Ministry of Higher Education and Scientific Research

Middle Technical University, College of Health and Medical Technique, Anaesthesia Technique Department, 4th stage.

Teaching package for ICU technique

By

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Recognition and management of the seriously ill patient

The aim of assessment of seriously ill patient

1) Identify the physiological abnormalities.

2) Identify the most appropriate way to correct those abnormalities.

3) Diagnose the underlying problem.

• The process involves taking a full history and thoroughly examining and investigating the patient occurs is different manner in the seriously ill patient.

The difference is due to the urgency with which treatment needs to be started .

• There is rarely time to take a full history or carry out a very detailed examination before initiating treatment.

• Tasks that are typically carried out sequentially often have to be carried out in parallel with history taking, examination and initial resuscitation often occurring simultaneously.

• Often it is necessary to restrict oneself to only the information required to guide the next treatment decision and to fill in missing components of history,

examination and investigation after treatment has been Initiated on a "best guess" basis.

• The working diagnosis needs to be repeatedly reassessed as more information becomes available and on the basis of response to treatment.

1) Initial assessment

• The first step in assessing a seriously ill patient is to estimate how ill the patient is and how much time is available for assessment and investigation before initiating treatment.

Warning signs of a severely ill patient

Parameter	Values
Blood pressure	Systolic <90 or mean<70mmHg
Heart rate	>150 or <50 bpm
Respiratory rate	>30 or <8 breaths/min
Conscious level	GCS<12
Oliguria	<0.5 ml/kg/h
Sodium	<120 mmol/l or >150 mmol/l
Potassium	<2.5 mmol/l or >6 mmol/l
pH	>7.2
Bicarbonate	<18 mmol/l
Worried nurse	Concerned experienced nurse

A patient with any of these features should be assessed urgently

By an experienced physician

Components of the initial assessment

• Key components of the Initial assessment are assessment of airway patency, breathing and circulation.

• Absence of any of these should prompt immediate resuscitation.

• The severity of illness is often judged by assessing the compensatory response to the primary abnormality. In most cases This compensation involves activation of the sympathetic nervous system, and the magnitude of the sympathetic response gives an indication of the severity of illness.

• It should, however, be noted that in the pre-terminal patient the compensatory response is exhausted and the patient may be bradycardiac and bradypnic.

• If the patient is already receiving supportive therapy it is important to note the intensity of such therapy.

• For example a patient with an arterial oxygen saturation of 92% on 2 L/min of oxygen through a nasal cannula is much less ill than a similar patient with a saturation of 92% on 15 L/min of oxygen.

Assess intensity of support

• Inspired oxygen fraction needed to maintain saturation above 90%

• Intensity of ventilatory support—positive end-expiratory pressure, minute ventilation

• Dose of vasopressor & inotrope needed to maintain mean arterial pressure above 60 mmHg

• Need for volume support to keep adequate urine output

• Need for blood transfusion to keep hemoglobin above 8 g/dL

• Need for sedation in agitated patients

• Need for dialysis support or Worsening biochemistry

Seek help for specific problems

• **Cardiologist**—complete heart block, acute coronary syndrome, cardiogenic shock, intra-aortic balloon pump insertion, pericardial tamponade, massive pulmonary embolism

• **Nephrologist**—dialysis

• **Neurologist**—acute stroke or undiagnosed depressed conscious level

• **Neurosurgeon**—intracranial hemorrhage, head injury, severe cerebral edema

• **Trauma surgeon**—polytrauma, abdominal trauma, thoracic trauma, compartment syndrome

• **Obstetrician**—ruptured ectopic pregnancy, postpartum hemorrhage

Construct a working diagnosis and plan for further management

• After initial resuscitation, assessment, investigation, and response, a differential diagnosis should be arrived at.

• Reassess the patient frequently to modify initial plan if needed.

Brief relatives

• After initial resuscitation, assessment, investigation, and response, the family should be briefed about the :

✓ likely diagnosis

✓ treatment plan

✓ approximate prognostication

✓ approximate duration of stay and

✓ consent should be taken for any invasive procedures.

Airway

• Assessment of airway patency is vital. Look, listen and feel for evidence of airway obstruction.

• Look for tachycardia, tachypnea, sweating, use of accessory muscles, drooling (epiglottitis), see-saw thoracoabdominal (paradoxical chest wall) movement and recession.

• Remember that chest movement can occur even in the presence of complete airway obstruction.

• Listen for gurgling or strider (note that stridor may be absent, particularly in severe cases and the presence of a normal oxygen saturation does not exclude a compromised airway).

• Hypercarbia and a resulting decrease in conscious level indicate that the compensatory mechanisms are exhausted. Bradycardia indicates impending cardiorespiratory arrest.

➢ Inspiratory stridor is a rasping sound heard during inspiration and is a result of obstruction above or involving the larynx

➢ Wheeze is usually heard on expiration as a result of the lower airways collapsing

➢ Gurgling occur when secretion or liquid is present in the upper airways

➢ Snoring occurs during partial occlusion of the oropharynx due to relaxation of the oropharyngeal muscles and tongue

Breathing

✓ Effectiveness of Breathing

✓ Work of Breathing

• Cyanosis, hypoxia ?

• Rate, depth, symmetry of chest movement ? Use of accessory muscles?

• Palpate chest wall for structural integrity

• Chest injury / flail / pneumothoraces

• O2 therapy / Assisted ventilation

• Manage injury / pnuemothoraces

• Note that marked tachypnea is a useful marker of a severely ill patient, regardless of whether the patient has respiratory failure.

• Detection of cyanosis is often difficult and tachypnea is usually a more obvious, although non-specific sign of a problem.

• As with airway problems the severity of the problem is often best judged from the magnitude of the compensatory response.

• Pulse oximetry is a useful bedside test investigation, but it should be noted that significant desaturation is often a late feature of ventilatory abnormalities.

• Absence of a defect in oxygenation in a breathless patient should also prompt a search for non-respiratory causes such as metabolic acidosis and sepsis.

Circulation

• Quick head to toe survey to note and control bleeding

• Skin color, moisture, temperature

• Pulse quality, rate, regularity, volume

• Blood pressure

• Capillary refill (should be < 2 seconds)

• Chest Compressions / Positioning etc.

• Rapid initial assessment of circulatory status should concentrate on tissue perfusion and not just blood pressure.

• As a result of compensatory mechanisms, hypotension is a late feature of cardiovascular dysfunction.

• Evidence of inadequate tissue perfusion (decreased conscious level, skin mottling, cold peripheries, poor capillary refill, oliguria and metabolic acidosis) indicates a severely ill patient, even in the absence of hypotension.

• An indication of the type of shock (cardiogenic, distributive etc.) can be obtained by feeling the pulse and the peripheries and examining the jugular venous pressure.

Conscious state

• A marked reduction in conscious level indicates either that compensatory homeostatic mechanisms have been overwhelmed or severe neurological disease.

• In either case, the patient is severely ill and requires urgent supportive therapy.

• The pupillary response should be checked frequently.

Investigations

• These should not delay initial resuscitation but can be carried out while the patient is being resuscitated.

• Useful screening investigations include :

pulse oximetry, arterial blood gases, electrolytes, renal function tests, complete blood count and clotting.

2) Subsequent assessment

Part or all of this assessment may be carried out before initiating any treatment in less severely ill patients.

History

• Often it is not possible to take a full history from the patient and therefore other sources of information became more important.

• These include medical, nursing and ambulance staff, relatives, and notes and charts. As well as revealing the history the notes and charts may give useful information on the rate of deterioration. In post-operative patients the operation note may be particularly helpful.

• The history will reveal whether the patient falls into a group that is difficult to assess.

These include :

1. Young adults
2. Elderly or immunocompromised.
3. Trauma patients

Groups of patients who are difficult to assess

• The history is invaluable in the assessment of physiological reserve.

• An assessment of cardiopulmonary reserve can be obtained from the patient exercise tolerance.

• In assessing exercise tolerance it is Important to enquire about both distance covered and speed. For example

there is a considerable difference in exercise tolerance between the patient who can walk up one flight of stairs at a normal pace and the patient who takes 5 minutes to walk up one flight of stairs.

Examination

• While the aim of the initial examination is to detect life threatening physiological abnormalities and to determine appropriate supportive therapy, the focus of

Group	Difficulty
Young adults	Compensatory mechanisms in young patients tend to mask signs of severe illness until the illness is very advanced. Significant physiological abnormalities in these patients therefore indicate very severe illness.
Elderly or immuncompromised patients	The inflammatory response may be damped, again hiding signs of severe illness. In addition the physiological reserve of these patients is often severely compromised
Trauma patients	Notoriously difficult to assess due the multitude of possible Injuries and the effect of distracting pain making injuries difficult to localize In these patients a detailed history of the mechanism of Injury provides vital clues to likely injuries.

subsequent examination is to determine the underlying cause, in order to determine the appropriate definitive therapy.

• Examination should be repeated frequently to determine the response to therapy and because the clinical signs may change.

Investigations

• In addition to the Investigations carried out during the initial assessment,

- ✓ Liver function tests,
- ✓ calcium,phosphate,magnesium estimation
- ✓ a chest X-ray

are useful screening tests. Other investigations should be ordered on the basis of the history and clinical findings.

• If advanced radiological imaging is indicated, consideration should be given to ultrasound scanning because of the low risk of adverse effects and because it may obviate the need to transport the patient to the radiology department.

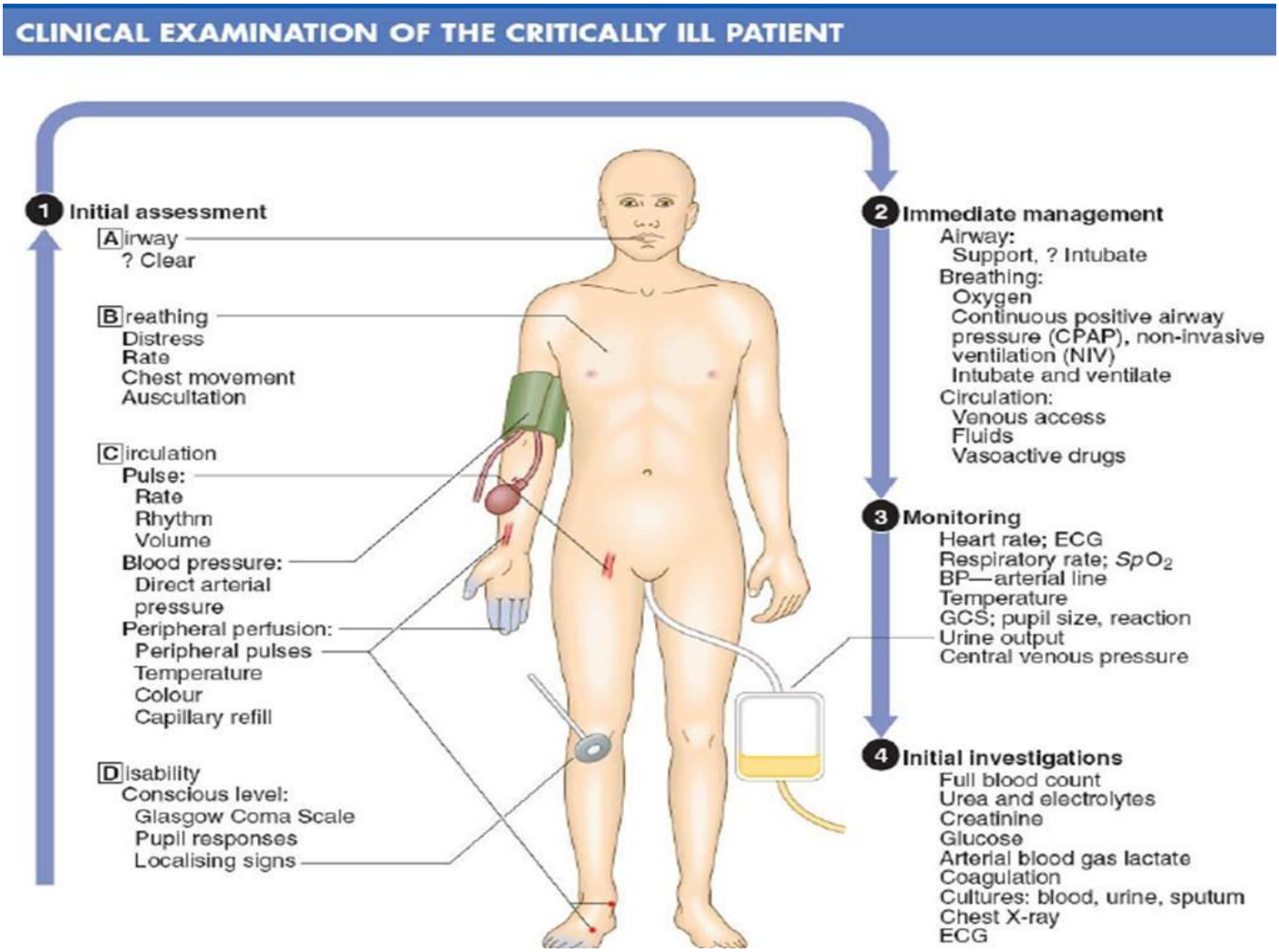
Clinical and laboratory features suggestive of severe illness

System	Feature
Cardiovascular	Tachycardia, hypotension, cold peripheries, skin mottling. Bradycardia may indicate a pre-terminal state.
Respiratory	Tachypnea, recession, use of accessory muscles of respiration, low oxygen saturation. Low respiratory rate may indicate impending respiratory arrest.
Renal	Oliguria
Nervous system	Decreased consciousness, confusion agitation, aggressive behavior
Metabolic	Acidosis, severe electrolyte abnormalities (particularly severe hyperkalemia and severe hyponatremia), severe anemia, thrombocytopenia, coagulopathy, elevated lactate.
Miscellaneous	Sweating

Review

• Following the primary assessment, initiation of emergency treatment and full assessment, a working diagnosis and plan for the subsequent management should be developed.

• This plan should include an ongoing review of the response to treatment, and a consideration of the appropriate placement of the patient, possibly in Intensive Care or another high care area.



ICU admission criteria

• Life support technology is intended to provide temporary support for patients with potentially reversible organ failure and is not a measure to conquer (defeat) death (advanced or terminally ill cases).

System approach

• Airway: compromise or impending compromise (e.g. burn).

• Respiratory failure: (not responding to conservative treatment).

- ✓ Type I Respiratory failure (hypoxia).
- ✓ Type II Respiratory failure (hypercapnia).

• Circulatory failure:

- ✓ Hypovolemic shock:
- ✓ Distributive shock: (e.g. septic, most common type in ICU).
- ✓ Cardiogenic shock: (usually treated in CCU).
- ✓ Obstructive shock:

• CNS emergency:

- ✓ Severe traumatic brain injury.
- ✓ Encephalitis.
- ✓ Refractory status epilepticus.
- ✓ Brain tumor (post operative).
- ✓ Stroke (post operative or post tPA).
- ✓ Altered LOC (poisoning or toxicity).

• Endocrine emergency:

- ✓ Diabetic: (severe DKA, HHS, recurrent hypoglycemia).
- ✓ Adrenal: (adrenal crisis).
- ✓ Thyroid: (thyrotoxicosis and myxedema coma).

• Visceral and metabolic:

- ✓ Acute liver failure or acute on chronic liver failure.
- ✓ Acute kidney injury (complicating sepsis or rhabdomyolysis).

- ✓ Severe electrolyte imbalance (requiring close monitoring).

• Obstetric emergency: (e.g. eclampsia & its complications).

• Postoperative cases:

- ✓ Major general surgery (e.g. large fluid shift).
- ✓ Major vascular surgery.
- ✓ Neurosurgery (brain tumor).

• Massive blood transfusion.

ICU Discharge Criteria

• Stable respiratory status.

• No or minimal oxygen support.

• Stable hemodynamic parameters.

• Inotropic support, vasodilators and antiarrhythmic drugs, and intracranial pressure monitoring are no longer required.

• Neurologic stability with control of Seizures.

• Close monitoring is no longer required.

MCQ TEST

1- Warning signs of a severely ill patient(all true except one)

a) Systolic blood pressure <90 or Meam<70mmHg

b) Heart rate <150 or <50 bpm
c) Conscious level GCS<12
d) Urine output 0.5 ml/kg/min
e) Potassium >6 mmol/l

2- Airway assessement in crically ill patient
a) Look for tachycardia
b) Look for tachypnea
c) Look for sweating
d) use of accessory muscles.
e) All the above

3- Which one is true regarding chest sounds
a) Inspiratory stridor is a rasping sound is a result of obstruction above or involving the larynx
b) Wheeze is usually heard on expiration as a result of the lower airways collapsing
c) Gurgling occur when secretion or liquid is present in the upper airways
d) Snoring occurs during partial occlusion of the oropharynx.
e) Crepitation is usually normal sound.

4- Tachypnea in critically ill patient(all true except one)
a) marked tachypnea is a useful marker of a severely ill patient
b) tachypnea is usually a more obvious than cyanosis.
c) It is a sign of compensatory mechanism
d) Could be caused by metabolic acidosis
e) Not caused by sepsis.

5- Evidence of inadequate tissue perfusion(all true except one)
a) decreased conscious level
b) skin mottling
c) hot peripheries
d) capillary refill more than 5 second
e) Oliguria

6- Which one is false regarding difficult assessing of this patient

a) In young adults compensatory mechanism is mask sign
b) In Elderly patient the inlammoatory response is inhibited
c) Immunocompromised also difficult to assess
d) Trauma patients difficult due to pain and multiple injury
e) Female more difficult than male

7- Clinical and laboratory features suggestive of severe illness(all true except one)
a) High lactate level
b) Tachypnea
c) Oliguria
d) Agitation
e) Sweating

8- All the following are ICU admission criteria except one
a) adrenal crisis
b) sever hyperkalemia
c) diabetic ketoacidosis
d) blood transfusion
e) eclampsia

9- discharge criteria from ICU(all true except one)
a) Stable respiratory status.
b) No or minimal oxygen support.
c) Stable hemodynamic parameters.
d) Inotropic support is longer required.
e) Neurologic stability with control of Seizures.

Patient Monitoring in ICU

Monitoring definition

Interpreting available clinical data to help recognize present or future mishaps or unfavorable system conditions

Monitoring goals

• Enhances (but not replaces) the vigilance of the intensivist.

• Provides means to assess physiological function.

• Provides information that improves the safety of patient.

Monitoring guidelines

• Qualified personnel should be present in the ICU

• Physical examination, Assessment & Diagnosis

remain the most important tools available to the intensivist

Basic Monitoring

1) Oxygenation

2) Ventilation

3) Circulation

4) Temperature

should be continually evaluated.

1) Oxygenation

Objective: To ensure adequate oxygen concentration in the delivered gas and in the blood.

Methods

1) Clinical: color, respiratory pattern (rate, rhythm, depth, etc.), equal air entry, wheezing, crackles.

2) Delivered gas: the concentration of oxygen in the patient breathing system shall be measured by an oxygen analyzer.

3) Blood oxygenation: a quantitative method of assessing oxygenation such as pulse oximetry shall be employed and ABGs show PaO2.

2) Ventilation

A. Every patient should have the adequacy of ventilation continually evaluated.

1) Clinical signs such as chest movement and auscultation of breath sounds are useful.

2)

* ABGs show PaCO2
* Continual end-tidal carbon dioxide analysis
* Monitoring of the volume of expired gas is strongly encouraged in mechanically ventilated patients.

B. When ventilation is controlled by a mechanical ventilator, there shall be in continuous use a device that is capable of detecting disconnection of components of the breathing system. The device must give an audible signal.

3) Circulation

• Every patient shall have the ECG continuously.

• Every patient shall have BP and HR determined and evaluated at close intervals.

• Other clinical evaluation methods like Palpation of a pulse, Auscultation of heart sounds & Oximetry

Other used monitors:

• Temperature [pharyngeal, axillary, esophageal]

• Urine output

• Central venous line: measuring CVP

• Arterial line:

– Continuous BP monitoring

– Easy access allowing for frequent ABGs

Less frequently used monitors:

• Swan-Ganz catheter, PCWP: pulmonary artery pressures, cardiac output

• ICP monitoring

• EEG

• Transesophageal echocardiography (TEE)

Central venous pressure (CVP)

is the pressure recorded from the right atrium or superior vena cava and is representative of the filling pressure of the right side of the heart?

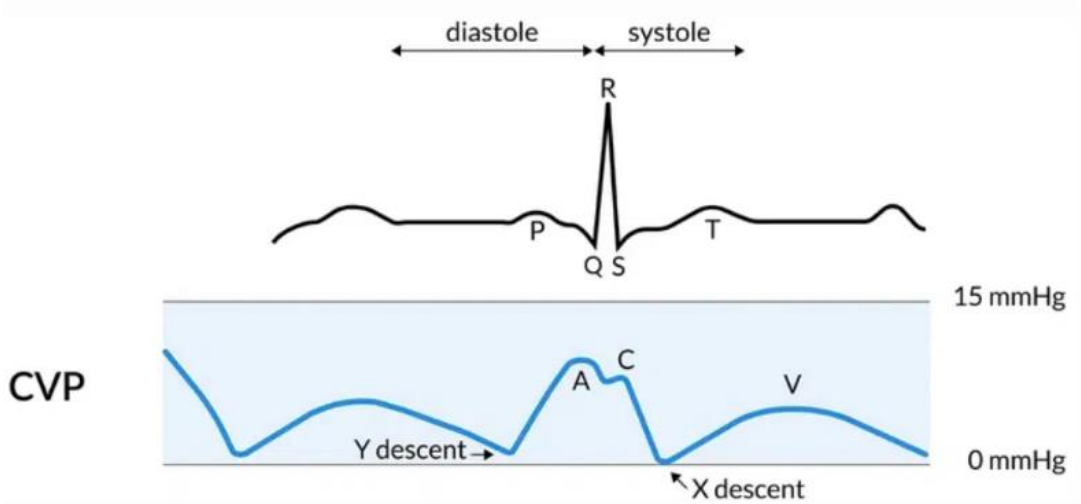
- CVP monitoring in the critically ill is established practice but the traditional belief that CVP reflects ventricular preload and predicts fluid responsiveness has been challenged by a large body of evidence
- CVP represents the driving force for filling the right atrium and ventricle
- normal is 0-6mmHg in a spontaneously breathing non-ventilated patient

MEASUREMENT

- recorded at the end of expiration
- measured by transducing the waveform of a central venous line
- electronic transducer placed & zeroed at the level of the RA (the "hemostatic axis" – usually the 4th intercostal space in the mid-axillary line is used)

CVP WAVEFORM

a = atrial contraction
c = closing and bulging of the tricuspid valve
x = atrial relaxation, with downward movement of the tricuspid valve during ventricular contraction
v = passive filling of atrium (tricuspid valve still closed)
y = ventricular filling with opening of the tricuspid valve



USE

Value and waveform assist with diagnosis of:

- right ventricular infarction
- right heart failure and cor pulmonale

- tamponade
- Tricuspid regurgitation or stenosis
- Complete heart block
- Constrictive pericarditis

Determining:

- mechanical atrial capture with AV pacing
- presence of P waves in cases of SVT
- differential diagnosis of shock state
- correct central line placement

CAUSES OF RAISED CVP

- Right ventricular failure
- Tricuspid stenosis or regurgitation
- Pericardial effusion or constrictive pericarditis
- Superior vena caval obstruction
- Fluid overload
- Hyperdynamic circulation
- High PEEP settings

Low Central Venous Pressure

Some factors that can decrease central venous pressure are hypovolemia or vasodilation. Either of these would decrease venous return and thus decrease the central venous pressure. A decrease in central venous pressure is noted when there is more than 10% of blood loss or shift of blood volume. A decrease in intrathoracic pressure caused by forced inspiration causes the vena cava to collapse which decreases the venous return and, in turn, decreases the central venous pressure.

CVP WAVEFORM ANALYSIS

Waveform abnormalities may indicate specific pathologies:

- Dominant a wave – pulmonary hypertension, tricuspid stenosis, pulmonary stenosis
- Cannon a wave – complete heart block, ventricular tachycardia with atrio-ventricular dissociation
- Dominant v wave – tricuspid regurgitation
- Absent x descent – atrial fibrillation
- Exaggerated x descent – pericardial tamponade, constrictive pericarditis
- Sharp y descent – severe tricuspid regurgitation, constrictive pericarditis
- Slow y descent – tricuspid stenosis, atrial myxoma
- Prominent x and y descent – right ventricular infarction

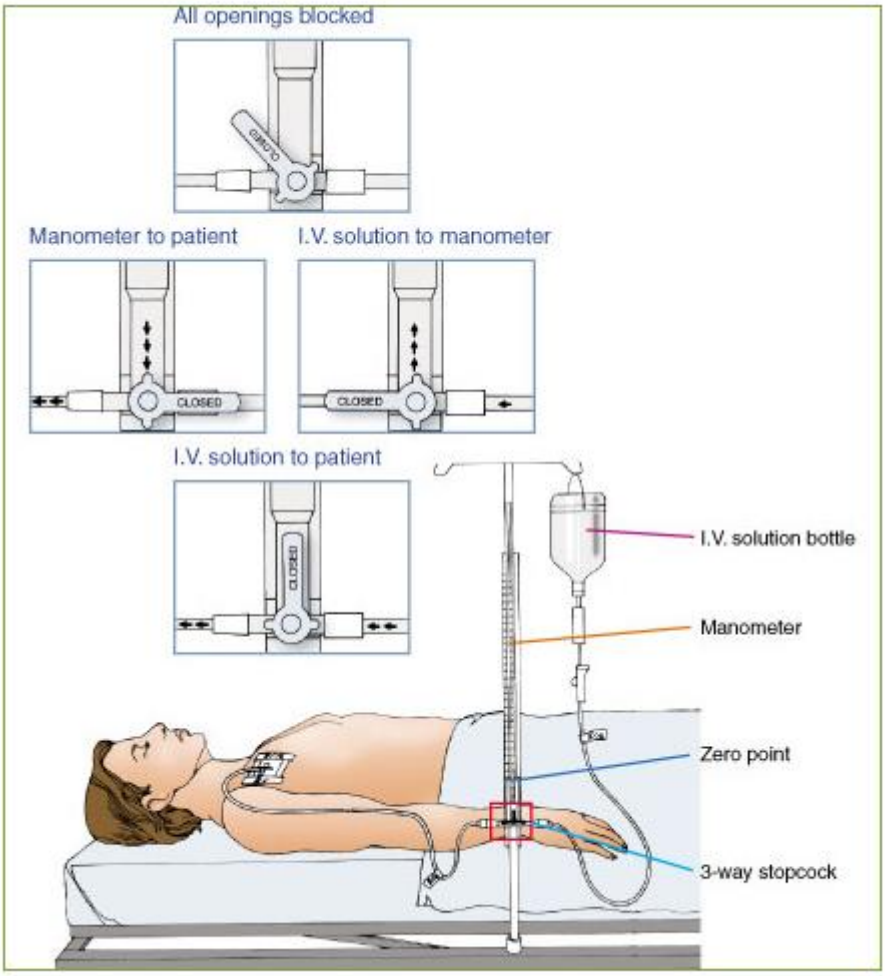
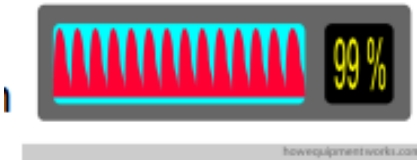


Fig 1: A water manometer setup.

Airway / Respiratory Axis

- Oxygenation
- Ventilation
- Correct ETT placement
- ETT cuff pressure (keep between 20-30 cm H2O)
- Airway pressure

Respiratory Monitoring



Various alarms by the ventilator:

– Low airway pressure: leakage, disconnection.

– High airway pressure: kink, biting of the tube, blocked tube, bronchospasm.

– Low expired tidal volume: leakage.

– Apnea alarm: disconnection.

– O2 sensor failure: (unfortunately common in many of our ventilators).

– Flow sensor failure: (unfortunately common in many of our ventilators).

NEVER ignore an alarm by the ventilator!

PEAK INSPIRATORY PRESSURE (PIP)

• Depends on: Airway resistance (Raw) & lung compliance (Cl).

• During controlled ventilation look for increase airway resistance (e.g., bronchospasm, kinked ETT) or decrease in pulmonary compliance (e.g., pulmonary congestion).

Oxygenation and ventilation

• Pulse oximetry (vital sign for Oxygenation)

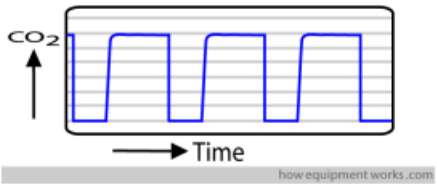
– Measures O2 saturation in blood

• Capnography (vital sign for ventilation &perfusion)

– Measures CO2 in the airway

– Provides a breath-to-breath status of ventilation

Cardiovascular Axis



1. Arterial Blood Pressure
2. Electrocardiography
3. Central Venous Catheterization
4. Pulmonary Artery Catheterization
5. Cardiac Output:

- Thermodilution
- Dye Dilution
- Pulse Contour Devices
- Esophageal Doppler
- Fick Principle
- Echocardiography
- Thoracic Bioimpedance

Electrolyte / Metabolic Axis

• Fluid balance

• Sugar

• Electrolytes

• Acid-base balance

• Nutritional status

Visual Monitoring

• Respiratory: patient color, respiratory pattern (accessory muscle use etc.)

• Patient monitor numbers and waveforms

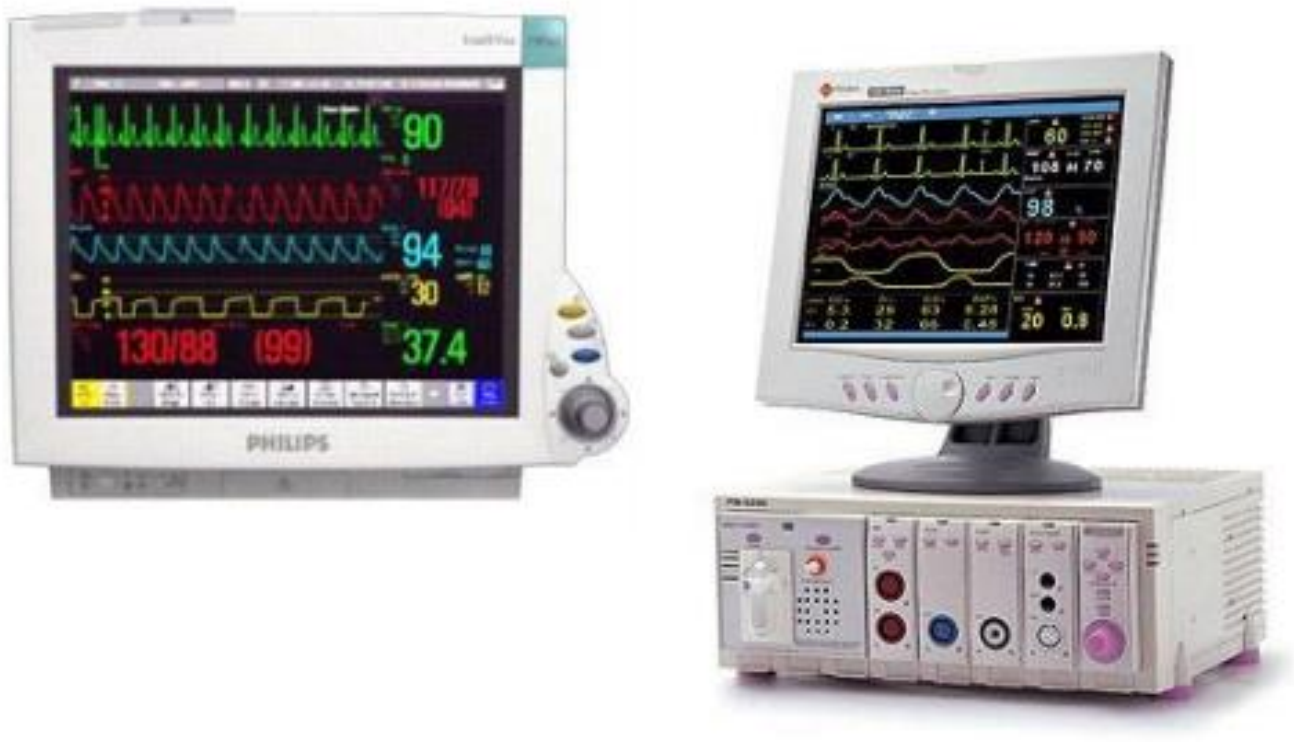
• Bleeding/coagulation

• Diaphoresis / movements

• Line quality (is my IV reliable?)

• Positioning safety review

High Tech Patient Monitoring



Examples of Multiparameter Patient Monitors

Clinical Tips

• ALWAYS remember that your clinical sense & judgement is superior to any monitor.

• You are a clinician, not a technician.

• The monitor is present to help you, not to be ignored and not to cancel your brain.

• Never panic Particularly when the patient is going to die and you have no idea why.

• If a monitor gives an abnormal value, such as low oxygen saturation, Check the patient first then the equipment.

• Know where the defibrillator is kept in the unit and how it works

• All 1 ml ampoules look the same (check very carefully)

• Always label all syringes

MCQ TEST

1- Alarms in ICU ventilator (all true except one)

a) Low airway pressure: leakage,
b) High airway pressure: disconnection.
c) Low expired tidal volume: leakage .
d) Apnea alarm: disconnection .
e) O2 sensor failure: (unfortunately common in many of our ventilators) .

2- PEAK INSPIRATORY PRESSURE (PIP) (all true except one)

a) Depends on airway resistance (Raw)

b) Not affected by lung compliance (Cl) .
c) During controlled ventilation look for increase airway resistance
d) Increased by bronchospasm
e) Increased by kinked ETT

3- All the following are for circulation monitoring except one
a) Urine output
b) Central venous line: measuring CVP
c) Arterial line Invasive BP mentoring :
d) ECG
e) Glasgow coma scale

4- Causes of high CVP
a) Right ventricular failure
b) Pericardial effusion
c) Superior vena caval obstruction
d) Fluid overload
e) low PEEP settings

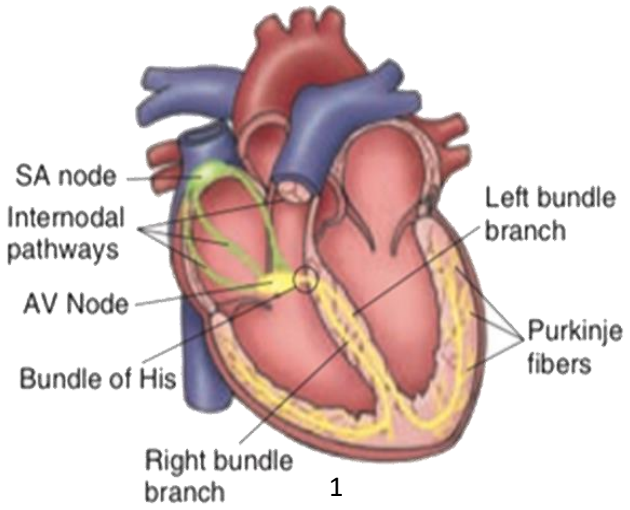
5- Waveform abnormalities of CVP and specific pathologies (all true except one)
a) Dominant a wave – pulmonary embolism
b) Cannon a wave – complete heart block
c) Dominant v wave – tricuspid regurgitation
d) Absent x descent – atrial fibrillation
e) Exaggerated x descent – pericardial tamponade

Electrocardiography (ECG)

Physiology of the heart

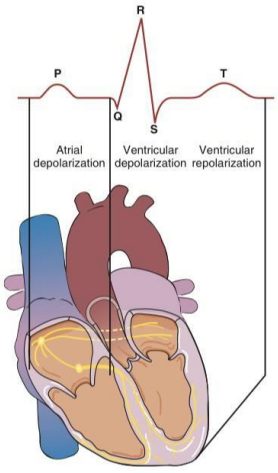
Properties of Cardiac Cells	
Property	**Ability**
Automaticity	Generates electrical impulse independently, without involving the nervous system.
Excitability	Responds to electrical stimulation.
Conductivity	Passes or propagates electrical impulses from cell to cell.
Contractility	Shortens in response to electrical stimulation.

Electrical Conduction System of the heart



Conduction System Structures and Functions	
Structure	**Function and Location**
Sinoatrial (SA) node	Dominant pacemaker of the heart, located in upper portion of right atrium. Intrinsic rate 60–100 bpm.
Internodal pathways	Direct electrical impulses between SA and AV nodes.
Atrioventricular (AV) node	Part of AV junctional tissue. Slows conduction, creating a slight delay before impulses reach ventricles. Intrinsic rate 40–60 bpm.
Bundle of His	Transmits impulses to bundle branches. Located below AV node.
Left bundle branch	Conducts impulses that lead to left ventricle.
Right bundle branch	Conducts impulses that lead to right ventricle.
Purkinje system	Network of fibers that spreads impulses rapidly throughout ventricular walls. Located at terminals of bundle branches. Intrinsic rate 20–40 bpm.

Electrophysiology	
Action	**Effect**
Depolarization	The electrical charge of a cell is altered by a shift of electrolytes on either side of the cell membrane. This change stimulates muscle fiber to contract.
Repolarization	Chemical pumps re-establish an internal negative charge as the cells return to their resting state.



Depolarization and repolarization of the heart.

♥ **Clinical Tip:** Mechanical and electrical functions of the heart are influenced by proper electrolyte balance. Important components of this balance are sodium, calcium, potassium, and magnesium.

ECG

1- Electrocardiography is the process of producing an electrocardiogram (ECG or EKG , a recording of the heart's electrical activity through repeated cardiac cycles.

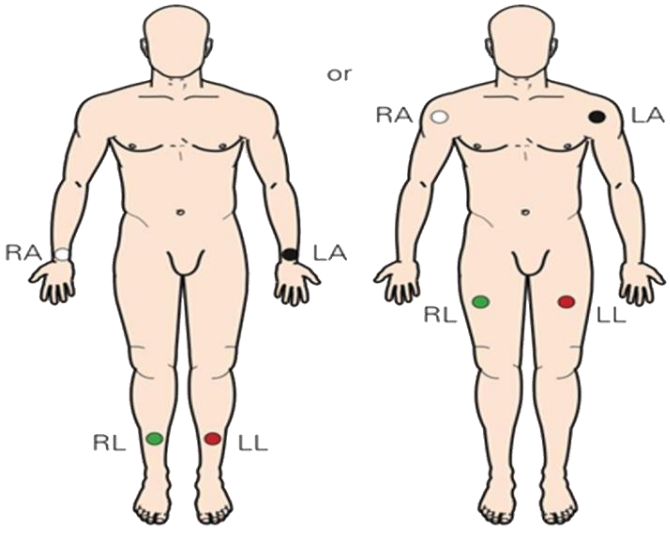
2- It is an electrogram of the heart which is a graph of voltage versus time of the electrical activity of the heart using electrodes placed on the skin.

3- These electrodes detect the small electrical changes that are a consequence of cardiac muscle depolarization followed by repolarization during each cardiac cycle (heartbeat).

Limb Lead

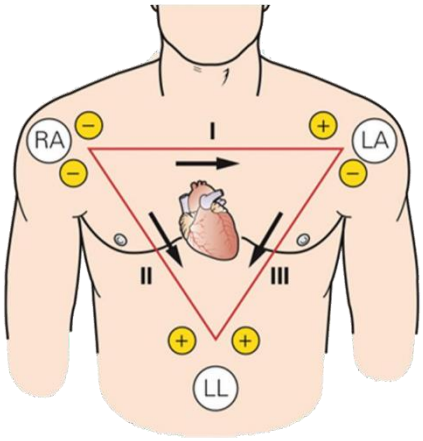
Electrodes are placed on the right arm (RA), left arm (LA), right leg (RL), and left leg (LL). With only four electrodes, six leads are viewed.

- Standard leads: I, II, III
- Augmented leads: aVR, aVL, aVF



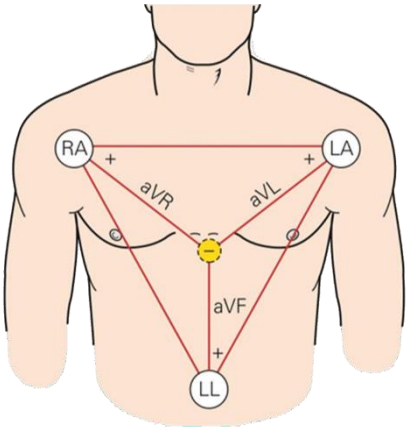
Standard Limb Lead

Elements of Standard Limb Leads			
Lead	Positive Electrode	Negative Electrode	View of Heart
I	LA	RA	Lateral
II	LL	RA	Inferior
III	LL	LA	Inferior



Augmented Limb Leads

Elements of Augmented Limb Leads		
Lead	**Positive Electrode**	**View of Heart**
aVR	RA	None
aVL	LA	Lateral
aVF	LL	Inferior



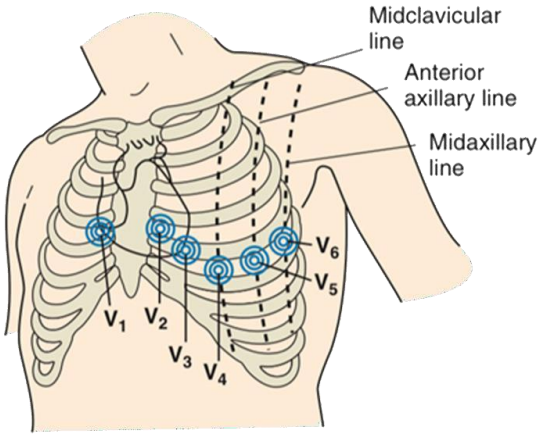
Limb Lead

1 - Leads I, II, and III are bipolar leads, which consist of two electrodes of opposite polarity (positive and negative). The third (ground) electrode minimizes electrical activity from other sources.

2 - Leads aVR, aVL, and aVF are unipolar leads and consist of a single positive electrode and a reference point (with zero electrical potential) that lies in the center of the heart's electrical field.

Chest Leads

Elements of Chest Leads		
Lead	**Positive Electrode Placement**	**View of Heart**
V_1	4th Intercostal space to right of sternum	Septum
V_2	4th Intercostal space to left of sternum	Septum
V_3	Directly between V_2 and V_4	Anterior
V_4	5th Intercostal space at left midclavicular line	Anterior
V_5	Level with V_4 at left anterior axillary line	Lateral
V_6	Level with V_5 at left midaxillary line	Lateral

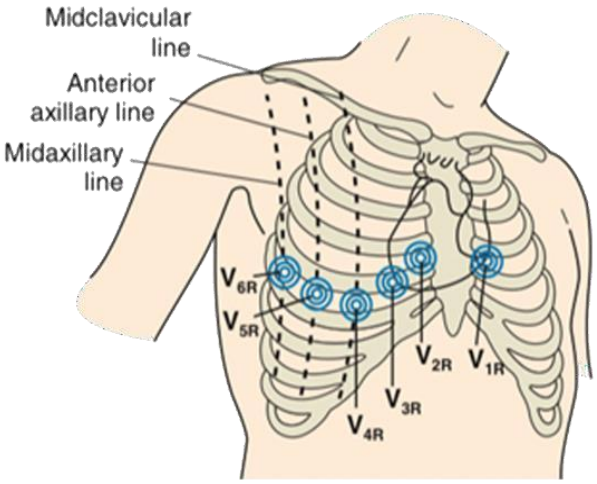


The Right-Sided 12-Lead ECG

- The limb leads are placed as usual but the chest leads are a mirror image of the standard 12-lead chest placement.
- The ECG machine cannot recognize that the leads have been reversed. It will still print "V_1–V_6" next to the tracing. Be sure to cross this out, and write the new lead positions on the ECG paper.

The Right-Sided 12-Lead ECG	
Chest Leads	**Position**
V_{1R}	4th Intercostal space to left of sternum
V_{2R}	4th Intercostal space to right of sternum
V_{3R}	Directly between V_{2R} and V_{4R}
V_{4R}	5th Intercostal space at right midclavicular line
V_{5R}	Level with V_{4R} at right anterior axillary line
V_{6R}	Level with V_{5R} at right midaxillary line

♥ **Clinical Tip:** Patients with an acute inferior MI should have right-sided ECGs to assess for possible right ventricular infarction.



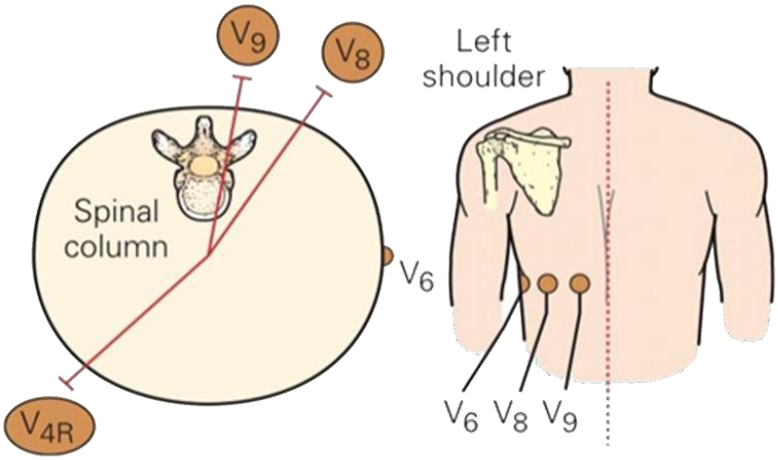
The 15-Lead ECG

Areas of the heart that are not well visualized by the six chest leads include the wall of the right ventricle and the posterior wall of the left ventricle. A 15-lead ECG, which includes the standard 12 leads plus leads V_{4R}, V_8, and V_9, increases the chance of detecting an MI in these areas.

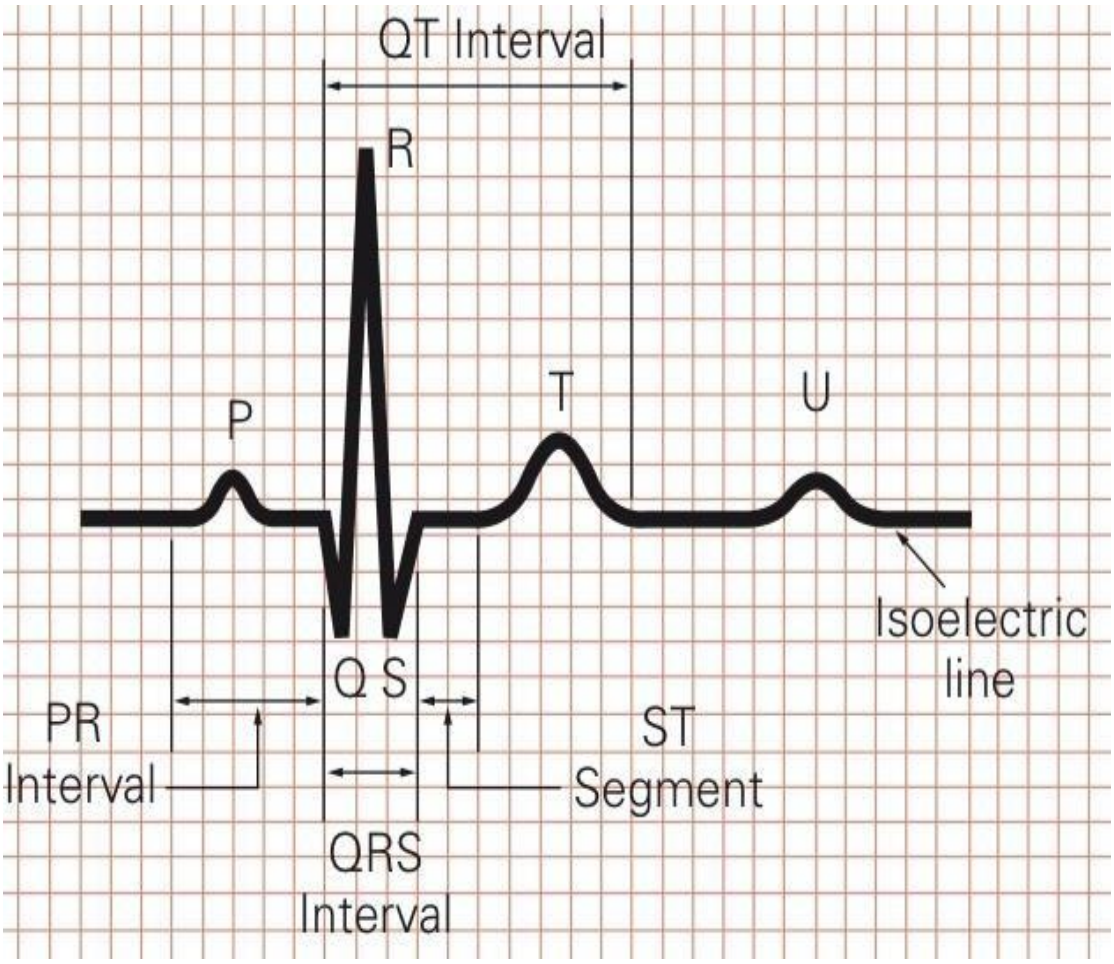
The 15-Lead ECG

Chest Leads	Electrode Placement	View of Heart
V_{4R}	5th Intercostal space in right anterior midclavicular line	Right ventricle
V_8	Posterior 5th intercostal space in left midscapular line	Posterior wall of left ventricle
V_9	Directly between V_8 and spinal column at posterior 5th intercostal space	Posterior wall of left ventricle

♥ **Clinical Tip:** Use a 15-lead ECG when the 12-lead is normal but the history is still suggestive of an acute infarction.



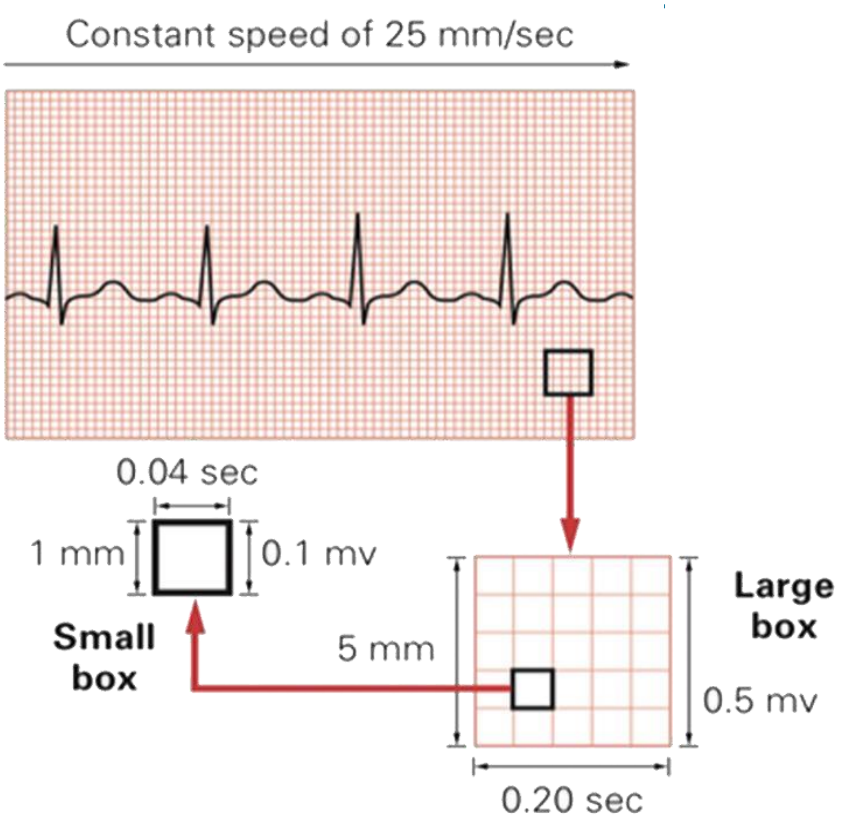
Components of an ECG Tracing



Components of an ECG

Electrical Components	
Deflection	**Description**
P Wave	First wave seen Small rounded, upright (positive) wave indicating atrial depolarization (and contraction)
PR Interval	Distance between beginning of P wave and beginning of QRS complex Measures time during which a depolarization wave travels from the atria to the ventricles
QRS Interval	Three deflections following P wave Indicates ventricular depolarization (and contraction) Q Wave: First negative deflection R Wave: First positive deflection S Wave: First negative deflection after R wave
ST Segment	Distance between S wave and beginning of T wave Measures time between ventricular depolarization and beginning of repolarization
T Wave	Rounded upright (positive) wave following QRS Represents ventricular repolarization
QT Interval	Measured from beginning of QRS to end of T wave. Represents total ventricular activity.
U Wave	Small rounded, upright wave following T wave Most easily seen with a slow HR. Represents repolarization of Purkinje fibers.

Recording of the ECG



Reporting an ECG recording

The reporting of an ECG recording is best done in a methodical manner to ensure that the report is comprehensive and doesn't overlook any potentially important details

Patient Data

Begin by checking key information on the ECG and/or request form relating to the patient:

- Patient name
- Date of birth
- Identification number (e.g. hospital number)
- Reason for the request
- Relevant past medical history

- Relevant medication

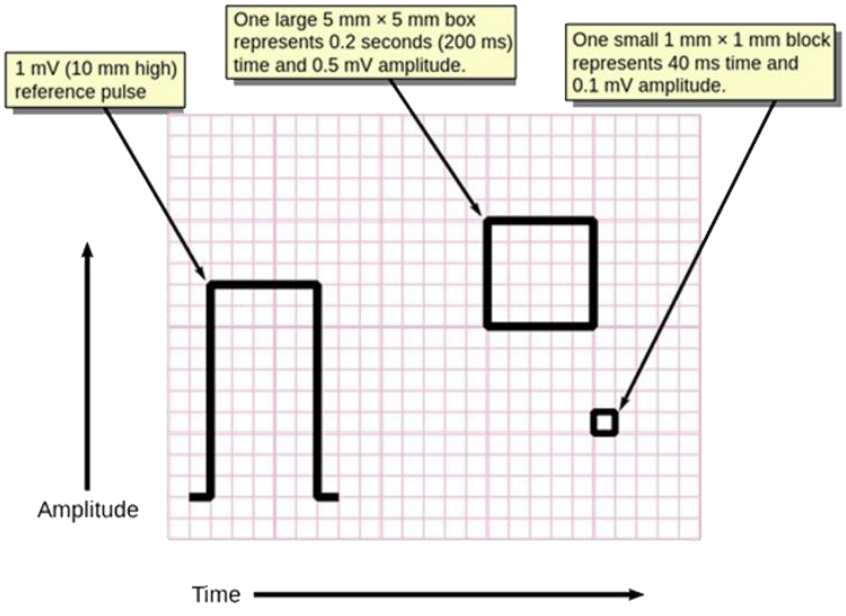
Technical Data

- ❖ Date and time of recording
- ❖ Paper speed and calibration
- ❖ Technical quality
- ❖ Any atypical settings
- ❖ Additional leads (e.g. posterior leads, right-sided chest leads)
- ❖ Physiological manoeuvres (e.g., ECG recorded during deep inspiration)
- ❖ Diagnostic or therapeutic manoeuvres (e.g. ECG recorded during carotid sinus massage)

Stander ECG

ECGs are normally printed on a grid. The horizontal axis represents time and the vertical axis represents voltage. The standard values on this grid are shown in the adjacent image at 25mm/sec:[58]

A small box is 1 mm × 1 mm and represents 0.1 mV × 0.04 seconds.
A large box is 5 mm × 5 mm and represents 0.5 mV × 0.20 seconds.



ECG Fundamentals

Next, report on the fundamental features of the ECG recording itself, namely:

1- **Rate**
2- **Rhythm**

- Supraventricular
- Ventricular
- Conduction problems

3- **Axis**

ECG Details

Next, review the individual features of the ECG using a step-by-stepapproach.
Describe:

- P wave
- PR interval
- Q wave
- QRS complex
- ST segment
- T wave
- QT interval
- U wave

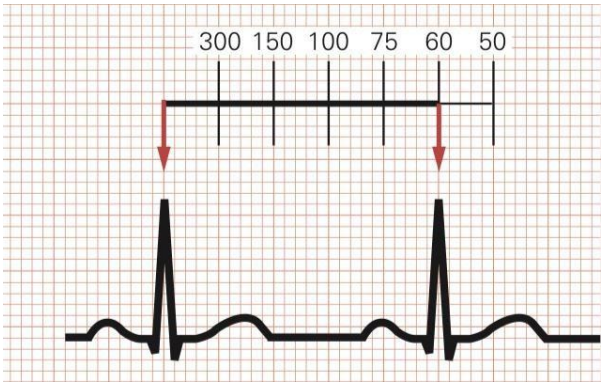
Heart Rate

Methods for Calculating Heart Rate:

Method 1: Count Large Boxes

Regular rhythms can be quickly determined by counting the number of large graph boxes between two R waves.

* That number is divided into 300 to calculate bpm.
* The rates for the first one to six large boxes can be easily memorized.
* Remember: 60 sec/min divided by 0.20 sec/large box 300 large boxes/min.



Method 2: Count Small Boxes

Sometimes it is necessary to count the number of small boxes between two R waves for fast heart rates.

* That number is divided into 1500 to calculate bpm.
* Remember: 60 sec/min divided by 0.04 sec/small box 1500 small boxes/min.
* Examples: If there are six small boxes between two R waves: 1500/6 250 bpm.
* If there are ten small boxes between two R waves: 1500/10 150 bpm

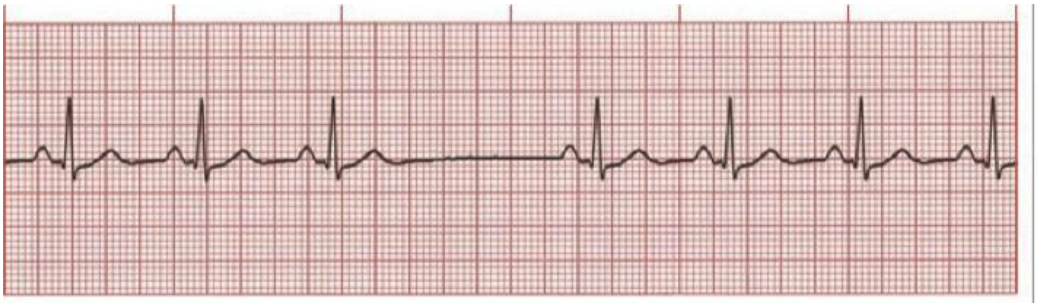
Methods 1 and 2 for Calculating Heart Rate

Methods 1 and 2 for Calculating Heart Rate			
Number of Large Boxes	Rate/Min	Number of Small Boxes	Rate/Min
1	300	2	750
2	150	3	500
3	100	4	375
4	75	5	300
5	60	6	250
6	50	7	214
7	43	8	186
8	38	9	167
9	33	10	150
10	30	11	136
11	27	12	125
12	25	13	115
13	23	14	107
14	21	15	100
15	20	16	94

Method 3: Six-Second ECG Rhythm Strip

* The best method for measuring irregular rates with varying R-R intervals is to count the number of R waves in a 6-sec strip and multiply by 10

* This gives the average number of bpm



Using 6-sec ECG rhythm strip to calculate heart rate. Formula: 7 × 10 = 70 bpm

Heart Rate

* Once you have measured the heart rate, you need to decide whether it is normalor abnormal.
* As a general rule, a heart rate between 60 and 100/min is normal.
* If the rate is below 60/min, the patient is said to be bradycardic. With a heart rate above 100/min, the patient is tachycardic.
* Therefore, the two questions you need to ask about heart rate are:

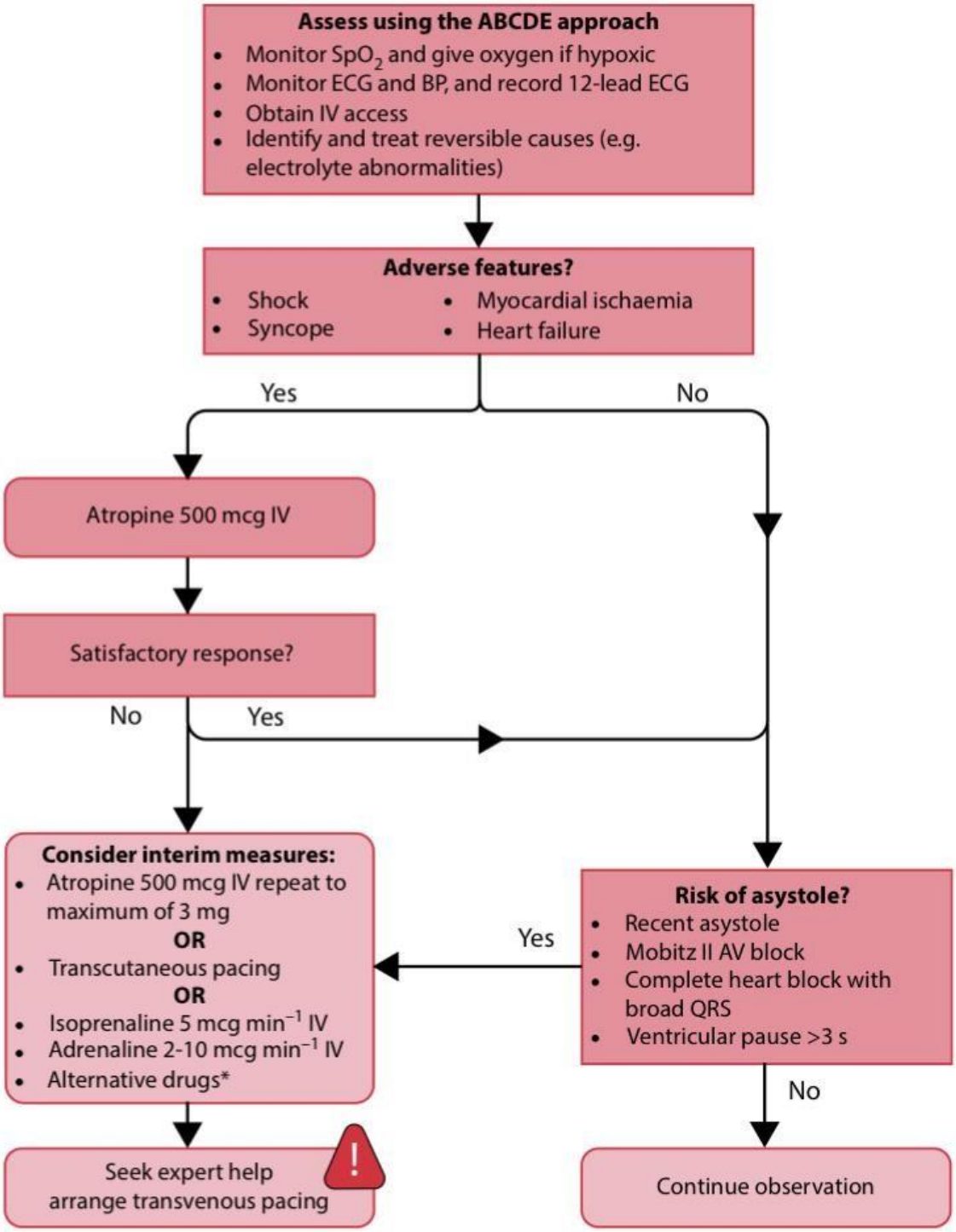
1. Is the heart rate below 60/min?
2. Is the heart rate above 100/min?

Is The Heart Rate Below 60/Min?

* Bradycardia is defined as a heart rate below 60/min. Identification of the cardiac rhythm and any conduction disturbances is essential, and this is discussed in the following chapters.
* Problems to consider in the bradycardic patient are:

1. Sinus bradycardia
2. Sick sinus syndrome

Management of patient with bradycardia



*** Alternatives include:**

- Aminophylline
- Dopamine
- Glucagon (if bradycardia is caused by beta-blocker or calcium channel blocker)
- Glycopyrrolate (may be used instead of atropine)

Is The Heart Rate Above 100/Min?

* Tachycardia is defined as a heart rate above 100/m
* When Begin the process of identification by checking whether the QRS complexes are:
    1. Narrow (<3 small squares)
    2. Broad (>3 small squares)

Narrow-complex tachycardias

always arise from above the ventricles – that is, they are supraventricular in origin.

The possibilities are:

• Sinus tachycardia

• Atrial tachycardia

• Atrial flutter

• Atrial fibrillation

• AV re-entry tachycardia (AVRT(

• AV nodal re-entry tachycardia (AVNRT)

Broad-complex tachycardia:

- Ventricular tachycardia
- Accelerated idioventricular rhythm
- Torsade's de pointes

Broad-complex tachycardia should also make you think of ventricular arrhythmias.

Management of tachycardia depends on the underlying rhythm

1. The first step, as with managing a bradycardia, is to assess the urgency of the situation – in the peri-arrest situation, use the ABCDE approach and assess the patient for adverse features

2. Clues to the nature of the arrhythmia may be found in the patient's history.

3. Ask the patient about:

- How any palpitations start and stop (sudden or gradual(
- Whether there are any situations in which they are more likely to happen (e.g. during exercise, lying quietly in bed(
- How long they last
- Whether there are any associated symptoms (dizziness, syncope, falls, fatigue, breathlessness and chest pain(
- Also ask the patient to 'tap out' how the palpitations feel – this will give you clues about the rate or slow) and rhythm (regular or irregular).

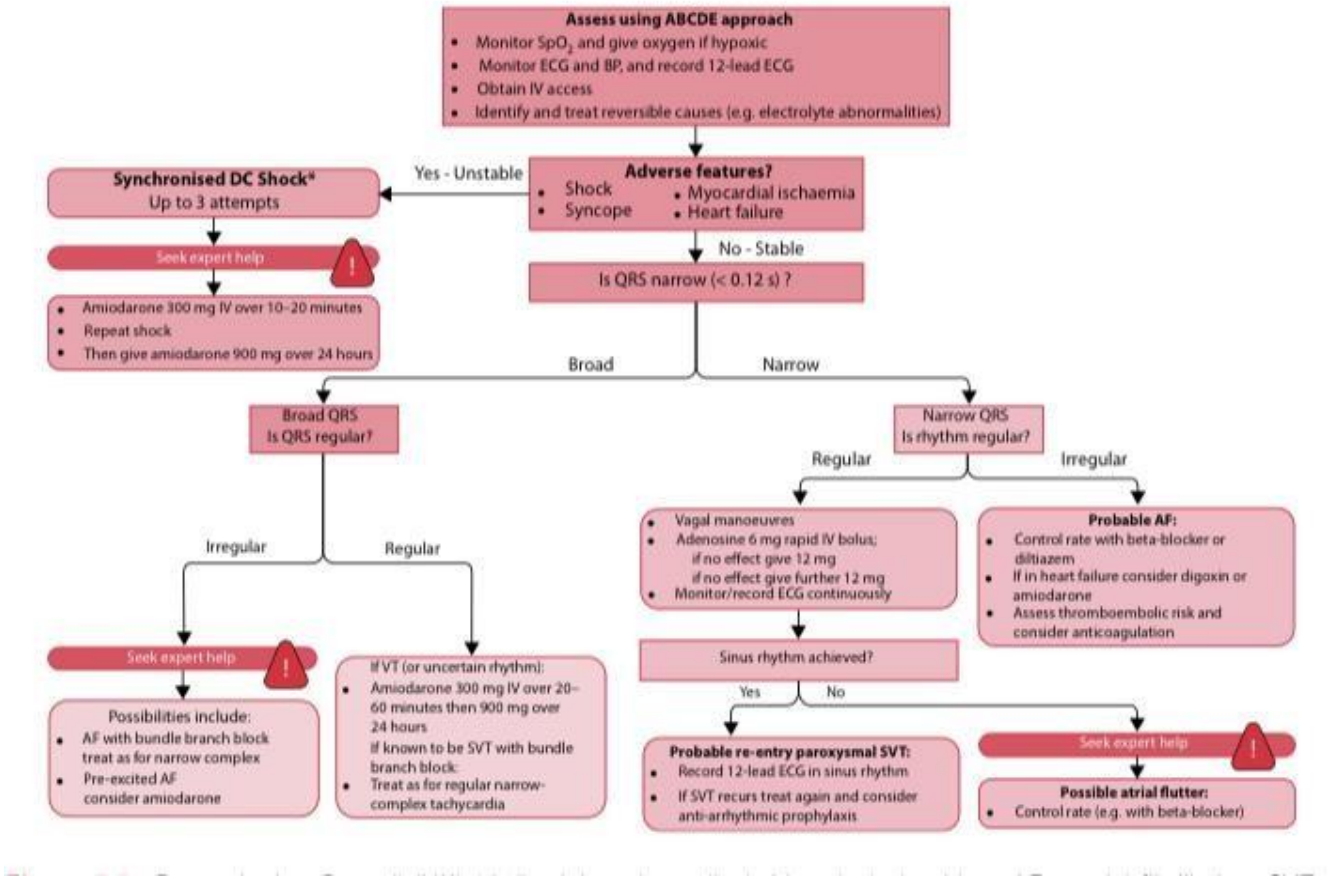
4. Also enquire about symptoms of related disorders (e.g. hyperthyroidism) and obtain a list of current medications.

5. Check for any drugs (e.g., salbutamol) that can increase the heart rate (positively chronotropic)

6. Do not forget to ask about caffeine intake (e.g., coffee, tea and energy drinks)

7. A thorough examination is always important, looking for evidence of hemodynamic disturbance (hypotension, cardiac failure and poor peripheral perfusion) and coexistent disorders (e.g., thyroid goiter)

8. Use the history, examination and further investigations (e.g., plasma electrolytes, thyroid function tests) to reach a diagnosis



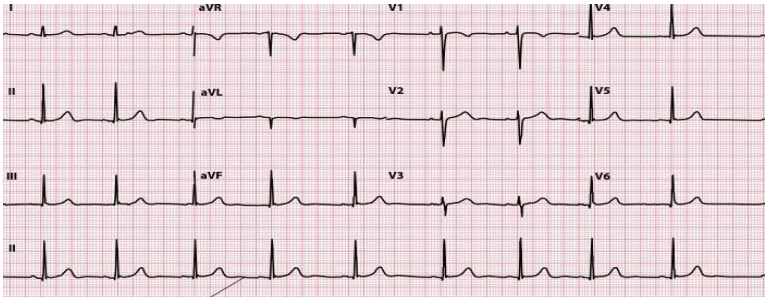
An Approach to Heart Rhythms

To identify the cardiac rhythm with confidence you need to begin with a rhythm

strip - a prolonged recording of the ECG from just one lead. Most ECG machines automatically include a rhythm strip at the bottom of a 12-lead ECG

The rhythm strip:

The standard lead used for the rhythm strip is lead II, but alternative leads can be selected if it helps to clarify the cardiac rhythm



Identifying The Cardiac Rhythm

When you analyze the cardiac rhythm, always keep in mind the two primary questions that you are trying to answer:

Where does the impulse arise from?

- Sinoatrial (SA) node
- Atria
- Atrioventricular (AV) junction
- Ventricles

How is the impulse conducted?

- Normal conduction
- Impaired conduction
- Accelerated conduction (e.g. Wolff-Parkinson-White [WPW] syndrome)

The following seven questions will help you to narrow down the possible diagnoses:

1. How is the patient?
2. Is ventricular activity present?
3. What is the ventricular rate?
4. Is the ventricular rhythm regular or irregular?
5. Is the QRS complex width normal or broad?
6. Is atrial activity present?
7. How are atrial activity and ventricular activity related?

1- How is the patient?

The clinical context will also help you decide how urgently to deal with an arrhythmia. When assessing a 'sick' patient, use the ABCDE approach:

- **Airway**: Check for any evidence of airway obstruction
- **Breathing**: Assess the patient's breathing, paying attention to respiratory rate, chest percussion and auscultation, and oxygenation
- **Circulation**: Assess the patient's circulation, including pulse rate, blood pressure and capillary refill time
- **Disability**: Assess level of consciousness and neurological status
- **Exposure**: Ensure adequate exposure to permit a full examination

As you assess a patient with an arrhythmia, be alert for 'adverse features' which indicate hemodynamic instability:

- Shock: As evidenced by hypotension (systolic blood pressure <90 mmHg), clamminess, sweating, pallor, confusion or reduced conscious level
- Syncope: As a consequence of cerebral hypoperfusion
- Myocardial ischemia: Indicated by ischemic chest pain and/or ischemic ECG changes
- Heart failure: Pulmonary oedema, elevated jugular venous pressure, peripheral/sacral oedema

2- Is Ventricular Activity Present ?

Look at the ECG as a whole for the presence of electrical activity. If there is none,

Assess:

- The patient (do they have a pulse?)
- The electrodes (has something become disconnected?)
- The gain setting (is the gain setting on the monitor too low?)

If QRS complexes are present, move on to the next question.

3- What Is the Ventricular Rate?

Ventricular activity is represented on the ECG by QRS complexes. Once you have calculated the ventricular rate, you will be able to classify the rhythm as:

- Bradycardia (<60 beats/min)

- Normal (60-100 beats/min)
- Tachycardia (>100 beats/min)

4- Is The Ventricular Rhythm Regular or Irregular?

Having determined the ventricular rate, go on to assess regularity.

Look at the spacing between QRS complexes - is it the same throughout the rhythm strip? Irregularity can be subtle, so it is useful to measure the distance between each QRS complex.

One way to do this is to place a piece of paper alongside the rhythm

strip and make a mark on it next to every QRS complex. By moving the marked paper up and down along the rhythm strip, you can soon see if the gaps between the QRS complexes are the same or vary.

Once you have assessed the regularity, you will be able to classify the ventricular rhythm as:

- Regular (equal spacing between QRS complexes)
- Irregular (variable spacing between QRS complexes)

Table 6.2 Regular and irregular cardiac rhythms

- Regular rhythms
  - Sinus rhythm
  - Sinus bradycardia
  - Sinus tachycardia
  - Atrial flutter (if constant AV block, e.g. 2:1)
  - Atrial tachycardia
  - AV re-entry tachycardia (AVRT)
  - AV nodal re-entry tachycardia (AVNRT)
  - Accelerated idioventricular rhythm
  - Monomorphic ventricular tachycardia (VT)
  - Polymorphic ventricular tachycardia (torsades de pointes)
  - Third-degree AV block (if regular escape rhythm)
- Irregular rhythms
  - Sinus arrhythmia (rate varies with respiration)
  - Ectopic beats (atrial, junctional, ventricular)
  - Atrial fibrillation
  - Atrial flutter (if variable AV block)
  - Sinus arrest and SA block
  - Mobitz type I second-degree AV block
  - Mobitz type II second-degree AV block

5- Is The QRS Complex Width Normal or Broad?

By answering this question, you will have narrowed down the origin of the impulse to one half of the heart.

This allows us to use the width of the QRS complex to try to determine how the ventricles were depolarized.

If the QRS complex is narrow (<3 small squares), the ventricles must have been rapidly depolarized by an impulse that came through the AV node - the only way into the His-Purkinje system. The patient is then said to have a supraventricular rhythm (arising from above the ventricles).

If the QRS complex is broad (>3 small squares), there are two possible explanations.

1. The impulse may have arisen from within the ventricles and thus been unable to travel via the HisPurkinje system (ventricular rhythm).

2. The impulse may have arisen from above the ventricles but not been able to use all the His-Purkinje system because of a conduction problem (supraventricular rhythm with aberrant conduction).

6- Is Atrial Activity Present?

- ❖ presence of P waves indicates atrial depolarization.

❖ However, it does not mean that the depolarization necessarily started at the SA node. P waves will appear during atrial depolarization regardless of where it originated

7- How Are Atrial Activity and Ventricular Activity Related?

- If every QRS complex is associated with a P wave, this indicates that the atria and ventricles are being activated by a common source. This is usually, but not necessarily, the SA node (e.g. AV junctional rhythms will also depolarize both atria and ventricles).

- If there are more P waves than QRS complexes, conduction between atria and ventricles is being either partly blocked (with only some impulses getting through) or completely blocked (with the ventricles having developed their own escape rhythm).

- More QRS complexes than P waves indicates AV dissociation, with the ventricles operating independently of the atria and at a higher rate

Table 6.3 Broad-complex versus narrow-complex rhythms

Rhythm origin	Rhythm conduction	QRS complex
Supraventricular	Normal	Narrow
Supraventricular	Aberrant (e.g. bundle branch block)	Broad
Ventricular	Myocyte to myocyte	Broad

Note: Only supraventricular rhythms with normal conduction can gain access to the His–Purkinje system to rapidly depolarize the ventricles.

Supraventricular rhythms

Supraventricular rhythms are those which arise above the level of the ventricles

The supraventricular rhythms we will consider are:

1. Sinus rhythm
2. Sinus arrhythmia
3. Sinus bradycardia
4. Sinus tachycardia
5. Sick sinus syndrome
6. Atrial ectopic beats
7. Atrial fibrillation
8. Atrial flutter
9. Atrial tachycardia
10. Focal atrial tachycardia
11. Multifocal atrial tachycardia
12. Atrioventricular re-entry tachycardia (AVRT)
13. Atrioventricular nodal re-entry tachycardia (AVNRT)

1- Sinus Rhythm

Sinus rhythm is the normal cardiac rhythm, in which the SA node acts as the natural pacemaker, discharging at a rate of 60-100/min

The characteristic features of sinus rhythm are:

- Heart rate is 60-100/min
- P wave morphology is normal (e.g. upright in lead II and inverted in lead aVR)
- Every P wave is followed by a QRS complex

Normal SINUS RHYTHM

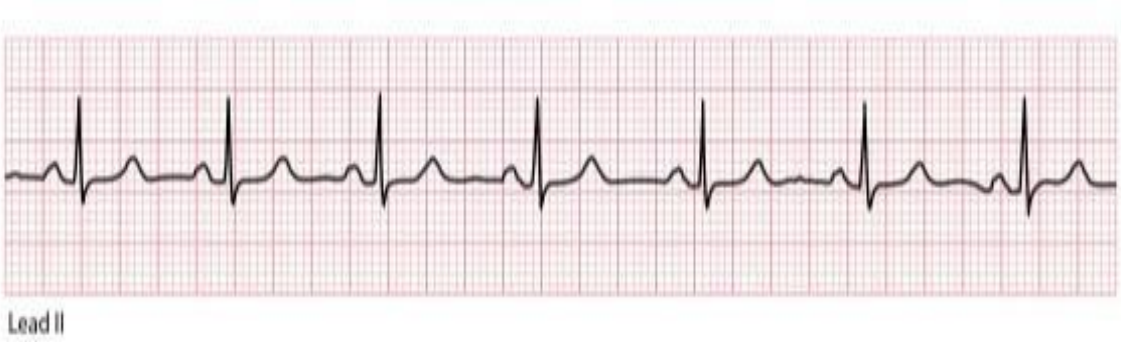


Figure 7.1 Normal sinus rhythm.

Key point: • The heart rate is 75/min, the P waves are upright (lead II) and every P wave is followed by a QRS complex.

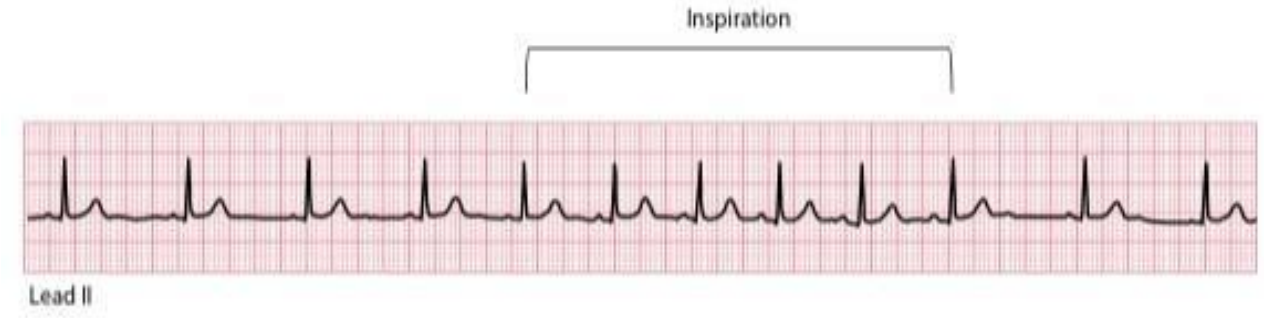
2- Sinus Arrhythmia

- Sinus arrhythmia is the variation in heart rate that is seen during inspiration and expiration The characteristic features of sinus arrhythmia are:

- The heart rate varies with respiration, with the difference between the longest and shortest P-P intervals being >0.12 s (3 small squares)

- During inspiration, the heart rate increases as a reflex response to the increased volume of blood returning to the heart (which triggers baroreceptors that inhibit vagal tone)

- During expiration, the heart rate decreases as a reflex response to the decreased volume of blood returning to the heart (vagal tone is no longer inhibited)



gure 7.2 Physiological sinus arrhythmia.

y point: • The heart rate increases during inspiration, the P waves are upright (lead II) and every P wave is followed by a QRS complex.

- P wave morphology is normal (e.g. upright in lead II and inverted in lead aVR)

- Every P wave is followed by a QRS complex

3- Sinus Bradycardia

Sinus bradycardia is sinus rhythm with a heart rate of less than 60/min

The characteristic features of sinus bradycardia are:

- The heart rate is less than 60/min
- P wave morphology is normal (e.g. upright in lead II and inverted in lead aVR)
- Every P wave is followed by a QRS complex

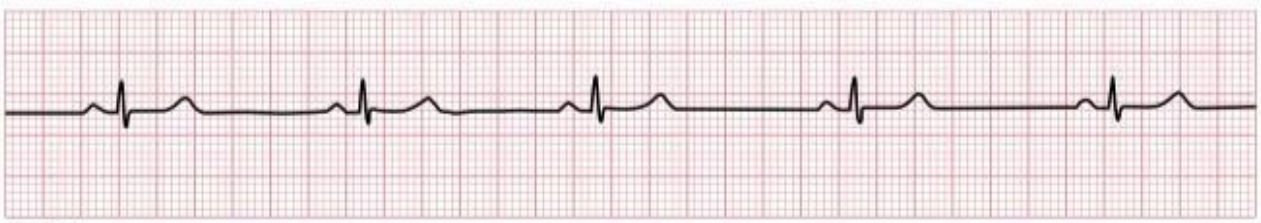


Figure 7.3 Sinus bradycardia.

Key point: • The heart rate is 46/min, the P waves are upright (lead II) and every P wave is followed by a QRS comple

always consider the following possible causes:

1- Drugs (e.g. digoxin, beta blockers - including beta blocker eye drops)
2- Ischemic heart disease and myocardial infarction
3- Hypothyroidism
4- Hypothermia
5- Electrolyte abnormalities
6- Obstructive jaundice
7- Uremia
8- Raised intracranial pressure
9- Sick sinus syndrome

4- Sinus Tachycardia

Sinus tachycardia is sinus rhythm with a heart rate of greater than 100/min

The characteristic features of sinus tachycardia are:

- The heart rate is greater than 100/min
- P wave morphology is normal (e.g. upright in lead II and inverted in lead aVR)
- Every P wave is followed by a QRS complex

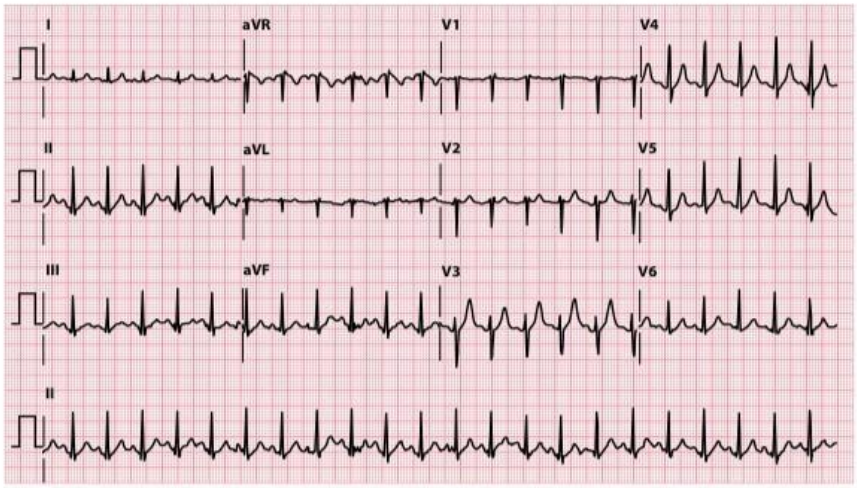


Figure 7.4 Sinus tachycardia.

Key point: • The heart rate is 136/min, the P waves have a normal orientation in each lead, and every P wave is followed by a QRS complex.

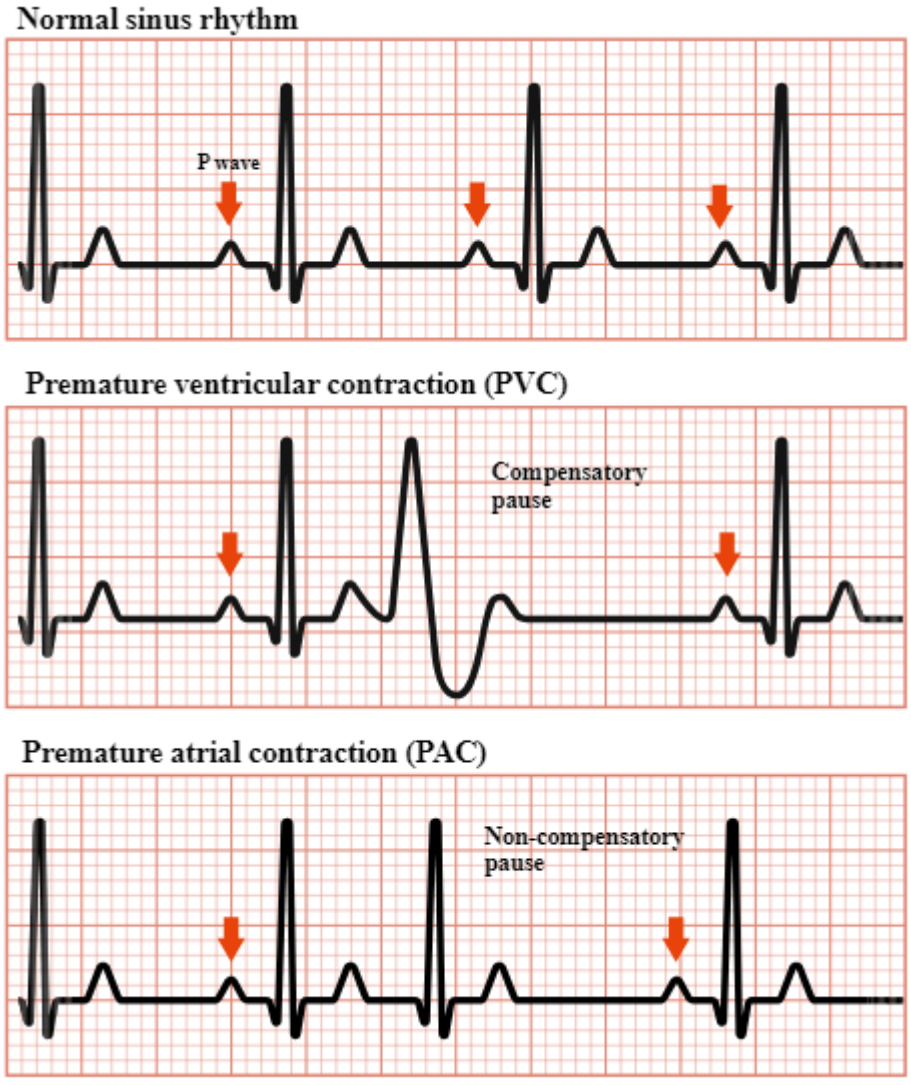
Always consider the following causes as well:

1- Drugs, e.g. adrenaline, atropine, salbutamol (do not forget inhalers and nebulizers), caffeine and alcohol
2- Ischemic heart disease and acute myocardial infarction
3- Heart failure
4- Pulmonary embolism
5- Fluid loss
6- Anemia
7- Hyperthyroidism

5- Atrial Ectopic Beats

Atrial ectopic beats are also called atrial extrasystoles, atrial premature complexes (APCs), atrial premature beats (APBS) or premature atrial contractions (PACs).

Atrial ectopic beats are identified by a P wave that appears earlier than expected and has a different shape to the normal P waves. Although atrial ectopic beats will usually be conducted to the ventricles and give rise to a QRS complex,



6- Atrial Fibrillation

- The basis of AF is rapid
- No P waves are seen
- ECG baseline consists of low-amplitude oscillations (fibrillation or f waves).
- Although 400-600 impulses reach the AV node every minute, only some will be transmitted to the ventricles.
- The ventricular rate is typically fast (100-180/min), although the rate can be normal or even slow. Transmission of the atrial impulses through the AV note is erratic, making the ventricular (QRS complex) rhythm 'irregularly irregular'.

Five categories of AF are recognized:

1. First-diagnosed AF: Namely, patients presenting in AF for the first time
2. Paroxysmal AF: Self-terminating episodes of AF, typically lasting <48 hours although they can last up to 7 days
3. Persistent AF: An episode of continuous AF lasting >7 days or requiring cardioversion
4. Long-standing persistent AF: Where AF has been present for at least one year, but there is still an aim to restore sinus rhythm
5. Permanent AF: Continuous AF where the arrhythmia is 'accepted' and there is no plan to restore sinus rhythm

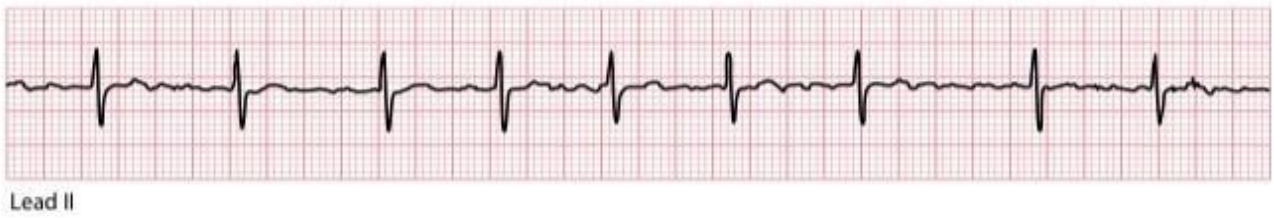


Figure 7.7 Atrial fibrillation.

Key point: • The ventricular rhythm is irregularly irregular, with an absence of distinct P waves.

Key issues to consider in managing patients with AF are:

- Reducing stroke risk
- Ventricular rate control
- Rhythm control

Reducing stroke risk

- The presence of AF increases a patient's stroke risk fivefold and one in five strokes occurs as a result of
- Strokes that occur in AF are more likely to be disabling or fatal.
- Reducing stroke risk in AF is therefore important, and the approach can be summarized as follows:

✓ For patients with valvular AF (including rheumatic valve disease and prosthetic valves), anticoagulation is recommended for all, unless there are contraindications

✓ For those with non-valvular AF, anticoagulation is recommended for all, except in those patients who are at low risk (aged <65 years and lone AF), or with contraindications

Ventricular rate control

- Commonly used drugs for ventricular rate control include beta blockers and non-dihydropyridine calcium channel blockers (verapamil or diltiazem).

- Digoxin is good for rate control at rest but is poor at rate control during exercise.
- Although amiodarone is effective for rate control,
- Amiodarone also carries the risk of chemical cardioversion to sinus rhythm, which could lead to thromboembolism unless the patient has been anticoagulated, where appropriate.
- Rate control is the preferred strategy (instead of rhythm control) in elderly patients and those with minimal symptoms
- aiming for a resting ventricular rate <110/min. If patients remain symptomatic, a stricter rate control strategy can be used, aiming for a resting heart rate <80/min (with a heart rate <110/min during moderate exercise).
- If drug therapy cannot attain satisfactory rate control in AF, and restoration of sinus rhythm cannot be achieved, an alternative strategy is to undertake ablation of the AV node plus permanent pacing.

Rhythm control

Patients with symptomatic AF despite adequate ventricular rate control should be considered for a rhythm control strategy, where the aim is to restore and maintain sinus rhythm.

Table 7.1 EHRA classification of AF symptoms

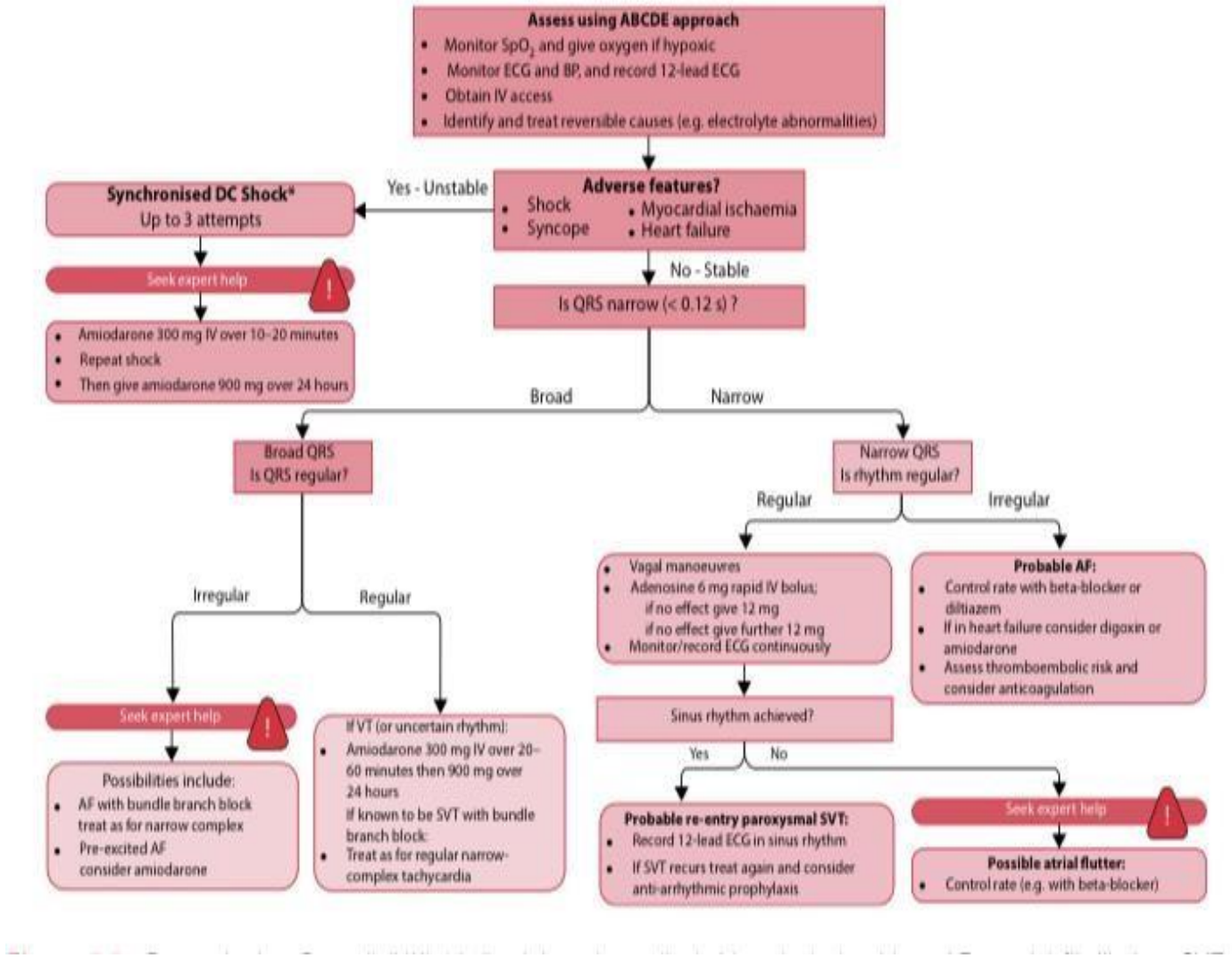
EHRA class	Severity of AF symptoms
I	Asymptomatic
II	Mild symptoms (not affecting daily activities)
III	Severe symptoms (affecting daily activities)
IV	Disabling symptoms (unable to undertake daily activities)

Cardioverting to sinus rhythm

- For those with recent onset AF and who are hemodynamically unstable, urgent electrical cardioversion is advised.

- Similarly, patients who e stable and who present within 48 hours of the onset of AF can have urgent electrical cardioversion

- In both cases, anticoagulant cover using intravenous unfractionated heparin is advised initially, followed by a minimum of 4 weeks' oral anticoagulation (unless the patient is aged <65 years and has had 'lone' AF).


Assess using ABCDE approach
Monitor SpO2 and give oxygen if hypoxic
Monitor ECG and BP, and record 12-lead ECG
Obtain IV access
Identify and treat reversible causes (e.g. electrolyte abnormalities)
Adverse features?
Shock
Syncope
Myocardial ischaemia
Heart failure
Yes - Unstable
Synchronised DC Shock*
Up to 3 attempts
Seek expert help
Amiodarone 300 mg IV over 10–20 minutes
Repeat shock
Then give amiodarone 900 mg over 24 hours
No - Stable
Is QRS narrow (< 0.12 s) ?
Broad
Narrow
Broad QRS
Is QRS regular?
Irregular
Regular
Seek expert help
Possibilities include:
AF with bundle branch block treat as for narrow complex
Pre-excited AF consider amiodarone
If VT (or uncertain rhythm):
Amiodarone 300 mg IV over 20–60 minutes then 900 mg over 24 hours
If known to be SVT with bundle branch block:
Treat as for regular narrow-complex tachycardia
Narrow QRS
Is rhythm regular?
Regular
Irregular
Vagal manoeuvres
Adenosine 6 mg rapid IV bolus;
if no effect give 12 mg
if no effect give further 12 mg
Monitor/record ECG continuously
Probable AF:
Control rate with beta-blocker or diltiazem
If in heart failure consider digoxin or amiodarone
Assess thromboembolic risk and consider anticoagulation
Sinus rhythm achieved?
Yes
No
Probable re-entry paroxysmal SVT:
Record 12-lead ECG in sinus rhythm
If SVT recurs treat again and consider anti-arrhythmic prophylaxis
Seek expert help
Possible atrial flutter:
Control rate (e.g. with beta-blocker)

7- Atrial Flutter

- In atrial flutter the atrial rate is usually 250-350/min
- and often almost exactly 300/min.
- The AV node cannot normally keep up with such a high atrial rate and AV block occurs.
- This is most commonly 2:1 block, where only alternate atrial impulses get through the AV node to initiate a QRS complex, although 3:1, 4:1 or variable degrees of block are also seen.
- Thus, the ventricular rate is less than the atrial rate, and is often 150, 100 or 75/min. You should always suspect atrial flutter with 2:1 block when a patient has a regular tachycardia with a ventricular rate of about 150/min.
- The rapid atrial rate gives a characteristic 'sawtooth' appearance to the baseline of the ECG, made up of flutter or 'F' waves.

The rapid atrial rate gives a characteristic 'sawtooth' appearance to the baseline of the ECG, made up of flutter or 'F' waves.

the characteristic features of atrial flutter are:

- Atrial rate around 300/min
- 'Sawtooth baseline
- AV block (commonly 2:1, but can be 3:1, 4:1 or variable)

Atrial flutter carries a risk of thromboembolism, and patients with atrial flutter are usually assessed for anticoagulant therapy according to the same guidelines as those used in AF (see earlier).

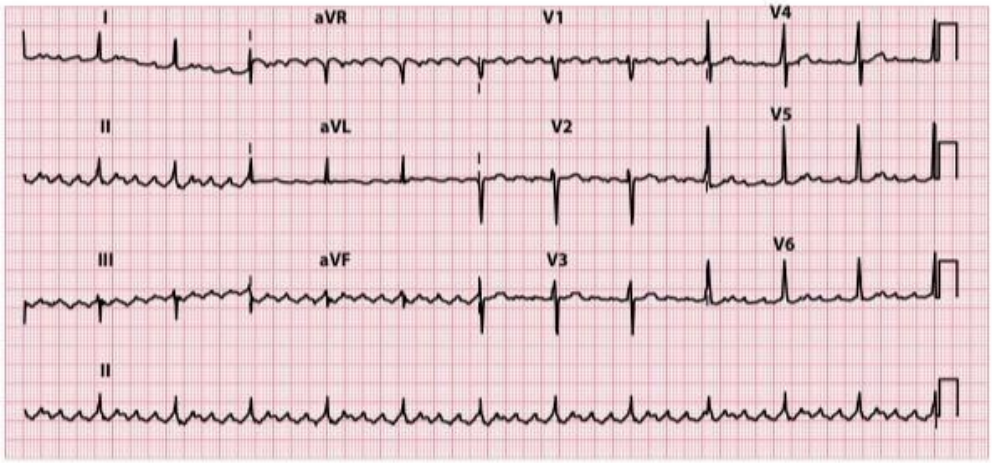


Figure 7.9 Atrial flutter.

Key point: • There is a 'sawtooth' pattern of atrial activity, with an atrial rate of 288/min and a ventricular rate of 72/ in (indicating 4:1 AV block).

Treatment of Atrial Flutter

Medicines for the treatment of atrial flutter include:

- **Medicines to slow down your heart rate:** Calcium channel blockers and beta-blockers.
- **Medicines to stop the abnormal rhythm:** Antiarrhythmic drugs.

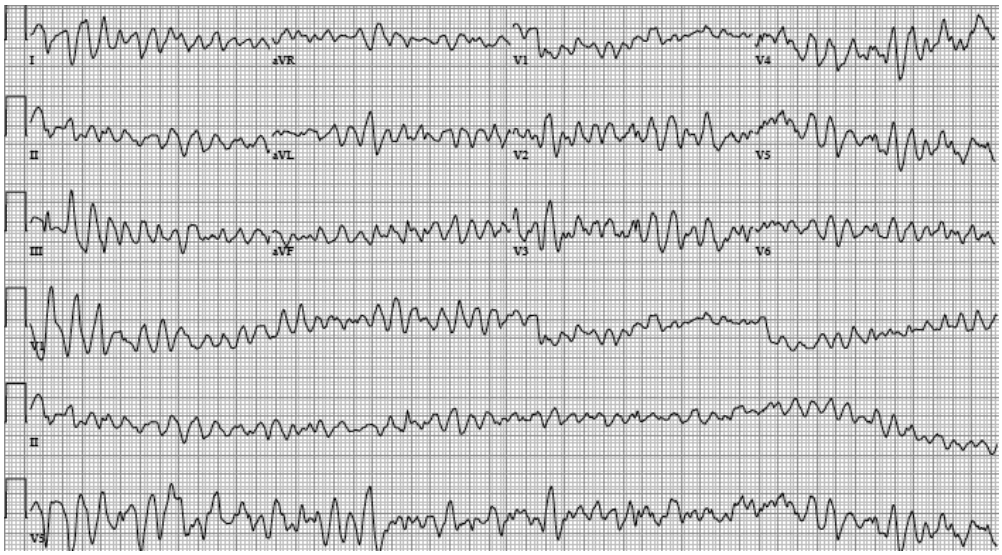
Procedures:

Procedures to treat atrial flutter include:

- Cardioversion via electrical shock.
- Catheter ablation, a procedure a provider can use to destroy the tissue that's creating abnormal signals.
- Temporary change in your pacemaker or implantable cardioverter defibrillator (ICD) setting.

Ventricular fibrillation

- ECG show irregular waves varying morphology and amplitude



<u>Causes</u>

- ✓ IHD
- ✓ Antiarrhythmic drugs
- ✓ Severe hypoxia

<u>Management</u>

- DC shock: immediate non synchronized DC shock at 200 J if ineffective repeated at 200-360 J
- IF DC shock fails start basic and advanced life support
- Drugs
- ✓ Amiodarone id drug of choice
- ✓ Others: lidocaine procainamide

MCQ test

1- Conduction system structures and its functions (all true except one)

a) SA node=pacemaker of the heart

b) Internodal pathway=direct impulse between SA and AV node

c) AV node=slows impulse

d) His bundle=transmits impulses between branches

e) Left bundle branch=conduct impulses that lead to right ventricles

2- Leads of ECG (all true except one)

a) Lead I=view of lateral heart

b) Lead II=view of inferior heart

c) Lead III=view of posterior of heart

d) aVL= view lateral of the heart

e) aVF= view inferior of the heart

3- the characteristic features of atrial flutter are (all true except one)

a) Atrial rate around 300/min

b) Sawtooth baseline

c) carries a risk of thromboembolism

d) treated by Cardioversion via electrical shock.

e) No response to calcium channel blockers

4- Atrial Fibrillation (all true except one)

a) The basis of AF is rapid

b) No P waves are seen

c) ECG baseline consists of low-amplitude oscillations (fibrillation or f waves).

d) The ventricular rate is typically fast (100-180/min)

e) No risk for thromboembolic events

5- Causes of sinus tachycardia (all true except one)

a) salbutamol

b) acute myocardial infarction

c) Heart failure

d) Pulmonary embolism

e) Fluid overload

6- Narrow complex tachycardia (all true except one)

a) Sinus tachycardia

b) Atrial tachycardia

c) Atrial flutter

d) Atrial fibrillation

e) Ventricle tachycardia

7- Located at 5^{th}. Intercostal space at left midclavicular line

a) V1

b) V2

c) V4

d) V5

e) V6

8- All the following drugs are used in management of bradycardia except one

a) Glycopyrrolate

b) Isoprenaline

c) Atropine

d) Glucagon

e) Atenolol

9- Ventricular fibrillation (all true except one)

a) Irregular waves

b) Caused by sever hypotension

c) Treated by DC shock

d) Alternative management is amiodarone

e) varying morphology and amplitude.

10- Rate control drugs (all true except one)

a) beta blockers

b) verapamil

c) diltiazem).

d) atropine

e) amiodarone

11- Dominant pacemaker of the heart is

a) AV node

b) SA node

c) Purkinje fiber

d) Bundle of His

e) None of the above

Defibrillation

Defibrillation is the passage of an electrical current across the myocardium in order to change the electrical activity of the heart from a chaotic to organized rhythm. It is a term usually used in relation to the treatment of ventricular fibrillation (VF) or ventricular tachycardia (VT); when used for the treatment of atrial fibrillation (AF) or atrial flutter, it is usually termed ‘cardioversion’.

What is Defibrillation?

The treatment for ventricular fibrillation and other life-threatening arrhythmias (abnormal heartbeats) is defibrillation. The heart stops pumping blood to the brain and body when it is in ventricular fibrillation. If not treated immediately, it will induce cardiac arrest and death within minutes. By shocking the heart with electricity, defibrillation restores a regular heartbeat.

When combined with CPR and specialized medical care, rapid defibrillation can save lives. Defibrillation does not treat the arrhythmia's underlying cause. It does not always work, especially in cases of severe, untreated cardiac disease or some end-stage conditions.

Defibrillator Machine

Defibrillators are devices that provide an electric pulse or shock to the heart to restore a regular heartbeat. They're used to prevent or treat arrhythmias, which are irregular heartbeats that are either too slow or too fast. If the heart abruptly stops beating, defibrillators can help restore it. Defibrillators are designed to work in a variety of ways. The goal of automated external defibrillators (AEDs), which are found in many public places, is to save the lives of those who have suffered a sudden cardiac arrest. In an emergency, even inexperienced people can operate these devices. Other defibrillators can help those who are at high risk of dying from a life-threatening arrhythmia. Implantable cardioverter defibrillators (ICDs) and wearable cardioverter defibrillators (WCDs) are two types of defibrillators. ICDs are surgically implanted inside the body, while WCDs are placed on the body. It takes time and effort to adjust to living with a defibrillator, and it's critical to be aware of the risks and issues that can arise.

Types of Defibrillator Machines

1. Manual Defibrillator

A healthcare professional's skill is required for manual external defibrillators. They're utilized in conjunction with an Electrocardiogram, which might be standalone or integrated. The voltage and timing for the electrical shock are manually determined after a healthcare provider assesses the heart rhythm. The majority of these units may be found in hospitals and on select ambulances. During or after cardiac surgery, such as a heart bypass, an internal defibrillator is frequently used to defibrillate the heart. Round metal plates are used as electrodes and come into direct touch with the myocardium. Paddles placed directly on the heart administer the shock in manual internal defibrillators. They're mostly employed in the operating room and, in rare cases, the emergency department during an open-heart surgery.

2. Automated External Defibrillators (AED Defibrillator)

Automated external defibrillators (AEDs) are a type of defibrillator designed for use by untrained persons. AEDs have equipment that can analyze cardiac rhythms. As a result, determining whether or not a rhythm is shockable does not necessitate the use of a qualified health expert. AEDs have improved outcomes for abrupt out-of-hospital cardiac arrests by making these units widely available.

Trained health professionals will have more limited use of automatic defibrillators than manual external defibrillators. AEDs do not enhance

outcomes in patients with in-hospital cardiac arrests, according to recent studies. AEDs have fixed voltages and do not allow the operator to adjust the voltage based on the situation. AEDs may also cause effective CPR to be delayed. AEDs frequently require the cessation of chest compressions and rescue breathing in order to diagnose rhythm. For these reasons, certain organizations, such as the European Resuscitation Council, advocate that if manual external defibrillators are readily available, they be used instead of AEDs.

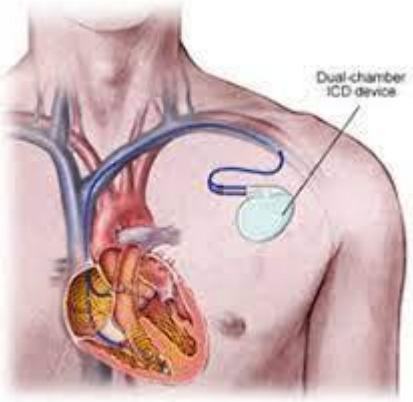
AEDs have been widely available in many easily accessible regions because early defibrillation can greatly improve VF outcomes. AEDs have been incorporated into the basic life support algorithm (BLS). They are carried by many first responders, including firefighters, police officers, and security guards.

AEDs are available in two types: completely automatic and semi-automatic. A semi-automatic AED diagnoses heart rhythms and determines whether or not a shock is required. If a shock is recommended, the user must press a button to deliver it. A fully automated AED detects the heart beat and instructs the user to stand back while the shock is delivered automatically. Few types of AEDs have advanced features, like manual override or an ECG display.

3. Cardioverter-Defibrillators

Automatic internal cardiac defibrillators (AICDs), also known as implantable cardioverter-defibrillators (ICDs), are pacemaker-like implants. According to the device's programming, they continuously monitor the patient's heart rhythm and automatically administer shocks for life-threatening arrhythmias. Many

current equipment's can discriminate between ventricular fibrillation, ventricular tachycardia, and other arrhythmias that are more benign, such as supraventricular tachycardia and atrial fibrillation. Prior to synchronized cardioversion, some devices may attempt overdrive pacing. When ventricular fibrillation is life-threatening arrhythmia, the device is programmed to deliver an unsynchronized shock right away.



In some situations, the patient's ICD may fire frequently or incorrectly. This is a medical emergency since it depletes the device's battery life, gives the patient severe discomfort and worry, and in extreme situations, can even produce life-threatening arrhythmias. Some emergency medical personnel now have a ring magnet to place over the device, which essentially inhibits the device's shock function while still allowing the pacemaker to work (if the device is so equipped). EMS workers may deliver sedation if the gadget shocks regularly but adequately.

A wearable defibrillator is a portable defibrillator which can be worn by risky patients. If VF or VT is identified, the equipment watches the patient 24 hours a day and can immediately deliver a biphasic shock. Patients who are not immediate candidates for ICDs should use this device.

Defibrillator Uses

In cardiac resuscitation, defibrillation is frequently used (CPR). CPR is a procedure that uses an algorithm to restore cardiac and pulmonary function. Only some types of cardiac dysrhythmias, such as ventricular fibrillation (VF) and pulseless ventricular tachycardia, require defibrillation. Defibrillation is not recommended if the heart has fully stopped, as in asystole or pulseless electrical activity (PEA). If the patient is cognizant and has a pulse, defibrillation

is not recommended. Electrical shocks administered incorrectly can result in serious dysrhythmias such as ventricular fibrillation.

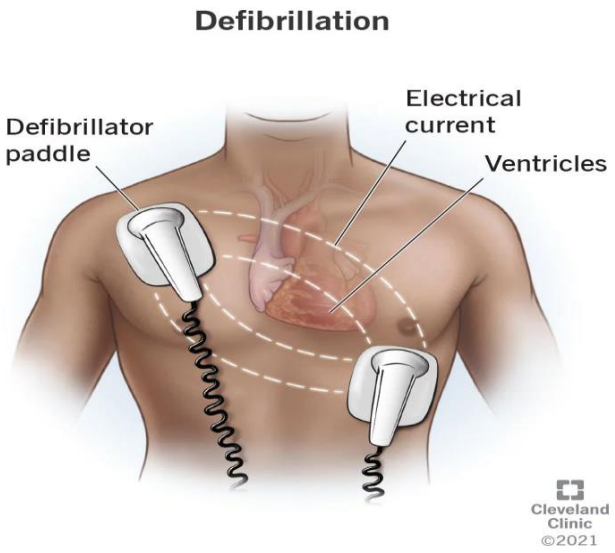
Out-of-hospital cardiac arrest survival rates are dismal, often less than 10%. In-hospital cardiac arrests have a greater success rate of 20%. The specific heart rhythm of persons who have experienced a cardiac arrest can have a major impact on survival rates. People with a shockable rhythm (such as VF or pulseless ventricular tachycardia) had better survival rates of 21-50 percent compared to people with a non-shockable rhythm (such as asystole or PEA).

Why is Defibrillation Performed?

Your heart is a pump-like muscle that pumps blood around your body. There are two upper chambers (atria) and two lower chambers in this structure (ventricles). Your heart, like other pumps, requires an energy source to work. The energy in your heart comes from an electrical conduction system built into it that transfers electrical signals through the four chambers. To produce a regular heartbeat, electrical signals coordinate the chambers. Certain signal faults result in a disordered, inefficient, quivering rhythm. Defibrillation sends an electrical shock through the heart, causing all of the cardiac cells to contract simultaneously. This brings the heart's abnormal beat to a halt and allows it to resume normal electrical activity. To be effective, defibrillation must be performed within minutes of the onset of a life-threatening ventricular arrhythmia.

Defibrillation is used to treat ventricular arrhythmias that are immediately life-threatening, such as:

- Ventricular fibrillation is a condition in which your heart's lower chambers, or ventricles, beat so quickly and irregularly that they quiver or shake. Your heart pumps very little or no blood to your brain and body as a result. Without defibrillation, death occurs in five to ten minutes.
- Without a pulse, ventricular tachycardia occurs when the ventricles beat excessively quickly. The heart's efficiency suffers as a result of this. It lowers the quantity of blood your heart can pump to your brain and other parts of your body. If there isn't enough blood to produce a pulse or you pass out, you'll need to be treated with defibrillation. Without a pulse, ventricular tachycardia can quickly progress to ventricular fibrillation.



How is Defibrillation Performed?

The technique for defibrillation differs based on the type of device. It usually entails the following steps:

- Until a heart defibrillator is available, the clinician will begin CPR.
- Two sticky defibrillator electrodes or paddles lubricated with special jelly will be applied to your upper right chest and lower left rib cage area by the provider. The upper right chest and upper left back are two possible positions. A doctor will create a thoracotomy incision in your chest and place the electrodes directly on your heart muscle in rare circumstances.
- The provider or the AED will assess your cardiac rhythm and, if necessary, shock your heart.
- The provider or the AED will re-analyze the resulting heart rhythm and, if necessary, administer further shocks. A team of healthcare professionals will offer CPR and advanced life support (ALS) treatments as needed during this period.

Complications of Defibrillation

Defibrillation comes with dangers and potential problems. Defibrillation's life-saving benefits significantly exceed the hazards. The following are some of the risks and potential complications:

- Burns on the skin
- Myocardial necrosis (death of heart muscle tissue)
- Various cardiac arrhythmias include asystole (no heart rhythm, also known as "flatlining"), ventricular fibrillation following pulseless ventricular tachycardia, and other less dangerous arrhythmias.

Safety

Oxygen concentrations as high as 60% have been measured in enclosed environments using oxygen-powered medical devices, 24% oxygen doubles the rate of combustion and 30% oxygen increases combustion rate 10-fold. In an oxygen-enriched atmosphere, sparking from poorly applied defibrillator paddles in an oxygen-enriched environment can cause a catastrophic fire.

The risk of fire during attempted defibrillation can be minimized by taking the **following precautions**:

◆ Remove any oxygen mask or nasal cannula and place ≥ 1 m away from the patient's chest

◆ Leave any bag-valve device connected to a tracheal tube or other airway adjunct (e.g., laryngeal mask airway, Combi tube, or laryngeal tube). Alternatively, disconnect any bag-valve device from the tracheal tube (or other airway adjunct), and remove it ≥ 1 m from the patient's chest during defibrillation.

◆ If the patient is connected to a ventilator, leave the ventilator tubing (breathing circuit) connected to the tracheal tube during defibrillation.

Application of defibrillation electrodes

Self-adhesive defibrillation pads are generally replacing defibrillation paddles. They improve safety by avoiding the need to lean over the patient during defibrillation and provide better electrical contact with the skin compared with the flat metal plates of defibrillation paddles, minimizing the risk of electrical arcing and fire. Optimal electrode position is one that results in greatest current flow across the myocardium. The standard position to achieve this is the sternal electrode placed to the right of the sternum, immediately below the clavicle and the apical paddle placed in the mid-axillary line (Fig. 63.5), level with the V6 ECG electrode position.

Acceptable alternative positions include:

- ◆ Bi-axillary.
- ◆ Anterior (left sternal edge): posterior (between the left clavicle and spine).
- ◆ Posterior (behind the right clavicle): apical

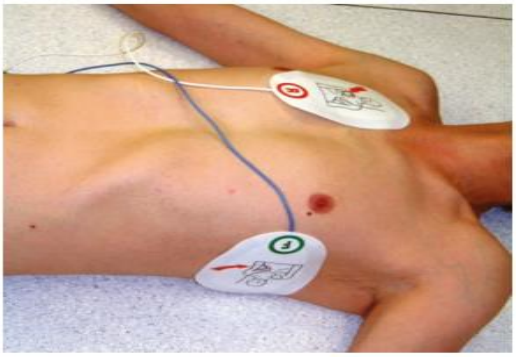


Fig. 63.5 Anterior-apical defibrillation electrode placement.
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Clinical aspects of defibrillation

Defibrillation is one of the few interventions that have been shown to improve survival from cardiac arrest and it is therefore a key link in the **chain of survival**. The probability of successful defibrillation and neurologically-intact survival to hospital discharge is time critical, with every minute that passes between collapse and defibrillation resulting in an increase in mortality of 7–10%, this increase is slowed marginally in patients receiving bystander resuscitation. In order to reduce delays in defibrillation, the introduction of public access defibrillators in areas of high population density (e.g., airports, shopping centers, railways stations, etc.) has resulted in significantly improved survival rates, particularly in some urban areas. In more rural areas rapid activation of trained community responders is also contributing to early defibrillation. CPR versus defibrillation as the initial treatment. Although early studies suggested that a period of cardiopulmonary resuscitation (CPR) for 1–3 minutes prior to defibrillation increased the defibrillation success rate, subsequent larger studies failed to repeat these observations. It is now recommended that when

treating both in- and out-of-hospital cardiac arrest, rescuers should provide good-quality CPR, while a defibrillator is quickly applied and charged, but routine preshock CPR (e.g. 2 or 3 minutes) is no longer recommended. One shock versus three shock sequence With first shock efficacy of biphasic waveforms exceeding 90%, failure to successfully defibrillate is more likely to suggest the need for a period of CPR, rather than a further shock. The previous recommendations for up to three shocks before resuming CPR have now been superseded by a single shock sequence. Only on the rare occasion of a monitored VF/non-pulsatile VT arrest, it is acceptable to deliver three stacked shocks using a manual defibrillator before commencing 2 minutes of external chest compression and ventilation if necessary. Automatic external defibrillators (AEDs) are all programmed to deliver a single shocks before recommencing the 2-minute CPR cycle.

Energy levels

First shock efficacy of the BIPHASIC SHOCK using 150–200 J has been reported as 86–98%. Ideally, the initial biphasic shock energy should be at least 150 J for all waveforms when defibrillating ventricular arrhythmias. If the first shock is unsuccessful, there is no evidence to suggest that either a fixed or escalating energy protocol is more effective. However, although an escalating strategy reduces the number of shocks required to restore an organized rhythm compared with fixed-dose biphasic defibrillation, rates of return of spontaneous circulation or survival to hospital discharge are not significantly different between strategies. Both fixed or escalating strategies are acceptable, but when using a manual defibrillator, it is reasonable to increase the energy for subsequent shocks. The lower efficacy of the monophasic waveform means the initial and all subsequent monophasic shocks should be delivered at 360 J

Detrimental effects of defibrillation

Interruptions to chest compressions adversely affect the outcome of a resuscitation attempt. One of the commonest causes of these interruptions is defibrillation, due to pre-shock and post-pauses in chest compressions associated with ECG analysis, delivery of the shock and a pulse check. In order

to minimize interruptions to chest compressions associated with defibrillation, it is now recommended to continue chest compressions whilst the defibrillator is charged. Immediate resumption of chest compressions following defibrillation is also emphasised, without pausing for a pulse check, which should only then take place after 2 minutes of CPR. Defibrillation should be achievable with an interruption in chest compressions of no more than 5 seconds.

Early defibrillation in conjunction with high quality minimally interrupted chest compressions is critical to survival following sudden cardiac arrest. Survival rates decrease 7–10% with each minute that passes without defibrillation and an initial shockable rhythm of ventricular fibrillation (VF) will then deteriorate into pulseless electrical activity (PEA) and asystole. It is important however, to realize that early defibrillation alone does not usually improve survival and that integration of high quality minimally interrupted chest compressions is essential for optimal outcome.

Defibrillation in children

Shockable rhythms occur in only 7–15% of paediatric and adolescent arrests with a much lower percentage than in adult cardiac arrest. *Common causes of VF in these children include* trauma, congenital heart disease, drug overdose, and hypothermia. Ideally, paediatric self-adhesive pads with electrical attenuators should be used for children aged less than 8 years, but when these are not available, adult pads are acceptable, as long as there is no direct contact between the two electrodes. The recommended energy levels for both monophasic and biphasic defibrillation are 4 J/kg for the initial and all subsequent shocks.

MCQ TEST

1- The recommended energy levels for both monophasic and biphasic defibrillation in pediatric are
    a) 2jouls/kg
    b) 4 joules/kg
    c) 6jouls/kg
    d) 8jouls/kg
    e) 10 joules/kg
2- The level of biphasic shock for defibrillating ventricular arrhythmias are
    a) 150-200 J
    b) 200-300 J
    c) 360 J
    d) More than 360 J
    e) Less than 150 J
3- All the following are types of defibrillators except one
    a) Automatic internal cardiac defibrillators
    b) Automated external defibrillator
    c) Implantable cardioverter defibrator
    d) Wearable defibrillator
    e) Ventricular defibrillator
4- the precautions during defibrillation
    a) Remove any oxygen mask or nasal cannula
    b) Leave any bag-valve device connected to a tracheal tube
    c) Leave any bag-valve device connected to laryngeal mask airway
    d) If the patient is connected to a ventilator, leave the ventilator tubing (breathing circuit) connected to the tracheal tube during defibrillation
    e) All the above
5- Optimal electrode position during defibrillation is
    a) the apical electrode placed to the right of the sternum, immediately below the clavicle and the apical paddle placed in the mid-axillary line.
    b) the sternal electrode placed to the right of the sternum, immediately below the clavicle and the apical paddle placed in the mid-axillary line
    c) the sternal electrode placed to the right of the sternum, immediately below the clavicle and the apical paddle placed in the mid-clavicular line

d) the sternal electrode placed to the left of the sternum, immediately below the clavicle and the apical paddle placed in the mid-axillary line
e) the sternal electrode placed to the right of the sternum, immediately above the clavicle and the apical paddle placed in the mid-axillary line