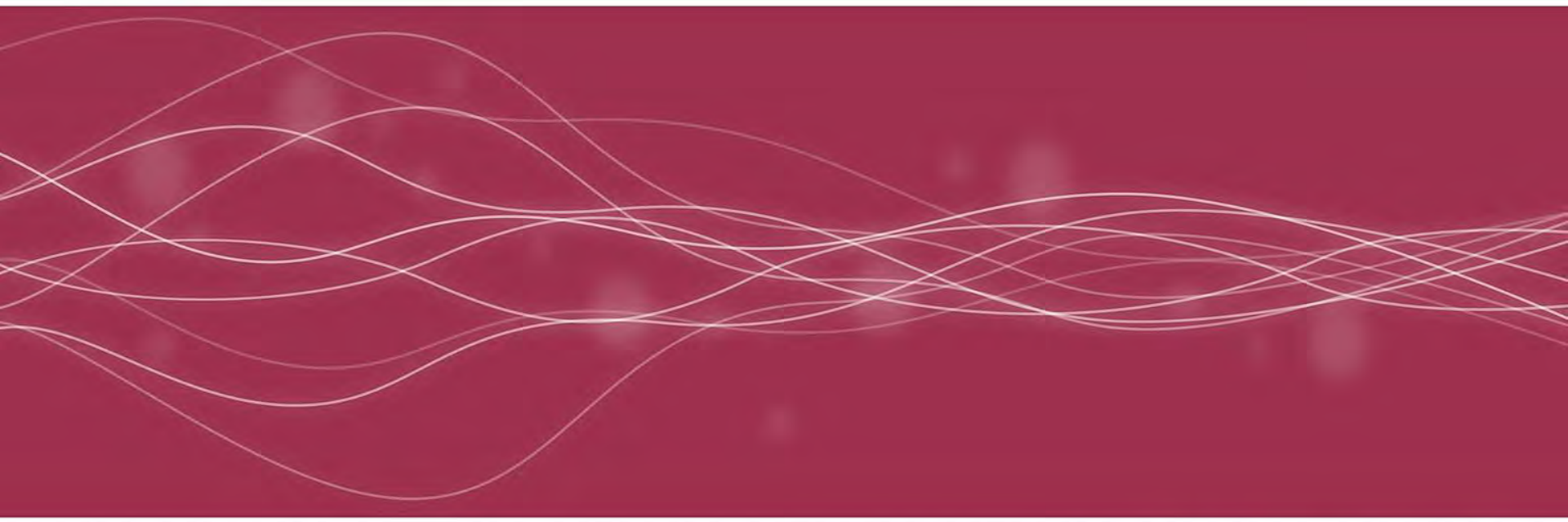
The ECG Atlas of Cardiac Rhythms



Rob Scott Millar

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About the book

This book contains high-quality 12-lead ECGs of all the common rhythms, and many of the less common, but important cardiac rhythm disturbances. The ECGs are reproduced full size, so that you can examine and interpret them as you would in a clinical situation. My analysis will be on the following page, so you can check your interpretation. Brief clinical information will be provided.

I have collected the ECGs over more than 20 years from my own cases and those of my colleagues at the Cardiac Clinic, Groote Schuur Hospital, Cape Town. Most are printouts from the electronic copies stored in the Marquette© ECG management system, installed in 1989, recently updated. Most of the more difficult diagnoses have been validated by intracardiac recordings during invasive electrophysiological studies (EPS).

This is not a textbook. Its core value depends on the variety of real ECG traces and their interpretation which will, hopefully, assist you in interpreting similar rhythms that you encounter in your own practice. I have, however, included some introductory chapters on ECG interpretation and rhythm diagnosis, in particular. I have included some extra tracings with a quiz to help you apply your skills. You should study and interpret the ECG on the left-hand page, before moving to the explanation on the facing page.

The ECG as we know it has been around for more than a century. A tracing published by Einthoven in 1906, showing atrial fibrillation, is readily interpretable in 2014. Far from being outdated, the value of the ECG continues to grow as more information from clinical sources, pathology, electrophysiology and molecular biology is used to refine ECG analysis.

Clinical cardiac electrophysiology, in particular, has revolutionised our knowledge and interpretation of arrhythmias. This has led to re-interpretation of the ECG and made possible the confident diagnosis of the mechanisms of many rhythms from the 12-lead surface ECG that were previously poorly understood. Successful ablation of the substrate of some arrhythmias has contributed enormously to this understanding. For example, the pattern of delta waves in one of the ECGs published by Rosenbaum in his 1945 article,[1] in which the 'type A and B' classification was proposed, would now be interpreted as being typical of a posterior epicardial accessory pathway, which can be cured by radiofrequency ablation applied via a catheter from within the coronary sinus.

The ECG is widely available, relatively cheap, non-invasive, and can be recorded repeatedly over time. I can think of no other investigation capable of giving as much valuable information in as wide a variety of circumstances or diversity of patients and conditions. To extract this information, you need a basic knowledge of ECG interpretation, intelligent application and practice. Experience helps.

Reference

1. Rosenbaum FF, Hecht HH, Wilson FN, Johnston FD. The potential variations of the thorax and the esophagus in anomalous atrioventricular excitation (Wolff–Parkinson–White syndrome). *Am Heart J* 1945; **29**: 281–326.

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1. Guide to the ECG analysis of rhythm

It is tempting to jump to a quick conclusion when assessing the rhythm on the ECG. It will usually be apparent at first glance that the rhythm is a problem when the overall rate is very slow or very fast. However, the rhythm may be abnormal even if the rate is normal. Avoid snap judgements and assess the tracing systematically:

- Make careful observations of the rate, regularity, QRS width, P waves and P–QRS relationship (see box).
- Consider the possible mechanisms responsible for any abnormalities detected.
- Come to a considered diagnosis of the rhythm and the most likely underlying pathology.

While many will recommend that you start with a search for P waves, these may be hidden and difficult to find. They may also lead to false conclusions as to the origin of the rhythm. The QRS complexes are the most useful place to start to determine whether the rhythm originates from above the bifurcation of the common His bundle and conducts to the ventricles via all or part of the His-Purkinje system (supraventricular), or originates in the ventricles.

The QRS complexes may also alert one to the presence of an accessory (extranodal) conduction pathway. Examination of the QRS complexes during supraventricular rhythm may reveal clues to structural heart disease, such as old myocardial infarction or ventricular hypertrophy.

Be systematic and aim to work out the mechanism of the rhythm before speculation as to the underlying pathology. I suggest that you follow the sequence as set out in the rhythm analysis tick sheet (page 10).

Primary observations

1. Regularity:
   - Regular
   - Irregular
2. Ventricular rate:
   - Normal (50–100 bpm)
   - Slow (< 50 bpm)
   - Fast (> 100 bpm)
3. QRS width
   - Normal (≤ 100 ms)
   - Intermediate (> 100 ms, < 120 ms)
   - Wide (≥ 120 ms)
     - Left bundle branch block
     - Right bundle branch block
     - Neither
4. P waves
   - Morphology (? upright in II, negative aVR)
   - Rate
   - Relationship to QRS complexes
5. PR interval

Secondary observations

6. QRS abnormalities
7. ST segment abnormalities
8. T-wave abnormalities
9. QT interval

Primary observations

Rhythm

Is the rhythm regular or irregular? (Fig. 1.01). If it is irregular, is there a pattern (group beating), intermittent pauses, or random irregularity (as in atrial fibrillation)? Are there extra (premature) complexes?

Ventricular rate

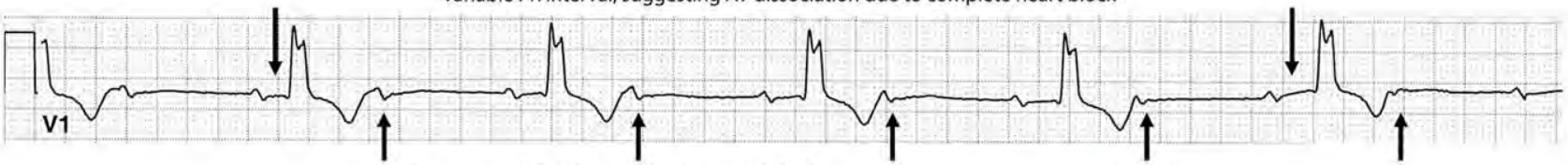
If the rhythm is irregular, do not attempt to use any of the methods to calculate the ventricular rate, which use one or two R–R intervals. Calculate the average by counting the number of beats over as long a period as is available. A modern 12-lead ECG recording on A4 or similar paper is 250 mm long – 10 seconds at the standard recording rate of 25 mm/s. Count the number of consecutive complexes on the page and multiply by six to get the ventricular rate per minute.

- **Normal**
  This is regarded as 50–100 beats per minute (bpm). While 60 bpm is the usual cut-off rate for sinus bradycardia, rates between 50 and 60 bpm are extremely common in normal individuals and are usually of no clinical significance.

- **Slow (bradycardia)**
  This is less than 50 bpm. At slow rates, pay particular attention to the atrial rate and the relationship of the P waves to the QRS complexes. If the rhythm appears to be sinus bradycardia, look carefully for hidden P waves (e.g. at the end of the T wave) that

Fig. 1.02 – Bradycardia

Variable PR interval, suggesting AV dissociation due to complete heart block



P waves partially obscured by the end of the T waves

Fig. 1.03 – ECG diagnosis of tachycardia

Regular

Narrow QRS

- Sinus tachycardia
- **Atrial flutter**
- **AV junctional re-entry tachycardia (AVNRT, AVRT)**
- Atrial tachycardia
- Junctional ectopic tachycardia

Irregular

Narrow QRS

- **Atrial fibrillation**
- **Atrial flutter/tachy + variable AV block**
- Multifocal atrial tachycardia

Wide QRS

- **Ventricular tachycardia**
- Supraventricular tachycardia with bundle branch block
- Antidromic AVRT
- Pre-excited SVT
- Paced rhythm

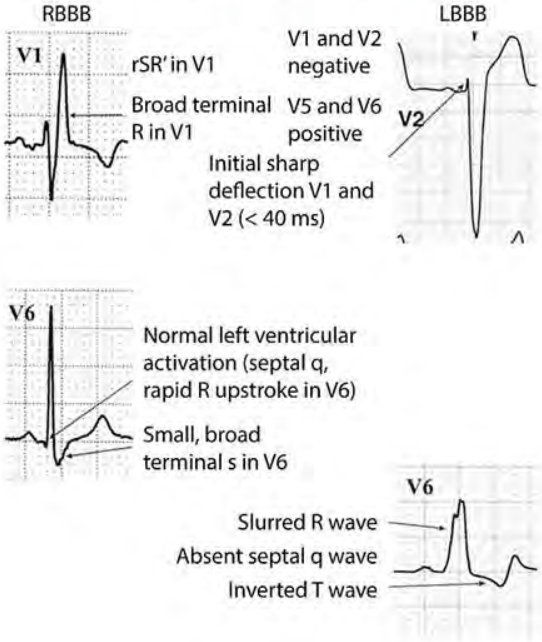
Regular

Wide QRS

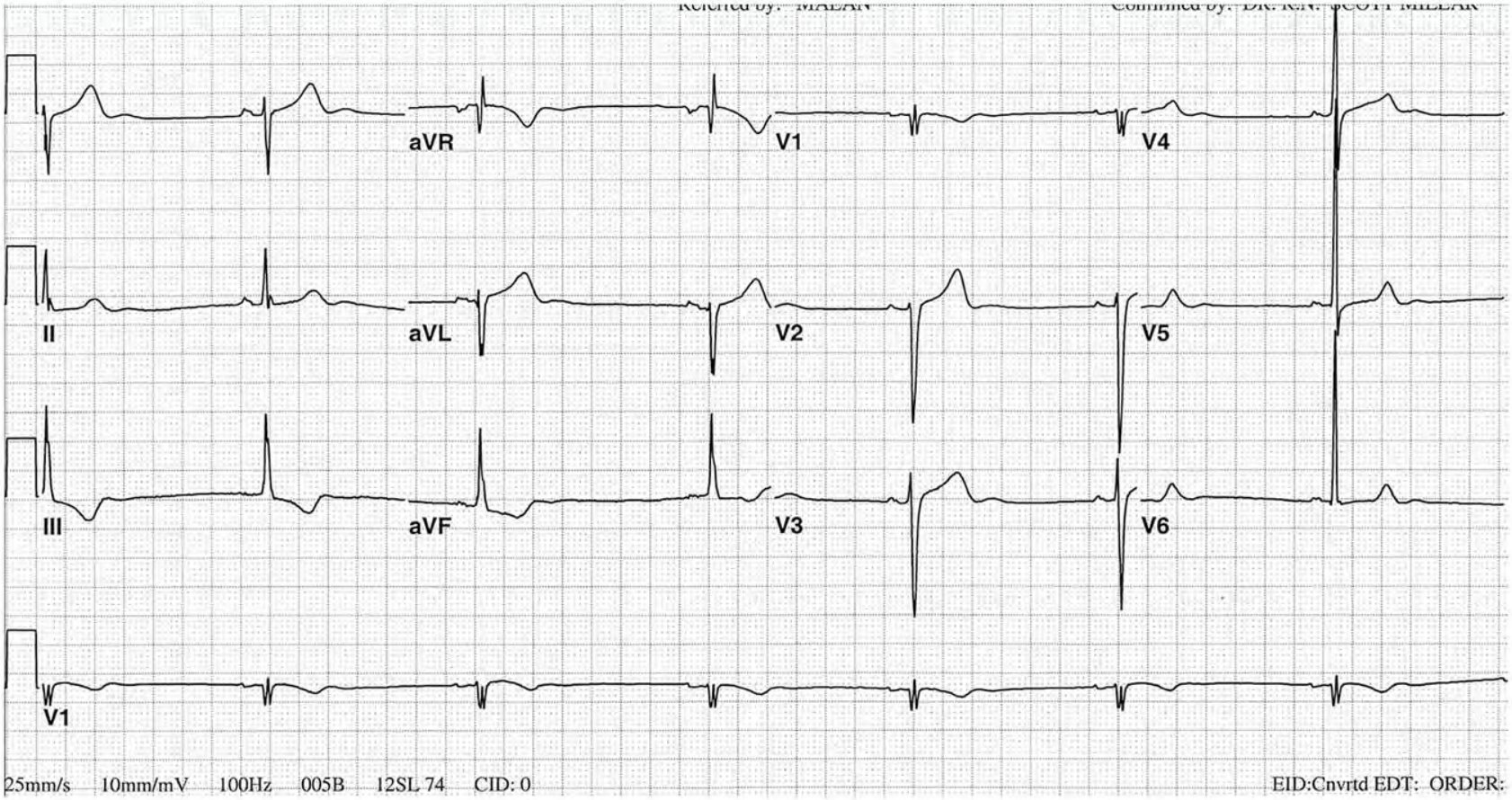
- **AF with bundle branch block**
- **Atrial flutter, variable AV block + bundle branch block**
- Pre-excited AF
- Polymorphic VT

Irregular

Fig. 1.04 – Typical patterns of right and left bundle branch block



ECG 1.02


aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
V1
25mm/s 10mm/mV 100Hz 005B 12SL 74 CID: 0
EID:Cnvrtd EDT: ORDER:

ECG 1.02: Left posterior fascicular block

Overview: Sinus bradycardia with right-axis deviation
Rhythm: Regular
Rate: 42 bpm (7 × 6)
P waves: P axis +30°
PR interval: 150 ms
QRS width: 90 ms
QRS axis: +120°

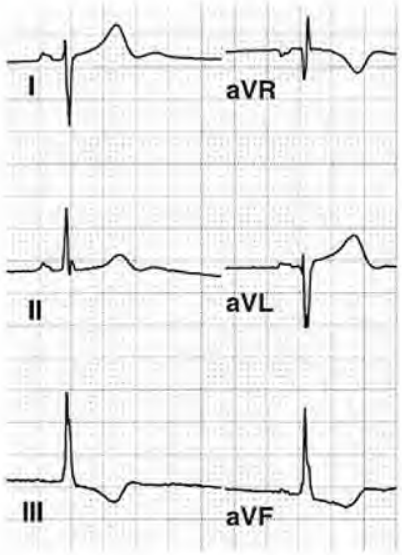
QRS morphology: The left ventricular voltage is increased; the QRS is splintered in V1, but there is a small initial r wave.

Comment: Right-axis deviation, but no other definite evidence for right ventricular hypertrophy. There is probably LVH, and there is non-specific inferior T-wave inversion.

Differential diagnosis: Right-axis deviation may be due to RVH, left posterior fascicular block (LPFB) or lateral myocardial infarction. Although there is a small initial q wave in aVL, this is not sufficient to account for the right axis. Unlike anterior fascicular block, LPFB is not a purely electrocardiographic diagnosis. RVH must be excluded on clinical or other grounds. In this case, however, another ECG taken two days later (B) showed a normal QRS axis, confirming transient LPFB.

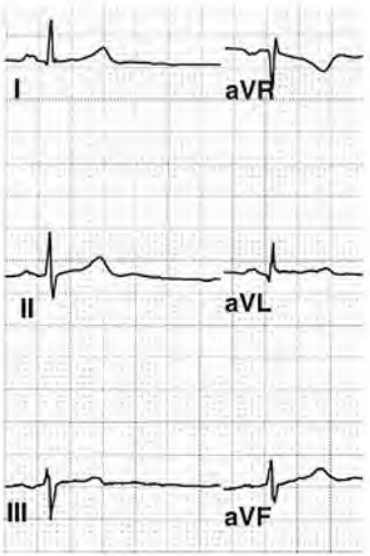
Clinical information: A 45-year-old man with hypertension and ischaemic heart disease. There was no clinical or other evidence of right ventricular hypertrophy.

A



Initial ECG, showing a QRS axis of +120°.

B



ECG taken two days later, showing a QRS axis of +15°.

Final diagnosis: Sinus bradycardia with transient left posterior fascicular block. Isolated LPFB is very uncommon.

2. Bradycardias and conduction disorders

By definition, a ventricular rate of less than 60 bpm is a bradycardia but rates of 50–60 bpm are common in normal people. Mechanisms of bradycardia include disorders of impulse formation (sinus node function) and atrio-ventricular (AV) conduction, and combinations of the two.

Clinical features

Symptoms

A patient with bradycardia may have no symptoms but the following are common:

- fatigue
- dizziness
- syncope
- heart failure.

Signs

Blood pressure: bradycardia does not cause hypotension in the absence of myocardial depression (e.g. acute myocardial infarction) or peripheral vasodilatation (e.g. vasovagal syncope). On the contrary, complete heart block in an otherwise healthy elderly person (most often the case) results in a high pulse pressure. This is caused by ejection of an increased stroke volume (compensating for the slow rate via the Starling principle) into a non-compliant arterial tree (resulting in a high systolic pressure), followed by prolonged runoff during the long diastole. The non-compliant arteries of an elderly subject exacerbate the low diastolic pressure because of lack of elastic recoil. In the presence of a ventricular escape rhythm of 36 bpm, it is common to find a blood pressure of 200/70 mmHg or more. If hypotension accompanies bradycardia, suspect one of the causes of 'sick bradycardia' (see below).

AV dissociation: look for signs of AV dissociation (irregular cannon waves in the jugular pulse, varying pulse volume, and variation in the first heart sound (due to changing stroke volume as the timing of atrial systole varies). If present, they suggest complete heart block, but be aware that AV dissociation may occur with marked sinus bradycardia (isorhythmic AV dissociation).

Age

Normal resting heart rate is a function of age and prevailing autonomic tone. Children have faster resting rates than adults. Intrinsic heart rate (measured after complete autonomic blockade by means of atropine + propranolol) decreases with advancing age. Fit young people with high vagal tone have slower resting heart rate and up to 7% have episodes of Wenckebach AV block while asleep.[1] Extremely fit endurance athletes may even develop Wenckebach AV block while awake.

While heart block may complicate acute myocardial infarction, particularly of the inferior wall, and other acute myocardial conditions such as myocarditis, chronic AV block affects mainly older people. The pathology is idiopathic degeneration of the conducting tissue, mainly His-Purkinje fibres, in the majority of cases, rather than ischaemia. As a result, myocardial function is usually normal for age and the prognosis is excellent once a permanent pacemaker has been implanted.

Congenital complete heart block presents in utero or at birth and carries a high mortality rate because of commonly associated structural abnormalities. Those who survive infancy may have few or no symptoms until later in life.

'Sick bradycardias'

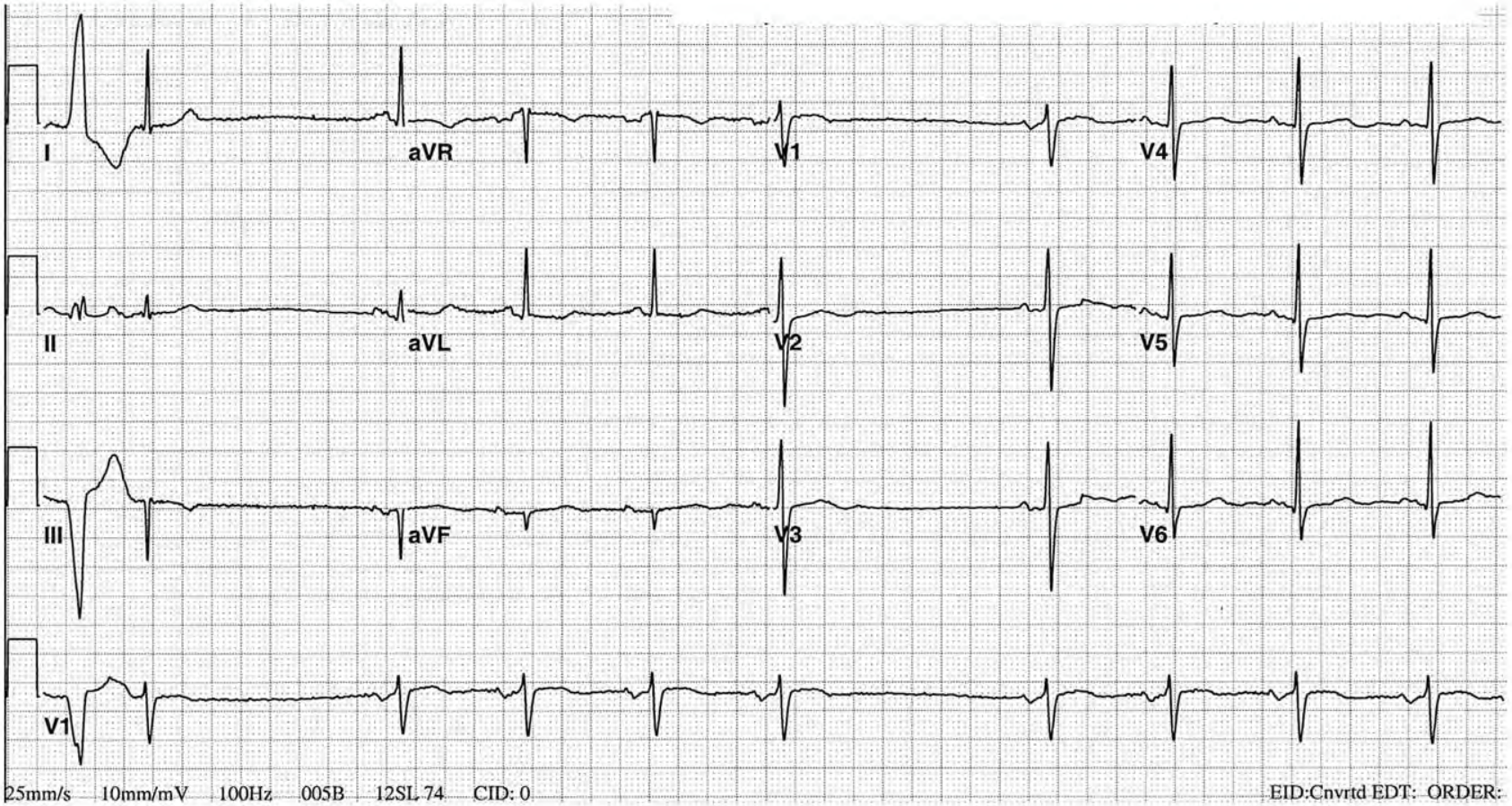
This is my term for patients who present to the emergency unit with bradycardia (often sinus bradycardia/sinus arrest, rather than AV block) and hypotension. They may have poor perfusion and be obtunded. The bradycardia is seldom, if ever, the primary cause of the problem but is a consequence of the underlying condition. While the ECG is not the primary means of diagnosis in such cases, it may show characteristic changes in hyperkalaemia (Fig. 2.01) and hypothermia (Fig. 2.02). It may, however, be misleading in cases of subarachnoid haemorrhage or other cerebral insult (Fig. 2.03), by suggesting myocardial ischaemia or infarction.

Bradycardia/tachycardia syndromes

Lack of regular sino-atrial discharge at a normal rate promotes atrial anarchy and allows competing rhythms, particularly atrial fibrillation, to take over. Atrial fibrillation may also reflect underlying atrial disease in patients with sick sinus syndrome. Tachycardia may follow an episode of sinus bradycardia or arrest. However, sinus arrest commonly follows termination of an atrial tachyarrhythmia in a patient with sinus node disease, due to overdrive suppression of sinus node discharge.

While this is a normal phenomenon, a normal sinus

ECG 2.02



This 71-year-old woman complained of dizzy spells.

ECG 2.02: Mobitz II sino-atrial exit block

Overview: Sinus rhythm with pauses
Rhythm: Irregular – group beating
Rate: 60 bpm (10 × 6)
QRS width: 100 ms
P waves: P axis +30° – probable left atrial enlargement.
PR interval: 170 ms
QRS axis: –15°

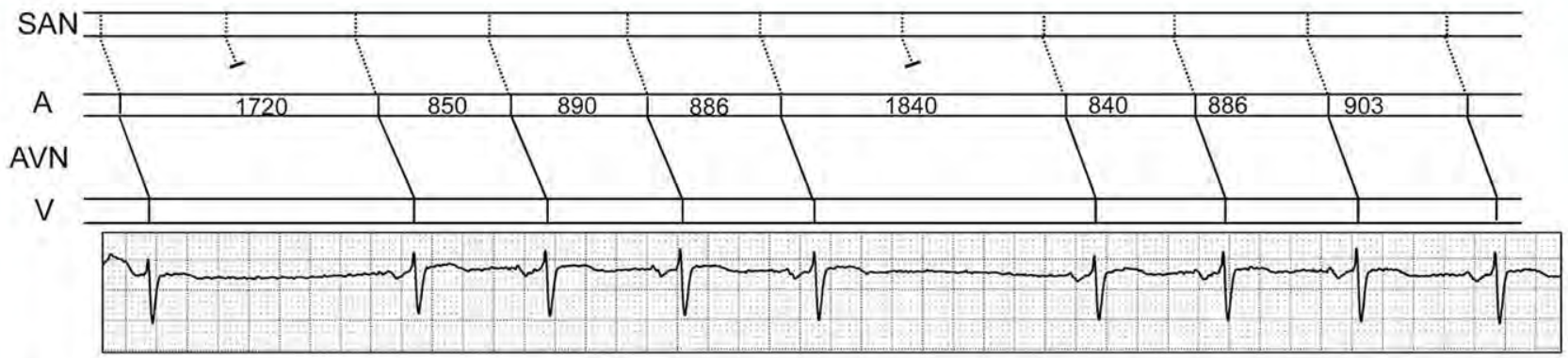
QRS morphology: Small q wave in II, larger in III, raises the possibility of old inferior MI, but there is a small r wave in aVF.

Comment: Apart from a PVC, the rhythm is sinus (normal P axis, 85 bpm) with two pauses. The second pause follows three sinus beats, and is slightly longer than twice the preceding P–P interval. This could be due to either transient sinus arrest or intermittent 2:1 sino-atrial exit block. The first pause is the same duration as the sum of the succeeding P–P intervals, making SA block more likely. There are slight variations in the P–P interval (sinus arrhythmia). However, the P–P interval does not shorten progressively before the dropped P wave, as would be the case with Wenckebach SA block.

Differential diagnosis: Sinus arrest or Mobitz II SA block.

Clinical information: No reversible cause for sinus node dysfunction was found. In view of her symptoms, a permanent pacemaker was implanted.

Final diagnosis: Mobitz II sino-atrial exit block.



Ladder diagram to illustrate intermittent Mobitz II sino-atrial block. SAN = sino-atrial node; A = atrial depolarisation; P–P intervals in milliseconds; AVN = AV node; V = ventricular depolarisation. The sinus node discharge is not visible on the ECG.

3. Abnormal rhythms with a normal rate

While a normal heart rate usually signifies sinus rhythm, many abnormal rhythms may occur at rates within the normal range (50–100 bpm). Many are atrial tachyarrhythmias with sufficient AV block to reduce the ventricular rate to within the normal range. Others are due to premature complexes ('ectopics'), or to acceleration of lower pacemakers. Artificial paced rhythms also fall into this category.

Clinical features

There are no clinical features specific to this group of arrhythmias. The normal ventricular rate usually prevents severe symptoms, but the patient may complain of palpitations related to an irregular rhythm. Some people are very intolerant of ectopics, particularly premature ventricular complexes. This is most often due to heightened awareness of the forceful beat which follows the post-ectopic pause. This forceful contraction is due to the increased ventricular filling allowed by the longer diastole (Starling principle), and the phenomenon of post-ectopic potentiation, probably related to increased calcium ingress.

Sometimes, ventricular bigeminy causes an effective bradycardia because the premature beat may occur too early for sufficient ventricular filling to occur, resulting in markedly reduced stroke volume and even failure to open the aortic valve. This may cause fatigue and dizziness.

Pacemaker syndrome

Some patients with artificial ventricular pacemakers develop a constellation of symptoms related to the loss of atrio-ventricular synchrony. These include postural dizziness, even syncope, reduced effort tolerance and a sensation of pulsation in the neck. The latter is due to cannon waves in the jugular venous pulse because of either AV dissociation or 1:1 ventriculo-atrial conduction. Symptoms usually subside if sinus rhythm resumes or AV synchrony is restored by upgrading the system to AV sequential pacing.

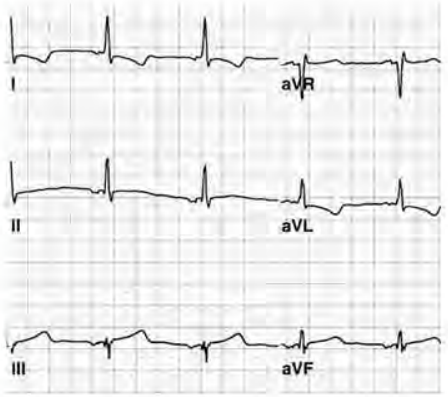
Regular narrow QRS rhythms

Atrial rhythm with abnormal P waves

Before accepting an atrial rhythm with P waves preceding each QRS complex as being of sinus origin, check the P-wave axis. The sinus node is situated around the junction of the superior vena cava with the right atrium. Atrial depolarisation starts from there and spreads downward and to the left, towards the AV junction, giving rise to P waves with a mean frontal plane axis of between 30° and 70°.

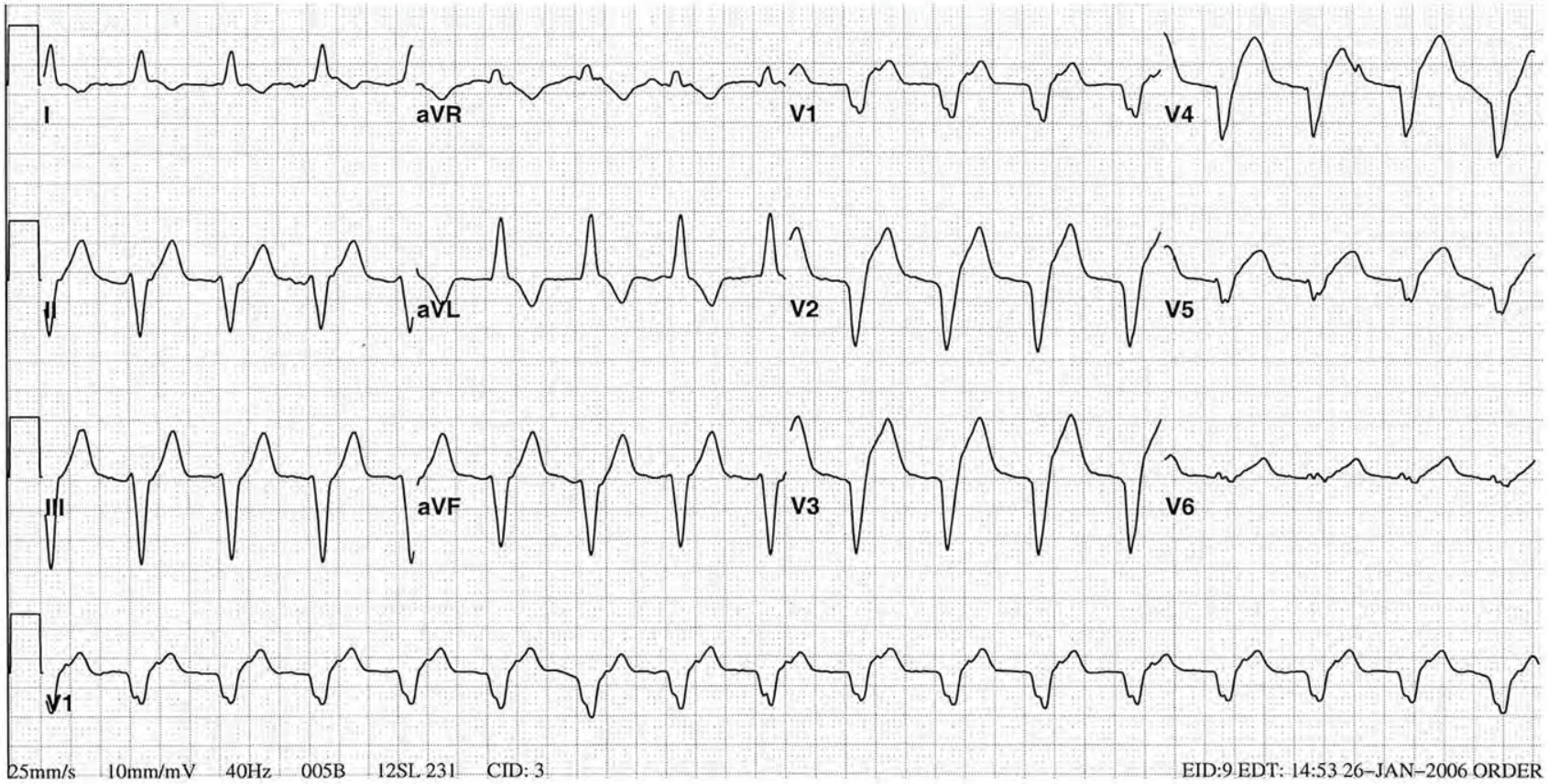
As a result, sinus P waves are always positive in standard lead II. If this is not the case, the atrial depolarisation must arise from a focus elsewhere in the atria (Fig. 3.01).

Fig. 3.01 – Atrial rhythm with abnormal P waves



Regular atrial rhythm at 70 bpm. The P waves are negative in standard lead II and therefore cannot arise from the sinus node. The P-wave axis is about –100°, indicating an origin low in the atria. The P–R interval is relatively short (130 ms), due to the proximity of the atrial pacemaker to the AV node.

ECG 3.03



This 67-year-old man presented with crushing central chest pain of about four hours' duration.

4. Narrow QRS tachycardias

If the QRS complex is 100 ms or less, a supraventricular origin is almost certain. Supraventricular rhythms arise above the bifurcation of the common His bundle and are therefore able to use the rapid transit properties of the His-Purkinje network to distribute the impulse rapidly to the endocardial surface of both ventricles.

Anatomically, narrow QRS tachycardias can arise in either atrium, in or around the AV junction, or in the common His bundle. It is, however, misleading to think of the tachycardia arising at a specific point. While some supraventricular tachycardias (SVTs) do have a focal origin (some atrial tachycardias and most junctional and His bundle rhythms) the majority are due to re-entry, with circuits involving small or large amounts of tissue.

Irregular versus regular narrow QRS tachycardia

The vast majority of sustained irregular narrow QRS tachycardias are due to atrial fibrillation. Other mechanisms include:

- Atrial flutter with varying block
- Multifocal atrial tachycardia.

The differential diagnosis of regular SVTs is wider:

- Atrial flutter
- AV junctional re-entry tachycardias
  - AV nodal re-entry tachycardia (AVNRT)
  - AV re-entry tachycardia (AVRT), using an accessory AV connection (WPW syndrome)
- Atrial tachycardias
- Junctional and His bundle tachycardias.

Clinical pointers

Structural heart disease

A history or signs of structural heart disease is common in patients with atrial fibrillation or flutter, whereas most AV junctional re-entry tachycardias occur in younger people with structurally normal hearts.

Patients, particularly young adults, with congenital heart disease are prone to atrial tachycardias. These may be classical (counter-clockwise) atrial flutter or re-entry around a surgical scar in the atrium.

The commonest underlying clinical condition in patients presenting in atrial fibrillation is hypertension. Atrial fibrillation commonly complicates heart failure from any cause and other conditions, which result in atrial stretch and fibrosis, notably rheumatic mitral stenosis.

Age

Regular narrow QRS tachycardias may occur at any age, but mechanisms differ. In infants up to about 12 months, around 90% are due to accessory pathways (AVRT). Atrial flutter (often starting in utero) and atrial tachycardias also occur. Junctional ectopic tachycardias may be especially troublesome in infants and young children in the early post-operative period after surgery for congenital heart defects. Beware of borderline 'narrow QRS' (100–120 ms) tachycardias in young children – ventricular tachycardias do occur in this age group and the QRS is much narrower than in adults (Fig. 4.01).

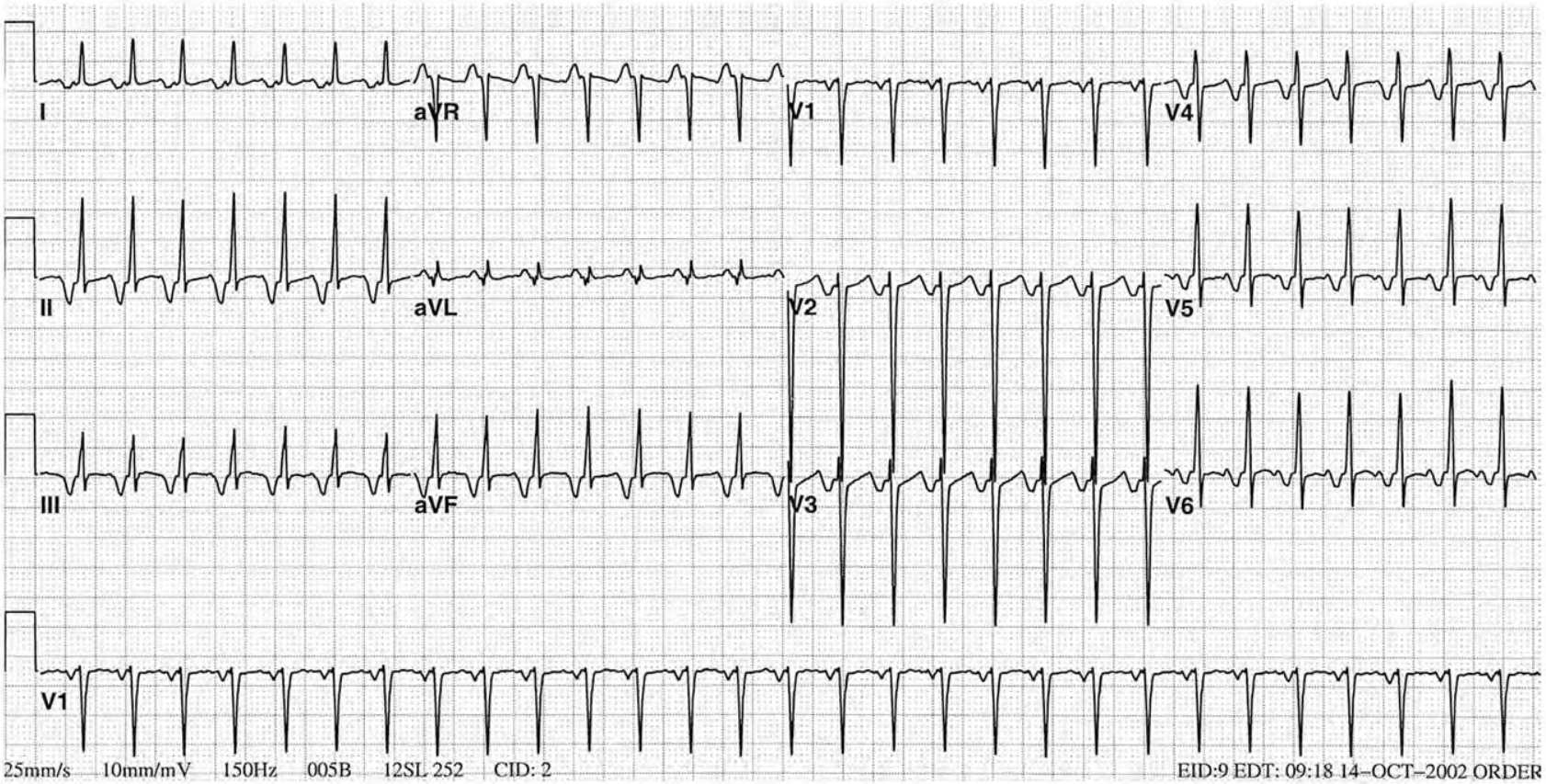
In the age group one to 10 years, atrial flutter is rare, as is AVNRT. AVRT remains the commonest cause, but ectopic atrial tachycardias are much more frequent in children than adults. They tend to be incessant and present insidiously as heart failure due to tachycardia-induced cardiomyopathy, rather than with symptoms of palpitations. Atrial tachycardia is often misdiagnosed as 'sinus tachycardia' secondary to the cardiomyopathy. It is rare for persistent sinus tachycardia to exceed 140 bpm, even in young children with cardiomyopathy.

In older children and young adults, AVNRT becomes increasingly common. Accessory pathways (WPW) remain important. Atrial fibrillation is rare in younger children, but may complicate WPW syndrome (irregular wide, bizarre QRS complex) and may occur in rheumatic heart disease. Idiopathic fascicular (ventricular) tachycardia in this age group often has a relatively narrow QRS (110–120 ms). It is often misdiagnosed as supraventricular tachycardia (SVT), particularly as it may terminate with intravenous adenosine or verapamil.

In young to middle-aged adults, any SVT may occur, including atrial fibrillation (AF), with or without structural heart disease. AF without clinical or echocardiographic evidence of structural heart disease ('lone AF') tends to be paroxysmal, lasting from minutes to days.

Beyond the age of 60 years, AF becomes increasingly common, such that 10% of people over the age of 75 years may be affected. Hypertension is the most common associated condition. In those with ischaemic heart disease, AF usually complicates heart failure due to left ventricular damage, rather than being related to ischaemia directly.

ECG 4.03



ECG from a 40-year-old female with recurrent palpitations and no evidence of heart disease.

5. Wide QRS tachycardias

Whereas for practical purposes, all narrow QRS tachycardias are supraventricular, i.e. they originate above the bifurcation of the common His bundle, wide QRS tachycardias can be produced by a number of different mechanisms. This causes confusion and diagnostic difficulty because of the need to distinguish ventricular tachycardia (VT) from supraventricular tachycardia (SVT) with bundle branch block (aberrant conduction). From a clinical point of view, this is obviously important, because of differences in prognosis and treatment. Much has been written on the subject and a number of algorithms have been developed.

Regular versus irregular wide QRS tachycardia

The vast majority of regular wide QRS tachycardias are ventricular in origin and *VT must therefore be the default diagnosis* from the point of view of clinical safety, particularly in an emergency. Most sustained irregular tachycardias, on the other hand, whether wide or narrow, are due to atrial fibrillation.

Wide QRS tachycardias

- **Regular**: most (> 80%) are ventricular. *VT is therefore the default diagnosis*
- **Irregular:**
  - Sustained (> 30 s): most are atrial fibrillation with bundle branch block or pre-excitation
  - Non-sustained: most are ventricular

Clinical pointers

Before discussing the ECG features, consider some of the clinical pointers which may be helpful in differentiating VT from SVT:

A history of previous myocardial infarction

The commonest anatomical basis for VT is the scar of a healed myocardial infarct. Such scars are seldom uniform. Trapped islands of viable muscle are commonly subject to slow conduction and enable the development of re-entry circuits. The probability that a wide QRS tachycardia is VT is extremely high in such a patient.[1]

Shock and clinical perfusion

Contrary to popular belief, VT does not necessarily result in shock or poor perfusion. Conversely, a rapid SVT may result in shock, particularly in a patient with poor left ventricular function. The presence or absence of shock should therefore not influence your assessment of the arrhythmia.

Clinical signs of AV dissociation

If circumstances permit, take time to examine the patient during tachycardia for signs of AV dissociation: irregular cannon waves in the jugular pulse, varying pulse volume, and variation in the first heart sound, due to changing stroke volume as the timing of atrial systole varies. Sometimes these signs are obvious, even though P waves may be difficult to distinguish on the ECG.

Age

Bundle branch block is rare in young people in the absence of congenital heart disease or cardiomyopathy. Ventricular tachycardia (often idiopathic) remains the commonest cause of a regular wide QRS tachycardia in the young but consider antidromic AVRT due to an accessory pathway.

Structurally normal heart

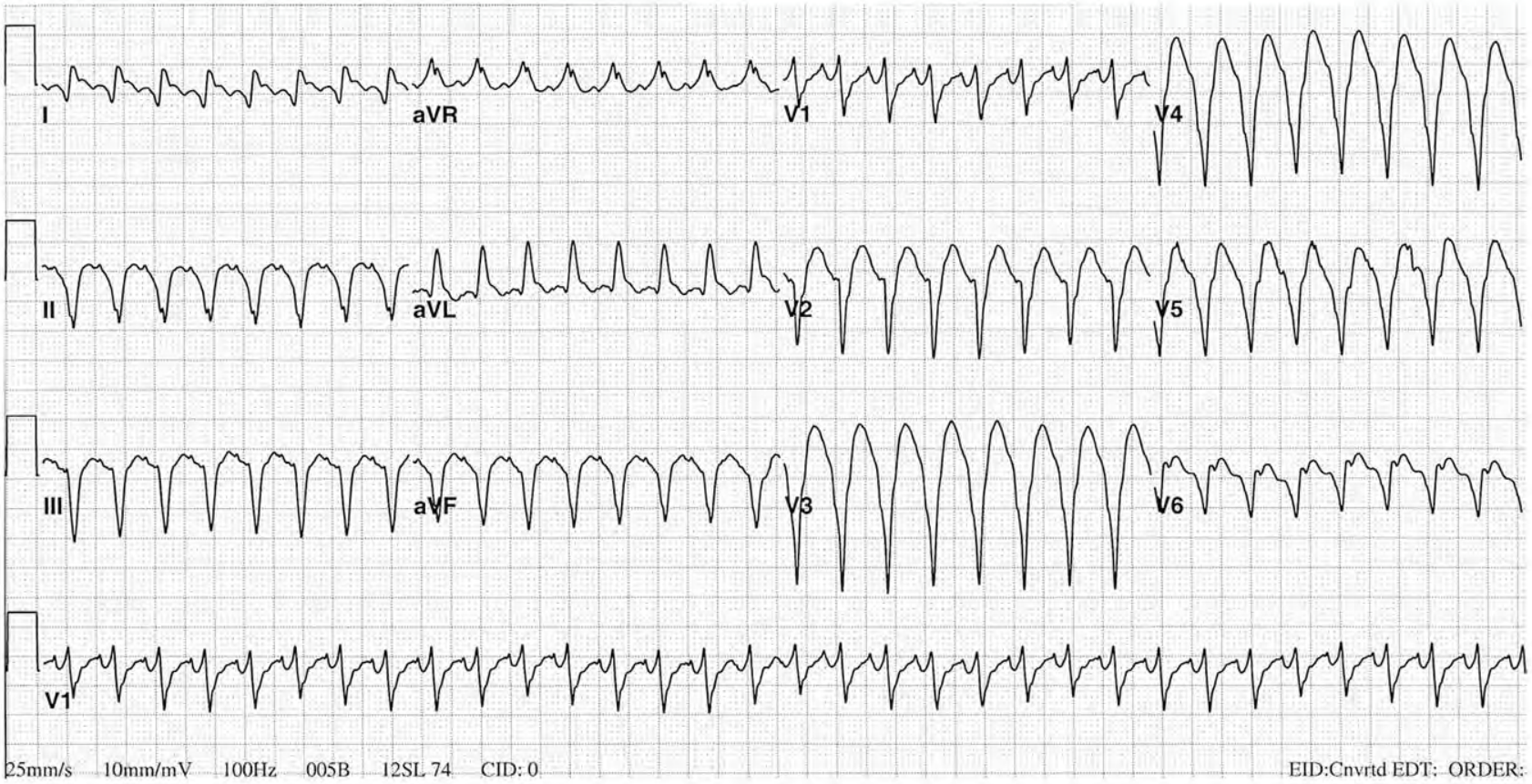
In the absence of a history and clinical evidence of structural heart disease, a regular wide QRS tachycardia is likely to be idiopathic VT, arising either from the right ventricular outflow tract (inferior axis, pseudo-LBBB pattern), or in the fascicles of the left bundle (left axis, pseudo-RBBB pattern). However, VT with a LBBB pattern may be due to occult arrhythmogenic right ventricular cardiomyopathy (ARVC).

Consider also antidromic AVRT and unusual forms of pre-excitation, such as Mahaim tachycardia (see below). If the tachycardia is irregular, particularly with bizarre QRS complexes, consider atrial fibrillation conducting via an accessory pathway.

Mechanisms of wide QRS tachycardias

Narrow QRS rhythms depend on normal conduction via an intact His-Purkinje system. This can only occur if the rhythm originates above the bifurcation of the common His bundle and the bundle branches are functionally intact.

ECG 5.10



ECG of a 35-year-old man who presented with dizziness, palpitations and chest pain.

ECG 5.10: Sustained monomorphic ventricular tachycardia

Overview: Regular wide QRS tachycardia with P waves before each QRS complex. The default diagnosis remains ventricular tachycardia.

Rhythm: Regular

Rate: 198 bpm (18 × 6)

QRS width: 140 ms

QRS axis: –80°

QRS morphology: Negative complexes V2–V6

P waves: Visible in V1 and the inferior leads (arrows)

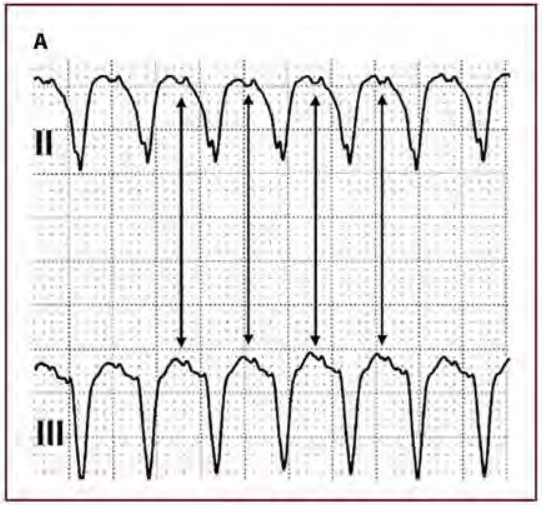
Comment: There appears to be a 1:1 relationship between the P waves and the QRS complexes (A).

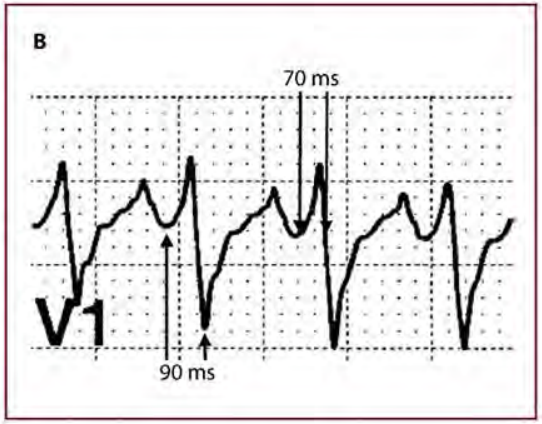
Differential diagnosis: This might suggest the possibility of SVT with LBBB aberration. However, the QRS morphology is not compatible with this diagnosis. The initial R wave in V1 is > 40 ms, too long for LBBB, and the onset to the nadir of the QRS is 90 ms (B). This indicates slow ventricular depolarisation of the ventricles from the outset, without the rapid initial depolarisation of the contralateral ventricle which characterises unilateral bundle branch block. Slow initial depolarisation occurs in ventricular rhythms and ventricular pre-excitation. Antidromic tachycardia of WPW syndrome is, however, excluded by the QS complexes from V4–V6, which indicate ventricular depolarisation starting from the apex. They are pathognomonic for VT. The P waves are the result of retrograde conduction from the ventricles.

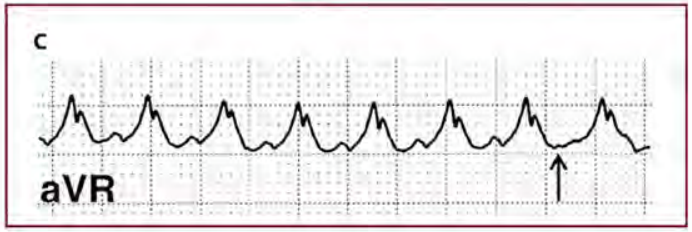
Clinical information: A 35-year-old man with previous inferior myocardial infarct.

Further comment: While there appears to be a 1:1 VA relationship, there is a single missing retrograde P wave, best seen in the last QRS complex in aVR (arrow, C). While this clinches the diagnosis of VT, it could easily be missed or not occur during the recording. The QRS morphology is sufficient to make a certain diagnosis in this case.

Final diagnosis: Sustained monomorphic ventricular tachycardia, proven at EPS.







6. The ECG and computers

Computer technology is now ubiquitous in most instruments, devices and appliances in modern use. The electrocardiogram is no exception. For the first quarter-century after its development by Einthoven, the ECG was recorded using a string galvanometer, the movement of which was amplified and recorded photographically. Later, direct writing machines were developed. The ECG was recorded directly onto heat-sensitive paper via a heated stylus. Modern ECG signals are digitised and then printed on heat-sensitive paper, or by ink-jet or laser. This has a number of advantages and disadvantages.

Advantages
- Storage and replication
- Variety of display formats
- ECG monitoring
- Noise reduction
- Measurement
- Transmission to distant sites

Disadvantages
- Incorrect interpretation
- Poor durability of paper records
- Computer-induced artefacts

Advantages

Storage and replication

Before the advent of computers, each ECG recording was unique. If the tracing was damaged or lost, it was gone forever. While ECGs directly recorded on heat-sensitive paper have proved remarkably durable, they are vulnerable to damage, particularly heat (e.g. when shown using an epidiascope), or if liquid is spilt on the trace.

Computerisation of ECGs began in the late 1960s. The first ECG computer at Mount Sinai Hospital, New York, occupied several rooms and had 64 KB of memory (less than a cheap calculator today). However, rapid developments in storage capacity have resulted in the ability to store hundreds of thousands of 12-lead ECG traces on the hard disk of a desktop PC.

Most of the traces used in this book come from that source. Provided that the ECGs are stored and backed up, they remain available for many years. If the original is lost or damaged, a fresh copy can be printed. Extra copies become available for teaching. Ready access to all ECGs recorded on an individual patient simplifies comparisons over time. At my institution, Groote Schuur Hospital, the ECG management system has stored all ECGs since it was installed in 1989.

Display formats

In the past, the 12 ECG leads were recorded sequentially on a single long strip of paper. Most ECGs today are recorded with at least three simultaneous channels on a single A4-sized sheet of paper. Usually, the 12 leads are recorded in four groups of three, with instantaneous switchover between groups, while the paper runs through the machine at 25 mm/s. A fourth channel records an uninterrupted 'rhythm strip', usually standard II or V1. The rhythm strip may be synchronous with the 12-lead recording (Fig. 6.01) or be recorded consecutively. Alternative configurations include:

- two groups of six leads
- 12 leads recorded simultaneously
- three-lead rhythm strip.

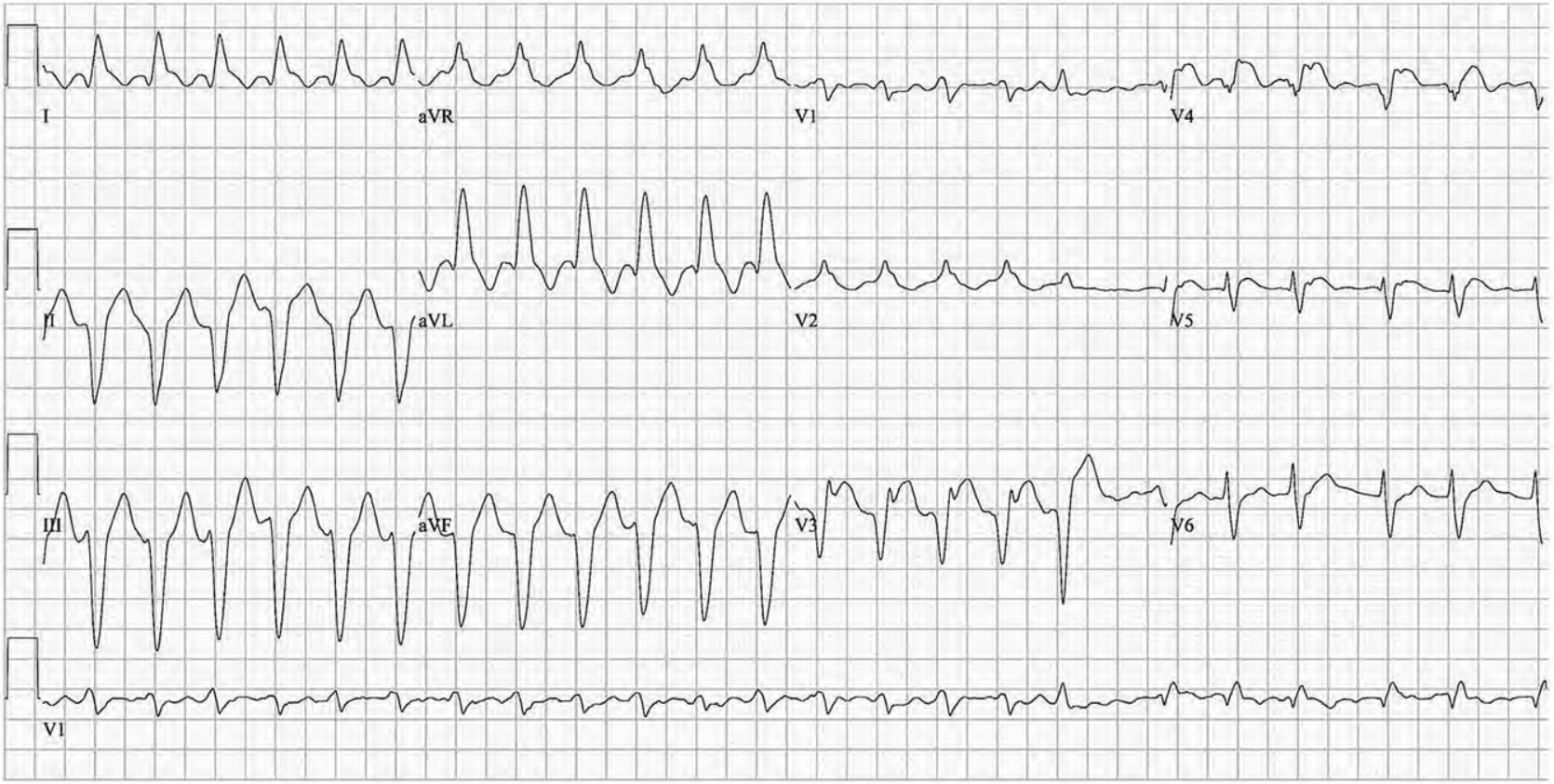
Some machines do not record the lead groups without interruption (Fig. 6.02). This needs to be taken into account when analysing rhythm and heart rate.

ECG monitoring

ECG monitoring in intensive care units has been computerised for many years. On modern systems, an individual patient's ECG from the monitor can be stored continuously for 72 hours or longer.

It is then possible to examine the trace from any particular time and to correlate it with a clinical event, much like ambulatory Holter monitoring. Typically, continuous computer surveillance of the rhythm occurs, with an alarm triggered by a pre-determined 'event'. Despite their increasing sophistication, false alarms are frequent, particularly with movement artefact (Fig. 6.03). Nevertheless, such systems may be very valuable for detecting infrequent events, such as intermittent AV block.

ECG 7.19 ECG recorded from a 68-year-old man who had been admitted four days earlier, after developing severe chest pain.



(A) Which of the following best describes this ECG:

1. Atrial fibrillation + LBBB; recent anterior myocardial infarct
2. Sustained monomorphic VT; recent anterior myocardial infarct
3. Monomorphic VT + atrial fibrillation; recent anterior myocardial infarct
4. Atrial fibrillation + RBBB
5. Atrial flutter + LBBB

(B) Which of the following is most likely to help with diagnosis:

1. Carotid sinus massage
2. Adenosine
3. Another ECG
4. A signal-averaged ECG

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