

# Ventricular Tachycardia: Recognition and Management for The Internist

Indranill Basu-Ray

Cardiac Arrhythmia Service, Divn. of Cardiology, Massachusetts General Hospital, Harvard Medical School, Harvard University, 55 Fruit Street, GRB 109, Boston, MA, USA 02114

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## A B S T R A C T

Cardiac arrhythmias particularly Ventricular Tachycardia often present as urgent medical conditions requiring immediate care. Patient presenting with a ventricular tachyarrhythmia is a common finding in the emergency room. They also occur commonly in patients undergoing non-cardiovascular procedures including surgeries. It is thus pertinent that the physician handling such cases must be appropriately trained to diagnose and provide emergency management till the case is referred to a specialist. Most cases present as narrow or a wide complex tachycardia. The differential diagnosis is arrived at by deciding on the ECG morphology along with relevant history and physical examination where feasible. This article describes the diagnosis and management of ventricular tachycardia in primary care and the emergency room settings.

### INTRODUCTION

Ventricular tachycardias (VT) are urgent medical conditions requiring immediate care. In the primary care settings it is often more important to first judge the hemodynamic effects of the arrhythmia rather than its actual nature. Thus the first step is the assessment of the vital signs, to differentiate between tachycardia with or without hemodynamic instability. The arrhythmia may be the primary cause of hemodynamic instability, or a concomitant symptom of another serious condition. In the case of hemodynamic instability at presentation, or at any time during the treatment of cardiac tachyarrhythmias, electrical cardioversion should be immediately considered as a life saving measure. Cardioversion is a safe and effective therapy for almost all tachyarrhythmias and certainly VTs. History taking and a comprehensive physical examination is important but can be done later after the patient stabilizes. If the patient does not require immediate cardioversion, a thorough physical examination along with relevant history taking is mandated along with a 12 lead ECG. The ECG interpretation should always be made in the light of history and physical examination. In the haste to treat the arrhythmia itself, concomitant treatment of the underlying cause and precipitating factors, as well as supportive care should not be delayed. Pharmacological antiarrhythmic therapy can be considered if the patient is hemodynamically stable, and/or electrical cardioversion is not feasible, desirable or successful. To determine the appropriate therapy, the next step would be to decide the nature of the abnormal arrhythmia seen.

### DIAGNOSIS

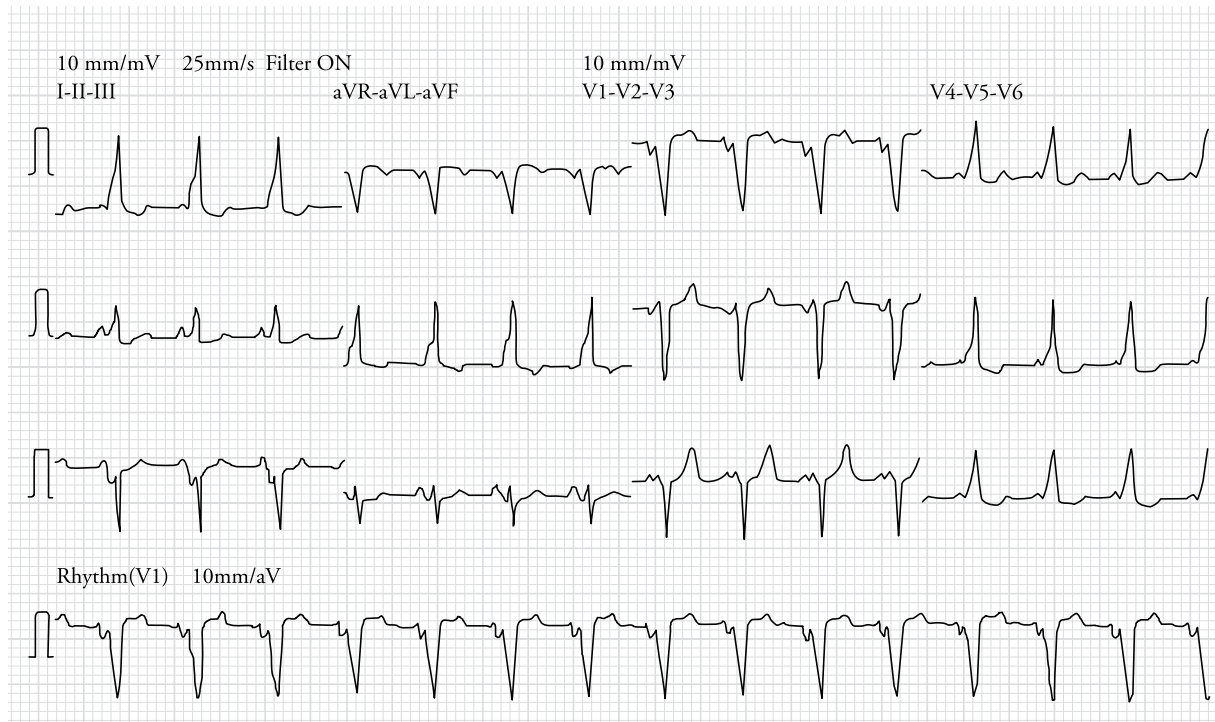
VT can present either with a narrow (NCT: QRS duration <120 msec) or wide complex tachycardia (WCT: QRS duration  $\geq$  120 msec). Certain features when present supports VT rather than supraventricular tachycardia (SVT) and these are outlined below:

#### Certain features which when present supports VT

- I. presence of fusion beats
- II. presence of capture beats
- III. AV dissociation
- IV. P and QRS rate and rhythm linked to suggest that atrial activation depends on ventricular discharge; e.g., 2:1 VA block.
- V. Concordant patterns: all the precordial leads have predominantly positive or negative QRS pattern.
- VI. Extreme Left axis deviation is more likely to be VT. Axis in "no man's land" (right superior i.e. between  $-90$  to  $\pm 180$ ) is almost always VT.
- VII. QRS duration  $> 140$  ms (RBBB QRS configuration) or  $> 160$  ms (LBBB QRS configuration) is suggestive of VT.
- VIII. Presence of Q waves indicates presence of old myocardial infarction and thus VT is the likely diagnosis.

### DIFFERENTIAL DIAGNOSIS

In the differential diagnosis of a wide complex tachycardia the dictum is to consider it as VT if one is not sure of its actual



**Fig. 1:** Sinus rhythm in a patient with pre-excitation demonstrating accessory pathway conduction with a short PR interval and a delta wave at the onset of the QRS complex. Antidromic AVRT using the accessory pathway for antegrade conduction.

nature as wide complex tachycardias are more often VTs than otherwise. However certain SVTs can present as WCT. These are discussed below:

### SVT with aberrancy

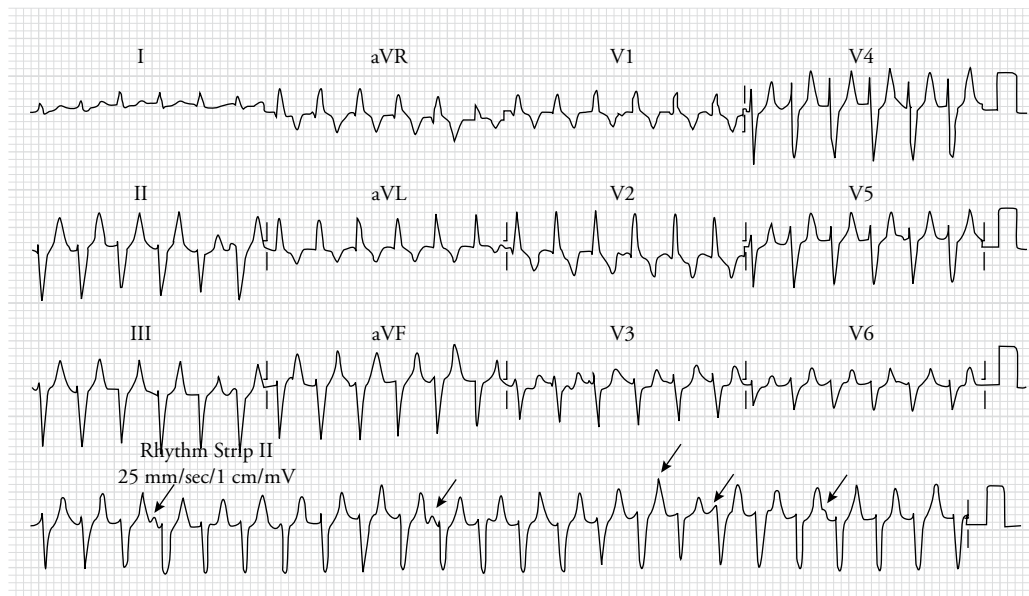
Any supraventricular tachycardia can present as a wide QRS complex tachycardia mimicking VT. In AVNRT, atrial tachyarrhythmias, atrial flutter with a fixed A:V relationship and AVRT involving a retrograde concealed accessory pathway, wide QRS complex tachycardia is the result of delayed or blocked conduction over a portion of the His-Purkinje system (bundle branches). If the tachycardia is known with certainty to be supraventricular in origin, it can be treated as a narrow QRS complex tachycardia.<sup>1</sup> In some cases it can prove challenging to differentiate VT from SVT with aberrancy. It must be remembered in this context that 80% of WCT are VTs and a minority are actually SVTs. Points that distinguish a VT are tabled below. However it is important to remember that at the bedside if it is difficult to differentiate between the two all such rhythms must be taken as VT and treated as such. Far more costly mistakes are made at the bedside by incorrectly diagnosing VT as SVT than conversely by incorrectly diagnosing SVT as VT. An arrhythmia that has been definitively diagnosed as SVT with aberrancy can be treated according to the typical care for that SVT; any WCT that does not have definitive features of SVT should be treated as VT until proven otherwise. It should be noted that underlying heart disease and heart rate during a tachycardia (rather than SVT versus VT) are the prime determinants of the hemodynamic stability of a rhythm. VT can be hemodynamically stable and even asymptomatic if the rate is sufficiently slow, while SVT with very rapid ventricular rate or

in the setting of structural heart disease can cause hemodynamic instability, syncope and even death.

### AVRT with a manifest accessory pathway

AVRT can present with a wide QRS complex, pre-excited tachycardia as a result of manifest anterograde accessory pathway conduction. These patients may have WPW syndrome that can be diagnosed by the resting sinus ECG, based on the delta wave and short PR interval (Fig. 1). It is also possible to have a normal ECG during sinus rhythm if the accessory pathway conduction is slow compared to the AV nodal conduction. Accessory pathways are anomalous extranodal connections which connect the epicardial surface of the atrium and ventricle along the atrioventricular groove. Accessory pathways which are capable only of retrograde conduction are concealed whereas those capable of antegrade conduction are manifest, demonstrating pre-excitation on a standard ECG. The term “Wolf-Parkinson-White syndrome” is reserved for patients who have both pre-excitation (wide QRS in sinus rhythm due to presence of delta wave) and symptomatic tachyarrhythmias. It is important to remember that even amongst patients with the Wolf-Parkinson-White syndrome, when they present with tachyarrhythmia; orthodromic atrioventricular reciprocating tachycardia (AVRT) with a NCT is the most common form, occurring in 75% of these patients.

AVRT is further subclassified into orthodromic and antidromic AVRT. During orthodromic AVRT the re-entrant impulse utilizes the atrioventricular node and specialized conduction system for conduction from the atrium to the ventricle, and utilizes the accessory pathway for conduction from the ventricle to the atrium. During antidromic AVRT the re-entrant impulse



**Fig. 2:** Monomorphic VT. AV dissociation is clearly seen. The p waves marked by arrows are clearly dissociated from the QRS complexes. Note that this VT has a relatively narrow QRS and might be misinterpreted as an SVT if not for the AV dissociation.

travels in the reverse direction with conduction from the atrium to the ventricle occurring via the accessory pathway. Orthodromic AVRT presents with NCT (unless there is aberrant ventricular conduction) whereas antidromic AVRT always produces WCT.

Atrial fibrillation is a potentially life-threatening arrhythmia in patients with the Wolf-Parkinson-White syndrome as it can result in a very rapid ventricular response, and rarely ventricular fibrillation. The incidence of sudden cardiac death in patients with the Wolf-Parkinson-White syndrome has been estimated to be 0.15% per patient year. Therapeutically it is important to identify AF occurring in the substrate of WPW for reasons explained later.

**ECG** Patients with WPW have a delta wave seen in a sinus rhythm ECG (Fig. 1). A wide complex tachycardia is generally seen when these patients present with antidromic conduction over the accessory pathway. Patients with WPW syndrome and atrial fibrillation can present with a broad complex irregular ECG mimicking polymorphic VT. If no previous ECG is present and it is difficult to be certain they should be treated as if they have VT.

### WPW with atrial fibrillation/flutter

This is basically a variant of the above described subgroup but described separately as these are potentially life threatening disorders requiring appropriate urgent management to prevent sudden death. WPW syndrome can be diagnosed by the resting sinus ECG, based on the delta wave and short PR interval. Irregular polymorphic wide QRS complex tachycardia can occur in these patients during atrial fibrillation or atrial flutter. Due to different degrees of preexcitation over the accessory pathway, beat to beat variation in the QRS complex can also be observed, and may be difficult to differentiate from PMVT. The fast ventricular rates can degenerate into ventricular tachycardia or fibrillation, and so WPW syndrome with atrial fibrillation or atrial flutter represents a medical emergency which requires prompt treatment. In patients with preexcited atrial fibrillation or flutter, the use

of AV nodal blocking agents (beta-blockers, calcium channel blockers, adenosine, and digoxin) should be avoided as they can lead to an acceleration of accessory pathway conduction, leading to an increase in the ventricular rate with potentially disastrous consequences. Procainamide and amiodarone (as well as flecainide, propafenone and sotalol) directly prolong the accessory pathway conduction, therefore slowing the ventricular response. Electrical cardioversion is always a safe option if one is not sure and a resting ECG is not available. Patients with WPW and atrial fibrillation once resuscitated need to be referred to an electrophysiologist for ablation of the abnormal tract as they remain susceptible to sudden cardiac death as long as the pathway is intact and functioning.

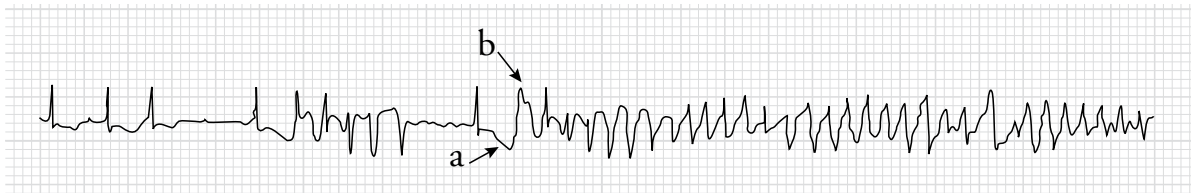
### Wide QRS complex tachycardia of uncertain origin

In patients with stable wide QRS tachycardia of uncertain origin, adenosine and lidocaine previously were recommended. This recommendation has recently been changed. Before giving any anti-arrhythmic medication, attempts should be made to determine the origin of the tachycardia. Signs of AV dissociation on the ECG, esophageal ECG, and the clinical characteristics (age, structural heart disease, history of WPW syndrome or BBB) can help in making a diagnosis.<sup>2</sup> Wide QRS complex tachycardia is most often due to VT, especially in the setting of structural heart disease. The most costly misdiagnosis is diagnosing VT as SVT with aberrancy. It is generally recommended to err in favor of VT than otherwise.

If electrical cardioversion is not feasible, desirable, or successful, iv procainamide or iv amiodarone are recommended.

### Ventricular Tachycardia

Acute ventricular tachycardia is a syndrome with diverse etiologies. There have been many classifications of ventricular tachycardia depending on the site of origin, etiology and the morphology of the ECG complexes produced. In this article we shall categorize VT based on morphology as monomorphic VT, polymorphic



**Fig. 3:** The typical initiation of a sequence of Torsade. a-note the long QT and b-long short initiating cycles with twisting QRS complexes typical of TdP.

VT with long QT, and polymorphic VT with normal QT. This type of classification has been found to be clinically very useful in managing these arrhythmias particularly in the emergency room settings. These ECG diagnoses are usually accompanied by typical clinical characteristics, and allow a focused treatment of the ventricular arrhythmia.

Pharmacological treatment can be considered in cases of hemodynamically stable sustained VT, especially if electrical cardioversion is not available, or desirable, or if the ventricular tachycardia recurs, despite electrical cardioversion.

### Monomorphic ventricular tachycardia

Monomorphic ventricular tachycardia is most frequently due to scar related reentry, typically in the setting of coronary artery disease, prior MI, and left ventricular dysfunction (Fig. 2).

Less frequently encountered (10% of sustained VT) is idiopathic ventricular tachycardia. This typically occurs in the setting of a structurally normal heart, and often responds to beta-blocker or non-dihydropyridine calcium channel blocker therapy. VT arising from the right or left ventricular outflow tract is especially common in this setting; clues to its diagnosis include VT in the setting of a structurally normal heart and tall R waves in the inferior leads. Other monomorphic VTs (bundle branch reentry, right ventricular dysplasia associated VT) should be treated acutely similarly to scar related VT.

### TREATMENT

In a patient with sustained hemodynamically unstable VT, electrical cardioversion is highly effective and recommended therapy<sup>3</sup>. Hemodynamically stable VT can first be approached with IV medications but with constant monitoring of the patient and with equipment immediately available for electrical cardioversion if the VT becomes unstable or does not respond to medical therapy. The risk of immediate recurrence after successful cardioversion is variable but is low to intermediate in most settings. The drugs commonly used to treat hemodynamically stable VT's are individually discussed.

#### Lidocaine

Intravenous lidocaine can be rapidly infused with minimal hemodynamic effects, and has been used for decades for the termination of sustained monomorphic VT. However its success rate in VT termination is approximately 15%, and there is no randomized, controlled studies to support its efficacy.<sup>4,5</sup> It is no longer recommended as first line therapy for the treatment of monomorphic VT.<sup>3</sup> Despite this recommendation, lidocaine continues to be used as first-line therapy with much vigor in clinical practice.

#### Procainamide

Intravenous procainamide has a success rate of 80-90% in termination of monomorphic VT.<sup>6</sup> It is administered as a slow infusion, and hypotension is a common side effect; it may not be effective in preventing recurrences.<sup>7</sup> Presence of CHF or poor LV function precludes its use.

#### Amiodarone

Intravenous amiodarone was tested in three randomized, controlled, double-blind studies in patients with electrical storm. Patient with recurrent VT/VF refractory to lidocaine and procainamide were included. Two of the studies compared different doses of amiodarone, while one study compared amiodarone to intravenous bretylium. The investigators found amiodarone to be as effective as bretylium, and to have significantly fewer adverse effects, requiring drug discontinuation.<sup>8-10</sup> Based on these findings amiodarone is recommended in the treatment of hemodynamically unstable, recurrent VT.

Intravenous amiodarone has not been tested in the treatment of hemodynamically stable VT. Extrapolating from the data available, it is a reasonable and safe therapy, especially in the setting of CHF, and poor LV function, when procainamide is not recommended.

### IRREGULAR POLYMORPHIC WCT

#### Polymorphic ventricular tachycardia with normal QT

Polymorphic ventricular tachycardia with a normal QT interval is most frequently caused by acute ischemia or myocardial infarction. It usually is initiated during sinus tachycardia, with a premature ventricular beat with a short coupling interval. It is usually poorly tolerated and tends to degenerate into VF quickly. Rarely, polymorphic ventricular tachycardia with a normal QT interval is caused by arrhythmogenic right ventricular cardiomyopathy, idiopathic polymorphic ventricular tachycardia ("short-coupled variant of torsade de pointes") or familial catecholaminergic polymorphic VT.

#### Polymorphic ventricular tachycardia with long QT

Torsade de pointes is a type of ventricular arrhythmia that is classically accompanied by certain characteristics including QT prolongation. The initiation of the tachycardia is pause-dependent, with a late coupled PVC (long-short initiating sequence). The tachycardia is frequently non-sustained, and patients may present with history of syncope. On the resting ECG long QTc and abnormally shaped T waves are characteristic. QT prolonging drugs, bradycardia, hypokalaemia and hypomagnesaemia are usually the precipitating factors (Fig. 3).

## TREATMENT

### Cardioversion

Sustained polymorphic VT is almost always an unstable rhythm with hemodynamic compromise and frequent degeneration to VF. Electrical cardioversion is generally the first line of therapy for sustained PMVT, with subsequent therapy as described below intended to reduce the risk of recurrence or as treatment for non-sustained PMVT.

### Beta-blockers

A recent non-randomized study followed 49 patients with recent MI and electrical storm. One group of 27 patients received sympathetic blockade (21 in the form of beta-blocker therapy, and six as left ganglion stellate block) and the other group of 22 patients was treated with lidocaine, epinephrine, with or without bretylium and procainamide, as recommended by the ACLS guideline. One week mortality was 82% in the 'ACLS guided' group, while in the sympathetic blockade group the mortality was 22%.<sup>11</sup>

Based on these and other studies, beta-blockers are recommended by most authorities for polymorphic VT with normal QT, especially, if ischemia is suspected or is the etiology.<sup>12</sup> Cardioversion is called for if VF is precipitated or if there are any signs of hemodynamic compromise.

### Other therapies

**Magnesium:** Although only case reports support its efficacy, intravenous magnesium is a recommended treatment for Polymorphic ventricular tachycardia with long QT.<sup>3</sup>

**Other:** Discontinuation of the precipitating drug, repletion of potassium (to 4.5-5 meq/L), temporary pacing (especially if PMVT is bradycardia or pause-related) with or without adjunctive beta-blockade, or isoproterenol (if temporary pacing is not available) are recommended.<sup>9,11</sup>

## VENTRICULAR FIBRILLATION

The most frequent etiology of ventricular fibrillation is coronary artery disease. Primary VF in the setting of acute MI can occur. However, scar related monomorphic ventricular tachycardia can also degenerate into VF, and so sudden cardiac death can occur outside the setting of an acute coronary event.

Shock resistant ventricular fibrillation (VF) is defined as ventricular fibrillation persisting after three defibrillation attempts. In approximately 10 to 25% of all cardiac arrests shock resistant VF develops. Importantly, up to 60% of patients suffer recurrent VF after initially successful defibrillation.<sup>13</sup> VF needs to be treated with immediate cardioversion. However it is important to understand the role of pharmacological agent's in the treatment of this fatal disorder. Antiarrhythmic drugs have played an important role despite the primary therapy being electrical. They have been used to improve defibrillation efficacy, and/or prevent ventricular fibrillation recurrence.

### Amiodarone

The ARREST (Amiodarone in the Resuscitation of Refractory Sustained ventricular Tachyarrhythmias) trial randomized patients with out-of-hospital cardiac arrest to receive amiodarone

or placebo.<sup>14</sup> Patients were eligible if ventricular fibrillation or pulseless ventricular tachycardia was present after receiving three or more shocks. The primary endpoint of survival to hospital admission, which was achieved in 44% of the patients in the amiodarone group versus 34% in the placebo group ( $p=0.03$ ). The proportion of patients who survived to hospital discharge did not differ in the two treatment groups; 13.4% in the amiodarone and 13.2% in the placebo group.

The ALIVE (Amiodarone versus Lidocaine in Prehospital Ventricular Fibrillation Evaluation) trial was a randomized study comparing amiodarone to lidocaine in patients with out-of-hospital ventricular fibrillation.<sup>15</sup> Patients were eligible if ventricular fibrillation was present after receiving 4 or more shocks. After treatment with amiodarone, 22.8% survived to hospital admission, as compared with 12% in the lidocaine group. The proportion of patients who survived until hospital discharge was 5% in the amiodarone group and 3% in the placebo group ( $p=0.34$ ). It should be noted that neither trial was powered to demonstrate a difference in survival to hospital discharge.

The ARREST and ALIVE studies represented the first instance of any proven benefit from a pharmacological antiarrhythmic intervention in randomized trials of cardiac arrest. Based on this data, an amiodarone bolus of 300 mg is recommended after the third shock in the treatment of refractory ventricular fibrillation.<sup>16</sup>

### Magnesium

Magnesium has been used for decades to treat arrhythmias without strong evidence. Two randomized, double blind, placebo controlled studies have tested its efficacy in out-of-hospital ventricular fibrillation refractory to three countershocks.<sup>17,18</sup> These studies failed to demonstrate any short or long-term benefit of the administration of magnesium. Based on this data, magnesium is no longer recommended routinely in refractory VF.<sup>16</sup>

### Beta-blockers

In the absence of a contraindication, beta-blockers should be given to all patients with AMI and unstable angina. Cardiogenic shock, severe congestive heart failure, bradycardia and hypotension are considered contraindications, and therefore beta-blockers are conventionally contraindicated for the treatment of VF during resuscitation.<sup>16</sup> However, in case of successfully treated ventricular fibrillation in the setting of acute myocardial infarction, or recurrent polymorphic VT accompanied with normal QT interval, beta-blockers are recommended.<sup>16</sup> Beta-blockers can be considered in shock refractory VF if other therapies fail, especially if myocardial ischemia or infarction are likely to be present, although there is stronger evidence in favor of amiodarone in this setting

## CONCLUSION

Monomorphic wide QRS complex tachycardia should be considered as ventricular tachycardia unless there is definite evidence of SVT, and treated with procainamide or amiodarone if stable or electrical cardioversion if unstable. Polymorphic wide QRS complex tachycardia should be classified, before any pharmacological treatment, into the subgroups of VT with normal QT, VT with long QT or atrial fibrillation with WPW

syndrome. The treatment in the first group is beta-blocker, in the second group magnesium, and procainamide or amiodarone in the last group, respectively. In regard to emergent management of tachycardia in the ER, it is best to consider any WCT to be VT until proven otherwise, and to be prepared for electrical cardioversion with any tachycardia which exhibits significant hemodynamic instability. Definitive identification of the precise mechanism of a tachycardia can always wait until the patient has been stabilized.

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