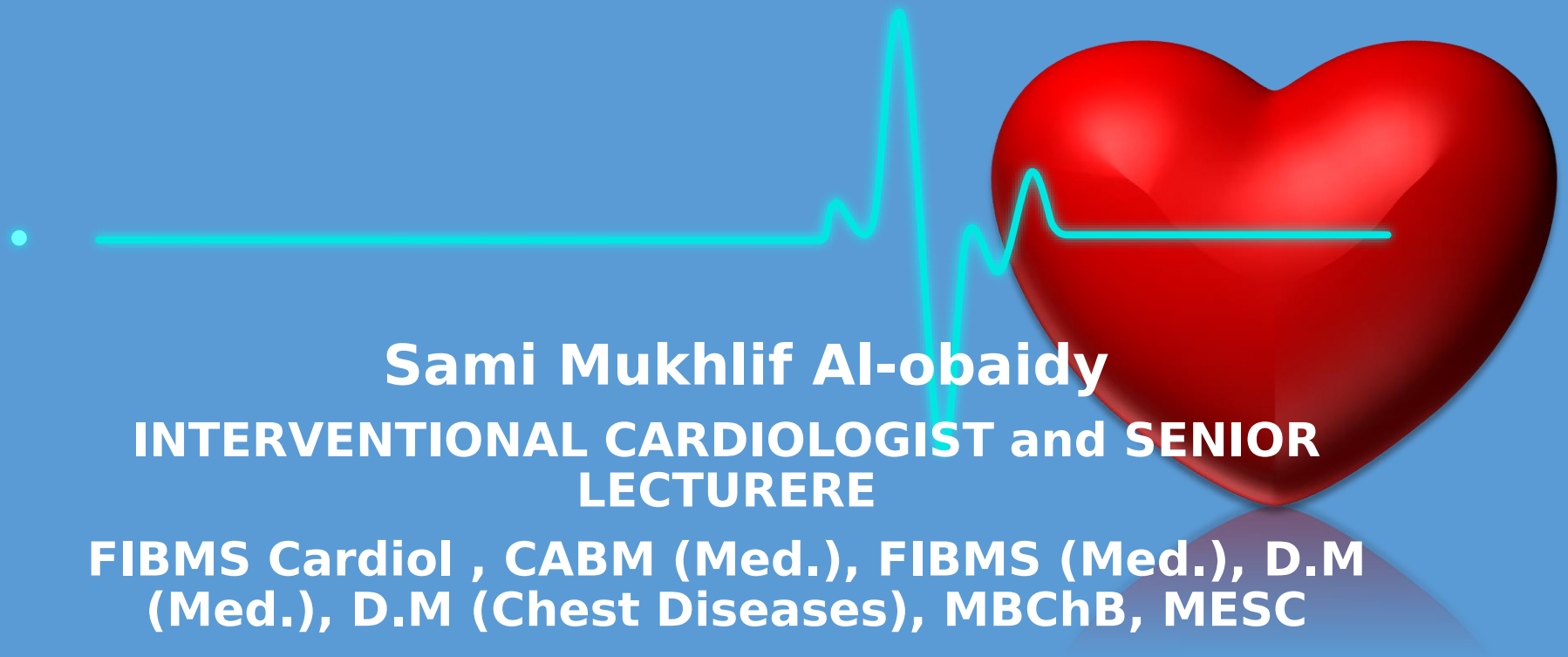
Presenting problems in cardiovascular diseases



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Presenting problems in cardiovascular disease

Cardiovascular disease gives rise to a relatively limited range of symptoms. Making the correct diagnosis depends on careful analysis of the factors that provoke symptoms, the subtle differences in how they are described by the patient, the clinical findings and the results of investigations. **A close relationship between symptoms and exercise is the hallmark of heart disease.** The New York Heart Association (NYHA) functional classification is used to grade disability.

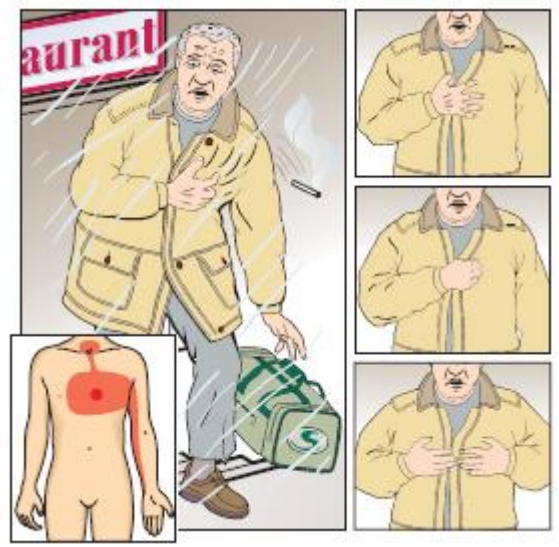
Chest pain on exertion

Exertional chest pain, which is a typical presenting symptom of coronary artery disease.

Clinical assessment

A careful history is crucial in determining whether chest pain is cardiac or not. Chest pain on effort is the hallmark of angina pectoris. The reproducibility, predictability and relationship to physical exertion (and occasionally emotion) of the chest pain are the most important features. The duration of symptoms should be noted because patients with recent-onset angina are at greater risk than those with long-standing and unchanged symptoms

i New York Heart Association (NYHA) functional classification
Class I
• No limitation during ordinary activity
Class II
• Slight limitation during ordinary activity
Class III
• Marked limitation of normal activities without symptoms at rest
Class IV
• Unable to undertake physical activity without symptoms; symptoms may be present at rest



Physical examination is often normal but may reveal evidence of risk factors for cardiovascular disease, such as xanthoma or xanthelasma indicating hyperlipidaemia. Signs of anaemia or thyrotoxicosis may be identified, both of which can exacerbate angina. Cardiovascular examination may reveal evidence of left ventricular dysfunction or cardiac murmurs in patients with aortic valve disease and hypertrophic cardiomyopathy. Other manifestations of arterial disease, such as bruits and loss of peripheral pulses, may also be observed.

Investigations

A full blood count, fasting blood glucose, lipids, thyroid function tests and a 12-lead ECG are the most important baseline investigations. An exercise ECG is helpful in identifying high risk patients who require further investigation and treatment but cannot reliably exclude the presence of coronary artery disease. In patients with chest pain where the exercise ECG is normal but where there is a suspicion of coronary artery disease, CT coronary angiography should be performed. If a murmur I found, echocardiography should be performed to check for valve disease or hypertrophic cardiomyopathy.

Severe prolonged chest pain

Severe prolonged cardiac chest pain may be due to acute myocardial infarction or to unstable angina. These are known collectively as the **acute coronary syndromes.**

Clinical assessment

Acute coronary syndrome is suggested by a previous history of stable angina but an episode of acute severe chest pain at rest can be the first presentation of coronary artery disease. Making the correct diagnosis depends on analysing the character of the pain and its associated features. Physical examination may reveal signs of risk factors for coronary artery disease as described for exertional chest pain, and pallor or sweating, which is indicative of autonomic disturbance and typical of acute coronary syndrome. Other features, such as arrhythmia, hypotension and heart failure, may occur. Patients presenting with symptoms consistent with an acute coronary syndrome require admission to hospital and urgent investigation because there is a high risk of avoidable complications.

Investigations

A 12-lead ECG is mandatory and is the most useful method of initial triage, along with measurement of serum troponin I or T. The diagnosis of an acute coronary syndrome is supported by ST segment elevation or depression on ECG and an elevated level of troponin I or T, which demonstrates that there has been myocardial damage. If the diagnosis remains unclear after initial investigation, repeat ECG recordings should be performed and are particularly useful if they can be obtained during an episode of pain. If the plasma troponin concentrations are normal at baseline, repeat measurements should be made 6–12 hours after the onset of symptoms or admission to hospital. New ECG changes or an elevated plasma troponin concentration confirm the diagnosis of an acute coronary syndrome. If the pain settles and does not recur, there are no new ECG changes and troponin concentrations remain normal, the patient can be discharged from hospital but further investigations may be indicated to look for evidence of coronary artery disease.

Breathlessness

Cardiac causes of breathlessness include cardiac arrhythmias, acute and chronic heart failure, acute coronary syndrome, valvular disease, cardiomyopathy and constrictive pericarditis.

16.5 New York Heart Association (NYHA) functional classification

Class I
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Syncope

The term ‘syncope’ refers to loss of consciousness due to reduced cerebral perfusion.

Cardiac causes?

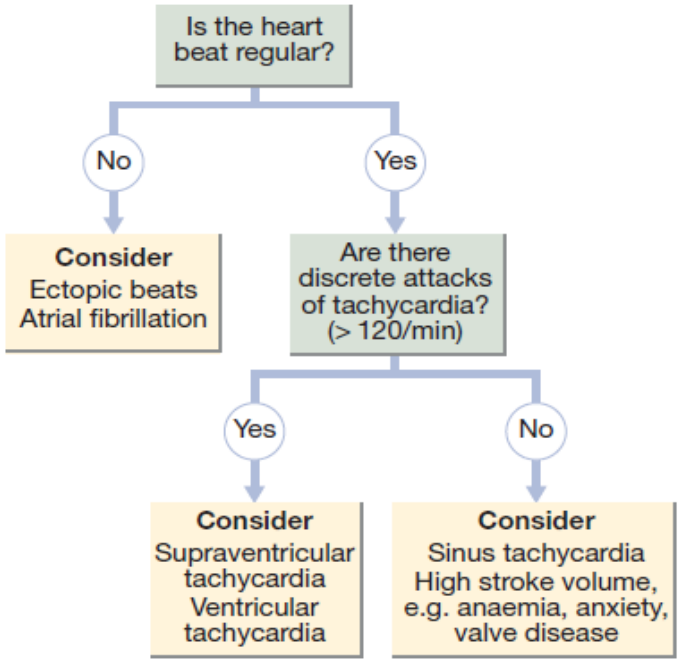
Palpitation

Palpitation is a common and sometimes frightening symptom that is usually due to a disorder of cardiac rhythm. Patients use the term to describe many sensations, including an unusually erratic, fast, slow or forceful heart beat, or even chest pain or breathlessness.

Clinical assessment

Initial evaluation should concentrate on determining the likely mechanism of palpitation and whether or not there is significant underlying heart disease. A detailed description of the sensation is essential and patients should be asked to describe their symptoms clearly, or to demonstrate the sensation of rhythm by tapping with their hand.

Some patients will describe the experience as a ‘flip’ or a ‘jolt’ in the chest, while others report dropped or missed beats. Extrasystoles are often more frequent during periods of stress or debility; they can be triggered by alcohol or nicotine. Episodes of a pounding, forceful and relatively fast (90–120/min) heart beat are a common manifestation of anxiety. These may also reflect a hyperdynamic circulation, such as anaemia, pregnancy and thyrotoxicosis, and can occur in some forms of valve disease such as aortic regurgitation.



Discrete bouts of a very rapid (over 120/min) heart beat are more likely to be due to a paroxysmal supraventricular or ventricular tachycardia. In contrast, episodes of atrial fibrillation typically present with irregular and usually rapid palpitation.

Investigation

If initial assessment suggests that the palpitation is due to an arrhythmia, the diagnosis should be confirmed by an ECG recording during an episode using an ambulatory ECG monitor or a patient-activated ECG recorder. Additional investigations may be required depending on the nature of the arrhythmia.

Management

Palpitation is usually benign and even if the patient's symptoms are due to an arrhythmia, the outlook is good if there is no underlying structural heart disease. Most cases are due to an awareness of the normal heart beat, a sinus tachycardia or benign extrasystoles, in which case an explanation and reassurance may be all that is required.

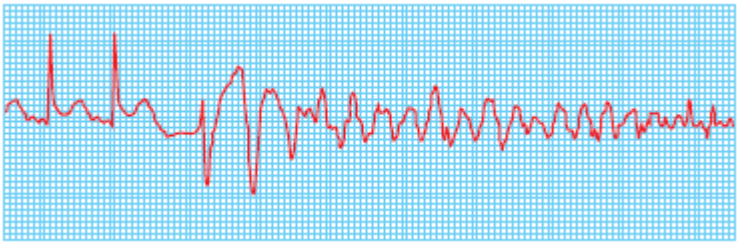
Palpitation associated with pre-syncope or syncope may reflect more serious structural or electrical disease and should be investigated without delay. Other arrhythmias may require treatment.

Cardiac arrest

Cardiac arrest describes the sudden and complete loss of cardiac output due to asystole, ventricular tachycardia or fibrillation, or loss of mechanical cardiac contraction (pulseless electrical activity). The clinical diagnosis is based on the victim being unconscious and pulseless; breathing may take some time to stop completely after cardiac arrest. Death is virtually inevitable, unless effective treatment is given promptly. Sudden cardiac death is usually caused by a catastrophic arrhythmia and accounts for 25–30% of deaths from cardiovascular disease, claiming an estimated 70 000 to 90 000 lives each year in the UK. Many of these deaths are potentially preventable.

Pathogenesis

Coronary artery disease is the most common cause of cardiac arrest. Ventricular fibrillation or ventricular tachycardia is common



i Causes of sudden arrhythmic death

Coronary artery disease (85%)

- Myocardial ischaemia
- Acute myocardial infarction
- Prior myocardial infarction with myocardial scarring

Structural heart disease (10%)

- Aortic stenosis (p. 521)
- Hypertrophic cardiomyopathy (p. 539)
- Dilated cardiomyopathy (p. 539)
- Arrhythmogenic right ventricular dysplasia (p. 540)
- Congenital heart disease (p. 531)

No structural heart disease (5%)

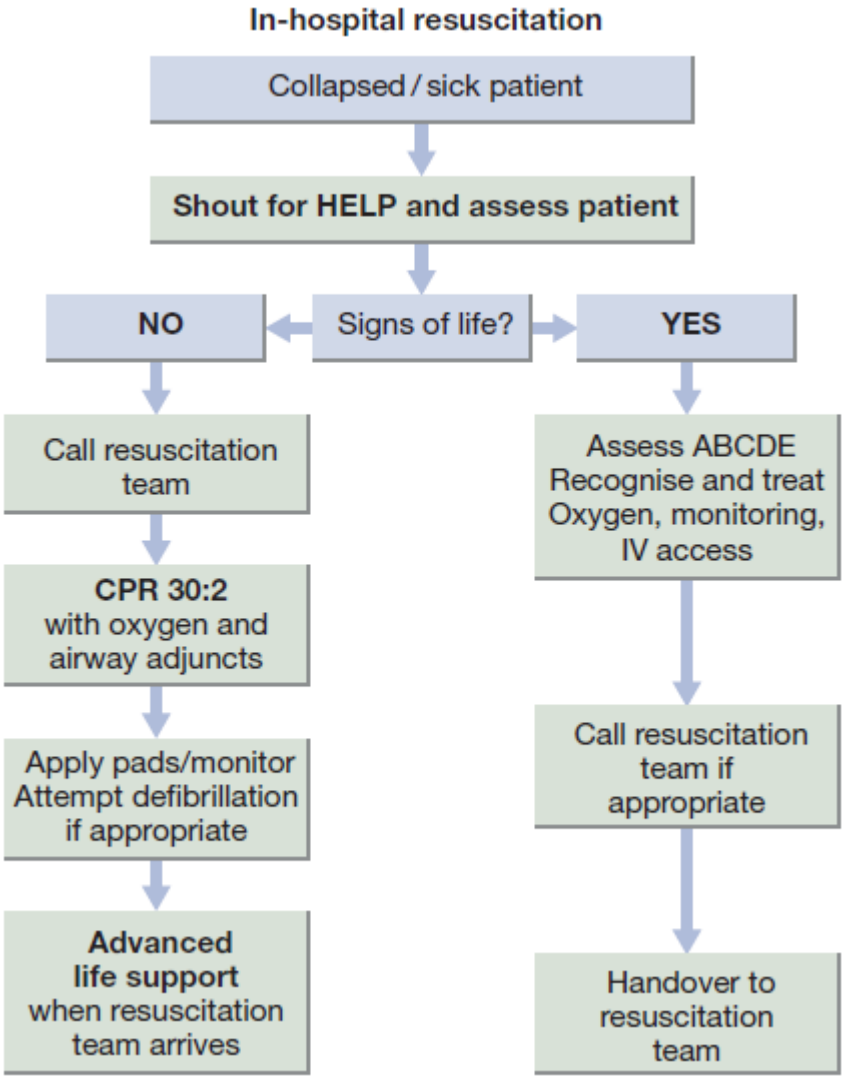
- Long QT syndrome (p. 476)
- Brugada syndrome (p. 477)
- Wolff–Parkinson–White syndrome (p. 474)
- Adverse drug reactions (torsades de pointes, p. 476)
- Severe electrolyte abnormalities

in the first few hours of MI and many victims die before medical help is sought. Up to one-third of people developing MI die before reaching hospital, emphasising the importance of educating the public to recognise symptoms and to seek medical help quickly. Acute myocardial ischaemia in the absence of infarction can also cause these arrhythmias, but less commonly. Patients with a history of previous MI are at increased risk of sudden arrhythmic death, especially if there is extensive left ventricular scarring and impairment, or if there is ongoing myocardial ischaemia. Cardiac arrest may be caused by ventricular fibrillation pulseless ventricular tachycardia asystole or pulseless electrical activity. These can complicate many types of heart disease, including cardiomyopathies, and sometimes can occur in the absence of recognised structural abnormalities. The causes Sudden death less often occurs because of an acute mechanical catastrophe such as cardiac rupture.

Clinical assessment and management

Basic life support

When a patient with suspected cardiac arrest is encountered, the ABCDE approach to management should be followed; this involves prompt assessment and restoration of the Airway, maintenance of ventilation using rescue Breathing (‘mouth-tomouth’ breathing), and maintenance of the Circulation using chest compressions; Disability, in resuscitated patients, refers to assessment of neurological status, and Exposure entails removal of clothes to enable defibrillation, auscultation of the chest, and assessment for a rash caused by anaphylaxis, for injuries and so on. The term basic life support (BLS)

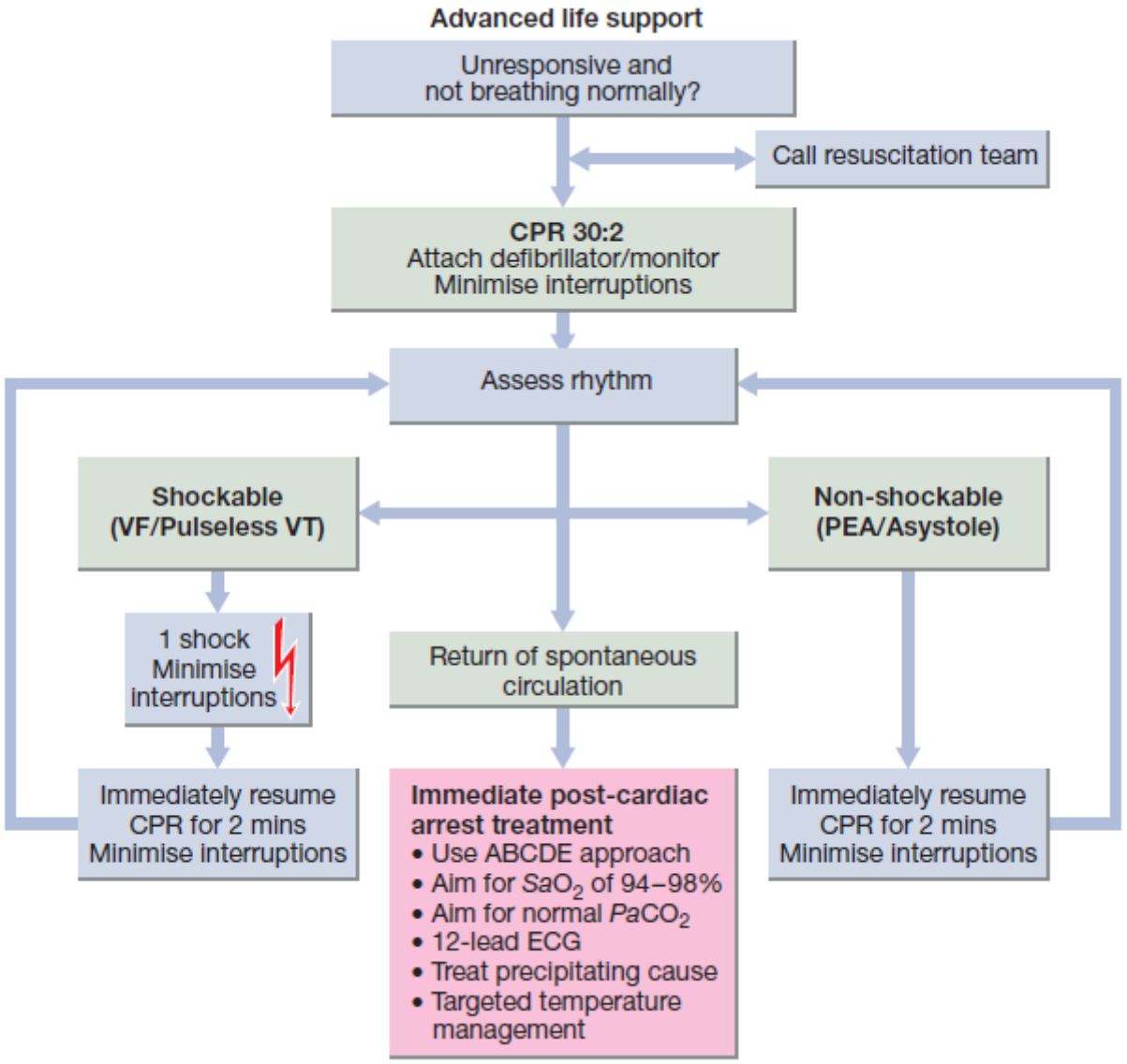


encompasses manoeuvres that aim to maintain a low level of circulation until more definitive treatment with advanced life support can be given. Chest compression only(‘hands-only’) cardiopulmonary resuscitation (CPR) is easier for members of the public to learn and administer, and is now advocated in public education campaigns.

Advanced life support

Advanced life support (ALS) aims to restore normal cardiac rhythm by defibrillation when the cause of cardiac arrest is a tachyarrhythmia, or to restore cardiac output by correcting other reversible causes of cardiac arrest. The initial priority is to assess the patient's cardiac rhythm by attaching a defibrillator or monitor. Once that this has been done, treatment should be instituted based on the clinical findings.
Ventricular fibrillation or pulseless ventricular tachycardia should be treated with immediate defibrillation. Defibrillation is more likely to be effective if a biphasic shock defibrillator is used, where the polarity of the shock is reversed midway through its delivery. Defibrillation is usually administered using a 150-joule biphasic shock, and CPR resumed immediately for 2 minutes without attempting to confirm restoration of a pulse because restoration of mechanical cardiac output rarely occurs immediately after successful defibrillation.

If, after 2 minutes, a pulse is not restored, a further biphasic shock of 150–200 joules should be given. Thereafter, additional biphasic shocks of 150–200 joules are given every 2 minutes after each cycle of CPR. During resuscitation, adrenaline (epinephrine, 1 mg IV) should be given every 3–5 minutes and consideration given to the use of intravenous amiodarone, especially if ventricular fibrillation or ventricular tachycardia re-initiates after successful defibrillation. Ventricular fibrillation of low amplitude, or ‘fine VF’, may mimic asystole. If asystole cannot be confidently diagnosed, the patient should be treated for VF and defibrillated. If an electrical rhythm is observed that would be expected to produce a cardiac output, ‘pulseless electrical activity’ is present. Pulseless electrical activity should be treated by continuing CPR and adrenaline (epinephrine) administration while seeking such causes. Asystole should be treated similarly, with the additional support of atropine and sometimes external or transvenous pacing in an attempt to generate an electrical rhythm. **There are many potentially reversible causes of cardiac arrest; the main ones can be easily remembered as a list of four Hs and four Ts.**


Advanced life support
Unresponsive and not breathing normally?
Call resuscitation team
CPR 30:2
Attach defibrillator/monitor
Minimise interruptions
Assess rhythm
Shockable (VF/Pulseless VT)
Non-shockable (PEA/Asystole)
1 shock
Minimise interruptions
Return of spontaneous circulation
Immediately resume CPR for 2 mins
Minimise interruptions
Immediate post-cardiac arrest treatment
• Use ABCDE approach
• Aim for SaO_2 of 94–98%
• Aim for normal $PaCO_2$
• 12-lead ECG
• Treat precipitating cause
• Targeted temperature management
Immediately resume CPR for 2 mins
Minimise interruptions

The Chain of Survival

This term refers to the sequence of events that is necessary to maximise the chances of a cardiac arrest victim surviving. Survival is most likely if all links in the chain are strong: that is, if the arrest is witnessed, help is called immediately, basic life support is administered by a trained individual, the emergency medical services respond promptly, and defibrillation is achieved within a few minutes. Good training in both basic and advanced life support is essential and should be maintained by regular refresher courses. In recent years, public access defibrillation has been introduced in places of high population density, particularly where traffic congestion may impede the response of emergency services, such as railway stations, airports and sports stadia. Designated individuals can respond to a cardiac arrest using BLS and an automated external defibrillator.

Survivors of cardiac arrest

Patients who survive a cardiac arrest caused by acute MI need no specific treatment beyond that given to those recovering from an uncomplicated infarct, since their prognosis is similar. Those with reversible causes, such as exercise-induced ischaemia or aortic stenosis, should have the underlying cause treated if possible. Survivors of ventricular tachycardia or ventricular fibrillation arrest in whom no reversible cause can be identified may be at risk of another episode, and should be considered for an implantable cardiac defibrillator and anti-arrhythmic drug therapy. In these patients, the risk is reduced by treatment of heart failure with â-adrenoceptor antagonists (â-blockers) and angiotensin-converting enzyme (ACE) inhibitors, and by coronary revascularisation.

During CPR

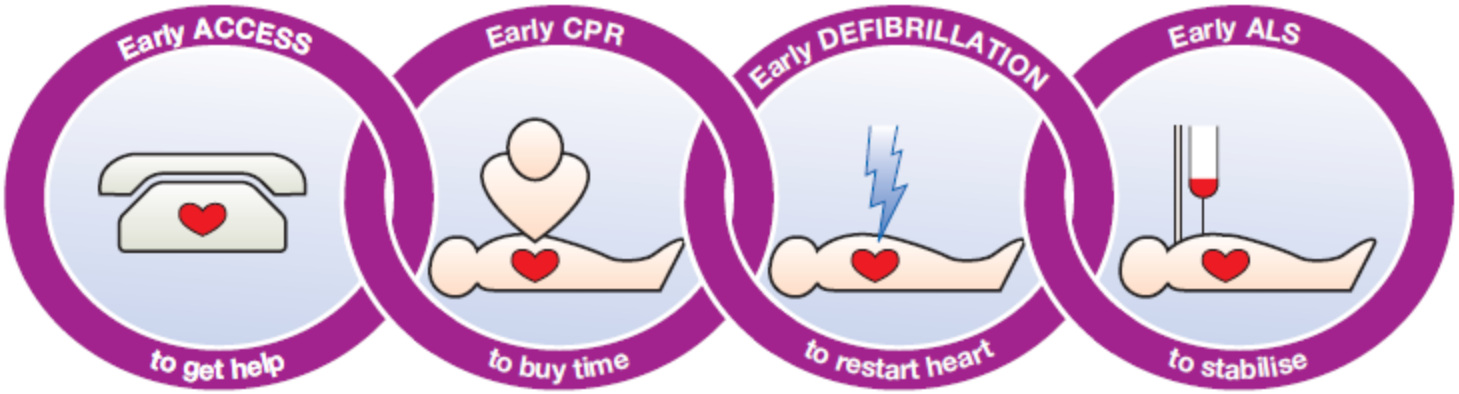
- Ensure high-quality chest compressions
- Minimise interruptions to compressions
- Give oxygen
- Use waveform capnography
- Perform continuous compressions when advanced airway in place
- Gain vascular access (intravenous or interosseous)
- Give adrenaline (epinephrine) every 3–5 min
- Give amiodarone after 3 shocks

Treat reversible causes

Hypoxia	Thrombosis – coronary or pulmonary
Hypovolaemia	Tension pneumothorax
Hypo-/hyperkalaemia/metabolic	Tamponade – cardiac
Hypothermia/hyperthermia	Toxins

Consider

- Ultrasound imaging
- Mechanical chest compressions to facilitate transfer/treatment
- Coronary angiography and percutaneous coronary intervention
- Extracorporeal CPR



Abnormal heart sounds

The first indication of heart disease may be the discovery of an abnormal sound on auscultation. This may be incidental – for example, during a routine childhood examination – or may be prompted by symptoms of heart disease.

Clinical assessment

The aims of clinical assessment are, firstly, to determine if the abnormal sound is cardiac; secondly, to determine if it is pathological; and thirdly, to try to determine its cause.

Is the sound cardiac?

Additional heart sounds and murmurs demonstrate a consistent relationship to a specific part of the cardiac cycle, whereas extracardiac sounds, such as a pleural rub or venous hum, do not.

Pericardial friction produces a characteristic scratching noise termed a pericardial rub, which may have two components corresponding to atrial and ventricular systole, and may vary with posture and respiration.

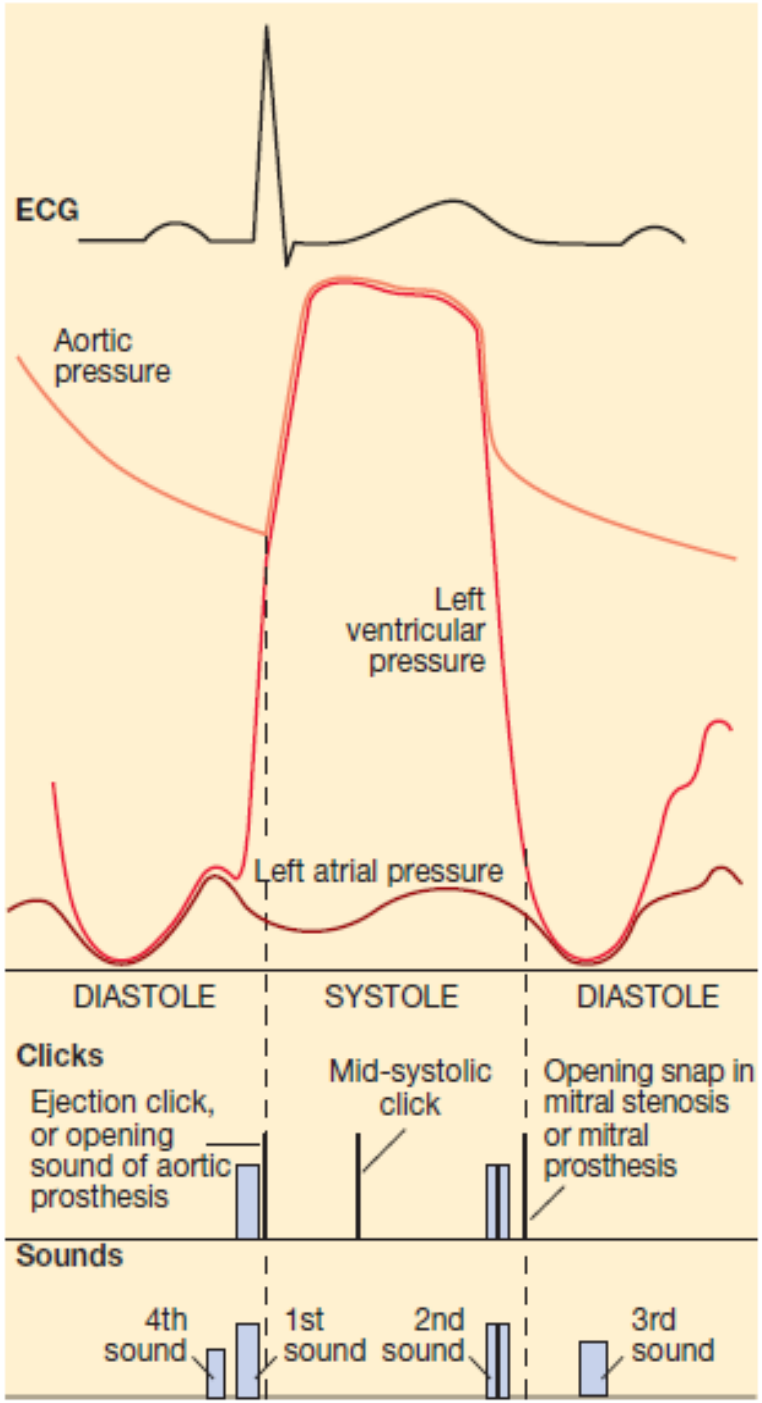
Is the sound pathological?

Pathological sounds and murmurs are the product of turbulent blood flow or rapid ventricular filling due to abnormal loading conditions. Some added sounds are physiological but may also occur in pathological conditions; for example, a third sound is common in young people and in pregnancy but is also a feature of heart failure. Similarly, a systolic murmur due to turbulence across the right ventricular outflow tract may occur in hyperdynamic states such as anaemia or pregnancy, but may also be due to pulmonary stenosis or an intracardiac shunt leading to volume overload of the RV, such as an atrial septal defect.

Benign murmurs do not occur in diastole and systolic murmurs that radiate or are associated with a thrill are almost always pathological.

What is the origin of the sound?

Timing, intensity, location, radiation and quality are all useful clues to the origin and nature of an additional sound or murmur. Radiation of a murmur is determined by the direction of turbulent blood flow and is detectable only when there is a high-velocity jet, such as in mitral regurgitation (radiation from apex to axilla) or aortic stenosis (radiation from base to neck). Similarly, the pitch and quality of the sound can help to distinguish the murmur, such as the ‘blowing’ murmur of mitral regurgitation or the ‘rasping’ murmur of aortic stenosis. The position of a murmur in relation to the cardiac cycle is crucial and should be assessed by timing it with the heart sounds, carotid pulse and apex beat .


ECG
Aortic pressure
Left ventricular pressure
Left atrial pressure
DIASTOLE
SYSTOLE
DIASTOLE
Clicks
Ejection click, or opening sound of aortic prosthesis
Mid-systolic click
Opening snap in mitral stenosis or mitral prosthesis
Sounds
4th sound
1st sound
2nd sound
3rd sound

Normal and abnormal heart sounds

Sound	Timing	Characteristics	Mechanisms	Variable features
First heart sound (S1)	Onset of systole	Usually single or narrowly split	Closure of mitral and tricuspid valves	Loud: hyperdynamic circulation (anaemia, pregnancy, thyrotoxicosis); mitral stenosis Soft: heart failure; mitral regurgitation
Second heart sound (S2)	End of systole	Split on inspiration Single on expiration (p. 447)	Closure of aortic and pulmonary valve A_2 first P_2 second	Fixed wide splitting with atrial septal defect Wide but variable splitting with delayed right heart emptying (right bundle branch block) Reversed splitting due to delayed left heart emptying (left bundle branch block)
Third heart sound (S3)	Early in diastole, just after S2	Low pitch, often heard as 'gallop'	From ventricular wall due to abrupt cessation of rapid filling	Physiological: young people, pregnancy Pathological: heart failure, mitral regurgitation
Fourth heart sound (S4)	End of diastole, just before S1	Low pitch	Ventricular origin (stiff ventricle and augmented atrial contraction) related to atrial filling	Absent in atrial fibrillation A feature of severe left ventricular hypertrophy
Systolic clicks	Early or mid-systole	Brief, high-intensity sound	Valvular aortic stenosis Valvular pulmonary stenosis Floppy mitral valve Prosthetic heart sounds from opening and closing of normally functioning mechanical valves	Click may be lost when stenotic valve becomes thickened or calcified Prosthetic clicks lost when valve obstructed by thrombus or vegetations
Opening snap (OS)	Early in diastole	High pitch, brief duration	Opening of stenosed leaflets of mitral valve Prosthetic heart sounds	Moves closer to S2 as mitral stenosis becomes more severe. May be absent in calcific mitral stenosis

Features of a benign or innocent heart murmur

- Soft
- Mid-systolic
- Heard at left sternal edge
- No radiation
- No other cardiac abnormalities

16.10 How to assess a heart murmur

When does it occur?

- Time the murmur using heart sounds, carotid pulse and the apex beat. Is it systolic or diastolic?
- Does the murmur extend throughout systole or diastole or is it confined to a shorter part of the cardiac cycle?

How loud is it? (intensity)

- Grade 1: very soft (audible only in ideal conditions)
- Grade 2: soft
- Grade 3: moderate
- Grade 4: loud with associated thrill
- Grade 5: very loud
- Grade 6: heard without stethoscope

Note: Diastolic murmurs are very rarely above grade 4

Where is it heard best? (location)

- Listen over the apex and base of the heart, including the aortic and pulmonary areas

Where does it radiate?

- Listen at the neck, axilla or back

What does it sound like? (pitch and quality)

- Pitch is determined by flow (high pitch indicates high-velocity flow)
- Is the intensity constant or variable?

Ejection systolic murmur (aortic stenosis, pulmonary stenosis, aortic or pulmonary flow murmurs)

Pansystolic murmur (mitral regurgitation, tricuspid regurgitation, ventricular septal defect)

Late systolic murmur (mitral valve prolapse)

Early diastolic murmur (aortic or pulmonary regurgitation)

Mid-diastolic murmur (mitral stenosis, tricuspid stenosis, mitral or tricuspid flow murmurs)

Systolic murmurs

Ejection systolic murmurs are associated with ventricular outflow tract obstruction and occur in mid-systole with a crescendo–decrescendo pattern, reflecting the changing velocity of blood flow.
Pansystolic murmurs maintain a constant intensity and extend from the first heart sound throughout systole to the second heart sound, sometimes obscuring it.
They occur when blood leaks from a ventricle into a low pressure chamber at an even or constant velocity.
Mitral regurgitation, tricuspid regurgitation and ventricular septal defect are the only causes of a pansystolic murmur.
Late systolic murmurs are unusual but may occur in mitral valve prolapse, if the mitral regurgitation is confined to late systole, and hypertrophic obstructive cardiomyopathy, if dynamic obstruction occurs late in systole.

Diastolic murmurs

These are due to accelerated or turbulent flow across the mitral or tricuspid valves. They are low-pitched noises that are often difficult to hear and should be evaluated with the bell of the stethoscope. A mid-diastolic murmur may be due to mitral stenosis (located at the apex and axilla), tricuspid stenosis (located at the left sternal edge), increased flow across the mitral valve (for example, the to-and-fro murmur of severe mitral regurgitation) or increased flow across the tricuspid valve (for example, a left-to-right shunt through a large atrial septal defect). Early diastolic murmurs have a soft, blowing quality with a decrescendo pattern and should be evaluated with the diaphragm of the stethoscope. They are due to regurgitation across the aortic or pulmonary valves and are best heard at the left sternal edge, with the patient sitting forwards in held expiration.

Continuous murmurs

These result from a combination of systolic and diastolic flow, such as occurs with a persistent ductus arteriosus, and must be distinguished from extracardiac noises such as bruits from arterial shunts, venous hums (high rates of venous flow in children) and pericardial friction rubs.

Investigations

If clinical evaluation suggests that the additional sound is cardiac and likely to be pathological, then echocardiography is indicated to determine the underlying cause.

Management

Management of patients with additional cardiac sounds depends on the underlying cause.