Cardiac Monitoring, Electrocardiography, and Rhythm Recognition

CHAPTER 8

Learning outcomes

To understand:

- **The reasons for ECG monitoring**
- **How to monitor the ECG**
- **The origin of the ECG**
- **The importance of recording the ECG**
- **The cardiac rhythms associated with cardiac arrest**
- **How to identify other common arrhythmias**

Introduction

During cardiac arrest, identification of the cardiac rhythm will help to determine the correct treatment. Establish cardiac monitoring as soon as possible during cardiac arrest. In many patients who have been resuscitated from cardiac arrest there is a substantial risk of further arrhythmia and cardiac arrest. Maintain cardiac monitoring in people who have been resuscitated from cardiac arrest until you are confident that the risk of recurrence is very low.

Some patients present with an arrhythmia that may lead to cardiac arrest or other serious deterioration in their condition. Early detection and treatment of the arrhythmia may prevent cardiac arrest in some patients and prevent life-threatening deterioration in others. Patients at risk include those with persistent arrhythmia associated with structural heart disease, chest pain, heart failure, reduced conscious level or shock. In all patients with persistent cardiac arrhythmia at risk of deterioration, establish cardiac monitoring and whenever possible record a good-quality 12-lead ECG. Monitoring alone will not always allow accurate rhythm recognition and it is important to document the arrhythmia for future reference if required.

Some people experience symptoms (usually syncope) caused by an intermittent cardiac arrhythmia that, if not documented and treated, could lead to cardiac arrest or sudden death. However, the arrhythmia may not be present at the time of initial assessment. In people who present with syncope undertake careful clinical assessment and record a 12-lead ECG. People who have experienced uncomplicated faints, situational syncope (such as cough syncope or micturition syncope) or syncope due to orthostatic hypotension do not require cardiac monitoring and do not usually require hospital admission. In those who have had unexplained syncope, especially during exercise, those who have had syncope and have evidence of structural heart disease, and those who have had syncope and have an abnormal ECG (especially a prolonged QT interval) start cardiac monitoring and arrange further expert cardiovascular assessment.

Single-lead ECG monitoring is not a reliable technique for detecting evidence of myocardial ischaemia (ST segment depression). Record serial 12-lead ECGs in people experiencing chest pain suggestive of an acute coronary syndrome.

During cardiac arrest, recognition of ventricular fibrillation/pulseless ventricular tachycardia (VF/VT) as shockable rhythms is crucial to the delivery of effective treatment. Automated external defibrillators (AEDs) and shock advisory defibrillators (SADs) can identify these rhythms reliably by electronic analysis. If a shockable rhythm is present, the defibrillator will charge to the appropriate energy level and instruct the operator that a shock is required. The introduction of AEDs has enabled resuscitation from VF/VT to be achieved by people who do not have skill in rhythm recognition, both in hospitals and in the community.

The accurate analysis of some cardiac rhythm abnormalities requires experience and expertise; however, the non-expert can interpret most rhythms sufficiently to identify the appropriate treatment. The main priority is to recognise that the rhythm is abnormal and that the heart rate is inappropriately slow or fast. Use the structured approach to rhythm interpretation, described in this chapter, to avoid errors. The need for immediate treatment will be determined largely by the effect of the arrhythmia on the patient rather than by the nature of the arrhythmia. When an arrhythmia is present, first assess the patient (use the ABCDE approach), and then interpret the rhythm as accurately as possible. Treat the patient, not the ECG!

Techniques for ECG monitoring

Cardiac monitors

Cardiac monitors display the ECG on a screen in real time. The signal is obtained from adhesive electrodes on the patient's skin and transmitted to the monitor either by wires or by telemetry. Many monitor systems have other features, such as the ability to print samples of the ECG rhythm display or to store samples of the ECG. Most monitors include a display of heart rate, and some have alarms that can be programmed to provide an alert when the heart rate goes below or exceeds preset limits.

Many systems enable monitoring of other values such as blood pressure and oxygen saturation, which are important in the assessment of patients at risk. Digital processing of the ECG offers the potential for electronic analysis of the cardiac rhythm. If a patient requires monitoring, make sure that the monitor is being observed so that immediate action can be taken if necessary, should the rhythm change.

How to attach the monitor

Attach ECG electrodes to the patient using the positions shown in Figure 8.1. These will enable monitoring using 'modified limb leads' I, II and III. Make sure that the skin is dry, not greasy (use an alcohol swab and/or abrasive pad to clean), and either place the electrodes on relatively hair-free skin or shave off dense hair. Place electrodes over bone rather than muscle, to minimise interference from muscle artefact in the ECG signal. Different electrode positions may be used when necessary (e.g. trauma, recent surgery, skin disease).

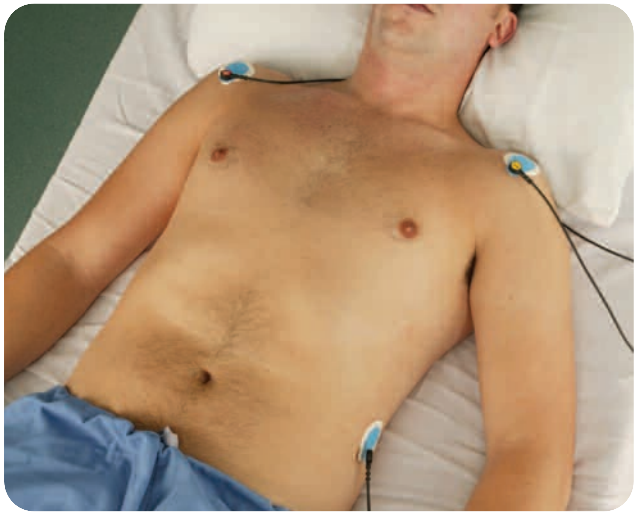


Figure 8.1 Position of electrode for monitoring the ECG using modified limb leads

Most leads are colour-coded to help with correct connection. The usual scheme (except in the United States) uses **R**ed for the **R**ight arm lead, ye**LL**ow for the **L**eft arm lead, **G**reen for the le**G** lead (usually placed on the abdomen or lower left chest wall) for modified limb leads.

Begin by monitoring in modified lead II as this usually displays good amplitude sinus P waves and good amplitude QRS complexes, but switch to another lead if necessary to obtain the best ECG signal. Try to minimise muscle and movement artefact by explaining to patients what the monitoring is for and by keeping them warm and relaxed.

Emergency monitoring

In an emergency, such as a collapsed patient, assess the cardiac rhythm as soon as possible by applying adhesive defibrillator pads, which can be used for monitoring and hands-free shock delivery (Figure 8.2). Apply the pads in the conventional positions, beneath the right clavicle and in the left mid-axillary line. Use anterior and posterior positions as an alternative if the conventional positions cannot be used (e.g. permanent pacemaker in right pectoral position, chest wall trauma). The rapid application of manual defibrillator paddles also enables the cardiac rhythm to be determined rapidly, but in most healthcare environments these paddles have been replaced with hands-free adhesive defibrillator pads.

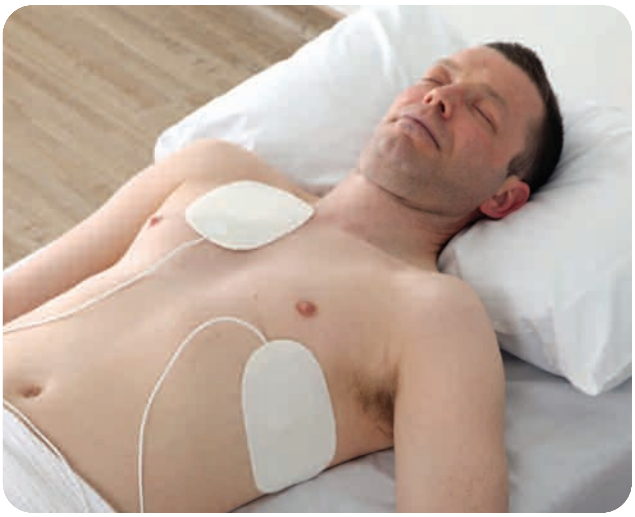


Figure 8.2 Defibrillator pads

Diagnosis from cardiac monitors

Use the displays and printouts from cardiac monitors only for rhythm recognition; do not attempt to interpret ST segment abnormalities or other more sophisticated elements of the ECG from monitors. When an arrhythmia is detected on a monitor, record a rhythm strip whenever possible.

If the arrhythmia persists for long enough, record a 12-lead ECG. It is not always possible to identify an arrhythmia from a single lead ECG recording. The heart is a three-dimensional organ and the 12-lead ECG examines the electrical signals from the heart in three dimensions. Sometimes, features that enable precise identification of cardiac rhythm are visible in only one or two leads of the 12-lead ECG and would not be seen on a single-lead recording of any other lead (Figure 8.3).

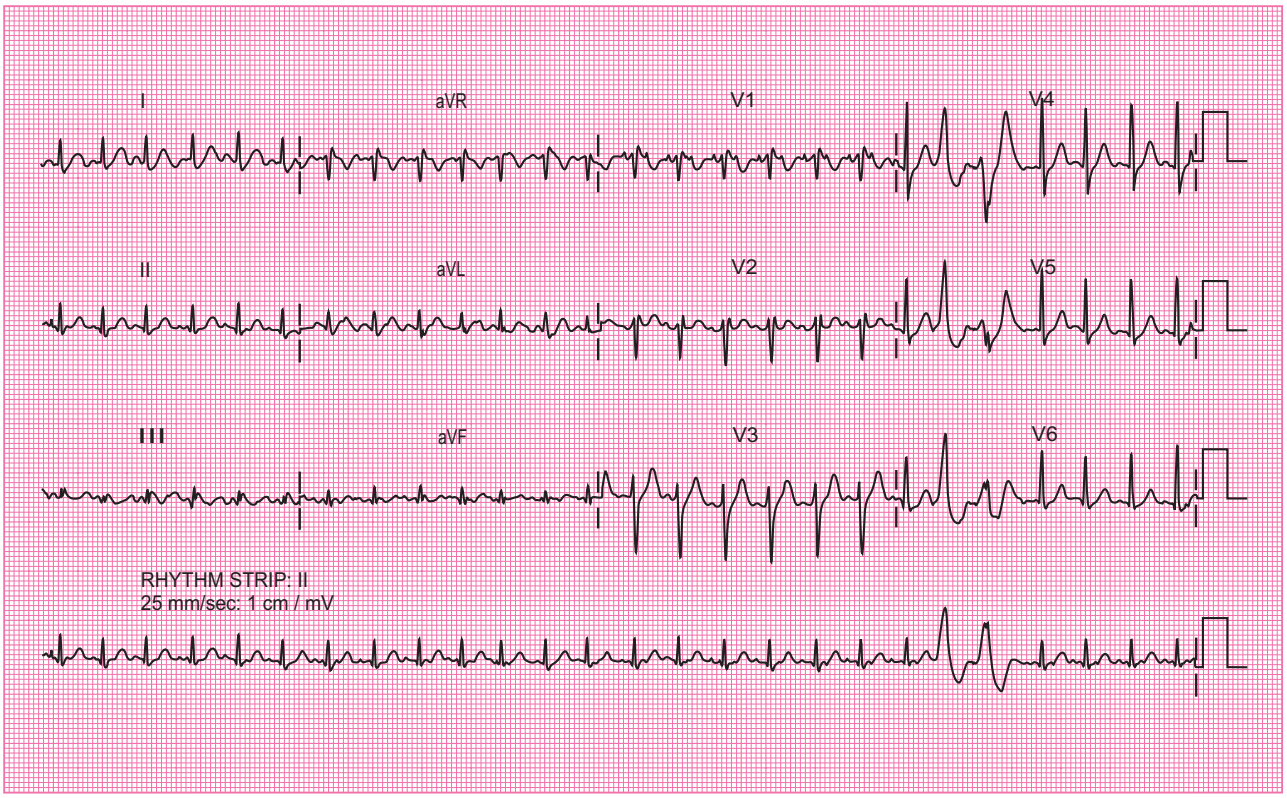


Figure 8.3 12-lead ECG showing atrial tachycardia, which is seen clearly only in lead V1

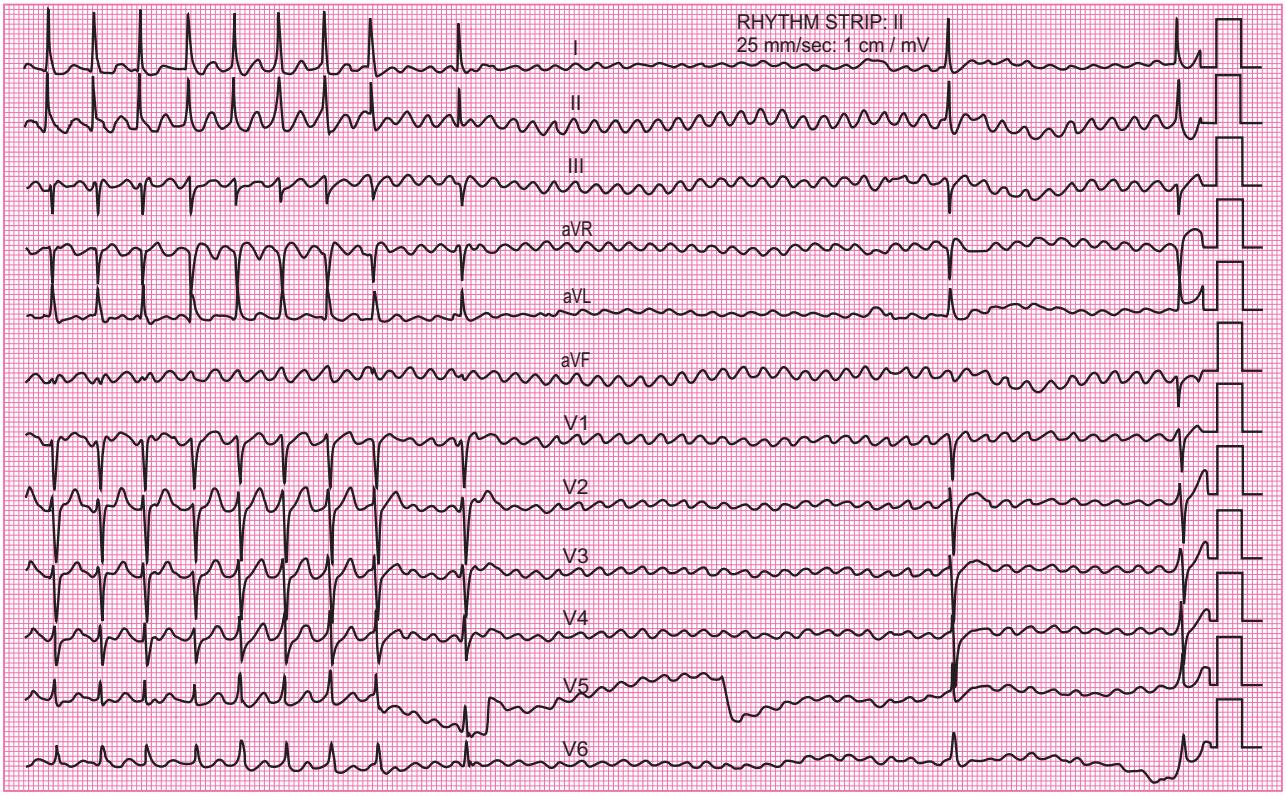


Figure 8.4 12-lead ECG showing the effect of adenosine in atrial flutter. Transient AV block demonstrates clearly that this regular narrow-complex tachycardia was atrial flutter with 2:1 AV conduction

These recordings may assist with rhythm interpretation at the time but are also useful for later examination and planning of treatment in the longer term. Therefore effective management of any arrhythmia, including a cardiac arrest arrhythmia, includes good quality ECG recording, as well as interpretation and treatment at the time.

Valuable information about the nature and origin of a tachyarrhythmia can also be obtained by recording the response to treatment (e.g. carotid sinus massage, adenosine). Whenever possible, the effect of any such intervention should be recorded on a continuous ECG recording, if possible using multiple leads (Figure 8.4).

Basic electrocardiography

At rest, the cells of the cardiac conducting system and myocardium are polarised. A potential difference of approximately 90 mV is present between the inside of the cell (which is negatively charged) and the extracellular space. A sudden shift of ions across the cell membrane triggers depolarisation, generating the electrical signal that travels through the conducting system and triggers contraction of myocardial cells.

In normal sinus rhythm, depolarisation begins in a group of specialised 'pacemaker' cells, called the sino-atrial (SA) node, located close to the entry of the superior vena cava into the right atrium. A wave of depolarisation then spreads from the SA node through the atrial myocardium.

This is seen on the ECG as the P wave (Figure 8.5). Atrial contraction is the mechanical response to this electrical impulse.

The transmission of this electrical impulse to the ventricles occurs through specialised conducting tissue (Figure 8.6).

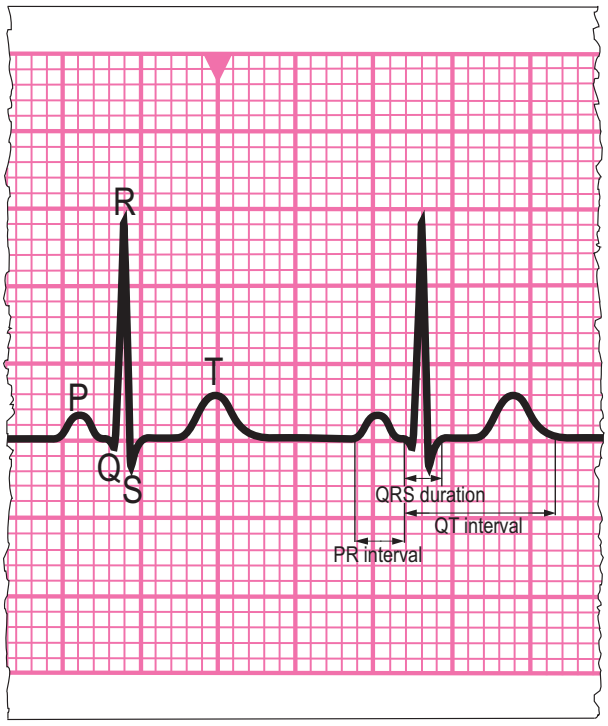


Figure 8.5 Components of the normal ECG signal

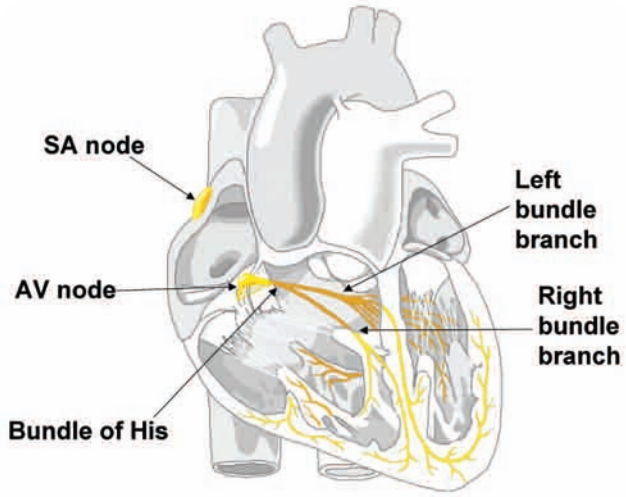


Figure 8.6 Electrical conduction in the heart

Firstly, there is slow conduction through the atrioventricular (AV) node, followed by rapid conduction to the ventricular myocardium by specialised conducting tissue (Purkinje fibres). The bundle of His carries these fibres from the AV node and then divides into right and left bundle branches, spreading out through the right and left ventricles respectively. Rapid conduction down these fibres ensures that the ventricles contract in a co-ordinated fashion.

Depolarisation of the bundle of His, bundle branches and ventricular myocardium is seen on the ECG as the QRS complex (Figure 8.5). Ventricular contraction is the mechanical response to this electrical impulse.

Between the P wave and QRS complex is a small isoelectric segment, which largely represents the delay in transmission through the AV node. The normal sequence of atrial depolarisation followed by ventricular depolarisation (P wave followed by QRS complex) is sinus rhythm (Rhythm Strip 1).

The T wave, which follows the QRS complex, represents recovery of the resting potential in the cells of the conducting system and ventricular myocardium (ventricular repolarisation).

Because the normal conducting system transmits the depolarising impulse rapidly to both ventricles, the normal QRS complex is of relatively short duration (normally < 0.12 s).

When one of the bundle branches is diseased or damaged, rapid conduction to the corresponding ventricle is prevented. The depolarising impulse travels more rapidly down the other bundle branch to its ventricle and then more slowly, through ordinary ventricular myocardium to the other ventricle. This situation is called bundle branch block. Because depolarisation of both ventricles takes longer than normal it is seen on the ECG as a broad QRS complex (0.12 s or longer).

How to read a rhythm strip

Experience and expertise may be needed to identify some rhythm abnormalities with complete precision. However, a simple, structured approach to interpreting the rhythm on any ECG recording will define any rhythm in sufficient detail to enable the most appropriate treatment to be chosen.

Apply the following 6-stage system to the analysis of any rhythm on an ECG:

1. Is there any electrical activity?
2. What is the ventricular (QRS) rate?
3. Is the QRS rhythm regular or irregular?
4. Is the QRS complex width normal or prolonged?
5. Is atrial activity present?
6. Is atrial activity related to ventricular activity and, if so, how?

Any cardiac rhythm can be described accurately (e.g. irregular narrow complex tachycardia, regular broad-complex bradycardia, etc.) and managed safely and effectively using the first four steps.

Is there any electrical activity?

If you cannot see any electrical activity, check that the gain control is not too low and that the electrodes and leads are connected to both the patient and the monitor.

Check the patient: is a pulse present? If the patient is pulseless and there is still no activity on the ECG this is asystole (Rhythm Strip 2). Atrial and ventricular asystole are often both present, resulting in a line with no deflections. A completely straight line indicates usually that a monitoring lead has become disconnected. During asystole the ECG usually shows slight undulation of the baseline, and may show electrical interference due to respiratory movement, or chest compression.

Atrial activity (usually P waves but occasionally atrial fibrillation (AF) or atrial flutter) may continue for a short time after the onset of ventricular asystole. The ECG will show the atrial activity but no QRS complexes - ventricular standstill (Rhythm Strip 3). Recognition of this is important because pacing is more likely to achieve a cardiac output in this situation than in most cases of complete asystole (Chapter 10).

If the patient is pulseless and electrical activity is present, decide whether recognisable QRS complexes are present. If not, and the ECG shows rapid, bizarre, irregular deflections of random frequency and amplitude, this is VF (Rhythm Strip 4). In VF all co-ordination of electrical activity is lost, and there is no effective ventricular contraction, and no detectable cardiac output.

Ventricular fibrillation is sometimes classified as coarse (Rhythm Strip 4) or fine (Rhythm Strip 5) depending on the amplitude of the complexes; If there is doubt about whether the rhythm is asystole or fine VF, do not attempt defibrillation; instead, continue chest compressions and ventilation. Fine VF that is difficult to distinguish from asystole is unlikely to be shocked successfully into a rhythm that produces a cardiac output. Continuing good-quality CPR may improve the amplitude and frequency of the VF and improve the chance of subsequent successful defibrillation and return of spontaneous circulation. Delivering repeated shocks in an attempt to defibrillate what is thought to be fine VF will increase myocardial injury both directly from the electric current and indirectly from the interruptions in coronary blood flow (Chapter 6).

If electrical activity is present and contains recognisable QRS complexes, continue with the following steps in rhythm analysis.

If the patient is pulseless and there are recognisable complexes on the ECG that would be expected to produce a pulse, this is pulseless electrical activity (PEA) and requires immediate CPR. Do not delay CPR whilst the cardiac rhythm is analysed further.

What is the ventricular (QRS) rate?

The normal heart rate (ventricular rate) at rest is 60 - 100 beats min^{-1}. A bradycardia has a heart rate slower than 60 min^{-1}. A tachycardia has a rate faster than 100 min^{-1}. ECG paper is calibrated in mm, with bolder lines every 5 mm. Standard paper speed in the UK is 25 mm s^{-1}. One second is represented by 5 large squares (25 small squares).

The best way of estimating the heart rate is to count the number of cardiac cycles that occur in 6 s (30 large squares) and multiply by 10. This provides an estimate of heart rate, even when the rhythm is somewhat irregular. For example, if 20 cardiac cycles occur in 30 large squares the rate is 200 min^{-1} (Figure 8.7). For shorter rhythm strips count the number of cardiac cycles in 3 s (15 large squares) and multiply by 20.

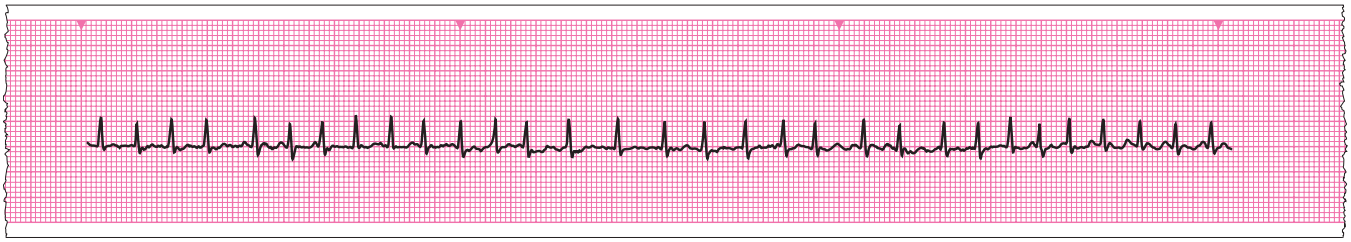


Figure 8.7 Calculation of heart rate from a rhythm strip (20 cardiac cycles occur in 30 large squares = 200 min^{-1}).

Is the QRS rhythm regular or irregular?

This is not always as easy as it seems; at faster heart rates beat-to-beat variation during some irregular rhythms appears less obvious. Some rhythms may be regular in places but intermittent variation in R-R interval makes them irregular. Inspect an adequate length of rhythm strip carefully, measuring out each R-R interval and comparing it to others to detect any irregularity that is not obvious at first glance. Dividers are very useful for comparing the R-R intervals. Alternatively, the position of two adjacent identical points in the cardiac cycle (such as the tips of the R waves) can be marked on a strip of paper; this can then be moved to another section of the rhythm strip. If the rhythm is regular the marks will align precisely with each pair of R waves.

If the QRS rhythm is irregular, decide:

- Is this totally irregular, with no recognisable pattern of R-R interval?
- Is the basic rhythm regular, with intermittent irregularity?
- Is there a recurring cyclical variation in the R-R intervals?

If there is a cyclical pattern, the relationship between the QRS waves and the P wave requires careful analysis, as described below. If the R-R intervals are totally irregular (irregularly irregular) and the QRS complex is of constant morphology, the rhythm is most likely to be AF (Rhythm Strip 6).

A regular underlying rhythm may be made irregular by extrasystoles (ectopic beats). Extrasystoles can arise from the atria or the ventricles, and the position or focus from which they arise will determine their morphology on an ECG.

If the QRS complex of ectopic beats is narrow (< 0.12 s), the beat is likely to have come from above the ventricular myocardium (i.e. from atrial muscle or the AV node).

Broad-complex ectopic beats may be of ventricular origin or may be supraventricular ectopic beats with bundle branch block.

Broad-complex atrial premature beats can sometimes be identified by a preceding ectopic P wave. Ventricular ectopic beats can be accompanied by a P wave occurring shortly after the QRS complex, caused by retrograde conduction from the ventricles to the atria.

Ectopic beats that occur early (that is before the next regular sinus beat was due to occur) are referred to as premature beats (Rhythm Strip 7).

A beat that arises from the AV node or from ventricular myocardium after a long pause, for example during sinus bradycardia or after sinus arrest, is referred to as an escape beat (Rhythm Strip 8). This implies that the focus in the AV node or ventricle that generates this beat is acting as a back-up pacemaker, because the normal pacemaker function of the sinus node is too slow or absent. Ectopic beats may occur singly, in pairs (couplets) or in threes (triplets). If more than three ectopic beats occur in rapid succession, this is regarded as a tachyarrhythmia.

An arrhythmia that occurs intermittently, interspersed with periods of normal sinus rhythm, is described as paroxysmal.

When ectopic beats occur alternately with sinus beats for a sustained period this is called bigeminy. It may be referred to as atrial bigeminy or ventricular bigeminy, depending on whether the ectopic beats are atrial or ventricular in origin.

Is the QRS complex width normal or prolonged?

The upper limit of normal for the QRS interval is 0.12 s (3 small squares). If the QRS width is less than this, the rhythm originates from above the bifurcation of the bundle of His and may be from the SA node, atria or AV node, but not from the ventricular myocardium. If the QRS duration is 0.12 s or more the rhythm may be coming from ventricular myocardium or may be a supraventricular rhythm, transmitted with aberrant conduction (i.e. bundle branch block).

Is atrial activity present?

Having defined the rhythm in terms of rate, regularity and QRS width, examine the ECG carefully for evidence of atrial activity. This may be difficult or impossible to identify, either because it is not visible or because atrial activity is partly or completely obscured by QRS complexes or T waves. Do not guess or try to convince yourself that you can identify atrial activity unless you are completely sure.

Depending on the nature of the arrhythmia and the ECG lead being examined, P waves may be present as positive deflections, negative deflections or biphasic deflections. When present, U waves may be mistaken for P waves. P waves may coincide with and cause distortion or variation of QRS complexes, ST segments, or T waves. Whenever possible, recording of a 12-lead ECG may enable P waves to be identified in one or more leads, even if they cannot be seen clearly in the initial monitoring lead. Lead V1 is often useful for clear demonstration of some types of atrial activity including sinus P waves and AF. Sinus P waves are usually seen clearly in lead II.

Other types of atrial activity may be present. During atrial flutter, atrial activity is seen as flutter waves - an absolutely regular repetitive deflection with a 'saw-tooth' appearance, often at a rate of about 300 min^{-1}. This is usually seen best in the inferior leads (II, III, aVF) (Figure 8.4).

During AF, circuits and waves of depolarisation travel randomly through both atria. There are no P waves. Atrial fibrillation waves may be seen as rapid deviations from the baseline of varying amplitude and duration, usually seen best in lead V1. In some patients this may be of such low amplitude that no atrial activity can be seen.

During a sustained tachycardia atrial activity may not be visible between the QRS complexes. If the rhythm is of atrial origin (e.g. atrial flutter or AF) it may be possible to reveal atrial activity by slowing the ventricular rate whilst recording an ECG, preferably in multiple leads. For example, if a regular tachycardia of 150 min^{-1} is due to atrial flutter with 2:1 conduction it may not be possible to identify flutter waves with confidence. A transient increase in AV block by vagal stimulation or by an intravenous bolus of adenosine will demonstrate the flutter waves and identify the rhythm accurately (Figure 8.4).

The shape and direction of P waves help to identify the atrial rhythm. For example, sinus P waves are upright in leads II and aVF. If retrograde activation of the atria is taking place from the region of the AV node (i.e. the rhythm is junctional or ventricular in origin), the P waves will be inverted in leads II and aVF because atrial depolarisation travels in the opposite direction to normal.

P wave rate and regularity (and flutter wave rate) are assessed in the same way as the rate and regularity of QRS complexes.

Is atrial activity related to ventricular activity and, if so, how?

If there is a consistent interval between each P wave and the following QRS complex, it is likely that conduction between atrium and ventricle is intact and that ventricular depolarisation is triggered by atrial depolarisation. Examine a long rhythm strip to make sure that subtle variation in the PR interval is not missed. Occasionally conduction between atria and ventricles is reversed (i.e. ventricular depolarisation is followed by retrograde conduction through the AV node and then by atrial depolarisation); the P wave occurs soon after the QRS complex. It may sometimes be difficult to distinguish between this situation and the presence of a very long PR interval.

In other circumstances careful inspection will detect no relationship between the timing of P waves and of QRS complexes. This will indicate that atrial and ventricular depolarisation is arising independently, sometimes referred to as atrioventricular dissociation. Examples of this include:

- Complete (third degree) AV block, where a normal sinus rate in the atria is accompanied by a regular bradycardia arising below the AV node.
- Some examples of VT in which regular broad QRS complexes are present and regular P waves can be seen at a different, slower rate, out of phase with the QRS complexes.

Difficulty may arise when the relationship between the P waves and the QRS complexes varies in a recurring pattern. This may be misinterpreted as atrioventricular dissociation. This is seen most commonly in one form of second degree AV block (called Wenkebach or Mobitz I AV block). Examine a long rhythm strip carefully for recurring patterns and plot and compare the timing of P waves and QRS complexes. In complete AV block, the QRS rhythm is usually completely regular.

In AF, the atrial activity is completely irregular, so there is no identifiable relationship between this atrial activity and the irregular ventricular rhythm that results from it. If AF is accompanied by a completely regular, slow ventricular rhythm this is likely to be due to complete AV block in the presence of AF in the atria.

In atrial flutter there may be a consistent relationship between the flutter waves and the QRS complexes, giving rise to 1:1, 2:1, 3:1 conduction etc. In some instances, there is a constantly varying relationship, producing an irregular QRS rhythm; this is atrial flutter with variable AV block.

Cardiac arrest rhythms

The rhythms present during cardiac arrest can be classified into 3 groups:

- ventricular fibrillation (VF) and some cases of ventricular tachycardia (VT);
- asystole;
- pulseless electrical activity (PEA).

Extreme bradycardia and rarely very fast supraventricular tachyarrhythmia may also cause such a severe fall in cardiac output to effectively cause cardiac arrest.

Ventricular fibrillation

The characteristic appearance of VF (Rhythm Strip 4) is usually easy to recognise, and this is the only rhythm that does not need the systematic rhythm analysis described earlier in this chapter. When a monitor appears to show VF check the patient immediately to establish whether this is VF requiring immediate defibrillation, or whether the appearance is due to artefact. If the patient has a pulse, the rhythm is not VF.

Two rhythm abnormalities may resemble VF in some circumstances, since both produce an irregular, broad-complex, fast rhythm:

One is polymorphic VT (Rhythm Strip 12). This may cause cardiac arrest, and when it does so the immediate treatment is the same as for VF, so failure to distinguish this immediately from VF would not lead to inappropriate treatment. However, it is important to document polymorphic VT and to recognise it following immediate resuscitation, so that the causes can be identified and corrected and appropriate treatment given to prevent recurrence.

The second possible source of confusion is pre-excited AF. This occurs in the presence of an accessory pathway connecting atrial and ventricular muscle in the Wolff-Parkinson-White (WPW) syndrome. Some of these accessory pathways can conduct very rapidly, transmitting atrial impulses to the ventricles, sometimes at 300 min^{-1} or faster. This produces an irregular broad complex tachycardia (Figure 8.8) that does not usually resemble VF but might be mistaken for polymorphic VT. Left untreated, this rhythm may lead to VT or VF causing cardiac arrest. If AF with WPW syndrome itself caused clinical cardiac arrest, the correct treatment would be immediate defibrillation (as for any broad-complex pulseless tachycardia) so misinterpretation as VT or VF would not lead to inappropriate treatment. Again, the importance of documenting and recognising the rhythm is to ensure that the patient receives immediate appropriate specialist referral for treatment to protect them against the risk of recurrence of this potentially dangerous arrhythmia.

Ventricular tachycardia

Ventricular tachycardia (VT) may cause loss of cardiac output resulting in cardiac arrest, particularly at faster rates or in the presence of structural heart disease (e.g. impaired left ventricular function, severe left ventricular hypertrophy, aortic stenosis). VT may degenerate suddenly into VF. Pulseless VT is treated in the same way as VF by immediate defibrillation.

In the presence of a cardiac output (i.e. palpable pulse), treatment of VT should follow the broad complex tachycardia algorithm described in Chapter 11.

The QRS morphology may be monomorphic or polymorphic. In monomorphic VT (Rhythm Strip 10), the rhythm is regular (or almost regular). The rate during VT may be anything from 100 to 300 min^{-1}, rarely faster. It is unusual to see more than slight variation in heart rate during any single episode of VT (other than in response to anti-arrhythmic drug therapy). Atrial activity may continue independently of ventricular activity; the identification of P waves, dissociated from QRS complexes during broad complex tachycardia, identifies the rhythm as VT. Occasionally these atrial beats may be conducted to the ventricles, causing capture beats or fusion beats (Rhythm Strip 11). A capture beat produces a single normal-looking QRS complex during monomorphic VT, without otherwise interrupting the arrhythmia. In a fusion beat, a wave of depolarisation travelling down from the AV node occurs simultaneously with a wave of depolarisation travelling up from the ventricular focus producing the arrhythmia. This results in a hybrid QRS complex caused by fusion of the normal QRS complex with the complex of the monomorphic VT.

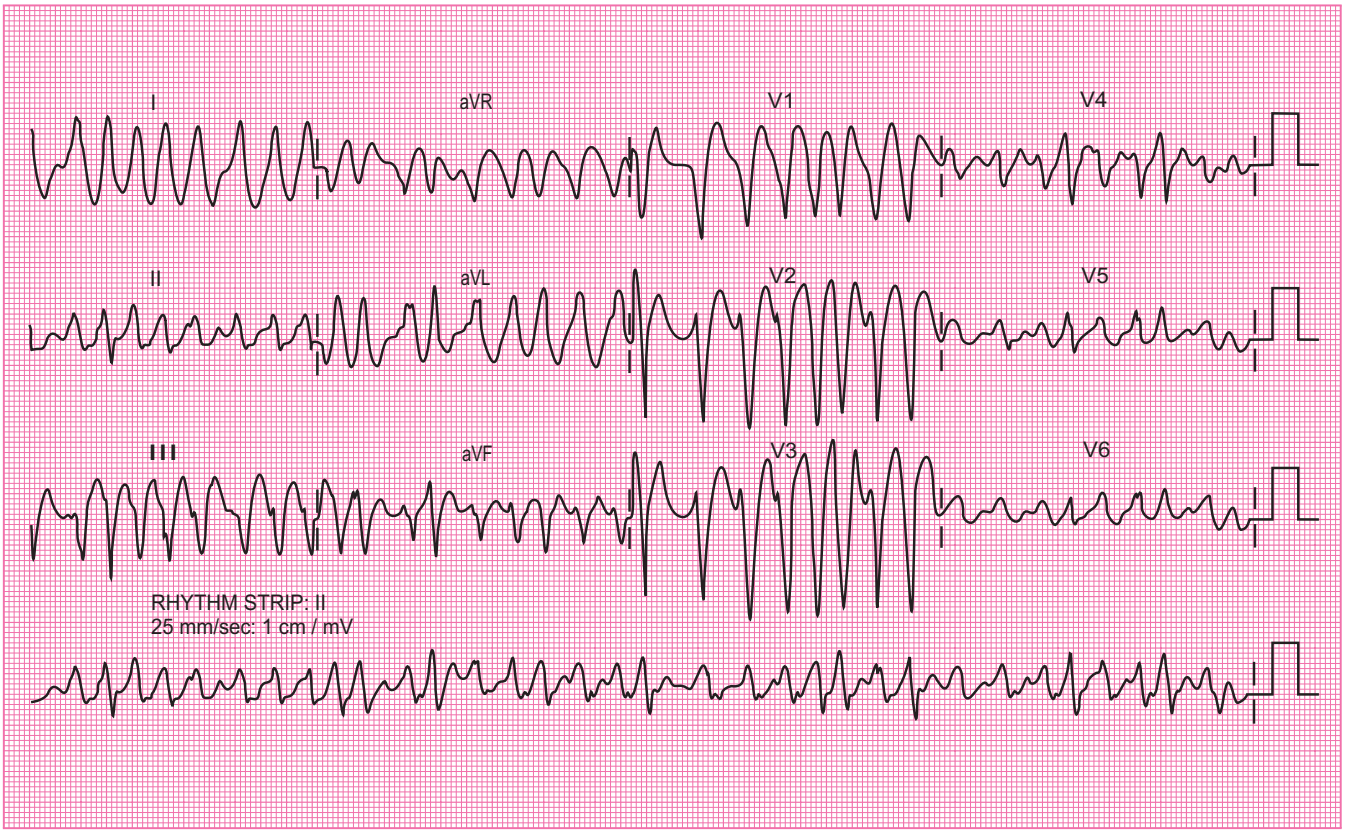


Figure 8.8 12-lead ECG showing pre-excited atrial fibrillation in a patient with Wolff-Parkinson-White syndrome

In the presence of bundle branch block, a supraventricular tachycardia (SVT) will produce a broad complex tachycardia. After myocardial infarction, most broad complex tachycardia will be ventricular in origin. The safest approach is to regard all broad complex tachycardia as VT until, or unless, proved otherwise.

One important type of polymorphic VT is torsade de pointes (TDP) in which the axis of the electrical activity changes in a rotational way so that the overall appearance of the ECG on a rhythm strip produces a sinusoidal pattern (Rhythm Strip 12). This arrhythmia usually arises in patients with a prolonged QT interval. This can occur as an inherited phenomenon in some families (long QT syndromes). In some people it is caused by drugs, including some anti-arrhythmic drugs, and it may occur less commonly as a manifestation of myocardial ischaemia. Many patients with TDP VT are also hypokalaemic and/or hypomagnesaemic. It is important to recognise TDP VT, because effective treatment (prevention of recurrent episodes) will require removal of any predisposing causes (i.e. drugs), treatment with intravenous magnesium and/or potassium, and may also require the use of overdrive pacing. Drugs that prolong QT interval (including amiodarone) should be avoided in patients with TDP VT. This arrhythmia can itself cause cardiac arrest (in which case it is treated by defibrillation) and it can also degenerate into VF.

Asystole

The appearance of asystole has been described already (Rhythm Strip 2). Sometimes it is not clear whether the observed rhythm is asystole or very fine VF. In this situation, immediate treatment is to provide high quality CPR. If fine VF was present, good CPR may increase the amplitude and frequency of the VF, making that diagnosis clear and increasing the probability of successful defibrillation.

Pulseless electrical activity

The term pulseless electrical activity (PEA) does not refer to a specific cardiac rhythm. It defines the clinical absence of cardiac output despite electrical activity that would normally be expected to produce a cardiac output. It generally has a poor prognosis especially when it is caused by a very large acute myocardial infarction. Potentially more treatable causes include massive pulmonary embolism, tension pneumothorax, cardiac tamponade and acute severe blood loss.

Peri-arrest arrhythmias

These are defined according to heart rate (bradyarrhythmia, tachyarrhythmia or arrhythmia with a normal rate), as this will dictate initial treatment (Chapter 11). In the unstable patient, concentrate on early treatment to prevent deterioration, rather than on prolonged attempts to identify the precise rhythm.

Bradyarrhythmia

A bradycardia is present when the ventricular (QRS) rate is < 60 min^{-1} (Rhythm Strip 13). Bradycardia may be a physiological state in very fit people or during sleep, or may be an expected result of treatment (e.g. with a beta-blocker). Pathological bradycardia may be caused by malfunction of the SA node or from partial or complete failure of atrioventricular conduction. Some patients with these rhythm abnormalities may need treatment with an implanted pacemaker (Rhythm Strip 14).

The emergency treatment of most bradycardia is with atropine and/or cardiac pacing. Occasionally it may be necessary to use sympathomimetic drugs such as isoprenaline or adrenaline. The need for treatment depends on the haemodynamic effect of the arrhythmia and the risk of developing asystole, rather than the precise ECG classification of the bradycardia. Extreme bradycardia may sometimes precede cardiac arrest and this may be prevented by prompt and appropriate treatment. In this context the most important bradyarrhythmia is acquired complete heart block (see below).

Heart block: first degree atrioventricular block

The PR interval is the time between the onset of the P wave and the start of the QRS complex (whether this begins with a Q wave or R wave). The normal PR interval is between 0.12 and 0.20 s. First degree atrioventricular (AV) block is present when the PR interval is > 0.20 s and is a common finding (Rhythm Strip 15). It represents a delay in conduction through the AV junction (the AV node and bundle of His). In some instances this may be physiological (for example in trained athletes). There are many other causes of first degree AV block, including primary disease (fibrosis) of the conducting system, various types of structural heart disease, ischaemic heart disease and use of drugs that delay conduction through the AV node. First degree AV block rarely causes any symptoms and as an isolated finding rarely requires treatment.

Heart block: second degree atrioventricular block

Second degree AV block is present when some, but not all, P waves are conducted to the ventricles, resulting in absence of a QRS complex after some P waves. There are two types:

Mobitz Type I AV block (also called Wenckebach AV block)

The PR interval shows progressive prolongation after each successive P wave until a P wave occurs without a resulting QRS complex. Usually the cycle is then repeated (Rhythm Strip 16).

Any condition that delays AV conduction can produce Wenkebach AV block. In some situations this may be physiological, for example in highly trained athletes with high vagal tone. Outside that setting Wenckebach AV block is usually pathological. Its many causes include acute myocardial infarction (especially inferior infarction). If asymptomatic, this rhythm does not usually require immediate treatment. The need for treatment is dictated by the effect of the bradyarrhythmia on the patient and the risk of developing more severe AV block or asystole.

Mobitz Type II AV block

There is a constant PR interval in the conducted beats but some of the P waves are not followed by QRS complexes. This may occur randomly, without any consistent pattern. People with Mobitz II AV block have an increased risk of progression to complete AV block and asystole.

2:1 and 3:1 AV block

The term 2:1 AV block describes the situation in which alternate P waves are followed by a QRS complex (Rhythm Strip 17). 2:1 AV block may be due to Mobitz I or Mobitz II AV block and it may be difficult to distinguish which it is from the ECG appearance. If bundle branch block is present as well as 2:1 block (broad QRS complexes) this is likely to be Mobitz II block. 3:1 AV block (Rhythm strip 18) is less common and is a form of Mobitz II AV block. Immediate decisions about treatment of these rhythms (see algorithm for treatment of bradycardia - Chapter 11) will be determined by the effect of the resulting bradycardia on the patient. After identifying and providing any necessary immediate treatment continue cardiac monitoring and arrange expert cardiological assessment.

Heart block: third degree atrioventricular block

In third degree (complete) AV block, there is no relationship between P waves and QRS complexes; atrial and ventricular depolarisation arises independently from separate 'pacemakers' (Rhythm Strip 19). The site of the pacemaker stimulating the ventricles will determine the ventricular rate and QRS width. A pacemaker site in the AV node or proximal bundle of His may have an intrinsic rate of 40 - 50 min^{-1} or sometimes higher and may produce a narrow QRS complex. A pacemaker site in the distal His-Purkinje fibres or ventricular myocardium will produce broad QRS complexes, often have a rate of 30 - 40 min^{-1} or less, and is more likely to stop abruptly, resulting in asystole.

Escape rhythms

If the normal cardiac pacemaker (SA node) fails, or operates abnormally slowly, cardiac depolarisation may be initiated from a 'subsidiary' pacemaker in atrial myocardium, AV node, conducting fibres or ventricular myocardium. The resulting escape rhythm will be slower than the normal sinus rate. As indicated above, subsidiary pacemakers situated distally in the conducting system tend to produce slower heart rates than those situated more proximally. Thus a ventricular escape rhythm will usually be slower than a 'junctional' rhythm arising from the AV node or bundle of His.

The term idioventricular rhythm is used to describe a rhythm arising from ventricular myocardium. This includes ventricular escape rhythms seen in the presence of complete AV block. The term accelerated idioventricular rhythm is used to describe an idioventricular rhythm with a normal heart rate (usually faster than the sinus rate but not fast enough to be VT). This type of rhythm is observed quite frequently after successful thrombolysis (or primary percutaneous coronary intervention) for acute myocardial infarction (a 'reperfusion arrhythmia'). Accelerated idioventricular rhythms do not influence prognosis unless they cause haemodynamic compromise or develop into VT or VF, which is uncommon. The QRS complex of an idioventricular rhythm will be broad (i.e. 0.12 s or greater), whereas a junctional rhythm may be narrow or broad, depending on whether conduction to the ventricles occurs normally, or with bundle branch block.

Agonal rhythm

Agonal rhythm occurs in dying patients. It is characterised by the presence of slow, irregular, wide ventricular complexes, often of varying morphology (Rhythm Strip 20). This rhythm is seen commonly during the later stages of unsuccessful resuscitation attempts. The complexes slow inexorably and often become progressively broader before all recognisable activity is lost.

Tachyarrhythmia

A pathological tachycardia may arise from atrial myocardium, the AV junction or ventricular myocardium. Sinus tachycardia is not an arrhythmia and usually represents a response to some other physiological or pathological state (e.g. exercise, anxiety, blood loss, fever etc).

Narrow-complex tachycardia

When a tachycardia arises from tissue situated above the bifurcation of the bundle of His, it is described as supraventricular (Rhythm Strip 21). The QRS complexes will be narrow if ventricular depolarisation occurs normally, but will be broad if bundle branch block is present. QRS complexes may be regular in many rhythms or may be irregular in the presence of atrial fibrillation or variably conducted atrial flutter. Most tachycardia with narrow QRS complexes has a favourable prognosis, but the outlook will vary with individual clinical circumstances. These rhythms may be tolerated poorly by patients with structural heart disease and may provoke angina, especially in patients with coronary artery disease.

Atrial fibrillation

Atrial fibrillation is the most common arrhythmia encountered in clinical practice. It is characterised by disorganised electrical activity in the atria. No recognisable P waves or co-ordinated atrial activity can be seen in any lead (Rhythm Strip 6). The baseline is irregular and chaotic atrial activity is best seen in lead V1 where the atrial waveform is irregular in both amplitude and frequency. The QRS rhythm is irregularly irregular (i.e. there is no consistent R-R interval from beat to beat). The ventricular rate will depend on the refractory period of the AV junction. In the absence of drug treatment or pre-existing disease affecting the AV node, the resulting ventricular rate will be rapid, usually 120 - 180 min^{-1} or faster.

Common causes of AF include hypertension, obesity, alcohol excess and structural heart disease. In coronary heart disease AF usually results from left ventricular impairment (acute or chronic) and not as a direct result of ischaemia of the atrial myocardium.

Atrial flutter

In atrial flutter, atrial activity is seen on the ECG as flutter or F waves at a rate of about 300 min^{-1} (Rhythm Strip 22). These are best seen in the inferior leads II, III and aVF where they have a 'saw-tooth' appearance (Figure 8.4). The ventricular rate depends on AV conduction but there is often 2:1 (Rhythm Strip 9) or 3:1 conduction (often referred to as atrial flutter with 2:1 or 3:1 block). If conduction is constant the ventricular rhythm will be regular, but variable conduction causes an irregular ventricular rhythm. Like atrial fibrillation, atrial flutter is often, but not always, associated with underlying disease. Atrial flutter usually arises in the right atrium so is a recognised complication of diseases that affect the right heart, including chronic obstructive pulmonary disease, major pulmonary embolism, complex congenital heart disease and chronic congestive heart failure of any cause.

Broad-complex tachycardia

Broad-complex tachycardia may be:

- a tachycardia arising in the ventricle below the bifurcation of the bundle of His, i.e. VT (Rhythm Strip 10); or
- a supraventricular tachycardia conducted aberrantly (right or left bundle branch block) to the ventricles.

The clinical consequences depend on:

- heart rate during the arrhythmia;
- the presence or absence of structural heart disease or coronary disease;
- duration of the arrhythmia.

Ventricular tachycardia may degenerate into VF, especially if the VT is very fast (e.g. 200 min^{-1} or faster) or if the heart is unstable as a consequence of acute ischaemia or infarction, or in the presence of electrolyte abnormality (hypokalaemia or hypomagnesaemia).

Treat all broad-complex tachycardia as ventricular tachycardia unless there is good evidence that it is supraventricular in origin.

Patients with WPW syndrome have accessory pathways connecting atrial and ventricular myocardium. Some atrioventricular conduction occurs through these pathways as well as through the AV node. This results in widening of the QRS complexes by so-called delta waves. In the presence of such an accessory pathway that bypasses the AV node, AF may result in a ventricular rate that is so fast that cardiac output decreases dramatically. The ECG appearances are of a very rapid, irregular, broad complex tachycardia that usually shows variability in the width of QRS complexes. This rhythm may be misdiagnosed as irregular VT or possibly as VF. Overall the rhythm is more organised than ventricular fibrillation and lacks the random chaotic activity of variable amplitude.

The QT interval

When identifying and treating rhythm abnormalities it is important to recognise likely underlying causes that may influence choice of effective treatment. These may be identified from clinical assessment (e.g. myocardial infarction), laboratory tests (e.g. electrolyte abnormality) or from the ECG. Prolongation of the QT interval predisposes people to ventricular arrhythmia, in particular TDP VT and VF.

The QT interval is measured from the start of the QRS complex to the end of the T wave. It can be difficult to measure accurately, mainly because it may be difficult to identify the end of the T wave. This may be especially difficult when prominent U waves are present, merging with the end of the T wave. U waves can be a feature of some abnormalities (e.g. hypokalaemia) but may be present in some healthy people with normal hearts.

The length of the QT interval may also vary between different leads of the same ECG. This may partly reflect variation in amplitude and direction of the T wave, making it more difficult to measure in some leads than others. Variation in the QT interval (QT dispersion) has also been shown to be associated with an increased risk of death in patients with ischaemic heart disease, but this finding has not been developed into a useful measurement for use in clinical practice.

The QT interval varies with age, with gender and in particular with heart rate. The QT interval shortens as the heart rate increases. A correction can be made to allow for this, using the measured QT interval and heart rate to

calculate the corrected QT interval (QTc). The upper end of the normal range for QTc is 0.42 s. Many modern ECG machines measure the QT and other intervals and calculate the QTc automatically. These measurements are accurate only if the ECG recording is of good quality. Most ECG machines cannot distinguish between T waves and U waves. Always look at the recording and make sure that the quoted measurements are not obviously inaccurate. If in doubt seek expert help with interpretation.

Abnormality of the QT interval can be seen in various situations. A shortened QT interval may be seen with hypercalcaemia and digoxin treatment. Hypokalaemia, hypomagnesaemia, hypocalcaemia, hypothermia, myocarditis and in some instances myocardial ischaemia can all cause QT prolongation. There is also a long list of drugs that may prolong the QT interval, including class I and class III anti-arrhythmic drugs.

There are several genetic abnormalities in which the QT interval is abnormal or there is abnormality of ventricular repolarisation (principally the long QT, short QT and Brugada syndromes). The abnormality of repolarisation places them at risk of ventricular arrhythmia and sudden death. These people require expert assessment to identify whether treatment is needed to reduce this risk. For some the only effective treatment is an implantable cardioverter-defibrillator to treat VF or VT immediately, if it occurs. It is especially important that patients with long QT syndromes are not given any drug that may cause further QT prolongation.

Key learning points

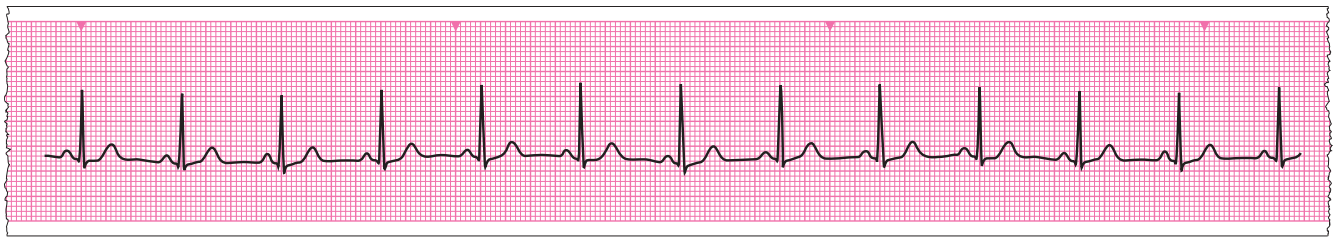
- A systematic approach to ECG rhythm analysis enables accurate assessment of any rhythm abnormality sufficiently to enable safe, effective treatment.
- Recordings of any rhythm abnormality and of the ECG in sinus rhythm provide valuable diagnostic information and help the correct choice of longer-term treatment.
- Accurate monitoring of the cardiac rhythm is essential for any patient at high risk of developing life-threatening arrhythmia.
- Accurate monitoring of the cardiac rhythm is essential in the management of cardiac arrest.

Further reading

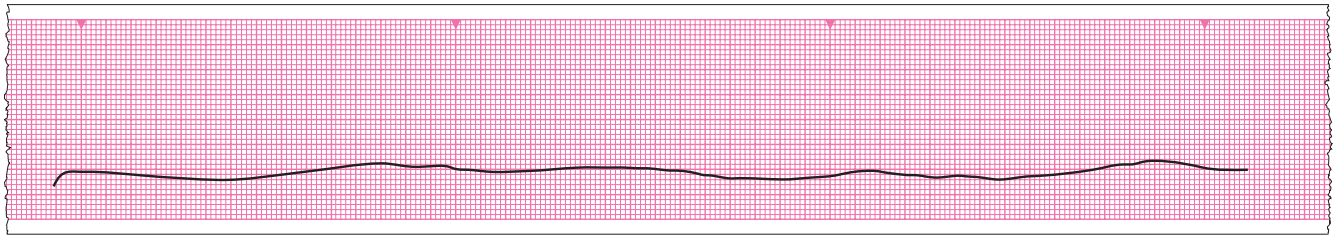
Blomstrom-Lundqvist C, Scheinmann M M (Co-Chairs). American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines. ACC/AHA/ESC Guidelines for the Management of Patients With Supraventricular Arrhythmias. European Heart Journal 2003;24:1857-1897. www.escardio.org

Fuster V, Ryden L E, Cannom DS, et al. ACC/AHA/ESC 2006 Guidelines for the Management of Patients with Atrial Fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Revise the 2001 Guidelines for the Management of Patients With Atrial Fibrillation): developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. Circulation 2006;114:e257-354. www.escardio.org

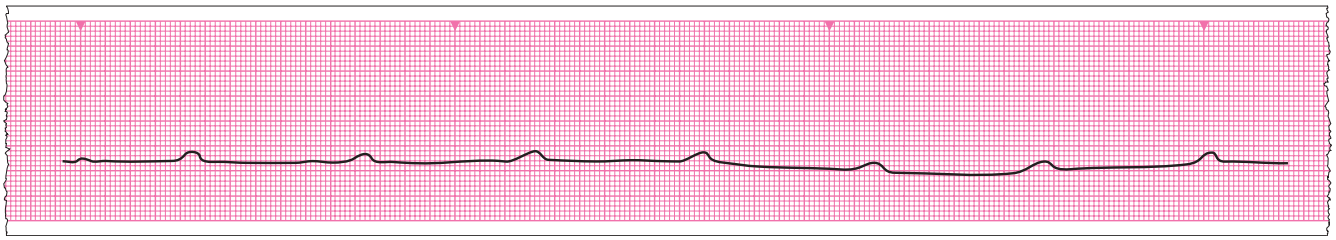
Zipes DP, Camm AJ, Borggrefe M, et al. ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines (Writing Committee to Develop Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death) J Am Coll Cardiol 2006;48:e247-e346. www.escardio.org



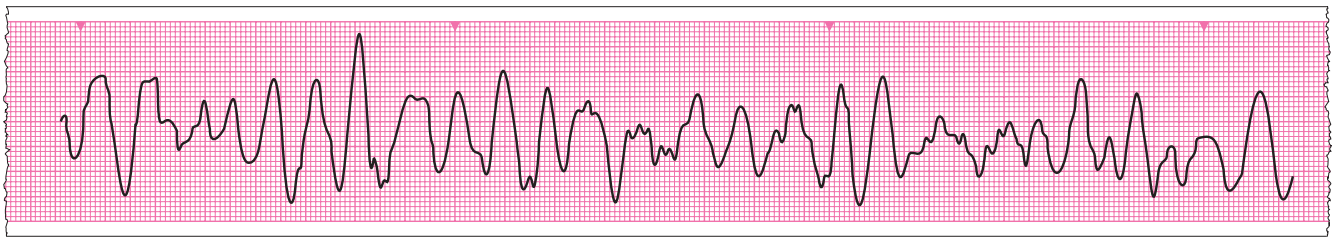
Rhythm Strip 1. Normal sinus rhythm



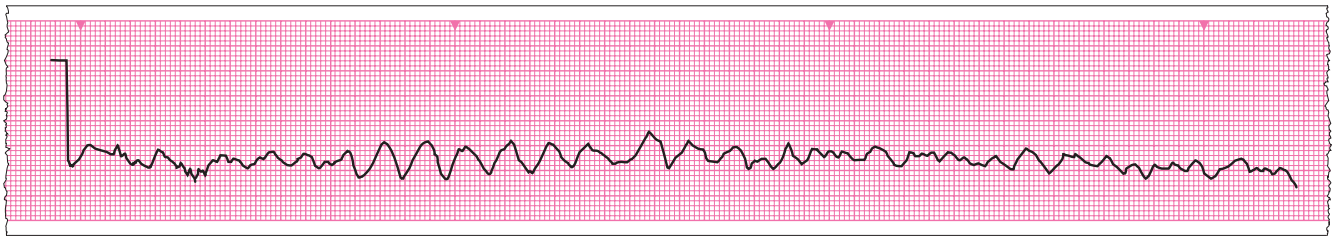
Rhythm Strip 2. Asystole



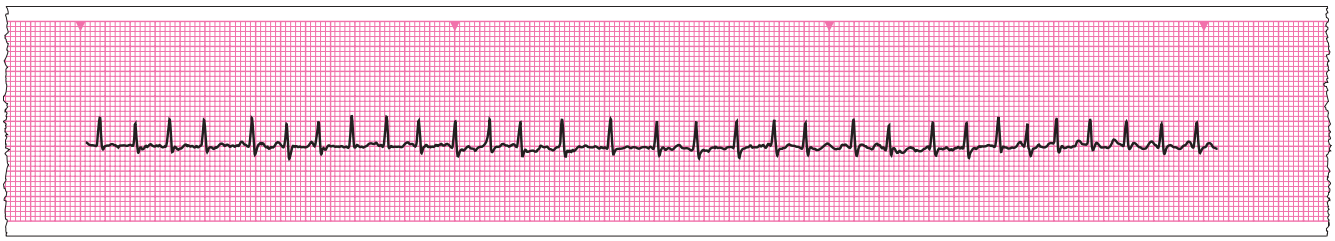
Rhythm Strip 3. P-wave asystole



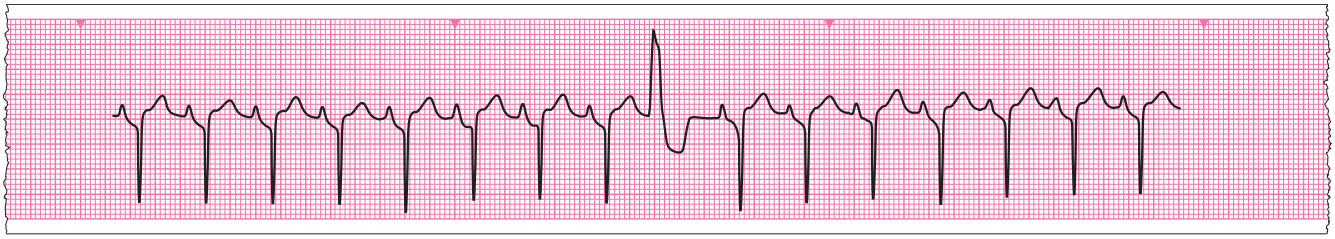
Rhythm Strip 4. Coarse ventricular fibrillation



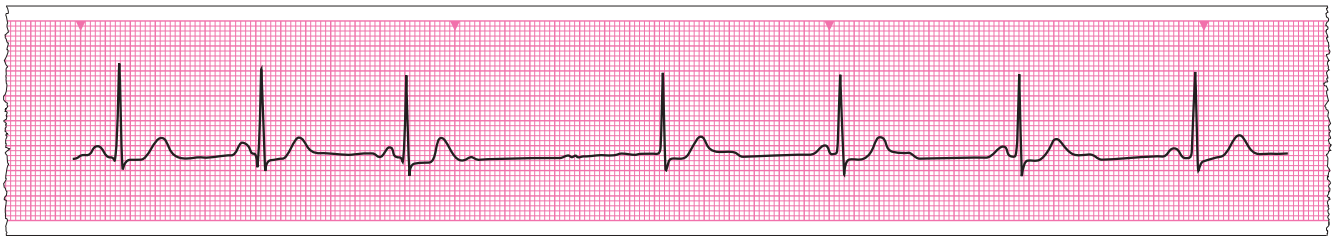
Rhythm Strip 5. Fine ventricular fibrillation



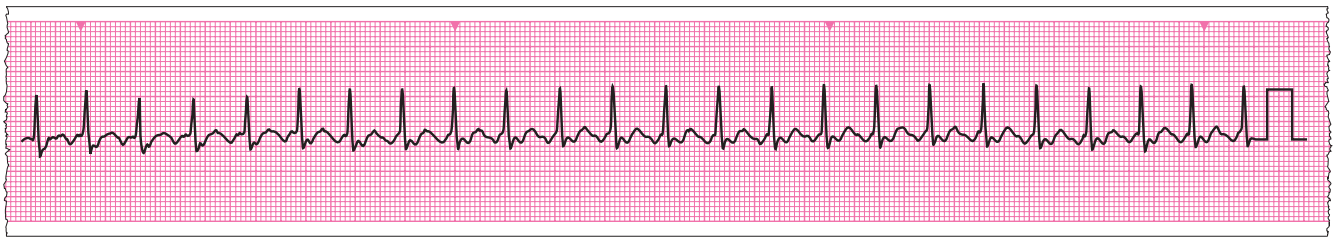
Rhythm Strip 6. Atrial fibrillation



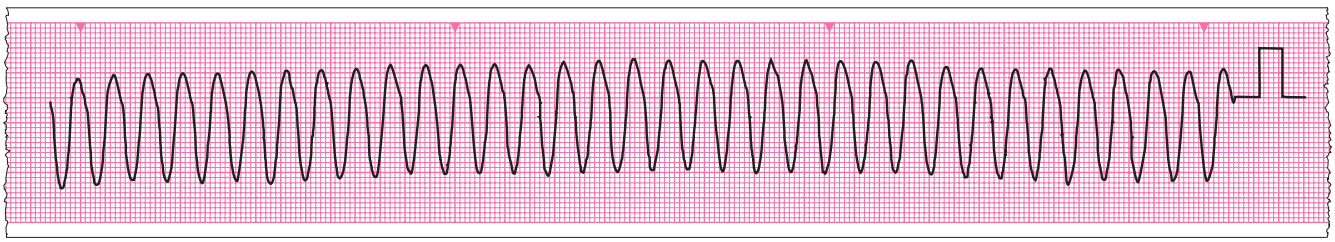
Rhythm Strip 7. Premature ventricular beat



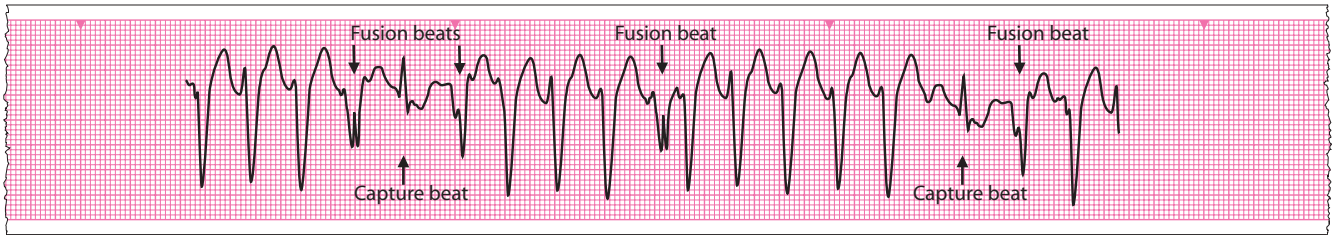
Rhythm Strip 8. Junctional escape beat



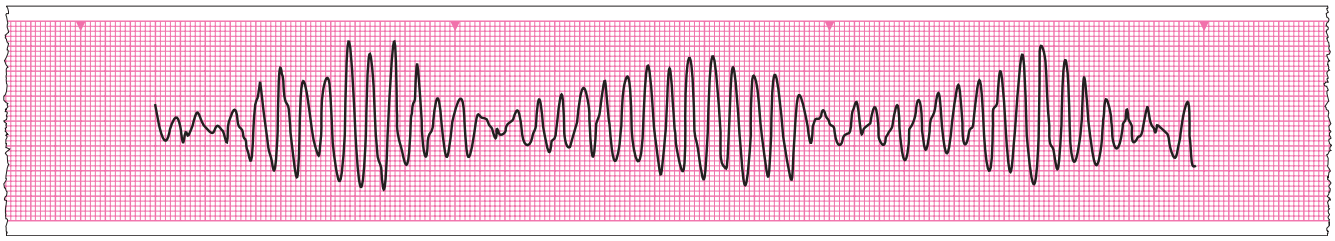
Rhythm Strip 9. Atrial flutter with 2:1 atrioventricular block



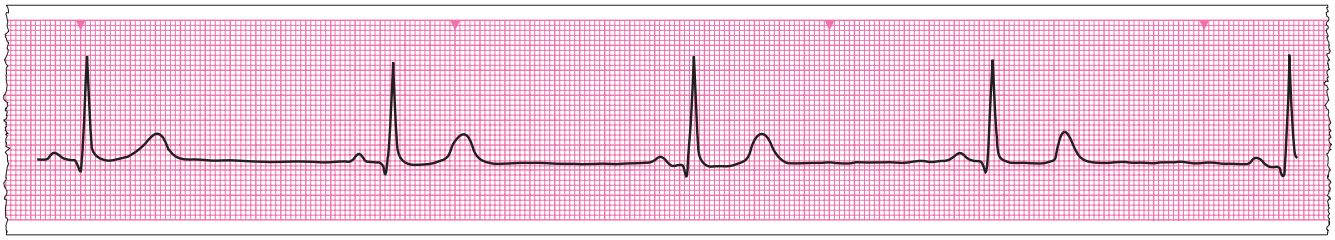
Rhythm Strip 10. Monomorphic ventricular tachycardia



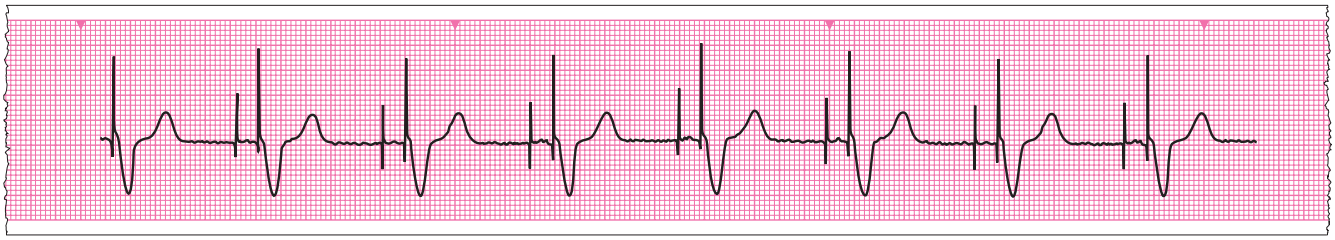
Rhythm Strip 11. Ventricular tachycardia with capture and fusion beats



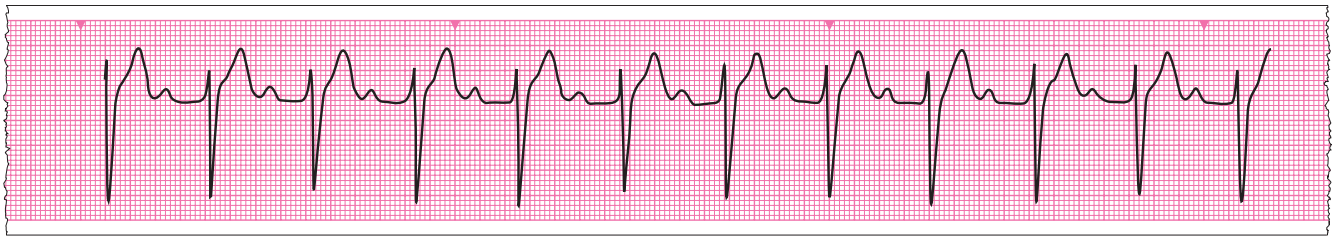
Rhythm Strip 12. Polymorhpic ventricular tachycardia - Torsade de Pointe



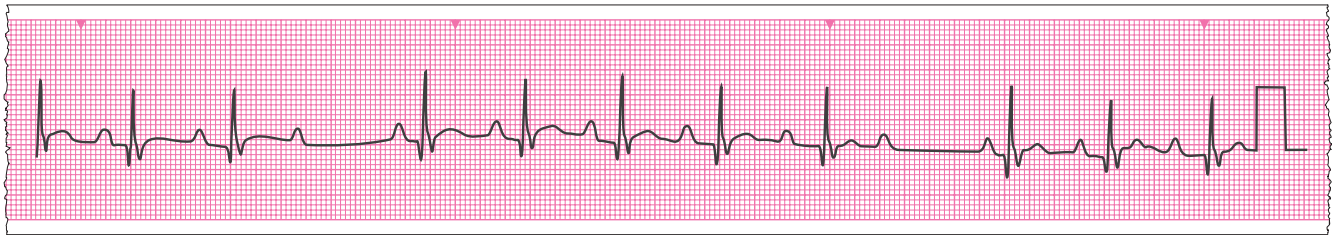
Rhythm Strip 13. Sinus bradycardia



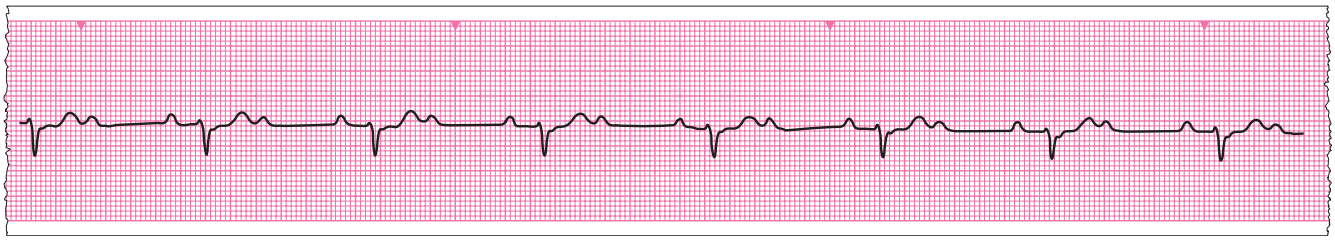
Rhythm Strip 14. Paced rhythm



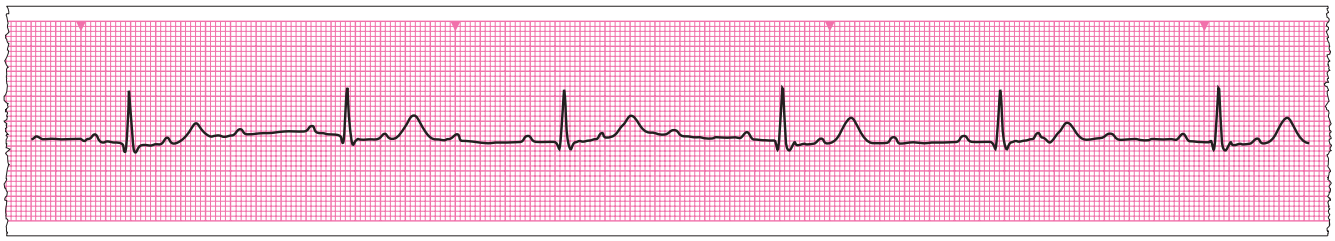
Rhythm Strip 15. First degree atrioventricular block



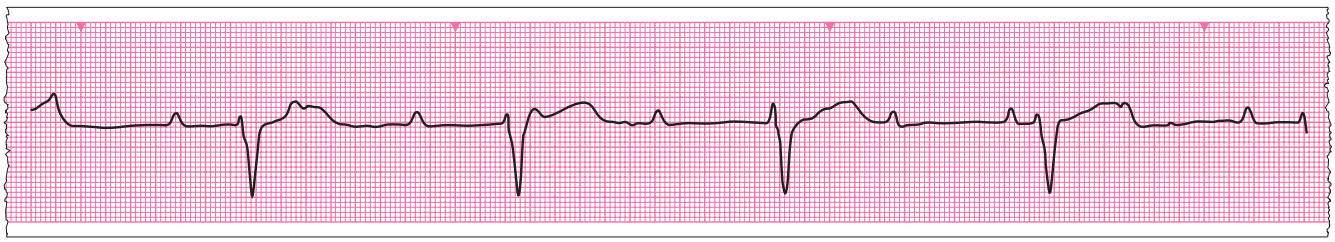
Rhythm Strip 16. Mobitz type I or Wenckebach block



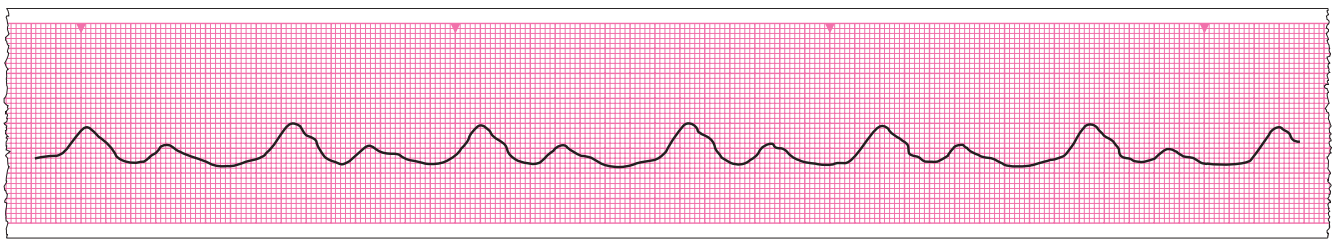
Rhythm Strip 17. Mobitz type II second degree atrioventricular block (2:1)



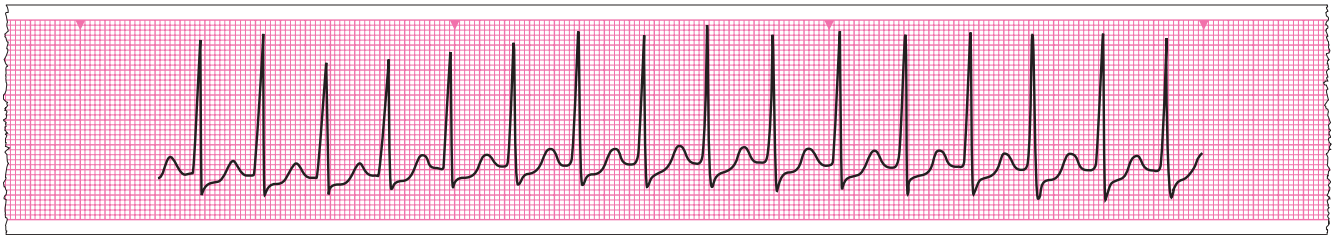
Rhythm Strip 18. Mobitz type II second degree atrioventricular block (3:1)



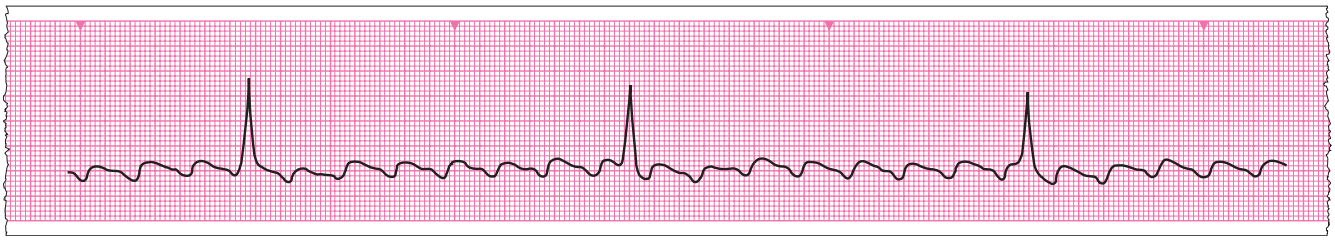
Rhythm Strip 19. Third degree (complete) atrioventricular block



Rhythm Strip 20. Agonal rhythm



Rhythm Strip 21. Supraventricular tachycardia



Rhythm Strip 22. Atrial flutter with a high degree of atrioventricular block