BMJ Best Practice

Assessment of tachycardia

Straight to the point of care

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Summary

Tachycardia, generally defined as a heart rate ≥100 bpm, can be a normal physiological response to a systemic process or a manifestation of underlying pathology.[1] [2] The normal heart rate varies with age. The normal sinus rate in infants is 110 to 150 bpm, which gradually slows with age.[3]

Classification of tachyarrhythmia

Several methods of classification of tachyarrhythmia are helpful in organising and assessing tachycardias. These include: sinus versus non-sinus causes; atrial versus ventricular arrhythmias; narrow- versus wide-complex tachycardias; regular versus irregular arrhythmias; and classification based on the site of origin of the arrhythmia.[1]

Sinus versus non-sinus causes

Sinus tachycardia is a common cause of tachycardia that can often be mistaken for an arrhythmia. Diagnosis depends on the P-wave morphology and the setting in which it occurs. Because each impulse originates in the sino-atrial node, the ECG shows a P wave preceding each QRS interval with a normal P-wave axis. In most cases, a secondary cause of sinus tachycardia can be identified. A careful assessment is important, to evaluate whether the sinus rate is appropriate for the clinical situation. Sinus tachycardia can be mistaken for other supraventricular arrhythmias, including atrial flutter, particularly with rapid tachyarrhythmias (when P waves are difficult to distinguish or when ectopic atrial foci originate near the sino-atrial node, such as near the superior vena cava or upper crista terminalis).

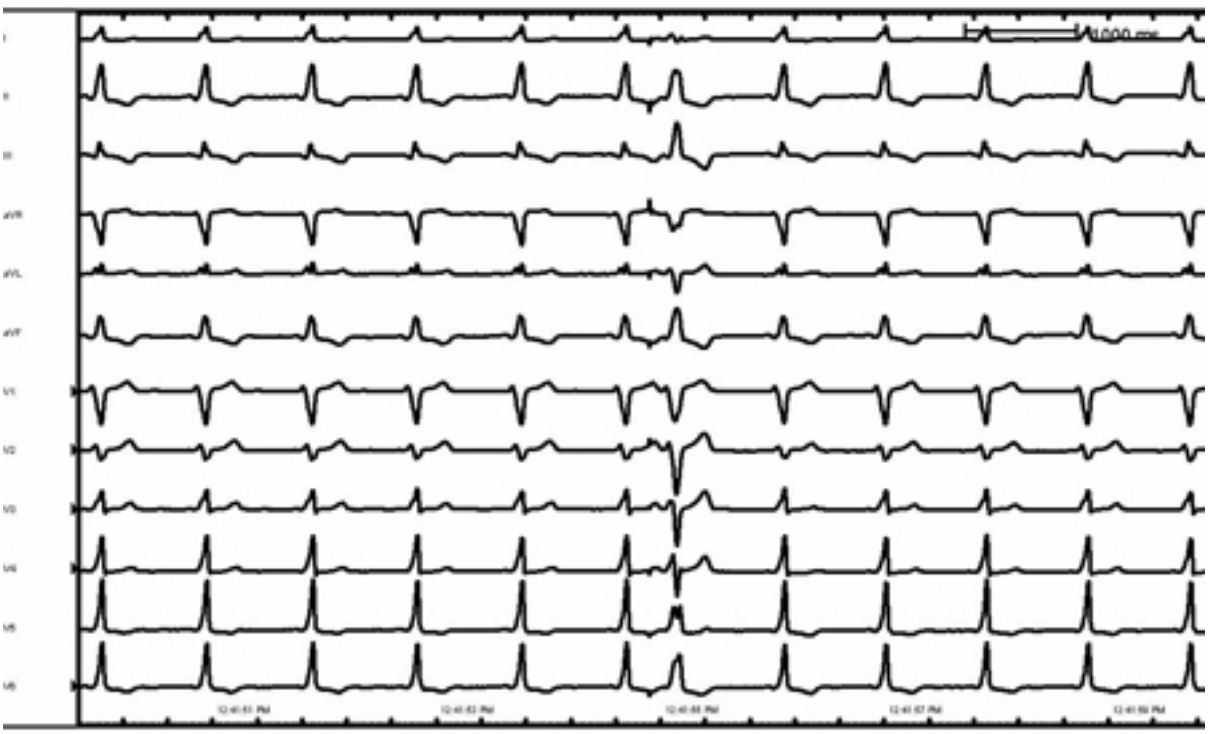
Atrial versus ventricular arrhythmia

Whether the arrhythmia originates from the atrium or the ventricle is usually dependent on whether the QRS complex is wide or narrow, and on the atrial:ventricular relationship. Atrial arrhythmias usually conduct to the ventricle through the His-Purkinje system and result in a narrow QRS complex. Some overlap may occur when conduction occurs with aberrancy (left or right bundle branch block), if there is pre-excitation, and in the presence of anti-arrhythmic agents that may slow conduction (sodium channel blockers). If P waves are discernible, an atrial:ventricular relationship of <1 is highly suggestive of a ventricular origin, whereas a relationship >1 is highly suggestive of an atrial origin. A 1:1 atrial:ventricular relationship can occur with both atrial and ventricular arrhythmias.

Narrow versus wide QRS complex

Classification can also be based on whether there is a narrow- (QRS interval <120 ms) or wide- (QRS interval >120 ms) complex tachycardia. Narrow-QRS-complex tachycardia suggests that anterograde conduction and thus depolarisation of the ventricle occurs through the atrioventricular (AV) node and His-Purkinje system. A wide-complex tachycardia suggests that conduction through the ventricle occurs through the slower myocyte-to-myocyte connections (because of either AV conduction via an accessory pathway or a ventricular origin) and can be seen even with sinus rhythm. However, atrial tachyarrhythmias that conduct aberrantly may present as a wide-complex tachycardia.

OVERVIEW



Sinus rhythm with pre-excitation

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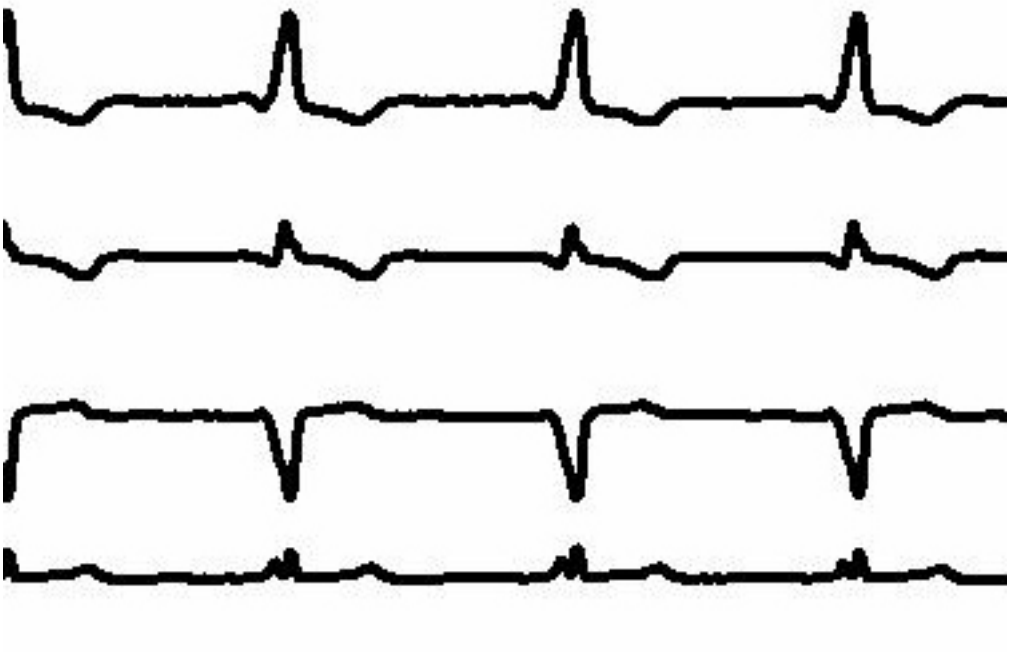

Sinus rhythm with pre-excitation (detail)

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Regular versus irregular rhythm

Whether a rhythm is regular or irregular is easy to determine clinically and can help guide diagnosis of the tachyarrhythmia. An irregular rhythm is defined as a beat-to-beat R-R variability of more than 30 ms. In

general, irregular narrow-complex arrhythmias include: atrial fibrillation, atrial flutter with variable conduction, and multifocal atrial tachycardia. An irregular wide-complex tachycardia may be due to pre-excited atrial fibrillation (due to a rapidly anterograde-conducting bypass tract), polymorphic ventricular tachycardia and atrial fibrillation, or multifocal atrial tachycardia conducting with aberrancy.

Site of origin

Tachyarrhythmias can be classified according to the site of origin: atrial, junctional, or ventricular:

- Atrial impulses are characterised by initial depolarisation of the atrium, from a single focus, such as sinus tachycardia or atrial tachycardia; macro re-entry around an anatomical obstacle, such as in typical atrial flutter; or from multiple wavelets of re-entry, such as atrial fibrillation.
- Arrhythmias that originate at the level of the junction of the atrium and ventricle (AV node and/or proximal His bundle), such as AV nodal re-entrant tachycardia or junctional ectopic tachycardia, are characterised by depolarisation of the ventricle and retrograde atrial activation (if present), manifested by a retrograde P wave.
- Arrhythmias that originate from the ventricle may originate from the distal His-Purkinje system or ventricular myocardium. The site of origin within the ventricle further defines some arrhythmias within the ventricle. Examples include right ventricular out-flow tract ventricular tachycardia and bundle branch re-entry ventricular tachycardia. Haemodynamic stability is not a helpful factor in differentiating atrial and ventricular arrhythmias. Some cases of ventricular tachycardia may be initially well tolerated haemodynamically. Misdiagnosis and inappropriate treatment (i.e., calcium channel blockers) may have catastrophic consequences.

When a patient with a wide-complex tachycardia is being evaluated and the diagnosis is not certain, the arrhythmia must be initially regarded as ventricular tachycardia until it is proven to be otherwise.

Epidemiology

Sinus tachycardia is the most common cause of sustained tachycardia, as it is usually a normal physiological response to emotional or physical stimulation.[4]

Atrial fibrillation is the most common arrhythmia in clinical practice, with an estimated global prevalence of 50 million in 2020.[5] [6][7] Up to 9% of patients aged 80 years or older have atrial fibrillation.[8] [9] As the proportion of older people in the population increases, the number with atrial fibrillation is likely to increase significantly.

In the US, the prevalence of atrial fibrillation is estimated to increase from approximately 5.2 million in 2010 to 12.1 million in 2030.[10] [11] In the European Union, prevalence of atrial fibrillation (in adults aged >55 years) is projected to increase from 8.8 million in 2010 to 17.9 million by 2060.[12]

The incidence of atrial flutter has been reported as 88 cases per 100,000 person-years; it is more common in men, with increasing age, and in those with heart failure or chronic obstructive pulmonary disease (COPD).[13] [14]

PSVT, defined as intermittent supraventricular tachycardia (AV node re-entry tachycardia, AV reciprocating tachycardia, or atrial tachycardia) has an incidence of 57.8 cases per 100,000 person-years and a prevalence of 1.26 million people in the US.[15] Females are twice as likely to develop PSVT, and the incidence is five times greater in people older than 65 years compared with younger people.[16] Most cases of supraventricular tachycardia are due to AV nodal re-entrant tachycardia (60% of cases); the remainder are due to AV reciprocating tachycardia (30%) and atrial tachycardia (10%).[17]

The prevalence of inappropriate sinus tachycardia is not well known and the underlying mechanisms are likely to be multifactorial, but patients are often young (age 15 to 50 years) and female.[1] [2] [18]

The prevalence of ventricular tachyarrhythmia is highly dependent on its type and duration. In patients with a history of previous MI, the incidence of sustained monomorphic ventricular tachyarrhythmia depends on the size of the infarction and the overall left ventricular function.

Aetiology

The aetiology of tachycardia is variable and often multifactorial.

The most common type is sinus tachycardia.

Cases of tachyarrhythmias not due to a heightened sinus rate may be due to focal islets of tissue that fire rapidly because of elevated automaticity, triggered activity, or re-entry within a tissue with heterogeneous conduction properties.

Secondary causes of tachyarrhythmia include ion channelopathies, myocardial scar, surgical scar, elevated atrial or ventricular wall tension and stretch due to elevated filling pressures, ischaemia, electrolyte abnormalities, elevated intrinsic catecholamines, myocarditis, or any combination of these causes. Prescribed, legitimate, and illicit drug use have been implicated.

Narrow QRS (duration <120 ms) with a regular ventricular rhythm

Sinus tachycardia

A rhythm that originates in the sino-atrial (sinus) node with a rate above 100 bpm. This is usually a normal response to physical, emotional, physiological, or pharmacological stress. Secondary causes of sinus tachycardia include physical deconditioning, hypoxia, pulmonary embolism, hypovolaemia, hyperthyroidism, anaemia, drugs (e.g., caffeine, alcohol, nicotine, amphetamines, cocaine), and prescribed medications (e.g., aminophylline, atropine, clozapine, catecholamines).[1] [2] [19]

Postural orthostatic tachycardia syndrome (POTS)

A chronic, multi-system disorder that is thought to be due to an autoimmune process. POTS is characterised by:[20] [21] [22] [23]

- frequent symptoms of orthostatic intolerance (that improve rapidly when the patient returns to a supine position) that interfere with daily living activities, and have continued for at least 3 months, and
- an increase in heart rate by ≥30 bpm (or ≥40 bpm in patients aged 12 to 19 years) within 10 minutes of standing from a supine position or head-up tilt (without orthostatic hypotension) that is not due to other causes of sinus tachycardia.

Inappropriate sinus tachycardia

A persistent increase in resting heart rate unrelated to or out of proportion to physical, emotional, pathological, or pharmacological stress (resting heart rate >100 bpm or average heart rate >90 bpm in 24-hour ECG monitoring). The precise aetiology is unknown and is likely to be multifactorial. Elevated sinus node automaticity and autonomic dysfunction have been proposed as possible causes.[1] [2] [21]

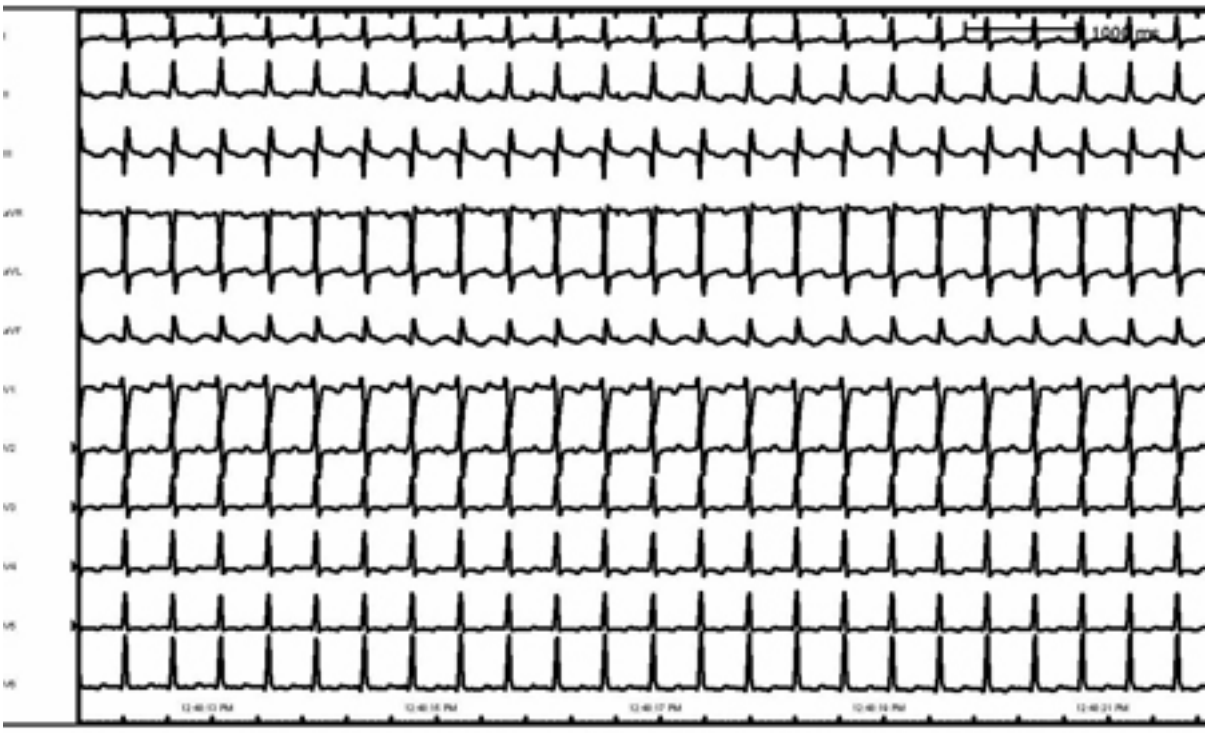
Atrial tachycardia

Rapid atrial activation from a region of the atria other than the sinus node with rates typically between 100 and 250 bpm. Multifocal atrial tachycardia is defined as three or more sites of atrial activation (commonly irregular rhythm). Focal atrial tachycardia can occur without cardiac disease. Atrial tachycardia with AV block should raise the suspicion for digitalis toxicity. Hypokalaemia can also exacerbate this condition.[24] Right-sided atrial tachycardias tend to originate from the crista terminalis, tricuspid annulus, or coronary sinus

ostium. Left-sided atrial tachycardias often originate from around the pulmonary veins, atrial septum, or mitral annulus.[1] [2]

Atrial flutter

Organised re-entrant rhythm with atrial rates typically 250 to 350 bpm and ventricular rates often 145 to 150 bpm (due to 2:1 block) involving large areas of the atrium. In typical atrial flutter, the macroentry atrial circuit rotates around the tricuspid valve and between the inferior vena cava and tricuspid annulus. This essential part of the circuit can be a target for catheter ablation.[1] [2]



Atrial flutter

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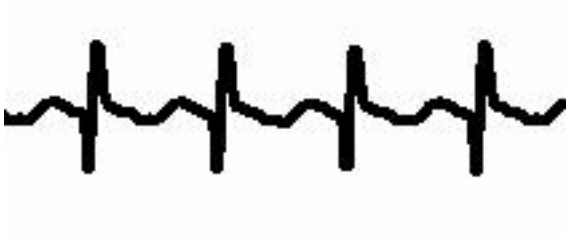

Atrial flutter (detail)

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Sinus node re-entry tachycardia

A tachycardia that originates from re-entry circuits that involve the sinus node and perinodal tissue. It is presumed to be secondary to heterogeneous conduction properties of the sinus node and perinodal tissue.[1] [2]

AV nodal re-entrant tachycardia (AVNRT)

A re-entrant tachycardia involving two pathways within the AV node or perinodal atrial tissue. One pathway has rapid conduction and a relatively long refractory period; the second pathway has slow conduction and a shorter refractory period. Following a premature atrial impulse, the fast pathway is still refractory from the previous impulse but anterograde conduction can occur through the slow pathway, which is no longer refractory. By the time slow-pathway conduction is complete, the fast pathway is no longer refractory and retrograde conduction can occur. If the slow pathway is no longer refractory following retrograde conduction, the cycle repeats. A less common form of AVNRT ('atypical' AVNRT) involves anterograde conduction down the fast pathway, resulting in a re-entry circuit that turns in the opposite direction to the more common 'typical' AVNRT described above.[1] [2]

AV reciprocating tachycardia (AVRT)

A re-entrant tachycardia circuit that involves an accessory pathway as well as the native AV node. The most common form (approximately 90% of cases) is orthodromic AVRT.[25] The arrhythmia results from anterograde conduction through the AV node and retrograde conduction through the accessory pathway. This results in a narrow-complex tachycardia because the ventricle is activated anterograde through the His-Purkinje system.[26]

Permanent junctional reciprocating tachycardia

A form of orthodromic AVRT involving anterograde conduction through the AV node and retrograde accessory-pathway conduction. The retrograde limb of this re-entry circuit is characterised by slow conduction, creating a very stable circuit (anterograde down the AV node and retrograde up the slowly conducting bypass tract). The descriptive term 'permanent' is added to reflect its stable nature and tendency to recur frequently and dominate sinus rhythm. For this reason, this arrhythmia can sometimes cause a tachycardia-mediated cardiomyopathy. This rhythm has rates between 120 and 200 bpm.[1] [2] It is most commonly seen in infants and children.[1] [2]

Junctional ectopic tachycardia

This rhythm is caused by abnormal rapid discharges from the junctional region (distal AV node or proximal His-Purkinje system) and does not require atrial or ventricular involvement to originate. The congenital form is insidious in presentation and is often identified in infants only after the development of tachycardia-induced cardiomyopathy. It is sometimes seen after cardiac surgery and may result in haemodynamic instability because of its rate and lack of AV synchrony. Clinical factors that may predispose to this arrhythmia include digitalis toxicity, hypokalaemia, myocardial ischaemia, cardiac surgery, and inflammatory myocarditis.[1] [2]

Narrow QRS (duration <120 ms) with an irregular ventricular rhythm

Atrial fibrillation

A supraventricular tachyarrhythmia characterised by an irregularly irregular rhythm due to the rapid and random conduction of irregular impulses to the ventricle. The irregular impulses are due to multiple wavelets of re-entry that rotate randomly within the atria and randomly bombard the AV node.[27] Risk of atrial fibrillation is increased in patients treated for hypertriglyceridaemia with medicine containing omega-3-acid ethyl esters, particularly at high doses.[28] [29] [30]

Atrial tachycardia or flutter with variable AV conduction

In contrast to atrial fibrillation, rapid atrial tachycardia and atrial flutter result in fast but regular impulses to the AV node. Conduction of impulses to the ventricle is variable but tends to be 'regularly irregular' and with a pattern.[1] [2]

Multifocal atrial tachycardia

This arrhythmia involves at least three distinct competing atrial foci. Multifocal atrial tachycardia is most often associated with pulmonary disease but has also been associated with cardiac disease (valvular, hypertensive, coronary) as well as a variety of other systemic conditions, including hypokalaemia, hypomagnesaemia, and sepsis, and certain medications (including isoprenaline [isoproterenol] and aminophylline).[31] [32]

Wide QRS (duration >120 ms) with a regular ventricular rhythm

Atrial tachycardia, atrial flutter, and common supraventricular tachycardias that conduct with aberrancy to the ventricle (left bundle branch block or right bundle branch block) are an important part of the differential diagnosis of aetiologies of wide-complex tachycardias with uniform QRS duration and morphology.

Idiopathic ventricular tachycardia (VT) (monomorphic VT associated with a structurally normal heart)

Repetitive monomorphic ventricular tachycardia: a focal arrhythmia that is thought to be due to triggered activity. Typically, it originates from the right ventricular outflow tract (when it is known as right ventricular outflow tract tachycardia). The aetiology is unknown. It is mostly seen in young and middle-aged patients of both sexes, with a structurally normal heart and is often provoked by exercise, emotion, stress, or hormone fluctuations.[33] Less commonly the site of origin may be in the LV outflow tract, the LV epicardium, above the pulmonary valve, or along the septal aspect of the mitral annulus. VT that maps to these regions behaves similarly to 'right ventricular outflow tract VT' and typically follows a benign clinical course.

Idiopathic left VT: occurs due to re-entry around the specialised conduction tissue and slow calcium-ion-sensitive myocardial tissue. The aetiology of this arrhythmia is unknown. It is sensitive to calcium channel blockers, which may terminate and control the arrhythmia. The re-entry circuit usually involves the left posterior fascicle and the VT is therefore relatively narrow, with a right bundle branch block and a superior axis on the 12-lead ECG. It is seen in patients aged 15 to 40 years old with structurally normal hearts. Seventy percent of cases are in males. In most cases, this VT is not associated with an elevated risk of sudden death.[34] [35]

Accelerated idioventricular rhythm

An automatic focus originating in the ventricular myocardium. Similar to VT, though rates are no more than 20% faster than the sinus rate (typically 80-120 bpm).[34]

Monomorphic VT associated with previous myocardial infarction

An arrhythmia that usually originates at the interface between healthy and damaged myocardium and is most commonly a re-entrant rhythm.[36] It is more commonly seen with larger infarctions and in patients with a depressed ejection fraction.

Monomorphic VT associated with non-ischaemic cardiomyopathy

Regardless of the aetiology of the cardiomyopathy, heterogeneous conduction properties within the ventricular myocardium due to cardiomyopathy can serve as a substrate for re-entry and result clinically in VT. Some specific cardiomyopathies merit special attention.[34]

- Arrhythmogenic right ventricular dysplasia: characterised by fatty right ventricular infiltration and fibrosis or thinning. VT is due to re-entry within this complicated substrate.
- Bundle branch re-entry VT: a common cause of sustained VT in patients with non-ischaemic dilated cardiomyopathy. The re-entry circuit usually conducts anterograde down the right bundle branch, across the ventricular septum, and conducts retrograde up the left bundle branch. The characteristic 12-lead ECG shows a left bundle branch block pattern during VT because of this pattern of activation.
- Cardiac sarcoidosis: may present with arrhythmias (such as advanced AV block or VT) and/or unexplained new onset heart failure without a history of systemic sarcoidosis.[37] [38] [39] The patchy formation of granulomas, fibrosis, and scarring leads to VT due to reentry within this complicated substrate.[37] Clinically overt cardiac sarcoidosis has been reported in 5% to 10% of cases with systemic sarcoidosis.[37] [40] [41] Cardiac sarcoidosis has been confirmed in approximately one third of middle-aged patients (37%) presenting with unexplained high-grade atrioventricular block.[42]
- Chagas cardiomyopathy: VT is predominantly seen in chronic Chagas cardiomyopathy, which is probably caused by an autoimmune response to infection with *Trypanosoma cruzi* . The mechanism is likely to be related to the resulting fibrosis of the myocardium. The mechanism of VT is re-entry within this complicated substrate.

Rapid atrial arrhythmias associated with a 'bystander' accessory pathway

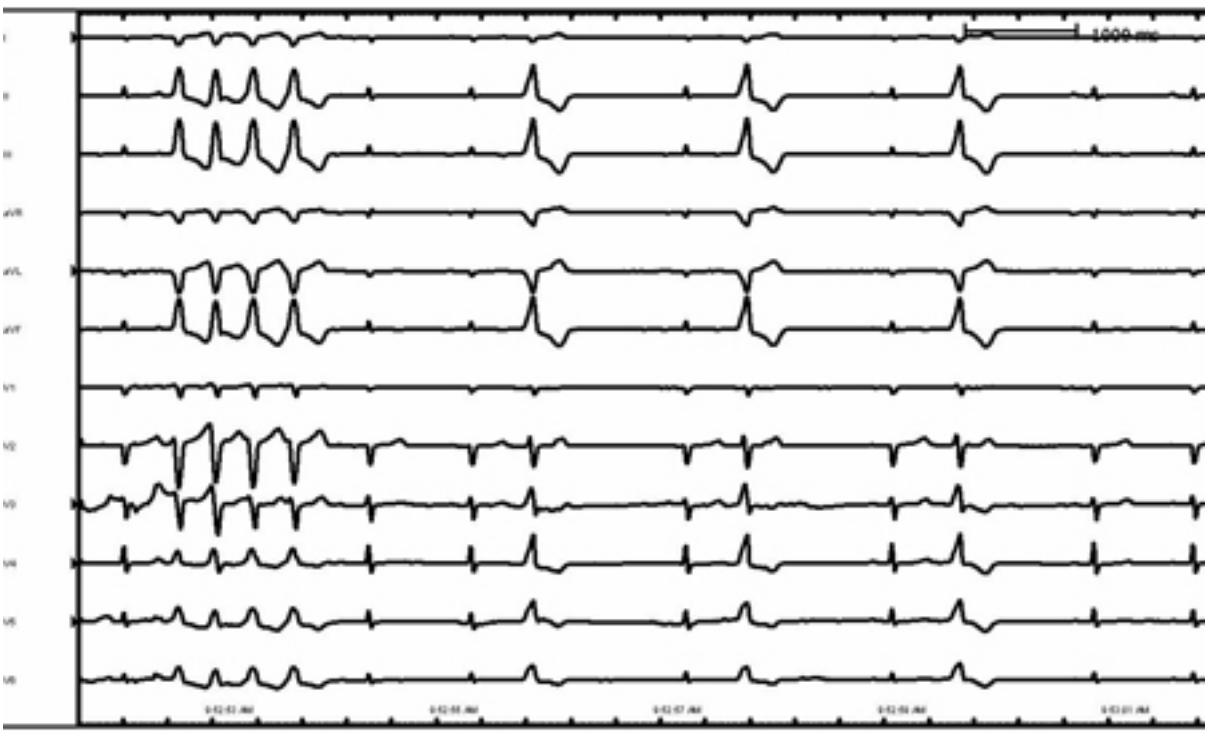
Any atrial arrhythmia (atrial flutter, supraventricular tachycardia) that normally conducts through the native His-Purkinje system and would manifest with a narrow QRS can instead present as a wide-complex tachycardia if there is also an associated accessory pathway.[1] [2] Additionally, with rapid supraventricular rhythms, a rate-related bundle branch may develop only at times of tachycardia, resulting in a wide-complex-tachycardia morphology.

Antidromic AV reciprocating tachycardia

Re-entrant circuit in which an anterograde circuit conducts down the accessory pathway and retrograde back up the AV node, resulting in a wide-QRS-complex morphology. The re-entry circuit in this arrhythmia is the same as that in orthodromic AV reciprocating tachycardia but in the opposite direction. Because activation is anterograde down the accessory pathway, the QRS complex is maximally pre-excited during tachycardia.[1] [2]

Paced rhythm

Patients with pacemakers can present with wide-complex tachycardia secondary to rapid ventricular pacing. Most commonly this is due to a dual chamber device tracking an atrial arrhythmia (atrial tachycardia and atrial flutter) with subsequent rapid ventricular pacing.



Right ventricular outflow tract ventricular tachycardia

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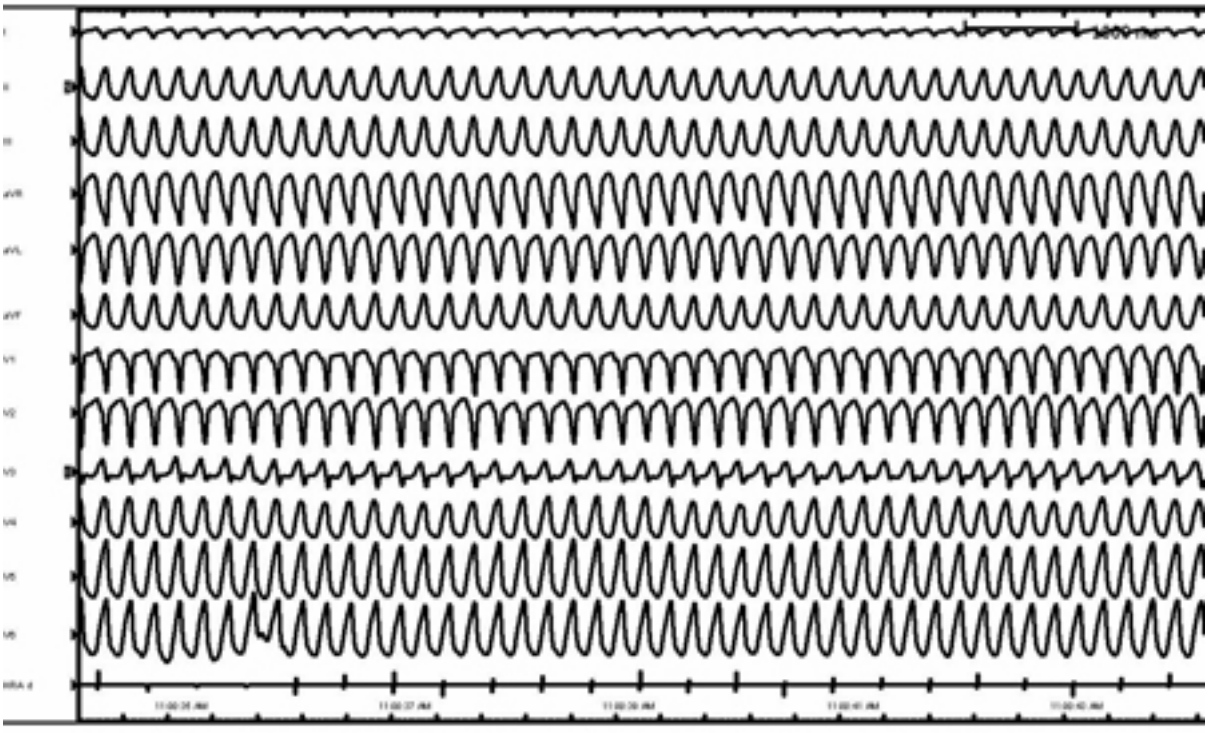

Ventricular tachycardia in a patient with arrhythmogenic right ventricular cardiomyopathy

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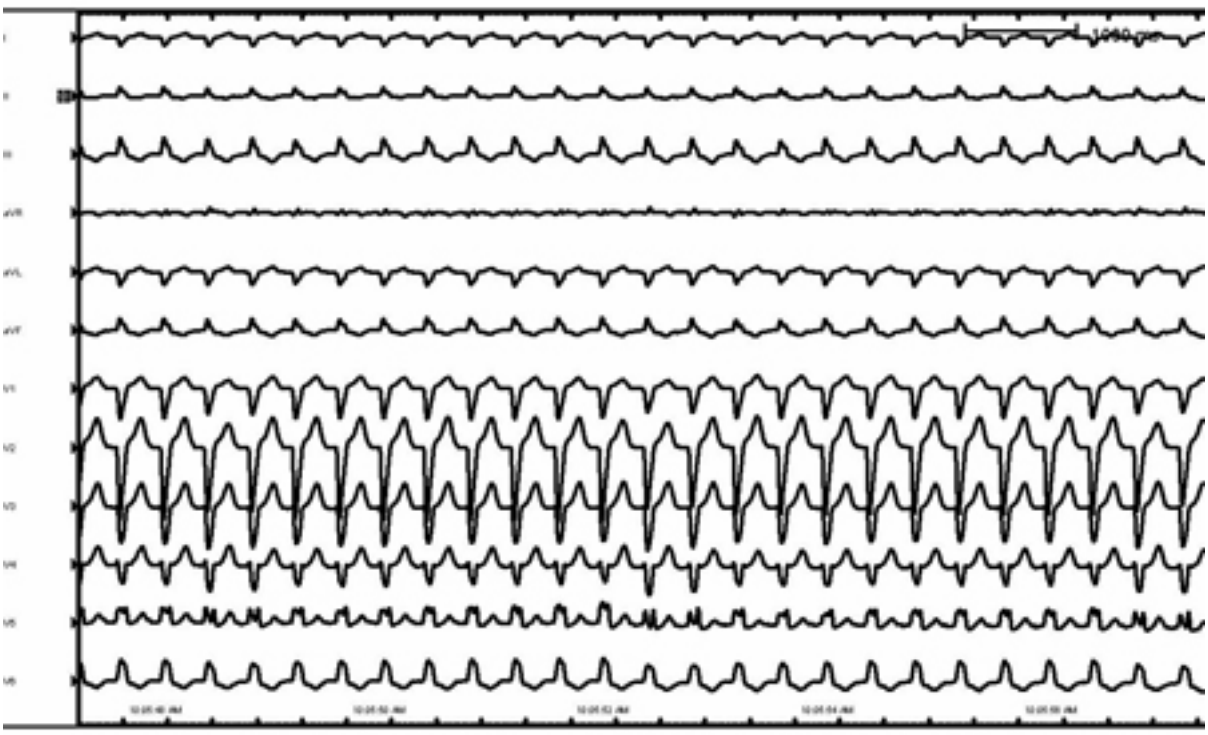

Supraventricular tachycardia with aberrancy and left bundle branch block

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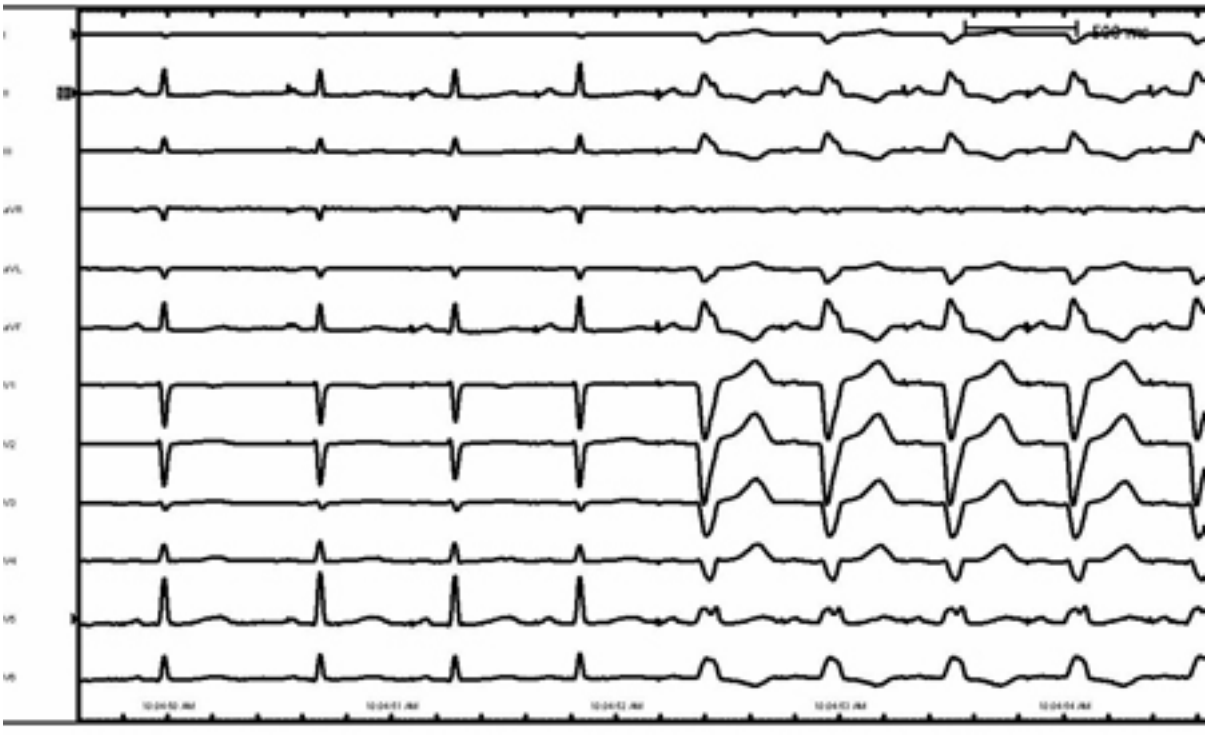

Rate-related left bundle branch block

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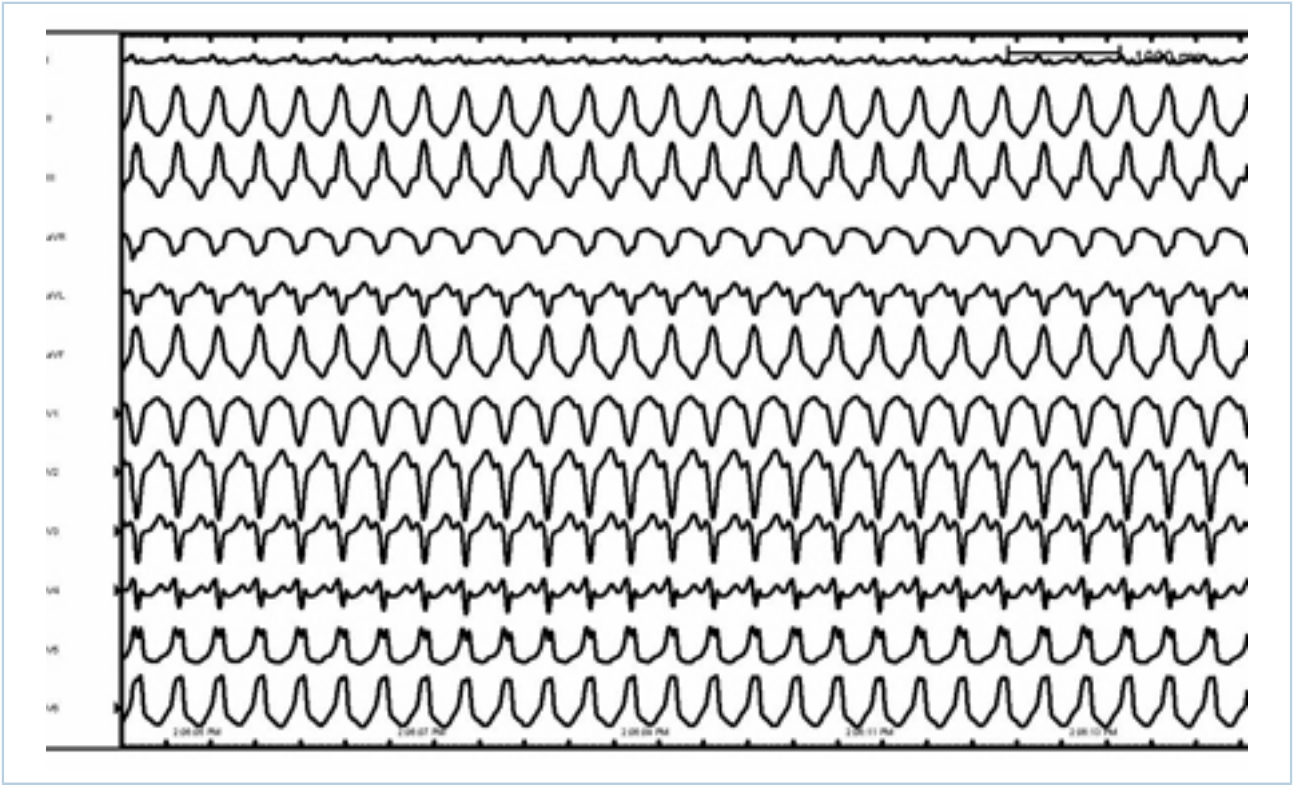

Antidromic re-entrant tachycardia

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Wide QRS (duration >120 ms) with an irregular ventricular rhythm

Causes include the following:

- Atrial fibrillation with a bundle branch block
- Multifocal atrial tachycardia with a bundle branch block
- Atrial tachycardia or flutter with variable AV conduction with a bundle branch block or accessory pathway.

Variable QRS duration with an irregular ventricular rhythm

Polymorphic VT with a normal QT interval

Most commonly seen in the context of acute coronary syndrome or myocardial ischaemia but can also be seen in structurally normal hearts. In the context of myocardial infarction, the development of polymorphic VT is suggestive of ongoing ischaemia; treatment is focused on the underlying ischaemia. Ion channelopathies can present with polymorphic VT. Catecholaminergic polymorphic VT occurs in the absence of structural heart disease and often presents as stress- or exercise-induced syncope, or sudden death in childhood or adolescence. A variety of genetic causes have been identified, one of which involves an autosomal dominant mutation in the gene for the cardiac ryanodine receptor.[43] Less commonly, an autosomal recessive mutation in the cardiac calsequestrin gene (CASQ2) has been associated with this condition. Both of these gene mutations are associated with the release of calcium from the sarcoplasmic reticulum.[44]

Torsades de pointes

Polymorphic VT associated with a prolonged QT (observed during sinus rhythm) with a characteristic 'twisting on axis' morphology. The prolonged QT may be congenital or acquired. Acquired long-QT syndrome is more commonly observed clinically. It may be secondary to medications known to prolong the QT interval, [AZCERT: QT drugs list] (https://www.crediblemeds.org/everyone/composite-list-all-qtdrugs) ischaemia, significant electrolyte abnormalities (hypokalaemia, hypomagnesaemia, hypocalcaemia), or massive injury to the central nervous system.[43] [45]

Bi-directional VT

A rare but potentially fatal arrhythmia most often caused by digitalis toxicity but also seen in catecholaminergic polymorphic VT and Andersen-Tawil syndrome (long QT 7). It is characterised by two alternating QRS morphologies (often a right bundle branch block with alternating left and right axis). Contrary to ventricular bigeminy, the R-R interval in bi-directional VT is regular.[34]

Atrial fibrillation with ventricular pre-excitation

Patients with atrial fibrillation and an anterograde-conducting accessory pathway may present with a wide-complex irregular tachycardia. Some patients may have anterograde conduction pathways with more rapid properties than others and are therefore at greater risk of this arrhythmia. Identification of this arrhythmia is vital because it can lead to ventricular fibrillation and haemodynamic collapse. Pre-excited atrial fibrillation is a potentially life-threatening arrhythmia.[5]

Ventricular fibrillation

Rapid (rate >300 bpm), grossly irregular, life-threatening ventricular rhythm characterised by variable QRS cycle length, morphology, and amplitude.[34]

Urgent considerations

(See **Differentials** for more details)

Cardiac arrest and haemodynamic instability: urgent cardioversion

Patients who suffer a cardiac arrest from ventricular fibrillation, polymorphic ventricular tachycardia (VT), or rapid VT require CPR and prompt defibrillation.[46] In this situation the chance of surviving the cardiac arrest decreases by 7% to 10% for every 1-minute delay in defibrillation.[47] [48] [49]

Atrial fibrillation with ventricular pre-excitation often requires immediate cardioversion because of the risk that the arrhythmia will degenerate into ventricular fibrillation. Long term anticoagulation should be considered based on thromboembolic risk.[5]

In any situation where a tachycardia (regardless of the mechanism) is the cause of haemodynamic instability, angina, syncope, or decompensated heart failure, the priority should be towards rapid termination of the arrhythmia. In many cases (atrial fibrillation with rapid ventricular response, supraventricular tachycardia, VT) electrical cardioversion is the most efficient and reliable way to achieve sinus rhythm. It is useful to obtain a rhythm strip during and immediately after cardioversion in the event of re-initiation of the rhythm.[47] [48]

Regular wide-complex or regular narrow-complex tachycardia with haemodynamic stability: adenosine administration

In patients who are stable with a regular wide- or narrow-complex tachycardia, administration of adenosine is a therapeutic intervention and can provide useful diagnostic information.[14]

Adenosine should be administered in a closely monitored setting, with the patient supine, and with continuous ECG and haemodynamic monitoring.

Adenosine is metabolised rapidly by red blood cells and must therefore be given as a rapid bolus to be effective.

Adenosine transiently slows the sinus node or atrial tachycardia and transiently slows or blocks conduction in the atrioventricular (AV) node. Depending on the arrhythmia mechanism, adenosine may unmask the underlying rhythm (atrial flutter, atrial tachycardia) or may terminate arrhythmias that are dependent on the AV node (AV nodal re-entrant tachycardia, AV reciprocating tachycardia).

Caution is required in the presence of atrial fibrillation and a possible accessory conduction pathway because adenosine can precipitate preferential rapid accessory tract conduction and degeneration to ventricular fibrillation.

Approach

The diagnostic approach to a patient with tachycardia focuses on rapid assessment of the clinical consequences and careful assessment to identify the mechanism of the arrhythmia and the setting in which it is occurring (drug toxicity, structural heart disease, ischaemia).

In the setting of haemodynamic instability, it is important that diagnostic manoeuvres do not delay therapy necessary to terminate the tachycardia.[1] [2] [50]

History

Often, patients with paroxysmal tachycardia will be asymptomatic at the time of assessment. Symptoms associated with tachycardia include palpitations, fatigue, lightheadedness, pre-syncope, chest discomfort, and dyspnoea.

Details of the pattern of the symptoms should be obtained, such as regular or irregular palpitations, number and frequency of episodes, potential triggers, whether the onset is abrupt or gradual, duration of symptoms, and how the tachycardia terminates.

The history should also include an assessment of potential stressors such as hypovolaemia, infection, ischaemia, heart failure; the patient's medication regimen (including herbal supplements); a thorough history of substance use or misuse (including caffeine, energy drinks, and other stimulants); and any family history of arrhythmias or sudden death.

Pre-syncope and syncope associated with tachyarrhythmia and structural heart disease have a poor prognosis and should prompt detailed questioning surrounding the event.

A history of gradual onset and termination is more common with sinus tachycardia and atrial tachycardia, whereas an abrupt onset and termination is more common for re-entrant tachycardias such as supraventricular tachycardia (SVT) and ventricular tachycardia (VT). The ability to terminate the rhythm abruptly with vagal manoeuvres suggests that the re-entrant rhythm circuit involves the AV node and is associated with AV nodal re-entrant tachycardia (AVNRT) or orthodromic AV reciprocating tachycardia (AVRT).[14]

It is important to note that a common misconception is that haemodynamic stability may help to 'rule out' VT. However, VT may commonly be tolerated haemodynamically and, in contrast, some atrial arrhythmias (for example, atrial fibrillation and atrial flutter conducting with a rapid ventricular rate) may be poorly tolerated. Therefore, haemodynamic stability should not be a factor in distinguishing VT from atrial arrhythmias.

Physical examination

The standard physical examination is often unrevealing, particularly if the tachycardia is episodic and not ongoing at the time of assessment.

The physical examination should include a detailed cardiac examination to assess for valvular, congenital, and other structural heart disease.

A careful assessment for signs of cardiomyopathy should be performed because of the worse prognosis of tachyarrhythmias associated with structural heart disease. This includes looking for S3 gallop, right ventricular (RV) heave, laterally displaced point of maximal impulse, and other signs of heart failure (elevated jugular venous pressure, lower-extremity oedema).

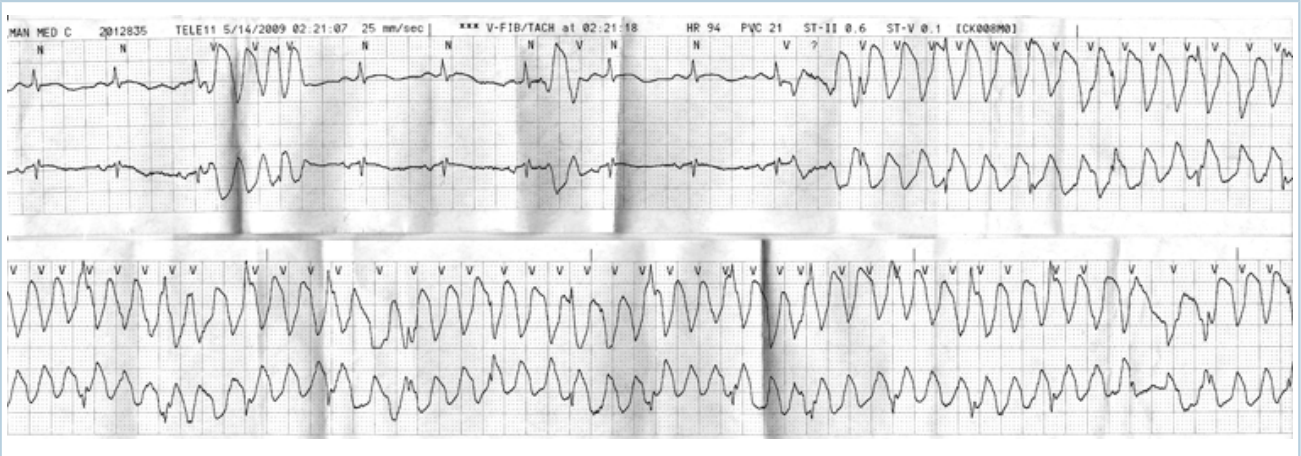
If the patient has the symptoms at the time of physical assessment, determining whether the pulse is regular or irregular can greatly assist with a diagnosis. Physical examination findings associated with AV dissociation such as cannon A waves or variability in the intensity of S1 are highly suggestive of VT.[51]

Diagnostic studies: 12-lead ECG

The resting 12-lead ECG is the cornerstone of the standard assessment of tachycardia.

Even if the patient does not have the tachyarrhythmia at the time of the ECG, one can assess for evidence of prior myocardial infarction (pathological Q waves), prolonged QT interval, ischaemia, atrial or ventricular enlargement or hypertrophy, and signs of pre-excitation.

Evidence of pre-excitation, evidenced by a widened QRS complex with a delta wave, should raise suspicion for AVRT. The ECG should also be closely assessed to rule out artifacts such as electrical interference or movement of telemetry monitoring leads as a cause for the observed abnormality.



Artifact overlying sinus rhythm

From the collection of Robert W. Rho, MD; used with permission

If the patient is experiencing the tachyarrhythmia at the time of the ECG, the findings can be diagnostic.

Having determined whether the tachyarrhythmia has a narrow or wide QRS interval, one can apply the appropriate diagnostic algorithm to develop a preliminary differential or diagnosis.[1] If P waves cannot be seen, sometimes an oesophageal lead can also be used to help determine the atrial:ventricular relationship during tachycardia.[1] [2]

During the assessment of a regular narrow-QRS-complex tachycardia (<120 ms), assessment of the response to elevated AV nodal blockade, either through carotid massage or with adenosine, can assist with the differential diagnosis. Intravenous adenosine should be administered in a monitored setting and with a continuous 12-lead ECG recording at the time of the manoeuvre. Absence of effect on ventricular rate, a temporarily gradual slowing of ventricular rate, or sudden termination of the tachycardia may be helpful in the diagnosis and can be therapeutic in some situations. Temporary AV block may unmask an atrial tachyarrhythmia. If this is ineffective or not feasible and the patient is haemodynamically stable, intravenous beta blocker or intravenous diltiazem or verapamil can be used.

For the assessment of wide-QRS-complex tachycardia (>120 ms), differentiation between SVT and VT is important because intravenous medications for SVT (verapamil or diltiazem) can have adverse and even fatal consequences in a patient with VT. If differentiation between SVT and VT cannot be made, one should suspect and treat as VT until shown otherwise, especially in a patient with structural heart disease.

A useful way to sub-categorise SVTs is to assess the relationship between the P wave and the preceding R wave. SVTs can be categorised into short-RP tachycardias (RP less than half the R-R interval) and long-RP tachycardias (RP greater than half the R-R interval). Short-RP tachycardia with retrograde P waves usually represents typical (slow-fast) AVNRT, AVRT, or atrial tachycardia with prolonged AV conduction. A long-RP tachycardia usually represents permanent junctional reciprocating tachycardia, atypical (fast-slow) AVNRT, or an atrial tachycardia conducting with brisk AV node conduction.[1] [2]

Further diagnostic studies

Monitoring devices

- If the patient has frequent episodes (several per day) of the presumed tachyarrhythmia but is not experiencing the rhythm at the time of assessment, an ambulatory 24- or 48-hour Holter recording can be used.
- If the episodes are less frequent and would be unlikely to occur during a 24- to 48-hour monitoring period, an event or wearable loop recorder can be used.[52] [53]
- If episodes occur rarely (<2 episodes per month) and are associated with haemodynamic instability or syncope (in which case activation of an event monitor is unlikely), an implantable loop recorder is appropriate and has been shown to be effective.[50]
- If the patient already has a pacemaker or defibrillator in place, interrogation of the device may greatly assist with diagnosis.[5]

Direct to consumer technologies

- Wearable and remote ECG-recording devices may be used by patients outside of a medical setting to provide single/multi-lead ECGs.[54] [55] Some devices may not be approved as medical devices.
- Clinicians should be prepared to discuss results generated using these technologies, and understand their risks and limitations.[56]
- The 12-lead ECG remains the gold standard for the detection and evaluation of tachycardia.

Imaging

- Imaging is not required in all cases and its use depends on the nature of the suspected tachyarrhythmia and the patient's overall clinical presentation.
- Indications for echocardiography include wide-complex tachycardia of unknown origin, documented sustained atrial arrhythmias (atrial fibrillation, atrial flutter, SVT), or findings on history or physical examination that suggest structural heart disease.[57]

Exercise testing

- Exercise testing may be helpful in defining the association of the arrhythmia to exercise, in provoking the arrhythmia, and in ruling out significant coronary artery disease, if appropriate.
- If the arrhythmia is provoked during the study, the onset, termination, and a full 12-lead ECG of the arrhythmia is provided.
- The exercise stress test is particularly helpful in provoking VT in patients suspected to have right ventricular outflow tract VT.

Electrophysiology testing

- A diagnostic electrophysiological study (EPS) is a useful tool for clarifying the mechanism of sustained and non-sustained supraventricular and ventricular arrhythmias. In wide-complex tachycardias where

the diagnosis is uncertain, a diagnostic EPS can be helpful in establishing whether the arrhythmia is supraventricular or ventricular in origin. In addition to the diagnosis of tachyarrhythmias, several arrhythmias can be successfully treated with radiofrequency ablation at the time of the EPS.

- Indications for referral to a cardiac arrhythmia specialist for EPS include, but are not limited to:
    - Unknown wide-complex tachycardias
    - Wolff-Parkinson-White syndrome (pre-excitation with arrhythmia)
    - History of MI with history or symptoms suggestive of VT.

Laboratory testing

- Baseline laboratory evaluation should be targeted toward the patient's overall clinical picture. But a number of tests should be included:
    - Blood electrolytes: particularly potassium, magnesium, and calcium; hypovolaemia, which can manifest as pre-renal azotaemia or orthostatic hypotension, can cause sinus tachycardia
    - FBC: to determine if anaemia is a contributing factor
    - Thyroid function tests: particularly if hyperthyroidism is in the differential diagnosis; results are often normal
    - Cardiac biomarkers: for patients who present with chest pain, have significant risk factors for ischaemic heart disease, or are otherwise unstable; can show whether ischaemia or infarction are contributing factors
    - Drug levels: drug toxicity should be considered, particularly for patients who are on digitalis or who present with closely associated rhythms such as bi-directional VT or atrial tachycardia with AV block
    - Toxicology screen: for stimulants such as cocaine or tricyclic antidepressants.

Differentials overview

Common

- Sinus tachycardia
- Acute atrial fibrillation
- Chronic atrial fibrillation
- Atrial flutter
- Atrial tachycardia
- AV nodal re-entrant tachycardia
- AV re-entry tachycardia/Wolff-Parkinson-White syndrome
- Multifocal atrial tachycardia
- Junctional ectopic tachycardia
- Monomorphic ventricular tachycardia with prior myocardial infarction
- Monomorphic ventricular tachycardia with non-ischaemic cardiomyopathy
- Ventricular fibrillation
- Polymorphic ventricular tachycardia with normal QT interval
- Idiopathic ventricular tachycardia: structurally normal heart

Uncommon

- Sinus node re-entry tachycardia
- Inappropriate sinus tachycardia
- Permanent junctional reciprocating tachycardia
- Torsades de pointes
- Bidirectional ventricular tachycardia
- Accelerated idioventricular rhythm

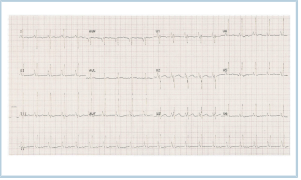
Uncommon

Postural orthostatic tachycardia syndrome

Differentials

Common

◊ Sinus tachycardia

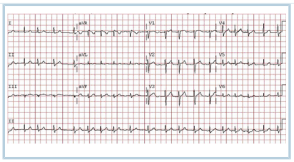
History	Exam	1st Test	Other tests
fever or other signs of infection; weight loss and/or agitation (suggestive of hyperthyroidism); causes of anxiety or stress; fatigue or malaise (suggestive of anaemia); drug use history; medication use and dosage changes; orthostatic symptoms (volume depletion); if palpitations are felt, they have a gradual onset and gradual resolution; postural orthostatic tachycardia syndrome (POTS) is characterised by an exaggerated heart rate and orthostatic symptoms in response to postural change, in the absence of orthostatic hypotension and cardiac causes of sinus tachycardia	regular tachycardic pulse; skin pallor (anaemia); lid lag, warm smooth skin (hyperthyroidism); hypotension or orthostasis (volume depletion); normal cardiac examination	»**12-lead ECG:** regular narrow-complex tachycardia (heart rate >100 bpm); P wave before every QRS complex  *ECG example of sinus tachycardia BMJ Learning/ Professor Kevin Channer; used with permission*	»**FBC:** leukocytosis if there is an infection; low Hb in anaemia »**TSH:** low in primary hyperthyroidism »**urea/creatinine ratio:** elevated urea/ creatinine ratio with volume depletion »**urine and blood toxicology:** positive if drug use is the aetiology; may be negative in the case of withdrawal (such as with alcohol)

◊ Acute atrial fibrillation

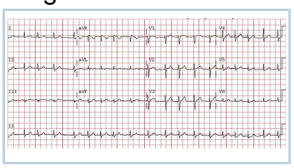
History	Exam	1st Test	Other tests
often asymptomatic but history can include irregular palpitations, malaise, fatigue, chest pain, or dyspnoea; may have history of alcohol misuse,	normal physical examination in the absence of other concomitant pathologies, except for the presence of an irregularly irregular	»**12-lead ECG:** P waves absent with an irregular ventricular rate	»**transthoracic echocardiogram:** rules out structural heart disease Left atrial enlargement, valvular disease, and left ventricular

Common

◊ Acute atrial fibrillation

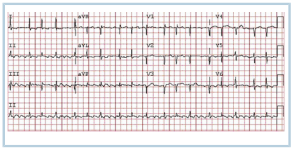
History	Exam	1st Test	Other tests
use of stimulants or illicit stimulants, use of omega-3-acid ethyl esters in patients treated for hypertriglyceridaemia, hyperthyroidism, pulmonary embolism, heart failure, lung disease, hypertension, or diabetes	pulse; signs of heart failure, lung disease, hyperthyroidism, hypertension, or diabetes may be found	 *Atrial fibrillation: P waves are not discernible; the ventricular (QRS complexes) rate is irregularly irregular* *From the collection of Dr Arti N. Shah*	dysfunction can predispose to atrial fibrillation. »**TSH:** low with hyperthyroidism »**urine and blood toxicology:** positive if drug use is the aetiology »**cardiac biomarkers:** positive with recent or ongoing atrial or ventricular ischaemia

◊ Chronic atrial fibrillation

History	Exam	1st Test	Other tests
history can include palpitations, shortness of breath, fatigue, chest pain, dizziness, and stroke; due to rapidity of ventricular response, cerebral hypoperfusion can result in pre-syncope; may be asymptomatic; may have history of hypertension, coronary artery disease, congestive heart failure, rheumatic valvular disease, alcohol misuse, hyperthyroidism, or recent cardiothoracic surgery	normal physical examination in the absence of other concomitant pathologies, except for the presence of an irregularly irregular pulse; signs of heart failure, lung disease, hyperthyroidism, hypertension, or diabetes may be found	»**12-lead ECG:** P waves absent with an irregular ventricular rate  *Atrial fibrillation: P waves are not discernible; the ventricular (QRS complexes) rate is irregularly irregular* *From the collection of Dr Arti N. Shah*	»**transthoracic echocardiogram:** rules out structural heart disease Left atrial enlargement, valvular disease, and left ventricular dysfunction can predispose to atrial fibrillation. »**TSH:** low with hyperthyroidism »**urine and blood toxicology:** positive if drug use is the aetiology »**cardiac biomarkers:** positive with recent or ongoing atrial or ventricular ischaemia

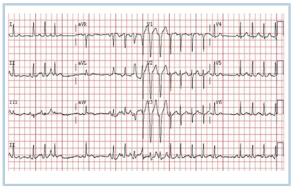
Common

◊ Atrial flutter

History	Exam	1st Test	Other tests
palpitations, dyspnoea, fatigue, chest discomfort, or worsening exercise tolerance, or symptoms of heart failure; history of congenital heart disease; previous heart surgery; structural heart disease	normal physical examination except for a rapid pulse (usually regular, but can be irregular with AV block); suggestive of heart failure: jugular venous distension, lung crackles, and lower-extremity oedema; hypotension in the context of rapid atrial flutter may provoke more urgent cardioversion	»**12-lead ECG:** typical atrial flutter characterised by regular narrow-complex tachycardia with regular sawtooth flutter waves best seen in leads II, III, aVF (type 1 flutter), atrial rates 240 to 340 bpm with ventricular rates most commonly 150 bpm (2:1 conduction); atypical atrial flutter characterised by flutter-wave morphology without the characteristic sawtooth pattern ECG example of atrial flutter:  *Atrial flutter: typical saw-tooth appearance of the flutter waves in the inferior leads (leads II, III, and aVF) indicates typical counterclockwise atrial flutter; the ventricular (QRS complexes) rate is variable* *From the collection of Dr Arti N. Shah*	»**transthoracic echocardiogram:** rules out structural heart disease

Common

◊ Atrial tachycardia

History	Exam	1st Test	Other tests
sudden-onset palpitations, dizziness, dyspnoea, lightheadedness, or chest pressure or tightness; may have symptoms of infection or hyperthyroidism; may be on digoxin; may have taken stimulants	normal physical examination except for a rapid pulse (if the rhythm is occurring at that time); no orthostatic hypotension	»**12-lead ECG:** regular narrow-complex tachycardia (rate 100-250 bpm); an abnormal P-wave axis suggests an ectopic atrial focus; at faster rates there may be variable AV block Atrial tachycardia with AV block should raise strong suspicion for digitalis toxicity.  *Atrial tachycardia: bursts of atrial tachycardia (10 beats in the middle section of the rhythm strip II at bottom) follows sinus complexes* *From the collection of Dr Arti N. Shah*	»**serum digitalis level:** elevated if digitalis toxicity Should be checked, particularly if ECG shows atrial tachycardia with AV block. »**TSH:** low in primary hyperthyroidism »**serum potassium:** low level can exacerbate atrial tachycardia »**toxicology screen:** stimulants such as cocaine can cause atrial tachycardia

◊ AV nodal re-entrant tachycardia

History	Exam	1st Test	Other tests
episodic tachycardia with abrupt onset and termination; can be associated with symptoms of chest discomfort, dyspnoea, dizziness, or anxiety; in the differentiation between narrow-complex regular tachycardias, a sensation of a regular	normal physical examination except for a rapid regular pulse	»**12-lead ECG:** regular narrow-complex tachycardia (rate 150-250 bpm) without apparent P waves before each QRS complex; a retrograde P wave may be seen negative in the inferior leads	»**transthoracic echocardiogram:** rules out structural heart disease

Common

◊ AV nodal re-entrant tachycardia

History	Exam	1st Test	Other tests
rapid pounding in the neck is highly suggestive of AV node re-entry		Pseudo R wave in V1 and pseudo S wave in the inferior leads may be present. Retrograde P waves are hidden in the terminal portion of the QRS complex.	

◊ AV re-entry tachycardia/Wolff-Parkinson-White syndrome

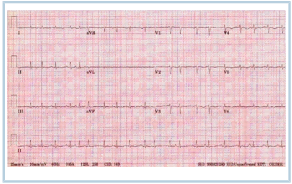
History	Exam	1st Test	Other tests
episodic tachycardia with abrupt onset and termination; can be associated with symptoms of chest discomfort, dyspnoea, dizziness, syncope, or anxiety	normal physical examination except for a rapid regular pulse; suggestive of secondary cardiomyopathy: S3 gallop, right ventricular (RV) heave, laterally displaced point of maximal impulse, and other signs of heart failure (elevated jugular venous pressure, lung crackles, lower-extremity oedema)	»**12-lead ECG:** when in sinus rhythm, a short PR interval with a delta wave, secondary ST-T changes, and a wide QRS complex is the classic Wolff-Parkinson-White (WPW) pattern; if this finding is associated with palpitations, it is called WPW syndrome; a bypass tract that conducts retrograde only is called a concealed bypass tract and will have a sinus-rhythm ECG with a normal PR interval, narrow QRS, and no pre-excitation (i.e., no delta wave at baseline) The same substrate is used in antidromic tachycardia where electrical activation propagates anterograde down the bypass tract and retrograde up the AV node. By contrast with AV reciprocating tachycardia (AVRT),	»**transthoracic echocardiogram:** rules out structural heart disease Ebstein's anomaly and hypertrophic cardiomyopathy are associated with WPW syndrome. Patients with Ebstein's anomaly often have multiple bypass tracts.

Common

◊ AV re-entry tachycardia/Wolff-Parkinson-White syndrome

History	Exam	1st Test	Other tests
		this rhythm is a wide, maximally pre-excited arrhythmia. During SVT, electrical activation goes down the AV node and retrograde up the bypass tract. Therefore, in WPW syndrome and in patients with a concealed bypass tract, supraventricular tachycardia is associated with a normal PR interval and no pre-excitation (i.e., no delta wave at baseline).	

◊ Multifocal atrial tachycardia

History	Exam	1st Test	Other tests
patients may report palpitations and malaise; history of pulmonary disease is highly suggestive of multifocal atrial tachycardia (MAT)	rapid irregular pulse; signs of pulmonary disease or hypoxia	»**12-lead ECG:** narrow-complex tachycardia with at least 3 discrete P-wave morphologies  *Multifocal atrial tachycardia* *From the collections of Arti N. Shah and Bharat K. Kantharia*	»**chest x-ray:** signs of obstructive pulmonary disease Pulmonary disease can be seen with other conditions, including intravenous cocaine use, or secondary to pulmonary embolism. »**serum potassium:** low level can predispose to MAT »**serum magnesium:** low level can predispose to MAT »**serum creatinine:** elevated; chronic renal

DIAGNOSIS

Common

◊ Multifocal atrial tachycardia

History	Exam	1st Test	Other tests
			failure can predispose to MAT

◊ Junctional ectopic tachycardia

History	Exam	1st Test	Other tests
postoperative junctional ectopic tachycardia (JET) is commonly seen following cardiac surgery and may at times lead to haemodynamic compromise due to the loss of A-V synchrony; congenital JET usually presents within the first 4 weeks of life and manifests with symptoms of heart failure; the tachycardia usually has a gradual onset or 'warm up' pattern	often a regular rapid pulse; intermittent cannon A waves can be seen with atrioventricular dissociation in either type of JET; congenital JET can have physical signs of congestive heart failure due to tachycardia-mediated cardiomyopathy within the first 4 weeks of life	»**12-lead ECG:** narrow-complex QRS morphology similar to baseline with gradual QRS acceleration beyond the sinus rate; may have intermittent atrial capture beats	»**transthoracic echocardiography:** depressed left ventricular systolic function »**chest x-ray:** cardiomegaly or pulmonary congestion

Monomorphic ventricular tachycardia with prior myocardial infarction

History	Exam	1st Test	Other tests
history of significant coronary artery disease or structural heart disease; symptoms are abrupt in onset or termination, and can be mild (such as dizziness, diaphoresis, dyspnoea, palpitations) or more severe, including syncope, angina, or cardiogenic shock	rapid regular pulse, often with variable intensity depending on the degree of atrioventricular (AV) dissociation; during haemodynamically tolerated slow VT, cannon A waves, resulting from AV dissociation, are highly suggestive of ventricular tachyarrhythmia; examine for signs of heart failure (right ventricular heave, laterally displaced point	»**12-lead ECG:** presence of AV dissociation; intermittent fusion or capture beats, concordance in the precordial leads and an initial R wave or a positive complex in lead aVR is highly suggestive of ventricular tachycardia; in sinus rhythm, Q waves or ST-segment changes suggestive of ischaemia or injury	»**transthoracic echocardiography:** depressed left ventricular systolic function or wall motion abnormalities can be seen »**serum potassium:** hypo- or hyperkalaemia can predispose to VT »**serum magnesium:** hypomagnesaemia can predispose to VT »**cardiac biomarkers:** elevated

Common

Monomorphic ventricular tachycardia with prior myocardial infarction

History	Exam	1st Test	Other tests
	of maximal impulse, increased jugular vein pressure, S3 gallop, lung crackles, peripheral oedema, ascites), which may predispose the patient to VT	Non-sustained ventricular tachycardia is defined as 3 or more beats with spontaneous termination. Sustained VT lasts longer than 30 seconds and/or is associated with haemodynamic compromise.	with new ischaemia or infarction »**exercise stress testing:** may indicate ischaemia To assess for ischaemia if the clinical suspicion is intermediate. Contra-indicated in recurrent VT and active ischaemia or infarction. Usually performed in conjunction with an echocardiogram or nuclear tracer to rule out ischaemia. »**event monitor:** intermittent tachyarrhythmias Can record intermittent tachyarrhythmias in a patient with concerning symptoms and a history of ischaemic heart disease. »**electrophysiological studies:** can demonstrate dissociation between atrial and ventricular depolarisation in addition to localisation of its origin Can assist with the diagnosis of monomorphic VT if the diagnosis is ambiguous, its haemodynamic consequences,

Common

Monomorphic ventricular tachycardia with prior myocardial infarction

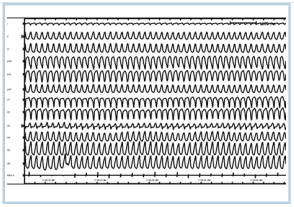
History	Exam	1st Test	Other tests
			responsiveness to anti-tachycardia pacing and mapping of the origin, and may potentially provide a therapeutic intervention.

Monomorphic ventricular tachycardia with non-ischaemic cardiomyopathy

History	Exam	1st Test	Other tests
symptoms are abrupt in onset or termination; intermittent palpitations can be associated with dizziness, diaphoresis, or dyspnoea; may be triggered by emotional stress or exercise; symptoms suggestive of ischaemic heart disease	rapid regular pulse, often with variable intensity depending on the degree of AV dissociation; during haemodynamically tolerated slow VT, cannon A waves, resulting from AV dissociation, are highly suggestive of ventricular tachyarrhythmia	»**12-lead ECG:** wide-complex monomorphic tachycardia (rate commonly 140-180 bpm) with evidence of AV dissociation; no ischaemic changes present Non-sustained VT is defined as 3 or more beats with spontaneous termination. Sustained VT lasts longer than 30 seconds and/or is associated with haemodynamic compromise.	»**transthoracic echocardiography:** may demonstrate cardiomyopathy Additional imaging modalities such as cardiac CT or MRI can be used to assess infiltrative cardiomyopathies. »**TSH:** can be elevated or low because both hyper- and hypothyroidism can result in non-ischaemic cardiomyopathy »**serum potassium:** hypo- or hyperkalaemia can predispose to VT »**serum magnesium:** hypomagnesaemia can predispose to VT

Common

Monomorphic ventricular tachycardia with non-ischaemic cardiomyopathy

History	Exam	1st Test	Other tests
		 *Ventricular tachycardia in a patient with arrhythmogenic right ventricular cardiomyopathy* *From the collection of Robert W. Rho, MD; used with permission*	»**exercise stress testing:** may induce ventricular tachyarrhythmias or demonstrate underlying ischaemia »**event monitor:** intermittent tachyarrhythmias Can record intermittent tachyarrhythmias in a patient with concerning symptoms. »**electrophysiological studies:** presence of inducible VT, multiple VT morphologies, fractionated diastolic electrograms during VT, or regions of low amplitude and prolonged duration suggest arrhythmogenic right ventricular cardiomyopathy (versus idiopathic right ventricular tachycardia) Can assist with the diagnosis of monomorphic VT and potentially provide therapeutic interventions.

Ventricular fibrillation

History	Exam	1st Test	Other tests
often seen, but not limited to, patients with associated ischaemic heart disease and ongoing ischaemia;	pulse absent; dramatic haemodynamic collapse and loss of consciousness	»**12-lead ECG:** rapid dysmorphic irregular rhythm without clear QRS morphologies	»**serum potassium:** hypo- or hyperkalaemia can predispose to VT

Common

Ventricular fibrillation

History	Exam	1st Test	Other tests
associated with rapid haemodynamic collapse and syncope; may have recent history of progressive angina, previous cardiac arrest, severe valvular disease, or depressed left ventricular systolic function		Patients in VF should be defibrillated immediately. Survival decreases by 7% to 10% for every 1-minute delay in defibrillation.[47] [48] [49]	»**serum magnesium:** hypomagnesaemia can predispose to VT »**cardiac biomarkers:** elevated with new ischaemia »**toxicology screen:** screen for cocaine or serum levels of antiarrhythmics »**transthoracic echocardiography:** may show depressed systolic function or wall-motion abnormalities suggestive of ischaemia or infarction »**coronary angiography:** coronary artery disease Should be performed in survivors of VF to assess for coronary artery disease. »**electrophysiological studies:** presence of inducible VT or VF can help identify higher risk patients Not usually indicated if cardiac arrest was within 48 hours of an acute myocardial ischaemic episode.

Polymorphic ventricular tachycardia with normal QT interval

History	Exam	1st Test	Other tests
dizziness, diaphoresis, dyspnoea, palpitations, syncope, and angina; a family history of juvenile sudden death or stress-	peripheral pulses may have variable intensity depending on degree of AV dissociation; often associated with severe	»**12-lead ECG:** wide-complex tachycardia with continuously varying QRS morphology;	»**TSH:** normal »**serum potassium:** hypo- or hyperkalaemia can predispose to VT

Common

Polymorphic ventricular tachycardia with normal QT interval

History	Exam	1st Test	Other tests
induced syncope should raise suspicion for catecholaminergic polymorphic VT	hypotension; cannon A waves, also resulting from AV dissociation, are highly suggestive of ventricular tachyarrhythmia	baseline ECG with a normal QT interval Repeating the ECG after termination of the arrhythmia can demonstrate ischaemia, QT interval, or repolarisation abnormalities.	»**serum magnesium:** hypomagnesaemia can predispose to VT »**cardiac biomarkers:** positive cardiac biomarkers with new ischaemia »**toxicology screen:** screen for cocaine, digitalis levels, and serum levels of tricyclic antidepressants »**transthoracic echocardiography:** may show depressed systolic function or wall-motion abnormalities suggestive of ischaemia or infarction »**exercise stress testing:** positive with catecholaminergic VT or can demonstrate ischaemia »**genetic screening:** can provide a diagnosis or help with familial screening for inherited mutations (such as mutations in genes for the cardiac ryanodine receptor or calsequestrin 2 in catecholaminergic VT)

◊ Idiopathic ventricular tachycardia: structurally normal heart

History	Exam	1st Test	Other tests
intermittent palpitations that can be associated with dizziness, diaphoresis, or dyspnoea; may be triggered by emotional stress, exercise, caffeine intake, and menstrual variation;	peripheral pulses are regular and may have variable intensity depending on degree of AV dissociation; cannon A waves are also due to AV dissociation and are highly suggestive	»**12-lead ECG:** wide-complex monomorphic tachycardia (rate commonly 90-120 bpm) with evidence of AV dissociation; no ischaemic changes present	»**transthoracic echocardiography:** normal »**TSH:** normal »**serum potassium:** normal »**serum magnesium:** normal

Common

◊ Idiopathic ventricular tachycardia: structurally normal heart

History	Exam	1st Test	Other tests
attention to symptoms that suggest ischaemic heart disease; often seen in postoperative states or after a acute coronary event followed by reperfusion	of ventricular tachyarrhythmia	Left bundle branch block morphology with an inferior axis is most common and suggests right ventricular outflow tract origin. Right bundle branch block with a left superior axis with a relatively narrow QRS (<140 ms) suggests idiopathic left ventricular tachycardia.	»**exercise stress testing:** may induce ventricular tachyarrhythmias during or after exercise, or demonstrate underlying ischaemia Inducibility with exercise is characteristic of right ventricular outflow tract VT. However, in some cases it may suppress VT. Idiopathic left ventricular tachycardia is not usually induced with exercise. »**event monitor:** can record intermittent tachyarrhythmias

Uncommon

◊ Sinus node re-entry tachycardia

History	Exam	1st Test	Other tests
rarely symptomatic, though patients may report intermittent rapid palpitations with abrupt onset or termination	normal physical examination, though the patient may have a rapid regular pulse	»**12-lead ECG:** abrupt onset of narrow-complex tachycardia (rate 100-150 bpm) with P-wave morphology similar to baseline Differentiated from atrial tachycardia and atrial flutter by P wave similar to sinus rhythm; differentiated from sinus tachycardia by abrupt onset and termination.	

Uncommon

◊ Inappropriate sinus tachycardia

History	Exam	1st Test	Other tests
often asymptomatic; symptoms can include palpitations, fatigue, exercise intolerance, anxiety, or panic attacks; no history suggestive of hyperthyroidism, infection, anaemia, volume depletion	normal physical examination except for a rapid pulse; specific attention to rule out causes of secondary sinus tachycardia such as hyperthyroidism, infection, anaemia, volume depletion (test for orthostatic hypotension)	»**12-lead ECG:** regular narrow-complex tachycardia (heart rate >100 bpm); P-wave morphology is the same as sinus rhythm.	»**transthoracic echocardiogram:** rules out structural heart disease »**24-hour Holter monitor:** elevated heart rate at rest, exaggerated heart rate elevation for degree of exertion, no change in P-wave morphology »**TSH:** normal »**FBC:** normal

◊ Permanent junctional reciprocating tachycardia

History	Exam	1st Test	Other tests
asymptomatic, though may present with palpitations or symptoms secondary to tachycardia-mediated cardiomyopathy, including malaise, oedema, and dyspnoea	normal physical examination, may have a rapid regular pulse; assess for impaired left ventricular systolic function, which may suggest a tachycardia-mediated cardiomyopathy with S3 gallop, right ventricular heave, laterally displaced point of maximal impulse, or other signs of heart failure (elevated jugular venous pressure, lower-extremity oedema)	»**12-lead ECG:** narrow-complex tachycardia (rate 120-200 bpm); negative P waves in the inferior leads, with a long RP interval due to slow retrograde atrial activation; usually initiated by a premature atrial contraction	»**transthoracic echocardiogram:** rules out structural heart disease

Torsades de pointes

History	Exam	1st Test	Other tests
may report intermittent palpitations, syncope, seizures, or cardiac arrest; may have family history of juvenile sudden death and/or a history of	variable peripheral pulse intensity and cannon A waves, resulting from AV dissociation, though often no pulse is palpable given	»**12-lead ECG:** wide-complex tachycardia with continuously varying QRS morphology; baseline ECG with a wide QT interval	»**TSH:** normal »**serum potassium:** hypo- or hyperkalaemia can predispose to VT

Uncommon

Torsades de pointes

History	Exam	1st Test	Other tests
using QT-prolonging medication; aetiology usually secondary to either congenital or acquired QT interval prolongation	haemodynamic compromise; sensorineural deafness is associated with Jervell and Lange-Nielsen syndrome (autosomal recessive long-QT syndrome); neurological examination may show focal deficits or other causes for elevated intracranial pressure	Patients with acquired long-QT syndrome with bradycardia and frequent premature ventricular contractions (PVCs) are at greatest risk of developing torsades de pointes (TdP). TdP is often initiated by a short-long-short sequence (sinus bradycardia, PVC, sinus pause, PVC, initiation of TdP). In patients experiencing recurrent bouts of TdP, temporary pacing may help prevent the short-long-short triggers of TdP. Many drugs can be associated with acquired-long QT syndrome, including anti-arrhythmic agents, macrolide antibiotics, non-sedating antihistamines, psychotropic medications, and certain gastric motility agents. [AZCERT: QT drugs list] (https://www.crediblemeds.org/everyone/composite-list-all-qtdrugs)	»**serum magnesium:** hypomagnesaemia can predispose to VT »**cardiac biomarkers:** positive cardiac biomarkers with new ischaemia »**transthoracic echocardiography:** heart failure or ventricular hypertrophy predispose to drug-induced TdP »**exercise stress testing:** lack of appropriate QTc interval shortening »**genetic screening:** genetic mutation specific to the syndrome Limited diagnostic value given numerous possible mutations, though it can be used to screen family members, to guide specific therapy (based on mutation type), or in cases with borderline clinical diagnostic criteria.

Uncommon

Bidirectional ventricular tachycardia

History	Exam	1st Test	Other tests
assessment is time-critical, as delay in treatment may be fatal; digitalis toxicity or history of syncope in patient; history of sudden death in family members	rapid regular pulse, if palpable, due to hypotension; signs of hypoperfusion may be present, including changes in mental status	»**12-lead ECG:** wide-complex tachyarrhythmia with alternating morphologies (often with an alternating axis shift) and a regular R-R interval Ventricular bigeminy will have varying R-R intervals.	»**digitalis level:** elevated

◊ Accelerated idioventricular rhythm

History	Exam	1st Test	Other tests
gradual onset and termination; symptoms consistent with acute myocardial infarction or history of angina; increased risk after thrombolytics or percutaneous coronary intervention for cardiac ischaemia; history of digitalis use should be investigated, as accelerated idioventricular rhythm (AIVR) can suggest digitalis toxicity	bradycardia or mild tachycardia is possible with possible irregular rhythm (intermittent sinus capture); patient may be hypotensive given the lack of atrioventricular synchrony, and there may be evidence of AV dissociation (cannon A waves or carotid pulse intensity variation)	»**12-lead ECG:** wide-complex rhythm (HR 40-120 bpm) with gradual acceleration beyond the sinus rate; may have intermittent sinus capture beats	»**transthoracic echocardiography:** may demonstrate regional wall-motion abnormalities or valvular dysfunction »**serum potassium:** hypokalaemia predisposes to AIVR »**serum magnesium:** hypomagnesaemia predisposes to AIVR »**serum urea:** if elevated can predispose to digoxin toxicity »**serum creatinine:** if elevated can predispose to digoxin toxicity »**digoxin level:** elevated Digoxin toxicity can be a cause.

Uncommon

Postural orthostatic tachycardia syndrome

History	Exam	1st Test	Other tests
symptoms of orthostatic intolerance include: palpitations, lightheadedness, blurred vision, exercise intolerance (which may also be a non-orthostatic feature of POTS), presyncope and syncope, tremor, generalised weakness, fatigue (which may also be a non-orthostatic feature of POTS); non-orthostatic symptoms include: dyspnoea, gastrointestinal symptoms, exercise intolerance, fatigue, headache, sleep disturbance, cognitive impairment, chest pain, bladder disturbance; may be symptoms of associated comorbidities, such as those of Ehlers-Danlos syndrome and autoimmune diseases, particularly Hashimoto's thyroiditis and celiac disease	irregular heart rate, tachycardia, increased respiratory rate, generalised weakness; may be signs of associated comorbidities, such as those of Ehlers-Danlos syndrome and autoimmune diseases, particularly Hashimoto's thyroiditis and celiac disease	»**10 minute standing test:** heart rate typically increases by ≥30 bpm (≥40 bpm in patients aged 12 to 19 years old) after changing position from supine to standing, and no orthostatic hypotension (sustained drop in systolic blood pressure by ≥20 mmHg) Allow at least 5 minutes of a supine position and at least 1 minute of standing before checking orthostatic vital signs. Standing heart rate and blood pressure check can be repeated at 3, 5, and 10 minutes as changes are often not apparent after 1 minute of standing in practice. In cases of hyperadrenergic POTS, patients will have orthostatic hypertension (increase in systolic blood pressure ≥10 mmHg after standing for 10 minutes).	»**24-hour Holter monitor:** demonstrates the association between tachycardia and orthostatic changes Tilt-table test may be used if the diagnosis is unclear following initial assessment or if the patient is not able to perform a 10 minute standing test. Referral to a center experienced with the autonomic testing of POTS may be considered for further investigation of underlying pathology.

Guidelines

United Kingdom

Atrial fibrillation: diagnosis and management (https://www.nice.org.uk/guidance/NG196)

Published by: National Institute for Health and Care Excellence
Last published: 2021

Europe

2022 ESC Clinical Practice Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death (https://www.escardio.org/Guidelines)

Published by: European Society of Cardiology
Last published: 2022

Management of supraventricular arrhythmias: a consensus document (https://www.escardio.org/Guidelines/Consensus-and-Position-Papers)

Published by: European Heart Rhythm Association
Last published: 2017

North America

2023 ACC/AHA/ACCP/HRS guideline for the diagnosis and management of atrial fibrillation: a report of the American College of Cardiology/American Heart Association Joint Committee on clinical practice guidelines (https://www.acc.org/guidelines)

Published by: American Heart Association; American College of Cardiology; Association Joint Committee on Clinical Practice Guidelines
Last published: 2023

2020 Canadian Cardiovascular Society/Canadian Heart Rhythm Society position statement on the management of ventricular tachycardia and fibrillation in patients with structural heart disease (https://www.chrsonline.ca/resources-publications/guidelines-clinical-updates)

Published by: Canadian Cardiovascular Society/Canadian Heart Rhythm Society
Last published: 2020

North America

The 2020 Canadian Cardiovascular Society/Canadian Heart Rhythm Society comprehensive guidelines for the management of atrial fibrillation (https://www.chrsonline.ca/resources-publications/guidelines-clinical-updates)

Published by: Canadian Cardiovascular Society/Canadian Heart Rhythm Society
Last published: 2020

2017 AHA/ACC/HRS guideline for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death (http://www.acc.org/guidelines)

Published by: American College of Cardiology; American Heart Association; Heart Rhythm Society
Last published: 2017

2015 ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia (http://www.acc.org/guidelines)

Published by: American College of Cardiology; American Heart Association; Heart Rhythm Society
Last published: 2015

Online resources

1. AZCERT: QT drugs list *(external link) (https://www.crediblemeds.org/everyone/composite-list-all-qtdrugs)*

Key articles

- Page RL, Joglar JA, Caldwell MA, et al. 2015 ACC/AHA/HRS guideline for the management of adult patients with supraventricular tachycardia: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. J Am Coll Cardiol. 2016 Apr 5;67(13):e27-115. Full text (https://www.sciencedirect.com/science/article/pii/S0735109715058404?via%3Dihub) Abstract

- Katritsis DG, Boriani G, Cosio FG, et al. European Heart Rhythm Association (EHRA) consensus document on the management of supraventricular arrhythmias, endorsed by Heart Rhythm Society (HRS), Asia-Pacific Heart Rhythm Society (APHRS), and Sociedad Latinoamericana de Estimulación Cardiaca y Electrofisiologia (SOLAECE). Europace. 2017 Mar 1;19(3):465-511. Full text (https://academic.oup.com/europace/article/19/3/465/2631183) Abstract

- Joglar JA, Chung MK, Armbruster AL, et al. 2023 ACC/AHA/ACCP/HRS guideline for the diagnosis and management of atrial fibrillation: a report of the American College of Cardiology/American Heart Association Joint Committee on clinical practice guidelines. Circulation. 2024 Jan 2;149(1):e1-156. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC11095842) Abstract

- Sheldon RS, Grubb BP 2nd, Olshansky B, et al. 2015 heart rhythm society expert consensus statement on the diagnosis and treatment of postural tachycardia syndrome, inappropriate sinus tachycardia, and vasovagal syncope. Heart Rhythm. 2015 Jun;12(6):e41-63. Full text (https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5267948)

- Al-Khatib SM, Stevenson WG, Ackerman MJ, et al. 2017 AHA/ACC/HRS guideline for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. Circulation. 2018 Sep 25;138(13):e272-391. Full text (https://www.ahajournals.org/doi/10.1161/CIR.0000000000000549) Abstract

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Images

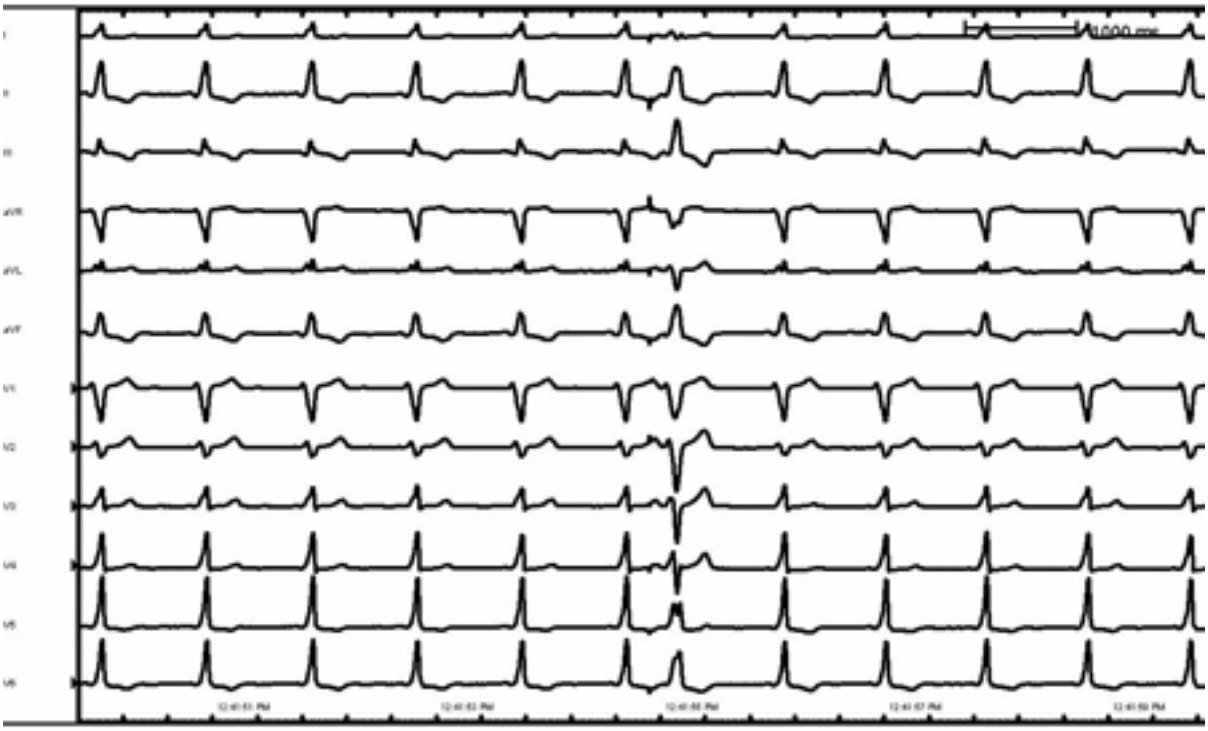

Figure 1: Sinus rhythm with pre-excitation

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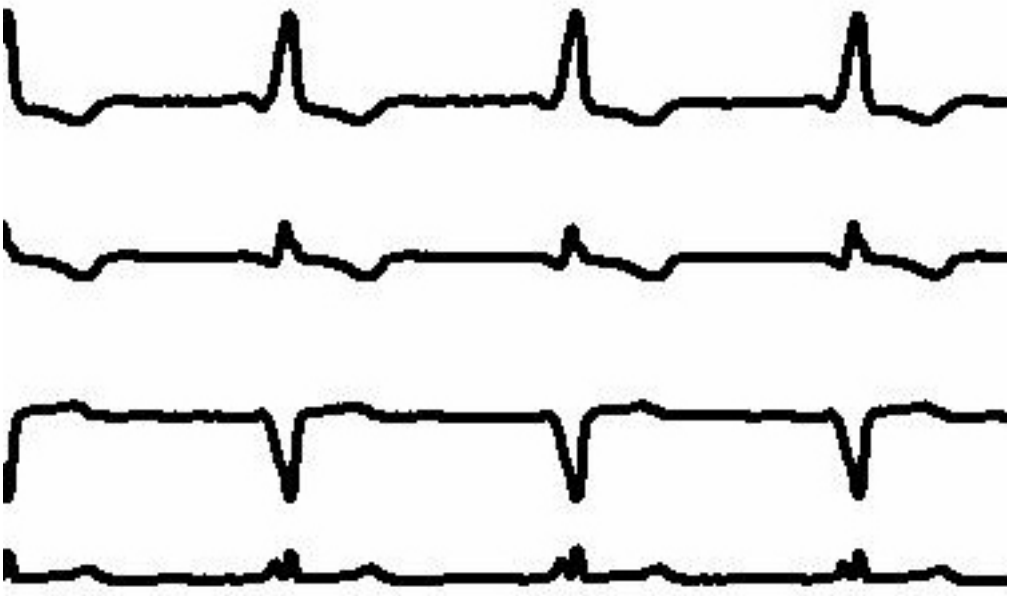

Figure 2: Sinus rhythm with pre-excitation (detail)

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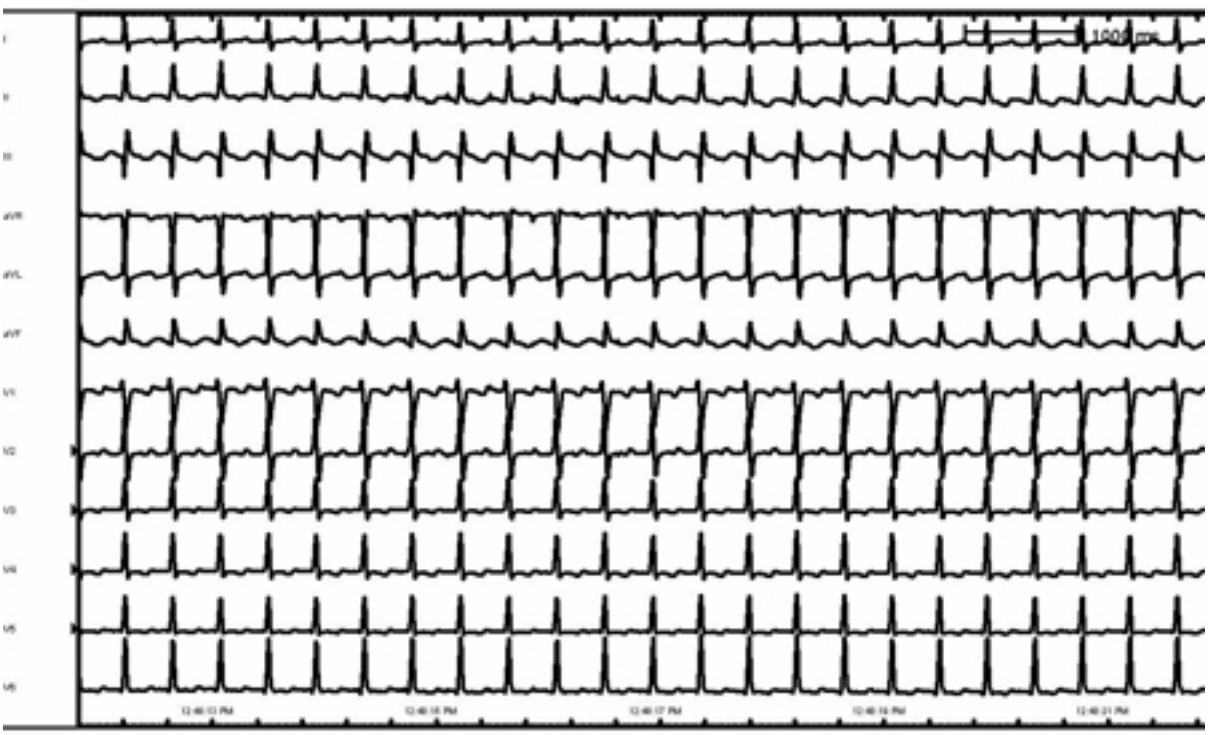

Figure 3: Atrial flutter

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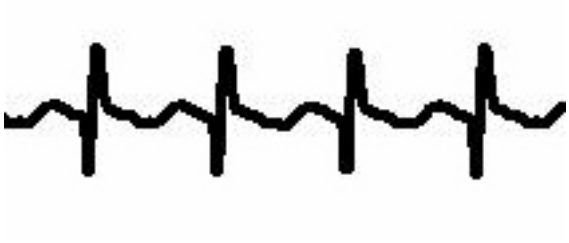

Figure 4: Atrial flutter (detail)

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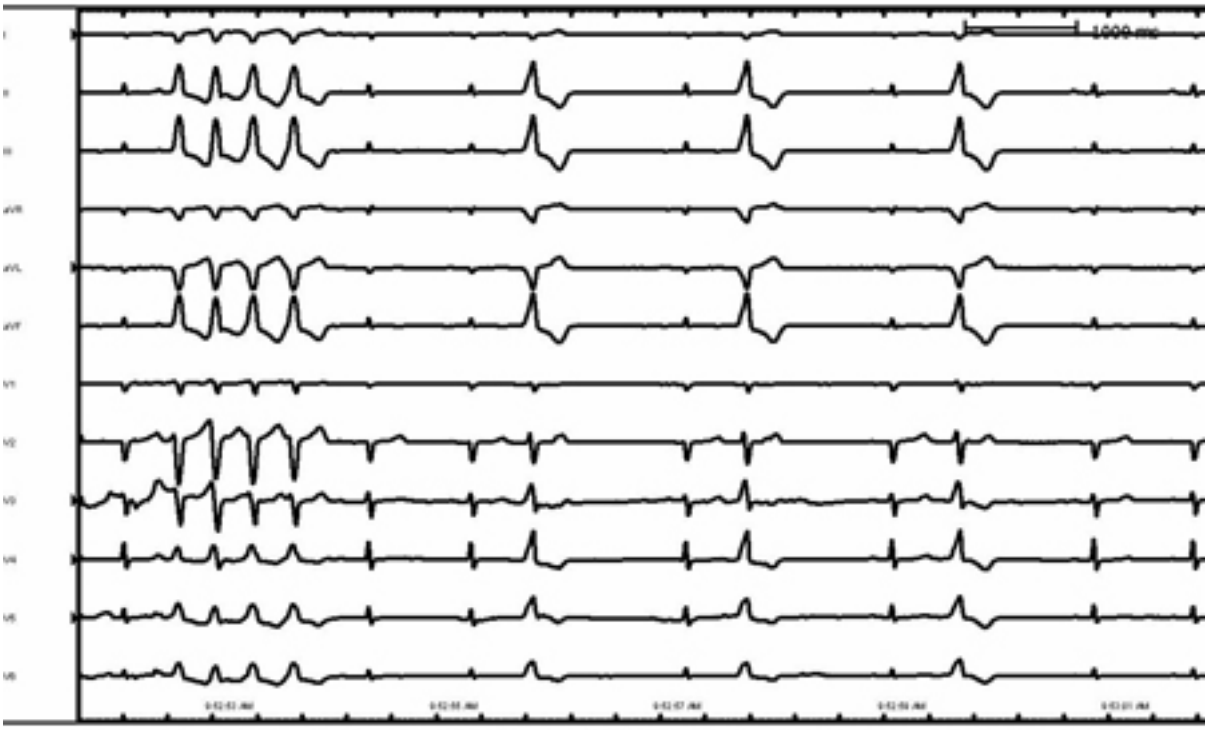

Figure 5: Right ventricular outflow tract ventricular tachycardia

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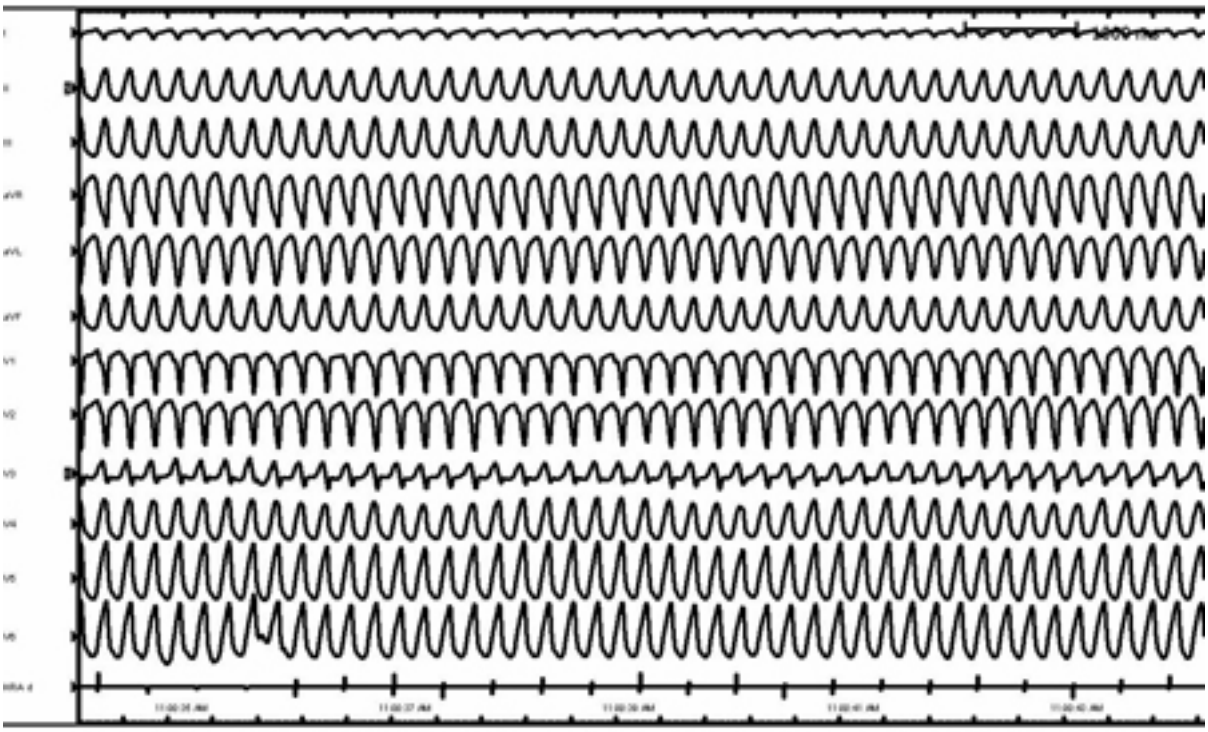

Figure 6: Ventricular tachycardia in a patient with arrhythmogenic right ventricular cardiomyopathy

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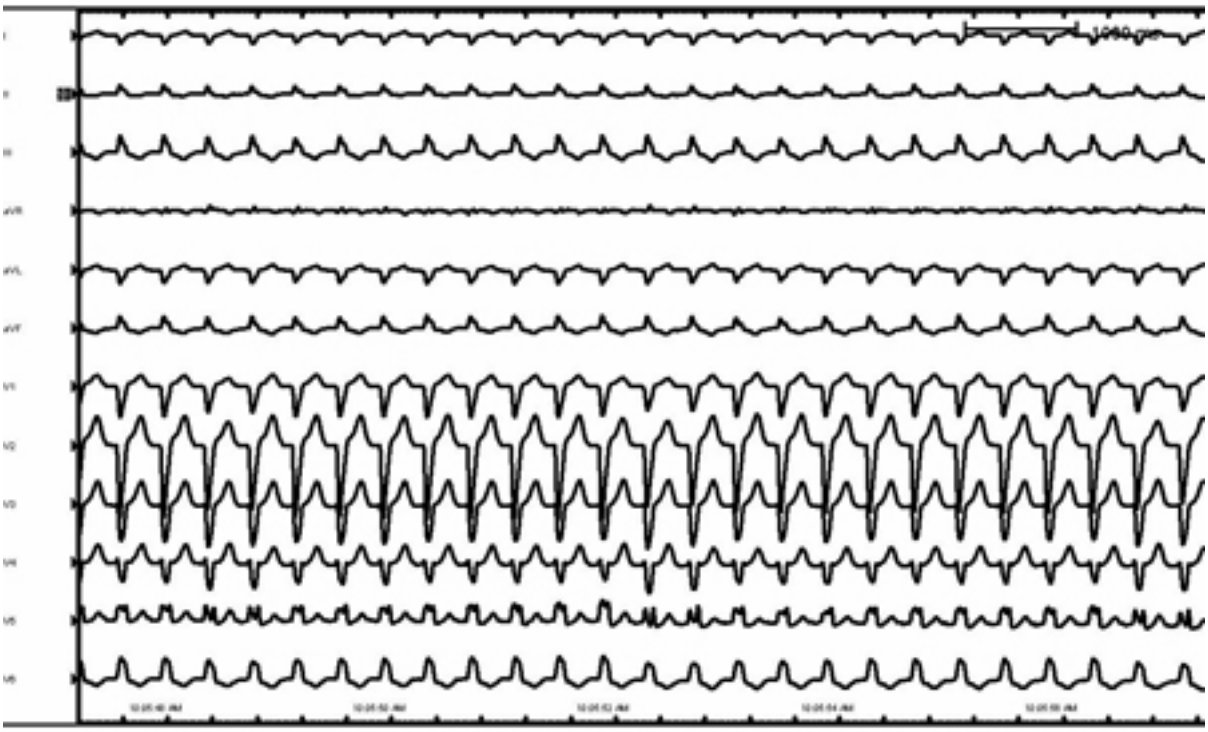

Figure 7: Supraventricular tachycardia with aberrancy and left bundle branch block

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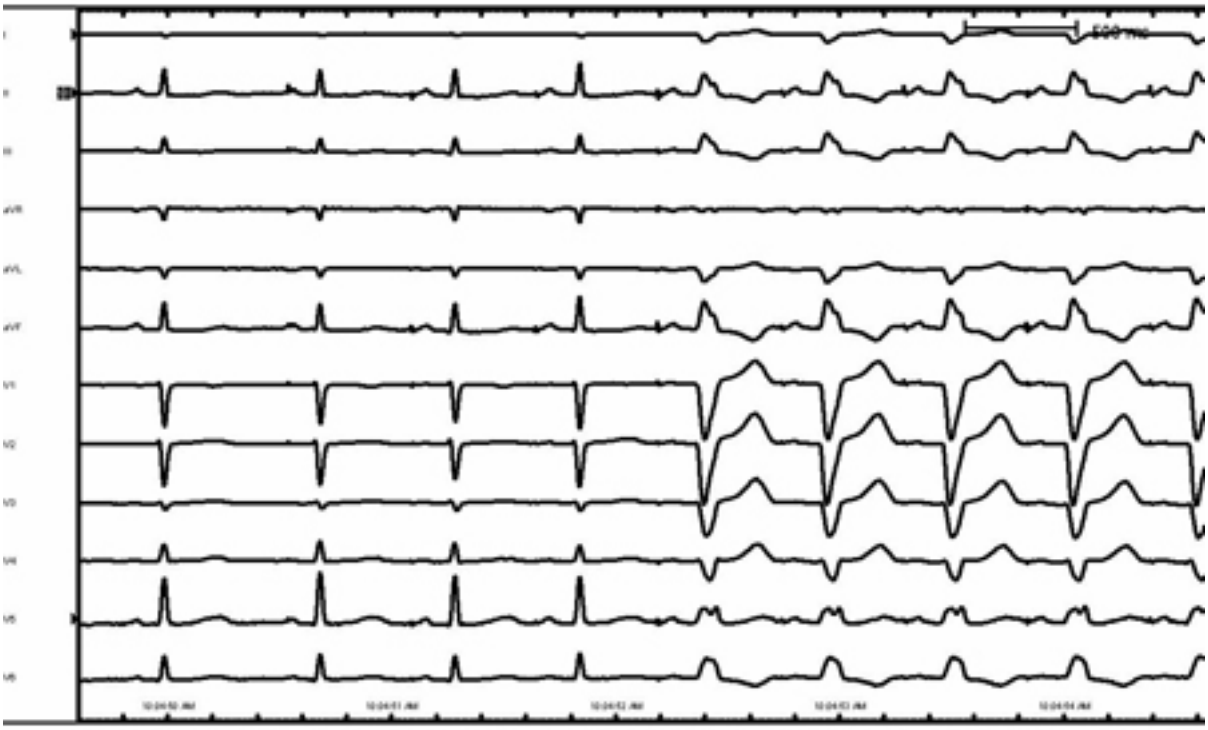

Figure 8: Rate-related left bundle branch block

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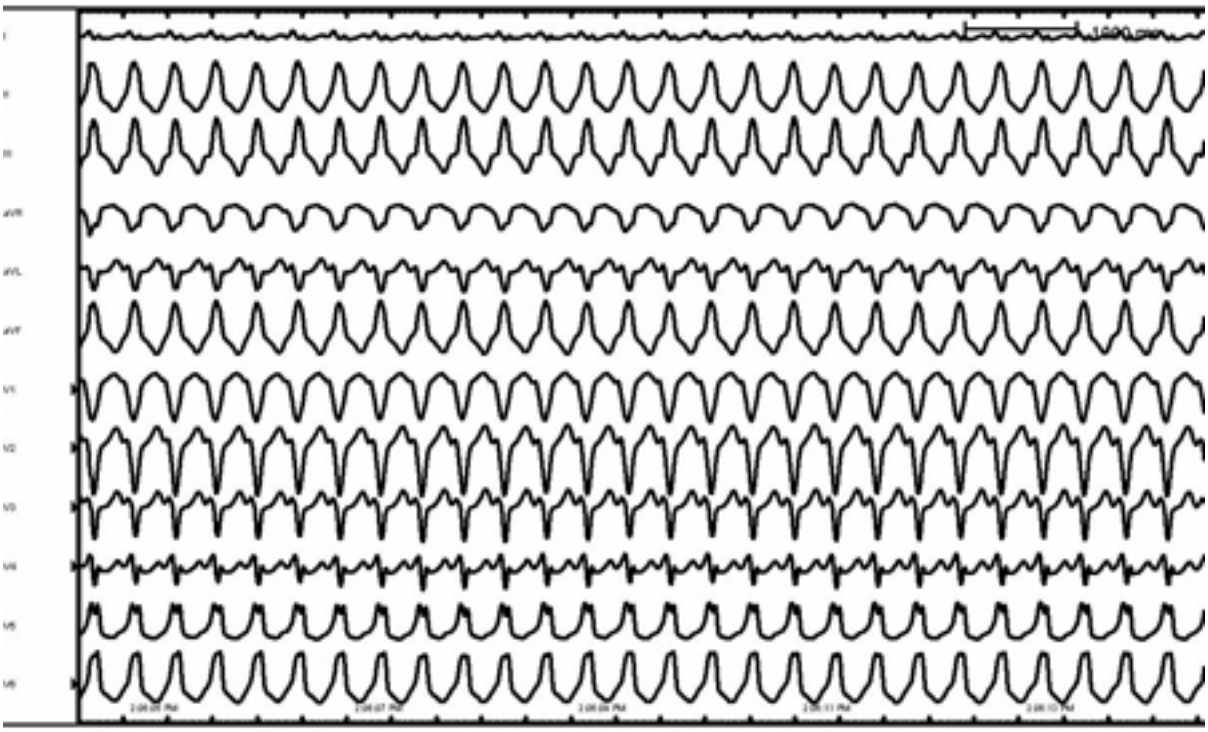

Figure 9: Antidromic re-entrant tachycardia

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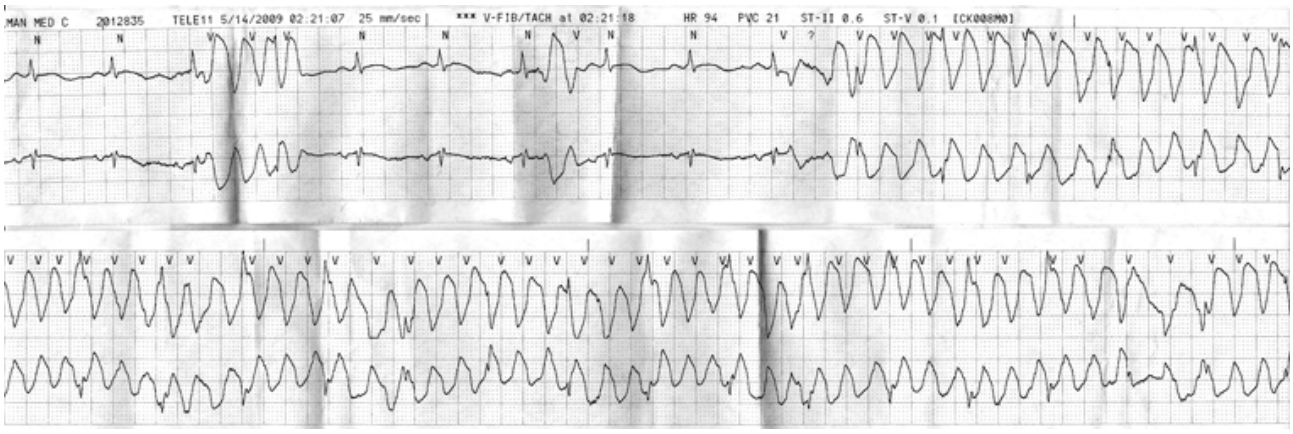

Figure 10: Artifact overlying sinus rhythm

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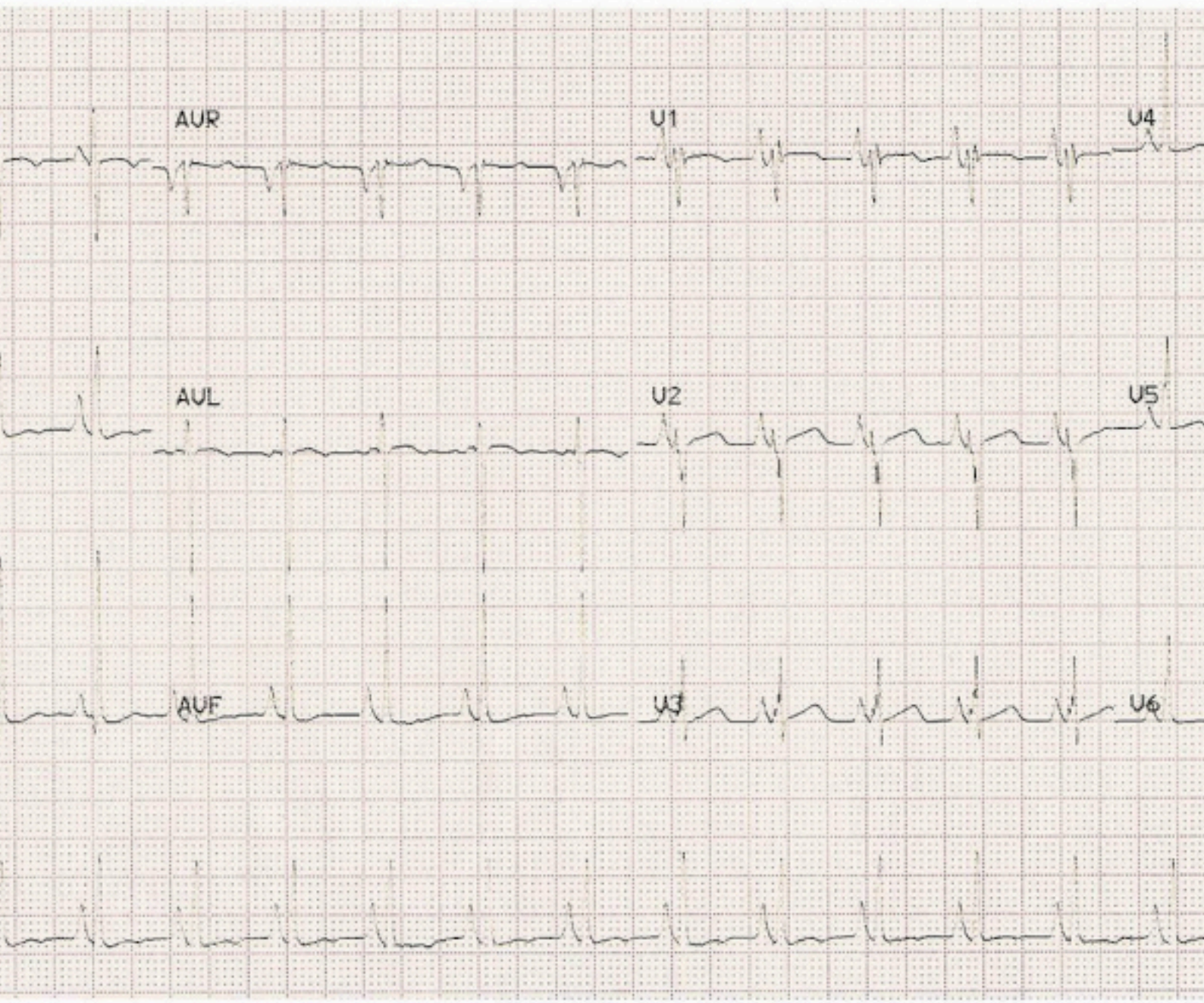

Figure 11: ECG example of sinus tachycardia

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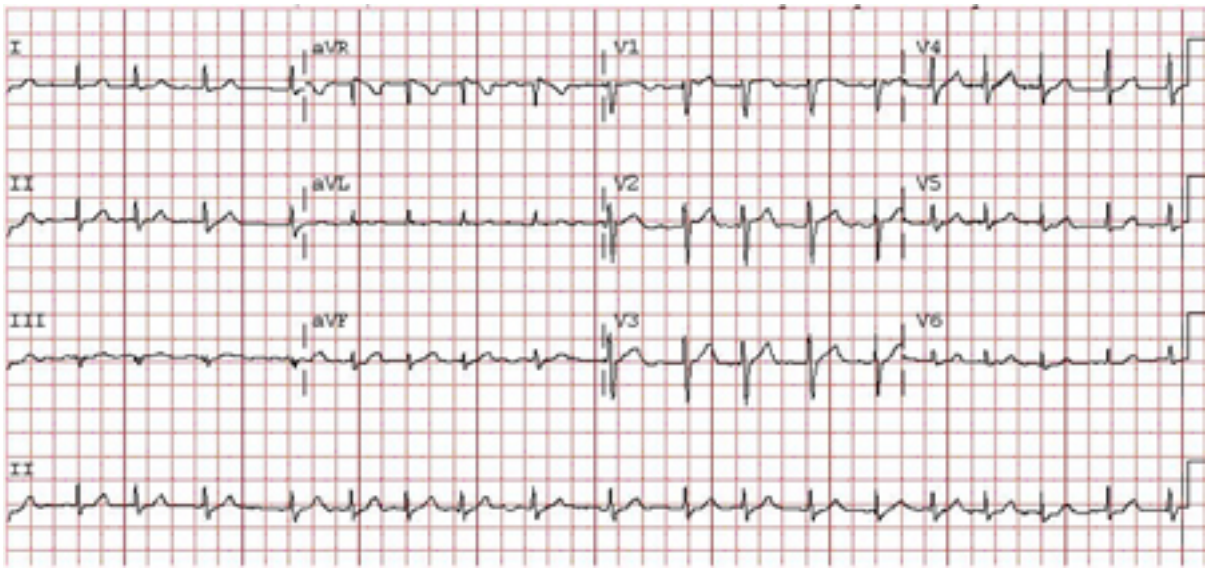

Figure 12: Atrial fibrillation: P waves are not discernible; the ventricular (QRS complexes) rate is irregularly irregular

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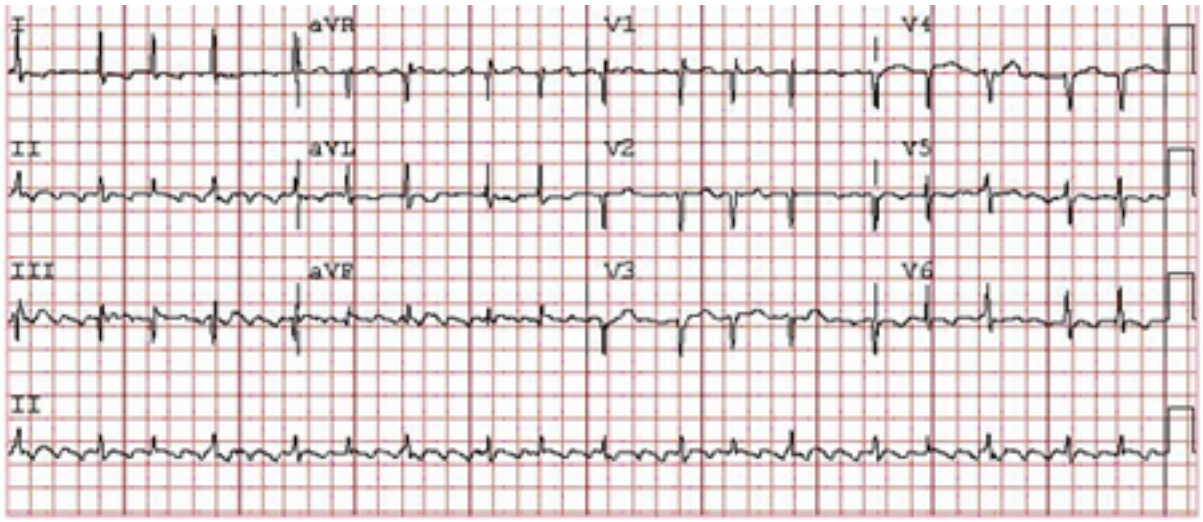

Figure 13: Atrial flutter: typical saw-tooth appearance of the flutter waves in the inferior leads (leads II, III, and aVF) indicates typical counterclockwise atrial flutter; the ventricular (QRS complexes) rate is variable

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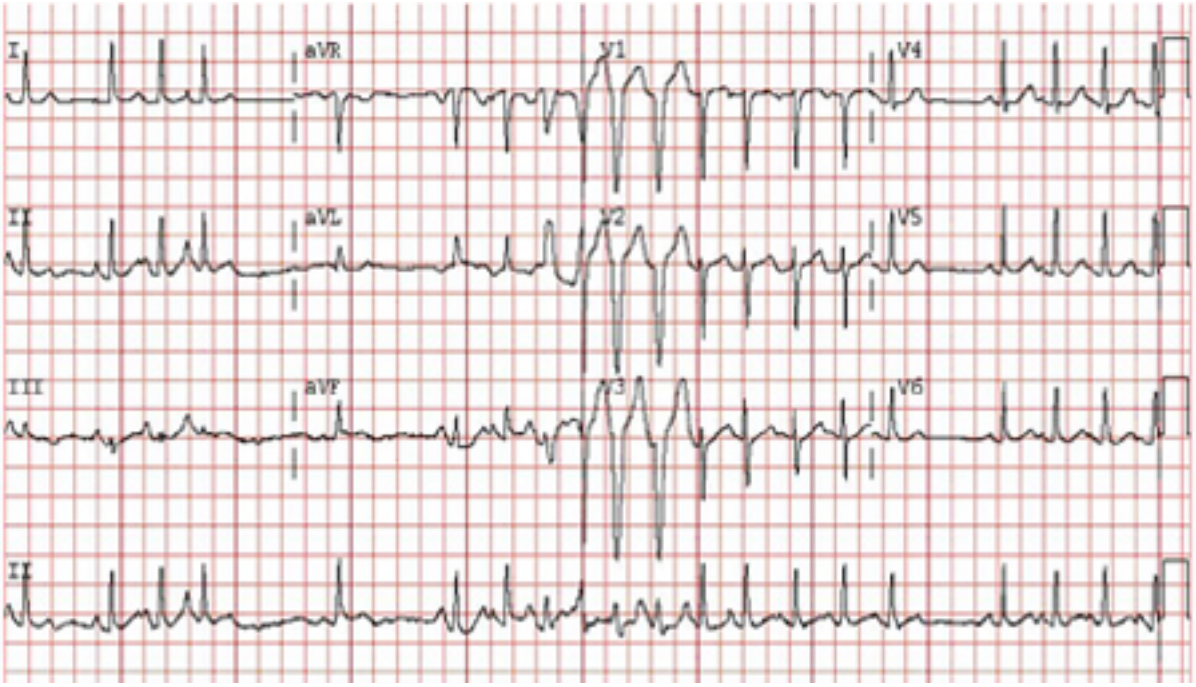

Figure 14: Atrial tachycardia: bursts of atrial tachycardia (10 beats in the middle section of the rhythm strip II at bottom) follows sinus complexes

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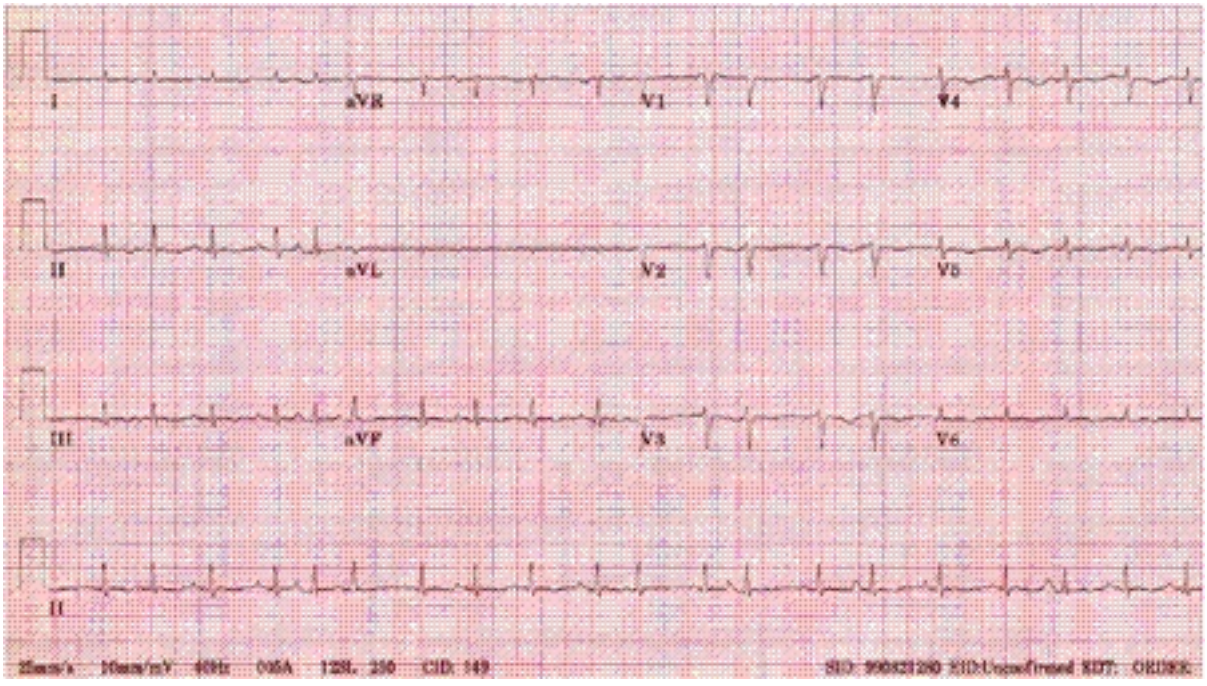

Figure 15: Multifocal atrial tachycardia

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Figure 1 – BMJ Best Practice Numeral Style

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