

Case 4 A 60-year-old man with a broad complex tachycardia

The doctor working in the accident and emergency department phones to refer a 60-year-old man who has been brought into hospital having collapsed at home. Mr Garth Dale became unwell while gardening, when he suddenly felt breathless with light-headedness and a feeling of chest tightness. On arrival of the ambulance Mr Dale was noted to be pale, sweaty and tachycardic with a pulse of 160 beats/min and blood pressure 100/50. The cardiac monitor has shown a broad complex tachycardia (BCT).

What is a broad complex tachycardia?

- BCT is defined as a pulse rate >100 beats/min, and where the QRS complexes are more than 120 ms in duration (>3 mm on standard ECG settings)
- BCT results from either ventricular tachycardia (VT) or supraventricular tachycardia (SVT) combined with a bundle branch block (BBB) (see Box 9)
- BCT is a potentially life-threatening medical emergency, particularly when caused by VT. Loss of cardiac output ('pulseless VT') or degeneration into ventricular fibrillation can result in cardiopulmonary arrest
- Although generally less serious than VT, tachycardia caused by SVT can also result in reduced cardiac output because of reduced cardiac filling time during diastole

On your arrival in the emergency department Mr Dale remains unwell. Mr Dale is conscious and breathing spontaneously with a respiratory rate of 20/min. He is complaining of central chest tightness and a feeling of breathlessness. Oxygen saturation is 99% while breathing oxygen via a re-breath mask. Mr Dale's pulse feels weak and thready, but regular with a rate >160 beats/min, which is confirmed on the cardiac monitor. Blood pressure is now 90/40.

What immediate management is required?

- **Administer oxygen** (15 L via a re-breath mask)
- **Establish intravenous access:** if a cannula has already been inserted, confirm that it is patent
- **Administer opiate analgesia if required** (e.g. morphine sulphate 1–5 mg i.v. until pain is controlled) combined with an antiemetic (e.g. cyclizine 50 mg i.v.)
- **Ensure continuous cardiac monitoring, pulse oximetry, regular measurement of blood pressure and assessment of conscious level**
- **Perform a 12-lead ECG**
- **Request a chest radiograph**
- **Send venous blood to the laboratory** for full blood count (FBC), urea and electrolytes (U&E), liver function tests (LFT), serum magnesium level, thyroid function and cardiac enzymes (e.g. troponin – may need to be repeated later if patient is presenting less than 12 h after the onset of symptoms)
- **Try to obtain any previous hospital records**, including any old ECG readings

Provided the patient's condition is not deteriorating it is important to obtain further history and conduct a clinical examination, including careful inspection of the ECG. This must be done quickly.

Mr Dale describes feeling well before the episode of collapse 2 h ago. He has been experiencing episodes of light-headedness, associated with a feeling of 'palpitations' every few days for the past month, although these usually last a few seconds only and have never resulted in collapse or loss of consciousness. Between the attacks Mr Dale has felt well. Mr Dale had a myocardial infarct one year ago, for which he was treated with thrombolytic drugs and subsequently underwent coronary angiography and stent insertion. Mr Dale has not experienced any chest pain or breathlessness since that time. He has no other significant past medical history. Mr Dale takes aspirin, clopidogrel, bisoprolol, ramipril and simvastatin. He is an ex-smoker and only occasionally drinks alcohol.

Box 9 Mechanisms of broad complex tachycardia

The QRS complex represents the wave of depolarisation travelling across the ventricular wall, resulting in contraction of the ventricle (ventricular systole). The width of the QRS complex is determined by the speed at which the wave travels through the ventricle.

Specialised conducting fibres in the interventricular septum (the left and right bundles of His) carry the wave at the maximum speed; when the impulse travels by this route the calibre of the complex is usually <120 ms. When the cardiac impulse arises 'above' the atrioventricular node (AVN), it is able to enter the bundle of His resulting in a narrow-calibre QRS complex.

- **For this reason narrow-calibre QRS complexes (and therefore narrow complex tachycardias) are almost always supraventricular in origin.**

On occasion, conduction of the impulse through the bundle of His is slower than normal. The usual cause for this is an abnormality of one of the bundles, termed a left

or right bundle branch block. The result is that the QRS complex becomes wider than normal, resulting in a 'broad' complex (see p. 24).

- **If a supraventricular tachycardia is combined with a left or right bundle branch block the result is a BCT.**

Broad complexes may also occur when the origin of the impulse is in the ventricular wall, outside the normal conducting system. All cardiac myocytes are capable of producing spontaneous electrical activity, although this is normally 'overridden' by the beats arising within the pacemaker cells. When a QRS complex arises from within the ventricular wall, the resulting wave of depolarisation travels relatively slowly across the myocardium, resulting in a broad complex. A tachycardia arising in the ventricular wall will therefore almost always comprise broad complexes.

- **Ventricular tachycardia is almost always a broad complex tachycardia.**

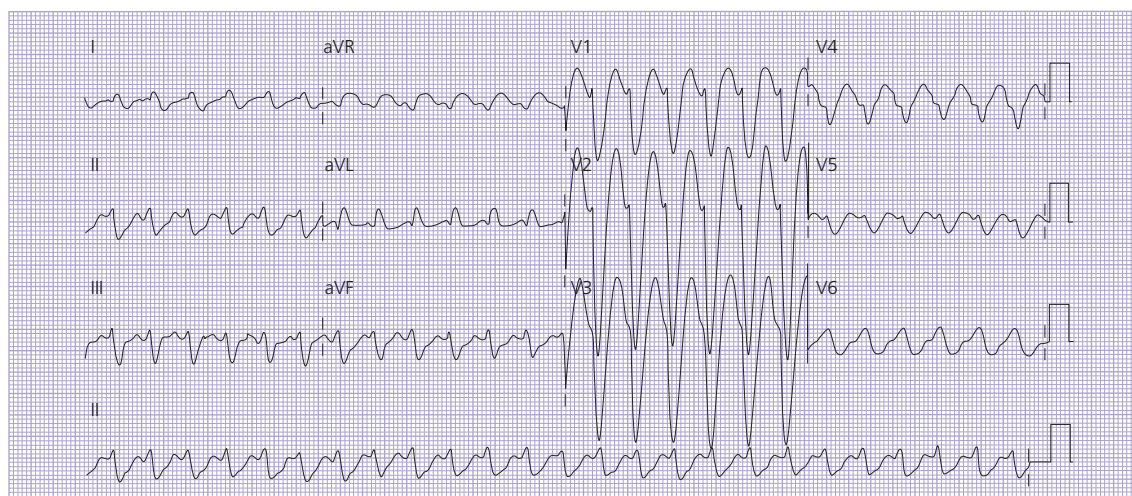


Figure 26 ECG taken on arrival; note the broad complexes and concordance of electrical polarity in leads v1–v6.

Examination reveals the persistent tachycardia with hypotension as described above. There are bibasal crepitations and mild pitting ankle oedema.

Mr Dale's serum potassium level is slightly low at 3.15 mmol/L; other blood results are normal, including serum magnesium.

The ECG is shown in Fig. 26.

Is the tachycardia caused by VT or SVT with bundle branch block?

Clinical clues to the cause of the tachycardia are described in Table 26.

- In this case the prior history of ischaemic heart disease combined with the significant haemodynamic compromise are both suggestive of VT as the cause for the BCT

Table 26 Clinical clues to distinguish ventricular tachycardia (VT) from supraventricular tachycardia (SVT) with bundle branch block

	VT	SVT	Comments
History	<p>Previous VT</p> <p>Recent MI, previous IHD or any other structural heart disease makes VT much more likely than SVT; BCT in patients with previous IHD or any other structural heart disease is VT in >95% of cases</p> <p>Often older age group (e.g. age >40)</p>	<p>Previous documented SVT or cardioversion of arrhythmia with AVN blocking agents (e.g. adenosine)</p> <p>Often younger age group (e.g. age <40)</p>	<p>Patients with VT tend to be more unwell than those with SVT, although this cannot be relied upon: patients with SVT can be severely compromised if the rate is very rapid, while some patients can tolerate VT for many hours without significant haemodynamic effects</p>
Examination	<p>Often severe haemodynamic compromise with hypotension, raised JVP and pulmonary oedema</p> <p>'Cannon' waves sometimes seen in JVP due to contraction of right atrium against closed tricuspid valve</p> <p>Pulse rate rarely >200/min</p>	<p>Usually better tolerated than VT, unless rate very rapid</p>	<p>Any patient who is haemodynamically compromised should be treated as having VT until proven otherwise</p> <p>Patients with a well-tolerated tachycardia can usually undergo diagnostic tests provided resuscitation equipment is rapidly available if required</p>

AVN, atrioventricular node; IHD, ischaemic heart disease; JVP, jugular venous pressure.

Further clues can be obtained from close inspection of the patient's ECG. Helpful distinguishing features of the ECG in VT and SVT with BBB are given in Table 27.

This patient's ECG would support the diagnosis of VT on the basis of:

- QRS complex width >150 ms
- concordance across chest leads

Administration of intravenous adenosine is worth considering if there is any ongoing doubt about the diagnosis (see Box 10), provided there are no contraindications.

Adenosine is not considered appropriate in this case, given the high likelihood of VT combined with the history of asthma.

What are the treatment options if the likely diagnosis is VT?

Restoration of sinus rhythm ('cardioversion') should be achieved as soon as possible if VT is considered likely, as in this case. Options to achieve this include:

- Pharmacological cardioversion, using drugs such as amiodarone
- Electrical cardioversion, by administration of a DC shock to the chest wall

Electrical cardioversion is the most rapid and effective method, and is therefore the required treatment when the

Table 27 ECG clues to distinguish ventricular tachycardia from supraventricular tachycardia with bundle branch block

Features which make ventricular tachycardia more likely

'Concordance' of polarity across the chest leads (all QRS complexes across chest leads point in the same direction – see Fig. 26)

Broader QRS complexes (especially if >150 ms)

P-waves visible but dissociated from QRS complexes

Capture beats

Fusion beats

Features which make supraventricular tachycardia more likely

QRS duration 120–150 ms

Rate >200 beats/min

Classic left or right bundle branch block morphology

P-wave visible (or inverted) and associated with QRS complex

Resolution following administration of adenosine

patient is pulseless, severely compromised or rapidly deteriorating. If patients are conscious they will usually require general anaesthesia, which will have some risk attached in this clinical setting. For this reason, if

Box 10 Administration of intravenous adenosine

Intravenous administration of adenosine results in rapid and total blockage of the atrioventricular node. Because of the short half-life of the drug this only lasts for a few seconds. However, if a tachycardia is supraventricular in origin the rate will usually be temporarily slowed. In cases where the tachycardia has arisen within the atrioventricular node (e.g. nodal re-entrant tachycardia), adenosine may result in cardioversion to sinus rhythm.

Procedure for administration

- confirm no contraindications (see below)
- site a cannula in a large peripheral vein (e.g. antecubital fossa)
- warn the patient that they may experience a feeling of extreme light-headedness or chest pain following administration (although this should be extremely short-lived)
- ensure the patient is connected to a cardiac monitor with the ability to print the trace for later review if required: press the 'print' button immediately before administration of the drug
- administer 6 mg by rapid intravenous bolus, followed by 20 mL of normal saline flush
- if there is no change in the rate or rhythm after 2 min, administer a further 12 mg of the drug.

If no response is seen after administration of 12 mg it is likely that the tachycardia is ventricular in origin; if there is still a strong clinical suspicion of supraventricular origin it may be worth administering an 18 mg bolus if there is no response after 12 mg.

Contraindications to adenosine

- severe haemodynamic compromise
- asthma: may cause acute bronchospasm
- Wolf–Parkinson–White (WPW) syndrome: may result in paradoxical increase in heart rate – discuss with local cardiology team before use of adenosine in patients with known or suspected WPW
- Denervated heart following cardiac transplantation.

a patient is more stable, as in this case, an attempt at pharmacological cardioversion is usually made in the first instance.

Intravenous amiodarone is the usual drug of choice for **pharmacological cardioversion**, and has the added advantage of increasing the sensitivity of the myocardium to, and therefore the effectiveness of, subsequent electrical cardioversion, if required.

In addition to these measures it is important to correct any potentially contributing factors (e.g. electrolyte abnormalities).

Mr Dale is given an intravenous infusion of 300 mg of amiodarone over 30 min via a cannula in the antecubital fossa. A second cannula is sited and an infusion of 500 ml of normal saline containing 3 g of potassium chloride is commenced, infusing over 3 h.

The heart rhythm is unchanged 10 min after completion of the amiodarone infusion. Furthermore, Mr Dale now appears more breathless. His oxygen saturation has fallen to 94% despite high-flow oxygen, and his blood pressure has fallen to 85/40.

The on-call anaesthetist is contacted and attends urgently. After informed consent, Mr Dale is anaesthetised and undergoes synchronised DC cardioversion. After two shocks at 200 J the sinus rhythm is restored.

What further investigation is required after restoration of sinus rhythm?

It is crucial to establish whether there is a treatable underlying cause for the arrhythmia. This will help to guide further treatment. If no clear reversible cause is identified, it is likely that the patient will require long-term anti-arrhythmic therapy or insertion of an implanted cardiac defibrillator (ICD).

- Repeat the ECG
- Recheck electrolytes
- Check cardiac enzymes (e.g. troponin I, 12 h after onset of the original collapse)
- Review other blood test results (e.g. thyroid function) when available

The repeat ECG shows sinus rhythm with left BBB at a rate of 70 beats/min. Mr Dale's blood pressure is now 130/70 and he looks and feels much better. The repeat serum potassium level is 4.1 mmol/L after completion of the saline/potassium infusion. Cardiac troponin is slightly elevated at 0.75 ng/mL (normal <0.15 ng/mL), 12 h after admission.

How should the elevated troponin be interpreted in this situation?

Elevated troponin indicates some degree of myocardial damage; this may have resulted from:

- Myocardial ischaemia or infarction prior to Mr Dale's admission

- Rate-related ischaemia: reduced coronary artery filling during the shortened diastole resulting from the tachyarrhythmia
- Minor myocardial damage during electrical cardioversion

It is likely that Mr Dale will require coronary angiography and electrophysiological studies, which may be required prior to discharge from hospital. This will help to determine his requirement for an ICD. Mr Dale should be referred for review by a cardiologist in the hospital or regional centre.

What further monitoring/treatment is required while awaiting cardiology review?

- Maintain the patient on continuous cardiac monitoring
- Restart the patient's regular medication (especially the antiplatelet therapy – aspirin and clopidogrel)

- Commence regular low molecular weight heparin (e.g. enoxaparin 1 mg/kg twice daily)
- Commence regular oral amiodarone (initially 200 mg tds for one week; this will then be reduced to 200 mg twice daily and then once daily after a further week)

Mr Dale is reviewed by the cardiologist, who agrees to transfer him to a bed on the cardiac unit as soon as possible. Mr Dale is prescribed oral amiodarone and enoxaparin as well as his regular medication. He remains in sinus rhythm on his cardiac monitor. Mr Dale undergoes cardiac catheterisation 24 h after admission, which reveals a significant stenosis in his left anterior descending artery; a stent is inserted with good effect. At the same time, an ICD is sited enabling Mr Dale to discontinue treatment with amiodarone. Mr Dale is discharged from hospital after five days of inpatient observation having had no further arrhythmias.

CASE REVIEW

A 60-year-old man with a past history of ischaemic heart disease is brought to the accident and emergency department after collapsing in his garden. The admission ECG reveals a broad complex tachycardia. Mr Garth Dale is hypotensive and sweaty with cool peripherae. Examination reveals signs consistent with mild pulmonary oedema. Examination of the ECG reveals broad complexes in a left BBB pattern and a heart rate of 160 beats/min. There is also concordance of polarity across the chest leads. Combined with the past history of ischaemic heart disease and haemodynamic compromise, it is felt that the BCT is likely to reflect ventricular tachycardia.

Mr Dale is noted to have low potassium and he is given infusions of amiodarone and saline with potassium via a separate cannula. Mr Dale's rhythm is unchanged and his condition appears to be deteriorating, with falling blood pressure and increasing respiratory rate. He therefore undergoes emergency synchronised DC cardioversion, which restores sinus rhythm on the second shock at 200 J.

Following restoration of sinus rhythm, Mr Dale is noted to have a left bundle branch block and slightly elevated

troponin 12 h after admission to hospital. He starts regular low molecular weight heparin and oral amiodarone, pending transfer to the cardiac ward. Mr Dale undergoes cardiac catheterisation 24 h after admission, revealing a significant stenotic lesion in his left anterior descending artery, which is stented by the cardiologists. An implantable cardiac defibrillator is also inserted.

This case highlights some of the diagnostic dilemmas encountered when managing a patient with a BCT. In some cases the distinction between VT and SVT with BBB is much less clear-cut. When the ECG is more suggestive of SVT or if the patient's condition is stable, it is often advisable to administer a short-acting atrioventricular node (AVN) blocking agent, such as adenosine. This is given by rapid intravenous bolus injection and produces temporary blockage of the AVN. Incremental doses of 6, 12 and, sometimes, 18 mg are given; if the patient has a supraventricular tachycardia, the rate will be slowed or reverted to sinus rhythm. There will be no response in VT, where the rhythm is generated from below the AVN. This drug therefore acts as both a diagnostic and therapeutic agent for patients with SVT.

KEY POINTS

- Broad complex tachycardia is a potentially life-threatening medical emergency.
- Broad complex tachycardia implies either ventricular tachycardia or supraventricular tachycardia with BBB.
- VT is more serious than SVT, mainly because of the risk of degeneration into pulseless VT or ventricular fibrillation.
- Distinction of VT from SVT with BBB requires careful consideration of the history, examination findings and ECG appearances.
- Where doubt remains, administration of adenosine may be helpful as a diagnostic agent: reversion to sinus rhythm or slowing of the rate implies a supraventricular source.
- When the patient is acutely unwell or deteriorating, DC cardioversion is the most rapid and effective means of re-establishing sinus rhythm.
- Underlying causes such as myocardial ischaemia, myocardial infarction and electrolyte imbalances should be sought and corrected where possible.
- Referral to the cardiology team for consideration of coronary angiography and insertion of an ICD is recommended for all patients when VT is the likely diagnosis.

Further reading

ACC/AHA/ESC. Guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death (2006). <http://www.escardio.org/guidelines-surveys/esc-guidelines/Pages/ventricular-arrhythmias-and-prevention-sudden-cardiac-death.aspx>

O'Neill MD, Davies DW. Cardiac arrhythmias part II: Broad complex tachycardia. *Acute Med* 2005; 4(1): 3–9

Resuscitation Council UK. Resuscitation guidelines (2005). <http://www.resus.org.uk/pages/guide.htm>