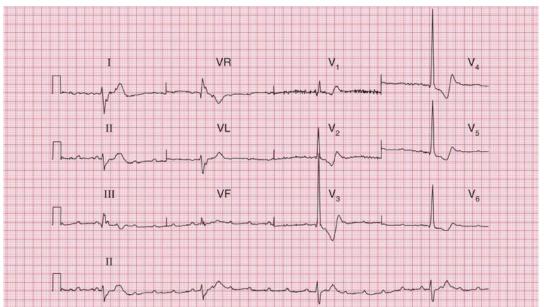
ECG CASES

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ECG 3 An 80-year-old woman, who had previously had a few attacks of dizziness, fell and broke her hip. She was found to have a slow pulse, and this is her ECG. The surgeons want to operate as soon as possible, but the anaesthetist is unhappy. What does the ECG show and what should be done?

Answer 3

- P wave rate 130 bpm
- Complete heart block

- Ventricular (QRS complex) rate 23 bpm
- The ventricular 'escape' rhythm has wide QRS complexes and abnormal T waves. No further interpretation of the ECG is possible.

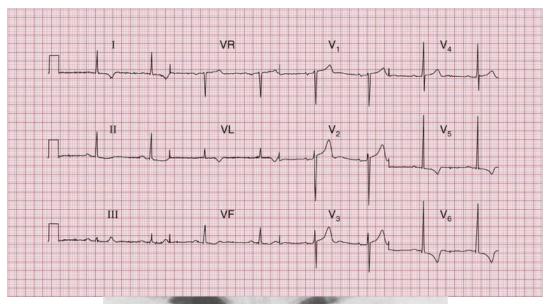
In complete heart block there is no relationship between the P waves (here with a rate of 120 bpm) and the QRS complexes.

What to do

In the absence of a history suggesting a myocardial infarction, this woman almost certainly has chronic heart block: the fall may or may not have been due to a Stokes–Adams attack. She needs a permanent pacemaker, ideally immediately. If permanent pacing is not possible immediately, a temporary pacemaker will be needed preoperatively.

Summary

Complete (third degree) heart block.







ECG 8 A 70-year-old retired orthopaedic surgeon telephones to say that he always gets dizzy playing golf. You find that he has a systolic heart murmur. His ECG and chest X-ray are shown. What is the diagnosis and what do you do next?

Answer 8

The ECG shows:

- Sinus rhythm, rate 48 bpm
- Normal axis
- QRS complex duration normal, but the R wave height in lead V₅ is 30 mm, and the S wave depth in lead V₂ is 25 mm
- Inverted T waves in leads I, VL, V₅–V₆.

The chest X-ray shows an enlarged left ventricle with 'post-stenotic' dilatation of the ascending aorta (arrowed).

Clinical interpretation

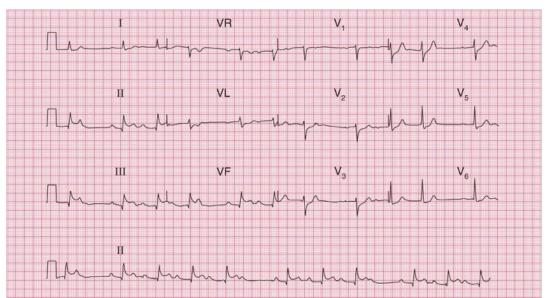
This is the classic ECG appearance of left ventricular hypertrophy.

What to do

The combination of dizziness on exercise, a systolic murmur and evidence of left ventricular hypertrophy suggests significant aortic stenosis. The next step is an echocardiogram: in this patient it showed a gradient across the aortic valve of 140 mmHg, indicating severe stenosis. He needed an urgent aortic valve replacement.

Summary

Left ventricular hypertrophy.



ECG 9 A 70-year-old man is admitted to hospital following the onset of severe central chest pain. This is his ECG. What does it show and what treatment is needed?

- Sinus rhythm, rate of sinus beats 75 bpm
- Second degree (Wenckebach) heart block (most obvious in the rhythm strip, recorded from lead II)
- Ventricular rate 70 bpm

- Normal axis
- Small Q waves in leads II, III, VF
- Raised ST segments in leads II, III, VF
- Depressed ST segments in leads V₅–V₆.

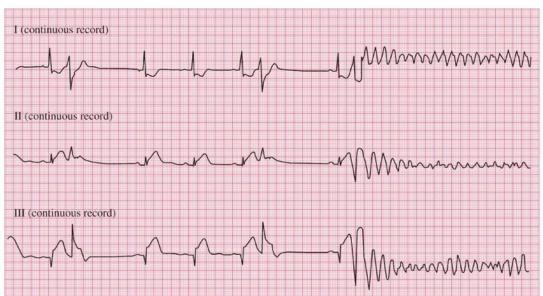
This patient has second degree block of the Wenckebach type (progressive lengthening of the PR interval followed by a nonconducted P wave, and then a return to a short PR interval and repeat of the sequence). There is also clear evidence of a recent acute inferior ST segment elevation myocardial infarction (STEMI).

What to do

The patient should be treated in the usual way for his acute myocardial infarction, with pain relief, dual antiplatelet therapy (with aspirin and a P2Y12 inhibitor) and immediate primary percutaneous coronary intervention (PCI). Wenckebach second degree block is usually benign when it occurs with an inferior infarction, and although he must obviously be monitored until sinus rhythm with normal conduction returns, temporary pacing is not necessary.

Summary

Second degree (Wenckebach) atrioventricular block with acute inferior STEMI.



ECG 10 A 50-year-old man, who had come to the Accident and Emergency (A&E) department with chest pain, collapsed while his ECG was being recorded. What happened and what would you do?

- Sinus rhythm initially 55 bpm, with ventricular extrasystoles
- The third extrasystole occurs on the peak of the T wave of the preceding sinus beat

- After three or four beats of ventricular tachycardia, ventricular fibrillation develops
- In the sinus beats there is a Q wave in lead III; and there are raised ST segments in leads II and III, and ST segment depression and T wave inversion in lead I

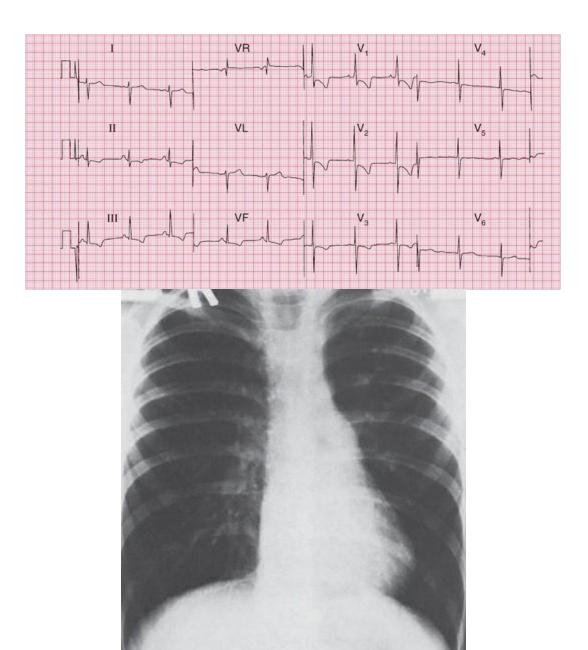
Although only leads I, II and III are available, it looks as if the chest pain was due to an inferior myocardial infarction. This was probably the cause of the ventricular extrasystoles, and an 'R on T' extrasystole caused ventricular tachycardia, which rapidly decayed into ventricular fibrillation. It might be argued that in lead III, and perhaps also in lead I, 'torsade de pointes' ventricular tachycardia is present, but this is not apparent in lead II.

What to do

Immediate defibrillation, but if no defibrillator is at hand then cardiopulmonary resuscitation should be performed according to advanced life support (ALS) guidelines. Following return of spontaneous circulation (ROSC) primary angioplasty is indicated.

Summary

Probable inferior myocardial infarction; R on T ventricular extrasystole, causing ventricular fibrillation.



ECG 13 A 40-year-old woman is referred to the outpatient

department because of increasing breathlessness. What do this ECG and chest X-ray show, what physical signs might you expect, and what might be the underlying problem? What might you do?

Answer 13

The ECG shows:

- Sinus rhythm, rate 65 bpm
- Peaked P waves, best seen in lead II
- Right axis deviation
- Dominant R waves in lead V₁
- Deep S waves in lead V₆
- Inverted T waves in leads II, III, VF, V₁–V₃.

The chest X-ray shows a slightly enlarged heart with a high cardiac apex and a prominent main pulmonary artery, suggesting right ventricular hypertrophy.

Clinical interpretation

This combination of right axis deviation, dominant R waves in lead V_1 and inverted T waves spreading from the right side of the heart is classic of severe right ventricular hypertrophy. Right ventricular hypertrophy can result from congenital heart disease, or from pulmonary hypertension, which may be idiopathic or secondary to mitral valve disease, lung disease or pulmonary embolism. The physical signs of right ventricular hypertrophy are a left parasternal heave and a displaced but diffuse apex beat. There may be a loud pulmonary second sound. The jugular venous pressure may be elevated, and a 'flicking A' wave in the jugular venous pulse is

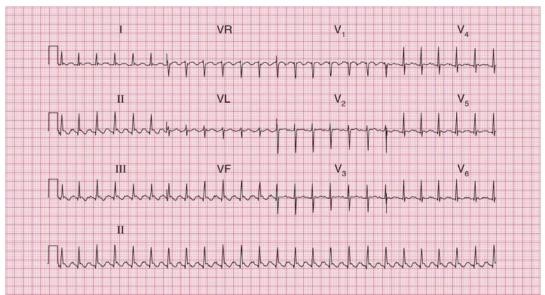
characteristic of pulmonary hypertension.

What to do

This woman requires urgent investigation to assess the underlying cause of her pulmonary hypertension. The first step will be to confirm the diagnosis by echocardiography. The two main causes of pulmonary hypertension of this degree in a 40-year-old woman are recurrent pulmonary emboli, and idiopathic (primary) pulmonary hypertension. Clinically, it is difficult to differentiate between the two, but a lung scan and computed tomography (CT) pulmonary angiography will help. In either case, anticoagulants are indicated. In fact, this patient had primary pulmonary hypertension, and treatment with high dose calcium channel blockers, prostanoids, endothelin receptor antagonists (bosentan) and phosphodiesterase inhibitors was tried, without success. Eventually she needed heart and lung transplantation.

Summary

Severe right ventricular hypertrophy.



ECG 15 This ECG was recorded from a 40-year-old man who was admitted to hospital as an emergency, with the sudden onset of the symptoms and signs of severe left ventricular failure. What does it show and what would you do?

- Atrial flutter with 2 : 1 block (best seen in leads II, III, VF)
- Normal axis
- Normal QRS complexes

• The T waves are difficult to identify because of the flutter waves.

Clinical interpretation

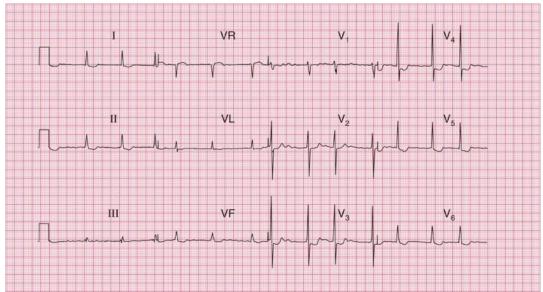
It is a bit unusual for the onset of atrial flutter in a young and otherwise fit male to cause severe left ventricular failure. Sometimes an asymptomatic tachyarrhythmia can lead over time to impaired left ventricular function (a tachycardiomyopathy). Alternatively there may be another cause of left ventricular impairment (such as a cardiomyopathy) which has only become manifest by the onset of arrhythmia. There is nothing on the ECG to suggest a cause for the arrhythmia.

What to do

Initial treatment depends on the severity of symptoms. Milder cases may be managed initially with rate control and anticoagulation. However, when an arrhythmia causes severe heart failure immediate treatment may be necessary. In a severely compromised patient treatment under sedation with direct current (DC) cardioversion Ideally this should be considered. should be guided bv transoesophageal ECHO unless the timing of onset of the arrhythmia is clear from the history and is <48 hours. This is to exclude left atrial appendage thrombus (which, if present, can be dislodged during cardioversion leading to stroke). In the long term, this patient will require further investigations and ablation therapy may be considered to prevent further episodes of atrial flutter.

Summary

Atrial flutter with 2:1 block.



ECG 26 An 80-year-old woman, who has apparently been treated for heart failure for years, complains of nausea and vomiting. No previous records are available. Does her ECG help her management?

- Atrial fibrillation, ventricular rate 80 bpm
- Normal axis
- Normal QRS complexes

- Downward-sloping ST segment depression, especially in leads V₄–V₆
- T waves probably upright
- Prominent U waves in leads V₂–V₃.

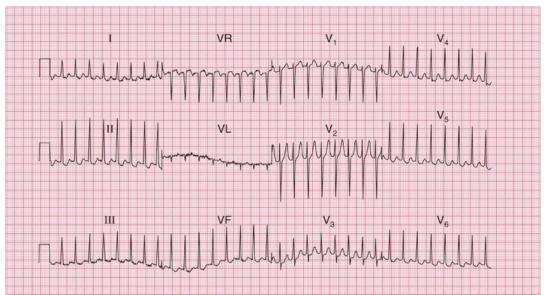
The ECG shows atrial fibrillation with a controlled ventricular rate. There is nothing on the ECG to suggest a cause for the arrhythmia or the patient's heart failure. The 'reversed tick' ST segment depression suggests that she is being treated with digoxin. The ECG does not suggest digoxin toxicity, but nevertheless this is a potential cause of her nausea. The U waves may be normal, but raise the possibility of hypokalaemia.

What to do

Digoxin is not generally used as first-line rate controlling treatment but it may be that alternatives have already been tried unsuccessfully. Measures of her plasma potassium and digoxin levels should certainly be checked and her treatment adjusted accordingly.

Summary

Atrial fibrillation and digoxin effect.



ECG 28 A 45-year-old woman had complained of occasional attacks of palpitations for 20 years, and eventually this ECG was recorded during an attack. What are the palpitations due to, and what would you do?

- Narrow complex tachycardia at 188 bpm
- No P waves visible
- Normal axis

- QRS complexes normal
- Some ST segment depression.

This ECG shows supraventricular tachycardia. This rhythm is usually due to a re-entry pathway within, or near to, the atrioventricular node, so the rhythm is properly called AV nodal re-entry tachycardia (AVNRT). Alternatively an AVRT where an aberrant pathway is not evident on the resting ECG may present similarly. The ST segment depression could indicate ischaemia, but the ST segments are not horizontally depressed, nor is the depression greater than 2 mm, so it is probably of no significance.

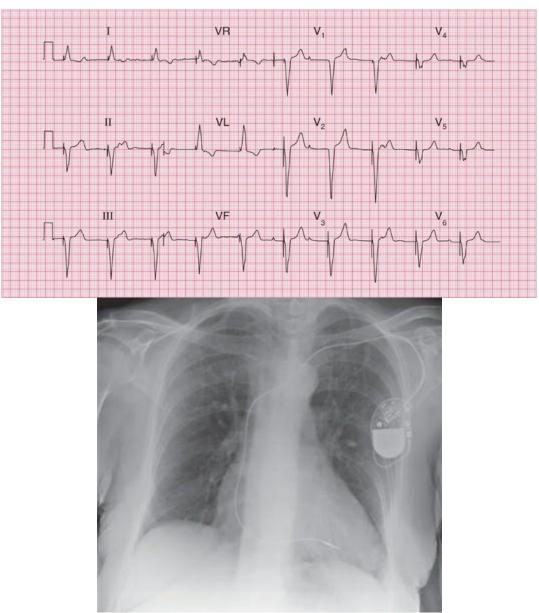
What to do

Valsalva's manoeuvre or carotid sinus massage may be tried and will sometimes terminate the attack. If this fails, it will almost certainly respond to intravenous adenosine. Once sinus rhythm has been restored, prophylactic medication may not be needed if attacks are infrequent. Patients with recurrent problematic episodes despite medical therapy may require specialist electrophysiological assessment to consider ablation therapy.

Summary

AV nodal re-entry (junctional) tachycardia (AVNRT).

9



ECG 30 The senior house officer in the A&E department is puzzled by this ECG, which was recorded from an 80-year-old

admitted unconscious with a stroke. What has the house officer missed? Perhaps he did not make a proper examination and did not look at the chest X-ray?

Answer 30

The ECG shows:

- Regular rhythm at 60 bpm
- Occasional P waves, not related to QRS complexes (e.g. in lead I)
- Left axis deviation
- QRS complexes preceded by a sharp 'spike'
- Broad QRS complexes (160 ms)
- Deep S wave in lead V₆
- Inverted T waves in leads I, VL.

The chest X-ray shows a permanent pacemaker, with a single lead in the right ventricle.

Clinical interpretation

The sharp spikes preceding each QRS complex are due to the pacemaker. The P waves that can occasionally be seen indicate that the underlying rhythm is complete heart block – presumably the reason why the pacemaker was inserted.

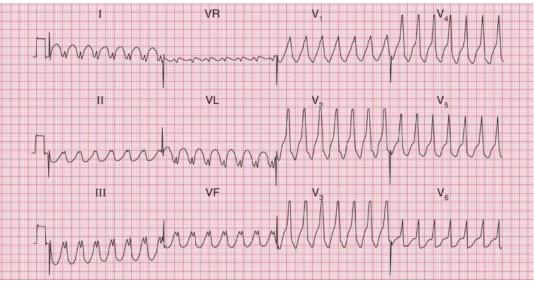
What to do

The house officer has missed the pacemaker, which is usually buried below the left clavicle. There is no particular reason why the pacemaker should be related to the stroke, except that patients with vascular disease in one territory usually have it in others – this man probably has both coronary and cerebrovascular disease.

Summary

Permanent pacemaker and underlying complete block.

10



ECG 31 A 60-year-old man complained of severe central chest pain, and a few minutes later became extremely breathless and collapsed. He was brought to the A&E department, where his heart rate was found to be 165bpm, his blood pressure was unrecordable and he had signs of left ventricular failure. This is his ECG. What has happened and what would you do?

Answer 31

- Broad complex tachycardia at 165 bpm
- No P waves visible

- QRS complex duration about 200 ms
- Concordance of QRS complexes (i.e. all point upwards) in the chest leads

A broad complex tachycardia can be ventricular in origin, or can be due to a supraventricular tachycardia with aberrant conduction (i.e. bundle branch block). Here the very broad complexes and the QRS complex concordance suggest a ventricular tachycardia. In a patient with a myocardial infarction it is always safe to assume that such a rhythm is ventricular. From the story, one would guess that this patient had a myocardial infarction and then developed ventricular tachycardia, but it is possible that the chest pain was due to the arrhythmia.

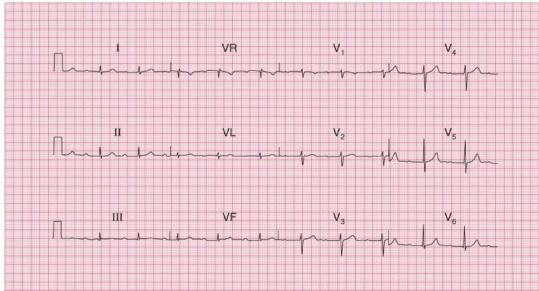
What to do

This patient has haemodynamic compromise – low blood pressure and heart failure – and needs immediate cardioversion. An anaesthetic will be required. Following restoration of a less malignant heart rhythm, he is likely to require emergency coronary revascularization with primary angioplasty.

Summary

Ventricular tachycardia.

11



ECG 33 This ECG was recorded from an asymptomatic 45year-old man at a 'health screening' examination. Is it normal, and what advice would you give him?

Answer 33

The ECG shows:

- Sinus rhythm, rate 64 bpm
- Prolonged PR interval (360 ms)
- Normal QRS complexes, ST segments and T waves.

Clinical interpretation

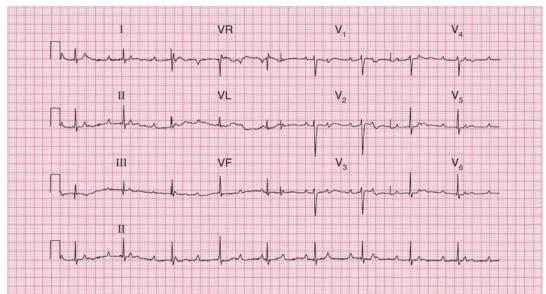
This ECG shows first degree atrioventricular block but is otherwise entirely normal.

What to do

Although the upper limit of the PR interval is usually taken to be 220 ms, longer durations (technically first degree block) are frequently seen in healthy people. Provided you can be sure that this patient has no symptoms, and provided the physical examination is normal, no further action is required. Some individuals in occupations that require a totally normal ECG may have to have an ambulatory ECG recording to demonstrate that there are no episodes of higher-degree block.

Summary

First degree atrioventricular block.



ECG 34 This ECG was recorded from a 70-year-old woman who had complained of attacks of dizziness for about a year. What is the problem, what might be its cause, and how should this woman be treated?

- Sinus rhythm with complete (third degree) block, rate 55 bpm
- Normal axis
- Normal QRS complexes and T waves.

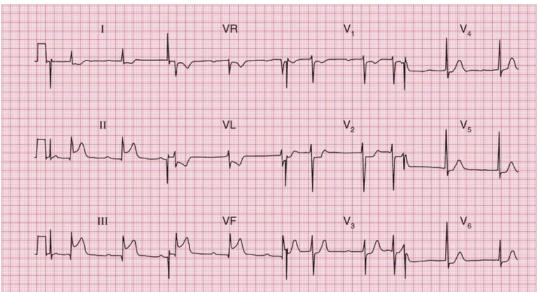
This ECG shows complete heart block with a relatively low ventricular rate. The attacks of dizziness may be due to further slowing of the heart rate. Although at times there appears to be second degree (2 : 1) block, the lead II rhythm strip shows that what could be the PR interval is continually changing, and that there is actually no relationship between the P waves and QRS complexes. The QRS complex is narrow, and so must originate in the His bundle.

What to do

The patient needs a permanent pacemaker. There are many causes of heart block, including ischaemia; association with aortic valve calcification; Lyme disease (*Borrelia burgdorferi*); His bundle interruption (due to surgery, trauma, parasites, tumours, abscesses, granulomata); and drugs (digoxin, beta-blockers, calcium-channel blockers). However, most cases of heart block are due to His bundle fibrosis, for which hypertension is a risk factor. An echocardiogram should be arranged to exclude structural heart disease.

Summary

Complete (third degree) block.



ECG 37 This ECG was recorded from a 55-year-old man who was admitted to hospital as an emergency with severe central chest pain that had been present for about an hour. He was pale, cold and clammy; his blood pressure was 100/80, but there were no signs of heart failure. What does this ECG show? Does anything about it surprise you?

- Sinus rhythm, rate 50 bpm
- First degree block (PR interval 350 ms)

- Normal axis
- Small Q waves in leads II, III, VF
- Raised ST segments in leads II, III, VF
- Depressed ST segments and inverted T waves in leads I, VL
- Slight ST segment depression in the chest leads.

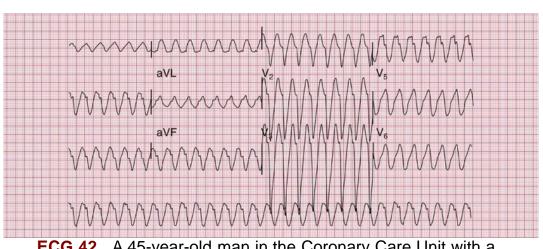
Acute inferior ST segment elevation myocardial infarction (STEMI) with anterolateral ischaemia, and first degree block. Patients who are in pain with an acute myocardial infarction usually have a sinus tachycardia, but here vagal overactivity is causing a bradycardia.

What to do

This patient should be treated as a STEMI emergency. Analgesia should be administered as required along with dual antiplatelet therapy (aspirin and P2Y12 inhibitor) in preparation for primary percutaneous coronary intervention (PCI). The first degree block does not require specific treatment nor does the relative bradycardia unless this deteriorates further.

Summary

Acute inferior STEMI with first degree block.



ECG 42 A 45-year-old man in the Coronary Care Unit with a suspected myocardial infarction suddenly becomes breathless and hypotensive, and this is his ECG. What does it show and what would you do?

This ECG shows:

- A broad complex tachycardia
- Heart rate 180 bpm
- No P waves visible
- QRS duration about 200 ms
- It is difficult to be sure whether the complexes are

pointing upwards or downwards, but the axis appears to be to the left.

Clinical interpretation

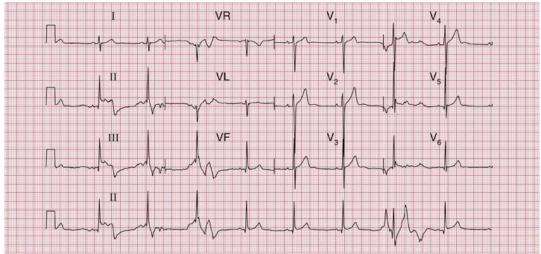
A broad complex tachycardia can in theory be due either to a supraventricular tachycardia with bundle branch block, or to ventricular tachycardia. The very broad complexes with left axis deviation suggest that this is ventricular tachycardia. More important, however, is the clinical context: in a patient with a suspected myocardial infarction with a first attack of tachycardia, a broad complex rhythm is almost certainly ventricular tachycardia.

What to do

Since the patient is breathless with a low blood pressure, immediate cardioversion under sedation of anaesthesia is indicated. Once a safe rhythm has been restored, attention should be turned to management of his myocardial infarction, including emergency coronary angiography and percutaneous intervention.

Summary

Ventricular tachycardia.



ECG 47 This was a routine ECG recorded pre-operatively in a 60-year-old woman due for a cholecystectomy. Can the operation go ahead?

This ECG shows:

- Three complexes in the middle of the record which are clearly sinus rhythm
- Rate 57 bpm
- Remaining complexes are a bizarre and variable shape and the T waves cannot be identified, but these complexes are at the same rate as those in

sinus rhythm.

Clinical interpretation

At first sight the abnormal complexes might be ventricular extrasystoles, but this is very unlikely because they are such an abnormal shape and have no obvious T waves. More importantly, they occur at the time that would be expected if the rhythm was sinus rhythm throughout. These complexes must be artefacts, due to poor electrode contact with the skin.

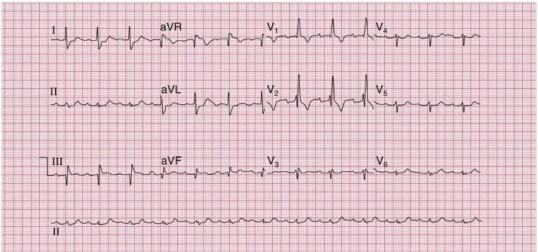
What to do

Although all the beats that clearly show sinus rhythm are normal, the ECG is incomplete and must be repeated.

Summary

Artefacts due to poor electrode contact.

16



ECG 50 This ECG was recorded from a 60-year-old man who had had an episode of chest pain a year previously, and now complained of breathlessness and occasional dizziness.

Answer 50

This ECG shows:

- Sinus rhythm, rate 80 bpm
- First degree block PR interval 250 ms
- Right bundle branch block (RBBB)
- Q waves in leads III and VF.

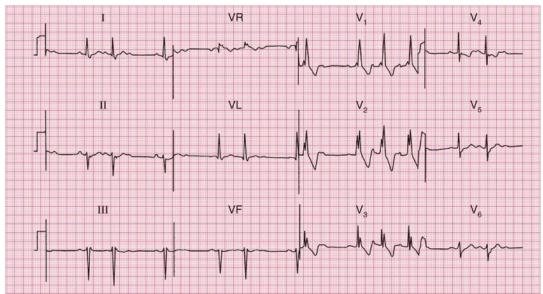
The Q waves in the inferior leads suggest an old myocardial infarction, which would fit with his history of chest pain. Without a previous ECG it is difficult to know whether the RBBB is new or old, but the combination of RBBB and first degree block raises the possibility that his dizziness is due to intermittent complete block.

What to do

He needs investigations to assess the extent of LV dysfunction, evaluate his coronary artery disease and to exclude arrhythmia as a cause for his dizziness. A cardiac stress MRI will determine LV function, the extent of infarction versus viability and the ischaemic burden. Ambulatory ECG recording will be required to investigate the dizziness, in the first instance with a 24 hour tape recording.

Summary

First degree block and RBBB.



ECG 51 A 70-year-old woman who complained of 'dizzy turns' was found to have an irregular pulse, and this ECG was recorded. There are three abnormalities. What advice would you give her?

- Sinus rhythm; sinus rate 100 bpm
- Normal and constant PR intervals in the conducted beats
- Occasional nonconducted P waves (best seen in lead

I)

- Left axis deviation
- Right bundle branch block (RBBB).

Clinical interpretation

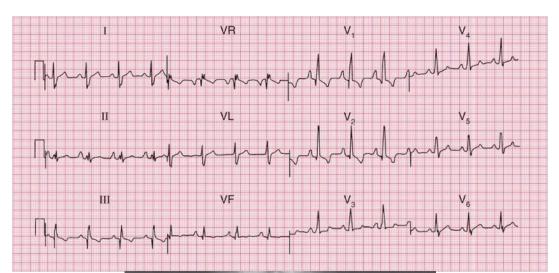
This ECG shows second degree block (Mobitz type 2) and bifascicular block – left axis deviation (left anterior hemiblock) and RBBB. This combination of conduction abnormalities indicates disease throughout the conduction system, and is sometimes called 'trifascicular' block.

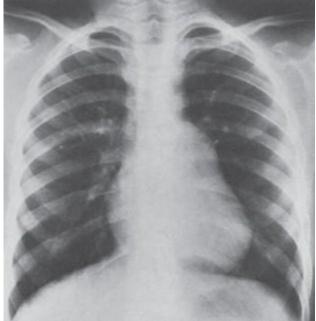
What to do

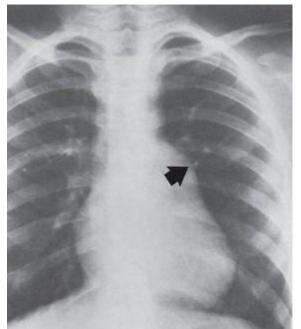
The 'dizzy turns' may represent intermittent complete block. Permanent pacing is essential.

Summary

Second degree block (Mobitz type 2) and bifascicular block.







ECG 54 This ECG and chest X-ray were recorded from a 17-year-old girl who was breathless, had marked ankle swelling with signs of right heart failure, and who had been known to have a heart murmur since birth. She was acyanotic. What ECG abnormalities can you identify, and can you suggest a diagnosis?

The ECG shows:

- Sinus rhythm, rate 81 bpm
- Markedly peaked P waves (best seen in leads II, V₁)
- Normal axis
- Dominant R wave in lead V₁.

The chest X-ray shows a high and prominent cardiac apex, consistent with right ventricular hypertrophy, and a prominent pulmonary artery (arrowed) which is due to post-stenotic dilatation as a result of pulmonary stenosis.

Clinical interpretation

The ECG shows right atrial and right ventricular hypertrophy.

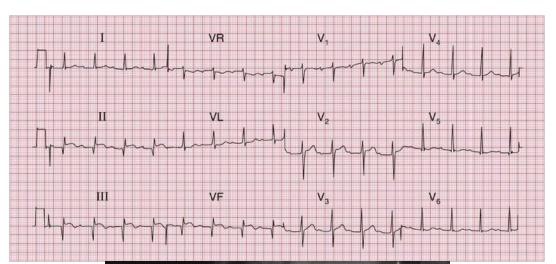
What to do

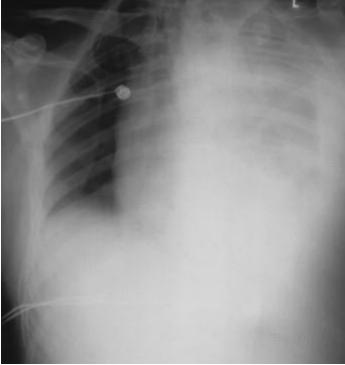
Right atrial hypertrophy is seen with pulmonary hypertension of any cause, tricuspid stenosis and Ebstein's anomaly. Right ventricular hypertrophy is seen with pulmonary stenosis and pulmonary hypertension. These conditions can all be diagnosed by echocardiography. This patient had pulmonary stenosis.

Summary

Right atrial and right ventricular hypertrophy.

19





ECG 58 This ECG and chest X-ray were recorded in the

A&E department from a 50-year-old man with severe central chest pain that radiated into his back. The pain had been present for 6h. What do the ECG and X-ray show, and what would you do?

Answer 58

The ECG shows:

- Sinus rhythm, rate 88 bpm
- PR interval 320 ms first degree block
- Q waves in leads II, III, VF
- Raised ST segments in leads II, III, VF
- Inverted T waves in leads III, VF.

The chest X-ray shows opacification of the left side of the chest, with probable shift of the mediastinum to the right.

Clinical interpretation

This ECG shows an acute inferior myocardial infarction, which often causes first degree block. The Q waves and raised ST segments are consistent with the story of 6 h of chest pain, and the first degree block is not important.

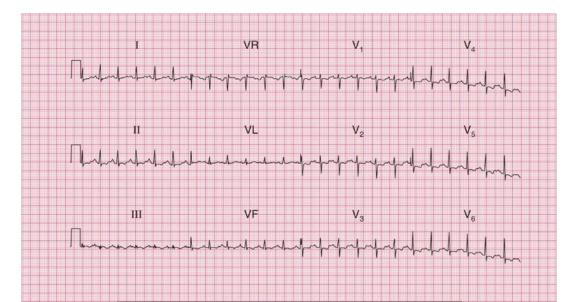
What to do

Chest pain radiating through to the back has to raise the possibility of aortic dissection, which can occlude the opening of the coronary arteries and so cause a myocardial infarction. However, it should be noted that this is relatively rare compared with back pain associated with myocardial infarction, which is common. In this case, the chest Xray suggests that blood has leaked into the left pleural cavity from a dissection of the aorta. The patient needs immediate investigation by computed tomography (CT) or magnetic resonance (MR) scanning and consideration of surgical repair of the dissection. Control of pain and avoidance of hypertension in the interim are important.

Summary

Acute inferior myocardial infarction with first degree block, due to dissection of the aorta.

ECG20





ECG 62 This ECG and pulmonary angiogram are from a 39-year-old woman who complained of a recent sudden onset of

breathlessness. She had no previous history of breathlessness, and no chest pain. Examination revealed nothing, other than a rapid heart rate. A pulmonary angiogram was carried out as part of a series of investigations immediately after admission. What is the diagnosis?

Answer 62

The ECG shows:

- Sinus rhythm, rate 140 bpm
- Normal conduction
- Normal axis
- Normal QRS complexes
- Slightly depressed ST segments in leads V₁–V₄
- Biphasic or inverted T waves in the inferior leads and all the chest leads.

Clinical interpretation

The ECG shows a marked sinus tachycardia, with no change in the cardiac axis and normal QRS complexes. The widespread ST segment/T wave changes are clearly very abnormal, but are not specific for any particular disease. However, the fact that leads V_1-V_3 are affected suggests a right ventricular problem.

The pulmonary angiogram shows a large central pulmonary embolus and occlusion of the arteries to the right lower lung.

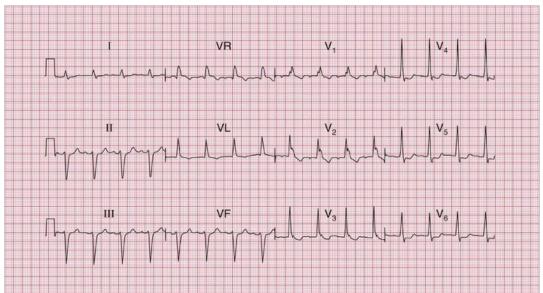
What to do

This is a case where the ECG must be considered in the light of the patient's history and physical signs (if any). Clearly something has

happened: the sudden onset of breathlessness without pain suggests a central pulmonary embolus – with pulmonary emboli that do not reach the pleural surface of the lung there may be little pain. In this patient, an echocardiogram and then a pulmonary angiogram demonstrated a large pulmonary embolus. Remember that sudden breathlessness with clear lung fields on a routine chest X-ray is always assumed to be due to a pulmonary embolus until proved otherwise. Anticoagulation is essential; thrombolysis should be considered.

Summary

Sinus tachycardia with widespread ST segment/T wave changes, suggesting pulmonary embolism.



ECG 63 A 70-year-old man is sent to the clinic because of rather vague attacks of dizziness, which occur approximately once per week. Otherwise he is well, and there are no abnormalities on examination. Does this ECG help with his management?

- Sinus rhythm, rate 94 bpm
- PR interval at the upper limit of normal (200 ms)
- Left axis deviation

- QRS complex duration prolonged (160 ms)
- RSR¹ pattern in leads V₁–V₂; wide S wave in lead V₆
- Inverted T waves in leads VL, V₁–V₄.

Clinical interpretation

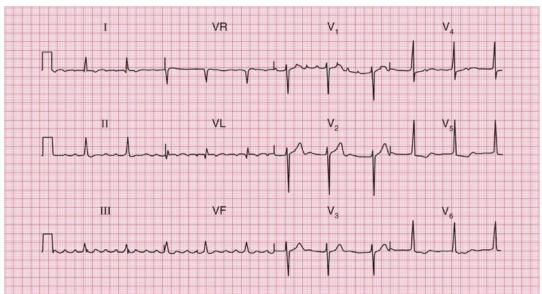
The left axis deviation is characteristic of left anterior hemiblock. There is also right bundle branch block (RBBB), so two of the main conducting pathways are blocked, resulting in 'bifascicular block'. The fact that the PR interval is at the upper limit of normal raises the possibility of delayed conduction in the remaining pathway; if the PR interval were definitely prolonged, this would indicate 'trifascicular block'.

What to do

Bifascicular block is not an indication for pacing if the patient is asymptomatic. The problem here is to decide if the attacks of dizziness are due to intermittent higher degree AV block causing bradyarrhythmias. Ideally an ECG would be recorded during an attack. Since they occur only every week or so, a longer period of ambulatory ECG recording will be required to capture an episode. A loop recorder (whether external or implanted) might be the best approach.

Summary

Left anterior hemiblock and RBBB – bifascicular block.



ECG 65 A 60-year-old woman is seen in the outpatient department, complaining of breathlessness. There are no abnormal physical findings. What does this ECG show, what might be the underlying problem and how would you treat her?

- Atrial flutter, best seen in lead III
- 4:1 block
- Normal axis

- Normal QRS complexes
- Sloping ST segment depression, best seen in leads V₅–V₆.

Clinical interpretation

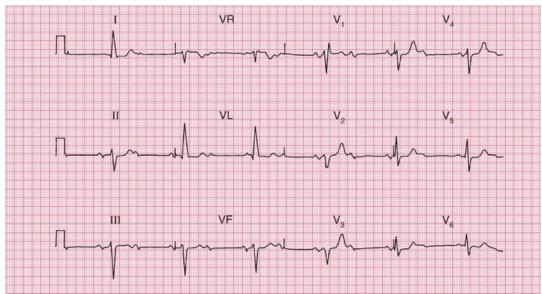
This shows atrial flutter with what appears to be a stable 4 : 1 block. The ST segment depression is non-specific or could relate to digoxin use.

What to do

The stable 4 : 1 block has caused a regular heartbeat, so the arrhythmia was not suspected at the time of the clinical examination. There is nothing in this ECG to indicate the underlying disease, which could be ischaemic or rheumatic heart disease, or a cardiomyopathy: echocardiography is needed. The downward-sloping ST segments suggest digoxin treatment. Digoxin is not generally the first-line medication for rate control but can still be useful where blood pressure limits use of other drugs (such as beta-blockers) or as a second-line agent where rate control is not achieved with a single drug. If managed without cardioversion, her CHA2DS2-VASc score (or similar assessment) should be used to assess the need for anticoagulation. However given her symptoms, cardioversion (following a period of anticoagulation) and even ultimately ablation therapy, should be considered.

Summary

Atrial flutter with 4:1 block.



ECG 66 An 80-year-old man is found at routine examination to have a slow heart rate and a harsh systolic murmur. This is his ECG. What does it show, and what is the likely diagnosis? Is treatment necessary?

- Sinus rhythm, P wave rate 75 bpm
- Second degree (2:1) block
- Left axis deviation

 QRS complex duration 140 ms, with an RSR¹ pattern in lead V₁, indicating right bundle branch block (RBBB).

Clinical interpretation

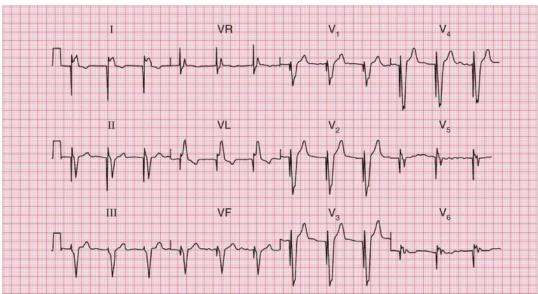
This is second degree block and not complete (third degree) block. Left axis deviation (left anterior hemiblock) and RBBB constitute bifascicular block, but the 2 : 1 block shows that there is also significant disease in either the His bundle or the remaining posterior fascicle.

What to do

This patient needs a pacemaker. The combination of a heart murmur and heart block suggests aortic stenosis which is associated with AV node disease. The severity of this can be assessed with echocardiography.

<u>Summary</u>

Second degree (2:1) block.



ECG 68 An elderly woman is admitted to hospital unconscious, evidently having had a stroke. No cardiac abnormalities are noted, but this is her ECG. What has been missed?

- No P waves; irregular baseline suggesting atrial fibrillation
- Regular QRS complexes; rate 73 bpm
- Left axis deviation

- Wide QRS complexes of an indeterminate pattern, with inverted T waves in some leads
- Each QRS complex is preceded by a deep and narrow spike.

Clinical interpretation

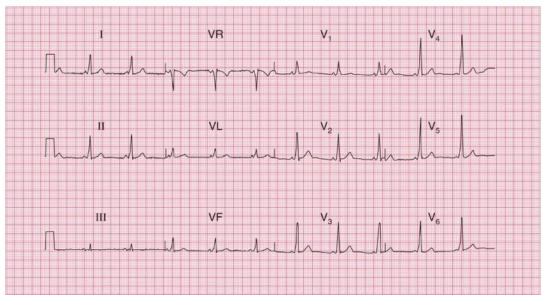
The narrow spike is due to a pacemaker, and someone has not noticed the permanent pacing battery, which is probably below the left clavicle. The pacing wire will be stimulating the right ventricle, giving rise to broad QRS complexes resembling a bundle branch block pattern. The underlying rhythm here is atrial fibrillation: the patient may have had atrial fibrillation with a slow ventricular response or complete AV block, or may have developed atrial fibrillation since pacing for another indication.

What to do

If this patient was not anticoagulated, the stroke may well have been due to an embolus arising in the left heart as a result of atrial fibrillation. If she was anticoagulated for her atrial fibrillation, an intracranial haemorrhage is possible. Either way she requires urgent Management brain imaging. for stroke may include acute thrombolysis or percutaneous intervention depending on local practice and in accordance with guidelines. Anticoagulation for her atrial fibrillation may be appropriate later depending on her response to initial treatment.

Summary

Ventricular-paced rhythm and atrial fibrillation.



ECG 69 A 30-year-old woman, who had a baby 3 months previously, complains of breathlessness, and this is her ECG. What is the problem?

- Sinus rhythm, rate 64 bpm
- Short PR interval at 100 ms
- Normal axis
- Normal QRS complex duration

- Slurred upstroke to QRS complexes (delta wave)
- Dominant R wave in lead V₁
- Normal ST segments and T waves.

Clinical interpretation

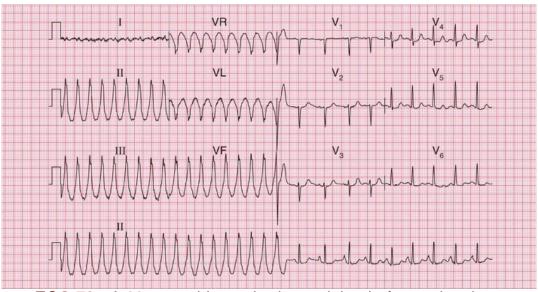
This ECG shows the Wolff–Parkinson–White (WPW) syndrome type A, which is characterized by a dominant R wave in lead V_1 . This is likely to be coincidental to her presentation.

What to do

The catch here is that the dominant R wave in lead V_1 may be mistakenly thought to be due to right ventricular hypertrophy. In a young woman who complains of breathlessness after a pregnancy, pulmonary embolism is obviously a possibility, and this might well cause ECG evidence of right ventricular hypertrophy – but in the presence of the WPW syndrome this would be very difficult to diagnose from the ECG. The only thing that might help the diagnosis in this scenario would be the appearance of right axis deviation, which is not usually present in WPW syndrome, but is not present here. The differential diagnosis remains broad and other causes of breathlessness, such as anaemia, should also be considered. Further investigation will be required both for her breathlessness and for her WPW which may require an electrophysiological assessment.

Summary

The WPW syndrome type A.



ECG 73 A 60-year-old man had complained of occasional episodes of palpitations for several years. Between attacks he was well, there were no physical abnormalities, and his ECG was normal. Eventually this ECG was recorded during one of his attacks. What is the arrhythmia and what would you do?

The lead II rhythm strip at the bottom of the record shows that the rhythm changes halfway through the recording, and this makes interpretation difficult. However, the ECG shows:

• Regular broad complex tachycardia, rate 160 bpm, followed by sinus rhythm, rate 120 bpm

- Normal axis during the tachycardia
- Broad QRS complexes, duration 160 ms
- QRS complexes normal during sinus rhythm
- During sinus rhythm there is ST segment depression in leads V₄–V₅.

Clinical interpretation

Without a full 12-lead record of the tachycardia it is difficult to be certain, but the complexes are very broad and have a totally different appearance from those in sinus rhythm, so this is almost certainly ventricular tachycardia. The ST segment depression in sinus rhythm is mild and not sufficient to make a confident diagnosis of ischaemia, but because the depression is horizontal, ischaemia seems likely.

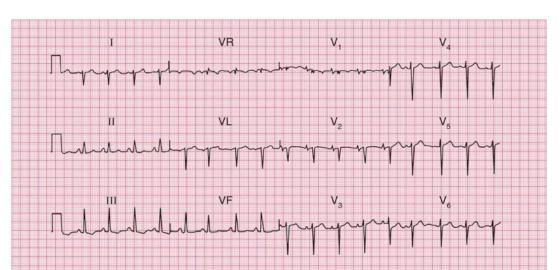
What to do

Patients who have only occasional episodes of an arrhythmia, and who are otherwise well, are always difficult to manage. This patient echocardiogram should have exclude certainly an to а cardiomyopathy, and a perfusion scan and possibly a coronary angiogram to investigate the possibility of ischaemia. The arrhythmia seems to be exercise-induced and ambulatory monitoring would be worthwhile to see how frequently it occurs, and a very cautious exercise test might be justified to see how easily it is provoked. A specialist electrophysiological assessment with a view to ablation therapy should be considered before initiation of antiarrhythmic therapy. If the episodes were causing syncope, an implanted defibrillator could be considered.

Summary

Paroxysmal ventricular tachycardia.

27





ECG 75 This ECG and chest X-ray were recorded from a

70-year-old man who complained of breathlessness. What abnormalities do they show and what is the most likely diagnosis? (X-ray reproduced with permission from Corne J & Pointon K (eds), *100 Chest X-Ray Problems*, Elsevier, 2007.)

Answer 75

The ECG shows:

- Sinus rhythm, rate 102 bpm
- Peaked P waves, best seen in leads V₁–V₂
- Right axis deviation (deep S waves in lead I)
- RSR¹ pattern with normal QRS complex duration in lead V₁ (partial right bundle branch block [RBBB])
- Deep S waves in lead V₆, with no left ventricular pattern.

The chest X-ray shows a long and thin mediastinum, with no increase in heart size but possible prominence of the pulmonary arteries. The lung fields appear essentially black, which is a feature of emphysema. This is the picture of chronic obstructive pulmonary disease (COPD).

Clinical interpretation

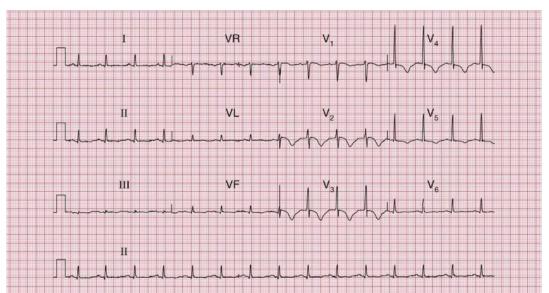
Peaked P waves suggest right atrial hypertrophy. The partial RBBB pattern is not significant. Right axis deviation may be seen in tall, thin people with normal hearts, but with the deep S waves in lead V_6 it suggests right ventricular hypertrophy. The lack of development of a left ventricular pattern in the V leads (i.e. deep S waves persisting into lead V_6) results from the right ventricle occupying most of the precordium. This is sometimes called 'clockwise rotation' (looking at the heart from below) and is characteristic of chronic lung disease.

What to do

Lung function tests may be more helpful than echocardiography but remember both COPD and heart disease are common and not infrequently occur in the same patient.

Summary

Right atrial hypertrophy and chronic obstructive pulmonary disease.



ECG 81 This ECG was recorded from a 15-year-old boy who collapsed while playing football. His brother had died suddenly. What does the ECG show and what clinical possibilities should be considered?

- Sinus rhythm, rate 91 bpm
- Normal PR interval
- Normal axis

- Normal QRS complexes
- Prolonged QT interval (QT=492 ms; QT_c=598 ms)
- Inverted T waves in leads V₂–V₅.

Clinical interpretation

This is clearly a very abnormal ECG, with a markedly prolonged QT interval and abnormal T waves.

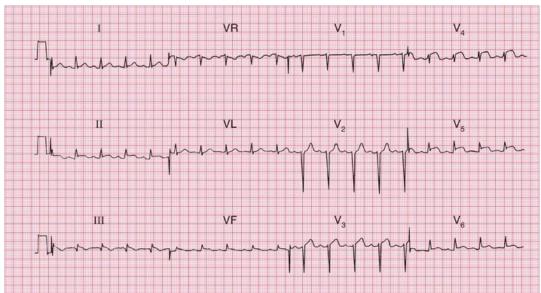
What to do

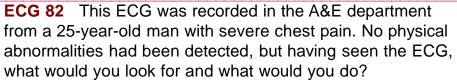
The family history suggests that this may well be an example of one of the congenital forms of prolonged QT interval. These are characterized by episodes of loss of consciousness in children, often at times of increased sympathetic nervous system activity, and betablockers are the immediate form of treatment. The insertion of a permanent defibrillator should be considered. Prolonged QT interval syndrome is also associated with antiarrhythmic drugs (including amiodarone and sotalol) and with other drugs such as the tricyclic antidepressants and erythromycin. Electrolyte abnormalities (low potassium, magnesium or calcium levels) also prolong the QT interval.

Summary

Marked prolongation of the QT interval – long QT syndrome.

See *ECG Made Practical*, 7th edition, Chapter 2





- Sinus rhythm, rate 105 bpm
- Normal axis
- Normal QRS complexes

• Raised ST segments in leads I–III, VF, V₄–V₆.

Clinical interpretation

The raised ST segments could indicate an acute infarction, but since the change is so widespread, is not in a particular coronary territory and shows no reciprocal ST-depression, pericarditis seems much more probable.

What to do

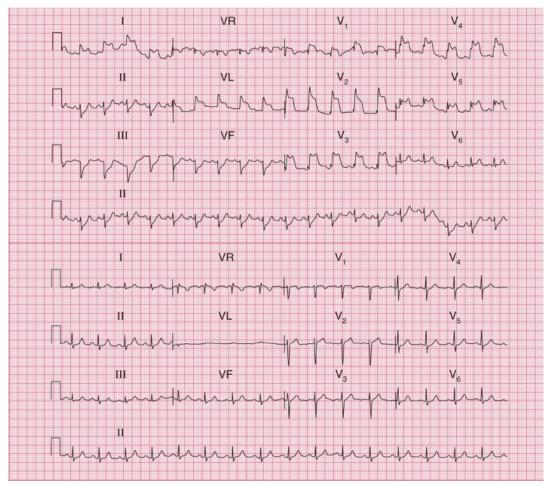
In a 25-year-old, pericarditis is a much more likely diagnosis than infarction. When lying the patient flat, a pericardial rub may become much easier to hear but is not always present. Echocardiography will confirm no myocardial regional wall motion abnormality and may show a pericardial effusion if one is present. Management is with nonsteroidal anti-inflammatory drugs and colchicine.

Summary

Widespread ST segment elevation, suggesting pericarditis.

See ECG Made Practical, 7th edition, Chapter 6

ECG 30



ECG 84 The upper ECG was recorded by paramedics from a 50-year-old woman who had had episodes of chest pain for several years, and who called an ambulance because of a severe attack. By the time she reached the A&E department, when the lower ECG was recorded, her pain had gone. What had happened?

The upper ECG shows:

- Sinus rhythm, average rate 111 bpm
- Left axis deviation
- QRS complexes probably normal, but partly obscured by the ST segments
- Raised ST segments in leads I, VL, V₁–V₅
- T waves probably normal. The lower ECG shows:
- Sinus rhythm, rate 97 bpm
- Normal axis
- Normal QRS complexes, ST segments and T waves.

Clinical interpretation

The first ECG seems to indicate an acute anterolateral myocardial infarction. An alternative explanation, given the widespread changes, would be pericarditis. The second ECG is normal. Because the ECG reverted to normal when the pain cleared, it seems likely that the changes in the initial ECG represent ischaemia. In this case coronary angiography showed no obstructive coronary disease. This was a case of Prinzmetal's variant angina.

What to do

Prinzmetal's variant angina was first described in 1959. It occurs at rest, and the characteristic raised ST segments seen in the ECG are not reproduced by exertion. It has been shown by angiography during pain to be due to spasm of one or more coronary arteries. Relatively

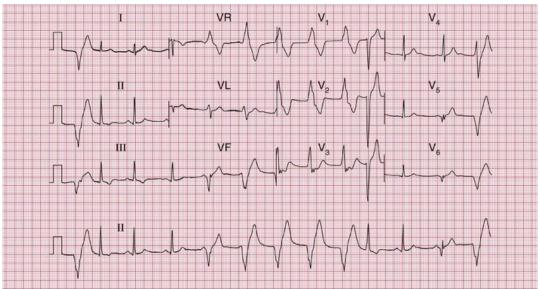
few patients with this type of angina have totally normal arteries, and spasm may occur at the site of atheromatous plaques. Vasodilator therapies may be helpful, but the condition can be difficult to treat.

Summary

Prinzmetal's variant angina.

See ECG Made Practical, 7th edition, Chapter 6

ECG 31



ECG 87 This ECG was recorded as part of the health screening of an asymptomatic 40-year-old man. How would you proceed?

Answer 87

The lead II rhythm strip at the bottom of the record shows that the rhythm changed during the recording, so it is necessary to try to identify the normal complexes (if any) in each lead. There are normal beats in the second and third complexes in leads I, II and III; in the first complex in leads VR, VL and VF; in the last complex in leads V_1 – V_3 ; and in the first complex in leads V_4 – V_6 . The ECG shows:

• Sinus rhythm at about 77 bpm, with ventricular

extrasystoles at the beginning and end of the record, and a six-beat run of a broad complex rhythm in the middle of the record

- The first complex of the run of broad complexes differs from the others, and is probably a fusion beat (a combination of a sinus beat and the ectopic rhythm)
- Normal axis when in sinus rhythm
- Normal QRS complexes in sinus rhythm
- Inverted T waves in lead III, but not VF.

Clinical interpretation

The run of broad complexes represents accelerated idioventricular rhythm. This is quite common following a myocardial infarction, but in a healthy subject it is probably of no significance. The T wave inversion in lead III is not important because the T wave is upright in lead VF.

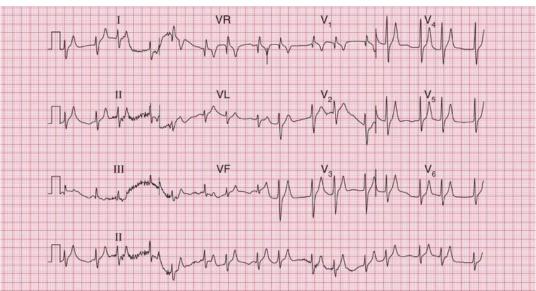
What to do

Ambulatory ECG monitoring over 24 hours and confirmation of a structurally normal heart by echocardiography or cardiac MRI will provide reassurance. If the individual has no symptoms and the physical examination is otherwise normal, no further action is needed. Accelerated idioventricular rhythm should not be treated.

Summary

Sinus rhythm and accelerated idioventricular rhythm.

ECG 32



ECG 88 This ECG was recorded from a 30-year-old woman admitted to hospital with diabetic ketoacidosis. Any comments?

Answer 88

This is not a technically good record, and exhibits considerable artefacts. However, the ECG shows:

- The rhythm is probably sinus, with coupled junctional extrasystoles
- P waves difficult to identify, but there are probably flattened P waves before the first of each pair of

QRS complexes, best seen in lead VR

- Probably normal PR interval
- Normal axis
- Narrow QRS complexes, so this is a supraventricular rhythm
- QRS complexes apparently in pairs, which are identical
- QRS complex duration at the upper limit of normal (120 ms)
- ST segment not easy to identify
- T waves sharply peaked in all leads.

Clinical interpretation

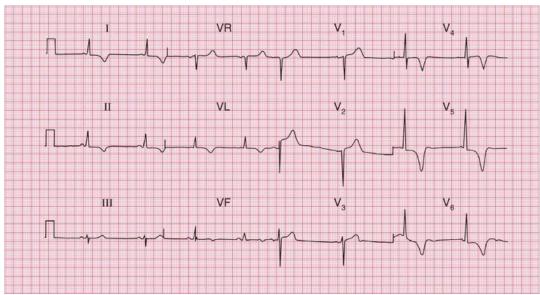
These changes are characteristic of hyperkalaemia, which of course is likely to be secondary to diabetic ketoacidosis.

What to do

This ECG should alert you to check the serum potassium level immediately: in this patient, it was found to be 7.1 mmol/l. This degree of hyperkalaemia with ECG changes requires emergency treatment with calcium gluconate or calcium carbonate as well as treatment of the underlying dehydration and hyperglycaemia.

Summary

Hyperkalaemia.



ECG 89 A 35-year-old white man is seen in the outpatient department complaining of chest pain on exertion, sometimes with exertion-induced dizziness, and this is his ECG. What is the likely diagnosis? What physical signs would you look for?

Answer 89

- Sinus rhythm
- Normal axis
- Normal QRS complexes

• Marked T wave inversion in leads I, II, VL, V₄–V₆.

Clinical interpretation

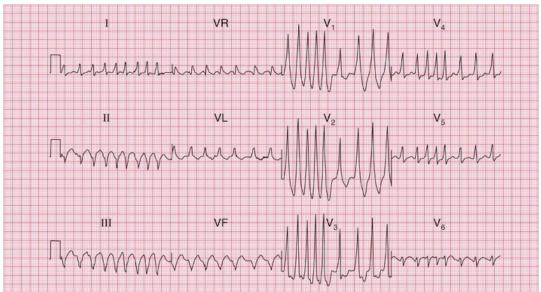
Anterolateral T wave inversion as gross as this may be due to a non-ST segment elevation myocardial infarction. However, on this trace the changes are due to left ventricular hypertrophy secondary to a hypertrophic cardiomyopathy. Myocardial infarction is uncommon in people of this age.

What to do

Physical signs of hypertrophic cardiomyopathy include a 'jerky pulse'; an aortic flow murmur which is characteristically louder after the pause that follows an extrasystole; and mitral regurgitation. Hypertrophic cardiomyopathy is best diagnosed by echocardiography or cardiac MRI, which will show asymmetric septal hypertrophy, systolic anterior movement of the mitral valve apparatus and sometimes associated left ventricular outflow tract obstruction and/or mitral regurgitation. This patient's echocardiogram showed all these features, confirming the diagnosis of hypertrophic cardiomyopathy.

Summary

Gross T wave inversion in the anterolateral leads, suggesting hypertrophic cardiomyopathy.



ECG 90 A 25-year-old woman, who had had episodes of what sound like a paroxysmal tachycardia for 10 years, produced this ECG when seen during an attack. What is the rhythm, and what is the underlying problem?

Answer 90

- Irregular tachycardia at about 200 bpm
- No consistent P waves visible
- Left axis deviation

- QRS complex duration varies between about 120 and 160 ms
- QRS complexes show a dominant R wave in lead V₁ and a prominent S wave in lead V₆
- After the longer pauses, the upstroke of the QRS complexes appears slurred.

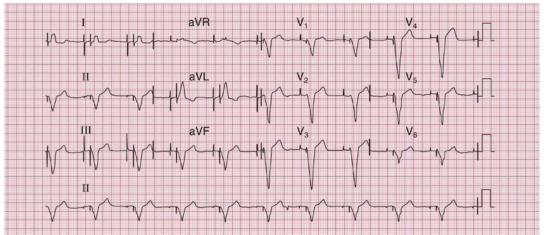
The marked irregularity of this rhythm must be explained by atrial fibrillation. The broad QRS complexes might be due to right bundle branch block, but the dominant R wave in lead V_1 , together with the slurred upstroke of the QRS complex in at least some leads, indicate the Wolff–Parkinson–White (WPW) syndrome type A.

What to do

A combination of the WPW syndrome and atrial fibrillation is very dangerous, because it can degenerate into ventricular fibrillation. The arrhythmia needs treating as an emergency with DC cardioversion under sedation of anaesthesia, whatever the clinical state of the patient. It is important not to use drugs that may block the atrioventricular node (such as adenosine, verapamil or beta-blockers) as these increase conduction through the accessory pathway and increase the risk of ventricular fibrillation. Following cardioversion, flecainide be can used antiarrhythmic as an pending an electrophysiological study to identify and ablate the accessory pathway.

Summary

Atrial fibrillation and the WPW syndrome type A.



ECG 91 An elderly man was admitted unconscious after a stroke, and this was his ECG. Does it help with diagnosis and management?

Answer 91

- A regular broad complex rhythm at 60 bpm
- There is a sharp "pacing" spike immediately before each QRS
- P waves are not easy to see except perhaps in V₂, but there are pacing spikes where P waves would be expected.

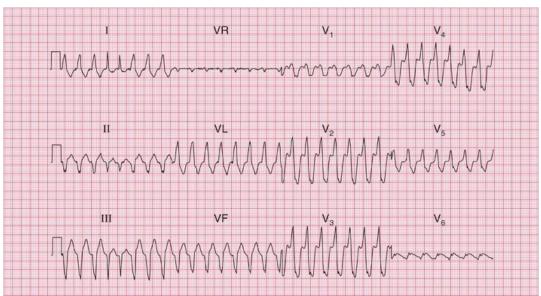
This ECG shows dual chamber (i.e., right atrial and right ventricular) pacing.

What to do

There is nothing on this ECG to suggest pacemaker malfunction and the underlying rhythm is probably sinus. The pacemaker is probably not related to the stroke.

Summary

Dual chamber pacemaker.



ECG 103 A 70-year-old woman, admitted to hospital because of increasing heart failure of uncertain cause, collapsed and was found to have a very rapid pulse and a low blood pressure. This is her ECG. She recovered spontaneously. What is this rhythm, and what would you do?

Answer 103

- Broad complex tachycardia at about 188 bpm
- No P waves visible
- Left axis deviation

- QRS complex duration about 140 ms
- Narrow fourth and fifth QRS complexes
- QRS complexes that are probably concordant (in the chest leads all point upwards) though it is difficult to be certain.

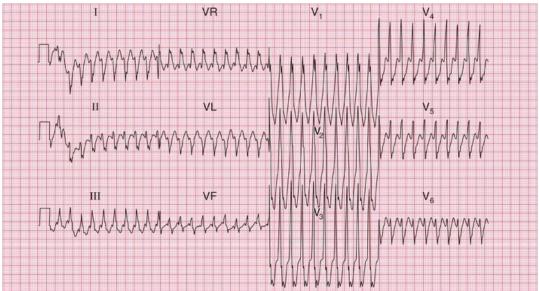
Broad complex tachycardias may be ventricular, supraventricular with bundle branch block, or due to the Wolff–Parkinson–White syndrome. We have no ECG from this patient recorded in sinus rhythm, which is always the most helpful thing in deciding between these possibilities. The complexes are not very wide, which would be consistent with a supraventricular origin with aberrant conduction, but the left axis deviation and (probable) concordance point to ventricular tachycardia. The key is the two narrow complexes near the beginning of the record: these are slightly early and are probably capture beats. They indicate that with an early supraventricular beat the conducting system can function normally; by implication, the broad complexes must be due to ventricular tachycardia.

What to do

In the first instance restoration of sinus rhythm by DC cardioversion under sedation or anaesthesia is indicated. An older patient with heart failure is more likely to have ischaemic disease than anything else, but all the possible causes of heart failure must be considered. The sudden onset of an arrhythmia could also be due to a myocardial infarction which should be evident on the post-cardioversion ECG. It is important to consider whether this rhythm change is related to treatment, in which case it could be due to an electrolyte imbalance or to the pro-arrhythmic effect of a drug the patient is taking.

Summary

Ventricular tachycardia.



ECG 107 A 30-year-old man, who had had brief episodes of palpitations for at least 10 years, was seen during an attack in the A&E department and this is his ECG. What is the rhythm, and what would you do immediately, and in the long term?

Answer 107

- Broad complex tachycardia at about 230–240 bpm
- No P waves visible
- Right axis deviation

- QRS complex duration of about 180 ms
- QRS complexes point upwards in lead V₁ and downwards in lead V₆ no concordance
- QRS complex configuration characteristic of right bundle branch block – but in lead V₁ the first R wave peak is higher than the second peak.

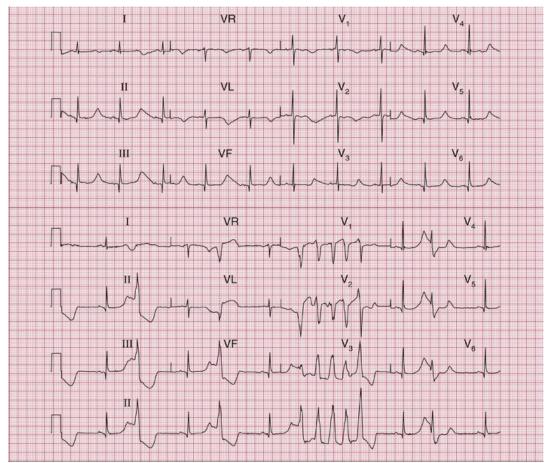
There are essentially three causes of a broad complex tachycardia: ventricular tachycardia, supraventricular tachycardia with bundle branch block and the Wolff–Parkinson–White (WPW) syndrome. The key to the diagnosis lies in the ECG when the heart is in sinus rhythm, but this is not always available. Patients with a broad complex tachycardia in the context of an acute myocardial infarction must be assumed to have a ventricular tachycardia, but that does not apply here. In this record, the QRS complexes are not very broad, the axis is to the right, and there is no concordance of the QRS complexes – all pointing to a supraventricular origin. In favour of a ventricular tachycardia is the fact that the height of the primary R wave in lead V_1 is greater than that of the secondary R wave. However, taking these features together with the clinical picture, the rhythm is probably supraventricular.

What to do

Carotid sinus pressure or adenosine is a reasonable first move. If there is severe haemodynamic compromise the patient may need urgent electrical cardioversion. In fact, in this case the arrhythmia terminated spontaneously, revealing a short PR interval and a QRS complex with a delta wave. So this patient had the WPW syndrome, and needed an electrophysiological study with a view to ablation of the accessory tract.

Summary

Broad complex tachycardia (eventually shown to be due to the WPW syndrome).



ECG 109 A confused 80-year-old woman was sent to hospital from a nursing home because of a collapse. No other history was available, except that she was said to be having treatment for her heart. There were no obvious physical signs. The upper ECG was recorded on admission, and the lower ECG was recorded shortly afterwards. What is going on?

Answer 109

The upper ECG shows:

- Sinus rhythm, with a rate of 60 bpm
- Narrow Q waves in leads II, III, VF, V₄–V₆
- Abnormally shaped T waves in most leads
- Prolonged QT interval (about 650 ms). The lower ECG shows:
- Sinus rhythm with multifocal ventricular extrasystoles
- A run of polymorphic (i.e. changing shape) ventricular tachycardia.

Clinical interpretation

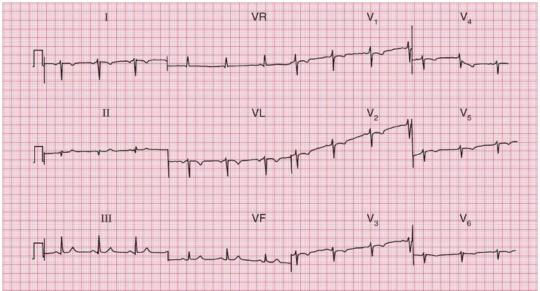
In the upper trace the inferolateral Q waves could represent an old infarction, but they are narrow and are probably septal in origin. The prolonged QT interval and abnormal T waves suggest either an electrolyte abnormality or that the patient is being treated with one of the many drugs that have these effects. A collapse in a patient with an ECG with a long QT interval suggests episodes of torsade de pointes ventricular tachycardia.

What to do

The electrolyte levels, including magnesium, must be checked, and in this case were found to be normal. It is essential immediately to establish what medication the patient is taking, and pending that information it would be sensible to leave her untreated and simply to monitor her for arrhythmias. It turned out that this woman was taking sotalol – a beta-blocker with class III antiarrhythmic activity which is known to cause QT interval prolongation. When this drug was stopped, her ECG returned to normal.

Summary

Drug-induced QT interval prolongation and polymorphic ventricular tachycardia.



ECG 114 This ECG was recorded from a healthy 25-yearold man during a routine medical examination. Any comments?

Answer 114

- A very odd appearance
- Sinus rhythm, rate 70 bpm
- Inverted P waves in lead I
- Right axis deviation

- Dominant R waves in lead VR
- No R wave development in the chest leads, with lead V₆ still showing a right ventricular pattern
- Normal-width QRS complexes.

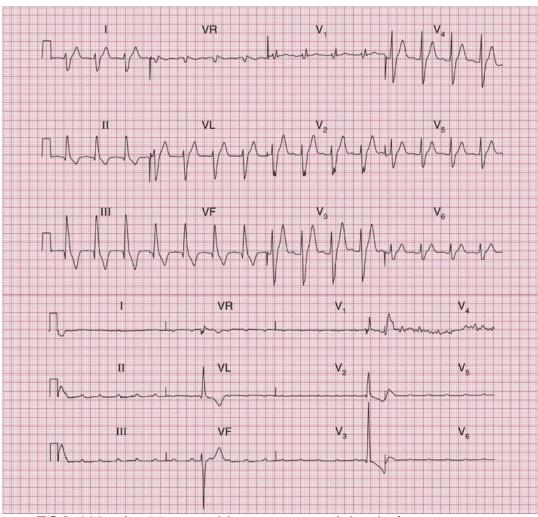
This is dextrocardia. A normal trace would be obtained with the limb leads reversed and the chest leads attached in the usual rib spaces but on the right side of the chest.

What to do

Ensure that the leads are properly attached – for example, inverted P waves in lead I will be seen if the right and left arm attachments are reversed. Of course, this would not affect the appearance of the ECG in the chest leads.

Summary

Dextrocardia.



ECG 118 An 80-year-old woman complained of breathlessness and frequent attacks of dizziness. This was her ECG when she attended the clinic. What does the ECG show, what might the dizziness be due to, and how would you manage her?

Answer 118

The ECG shows:

- Sinus rhythm, rate 90 bpm
- Right axis deviation
- Right bundle branch block (RBBB).

Clinical interpretation

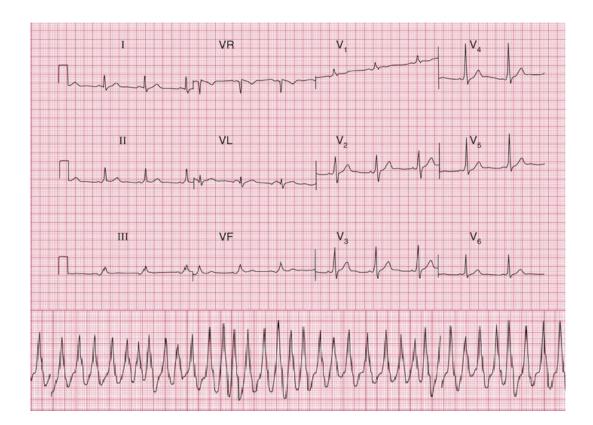
The right axis deviation suggests left posterior hemiblock, and, combined with RBBB, this suggests bifascicular block. The patient is therefore at risk of complete (third degree) block, which could cause a Stokes–Adams attack.

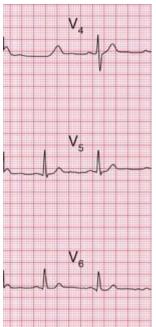
What to do

Ambulatory monitoring is required to confirm bradyarrhythmias. This woman was in fact admitted to hospital and monitored, and had a severe attack of dizziness and fainting. During this attack, another ECG was recorded (see below). This ECG shows complete heart block with a ventricular rate of about 15 bpm. The patient was immediately given a permanent pacemaker.

Summary

Left posterior hemiblock and RBBB – bifascicular block, followed by complete heart block.





ECG 123 A 30-year-old woman complains of episodes of palpitations associated with dizziness and breathlessness. These begin and stop suddenly. She has had them for many years, but they are becoming more frequent and more severe. The upper ECG was recorded at rest; the lower ECG is part of an ambulatory record, during which she had a typical attack. What do these ECGs show and what would you do?

Answer 123

The upper ECG shows:

- Sinus rhythm, rate 64 bpm
- Short PR interval, best seen in leads V₄–V₅
- Normal axis
- Dominant R waves in lead V₁
- Slurred upstroke (delta wave) in the QRS complexes. The lower ECG (rhythm strip) shows:

- A broad complex tachycardia
- Rate about 230 bpm
- The rhythm is irregular
- There is a slurred upstroke in some of the beats, suggesting pre-excitation.

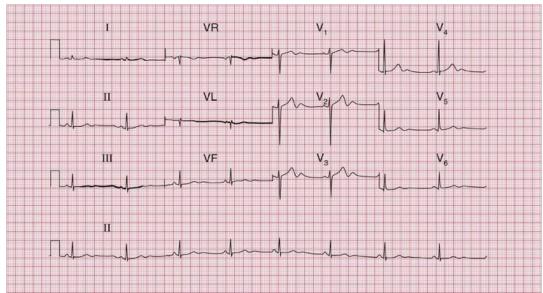
This is the Wolff–Parkinson–White (WPW) syndrome, involving a short PR interval and a widened QRS complex. This pattern, with a dominant R wave in lead V_1 and where there is a left-sided accessory pathway, is called 'type A'. It can easily be mistaken for right ventricular hypertrophy. The patient's palpitations are due to atrial fibrillation; an irregular broad complex tachycardia is characteristic of atrial fibrillation in the WPW syndrome.

What to do

Atrial fibrillation in association with the WPW syndrome is extremely dangerous. The patient should be treated with Flecainide pending an urgent electrophysiological study with a view to ablation of the accessory pathway. An ECG was recorded after the ablation (see right – leads V_4 – V_6 shown): the PR interval is now normal and there is no widening of the QRS complex.

Summary

The WPW syndrome type A, with paroxysmal atrial fibrillation.



ECG 124 This ECG was recorded from a 30-year-old man at a medical examination required by the Civil Aviation Authority. Is it normal?

Answer 124

The ECG shows:

- Sinus rhythm, rate 52 bpm
- Prominent U waves, especially in leads V₂–V₄.

Clinical interpretation

U waves can indicate hypokalaemia, but when associated with normal

T waves (as here) they are a normal variant.

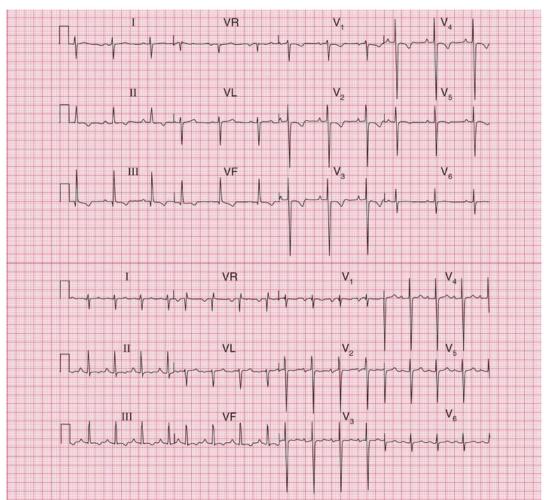
What to do

Provide reassurance – on the basis of his ECG at least, he is fit to fly.

Summary

Normal ECG, with prominent U waves.

43



ECG 126 A 70-year-old man gave a history of several years of chest pain on exertion. These are his ECGs at rest (upper trace) and on exercise (lower trace). What do they show?

Answer 126

The upper ECG shows:

- Sinus rhythm, rate 68 bpm
- Right axis deviation
- Small Q waves in leads III, VF
- Persistent S wave in leads V₅–V₆
- Inverted T waves in leads II, III, VF, V₁–V₅.

The lower record was taken during stage 2 of the Bruce protocol. It shows:

- Sinus rhythm at 100 bpm
- T wave inversion persists in leads II, III, VF; but the T waves are now upright in the chest leads.

Clinical interpretation

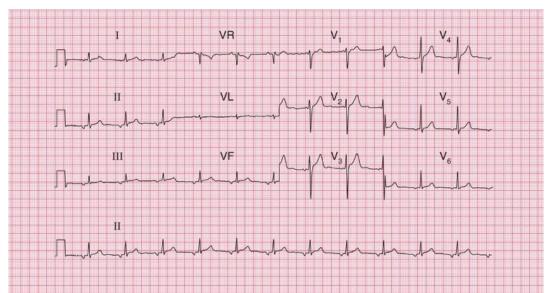
Exercise testing is no longer a central element of guideline-based assessment of stable chest pain syndromes which now largely favour imaging-based investigations. However it is still performed in many centres. The widespread T wave inversion suggests a non-ST segment elevation myocardial infarction, although there is nothing in the history to suggest when this occurred. The S wave in lead V₆ suggests the possibility of chronic lung disease. The change in the T waves in the anterior leads, from inverted at rest to normal on exercise, is an example of 'pseudonormalization', which is an indication of ischaemia.

What to do

'Pseudonormalization' must be regarded in the same way as the usual ST segment response to ischaemia, which is depression. This patient's exercise test was positive (i.e. indicates ischaemia) at a relatively low level – he requires guideline-based treatment for ischaemic heart disease and anti-anginal therapy pending a coronary angiogram with a view to revascularization.

Summary

Ischaemia with 'pseudonormalization' of the ECG on exercise.



ECG 133 This ECG was recorded from a 30-year-old woman who complained of palpitations. Does it help in making a diagnosis?

Answer 133

- Ectopic atrial rhythm, with inverted P waves in leads II, III, VF, V₃–V₆; ventricular rate 69 bpm
- Normal axis
- Normal QRS complexes and T waves.

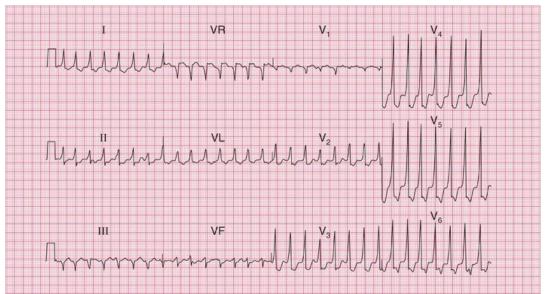
This appears to be a stable rhythm originating in the atrial muscle rather than the sinoatrial (SA) node – hence the abnormal P wave and the slightly short PR interval (130 ms). This rhythm is not uncommon, and is usually of no clinical significance. It is unlikely to be the cause of her symptoms unless at times she has a paroxysmal atrial tachycardia.

What to do

Take a careful history and attempt to determine whether her symptoms sound like a paroxysmal tachycardia – ask about any sudden onset and ending of the palpitations; associated symptoms such as breathlessness; precipitating and terminating factors; and so on. If in doubt, some sort of ambulatory recording will be needed.

Summary

Ectopic atrial rhythm.



ECG 140 A 35-year-old woman, who had had attacks of what sounded like a paroxysmal tachycardia for many years, was seen in the A&E department, and this ECG was recorded. What is the diagnosis?

Answer 140

- Narrow complex tachycardia at about 170 bpm
- No P waves visible
- Normal axis

- QRS complex duration 112 ms
- Slurred upstroke to QRS complexes, best seen in leads V_3-V_6
- Depressed ST segments in leads V₃–V₆
- Inverted T waves in the lateral leads.

This is a narrow complex tachycardia, so it is supraventricular. The slurred upstroke to the QRS complex suggests the Wolff–Parkinson–White (WPW) syndrome, so this is a re-entry tachycardia, with depolarization spreading down the accessory pathway. The absence of a dominant R wave in lead V_1 indicates that this is WPW syndrome type B. This diagnosis is consistent with the patient's history.

What to do

Carotid sinus pressure is always the first thing to try in patients with a supraventricular tachycardia. In most such patients, adenosine is the first drug to use provided you are absolutely certain the rhythm is not pre-excited atrial fibrillation (when adenosine could be dangerous). If

in doubt DC cardioversion under sedation or anaesthesia is an alternative. An electrophysiological ablation procedure is the definitive management; in the interim drugs which block the atrioventricular node (such as beta blockers, calcium channel blockers and digoxin) should be avoided but flexainide can be used as prophylaxis against further arrhythmia.

Summary

Supraventricular tachycardia and the WPW syndrome type B.