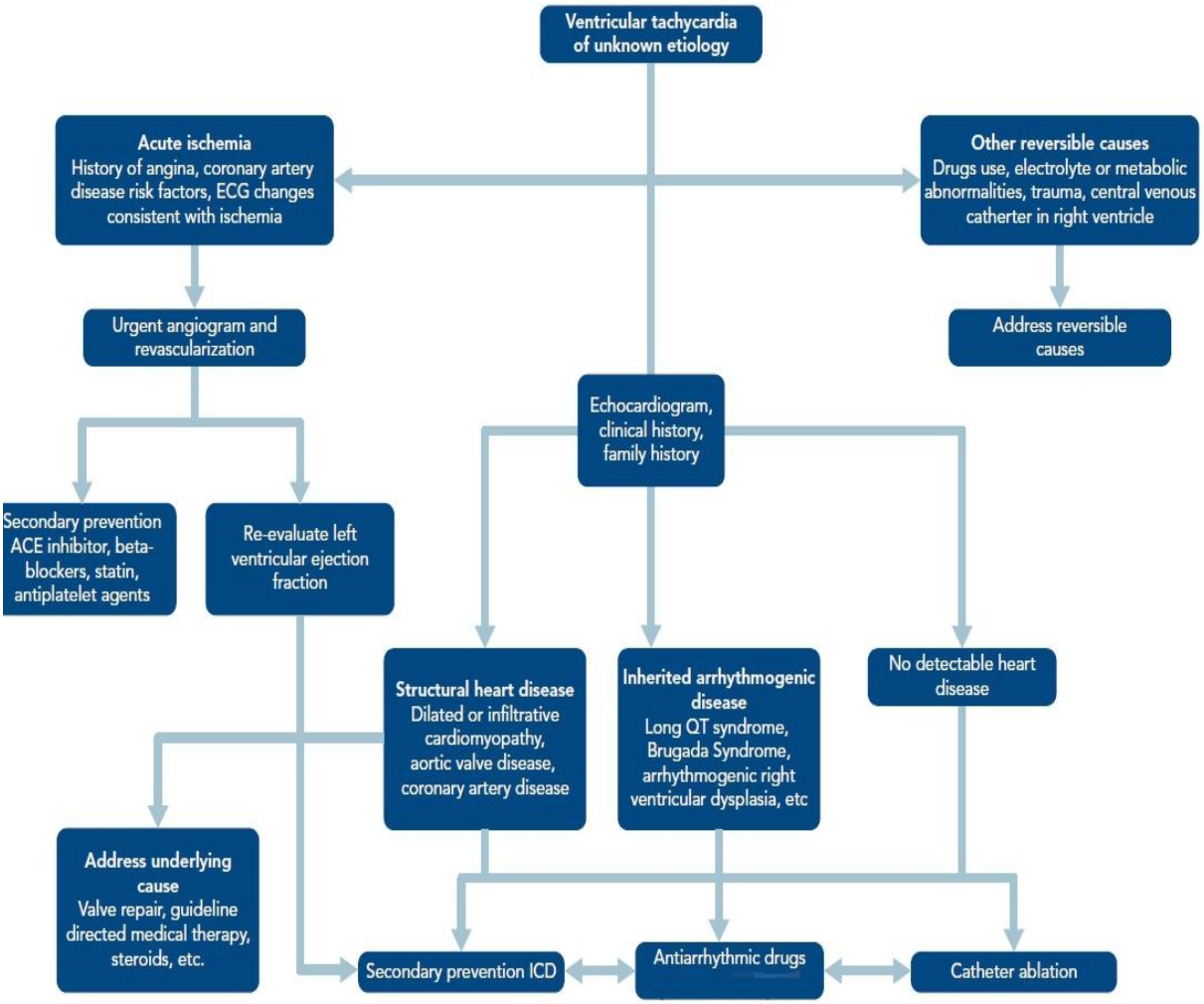
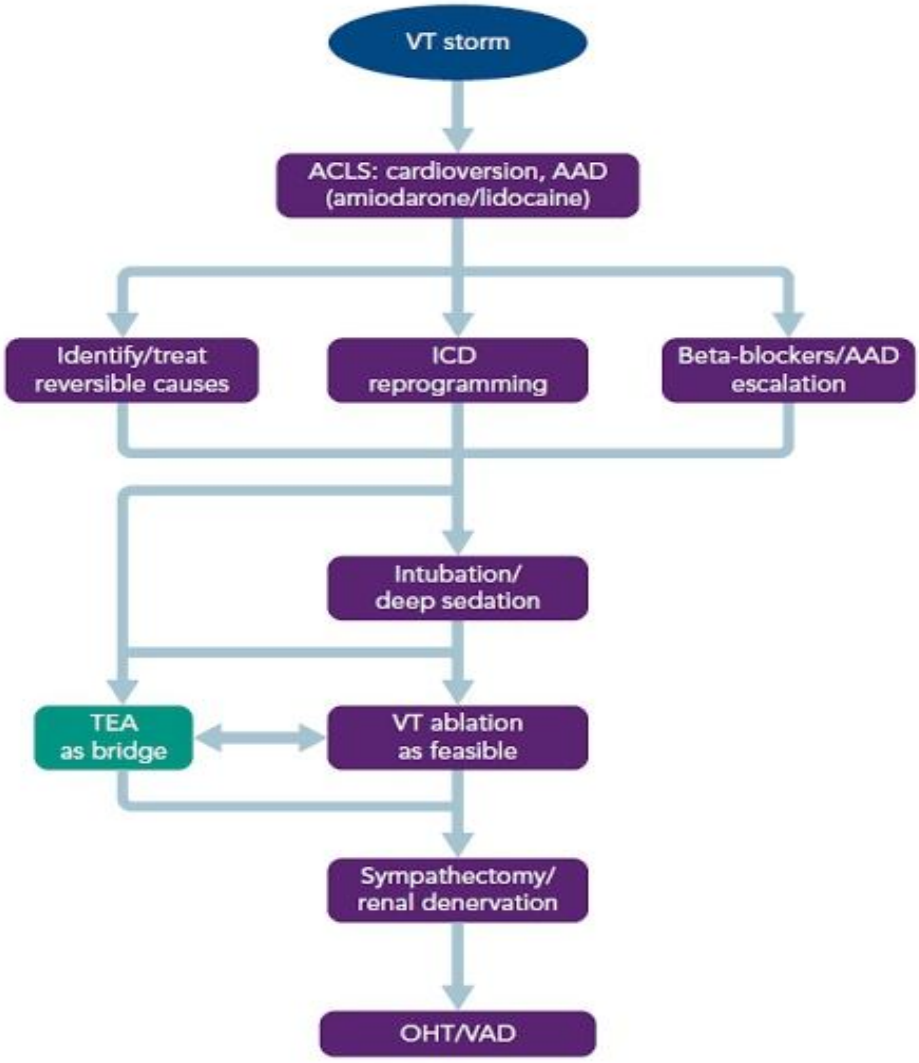


Figure 3: Ventricular Tachycardia Management Algorithm [22]

	Route	Common dosing	Contraindications or warnings	Important Adverse Effects
Beta-blocker	PO or IV	Varies by individual agent	Severe bradycardia or heart block without a pacemaker, decompensated heart failure, Prinzmetal's variant angina	Hypotension, sinus bradycardia, AV block, bronchospasm, fatigue, depression, sexual dysfunction
Amiodarone	PO or IV	PO: 400–1200 mg daily in divided doses IV: 150 mg bolus; 0.5–1.0 mg/min infusion	Severe bradycardia or heart block without a pacemaker, decompensated heart failure, prolonged QT interval	Hypo- and hyper-thyroidism, pulmonary fibrosis, hepatotoxicity, bradycardia, QT interval prolongation, photosensitivity and skin discoloration, corneal deposits, neuropathy
Lidocaine	IV	1.0–1.5 mg/kg bolus, repeat 0.5–0.75 mg/kg every 5–10 min as needed up to 3 mg/kg; 1–4 mg/min infusion	Severe bradycardia or heart block without a pacemaker, Wolf–Parkinson–White syndrome	Gastrointestinal disturbances, bradycardia, hypotension, agitation, seizures, dizziness, altered sensorium, dysarthria, psychosis
Mexiletine	PO	450–900 mg daily	Severe bradycardia or heart block without a pacemaker, reduced left ventricular ejection fraction or heart failure, inherited long QT syndrome (except long QT syndrome 3)	Gastrointestinal disturbances, bradycardia, hypotension, tremor, dizziness, dysarthria
Procainamide	PO or IV	PO: 1000–4000 mg daily in divided doses IV: 100–1000 mg load; 2–6 mg/min infusion	Severe bradycardia, heart block, or intraventricular conduction delay without a pacemaker, coronary artery disease, reduced left ventricular ejection fraction or heart failure, hypotension, Brugada syndrome	Drug-induced lupus, rash, myalgia, bone marrow suppression, vasculitis, bradycardia, hypotension, QT interval prolongation
Sotalol	PO or IV	PO: 160–320 mg in divided doses IV: 75–300 mg twice daily	Severe bradycardia or heart block without a pacemaker, decompensated heart failure, Prinzmetal's variant angina, prolonged QT interval	Hypotension, sinus bradycardia, AV block, bronchospasm, fatigue, depression, sexual dysfunction, QT interval prolongation

Figure 4: Antiarrythmic Drugs for the Treatment of Ventricular Tachycardia [22]



AAD = antiarrhythmic drugs; ACLS = advanced cardiac life support; OHT = orthotopic heart transplant; TEA = thoracic epidural anaesthesia; VAD = ventricular assist device; VT = ventricular tachycardia.

Figure 5: An overview of management for patients presenting with VT storm

Conclusion

Sustained VT is a ventricular rhythm faster than 100 bpm typically lasting at least 30 seconds or requiring termination earlier due to hemodynamic instability. At a rate of 100 bpm or more, it is a broad complex tachycardia (QRS 120 milliseconds or larger) that originates from one of the ventricles and is not brought on by abnormal conduction (such as from bundle branch block). "Idiopathic" VT occurs in the absence of apparent structural heart disease (e.g., prior myocardial infarction, active ischemia, cardiomyopathy, valvular disease, arrhythmogenic right ventricular cardiomyopathy, cardiac sarcoid, left ventricular noncompaction, or other disorders of the myocardium), known channelopathy (e.g., long QT syndrome, Brugada syndrome, catecholaminergic polymorphic VT, short QT syndrome), drug toxicity, or electrolyte imbalance. VT can be classified as either monomorphic or polymorphic. Torsades de pointes is a polymorphic VT with a distinctive twisting morphology that occurs when the QT interval is prolonged. Although sustained VT frequently causes hypotension and symptoms of weakness,

syncope, or palpitations, the arrhythmia can also exist in patients with no symptoms or normotension.

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