

CARDIO PULMONARY RESUSCITATION

CPR

Definition of CPR according to WHO

Is an emergency life saving procedure performed when heart stops beating or impending stop for the purpose of oxygenation of the heart, lungs & brain until restore the normal cardiopulmonary function

CPR IS EASY AS CAB C=Compression...A= Airway....B =Breathing

Compressions Push hard and fast on the center of the victim's chest

Airway Tilt the victim's head back and lift the chin to open the airway

Breathing Give mouth-to-mouth rescue breaths

Types of C P R

1- B L S

2- A L S

Definition of B L S according to WHO

Is an emergency life saving procedure performed when heart stops beating or impending stop for the purpose of oxygenation of the heart, lungs & brain until restore the normal cardiopulmonary function WITHOUT EQUIPMENTS

It involves by giving strong , rapid pushes to the chest to keep blood moving through the body and blows air into the mouth to help with breathing and send oxygen to the patient's lung

So its C A B C=Compression...A= Airway....B =Breathing

Goals of BLS

1. Maintain oxygen and blood supply to vital organs during cardiac arrest

Restore spontaneous circulation) Cardiac output during CPR with effective ,uninterrupted chest compression is at best GOALS OF BLS

Maintain oxygen and of the normal spontaneous circulation .

- 2- Minimize post resuscitation organ injury**
- 3- Improve the patient's survival and neurologic outcome**

Newly concept for training

- 1. CPR coach to help team leader .**
- 2. CPR coach ensures high quality BLS, while team leader focuses on other aspects like ACLS .**
- 3. Double sequential defibrillation (Two defibrillators set one anterolateral and other antero posterior electrodes**
- 4. ,Biphasic 120: 200 J but monophasic 360 J**
- 5. In Situ training (simulation and training in the clinical setting to improve professional skills ,team function & clinical care**
- 6. Booster training to refresh participants knowledge ,skills & attitudes around the key of course components**
- 7. Spaced learning approach (outside clinic ,hospital &classroom)**

CHAIN OF SURVIVAL

- Identifies a sequence of **SIX critical actions** that increase survival rates from sudden cardiac arrest (SCA)

OUTSIDE HOSPITAL CARDIAC ARREST



INSIDE HOSPITAL CARDIAC ARREST

IHCA



Recovery (including additional treatment, observation, rehabilitation, and psychological support)

CPR Components for BLS Providers

Component	Adults and Adolescents	Children (Age 1 Year to Puberty)	Infants (Age Less Than 1 Year, Excluding Newborns)
1-Safety	Make sure the environment is safe for rescuers and victim		
Scene			
Rescuer			
Victim			
Bystanders			



UNWITNESSED COLLAPSE



A-CHECK RESPONSE



Shake shoulders gently

Ask “Are you all right?”

If he responds

- Leave as you find him.
- Find out what is wrong.
- Reassess regularly.

Rapid neurological assessment of patient response (AVPU)

- **Alert**
- **Voice**
- **Pain**
- **Unresponsive**

B-Check pulse Central not peripheral pulse Age & site of pulse check in CPR

CATEGORY	AGE	SITE FOR PULSE CHECK
NEONATE	1 ST 30 DAYS AFTER BIRTH	<u>PRECORDIAL</u> <u>AUSCULTATION</u> <u>LEAD ECG</u>
INFANT	30 DAYS TO 1 YEAR AFTER BIRTH	<u>BRACHIAL ARTERY</u>
CHILD	1 YEAR TO PUBERTY	<u>FEMORAL</u> <u>CAROTID ARTERY</u>
ADULT \ ADOLOCENT	AFTER PUBERTY	<u>CAROTID ARTERY</u>

- PUBERTY: **FEMALE**: BREAST DEVELOPMENT
MALE: AXILLARY HAIR

C - CHECK BREATHING

- Look, listen and feel for NORMAL breathing



- Do not confuse agonal breathing with NORMAL breathing

No response
No pulse
No breathing
SHOUT FOR HELP



Component	Adults and Adolescents	Children (Age 1 Year to Puberty)	Infants (Age Less Than 1 Year, Excluding Newborns)
3 - Shout for help -Mobile phone technology -Activation of emergency response system	<ul style="list-style-type: none"> -If you are alone with no mobile phone, leave the victim to activate the emergency response system and -Get the AED before beginning CPR - If you have other personell , send someone for activation And begin CPR immediately; -use the AED as soon as it is available 	<p style="text-align: center;">Witnessed collapse</p> <p>One was present at time of the patient collapsed</p> <p>Follow steps for adults and adolescents on the left</p> <p style="text-align: center;">Unwitnessed collapse</p> <p>the patient is found without pulse and no one was present at the time the patient collapsed</p> <p>Give 2 minutes of CPR Leave the victim to activate the emergency response team</p>	

Air way

4-Open airway

- head tilt
 - chin lift or head tilt
 - jaw thrust manoeuvre
 - and mask bag ventilation is recommended for initial airway control in most circumstances. If not available do mouth to mouth breathing
- **Triple manoeuvre:** head tilt-chin lift , mouth open, jaw thrust



Head Tilt-Chin Lift Maneuver

- **Indications:**
 - Unresponsive
 - No spinal injury
 - Unable to protect airway
- **Contraindications:**
 - Responsive
 - Possible spinal injury



- **Advantages**
 - No equipment
 - Noninvasive
- **Disadvantages**
 - Hazardous to spinal injury
 - No protection from aspiration

Jaw-Thrust Maneuver

- **Disadvantages**

- Cannot maintain if patient becomes responsive or combative
- Difficult to maintain for an extended time
- Difficult to use with bag-mask ventilation

- Thumb must remain in place
- Requires second rescuer
- No protection against aspiration



Clean airway



- Removes material from the mouth or throat quickly and efficiently
 - Ventilating with secretions in the mouth will result in upper airway obstruction or aspiration.
- Next priority after opening airway manually

CHECK BREATHING

5-Check breathing

- Look
- listen
- Feel
- for NORMAL breathing
- Do not confuse agonal breathing with NORMAL breathing



AGONAL BREATHING

- Occurs shortly after the heart stops
in up to 40% of cardiac arrests
- Described as barely, heavy, noisy or gasping breathing
- **Recognise as a sign of cardiac arrest**

Not breathing

- Call for help
- Start CPR and Rescue breaths

Age & site of pulse check in CPR

CATEGORY	AGE	SITE FOR PULSE CHECK
NEONATE	1 ST 30 DAYS AFTER BIRTH	<u>PRECORDIAL</u> <u>AUSCULTATION</u> <u>3</u> <u>LEAD ECG</u>
INFANT	30 DAYS TO 1 YEAR AFTER BIRTH	<u>BRACHIAL ARTERY</u>
CHILD	1 YEAR TO PUBERTY	<u>FEMORAL</u> <u>CAROTID ARTERY</u>
ADULT \ADOLESCENT	AFTER PUBERTY	<u>CAROTID ARTERY</u>

- PUBERTY: FEMALE: BREAST DEVELOPMENT
MALE: AXILLARY HAIR

- No pulse
- Call for help
- Start CPR

No response
No breathing
No pulse
Shout for help
START C P R



Component	Adults and Adolescents	Children (Age 1 Year to Puberty)	Infants (Age Less Than 1 Year, Excluding Newborns)
6-Compression-ventilation ratio without advanced airway	1 or 2 rescuers 30:2		<i>One rescuer</i> 30:2 <i>Two or more rescuers</i> 15:2
-Compression-ventilation ratio with advanced airway	Continuous compressions at a rate of 100-120/min Give 1 breath every 6seconds(10 breaths/mm)	Continuous compressions at a rate of 100-120/min Give 1 breath every 2-3 seconds (20-30 breaths/mm)	

Check circulation

- Check the pulse
- Check the bleeding

Age & site of pulse check in CPR

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- PUBERTY: FEMALE: BREAST DEVELOPMENT
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- No pulse
 - Call for help
 - Start CPR

No response
No breathing
No pulse
Shout for help
START C P R



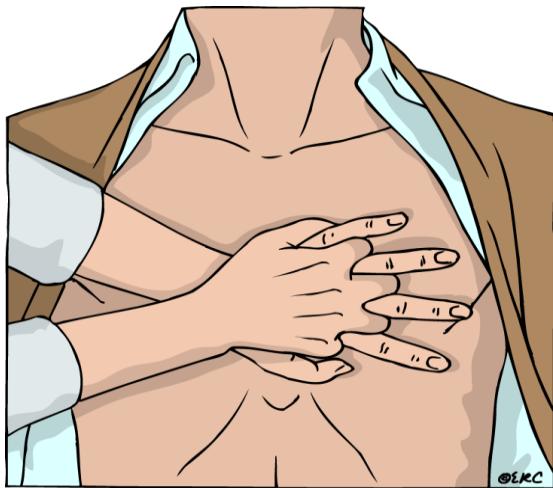
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Compression rate	100-120/min		
Compression depth	At least 2 inches (5 cm)*	At least one third AP diameter of chest About 2 inches (5 cm)	At least one third AP diameter of chest About 1 : 1 1/2 inches (4 cm)
Hand placement	2 hands on the lower half of the breastbone (sternum) Push hard In the center of chest 100 to 120 per minute  shutterstock.com • 519158911	2 hands or 1 hand (optional for very small child) on the lower half of the breastbone (sternum)	1 rescuer 2 fingers in the center of the chest, just below the nipple line 2 or more rescuers 2 thumb-encircling hands in the center of the chest, just below the nipple line
Chest recoil	Allow full recoil of chest after each compression; do not lean on the chest after each compression		
Minimizing interruptions	Limit interruptions in chest compressions to less than 10 seconds		



CHEST COMPRESSIONS

- Place the heel of one hand in the centre of the chest
- Place other hand on top
- Interlock fingers
- Compress the chest
 - Rate 100 min^{-1}
 - Depth 4-5 cm
 - Equal compression : relaxation
- When possible change CPR operator every 2 min



CHEST COMPRESSIONS

Table 1

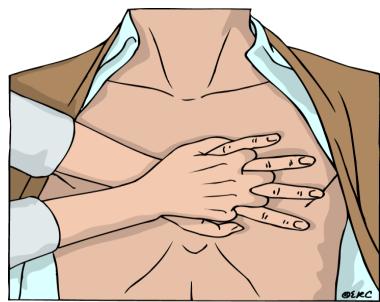
BLS Dos and Don'ts of Adult High-Quality CPR

Rescuers Should	Rescuers Should Not
Perform chest compressions at a rate of 100-120/min	Compress at a rate slower than 100/min or faster than 120/min
Compress to a depth of at least 2 inches (5 cm)	Not less than 4 cm and not more than 7 cm
Allow full recoil after each compression	Lean on the chest between compressions
Minimize pauses in compressions	Interrupt compressions for greater than 10 seconds
Ventilate adequately (2 breaths after 30 compressions, each breath delivered over 1 second, each causing chest rise)	Provide excessive ventilation (ie, too many breaths or breaths with excessive force)





CONTINUE CPR



DIFFERENCE IN ADULT AND PEDIATRIC CPR

POINT OF DIFFERENCE	ADULTS	CHILDREN\INFANT
1: activation of emergency response system	1 st : Activate ERS & get AED 2 nd : Start CPR	<u>Witnessed collapse</u> : same as adult <u>Unwitnessed collapse</u> : 1 st : 2mins of CPR 2 nd : leave the victim to active ERS
2:compression to ventilation ratio without advanced airway	One or more rescuer. Always 30:2	1 rescuer: 30:2 2 rescuer: 15:2
3:compression to ventilation ratio with advanced airway	1breath every 6secs (10 breaths\min(1breath every 2-3 secs 30-20)breaths \mins(
4: depth of compression	2to 2.4inches	Children: about 2inches Infants: about 1.5inches
5: hand placement	Lower half of sternum	Infants: 1 rescuer: 1finger technique

AUtomated Electrical Defirilator AED



Some AEDs will automatically switch themselves on when the lid is opened •

ATTACH PADS TO CASUALTY'S BARE CHEST



**ANALYSING RHYTHM
DO NOT TOUCH VICTIM**



SHOCK INDICATED



**SHOCK DELIVERED
FOLLOW AED INSTRUCTIONS**

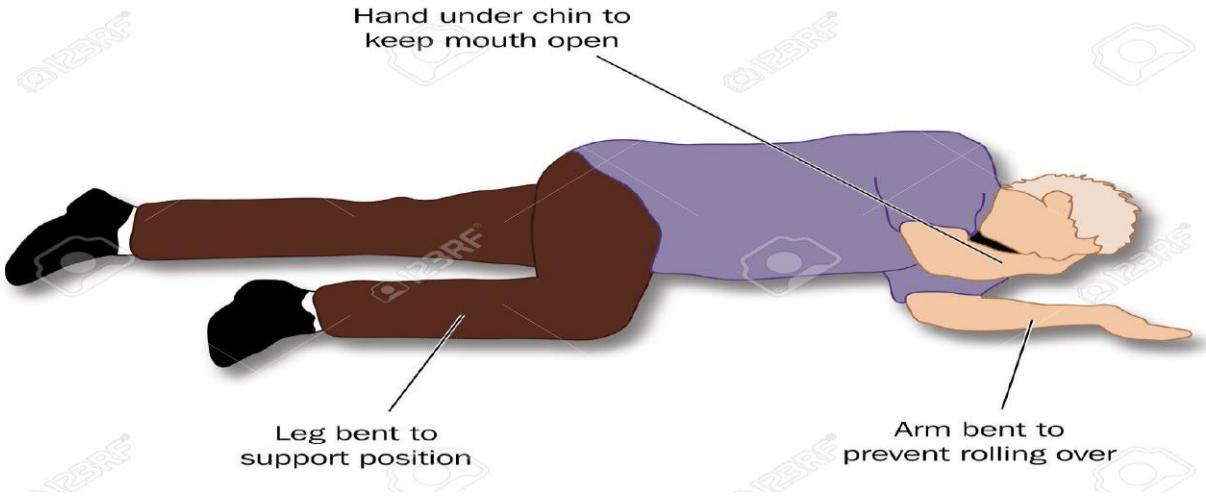


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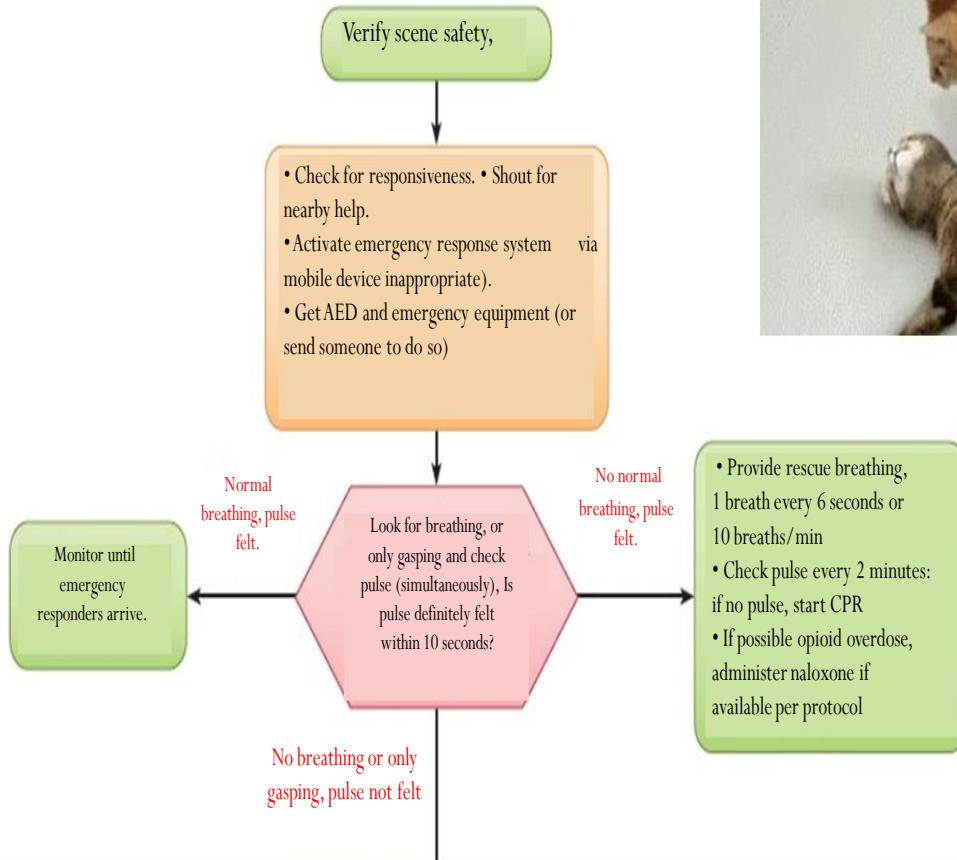


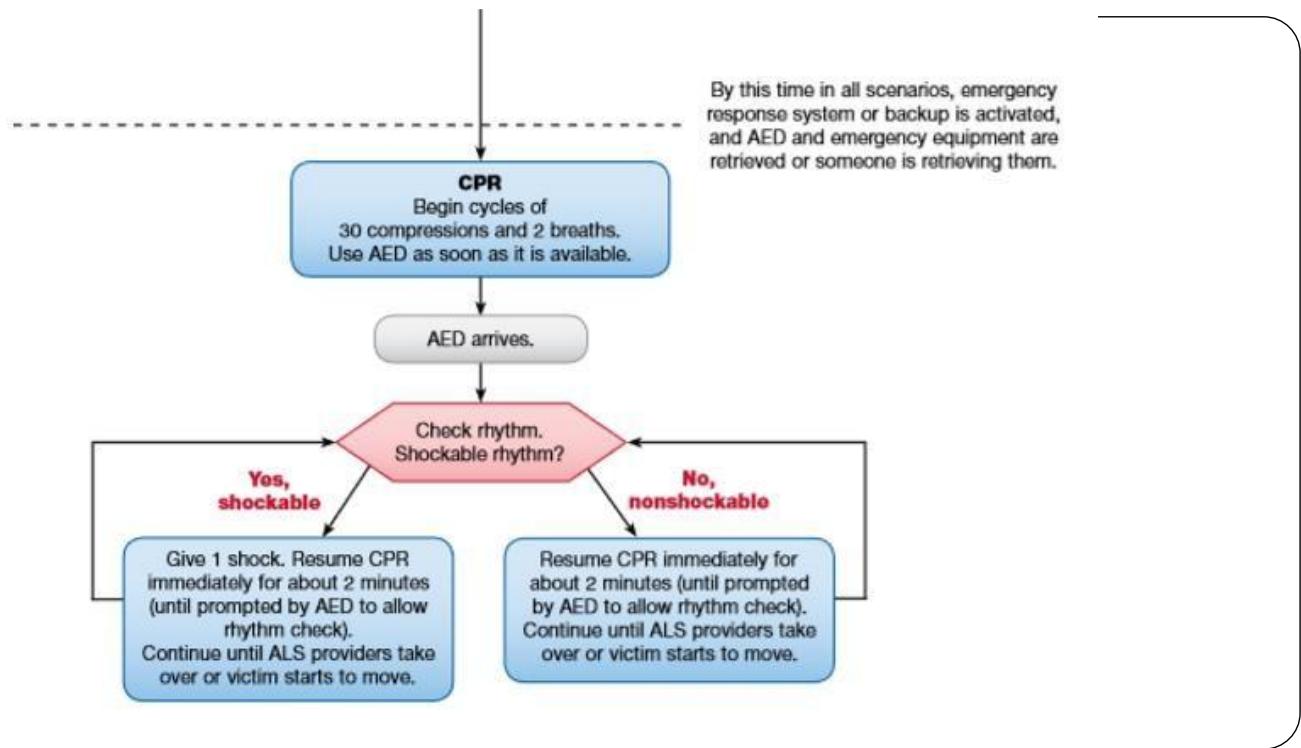
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Adult Basic Life Support Algorithm for Healthcare Providers





ADVANCED LIFE SUPPORT

Definition of A L S according to WHO

Is an emergency life saving procedure performed when heart stops beating or impending stop for the purpose of oxygenation of the heart, lungs & brain until restore the normal cardiopulmonary function with EQUIPMENTS

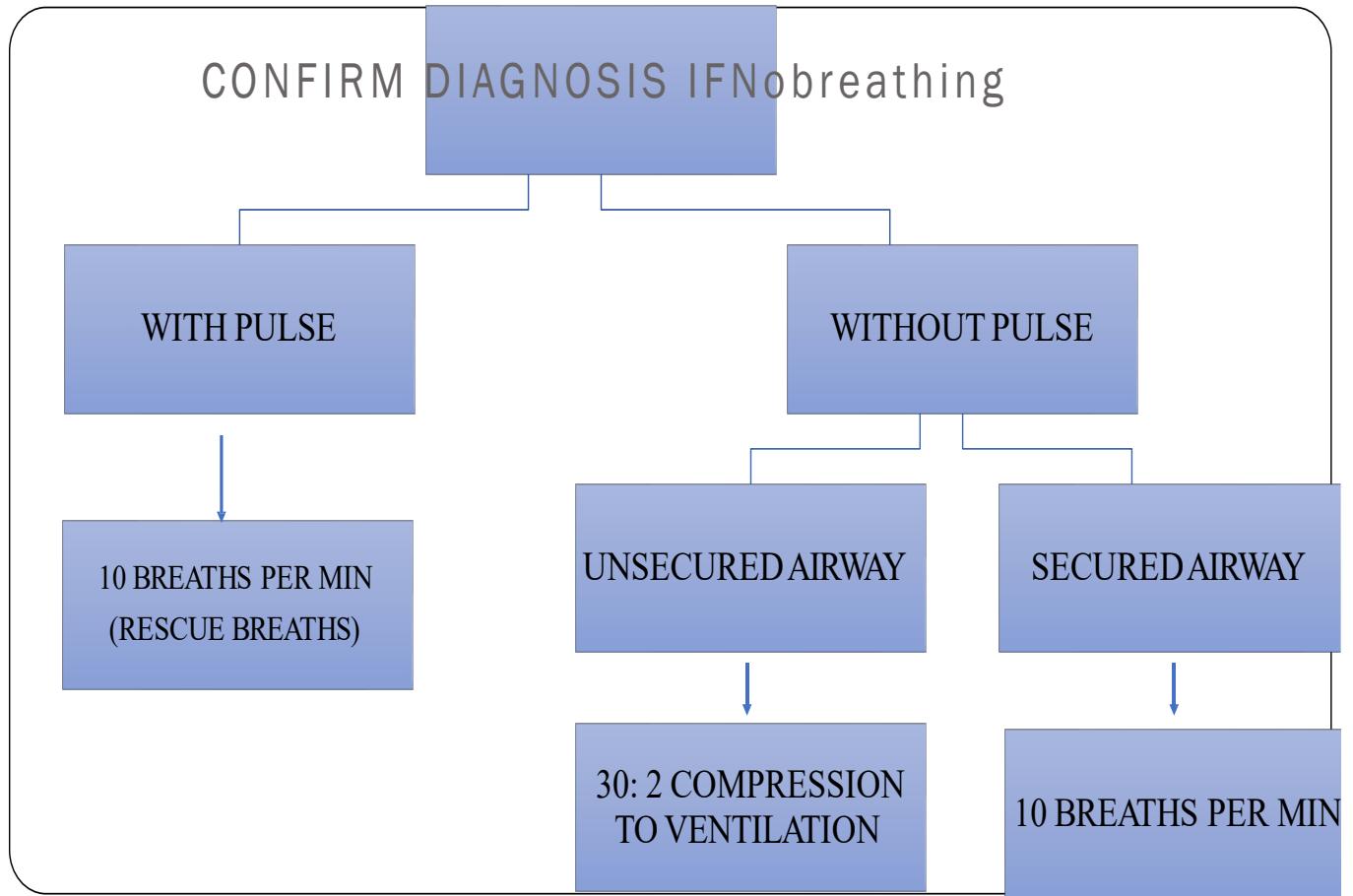
So its A B C..... A=Airway....B =Breathing....C =Circulation

ADVANCED CARDIAC LIFE SUPPORT

- TEAM OF TRAINED PROFESSIONALS
- COMPRISES OF DOCTORS, TRAINED PARAMEDICAL STAFF, NURSES
- **MEMBERS:** MINIMUM 6 AND MAXIMUM 10
- 6 rescuer role distribution
 - ✓ 1 TEAM LEADER
 - ✓ 1 COMPRESSOR & 1 VENTILATOR (CHANGE ROLES EVERY 2MINS)
 - ✓ 1 DEFIBRILLATOR
 - ✓ 1 INTRAVENOUS DRUG DELIVERY
 - ✓ 1 TIME KEEPER

Comparison between BLS & ACLS

BASIC LIFE SUPPORT	ADVANCED CARDIAC LIFE SUPPORT
CAN BE PERFORMED BY ANYONE	TRAINED MEDICAL \PARAMEDICAL STAFF
RHYTHM IDENTIFICATION BY AED	RHYTHM IDENTIFICATION BY RESUSICATOR
AED FOR DEFIBRILLATION	MANUAL DEFIBRILLATOR
NO IV LINE \DRUG USE	IV LINE AND DRUG MANAGEMENT
NO ADVANCED AIRWAY	ADVANCED AIRWAY USE
NO OXYGEN SUPPORT	OXYGEN SUPPORT



ADVANCED AIRWAY

- DEPENDING ON THE LEVEL OF EXPERTISE OF THE CPR PROVIDER
 - ENDOTRACHEAL TUBE
 - LARYNGEAL MASK AIRWAY
 - COMBITUBE
-
- “*UNDER NO CIRCUMSTANCES SHOULD THE INSERTION OF ADVANCED AIRWAY COMPROMISE THE CHEST COMPRESSIONS*” due to consumed long time.

DEFIBRILLATION

- Delivery of an **electrical current** through the myocardium to interrupt disorganized cardiac activity and restore an organized cardiac rhythm
- **Monophasic defibrillator** :a **single 360joule (J)** shock is delivered. (old –not used anymore)
- **Biphasic defibrillator 200-120(J)** is usually sufficient to terminate the arrhythmia)new & better(
- If the rescuer is unfamiliar :**maximal available energy** should be used as the default energy
- **Pediatric patients:** 1st shock : 4-2J/kg--- subsequent shocks 4 J/kg)MAX 10J/kg(



DEFIBRILLATOR



PLACEMENT OF ELECTRODES:

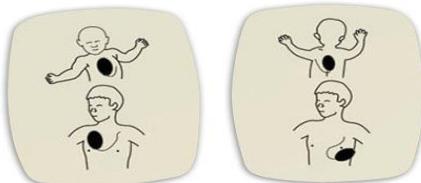
ADULT:

- **Upper right sternal border**, just below the clavicle
- Lateral to the **left nipple**.

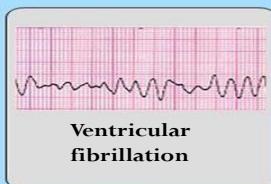


CHILD OR INFANT:

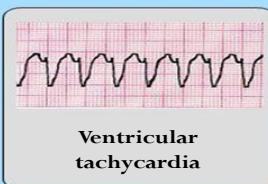
- Anterior and Posterior.



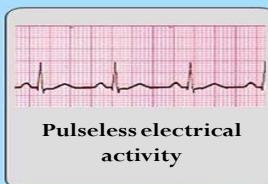
Cardiac arrest heart rhythms summary



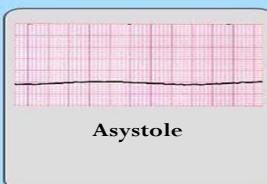
Ventricular fibrillation



Ventricular tachycardia



Pulseless electrical activity



Asystole

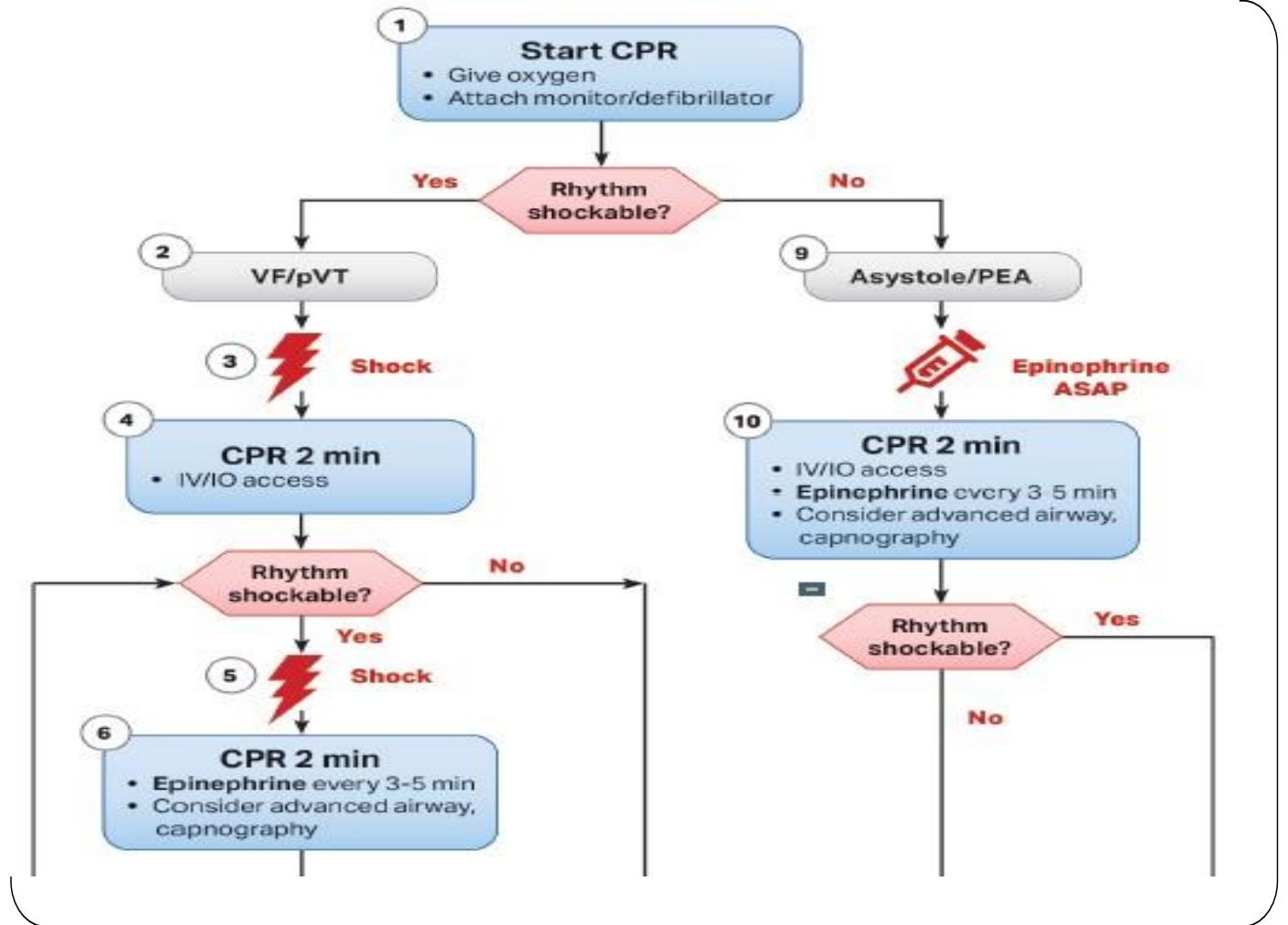


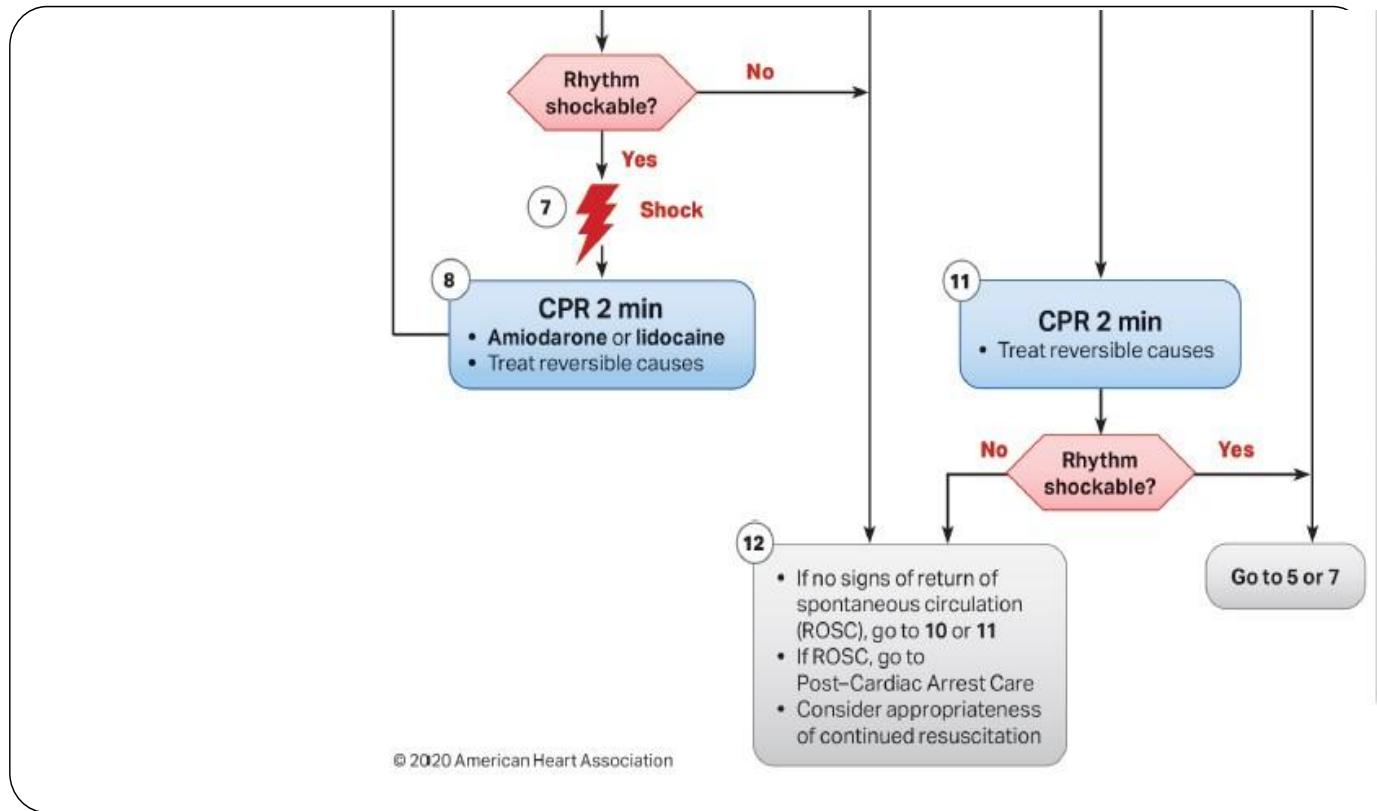
Shockable rhythms ; need to defibrillate



Non shockable

VF and VT are very common for cardiac arrest in the home or in public places





CPR Quality

- Push hard (at least 2 inches [5 cm]) and fast (100-120/min) and allow complete chest recoil.
- Minimize interruptions in compressions.
- Avoid excessive ventilation.
- Change compressor every 2 minutes, or sooner if fatigued.
- If no advanced airway, 30:2 compression-ventilation ratio.
- Quantitative waveform capnography
 - If PETCO₂ is low or decreasing, reassess CPR quality.

Return of Spontaneous Circulation (ROSC)

- Pulse and blood pressure
- Abrupt sustained increase in PETCO₂ (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Shock Energy for Defibrillation

- **Biphasic:** Manufacturer recommendation (eg, initial dose of 120-200 J); if unknown, use maximum available. Second and subsequent doses should be equivalent, and higher doses may be considered.
- **Monophasic:** 360 J

Advanced Airway

- Endotracheal intubation or supraglottic advanced airway
- Waveform capnography or capnometry to confirm and monitor ET tube placement
- Once advanced airway in place, give 1 breath every 6 seconds (10 breaths/min) with continuous chest compressions

Drug Therapy

- **Epinephrine IV/IO dose:** 1 mg every 3-5 minutes
- **Amiodarone IV/IO dose:** First dose: 300 mg bolus. Second dose: 150 mg.
or
- **Lidocaine IV/IO dose:** First dose: 1-1.5 mg/kg. Second dose: 0.5-0.75 mg/kg.

ROUTES OF ACCESS

NCU ACADEMY OF MEDICAL

Intravenous	Interosseous	Endotracheal
Intravenously push bolus injection	Most common: lower end of femur or upper end of tibia	least preferred
Flush with 20mL of fluid or saline		Dose is 2-2.5 times the iv dose
Raise extremity for 10 to 20 seconds to enhance delivery of drug to circulation		5 drugs:(NAVAL) Naloxone Adrenaline Vasopressin Atropine Lignocaine

DRUG	ADULT	PEDIATRIC
ADRENALINE\EPINEPHRINE	1mg (1:10000) bolus every 5-3mins <u>Endotracheal:</u> 2.5-2times dose (1:1000) diluted in 10ml normal saline	0.01Mg/kg or 0.1 ml/kg of 1:10000 concentration(Max dose:1 mg Repeat every 3-5mins. <u>Endotracheal:</u> 0.1mg/kg or 0.1 ml/kg of 1:1000concentration
AMIODARONE	1 st dose: 300mg 2 nd dose: 150mg	5mg/kg bolus during cardiac arrest. May repeat 3 doses for refractory VF\pulseless VT
LIGNOCAINE	1 st dose: 1-1.5mg/kg 2 nd dose: 0.75-0.5mg/kg	1mg/kg bolus dose

Return of Spontaneous Circulation (ROSC)

- Pulse and blood pressure
- Abrupt sustained increase in PETCO₂ (typically ≥40 mm Hg)
- Spontaneous arterial pressure waves with intra-arterial monitoring

Reversible Causes

- | | |
|---|--|
| <ul style="list-style-type: none">• Hypovolemia• Hypoxia• Hydrogen ion (acidosis)• Hypo-/hyperkalemia• Hypothermia | <ul style="list-style-type: none">• Tension pneumothorax• Tamponade, cardiac• Toxins• Thrombosis, pulmonary• Thrombosis, coronary |
|---|--|

POST CARDIAC ARREST CARE

THERAPEUTIC HYPOTHERMIA

- Recommended for comatose individuals with return of spontaneous circulation after a cardiac arrest event.
- Individuals should be cooled to 89.6 to 93.2 degrees F (32 to 36 degrees C) for at least 24 hours.

OPTIMIZATION OF HEMODYNAMICS AND VENTILATION

- 100% oxygen is acceptable for early intervention but not for extended periods of time.
- Oxygen should be titrated, so that individual's pulse oximetry is greater than 94% to avoid oxygen toxicity.
- Do not over ventilate to avoid potential adverse hemodynamic effects.
- Ventilation rates of 10 to 12 breaths per minute to achieve ETCO₂ at 35 to 40 mmHg.
- IV fluids and vasoactive medications should be titrated for hemodynamic stability.

CARDIAC ARREST MEDICATIONS

- Epinephrine
- Amiodarone
- Atropine
- Sodium Bicarbonate
- Calcium (Chloride or Gluconate)
- Lidocaine

EPINEPHRINE

- Both an alpha- and beta-adrenergic agent
- During an cardiac arrest, most think it has the greatest benefit by alpha-adrenergic actions, increasing afterload and thus diastolic blood pressure, leading to improved coronary artery perfusion.

EPINEPHRINE

- Indications:
 - Cardiac arrest
 - Severe bronchospasm
 - Anaphylactic reactions
- Route of Administration
 - IV or IO
 - SQ or IM (for bronchospasm)

ET (cardiac arrest without IV or IO access) ●

EPINEPHRINE

- Dosage:
 - initial (low) dose: 0.01 mg/kg
= 0.1 cc/kg of 1:10,000
 - subsequent (high) doses: 0.1 mg/kg
= (0.1 cc/kg of 1:1,000)
- PALS recommendations for high dose under review - please check for updates

Amiodarone

- Class 3 antiarrhythmic drug as well as alpha & beta receptors blockers
- Given 300 mg bolus then 150 mg loading dose
- Used for ventricular arrhythmias and atrial fibrillation or flutter

ATROPINE

- Parasympathetic (not an alpha- or beta-adrenergic) agent--acts by blocking cholinergic stimulation of the muscarinic receptors of the heart.
- Results in an increase in the sinus rate of the heart.
- Little effect on systemic vascular resistance or myocardial contractility.

ATROPINE

- Indications:
 - Bradycardia
 - Second or third degree heart block
 - Asystole
 - Pulseless electrical activity (electrical mechanical dissociation)
- Route of Administration
 - IV, IO, ET, SQ, IM, nebulization

ATROPINE

- Dosage:
 - 10 to 20 mcg/kg
 - minimum dose is 0.1 mg--smaller doses may cause reflex bradycardia (central stimulatory effect on the medullary vagal nuclei)
 - maximum (adult) dose is 2 mg

SODIUM BICARBONATE

- Use during CPR remains a controversial issue due to lack of evidence showing benefit from receiving bicarbonate.
- Elevates blood pH by binding with hydrogen to form water and CO₂
- HCO⁻₃ + H⁺ => H₂CO₃ => H₂O + CO₂
- Must have adequate ventilation to remove CO₂ or respiratory acidosis will worsen

SODIUM BICARBONATE

- Adverse effects of acidosis:
 - Cardiac
 - Decrease contractility
 - Lower threshold for ventricular fibrillation
 - Decrease responsiveness to catecholamines
 - Vascular
 - Decrease systemic vascular resistance
 - Decrease systemic vascular responsiveness to catecholamines
 - Increase pulmonary vascular resistance

SODIUM BICARBONATE

- Indications:
 - Pre-existing acidosis
 - Prolonged CPR (after 10 minutes)
 - Pulmonary hypertensive crisis
 - Hyperkalemia
- Route of administration:
 - IV, IO
- Dosage
 - 1-2 meq/kg/dose (1 meq/cc or 0.5 meq/cc)

CALCIUM

- Current recommendations for the use of calcium during CPR are restricted to a few specific situations.
- Intracellular calcium plays an important role in the process of cell death, but no studies have shown that transient hypercalcemia worsens outcome after cardiac arrest.

CALCIUM

- Adverse Effects of Hypocalcemia
 - Decreased myocardial contractility
 - Decreased systemic vascular resistance
 - Decreased catecholamine release
 - Decreased cardiovascular response to catecholamines

CALCIUM

- Indications:

- Hypocalcemia
 - Ionized hypocalcemia may result from severe alkalosis or after large transfusions of citrated blood products.
- Hyperkalemia
- Hypermagnesemia
- Calcium channel blocker overdose

LIDOCAINE

- Indications:
 - Ventricular Tachycardia
 - Ventricular Fibrillation
 - Frequent PVCs
- Route of Administration:
 - IV, IO, ET
- Dosage:
 - 1 mg/kg/dose (may need up to 2.5 mg/kg ET)

ENDOTRACHEAL MEDICATIONS

- LEAN
 - Lidocaine
 - Epinephrine
 - Atropine
 - Naloxone (Narcan)

CALCIUM

- Route of administration:
 - IV, IO only
 - Calcium chloride--central venous line
 - Calcium gluconate--peripheral venous line
- Dosage:
 - Calcium chloride = 10-20 mg/kg
 - Calcium gluconate = 100-200 mg/kg

LIDOCAINE

- Class 1B antiarrhythmic
- Decreases automaticity threshold and ventricular fibrillation threshold.
- Effective in terminating PVCs.
- Rarely used in pediatric arrests as ventricular tachycardia and ventricular fibrillation are not commonplace.

Defibrillation and Cardioversion

Objectives

- Defibrillator & Cardio version in your hands
- Definitions & Types
- Difference between defibrillator & Cardio version
- Different sites of plades
- Clinical application
- When don't process

What is Defibrillator?

DC shock = AC shock = defibrillator

- Its an electric and electronic equipments used in the cardiac emergencies and dysrhythmic cardiac arrest by releasing the stored electric charges in controlled fashion causing simultaneous depolarization of all abnormally excitable myocardial cells and interruption of abnormal pathways and electric foci

Types of defibrillators

1- according to nature of electric source

a- alternative current (AC)

b- direct = constant current (DC)

AC current more stability & accuracy

2- according to mode of action

a- manual

The trained personell determines the cardiac rhythm by monitor & ECG , voltage supply , timing of shock through external paddles applied to the patient chest wall

b- automatic

The device can identify rhythm and suitable electric shock through electronic programmed monitor insitu .

3- according to site of defibrillators & electrodes

a- External

i. Manual (MED)

ii. Automatic (AED)

b- Internal

i. Manual (MID)

During open heart surgery in operating theatre

ii. Automatic (AICD) = (ICD)

iii. Wearable cardiac defibrillator (WCD)

Portable external defibrillator used in high risk frequent arrhythmia by computer technology and used during preparation for AICD

4- according to relation to QRS complex

a- unsynchronized = defibrillator

Shock delivery at anytime of cardiac cycle used in pulseless V tac , ventricular flutter and defibrillation

b- synchronized = cardioversion

Asynchronization means it's the timing of shock delivery during R wave and away of T wave and ST segment

Used in all life-threatened tachy-arrhythmias as SVT, AFt, AFib, Vtac with pulse

No shock in Asystole & PEA

5- according to electric phases

a- Monophasic

where electric current travels in one direction through the chest
High energy level is required with skin hazards .

b- Biphasic defibrillator

where electric current travels in two directions through the chest

In the first phase the current moves from one paddle to other as monophasic

In the second phase the current flows in the reverse direction

Low energy level is required with very low skin hazards 120 : 200 J (2 : 3 J / Kg)

c-Double sequential defibrillator

Provision of rapid sequential shocks via two defibrillators with defibrillation pads placed in both planes (antero-lateral & antero-posterior)

Can be used in refractory ventricular fibrillation

Important Buttons of DC

2- synchronization

3- energy adjustment

4- energy store

5- charge

6- strip ECG

7-record

8- data base

9-paddles charge

Placement of External Paddles

1- the paddles should not placed over hard thick large bones (sternum , scapula ,vertebra)

2- the paddles should not placed within 12 cm of permanent pacemaker

3- paddles maybe self-adhesive single used or reusable with K-Y jel

4- skin at paddles must protected by K-Y jel to prevent skin burn & decrease trans-thoracic impedance.

5- excessive chest hair at the paddle area may need to be shaved to achieve better electrode contact

6- better to deliver shock during expiration to avoid air space between paddles & heart

External Paddle sizes

a- adult size 8-13 cm

b- children size 8 cm

c- infant size 4.5 cm

N.B : larger possible paddle is preferred than the smaller possible why ??

External Paddle sites

a- antrolaterally

Patient in supine position :

one paddle placed on the rt 2nd intercostal space next to sternum . The 2nd paddle placed on the 5th intercostal space in the left midclavicular line (apex)

b- anteroposteriorly

the patient in the lateral position :

one paddle placed on the left 5th intercostal space in the midclavicular line. The other paddle placed posteriorlly in the left infrascapular region

Internal Paddle site

a-Atrioventricular : one paddle placed on left ventricle and the other on the right atrium

b-Biventricular : one paddle placed on the left ventricule and the other on the right ventricle

N.B: cardioversion 5-10 j

N.B : defibrillation 30-50 j

Whats the difference ?? And why ??

Doses of electric shock

a-Cardioversion :

0.5-1 j / kg

b-Defibrillator :

Monophasic 4-6 j / kg

Biphasic 2-4 j/kg

Common cardiac arrhythmias shock (Adult)

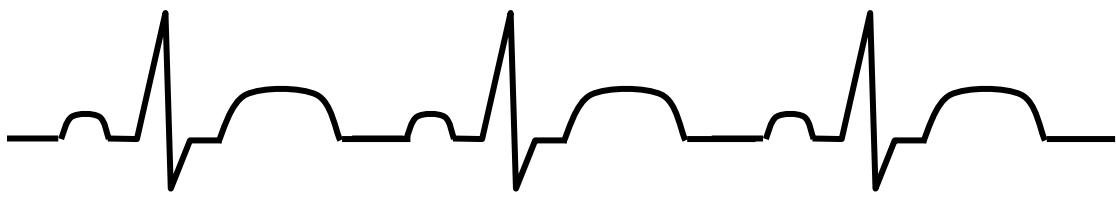
Atrial flutter : 50-100 j in adult

Atrial fibrillation 120-200 j in adult

SVT & Vtac : 120-200 j

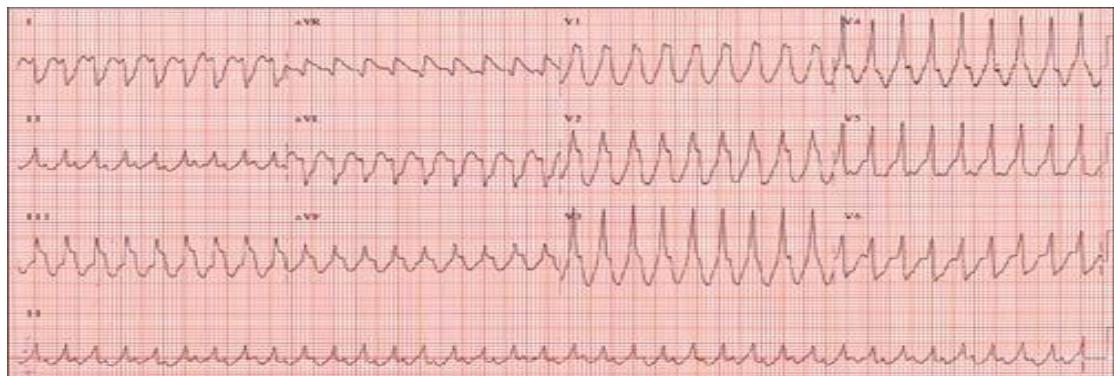
Ventricular flutter & fibrillation : 360 j

Normal sinus rhythm

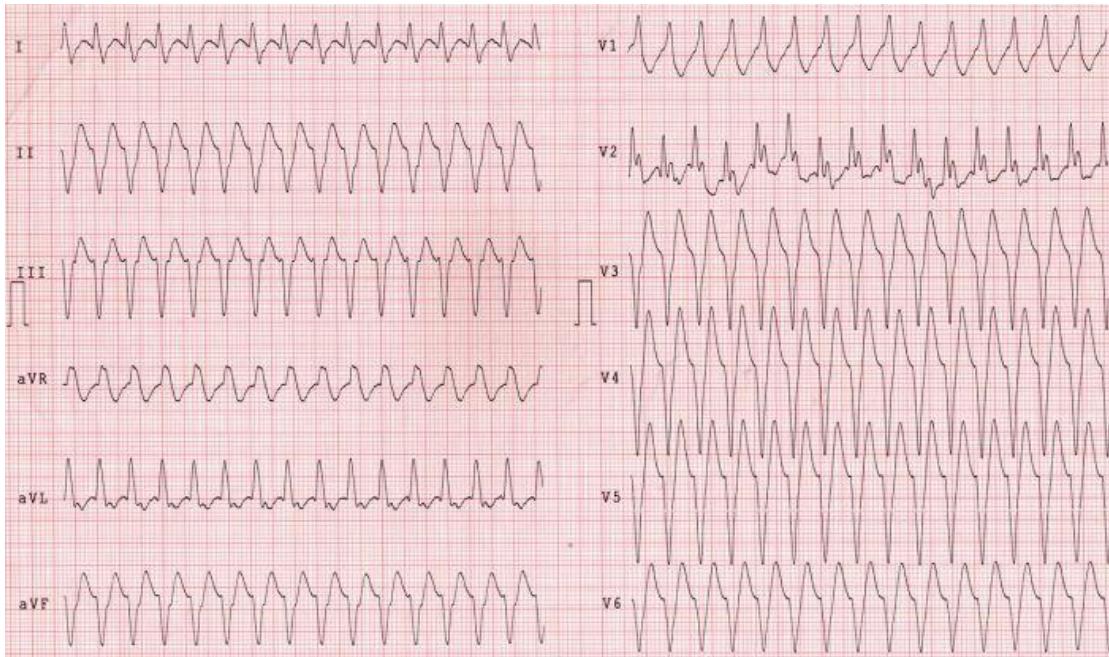


SVT

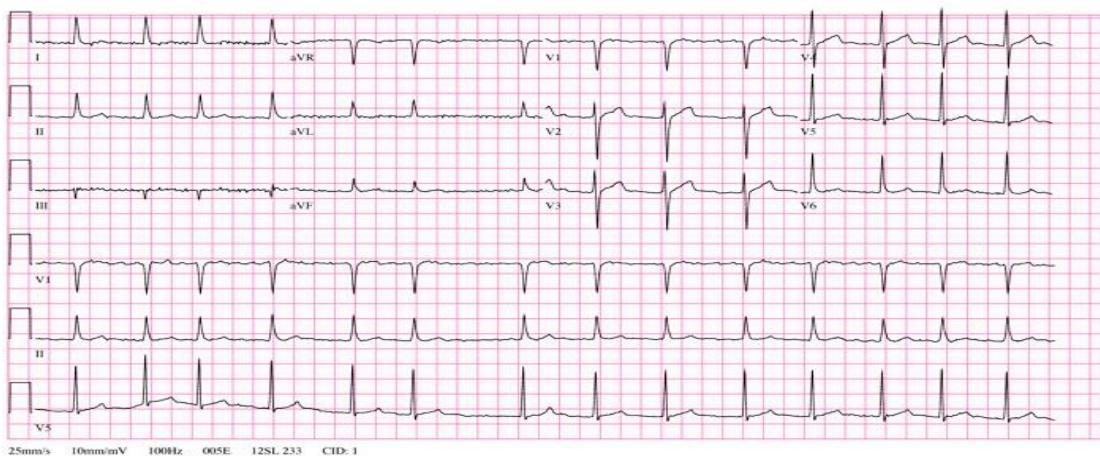
Vtac



Ventricular fibrillation



Atrial fibrillation



Atrial flutter



Acute cardiac ischemia (Acute Coronary Syndromes)

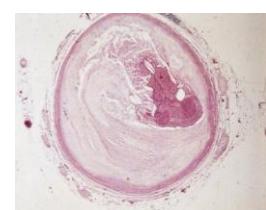
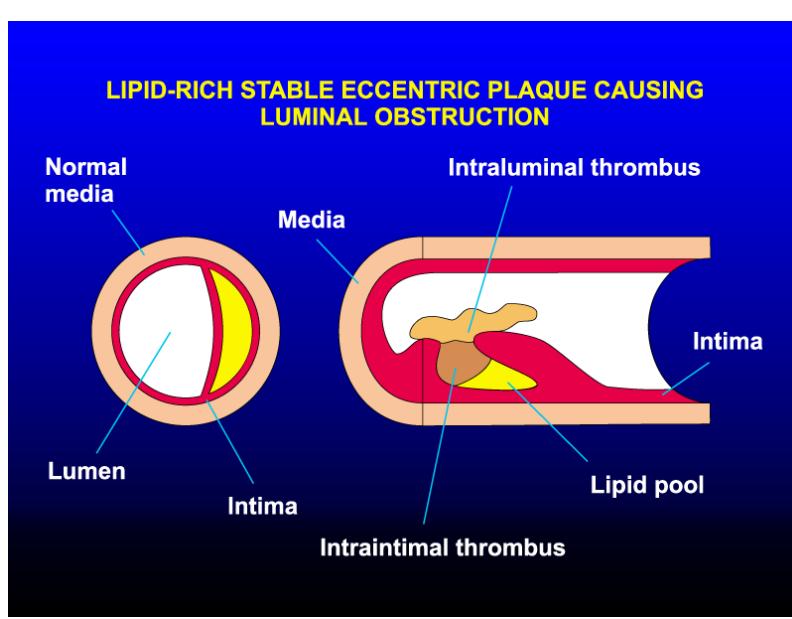
Learning Outcomes

- To understand the clinical spectrum of coronary disease
- To recognise different presentations of the disease process
- To be aware of the different treatment options for each clinical presentation

Acute Coronary Syndromes " ACS"

Spectrum of clinical presentation caused by:

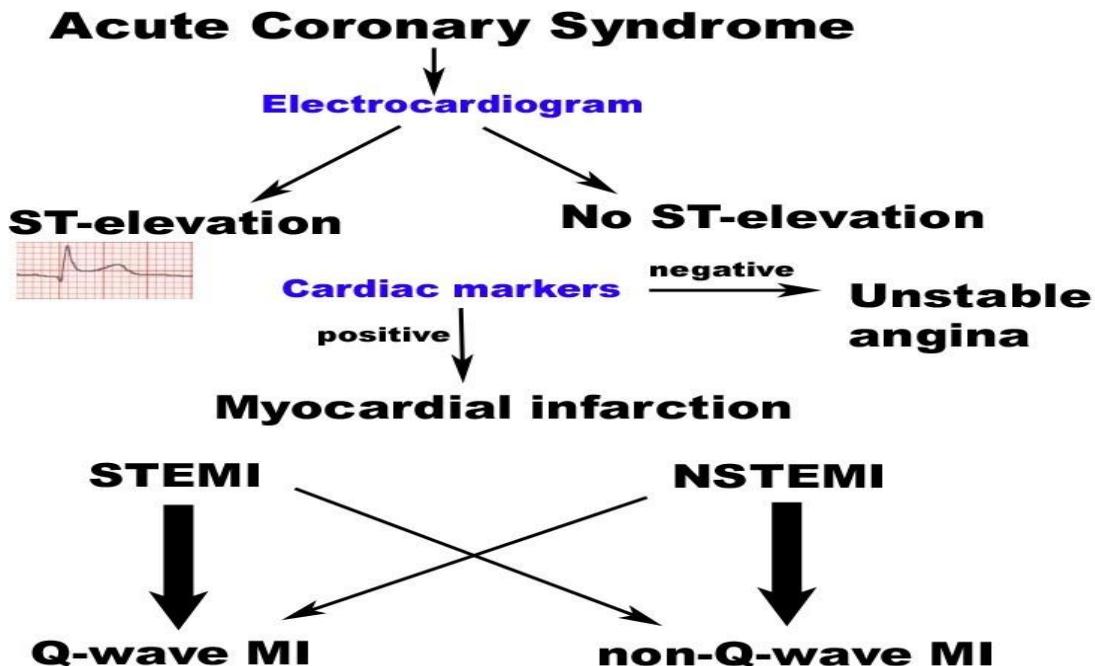
- Atherosclerotic plaque rupture
- Smooth muscle constriction
- Thrombus formation



Classification of ACS

- 1- Unstable angina
- 2- Non-ST-elevation myocardial infarction
- 3- ST-elevation myocardial infarction

Diagrammatic classification



Stable angina

Pain or discomfort from myocardial ischemia:

- Tightness , chest ache
- May radiate to throat, arms, shoulder, back,epigastrium
- Consistently provoked by exercise
- Settles when exercise stops

NOT an acute coronary syndrome

Unstable Angina

1. Angina on exertion with increasing frequency over a few days, provoked by less exertion

OR

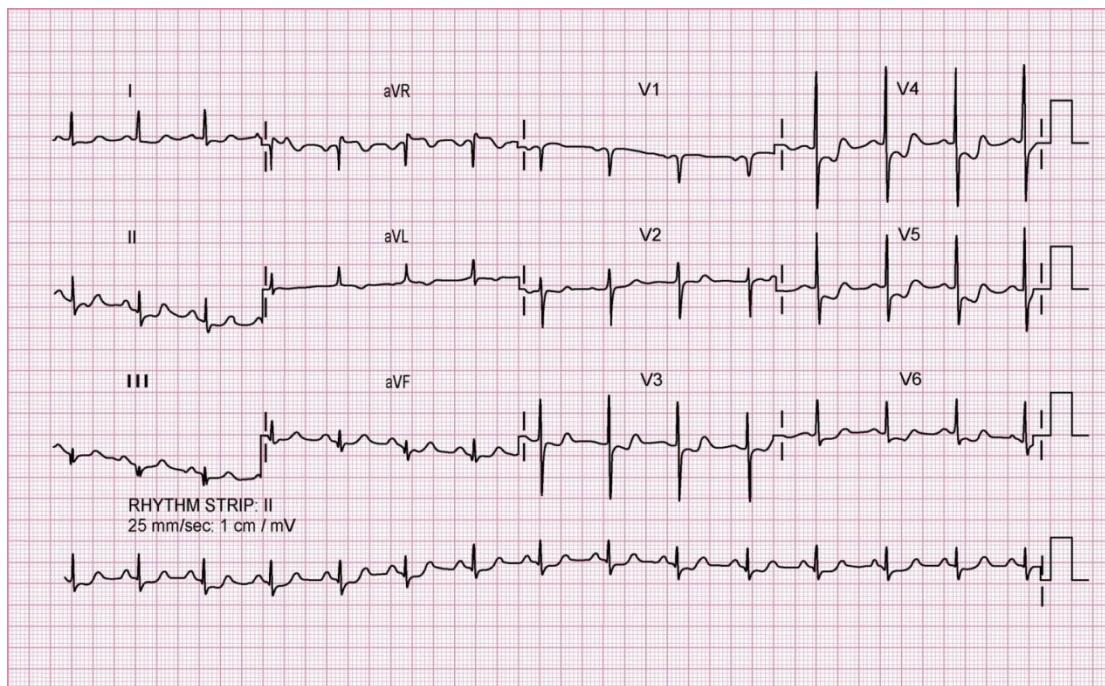
1. Angina occurring recurrently and unpredictably - not specific to exercise

OR

1. Unprovoked and prolonged episode of chest pain

- ECG may be normal
- ST segment depression suggests high risk
- No troponin release
- Cardiac enzymes usually normal

Acute ST depression

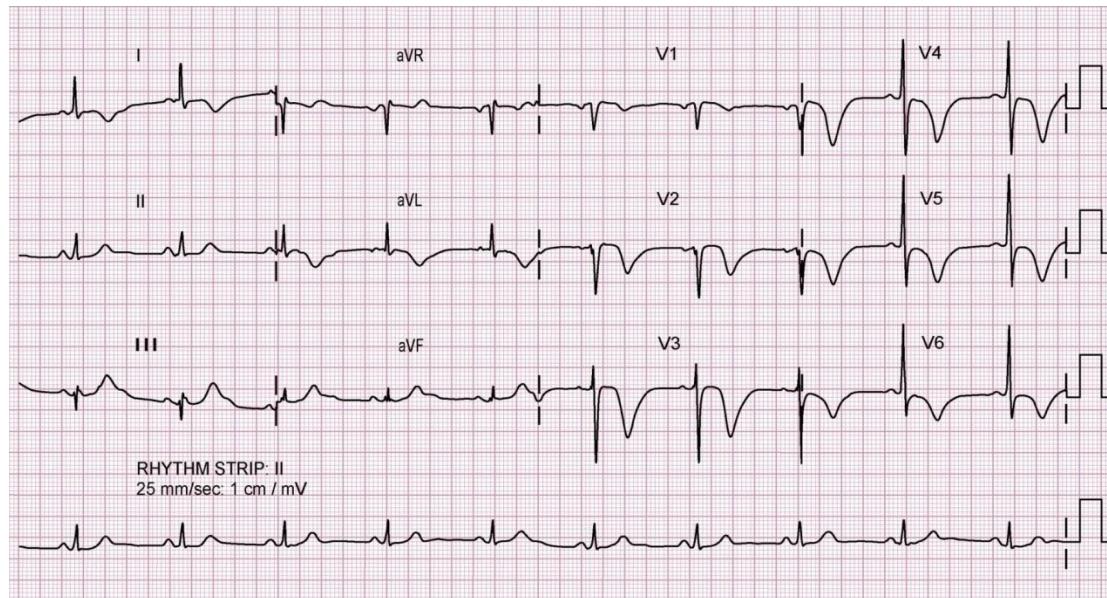


Non-ST-elevation myocardial infarction “NSTEMI”

- Symptoms suggesting acute MI
- Non-specific ECG abnormalities

- ST segment depression
- T wave inversion
- Troponin release
- Usually elevated cardiac enzymes
 - e.g. creatine kinase (CK)

NSTEMI

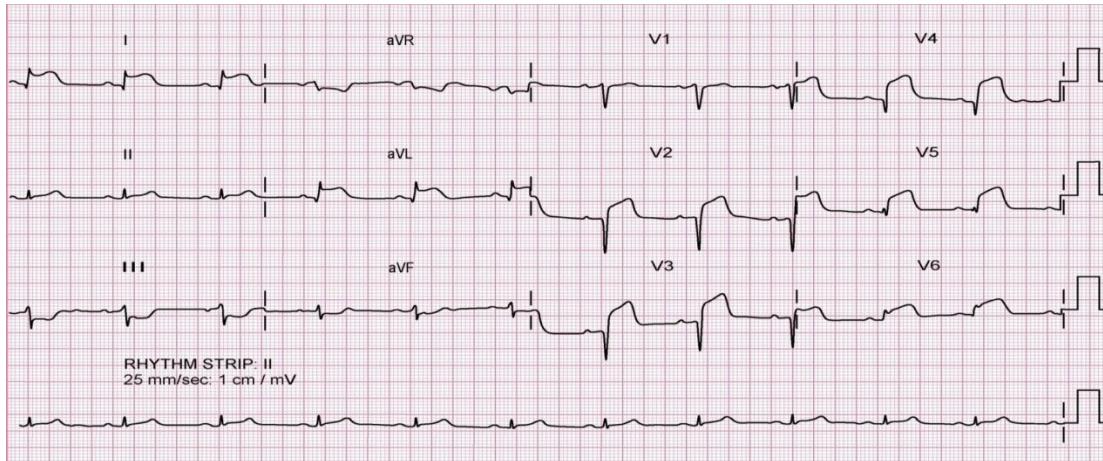


STEMI

- Symptoms suggesting acute MI
- Acute ST segment elevation
- Q waves likely to develop
- Troponin release
- Usually elevated cardiac enzymes (e.g. CK)

- **Early effective treatment may limit myocardial damage and prevent Q wave development**

STEMI



General diagnosis of MI

- The onset is usually gradual.
- Chest pain is often tightness, pressure, or squeezing.
- Radiating mostly to the left arm, the lower jaw, neck, right arm, back, and epigastrium.
- Levine's sign, thought to be predictive of chest pain, where the patient localizes the chest pain by clenching his fist over the sternum.
- Dyspnea
- Diaphoresis
- Increasing of cardio-specific enzymes – Troponin.
- Echocardiography :akinesia, hypokinesia.
- Autopsy of myocardium (thrombus ,necrosis.)
- ECG: Acute changes segment ST, acute left bundle- branch block.
- ECG: Pathological Q.

- New elevation of ST at 2 or more leads .
- In ECG Q wave MI – pathological Q and inversion T.
- In ECG non Q wave MI – complex QRS is normal, no pathological Q, but it is inversion T.

ECHO Criteria

- New regional wall motion abnormalities on an Echo are also suggestive of MI .
- Akinesia, hypokinesia, post infarction cardiosclerosis, myocardial aneurysm
- , intracardiac thrombus, systolic or diastolic dysfunction of myocardium, EF , pulmonary hypertension, hypertrophy of LV, LA etc.

Treatment

Immediate ttt for ACS “all types”

ABCDE approach

- Aspirin 300 mg orally (crush/chew)
- Nitrate (GTN spray or tablet)
- Oxygen if appropriate ****
- Morphine (or diamorphine)
- Anti-thrombotic
 - Aspirin
 - Clopidogrel or prasugrel
 - LMW heparin or fondaparinux
 - If very high risk: glycoprotein IIb/IIIa inhibitor
- Pain relief
 - Nitrate "2x1"
 - Morphine
- Oxygen if appropriate ***

- Myocardial protection

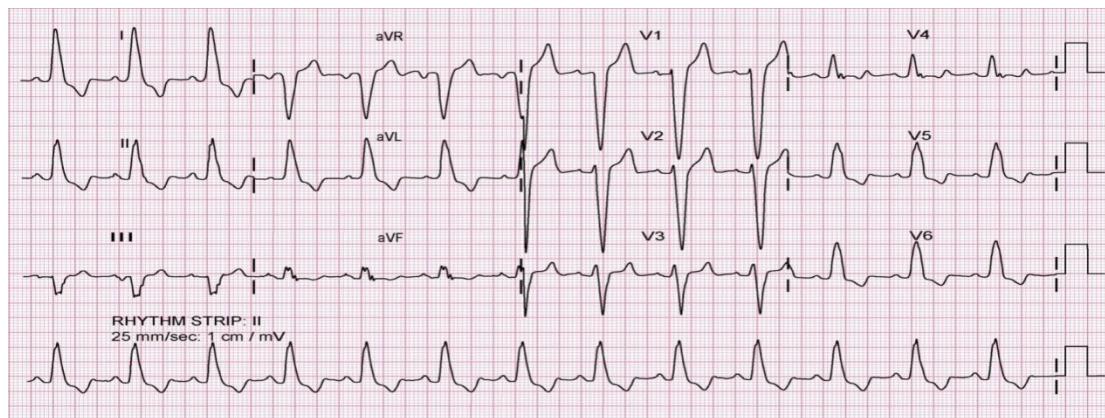
- Beta blocker
- Coronary angiography/PCI in most patients

Emergency reperfusion therapy:

- Percutaneous coronary intervention (PCI)

Fibrinolytic therapy Avoid delay – “Time is muscle”

LBBB



Absolute contraindications to fibrinolytic therapy

- Previous haemorrhagic stroke
- Other stroke or CVA within 6 months
- CNS damage or neoplasm
- Active internal bleeding
- Aortic dissection
- Recent major surgery or trauma
- Known bleeding disorder

STEMI – further management

- Anti-thrombotic therapy

- Beta blocker
- ACE inhibitor
- Coronary angiography and reperfusion strategies e.g. PCI

Brady arrhythmia

Learning outcomes

At the end of this subject you should:

- Be able to recognize bradycardia and differentiate between the different degrees of heart block
- Understand the principles of treating bradycardia
- Understand the indications for cardiac pacing
- Be aware of the different methods available for cardiac pacing
- Know how to apply non-invasive, transcutaneous electrical pacing safely and effectively

Normal Impulse Conduction

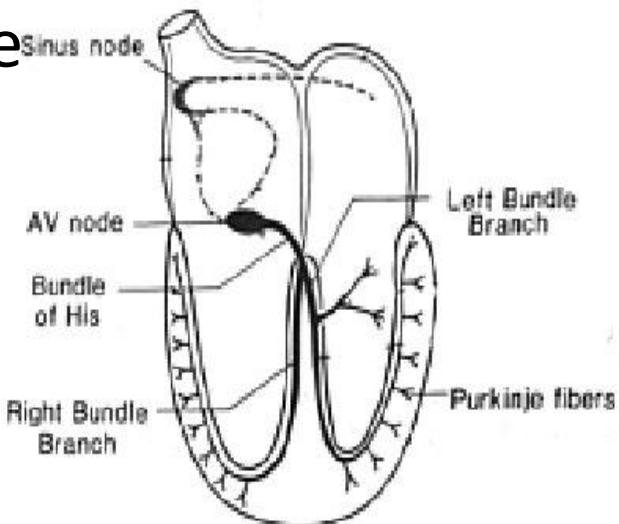
Normal Impulse Conduction

Sinoatrial node

AV node

Bundle of His

Bundle Branches



P wave = Atrial depolarization

QRS = Ventricular depolarization

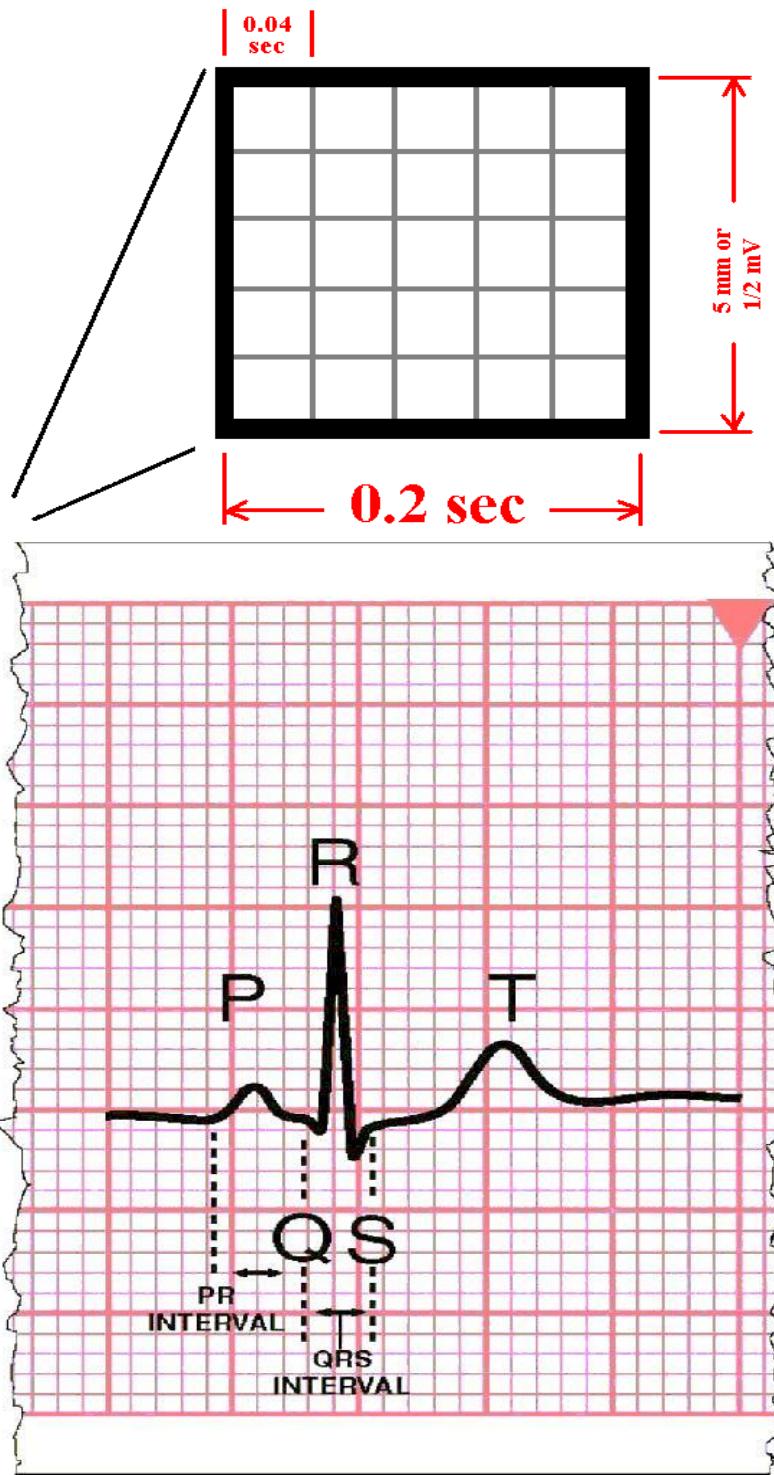
T wave = Ventricular repolarization

Pacemakers of the Heart

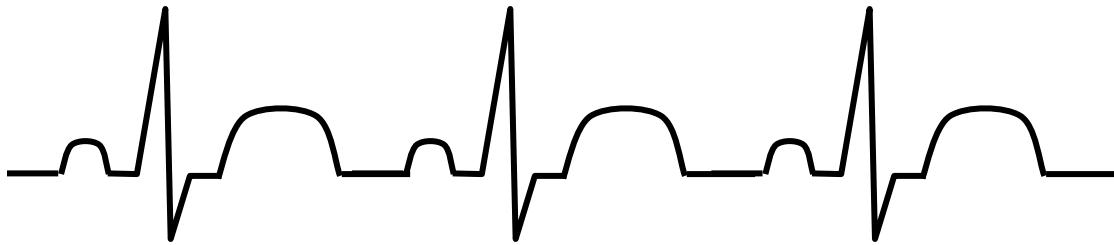
- SA Node - Dominant pacemaker with an intrinsic rate of 60 - 100 beats/minute.
- AV Node - Back-up pacemaker with an intrinsic rate of 40 - 60 beats/minute.
- Ventricular cells - Back-up pacemaker with an intrinsic rate of 20 - 45 bpm.

The ECG Paper

- Horizontally
 - One small box - 0.04 s
 - One large box - 0.20 s
- Vertically
 - One large box - 0.5 Mv



Normal sinus rhythm



- o each P wave is followed by a QRS
- o P waves normal for the subject
- o P wave rate 60 - 100 bpm with <10% variation
- o rate <60 = sinus bradycardia
- o rate >100 = sinus tachycardia
- o variation >10% = sinus arrhythmia

Rate calculation

Normal rate 60 : 100 b/min
If regular 300/n.of large squares between R.....R
If irregular n.of R in 15 large squares x 10

Abnormal rhythms from the Sinus Node

Sinus Tachycardia:

HR > 100 b/m

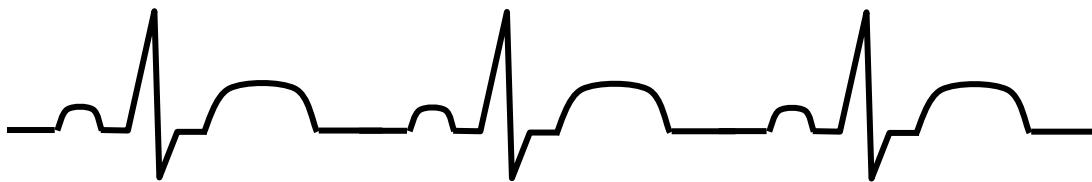
- Causes:
 - Withdrawal of vagal tone & Sympathetic stimulation (exercise, fight or flight)
 - Fever & inflammation
 - Heart Failure or Cardiogenic Shock (both represent hypoperfusion states)

- Heart Attack (myocardial infarction or extension of infarction)
- Drugs (alcohol, nicotine, caffeine)

Sinus Bradycardia:

HR < 60 b/m

- Causes:
 - Increased vagal tone, decreased sympathetic output, (endurance training)
 - Hypothyroidism
 - Heart Attack (common in inferior wall infarction)
 - Vasovagal syncope (people passing out when they get their blood drawn)
 - Depression



Sinus Arrhythmia Variation in HR by more than .16 seconds

- Mechanism:
 - Most often: changes in vagal tone associated with respiratory reflexes
 - Benign variant
- Causes
 - Most often: youth and endurance training



Sick Sinus Syndrome: Failure of the heart's pacemaking capabilities

- Causes:
 - Idiopathic (no cause can be found)
 - Cardiomyopathy (disease and malformation of the cardiac muscle)
- Implications and Associations
 - Associated with Tachycardia / Bradycardia arrhythmias
 - Is often followed by an ectopic “escape beat” or an ectopic “rhythm”



Rhythm Analysis



- Step 1: Calculate rate.
- Step 2: Determine regularity.
- Step 3: Assess the P waves.
- Step 4: Determine PR interval.
- Step 5: Determine QRS duration

How to read normal and abnormal rhythm strip

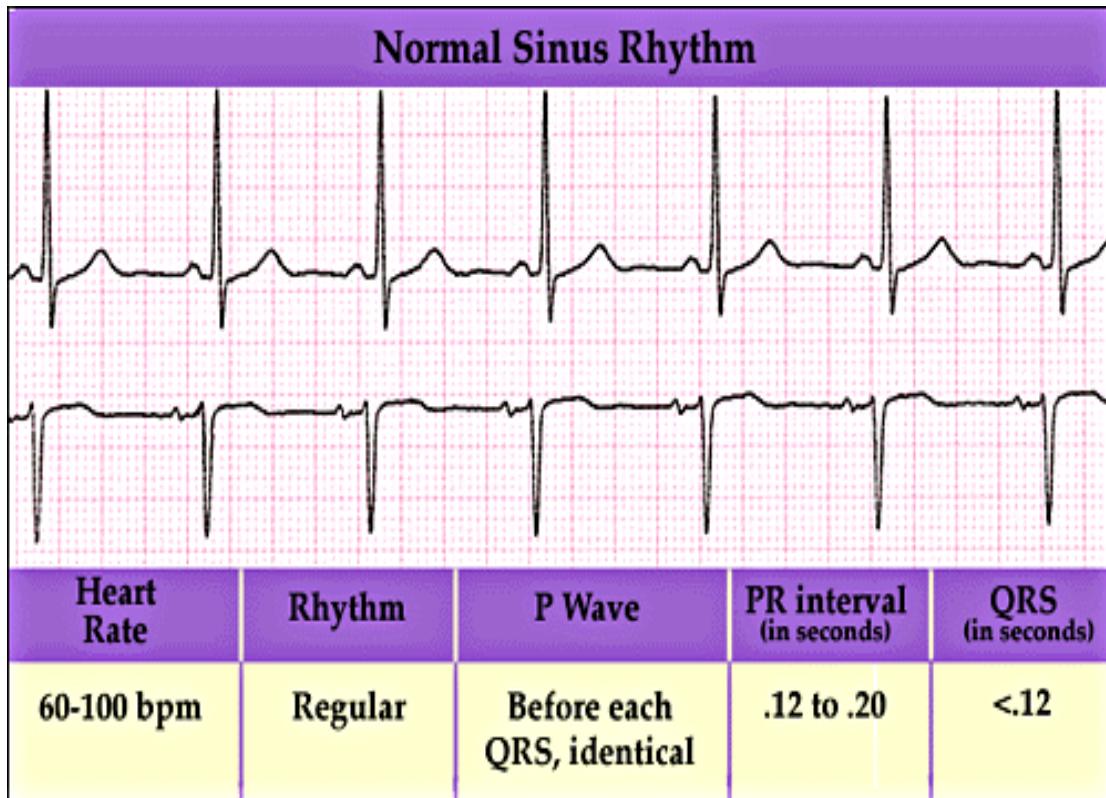
Is there any electrical activity? YES

2. What is the ventricular (QRS) rate?
3. Is the QRS rhythm regular or irregular?
4. Is the QRS width normal or prolonged?

5. Is atrial activity present?

(If so what is it? Normal P waves? Other atrial activity?)

6. How is atrial activity related to ventricular activity?



Rhythm 1 sinus brady cardia



Rate? 30 bpm

Regularity? regular

P waves? normal

PR interval? 0.12 s

QRS duration? 0.10 s

Interpretation? *Sinus Bradycardia*

So , Sinus Bradycardia is :-

- Deviation from NSR
- Rate < 60 bpm
- Etiology: SA node is depolarizing slower than normal, impulse is conducted normally (i.e. normal PR and QRS interval).

AV Nodal Blocks

- *1st Degree AV Block*
- *2nd Degree AV Block, Type I*
- *2nd Degree AV Block, Type II*
- *3rd Degree AV Block*

first degree block

- There is a delay in conduction of the atrial impulse to the ventricles, usually at the level of the AVN.
- This results in prolongation of the PR interval to > 0.2 s. QRS complex follows each P wave, and the PR interval remains constant.

Rhythm 2 first degree block



Rate? 60 bpm

Regularity? regular

P waves? normal

PR interval? 0.36 s

QRS duration? 0.08 s

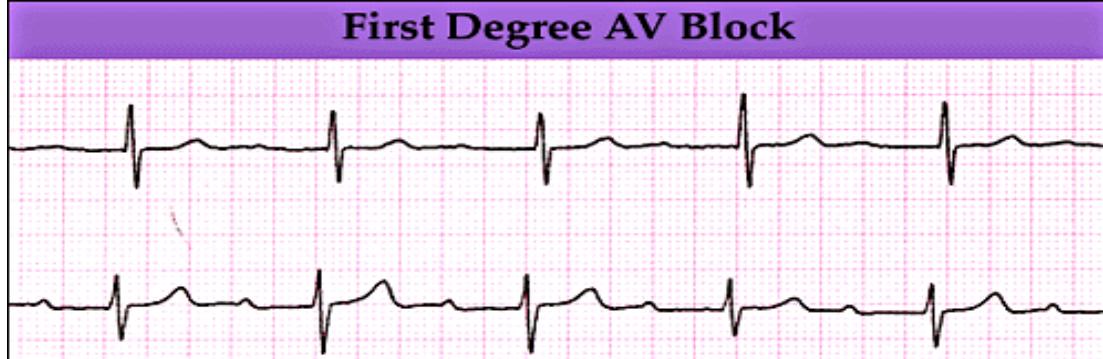
Interpretation? *1st Degree AV Blockz*



So, 1st Degree AV Block is:-

- Deviation from NSR
 - PR Interval > 0.20
- Etiology: Prolonged conduction delay in the AV node or Bundle of His.

First Degree AV Block			
P Wave	PR Interval (in seconds)	QRS (in seconds)	Characteristics
Before each QRS, identical	>.20	>.12	Regular rhythm



second degree block

- There is intermittent failure of conduction between the atria and ventricles. Some P waves are not followed by a QRS complex.

Mobitz type I block (Wenckebach phenomenon)

- The initial PR interval is normal but progressively lengthens with each successive beat until eventually atrioventricular transmission is blocked completely and the P wave is not followed by a QRS complex.
- The PR interval then returns to normal, and the cycle repeats.

Rhythm 3 2nd Degree AV Block, Mobitz Type I



Rate? 50 bpm

Regularity? regularly irregular

P waves? nl, but 4th no QRS

PR interval? lengthens

QRS duration? 0.08 s

Interpretation? 2nd Degree AV Block, Type I

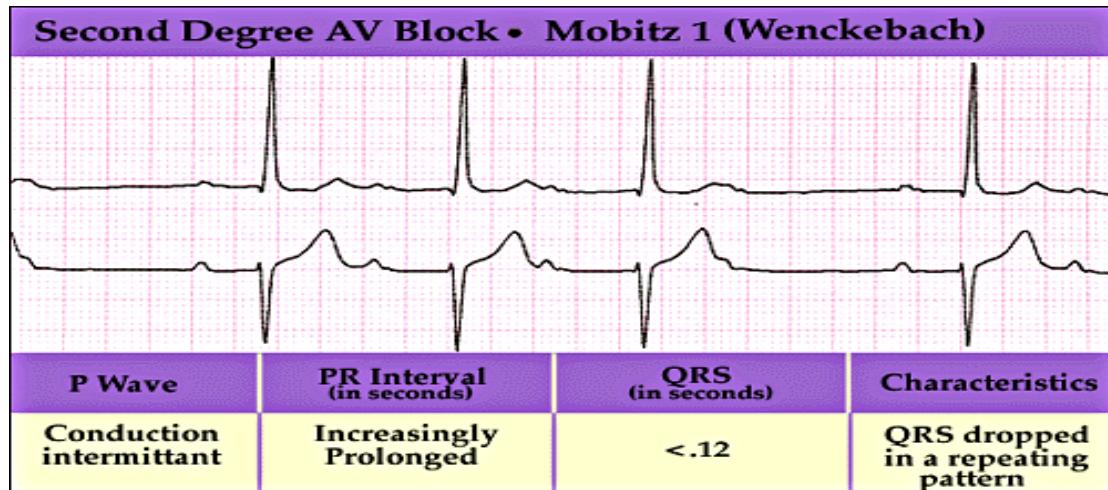
So 2nd Degree AV Block, Mobitz Type I is :-



- *Deviation from NSR*

- *PR interval progressively lengthens, then the impulse is completely blocked (P wave not followed by QRS).*

- Etiology: Each successive atrial impulse encounters a longer and longer delay in the AV node until one impulse (usually the 3rd or 4th) fails to make it through the AV node.



2nd degree heart block Mobitz type II

less common but is more likely to produce symptoms. There is intermittent failure of conduction of P waves. The PR interval is constant, though it may be normal or prolonged. The block is often at the level of the bundle branches and is therefore associated with wide QRS complexes.

Progress to complete third degree atrioventricular block.

Rhythm 4 2nd degree heart block Mobitz type II

Rate? 40 bpm

Regularity? regular

P waves? nl, 2 of 3 no QRS

PR interval? 0.14 s

QRS duration? 0.08 s

Interpretation? 2nd Degree AV Block, Type II

So 2nd Degree AV Block, Type II is :-



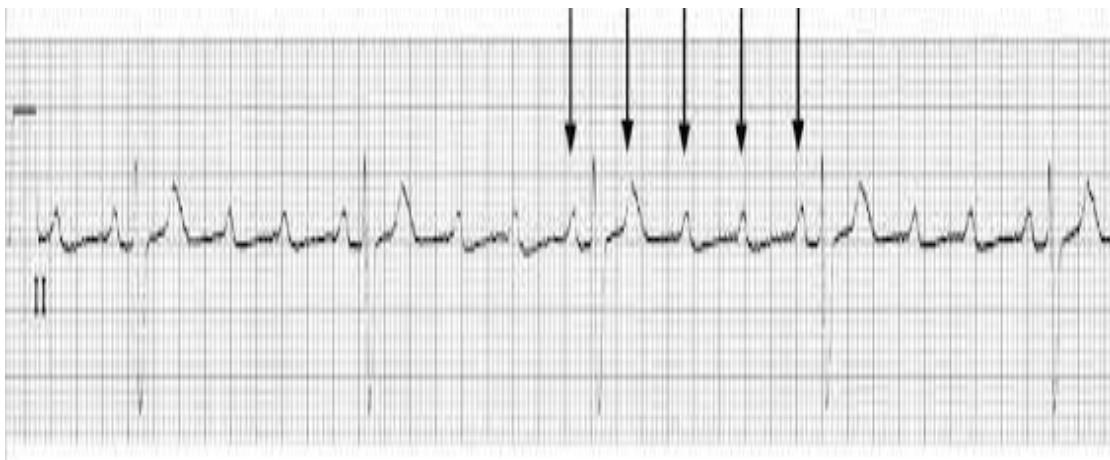
■ **Deviation from NSR**

- Occasional P waves are completely blocked (P wave not followed by QRS).
- **Etiology:** Conduction is all or nothing (no prolongation of PR interval); typically block occurs in the Bundle of His.

2:1 AV Block



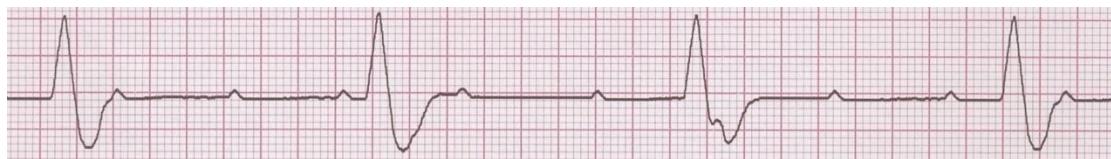
A V Block 2nd degree fixed ratio



Third degree AV block

- There is complete failure of conduction between the atria and ventricles, with complete independence of atrial and ventricular contractions. The P waves bear no relation to the QRS complexes and usually proceed at a faster rate.(A V dissociation)

Rhythm 3 Third degree AV block



Rate? 40 bpm

Regularity? regular

P waves? no relation to QRS

PR interval? none

QRS duration? wide (> 0.12 s)

Interpretation? 3rd Degree AV Block

So, 3rd Degree AV Block



- Deviation from NSR

- The P waves are completely blocked in the AV junction; QRS complexes originate independently from below the junction.

- Etiology: There is complete block of conduction in the AV junction, so the atria and ventricles form impulses independently of each other. Without impulses from the atria, the ventricles own intrinsic pacemaker kicks in at around 30 - 45 beats/minute.

- Remember

When an impulse originates in a ventricle, conduction through the ventricles will be inefficient and the QRS will be wide and bizarre.



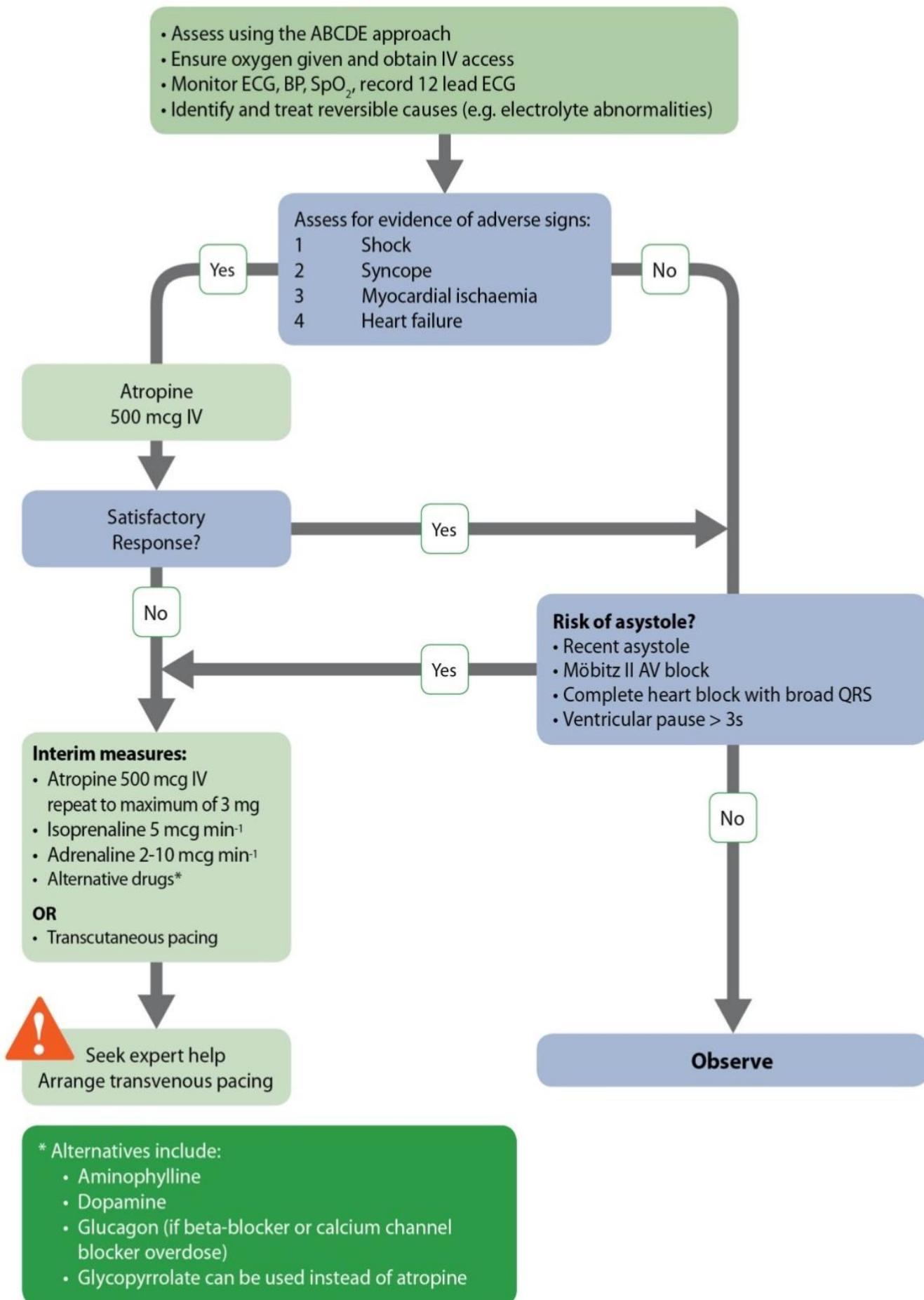
Idioventricular Rhythms



Heart Rate	Rhythm	P Wave	PR interval (in seconds)	QRS (in seconds)
20-40	Regular	Absent or not related	N/A	$\geq .12$

Bradycardia algorithm

Includes rates inappropriately slow for haemodynamic state

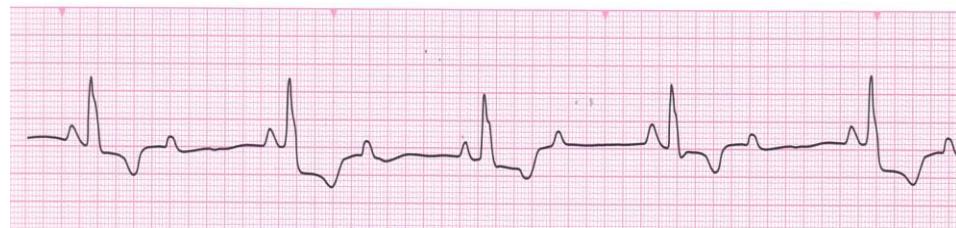


Management of brady cardia

Case study

■ Clinical setting and history

- 60-year-old man referred to admissions unit by GP
- Long-term history of heart disease
- Feeling light-headed and breathless



Clinical course

- ABCDE
 - A : Clear
 - B : Spontaneous breathing, rate 18 min^{-1}
 - C : Looks pale, P 45 min^{-1} , BP $90/50 \text{ mmHg}$, CRT 3 s

Initial rhythm?

- D : Alert, glucose 4.5 mmol l^{-1}
- E : Nil of note

What action will you take for management

1- Atropine

Indication

- Symptomatic bradycardia

Contraindication

- *Do not give to patients who have had a cardiac transplant*

Dose

- *500 mcg IV, repeated every 3 - 5 min to maximum of 3 mg*

Actions

- *Blocks vagus nerve*
- *Increases sinus rate*
- *Increases atrioventricular conduction*

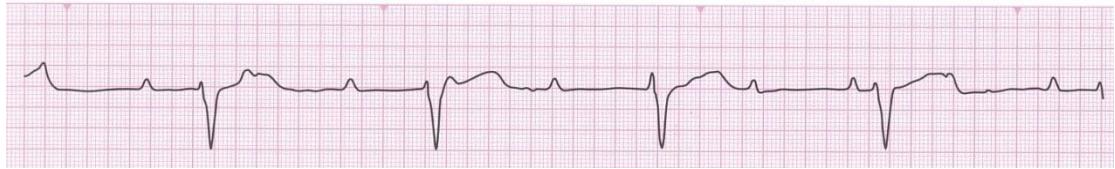
Side effects

- *Blurred vision, dry mouth, urinary retention*
- *Confusion*

No response to atropine

- *Patient becomes more breathless, cold, clammy and mildly confused*
- *Change in rhythm*
- *ABCDE*
 - *A : Clear*
 - *B : Spontaneous breathing, rate 24 min⁻¹
widespread crackles on auscultation*
 - *C : Looks pale, HR 35 min⁻¹, BP 80/50 mmHg, CRT 4 s*
 - *D : Responding to verbal stimulation*
 - *E : Nil of note*

What will you do now?



2- Pacing

NOT

NOT READY YET

3- Adrenaline

Infusion of 2-10 mcg min⁻¹ titrated to response

OR 4- Isoprenaline infusion 5 mcg min⁻¹ as starting dose

OR 5-Dopamine infusion 2-5 mcg kg⁻¹ min⁻¹

6-Consider need for expert help

7-Prepare for transcutaneous pacing

8- Consider percussion pacing as interim measure

9-Confirm electrical capture and mechanical response once transcutaneous pacing has started

Pace maker

