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BEST CLINICAL PRACTICE: EMERGENCY MEDICINE MANAGEMENT OF STABLE MONOMORPHIC VENTRICULAR TACHYCARDIA

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☐ **Abstract—Background: Ventricular tachycardia (VT) and ventricular fibrillation are the causes of approximately 300,000 deaths per year in the United States. VT is classified based on hemodynamic status and appearance. Stable, monomorphic VT treatment is controversial. Objective: Our aim was to provide emergency physicians with an evidence-based review of the medical management of stable, monomorphic VT. Discussion: Stable, monomorphic VT is part of a larger class of ventricular dysrhythmias defined by a rate of at least 120 beats/min with QRS > 120 ms without regularly occurring P:QRS association. Little controversy exists for the treatment of hemodynamically unstable VT. The medical management of hemodynamically stable monomorphic VT is surrounded by controversy. Direct current cardioversion is most efficacious. Guidelines for the treatment of stable VT from the American Heart Association provide a IIa recommendation for procainamide, compared with a IIb recommendation for both amiodarone and sotalol. Studies evaluating procainamide, lidocaine, amiodarone, and sotalol suffer from poor design, difference in inclusion and exclusion criteria, small sample size, and outcome determination. Procainamide demonstrates the greatest efficacy. If procainamide is selected, a maximum dose of 10 mg/kg at 50–100 mg/min intravenous (IV) over 10–20 min should be provided with monitoring of blood pressure and electrocardiogram. Monomorphic VT with acute myocardial ischemia requires further study. Conclusions: Optimal management of stable, monomorphic VT includes direct current cardioversion. If medical management is chosen, procainamide is most efficacious, though current literature suffers from poor design. Published by Elsevier Inc.**

☐ **Keywords—ventricular tachycardia; dysrhythmia; procainamide; amiodarone; electrocardiogram; wide complex tachycardia**

INTRODUCTION

Ventricular dysrhythmias are challenging, as they are unpredictable and potentially lethal. Ventricular tachycardia (VT) and ventricular fibrillation (VF) are the causes of approximately 300,000 deaths per year in the United States. One prospective study found a sudden death incidence of 53 per 100,000 people over 1 year (1). This accounted for 5.6% of all mortality in the population. The true incidence is difficult to quantify, as VT and VF overlap and patients may not die from the dysrhythmic event; however, VT is considered the most common regular wide complex tachycardia (WCT), followed by supraventricular tachycardia with aberrant conduction (2–6). One study found 178 patients with VT among 82,559 visits during a 2-year period, which approximates 7.4 visits per month, though this is greater than the majority of studies (7). VT is associated with patients with known cardiac ischemia, male sex, and older age. Causes of VT include ischemic heart disease, structural heart disease (cardiomyopathy), congenital heart disease, channelopathy, electrolyte derangements, sympathomimetic agents, digitalis toxicity, and infiltrative cardiomyopathy (2–6,8).

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This rhythm is part of a larger class of regular WCTs, defined by a rate > 120 beats/min and QRS complex > 120 ms on electrocardiogram (ECG). The differential diagnosis of WCT consists of supraventricular tachycardia (SVT) with aberrant ventricular conduction, sinus tachycardia with aberrant conduction, pre-excited tachycardia in patients with Wolff-Parkinson-White syndrome, ventricular tachycardia, toxic/metabolic derangement, pacemaker-related WCT, and artifact (3–6). It is thought that VT accounts for 80% of regular WCTs, with close to the remaining 20% SVT or sinus tachycardia with aberrant conduction. Two studies evaluating patients with WCT referred to an electrophysiologist found 80%–85% were diagnosed with VT (9,10). However, the majority of these studies were conducted in patients evaluated in a cardiology setting, outpatient or in-hospital, and not the emergency department (ED).

VT is defined by any rhythm > 120 beats/min that originates distal to the bundle of His in the ventricular myocardium external to the actual conduction system. Due to the conduction system, VT will have a wide QRS interval > 120 ms with no regularly occurring P:QRS association (3–6). VT is defined in this manner for the rest of this article. The site of origin may include the ventricular myocardium, distal conduction system beyond the bundle of His, or both. The majority of patients with VT possess an area of fibrotic tissue in the myocardium with small islands of normal, viable tissue (3–6,10). This scar usually results from coronary artery disease with prior myocardial infarction (MI) or ischemic cardiomyopathy. A small minority of patients may have normal myocardium with no structural disease (10,11).

The classification of VT is based on several factors: electrocardiographic (ECG) appearance, duration, and, most importantly, patient hemodynamic status. Monomorphic VT refers to a wide complex rhythm with QRS > 120 ms that is identical from beat to beat, originating from a single focus with no P:QRS association. Polymorphic VT is a rhythm that varies from beat to beat. Nonsustained VT occurs for <30 s, while sustained VT lasts >30 s (3–5,12). Finally, an unstable patient demonstrates evidence of hemodynamic compromise, including hypotension, altered mental status, chest pain, or heart failure, but is awake with a pulse. If the patient is unresponsive and pulseless, immediate defibrillation is required. A stable patient shows no signs of hemodynamic compromise (3–6,13).

DISCUSSION

The medical management of ventricular tachycardia is controversial, specifically for stable, monomorphic VT. Multiple options exist, and expert recommendations have undergone multiple modifications during the past several decades.

Review of VT Management Guidelines

The 2000 American Heart Association (AHA) guidelines for Advanced Cardiac Life Support (ACLS) recommended procainamide or sotalol (class IIa recommendation) over the use of amiodarone or lidocaine (IIb) in the setting of stable, monomorphic VT with preserved ejection fraction (EF) (13). However, in patients with decreased EF, amiodarone and lidocaine received class IIb recommendations, with no recommendation for procainamide or sotalol. Amiodarone's increased popularity was demonstrated in the American College of Cardiology/AHA/European Society of Cardiology 2006 modifications, where amiodarone received an upgrade to class IIa recommendation for stable VT resistant to procainamide (14,15). Per the AHA algorithm, amiodarone was the only antidysrhythmic medication for use (15). The European resuscitation guidelines recommend amiodarone for treatment of stable, monomorphic VT as well (16). In 2010, further modifications were made by the AHA, where procainamide was given a IIa recommendation compared with a IIb recommendation for both amiodarone and sotalol (17). The 2015 update does not change the recommendations for procainamide and amiodarone (18). Direct-current cardioversion is the most effective therapy, supported by numerous studies (4,6,18–20).

Management of Unstable VT

The management of patients with unstable monomorphic VT, as defined here, warrants immediate synchronized direct-current cardioversion. However, pulseless VT or unstable polymorphic VT is treated with defibrillation. If recurrent VT occurs, additional electrical cardioversion is required. A patient with shock-resistant unstable VT should receive amiodarone 300 mg IV with a second bolus of 150 mg IV per the 2015 ACLS guidelines (4,6,15–18,20).

Management of Stable VT

Hemodynamically stable patients, defined by adequate end-organ perfusion with no symptoms, should be approached by first evaluating whether the VT is monomorphic or polymorphic. Of note, QT interval baseline cannot be assessed during an episode VT, therefore, assessment of QT baseline should occur on an ECG obtained before the VT if possible or after conversion. If polymorphic with normal QT interval, treatment is the same as monomorphic VT (18,20). Polymorphic VT often will terminate, but may recur. Prolonged QT with