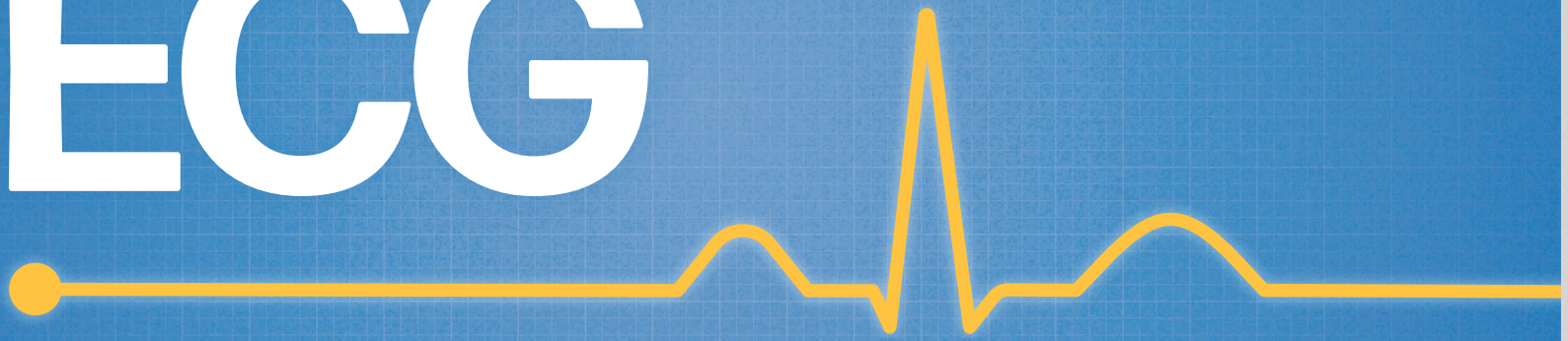
Essential ECG



Mark Mills, Akshay Gaur and David Warriner

A practical guide to recording, interpreting, and reporting ECGs with confidence

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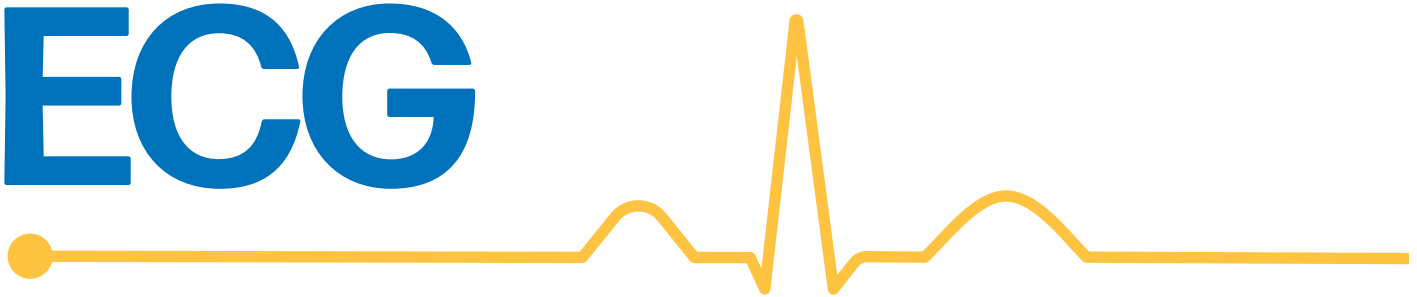

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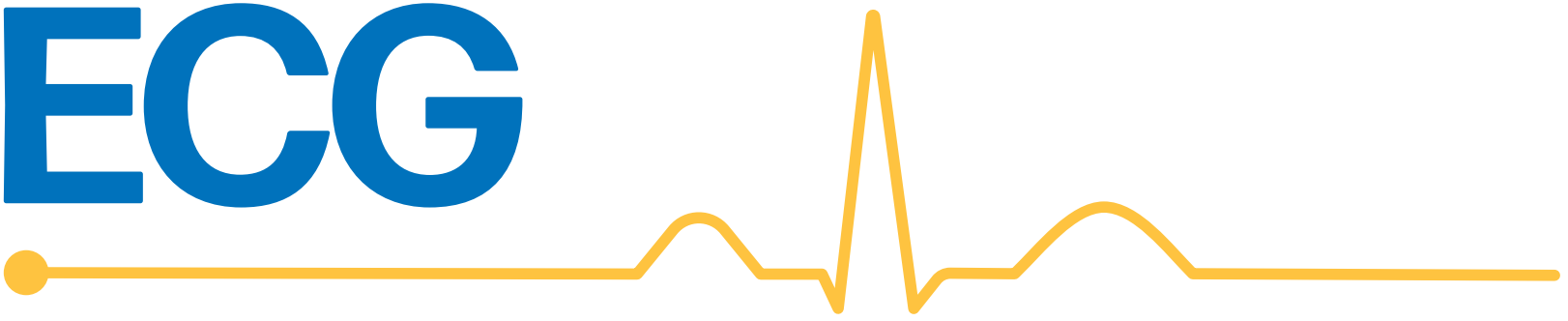

A practical guide to recording, interpreting and reporting ECGs with confidence

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Case scenarios 20.31 to 20.50 of the test yourself ECGs are available online; to view these additional ECGs see www.scionpublishing.com/Essential-ECG then go to the "Resources" tab.

Foreword

While the first heartbeat is detected between the 6th and the 7th week of fetal life, the first rhythmic beat starts a week or two earlier and continues throughout one's life.

The detection of electrical activity from the heart arose from the discoveries of electrical activity in a frog's muscle (Galvani, 1786), electrical current that accompanies each heartbeat in a frog (Matteucci, 1842) and the recording from the body surface of electrical cardiac activity (Waller, 1887). It was Willem Einthoven who established the principles of electrocardiography, giving it its nomenclature, establishing the electrical axis and creating the first machine to record the electrocardiogram (ECG). Einthoven was deservedly awarded the 1924 Nobel prize in Physiology and Medicine. One of his contemporaries, Sir Thomas Lewis, defined atrial fibrillation and wrote the first textbook on electrocardiography.

Since then, many more books on the ECG have been written, ranging from the highly authoritative *The Introduction to Electrocardiography* by Leo Schamroth in 1957 to the more accessible *ECG Made Easy* by John Hampton in 1973.

It is with these facts in mind that I have the pleasure of introducing *Essential ECG* by Drs Warriner, Mills and Gaur. This is a textbook that combines the authority of knowledge and excellence, with the ease of reading and practicality that makes it readily accessible. This book is an essential read for all those who need to know and understand the ECG to be able to provide the best possible care for their patients. The book provides the reader with the knowledge to deal with a wide variety of circumstances and patient complaints: from those presenting in a cardiac emergency to those with common complaints such as palpitations, chest discomfort, breathlessness, and syncope. The coverage also extends to the electrocardiographic changes in patients with, for example, kidney disease, endocrine disorders or electrolyte imbalance, and to those who are asymptomatic but need cardiac screening. With the expansion of our cardiology knowledge, screening has expanded exponentially in the last few decades to include exclusion of cardiac, neuromuscular or other inheritable conditions amongst relatives of sufferers of such conditions; or those starting a career in professional competitive sports; those embarking on work in unforgiving environments such as divers or offshore workers; or those whose work could put significant stresses on their cardiovascular system through aggressive G forces such as military pilots and astronauts.

The book is written with unparalleled clarity. This is helped by good use of excellent illustrations that combine anatomical, physiological and pathological concepts in the same figure, making potentially complex concepts easily accessible to the reader. It is good to see the authors starting from primary principles, thus not relying on prior knowledge; this makes the book accessible to the wide variety of readers it is intended to serve, including the novice who wants to attain competence in the reading and the interpretation of ECGs. This is important because ECG interpretation is no longer a specialist test limited to cardiologists and electrophysiologists.

In the second part of the book, the authors provide the reader with the opportunity to test their new-found knowledge on real-life ECGs. This is invaluable in helping to consolidate the learning from the first section. Readers are encouraged to add their own notes as they interpret the ECGs presented before being provided with the expert assessment of the authors.

By democratising understanding of an essential skill for many healthcare workers, *Essential ECG* is a book that I would highly commend to medical and nursing students, to paramedics, physician assistants, physicians and surgeons in training, and to practising nurses and doctors in primary and secondary care.

I would like finally to congratulate the authors, three fine cardiologists and teachers, for completing and publishing their brilliant textbook, *Essential ECG*.

Abdallah Al-Mohammad
Professor of Cardiology, Sheffield

Preface

We have written this book for medical students, allied health professionals and clinicians wanting a comprehensive introductory guide to electrocardiograms (ECGs). We are all enthusiastic teachers and wanted to take readers on a journey from complete novice to skilled ECG reader – the book assumes no prior knowledge of ECG and breaks everything down step by step.

No matter how unusual or uninterpretable an ECG may look at first glance, a simple structure can be used to work out what is going on. With repeated exposure to lots of ECGs, the simple structure we introduce is gradually replaced by pattern recognition (i.e. from unconscious incompetence to unconscious competence). This will be key in emergency situations, allowing you to give your full attention to other more complex, dynamic and pressing issues.

To help you on this journey, the first part of the book:

- starts by introducing basic cardiac anatomy and physiology
- then proceeds to describe how to perform and read ECGs – we have included plenty of figures and explanations to build understanding
- and guides you into differentiating normal from the abnormal – the unique format helps by allowing side-by-side comparison.

All major cardiac disease processes are covered including, for example, ischaemic, arrhythmic and structural heart disease.

The second part of the book is dedicated to 50 ECGs that everyone interpreting ECGs in clinical practice should be able to recognise correctly. This includes where in the heart a myocardial infarction is occurring and how to identify different types of narrow and broad complex tachycardia.

By the end of the book we are confident that you will feel comfortable reading ECGs – we hope that you might even enjoy it!

David, Mark and Akshay

About the authors

Dr Mark Mills is a cardiology registrar with a subspecialty interest in cardiac electrophysiology and implantable cardiac devices. He holds an MSc in Cardiovascular Research and a PhD focused on atrial fibrillation catheter ablation. Mark has served as the trainee representative on the British Heart Rhythm Society council and as a member of the European Heart Rhythm Association (EHRA) Scientific Initiatives Committee. Outside of medicine, he enjoys playing the piano, keeping active and spending time with his family.

Dr Akshay Gaur is a cardiology registrar in training with a deep passion for medical education. He serves as a Visiting Lecturer at the University of Leeds and continues to deliver lectures on ECGs at his alma mater, the University of Sheffield. Dedicated to advancing the practice of cardiology, Akshay strives to make complex concepts accessible and meaningful for students, trainees and medical professionals alike. His passions includes tennis and road trips.

Dr David Warriner is a consultant cardiologist specialising in imaging (echocardiography and cardiac computer tomography) and adult congenital heart disease, working between Doncaster and Leeds in South and West Yorkshire respectively. He is an honorary senior lecturer at the University of Sheffield and programme director for higher speciality cardiology training in South Yorkshire. He is passionate about undergraduate and postgraduate medical education and, as such, this is his fourth textbook. Two of his other books have been translated into French and Mandarin.

Acknowledgements

I am deeply grateful to my parents for their enduring support and guidance. My heartfelt thanks also go to my wife, Jodie, for her patience and encouragement, and to our son, Henry, whose presence brings constant joy and balance to life. I dedicate this book to them.

Mark Mills

My heartfelt gratitude goes to my parents, whose love, sacrifices, blessings, and unwavering faith have been the foundation of everything I do. Their strength and kindness continue to guide me in all aspects of life. Above all, I owe a special debt of love to my elder sister, Priya – my constant pillar of support and my greatest source of encouragement. Her belief in me has carried me through every doubt and every milestone. This book is as much hers as it is mine.

Akshay Gaur

Dedicated to Beth, Agnes and Felix. And I quote, "This might be my last book, might…"

David Warriner

The original ECGs were standardised and digitised using PMcardio. You can get PMcardio here: https://bit.ly/pmcardio-pub.

PMcardio was created by Robert Herman, MD: Department of Advanced Biomedical Sciences, University of Naples Frederico II, Naples, Italy; Cardiovascular Center, OLV Hospital, Aalst, Belgium; Powerful Medical, Bratislava, Slovak Republic.

Abbreviations

ACM	arrhythmogenic cardiomyopathy
ACS	acute coronary syndrome
AF	atrial fibrillation
AIVR	accelerated idioventricular rhythm
ARVC	arrhythmogenic right ventricular cardiomyopathy
AV	atrioventricular
AVB	AV block
aVF	augmented vector foot
aVL	augmented vector left
AVN	atrioventricular node
AVNRT	atrioventricular nodal re-entrant tachycardia
aVR	augmented vector right
AVRT	atrioventricular re-entry tachycardia
BBB	bundle branch block
BCT	broad complex tachycardia
bpm	beats per minute
CB	capture beat
CRT	cardiac resynchronisation therapy
DC	direct current
DCCV	direct current cardioversion
ECG	electrocardiogram
FB	fusion beat
HCM	hypertrophic cardiomyopathy
ICD	implantable cardioverter defibrillator
ICP	intracranial pressure
IVCD	interventricular conduction delay
LA	left arm
LAD	left anterior descending
LAFB	left anterior fascicular block
LBBB	left bundle branch block
LCx	circumflex artery
LL	left leg
LPFB	left posterior fascicular block
LV	left ventricle
LVH	left ventricular hypertrophy
LVOTO	left ventricular outflow tract obstruction
MAT	multifocal atrial tachycardia
MI	myocardial infarction
NCT	narrow complex tachycardia
NSAID	non-steroidal anti-inflammatory drug
NSTEMI	non-ST elevation myocardial infarction
PAC	premature atrial complex
PDA	posterior descending artery
PE	pulmonary embolism
PPCI	primary percutaneous coronary intervention
PVC	premature ventricular complex
RA	right arm
RBBB	right bundle branch block
RCA	right coronary artery
RL	right leg
RV	right ventricle
RVH	right ventricular hypertrophy
SCD	sudden cardiac death
STEMI	ST elevation myocardial infarction
SVE	supraventricular ectopic
SVT	supraventricular tachycardia
VE	ventricular ectopic
VF	ventricular fibrillation
VT	ventricular tachycardia

5.1 Having a framework

- When interpreting an ECG, it is important to have a clear framework in mind. Sticking to a framework will ensure that you consistently and systematically review all aspects of the ECG, reducing the probability of missing important findings.
- We recommend the following framework:
  - confirming the basics (see *Section 5.2*):
    - patient demographics
    - paper speed and voltage
    - checking the quality of the recording
  - heart rate (see *Section 5.3*)
  - heart rhythm (see *Section 5.4*)
  - heart axis (see *Section 5.5*)
  - P waves (see *Section 4.2*)
  - PR interval (see *Section 4.8*)
  - QRS complexes (see *Section 4.6*)
  - ST segments (see *Section 4.11*)
  - QT interval (see *Section 4.9*)
  - T waves (see *Section 4.7*)
  - bringing it all together: making a diagnosis (see *Section 5.7*).

5.2 The basics

- Demographics:
  - before reviewing the results of any clinical test, it is crucial to check that they are from the right patient from the right location
  - best practice includes checking that the date, time, ward, patient name, date of birth and hospital number are correct. This is particularly important if you were not present when the ECG was performed
  - assigning the results of a test to the wrong patient could have significant consequences for both you and the patient
  - if there is any doubt, or if the result itself is at odds with the clinical picture, then repeat it
  - similarly, as is often the case in clinical practice, if any of this information is missing, you review the ECG at your own risk.
- Paper speed and voltage:
  - it is important to check that both paper speed and voltage are set as standard (*Fig. 5.1*). This information can be found at the bottom of the ECG
  - using a standard paper speed (25 mm/s):
  - 1 small square (1 mm) is 0.04 s (or 40 ms)
  - 1 large square (5 mm) is 0.2 s (or 200 ms)
    - 50 large squares is 10 s
    - 300 large squares is 60 s
  - using a standard paper voltage (0.1 mV/mm):
  - 2 large squares (10 mm) is 1 mV
  - this information doesn't need to be included in your report, unless it has been altered for any reason (intended or otherwise)
  - if the QRS complexes are large and overlapping (e.g. in left ventricular hypertrophy; see *Section 6.3.1*) then the voltage settings may necessarily be reduced
  - if voltage settings are incorrectly reduced, QRS complexes may appear small when they are actually normal, raising erroneous concerns regarding myocardial infiltration
  - if voltage settings are incorrectly increased, QRS complexes may appear large when they are actually normal, raising erroneous concerns regarding left ventricular hypertrophy
  - if paper speed is incorrectly set (e.g. 50 mm/s or 2.5 mm/s) then the heart rate may appear erroneously slow or fast, respectively.
- Other checks:
  - it is worth commenting on the quality of the ECG, e.g. presence of artefact, tremor or baseline wander (see *Section 16.3*).

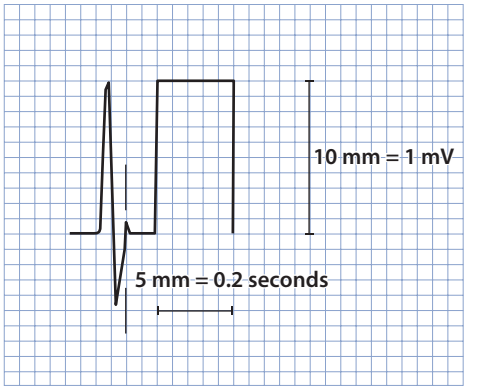

Fig. 5.1: Standardising wave for an ECG, showing the correct paper speed and voltage.

5.3 Heart rate

- The normal heart rate is between 60 and 100 beats per minute (bpm), which corresponds with the number of QRS complexes on the ECG.
- Any heart rate above 100 bpm is a tachycardia and any heart rate slower than 60 bpm is a bradycardia (*Table 5.1*).
- Calculating the heart rate is the first step in interpreting the ECG, i.e. is the heart rate too slow, too fast or just right?
- The 'rhythm strip' is the long trace (50 large squares; 10 s), usually of lead II, at the bottom of a 12-lead ECG and it serves as a reference. The heart rate is best judged on this strip because it shows the time interval between each QRS complex, specifically the R wave: the RR interval.

Table 5.1: Causes of slow and fast heart rates

Rate (bpm)	Term	Causes
<60	Bradycardia	Sleep, hypothermia, hypothyroidism, athletic training, arrhythmia and certain drugs (e.g. beta-blockers and calcium channel blockers)
60–100	Normal	
>100	Tachycardia	Pain, anxiety, fever, exercise, arrhythmia, shock and hyperthyroidism

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Table 5.2: Relationship between large squares on ECG (0.2 s) and successive R waves and heart rate

RR interval (large squares)	Heart rate (bpm)
1	300
2	150
3	100
4	75
5	60
6	50
7	42
8	38
9	33
10	30

- There are two methods to calculate heart rate (assuming the paper speed is set to 25 mm/s).
  - method 1 – for regular rhythms:
    - the quickest and easiest method is to divide 300 by the number of large squares between subsequent R waves (the RR interval)
    - 300 large squares represent 1 minute (300 × 0.2 s) and so dividing 300 (1 minute) by the RR interval (beat) = rate in bpm (*Table 5.2*)
    - for example, if the RR interval is 5 large squares (i.e. 1 s), then 300 divided by 5 is 60 bpm.

Table 5.3: Causes of regular and irregular cardiac rhythms

Rhythm	Causes
Regularly regular	Sinus rhythm 1st- or 3rd-degree heart block
Regularly irregular	Bigeminy/trigeminy 2nd-degree heart block
Irregularly irregular	Atrial and ventricular ectopic beats Atrial fibrillation Atrial flutter with variable block Multifocal atrial tachycardia

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  - method 2 – for irregular rhythms (*Table 5.3*) such as atrial fibrillation:
    - as a 12-lead ECG rhythm strip is 10 seconds, counting the number of QRS complexes (beats) within a 10-second rhythm strip and multiplying by 6 = rate in bpm. This also works with ad hoc rhythm strips, e.g. from a defibrillator or a portable cardiac monitor
    - alternatively, count the number of QRS complexes and multiply by an appropriate factor to calculate the heart rate in bpm (*Figure 5.2*)
    - for example, if 9 QRS complexes are counted over 15 large squares:
      - 15 large squares = 15 × 0.2 s = 3 s
      - to convert this to rate per minute = 60 s / 3 s = 20; so 20 x 9 = 180 bpm.

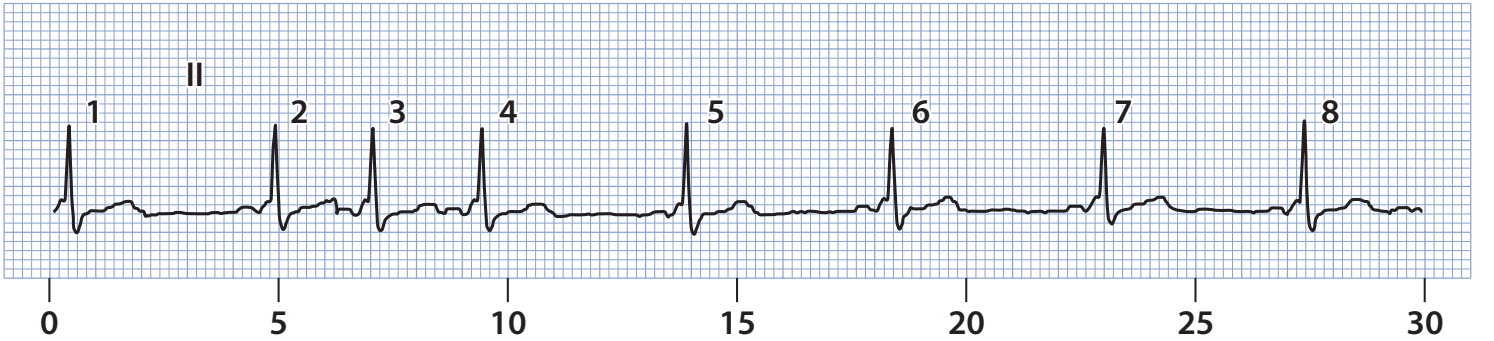

Fig. 5.2: Method for calculating heart rate from an irregular rhythm. In this example, 8 QRS complexes are counted in 30 large squares (30 x 0.2 s = 6 s), so 8 in 6 s is equivalent to 8 x 10 = 80 in 60 s = 80bpm.

5.4 Heart rhythm

- The next step to interpreting the ECG is assessing the heart rhythm, guided by the regularity of the pulse:
  - regularly regular rhythm: the period between each pulse wave is the same consistently, e.g. sinus rhythm
  - regularly irregular rhythm: the period varies but has a recurring pattern, e.g. ventricular bigeminy (sinus beat then premature ventricular contraction) (see *Section 11.3*)
  - irregularly irregular rhythm: there is no pattern and the pulse feels chaotic, e.g. atrial fibrillation (see *Section 9.3*).
- Specifically, cardiac rhythm refers to whether the RR interval is fixed (i.e. regular) or varying (i.e. irregular) (*Table 5.3*).
- Sinus rhythm refers to when the P wave (originating from the sinus node) precedes each and every QRS complex (*Fig. 1.1*). Any other variation is therefore by definition not sinus rhythm, but instead an arrhythmia.
- Arrhythmias can be divided into tachyarrhythmias (heart rate >100 bpm) and bradyarrhythmias (heart rate <60 bpm):
  - tachyarrhythmias:
    - narrow complex tachycardias (see *Chapter 9*): often referred to as 'supraventricular tachycardias' because they are usually due to tachycardias originating above ('supra') the ventricles (i.e. within the atria); common causes include (but are not limited to):
      - atrial fibrillation: no organised atrial activity seen with narrow, irregular and fast rhythm
      - atrial flutter: organised atrial activity with characteristic flutter waves and a narrow, regular, often fast ventricular response
      - AV nodal re-entrant tachycardia: atrial activity often hidden within or just after the QRS complex with a narrow, regular, fast rhythm
    - broad complex tachycardias (see *Chapter 10*): referring to tachycardias originating within the ventricles; common causes include:
      - ventricular tachycardia: no obvious organised atrial activity seen with broad, regular and fast rhythm
      - ventricular fibrillation: no organised atrial or ventricular activity seen with broad, irregular and fast rhythm
  - bradyarrhythmias (see *Chapter 8*):
    - this refers to bradycardia and the presence of an arrhythmia. The most common causes of bradyarrhythmia include:
      - sinus node disease
      - atrioventricular node dysfunction.

5.5 Heart axis

- The cardiac axis refers to the dominant direction of electrical activity within the heart and can be reported using an angle in degrees.
- The electrical deflection measured by the ECG represents both the amount and the direction of the heart's electrical activity.
- The normal heart axis in healthy individuals is between −30° and +90° (*Fig. 5.3*).
- In disease, the heart axis may swing abnormally in either direction (*Fig. 5.4*):
  - right axis deviation is defined as an axis between +90° and +180°
  - left axis deviation is defined as an axis between −90° and −30°
  - extreme axis deviation (so called 'north west') is defined as an axis between +180° and −90°.
- Causes of cardiac axis deviation are summarised in *Table 5.4*.
- As discussed in *Section 3.3*, the 12-lead ECG actually comprises only 10 physical ECG leads attached to the patient (*Fig. 5.5*). Each ECG lead displays a signal moving towards it as an upwards ('positive' or '+ve') deflection, and moving away as a downwards (negative or '−ve') deflection. The Q wave is the first negative deflection, the R wave the first upwards deflection and an S wave is any negative deflection after an R wave (*Fig. 5.6*).
- There are a variety of methods used to determine the heart axis. Ultimately, it is important to know if the axis is normal or not. If not normal, is it left, right or extreme 'north west' (NW) axis?

Table 5.4: Causes of right and left axis deviation

Deviation	Example causes
Right axis (+90° to +180°)	Right bundle branch block Right ventricular hypertrophy Pulmonary embolism Cor pulmonale Often also seen in healthy people, especially if they are tall and thin
Left axis (−30° to −90°)	Left bundle branch block Left ventricular hypertrophy Cardiac pacemaker Left anterior hemiblock

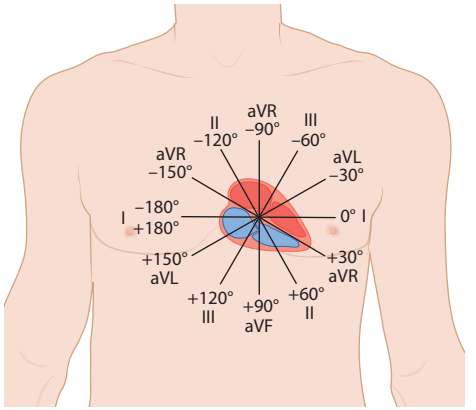

Fig. 5.3: The hex-axial reference system is a representation of the 12-lead ECG to assess the cardiac axis.

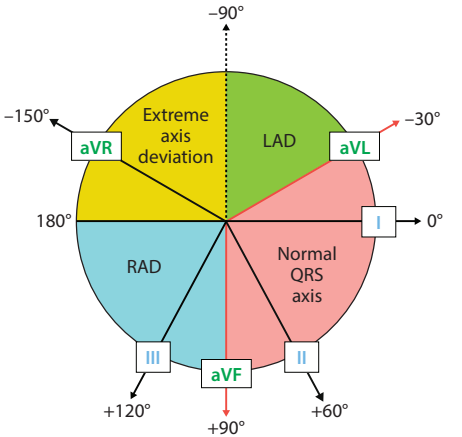

Fig. 5.4: Deviation of the cardiac axis.

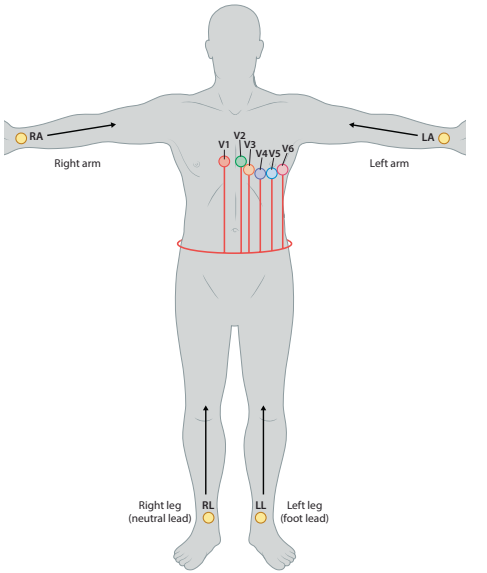

Fig. 5.5: Placement of the 10 ECG electrodes to create a 12-lead ECG. The six chest leads 'look' at the heart in an axial (horizontal) plane, and the four limb leads 'look' at it in a coronal (vertical) plane.

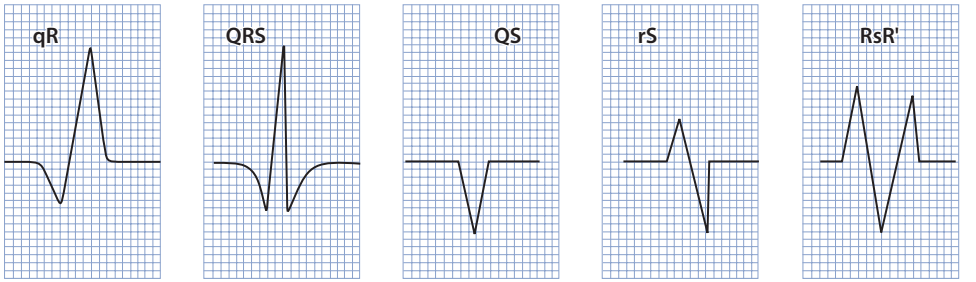

Fig. 5.6: The various forms of the QRS complex.

- Method 1 (looking for the lead with the maximum QRS deflection):
  - the axis corresponds with the lead containing the QRS complex with the largest positive deflection, e.g. maximal deflection above the isoelectric line
  - once you've identified this QRS complex, use the hex-axial reference to determine the axis (*Fig. 5.3*)
  - for example, if lead II has the largest +ve QRS complex, the axis is +60°.

NOTES

- Method 2 (the 'rule of thumbs' (*Fig. 5.7*):
  - imagine the QRS complexes as thumbs pointing up (i.e. R > S) or down (i.e. S > R)
  - when the axis is normal, all leads (except occasionally lead III) usually point up and therefore thumbs are all up
  - if the QRS in lead I points up (+ve) but the QRS in leads II and III point down (–ve), there is left axis deviation. This is because the predominant direction of depolarisation is opposite to leads II (+60°) and III (+120°)
  - if the QRS in lead I points down (–ve), but the QRS in leads II and III point up (+ve) there is right axis deviation. This is because the predominant direction of depolarisation is towards leads II (+60°) or III (+120°)
  - the easiest way to remember this, is to use the '**R**eaching towards' and '**L**eaning away' method:
    - if the thumbs in leads I and III are 'Reaching towards' each other, this is right axis deviation;
    - if the thumbs in lead I and III are 'Leaning away' from each other, this is left axis deviation.

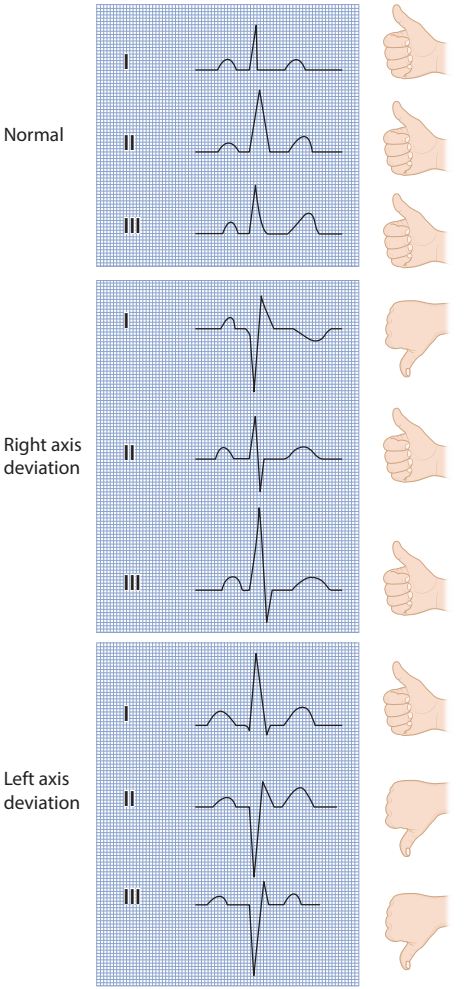

Fig. 5.7: The 'rule of thumb' method for determining cardiac axis.

5.6 Waves, complexes, intervals and segments

- Next, you should examine individual waves, complexes, intervals and segments.
- The most important ones to comment on in all ECGs are:
  - P waves
  - PR interval
  - QRS complexes
  - ST segments
  - QTc interval
  - T waves.
- *Chapter 4* provided a detailed breakdown of the meaning and interpretation of these features of the ECG.

NOTES

5.7 Bringing it all together

- As with any diagnostic test, the significance of the finding is influenced by the clinical context.
- Modern ECG machines will provide an automated report including diagnosis, heart rate and intervals (usually found at the top of the ECG). However, these are not always accurate and it is therefore best to assess them yourself.
- You should review the rate, rhythm, cardiac axis, conduction intervals, QRS complex and ST segment and T waves. All these do not need to be documented if they are normal, but a concise one-line interpretation should be included in the notes.
- Simply writing down a technical report without interpreting the ECG in light of the patient's symptoms is not helpful. Specific symptoms should prompt commenting on the presence or absence of certain abnormalities:
  - **chest pain:**
    - a report should comment on the presence or absence of myocardial ischaemia or infarction (see *Chapter 13*) along with differentials, e.g. pericarditis (see *Section 14.2*) or pulmonary embolism (see *Section 16.1*)
    - if present, report the abnormality and region affected (e.g. anterior T wave inversion or inferior ST elevation). Also report the likely diagnosis (e.g. non-ST elevation myocardial infarction (NSTEMI) or ST elevation myocardial infarction (STEMI), respectively)
    - or, if pathological changes are not present, include "no evidence of any ischaemic changes" in the report
  - **breathlessness:**
    - whilst there are many causes (cardiac or otherwise), there are a few key abnormalities to look out for on the ECG. Specifically, look for evidence of ischaemia (see *Chapter 13*), pulmonary embolism (see *Section 16.1*), arrhythmia, small complexes, left ventricular hypertrophy (see *Section 6.3.1*) and bundle branch block (see *Chapter 12*). If present, these should be included in the report along with the possible diagnosis (e.g. "left bundle branch block causing left ventricular systolic dysfunction" (see *Section 12.3*))
    - if changes are not present, include "no evidence of ECG changes causing dyspnoea" in the report
  - **palpitations:**
    - a report should include the presence or absence of any bradycardia (see *Chapter 8*) or tachycardia (see *Chapters 9* and *10*), along with pauses (see *Chapter 8*) or extra beats (see *Chapter 11*)
    - if present, this should be included in the report along with the possible diagnosis (e.g. "premature atrial contractions causing palpitations" (see *Section 11.2*))
    - if changes are absent, include "no evidence of ECG changes causing palpitations" in the report
    - a prolonged period of cardiac monitoring may be required if the ECG is normal and the patient was asymptomatic at the time of recording
  - **loss of consciousness:**
    - whilst there are many causes (cardiac and non-cardiac), there are a few key abnormalities to look out for on the ECG
    - specifically, look for evidence of myocardial ischaemia (see *Chapter 13*), pulmonary embolism (see *Section 16.1*), arrhythmia, prolonged QTc (see *Section 7.4*), left ventricular hypertrophy (see *Section 6.3.1*) and bundle branch block (see *Section 12.3*)
    - if present, this should be included in the report along with the possible diagnosis (e.g. "third-degree heart block causing cardiac syncope" (see *Section 8.3.4*)
    - if no changes are present, include "no evidence of ECG changes causing syncope" in the report
    - true cardiac syncope is relatively rare; because episodes are often short and transient, the 12-lead ECG only has a 10% diagnostic yield in this scenario.

5.7.1 Example of a normal ECG

Here is a normal 12-lead ECG (*Fig 5.8*). Below, we describe how to systematically assess this ECG using the framework presented in this chapter.

- Rate: normal (85 bpm)
  - explanation:
    - each QRS complex is separated by 3.5 large squares (*Fig. 5.9*)
    - using 'Method 1' described in *Section 5.3*, the heart rate = 300 / 3.5 = 85 bpm.

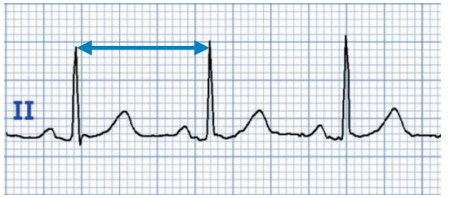

Fig. 5.9: The number of large squares (blue arrow; 3.5 large squares) between QRS complexes can be used to calculate the heart rate.

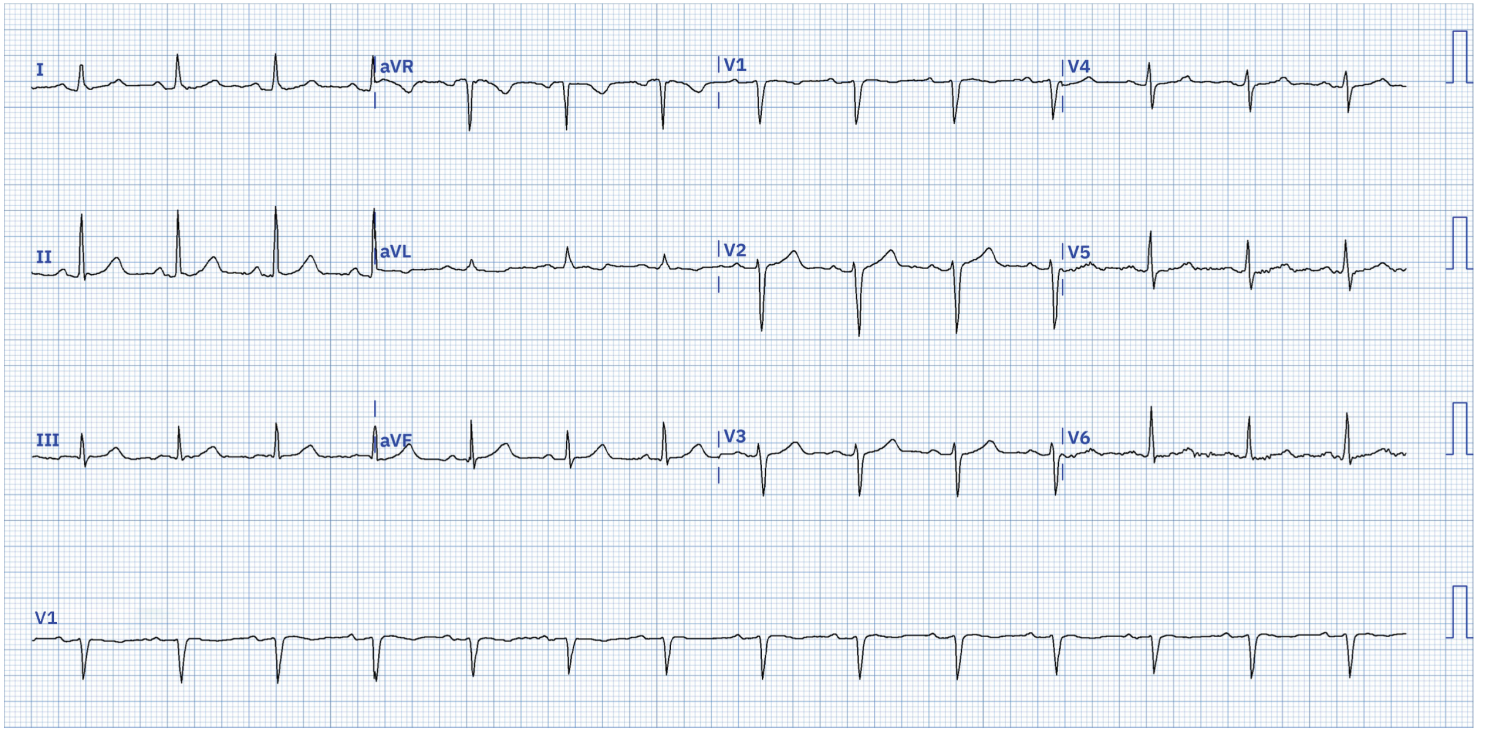

Fig. 5.8: A normal 12-lead ECG.

- Rhythm: regular (sinus rhythm)
  - explanation:
    - there are P waves, QRS complexes and T waves in a regular, recurring pattern (*Fig. 5.10*)
    - the interval between each QRS complex is regular (*Fig. 5.10*)
    - by definition, this is a normal ('sinus') rhythm.

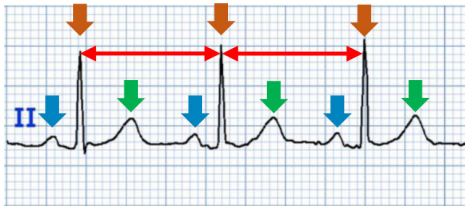

Fig. 5.10: P waves (blue arrows) are followed by QRS complexes (orange arrows) and T waves (green arrows) in a recurring pattern. The interval between QRS complexes (red arrows) is regular.

- Axis: normal
  - explanation:
    - the axis corresponds with the lead containing the QRS complex with the largest positive deflection
    - in this ECG, this is lead II (*Fig. 5.11*)
    - using the hex-axial reference (*Fig. 5.3*), the cardiac axis is therefore +60°, which is normal.

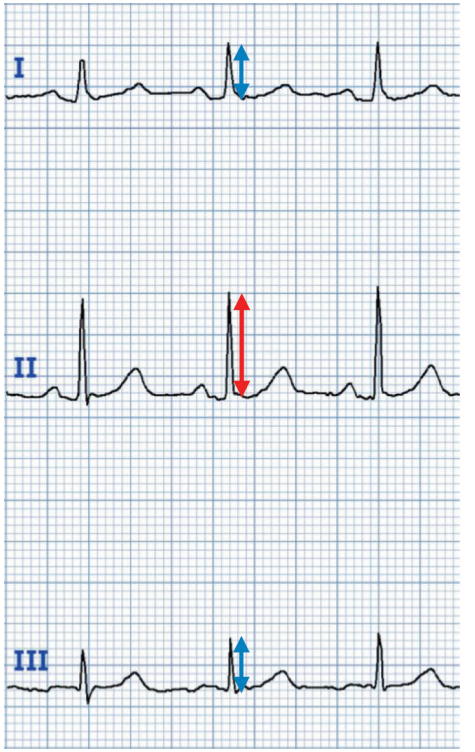

Fig. 5.11: Lead II contains the QRS complex with the largest positive deflection (red arrow). The QRS complexes are smaller in the other leads (for example, in leads I and III represented by blue arrows).

- P waves: normal
  - explanation:
    - a P wave is seen before every QRS complex, and each P wave is less than 120 ms (3 small squares) in duration (*Fig. 5.12*).

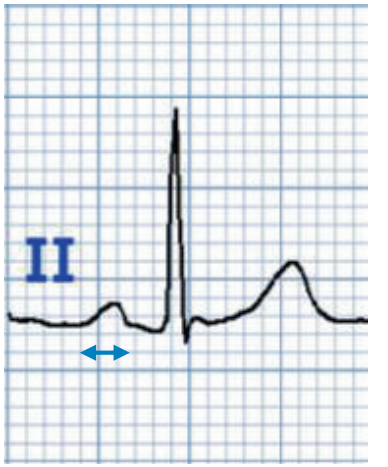

Fig. 5.12: The P wave is seen before the QRS complex and is less than 3 small squares wide (blue arrow).

- PR interval: normal
  - explanation:
    - this is the interval between the start of the P wave and the start of the QRS complex (*Fig. 5.13*)
    - a normal PR interval is 120–200 ms (3 to 5 small squares)
    - in this case, it is 180 ms (4.5 small squares).

- QRS complexes: normal
  - explanation:
    - a normal QRS complex duration is less than 120 ms (3 small squares)
    - in this case, it is 80 ms (2 small squares) (*Fig 5.14*).

- QTc interval: normal
  - explanation:
    - the QTc can be calculated using Bazett's formula:

$$QT_cB = \frac{QT}{\sqrt{RR}}$$

where QTcB = corrected QT interval; QT = measured QT interval (in ms); RR = the interval between subsequent R waves (in s)

- in this example, QT = 360 ms; RR = 0.72 s (*Fig. 5.15*)
- therefore, QTc = 360 / √ 0.72 = 424 ms.

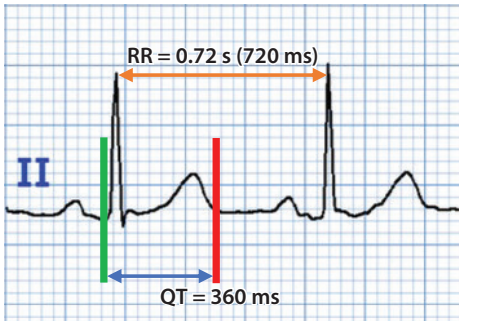

Fig. 5.15: The QT is measured from the start of the QRS complex (green line) to the end of the T wave (red line); in this case, it is 360 ms (9 small squares; blue arrow). The RR interval is 0.72 seconds (18 small squares).

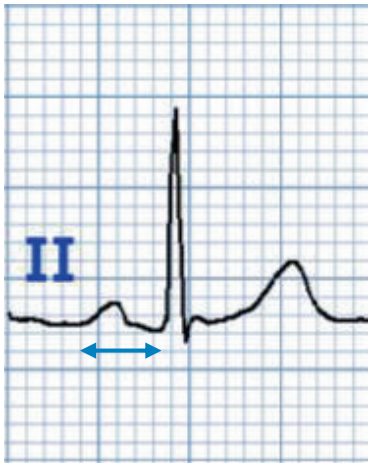

Fig. 5.13: The PR interval is 4.5 squares wide (blue arrow).

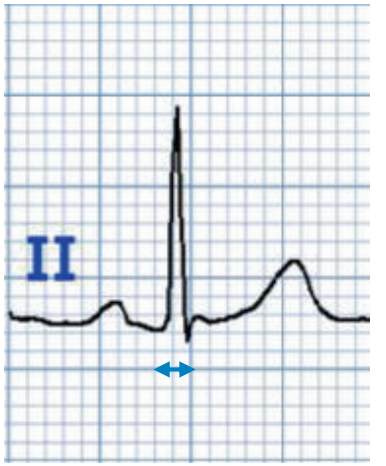

Fig. 5.14: The QRS complex is 2 small squares wide (blue arrow).

NOTES

- ST segments: normal
  - explanation:
    - the ST segment is flat (or 'isoelectric') in all leads (see example of three leads in *Fig. 5.16*).

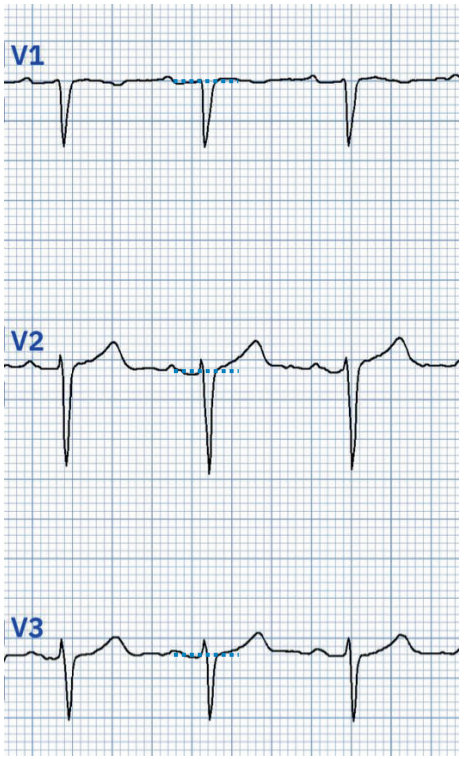

Fig. 5.16: Isoelectric ST segments (dotted blue lines representing the electric baseline).

- T waves: normal
  - explanation:
    - the T waves are positive in all leads (see example in *Fig. 5.17*), except in leads aVR and V1 (*Fig. 5.18*) where they are negative
    - negative T waves in aVR and V1 is a normal finding.

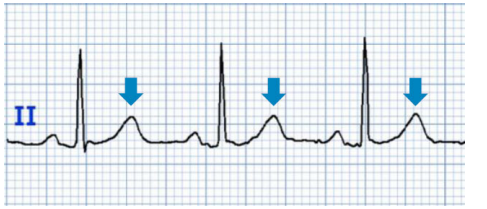

Fig. 5.17: Positive T waves in lead II (blue arrows).

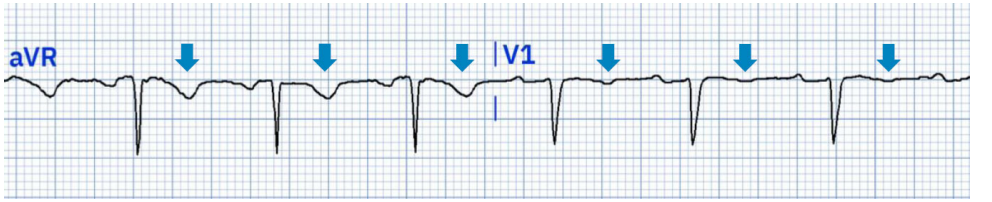

Fig. 5.18: Negative T waves in leads aVR and V1 (blue arrows).

NOTES

5.7.2 Example of an abnormal ECG

Here is an abnormal 12-lead ECG (*Fig 5.19*). Below, we describe how to systematically assess this ECG using the framework presented in this chapter.

- Rate: normal (64 bpm)
  - explanation:
    - each QRS complex is separated by 4.7 large squares (*Fig. 5.20*)
    - using 'Method 1' described in *Section 5.3*, the heart rate = 300 / 4.7 = 64 bpm.
- Rhythm: regular (but not sinus rhythm)
  - explanation:
    - there are P waves, QRS complexes and T waves, but not in a regular, recurring pattern (*Fig. 5.21*)
    - the interval between each QRS complex is regular (*Fig. 5.21*)
    - by definition, this is an abnormal rhythm.
- Axis: normal
  - explanation:
    - the axis corresponds with the lead (or leads) containing the QRS complex with the largest positive deflection
    - in this ECG, this is leads II and III (*Fig. 5.22*)
    - using the hex-axial reference (*Fig. 5.3*), the cardiac axis is therefore between +60° and +90°, which is normal.

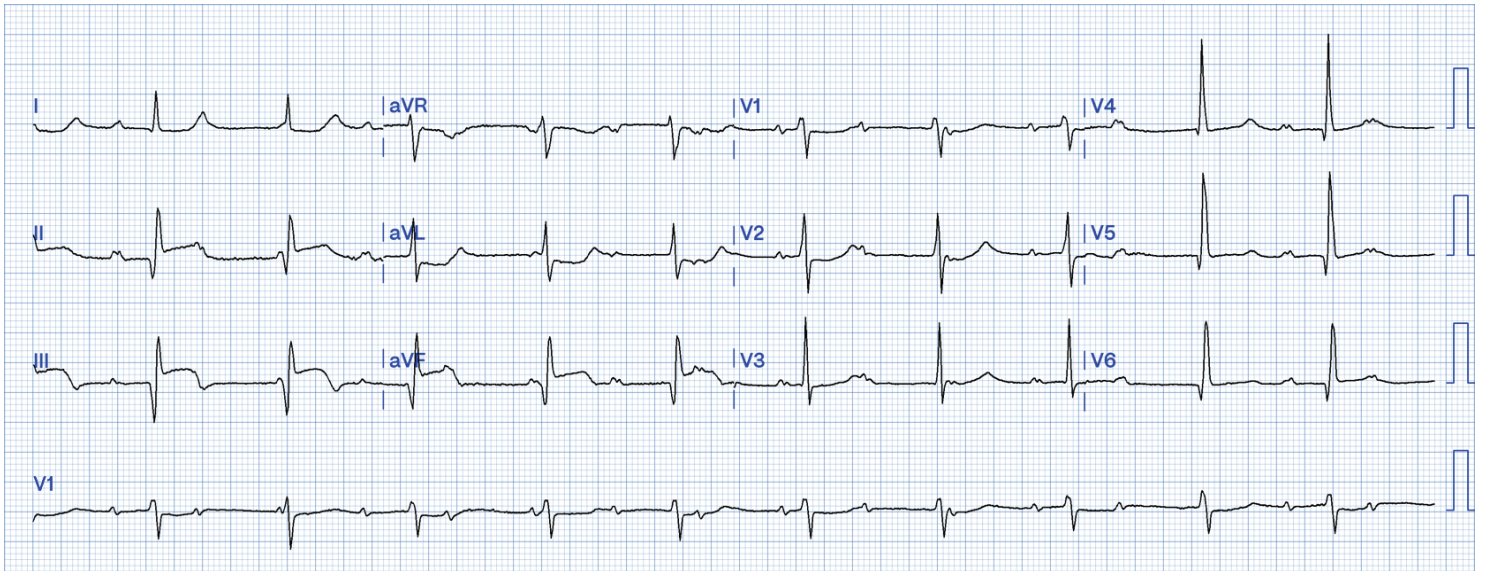

Fig. 5.19: An abnormal 12-lead ECG.

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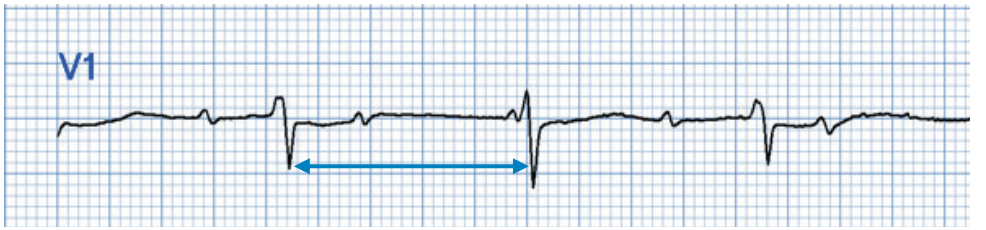

Fig. 5.20: The number of large squares (blue arrow; 4.7 large squares) between QRS complexes can be used to calculate heart rate.

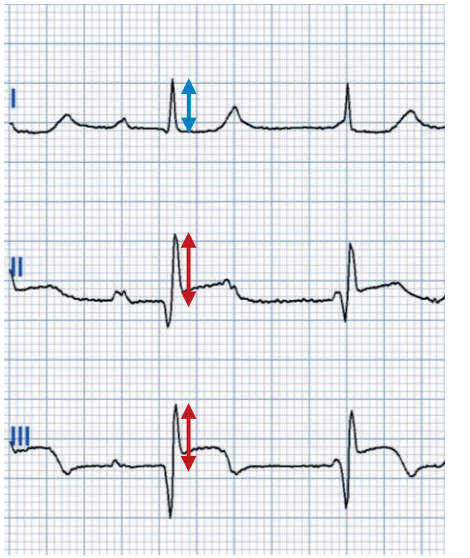

Fig. 5.22: Leads II and III contain the QRS complexes with the largest positive deflections (red arrows). The QRS complexes are smaller in the other leads (for example, in lead I represented by the blue arrow).

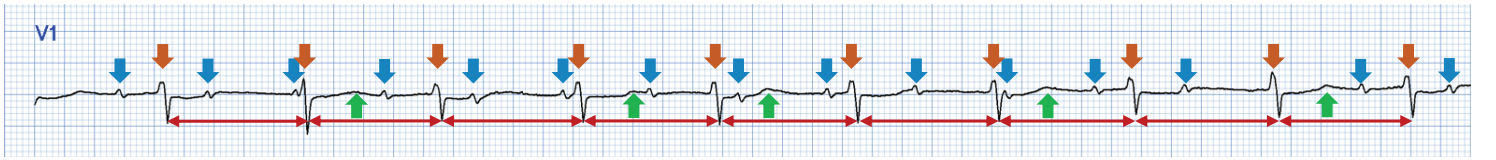

Fig. 5.21: P waves (blue arrows) and QRS complexes (orange arrows) are seen throughout, but there is no clear relationship between these. T waves (green arrows) are seen after some QRS complexes (after other QRS complexes they are not visible due to coinciding P waves). The interval between QRS complexes (red arrows) is regular.

- P waves: normal
  - explanation:
    - as highlighted above (*Fig. 5.21*), there is no clear relationship between P waves and QRS complexes. However, each P wave is less than 120 ms (3 small squares) in duration, which is normal (*Fig. 5.23*).
- PR interval: unable to comment
  - explanation:
    - unable to measure as no consistent relationship between P waves and QRS complexes
- QRS complexes: normal duration
  - explanation:
    - a normal QRS complex duration is less than 120 ms (3 small squares)
    - in this case, it is 80 ms (2 small squares) (*Fig 5.24*).

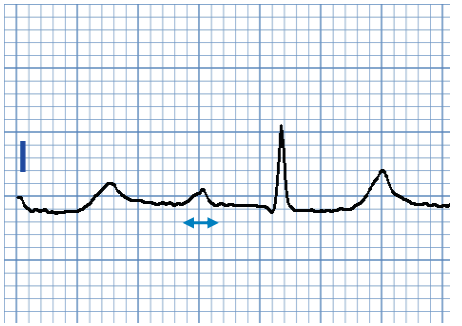

Fig. 5.23: The P wave is less than 3 small squares wide (blue arrow).

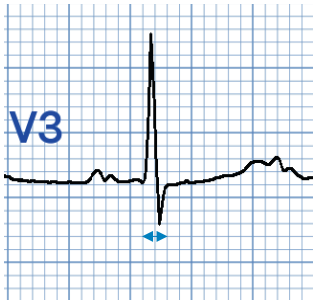

Fig. 5.24: The QRS complex is 2 small squares wide (blue arrow).

- QTc interval: normal
  - explanation:
    - the QTc can be calculated using Bazett's formula:

$$QT_cB = \frac{QT}{\sqrt{RR}}$$

where QTcB = corrected QT interval; QT = measured QT interval (in ms); RR = the interval between subsequent R waves (in s)

- in this example, QT = 440 ms; RR = 0.92 s (*Fig. 5.25*)
- Therefore, QTc = 440 / √ 0.92 = 459 ms.

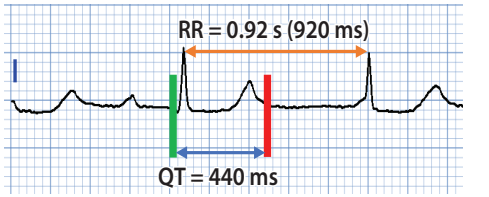

Fig. 5.25: The QT is measured from the start of the QRS complex (green line) to the end of the T wave (red line); in this case, it is 440 ms (11 small squares; blue arrow). The RR interval is 0.92 s (23 small squares).

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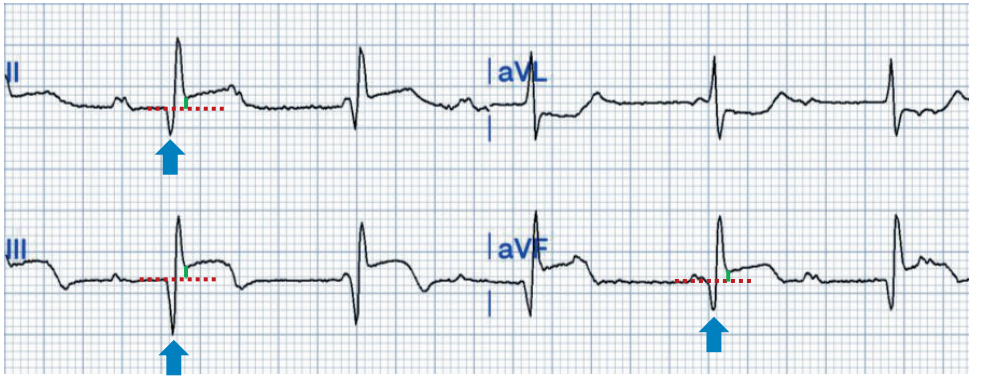

Fig. 5.26: ST elevation in leads II, II and aVF (the ST elevation is represented by the green lines above the red electric baseline). Deep Q waves (blue arrows).

- ST segments: abnormal
  - explanation:
    - ST elevation in leads II, III and aVF, with deep (pathological) Q waves (*Fig. 5.26*)
    - ST depression in leads V1, V2 and aVL (*Fig. 5.27*).
- T waves: normal
  - explanation:
    - the T waves are positive (*Fig. 5.28*).

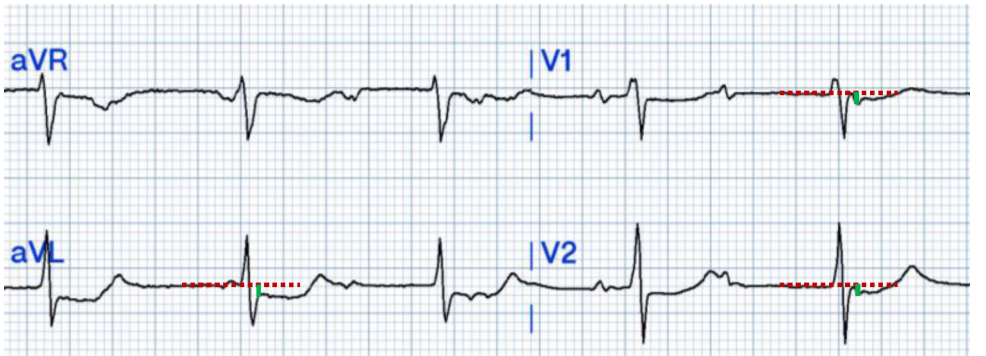

Fig. 5.27: ST depression in leads aVL, V1 and V2 (the ST depression is represented by the green lines below the red isoelectric baseline).

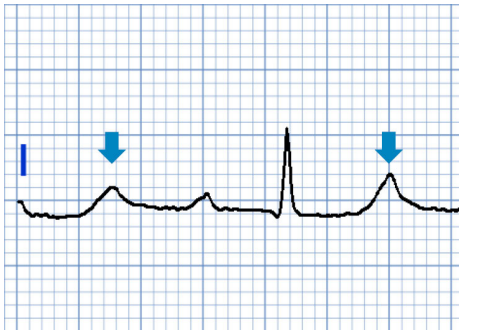

Fig. 5.28: Example of positive T waves in lead I (blue arrows).