Diagnosis of ventricular tachycardia

Ventricular tachycardia has three typical features: a rate greater than 100 bpm, a wide QRS (generally >120 ms), and dissociation with atrial activity. In its non-sustained form (<30 seconds), it may go unnoticed, or be associated with transient palpitations or pre-syncope. Its importance lies in its sustained forms where it can lead to pulmonary congestion, syncope and cardiogenic shock. In addition to a tachycardia, clinical findings may include hypotension, a raised jugular venous pressure with cannon A waves, and a variable intensity of S1. *Figure 1* describes some of the common terms used in association with ventricular tachycardia.

Causes of ventricular tachycardia

Myocardial scar

Following an acute myocardial infarction, infarcted tissue is replaced by fibrous scar. Fibrous tissue is electrically inactive and forms an area of fixed conduction block. However, within the scar, bundles of viable tissue can occasionally be found that are still electrically active, known as conduction channels (Peters and Wit, 1998). These channels can form a closed loop electrical circuit with a limb of healthy tissue around the scar, resulting in a re-entrant ventricular tachycardia (Stevenson et al, 1993). Ventricular tachycardia here tends to be monomorphic – the QRS morphology is the same from beat to beat. These conduction channels are targeted in scar-related ventricular tachycardia ablation.

Scar formation is not exclusive to the post-infarct patient. Ventricular arrhythmia can be seen in young adults with congenital heart disease as an unintended consequence of scar formation post-corrective cardiac surgery. This is often a cause of tragic sudden death in this patient population (Gatzoulis et al, 2000). Scar-related ventricular tachycardia is also seen in several forms of non-ischaemic cardiomyopathy, including dilated cardiomyopathy, hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy and cardiac sarcoidosis (Wissner et al, 2012).

Acute myocardial ischaemia

Patients presenting with an acute coronary syndrome can experience ventricular arrhythmia. However, this is not associated with scar formation, but rather accelerated impulse formation (known as enhanced automaticity) within a region of ischaemic ventricular tissue (Riley and Marchlinski, 2008). Ventricular tachycardia here tends to be polymorphic – there is variation in the QRS morphology between each beat.

Bundle–branch re-entry

Severe ventricular dilatation and dysfunction causing slowing within the conduction system may permit re-entry within the His–Purkinje system itself, causing an uncommon type of rapid ventricular tachycardia known as bundle–branch re-entry ventricular tachycardia. There is often partial or complete left bundle–branch block on the resting electrocardiogram and the close similarity of the re-entry 12-lead electrocardiogram QRS to that seen during tachycardia may give rise to the erroneous diagnosis of supraventricular tachycardia with aberrant conduction.

Dr Sukhjinder S Nijjer, Consultant in Interventional Cardiology, Hammersmith Hospital, Imperial College Healthcare NHS Trust, London

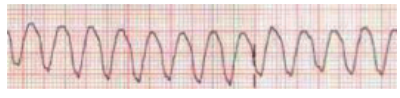
Dr Vishal Luther, BHF Clinical Research Fellow and Cardiology Specialist Registrar, Augustus Waller Department of Electrophysiology, Hammersmith Hospital, Imperial College Healthcare NHS Trust, London W12 0HS

Dr David C Lefroy, Consultant in Cardiology and Cardiac Electrophysiology, Augustus Waller Department of Electrophysiology, Hammersmith Hospital, Imperial College Healthcare NHS Trust, London

Correspondence to: Dr V Luther (v.luther14@imperial.ac.uk)

Figure 1. Definitions.

Ventricular tachycardia: a broad-complex tachycardia with discrete QRS complexes on the electrocardiogram.



There are a number of associated terms:

Pulsed ventricular tachycardia: ventricular tachycardia that is able to produce a cardiac output – may still require a synchronised shock

Pulseless ventricular tachycardia: ventricular tachycardia that causes cardiac collapse with no pulse and requires immediate defibrillation

Non-sustained: three or more beats but lasts <30 seconds

Sustained: lasts >30 seconds; may require chemical or electrical cardioversion depending on clinical state

Ventricular tachycardia storm: three or more episodes of sustained ventricular tachycardia over 24 hours, each requiring intervention

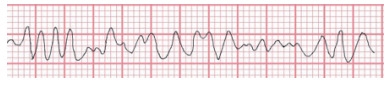
Incessant: sustained ventricular tachycardia that recurs despite intervention (e.g. defibrillation) over several hours.

Monomorphic: all QRS complexes have the same morphology

Polymorphic: QRS complexes that markedly change during the episode of ventricular tachycardia

Torsades de pointes: a special type of polymorphic ventricular tachycardia that is associated with a long QT interval duration. The QRS complexes appear to 'twist around the isoelectric line'

Ventricular fibrillation: a broad-complex tachycardia without a clear pattern of QRS complexes. There is no coordinated mechanical cardiac contraction resulting in a loss of output and cardiac arrest.



Ion channelopathies

QT interval prolongation

QT interval prolongation on 12-lead electrocardiogram is associated with an increased risk of ventricular arrhythmia and sudden cardiac death. This may be acquired (commonly as a result of drugs) or genetic (long QT syndromes caused by sodium or potassium channel abnormalities). Ventricular tachycardia is 'triggered' by early or delayed after depolarizations within the QT interval (Tomaselli et al, 1994), resulting in a form of polymorphic ventricular tachycardia known as torsades de pointes (*Figure 2*). *Table 1* lists common causes of QT interval prolongation.

Brugada syndrome

Brugada syndrome is a cause of ventricular tachycardia and sudden death. It presents a characteristic electrocardiogram abnormality (*Figure 3*) involving a partial right bundle–branch block pattern with ST elevation in V1–V3. The electrocardiogram changes may be intermittent and sometimes only seen when provoked, for instance during a febrile illness, or during diagnostic testing with sodium-channel blockers such as ajmaline or flecainide. The heart is structurally normal but the patient has a high risk of polymorphic ventricular tachycardia, syncope and sudden death. In some patients it is inherited, and mutation of the cardiac sodium channels has been identified (Gussak et al, 1999).

Catecholaminergic polymorphic ventricular tachycardia

Catecholaminergic polymorphic ventricular tachycardia is another genetic defect associated with mutation in the ryanodine receptor. In this condition, polymorphic ventricular tachycardia can be induced in situations of high adrenergic stimulation, including exercise (Laitinen et al, 2004). In some patients it is inherited, and mutation of cardiac sodium channels has been identified.

Idiopathic ventricular tachycardia

Ventricular tachycardia occurring in the absence of any underlying structural or electrical disease is termed idiopathic. This form of ventricular tachycardia is considered to carry a more benign prognosis than other types of ventricular tachycardia. There are two common forms of idiopathic ventricular tachycardia, which can be distinguished via their specific electrocardiogram characteristics.

Figure 2. Characteristic short long short initiation sequence triggering torsades de pointes – a 'polymorphic ventricular tachycardia' with a characteristic appearance, 'twisting on its baseline'.

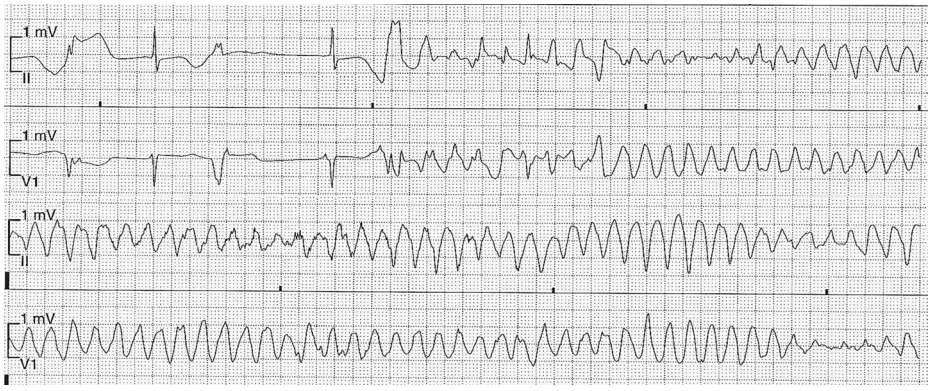


Table 1. Causes of QT interval prolongation

Genetic long QT syndrome (LQTS)	Potassium channel mutations	LQTS 1, 2, 5, 6
	Sodium channel mutations	LQTS 3
	Ankyrin B mutations	LQTS 4
Drug-induced long QT syndrome	Antibiotics	Erythromycin, clarithromycin, ciprofloxacin
	Antifungals	Ketoconazole, cotrimoxazole
	Antidepressants	Amitriptyline, prochlorperazine, citalopram
	Stimulants	Amphetamines, cocaine, ephedrine
	Anti-arrhythmics	Amiodarone, sotalol or quinidine
	Opiate substitutes	Methadone
Other predisposing conditions	Baseline prolongation of QTc	
	Bradycardia	
	Electrolyte abnormalities (hypokalaemia, hypomagnesaemia)	
	Hypothermia (including deliberate cooling on intensive treatment unit)	

Outflow tract ventricular tachycardia

Outflow tract ventricular tachycardia has an inferior QRS axis, with QRS complexes being strongly positive in LII, III and AvF. The tachycardia has a focal origin. The focus most often lies in the right ventricular outflow tract (*Figure 4*), just below the pulmonary valve, but may also lie within the left ventricle, or even outside the heart just above the aortic valve in one of the sinuses of Valsava. Patients may experience repetitive self-terminating bouts of monomorphic tachycardia, and frequently have a high burden of isolated ventricular ectopic beats with a similar QRS morphology (Badhwar and Scheinman, 2007).

Fascicular ventricular tachycardia

Fascicular ventricular tachycardia is positive in V1, typically a left axis deviation, and narrow QRS complex. It is thought to arise from elements of the Purkinje network of the left posterior fascicle.

Idiopathic ventricular tachycardias are highly amenable to curative ablation.

Work up for the patient with ventricular tachycardia

Resting 12-lead electrocardiogram

Q waves indicate completed transmural infarction and underlying myocardial scar which can set up monomorphic ventricular tachycardia. Bundle–branch block might indicate an underlying cardiomyopathy. QT interval prolongation is associated with torsades de pointes. Arrhythmogenic right ventricular cardiomyopathy has a characteristic T-wave inversion between V1–V3, or an epsilon wave. Brugada syndrome can demonstrate coved ST elevation pattern in V1–V2 (*Figure 3*).

Figure 3. Brugada syndrome. There is abnormal and highly characteristic ST elevation with upward convexity and right bundle–branch block pattern seen in V1 and V2.

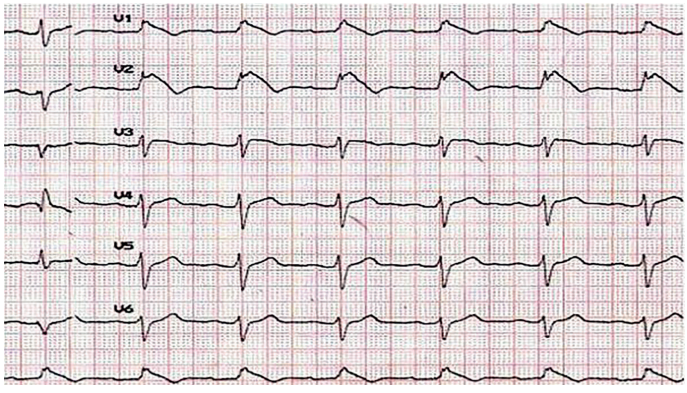
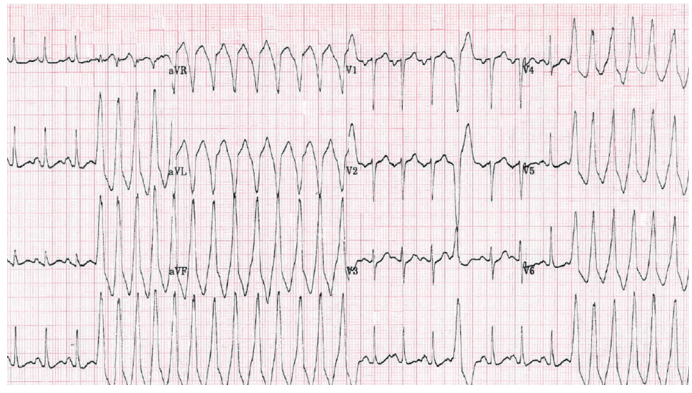


Figure 4. Intermittent runs of ventricular tachycardia (left bundle–branch block pattern seen in V6), inferior axis (positive in LII, LIII, AvF), in keeping with right ventricular outflow tract-ventricular tachycardia.



Blood testing

Significant abnormalities in serum potassium, calcium and magnesium levels can result in changes in ventricular repolarization and arrhythmogenesis.

Imaging

Transthoracic echocardiography is the first-line investigation to define cardiac anatomy. Ejection fraction is also used in the risk stratification for primary prevention via implantation of an implantable cardioverter-defibrillator.

If echocardiography is normal, more detailed imaging using cardiac magnetic resonance imaging can exclude less clearly evident myocardial scar.

Coronary angiogram

As coronary artery disease is the most common cause of ventricular tachycardia, invasive angiography with a view to revascularization is generally considered in all patients above 50 years of age or with risk factors for atherosclerotic coronary artery disease. In addition, it allows identification of coronary vasospasm and the presence of anomalous malignant courses of the coronary arteries.

Exercise testing

Exercise-induced ST depression might indicate myocardial ischaemia. Patients with non-ischaemic cardiomyopathy (e.g. hypertrophic cardiomyopathy) are exercised to assess for induced arrhythmia or abnormal blood pressure response, all of which risk stratify their need for a defibrillator implant. In patients with frequent ventricular ectopy, the suppression of ectopy with exercise generally indicates that their condition is benign. On the contrary, ectopy exacerbated by exercise is suggestive of triggered activity primed by catecholamines. This may be seen in arrhythmogenic right ventricular cardiomyopathy or catecholaminergic polymorphic ventricular tachycardia (Laitinen et al, 2004).

Non-invasive provocative tests

Ajmaline or flecainide are sodium-channel blockers that can induce the phenotypic Brugada pattern in at-risk individuals. Adrenaline infusions can induce ventricular tachycardia in patients with catecholamine polymorphic ventricular tachycardia and also prolong the QT interval in patients with type 1 long QT syndrome (Priori et al, 2013).

Electrophysiological testing

A diagnostic electrophysiology study can be useful in patients who present with syncope or sustained palpitations who have evidence of underlying structural heart disease or a broad complex tachycardia of unclear aetiology. Electrodes can be placed transvenously in the right heart to record local electrograms and pace the heart. A series of ventricular beats faster than the resting heart rate are delivered in the right ventricle to determine whether ventricular tachycardia can be induced and studied.

Interpreting the 12-lead electrocardiogram

Ventricular tachycardia accounts for 80% of all broad complex tachycardias (Akhtar et al, 1988). However, a broad complex tachycardia can also represent:

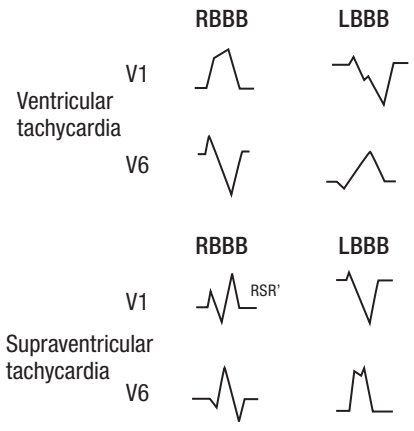
1. Supraventricular tachycardia or atrial flutter with abnormal inter-ventricular conduction
2. Antidromic atrioventricular reentrant tachycardia: this can be seen in patients with an accessory pathway, where ventricular activation travels down the accessory pathway from atria to ventricle, resulting in a broad complex tachycardia
3. Ventricular paced rhythm. This can include pacemaker-mediated tachycardia or ventricular tracking of high atrial rates.

Figure 5 describes features that can help distinguish between ventricular tachycardia and supraventricular tachycardia with abnormal inter-ventricular conduction (Miller et al, 2004).

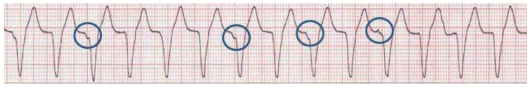
The ventricular paced rhythm is usually obvious, with prominent unipolar pacing spikes marching across the surface electrocardiogram capturing an adjacent ventricular complex. However, in a bipolar set up, pacing spikes are typically not visible – in a dual chamber pacemaker, device interrogation may be required to exclude ventricular tracking of high atrial rates, or a pacemaker-mediated tachycardia. Pacemaker-mediated tachycardia is another form of re-entrant tachycardia set up by a ventricular ectopic beat travelling repeatedly up the atrioventricular node to the atrium and down the pacemaker itself to the ventricle. Atrial arrhythmias (supraventricular tachycardia, atrial flutter) with aberrant conduction can be distinguished from ventricular tachycardia by the manoeuvres detailed in the authors' previous article (Nijjer et al, 2014). Adenosine administration leads to atrioventricular nodal block – it may terminate a supraventricular tachycardia involving the atrioventricular node and demonstrate typical atrial flutter waves, but will generally have no effect on ventricular tachycardia.

Figure 5. Distinguishing between ventricular tachycardia and supraventricular tachycardia with abnormal inter-ventricular conduction.

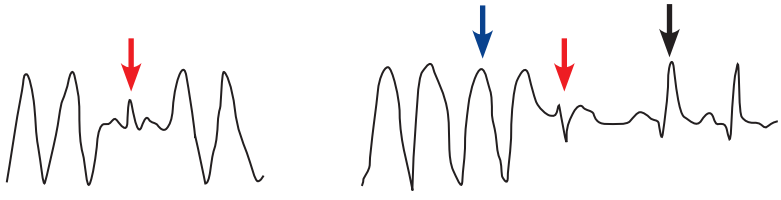
QRS morphology (right): ventricular tachycardia is suggested if the QRS is very broad (>140 ms in right bundle–branch block (RBBB) or >160 ms in left bundle–branch block; LBBB). A typical bundle–branch block pattern is more in keeping with supraventricular tachycardia than ventricular tachycardia – if there is a right bundle–branch block pattern present, an RSR' in V1 is more suggestive of supraventricular tachycardia with aberrant conduction, while a monophasic broad R wave in V1 is suggestive of ventricular tachycardia. If there is a left bundle–branch block pattern present: a broad R wave in V6 suggests ventricular tachycardia.



Atrioventricular dissociation (right): The atria continue to have activity but they are independent of the ventricles. Small p-waves (circled) may be seen hidden within the broad ventricular tachycardia complexes.



Dissociated atrial complexes during ventricular tachycardia (below) may occasionally be conducted to the ventricle to give a 'capture beat' or a QRS morphology that is a fusion of a sinus originating beat and ventricular tachycardia morphology (a 'fusion beat'). These are highly specific for ventricular tachycardia.



Capture beat
Normal QRS within runs of ventricular tachycardia

Fusion beat
QRS with intermediate morphology – it differs from ventricular tachycardia (blue) and normal beats (black)

QRS axis: A frontal axis between 90° and ±180° suggest ventricular tachycardia as do predominantly negative QRS complexes in I, II and III. Extreme left axis deviation suggests ventricular tachycardia. Extreme right axis deviation in the presence of left bundle–branch block also suggests ventricular tachycardia.

Conclusions

Ventricular tachycardia can become pulseless or degenerate into ventricular fibrillation at any point. A history of heart disease such as prior myocardial infarction or heart failure increases the likelihood of a broad complex tachycardia representing ventricular tachycardia. In these patients, all broad complex tachycardias should be considered as ventricular tachycardia until proven otherwise. The second article of this pair (https://doi.org/10.12968/hmed.2017.78.1.C6) reviews the treatment strategies for ventricular tachycardia. **BJHM**

Conflict of interest: none.

KEY POINTS

- Ventricular tachycardia is suggested by a rate greater than 100 bpm, a QRS width >120 ms and dissociation with atrial activity. It can lead to pulmonary congestion, syncope, cardiogenic shock and even sudden death.
- Ventricular tachycardia is caused by focal triggered or re-entrant circuits secondary to scar post myocardial infarction or secondary to underlying cardiomyopathy, acute myocardial ischaemia, electrolyte imbalances and ion channelopathies.
- The underlying cause of ventricular tachycardia can be identified from the 12-lead electrocardiogram, blood testing, cardiac imaging, exercise testing, coronary angiography, provocative tests (ajmaline, flecanide or adrenaline) and electrophysiological studies.
- Ventricular tachycardia can be differentiated from other broad complex tachycardias on the basis of atrioventricular dissociation, QRS morphology and QRS axis. In patients with a history of prior myocardial infarction or heart failure assume it is ventricular tachycardia until proven otherwise.

Akhtar M, Shenasa M, Jazayeri M et al (1988) Wide QRS complex tachycardia. Reappraisal of a common clinical problem. *Ann Intern Med* **109**: 905–12

Badhwar N, Scheinman MM (2007) Idiopath9ic ventricular tachycardia: diagnosis and management. *Curr Probl Cardiol* **32**: 7–43. https://doi.org/10.1016/j.cpcardiol.2006.10.002

Gatzoulis MA, Balaji S, Webber SA et al (2000) Risk factors for arrhythmia and sudden cardiac death late after repair of tetralogy of Fallot: a multicentre study. *Lancet* **356**(9234): 975–981. https://doi.org/10.1016/S0140-6736(00)02714-8

Gussak I, Antzelevitch C, Bjerregaard P, Towbin JA, Chaitman BR (1999) The Brugada syndrome: clinical, electrophysiologic and genetic aspects. *J Am Coll Cardiol* **33**(1): 5–15

Laitinen PJ, Swan H, Piippo K, Viitasalo M, Toivonen L, Kontula K (2004) Genes, exercise and sudden death: molecular basis of familial catecholaminergic polymorphic ventricular tachycardia. *Ann Med* **36** (Suppl 1): 81–86

Miller JM, Das MK, Arora R et al (2004) Differential diagnosis of wide QRS complex tachycardia. In: Zipes DP, Jalife J, eds. *Cardiac Electrophysiology from Cell to Bedside.* 4th edn. Saunders, Philadelphia, PA: 747–757

Nijjer SS, Sohaib SM, Whinnett ZI, Lefroy DC (2014) Management of supraventricular tachycardias. *Br J Hosp Med (Lond)* **75**(2): C26–C28. https://doi.org/10.12968/hmed.2014.75.Sup2.C26

Peters NS, Wit AL (1998) Myocardial architecture and ventricular arrhythmogenesis. *Circulation* **97**: 1746–1754

Priori SG, Wilde AA, Horie M et al (2013) HRS/EHRA/APHRS expert consensus statement on the diagnosis and management of patients with inherited primary arrhythmia syndromes. *Europace* **10**: 1932–1963. https://doi.org/10.1093/europace/eut272

Riley MP, Marchlinski FE (2008) ECG clues for diagnosing ventricular tachycardia mechanism. *J Cardiovasc Electrophysiol* **19**: 224–229. https://doi.org/10.1111/j.1540-8167.2007.01007.x

Stevenson WG, Khan H, Sager P et al (1993) Identification of re-entry circuit sites during catheter mapping and radiofrequency ablation of ventricular tachycardia late after myocardial infarction. *Circulation* **88**: 1647–1670

Tomaselli GF, Beuckelmann DJ, Calkins HG et al (1994) Sudden cardiac death in heart failure. The role of abnormal repolarization. *Circulation* **90**: 2534–2539

Wissner E, Stevenson WG, Kuck KH (2012) Catheter ablation of ventricular tachycardia in ischaemic and non-ischaemic cardiomyopathy: where are we today? A clinical review. *Eur Heart J* **33**(12): 1440–1450. https://doi.org/10.1093/eurheartj/ehs007