CPR Manual

Based on American Heart Association 2015 Guidelines

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VC'S FOREWORD

Message from the Vice Chancellor,

I am Pleased to know that the Medical Simulation Centre, MGMCRI plans to bring out second version of simplified manual on CPR, based on AHA 2015 recommendations. Medical Simulation Centre has been conducting AHA approved courses on BLS, ACLS and PALS, training hundreds of health care providers from different backgrounds . The centre has realized the need for a simplified CPR manual with flow charts, algorithms that are easily understandable which helps in rapid decision making .

The new version incorporated drugs used during resuscitation . The team of Medical Simulation Centre deserves appreciation for bringing up this second version.

Prof. Subhash Chandra Parija

Foreword

Message from the Director Simulation Centre,

Basic Life Support and Advanced Cardiac Life support are today's essential skills for all doctors. This training helps to tackle immediate life threats and saves countless lives worldwide. The MSC, MGMCRI is proud to be one of the International Training Centres for these courses in India. This in house manual edited by Prof. HemanthKumar VR. HOD of Anaesthesia, ITC Coordinator is a simplified algorithmic approach to a very confusing subject and I am sure it will be immensely helpful for all participants in these courses. I commend the entire authorship of this booklet for an excellent job.

Prof. Dinker R Pai

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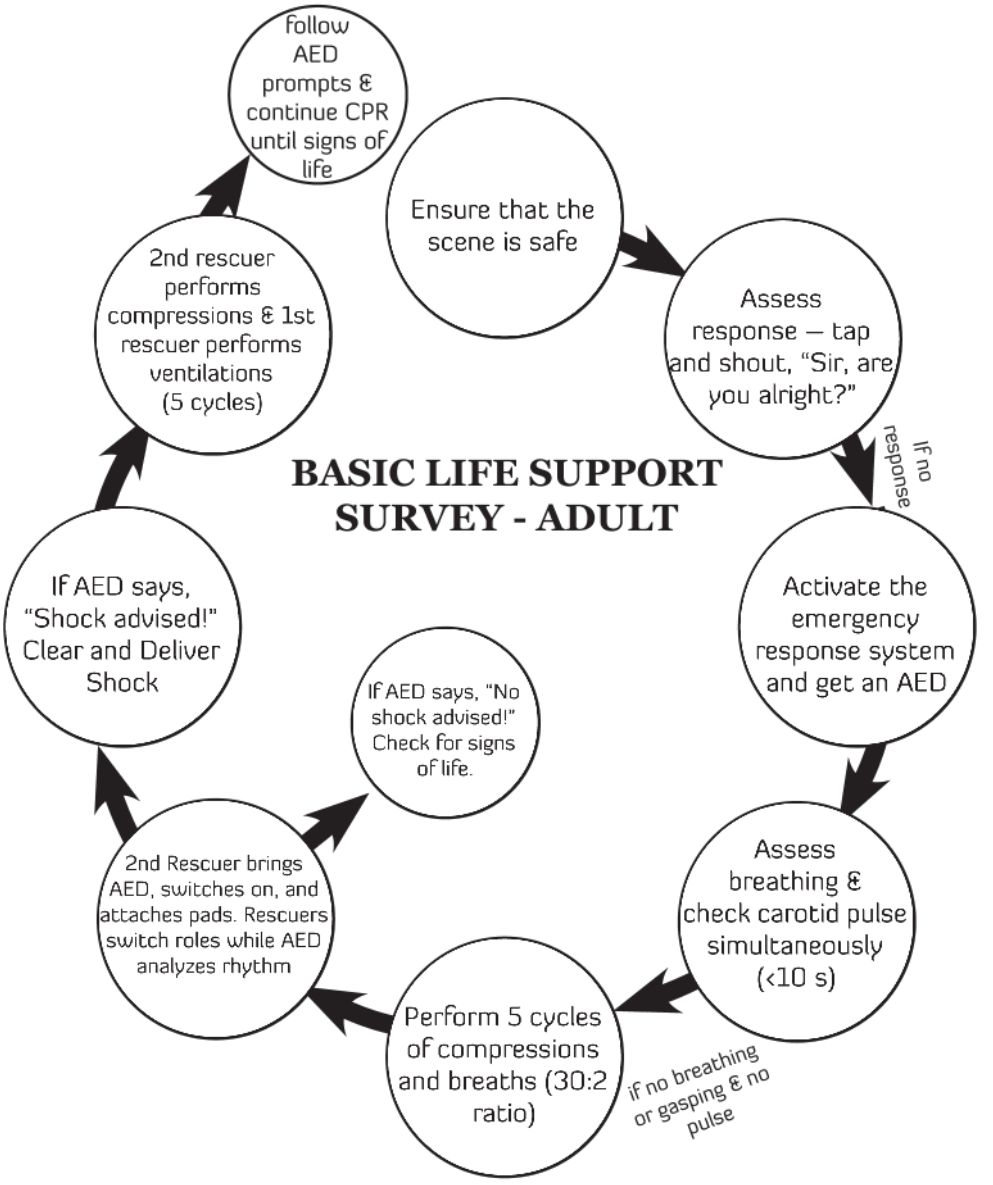
Basic Life Support (BLS)

Advanced Cardiovascular Life Support (ACLS)

Basic Life Support (BLS)

BLS Survey – Adult Algorithm



Assessing Response

* Tap: on the shoulder
* Shout: sir, are you okay?

(Call for help) Activating Emergency Response System

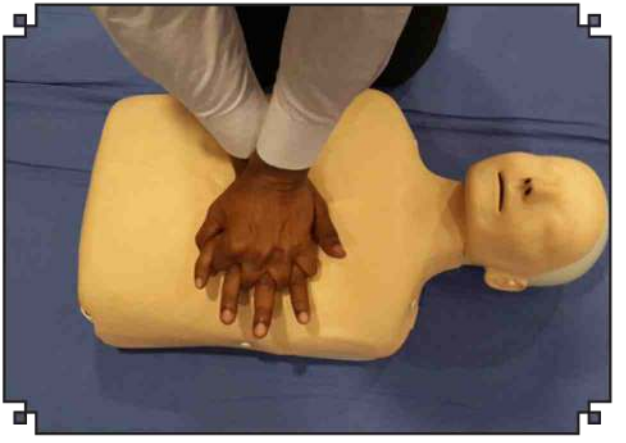
* Shout for help
* Call 108 & ask for AED

Check pulse and breathing

* Simultaneously Scan the chest movement for breathing & check carotid pulse (in the groove between trachea & sternocleidomastoid muscle) atleast 5sec & not more than 10 sec

Hand Position

* Heel of the palm of one hand on the center of lower half of sternum (i.e) center of two nipples
* Support with other hand



Performing CPR

* Wrist,elbows,shoulders in a straight line
* Movement should come from hip joint

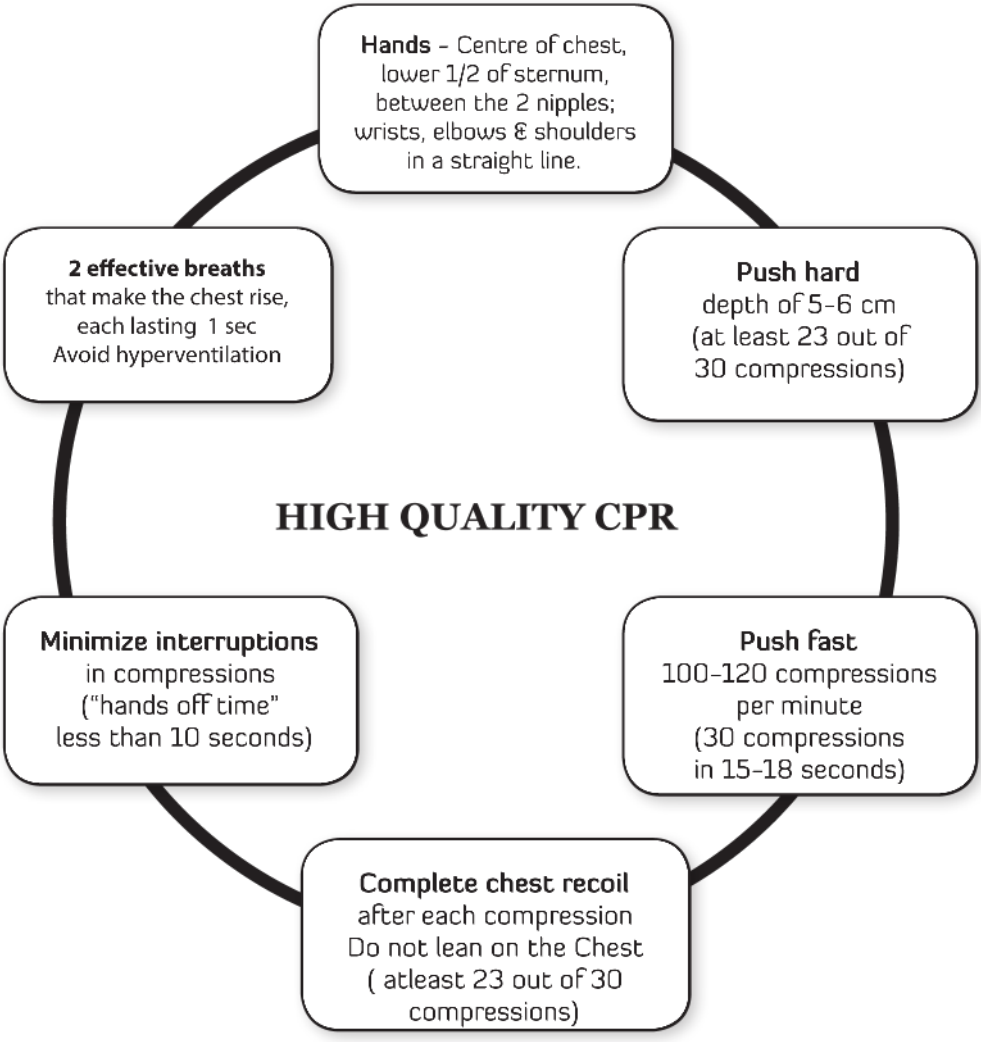
Breath using a pocket mask

* Keep the pocket mask over the face tightly
* Do head tilt & chin lift if no suspected neck injury
* Give 2 breaths (1 breath over 1 sec)
* Do not hyperventilate
* Watch for chest rise

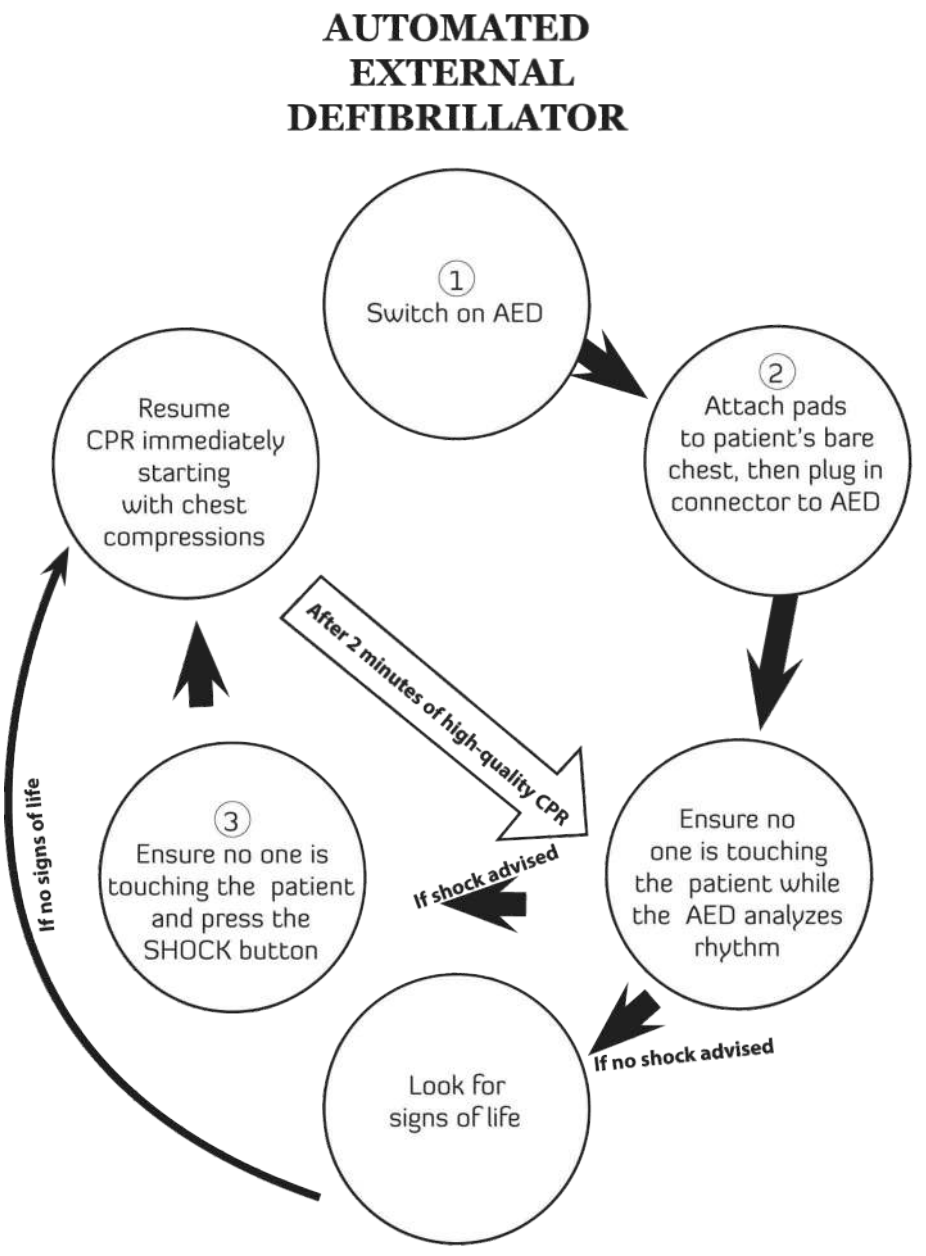
2 Rescuer CPR with Bag mask ventilation

* 1st rescuer gives 30 compression
*2nd rescuer positioned at head end of victim & gives 2 breaths using bag mask ("C &E" technique to hold the mask, head tilt & chin lift)
* 1breath over 1 sec
* Do not hyperventilate
* Watch for chest rise

Critical Concepts of High Quality CPR



AED Algorithm



Use of AED on infants

1. A manual defibrillator is preferred
2. If not available, an AED with a paediatric dose attenuator is preferred.
3. Use an AED without a paediatric dose attenuator If neither is available.

Use of AED pads in children less than 8 years of age

1. An AED with child pads.
2. If child pads are not available, use adult pads (but not touching each other).

1. Switching on AED

Rationale:

To listen & Follow the AED prompts

2.a. Applying AED pads to patient's chest

Rationale:

To analyse the rhythm.

*Attach the apex pad just below the left nipple

*Attach the sternal pad just below the right clavicle

2.b. Plugging in pads connector to AED

Rationale:

This step should not be done before attaching the pads since AED will start analyse the rhythm even if the pads are in the air

2.c. Clearing the patient before rhythm analysis

Rationale:

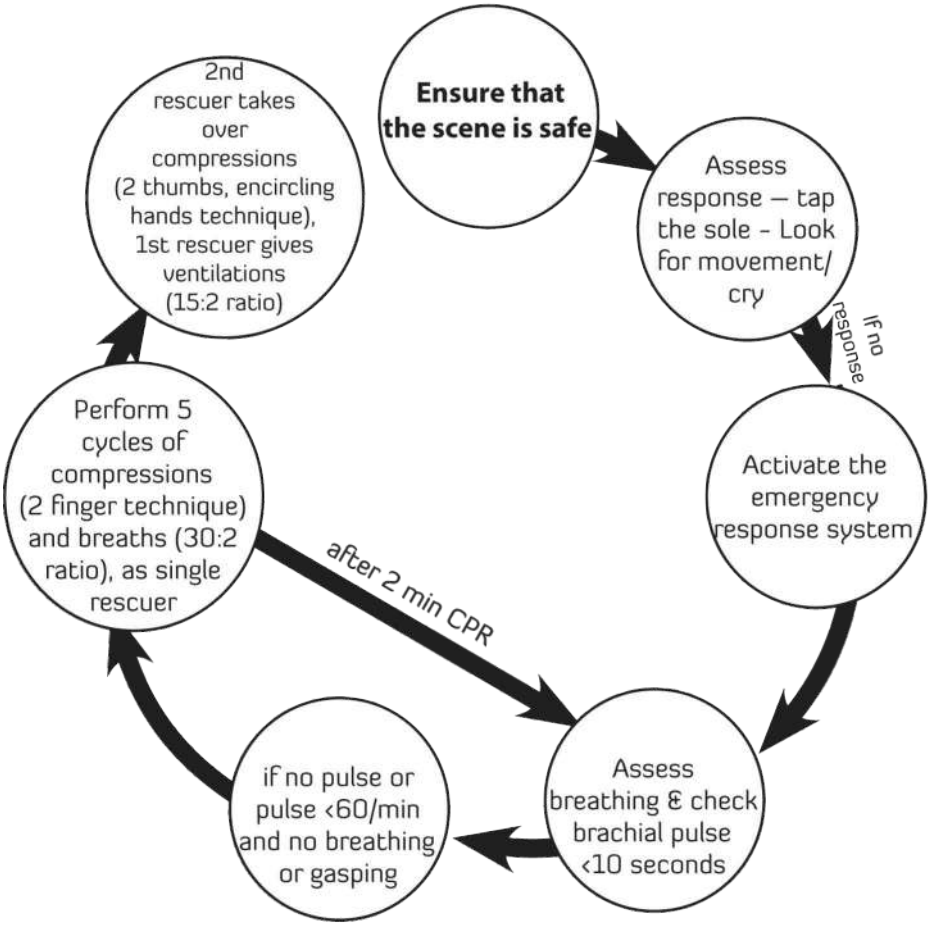
To avoid misinterpretation of rhythm by the AED

3. Delivery of shock

Rationale:

Press the shock button manually after ensuring that no one is touching the victim

BLS Survey – Infant Algorithm



* If only one rescuer is doing CPR after 2 min. of CPR look for signs of life. If absent continue 5cycles of CPR.

Checking response

* Tap: on the sole
* Shout : hey Papa..

Activating Emergency Response System (Calling for help)

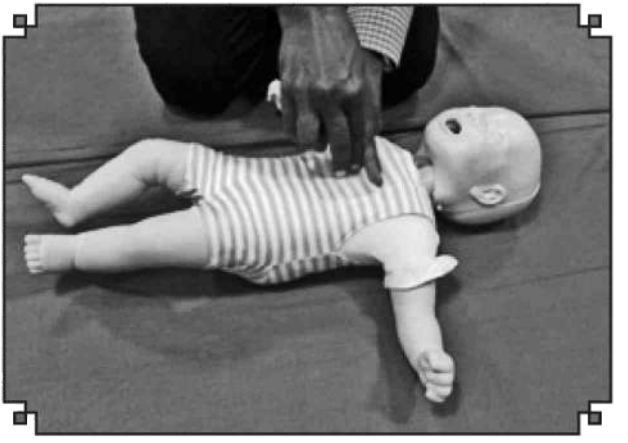
* Shout for help
* Call 108 & ask for AED

Pulse and breathing

Simultaneously Scan the chest movement andcheck brachial pulse bygently compressing the brachial artery with the pulp of 3 or 4 fingers in the inner aspect of arm, midway between shoulder & elbow,atleastfor 5sec
and not more than 10 sec.

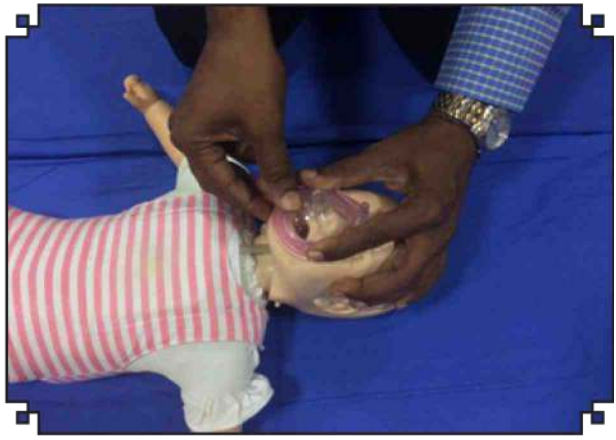
2 finger technique

* 2 fingers perpendicular to the center of the lower half of sternum (center of 2 nipples)
* 30 compressions with the depth of one and half inches or 4cm
* Allow complete chest recoil after each compression



Using a pocket mask

* Keep the pocket mask over the face tightly
* Head should be in the neutral position
* Do not hyper extend the neck
* Give 2 breaths (1 breath over 1 sec)
* Do not hyperventilate
* Watch for chest rise

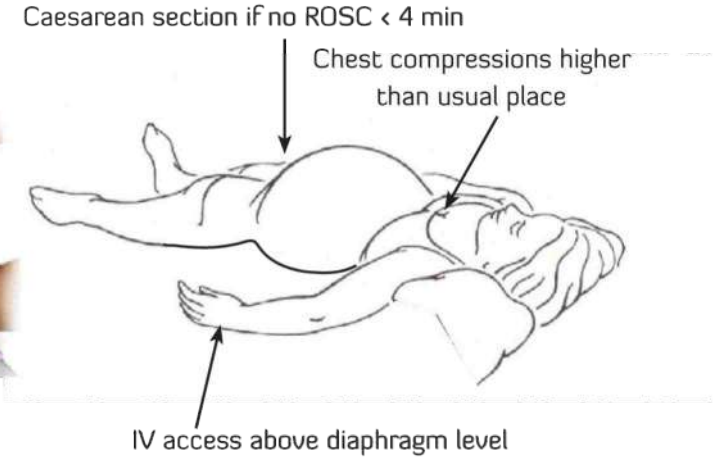


Two thumbs hands encircling technique(2 Rescuer CPR)

* 1 rescuer: positioned at foot end of infant
* 2 thumb hand encircling technique
* 15 compression
* 2 rescuer: positioned at head end of infant
* Using bag mask ("C & E technique" to hold the mask)
* Give 2 breaths (1 breath over 1 sec)
* Do not hyperventilate
* Watch for chest rise

Pregnancy and CPR

- Follow BLS and ACLS guidelines
- Modified Position – do manual uterine displacement.
- Chest compressions should be performed slightly higher on the sternum than normally recommended to adjust for elevation of diaphragm
- Intravenous access to be secured above diaphragm level
- If no Return Of Spontaneous Circulation by 4 min perform emergency caesarean section
- Aim for delivery within 5 min of onset of resuscitation
- Continue resuscitative efforts during and after caesarean section



Summary of High Quality BLS

Component		Adults	Children	Infants (age<1 yr)
Scene safety		Ensure "Scene is safe"		
Check Response		Tap and Shout, "Are you all right?" Look for movement / speech		Flick the soles of the feet & look for move-ment / cry
Call for Help		Activate Emergency Response System; Get AED		
Check Pulse & Breathing Simultaneously		Check pulse and observe for chest rise (>5 sec but <10 sec)		
		Carotid pulse		Brachial pulse
Hand placement for compressions		Two hands – centre of chest; lower ½ of sternum (between the 2 nipples)	As in adult. One hand alone may be used for small children	Two finger technique (If one rescuer)
				Two thumb-encircling hands technique (if 2 rescuers)
Compression rate		100-120 per min		
Compression depth		At least 5 cm	At least 1/3rd the AP diameter of chest	
			About 5 cm	About 4 cm
Compression / ventilation ratio	Without advanced airway	30:2 for one or two rescuers	30:2 if one rescuer 15:2 if two rescuers	
	With advanced airway	Continuous compressions @ 100-120 per min 1 breath every 6 seconds		

Chest recoil		Allow full re-expansion of chest before next compression	
Minimize interruptions		All interruptions should be <10 seconds (hands off time <10 sec)	
Opening the airway	**No suspicion of cervical spine injury**	Head tilt-chin lift	
		Maximize extension of neck	Avoid hyperextension
	Suspicion of cervical spine injury	Jaw thrust (No head extension)	
Device for giving artificial breaths	**1 rescuer**	Mouth-to-mask / Mouth-to-mouth	
	2 rescuer	Bag-mask device	
Breaths during respiratory arrest		1 breath every 5-6 seconds	1 breath every 3-5 seconds

Activation of Emergency Response System

Adults — If you are alone with no mobile phone, leave the victim to activate the emergency response system and get the AED before beginning CPR. Otherwise, send someone and begin CPR immediately. Use the AED as soon as it is available.

Children & Infants

Witnessed collapse — If you are alone with no mobile phone, leave the victim to activate the emergency response system and get the AED before beginning CPR. Otherwise, send someone and begin CPR immediately. Use the AED as soon as it is available.

Unwitnessed collapse – Give 2 minutes of CPR. Then Leave the victim to activate the emergency response system and get the AED. Return to the child or infant and resume CPR. Use the AED as soon as it is available.

Advanced Cardiovascular Life Support (ACLS)

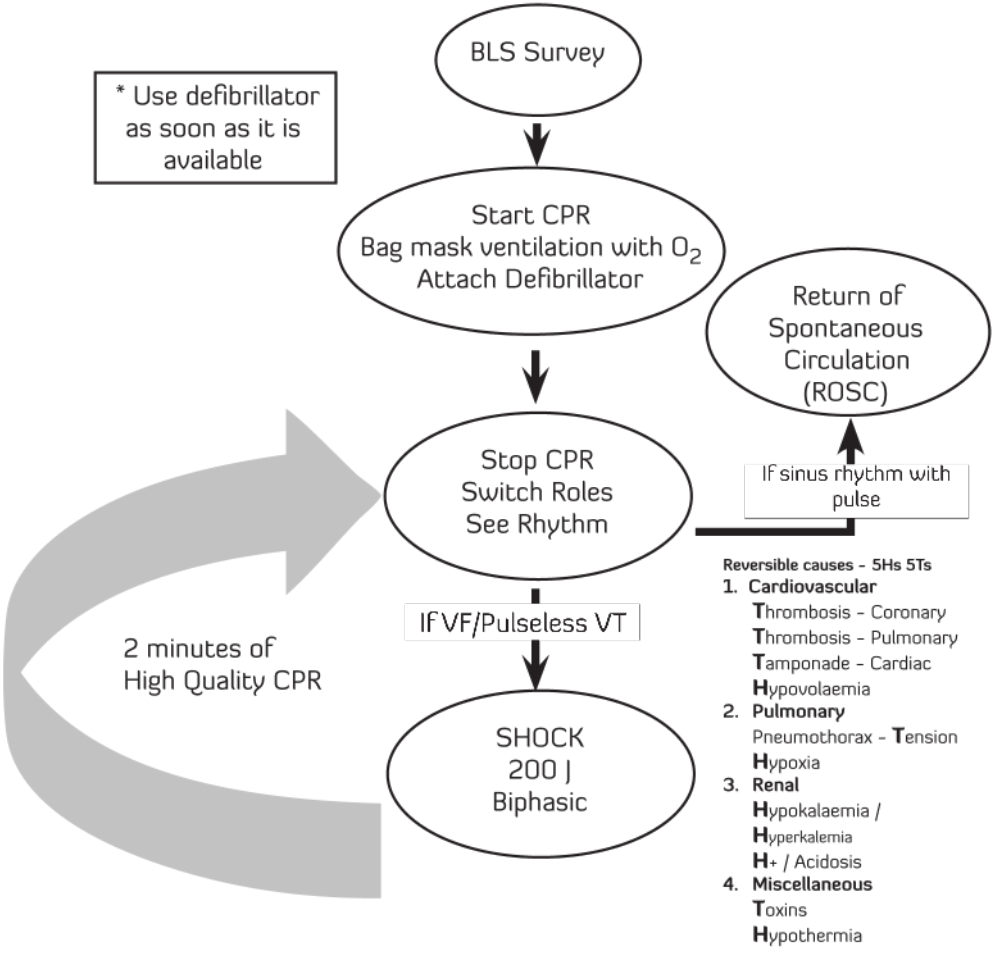
Megacode Resuscitation Team Concept

Effective resuscitation requires coordination between the team leader and team members. The coordination is discussed in the following 8 priniciples.

Principles of team resuscitation

No.	Principle	Example
1	Closed-loop communication	Leader – "Now that we have a shockable rhythm (VF) on the monitor, give 200 J biphasic shock." Member – After delivering the shock, says, "200 J biphasic shock delivered."
2	Clear messages	The leader instead of just saying "Give shock" should say, "Give 200 J biphasic shock."
3	Clear roles and responsibilities	The leader assigns clearly the following roles to team members – Compression, Ventilation, Monitor & Defibrillation, IV Access and drugs, Code Recorder.
4	Knowing one's limitations	If a team member assigned for defibrillation does not know how to use the defibrillator, the team leader assigns the role to a member who is capable of using the defibrillator.
5	Knowledge sharing	Team leader tells team member to apply conductive gel properly and apply sufficient pressure with the paddles on the chest before delivering shocks
6	Constructive intervention	If a team member fails to synchronize the defibrillator for a patient requiring cardioversion, another team member intervenes and reminds about synchronization.
7	Reevaluation and summarizing	The patient continues to have persistent VF, and we have now given 3 shocks, one dose of Epinephrine and Amiodarone each.
8	Mutual respect	The team leader gives commands with respect to team members, without shouting or criticism.

Cardiac arrest – VF/VT (Algorithm)



After 1st shock – Ensure iv/io access

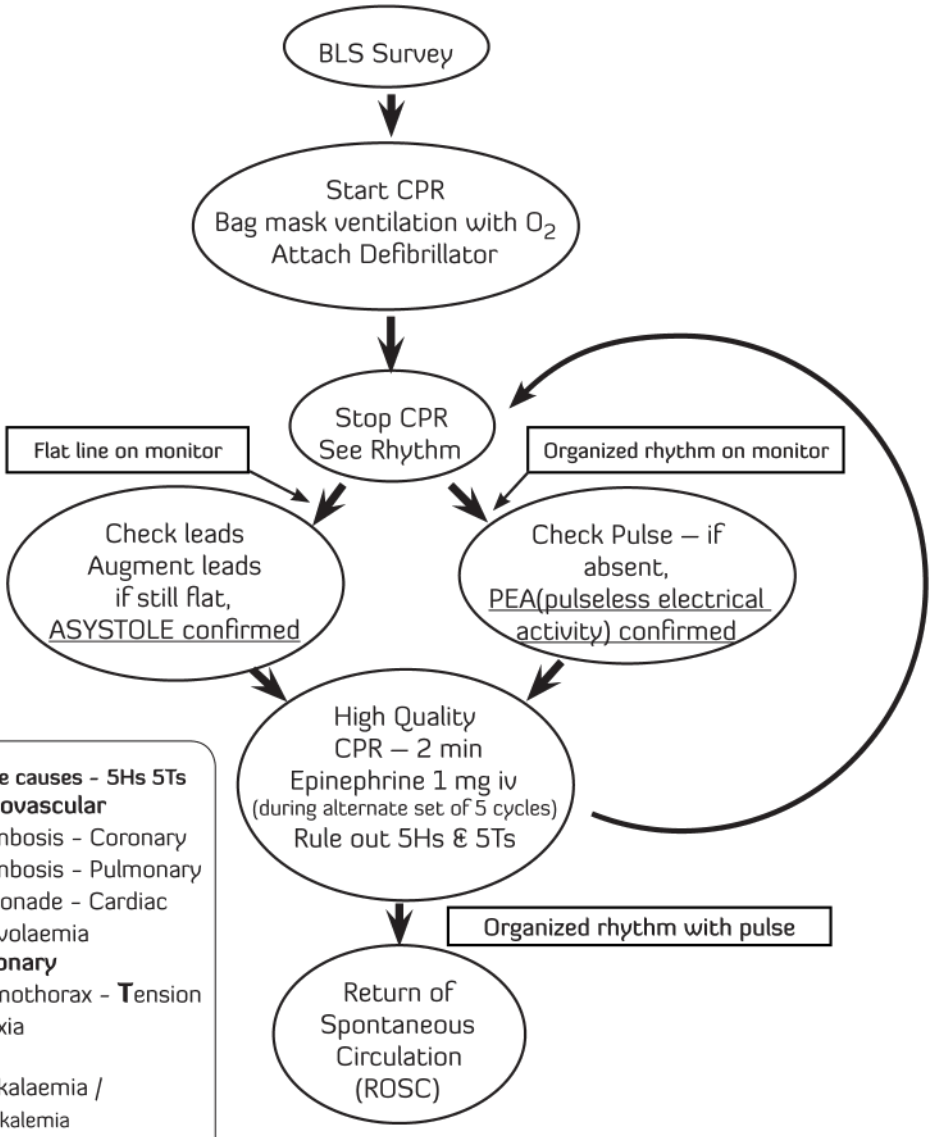
After 2nd shock – Epinephrine 1 mg iv bolus, consider advanced airway

After 3rd shock – Amiodarone 300 mg iv, Treat reversible causes - 5Hs 5Ts

After 4th shock – Repeat Epinephrine 1 mg iv after every even shock

After 5th shock – Amiodarone 150 mg iv, second dose

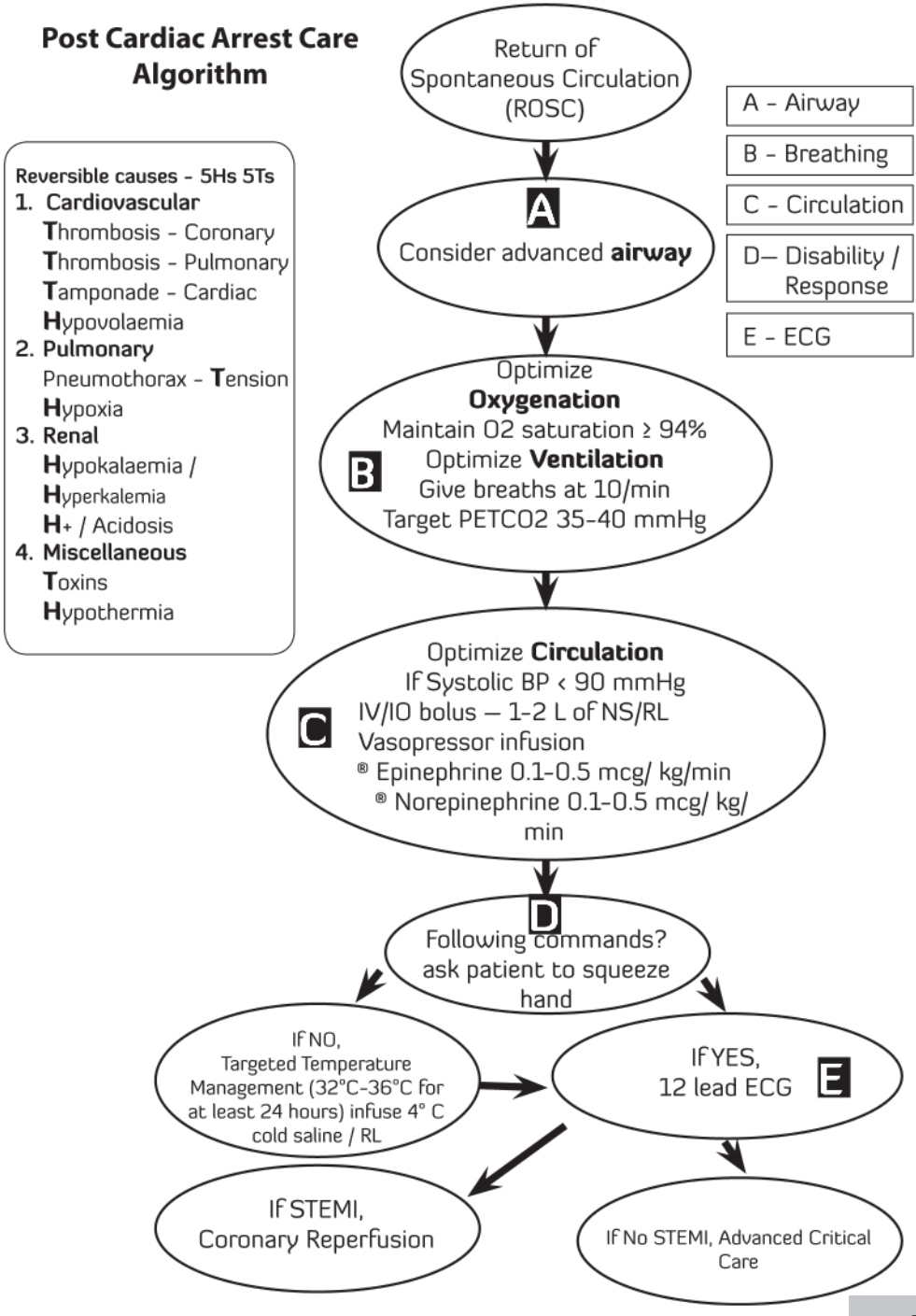
Cardiac Arrest – Asystole / PEA Algorithm



Reversible causes - 5Hs 5Ts

1. **Cardiovascular**
   - **T**hrombosis - Coronary
   - **T**hrombosis - Pulmonary
   - **T**amponade - Cardiac
   - **H**ypovolaemia
2. **Pulmonary**
   - Pneumothorax - **T**ension
   - **H**ypoxia
3. **Renal**
   - **H**ypokalaemia /
   - **H**yperkalemia
   - **H**+ / Acidosis
4. **Miscellaneous**
   - **T**oxins
   - **H**ypothermia

Post Cardiac Arrest Care Algorithm



Peri-arrest Management

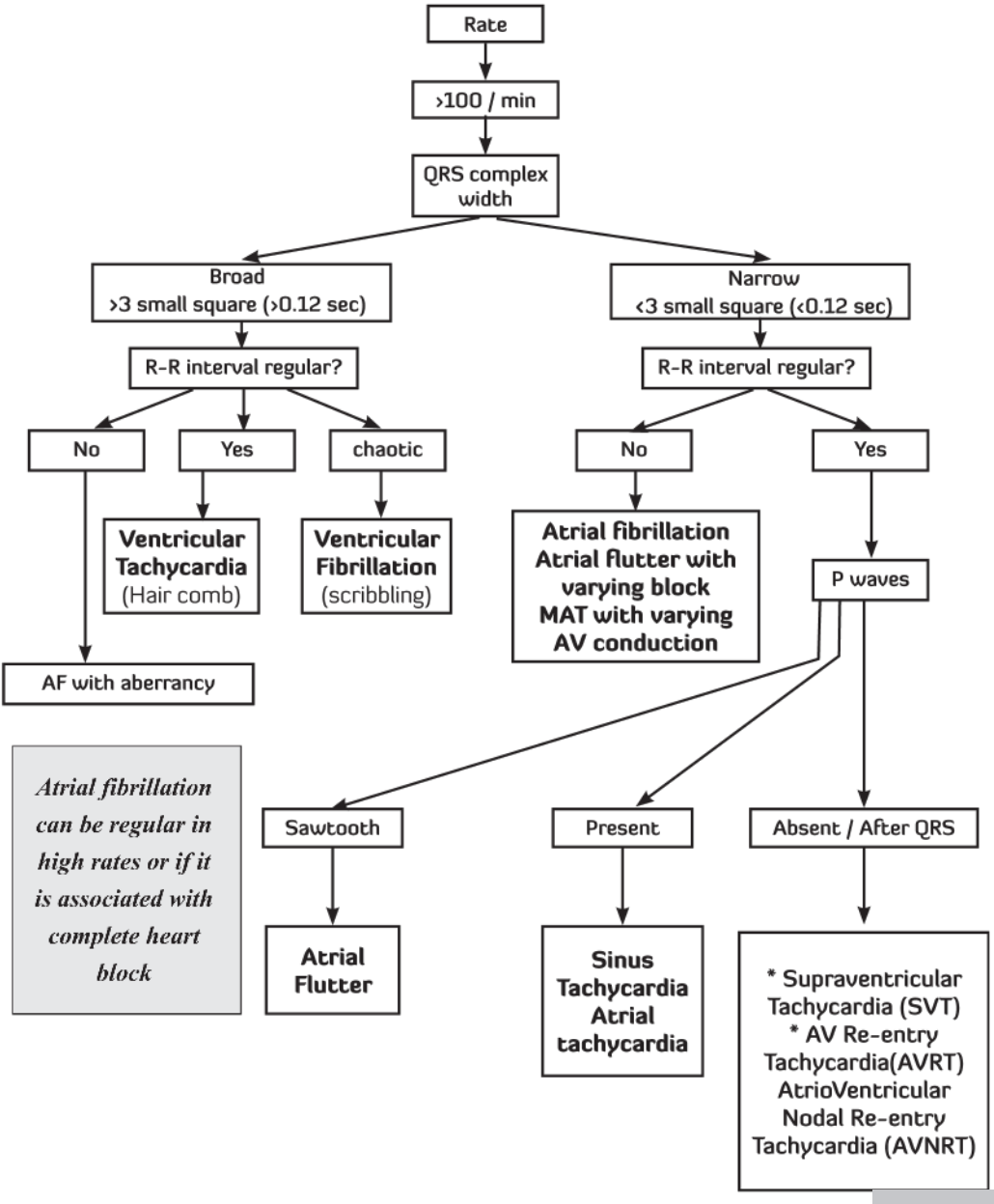
Responsive patients (AB VOMIT)

1. **Airway - Ensure it is patent**
2. **Breathing - Ensure rate & pattern is normal**
3. **Vitals**
4. **O_2**
5. **Monitor – See rate & rhythm**
6. **IV Access**
7. **Treat as below after considering heart rate,ECG rhythm & blood pressure.**

	Systolic BP > 90 mmHg	**Systolic BP < 90 mmHg**
Heart Rate <50/min	**Observe and monitor**	Atropine 0.5 mg iv, every 3-5 minutes, maximum 3 mg (cumulative) ↓ (If no response) Transcutaneous Pacing) ↓ (If not available or not effective) Dopamine infusion 2-10 mcg/kg/min (or) Epinephrine infusion 2-10 mcg/min
Heart Rate >150/min	**12 Lead ECG** **Regular, Narrow QRS** 1. Vagal manoeuvres 2. Adenosine 6 mg IV, repeat with 12 mg 3. Beta blockers / Calcium channel blockers 4. Expert consultation **Irregular, Narrow QRS** 1. Beta blockers / Calcium channel blockers 2. Expert consultation **Broad QRS** 1. Adenosine 6 mg IV, if regular and monomorphic (re-entry SVT) 2. Amiodarone 150 mg slow IV over 10 minutes 3. Expert consultation	**Synchronized cardioversion** • Sedate (IV Midazolam 1-2 mg) / Fentanyl 50-100mg • Synchronize defibrillator • Select energy (according to rhythm) (Initial recommended energy doses in biphasic current) • Atrial Flutter & SVT: 50-100 J • VT: 100 J • Atrial Fibrillation: 120 J

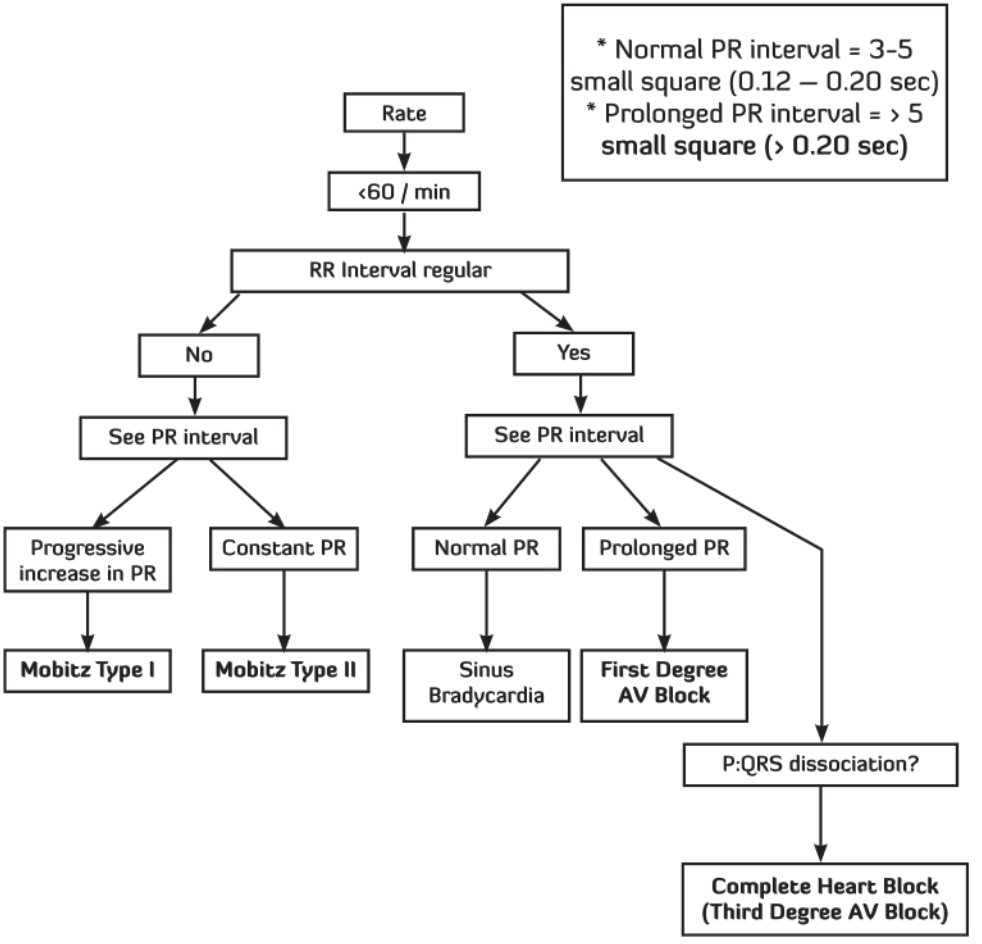
** Manage Peri-Arrest by correlating patients heart rate against blood pressur*

ECG algorithm (Tachyarrhythmias)



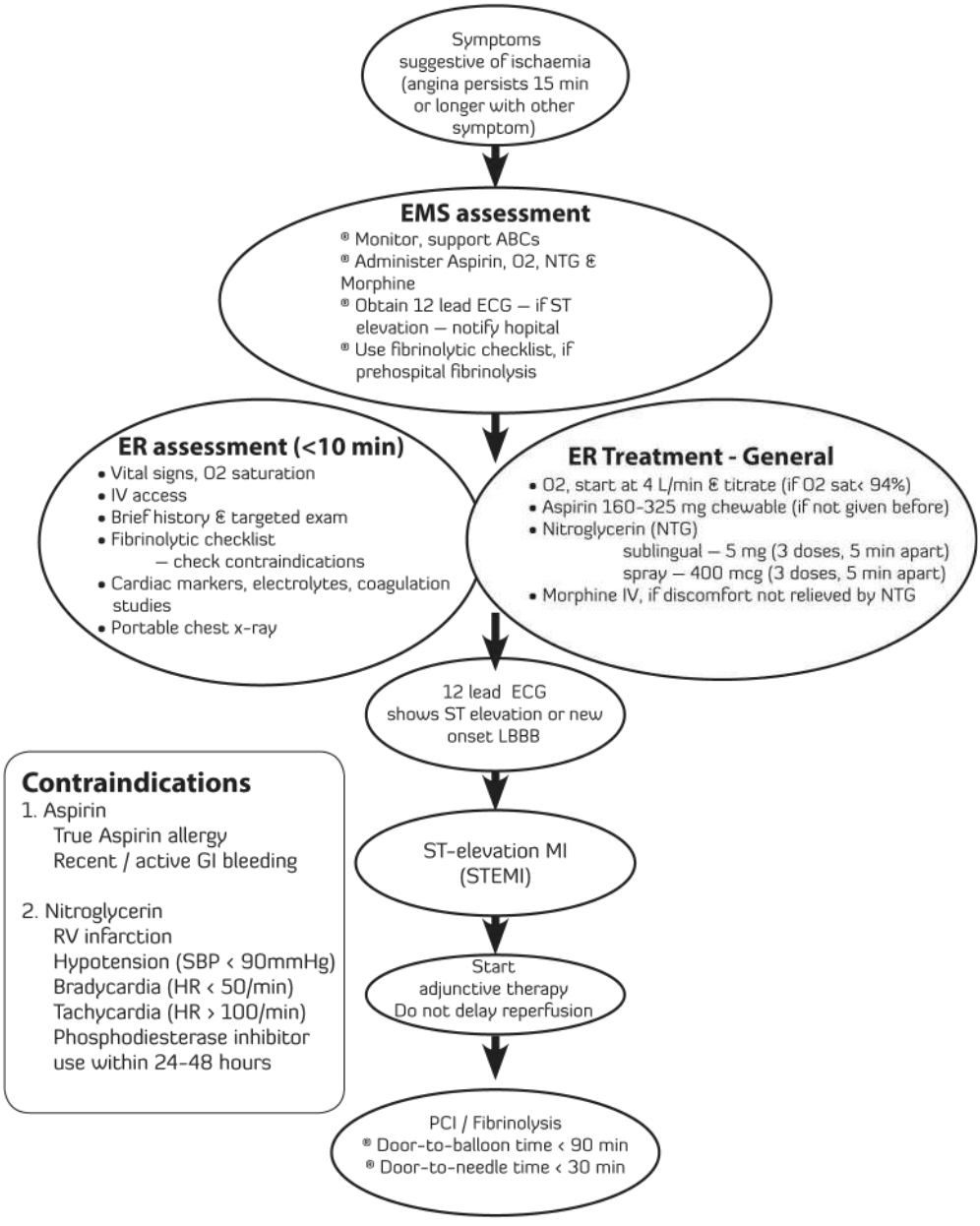
Atrial fibrillation can be regular in high rates or if it is associated with complete heart block

ECG algorithm (Bradyarrhythmias)



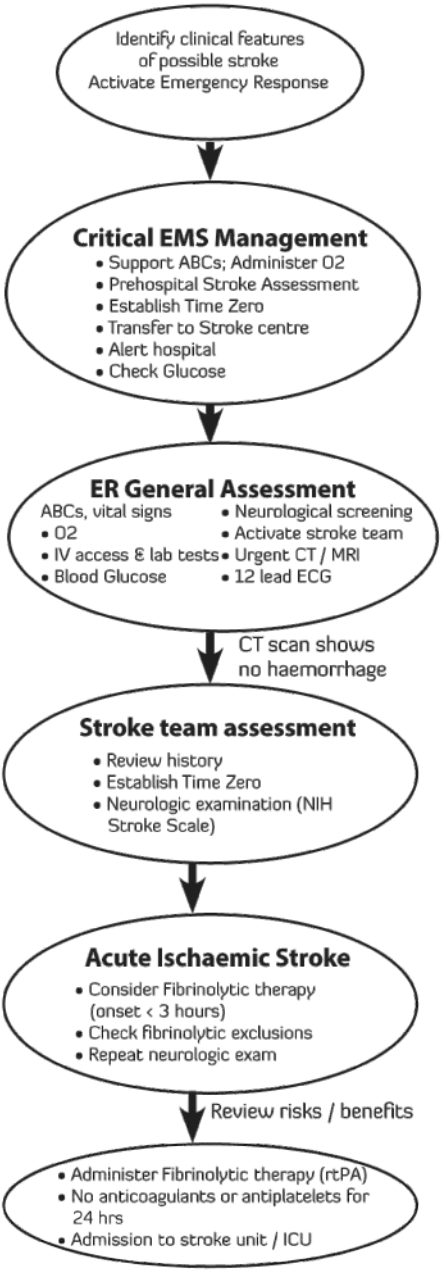
Bradycardia with retrograde or merged P after QRS is junctional

Acute Coronary Syndrome (ACS) Algorithm



*ABC –Airway, Breathing, Circulation
*ER – Emergency department
*EMS – Emergency Medical Services
*PCI- Percutaneous Coronary Intervention

Stroke Algorithm



Cincinnati Prehospital Stroke Scale

F-A-S-T

1. Facial droop
2. Arm Drift
3. Slurred Speech
4. Transport to Stroke Centre

Critical Time Periods

Action / Intervention	Perform within
General assessment	10 min
Neurologic assessment	25 min
CT Scan	25 min
Fibrinoloysis (from ER arrival)	60 min
Fibrinolysis (from Time Zero)	3 hours
Admission to stroke unit/ICU	3 hours

* NIH Stroke Scale – National Institute of HealthStroke Scale

Electrical therapies used in ACLS

1) Defibrillation (Unsynchronized shock)
2) Synchronized Cardioversion
3) Transcutaneous Pacing

Defibrillation

Indication: Cardiac arrest patient with VF / Pulseless VT on monitor VF/Pulseless VT on monitor

Energy used:

120-200 J Biphasic current (based on manufacturer recommendations; if not known, then use 200 J) 360 J Monophasic.

Steps:

1) Select energy
2) Apply conductive gel on paddle placement sites
3) Place paddles on the patient's chest - sternal paddle below the right clavicle, apex paddle on the left 5th intercostal space on the midaxillary line
4) Charge the defibrillator (pressing the charge button under the right thumb on the apex paddle)
5) Clear the patient by warning loudly - "Shocking - everybody stay clear"
6) Apply pressure (enough to cause mild indentation) on the chest and press shock buttons on both paddles simultaneously.
7) Resume chest compressions immediately (do not delay by checking for pulse or analyzing rhythm)

Transthoracic impedence:

For shock energy to be delivered maximally to the heart, transthoracic impedence has to be kept to a minimum. This can be done by

1) Using conductive gel - ensures maximum energy is delivered to the myocardium by decreasing impedence, ensures better contact between the paddles and chest wall.
2) Pressing the paddles and ensuring adequate contact with chest wall - until the indicator on the sternal paddle goes green (red and yellow indicate insufficient contact)
3) Pressing the paddles until there is indentation of the chest wall

Defibrillator safety:

1) Avoid O2 flowing across the chest by avoiding giving ventilations during defibrillator use.
2) Charge the defibrillator only after the paddles are placed on the chest,
3) Avoid holding the defibrillator paddles in your hands for long
4) Warn loudly – "Shocking, everybody stay clear!" and checking visually before delivering shock
5) Put the paddles back on the defibrillator immediately after the shock is delivered
6) Do not hold both paddles on one hand

Synchronized cardioversion

Indications:

A patient with tachycardia and haemodynamically unstable (hypotension, and signs and symptoms of poor perfusion)

Initial energy doses recommended
Differs according to the rhythm on the monitor

1) Atrial flutter and Supraventricular tachycardia – 50 to 100 J Biphasic
2) Ventricular tachycardia – 100 J
3) Atrial fibrillation – 120 J and above If the rhythm does not convert, increase energy levels for subsequent shocks.

Steps:

1) Sedate the patient – IV Midazolam 1 to 2 mg / Fentanyl 50-100 mg
2) Select energy dose according to the rhythms mentioned above
3) Set the defibrillator to "Synchronization" mode – this is confirmed by "Sync" display on the monitor, and by dots or dashes identifying R waves
4) Apply gel
5) Place paddles
6) Charge the defibrillator
7) Clear the patient
8) Shock – since the shock is delivered only at the next synchronization (with R wave), you may need to hold the paddles a little longer to confirm that shock has been delivered

Transcutaneous pacing

Indications - A patient with bradycardia and showing symptoms and signs of poor perfusion (including hypotension)

Steps:

1) Sedate the patient
2) Apply adhesive pacing pads on the patient's chest
3) Set the defibrillator to pacing mode
4) Select the desired pacing heart rate (80/min or above)
5) Increase the pacing current by 5 mAmp at a time while watching the monitor for the pacing impulses to appear (pacing current spike followed by broad QRS complex at the set heart rate) - electrical capture
6) After the impulses appear, increase the current by another 5-10 mAmp as a safetly margin above the threshold
   Check the vitals - pulse rate should match the set pacing rate (mechanical capture), BP should recover

	SYNCHRONIZED CARDIOVERSION	DEFIBRILLATION
DEFINITION	"CARDIOVERSION" is the application of electricity to terminate a *still perfusing rhythm* (e.g., ventricular tachycardia with a pulse, supraventricular tachycardias including atrial arrhythmias) to restore the normal Sinus Rhthym	"DEFIBRILLATION" is the application of electricity to terminate a *non perfusing rhythm* (Pulseless ventricular tachycardia, Ventricular Fibrillation) to restore the normal sinus Rhythm.
MECHANISM OF ACTION	By depolarising all excitable tissue of the circuit and making the tissue refractory, the circuit is no longer able to propagate or sustain re -entry.	By depolarising a critical mass of the heart muscle, terminates the arrhythmia, and allows normal sinus rhythm to be re-established by the body's natural pacemaker, in the sino- atrial node of the heart.
LEVEL OF CONSCIOUSNESS	Conscious	Unconscious
SYNCHRONICITY	Synchronous By pressing the "SYNC" soft key, the defibrillator will enter "SYNC" mode and the synchronising circuit within the defibrillator will detect the patient's R-waves. When the shock button is pressed and held, the unit discharges with the next detected R-wave, thus avoiding the vulnerable T-wave segment of the cardiac cycle.	Non synchronous The shock may fall randomly anywhere within the cardiac cycle (QRS complex). Unsynchronized cardioversion (defibrillation) is used when there is no coordinated intrinsic electrical activity in the heart (pulseless VT/VF) or the defibrillator fails to synchronize in an unstable patient.
ENERGY LEVEL	Starts at 25J – 120 J	Highest energy as per manufactured recommendation 120J-200J
INDICATIONS	Tacchyarrhythmia's causing hemodynamic compromise 1. Ventricular Tachycardia with pulse 2. Supraventricular Tachycardia 3. Atrial Flutter	1. Ventricular Fibrillation 2. Pulseless Ventricular Tachycardia

	4. Atrial Fibrillation 5. Atrial Tachycardia 6. Junctional Tachycardia	
NEED FOR PROCEDURAL SEDATION	Patient needs Sedation because patient is conscious.	Patient does not need sedation because patient is Unconscious.
PRACTICAL PROCEDURE	• Check the availability of full Resuscitation Equipments and Drugs • Secure IV access • Connect ECG Monitoring and Pulse Oximetry • Sedate with Midalzolam and Fentanyl • Pre-oxygenation • **ENSURE SYNCHRONISATION is ON** and marker on "R wave" • **Select Energy Level – 25J-120J** • Follow the steps as in page 39	• Check the availability of full Resuscitation Equipments and Drugs • Secure IV access • Connect ECG Monitoring and Pulse Oximetry • **ENSURE SYNCHRONISATION is OFF** • **Select Energy Level – High Joules** (Highest as per manufacturer recommendation) • Follow the steps as in page 38

ENERGY AND CURRENT FLOW

Adequate current flow through the heart is required for successful defibrillation. The current delivered to the myocardium with a given energy is dependent on the "**Transthoracic Impedance**", which can vary widely among patients. Thus, the same energy dose can potentially deliver varying current to a patient. Additionally, the percentage of current shunted through the thorax, away from the myocardium, influences the net current a patient receives.

MONOPHASIC DEFIBRILLATORS

Electrical current flows in a single direction from an electrode on one side of the patient's chest to a second electrode on the other side. The waveform associated with monophasic defibrillations contains a Single peak. The highest part of the waveform, the peak current, is a key determinant of successful defibrillation. There must be enough current to reach the heart to defibrillate (terminate the lethal rhythm), but not so much peak current that the heart is damaged.

BIPHASIC DEFIBRILLATORS

Electricity is sent from one electrode to the other in the first phase of this waveform, followed by a return back to the originating electrode in the second phase. Biphasic technology requires a much lower current to achieve successful termination of fibrillation. This may result in less damage to the myocardium and a reduced frequency of postshock contractility and dysrhythmias.

PADDLE / PADS SIZE

- Larger Size associated with higher success rates and less Myocardial Damage
- Paddles/pads of 10–13 cm optimally reduce transthoracic impedance

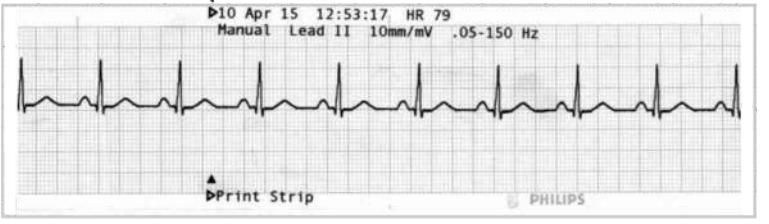
METHOD OF USE

There are two accepted positions to optimize current delivery to the heart:

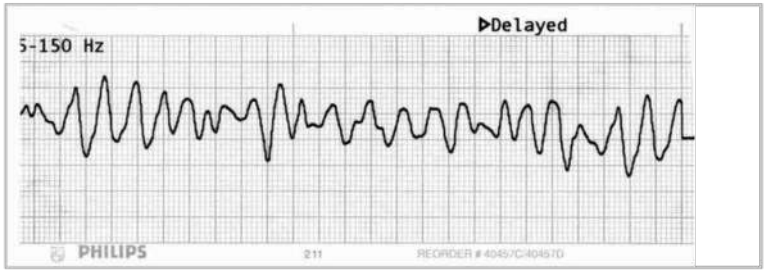
- **ANTEROAPICAL** – one pad/paddle is placed to the **right of the sternum just below the clavicle**, and the other is centred lateral to the normal cardiac apex in the anterior or midaxillary line (**V5–6**)
- **ANTEROPOSTERIOR** – the anterior pad/paddle is placed over the **precordium or apex**, and the posterior pad/paddle is placed on the **back in the left or right infrascapular region**.

ARRHYTHMIAS

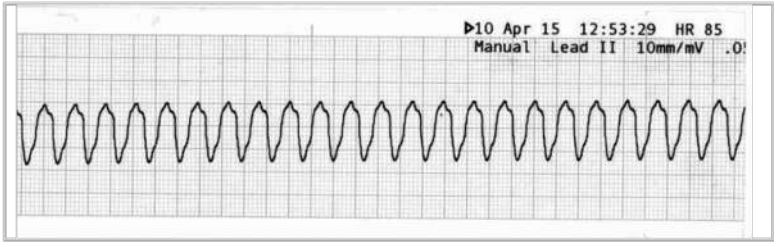
Normal Sinus Rhythm



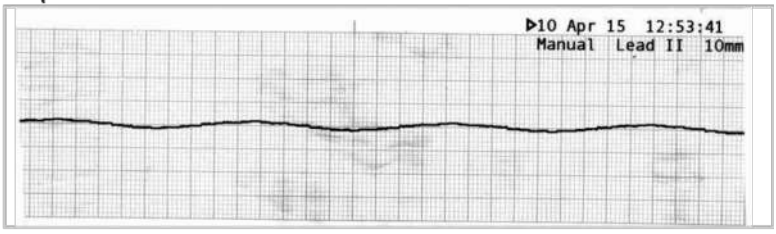
Ventricular Fibrillation



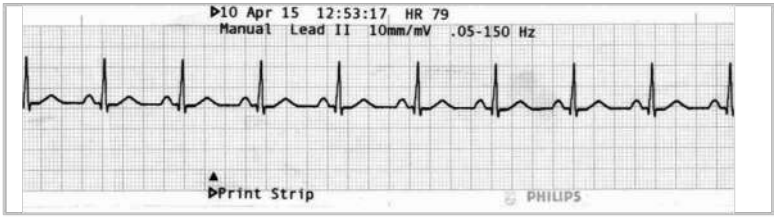
Ventricular Tachycardia (Monomorphic)



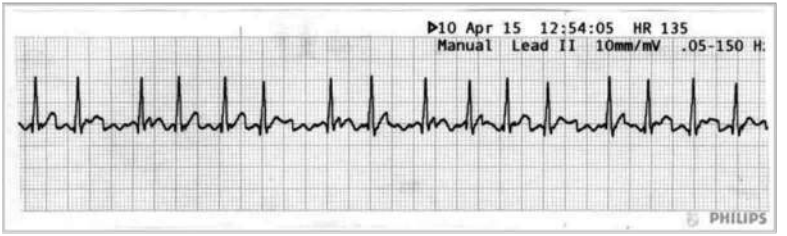
Asystole



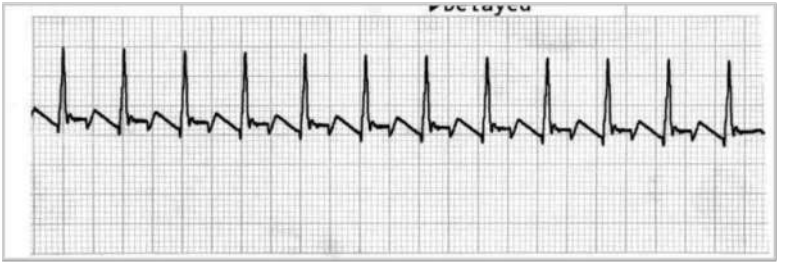
Pulseless electrical activity



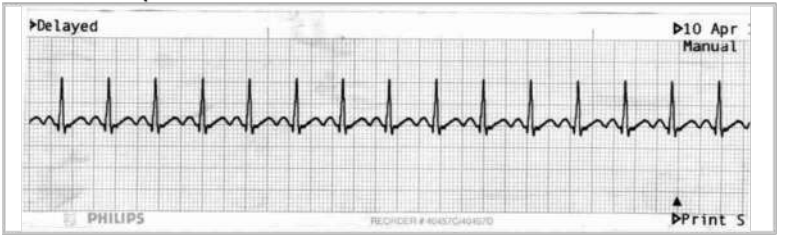
Atrial Fibrillation



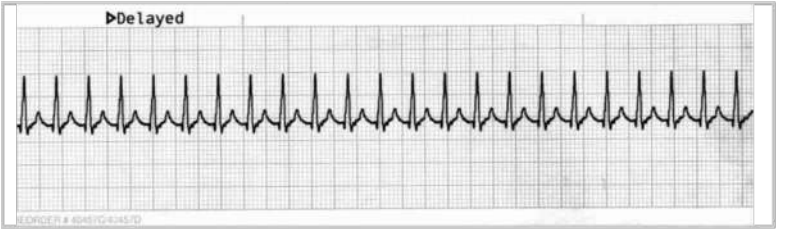
Atrial Flutter (with 2:1 AV block)



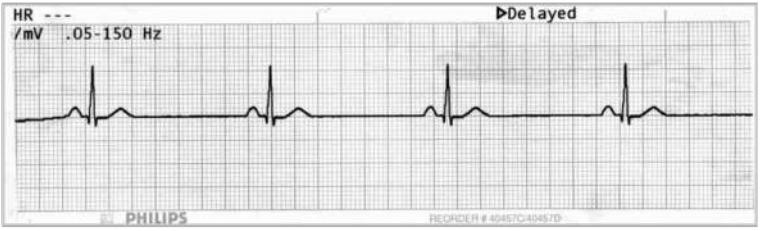
Sinus Tachycardia



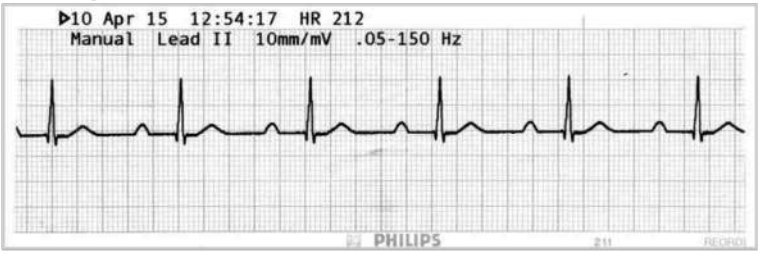
Supraventricular Tachycardia



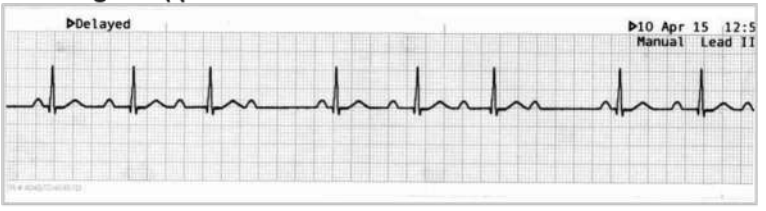
Sinus Bradycardia



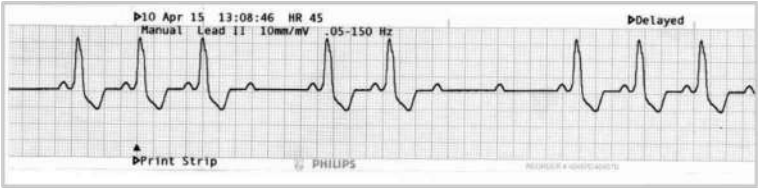
1st degree AV Block



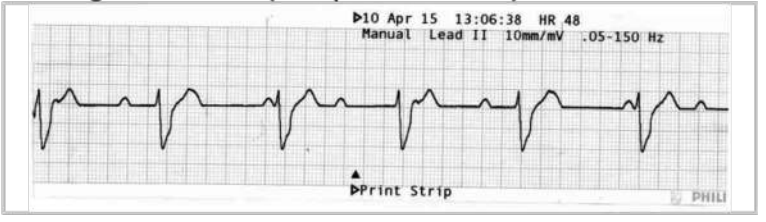
2nd degree Type 1 AV Block



2nd degree Type 2 AV Block



3rd degree AV Block (Complete AV Block)



Drugs

Anaesthesiology Postgraduates

Dr. Janani .N
Dr. Saranya .N

DRUG NAME	MOA and effects	ONSET and DURATION	DOSAGE & ROUTE OF ADMINISTRATION	INDICATIONS	CONTRAINDICATIONS	SIDE EFFECTS	PRECAUTIONS
Epinephrine (naturally occurring catecholamines)	• Beta & Alpha adrenergic agonist • $\alpha_1 = \alpha_2$; $\beta_1 = \beta_2$ • Myocardial stimulation (beta1) • Peripheral vasoconstriction • Bronchodilator (beta 2) • Mydriatric • Hyperglycemic	Onset: IV – 1 -2 min SC- 5-10min Duration – 5-10 min	**Adult:** 1 mg IV/IO every 3–5 minutes **Pediatric dose**: 0.01 mg/kg (0.1 mL/kg of 1:10,000 solution)IV/IO every 3 to 5 min **Anaphylaxis :** SC/IM :0.5mg IV :100mcg every 3 -5 mins followed by infusion. **Infusion dose:** Range – 0.03-3 mcg/kg/min **Nebulisation : (adult andpediatric)**0.5mg/kg of 1:1000 dose dilution(max 5mg) **Epidural test dose:** 1:200000(5 mcg/ml	• Cardiac arrest (asystole, pulseless electrical activity, ventricular fibrillation), shock states Anaphylaxis 99 • Test dose in epidurals and peripheral nerve blocks to rule out vascular placement • Co-administration with local anesthetics	Based on situation	• Ventricular arrhythmias • Cerebrovascular heamorrhage Myocardial ischemia.	Halothane (inhalational anaesthesia) – ventricular arrhythmias Limit- 0. 15mg

DRUG NAME	MOA and effects	ONSET and DURATION	DOSAGE & ROUTE OF ADMINISTRATION	INDICATIONS	CONTRAINDICATIONS	SIDE EFFECTS	PRECAUTIONS
Amiodarone	• Class III Antiarrhythmic drug. • Possesses characteristics of all 4 classes. • Inhibits sodium channels at rapid pacing • frequencies (Class I); • Suppresses AV node via sympatholytic activity (Class II); • Blocks potassium channels, which prolongs the cardiac action potential(Class III); Negative inotropy by blocking L-type calcium channels (Class IV).	Onset :IV - < 30min Duration : variable Elimination Half life of single dose – 56 days	**Cardiac arrest:** **Adult:** 300 mg initial dose; 150 mg second dose after 2nd shock. **Pediatric:** 5 mg/kg IV or IO; may repeat twice at same dose; maximum of 15 mg/kg **Refractory tachyarrythmias:** 150mg iv bolus over 10 mins followed by Infusion : 1mg/min for 6 hrs 0.5mg/min for 18hrs	• Ventricular tachycardia. • Ventricular fibrillation refractory to defibrillation; second-line after epinephrine. Atrial fibrillation /flutter and other supraventricular tachycardias.	• Prolonged QT interval. • Second degree AV block • Complete AV block. • Hepatotoxicity Porphyria	• Bradycardia Hypotension	

DRUG NAME	MOA and effects	ONSET and DURATION	DOSAGE & ROUTE OF ADMINISTRATION	INDICATIONS	CONTRAINDICATIONS	SIDE EFFECTS	PRECAUTIONS
Lidocaine	• Antiarrhytmic class 1b, weak sodium channel blocker Delays spontaneous phase 4 depolarisation.	Onset :IV– 45-90secs (bolus) Duration – 10 - 20 min (IV bolus) Context sensitivity time (after 3days infusion) – 20 – 40 min Elimination half life – 1.5 -2hrs	**Adult**: 1-1.5mg/kg initial dose IV **Repeat doses**: 0.5-0.75mg/kg IV with total maximum loading dose of 5mg/kg. **Pediatric Dose:** Give 1mg/kg IV bolus. **Local anaesthetic toxic dose:** 5mg/kg 7mg/kg (with adrenaline) **Suppress pressor response**: 1.5mg/kg (30-60 secs before intubation)	• Monomorphic VT • Refractory Ventricular fibrillation (VF) • Pulseless ventricular tachycardia (pVT) • Prevention of intubation response • Stress response	• Wolff-Parkinson-White Syndrome • II degree or III degree Heart Block • Adams Stokes Syndrome	Local anesthestic systemic toxicity	• Atrial arrhythmias Respiratory Depression

DRUG NAME	MOA and effects	ONSET and DURATION	DOSAGE & ROUTE OF ADMINISTRATION	INDICATIONS	CONTRAINDICATIONS	SIDE EFFECTS	PRECAUTIONS
Atropine	• Anticholinergic(muscarinic antagonist) • Blocks the neurotransmitte r acetylcholine in the central and peripheral nervous systems through competitive inhibition	Onset: IV – < 30 secs Duration : 30 min	**Adult :** **Bradycardia:** 0.5mg IV/IO. Repeat every 3 to 5 minutes with a maximum dose of 3mg. **Neuromuscular blockade reversal:** IV: 25–30 mcg/kg 30–60 seconds before neostigmine **vagolytic dose**: 2-3mg	• Symptomatic Bradycardia • II degree Heart block Mobitz type 1 / III degree heart block (in presence of AV Node escape rhytm) OPC Poisoning	• Acute MI • Tachycardia • Post Heart Transplant Glaucoma	• Central cholinergic syndrome. Reflex bradycardia when given in lower doses	• Use cautiously in myocardial ischemia because it increases myocardial oxygen demand. Not effective for Hypothermic Bradycardia

DRUG NAME	MOA and effects	ONSET and DURATION	DOSAGE & ROUTE OF ADMINISTRATION	INDICATIONS	CONTRAINDICATIONS	SIDE EFFECTS	PRECAUTIONS
Magnesium (M gSO4 1 gram = 4 mmol, 8 mEq, or 98 mg of elemental magnesium.)	• Non competitive NMDA antagonism, • Ca ++ antagonist and blunts the release of catecholamines Effects : • Potentiates Non depolarizing muscle relaxants • Tocolysis (calcium antagonism) • Preeclampsia Neuroprotective in cerebral palsy	Onset – IV immediate IM – 1hr Duration : IV – 30 min IM- 3 -4 hrs	**Adult :** **With a pulse**: 1-2g slow IV/IO infusion over 5-60 minutes, followed with a maintenance infusion of 0.5-1g/hr. (Magnesium should be diluted in 50-100ml of D_5W.) **Cardiac arrest**: 1-2gm slow IV/IO infusion over 5-20 minutes. (Magnesium should be diluted in 50-100ml of D_5W.) In instances of severe renal impairment do not give more than 20g in 48 hours. **Pediatric :** Give 25-50mg/kg IV/IO over 15-30 minutes with a max dose of 2g. (Magnesium should be diluted in 10mg/ml of D_5W.) Therapeutic range is a serum level of 4–7 mEq/L.	• Torsades de pointes during cardiac arrest • Atrial fibrillation • Hypomagnesemia • Digitalis Toxicity • Asthma attacks resilient to first line therapy • Preeclampsia • Obtund pressor response during intubation • Postoperative analgesia • Preterm labour • Shivering	• Hypermagnesemia • Hypocalcemia • Neuromuscular disorders such as myasthenia gravis or Eaton-Lambert syndrome	• Magnesium intoxication-Hypotension, CNS depression • Respiratory paralysis	• Monitor renal function, blood pressure, respiratory rate, and deep tendon reflex when magnesium sulfate is administered parenterally. • Attenuates Hypoxic Pulmonary Vasoconstriction • Crosses the placenta and cause neonatal hypotonia and neonatal depression in severe magnesemia.

DRUG NAME	MOA and effects	ONSET and DURATION	DOSAGE & ROUTE OF ADMINISTRATION	INDICATIONS	CONTRAINDICATIONS	SIDE EFFECTS	PRECAUTIONS
Adenosine	• Prolongs AV node conduction, by inhibiting L-type calcium channels in cardiac tissue, nodes. • Antagonizes cAMP mediated norepinephrine stimulation of ventricular muscle. Pharmacological stress during nuclear stress tests	Onset: IV – 20-30 Secs Elimination half life is 10min	**Adults : 6mg-12mg-12mg** 1st dose:6mg IV/IO over 1-3 seconds, immediately followed by 20ml of NS by rapid IV/IO. 2nd dose: If the patient still has an SVT rhythm 1-2 minutes later give 12mg IV/IO over 1-3 seconds, immediately followed by 20ml of NS by rapid IVP/IO. Followed by an additional 12 mg IV fast bolus 1–2 minutes **Stress test:** 0.14 mg/kg/min for 5–6 minutes.	• Paroxysmal SVT Stable narrow complex SVT/ Monomorphic VT	• Polymorphic wide complex Tachycardia • Unstable VT II or III degree Heart block	• Brief period of Asystole or Bradycardia, • Ventricular ectopy • Transient AV block Flushing , headache , dyspnea , bronchospasm	• Bronchial Asthma • Lower dose of 3mg for patients receiving carbamazepine,dipyridamole • CNS - Low-dose adenosine induces neuropathic pain, hyperalgesia, and ischaemic pain.

DRUG NAME	MOA and effects	ONSET and DURATION	DOSAGE & ROUTE OF ADMINISTRATION	INDICATIONS	CONTRAINDICATIONS	SIDE EFFECTS	PRECAUTIONS
Dopamine (Endogenous catecholamine)	• Beta adrenergic and dopaminergic agent • **D_1 = D_2>>β>>α** • Positive chronotropic and inotropic effects on myocardium • D1 – diuresis (<5 mcg/kg/min) • Alpha and beta – 5-10mcg/kg/min Alpha - >15mcg/kg/min	IV – 2-4 mins Duration -10 min Elimination half life – 2min	**Infusion dose**: 5 to 20µg/kg/min IV/IO	• Second line drug for Symptomatic Bradycardia (after atropine) Severe Hypotension	NIL	• Sinus tachycardia • Arrythmias • Euthyroid sick syndrome(inhibition of thyrotropin releasing hormone)	• Correct Hypovolemia before initiating Dopamine • Caution in cardiogenic shock with CHF • Inactivated by alkaline solutions • Patients on MAOIs

DRUG NAME	MOA and effects	ONSET and DURATION	DOSAGE & ROUTE OF ADMINISTRATION	INDICATIONS	CONTRAINDICATIONS	SIDE EFFECTS	PRECAUTIONS
Nor adrenaline (Endogenous catecholamine)	• $\alpha_1 = \alpha_2$; $\beta_1 >> \beta 2$ • Arterial and venous vasoconstriction	Onset :1-2 min Duration : Elimination half life - 2 min	**Infusion dose :** 0.01–3 mcg/kg/min	• Cardiogenic , anaphylactic, and septic shock	NIL	• Bradycardia • Tissue Hypoxia &Ischaemic injury	Extravasation through peripheral veins.
Dobutamine Synthetic catecholamine)	• Racemic mixture(50% (+) and 50% (-) . • $\beta_1 > \beta_2 >>>> \alpha$ • (–) isomer is $\alpha 1 >> \beta 1$ and $\beta 2$ • (+) is competitive antagonist at $\alpha 1$, potent $\beta 1$ and $\beta 2$ agonist Positive inotropic agent	Onset :IV – 1-2 min Peak – 10 min Duration : Elimination half life - 2 min	**Infusion dose :** 5-15mcg/kg/min	• Resting cardiac stress test Heart failure		• Tachyarrythmias • Prolonged infusion - Eosinophilic myocarditis • Peripheral eosinophilia	Extravasation through peripheral veins.

DRUG NAME	MOA and effects	ONSET and DURATION	DOSAGE & ROUTE OF ADMINISTRATION	INDICATIONS	CONTRAINDICATIONS	SIDE EFFECTS	PRECAUTIONS
Midazolam	• Facilititates GABA - increased frequency of chloride channel opening, hyperpolarization. Imidazole ring is open in acidic solutions , ionized and hydrophilic. In physiologic pH, ring is closed , nonionised and lipophilic	Onset IV – immediate (peak effect – 3-5min) IM – 3-5min Oral -5-15 min Nasal – 3-5 min Rectal – 5-10 min Duration : Elimination half life - IV - 3hrs IM – 4.2hrs	0.05- 0.15mg/kg IV 0.1-2 mg/kg IM 0.25-0.75 mg/kg oral 0.1-0.2 mg/kg nasal 0.75 -1 mg/kg rectal	• Sedation for cardioversion • Premedication - Anxiolysis and amnesia; • Emergence delirium; • Withdrawal of abuse drugs • Anticonvulsant • PONV • Antipruritic	Situational	Respiratory depression	• Ceiling effect • Cytochrome P450 inhibition prolongs the duration Pregnancy – category D – floppy baby syndrome

DRUG NAME	MOA and effects	ONSET and DURATION	DOSAGE & ROUTE OF ADMINISTRATION	INDICATIONS	CONTRAINDICATIONS	SIDE EFFECTS	PRECAUTIONS
Fentanyl	• Lipophilic Opioid Agonist. binds mu opioid receptor, , G protein coupling regulates adenylatecyclase, reducing concentrations of cAMP.	Onset IV – 2-5- min IM – 10- 15 min Duration : 1-2 Hrs Context sensitivity half time (4hr infusion) – 260 min Elimination half life:7- 12 hrs	**IV induction dose** :1.5- 3 mcg/kg **Epidural** 50–100 mcg; **Spinal** 10–25 mcg **Infusion dose**: 1-2 mcg /kg/hr infusion 75-150mcg per hr. PCA – 10mcg IV with 5-10mins lockout time with basal rate< 50mcg/hr with demand dose of 20mcg. Maximum dose in 4 hrs 300mcg	• Intra and post operative analgesia • Adjunct in regional anesthesia • Sedation		• Respiratory depression • Chest wall rigidity (the 'wooden chest' phenomenon)	
Morphine	• Agonist at mu and kappa opioid receptor. • Hydrophilic opioid	**Onset** : IV IM Duration :4 – 5 hrs (iv) Context sensitivity half life: Elimination half life: 2-3 hrs	IV – 0.05 – 0.1 mg/kg IM – 0.1-0.2mg/kg Sublingual – 0.2 – 0.4mg Q8H	• Premedication • Analgesic • Left ventricular failure • Pulmonary edema	• hepatic failure	• nausea vomiting , constipation • histamine release - pruritis • delayed respiratory depression • miosis • urinary retention	• Hypopitutarism • Hypothyroidism • Bronchial asthma

DRUG NAME	MOA and effects	ONSET and DURATION	DOSAGE & ROUTE OF ADMINISTRATION	INDICATIONS	CONTRAINDICATIONS	SIDE EFFECTS	PRECAUTIONS
Diltiazem	• Nondihydropyridine Calcium channel blockers(class IV Antiarrythmic) • Slows AV node conduction and increase AV Node refractoriness • Potent peripheral and coronary vasodilator • negative inotrope	Onset :IV – 3min Duration : 1-10 hr Elimination half life:3-9hrs	**For paroxysmal svt , atrial flutter, atrial fibrillation:** Initial Dose :15- 20 mg (0.25mg/kg) IV for 2 minutes Additional dose : In 15 minutes 20-25mg iv over 2 mins Maintenance dose :5-10mg/hr IV infusion not more than 15 mg/hrupto 24hrs	• Refractory reentry SVT in patients with narrow QRS complex & adequate BP • Atrial flutter • A .fibrillation • Angina pectoris • Hypertension • Migraine prophylaxis	• Hypotension (systolic BP < 90mmhg) • WPW syndrome • Sick sinus syndrome except in patients with functioning ventricular pacemaker. • AV Block without pacemaker • Prinzmetal's angina • COPD • CHF	• Headache • Hypotension • Dizziness • Bradycardia	• Patients receiving oral Beta Blockers

DRUG NAME	MOA and effects	ONSET and DURATION	DOSAGE & ROUTE OF ADMINISTRATION	INDICATIONS	CONTRAINDICATIONS	SIDE EFFECTS	PRECAUTIONS
Verapamil	• Nondihydropyridine L - TYPE Calcium channel blockers(class IV Antiarrythmic agent) • Suppresses SA node and ventricular automaticity • prolongation of AV nodal ERP	Onset IV – 1-5min Duration : 1-6 hr Elimination half life : 6-12 hrs	Initial Dose 2.5-5 mg; IV for 2 min: Repeat as 5 -10 mg every 15- 30 mins / total dose of 20 to 30 mg	• Control ventricular rate in Atrial fibrillation or atrial flutter • PSVT • Hypertension • Angina	• Broad QRS complex • WPW • Ventricular tachycardia • Post MI • Partial Heart block • Sick Sinus Syndrome	• Symptomatic Hypotension • Sinus Bradycardia , • Cardiac arrest • A-V block • Non obstructive Paralytic Ileus	
Digoxin	• Cardiac Glycoside with positive inotropic effects by intracellular calcium accumulation. • Binds to Na+ /K + ATPase channel in cardiac myocytes,decreasing its function. • slows Av node conduction	Onset IV– 5-30min Slow onset Peak effect – 1.5 – 4hrs Duration : 2-4 days Elimination half life: 36 hrs	Total Loading Dose: 8 – 12 mcg/kg; half should be administered initially over 5 mins; remaining portion as 25% fractions at 4-8 hr intervals.	• Ventricular rate control in Atrial fibrillation & Atrial flutter • Alternative drug for Reentry SVT. • Mild to moderate CHF	• Hypokalemia • Renal & Hepatic Disease • Thyrotoxicosis • VT • WPW Syndrome	• Pulsusbigemini • Ventricular Extrasystoles • VT, VF • Cardiac toxicity • AV Block • Nausea • Vomiting	• With amiodarone; decrease the digoxin dose to 50% • Avoid electrical cardioversion unless it is life threatening , give low energy shock (10 -20 J)

DRUG NAME	MOA and effects	ONSET and DURATION	DOSAGE & ROUTE OF ADMINISTRATION	INDICATIONS	CONTRAINDICATIONS	SIDE EFFECTS	PRECAUTIONS
Metoprolol **Esmolol** **Propranalol** **Labetolol**	• Metoprolol and esmolol -Selective Beta 1 receptor blocker • Propranalol – beta 1 and beta 2 • Labetolol – Beta Blocker with Alpha blocking activity	Onset:Metoprolol-immediate (iv) Elimination half life -: 3-4 hrs Esmolol- 90 seconds (rapid onset) Elimination half life – 2 min Propranalol – 2-10 min(iv) Duration : 5min	**Metoprolol-** 1-15mg iv over 5mins **Esmolol**– 50 – 300mcg/kg/min **Propranolol** – 0.5-1 mg to max 3 mg **Labetolol – 10 to 20 mg IV** over 2mins (double the dose maximum – **80mg/dose)** **Total maximum dose – 300 mg**	• Angina • Hypertension • To control heart rate	• Heart block • Sick sinus syndrome • Bradycardia • Cardiogenic Shock • Bronchial Asthma	• dizziness, tired feeling • depression • Confusion • memory problems • nightmares • trouble sleeping • diarrhea	

DRUG NAME	MOA and effects	ONSET and DURATION	DOSAGE & ROUTE OF ADMINISTRATION	INDICATIONS	CONTRAINDICATIONS	SIDE EFFECTS	PRECAUTIONS
Calcium	• Calcium gluconate (10%) 1gm containing 4.65 mEq has elemental calcium of 93mg • Calcium Chloride 1g containing 13.6 mEq has elemental calcium of 273 mg	Onset: IV – immediate Duration : 30 min – 2 hr for 1gm.	**CARDIAC ARREST due to hyperkalemia:** 1- 3 gm iv over 2-5 mins **HYPOCALCEMIA:** **Mild** (ionized calcium: 4 to 5 mg/dL [1 to 1.2 mmol/L]): 1 to 2 g over 2 hours; asymptomatic patients may be given oral calcium **Moderate to severe** (without seizure or tetany; ionized calcium: <4 mg/dL [<1 mmol/L]): 4 g over 4 hours **Severe symptomatic** (eg, seizure, tetany): 1 to 2 g over 10 minutes; repeat every 60 minutes until symptoms resolves	• Cardiac arrest due to hyperkalemia. • Hyperkalemia treatment • Hypotension treatment • Magnesium toxicity in preeclampsia or eclampsia	• Hypercalcemia • Ceftriaxone	• Bradycardia • Hypotension • Constipation • Extravasation necrosis • Hyperphosphatemia • Hypokalemia • Hypomagnesemia	

DRUG NAME	MOA and effects	ONSET and DURATION	DOSAGE & ROUTE OF ADMINISTRATION	INDICATIONS	CONTRAINDICATIONS	SIDE EFFECTS	PRECAUTIONS
Sodium bicarbonate (8.4% = 84mg/ml)	1ml =1 mEq 1g of NaHCO3 provides ~12 mEq each of sodium and bicarbonate ions	Onset IV – 15 min	**Cardiac arrest due to metabolic acidosis** : IV: Initial: 1 mEq/kg/dose; repeat doses should be guided by arterial blood gases HCO3-(mmol) = 0.3 x weight (kg) x base deficit(mmol/l) Administer 1/2 dose initially over 30 mins to 1 hr, then remaining 1/2 dose over the next 24 hours; monitor pH, serum HCO3-, and clinical status	• Cardiac arrest • Severe Metabolic acidosis (PH < 7.15 and HCO3 < 10 mEq) • Hyperkalemia • Renal tubular acidosis • Overdose of TCA.	• Gastrointestinal loss (severe vomiting) and patients on diuretics - risk of hypochloremic alkalosis	• Hypernatremia • Hyperosmolarity • severe pulmonary edema • hypocalcemia • gastric distension • intracranial acidosis • rebound alkalosis	• rapid IV injection may cause intracranial bleed • elderly • CHF • Cirrhosis • Edema • Heart failure • Peptic ulcer disease • Renal impairment

DRUG NAME	MOA and effects	ONSET and DURATION	DOSAGE & ROUTE OF ADMINISTRATION	INDICATIONS	CONTRAINDICATIONS	SIDE EFFECTS	PRECAUTIONS
Potassium chloride	• Availability - 150mg/ml • 10ml – 20 mEq of K+ and Cl • 10 mEq of potassium chloride increases serum potassium by 0.1mEq/L	Variable	**Serum potassium >2.5 to 3.5 mEq/L:** 10 mEq/hour; (peripheral line) maximum 24-hour dose:200 mEq **Serum potassium <2.5 mEq/L or symptomatic hypokalemia:** (**central line only**): 40 mEq/hour; up to 400 mEq/24 hours.	• Hypokalemia	• Hyperkalemia • Ckd on renal replacement therapy • hypersensitivity	• Asystole • Hyperkalemia • Abdominal pain • Abdominal distress • Dyspnea	• Cardiovascular disease • Hepatic impairment • Renal impairment • Thrombophlebitis