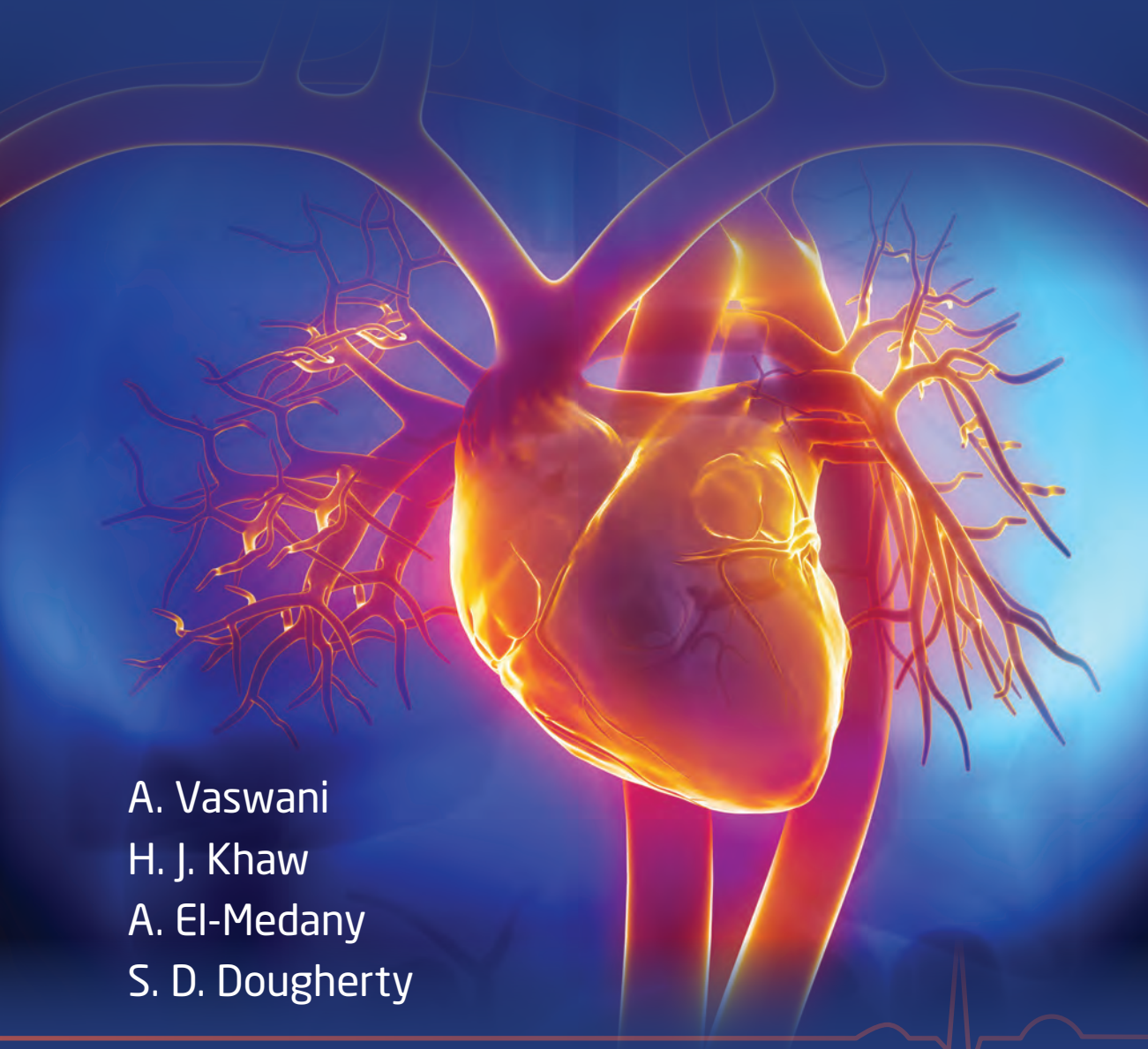
SECOND EDITION

Cardiology

in a Heartbeat



A. Vaswani
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Cardiology

in a Heartbeat

Dedicated to our patients;
And to the students about to serve them

SECOND EDITION

Cardiology in a Heartbeat

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Preface to the Second Edition

We would like to thank everyone for their overwhelming support and love for the first edition of *Cardiology in a Heartbeat*.

The rapidity with which the field has progressed, with new evidence and randomised controlled trials being added weekly, has only intensified in the years following our first edition.

This is a scary proposition for many students, and we remember being similarly overwhelmed when we were students.

In the first edition of *Cardiology in a Heartbeat* we based many of the topics around tutorials, and the demand and love for the content has only strengthened the work moving forward.

To that end, we have attempted to summarise the latest RCTs and evidence into manageable formats, with key takeaways for the time-strapped medical student. We hope you find this useful in your studies this year.

The core principles of the book have not changed, and rest on three key pillars:

1. **To our knowledge, there was huge demand for information presented in this format**
   We are very passionate about ensuring that students learn the material in an enjoyable way. Personally, we love the field because it combines sound physiological principles with practical hands-on intervention.
2. **We want to encourage students to pursue a career in cardiovascular medicine**
   Cardiovascular disease is the number one killer in the developed world. More than seven million people die from heart attacks each year. In addition to having a good grasp of cardiology (because of the sheer number of patients affected), we want to encourage the best and brightest to enter this field to develop novel therapies and conduct what will hopefully be ground-breaking research. In some small way, we hope that our book makes cardiology that little bit more attractive to study and motivates students to consider the field.
3. **Giving back**
   We joined medicine to help others, and to give back. In addition to encouraging the next generation of medical students, we also wanted to help raise funds for Vaccinaid (www.vaccinaid.org) to help against the ongoing COVID-19 pandemic, and the fallout expected thereafter. We encourage you to care for yourselves, as you in turn care for others during this time.

Whence you are called to sacrifice;
In your life and in your art,
Though trouble and toil may pile on high;
Serve with all your heart

To that end, we wish you the very best in your clinical rotations and we hope this text serves as a useful tool that allows you to excel in cardiology, gain a greater appreciation for the subject and serve your patients to the best of your ability.

A.V., H.J.K., S.D., A. El-M.

Foreword to the First Edition

Cardiology in a Heartbeat is a remarkable and impressive achievement. It is remarkable by any standard for the clarity of its presentation, superb illustration and succinct summaries with key points highlighting the essentials of cardiology.

It is designed to fulfil the needs of medical students across a spectrum, with highlighted 'pro-tip' boxes for those who want to know more and tackle more in-depth examination questions. The key information is also linked to the latest guideline recommendations – an important feature as questions in the medical school curriculum tend to test knowledge on these essential points.

Furthermore, *Cardiology in a Heartbeat* is designed and written for medical students primarily by medical students – encompassing a major collaborative effort between twenty medical student chapter contributors and eighteen senior clinicians and illustrators with excellent structure and editorial oversight.

I believe that this publication will succeed in the electronic era because there is still a need for a summary of the essentials of cardiology for modern medical students. This is the era of information overload, and web searches do not necessarily help a student keep perspective as to what is truly relevant.

One might think of this book as the equivalent of an amalgam of notes of the very best students – it is certainly better than the handouts of some lecturers!

Keith A. A. Fox
Past President British Cardiovascular Society
Chair: European Society of Cardiology
Congress Programme Committee 2012–14
Professor of Cardiology (Emeritus), University of Edinburgh
August 2015

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The authors would like to extend their thanks to our team of students and physicians for their hard work and dedication in bringing this project to life. We are also grateful to Mr Vipin Zamvar and Professor Chim Lang for painstakingly looking through the entire book for us. We would also like to extend our gratitude to Dr Nicholas Mills, Dr Neal Uren and Professor Keith Fox for their advice and direction.

We would also like to thank Dr Jonathan Ray, Mr Simon Watkins and Ms Clare Boomer at Scion Publishing for their continued support and belief in the project, as well as their guidance during the publishing process.

We are also indebted to the students who have kindly provided us with valuable feedback over the last year, and this has no doubt tremendously improved the finished article. Special thanks go to all of the following: Trishan Bali, Clare Boyle, Fraser Brown, Marcus Cabrera-Dandy, Stephanie Callaghan, Wei-Yee Chan, Ben Dallyn, Naomi Foster, Giles Goatly, Katherine Hurndall, Anna Kane, Jane Lim, Prasanna Partha Sarathy, Henry Roscoe, Sushant Saluja, Sandip Samanta, Alex Scott, Nick Smith, Rupert Smith, Charan Thandi, Hannah Theobald, Daniah Thomas, Sayinthen Vivekanantham, David Walker, Rachel Wamboldt and Philip Wright.

Last, but certainly not least, the authors wish to especially thank their families, friends, and long-suffering better halves for their unconditional support and encouragement throughout the writing process.

List of Abbreviations

AAA	Abdominal aortic aneurysm
ABC	Airway, breathing, circulation
ABPI	Ankle–brachial pressure index
ABPM	Ambulatory blood pressure monitoring
ACE	Angiotensin-converting enzyme
ACEi	ACE inhibitor
ACS	Acute coronary syndrome
AD	Aortic dissection
ADH	Antidiuretic hormone
ADP	Adenosine diphosphate
AF	Atrial fibrillation
AKI	Acute kidney injury
ALS	Advanced life support
ANCA	Anti-neutrophil cytoplasmic antibody
ANP	Atrial natriuretic peptide
AP	Action potential
AR	Aortic regurgitation
ARB	Angiotensin receptor blocker
ARDS	Acute respiratory distress syndrome
ARNI	Angiotensin receptor-neprilysin inhibitor
ARVC	Arrhythmogenic right ventricular cardiomyopathy
AS	Aortic stenosis
ASCVD	Atherosclerotic cardiovascular disease
ASD	Atrial septal defect
ATP	Adenosine triphosphate
AV	Atrioventricular
AVNRT	Atrioventricular nodal re-entrant tachycardia
AVR	Aortic valve replacement
AVRT	Atrioventricular re-entrant tachycardia
AVSD	Atrioventricular septal defect
AXR	Abdominal X-ray
BNP	Brain natriuretic peptide
BP	Blood pressure
bpm	Beats per min
BT shunt	Blalock–Taussig shunt
Ca^{2+}	Calcium
CABG	Coronary artery bypass graft
CAD	Coronary artery disease
CBP	Clinic blood pressure
CCB	Calcium channel blocker
CHD	Congenital heart disease
CHF	Congestive heart failure
CK	Creatine kinase
CLI	Critical limb ischaemia
CMRI	Cardiac magnetic resonance imaging
CMV	Cytomegalovirus
CNS	Central nervous system
CO	Cardiac output
COPD	Chronic obstructive pulmonary disease
COX	Cyclo-oxygenase
CPR	Cardiopulmonary resuscitation
CRP	C-reactive protein
CRT	Cardiac resynchronisation therapy
CT	Computerised tomography
CV	Cardioversion
CVD	Cardiovascular disease
CXR	Chest X-ray
DC	Direct current
DCM	Dilated cardiomyopathy
DHP	Dihydropyridine
DVT	Deep vein thrombosis
EBV	Epstein–Barr virus
ECG	Electrocardiogram
EDV	End diastolic volume
EF	Ejection fraction
EH	Essential hypertension
ESM	Ejection systolic murmur
ESR	Erythrocyte sedimentation rate
ESV	End systolic volume
EUCVS	Edinburgh University Cardiovascular Society
EVAR	Endovascular aneurysm repair
FAST scan	Focused Assessment with Ultrasonography in Trauma
FBC	Full blood count
GAS	Group A streptococcal
GCA	Giant cell arteritis
GI	Gastrointestinal
GPI	Glycoprotein IIb/IIIa inhibitor
GRACE	Global Registry of Acute Coronary Events
GTN	Glyceryl trinitrate
GU	Genito-urinary
HBPM	Home blood pressure monitoring

HCM	Hypertrophic cardiomyopathy
HDL	High density lipoprotein
HF	Heart failure
HIT	Heparin-induced thrombocytopenia
HIV	Human immunodeficiency virus
HLA	Human leukocyte antigen
HMG CoA	3-hydroxy-3-methylglutaryl coenzyme A
HOCM	Hypertrophic obstructive cardiomyopathy
HR	Heart rate
HRT	Hormone replacement therapy
HTN	Hypertension
IC	Intermittent claudication
ICD	Implantable cardioverter defibrillator
ICE	Ideas, concerns and expectations
IE	Infective endocarditis
IHD	Ischaemic heart disease
IM	Intramuscular
INR	International normalised ratio
IV	Intravenous
IVC	Inferior vena cava
IVDU	Intravenous drug user
JVP	Jugular venous pressure
LAA	Left atrial appendage
LAD	Left anterior descending artery
LBBB	Left bundle branch block
LCX	Left circumflex artery
LDH	Lactate dehydrogenase
LDL	Low density lipoprotein
LFT	Liver function test
LHF	Left heart failure
LMWH	Low molecular weight heparin
LV	Left ventricle
LVAD	Left ventricular assist device
LVF	Left ventricular failure
LVH	Left ventricular hypertrophy
LVOT	Left ventricular outflow tract
MAP	Mean arterial pressure
MC&S	Microscopy, culture and sensitivity
MI	Myocardial infarction
MO	Marginal obtuse
MPA	Microscopic polyangiitis
MPS	Myocardial perfusion scanning
MR	Mitral regurgitation
MRI	Magnetic resonance imaging
MS	Mitral stenosis
NDHP	Non-dihydropyridine
NICE	National Institute for Health and Care Excellence
NO	Nitric oxide
NOAC	Non-vitamin K antagonist oral anticoagulant
NSAIDs	Non-steroidal anti-inflammatory drugs
NSTEMI	Non-ST elevation myocardial infarction
PAD	Peripheral arterial disease
PAN	Polyarteritis nodosa
PCI	Percutaneous coronary intervention
PCR	Polymerase chain reaction
PCSK-9	Proprotein convertase/subtilisin/kexin type 9
PDA	Patent ductus arteriosus
PE	Pulmonary embolism
PEA	Pulseless electrical activity
PMBC	Percutaneous mitral balloon commissurotomy
PND	Paroxysmal nocturnal dyspnoea
PPI	Proton pump inhibitor
PPM	Permanent pacemaker
PVC	Premature ventricular contraction
PVD	Peripheral vascular disease
PVR	Pulmonary vascular resistance
RAAS	Renin–angiotensin–aldosterone system
RAD	Right anterior descending artery
RBBB	Right bundle branch block
RCA	Right coronary artery
RHF	Right heart failure
RVH	Right ventricular hypertrophy
SA	Sinoatrial
SBP	Systolic blood pressure
SC	Subcutaneous
SGLT-2	Sodium glucose cotransporter 2
SL	Sublingual
SLE	Systemic lupus erythematosus
SNS	Sympathetic nervous system
SOB	Shortness of breath
SR	Sarcoplasmic reticulum
STEMI	ST elevation myocardial infarction
SV	Stroke volume
SVC	Superior vena cava
SVR	Systemic vascular resistance
SVT	Supraventricular tachycardia
TAVI	Transcatheter aortic valve implantation
TB	Tuberculosis
TCA	Tricyclic antidepressant
TFT	Thyroid function test
TGA	Transposition of the great arteries

TIA	Transient ischaemic attack
TOE	Transoesophageal echocardiography
TSH	Thyroid-stimulating hormone
TTE	Transthoracic echocardiography
TVR	Transcatheter valve replacement
TWI	T-wave inversion
U&Es	Urea and electrolytes
UA	Unstable angina
UFH	Unfractionated heparin
URL	Upper reference limit
USS	Ultrasound scan
VF	Ventricular fibrillation
VSD	Ventricular septal defect
VT	Ventricular tachycardia
VTE	Venous thromboembolism
VV	Varicose veins
WCC	White cell count
WHO	World Health Organization
WPW	Wolff–Parkinson–White syndrome

How to Use This Book

This book aims to build upon relevant concepts from the pre-clinical years and bring you up to speed on information that will be particularly useful to you in clinical cardiology. It will incorporate basic anatomy, physiology and biochemistry, bridging the gap between theoretical and applied science in the initial chapters, and introduce you to clinical principles once the basics have been consolidated.

The book is divided into concept and clinical sections. If this is your first time approaching cardiology as a subject, or if you need your knowledge refreshed, this book is best read from cover to cover, as concepts discussed in the first half of the book will build on one another as you progress through each chapter.

Some chapters are particularly synergistic, e.g. *Chapter 4: The Electrocardiogram* and *Chapter 11: Arrhythmias*. Alternatively, if you're using this book as a revision tool, or if you've already been through the material once already, you may simply decide to jump into whichever chapter you're ready to begin revising.

Clinical chapters are arranged in a concise manner, focusing on definitions, aetiology, epidemiology, pathophysiological principles, key features and then an emphasis on investigation and management, which have been arranged in a step-wise fashion to help you decide what to do next.

We have also added in 'Pro-tip' boxes, which provide you with that little bit of extra knowledge, 'Exam Essential' boxes, which give you an idea of what you *must* know, 'Why?' boxes, which explain the pathophysiology and rationale behind certain decisions and processes, and last but not least, guidelines in red summarising the relevant information in a bite-sized snippet.

We have also added 'In a Heartbeat' summaries at the beginning of each chapter to help you consolidate your knowledge. We trust that you will accomplish much with what little we have provided you, and that your patients' needs will continue to remain at the forefront of your minds.

"A physician's understanding of physiology has much the same relation to his power of healing as a cleric's divinity has to his power to influence change in conduct."

Samuel Butler

"The life so short; the craft so long to learn."

Geoffrey Chaucer
cf. Hippocrates, *Aphorisms*

Chapter 11
Arrhythmias

by H.J. Khaw and C. Lang

11.1 Introduction

Arrhythmias are disorders of heart rhythm, representing some of the most common maladies seen in cardiovascular medicine. The majority of these conditions are benign, but there are important life-threatening arrhythmias to be aware of.

Arrhythmias *In A Heartbeat*

- Check the rate of the ECG – bradycardia (<60 bpm) or tachycardia (>100 bpm)
- Bradycardia – sinus bradycardia or heart block
- Tachycardia – narrow complex (QRS <0.12 seconds) or broad complex (QRS >0.12 seconds)
  - narrow complex – SVTs
  - Broad complex should be considered to be VT/VF until proven otherwise
- Extra beats or missing beats

How do arrhythmias arise?

As described in *Chapter 1*, the conducting system of the heart is highly specialised, and electrical impulses are produced by the heart's intrinsic pacemaker cells. When the conducting system is functioning as it should, each heartbeat is generated in a regular pattern and this is known as sinus rhythm. Arrhythmias arise as a result of problems in the conducting system and are broadly caused by:

1. Altered production
   - **Automaticity** – the ability of a cell to spontaneously depolarise and generate an action potential. In a healthy heart, only pacemaker cells possess this natural automaticity. It is important to note that although the SA node is the native pacemaker of the heart, the AV node and His-Purkinje system may also function as a pacemaker if necessary. The autonomic nervous system has significant control over SA node automaticity, hence altered sympathetic and parasympathetic activity will affect the heart rate (refer to *Chapter 1*).
     - **enhanced automaticity –** up-regulation of SA node activity most commonly by sympathomimetic drugs results in a tachyarrhythmia. Normal physiological sympathetic activation such as during stress and exercise may also result in increased automaticity.
     - **reduced automaticity –** down-regulation of SA node either by the activation of the parasympathetic nervous system or suppression of the sympathetic system results in bradyarrhythmias, the most common cause of which is beta-blockers.

// EXAM ESSENTIALS //

Some drugs, e.g. beta-blockers, anti-arrhythmics and anti-cholinergics in particular, are important causes of arrhythmias.

- **Triggered activity** – occurs as a result of membrane voltage instability. Ionic disturbances, specifically calcium and sodium ions, may alter the membrane potential of cardiac myocytes resulting in abnormal depolarisations (early or delayed) occurring after an action potential. This is known as *afterdepolarisations*. Long QT syndrome and ventricular arrhythmias may arise due to this mechanism.

2. Altered conduction

- **Conduction block** occurs when the conducting wave of depolarisation is terminated as it is blocked by unexcitable myocardial tissue. There are two ways in which this can happen.
  - **fixed block** – the wave of conduction is inhibited by a physical barrier i.e. scarred tissue
  - **functional block** – the conducting impulse encounters refractory tissue, preventing further wave propagation. This is often caused by drugs that prolong the action potential of myocytes (class III anti-arrhythmics e.g. amiodarone)
- **Re-entry** occurs when the conducting impulse travels around an abnormal re-entrant loop circuit. In order for re-entry to happen, there must be two distinct conduction pathways with different conduction velocities. This can occur as a large anatomical circuit as seen in Wolff–Parkinson–White syndrome via the normal conduction system and the accessory pathway, or as smaller circuits in the AV node and spiral micro-circuits seen in atrial fibrillation. The mechanism of re-entry is shown in *Figure 11.1.*

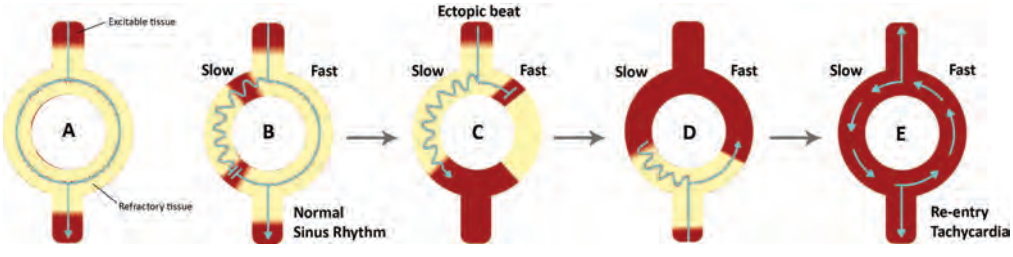

Figure 11.1 – Mechanism of re-entry.

A. In a normal heart, the wave of depolarisation travels down both pathways at similar velocities. They eventually meet and the resultant impulse is propagated distally.

B. When there are two pathways with different conduction velocities, the time taken for the wave of depolarisation to travel down the slow pathway is longer than that of the fast pathway. This may allow retrograde transmission of impulses of the fast pathway up the slow pathway. However, when this retrograde impulse encounters refractory tissue of the slow pathway, both conduction waves are terminated. As a result, only impulses travelling down the fast pathway are propagated distally. This process is known as *unidirectional block*.

C. When an ectopic beat reaches the circuit while the fast pathway is still in its refractory period, the wave of depolarisation is only allowed to travel down the slow pathway.

// WHY? //

The **refractory period of the slow pathway is shorter than that of the fast pathway**. This is a fundamental feature in the mechanism of re-entry.

D. As the impulse reaches the distal end of the circuit, the fast pathway has repolarised, allowing retrograde propagation of the impulse. *Re-entry* has now occurred.

E. The conduction wave may then loop around the circuit, initiating a tachycardia as the impulse is propagated distally after each loop. This re-entrant circuit may continue indefinitely until it is interrupted by a change in electrical depolarisation.

Investigations

A **12-lead electrocardiogram** should be performed in all patients with suspected arrhythmias (refer to *Chapter 4*). Further investigations may be useful in confirming the diagnosis and identifying the underlying cause.

- Blood test: **FBC** (anaemia may cause a sinus tachycardia), **U&Es** (hyperkalaemia and hypercalcaemia may predispose to arrhythmias), **TFTs** (hyperthyroidism is an important cause of atrial fibrillation and other tachyarrhythmias)
- Ambulatory 24-hour Holter recording: may be useful in patients with frequent but transient episodes
- Echocardiography: performed when there is evidence of ischaemic or structural heart disease
- Electrophysiology (EP) studies: performed in two groups of patients:
  - patients with paroxysmal episodes suitable for ablation therapy
  - high-risk patients with disabling symptoms and evidence of ischaemic heart disease.

Therapeutic modalities

The management of patients with arrhythmias can be fairly complex. Pharmacological modalities, particularly anti-arrhythmic drugs, were once the mainstay treatment for arrhythmias but their use has declined since the advent of percutaneous interventional electrophysiological procedures. However, anti-arrhythmic drugs are still used in clinical practice, mainly as an adjunct to other therapies. Pharmacological therapy of arrhythmias has been discussed in *Chapter 5*.

Devices *In A Heartbeat*

	Implantable cardioverter defibrillator (ICD)	**Permanent pacemaker (PPM)**	**Cardiac resynchronisation therapy (CRT)**
Purpose	Delivers overdrive pacing and/or DC shocks to restore sinus rhythm	Prevent bradycardia	Co-ordinate and synchronise atrial, RV and LV contractions
Indications	Used to treat ventricular tachyarrhythmias	Sinus node disease and AV node disease	Heart failure
Variation	Single chamber (RV) Dual chamber (RA and RV)	Single chamber (RA or RV) Dual chamber (RA and RV)	CRT pacemaker CRT ICDs for patients at high risk of ventricular arrhythmias

Interventional electrophysiology

Electrical therapy for arrhythmias involves delivering low-voltage electrical impulses to stimulate cardiac myocyte depolarisation, particularly in the case of bradycardias (**pacing**) or higher voltage shocks to globally and transiently depolarise the heart, resetting its rhythm, in particular during life-threatening tachyarrhythmias (**cardioversion**).

Pacing. There are two forms of pacing that are usually performed for the treatment of bradycardias.

- *Temporary pacing* is used in the acute setting to stabilise patients who are haemodynamically unstable.
- *Permanent pacemaker (PPM)*
  - sub-dermal implantation of a pulse generator and a battery, most commonly inferior to the clavicle as well as placement of pacing electrodes either in the right atrium or right ventricle (*single chamber*) or both (*dual chamber*)
  - possesses 'sense' and 'pace' functions that detect the cardiac rhythm and only delivers electrical pulses when necessary
  - common complications include lead displacement, wound infection and pneumothorax.

// PRO-TIP //

Temporary pacing can be performed in one of two ways:

- **Trans-thoracic external pacemaker**
  - delivered through large gel electrodes over the chest wall
  - rapid administration may be uncomfortable, as high currents are required to penetrate the thoracic wall
- **Trans-venous pacing**
  - percutaneous catheter insertion via the internal jugular, subclavian or femoral vein into the heart. The pacing wire is usually placed in the right ventricle.
  - more complex procedure but can be used for longer periods (few days)

GUIDELINES: Indications for permanent pacemaker insertion (ESC, 2021)

Pacing required	Pacing may be considered	Pacing not required
Symptomatic sinus node dysfunction or AV block	Symptoms likely related to sinus node dysfunction or AV block	Asymptomatic sinus node dysfunction
Asymptomatic Mobitz II and complete heart block	Recurrent unexplained syncope	Acquired sinus node dysfunction or AV block due to reversible causes

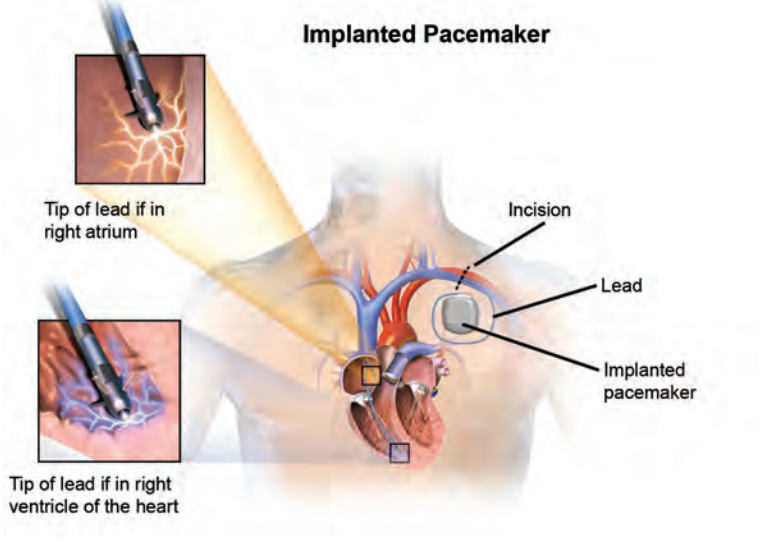

Figure 11.2 – Dual chamber permanent pacemaker and the location of its pacing electrodes.

Cardioversion *(also known as* ***defibrillation****).* The aim of cardioversion is to restore normal sinus rhythm. Electrical cardioversion involves the delivery of a high voltage direct current (DC) biphasic shock to completely depolarise the heart, terminating the tachyarrhythmia.

// WHY? //

A biphasic shock is used to minimise the total amount of shock delivered to the patient. The polarity of the shock is reversed (i.e. positive to negative or vice versa) halfway through. This effectively halves the energy needed.

// EXAM ESSENTIALS //

In patients with organised ventricular rhythms (e.g. VT) or patients with AF or flutter, it is important to synchronise the shock with the early part of the QRS complex. This is because delivery of a shock during the T wave on the surface ECG might result in initiation of ventricular fibrillation.

Cardioversion is primarily used in the treatment of ventricular arrhythmias, atrial fibrillation or atrial flutter. There are two types of cardioversion, electrical and chemical (pharmacological) cardioversion.

- *Electrical (external) cardioversion* – also known as DC cardioversion
- *Chemical (internal) cardioversion* – involves the use of intravenous anti-arrhythmic drugs (e.g. amiodarone, procainamide) and may be indicated in patients with AF and atrial flutter.

Implantable cardioverter defibrillators (ICDs)

- ICDs are very similar to permanent pacemakers and as such, they detect life-threatening tachyarrhythmias and deliver electrical shocks to terminate them
- It is important to note that in addition to their defibrillation function, ICDs also have pacing functions
- They are used in both primary and secondary prevention of sudden cardiac death from ventricular arrhythmias (see below).

GUIDELINES: Indications for implantable cardioverter defibrillators (ESC, 2015)

Secondary prevention in patients with:

- Documented VF or haemodynamically unstable VT in the absence of reversible causes or more than 48 hours after myocardial infarction who are receiving chronic optimal medical therapy and have a reasonable expectation of survival with a good functional status >1 year
- Recurrent sustained VT (more than 48 hours after myocardial infarction) who are receiving chronic optimal medical therapy, have a normal LVEF and have a reasonable expectation of survival with good functional status for >1 year.

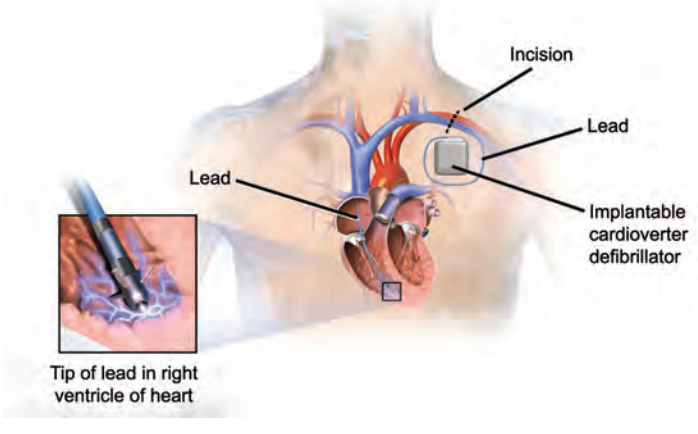

Figure 11.3 – Implantable cardioverter defibrillator.

Primary prevention of sudden cardiac death (SCD) in patients with:

- Symptomatic HF (NYHA class II–III) and LVEF ≤35% after ≥3 months of optimal medical therapy who are expected to survive for at least 1 year with good functional status (both ischaemic and non-ischaemic aetiology)
- Prior MI (>6 weeks prior) and LVEF <35%
- Hypertrophic cardiomyopathy and an estimated 5-year risk of sudden death ≥6% and a life expectancy >1 year following detailed clinical assessment that takes into account the lifelong risk of complications and the impact of an ICD on lifestyle, socioeconomic status and psychological health
- Congenital long QT syndrome with recurrent symptoms despite optimal beta blockade
- High risk of SCD with Brugada syndrome, ARVC and cathecholaminergic polymorphic VT.

GUIDELINES: Implantable cardioverter defibrillators for arrhythmias (NICE, 2014)

NICE recommends ICDs as **primary** prevention for patients with:

- Strong family history of a heart condition with a high risk of sudden death (e.g. long QT syndrome, Brugada syndrome, etc.)
- Congenital heart disease that has been repaired surgically.

NICE also recommends **secondary** prevention for patients who:

- Survived a cardiac arrest caused by a ventricular arrhythmia
- Have spontaneous sustained VT causing syncope or severe haemodynamic instability
- Have sustained VT without syncope and a LVEF of less than 35%.

Radiofrequency catheter ablation

This technique involves the utilisation of radiofrequency waves to heat and ablate the focus/foci of enhanced automaticity or re-entrant circuits causing tachyarrhythmias. EP studies are usually performed prior to the procedure to localise the abnormal tissue.

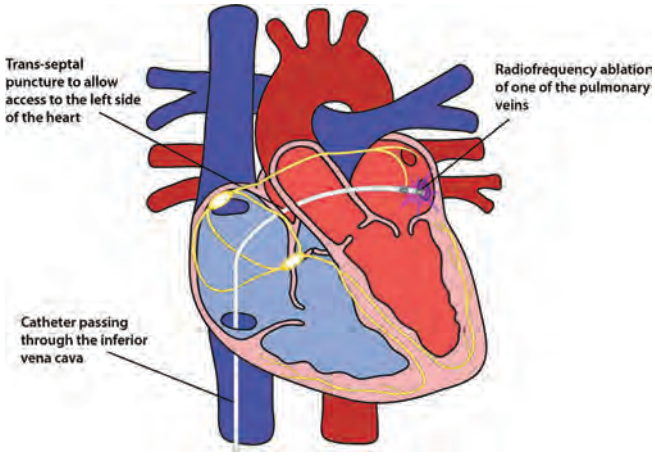

Figure 11.4 – Radiofrequency ablation.

Catheter ablation has become the definitive treatment for patients with SVTs, in particular atrioventricular nodal re-entrant, atrioventricular re-entrant tachycardias and atrial flutter. Cure rates of over 90% are expected in such patients.

11.2 Systematic approach to ECG rhythm abnormalities

As discussed in *Chapter 4*, an ECG can be interpreted in a few easy steps. Similarly, arrhythmias can be systematically approached with the following algorithm:

1. First, take a look at the **heart rate** – determine whether it is a **bradycardia** (<60 bpm) or a tachycardia (>100 bpm)
2. If it is a **bradycardia**,
   - and asymptomatic – it will most likely be **sinus bradycardia** but heart block should be ruled out
   - and symptomatic/haemodynamically unstable – **atrioventricular (AV) block** should be suspected and managed appropriately
3. If it is a **tachycardia**, determine whether it is a **narrow complex** (QRS <0.12 s) or a **broad complex** (QRS >0.12 s)
   - **Narrow complex tachycardias** are supraventricular in origin i.e. **SVT** and can be distinguished from one another based on their specific ECG patterns (e.g. saw tooth in atrial flutter)
   - All **broad complex tachycardias** should be considered ventricular in origin (**VT** or **VF**) until proven otherwise as these patients are commonly haemodynamically unstable. It is important to treat broad complex tachycardia as VT because treatments for SVT could make a patient with VT become haemodynamically unstable.
   - An **SVT with aberrant conduction** (e.g. atrial fibrillation with a bundle branch block) can also present with a broad complex tachycardia appearance. *As a very general rule of thumb*:
     - *Regular* rhythms are likely to be **VT**
     - *Irregular* rhythms are more likely to be **AF with aberrant conduction**
4. Check to see if there are any *Extra Beats* or *Missing Beats*

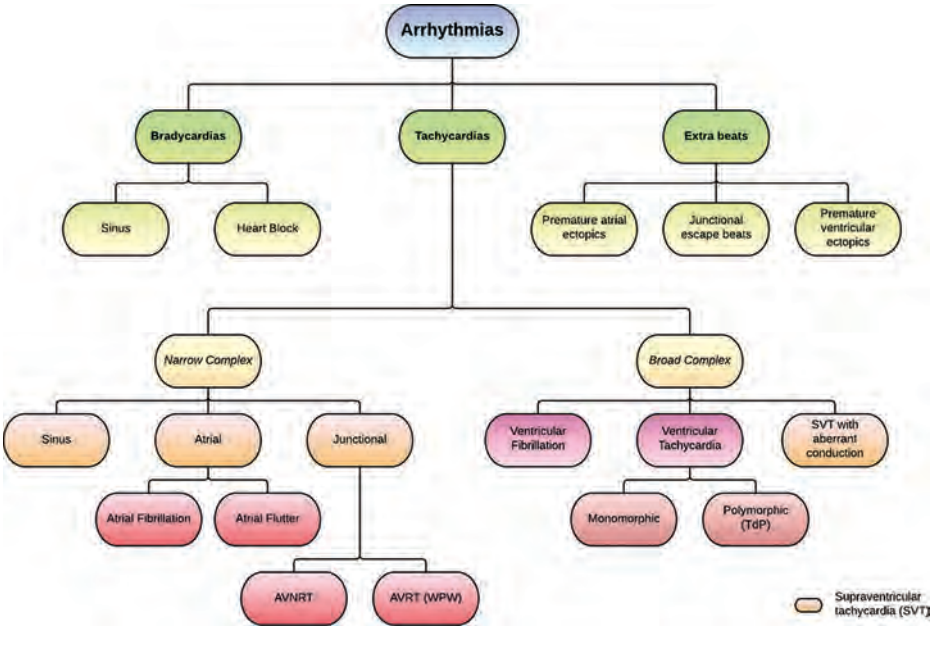

Figure 11.5 – Schematic representation of arrhythmias.

Extra beats
- **Premature atrial ectopic** (early conducted P wave before QRS)
- **Premature ventricular ectopic** (no P wave before QRS)
- **Escape beats** (no P wave before QRS but normally after SA pause)

Missing beats
- **SA pause** (no preceding P wave)
- **Non-conducted premature atrial contraction** (P wave conducted too early with no subsequent QRS)
- **2nd-degree heart block** (normal P wave conducted but no subsequent QRS)

11.3 Bradycardias

Bradyarrhythmias are disorders of slow heart rhythm. They occur as a result of reduced impulse production (e.g. sinus bradycardia) or failure of impulse propagation due to failure of the conducting system (e.g. atrioventricular blocks or bundle branch blocks). Each of these conditions will be discussed further in the sections below.

11.3.1 Sinus bradycardia

Definition
A decrease in the heart rate to less than 60 beats per minute in adults. Normal sinus rhythm is observed.

Epidemiology
- Affects 20–25% of under 25 year olds.

Aetiology
Common causes:
- Physiological – during sleep, athletes, young people
- Cardiac – inferior myocardial infarction, sick sinus syndrome (see below)

// WHY? //

The SA nodal artery is a branch of the right coronary artery (RCA) in 90% of the population. In an inferior myocardial infarction, the RCA could be involved, and blood flow to the SA node is also affected, leading to bradycardia.

- Drugs – beta-blockers, calcium channel blockers, digoxin, opiates.

Other causes:
- Metabolic – hypothyroidism, hyperkalaemia, hypothermia
- Neurological – brain stem pathology (e.g. raised intracranial pressure, infarction)

// PRO-TIP //

Increased intracranial pressure (from a haemorrhage or hydrocephalus) causes an increase in blood pressure to maintain cerebral perfusion. However, aortic baroreceptors are stimulated in response to the increased blood pressure and in turn, trigger a parasympathetic response. In addition, vagal compression as a result of the increased intracranial pressure further activates this response and ultimately induces bradycardia. This is known as the 'Cushing's reflex'. This is a sign of impending brain herniation and death.

Pathophysiology

In **intrinsic** SA node disease, the automaticity of the node is depressed due to ageing or any disease that affects the atrium such as ischaemic heart disease and cardiomyopathy. On the other hand, **extrinsic** factors such as drugs and metabolic imbalances suppress SA node activity by reducing its automaticity via vagal activation.

Clinical features

- Symptoms range from fatigue to syncope.

ECG appearance

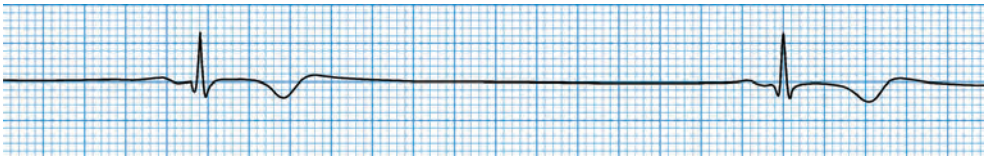

Figure 11.6 – An ECG showing an RR interval of 2.8 s (14 large boxes; 0.2 s) equivalent to a rate of 21 bpm.

Management

- Treatment not usually required for **asymptomatic patients**
- Treat underlying cause – stop offending medication
- Atropine may be indicated in acute setting in patients with severe bradycardia
- If patients are unresponsive to atropine, consider isoprenaline and temporary pacing.

11.3.2 Sinus node dysfunction (sick sinus syndrome)

Definition

A syndrome of SA nodal dysfunction that encompasses sinus bradycardia, sinus pause and sinoatrial block.

// EXAM ESSENTIALS //

Sick sinus syndrome is often associated with other SVTs, in particular atrial fibrillation and atrial flutter. When the atrial arrhythmia terminates, there is a prolonged sinus node recovery time, which can result in syncope. This combination is termed 'tachycardiac-bradycardia syndrome'.

Epidemiology

- More common in elderly patients
- Seen in 0.2% of patients over the age of 50

Aetiology

Common causes:

- **Idiopathic fibrosis** of the SA node (as a result of ageing)
- Myocardial ischaemia

Other causes:

- Infiltrative conditions – sarcoidosis, amyloidosis
- Drugs – beta-blockers, digoxin, calcium channel blockers, amiodarone
- Metabolic – hypothyroidism, hyperkalaemia
- Cardiomyopathies.

Pathophysiology

Degenerative and ischaemic changes in the SA node, nerve supply, and the surrounding atrial tissue comprise the underlying pathology of sick sinus syndrome. This will either result in abnormalities of impulse formation, impulse conduction or both. The failure to produce a sinus impulse, i.e. **sinus arrest or pause**, occurs as a result of reduced automaticity. In addition, fibrosis of the SA node and atrial myocytes may also result in conduction blocks (commonly presenting as **sinoatrial block**).

// PRO-TIP //

Fibrosis of atrial tissue contributes to the arrhythmogenicity of atrial fibrillation and atrial flutter.

Clinical features

- Chronic frequent episodes of intermittent bradycardia and tachycardia
- Palpitations
- Dizziness or syncope.

ECG appearance

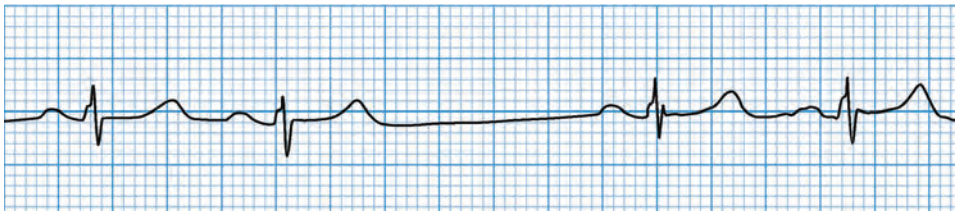

Figure 11.7 – **Sinus node exit block**. There is a missing P wave. The sinus node has 'fired' but the impulse has failed to propagate into the atrium.

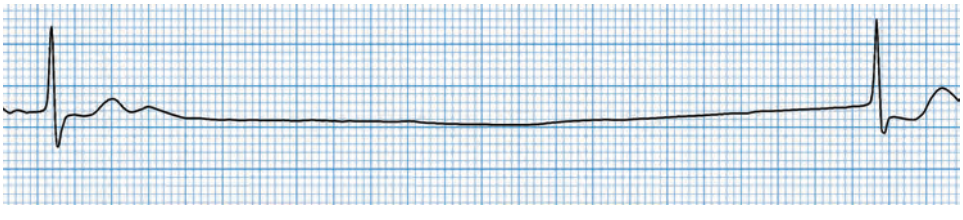

Figure 11.8 – A long **sinus pause** (>3 s) is seen as a result of SA node failure; the subsequent beat is a junctional escape.

Management

- Treat underlying cause if present
- Intravenous **atropine** may be useful in patients with severe symptoms
- **Dual chamber pacemaker** implantation recommended for patients with symptomatic chronic disease.

GUIDELINES: Dual-chamber pacemakers in sick sinus syndrome (NICE, 2014)

NICE recommends dual-chamber pacemaker implantation for all patients with symptomatic bradycardia due to sick sinus syndrome with or without the presence of an atrioventricular conduction block.

11.4 Atrioventricular conduction blocks

Atrioventricular (AV) conduction blocks refer to a disturbance in impulse conduction between the atria and ventricles. This can be permanent or transient depending on the aetiology of the block. Disturbances at different sites within the AV conduction system (AV node and the His-Purkinje system) produce AV blocks of varying severity. Classically, AV blocks have been split into three categories:

11.4.1 First-degree heart block

Definition

Delayed atrioventricular conduction resulting in a constant prolonged PR interval (>0.2 s) on ECG.

Epidemiology

- Commonly affects patients over the age of 65.

Aetiology

Common causes:

- Idiopathic degeneration (fibrosis) of the conduction system
- **Increased vagal tone** (e.g. athletes, during sleep)
- Myocardial ischaemia (RCA supplies the AV node)
- Drugs (beta-blockers, calcium channel blockers, digoxin)

Other causes:

- Myocarditis
- Metabolic disturbances (hypokalaemia, hypomagnesaemia)

Pathophysiology

First-degree heart block tends to involve the AV node itself. Structural causes such as fibrosis or damage to the AV nodal inputs will delay impulse conduction. Furthermore, the AV node is richly innervated by the autonomic nervous system. Therefore, vagal (parasympathetic) activation may also prolong AV conduction time.

Clinical features

- Usually asymptomatic.

ECG appearance

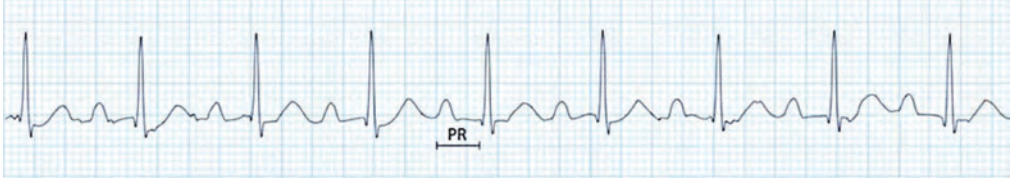

Figure 11.9 – ECG with a prolonged PR interval (240 ms) but otherwise normal.

- PR interval (>0.2 s or 5 small squares)
- Sinus rhythm (each P wave is followed by a QRS complex).

Management

- Benign condition; treatment is not usually required.

Prognosis

- Normal, although some patients will progress to higher degrees of AV block over time.

11.4.2 Second-degree heart block: Mobitz type I (also known as the Wenckebach block)

In second-degree heart block, there is intermittent failure of atrioventricular conduction resulting in occasional dropped beats. There are two forms of second-degree heart block, Mobitz Type I and Mobitz Type II. The mechanism and pathophysiology of both types are quite dissimilar and so are their clinical presentation and findings.

Definition

An atrioventricular conduction deficit resulting in progressive prolongation of the PR interval until a beat is dropped.

Epidemiology

- Occurs in 4% of post-inferior MI
- More common than Mobitz type II.

Aetiology

Common causes:

- Idiopathic fibrosis of the conduction system
- Drugs (beta-blockers, calcium channel blockers, digoxin, procainamide)
- Increased vagal tone – athletes, children, during sleep

Other causes:

- Iatrogenic – transcatheter aortic valve implantation (TAVI)
- Inferior MI
- *Other causes are similar to those of first-degree heart block.*

Pathophysiology

Mobitz type I heart block is caused by progressive conduction block, more commonly within the **AV node itself** (70%) and sometimes more distally (30%) in the conduction system. Mobitz type I can also be vagally mediated as a result of normal physiology or drugs and rarely caused by structural abnormalities. Mobitz type I differs from first-degree heart block in that there is progressive AV nodal cell **fatigue**, eventually resulting in a dropped beat.

Clinical features

- Majority of patients are asymptomatic
- May present with light-headedness, dizziness and syncope or exertional fatigue.

ECG appearance

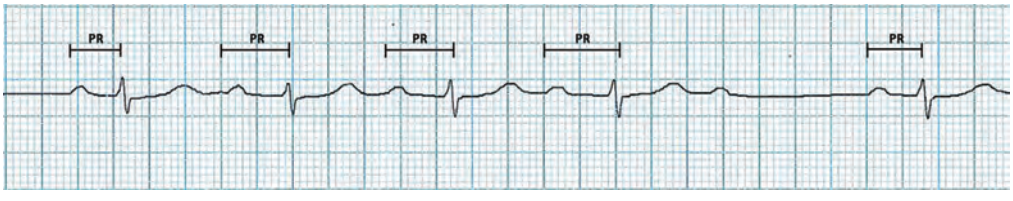

Figure 11.10 – Wenckebach phenomenon – prolongation of four preceding PR intervals before the 5th beat is dropped.

- Progressive prolongation of the PR interval until a beat is dropped
- Narrow QRS complexes
- PR interval longest before the dropped beat and shortest after.

Management
- Treatment not usually required unless symptomatic
- IV atropine may be used in emergency situations or in very severe bradycardia
- Permanent pacemaker implantation is indicated in patients with non-resolving symptomatic block. This is shown to have **mortality benefits** in patients above the age of 45.

11.4.3 Second-degree heart block: Mobitz type II – non-Wenckebach block

Definition
An atrioventricular conduction deficit resulting in intermittent dropped beats without changes in the PR interval.

Aetiology
Common causes:
- Idiopathic fibrosis of the conduction system
- Anterior MI

// WHY? //

An anteroseptal myocardial infarction may damage the His-Purkinje system as the conduction bundle lies within the septum.

Other causes:
- Drugs (beta-blockers, calcium channel blockers, digoxin)
- Infiltrative disease – haemochromatosis, sarcoidosis, amyloidosis
- *Other causes are similar to those of first-degree heart block.*

Pathophysiology
In contrast to Mobitz type I, the conduction block in Mobitz type II tends to occur infra-nodally, in the His bundles (20%) or Purkinje fibres (80%) and is more likely to be caused by structural abnormalities of the conduction system.

Clinical features
- Dizziness and syncope
- May present with haemodynamic instability and sudden cardiac death in some cases.

ECG appearance

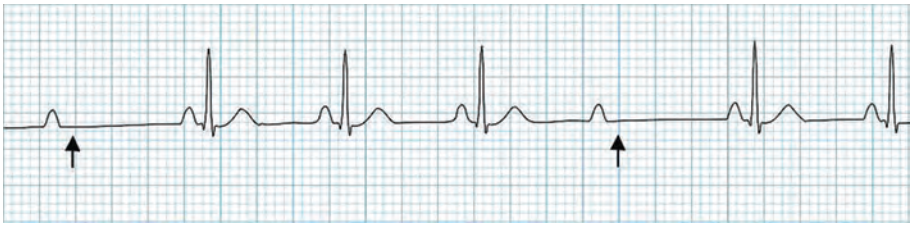

Figure 11.11 – Mobitz type II with a 3:1 block; arrows showing missing QRS complexes where the P wave is not propagated.

- Constant PR interval
- QRS complexes may be broad if the AV block occurs at the Purkinje system

// WHY? //

When the AV block occurs distally in the Purkinje system, there is often pre-existing bundle branch block. This produces the classic wide QRS complexes of bundle branch blocks as there is a delay in depolarisation of each ventricle.

- May be associated with a fixed ratio block (2:1, 3:1, etc.).

// EXAM ESSENTIALS //

A fixed ratio block may occur in both Mobitz type I and Mobitz type II. It is important to note that this is more of a descriptive term of an ECG appearance rather than a different type of second-degree heart block.

Management

- Treat haemodynamic compromise with:
  1. Intravenous atropine
  2. Intravenous adrenaline
  3. Intravenous isoprenaline infusion may stabilise rhythm in a patient with profound bradycardia
  4. If other measures fail, and permanent pacing is not immediately available consider temporary external pacing
  5. Temporary trans-venous pacing
- Permanent pacemaker implantation is indicated for all patients (even asymptomatic patients).

// PRO-TIP //

Patients with Mobitz type II heart block who have been treated with dual chamber pacemakers have been shown to have better exercise tolerance when compared with patients with single chamber pacemakers.

Prognosis

- **Commonly progresses to third degree heart block**
- PPM implantation has been shown to improve 5 year survival rates.

11.4.4 Third-degree heart block

Definition

Third-degree heart block, also known as complete heart block, refers to the complete failure of AV conduction resulting in loss of communication between the atria and ventricles, causing them to beat independently of one another.

Aetiology

Common causes:

- Idiopathic degeneration of the conduction system (ageing)
- **Anterior** and **inferior MI** – due to interruption of the blood supply to the AV node. Often resolves within 7 days
- Drugs (beta-blockers, calcium channel blockers, digoxin)

Other causes:

- Congenital – maternal systemic lupus erythematosus
- Iatrogenic – cardiac surgery, cardiac catheterisation
- *Other causes are similar to those of first-degree heart block.*

Pathophysiology

Complete failure of the AV conduction system results in a complete AV block. In this case, there is no relationship between the electrical activity in the atria and the ventricles. This is known as *AV dissociation.* Instead, latent pacemaker cells in the His-Purkinje system will resume the role of the AV node as a physiological compensatory mechanism to ensure ventricular contractions and maintain cardiac output. This escape (junctional or sub-junctional) rhythm is often slow (40 to 60 bpm).

Clinical features

- Symptoms of low cardiac output – dizziness, breathlessness, fatigue
- Stokes–Adams attacks

// EXAM ESSENTIALS //

Stokes–Adams attacks are episodes of syncope characterised by a sudden unexpected collapse, accompanied by a transient loss of consciousness (less than a minute). Patients are often described to have 'death-like' pallor with immediate flushing on awakening.

- Palpitations
- Intermittent **cannon 'A' waves** may be seen on examination. These are due to contraction of the atria at a time when the AV valves are closed, causing regurgitation of blood into the venae cavae.

// WHY? //

During examination of the JVP, cannon A waves indicate the presence of AV dissociation due to complete heart block. The intermittently prominent (cannon) A waves occur when the right atrium contracts against a closed tricuspid valve.

ECG appearance

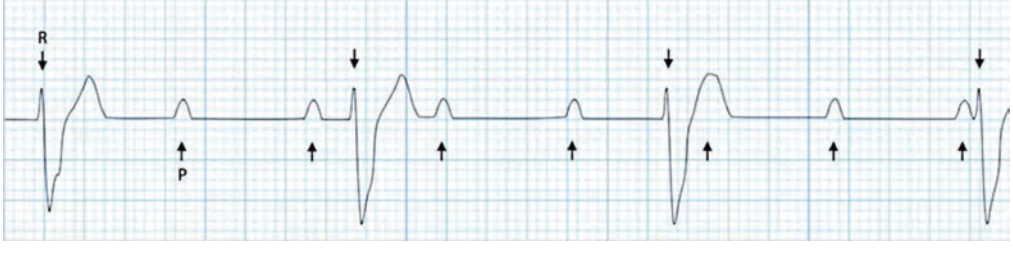

Figure 11.12 – Complete AV dissociation; atrial rate (bottom arrows) of 100 bpm and ventricular rate (top arrows) of 40 bpm.

- Rate tends to be less than 50 bpm
- Constant P–P and R–R intervals but apparent AV dissociation
- QRS complexes may be narrow (junctional escape rhythm) or wide (subjunctional escape rhythm).

Management

- Correct reversible causes
- If patient is haemodynamically compromised, emergency IV atropine may be used. However, atropine is short acting and an IV isoprenaline infusion may be more useful
- Temporary pacing may be indicated as a bridge to PPM
- Permanent pacemaker implantation is indicated for **all patients** to prevent recurrence.

Prognosis
- Patients have a 28% mortality if they develop third-degree block during an acute MI.

11.5 Bundle branch blocks

The bundle of His splits into the left and right bundle branches, and these subsequently divide into Purkinje fibres which transmit electrical impulses to ventricular myocytes. Bundle branch blocks occur as a result of interruptions to the conduction pathway along the His-Purkinje system and result in asynchronous activation of the ventricles.

11.5.1 Right bundle branch block

Definition
A conduction deficit in the His-Purkinje system resulting in a delay in right ventricular depolarisation.

Epidemiology
- Incidence increases with age.

Aetiology
Common causes:
- **Right ventricle hypertrophy**
- **Normal variant** in young fit people
- RV flow obstruction (right heart strain; pulmonary stenosis, pulmonary embolus)

Other causes:
- Ischaemic heart disease
- Iatrogenic – right heart catheterisation (5%).

Pathophysiology
Right bundle branch block (RBBB) tends to occur proximally. This is because of its close proximity to the subendocardial surface along which the proximal two-thirds of right bundle branch travels. This makes it highly susceptible to damage and any structural abnormalities (i.e. increase in right ventricular pressure as a result of right ventricular hypertrophy or right heart strain) can lead to RBBB.

Clinical features
- Usually asymptomatic
- Rarely presents with syncope or bradycardia.

ECG appearance

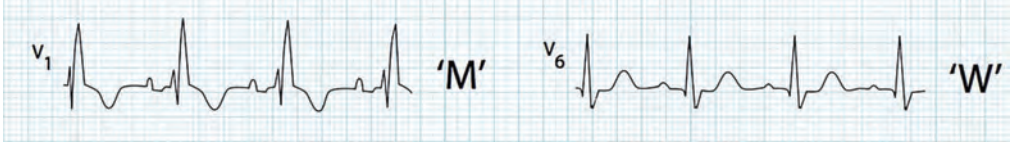

Figure 11.13 – Typical RSR' in V_1 and wide slurred S wave in V_6 (MarroW).

- Wide QRS complexes (>0.12 s)
- RSR' pattern in V_1–V_3 (M pattern)
- Long S wave duration in V_6, I.

Management
- Treatment not usually required as patients tend to be asymptomatic.

Prognosis
- Good prognosis if not associated with other cardiac conditions.

11.5.2 Left bundle branch block

Definition
A conduction deficit in the His-Purkinje system resulting in a delay in left ventricular depolarisation.

// PRO-TIP //

Left bundle branch blocks can be subdivided into left anterior fascicular block (LAFB) and left posterior fascicular blocks (LPFB) (also known as left anterior/posterior hemiblocks). A bifascicular block is a right bundle branch block *plus* either LAFB/LPFB, while a trifascicular block may refer to bifascicular block *plus* 1st/2nd/3rd-degree AV block.

// EXAM ESSENTIALS //

A new left bundle branch block on ECG, associated with chest pain, should raise clinical suspicion of an acute myocardial infarction.

Epidemiology
- Prevalence increases with age
- Affects less than 1% of the general population.

Aetiology
Common causes:
- **Aortic stenosis**
- Large anterior MI
- Hypertension

Other causes:
- Cardiomyopathies
- Idiopathic degeneration of the conduction system.

Pathophysiology
Left bundle branch block (LBBB) is often associated with underlying heart disease and rarely occurs in patients with structurally normal hearts. It may be present as a first sign of cardiomyopathy before the ventricular function declines. If it is present in a young person, follow-up is recommended.

// WHY? //

Ischaemia or degenerative damage can interrupt conduction through the right or left bundle branches. When this happens, the affected ventricle is unable to depolarise in the normal sequence (through rapid uniform stimulation via the Purkinje fibres). Instead, the cells of that ventricle have to depend on myocyte-to-myocyte spread of electrical activity travelling from the unaffected ventricle, which is comparably slower. This delayed process results in prolonged depolarisation and widening of the QRS complex.

Clinical features
- Usually asymptomatic
- May present with symptoms of underlying disease.

ECG appearance

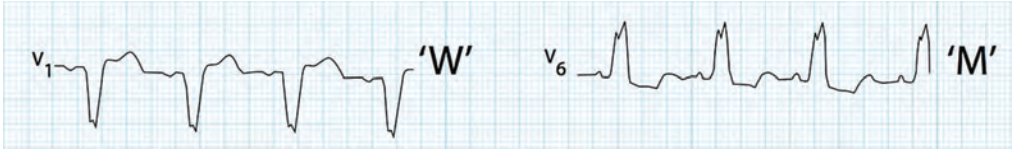

Figure 11.14 – Dominant broad S wave in V_1 and RSR' pattern in V_6 (WilliaM).

- Wide QRS complexes (>0.12 s)
- Deep S wave in V_1 and 'M-shaped' R wave in V_6
- Poor R wave progression in chest leads.

Management
- Echocardiography is always necessary to assess LV structure and function
- Specific treatment not usually required although treatment for e.g. cardiomyopathy, where present, is indicated.

Prognosis
- Patients with concomitant MI and LV dysfunction with LBBB are associated with higher mortality rates.

// PRO-TIP //

Sgarbossa criteria
A score of ≥3 has a 90% specificity for diagnosing an infarct.
- **Concordant ST elevation >1 mm** in leads with a positive QRS complex (score 5)
- **Concordant ST depression >1 mm** in V1–V3 (score 3)
- **Excessively discordant ST elevation >5 mm** in leads with a negative QRS complex (score 2)

11.6 Narrow complex tachycardias

The majority of tachycardias seen in clinical practice are narrow complex in nature. As previously discussed, all narrow complex tachycardias are generally supraventricular in origin, i.e. they either occur in the SA node, atria or AV node. Most supraventricular tachycardias are benign and rarely life-threatening.

11.6.1 Inappropriate sinus tachycardia

Definition
An increase in the heart rate to more than 100 beats per minute in adults. Normal sinus rhythm is observed.

Epidemiology
- Majority of patients (90%) are female
- Peak incidence of 38 years old.

Aetiology

Common causes:

- **Physiological** – exercise, pain, anxiety, pregnancy
- Drugs – adrenaline, salbutamol, anti-histamines, tricyclic antidepressants
- **Pulmonary embolism**

// EXAM ESSENTIALS //

Sinus tachycardia is the most common ECG finding in patients with pulmonary embolism. The second most common finding is right axis deviation with or without right bundle branch block. It is important to note that the $S_1Q_3T_3$ pattern (deep S wave in lead I and Q waves and T wave inversion in lead III) is rarely seen in clinical practice.

Other causes:

- Cardiac – heart failure, ischaemic heart disease, cardiomyopathies
- Metabolic – hyperthyroidism, anaemia
- Infection
- Substances – cocaine, amphetamines, cannabis, caffeine
- Psychological – anxiety

Pathophysiology

Increased automaticity as a result of sympathetic activation and/or parasympathetic inhibition.

Clinical features

- May present with palpitations, chest pain, breathlessness and light-headedness.

ECG appearance

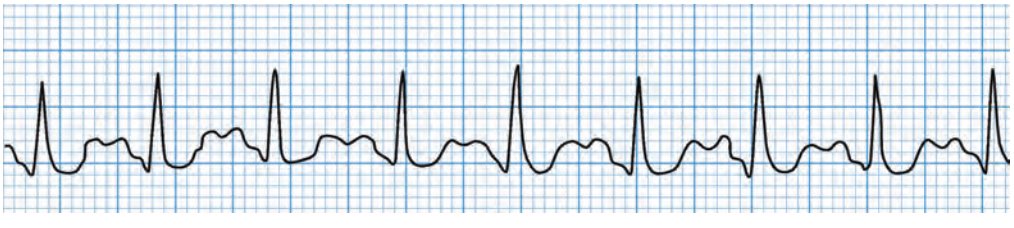

Figure 11.15 – ECG shows sinus rhythm with a rate of 150 bpm.

- Rate of more than 100 beats per minute
- Normal visible P waves but often buried in preceding T wave ('camel hump' appearance).

// PRO-TIP //

Sinus tachycardia is commonly mistaken for paroxysmal SVT and atrial flutter. These conditions can be distinguished using vagal manoeuvres and/or adenosine. A temporary heart block is induced, terminating the underlying SVT and allowing visualisation of P waves.

Management

- **Treat underlying cause** or remove offending agent
- Pharmacological therapy: **Beta-blockers** or ivabradine may be used in symptomatic patients with chronic inappropriate sinus tachycardia
- Surgical: Radiofrequency ablation of SA node is performed if patient is resistant to medical therapy. Note that this treatment modality for sinus tachycardia is exceedingly rare.

11.6.2 Atrial fibrillation

Atrial fibrillation *In A Heartbeat*	
Epidemiology	Affects 1% of the population, seen in 10% of those over 70 years Male predominance
Aetiology	ATRIALE PhIB (mnemonic) Ischaemic heart disease, hypertension and mitral pathologies are the most common causes. Also consider thyrotoxicosis as a cause.
Clinical features	Asymptomatic (25%), palpitations, exercise intolerance Irregularly irregular pulse
Management	Rate control, rhythm control and anticoagulation

Definition

An atrial tachyarrhythmia characterised by an irregularly irregular heart rhythm and indiscernible P waves on the ECG.

Epidemiology

- Most common atrial tachyarrhythmia
- Seen in 1% of the entire population
- Affects 10% of those over 70 years of age; prevalence increases with age
- Male predominance

Aetiology (ATRIALE PhIB)

- **A**lcohol and caffeine
- **T**hyrotoxicosis
- **R**heumatic fever and mitral valve pathologies
- **I**schaemic heart disease
- **A**trial myxoma
- **L**ungs (pulmonary hypertension, pneumonia)
- **E**lectrolyte disturbances
- **Ph**armacological
- **I**atrogenic (drugs, surgery)
- **B**lood pressure
- Others: infection

// WHY? //

Thyroxine, a thyroid hormone, has major effects on the heart. It shortens the duration of action potential and increases the automaticity of cardiac myocytes. This predisposes to formation of tachyarrhythmia.

// EXAM ESSENTIALS //

The three most common causes of atrial fibrillation (AF) are **ischaemic heart disease**, **hypertension** and **mitral valve pathologies**.

Pathophysiology

AF is often initiated by an area of ectopic focal activity with increased automaticity. This area typically originates at or around the pulmonary veins. The rapid activation of these foci propagate ectopic beats that create **micro-re-entrant circuits** throughout the atrial muscle. The various mechanisms of re-entry are explained in *Figure 11.16*. As there is no organisation of atrial electrical activity, the atrial myocytes are not able to contract simultaneously. This results in blood pooling

in the atria, predisposing to thrombus formation. In addition, loss of atrial contraction also leads to inadequate ventricular filling and, as a result, poor cardiac output.

Clinical features

- Asymptomatic (25% of patients)
- Palpitations, breathlessness on exertion and lightheadedness
- **Irregularly irregular pulse** with or without **pulse deficit** (see below)

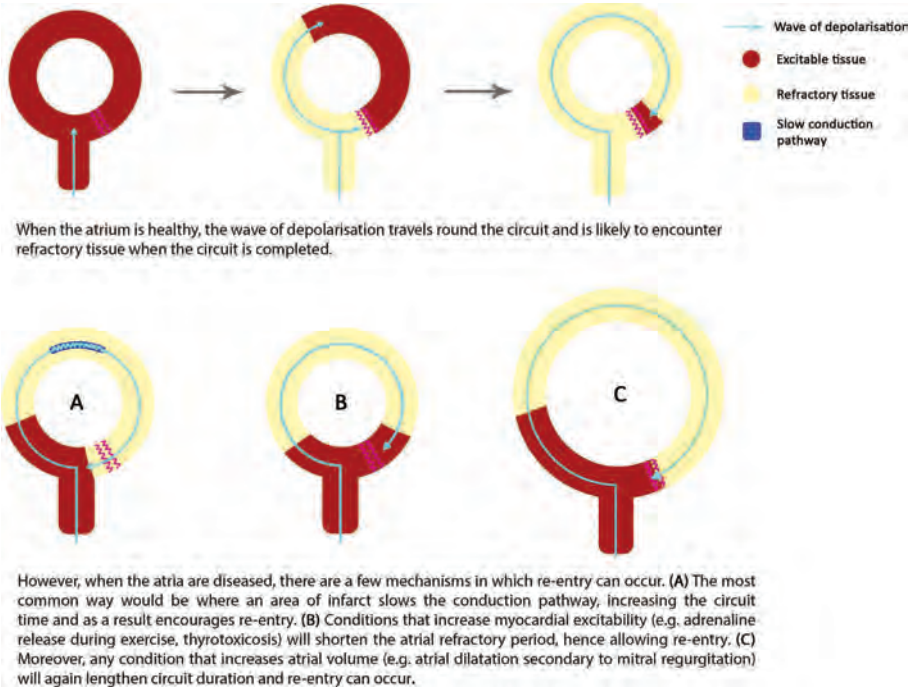

Figure 11.16 – Re-entry mechanism of AF.

// WHY? //

A pulse deficit refers to the difference in apical and peripheral pulse rate. In fast AF (rapid ventricular rates), there is inadequate diastolic filling. The subsequent stroke volume will be reduced and this might not be sufficient to generate a palpable peripheral pulse.

Clinical assessment

1. Initial investigations:
   - **ECG** (see below)
     - Holter ambulatory monitoring may be used for patients with suspected paroxysmal AF episodes
   - **Blood test** – identify underlying cause; FBC (anaemia, infection), TFTs (hyperthyroidism), U&Es (electrolyte disturbances), glucose
   - **Echocardiography** (transthoracic):
     - consideration of cardioversion for rhythm control management
     - high suspicion of structural heart disease
     - if additional information is needed for anticoagulation risk stratification

2. Assess duration of symptoms and episodes:

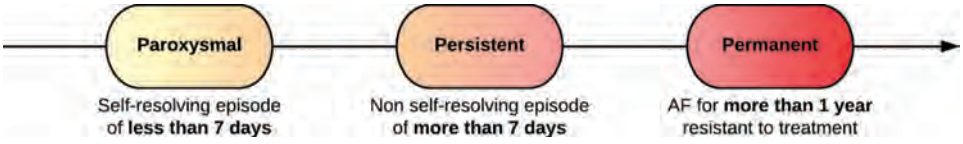

Figure 11.17 – Types of AF.

3. Controlling heart rate and rhythm:
   - Rate control is indicated as a first-line option unless:
     - there is a reversible cause
     - AF is secondary to heart failure
     - new-onset AF
   - Refer to algorithm (*Figure 11.18*) for full details on rate and rhythm control
   - Rapid AF (AF with rapid ventricular response) is a **medical emergency** (refer to *Chapter 17*)

// PRO-TIP //

The AFFIRM and RACE trial have shown similar outcomes (in both mortality benefit and stroke risk) with either rate or rhythm control.

   - As a **very general rule of thumb**
     - asymptomatic or mildly symptomatic patient above the age of 65, consider rate control
     - symptomatic patients under 65 year olds or concomitant heart failure, consider rhythm control

4. Assessment for anticoagulation:
   - All patients with AF should have their stroke risk calculated using the CHA_2DS_2-VASc score. However, the decision to initiate anticoagulation therapy should be weighed up with the patient's bleeding risk using the HAS-BLED score.
   - Bleeding risk scores are not used to determine whether anticoagulation is withheld.

CHA_2DS_2-VASc (stroke risk)
C – CHF History (1)
H – Hypertension (1)
A – Age (65–74 = 1, ≥75 = 2)
D – Diabetes (1)
S – Stroke, VTE history (2)
VA – Vascular disease (1)
Sc – Sex (F = 1)

- If score of ≥1 in **men**, consider anticoagulation
- If score of ≥2, offer anticoagulation

HAS-BLED (bleeding risk)
H – Hypertension (SBP >160 mmHg)
A – Abnormal renal/liver function
S – Stroke history
B – Bleeding history/predisposition
L – Labile INR (time in therapeutic range <60%)
E – Elderly (>65 years old)
D – Drugs (NSAIDs, antiplatelets) or alcohol

   - The choice of anticoagulant is based on the clinical picture and patient preference:
     - direct thrombin inhibitor – dabigatran
     - direct factor Xa inhibitors – apixaban, rivaroxaban
     - vitamin K antagonist – warfarin (target INR 2.0–3.0)
     - refer to *Chapter 5* for their side-effects and contraindications.

Management

This section is based on ESC 2020 AF guidelines.

An integrated 'ABC' holistic pathway has been recommended to streamline care of AF patients.
A – Anticoagulation
B – Better symptom control
C – Cardiovascular risk factor and comorbidities management

1. **Anticoagulation**
   - All patients with AF should have their stroke risk calculated using the CHA_2DS_2-VASc score. However, the decision to initiate anticoagulation therapy should be weighed up with the patient's bleeding risk using the HAS-BLED score (see opposite page).
   - The choice of anticoagulation depends on the clinical picture and patient preference
   - Direct oral anticoagulants (DOACs) have superseded VKAs due to their better pharmacokinetic profile and favourable safety and efficacy (see *Chapter 5*)
   - DOACs are at this point not suitable for use in patients with mechanical heart valves, nor in patients with moderate to severe mitral stenosis (previously termed valvular AF), and are less well established in patients with end-stage renal disease; studies in these groups of patients are ongoing.
   - Antiplatelet monotherapy is ineffective for stroke prevention and is potentially harmful
   - Patients with AF often have other comorbidities such as IHD, PVD and CVA requiring antiplatelet therapy. Combination therapy with oral anticoagulant and antiplatelet agents is a niche area and clinical practice remains patient- and clinician-specific.
   - Left atrial appendage occlusion or excision may be considered in patients with contraindications to or unable to tolerate OACs. This is done percutaneously with an occlusion device or surgically excised in patients undergoing cardiac surgery for other indications.
2. **Better symptom control**
   - Rate and rhythm control strategies are usually used to improve AF-related symptoms
   - Multiple studies over the years have shown similar outcomes in both mortality and stroke risk with either approach.

// PRO-TIP //

The EAST-AFNET4 study (2020) showed that in patients with recently diagnosed AF (<12 months) and at high risk for cardiovascular complications, early rhythm control strategy was associated with reduced mortality, stroke and hospitalisations.

- Rate control
  - typically used as first-line therapy in patients who are elderly and have multiple comorbidities
  - beta-blockers, digoxin and non-dihydropyridine calcium channel blockers are common agents used to achieve pharmacological rate control
  - in patients with concomitant HFrEF, beta-blockers and digoxin are recommended
  - the choice of agent depends on symptoms, comorbidities and side-effect profile
  - combination therapy is often required to achieve adequate rate control
  - a lenient HR target of <110 bpm is an acceptable initial approach for most patients
  - patients with refractory symptoms despite maximal therapy may be considered for an AV node ablation and pacemaker implantation ('pace and ablate' strategy)

// WHY? //

The risk of thromboembolism increases significantly after 48 hours of AF onset. It is important to establish adequate anticoagulation therapy prior to performing cardioversion. However, this risk can be mitigated by performing a TOE beforehand to exclude an LA/LAA thrombus.

- Rhythm control
  - a rhythm control strategy attempts to restore and maintain sinus rhythm
  - this involves 2 stages:
    - establishing sinus rhythm – electrical or pharmacological cardioversion
    - maintaining sinus rhythm – anti-arrhythmic medication and/or electrophysiological ablation
  - factors favouring rhythm control include:
    - younger age
    - 1st AF episode or short history
    - tachycardia-mediated cardiomyopathy
    - no or minimal comorbidities
    - failure of rate control
    - normal/mildly dilated atrial size
  - cardioversion
    - acute AF with rapid ventricular response and haemodynamic instability is a medical emergency; immediate electrical cardioversion is recommended (see *Chapter 17*)
    - the decision for early vs. elective cardioversion depends on the duration of symptom onset
    - early cardioversion may be considered in patients with AF onset <48 hours. Electrical (synchronised DC) or pharmacological can be attempted; however, pharmacological is less effective but does not require sedation.
    - if symptom onset >48 hours, an elective cardioversion may be attempted. This is usually performed at least 3 weeks after therapeutic OAC commencement.
    - pre-treatment with anti-arrhythmic agents (such as amiodarone, flecainide) should be considered to facilitate the success of electrical cardioversion
    - the choice of agent for pharmacological cardioversion depends on the presence of structural heart disease: if no structural heart disease – flecainide, propafenone; if structural heart disease – IV amiodarone
    - in selected patients with infrequent paroxysmal episodes, a 'pill in pocket' approach with flecainide may be effective
  - long-term anti-arrhythmic medication
    - AADs are often used after successful cardioversion to reduce AF recurrences and maintain sinus rhythm
    - the selection of AADs should primarily be guided by safety considerations rather than efficacy
    - amiodarone – most effective but highest risk of extra-cardiac complications; can be used in all patients regardless of structural disease or HF
    - flecainide and propafenone – contraindicated in patients with structural heart disease, IHD and HF, limiting their therapeutic use
    - dronedarone – less effective than amiodarone but better side-effect profile
    - sotalol – high risk of QT prolongation and hyperkalaemia; regular monitoring for both is recommended
  - catheter ablation
    - well-established treatment modality for prevention of AF recurrences
    - superior to and safer than AADs
    - reduces mortality and hospitalisation in patients with AF and HFrEF (CASTLE-AF trial)

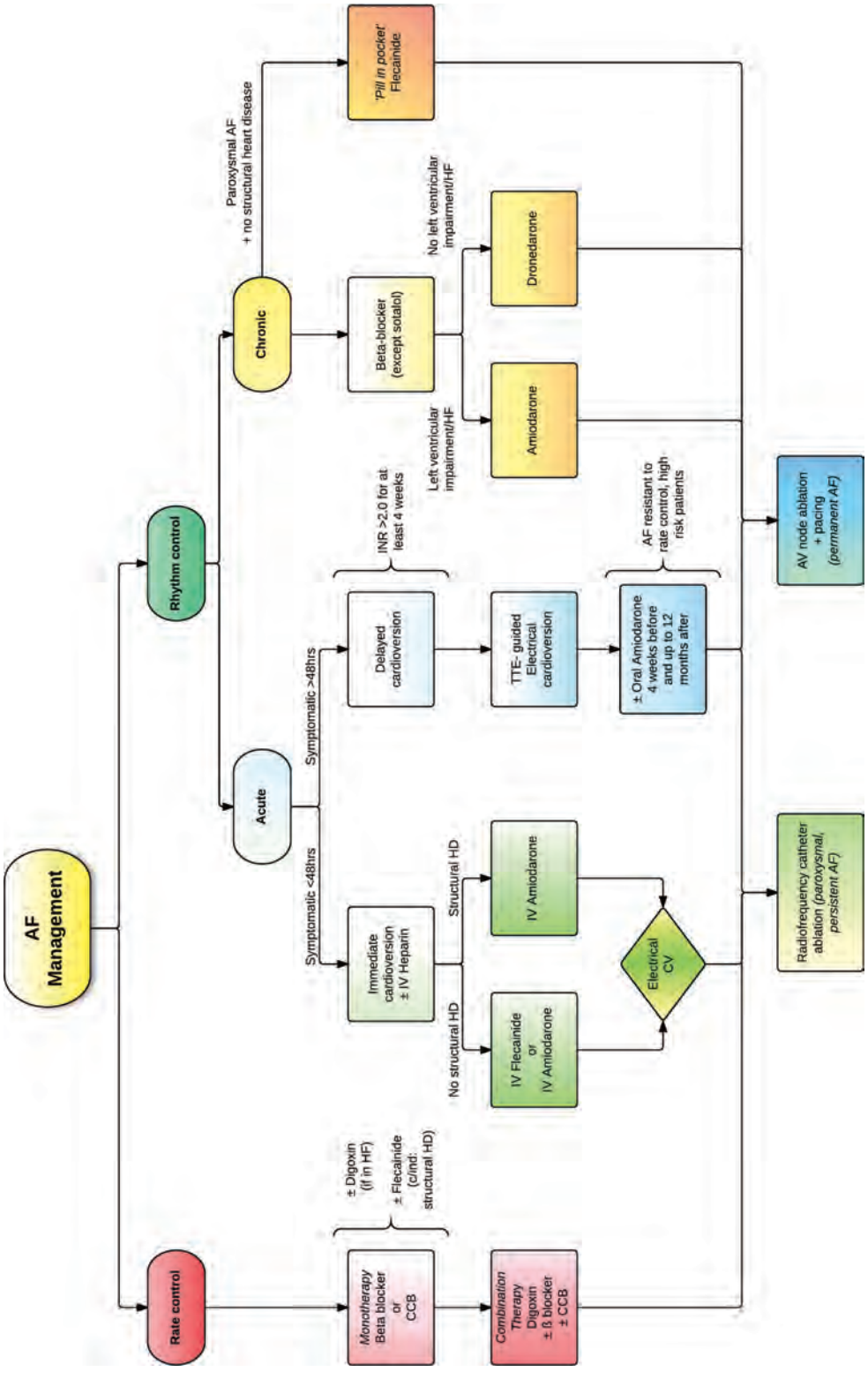

Figure 11.18 – Management of atrial fibrillation (based on NICE CG180).

  - indications are based on patient choice, failed medical therapy and low risk factors for recurrences post-ablation
  - complication rates are low (2–3%), mainly occurring in the first 24 hours. These include vascular access complications, asymptomatic CVA and tamponade (~1%).

3. **Cardiovascular risk factor and comorbidities management**
   - All patients with AF should be screened for other cardiovascular risk factors and comorbidities/lifestyle factors which may contribute to AF development
   - Lifestyle modification
     - alcohol reduction and abstinence
     - weight reduction
     - moderate intensity exercise; avoid chronic excessive endurance exercise as this may worsen AF
   - Comorbidities management
     - hypertension – most common risk factor (1.7x); see *Chapter 13*
     - HF – see *Chapter 10*
     - diabetes mellitus – glycaemic control to prevent autonomic neuropathy that may drive AF development
     - obstructive sleep apnoea
       - OSA shown to drive and worsen AF, reduce success rates of AAD, cardioversion and catheter ablation
       - reasonable to screen for OSA prior to initiation of rhythm control therapy
       - continuous positive airway pressure (CPAP) may improve rhythm control.

ECG appearance

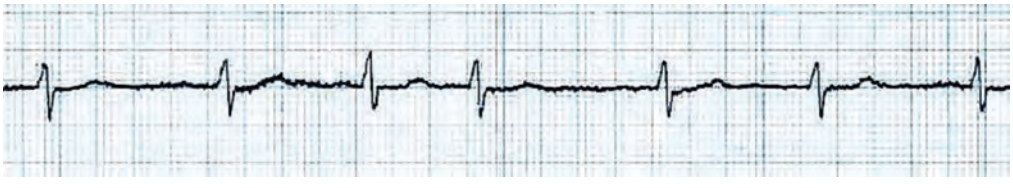

Figure 11.19 – Classic ECG showing an irregularly irregular rhythm with no visible P waves characteristic of AF.

- Irregularly irregular rhythm
- Absent P waves.

// EXAM ESSENTIALS //

When the rhythm of an ECG is in doubt, use a paper to mark an RR interval and move it along the rhythm strip to check whether it is truly irregular.

11.6.3 Atrial flutter

Definition

Atrial flutter is an atrial tachyarrhythmia which is characterised by a regular, rapid atrial rate.

// EXAM ESSENTIALS //

Atrial flutter should always be suspected in tachycardias with a fixed atrioventricular conduction ratio (2:1). Atrial flutter classically produces an atrial rate of approximately 300 bpm and a ventricular rate of 150 bpm.

Epidemiology

- Less common than atrial fibrillation
- The prevalence ratio in males to females is 5:2
- Incidence increases with age.

Aetiology

Common causes:

1. **Right atrial dilatation** – pulmonary embolus, mitral and/or tricuspid pathologies, congestive heart failure
2. **Ischaemic heart disease**
3. Idiopathic – no underlying heart disease
4. Normal variant – tall males
5. Patients with a history of endurance sports (causing atrial enlargement)

Other causes:

- Drugs – flecainide, propafenone (15% post-AF therapy)
- Metabolic disturbances – hyperthyroidism, alcohol
- Iatrogenic – previous catheter ablation, cardiac surgery.

Pathophysiology

Atrial flutter is characterised by a **macro re-entrant circuit** within the atrium, most commonly around the tricuspid annulus in an anti-clockwise fashion. This results in a rapid, regular atrial activity at a rate of around 300 bpm.

Many of these fast impulses are unable to pass through the AV node secondary to the refractory period. This results in a decreased rate of firing of the ventricles. The re-entrant circuit encompasses a substantial surface area and appears to have a 'sawtooth' pattern on ECG.

Clinical features

- Commonly presents with breathlessness and palpitations
- Syncope and severe dyspnoea (at very rapid rates).

// EXAM ESSENTIALS //

Vagal manoeuvres (e.g. carotid sinus massage, adenosine) may be useful in diagnosing atrial flutter as it causes a transient AV block and reduces the ventricular rate. This might reveal the classic 'sawtooth' flutter waves.

ECG appearance

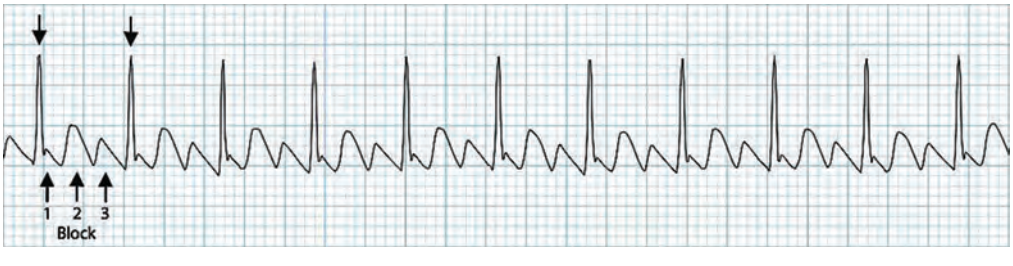

Figure 11.20 – ECG showing the characteristic sawtooth pattern seen in atrial flutter with a 3:1 conduction ratio (arrows).

- Narrow complex tachycardia with a classic atrial rate of 300 bpm
- **Regular** 'sawtooth' pattern of flutter waves
- Fixed conduction ratios (2:1, 3:1, etc.).

Management

- Management of atrial flutter is similar to that of AF, but it should be noted that achieving rate control in flutter is more difficult.
- 60% of patients with flutter present acutely and cardioversion is recommended in this group
- Radiofrequency ablation is highly recommended in patients with chronic atrial flutter as therapy can induce high rates of remission (90%).

11.6.4 Atrioventricular nodal re-entrant tachycardia

Definition

A type of paroxysmal supraventricular tachycardia (SVT) caused by an aberrant circuit within the AV node.

Epidemiology

- Most common cause of paroxysmal SVT (40–50%)
- Female predominance
- Incidence: 35 per 100 000 people.

Aetiology

Common causes:

- **Idiopathic** (occurs most commonly in structurally normal hearts).

Pathophysiology

In atrioventricular nodal re-entrant tachycardia, a re-entrant circuit occurs within the AV node. These re-entrant circuits tend to be functional (non-anatomical) in nature, most commonly involving dual pathways of different conduction velocities (i.e. a slow and a fast pathway). Typically, conduction via the slow pathway will be anterograde and retrograde via the fast pathway (refer to the *Re-entry* section above). This is known as the *slow–fast* type and is the most common form of AVNRT (90%) observed in clinical practice. AVNRT accounts for 60% of all regular supraventricular tachycardias.

Clinical features

- Sudden onset of rapid palpitations
- Dizziness
- Breathlessness
- Syncope occasionally occurs.
- Regular narrow complex tachycardia
- P waves may not be visible as they are buried in QRS complexes.

ECG appearance

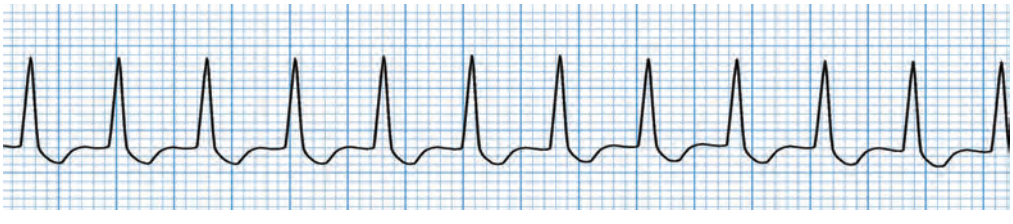

Figure 11.21 – AVNRT with narrow QRS complexes and no visible P waves.

Management

- Some AVNRTs can be terminated with **vagal manoeuvres** (e.g. Valsalva manoeuvres, carotid sinus massage)
- Intravenous adenosine or rate-limiting calcium channel blockers may be used if vagal manoeuvres fail
- In emergencies, when patients present with haemodynamic compromise, DC cardioversion is advised
- Catheter ablation – indicated in patients with recurrent episodes; associated with a less than 1% risk of heart block.

11.6.5 Atrioventricular re-entrant tachycardia

Definition

Atrioventricular re-entrant tachycardia (AVRT) refers to a form of paroxysmal SVT caused by an anatomically defined re-entrant circuit involving one or more accessory pathways. Wolff–Parkinson–White (WPW) syndrome, a pre-excitation syndrome which can often lead to AVRT, is characterised by a short PR interval and delta waves on ECG.

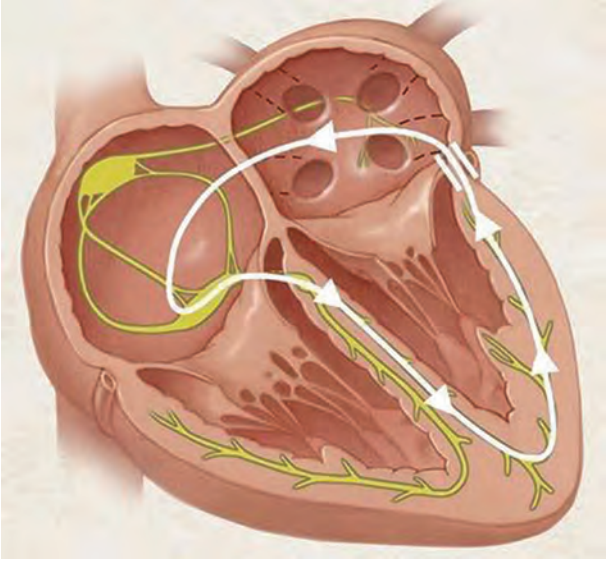

Figure 11.22 – Re-entrant circuit involving an accessory pathway (Bundle of Kent) as seen in WPW.

Epidemiology

- Prevalence: 0.1 to 30 per 1000
- 60–70% of patients with WPW have no evidence of heart disease.

Aetiology

- **Congenital**
- Associated with hypertrophic cardiomyopathy and Ebstein's anomaly.

Pathophysiology

Accessory pathways (e.g. Bundle of Kent in WPW) are fibres of abnormal myocytes extending across the atrioventricular groove forming an aberrant connection between the atria and ventricles. This results in an anatomical re-entrant circuit comprising the normal AV conduction

system and the accessory pathway. As electrical impulses travel faster through the accessory pathway, the re-entrant circuit tends to occur in an anti-clockwise direction.

// PRO-TIP //

There are two forms of AVRT:
- *Orthodromic* – the most common type (95%) that involves anterograde conduction via AV node and retrograde conduction via the accessory pathway
- *Antidromic* – anterograde conduction occurs via the accessory pathway and retrograde conduction through the AV node.

Clinical features
- Palpitations
- Chest pain
- Syncope.

ECG appearance

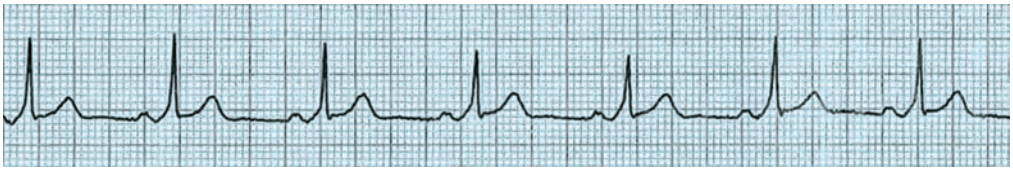

Figure 11.23 – Shortened PR interval and upslurring 'delta' wave characteristic of WPW.

- Short PR interval (<0.2 s)
- Broad QRS (>0.12 s)
- **'Delta' wave** – slurring of the QRS upstroke.

// WHY? //

Conduction via the accessory pathway is faster than that of the native Purkinje system. Therefore there is premature activation of the ventricles, eliminating the delay through the AV node. The QRS complex is widened and slurred because it represents a fusion of both excitation waves.

Management
- In the acute setting, vagal manoeuvres, IV adenosine and procainamide or, rarely, DC cardioversion may be used to terminate the tachyarrhythmia
- Flecainide and amiodarone may also be useful in the acute setting
- **Catheter ablation** of the accessory pathway is the definitive therapy.

Prognosis
- Associated with a small risk of sudden death.

11.7 Broad complex tachycardias

Broad complex tachycardias appear with wide QRS complexes of more than 0.12 seconds (3 small squares). They are often ventricular in origin, but may also be supraventricular with an aberrant conduction (usually a bundle branch block). They may be regular (monomorphic ventricular tachycardia) or irregular (torsades de pointes, polymorphic ventricular tachycardia) in nature.

Ventricular tachycardia and ventricular fibrillation *In A Heartbeat*	
Epidemiology	Most common cause of sudden cardiac death Most often occurring during and after an MI
Aetiology	Ischaemic heart disease Electrolyte disturbances Long QT syndrome
Clinical features	May be haemodynamically unstable – severe dyspnoea, shock and cardiac arrest
Management	Immediate ALS and DC cardioversion Pharmacological therapy (amiodarone) ICD insertion to prevent further episodes

11.7.1 Ventricular tachycardia

Definition

A ventricular tachycardia (VT) refers to a tachyarrhythmia that originates from the ventricles producing three or more successive broad QRS complexes at a rate of more than 100 beats per minute.

Epidemiology

- Peak incidence in middle-aged patients
- VT and ventricular fibrillation (VF) account for the most common causes of sudden cardiac death.

Aetiology

Common causes:

- **Ischaemic heart disease** – scarring post-MI
- Structural heart disease – **cardiomyopathies**, valvular heart disease
- Electrolyte disturbances – hyper-/hypokalaemia, hyper-/hypomagnesaemia

Other causes:

- Drugs and substances – digoxin toxicity, cocaine
- Channelopathies – long QT syndromes, Brugada syndrome
- Idiopathic

Classification:

- Morphology:
  - *monomorphic* – uniform QRS complexes in most leads (most common form)
  - *polymorphic* – QRS of varying amplitudes, axis and duration across the leads

// WHY? //

Monomorphic VT occurs as a result of a single distinct anatomical re-entrant circuit usually centred around an old myocardial infarct. Polymorphic VT, on the other hand, is caused by functional re-entry with varying circuits.

- Duration:
  - *sustained* – occurs for more than 30 seconds
  - *non-sustained* – self-terminating episodes (<30 s)

Pathophysiology

The mechanisms underlying VTs include:

- Re-entrant circuit (most common) – usually due to myocardial scarring post-MI
- Triggered activity – as seen in long QT syndromes
- Increased automaticity

Clinical features

- Haemodynamically stable patients present with:
  - palpitations
  - dizziness/light-headedness
  - syncope (inadequate cerebral perfusion)
- Haemodynamically unstable patients (severely hypotensive and tachycardic)
  - **low cardiac output symptoms** – severe dyspnoea, dizziness (altered consciousness), syncope
  - eventually leading to cardiogenic shock and cardiac arrest.

Examination findings:

- JVP may be elevated; intermittent cannon A waves may be seen.

// WHY? //

Intermittent cannon 'A' waves occur as a result of retrograde blood flow into the jugular vein resulting from right atrial contraction against a closed tricuspid valve; this is due to AV dissociation.

ECG appearance

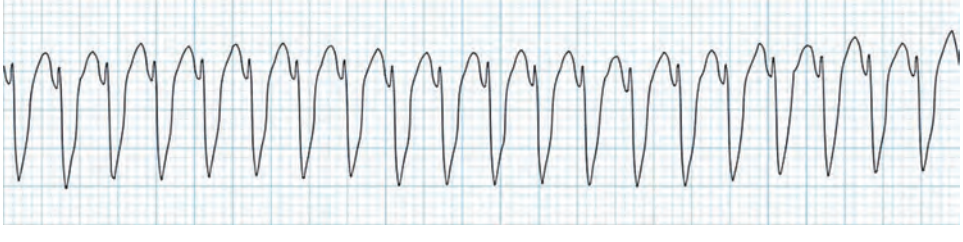

Figure 11.24 – Monomorphic VT with regular and uniform broad QRS complexes.

- Broad QRS complexes (>0.12 s).

// EXAM ESSENTIALS //

As a very general rule of thumb, all broad complex tachycardias should be suspected to be a VT until proven otherwise. The other important differential is an SVT with aberrant conduction (e.g. atrial tachyarrhythmia with a co-existing LBBB or WPW).

Management

Immediate management:

- If the patient is haemodynamically unstable:
  1. Immediate resuscitation
  2. Emergency DC cardioversion – synchronised shock is recommended
- If the patient is haemodynamically stable, the management depends on the underlying cause:
  1. Intravenous amiodarone (non-idiopathic VT)
  2. Elective synchronised DC cardioversion may be indicated if resistant to medical therapy

Further management:

- Identification of underlying aetiology
  - features of acute ischaemia (ST changes) – consider urgent coronary angiography
  - cardiac imaging (echocardiography or CMR if available) to evaluate for structural heart disease
  - exercise stress testing may be useful in patients with immediate risk of coronary artery disease
- Prevention of recurrence
  - anti-arrhythmic therapy
    - beta-blockers are used as first-line therapy as they have been shown to reduce mortality
    - amiodarone is often used due to its efficacy and low pro-arrhythmic effects
    - other agents such as procainamide, sotalol and mexiletine may be used as adjunct therapies
  - for indications of ICD implantation see Guidelines just above *Figure 11.3*
  - catheter ablation may be useful in patients with recurrent VT despite medical therapy

// EXAM ESSENTIALS //

Patients should not drive for up to 6 months after an unstable VT/VF event.

11.7.2 Torsades de pointes

Definition

A form of polymorphic VT associated with a prolonged QT interval. Torsades de pointes is French for '*twisting of the points*'.

Epidemiology

- Female predominance (females have longer baseline QT intervals).

Aetiology

- Congenital long QT syndrome – monogenic disorders inherited in an autosomal dominant fashion
- Acquired long QT syndrome
  - electrolyte imbalance
    - hypocalcaemia
    - hypomagnesaemia
    - hypokalaemia
  - drugs (refer to *Chapter 4*)

Pathophysiology

Derangement of cardiac ions, particularly sodium, potassium and calcium, increases the duration of action potential, resulting in early after-depolarisations (*triggered activity*). In congenital forms of this condition, early after-depolarisations may be triggered by sympathetic stimulation (e.g. exercise, sudden loud noises).

Clinical features

- Usually symptomatic, commonly presents with self-limiting episodes of palpitations, light-headedness or syncope.

ECG appearance

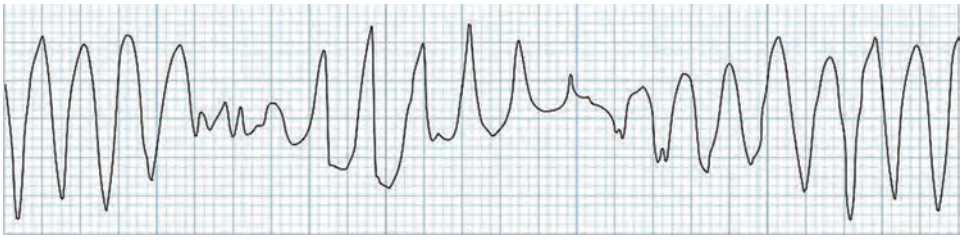

Figure 11.25 – Polymorphic VT (torsades de pointes); notice the change in amplitudes of the QRS complexes.

- Peaks of the QRS twist around the isoelectric line
- Irregular RR intervals
- Tachycardia with ventricular rates of 160 to 250 bpm
- Prolonged QTc (male >0.43 s, female >0.45 s).

Management

- Treat underlying cause – stop precipitating drug or correct electrolyte abnormalities
- IV **magnesium sulphate** (given as a slow infusion)
- If resistant to medical therapy, consider temporary pacing
- Consider IV isoproterenol as a bridge to pacing but pacing is preferable
- If patient is haemodynamically unstable, non-synchronised electrical cardioversion may be indicated.

Prognosis

- May degenerate into VF if heart rate exceeds 220 bpm.

11.7.3 Brugada syndrome

Brugada syndrome is an autosomal sodium channelopathy associated with sudden cardiac deaths. It is **most prevalent in Asia** and has a high male predominance (8:1). Defective sodium channels impair influx of sodium ions, resulting in shorter action potentials. Brugada syndrome classically presents with **syncope** and **sudden cardiac arrest** in an otherwise normal asymptomatic patient (33%). ICD implantation is the only definitive treatment.

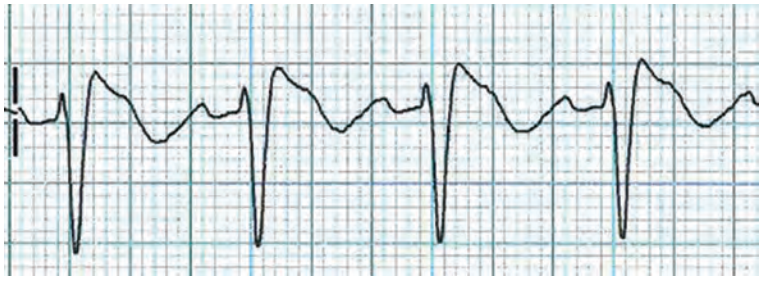

Figure 11.26 – An ECG of Brugada syndrome showing 'Brugada' sign (downsloping coved ST elevation followed by an inverted T wave); note that this is only present in leads V1 and V2.

11.7.4 Ventricular fibrillation

Definition

A rapid, unco-ordinated and life-threatening ventricular arrhythmia resulting in poor myocardial contraction, eventually leading to cardiac death.

Epidemiology

- Incidence: 6 per 10 000
- Has a bimodal distribution peaking at under 6 months and 45 to 75 years
- 1–8% occur out of hospital and are usually fatal.

Aetiology

// EXAM ESSENTIALS //

Ventricular fibrillation is usually a progression from ventricular tachycardias.

Common causes:

1. **Ischaemic heart disease** – relatively common following an acute MI
2. Electrolyte abnormalities (particularly hyperkalaemia)
3. Idiopathic

Other causes:

- Long QT syndromes
- Structural heart disease – cardiomyopathies, valvular heart disease
- Systemic – pulmonary embolus, sepsis

Pathophysiology

- Multiple wavelets – continuous micro re-entrant circuits are formed within the ventricles
- Very rapid (up to 500 bpm) irregular electrical activity results in unsynchronised ventricular contractions
- Because of the unsynchronised and ineffective ventricular contraction, the onset of VF leads to a precipitous drop in cardiac output, which is rapidly followed by cardiac arrest.

ECG appearance

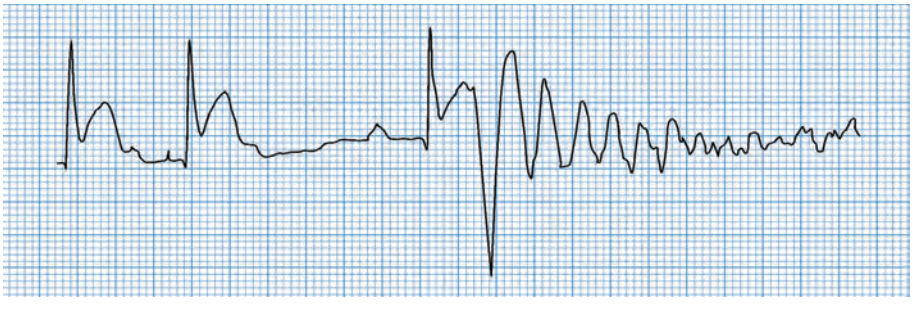

Figure 11.27 – A chaotic rhythm strip showing rapid progression from coarse to fine VF.

- Chaotic waveforms with varying amplitudes
- Unidentifiable P-waves, QRS complexes or T waves.

Management

Immediate management:

1. Advanced cardiac life support (refer to *Chapter 17*)
2. IV amiodarone, IV lignocaine can be considered.

Long-term management:

- ICD insertion
- Amiodarone may be used when ICDs are contraindicated.

Prognosis

- The community survival rate is 4–33% depending on factors such as prompt bystander CPR and duration of CPR to defibrillation time
- 20% recurrence rate per year.

11.8 Extra beats

11.8.1 Premature atrial ectopics

Premature atrial ectopics are beats that originate from an ectopic focus within the atria. They are more common in patients with conditions that cause elevated atrial pressures such as mitral valve disease, hypertension and heart failure but may also be related to electrolyte abnormalities and drugs. These beats represent a normal electrophysiological phenomenon, and the majority of patients are asymptomatic. Symptomatic individuals may experience palpitations or dizziness.

ECG appearance

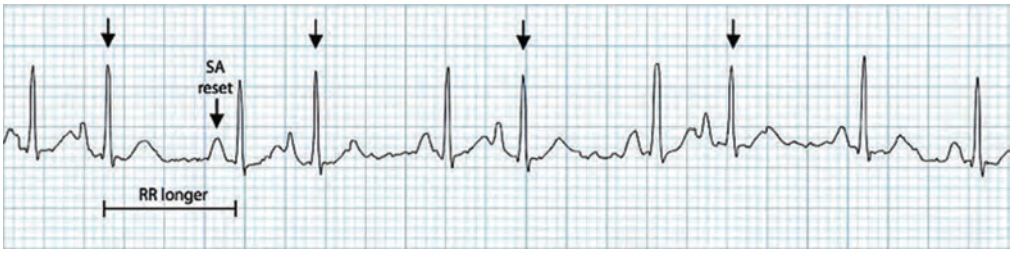

Figure 11.28 – This rhythm strip shows premature atrial ectopics (arrows) followed by compensatory pauses.

- Abnormal P wave followed by normal QRS complex
- P waves often hidden in preceding T wave – abnormal-looking T wave
- Reduced PR interval (<0.12 s).

Management

- Usually benign and treatment is symptomatic
- Beta-blockers, verapamil or flecainide may be considered.

Prognosis

- Increased risk of CVS mortality (stroke, sudden cardiac death), associated with new AF.

11.8.2 Premature ventricular ectopics

Premature ventricular ectopics, on the other hand, refer to complexes originating from an ectopic focus within the ventricles. The incidence of this phenomenon increases with age, and is more common in patients with concomitant IHD. These are usually benign as well, unless associated with a prolonged QT interval. The majority are asymptomatic, but as in premature atrial ectopics, these ectopics may present with palpitations in symptomatic patients.

ECG appearance

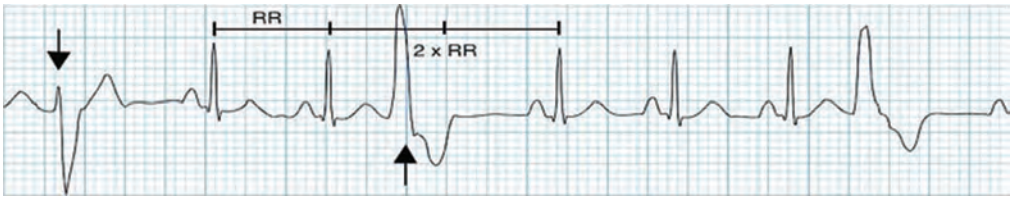

Figure 11.29 – Multifocal PVCs (arrows) followed with a prolonged compensatory pause (2× RR).

- Broad QRS complexes (>0.12 s)
- Premature beats
- Usually followed by a compensatory pause.

Management

- No treatment required if asymptomatic
- Beta-blocker or verapamil if symptomatic
- Radiofrequency catheter ablation if severe (more effective than anti-arrhythmics).

Prognosis

- Excellent prognosis if structurally normal heart
- Increased risk of sudden death in patients with LVEF<40%.