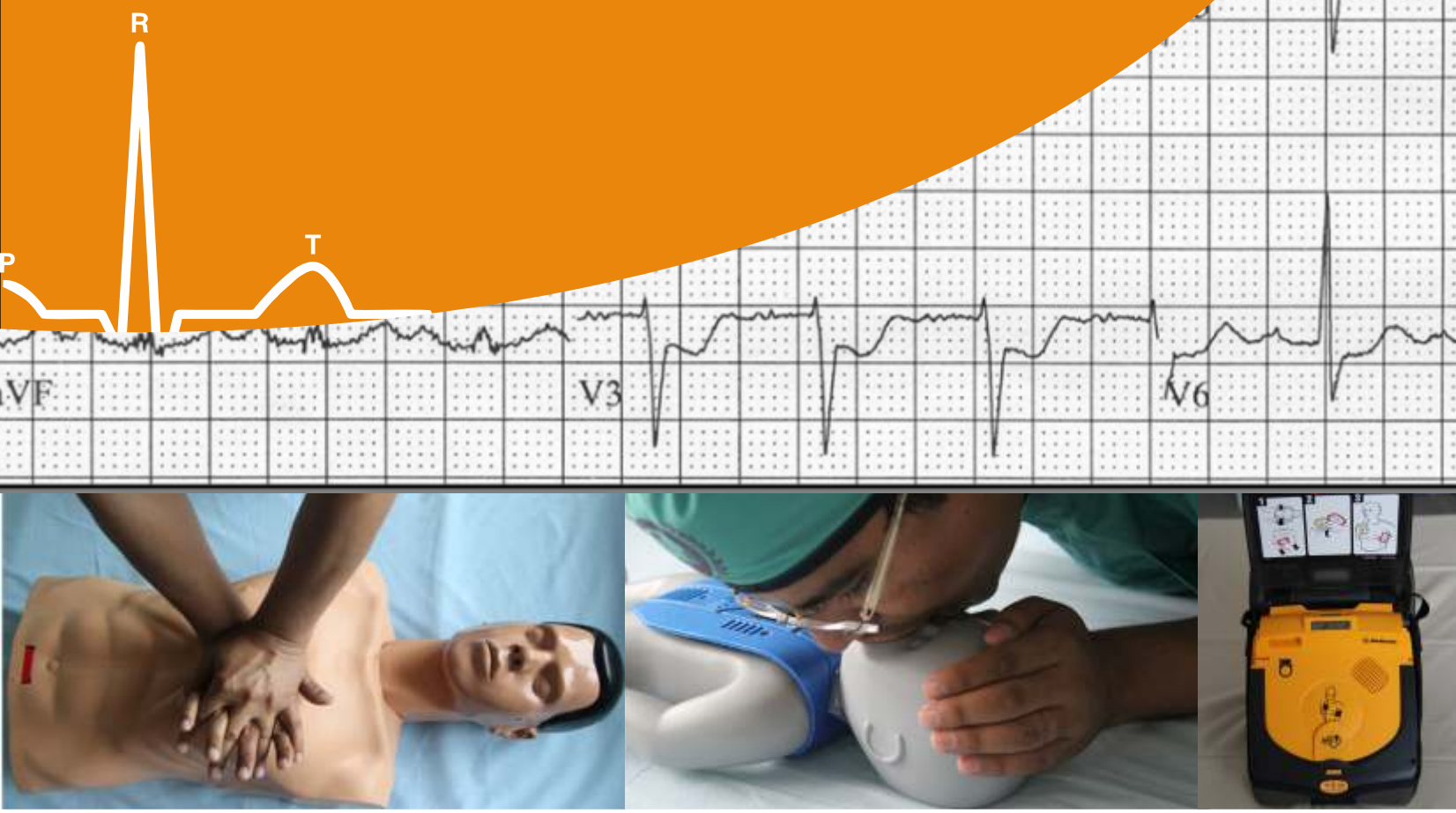
# MULTIDISCIPLINARY TRAINING IN
# EMERGENCY and TRAUMA (MTEC)
## Basic Emergency Care










Developed by
**All India Institute of Medical Sciences Bhopal**

with assistance from
**Department of Public Health and Family Welfare**
**Government of Madhya Pradesh**

# Multidisciplinary training in Emergency and Trauma (MTEC)

## AIIMS Bhopal
## Certification in
## Basic Emergency care



**Developed by**
**All India Institute of Medical Sciences Bhopal**
**with assistance from**
**Department of Public Health and Family Welfare**
**Government of Madhya Pradesh**

# AIIMS Bhopal Certification in Basic Emergency Care

**A certificate course**

**developed and conducted by**

**All India Institute of Medical Sciences Bhopal**

**2014**

Patron: Prof Sandeep Kumar, Director AIIMS Bhopal

Chairperson: Prof Vijaysheel Gautam, AIIMS Patna

Course Director: Dr JP Sharma

Writing team:


**Dr Saurabh Saigal**
Department of Trauma and Emergency Medicine
AIIMS Bhopal

**Dr Bhavna Dhingra Bhan**
Department of Pediatrics
AIIMS Bhopal

**Dr Nirendra Rai**
Department of Neurology
AIIMS Bhopal

**Dr Vaibhav Ingle**
Department of General Medicine
AIIMS Bhopal

**Dr Jai Prakash Sharma**
Department of Anesthesiology
AIIMS Bhopal

**Dr Rajnish Joshi**
Department of General Medicine
AIIMS Bhopal

**Dr Girish Bhatt**
Department of Pediatrics
AIIMS Bhopal

**Dr Sagar Khadange**
Department of General Medicine
AIIMS Bhopal

## TABLE OF CONTENTS

CHAPTER 1

# PRIMARY LIFE SUPPORT

## Introduction :

Being a health care provider you are expected to take remedial action when you encounter a emergency situation in Hospital or out of hospital on a road side or in your own house. You will be able to do justice to your patient or family member only when you know the life saving knowledge and skills. So there are few set of skills with clinical knowledge which one should imply in practice what one requires in emergency scenario like Cardiac arrest or choking or respiratory arrest. Therefore, this a module based course for this called as "Primary Life support"(BLS) in this course you are taught how to deal with these life threatening emergency.

Despite important advances in prevention, cardiac arrest remains a substantial public health problem and the leading cause of death. So at the end of this course you should be able to know how to response if you encounter a cardiac arrest in hospital or out of hospital.

## Objectives of this course :

This course will target all health care providers of every level ( paramedical to the super-specialist) who need to know to perform Cardio-respiratory Resuscitation (CPR) in a wide variety in any of the life threatening condition.

- Initiating the Chain of Survival
- Performing prompt, high quality, chest compression for adult, child and infant patients
- Using Automated External Defibrillator (AED) as early as possible.
- Imparting appropriate rescue breaths
- Practicing 2 rescue team CPR
- Relieving Choking

**Critical points :** High quality CPR improve's chances of survival. The most important features of **high quality CPR are:**

- Start chest compressions within 10 seconds of recognition of cardiac arrest.
- Push hard, push fast: Compress at a rate of at least 100/min with a depth of at least 5 cm ( 2 inches) for adults, approximately 5 cm for children and approximately 4 cm (1.5 inches) for infants.
- Allow complete chest recoil after each compression.
- Minimize interruptions in compressions ( try to limit interruptions to < 10 seconds)
- Give effect breaths that make the chest rise.
- Avoid excessive ventilation

## The Chain of Survival :

The adult chain of survival is described below in Figure 1.

Figure 1. Adult Chain of Survival

## Importance of the Adult Chain of Survival:

The term Chain of Survival provides a useful metaphor for the elements of the emergency cardiovascular care (EEC) system concept. The 5 links in the adult chain of survival are:

- Prompt recognition of cardiac arrest and activation of the emergency response system
- Early CPR with an emphasis on chest compressions

- Rapid defibrillation
- Effective advance life support
- Integrated post cardiac arrest care.

Although basic life support is taught as a sequence of distinct steps to enhance skills retention and clarify priorities, several actions should be accomplished simultaneously ( begin CPR and activate the emergency response system ) when multiple rescuers are present.

**Importance of Pediatric Chain of Survival:**

Because cardiac arrest in children and infants is more commonly due to respiratory failure not because of cardiac cause . So identifying children with these problems is essential to reduce the likelihood of pediatric cardiac arrest and maximize survival and recovery. Therefore, a prevention link is added in the pediatric Chain of Survival. ( Figure 2)

Figure 2 Pediatric Chain of Survival

- Prevention of arrest
- Early high quality bystander CPR
- Rapid activation of the EMS ( or Other emergency response ) system
- Effective advanced life support ( Including rapid stabilization and transport to definitive care and rehabilitation.)
- Integrated Post-cardiac arrest care.

**Learning Objectives:**

After reading this section of this manual you will be able to tell the sequence of CPR according to 2010 AHA guideline.

**Sequence of CPR :**

The 2010 AHA guideline for CPR makes a slight change in the sequence of A-B-C to C-A-B ( chest compression, Airway, and Breathing) for adults, children and infants.

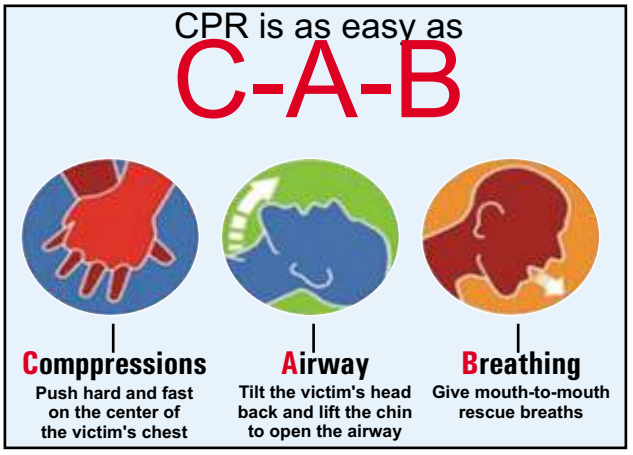



Figure 3. Sequence of CPR

Cause of change in sequence is because earlier lots of time used to waste in opening the airway, providing breathing by mouth to mouth breaths, retrieved a barrier device, or gathered and assembled ventilation equipment. Initiation of chest compressions were used to be delayed . So by changing the Sequence from A-B-C to C-A-B, the high quality chest compression can be initiated at an earlier stage of cardiac arrest.

The first cycle of 30 compressions or approximately 18 seconds or less; for 2 rescuers infant or child CPR the delay will be even shorter.

**Emphasis on High Quality CPR :**

- A Compression rate of **atleast 100/min ( Not approximately)**
- A compression depth of **atleast 5 cm ( 2 inches)** in adults and a compressions depth of atleast one third of anterior-posterior diameter of the chest in infants and children. This is approximately 4 cm (1.5 inches) in infants and 5 cm (2inches) in children.
- Allowing complete chest recoil, minimizing interruption in compressions and avoiding excessive ventilation continue to be important components of high quality CPR.

**The Team approach for CPR:**

In 2010 AHA guidelines the stress is on high quality chest compressions CPR this can be achieved by the team approach to the CPR.

So there is a increased focus on Team CPR because resuscitations in most EMS and health care system involve teams of rescuers, with rescuers performing several actions simultaneously. One rescuer activates the emergency response and second begins chest compressions, a third is either providing ventilations or retrieving the bag-mask for rescue breathing and a fourth is retrieving a defibrillator and preparing to use it.

**No Look, Listen and Feel :**

Look listen and feel has been removed from the PLS as this activity used to delay the initiation of chest compressions specially in the setting of child and infant. The new sequence of PLS : CAB allow the early initiation of chest compression. In case of adults, first step is to activate the EMS and start chest compression. While in case of child, infant first do one cycle of chest compression and rescue breathing and then activate EMS.

**Additional actions for CPR :**

No **Cricoid pressure during ventilation :** The routine use of cricoid pressure in cardiac arrest is not recommended because cricoid pressure can block the airway and interrupt the ventilation. The advantage of cricoid pressure is that it can prevent gastric insufflation and reduce the risk of regurgitation.

**Do not check the pulse too frequently:**

It could be difficult to determine the presence or absence of a pulse within 10 seconds ,specially in an emergency situation and studies show that in emergency situation, even health care providers can not determine absence and presence of pulse within 10 seconds.

**Use of an AED for infants:**

For infants, a manual defibrillator is preferred to an AED for defibrillation. If a manual defibrillator is not present is not available, an AED equipped with a pediatric dose attenuator is preferred. If neither is available, you may use an AED without a pediatric dose attenuator.

CHAPTER 2

# INTRODUCING LIFE SUPPORT FOR ADULTS

## BLS/ CPR for Adults

This part of manual will deal with basic steps of CPR for adults. Adults will include adolescent ( i.e after the onset of puberty) . Signs of puberty include chest or underarm hair in males and any breast development in females.

**Learning Objectives :**

At the end of this part of manual the participants of this course will be able to

- Tell the basic steps of CPR for adults
- Show he basic steps of CPR for adults.

**Components of BLS:**

BLS consists of these main parts:

- Chest Compressions
- Airway
- Defibrillation

There are two approach to CPR- single rescuers or a team of rescuers. Single rescuers will do all the sequence one by one alone while the team members can do the simultaneous action like :

- First will activate the EMS

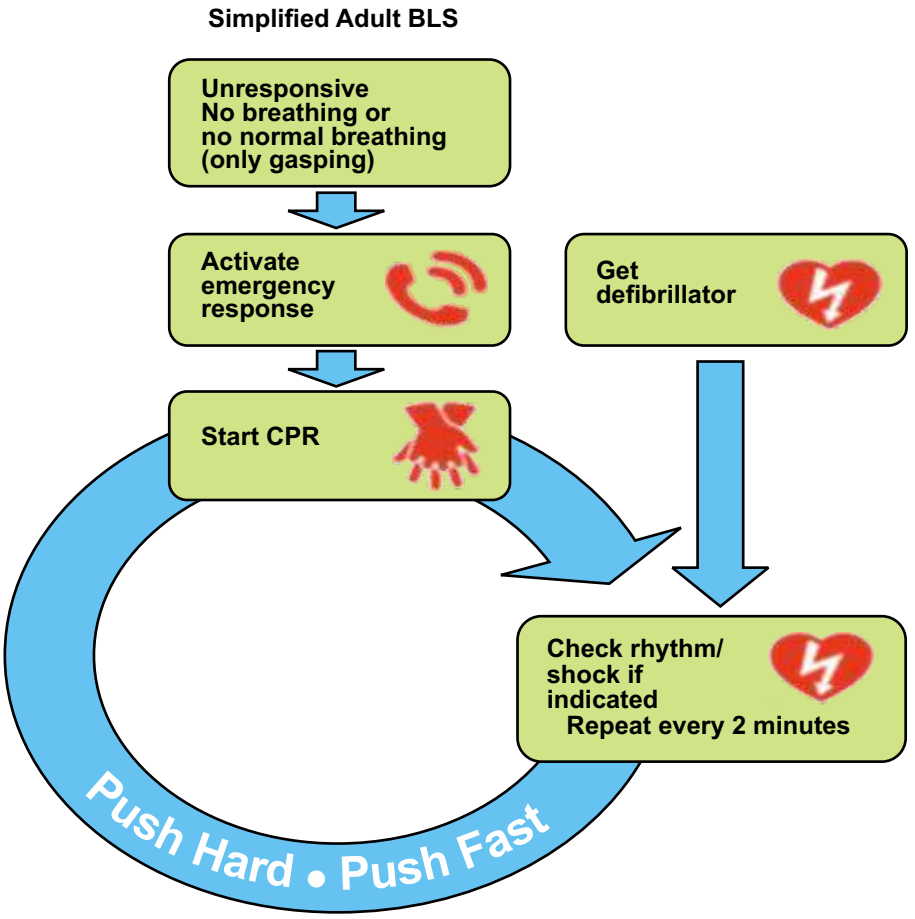



Figure 1 : Simplified form of Adult BLS

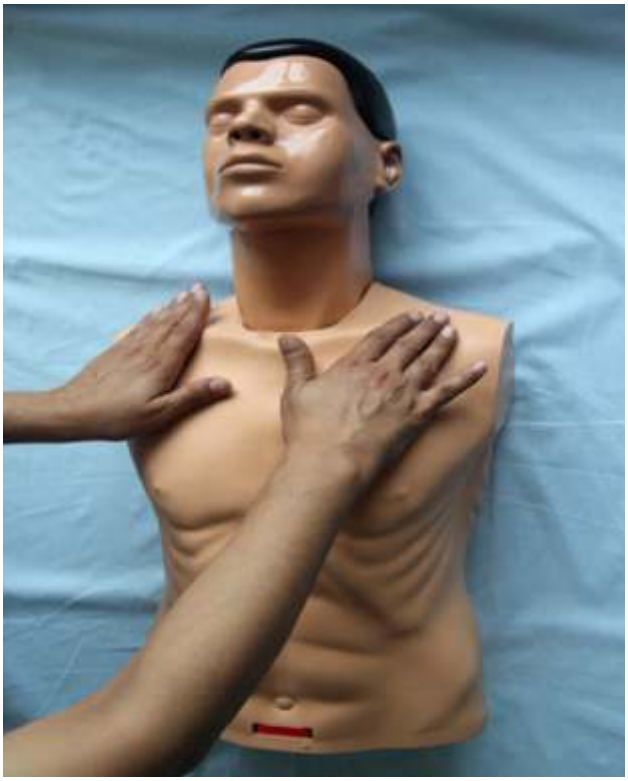


Figure 2 : Check for response and Breathing. (a) Check the patient's shoulder and shout " are you all right?" and simultaneously, look for breathing. (b) if patient does not respond and there is no breathing. Shout for help or call for help.

Figure 3 : Shout for help and if other rescuer responds, send him or her to activate the emergency system. Get the AED or defibrillation if available. If no one responds, activate emergency system, get AED or defibrillation and return to the patient to check a pulse and begin CPR (C-A-B) sequence.

- 2nd will start the chest compression
- Third will do the breathing and ventilation
- Fourth will retrieve the bag and mask, AED and will take the responsibility of applying the electrodes and switching on the AED.
- If fifth rescuers is there, he can be given a responsibility of taking focused history of relevance and can record the events of BLS.

**Overview of BLS Steps :**

1. Assess the patient for response. If no response and patient is also not breathing, then shout for help
2. If alone, activate the emergency response system and get an AED ( or defibrillator)
3. Check patient's pulse (Carotid pulse in adult, femoral or brachial in child / Infant) take at least 5 second but not more than 10 seconds.
4. If pulse not felt within 10 seconds, perform 5 cycles of compressions and breaths (30:2 ratio), starting the sequence as C-A-B (Circulation - Airway - Breathing).

**STEP 1:** To assess the patient and scene safety

1. The first rescuer who arrives at the scene must quickly assess the patient and safety of the site or scene.
2. Tap the patient's shoulder and shout, " Are you all right" ( this should be in the mother tongue of the patient)
3. Check the patient, "is he breathing " if he is not breathing normally or gasping , then activate the emergency response system. (Figure 2)

**Cautions :** Agonal gasp are not normal breathing. Agonal gasps may be present in first few minutes after sudden cardiac arrest. The gasp may sound like a snort, snore, or groan. Gasping is not normal breathing. It is a sign of cardiac arrest in someone who does not respond.

If the patient is not breathing or there is no breathing, one must activate the emergency response system, check the pulse and start CPR.

Figure 4 : Finding the pulse over Carotid artery (a) Locate the trachea first and slightly slip down laterally , gently feel the carotid pulse – don't press hard.

**STEP 2.** Activate the emergency response system and get an AED.

If alone and you find an unresponsive patient not breathing, shout for help. If no one responds, activate the emergency response system, get an AED if available. (Figure 3)

**STEP3.** Pulse check.

Health care provider should not take more than 10 seconds to determine the presence or absence of pulse.

Locating the carotid artery pulse: To perform a pulse check in the adults, palpate a carotid pulse by locating the trachea and gently feel for carotid pulse. (Figure 4)

1. Locate the trachea with 2 or 3 fingers.
2. Slide these 2 or 3 fingers into the groove between the trachea and the muscles at the side of the neck, where one can feel the carotid pulse.

Figure 5 : Place your hands over lower 1/3rd of sternum right in the center of the chest (b) Correct position of the rescuer during chest compressions.

3. Feel for a pulse for at least 5 but not more than 10 seconds. If you don't definitely feel a pulse, begin CPR, starting with chest compressions.

**STEP 4. Start CPR in cycles of 30 chest compression and 2 breaths (30:2, CPR)**

When chest compressions are given to the patient, it is important to **push chest hard and fast, at a rate of atleast 100/min, allow the chest to recoil completely after each compression and minimize interruptions in compressions.** The compression to ventilation ratio should be 30:2 i.e. one cycle of CPR. 5 cycles of CPR should be completed within 2 minutes.

**STEP 5 : Chest Compression Technique:**

Chest compressions are most important component of CPR, so follow these steps to perform chest compressions in an adult.

1. Extricate the patient at a safe site and position yourself at the patient's side.
2. Make sure that the patient is lying face up on a firm /hard, flat surface. If the patient has a lying facedown, carefully roll him face up. If you suspect the patient has a head or neck injury, try to keep the head, neck, and body in line when rolling the patient to a face up position.
3. Put the heel of the palm over the center of the patient's chest on the lower half of breast bone.
4. Put the heel of your other hand on the top of hand placed on patients chest. (Figure 5)

5. Straighten your arm and position yourself directly over your hands.

**STEP 6 : Push, hard and fast.**

- Press down or depress at least 5 cm ( 2 inches) with each compression, make sure you push straight down on the patient's breastbone.
- Deliver compressions in a smooth fashion at rate of at least 100/min. Compressions should not be jerky and the force needed to give compression should not generate from wrist or elbow of the hand. So the elbow should not flex at the time of delivering chest compressions.

Figure 6 : Alternative (one hand) technique for chest compressions for rescuers with arthritis.

**STEP 7** : At the end of each compression, **make sure you allow the chest to recoil or** re-expand completely. Chest recoil, allows the blood to flow into the heart and is necessary for chest compressions to create blood flow. This phase of chest compression is correlated to the diastole phase of heart beat, which is responsible for coronary circulation needed for the myocardial function.

8. There should be **a minimum interruption of chest compressions.** (i.e interruption should be only for using AED, repositioning of patient, intubating trachea and transporting the patient.

## Clinical facts of high quality CPR:

**Importance of firm or hard down surface under patient:**

Figure 7. The head tilt-chin lift opens up the airway obstruction in unresponsive patient (a) airway obstruction is caused by tongue in unconscious and unresponsive patient (b) The head tilt-chin lift maneuver lifts up the tongue and thus opening the obstructed airway.

Chest compressions compresses heart between the thoracic vertebra posteriorly and sternum anteriorly, which leads to pumping out of the blood from heart to rest of body. If a firm surface is under the patient, the force used is more likely to compress the chest and heart and create blood flow rather than simply push the patient into the mattress or other soft surface.

**CPR technique for rescuers with arthritis (Figure 6):**

Put one hand on the lower 1/3rd of sternum right on the center of chest to push on the chest. Grasp the wrist of hand with your other hand to support the first hand as it pushes the chest.

## Opening the Airway for breath

There are two maneuver which can be used to open the airway to provide the breaths. When there are two rescuers, one perform the jaw thrust and provide breaths with a bag-mask device. Use jaw thrust only if you suspect the hand or neck injury, as this technique for opening airway reduce neck and spine movement. Switch to head tilt- chin lift maneuver if the jaw thrust does not open the airway.

**A. Head tilt- chin lift :**

1. Place one hand on the patient's forehead and push with your palm to tilt the head back.
2. Place the fingers of the other hand under the bony part of the lower jaw near the chin.

3. Lift the jaw to bring the chin forward.

Don't press deeply into the soft tissue under chin because this might block the airway. Don't use thumb to tilt the chin. Do not close the patient's mouth completely.

**Open the airway for Breaths: Jaw thrust.**

If the patient is suspected to have head or neck injury. Two rescuers may use another method to open the airway i.e. jaw thrust.

**Method of doing Jaw thrust (Figure 8):**

1. Place one hand on each side of the patient's head, resting your elbows on the surface on which the patient is lying.
2. Place your fingers under the angles of the patient's lower jaw and lift with both hands, displacing the jaw forward.
3. If the lips close, push the lower lip with your thumb to open the lips.

**Mouth to Mouth Breaths:**

Figure 8. Jaw thrust to open the obstructed airway in the patient's of suspected cervical spine injury.

Because many of the Cardiac arrest happen to be at home, you may need to give breaths to a family member or close friend. This part of manual will describe you, how to give Mouth to Mouth breaths when you do not have a pocket mask or bas-mask.

**Learning Objectives:**

At the end of this portion of the course you should be to give mouth to mouth breaths.

Adult Mouth to Mouth Breathing: This is a quick method of giving breathing (effective oxygen –Know as Expired air ventilation) to the patient in absence of pocket mask or bag-mask. The rescuer's expired air containes approximately 17 % oxygen and 4 % CO2. This is enough oxygen to help the patient at this hour of crisis.

Following are the steps of providing the mouth to mouth breaths.

1. Hold the patient's airway open with a head tilt-chin lift.
2. Pinch the nose closed with your thumb and index finger ( Using hand on the forehead).
3. Take a regular (not deep) breath and seal your lips around the patient's mouth, creating an airtight seal.
4. Give 1 breaths ( blow for about 1 second). Watch for the chest to rise as you give the breath.
5. If the chest does not rise, repeat the head tilt – chin lift.
6. Give a second breath and watch for the chest to rise.
7. If you are unable to ventilate the patient after 2 attempts, promptly return to chest compressions.

**Clinical facts:**

Following is the clinical fact which has to be taken care off.

**Risk of Gastric insufflation:**

During mouth to mouth breaths, if you provide breaths too quickly or with too much force, air is likely to enter the stomach rather than the lungs. This leads to gastric insufflation.

Gastric insufflation frequently results during mouth to mouth, mouth to mask or bag-mask ventilation. Gastric insufflation can result in serious complications such as vomiting, aspiration, or pneumonia. Rescuers can reduce the risk of gastric insufflation by avoiding giving breaths too rapidly, too forcefully or with too much volume. During CPR, however, gastric insufflation may develop even when rescuers give breaths correctly.

To Reduce the risk of gastric insufflation:

- Take 1 second to deliver each breath.
- Deliver air until you make the patient's chest rise.

**Adult Mouth to Barrier device breathing:**

In this era of the infections spreading via body fluids, it advisable to use the barrier methods to ventilate the patient. This includes face mask (Pocket mask) or bag-mask device or Brookes airway- as all these devices have one way valve that diverts exhaled air, blood or bodily fluids away from the rescuers. (So rescuers are advised to take all universal precautions while doing mouth to barrier device breathing.)

The advantage of bag-mask ventilation (BMV) is that it has an oxygen inlet which can be used to administer oxygen to enhance oxygenation of the patient.

**Providing Mouth to Mask Breaths:**

To provide breath with face mask, the lone rescuer is at the patient's side, so that he can give breath and perform CPR . The lone rescuers holds the mask against the patient's face and opens the airway with a head tilt- chin lift.

Following are the Steps to open the airway with head tilt-chin lift.

1. Position yourself at the patient's side.
2. Place the mask on the patient's face, using the bridge of the nose as a guide for correct position.
3. Seal the mask against the face:

   - Using the hand that is closer to the top of the patient's head, place your index finger and thumb along the edge of the mask.

   -Place the thumb of your second hand along the bottom, edge of the mask.
4. Place the remaining fingers of your second hand along the bony margin of the jaw and lift the jaw. Perform a head tilt- chin lift to open the airway.
5. While you lift the jaw, press firmly and completely around the outside edge of the mask to seal the mask against the face.
6. Deliver breath air over 1 second to make the patient's chest rise. And don't take deep breath while you are going to blow the exhaled air into the mouth of the patient's mouth. So you should take normal tidal volume breaths while doing mouth to mask breathing.

**Bag-Mask Device Breathing :**

Here the face mask is attached with self inflating bag. The face mask contain 1 way valve to prevent the exhaled air, blood or body fluid from being to wards the rescuers. Bag-mask devices are the most common method that health care provider use to give positive-pressure ventilation during CPR. This device is used only when there are at least 2 rescuers.

Method of Bag-mask ventilation by 2 rescuers (Figure 9):

Follow these steps to open the airway with a head tilt – chin lift and use a bag-mask to give breaths to the patient.

1. Position yourself directly above the patient's face.

Figure 9. : Mouth to mask E-C clamp technique of holding mask while lifting the jaw and delivering breaths.

2. Place the mask on the patient's face, using the bridge of the nose as a guide for correct position.
3. Use E-C clamp technique to hold the mask in it's place while lifting the jaw to enable patent airway.

- Perform the head tilt

-Place the mask on the face with the narrow portion at the bridge of the nose.

-Use the thumb and index finger of one hand to make a "C" on the side of the mask, pressing the edges of the mask to the face.

- Use the remaining fingers to tilt the angles of jaw ( 3 fingers form an "E") open the airway, and press the face to the mask.

4. Squeeze the bag to give breaths (1second each) while watching for chest rise.

**Clinical Fact:**

**Giving Breaths with supplementary oxygen:**

During bag-mask Ventilation, you should use supplementary oxygen. You should still deliver each breath over 1 seconds. If you use only 1 second per breath for any method of delivery, you can help minimize the interruptions in chest compressions needed for breath and avoid excessive ventilation.

## Two-Rescuers Adult BLS/Team CPR Sequence :

This part of manual will teach you that how to perform 2 rescuers team CPR for adults.

**Learning objective:**

At the end of this part of manual you will be able to demonstrate how to perform 2 rescuers team CPR.

**When more rescuers are present:**

When a second rescuer is available to help, that second rescuer should activate the emergency response system and get the AED. The first rescuer should start CPR immediately and the first rescuer should use the AED as soon as it is available. The rescuers will then give compressions and breaths but switch roles after every 5 cycles of CPR i.e. after 2 minutes.

As additional rescuers arrive , they can help with the bag-mask ventilation, use of AED or defibrillator and crash cart.

**Tasks for Each rescuer's :**

**Rescuer 1:** Position himself at the side of patient and perform chest compressions. Compress the chest atleast 5 cm ( 2 inches) and compress at the rate of atleast 100/min and allow the chest to recoil completely after each compression. Minimize interruptions in compressions ( try to limit any interruption in chest compressions < 10 seconds.) Use a compressions to breaths ration of 30:2. Count compressions aloud. And switch duties with the second rescuers every 5 cylces or about 2 minutes, taking <5 seconds to switch.

**Rescuer 2 :** The 2nd rescuers stands at the patients head and maintains an open airway using either head tilt-chin lift or jaw thrust. Give breaths, watching for the chest rise and avoiding excessive ventilation and encourage the first rescuer to perform compressions that are deep enough and fast enough and to allow complete chest recoil between compressions. Switch duties after every 5 cycles or about 2 minutes, taking less than 5 seconds.

**Clinical facts:**

**Effective team performance to minimize interruptions in compressions:**

Effective teams communicate continuously. If the compressor counts out loud, the rescuers providing breaths can anticipate when breaths will be given and prepare to give them efficiently to minimize interruptions in compressions. Count will also help each rescuers to know when the time for a switch is approaching.

It is hard work to deliver effective chest compressions. If the compressor tires, chest compressions wont be effective. To reduce rescuer fatigue, switch compressor every 5 cycles to minimize interruption, perform the switching when the AED is analyzing the rhythm and it should take no more than 5 seconds to switch.

## Two Rescuers Using the Bag-Mask breathing (Figure 9) :

When 3 or more rescuers are present, 2 rescuers can provide more effective bag- mask ventilation than 1 rescuer. When 2 rescuers use the bag mask system, one rescuer opens the airway with head tilt- chin lift or jaw thrust and holds the mask to the face while the other rescuer squeezes the bag. All professional rescuers should learn both the 1 and 2 rescuer bag-

mask ventilation techniques. When possible in the course, practice with devices for both bag-mask and mouth to mask ventilation.

(III) Automated External Defibrillator for adults and children 8 years of age and older.

Overview: The interval from collapse to defibrillation is one of the most important determinants of survival from sudden cardiac arrest with ventricular fibrillation or pulseless ventricular tachycardia.

Automated external defibrillators (AED) are computerized devices that can identify cardiac rhythm that needs a shock and they can then deliver the shock. AEDs are simple to operate, allowing laypersons and healthcare providers to attempt defibrillation safely.

**Learning Objective :** At the end of this part of manual you will be able to

- List the steps common to the operation of all AEDs.

Figure 9 : Two rescuer's bag- mask ventilation. The rescuer at the patient's head tilts the patient's head and seals the mask against the patient's face with the thumb and first finger of each hand, creating a "C" to provide a complete seal around the edges of the mask. Rescuer uses three fingers (the "E") to tilt the jaw (this holds the airway open) and holds the face up against the mask. The second rescuer slowly squeezes the bag (over 1 second) until chest rises.

- Apply proper placement of the AED pads.
- Recall when to press the shock button when using an AED.
- Explain why no one should touch the patient when prompted by the AED during analysis and shock delivery.
- Describe the proper actions to take when the AED gives a "no shock indicated" ( or no shock advised") message
- Their must be coordination of CPR and AED to minimize

  -Interruptions in chest compressions.

  -Time between last compression and shock delivery

  -Time between shock delivery and resumption of chest compressions.

**AED Arrival :** Once the AED arrives, place it next to the rescuer who will operate it. This position provides ready access to the AED controls and easy placement of AED pads. It also allows a second rescuer to perform CPR from the opposite side of the patient without interfering with AED operation.

**Note :** If multiple rescuers are present, one rescuer should continue chest compressions while another rescuer attaches the AED pads.

**Clinical Facts :**

Defibrillation- When ventricular defibrillation is present, the heart muscle fibers quiver and do not contract together to pump blood. The defibrillation allows the muscle fibers of the heart to "reset" so that they can begin to contract at the same time. Once an organized rhythm occurs, the heart muscle may begin to contract effectively and begin to generate a pulse ( called as a return of spontaneous circulation or ROSC).

There are four universal steps for operating an AED:

1. **POWER ON AED** (the AED will guide you through the next step)

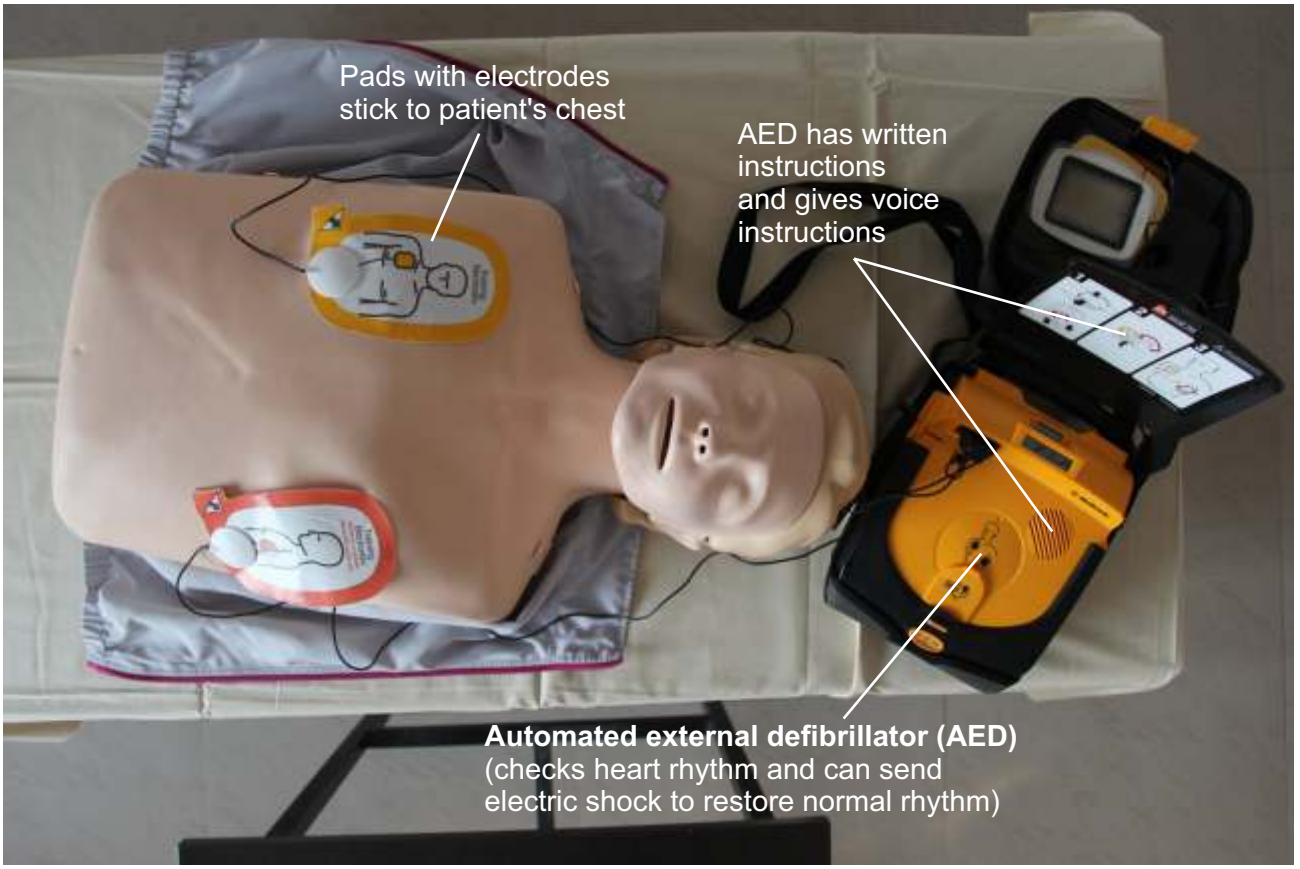



Figure11. AED pads placement on the patient. Once AED is on, it starts prompting instruction. You just have to follow the instruction given by AED.

- Open the carrying case or the top of the AED
- Turn the power on ( some devices will " power on" automatically when you open the lid or case)

**2. ATTACH AED pads to the patient's bare chest.**

- Choose adult pads ( not child pads or a child system) for patient's 8 year of age and older.
- Peel the backing away from the AED pads
- Attach the adhesive AED pads to the patient's bare chest.

- Place one AED pad on the patient's upper right chest (directly below the collar bone)

- Place the other pad to the side of the left nipple, with the top edge of the pad a few centimeters below the armpit.

Attach the AED connecting cables to the AED box (some are pre-connected)

**3. "Clear" the patient and ANALYZE the Rhythm.**

- If the AED prompts you, clear the patient during analysis, be sure to no one is touching the patient, not even the rescuer in charge of giving breaths.
- Some AEDs will tell you to push a button to allow the AED to begin analyzing the heart rhythm, others will do that automatically. The AED may take about 5 to 15 seconds to analyze.
- The AED then tells you if a shock is needed.

**4. If the AED advises a shock, it will tell you to clear the patient.**

- Clear the patient before delivering the shock: be sure no one is touching the patient.
- Loudly state a " clear the patient" message, such as" Everybody clear" or simply "clear"

- Look to be sure no one is in contact with the patient.
- Press the SHOCK button
- The shock will produce a sudden contraction of the patient's muscles.
- If no shock is needed, and after any shock delivery, immediately resume CPR, starting with chest compressions.
- After 5 cycles or about 2 minutes of CPR, the AED will prompt you to repeat steps 3 and 4. If "no shock advised"immediately restart CPR beginning with chest compressions.

**Clinical Facts:**

**Importance of Minimising time between last compression and shock delivery.** If the rescuer is able to keep the time between last chest compression and shock delivery to less than 10 seconds, shock is much more effective in eliminating ventricular fibrillation thus resulting in return of spontaneous circulation.

Effectiveness of shock delivery decreases significantly for every 10 additional seconds that elapse between the last compression and shock delivery. To minimize this interval one need to practice team coordination, particularly between compressor and the rescuer operating the defibrillator.

**Don't move the patient unnecessarily :** The patient should not move at all while the AED is analyzing the rhythm and delivering shock. As movement will lead to error ( by producing artifacts) in analyzing rhythm.

**Special Situations:**

There are some situations that may require the rescuer to take additional actions when using an AED.

- The patient has a hairy chest
- The patient is immersed in water or water is covering the patient's chest.
- The patient has an implanted defibrillator or pacemaker.
- The patient has a transdermal medication patch or other object on the surface of the skin where the AED pads are placed.

*Hairy chest :* If a teen or adult patient has a lot of chest hair, the AED pads may not stick to the skin on the chest. If this occurs , the AED will not be able to analyze the patient's heart rhythm. The AED will then give a "check electrodes" or "check electrode pads"message.

**Steps:**

1. If the pads sticks to the hair instead of the skin, press down firmly on each pad.
2. If the AED continues to prompt you to " check pads or check electrodes" quickly pull of the pads. This will remove a large amount of hair and should allow the pads to stick to the skin.
3. If a large amount of hair still remains where you will put the pads, shave the area with the razor in the AED carrying case.
4. Put on a new set of pads. Follow the AED voice prompts.

*Water* is a good conductor of electricity. Do not use an AED in water. If the patient is in water, pull the patient out of the water. If the patient is lying in water or the chest is covered with water, the water may conduct the shock electricity across the skin of the patient's chest. This prevents the delivery of an adequate shock dose to the heart. If water is covering the patient's chest, quickly wipe the chest before attaching the AED pads.

If the patient is lying on snow or in a small puddle, you may use the AED.

The patient in cardiac arrest may have in situ *implanted defibrillator/ pacemakers* that automatically deliver shocks directly to the heart. You can immediately identify these devices because they create a hard lump beneath the skin of the upper chest or abdomen. The lump is half the size of a deck of cards, with an overlying scar. If you place an AED pad directly over an implanted medical device, the device may block delivery of the shock to heart.

If you identify an implanted defibrillator/ pacemaker:

- If possible avoid placing the AED pad directly over the implanted device.
- Follow the normal steps for operating an AED.

Occasionally, the analysis and shock cycles of implanted defibrillator and AEDs will conflict. If the implanted defibrillator is delivering shocks to the patient ( the patient's muscle contract in a manner like that observed after an AED shock), allow 30 to 60 seconds for the implanted defibrillator to complete the treatment cycles before delivering a shock from the AED.

Don't place AED pads directly on the top of a *drug patch* ( e.g a patch of nitroglycerine, nicotine, pain medication, hormone replacement therapy or antihypertensive drugs). The drug patch may block the transfer transfer of energy from the AED pad to the heart and may cause small burns to the skin.

**Two Rescuer BLS Sequence With an AED:**

Following are the steps for 2 rescuers BLS With an AED.

1. **Check the response and check breathing :** If the patient does not respond and is not breathing or not breathing normally ( i.e only gasping):
   - The first rescuer stays with the patient and performs the next steps until the second rescuer returns with the AED.
   - The second rescuer activates the emergency response system and gets the AED.
2. **Check for pulse :** If a pulse is not definitely felt in 10 seconds.
   - The first rescuer removes or moves clothing covering the patient's chest ( this will allow rescuers to apply the AED pads when the AED arrives) and starts CPR, beginning with chest compressions.
3. **Attempt defibrillation with the AED:**

When AED arrives, place it at the patient's side near the rescuer who will be operating it. The AED is usually placed in the side of the patient opposite the rescuer who is performing chest compressions.

4. **Power on the AED :** ( The AED will guide you through the next steps)
   - Open the carrying case or top of the AED.
   - Turn the power on ( some devices will "Power on" automatically when you open the lid or case.)
5. **Attach AED pad to the patient's bare chest.**
   - Choose adult pads ( not child pads or child system) for patients 8 years of age and older.
   - Peel the backing away from the AED pads.
   - Attach the adhesive AED pads to the patient's bare chest.
     - Place one AED pad on the patient's upper right chest ( directly below the collarbone.)
     - Place the other pad to the side of left nipple, with the top edge of the pad a few centimeters below the armpit.
   - Attach the AED connecting cables to the AED box ( some are pre-connected).
6. **"Clear" the patient and analyze the rhythm**
   - If the AED prompts you, clear the patient during analysis. Be sure no one is touching the patient, not even the rescuer in charge of giving breaths.
   - Some AEDs will tell you to push a button to allow the AED to begin analyzing the heart rhythm; others will do that automatically. The AED may take about 5 to 10 seconds to analyze.
   - The AED then tells you if a shock is needed.
7. **If the AED advises a shock, it will tell you to clear the patient:**
   - Clear the patient before delivering the shock be sure no one is touching the patient.

- Loudly state a " clear the patient" message, such as" Everybody clear" or simply" clear".
- Look to be sure no one is in contact with the patient.
- Press the shock button.
- The shock will produce a sudden contraction of the patient's muscles.

8. After any shock delivery, immediately resume CPR, starting with chest compressions.
9. After 5 cycles or about 2 minutes or CPR, the AED will prompt you to repeat steps 6 and 7.
   - If "No shock is advised" immediately restart CPR beginning with chest compressions.

## Technique of Rescue breathing when patient is in Respiratory arrest but no cardiac arrest:

**Learning objectives :** This part of manual will describe you that how to provide rescue breathing for adult, child and infant.

When you see an adult, child or infant is having pulse but is not breathing effectively, rescuers should **give breaths without chest compressions.** This is called rescue breathing.

**Rescue Breathing For Adults :** Give 1 breaths every 5-6 seconds ( about 10-12 breaths/ minute) while in case of infants or children give 1 breaths every 3-5 seconds (about 12-20 breaths per minute). Each breath should be give over 1 second, and should result in visible chest rise. During this activity, pulse should only be checked about every 2 minute.

** In infants or children, if despite adequate oxygenation and ventilation, the pulse is < 60/minute with signs of poor perfusion, start CPR.

**Clinical Facts :**

Respiratory arrest : Respiratory arrest is the absence of respiration (i.e apnoea). During both respiratory arrest and inadequate ventilation, the patient has cardiac put (blood flow to the body) detectable as a palpable central pulse. The heart rate may be slow, and cardiac arrest may develop. Therefore, Health care providers should be able to identify respiratory arrest.

**CPR With an Advanced Airway (Table 1).**

This part of manual explains how to do CPR with an advanced airway.

**Compression Rate and Ratio during 2 Rescuer CPR with and Without an Advanced Airway in place:**

The compression rate for 2 rescuer CPR is at least 100/min. Until an advanced airway (e.g laryngeal mask airway, supra-glottic or endotracheal tube) is in place, rescuer must pause compressions to provide breaths.

Following are the comparative of advance airway CPR in adult, children and infants.

When an advanced airway is in place during 2 rescuer CPR, do not stop compressions to give breaths. Give 1 breath every 6-8 seconds ( 8-10 breaths per minute, without attempting to deliver breaths between compressions. There should be no pause in chest compressions for delivery of breaths.

Table 1 : Compression to ventilation ratio in adult and child with or without advance airway

| Ventilation technique | Compression to the breaths (adult) | Compressions to the breaths (child &infant) |
|---|---|---|
| No advance airway.( mouth to mouth , mouth to mask, bag-mask) | -30 compressions to 2 breaths - Compression rate at least 100/min | 15 Compressions to 2 breaths Compressions rate of at least. 1oo/min. |
| Advance airway ( endotracheal tube, laryngeal mask airway, supra-glottic) | - Compression rate of at least 100 / min without pauses for breaths. - 1 breath every 6-8 seconds (8 to 10 breaths per min | |

Table 2 : Summary of Steps of CPR for Adults, Children, and Infants.

| Component | Adults | Children | Infants |
|---|---|---|---|
| **Recognition of cardiac arrest** | Unresponsive ( tap the shoulder)<br>No breathing, No pulse felt within 10 seconds | Unresponsive<br>No breathing, No pulse felt within 10 seconds | Unresponsive ( Tap the sole of foot)<br>No breathing, No pulse felt within 10 seconds |
| **CPR Sequence** | Chest compressions, Airway, Breathing ( C-A-B) | | |
| **Compression Rate** | At least 100/ minute | | |
| **Compressions Depth** | At least5 cm ( 2 inches) | At least 1/3 AP diameter about 5 cm | At least 1/3 AP diameter about 4 cm ($1^{1/2}$ inches) |
| **Chest wall recoil** | Allow complete recoil between compressions. Rotate compressor every 2 minutes. | Allow complete recoil between compressions. Rotate compressor every 2 minutes. | Allow complete recoil between compressions. Rotate compressor every 2 minutes. |
| **Compression interruptions** | Minimize interruptions in chest compressions. Attempt to limit interruptions to < 10 seconds | Minimize interruptions in chest compressions. Attempt to limit interruptions to less than10 seconds | Minimize interruptions in chest compressions. Attempt to limit interruptions to less than 10 seconds |
| **Airway** | Head tilt-chin lift ( Suspected trauma: jaw thrust) | | |
| **Compressions-ventilation ratio (Until advanced airway placed)** | 30:2 1 or 2 rescuers | 30:2 single rescuer or 15:2 for 2 rescuers | |
| **Ventilations with advanced airway.** | 1 breath every 6-8 seconds ( 8-10 breaths/ min)<br>Asynchronous with chest compressions about 1 second per breath.<br>Visible chest rise. | | |
| **Defibrillation** | Attach and use AED as soon as available.<br>Minimize interruptions in chest compressions before and after shock;<br>Resume CPR beginning with compressions immediately after each shock. | | |

CHAPTER 3

# BASIC LIFE SUPPORT FOR CHILDREN AND INFANTS

## BLS/CPR for Children

High quality Cardio Pulmonary Resuscitation Following are the six components of high quality CPR in children

1) Start compression within 10 seconds of recognition of cardiac arrest.
2) Push hard, push fast compress at a rate of at least 100/min with a depth of at least 2inches (5cms) in adults, approximately 2 inches (5cms) for children and 11/2 inches (4cm) for infants.
3) Allow complete chest recoil
4) Minimize interruptions in compressions (limit interruptions to less than 10 seconds)
5) Give effective breaths that make the chest rise.
6) Avoid excessive ventilation.

The potential places where interruptions usually occur during the process of CPR are

a) During pulse check. Take minimum 5 seconds to feel for the pulse but not more than 10 seconds. If in 10 seconds you are not able to feel pulse or there is no pulse start chest compression.

b) During switch over of roles while performing CPR. To avoid interruptions it is recommended to switch over the position while the AED is analyzing the rhythm.

**The Pediatric chain of survival**

In children the cardiac arrest is often secondary to respiratory failure and shock. This is opposite to that of adults in which the cardiac arrest is often sudden and results from a cardiac cause. Therefore, prevention of cardiac arrest is the first step in pediatric chain of survival.

The key differences between adult and child CPR are:

1. A change in compression ventilation ratio of 15:2 for two rescuer child CPR
2. Compression depth of at least one third the depth of the chest, approximately 2 inches(5cm)
3. Compression technique: May use 1 or 2 handed chest compressions for very small children

**Activation of emergency response system in children**

1. If you did not witness the arrest and you are alone, give 2 minutes of CPR before leaving the child to activate the emergency response system and get the AED9or defibrillator)
2. If the arrest is sudden and witnessed, leave the child to activate the emergency response system and get the AED (or defibrillator), and then return to the child.

**Compression depth, Adult vs Child**

Recommended depth of compressions:

- For adults: at least 2 inches
- For child: At least one third of the anterior-posterior depth of the chest or approximately 2 inches(5cms)

**Compression rate and ratio for lone rescuer**

The lone rescuer should use **the universal compression-ventilation ratio of 30:2.** when giving CPR to patients of all ages (except new born infants).

**One handed chest compression:**

For very small children one may use either 1 or 2 hands for chest compressions. Compression depth should be one third of the anterior posterior diameter.

**Single rescuer child BLS sequence :**

1. Check for scene safety
2. Check the child for response and check breathing. If there is no response and no breathing or only gasping, shout for help.
3. If someone responds, send that person to activate the emergency response system and get the AED. However, if the child collapsed suddenly and you are alone, leave the child to activate the emergency response system and get the AED and then return to the child.
4. Check the child's pulse (take at least 5 seconds but not more than 10 seconds). One may try to feel the child's carotid or femoral pulse.

Figure 1 : One handed chest compression:

5. If within 10 seconds, a definite pulse is not felt or if , despite adequate oxygenation and ventilation the heart rate is <60/min with signs of poor perfusion, perform cycles of compression and breaths(30:2 ratio), starting with compressions.
6. After 5 cycles, if someone has not already done so, activate the emergency response system and get the AED. **Use the AED as soon as it is available.**

**Locating the carotid or femoral artery pulse :**

To perform a pulse check in the child, palpate a carotid or femoral pulse. If you do not definitely feel a pulse within 10 seconds, start chest compressions. Follow the following steps to locate the carotid and femoral pulse:

**For carotid pulse**

- Locate the trachea, using 2 or 3 fingers
- Slide fingers into the groove between the trachea and the muscle at the side of the neck, where you can feel the carotid pulse.

**For femoral pulse**

- Place two fingers in the inner thigh, midway between the hipbone and the pubic bone and just below the crease where the leg meets the abdomen(fig)

  Feel for pulse for at least 5 but not more than 10 seconds. If you do not pulse, begin CPR, starting with chest compressions(C-A-B sequence)

**2. Rescuer child BLS sequence**

1. Check for scene safety
2. Check the child for response and check breathing. If there is no response and no breathing or only gasping, the second rescuer activates the emergency response system.
3. Check the child's pulse (take at least 5 seconds but not more than 10 seconds). One may try to feel the child's carotid or femoral pulse.
4. If within 10 seconds, a definite pulse is not felt or if, despite adequate oxygenation and ventilation the heart rate is <60/min with signs of poor perfusion, perform cycles of compression and breaths (30:2 ratio), starting with compressions. When the second rescuer arrives, use a compression to breath ratio of 15:2.

**Why Breaths are important for infants and children in cardiac arrest**

In case of infants and children the cardiac arrest occurs secondary to respiratory failure or shock. As a result, the oxygen content of the blood is reduced even before the onset of arrest. Due to this, for most

children and infants chest compressions alone are not as effective for delivering oxygen to the heart and brain as the combination of compressions plus breaths. For this reason, it is very important to give both compressions and breaths for infants and children during CPR.

In contrast, cardiac arrest occurs in adults secondary to cardiac disease and the oxygen content of the blood is typically normal. As a result, compressions alone may maintain adequate oxygenation to the heart and brain for first few minutes after arrest.

## BLS/CPR for Infants

For the purpose of the BLS sequence described in the Pediatric BLS algorithm, the term infant means 28 days to 12 months of age, excluding newly born infants in the delivery room.

The Key differences for infants BLS are:

- Location of pulse check; brachial artery in infants
- Technique of delivering compressions : 2 fingers for single rescuer and 2 thumb encircling hands technique for 2 rescuers
- Compression depth: at least one third the chest depth, approximately 1 ½ inches(4 cms)
- Compression ventilation rate and ratio for 1 rescuer 30:2 and for 2 rescuer 15:2 ratio.

**Locating the Brachial Artery Pulse (Figure 2) :**

To perform a pulse check in infant, palpate a brachial pulse. It can be difficulties for healthcare providers to determine the presence or absence of a pulse in any patient, but it can be particular difficult in an infant. If an infant is unresponsive and not breathing or only gasping and a definite pulse is not felt within 10 seconds, start CPR. Begin chest compression if you do not definitely feel a pulse within 10 seconds.

Steps to locate the brachial artery pulse:

1. Place 2 or 3 fingers on the inside of the upper arm, between the infant's elbow and shoulder.
2. Press the index and middle fingers gently on the inside of the upper arm for at least 5 but not more than 10 seconds when attempting to feel the pulse.

Figure 2 : Locating brachial artery in an infant

**One Rescuer Infant CPR**

The lone rescuer should use the universal compression ventilation ratio of 30:2 when giving CPR to patients of all ages.

**Two finger chest compression technique (Figure 3)**

- Place the infant on a firm, flat surface
- Place 2 fingers in the centre of the infant's chest just below the nipple line. Do not press on the bottom of the breastbone (Figure 20).
- Push hard and fast. Press the infant's breastbone down at least one third the depth of the chest (approximately 11/2 inches or 4 cms). Deliver compressions in a smooth fashion at a rate of at least 100/min.
- At the end of each compression allow complete chest recoil. Chest recoil allows blood to flow into the heart and is necessary to create blood flow during chest compressions.
- Minimize interruptions in chest compressions.

**Techniques for giving breaths to infants**

**Mouth to mouth and nose (preferred method) (Figure 4)**

- Maintain head tilt chin lift to keep the airway open
- Place your mouth over infant's mouth and nose to create an airtight seal.

- Blow into the infant's nose and mouth to make the chest rise with each breath.
- In case, chest does not rise repeat head tilt chin lift to reopen the airway and try to give a breath that makes the chest rise. When the airway is open, give 2 breaths that make the chest rise (Figure 5)

**Mouth to mouth (used if you can't cover the nose and mouth with your mouth)**

- Maintain a head tilt-chin lift to keep the airway open
- Pinch the patient's nose tightly with thumb and forefinger.
- Make a mouth to mouth seal.
- Provide 2 mouth to mouth breaths to make the chest rise.

**Bag and mask ventilation**

Bag and mask device consist of a bag attached to a face mask. These are the most common devices used by the healthcare providers to give positive pressure ventilation. **This technique requires expertise and is not recommended for lone rescuer during CPR.**

Figure 3 : Two thumb technique of chest compression for single rescuer

Using bag and mask device during 2-rescuer CPR.

- Position yourself directly above the patient's head.

Figure 4 : Mouth to mouth and nose technique of giving breath in an infant

- Place the mask on the patient's face, using the bridge of the nose as a guide for correct position.
- Use the EC clamp technique to hold the mask in place while you lift the jaw to hold the airway open

Figure 5 : Head tilt chin lift maneuver in an infant

- o Perform a head tilt
- o Place the mask o the face with the narrow portion at the bridge of the nose.
- o Use the thumb and index finger of one hand to make a "C"on the side of the mask, pressing the edges of the mask to the face.
- o Use the remaining fingers to lift the angles of the jaw (3 fingers form an "E"),

open the airway and press the face to the mask (Figure 6).

- o Squeeze the bag to give breaths (1 second each) while waiting for chest rise give all breaths over 1 second whether or not you use supplemental oxygen.

## Two rescuer infant CPR

- Place both thumbs side by side in the centre of the infant's chest on the lower half of the breastbone. The thumbs may overlap in very small infants.
- Encircle the infant's chest and support the infant's back with the fingers of both hands
- With hands encircling the chest, use both thumbs to depress the breastbone approximately one third the depth of infant's chest (Figure 7).
- Deliver compression in a smooth fashion at a rate of at least 100/min.
- Allow full chest recoil after each compression. After every 15 compressions stop briefly for the second rescuer to open the airway with a head tilt chin lift and give 2 breaths. The chest should rise with each breath.
- Continue compressions and breaths in ratio of 15:2(for 2 rescuers), switching roles every 2 minutes to avoid rescuer fatigue (Figure 8).

Automated External Defibrillator for Infants and for Children from 1 to 8 Years of Age

- As in adults, use the AED as soon as it is available. Use child pads and a child system if it is available.
- For infants a manual defibrillator is preferred to an AED for defibrillation. If manual defibrillator is not available, an AED equipped with Pediatric dose attenuator (means having separate pads for pediatric age) is preferred. If neither is available, you may use an AED without a pediatric dose attenuator.

**Use of adult dose is better than no attempt at defibrillation**

Figure 7 : Two thumb encircling hands technique during 2 rescuer infant CPR

**CPR with an Advanced Airway**

When an advance airway is in place during 2 rescuers CPR do not stop compressions to give breaths. Give one breath every 6 to 8 seconds (8 to 10 breaths). The compression rate should be given at a rate of at least 100/min without pauses for breaths.

Figure 6 : E-C technique of giving bag & mask ventilation

Figure 8 : Two rescuer chest compression and bag and mask ventilation

**Rescue Breathing**

When an adult, child or infant has a pulse but is not breathing effectively ( apneic or gasping), rescuer should give breaths without chest compressions. This is called as rescue breathing.

Table 1 : Rescue breathing for adults, infant and children

| Rescue breathing for adults | Rescue breathing for infants and children |
|---|---|
| Give 1 breath every 5 to 6 seconds(about 10 to 12 breaths per minute) | Give 1 breath every 3 to 5 seconds(about 12 to 20 breaths per minute) |
| Give each breath in 1 second<br>Each breath should result in visible chest rise<br>Check the pulse about every 2 minutes. | |

**Relief of choking in children and infants** (Figure 9)
Choking is when food or another object gets stuck in the airway or throat. Choking could be mild or severe.

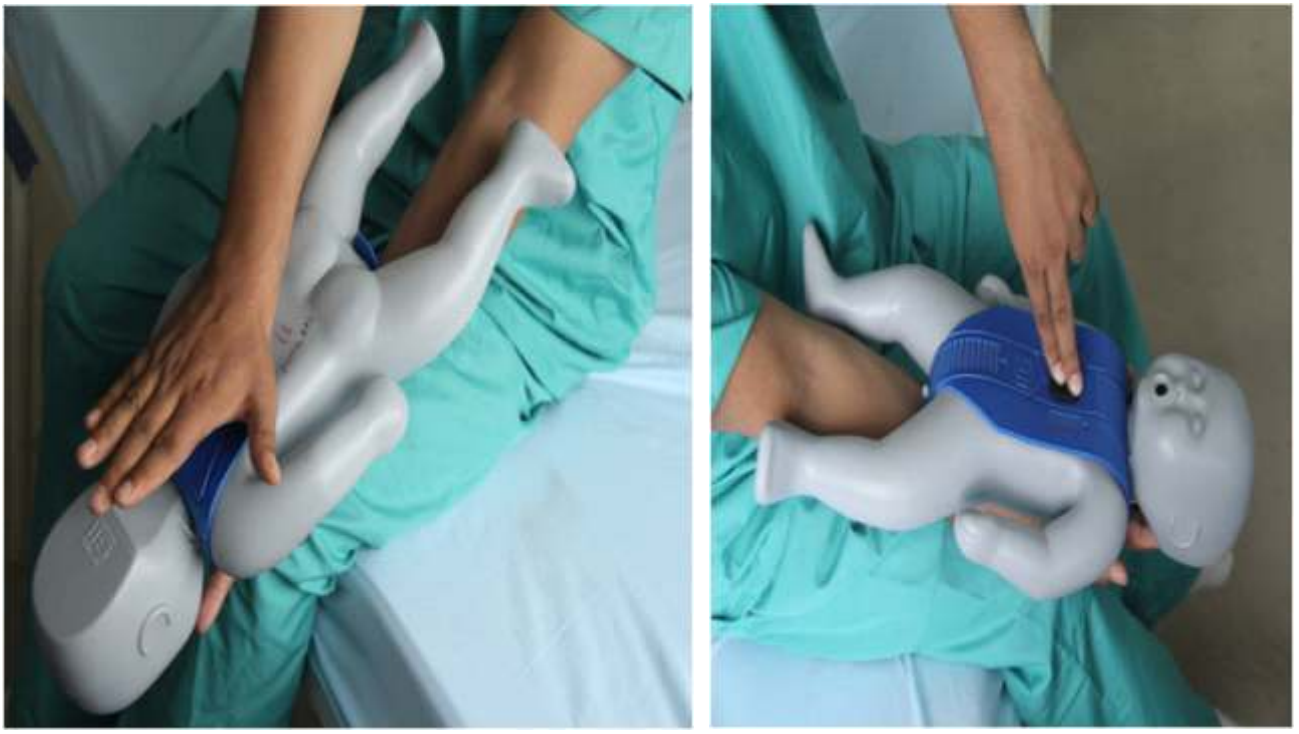


Figure 9 : Back slaps and chest thrusts to relieve chocking in an infant

Table 2 : Management of choking in a child & Infant.

| **Mild choking** | **Child** | **Infant** |
|---|---|---|
| There is good air exchange, patient can make sounds, can cough loudly and may wheeze between cough. | Encourage the patient to continue spontaneous breathing efforts and coughing | Encourage the patient to continue spontaneous breathing efforts and coughing |
| **Severe choking**<br><br>patient cannot breath or has increased respiratory difficulty, coughs without sound, cannot talk or make a sound. Has cyanosis and makes choking sign.<br><br>Infant with severe choking is unable to cry and shows of cyanosis | Ask the patient if he/she is choking. If the patient nodes yes, cannot talk severe airway obstruction is present.<br><br>**Steps to relieve severe choking in child victim:**<br><br>Get behind the patient. Wrap your arms around him so that your hands are in front.<br><br>Make a fist with one hand<br><br>Put the thumb side of your fist slightly above the belly button and well below the breastbone.<br><br>Grasp your fist with your other hand and give quick upward thrusts into the belly.<br><br>Give thrusts until the object is forced out and patient can breath, cough or talk or until he stops responding(Same as adult)<br><br>**If patient becomes unresponsive**In thiscircumstance activate the emergency response system. Lower the patient to the ground and begin CPR, starting with chest compression **(do not check for a pulse).**For adult or child patient look for foreign body every time you open the airway to give breaths. If you see an object that cans easily be removed, remove with your fingers. If you do not see an object, keep doing CPR. | If the infant has signs of severe obstructions, follow the following steps:<br><br>**Steps to relieve severe choking in infant patient:**<br><br>Kneel or sit with the infant in your lap.<br><br>Remove clothing, if it is easy to do.<br><br>Hold the infant face down with the head slightly lower than the chest, resting on forearm. Support the infants head and jaw with your hand. Take care to avoid compressing the soft tissues of the infant's throat. Rest your forearm on your lap or thigh to support the infant.<br><br>Give 5 back slaps forcefully between infant's shoulder blades, using the heel of your hand. Deliver each slap with sufficient force to dislodge the foreign body.<br><br>After giving 5 back slaps, place your free hand on the infant's back, supporting the back of the infant's head with the palm of your hand. The infant will be adequately cradled between your 2 forearms with the palm of one hand supporting the face and jaw while the palm of other hand supports the back of the infant's head.<br><br>Turn the infant as a unit while carefully supporting the head and neck. Hold the infant's face up, with your forearm resting on your thigh. Keep the infant's head lower than trunk.<br>Provide 5 quick downward chest thrusts in the middle of the chest over the lower half of the breastbone (same as for chest compression during CPR**). Deliver chest thrusts at a rate of about 1 per sec** each with the intention to create a force sufficient to dislodge the object.<br><br>Repeat the sequence of up to 5 back slaps *and* upto 5 chest thrusts until the object is removed or the infant becomes unresponsive.<br><br>If the infant becomes unresponsive, stop giving back slaps, call for help and begin CPR. |

CHAPTER 4

# NORMAL ELECTROCARDIOGRAM

Electrocardiogram (ECG) is a recording of electrical signals obtained from the conduction system of the heart. In a standard ECG, since recording of these signals is performed by using leads on the surface of body, it is also known as "surface ECG".

Conduction system of the heart generates and conducts electric current from Sinoatrial node (SA node) to the atria and then to atrioventricular node (AV node) , and further to the ventricular muscle. This electrical current makes ventricles to contract in a manner that they are able to pump blood into the blood vessels. This ventricular contraction is also known as "systole", and outflow of blood into the vessels generates a pulse, as well as a blood pressure.

In order to understand if heart is working well, we use various tools. A normal blood pressure and peripheral oxygenation (judged by pulse oxymetry) help us determine if heart is able to maintain an adequate outflow. Ventricles act as a pump, responsible for this outflow.

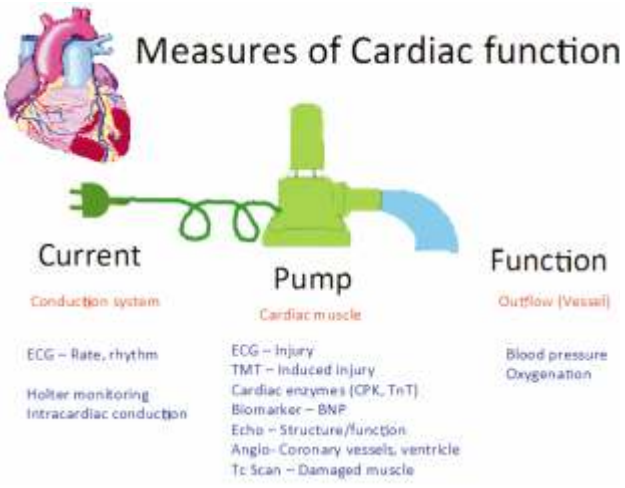



Figure 1: Measures of cardiac function in conduction system (current), pump (cardiac muscle) and function (outflow). ECG = electrocardiogram; TMT = Treadmill test; CPK = Creatinine phosphokinase; BNP (B-type natriuretic peptide); Tc Scan = Technetium scan.

Many a times even if this pump is not entirely normal (or injured), it can stress itself to maintain a normal outflow and oxygenation. If cardiac injury is evident in a resting situation, it can be determined by an ECG, as injured cardiac muscle can lead to specific patterns in ECG. Abnormalities in the conduction system are more readily detected by a resting surface ECG. However a normal ECG may not always suggest a normal heart, as various other tests as shown in the figure 1 below are used to detect an abnormality.

ECG is obtained on a grid-paper, known as ECG paper. The horizontal axis denotes time, and vertical axis denotes amplitude. Standard ECG is obtained with a paper speed of 25mm/sec, and an amplitude of 10mm/mV. ECG paper grid is in small and large squares (small square = 1mm, and a large square =5 mm). Two vertical large squares are equal to 1mV amplitude, and five horizontal biq squares are equal to one second (See Figure 2).

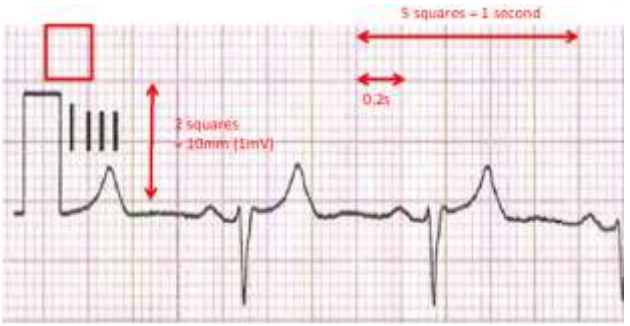



Figure 2 : ECG paper and its measurements.

Normal ECG has three principal waves P, QRS, and T. Hence the waveform is also known as P-QRS-T waveform (See Figure 3).

QRS interval is the most prominent wave, seen in all the leads of the ECG (See figure 6). However it may not always be in a QRS fashion. It may be a RS wave,

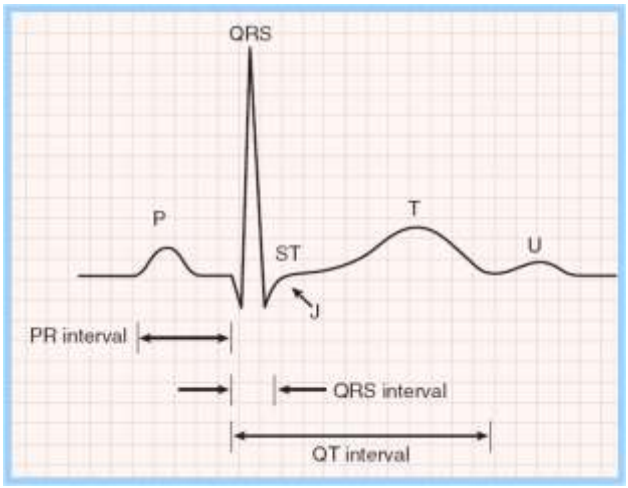



Figure 3: ECG waveforms. P-wave is first positive wave and leads to atrial contraction; QRS complex is made of a first negative (Q), first positive (R) and second negative (S )waves and leads to ventricular contrcation; T wave is a positive wave after QRS complex and is responsible for ventricular relaxation.

QR wave, or a RS waveform. This difference in QRS configuration is due to the fact that this waveform is obtained from ECG leads which are looking at the conduction system of the heart from different directions.

Standard ECG has 12 leads, six of these leads are limb leads (I, II, III, aVR, aVL, and aVF). Remaining six are chest leads (V1, V2, V3, V4, V5, and V6). These 12 leads are obtained by attaching 4 electrodes to the limbs (both arms, and both legs) and 6 electrodes to the chest wall. (Figure 4)

By virtue of their location, chest leads capture electric current from the part of the cardiac muscle which lies directly below the location where elctrode is placed. Thus V1 & V2 capture current from rightventricle and the inter-ventricular septum (Septal leads), V3 & V4 from anterior wall (anterior), and I, aVL, V5 & V6 from the lateral wall (lateral leads). Leads II, III, aVF inform about the inferior surface of the heart (inferior). Thus ECG waves are interpreted in groups. By looking at these combinations of leads we will be able to diagnose which muscle of heart is injured. (Figure 5)

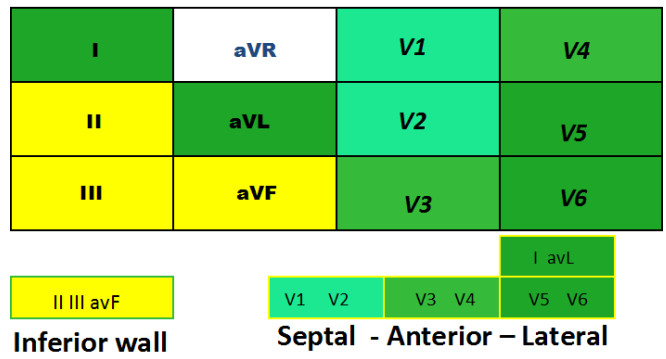



Figure 5: ECG leads and their relation to the heart.

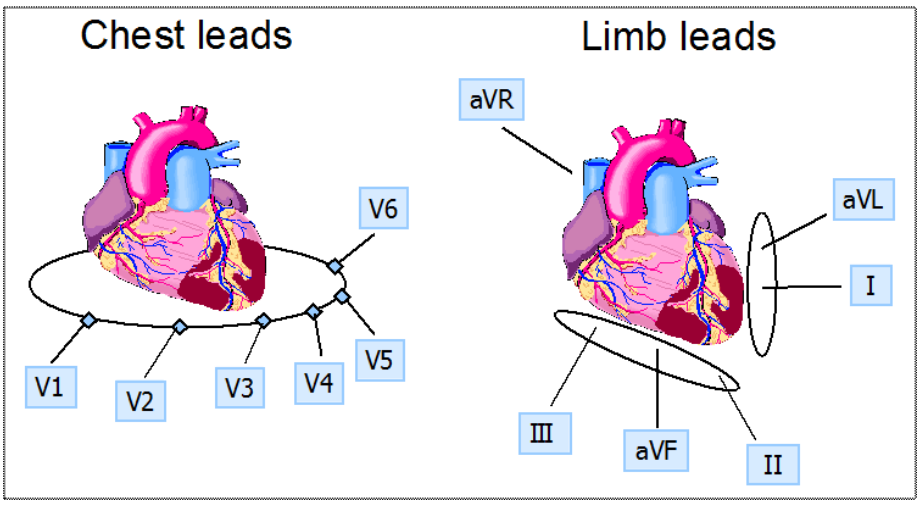



Figure 4: ECG leads and their relation to the heart.

On the other hand conduction abnormalities are detected in all the leads. We usually use Lead II, as a standard lead to identify such abnormalities in cardiac rhythm (known as rhythm strip).

An understanding of grid, waveforms, intervals, and leads is important to define a normal electrocardiogram. Rules for a normal electrocardiogram are in Table 1.

Table 1: Rules for a normal ECG

| S.No. | Which waveform | Rule description | Which leads |
|---|---|---|---|
| 1 | P wave | P wave is upright, and all P-waves are similar | I, II, V2-V6 |
| 2 | PR interval | PR interval should be 120 to 200 milliseconds or 3 to 5 little squares | All |
| 3 | QRS | The QRS complex should be dominantly upright | I, II |
| 4 | QRS | The width of the QRS complex should not exceed 110 ms, less than 3 little squares | All |
| 5 | QRS | The R wave in the precordial leads must grow (Progression of R waves) | V1 to V6 |
| 6 | QRS | There should be no Q wave or only a small q less than 0.04 seconds in width | I, II, V2 to V6 |
| 7 | QRS & T | QRS and T waves tend to have the same general direction in the limb leads | All Limb leads |
| 8 | ST segment | The ST segment should start isoelectric | All except V1/V2 |
| 9 | T | The T wave must be upright | I, II, V2 to V6 |
| 10 | P, QSR, T | All waves are negative in lead aVR | aVR |

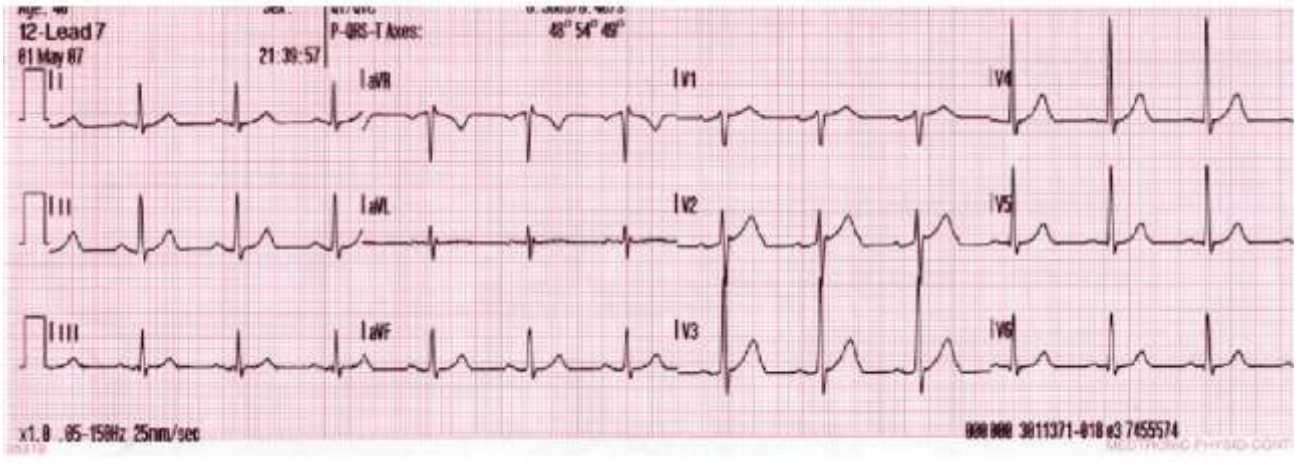



Figure 6: Please note features of a normal ECG. P wave is upright in all leads (except V1); PR interval (from beginning of P to beginning of QRS complex) is 4 small squares; QRS interval is narrow (3 small squares), dominantly upright, no Q-waves and R wave becomes progressively larger from V1 to V5; T wave is in same direction as QRS in all leads; ST segment is isoelectric; All waves are negative in aVR.

CHAPTER 5

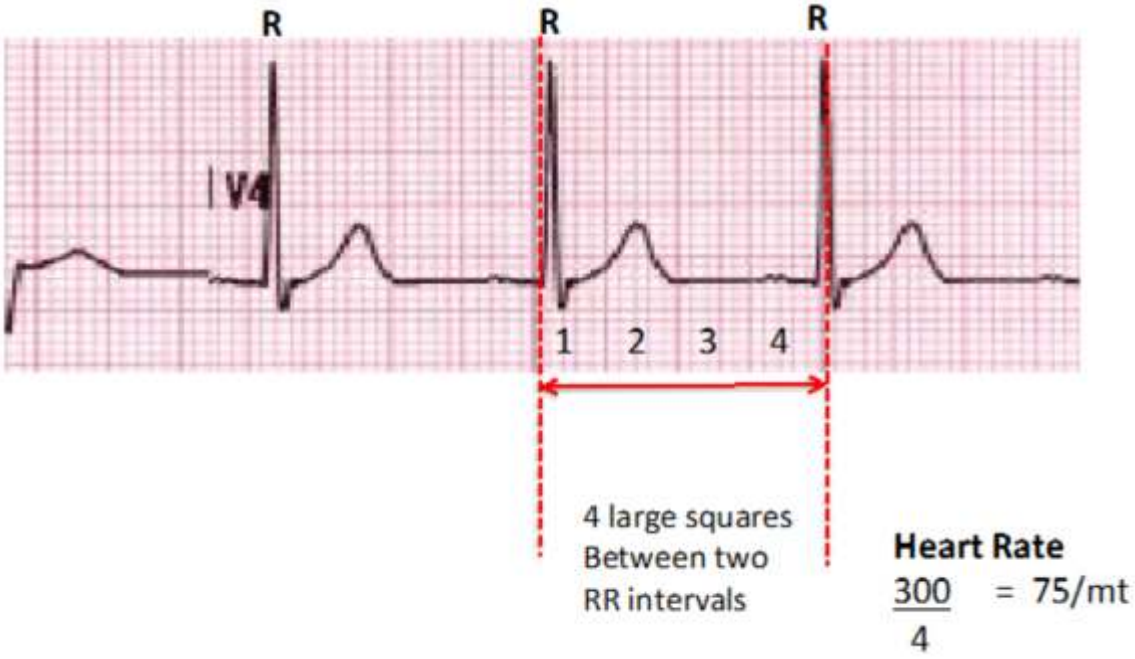
# HEART RATE AND RHYTHM

Heart rate is defined as number of cardiac contractions in a minute. Since we cannot see cardiac contractions directly, we can either measure pulse-rate or auscultatory heart-beat as indirect indicators of ventricular contractions. If we have ECG, we measure rate of generation of RR waves, as an indicator of heart rate. Under normal circumstances these rates are equal to each other. However if some ventricular contractions donot result in an aqequate outflow, pulse rate will be lower or if an electrical impulse does not lead to effective ventricular contraction, RR-rate will be higher than true heart rate. Hence "heart-rate" measured from ECG is better known as "RR-rate". Its calculation is shown in Figure 1.

Conduction system of the heart generates an impulse in a regular manner. This regular impulse generation is known as a rhythm. Normally the rhythm is generated by sinoatrial node (SA node), and this is known as a **sinus rhythm**. Sinus rhythm is generated at a rate of 60 to 100 per minute. While sinus impulse is not seen by itself on a surface ECG, we call a waveform complex (P-QRS-T) as sinus, if the following features are present:

a) P-wave is positive and preceeds QRS complex

If RR-waves are at a regular interval, then :
RR rate=Heart rate = 300/x (x=number of large squares between two R waves)




If RR-waves are at a irregular interval, then :
Count number of R waves in six seconds (30 large squares) and multiply by 10

Figure 1: Calculation of heart rate using ECG. Please note that when cardiac rhythm is recorded on a monitor, heart-rate is calculated and displayed by the monitor.

b) Resultant QRS complex is narrow (does not exceed 3 small squares), given that there is no conduction block distal to AV node.

Such sinus waveforms are generated at regular rate, and if this rate is between 60 to 100 per minute, it is known as a "Normal Sinus Rhythm." Similarly sinus bradycardia is rate of less than 60/min., and simus tachycardia is rate greater than 100 per minute. (Figure 2)

Normal sinus rhythm generates a normal regular pulse of the same rate. However if such an electrical activity does not generate any ventricular contractions and hence no pulse it is known as pulseless electrical activity (PEA).

Sometimes when sinus rhythm is not gerenerated, conducting tissue around AV node (or AV junction) or conduction tissue in the ventricles may assume a pacemaker role. These distal pacemakers normally remain inhibited. (Figure 2)

Rhythm generated by conduction system a round AV junction is known as **Junctional rhythm**. This rhythm is characterized on ECG by narrow QRS complex, and P-waves which are inverted, and appear after the QRS complex. Junctional rhythms with a higher rate are known as accelerated junctional rhythm (rate 60-100/mt) or junctional tachycardia (rate >100/mt). Rhythm generated by conduction tissue in the ventricles is known as **ventricular rhythm.** It has a broad QRS complex, there are no P-waves, and inherant rate is 20-40 per minute. Ventricular rhythm with enhanced rates are known as Accelerated ventricular rhythm (rate 40-100/mt) or Ventricular tachycardia (rate >100/mt). These are again described later.

There are situations when SA node is normal but AV node is partially or completely blocked. These situations are known as AV-blocks. (Figure 3)

a) **1st degree AV block** (PR interval is longer than 5 small squares)

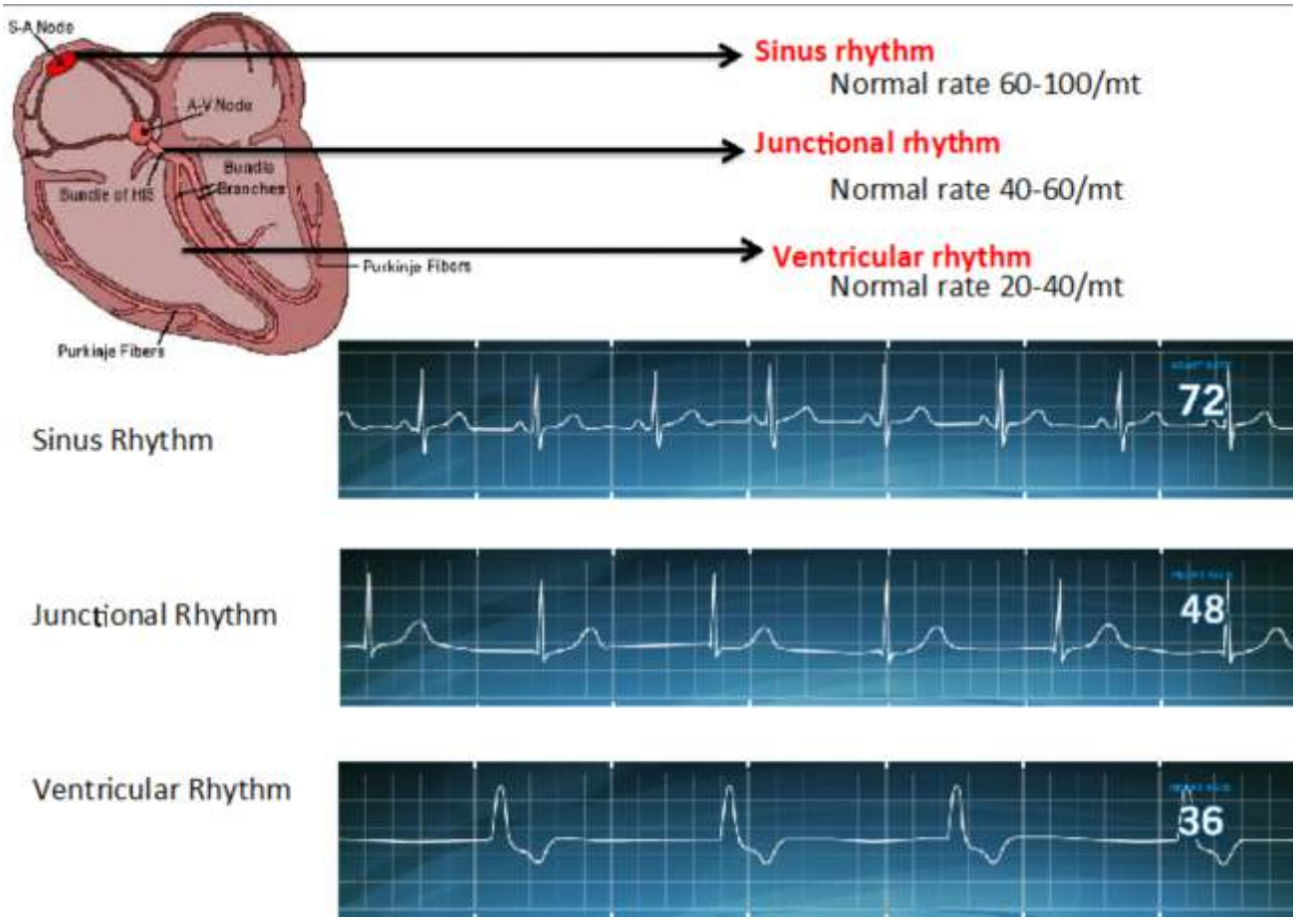



Figure 2 : Basic rhythm patterns

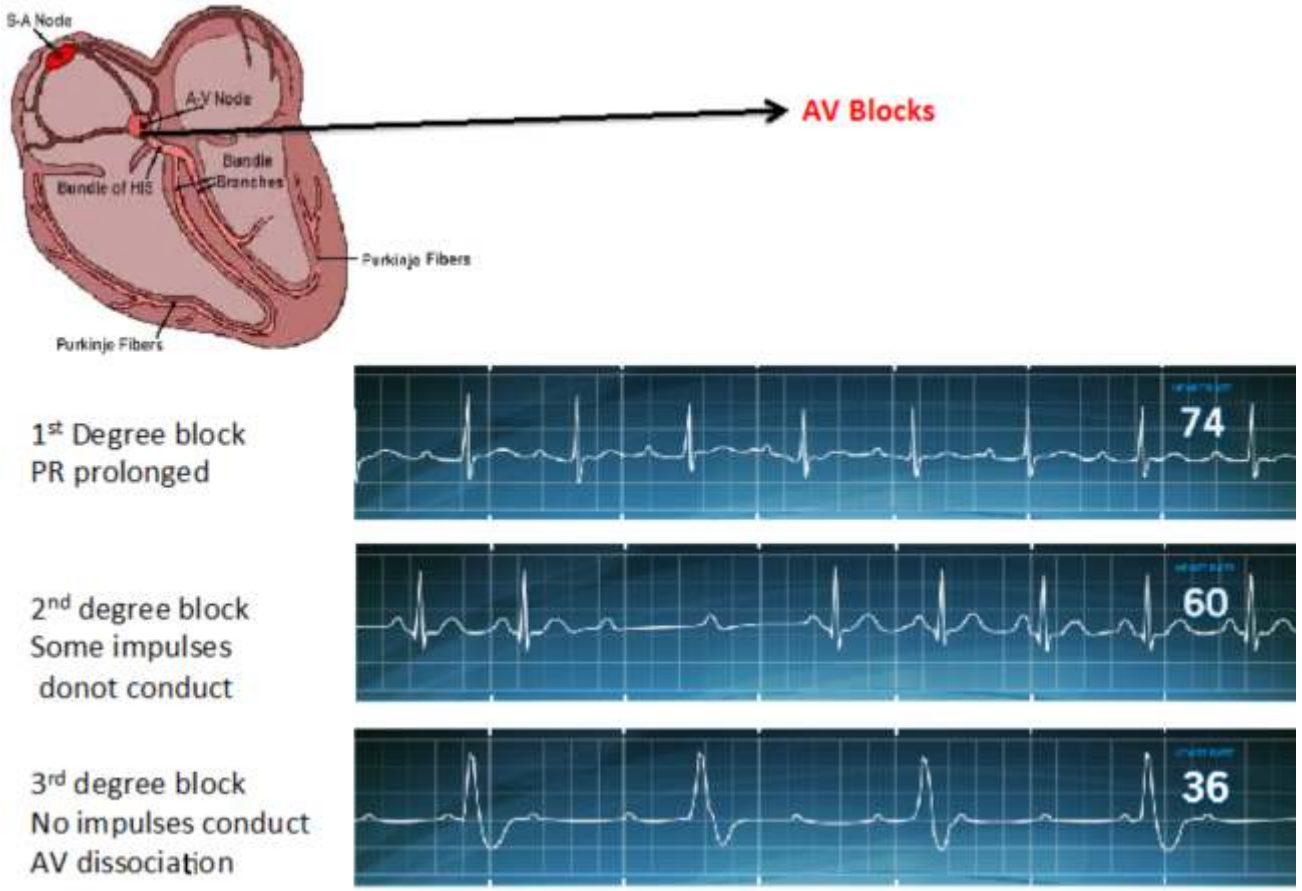



Figure 3: AV blocks

**b) 2nd degree AV block** (some P-waves donot conduct and hence donot lead to a QRS complex)

**c) 3rd degree AV block** (P waves are generated, and none of them conduct). Here we can see sinus rhythm and a ventricular rhythm in the same ECG. Sinus rhythm is evident by P-waves which are generating in a regular manner, and a Ventricular rhythm (generated by conduction system below AV node such as bundle of His) which is evident by broad QRS complexes. Sinus and ventricular rhythms are independent of each other (AV dissociation). RR-rate is regular, and so is heart rate and pulse rate, and these follow the ventricular rate (between 20-40 per minute).

Other supraventricular rhythms which are emergency situations include: (Figure 4)

**a) Atrial Flutter**

a. It is seen as multiple saw tooth like waves between two RR-complexes. These are flutter waves due to ectopic atrial contraction. While basic rhythm is sinus, multiple atrial contractions (at a atrial rate of 300/min or more)

b. Hall mark of atrial flutter is regular RR-interval, and multiple saw tooth like atrial waveforms.

**b) Atrial Fibrillation**

a. This is a situation where either small fibrillatory waves (or no waves) are seen between two RR-intervals. RR-rate is irregularly irregular (corresponding with irregularly irregular pulse). This is because of disturbance in atrial conduction.

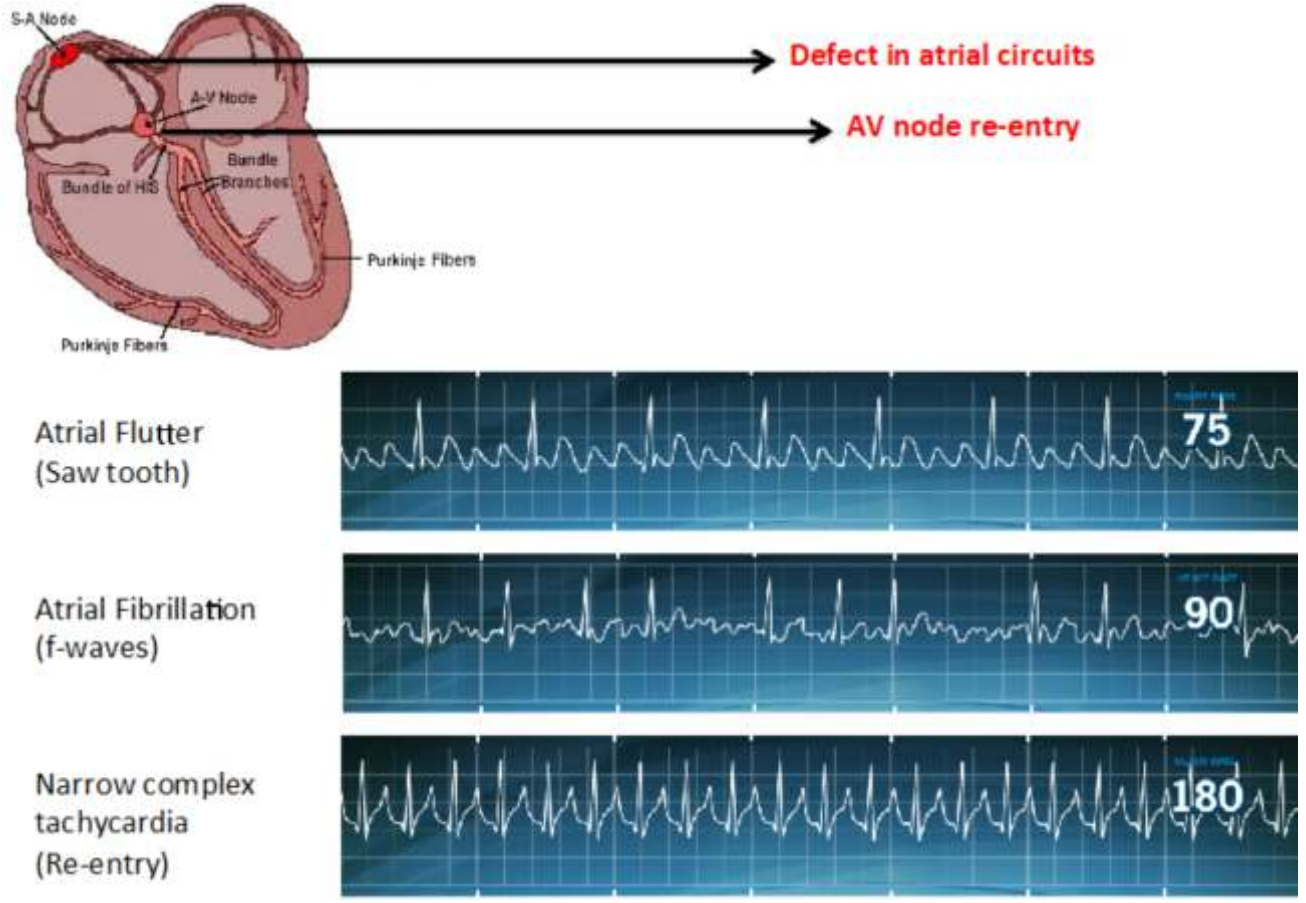



Figure 4 : Supra-ventricular rhythm disturbances.

a. Some QRS complexes are not able to achieve an effective ventricular contraction (as a new impulse may arrive when cardiac muscles in vntricles are refractory (not ready to contract). This leads to a pulse deficit.

b. Hallmark of Atrial fibrillation is irregular RR-interval, with no distinct P-waves.

**c) Narrow complex tachycardia**

a. This is a defect where either AV-node or tissue around AV-node has an abnormal re-entry pathway, so that multiple QRS complexes are generated after an initial sinus impulse.

b. This typically results in a narrow complex rhythm, with RR-rates which are greater than 150/minute. Usually these rates may exceed 180-200/minute.

c. This condition encompasses paroxysmal supraventricular tachycardia (PSVT), AV nodal re-entry tachycardia (AVNRT) or AV-re-entry tachycardia (AVRT).

The above rhythms are always with a pulse. Atrial flutter and narrow complex tachycardias are emergency rhythms. Atrial fibrillation in an unstable patient is an emergency rhythm.

**Ventricular rhythms** (Ventricular rhythm, accelerated ventricular rhythm, or ventricular tachycardia) may or may not generate a pulse. Ventricular tachycardia is an emergency rhythm, and since in this rhythm QRS complexes are broad, it is also known as broad complex tachycardia. This is an emergency rhythm (Figure 5&6).

Ventricular tachycardia with a pulse is managed using cardioversion. Ventriculartachycardia without pulse is managed with defibrillation.

Absence of a any identifable pattern in ECG is ventricular fibrillation, which is always without a

pulse. Ventricular fibrillation suggests some uncoordinated electrical activity in the heart, which is always insufficient to generate ventricular contractions.

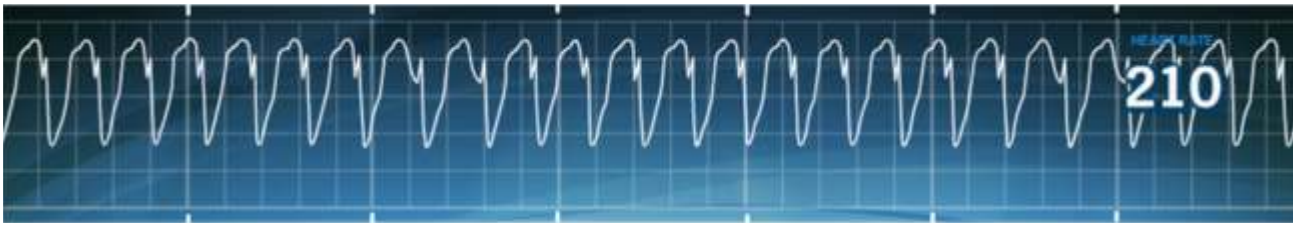



Figure 5 : Broad complex tachycardia (Ventricular tachycardia)

Figure 6 : Ventricular fibrillation

CHAPTER 6

# ADVANCED CARDIAC LIFE SUPPORT (ACLS) SURVEY

**Overview:**

- Healthcare provider should conduct the ACLS survey after completing the BLS survey.

For conscious patients who may need more advanced assessment and management techniques.

- Healthcare providers should conduct the ACLS survey.

An important component of this survey is the differential diagnosis, where identification and treatment of the underlying causes may be critical to patient to patient outcome.

In ACLS survey you keep on assess and perform an action as appropriate until transfer to the next level of care. Many times, team members perform assessments and actions in ACLS simultaneously.

**Remember: Assess..... Then perform appropriate actions.**

**Airway Management:**

When unresponsive patient is found first confirm whether patient is having respiratory arrest or cardiac arrest. The health care provider should conduct the ACLS survey after completing the BLS survey. An important component in ACLS survey is the differential diagnosis, where identification and treatment of underlying cause may be critical to patient outcome. Main dictum of this survey is First Assess and then perform appropriate action.

**Assessment of Airway during ACLS Survey:**

**TEAM CONCEPT OF CPR :** As we know "Together Everybody Achieve More (TEAM). This stand true during CPR also. Effective Resuscitation can be achieved by the good team Dynamics among team members. In CPR, healthcare providers need to do simultaneously a variety of interventions. Although a CPR trained bystander working alone can resuscitate a patient within the first moments after collapse, most attempts require the concerted efforts of multiple healthcare providers. Effective team work divide the tasks while multiplying the chances of a successful outcome.

**Learning Objective :** At the end of This part of manual you will be able to

- Describe team leader's and team member's roles
- Explain the importance of the team leader and team members understanding their specific roles.
- Describe how skill mastery combined with team dynamics may lead to increased success in resuscitation
- Coordinate team functions while ensuring continuous high quality CPR, defibrillation, and rhythm assessment.

**Basic Facts:**

Understanding team roles: Everybody needs to understands the role of team leader or team members. This awareness will help you anticipate.

- What action will be performed next
- How to communicate and work as a member or leader of the team.

Role of Team Leader: The role of the team leader is multifaceted. The team leader

- Organize the group
- Monitors individual performance of team members
- Backs up team members
- Models excellent team behavior
- Trains and coaches

Table 1 : Components of ACLS Survey

| Assess | Appropriate action |
|---|---|
| **Airway:**<br>- Is the airway patent.?<br>- Is an advanced airway is indicated.?<br>- Is proper placement of airway device confirmed?<br>- Is the airway device (endotracheal tube or LMA) secured and placement reconfirmed frequently?. | · **Maintain Air way Patency in unconscious patient by use of head tilt chin lift, oropharyngeal airway** (OPA) or Nasopharyngeal airway (NPA)<br>· **Use advanced airway management if needed (e.g** laryngeal mask airway, laryngeal tube esophageal-tracheal tube, endotracheal tube.)<br>If using advanced airway devices:<br>· **Confirm proper integration of CPR and ventilation.**<br>· **Confirm proper placement of advance airway devices by** |
| | - physical examination<br>- Quantitative wave form Capnography.<br>· **Secure the device to prevent dislodgement..**<br>· **Monitor airway placement with continuous quantitative wave form Capnography.** |
| **Breathing**<br>- **Are ventilation and oxygenation adequate?**<br>- **Are quantitative waveform capnography and oxyhemoglobin saturation moitored.** | · **Give supplementary oxygen when indicated**<br>-For cardiac arrest victim, administer 100% oxygen<br>-For others, titrate oxygen administration to achieve oxygen saturation value of ≥ 94% by pulse oximetry.<br>· **Monitor the adequacy of ventilation and oxygenation by**<br>-Clinical criteria ( chest rise and cyanosis)<br>-Quantitative waveform Capnography<br>-Oxygen saturation<br>· **Avoid excessive ventilation.** |
| **Circulations:**<br>-Are chest compression adequate<br>-what is cardiac rhythm<br>-is defibrillation or cardiac version<br>-Has IV/IO access been established<br>-Is the patient with a pulse unstable.<br>-Are medication needed for rhythm or BP<br>-Does the patient need volume( fluid) resuscitation. | · **Monitor CPR Quality.**<br>- **Quantitative wave capnography (** if PETCO2 >10mmHg. Attempt to improve quality of CPR**)**<br>- **Intra-arterial pressure (**if relaxation phase-diastolic pressure is <20mmHg. Attempt to improve CPR<br>· **Attach monitor /defibrillation/cardiac version for arrhythmias or cardiac arrest rhythms. (**e.g VF,Pulseless VT, Asystole, PEA)<br>· **Provide defibrillation or cardiac version**<br>· **Obtain IV/IO Access**<br>· **Give Appropriate drugs** to manage rhythm and blood pressure.<br>· **Give IV/IO fluids** |
| **Differential Diagnosis**<br>- Why did this patient develop arrest<br>- Is there any cause which can be reversed by the treatment. | **5 Remind youself about 5 H, and 5 Ts. And go for definitive care.** |

- Facilitates understanding
- Focuses on comprehensive patient care.

Team leader should be able to explain why it is essential to

- Push hard and fast
- Ensure complete chest recoil
- Minimize interruption in chest compressions
- Avoid excessive ventilations.

**Role of Team members :** Team members must be proficient In performing the skills authorized by their scope of practice. It is essential to the process of the resuscitation attempt that team members are.

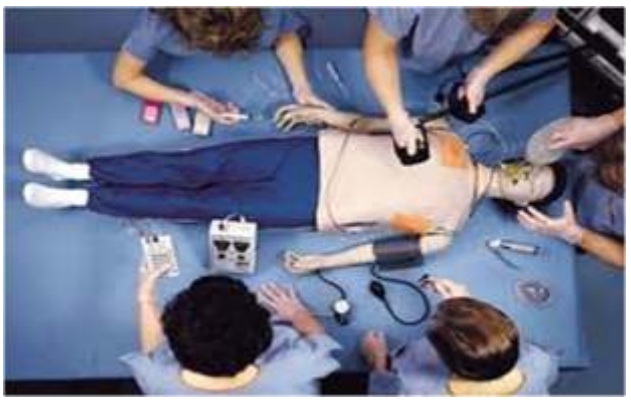
- Clear about role assignments
- Prepared to fulfill their role responsibilities
- Well practiced in resuscitation skills.
- Knowledgeable about the algorithm
- Committed to success.

**Components of efficient Resuscitation Team Dynamics:**

**1. Closed-Loop Communications:**

When communicating with resuscitation team members, the team leader should use closed loop communication

- The team leader gives a message, order or assignment to team member.
- By receiving a clear response and eye contact, the team leader confirms that the team members heard and understood the message.



- The team leader listens for confirmation of task performance from the team member before assigning another task.

**2. Clear Messages :** Whenever members are working together, messages should be clear and loud . One should always confirm that messages have been received.

**3. Clear Roles and Responsibilities :** Every-member of the team should know his or her role ad responsibilities. Each team member's role is unique and critical to the effective performance of the team. There should be at least 6 members for team efficiency . If members are less than 6 than, then all role must be assigned to the present team members.

When roles are unclear, team performance suffers. Signs of unclear roles includes.

- Performing the same task more than once
- Missing essential tasks
- Free lancing of team members

**4. Knowing Once's Limitation :** Only members who are well trained in a particular skill should be assigned the same skill task and during CPR no new skill task should not be given to a team members.

**5. Knowledge Sharing :** Sharing information is a critical component of effective team performance. Everymember has to participate actively in contributing the information in order to delineate the diagnosis of condition which may have led to cardiac arrest. Examples of three common types of fixation errors are

- "Everything is Okay"
- "This and only this is the correct path".
- "Anything but this"

When resuscitative measures are ineffective, go back to the basics and talk as a team members " well we have gotten the following on ACLS survey …. Have we missed something?

**6. Constructive intervention :** During CPR team leader or team member may need to intervene if an action that is about to occur may be inappropriate at that time. Although constructive intervention is necessary, it should be tactful. Team leader should

avoid confrontation with team members. Instead, conduct a debriefing afterward, if constructive criticism is needed.

**7. Reevaluation and summarizing :** An essential role of leader is monitoring and reevaluating

- The patient status
- Interventions that have been performed
- Assessment finding

A good habit of team leader is to summarize the information out loud in a periodic update to team. Review the status of the resuscitation attempt and announce the plan for next}\step. Rem\ember patient's condition can change with time.

**8. Mutual Respect :** Best teams are composed of members who share a mutual respect for each other and work together in a collegial, supportive manner. To have high performing team, everyone must abandon ego and respect each other during the resuscitation attempt.

**Systemic Approach :** Successful resuscitation following cardiac arrest requires an integrated set of coordinated action represented by the links in the Adult Chain of Survival. The links

Include:

- Immediate recognition of cardiac arrest and activation of the emergency response system.
- Early CPR with an good quality of chest compressions.
- Early defibrillation
- Effective advance life support
- Integrated post cardiac arrest care.

Effective resuscitation requires an integrated response known as a system of care. Fundamental to a successful resuscitation system of car is the collective appreciation of the Challenges and opportunities presented by the Chain of Survival. (Fig.)

To improve care, leaders must assess the performance of each system component. This process of quality improvement consists of an iterative and continuous cycle of

- Systematic evaluation of resuscitation care and outcomes .
- Benchmarking with stakeholder feedback.
- Strategic efforts to address identified deficiencies.

CHAPTER 7

# MANAGEMENT OF RESPIRATORY ARREST

Management of respiratory arrest includes both BLS and ACLS intervention. These interventions may include.

I) Giving supplementary oxygen

II) Opening the airway

III) Providing basic ventilation

IV) Using basic airway adjuncts ( OPA and NPA)

V) Suctioning

VI) Ventilating the patient with advance airway

**According to AHA 2010 for patient with perfusing rhythm i.e. only respiratory arrest, deliver 1 breath every 5-6 seconds ( 10-12 breaths / minute).**

**Critical Point:**

Avoiding Excessive Ventilation: When using any form of assisted ventilation, one should avoid providing too many breaths/min or too large a volume per breath. Excessive ventilation can be harmful because it increases intra-thoracic pressure, decreases venous return to the heart, and decrease cardiac output. It may also cause gastric inflation and predisposes the patient to vomiting and aspiration of gastric contents.

**I) Giving Supplementary Oxygen :** Give oxygen to patients with acute cardiac symptoms or respiratory diseases. Monitor oxygen saturation and titrate supplementary oxygen to maintain a saturation of ≥ 94%.

**II) Opening Airway:**

**Common causes of Airway Obstruction :** The most common cause of upper airway obstruction in the unconscious/unresponsive patient is loss of tone of throat muscles. In this case the tongue falls back and occludes the airway at the level of the pharynx.

Basic Airway opening Techniques: Basic techniques will effectively relieve airway obstruction caused either by the tongue or from relaxation of muscles in the upper airway. The basic airway opening technique is head tilt with anterior displacement of the mandible i.e head tilt-chin lift.

In trauma patient with suspected neck injury, use a jaw thrust manouvere.

Airway Management: Proper airway positioning may be all that is required for patients who can breathe spontaneously. A patient who has choked, known to be choking and is now unresponsive and in respiratory arrest, open the mouth wide and look for a foreign object. Each time you open the airway to give breaths, open the mouth wide and look for a foreign object. Remove it with your fingers if present. If there is no foreign object resume CPR.

**III) Providing Basic Ventilation:**

**Basic airway skills used to ventilate a patient are**

- Head tilt- chin lift
- Jaw thrust without head extension (suspected cervical spine trauma)
- Mouth to mouth ventilation
- Mouth to barrier device (using a pocket mask) ventilation
- Bag and mask ventilation

Bag and Mask: A bag and mask device consists of a ventilation bag attached to a face mask. These devices have been a mainstay of emergency ventilation decades. Bag-mask devices are the most common method of providing positive pressure ventilation. When using a bag mask device, these devises deliver approximately 600 ml of tidal volume which is sufficient to produce chest rise over 1 second.

Figure 1. Showing how oro-pharyngeal airway accommodates in oral cavity.

## IV) Basic Airway Adjuncts:

**Oro-pharyngeal Airway (OPA) :** The OPA is used in patient who are at risk for developing airway obstruction from tongue or from relaxed upper airway muscles. This J shaped device fits over the tongue to hold it and the soft hypo pharyngeal structures away from the posterior wall of pharynx.

The OPA is used in unconscious patients if procedures to open the airway (head tilt-chin lift or jaw thrust) fail to provide and maintain a clear, unobstructed airway. AN OPA should not be used in a conscious or semiconscious patient because it may stimulate gagging and vomiting. The key assessment is to check whether the patient has an intact cough and gag reflex. If so, do not use an OPA.

OPA is also used during suctioning of the mouth and throat, in an intubated patient to prevent them from biting and occluding the ET tube.

**Technique of OPA insertion :**

- Clear the mouth and pharynx of secretions, blood or vomit using a rigid pharyngeal suction tip if possible.
- Select the proper size OPA: Place the OPA against the side of face. When the tips of OPA is at the corner of mouth and the flange is at the angle of the mandible or tragus. A properly sized and inserted OPA results in proper alignment with glottis opening (Figure 2).
- Insert the OPA so that so that it curves upward toward the hard plate as it enters the mouth.
- As the OPA passes through the oral cavity and approaches the posterior wall of the pharynx, rotate it $180^0$ into the proper position. The OPA can also be inserted at a $90^0$ angle to mouth and then turned down toward the posterior pharynx as it is advanced. In both methods, the goal is to curve the device around the tongue so that the tongue is not inadvertently pushed back in to the pharynx rather than being pulled forward by the OPA.

An alternative method is to insert the OPA straight in while using a tongue depressor or similar device to hold the tongue forward as the OPA is advanced.

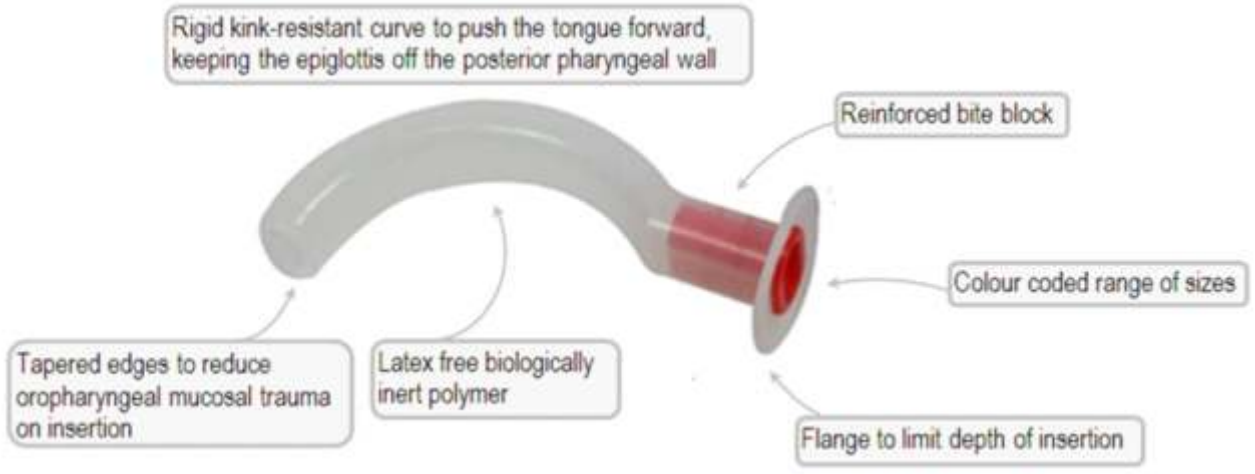



Figure 2. Despcription of Oro-pharyngeal Airway

**Caution :**

- OPAs that are too large may obstruct the larynx or cause trauma to the laryngeal structures.
- OPAs are too small or inserted improperly may push the base of the tongue posteriorly and obstruct the airway.
- Insert the OPA carefully to avoid soft tissue trauma to the lips and tongue.

**Nasopharyngeal Airway:**

The NPA is used as an alternative to OPA in patients who need a basic airway management adjunct. The NPA is a soft rubber or plastic uncuffed tube that provides a conduit for airflow between the nares and the pharynx (See Figure 3).

Unlike oral airways, NPAs may be used in conscious or semiconscious patients ( patients with an intact cough and gag reflex.) The NPA is indicated when insertion of an OPA is technically difficult or dangerous such as in patients with gag reflex, trismus, massive trauma around the mouth or wiring of the jaws. The NPA may also be used in the patients who are neurologically impaired with poor pharyngeal tone leading to upper airway obstruction.

**Technique of NPA Insertion:**

**Select the proper size NPA.**

- Compare the outer circumference of the NPA with the inner aperture of the nares. The NPA should not be so large that it causes sustained blanching of the nostrils. Some providers use the diameter of the patient's smallest finger as a guide to selecting the proper size.
- The length of the NPA should be the same as the distance from the tip of the patient's nose to the earlobe.
- Lubricate the airway with a water soluble lubricant or anesthetic jelly.
- The NPA is inserted though nostril in a posterior direction perpendicular to the plane of the face. Pass it gently along the floor of the nasopharynx.

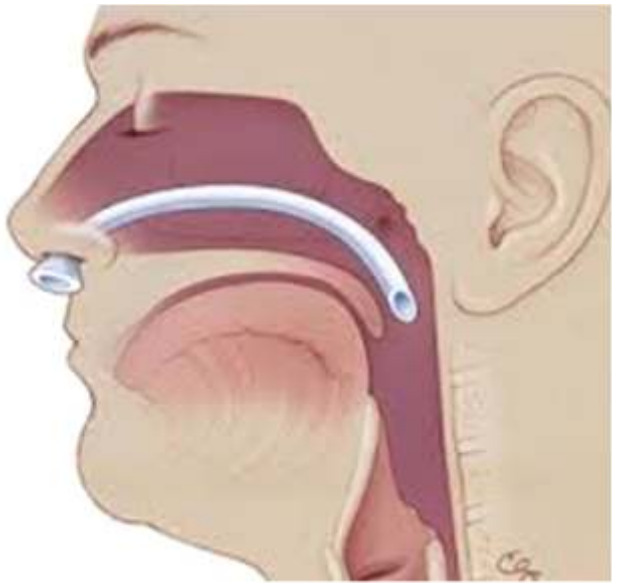


Figure 3. : Showing how do Nasopharyngeal Airway accommodates in naso-oropharynx.

If you encounter resistance :

-Slightly rotate the tube to facilitate insertion at the angle of the nasal passage and naso-pharynx.

- Attempt placement through the other nostril because patients have different sized nasal passages.

Mucus, blood, vomit, or the soft tissues of the pharynx can obstruct the NPA, which has as a small internal diameter.

**Cautions:**

- Gently insert the airway avoiding complication. The airway can irritate the mucosa or lacerate adenoidal tissue and cause bleeding, with possible aspiration of clots into the trachea. Suction may be necessary to remove blood or secretions.
- An improperly sized NPA may enter the esophagus, with active ventilation, such as bag-mask ventilation, the NPA may cause gastric inflation.

- An NPA may cause laryngospasm and vomiting even though it is commonly tolerated by semiconscious patients.
- To be used with caution in patients with facial trauma because of the risk of misplacement into the cranial cavity through a fractured cribriform plate.

## V) Suctioning:

- Suctioning is an essential component of maintaining a patent airway. Providers should suction the airway immediately if there are copious secretions, blood or vomit.

Suction devices consist of both portable and wall mounted units.

- Portable suction devices are easy to transport but may not provide adequate suction power, A suction force of – 80 to – 120 mmHg is generally necessary.
- Wall mounted suction units should be capable of providing an airflow of > 40 L/Min at the end of the delivery tube and a vacuum of more than – 300 mmHg when the tube is clamped at full suction.
- Adjust the amount of suction force for use in children and in intubated patients.

**Soft vs Rigid Catheters.**

Both soft flexible and rigid flexible catheters are available.

**Flexible** catheters may be used for suctioning of mouth or nose. Soft flexible catheters are available in sterile wrappers and can also be used for ET tube deep suctioning. **Rigid Catheters** (e.g Yankauer) are used to suction the oropharynx. These are better for suctioning thick secretions and particulate matter.

**Rigid Catheters :** More effective suctioning of the oropharynx, particularly if there is thick particulate matter.

**Oropharyngeal Suctioning Procedure:**

Follow the steps below to perform oro-pharyngeal suctioning.

- Measure the catheter before suctioning and donot insert it any further than the distance form the tip of the nose to the earlobe.
- Gently insert the suction catheter or device into the oropharynx beyond the tongue.
- Apply suction by occluding the side opening of the catheter while withdrawing with a rotating or twisting motion.
- If using a rigid suction device place the tip gently into the oral cavity. Advance by pushing the tongue down to reach the oropharynx if necessary.

**Endotracheal tube suctioning procedure:**

- Use sterile technique to reduce the likelihood of airway contamination.
- Gently insert the catheter into the ET tube. Be sure the side opening is not occluded during insertion.
- Insertion of the catheter beyond the tip of the ET tube is not recommended because it may injure the endotracheal mucosa or stimulate coughing or bronchospasm.
- Apply suction by occluding the side opening only while withdrawing the catheter with a rotating or twisting motion.
- Suction attempts should not exceed 10 seconds. To avoid hypoxemia precede and follow suctioning attempts with a short period of administration of 100 % oxygen.

Monitor the patient's heart rate, pulse, oxygen saturation and clinical appearance during suctioning. If bradycardia develops, oxygen saturation drops or clinical appearance deteriorates, interrupts at once. Administer high flow oxygen until the heart rate return to normal and the clinical condition improves. Assist ventilation as and when needed.

## VI) Ventilating a patient with an Advanced Airway:

**Introduction :** This part of manual will familiarize you about advance airway device, scope of practice and equipment of the providers on the resuscitation

team. Advanced airway include

- Endotracheal Tube
- Laryngeal Airway Tube
- Laryngeal Tube
- Esophageal-Tracheal Combi Tube

**Ventilation Rates:**

**1. Endotracheal (ET) Tube : Following are the steps to be done for intubation**

- Prepare for intubation by assembling the necessary equipment
- Perform endotracheal intubation
- Inflate cuff
- Attach it to oxygen enriched device which is AMBU bag.
- Confirm correct placement by physical examination. The best way of confirmation is continuous wave form capnography. It is recommended as the most reliable method of confirming and monitoring correct placement of ET tube.
- Healthcare providers may also use calorimetric and non waveform carbon dioxide detector when waveform capnography is not available.
- Secure the tube in place.
- Monitor for dislodgement.

Only experience providers should perform endotracheal intubation.

**Role Cricoid pressure :** Cricoid pressure in non-arrest patient may give some measure of protection to the airway from aspiration and gastric insufflation during bag-mask ventilation. However, this may also impede ventilation. This may interfere with placement of a supra-glottic airway or intubation.

**Guidelines of Cricoid Pressure :** The role of cricoid pressure during out of hospital or in hospital cardiac arrest, has not been studied. If cricoid pressure is used in a few special circumstances during cardiac arrest the pressure should be adjusted, relaxed or released if it impedes ventilation or advanced airway placement. The routine use of cricoid pressure in cardiac arrest is not recommended.

**2. Laryngeal Mask Airway ( LMA) :** LMA is an advanced airway alternative to endotracheal intubation and provides comparable ventilation. This is specially meant for paramedics. This should be used by a experienced paramedical.

**3. Laryngeal Tube :** The laryngeal tube is more compact and less complicated to insert. The advantage of this tube is same as with esophageal-tracheal or LMA. Healthcare provider trained in the use of laryngeal tube may also consider it as an alternative to bag-mask ventilation.

**4. Esophageal-Tracheal Tube (Combi-Tube) :** The esophageal tracheal tube is an advanced airway alternative to ET tube. This device provide adequate ventilation comparable to an ET tube. It is acceptable to use the esophageal-tracheal tube as an alternative to an ET tube for airway management.

**Rescue breathing with advanced airway :**

-During CPR the compression to Ventilation ratio 30:2. But once an advanced airway is in place, chest compressions are no longer interrupted for ventilations.

-When ventilating through a properly placed advanced airway give 1 breath every 6-8 seconds ( app. 8-10 breaths/min) without trying to synchronize breaths to compressions. Ideally deliver breath during chest recoil between compressions.

In patient who has a pulse and compressions are not indicated. Give 1 breath every 5 to 6 seconds ( 10-12 breaths/min.)

Table 2 : Airway Devices and frequency of ventilation

| Device for Airway | Ventilations during cardiac arrest | Ventilation during respiratory Arrest |
|---|---|---|
| Bag-mask | 30:2 | 1 ventilation every 5-6 seconds |
| Advanced airway | 1 Ventilation every 6-8 seconds ( 8-10 / min) | (10-12 breaths/min) |

CHAPTER 8

# RECOGNITION AND MANAGEMENT OF ACUTE CORONARY SYNDROMES

Acute coronary syndromes are group of disorders, commonly identified as "Heart-attack." This is an emergency situation, which needs to be identified and acted upon. We will first understand how acute coronary syndromes are identified, and what to do when they are recognized.

## What is an Acute Coronary Syndrome (ACS)

Muscles of heart receive their blood supply through coronary arteries. Heart Attacks are caused by Coronary Artery Disease (CAD) which causes blockage of the blood supply to the Heart.

Acute coronary syndrome (ACS) is a term used for any condition brought on by sudden, reduced blood flow to the heart. Patient with coronary syndromes present with spectrum of clinical syndromes representing varying degrees of coronary artery occlusion. These syndromes include Unstable angina (UA), Non ST elevation myocardial infarction (NSTEMI), and ST elevation myocardial infarction (STEMI). Sudden cardiac death may occur with each of these syndromes.

It is possible to recognize warning symptoms of ACS early and prevent myocardial infarction. It is also very important to recognize myocardial infarction as early as possible, and save lives. Myocardial infarction and heart attack is the same disease. Up to half the patients who have a heart attack die in the early stage. It is therefore important to correctly diagnose the risk of Heart Attack (Myocardial infarction) as soon as possible. It is important to recognize ACS early, so that we can focus on rapid referral once these conditions are recognized. Simple treatment and rapid referral to specialist will save lives.

Three pillars for diagnosis of acute coronary syndrome (ACS) are:

a) Symptoms (Angina)

b) Electrocardiography (ECG)

c) Cardiac enzymes (Troponin T, or CPK-MB)

## Symptoms:

The most common symptoms of myocardial ischemia & infarction is retrosternal chest discomfort. The patient may perceive this discomfort more as pressure or tightness than actual pain.

Symptoms suggestive of ACS may also include:

1. Uncomfortable pressure, fullness, squeezing, or pain in the center of the chest lasting several minutes ( Usually more than a few minutes).
2. Chest discomfort spreading to the shoulders, neck, one or both arms, or jaw.
3. Chest discomfort spreading into the back or between the shoulder blades
4. Chest discomfort with light headedness, dizziness, fainting, sweating, nausea or vomiting.
5. Unexplained, sudden, shortness of breath, which may occur with or without chest discomfort.

Any acute onset chest discomfort should be taken seriously. Above description of chest pain is known as angina, (central / precordial chest discomfort, a severe crushing, pressing, or tightness like pain which may radiate to shoulders, arms, and jaw.), which becomes more severe with increasing physical activity and is relieved with rest. When someone has myocardial infarction, anginal pain occurs at rest.

Some patients with acute coronary syndrome, may have an atypical pain, such as burning, gaseous distention, or breathlessness. While chest pain has various other etiologies (such as gestro-esophageal reflux, gastritis, peptic ulcer disease, pleural

disesaes etc) but cardiac origin of any chest pain should be ruled out, especially in individuals who are at risk for acute coronary syndrome (such as those with hypertension, diabetes mellitus, smokers, a previous stroke or a heart attack).

Every person with an acute onset chest discomfort, or with angina must be evaluated for Acute coronary syndrome. ECG must be obtained in this situation.

**Electrocardiography :** It is an important tool to identify if chest pain is due to Acute Coronary Syndrome (ACS) or due to any other etiology. We must obtain a resting ECG in all cases. To evaluate if ECG is indicative of ischemic changes, consistent with acute coronary syndrome, we must evaluate the ST-segment to identify if it is normal, elevated or depressed.

Normally ST segment (between QRS complex and T wave) is an isoelectric line, at same level as PR or TP intervals. If it is elevated (greater than 1 millimeter elevated in the limb leads or 2 mm elevated in the chest leads we infer that ST-segment is elevated.

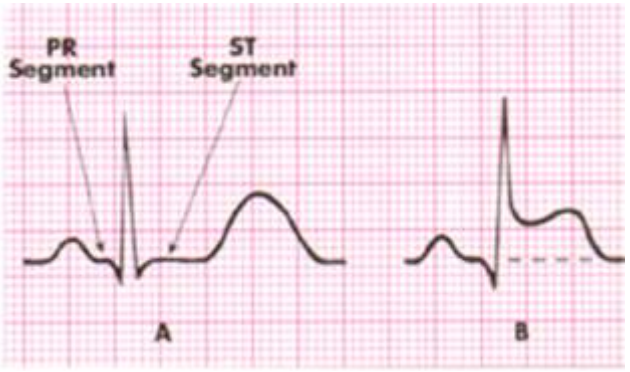



Figure 1: (A) Normal (B) ST elevation

This elevation should be in two or more contigious leads. (See figure 1)

**In presence of an acute chest pain, elevation of ST segment signifies ST-elevation myocardial infarction (STEMI)**

ST-elevation in inferior leads (II, III, aVF) suggests an

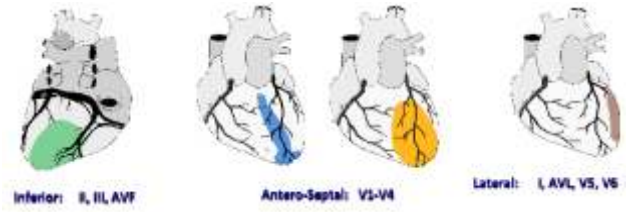



Figure 2 : Localization of myocardial infarction

Inferior wall STEMI, usually due to occlusion right coronary artery. ST-elevation in V1-V4 suggests Anterior wall STEMI, usually due to left anterior descending coronary artery, and in V5-V6,I aVL suggests Lateral wall STEMI usually due to left circumflex coronary artery.

If ST-segment is elevated then the patient is likely to be experiencing inadequate blood supply to the heart and in most cases must receive urgent treatment, which is detailed in the subsequent section.

If ST-segment is not elevated, we need to evaluate if it is depressed. ST-depression greater than 1 millimeter in the limb leads or 2 mm in the chest leads is considred as significant. In addition to ST-depression, other ischemic changes in ECG are:

a) T wave inversion

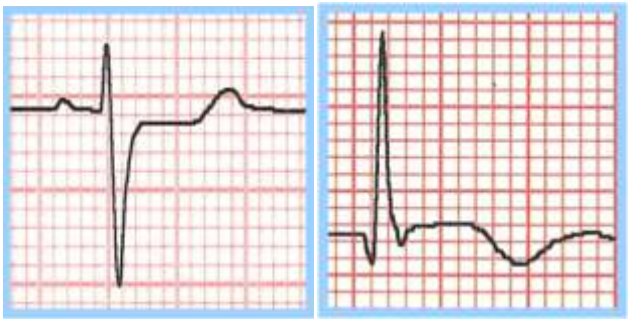


Figure 3: ST depression and T-inversion

b) Q waves (At least 0.04 seconds in duration or more than 25% deep as compared to subsequent R wave)

c) A new onset rhythm disturbance (See chapter 6) or Left Bundle Branch Block.

Presence of ischemic changes on ECG other than ST segment elevation may suggest either a Non-ST

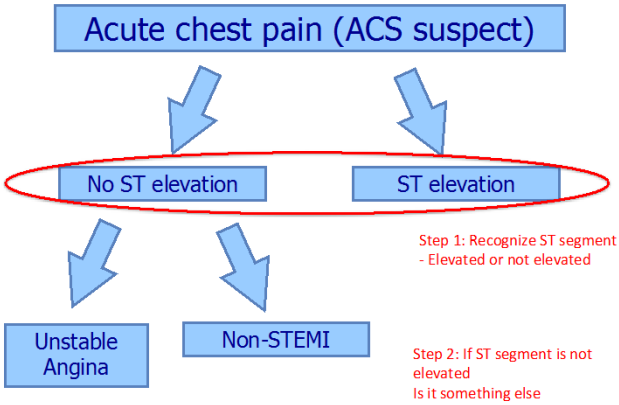



Figure 4: Approach to acute achest pain, after ECG is obtained.

elevation myocardial infarction (NSTEMI) or nstable angina (UA).

Even if ECG is normal, following patients are at high risk for ACS. They must be evaluated with serial ECGs obtained after 30 minutes, or when pain recurs.

a. Patients who take tablets to treat blood pressure, heart disease, angina, heart attack, diabetes, blood clots or who have undergone treadmill test, ECG or Coronary Angiography previously.

b. Patients with elevated blood pressure, or blood sugar at the time of presentation or those who are overweight or obese.

c. Patient with a family history of heart attack, angina or heart disease?

d. Patients who smoke or chew tobacco.

Serial ECGs represent dynamic changes which if present are highly suggestive of acute coronary syndomes.

**Cardiac enzymes :** Positive cardiac enzymes (Troponin T or CPK-MB) are specific for diagnosis of acute coronary syndrome (STEMI or NSTEMI). Cardiac enzymes are negative in unstable angina.

Cardiac enzymes become detectable only 4-6 hours after onset of chest pain. Hence during acute chest pain, symptoms and ECG are me mainstay for diagnosis.

By the time Troponin T or CPK-MB become positive, myocardial muscle has already moved to the stage of necrosis. Please see some example ECGs during acute chest pain in following pages:

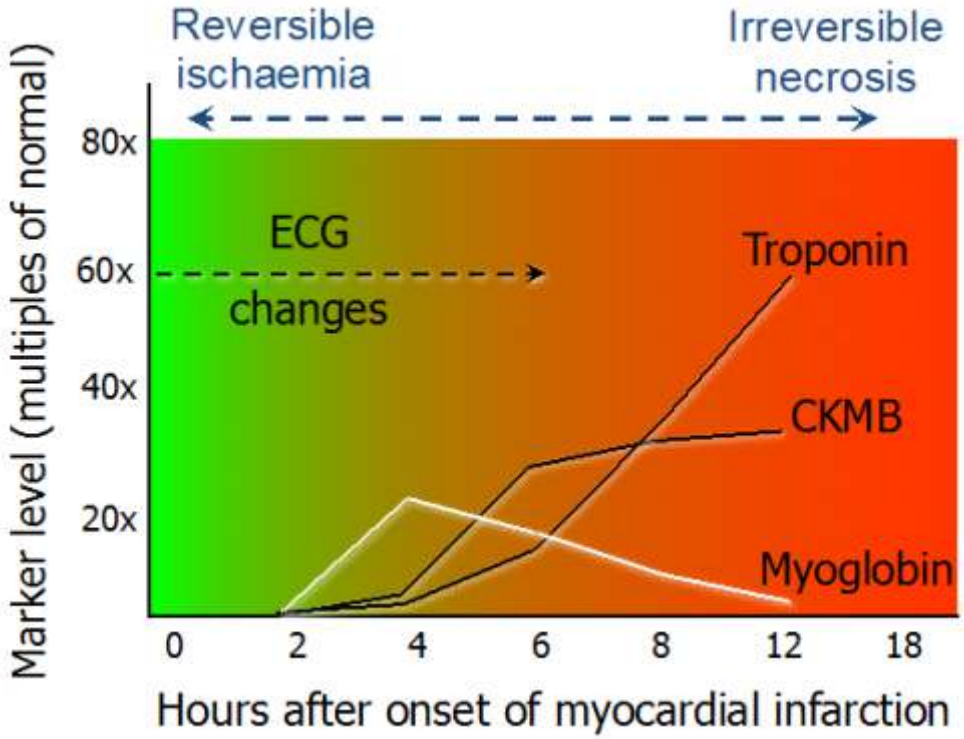
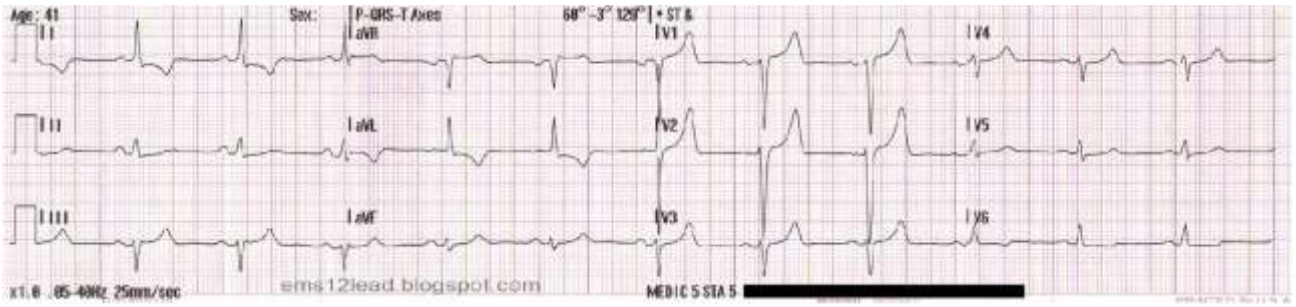


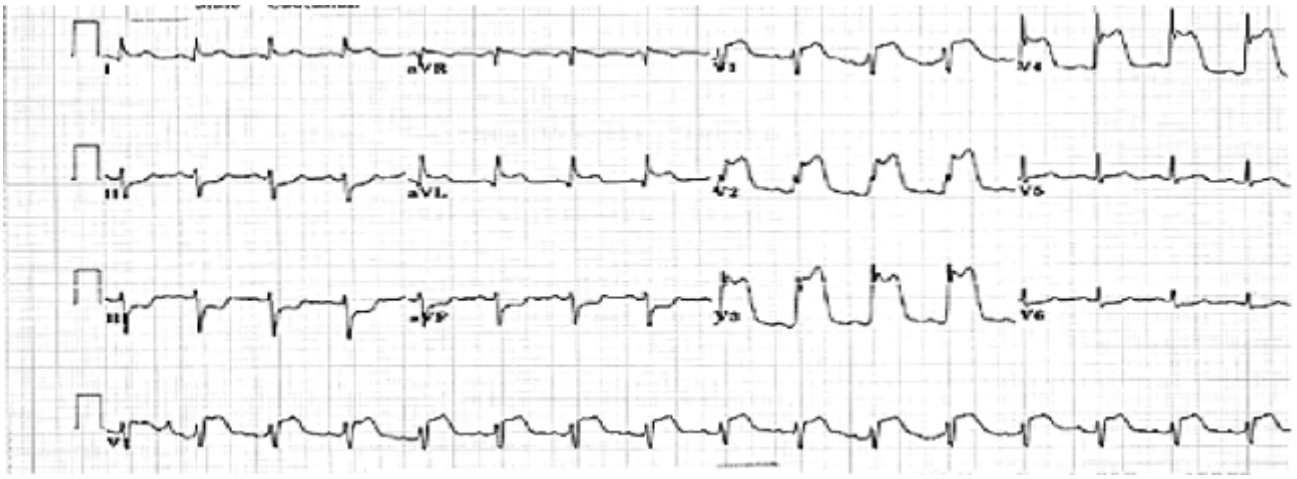

Figure 5 : Levels of cardiac enzyme and its association with MI

ECG Quiz please interpret these ECGs

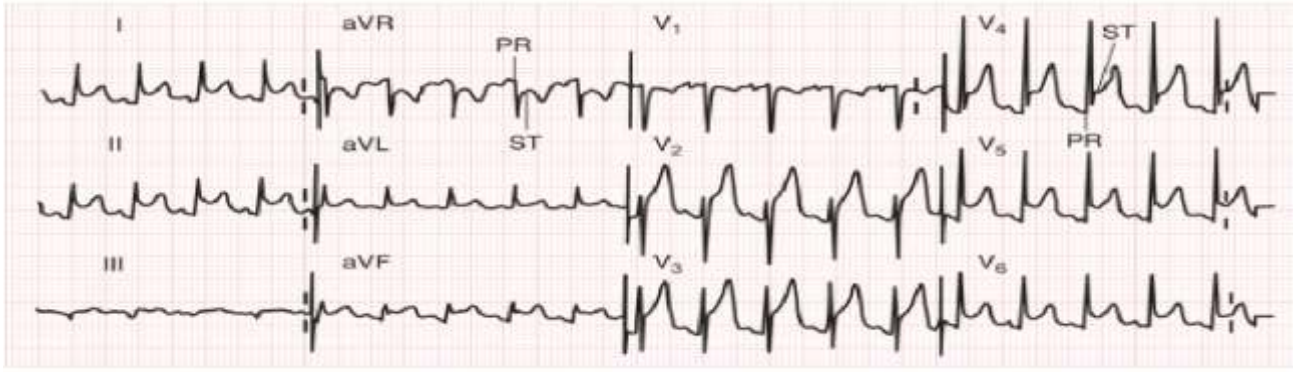
ECG : A



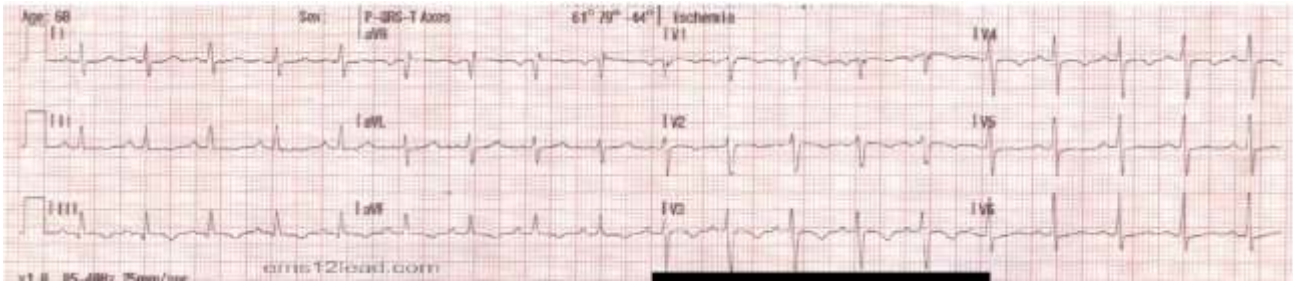

ECG : B



ECG : C




ECG : D




Answer on Page 48

**Management of ACS starts early, at the time of initial recognition.**

**ACS should be treated early so that:**

- We can prevent death
- We can prevent or reduce the amount of myocardial necrosis that occurs in patients with acute myocardial infarction (AMI and preserve left ventricular (LV) function
- We can prevent life-threatening complications of ACS, such as ventricular fibrillation (VF), pulseless ventricular tachycardia (VT), unstable tachycardias, symptomatic bradycardias, pulmonary edema, or cardiogenic shock and mechanical complications of AMI

**Management of suspected ACS, when ECG is not available (Pre-hospital care)**

In situations where ACS is suspected because of typical symptoms, or person is at a high risk for ACS and symptoms are typical or atypical we need to:

a) Arrange for prompt transportation where diagnosis and treatment can happen

b) Tab Aspirin 300mg to chew stat, to be given as an antiplatelet drug

c) Tab Isosorbide dinitrate (Sorbitrate) 10mg sublingual, as a vasodialator to relieve pain, if patient is not in hypotension.

d) If available, give oxygenation during transport.

Prompt transportation is the key so that if this is STEMI, revascularization (thrombolyisis or percutaneous interventions) is the key to survival and prevention of complications.

Do not give drugs like steroids or derriphyllin at this time. These drugs can do more harm. Donot give a diuretic like frusemide (Lasix) unless the patient is in acute pulmonary edema.

**Do not waste time and transport the patient to next level of facility**

**Management of suspected ACS, when ECG is available (in a hopsital, or during transportation)**

- Immediately assess the following (within 10 minutes of arrival to emergency room)
  - o A brief focussed history and physical exam
  - o Pulse, Respiratory rate, Blood pressure
  - o Oxygen saturation
  - o Obtain ECG and evaluate ST segment.
- If ST segment is elevated (greater than 1 millimeter in the limb leads or greater than 2 mm in the chest leads) in two or more contigious leads, STEMI is diagnosed and STEMI treatment protocol is initiated.
- If no ST elevation is there look for other features of ischemia (ST-depression, pathologic Q waves, inverted T waves). If such features are present a diagnosis of NSTEMI or UA treatment protocol is initiated.
- Obtain a blood sample for cardiac enzymes, blood glucose, and electrolytes.

**Initial management in STEMI / NSTEMI / UA**

Initial management of ACS of all types is same and involves the initial use of drugs to relieve ischemic discomfort, dissolve clots and inhibit thrombin and platelets. Initial management includes following four interventions:

a) Anti-platelet drugs (two drugs to be used)
   a. Tab Aspirin 300mg to chew, and
   b. Tab Clopidogrel 300mg to swallow

b) Nitrates (only if SBP >90mm Hg)
   a. Tab isosorbide dinitrate 10mg sublingual

c) Oxygen

d) Anti-coagulant
   a. Inj Enoxaparin (or another LMWH) 0.6mg subcutaneous stat

## Initial management in STEMI

Patient with STEMI usually have complete occlusion of an epicardial coronary artery. **The mainstay of treatment for STEMI is early reperfusion therapy.** Options for reperfusion therapy are:

### Percutaneous interventions (PCI)

PCI is treatment of choice for STEMI. Primary PCI is treatment of choice (Door to balloon time of 90 minutes). Percutaneous interventions include **Angioplasty alone or angioplasty with stenting.** In Angioplasty a wire with a deflated balloon is passed through the catheter to the narrowed area. The balloon is then inflated, compressing the deposits against artery walls. Stent is a long, thin tube (catheter) which is inserted into the blocked or narrowed part of artery. A mesh tube (stent) is usually left in the artery to help keep the artery open. Stents may be bare metal stents (BMS) or medicated stents.

If Primary PCI is not possible (Either hospital is not PCI enabled, there is no alternate facility in vicinity where PCI can be done within 90 minutes of recognition of STEMI, or it is logistically not possible to do a PCI), we move to next revascularization strategy of Thrombolysis.

**Thrombolysis** (or Fibrinolysis)

Thrombolytics are indicated in STEMI where Primary PCI is not possible.

Absolute contraindications for thrombolysis use in STEMI include the following:

- Prior intracranial hemorrhage (ICH)
- Known structural cerebral vascular lesion
- Known malignant intracranial neoplasm
- Ischemic stroke within 3 months
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Significant closed head trauma or facial trauma within 3 months
- Intracranial or intraspinal surgery within 2 months
- Severe uncontrolled hypertension (unresponsive to emergency therapy)
- For streptokinase, prior treatment within the previous 6 months

Relative contraindications for fibrinolytic use in STEMI include the following:

- History of chronic, severe, poorly controlled hypertension
- Significant hypertension on presentation (systolic blood pressure > 180 mm Hg or diastolic blood pressure > 110 mm Hg
- Traumatic or prolonged (> 10 minutes) cardiopulmonary resuscitation (CPR) or major surgery less than 3 weeks previously
- History of prior ischemic stroke not within the last 3 months
- Dementia
- Recent (within 2-4 weeks) internal bleeding
- Noncompressible vascular punctures
- Pregnancy
- Active peptic ulcer

Drugs used for thrombolysis are:

### Streptokinase

The adult dose of streptokinase for AMI is 1.5 million U in 50 mL of 5% dextrose in water (D5W) given IV over 60 minutes. Allergic reactions force the termination of many infusions before a therapeutic dose can be administered.

### Reteplase

First, reconstitute two 10-U vials with sterile water (10 mL) to 1 U/mL. The adult dose of reteplase for AMI consists of 2 IV boluses of 10 units each; there is no weight adjustment. The first 10 U IV bolus is given over 2 minutes; 30 minutes later, a second 10 U IV bolus is given over 2 minutes. Administer normal saline (NS) flush before and after each bolus.

## Acute Coronary Syndromes

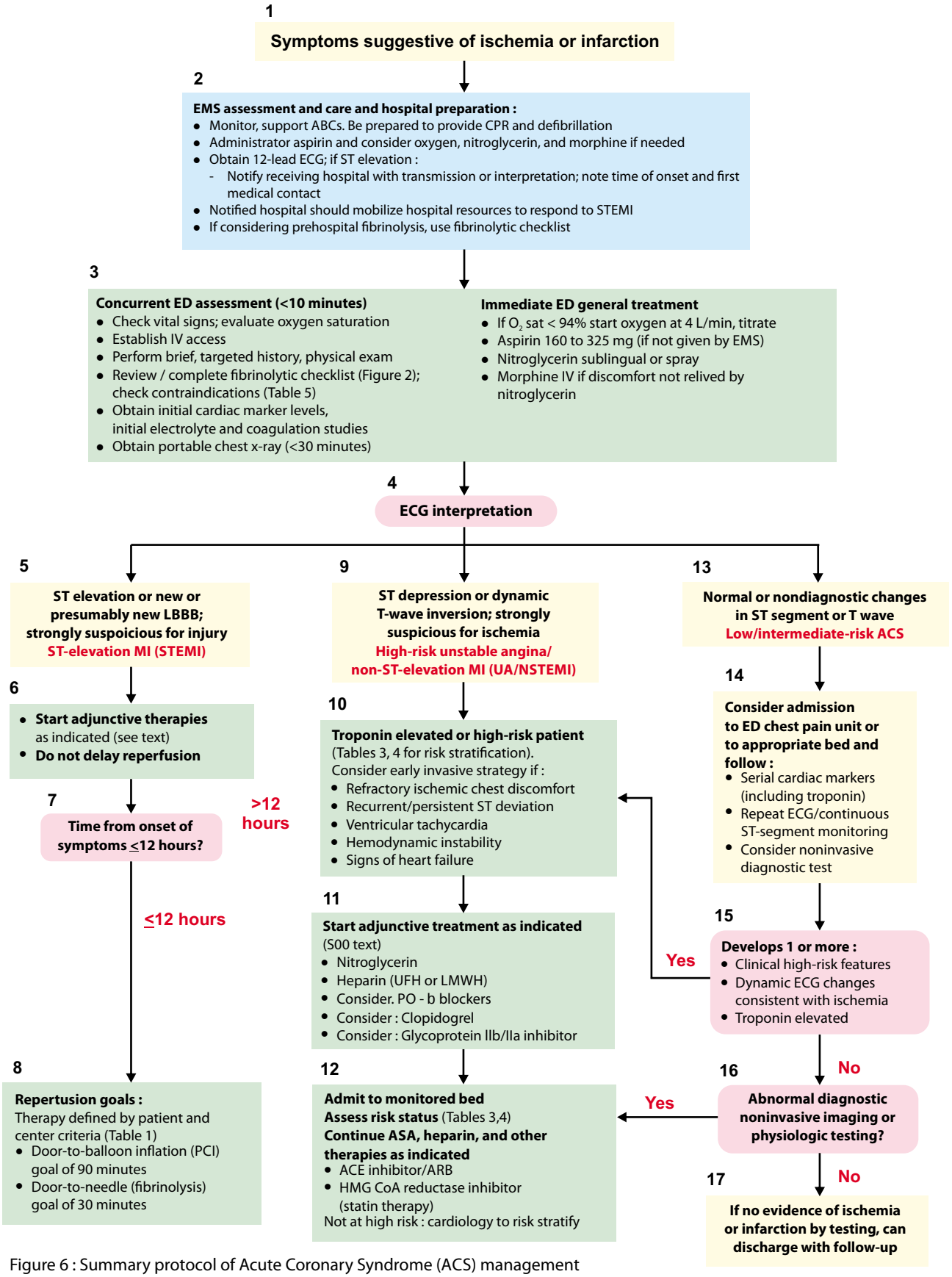



Figure 6 : Summary protocol of Acute Coronary Syndrome (ACS) management

**Subsequent therapies in all patients with ACS (STEMI / NSTEMI / UA)**

a) Anti-platelet drugs (two drugs to be used)
   a. Tab Aspirin 75-150 mg once daily
   b. Tab Clopidogrel 75mg once daily

b) Beta-blockers (unless contraindicated)
   a. Tab Metoprolol 50mg twice daily

c) Anti-coagulant
   a. Inj Enoxaparin (or another LMWH) 0.6mg SC twice daily x 5 days

d) ACE inhibitors (to start within 24 hrs)
   a. Tab Ramipril / Enalapril 5mg once daily and escalate

e) Statin (Lipid lowering)
   a. Tab Atrovastatin 40-80 mg once daily

f) Nitrates
   a. IV infusion of signs of ischemia persist

Specific contraindications to usage of beta-blockers include: (1) signs of heart failure, ( 2) evidence of a low output state, (3) increased risk for cardiogenic shock, (4) PR interval greater than 0.24 seconds (second- or third-degree heart block, and (5) active asthma or reactive airway disease.

---

**ECG Quiz Answers**

A T inversion in I, aVL and V6 (Consider ACS)

B ST elevation V1 to V4, reciprocal ST depression in II, III, aVF (Localizable to Anterolateral wall)

C ST elevation in almost all leads (Not Localizable), Also consider other causes such as acute pericarditis / Stroke.

D T inversion V1 to V5, and II, III. No ST changes. S wave in lead I, Q wave in III. These changes may suggest ACS or Pulmonary embolism (S1Q3T3)

CHAPTER 9

# MANAGEMENT OF RHYTHM DISTURBANCES

**In this chapter we will be discussing**

**A) Pulseless rhythms**

**B) Rhythm with pulse**

**(i) Tachy arrythmias (ii) Brady arrthmias**

## [A] Pulseless rhythms

These rhythms are characterised by absence of a pulse. Absence of pulse means absence of cardiac contractions, this is also refferred as cardiac arrest . However there may be an electric rhythm in a pulseless situation.

**Four common pulseless rhythms are -**

1. Ventricular fibrillation.
2. Ventricular Tachycardia without Pulse
3. Pulseless electrical activity.
4. Asystole.

**1. How to recognize them?**

**ECG is the gold standard tool to recognize these rhythms**

a) Ventricular Fibrillation (Figure 1) : It is a pre-terminal event. It is seen almost solely in dying hearts. It is most frequently encountered arrhythmia in adults who experience sudden cardiac death. ECG complexes are chaotic and no QRS complexes are seen. The heart generates no cardiac output and CPR along with defibrillation is a must.

b) Ventricular Tachycardia (Figure 2): Recognized by wide QRS complexes run of three or more PVC constitutes a VT. Ventricular tachycardia without pulse is an emergency. Immediate CPR along with defibrillation is a must.

c) Asystole (Figure 3) : It is the arrest of all the

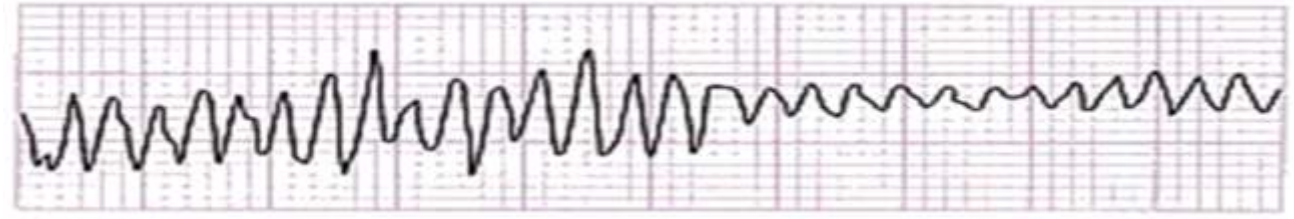


Figure 1: Ventricular Fibrillation

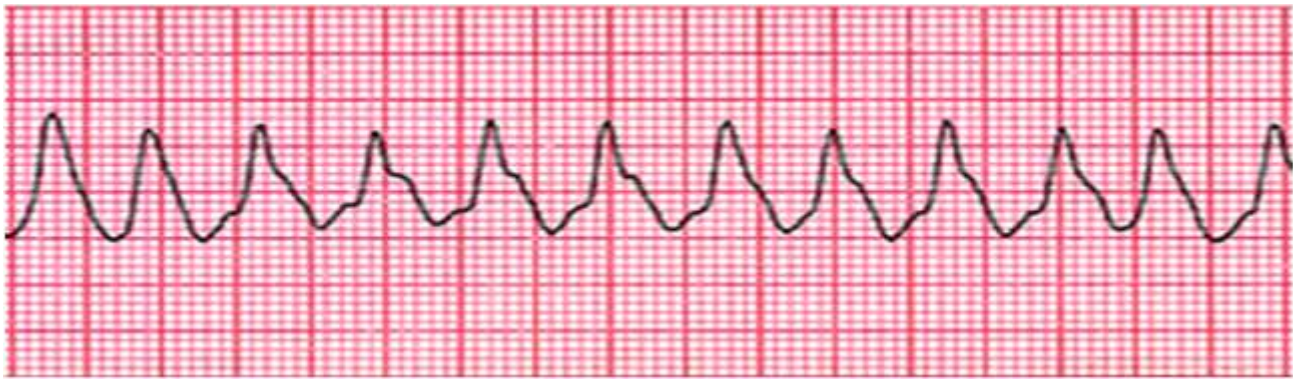


Figure 2: Ventricular Tachycardia

electrical and mechanical activities of heart. It is characterized by flat line. Immediate CPR is mainstay of this type of rhythm. There is no role of defibrillation.

d) Pulseless Electrical Activity (Figure 4): It is a type of electromechanical dissociation, there is some type of electrical activity but without any pulse. The treatment is like asystole where CPR is must but no role of defibrillation.

**2. Causes of Pulseless state (Cardiac Arrest):**

Ten causes to be ruled out in every arrest. These are reversible causes and must be kept in mind while resuscitating these patients.

i. Hypoxia
ii. Hypovolemia
iii. Hypothermia
iv. H+ ions- Acidosis/Alkalosis
v. Hypokalemia/ Hyperkalemia
vi. Toxins
vii. Tension pneumothorax
viii. Tamponade
ix. Thrombosis- Coronary
x. Thrombosis-Pulmonary

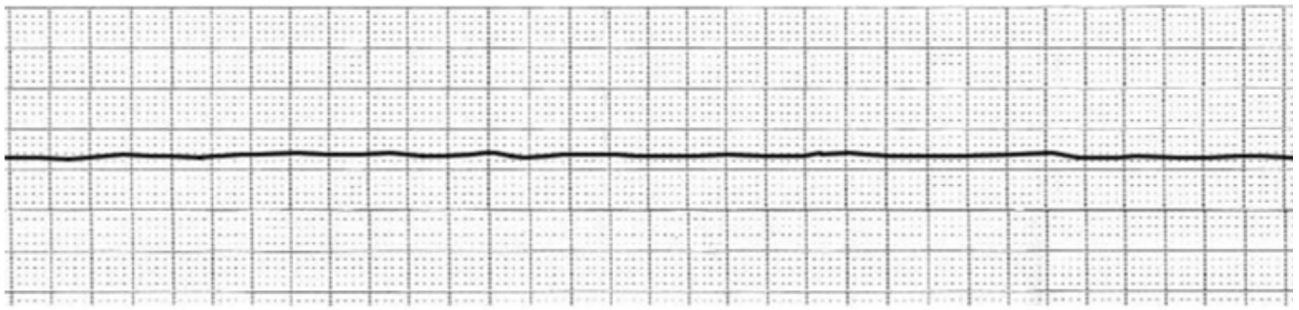


Figure 3: Asystole

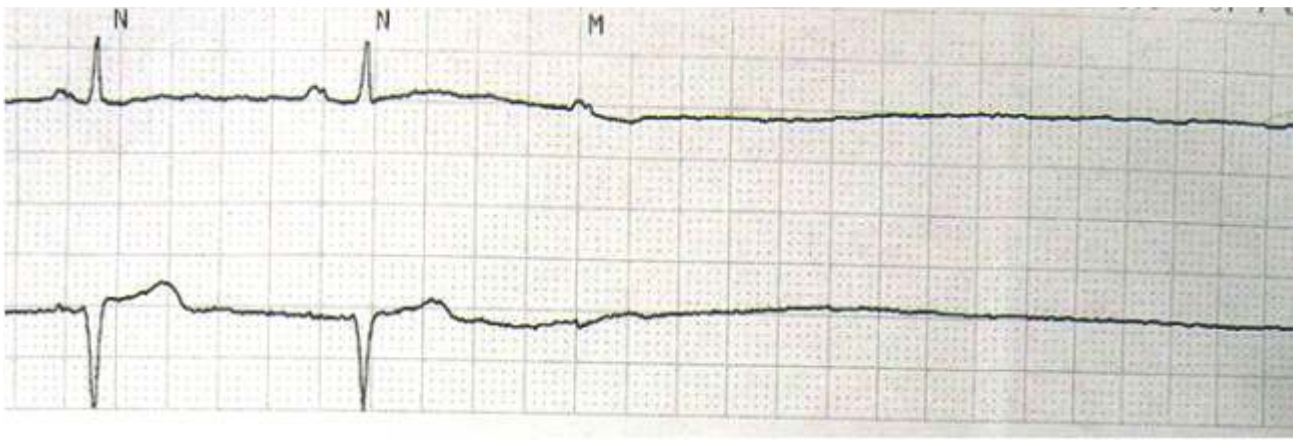



Figure 4: Pulseless Electrical Activity

**Management of Cardiac arrest**

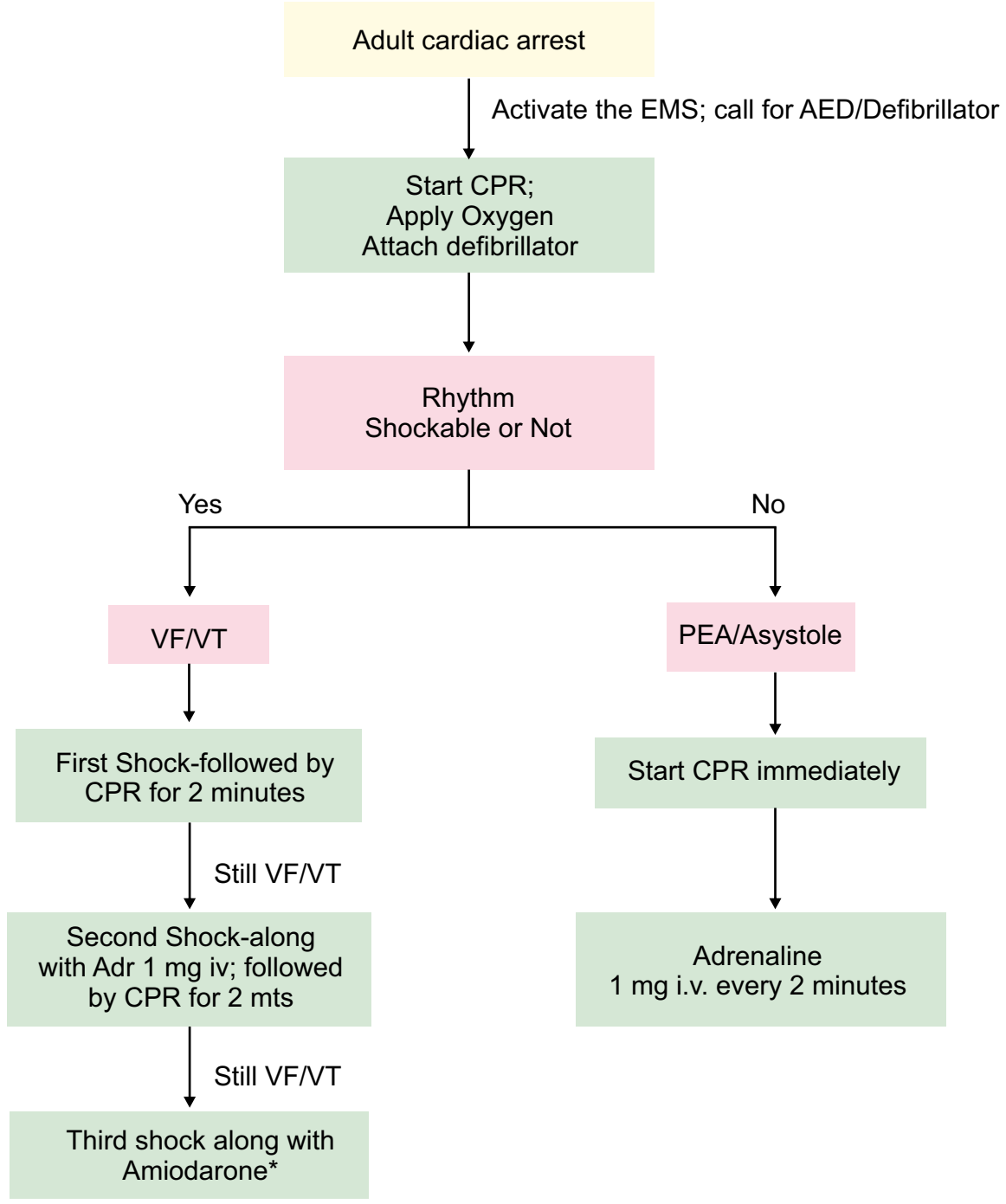



Fig 5: Algorithmic approach of Pulseless rythms

*Amiodarone 300mg iv bolus over 10min., followed by infusion 1 mg/minute X 6 hrs. followed by 0.5 mg/min. for next 18 hrs.

The primary life support to be started at scene, good good primary life support leads to better outcomes. Whatever be the rhythm, basic life support should be initiated as early as possible which has following steps:

(i) Recognition of arrest- By loss of consciousness, breathing absent or abnormally (like gasping)

(ii) Call for help- activation of 108 and call for defibrillator/AED

(iii) Pulse check for not more than 10 seconds

(iv) Immediate CPR- Push hard and fast, at least 100 compressions /mt; Compression depth of at least 2 inches.

A. Single Rescuer Compression: Ventilation ratio- 30:2

B. Two rescuers : First rescuer does CPR at rate of 100/mt

Second rescuer does ventilation at rate of 6-8 breaths / minute

5 cycles to be completed within 2 minutes.

(1 cycle – 30 Compression:2 Ventilation )

**VF/VT without pulse** (See Figure 5)

A. VT without pulse/ VF : Early and rapid defibrillation forms the key. After defibrillation immediate CPR should be initiated which has to be done for atleast 2 minutes. After second shock comes the role of Adrenaline, if still VF/VT persists then after third shock comes the role of Anti-arrhythmic drug i.e. Amiodarone.

B. Consider amiodarone for ventricular fibrillation/pulseless ventricular tachycardia after 3 attempts at defibrillation, as there is evidence it improves response in refractory VF / VT. (Note: As of the 2010 guidelines, Amiodarone is preferred as the first-line antiarrhythmic, moving lidocaine to a second-line backup if Amiodarone is unavailable.)

C. In the 2010 ACLS pulseless arrest algorithm, vasopressin may replace the first or second dose of Adrenaline.

D. Torsades de pointes : Refractory VF in patients with digoxin toxicity or hypo-magnesemia, give IV magnesium sulfate 8 mmol (4mL of 50% solution).

E. Exercise caution before using epinephrine in arrests associated with cocaine or other sympathomimetic drugs.

F. Epinephrine is not required until after the second DC shock in standard ACLS management as DC shock in itself releases significant quantities of epinephrine.

**PEA/Asystole**

A. Treatment of choice is immediate CPR followed by Adrenaline 1mg IV bolus which has to be repeated every 2 minutes.

A. Atropine 1 mg dose (IV) bolus for asystole no longer recommended.

B. In PEA arrests associated with hyperkalemia, hypocalcemia or $Ca^{2+}$ channel blocking drug overdose, give 10mL 10% calcium gluconate chloride (IV)

C. In the 2010 ACLS pulseless arrest algorithm, vasopressin may replace the first or second dose of epinephrine.

PEA/Asystole: Early and effective CPR forms the key to success. There is no role of defibrillation and neither Atropine. Only effective agent is Adrenaline.

**Defibrillators**

i. AED : These are automated external defibrillators, where they automatically detect whether the rhythm is shockable or not. If the rhythm is shockable – a prompt appears and shock is delivered.

ii. Manual defibrillators :

a) Monophasic- Maximum energy is to delivered which is displayed on the monitor which is upto 360 J.

b) Biphasic- Maximum energy which is displayed on monitor which ranges from 120 J- 200J.

## [B] Rhythms with pulse

a. Tachyarrythmias

b. Bradyarrythmias

### a. Tachyarrhythmia's

Tachycardia's can be classified in several ways, based on the appearance of the QRS complex, heart rate, and regularity. Tachycardia is defined as heart rate of 100 beats per minute. The rate of a tachycardia takes on clinical significance at it's greater extremes and is more likely attributable to an arrhythmia at a rate of 150 beats per minute. Tachyarrhythmia's for our knowledge are:

1. Sinus tachycardia
2. Atrial fibrillation
3. PSVT
4. Ventricular tachycardia

When dealing with Tachyarrhythmia's important thing is to search for the underlying cause.

In patients with Tachyarrhythmia's following basic steps are to be followed (Figure 6)

1. Check for Pulse : Check whether the pulse is present and absent In cases of absent pulse, pulseless algorithm has to be followed as patient would be suffering from VF/VT and for which treatment of choice is defibrillation.
2. Look for Signs and symptoms of hypo-perfusion : which are *altered mental status,*

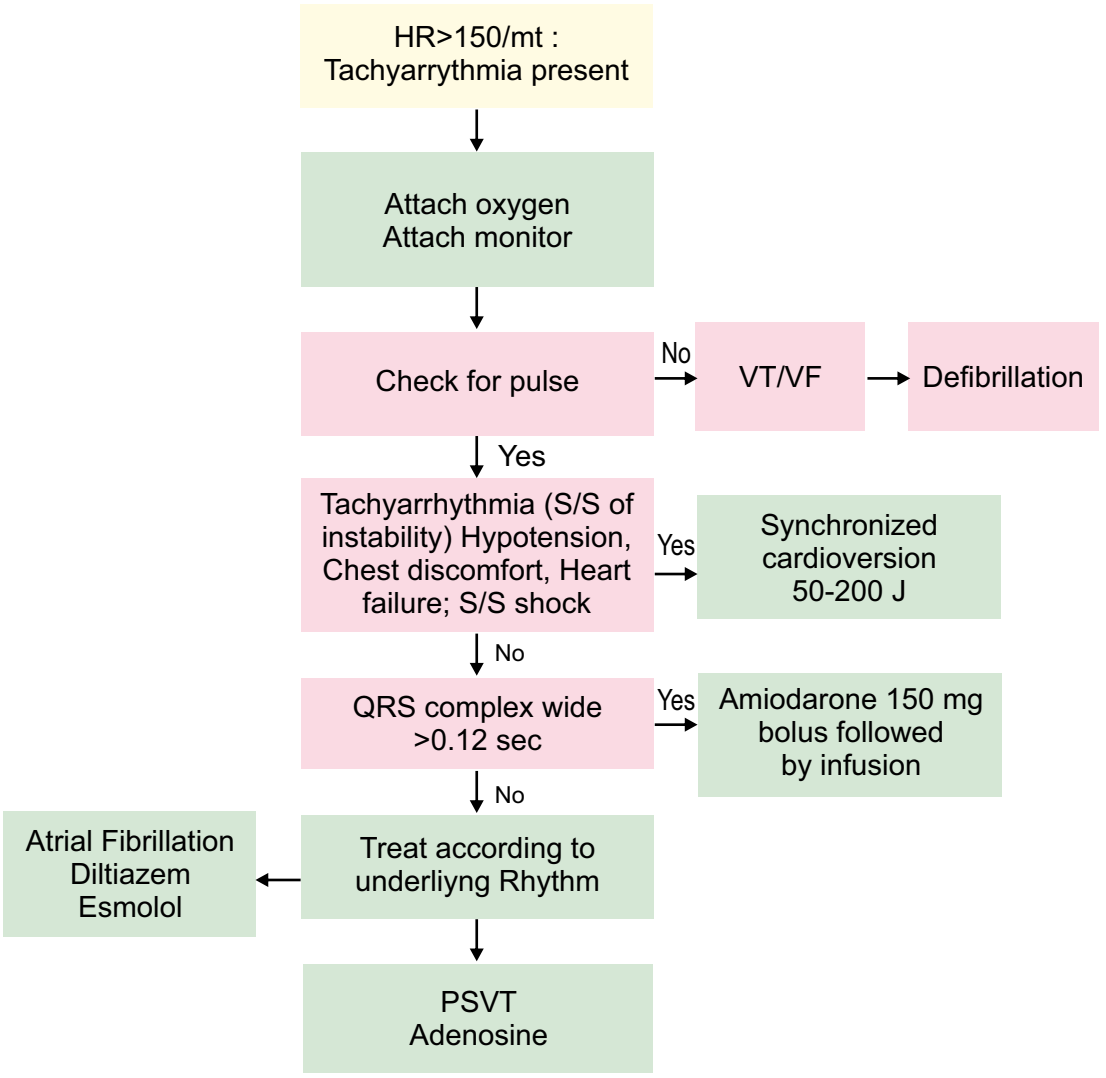



Figure 6 : Stepwise management of patient with tachyarrythmia

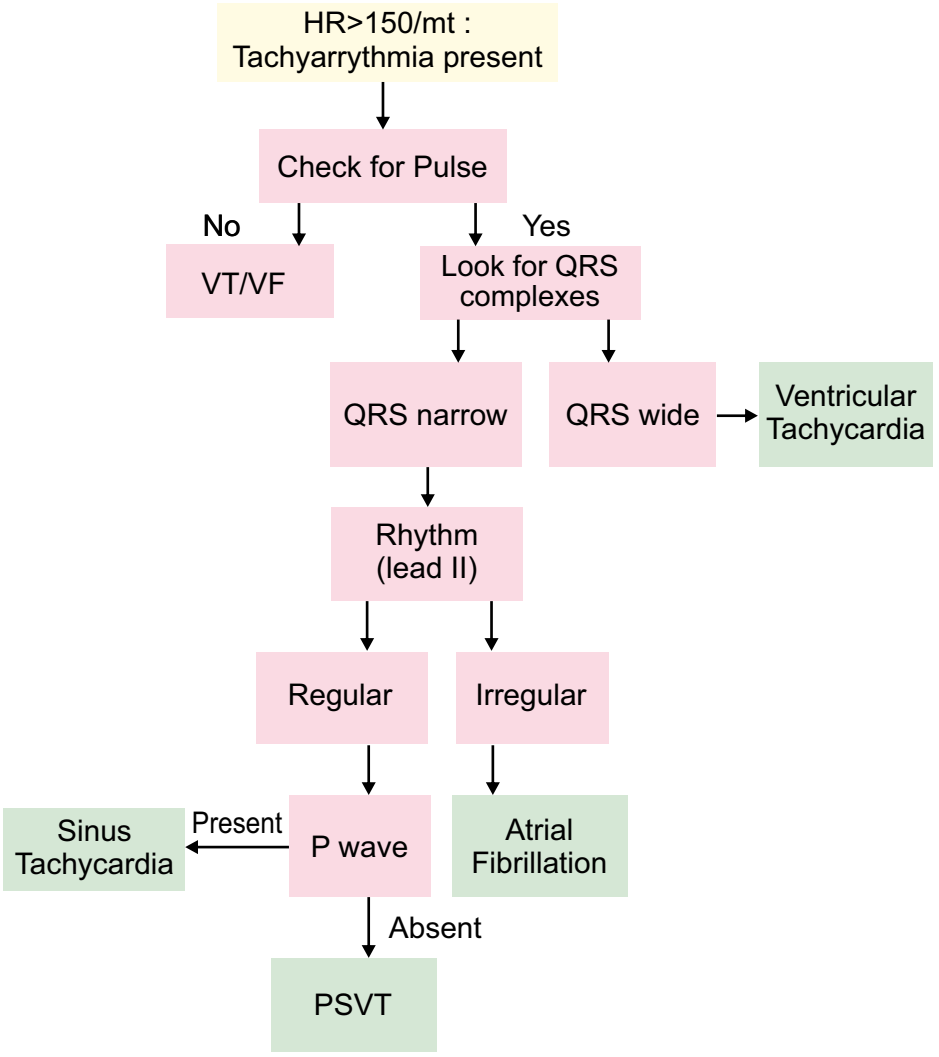



Figure 7 : Approach to a patient with Tachyarrhythmia

*hypotension, cool extremities, chest discomfort*. If any one of the above S/S are present cardioversion has to be initiated immediately.

3. Look for QRS complex and see whether QRS is wide or narrow : Treatment as per QRS complex

4. Regular/irregular : Regular-PSVT/

   Irregular - AF

**I. Classification of Tachy arrhythmias on the basis of: (Figure 7)**

**i. QRS complex :**

**a) Narrow complex**

**b) wide complex**

**ii) Regularity**

**a) Regular**

**b) Irregular**

**i. QRS Complex**

Look QRS complex see whether QRS is narrow i.e.QRS≤0.12 sec then rhythm is supraventricular and when QRS≥0.12 sec then rhythm is ventricular.

**a) Narrow complex tachy arrhythmias:**

1. Sinus tachycardia

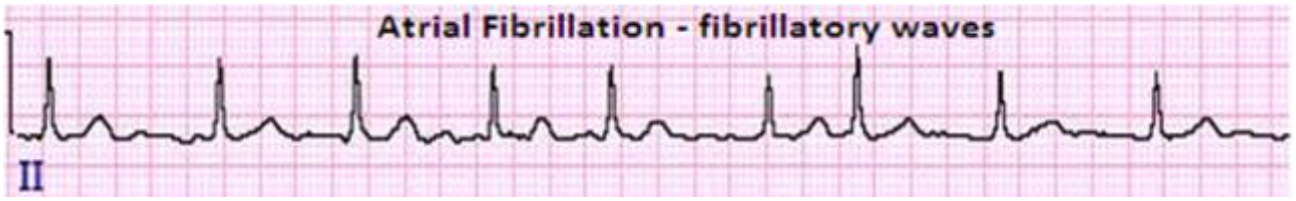



Figure 8: Atrial Fibrillation and Atrial flutter

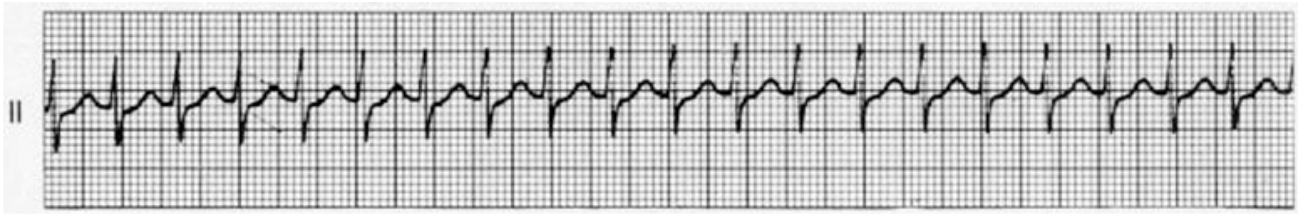



Figure 9 : Narrow complex tachycardia

2. Atrial fibrillation (Figure 8)
3. Narrow complex tachycardia (Figure 9)

**b) Wide complex rhythms:**

1. Ventricular tachycardia (Figure 2)

**ii) Classification on the basis of Regularity**

**a) Regular**

1. Sinus tachy cardia
2. Supra ventricular tachy cardia (SVT)

**a) Irregular rhythms:**

1. Atrial fibrillation (Figure 8)

**Individual rhythms**

1. **Atrial Fibrillation:** It is one of the most common supraventricular arrhythmia with QRS duration ≤0.12 secs. It is characterized by irregular rhythm. It is commonly due to sympathomimetic stimulation, electrolyte imbalances and can be drug induced. General management of atrial fibrillation should focus on control of the rapid ventricular rate (rate control), or conversion of hemodynamically unstable atrial fibrillation to sinus rhythm (rhythm control). Patients with atrial fibrillation duration of 48 hours are at increased risk for cardio-embolic events, although shorter durations of atrial fibrillation do not exclude the possibility of such events.

   Electric or pharmacologic cardioversion (conversion to normal sinus rhythm) should not beattemptedin these patients unless the patient is unstable. The management depends on hemodynamic status of the patient. If the patient is unstable then immediate cardioversion has to be done. On other hand if the patient is stable, then rate control with Diltiazem. (Table 1)

2. **Narrow complex tachycardia :** It is type of supraventricular arrhythmia which is regular in rhythm with QRS duration ≤0.12 sec. In this type of arrhythmia P waves are absent or merged in preceding T wave. The vagal manoeuvres may be tried. Adenosine is drug of choice. Adenosine 6 mg rapid i.v. push, followed with normal saline i.v. flush. A second dose of 12 mg if required is given.

3. **Ventricular Tachycardia :** Recognized by wide QRS complexes run of three or more PVC constitutes a Ventricular Tachycardia. Ventricular tachycardia without pulse is an emergency. Immediate CPR along with defibrillation is a must. In patients with VT with pulse the drug of choice is Amiodarone 150mg IV bolus over 10 minute.

**Cardioversion or Synchronized shocks:**

It uses a sensor that delivers a shock that is synchronized with peak of QRS complex (highest point of R wave). Synchronization avoids delivery of shock during cardiac repolarization i.e. during T

wave which in turn would precipitate VF. These are low energy shocks and must be synchronized in order to prevent VF. In addition to cardioversion, drug therapies are used as follows:

**b. Brady-arrhythmias (Figure 10) :** It is defined as HR<50/mt. If HR<50 /mt is associated accompanied with sign and symptoms of poor perfusion i.e. Chest pain, S/S of heart failure, altered sensorium and hypotension the immediate treatment in form of drug therapy is recommended or if available transcutaneous pacing is recommended.

Drug therapy in form of

i. Atropine 0.6 mg; max upto 3 mg is recommended.

ii. Dopamine in dose of 5-10 µg/kg/mt is recommended

iii. Epinephrine in dose of 0.05 µg/kg/mt-1µg/kg/mt is recommended.

**Conclusion :**

An algorhythmic approach to a patient with rhythm described in Figure 11.

Table 1 : Drug therapy for individual cardiac rhythms

| | Drug therapy | Side effects |
|---|---|---|
| **1. Atrial fibrillation** | **Rate control**<br>Diltiazem: Initial dose 15 to 20 mg (0.25 mg/kg) i.v. over 2 minutes; additional 20 to 25 mg (0.35 mg/kg) i.v. in 15 minutes if needed; 5to15 mg/h iv maintenance infusion (Titrated to AF heart rate if given for rate control). | Hypotension, Bradycardia with precipitation of heart failure. |
| **2. Narrow complex tachycardia** | Adenosine 6 mg iv bolus followed by flush with 20 ml NS. Repeat Adenosine 12 mg. | Hypotension, bronchospasm |
| **3. Ventricular Tachycardia with pulse** | Amiodarone 150 mg given over 10 minutes and repeated if necessary, followed by a 1 mg/min infusion for 6 hours, followed by 0.5 mg/min. Total dose over 24<br>hours should not exceed 2.2 g. | Hypotension, Bradycardia |

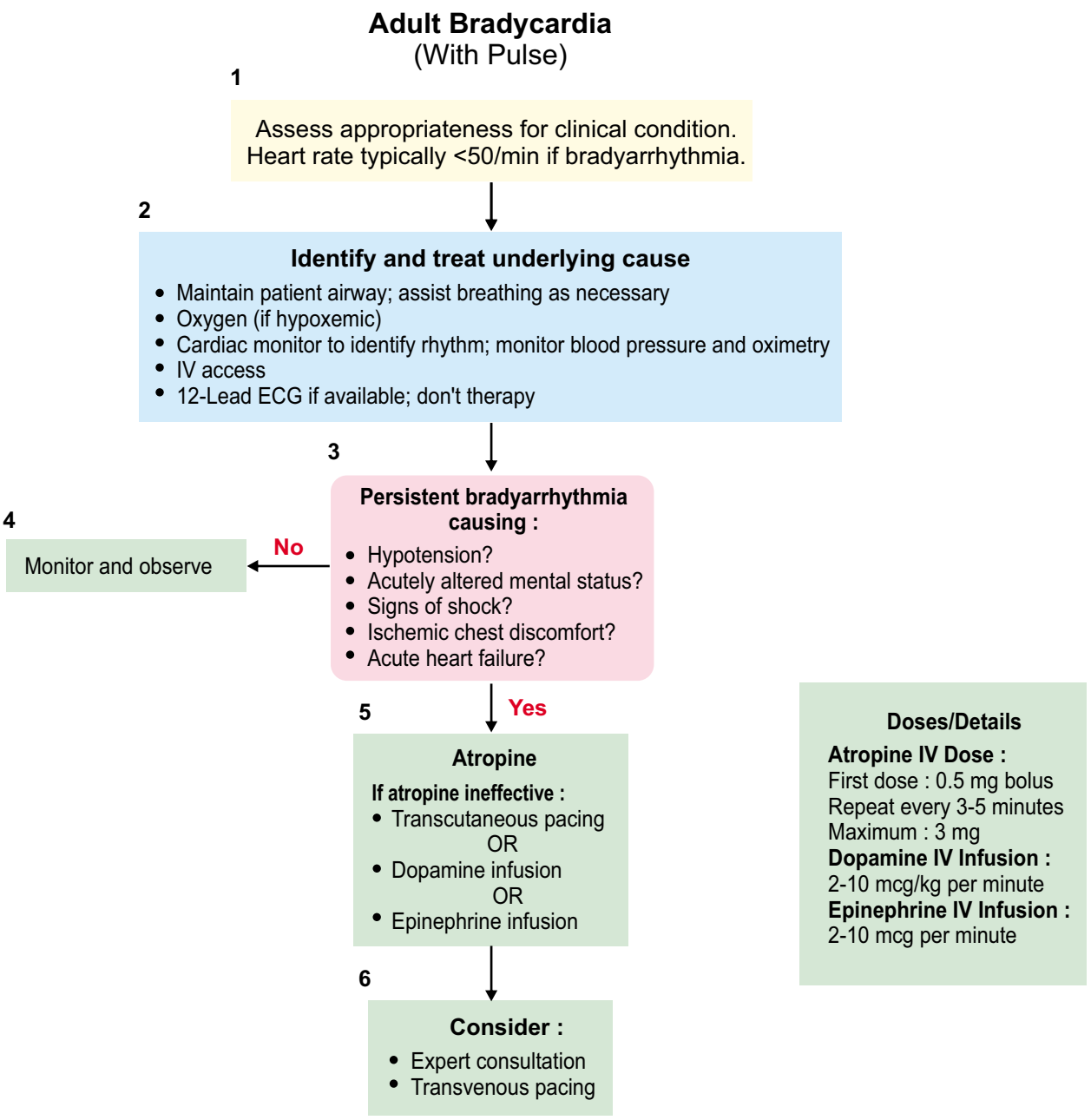



Figure 10 : Approach to a patient with Brady arrhythmia

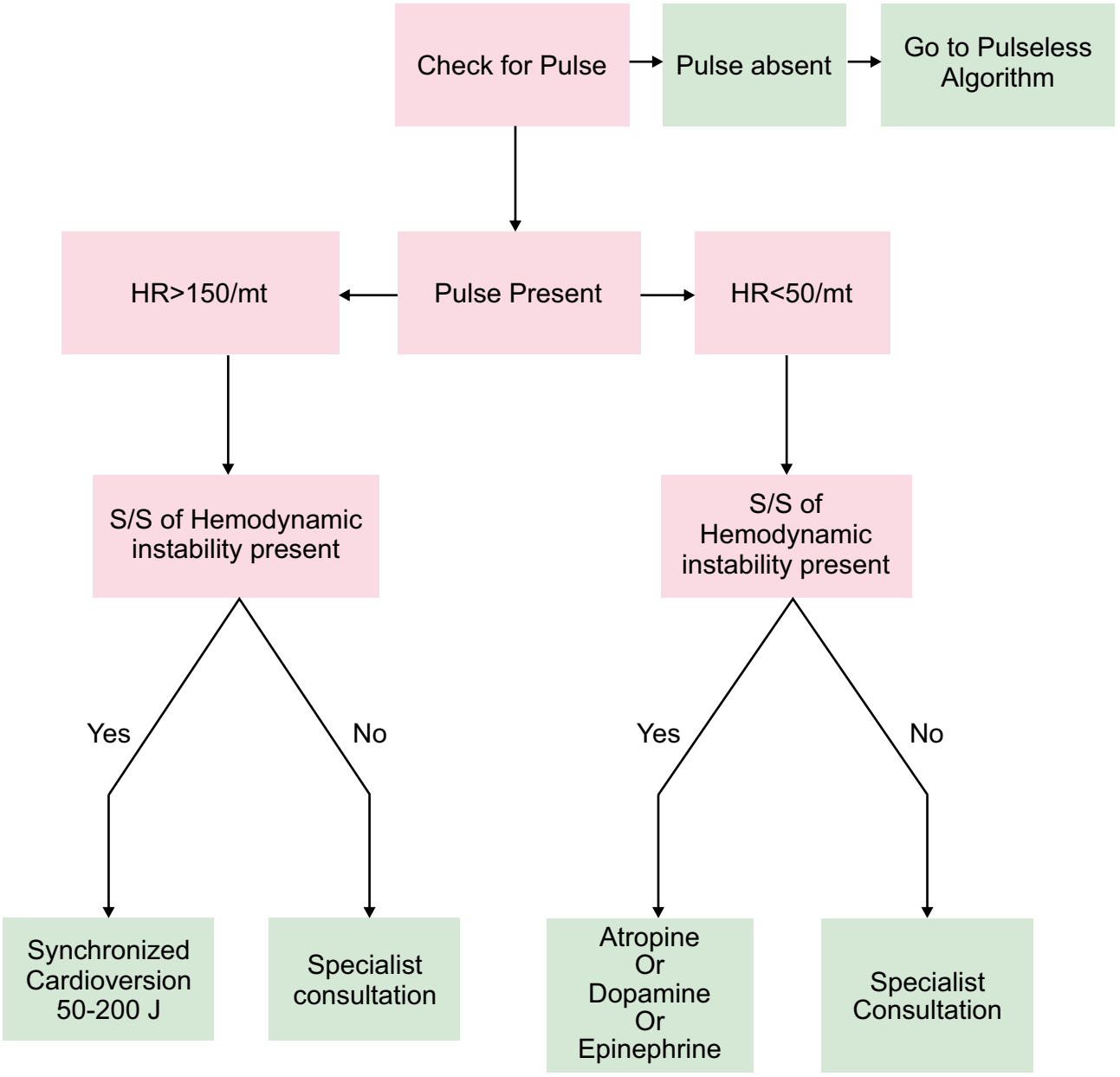



Fig 11 : Common Algorithmic approach – Pulseless rhythms and unstable patient with pulse

CHAPTER 10

# EARLY RECOGNITION AND MANAGEMENT OF STROKE

Stroke is an acute onset neurological deficit, which occurs due to interruption in blood supply of brain. About 85% of strokes occur due to occlusion in blood supply of the brain (thrombosis), and remaining 15% occur due to rupture of a blood vessel supplying brain (hemorrhage). It is important to recognize stroke early, and distinguish if it is due to hemorrhage or infarction, which requires brain imaging (CT scan). Subsequent management of stroke requires prompt specialist care including thrombolysis for ischemic stroke within a 3 hr window. Since early brain imaging is important, focus of this module is on early recognition (Detection) and prompt referral (Dispatch, and Delivery to facility with CT scan capability). Remaining steps in early stroke care are appropriate triage to a stroke center (Door), performance of CT scan and clinical evaluation (Data), stroke expertise and therapy selection (Decision), Use of fibrinolytic therapies (Drug) and rapid admission to stroke units (Disposition).

About 76% of all strokes can be detected by using FAST technique, which consists of asking three simple questions:

i. Ask person to smile, does one side of face droop on smiling ?

ii. Ask person to raise both arms, does one arm drift downward ?

iii. Ask person to repeat a simple phrase. Is their speech slurred or strange ?

Once stroke is recognized, idea is to promptly refer to a facility where CT scan is available.

## Guidelines for management of stroke

This clinical guideline for stroke management is based on the National Clinical Guidelines for stroke, published in annals of Indian academy in neurology. It includes the management of patients with acute stroke and the secondary prevention of stroke.


These guidelines cover the management of stroke in adults (over 18 years) from onset to chronic care and focus on patients with a new clinical event (first stroke or recurrent stroke).

## 1. Acute Phase Care (First 24-48 hrs.)

### Admission to hospital

Most patients with stroke should be admitted to a hospital because their neurological condition may worsen over the first few days, they may develop non-neurological complications (e.g., aspiration pneumonia), and urgent investigations (like CT scan) may be required.

The following assessments need to be performed in a hospital :-

a) Responsiveness

b) Adequacy of airway, breathing and circulation

c) Typing of stroke as Ischemic stroke or a hemorrhage. This needs to be done by performing brain imaging (CT scan)

On brain imaging intra-cerebral hemorrhage will be visible with first few hours of symptoms. In case of ischemic stroke brain imaging may be normal upto first 24 hours. In a person with symptoms of stroke, even if brain imaging is normal, it is considered as ischemic stroke.

Aspirin should not be given to patients who have intra-cerebral hemorrhage or where surgery is planned (decompressive craniotomy)

**Immediate specific management of ischemic stroke**

All patients with disabling acute ischemic stroke should be evaluated without delay to determine their eligibility for treatment with intravenous tissue plasminogen activator (alteplase). This drug must be given within 3 hours of symptom onset to be useful.

All acute stroke patients should be given at least 150 mg of aspirin immediately after brain imaging has excluded intracranial hemorrhage. In patients in whom t-PA is planed aspirin should be delayed until after the 24-hour post-thrombolysis. In patients with large hemispheric infarct (malignant MCA territory infarct), aspirin may be delayed until surgery or decision is made not to operate. In dysphagic patients, aspirin may be given by enteral tube.

**Management of blood pressure in Ischemic and hemorrhagic store**

Ischemic stroke

- Antihypertensive medication should be withheld in ischemic stroke patients unless SBP is >220 mmHg or the mean arterial blood pressure (MAP) is >120 mmHg.
- Lowering of blood pressure by approx 15% during the first 24 hours is recommended.
- Except in hypertensive emergency, lowering of blood pressure should be slow and with use of oral medications.
- Sublingual use of antihypertensives is not recommended.
- Blood pressure reduction to 185/110 mmHg or lower should be considered in people who are candidates for thrombolysis.

Hemorrhagic Stroke

- If SBP is >200 mmHg or MAP is >150 mmHg (recorded twice, two or more minutes apart), then blood pressure should be aggressively treated with parenteral antihypertensives (e.g., labetolol or nitroglycerin).
- If SBP is >180 mmHg or MAP is >130 mmHg (up to 150 mm Hg), then a modest reduction is advised with rapidly acting oral or parenteral medication or nitroglycerin patch.
- Target BP should be 160/90 or MAP of 110 mmHg.

**Management of blood glucose**

- The blood glucose level should be maintained between 70 and 190 mg/dL. Elevated blood glucose >140 mg/dL should be managed with insulin administration using the sliding scale in the first week of stroke onset.
- Hypoglycemia should be monitored and accordingly 20% glucose (50 ml bolus) should be administered.

**Cerebral edema and increased intracranial pressure**

- Corticosteroids are not recommended for reduction in intra-cronial pressure.
- In patients whose condition is deteriorating secondary to increased ICP (to reduce ICP): Hyperventilation, mannitol, furosemide, CSF drainage and surgery.
- If CT scan (first or repeat one after deterioration) suggests hydrocephalus as the cause of increased ICP, then continuous drainage of CSF can be advised.

- Initial care includes mild restriction of fluids, elevation of head end of the bed by 20 to 30 degrees and correction of factors that might exacerbate increased ICP (e.g. hypoxia, hypercarbia and hyperthermia).
- Hyperventilation acts immediately (reduction of the pCO 2 by 5-10 mmHg lowers ICP by 25-30%) but should be followed by another intervention to control brain edema and ICP. Hyperventilation can cause vasoconstriction that might aggravate ischemia.
- An intravenous bolus of 40 mg furosemide may be used in patients whose condition is rapidly deteriorating. If required, furosemide 20 mg (once daily) may be continued for the first week,
- Acetazolamide 250 mg (BD) may be added in those not responding to other treatment methods.
- Strict intake-output chart must be maintained to avoid dehydration.
- Mannitol (0.5 gm/kg IV given over 20 minutes) can be given every 6-8 hours. If clinically indicated, dose frequency may be increased to every 4 hours, but then the central venous pressure should be monitored and kept between 5 and 12 mmHg to prevent hypovolemia. This may be continued for 3-5 days.

**Supplemental oxygen therapy**

- Patients should receive supplemental oxygen if their oxygen saturation drops below 95%.

**Management of body temperature**

- Temperature should be monitored every 4 hours for at least first 48 hours and preferably as long as the patient is in the ward.
- Fever (>37.5 C) should be treated with paracetamol. The search for possible infection (site and cause) should be made.
- Hypothermia <34 C should be avoided as it can lead to coagulopathies, electrolyte imbalance, infection and cardiac arrhythmias.

**Management of seizures**

- Patients with seizure, even single should be treated with loading dose of phenytoin (15-20 mg/kg) followed by maintenance dose 5 mg/kg per day for a period of at least 3 months.
- If needed, carbamazepine or sodium valproate may be added.
- Status epilepticus should be treated as per its guidelines.
- Prophylactic anticonvulsants in recent stroke may not be required.

**Caveats in initial management:**

- A person with stroke may be semi-conscious or unconscious. Focus on maintainance of Airway, Breathing, and Circulation. Check blood glucose, and correct hypoglycemia if present.
- Patients with stroke have elevated blood pressure. Donot administer blood pressure lowering drugs prior to transport. Donot lower blood pressure to below 180/110 mm Hg.
- Do not administer Aspirin, before it is established that hemorrhage is not cause of stroke.
- Do not use steroids / bronchodialators / antibiotics as a routine in management of stroke.

## 2. Management after initial 24-48 hrs

- Patients should be advised to undertake activities like sitting, standing or walking only with caution. An occasional patient, who deteriorates on assuming sitting or standing posture, should be advised bed rest.
- Non-ambulatory patient should be positioned to minimize the risk of

complications such as contractures, respiratory complications, shoulder pain and pressure sores etc.

**Swallowing**

- All conscious patients should have assessment of the ability to swallow. A water swallow test performed at the bedside is sufficient (e.g. 50 ml water swallow test )
- Testing the gag reflex is invalid as a test of swallowing.
- Patients with normal swallow should be assessed for the most suitable posture and equipment to facilitate feeding. Any patient with abnormal swallow should be fed using a nasogastric tube.
- Gastrostomy feeding should be considered for patients who are unable to tolerate nasogastric tube.
- Patients with altered sensorium should be given only intravenous fluids (Dextrose saline or normal saline) for at least 2-3 days.

**Oral care**

- All stroke patients should have an oral/dental assessment including dentures, signs of dental disease, etc., upon or soon after admission.
- For patients wearing a full or partial denture, it should be determined if they have the skills to safely wear and use the appliance(s).
- An appropriate oral care protocol should be used for every patient with stroke, including those who use dentures.
- The oral care protocol should address areas including frequency of oral care (twice per day or more), types of oral care products (toothpaste, floss and mouthwash) and specific management for patients with dysphagia.
- If concerns are identified with oral health and/or appliances, opinion of dentist as soon as possible.

**Early mobilization**

- Passive full-range-of-motion exercises for paralyzed limbs can be started during the first 24 hours.
- Physiotherapist/rehabilitation team should be involved as soon as possible, preferably within 48 hrs.

**Nutrition**

- Every patient should have his/her nutritional status determined using valid nutritional screening method within 48 hours of admission.
- Nutritional support should be considered in any malnourished patient.

**Venous thromboembolism**

*Prophylaxis*

- Patients with paralyzed legs (due to ischemic stroke) should be given standard heparin (5000 units subcutaneous b.d.) or low-molecular weight heparin to prevent deep vein thrombosis (DVT).
- For those who cannot tolerate heparin, aspirin given for treatment is of some prophylactic value.
- In patients with paralyzed legs (due to ICH), routine physiotherapy and early mobilization should be carried out to prevent leg vein thrombosis.
- Early mobilization and optimal hydration should be maintained for all acute stroke patients.

**Treatment**

- Standard heparin (5000 U i.v.) or low molecular weight heparin (with appropriate therapeutic doses as per agent) should be started initially.
- When standard heparin is used, a prior baseline complete blood count and a-PTT should be done and a bolus (80 U/kg/h) and maintenance infusion (18 U/kg/h) should

be given (target a-PTT of 1.5 times the control value).

- Anticoagulation (warfarin 5 mg once daily) should be started simultaneously unless contraindicated and the dose should be adjusted subsequently to achieve a target INR of 2.5 (range 2.0-3.0), when heparin should be stopped.

**Bladder care**

- An indwelling catheter should be avoided as far as possible and if used, indwelling catheters should be assessed daily and removed as soon as possible.
- Intermittent catheterization should be used for urinary retention or incontinence.
- The use of portable ultrasound is recommended as preferred non-invasive method for assessing post-void residual urine.

**Bowel care**

- Patient with bowel incontinence should be assessed for other causes of incontinence including impacted feces with spurious diarrhea.
- Patients with severe constipation should have a drug review to minimize use of constipating drugs, be given advice on diet, fluid intake and exercise (as much as possible),
- Oral laxatives and be offered rectal laxatives only if severe problems remain.

**Infections**

- Development of fever after stroke should prompt a search for pneumonia, urinary tract infection or DVT.
- Prophylactic administration of antibiotics is not recommended.
- Appropriate antibiotic therapy should be administered early (after taking relevant culture specimens).

**Discharge planning**

- Discharge planning should be initiated as soon as a patient is stable.
- Patients and families should be prepared and fully involved.
- Care givers should receive all necessary training in caring for it.
- Patients should be given information about discharge issues and explained the need for and timing of follow up after discharge.

## 3. Diagnosis and management of resolved or rapidly resolving acute neurological event

- Patients with fully resolved or rapidly resolving neurological symptoms need diagnosis to determine whether cause is vascular (about 50% are not)
- Treatable causes of TIA to be identified to reduce the risk of stroke (greatest in first 7-14 days).
- Any patient with transient symptoms suggestive of a cerebrovascular event should be considered to have had a transient ischemic attack (TIA), unless neuroimaging reveals an alternative diagnosis.
- All such patients except those with transient monocular blindness should have imaging of brain, either CT scan or MRI.
- Patients presenting with transient monocular blindness (amaurosis fugax) must have a complete ophthalmological examination to exclude primary disorders of the eye before diagnosis of TIA.
- Patients TIA should be assessed as soon as possible for their risk of subsequent stroke using a validated scoring system.
- All patients with history of TIA should be started on aspirin (150 or 300 mg) daily or Clopidogrel (75 mg) once a day in case of aspirin allergy.

- All patients with TIA should be managed as indicated under the heading 'Secondary Prevention'.

ABCD and ABCD2 Prognostic score to identify people at high risk of stroke after a TIA. It is calculated based on :

A-age (>60 years, 1 point).

B-blood pressure at presentation (> 140/90 mmHg. 1 point)

C-clinical features (unilateral weakness, 1 points or speech disturbance without weakness, 1 point).

D-duration of symptoms (>60 minutes, 2 points or 10-59 minutes, 1 point)

The calculation of ABCD2 also includes the presence of diabetes (1 point). Total scores range from 0 (low risk) to 7 (high risk)

**History, physical examination and common investigations**

- Special attention to onset of symptoms, recent stroke, myocardial infarction, seizure, trauma, surgery, bleeding, pregnancy and use of anticoagulation/insulin/ antihypertensive,
- History of modifiable risk factors: Hypertension, diabetes, smoking, heart disease, hyperlipidemia, migraine and history of headache or vomiting, recent child birth and risk of dehydration.
- Physical examination should be on usual lines with special attention to ABC (airway, breathing, circulation), temperature, oxygen saturation, sign of head trauma (contusions), seizure (tongue laceration), carotid bruits, peripheral pulses, cardiac auscultation, evidence of petechiae, purpura or jaundice.
- All patients should have neuroimaging, complete blood count, blood glucose, urea, serum creatinine, serum electrolytes, ECG and markers of cardiac ischemia.
- Selected patients may require liver function tests, chest radiography, arterial blood gases, EEG, lumbar puncture, blood alcohol level, toxicology studies or pregnancy test.
- All patients should have their clinical course monitored and any patient whose clinical course is unusual for stroke should be reassessed for possible alternative diagnosis.

Brain imaging should be performed immediately for patients with persistent neurological symptoms if any of the following apply:

- Indication for thrombolysis or early anticoagulation.
- On anticoagulant treatment.
- A known bleeding tendency.

Patients with acute stroke without the above indications for immediate brain imaging, scanning should be performed within 24 hours after onset of symptoms.

CHAPTER 11

# DROWNING AND ELECTROCUTION

## Drowning

Drowning is defined as a mortal submersion event in which the patient dies within 24 hours, while the term submersion injury is used up until the time of drowning-related death. Death occurs due to denial of access of air to lungs by the submersion of body in water or other fluids.

Near-drowning generally is defined as survival, at least temporarily, after aspiration of fluid into the lungs ("wet near-drowning") or after a period of asphyxia secondary to laryngospasm ("dry near-drowning").

Children less than 5 years of age who are inadequately supervised in swimming pools or bathtubs, and males between 15 and 25 years old with mishaps occurring at rivers, lakes, and beaches are commonly effected.

### Pathophysiology

Near-drowning result in decreased lung compliance, ventilation-perfusion mismatching, and intrapulmonary shunting, leading to hypoxemia that causes diffuse organ dysfunction. The temperature of the water and the presence of contaminants may impact patient outcomes (Figure 1, Table 1)

### Management

a. **Prehospital care —** cardiopulmonary resuscitation (CPR) as soon as possible after assessing scene safety and removal of the patient from the water. In BLS the recommended sequences CAB but in cases of drowning the sequence is ABC. Two rescue breaths are given first thereafter the CPR is initiated. As in most of the cases there

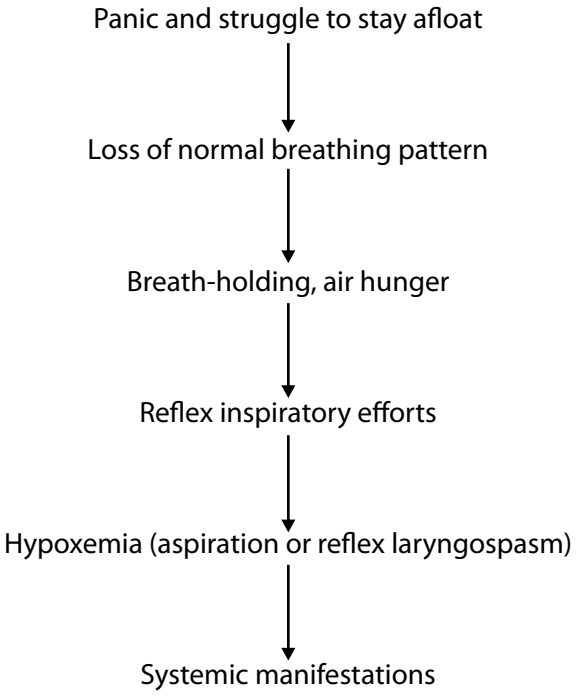



Figure 1 : Pathophysiology of Drowning

Table 1 : Systemic effects of Drowining

| | | |
|---|---|---|
| 1. | Respiratory | Non cardiogenic pulmonary edema, Acute respiratory distress syndrome |
| 2. | Cardiovascular | Hypothermia, Arrhythmias- sinus bradycardia, atrial fibrillation, ventricular arrhythmias and fibrillation (individuals with congenital long Q-T syndrome) |
| 3. | Neurologic | Neuronal damage, Cerebral edema |
| 4. | Metabolic | Metabolic/respiratory acidosis, hypernatraemia, hypermagnesemia and hypercalcaemia in sea water drowning |
| 5. | Renal | Haemoglobinuria, Myoglobinuria, Renal Failure |

is laryngospasm so by giving rescue breaths this laryngospasm is broken off.

Neck should be supported in a neutral position as chances of cervical spine injury may be high.

Administer high flow supplemental oxygen to the spontaneously breathing patient by facemask, while the apneic patient should be intubated.

Initiate rewarming in all hypothermic patients with a core temperature <33ºC either by passive or active means as available.

Heimlich manoeuvre delays the initiation of resuscitation and increases the risk of aspiration and should not be done.

**b. Emergency department management —** Prehospital resuscitative efforts should continue. Head and spinal cord injuries should be actively sought. Look for injuries to the axial skeleton as well as internal injuries to the abdomen and chest.

Remove wet clothing and continue rewarming with passive techniques (blankets or other insulators to reduce heat loss), active external measures ( warm blankets, heating pads, radiant heat, forced warm air, or warm baths applied directly to the patient's skin), and/or active internal rewarming (pleural and peritoneal irrigation with warm saline, continuous arteriovenous rewarming, hemodialysis, and cardiopulmonary bypass) until the temperature is 32 to 35ºC .

Electrocardiography and measurement of serum electrolytes should be done in all patients. Asymptomatic patients should be observed at-least for 8 hours and admitted if deterioration occurs.

Indications for intubation:

- neurologic deterioration and
- inability to maintain a PaO2 >60 mmHg on high fractions of supplemental oxygen.

**c. Inpatient management —** Ongoing supportive care and treatment of organ specific complications.

i. Neurologic injuries — the duration of loss of consciousness and the neurologic state of the patient upon presentation are major determinants of outcome.

The goal is to prevent secondary neurologic injuries due to ongoing ischemia, cerebral edema, hypoxemia, fluid and electrolyte imbalances, acidosis, and seizures.

- Maintain a PaCO2 of approximately 30 to 35 mmHg - Mild hyperventilation may reduce intracranial pressure by decreasing intracranial blood volume. Excessive hyperventilation can cause cerebral vasoconstriction, leading to decreased cerebral blood flow and worsening cerebral ischemia.
- Careful use of diuretics to avoid volume depletion which can lower cardiac output and cerebral perfusion.

- Control of Seizures- Phenytoin is the preferred anticonvulsant because it does not depress consciousness and neighther interferes with neurologic assessment. Neuromuscular blocking agents are to be avoided.
- Euglycemia and euthermia should be meticulously maintained.

ii. Respiratory failure — Chest radiographs may not reflect the severity of the pulmonary involvement.

- Bronchospasm is common in near-drowning patients, most cases rapidly improve with inhaled beta-adrenergic agonists.
- Antibiotics should be used only in cases of clinical pulmonary infection or if the patient was submerged in grossly contaminated water where a high suspicion for water-borne pathogens such as Aeromonas, Pseudomonas, and Proteus.
- Mechanical ventilation as in other cases of acute lung injury.
- No role of prophylactic steroids and surfactant therapy.

**iii. Hypotension** — hypothermia and hypovolemia can lead to hypotension due to a "cold diuresis" during the early phase of vasoconstriction resulting in decreased antidiuretic hormone production.

Hypoxic cardiomyopathy may occur in near-drowning patients

**Poor Prognostic Indicators:**

- Duration of submersion >10 minutes
- Time to effective basic life support >10 minutes
- Resuscitation duration >25 minutes
- Glasgow coma scale <5 (comatose)
- Age <3 years
- Persistent apnea and
- Requirement of CPR in the emergency department
- Arterial blood pH <7.1 upon presentation
- Water temperature >10°C (50°F) - Cold water is beneficial as it decreases the metabolic demands of the body and activates the diving reflex that shunts blood to the vital organs.
- The lack of spontaneous, purposeful movements at 24 hours is an ominous sign.

**Prevention**

Do not leave children, elderly and persons with seizure disorder unattended near swimming pools, ponds, full buckets in bathroom and in bath tubs.

Secure gating, fencing of swimming pools and availability of life guards at all times.

Always use safety gear, and personal floatation devices and follow instructions in the swimming pools and water parks.

Avoid use of alcohol and intoxicating drugs before venturing into water.

## Electrocution (Electrical Injuries)

Electrical injuries result when there is passage of electric current through any part of the body.

Electrocution is defined as sudden death due to delivery of electric current at the time of ventricular depolarisation. Immediate death may occur either from current-induced ventricular fibrillation or asystole or from respiratory arrest secondary to paralysis of the central respiratory control system or due to paralysis of the respiratory muscles. Presence of severe burns (common in high-voltage electrical injury), myocardial necrosis, the level of central nervous system injury and multi organ failure determine the subsequent morbidity and long-term prognosis.

Electrical current has an entry and an exit wound, whose location depends on the path taken by the current in the body and these should always be looked for. The extent of injury may not be fully apparent externally with significant damage to the

deep tissues and muscles.

The mechanism of injury is:

- Direct effect of electrical current on body tissues
- Conversion of electrical energy to thermal energy, resulting in deep and superficial burns
- Blunt mechanical injury from lightning strike, muscle contraction or as a complication of a fall after electrocution

The extent of injury depends upon:

- Type of current- Alternating current causes more damage than direct current due to stimulation of muscular contraction,
- Electrical Source- Low voltage, high voltage, lightning or electrical arc exposure.

  High-voltage electrical exposures usually cause severe burns whereas patients of

Table 2 : Comparison among lightning, high-voltage, and low-voltage electrical injuries

| | **Lightning** | **High voltage** | **Low voltage** |
|---|---|---|---|
| Voltage | V >30 x 10 6 | >1,000 | <600 |
| Current | A >200,000 | <1,000 | <240 |
| Duration | Instantaneous | Brief | Prolonged |
| Type of current DC | | DC or AC | Mostly AC |
| Cardiac arrest | Asystole | Ventricular fibrillation | Vent. fibrillation |
| Respiratory arrest | Direct CNS injury | Indirect trauma or tetany of resp. Muscles | Tetany of resp. Muscles |
| Muscles contraction | Single | Single (DC);Tetanic (AC) | Tetanic |
| Burns | Rare, superficial | Common, deep | Usually superficial |
| Rhabdomyolysis | Uncommon | Very common | Common |
| Blunt injury (cause) | Blast effect | Fall (muscle contraction) | Fall (uncommon) |
| Acute mortality | Very high | Moderate | Low |

Table 3 : Systemic manifestation of Electric injuries.

| | | |
|---|---|---|
| 1. | Cutaneous | Flash, thermal, linear, contact, feathering or arc burns |
| 2. | Musculoskeletal | Tetany, Myonecrosis, rhabdomyolysis, compartment syndrome |
| 3. | Vascular | Vasoconstriction, vasomotor spasm, thrombosis, ischaemia, necrosis, |
| 4. | Cardiac | Arrhythmias, Myocardial necrosis, Asystole, Fibrillation, Arrest |
| 5. | Respiratory | Pulmonary edema, respiratory paralysis/arrest |
| 6. | Gastrointestinal | Persistent ileus, visceral injuries, Curling's ulcers |
| 7. | Auditory | Tympanic membrane perforation, haemotympanum, CSF otorrhea |
| 8. | Ocular | Cataracts, Retinal detatchment, vitreous haemorrhage, optic nerve injury |
| 9. | Nerves | Paresis, paralysis, paraesthesias, chronic pain |
| 10. | Brain | Seizures, memory loss, personality changes, irritability, sleep disorders, infarcts, paraplegia, quadriplegia, post-traumatic stress disorder. |

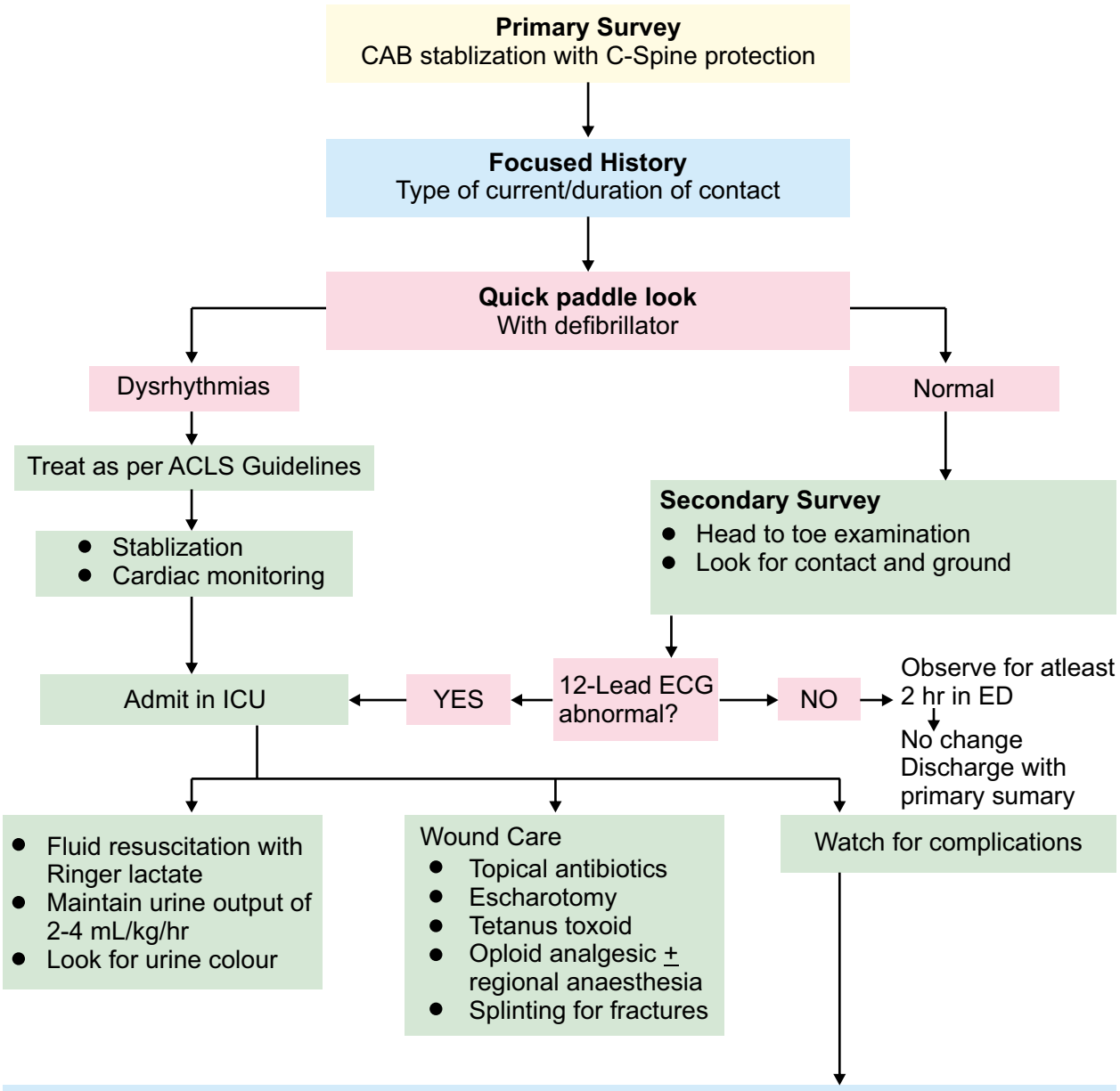



Figure 2 : Management of Electrical Injuries

lightning strikes may have no obvious physical injury but may present with cardiopulmonary arrest.

Low voltage injuries usually occur at home where is high voltage injuries are usually work related and may cause serious tissue destruction and secondary injuries.

Lightning injuries have a high mortality. Two-thirds of lightning-associated deaths occur within one hour of injury due to fatal arrhythmia or respiratory failure

- Path traversed by the current
- Resistance offered by the patient which depends on area of contact, pressure applied and the presence of moisture. Tissues with higher resistance heat up and coagulate rather than transmit current. Skin, bone, and fat have high resistances, while nerves and blood vessels have lower resistances. So skin bone and fat will coagulate where as nerve and blood vessel will transmit current Tetany may lead to sustained contact, and cardiac arrhythmias. Moisture lowers the resistance and facilitates transmission of current.

**Comparison among lightning, high-voltage, and low-voltage electrical injuries (Table 2)**

**Systemic Manifestations of Electrical Injuries**

The systemic manifestations of electrical injuries are described in Table 3

**Management of electrical Injuries**

Immediate switching off of the source of current with a non-conductor material like dry wooden stick or removal from the source.

Indications for Hospitalisation includes -

- Unconsciousness
- Arryhthmias
- Suspicion of deep tissue damage
- Metabolic acidosis
- Myoglobinuria or Renal failure
- Associated Traumatic injuries

Flow Chart for Management of Electrical Injuries (Figure 2)

**Prevention**

Use of high quality and safe electric points at home, to prevent children from inserting objects into them.

Maintenance of all electrical points and wires in good condition. Remove electrical equipment from plugs when not in use.

Keep electrical cables and switches away from the reach of children.

Use of protective gear at workplaces and adherence to safety norms.

Avoid touching any electrical point/cable or equipment with wet hands.

Educating children and adolescents about dangers of exploring electrical cables and connections.

# CHAPTER 12 POISONING

*"All substances are poisons; there is none that is not a poison; the right dose differentiates a poison from a remedy" – Paracelsus.*

The general management of these cases is supportive and herelies the importance of assessing airway, breathing and circulation of these patients.

## Introduction :

It is estimated that there are more than nine million synthetic and natural chemicals available today. Poisoning and deaths due to poisoning are on the rise over the years, despite advanced knowledge regarding their pharmacokinetics and pathology, and newer and better techniques being developed for the management of poisoning cases. The commonest agents in India are the pesticides, followed by sedatives, drugs, chemicals, alcohols, plant toxins and house-hold cleansing agents. Of late, aluminum phosphide has emerged as the commonest suicidal agent in Northern India.

## General Principles:

The general principles of management of poisoning cases, as we know are:

1. **Stabilization which includes assessment and management of**
   a) Airway and breathing
   b) Circulation, and
   c) Depression of the central nervous system
2. **Decontamination** including skin/eye decontamination, gut evacuation, etc.
3. **Poison Elimination** diuresis, peritoneal/ hemodialysis, haemoperfusion etc.
4. **Antidote administration:** As of now, antidotes are available for < 5% poisons
5. **Nursing and Psychiatric care:** Once the patient is stablized they are offered psychiatric care.

Gastrointestinal evacuation in use for centuries, is undergoing critical appraisal. The role of ipecac and gastric lavage are being questioned while activated charcoal is gaining importance in the management of such cases. Antidotal therapy is no more the mainstay of the management and the fact that we have antidotes for only about 5% poisons. Grouping the signs and symptoms produced by the poisons in to various toxidromes helps in rapid and effective management of the case.

### Gastric Decontamination (See Table 1):

Interference with absorption of ingested poison from the gastrointestinal tract is the mainstay of poison management. Because few specific antidotes are available to treat poisonings, absorption prevention, observation, and supportive care are the clinician's greatest assets. The challenge for clinicians managing poisoned patients is to identify those who are most at risk of developing serious complications and who might potentially benefit from gastrointestinal decontamination.

### a) Gastric Lavage

Stomach emptying by gastric lavage has been in use in the management of poisoning by ingestion, for almost 200 years. The effectiveness of gastric lavage in removing stomach contents is time dependent and best results are obtained when performed within one hour. Unfortunately, many overdose patients do not arrive to the emergency department within this valuable one hour. Although emptying the stomach in the first hour generally works and may be beneficial for up to a certain period of time thereafter, it is usually not helpful beyond 4hours of ingestion. Sadly, the same is not applied in our country. Irrespective of the time gap, gastric lavage is performed in most of the hospitals, as the initial part of the treatment. Legal

requirement, necessitating the preservation of a sample of the return lavage fluid for toxicological analysis, plays an important role in the continued use of this technique, despite its efficacy being highly questionable. Gastric lavage carries potential complications, including aspiration pneumonitis and, rarely, esophageal perforation. It can also promote the rapid passage of tablets into the small bowel rather than removing them. Studies have now shown that gastric lavage did not prove any more beneficial than activated charcoal, alone.

**b) Activated Charcoal:**

Produced by pyrolysis of carbon containing materials and activated by oxidation with steam at a high temperature, these processed carbon products adsorb many drugs. In addition to direct intra-luminal binding, activated charcoal can also decrease the resorption of agents that undergo enterohepatic or enterogastric cycling. It has a "gastrointestinal dialysis" effect, whereby the charcoal serves as a large "sink" with movement of toxin molecules across semi-permeable membranes from splanchnic circulation. During the last decade, activated charcoal has became increasingly popular as a first-line agent for the treatment of poisonings, particularly if more than several hours have passed since ingestion. It is generally considered ineffective against caustics, ethanol, ethylene glycol, methanol, iron lithium, metals, and petroleum distillates (Table 2). Usually complications of its use are rare, but they include aspiration of activated charcoal and gastric contents, as well as intestinal obstruction, particularly when repeated doses of activated charcoal are given. The use of multiple-dose activated charcoal (MDAC) is now recommended for the clearance of drugs such as -- carbamazepine, digitoxin, glutethimide, nadolol, phenobarbital, phenylbutazone, theophylline, and others. Multiple dosing appears to decrease both the absorption and blood concentration of many drugs. The multiple-dose regimen consists of an initial dose of 50-100g followed by maintenance doses of 30-50 g every 2-6 h with or without the administration of a cathartic agent. Maintaining a constant amount of activated charcoal in the gut adsorbs the toxin as it is secreted back in to the gut, thereby preventing a delayed peak in the serum concentration.

**3) Cathartics:**

Catharsis actually means purification and this is achieved in the poisoning cases by purging the gastrointestinal tract of all the poisonous material. This is the premise that promotes their use for the rectal evacuation of gastric contents - both the drug and the drug charcoal complex. Despite their widespread use, however, little evidence exists that cathartics alter the outcome of poisoned patients. The most commonly used cathartics are magnesium sulfate, magnesium citrate and sorbitol. Sorbitol works most quickly; causing bowel movements within one hour. Contraindications to cathartics include caustic ingestions and signs of intestinal obstruction. If being considered, one dose is generally sufficient.

**Substances not readily adsorbed to activated charcoal**

- Ferrous salts
- Acids
- Lithium preparations
- Alkalies
- Potassium salts
- Fluorides
- Ethanol
- Organic solvents
- Methanol
- Mercury and its salts
- Ethylene glycol
- Lead and its salts

**Indications for multiple dose activated charcoal (MDAC)**

- Slow release preparations such as theophylline (but not lithium)
- Carbamazepine
- Dapsone
- Digoxin
- Paraquat
- Phenobarbitone
- Quinine
- Amanita phalloides

**d) Whole - Bowel Irrigation:**

Polyethylene glycol-electrolyte solutions, which once were used for bowel cleansing before surgical

Table 1 : Techniques of gastric decontamination.

| Treatment | Indications |
|---|---|
| Gastric Lavage | Ingested a potentially life – threatening amount of a poison and the procedure can be undertaken within 60 minutes of ingestion. |
| Activated charcoal | May be considered if a patient has ingested a potentially toxic amount of the poison (known to be adsorbed by charcoal) up to 1 hour previously; there are insufficient data to support or to exclude its use after 1 hr of ingestion. |
| Ipecacuanha | Its routine administration in the emergency department should be abandoned; there are insufficient data to support or to exclude its administration soon after ingestion. |
| Whole–bowel irrigation | May be considered for potentially toxic ingestion of sustained release or enteric coated drugs; there are insufficient data to support or to exclude its use for potentially toxic ingestion of iron, lead, zinc, or packet of illicit drugs (body – packer) |
| Cathartics | The administration of a cathartic alone has no role in the treatment of a poisoned patient and is not recommended as a method of gut decontamination |

procedures are now being applied for gastrointestinal decontamination. These iso-osmotically balanced, non-absorbable solutions are safe, causing no fluid retention or electrolyte disturbances. The procedure has been advocated for overdoses of agents such as iron, lithium, arsenic, lead-oxide and enteric-coated or sustained-release medications. In practice, hemodynamically stable and cooperative patients are best suited for this intensive cathartic treatment. Adults should be given the solution at a rate of 2 L/h, children at 500 ml/h, either orally or through a nasogastric tube. The endpoint of treatment is a clear effluent, which may take 4-6 hours to appear. Contraindications include ileus or bowel obstruction, hemodynamic instability or where airway cannot be protected.

**e) Emesis :**

Ipecac syrup has long been used as a first-line agent for prevention of toxicity from ingested poisons, especially in children. However, this is not freely available in our country and its effectiveness in recovering ingested substances is poor and its ability to reduce the severity of poisoning has never been demonstrated. Moreover, the emesis induced by ipecac may preclude the use of other oral treatment options. It is contraindicated in ingestion of caustic substances and volatile hydrocarbons, in patients who have decreased gag reflex or altered mental status, and in patients at risk for rapid alteration in consciousness. Complications of ipecac include aspiration pneumonia, lethargy, diaphragmatic rupture, Mallory-Weiss esophageal tears and cerebral hemorrhages. Ipecac is also still recommended by poison control centers for usein the home, where early administration can be assured.

**Laboratory Tests:**

Although laboratory analysis of various body fluids of overdose patients frequently identifies substances that are clinically unsuspected, these additional findings rarely alter the patient's clinical course. This is largely because of the presence of a substance that does not necessarily correlate with acute toxicity. Moreover, analysis can be time-consuming and in most clinical settings falls short of being comprehensive. Most poisoned or overdose patients do well with supportive care alone. Again, no rapidly available universal screening tool exists and many patients require little, if any, laboratory investigation.

**Rational use of Antidotes :**

Antidotes are chemical or physiological antagonists that prevent the toxicological effect of specific poisons. In most toxicological emergencies, effective antidotes are not available. Symptomatic treatment and supportive care are still the primary approach to treatment; antidotal therapy often plays a relatively minor role. When appropriately used in specific situations, however, antidotes can substantially reduce morbidity and mortality in the poisoned patient. The coma cocktail (which include 50% dextrose, naloxone, thiamine) is common and appropriate to be used in coma patients.

**Organophosphorus poisoning**

Organo-phosphorus pesticide poisoning is an important clinical problem in rural regions of the developing world, and kills an estimated 2,00,000 people every year. Medical management is difficult, with case fatality generally more than 15%. 50 years after first use, we still do not know how the core treatments—atropine, oximes, and diazepam—should best be given. Important constraints in the collection of useful data have included the late recognition of great variability in activity and action of the individual pesticides.

Table 2 - Antidotes

| Drug/poison | Antidotes |
|---|---|
| Acetaminophen | N-acetylcysteine |
| Anticholinergics | Physostigrnine |
| Anticholinesterases | Atropine |
| Benzodiazepines | Flumazenil |
| Black widow spider bite | Equine-derived antivenom |
| Carbon monoxide | Oxygen |
| Coral snake (Eastern and Texas) bite | Equine-derived antivenom |
| Cyanide | Amyl nitrite, sodium nitrite, sodium thiosulfate, hydroxycobalamin |
| Digoxin | Digoxin-specific antibodies |
| Ethylene glycol | Ethanol/fomepizole, thiamine, and pyridoxine |
| Heavy metals (arsenic, copper, gold, lead, mercury) | Dimercaprol (BAL), EDTA, penicillamine |
| Hypoglycemic agents | Dextrose, glucagon, octmotide |
| Iron | Deferoxamine mesylate |
| Isoniazid | Pyridoxine |
| Methanol | Ethanol or fomepizole, folic acid |
| Methemoglobinemia | Methylene blue |
| Opicids | Naloxone |
| Organophosphalc | Atropine, pralidoxime |
| Rattlesnake bite | Equine-derived antivenm |

*EDTA-othylenediamine tetra acetic acid.

However, consensus suggests that early resuscitation with atropine, oxygen, respiratory support, and fluids is needed to improve oxygen delivery to tissues. The role of oximes is not completely clear; they might benefit only patients poisoned by specific pesticides or patients with moderate poisoning. Small studies suggest benefit from new treatments such as magnesium sulphate, but much larger trials are needed. Gastric lavage could have a role but should only be undertaken once the patient is stable. For understanding the basic principle of management of OP poisoning the activity of the autonomic nerves system of the body is essential this is described in Table 5.

**Pathophysiology**

Organo-phosphorus pesticides inhibit esterase enzymes, especially acetylcholinesterase in synapses and on red-cell membranes, and butyrylcholinesterase in plasma. Acetylcholinesterase inhibition results in accumulation of acetylcholine and overstimulation of acetylcholine receptors in synapses of the autonomic nervous system, CNS, and neuromuscular junctions.

The subsequent autonomic, CNS, and neuromuscular features of organo-phosphorus poisoning are well known as describe previously. Diagnosis is made on the basis of clinical suspicion, the characteristic clinical signs, smell of pesticides or solvents, and reduced butyrylcholinesterase or acetylcholinesterase activity in the blood. Patients with severe Organo-phosphorus poisoning typically present with pinpoint pupils, excessive sweating, reduced consciousness, and poor respiration. The major differential diagnosis is carbamate poisoning, which is clinically indistinguishable.

**Cholinesterase assays**

Studies suggest that red-cell acetylcholinesterase is a good marker of synaptic function and atropine needs in patients poisoned with organo-phosphorus, and is therefore probably a good marker of severity. Patients with red-cell acetylcholinesterase activity of at least 30% had normal muscle function and no need for atropine. By contrast, patients with less than 10% of normal red-cell acetylcholinesterase activity had grossly deranged muscle function and needed high doses of atropine. Acetylcholinesterase activity between these values was associated with moderate impairment of muscle function and subsequent need for atropine.

Table 5 : Actions of sympathtic/cholinergic nervous system on various body organs

| | Adrenergic | Anticholinergic | Cholinergic syndrome |
|---|---|---|---|
| **Vitals** | | | |
| RR | Increased | No change | Increased |
| HR | Increased | Increased | Decreased |
| Temperature | Increased | Increased | No change |
| BP | Increased | Increased/no change | No change |
| **Physical-Examination** | | | |
| Mental status | Alert/Agitated | Depressed/confused/ | Depressed/confused |
| Pupil | Dilated | hallucinating | Constricted |
| Mucous membranes | Wet | Dilated | Wet |
| Skin | Diaphoretic | Dry | Diaphoretic |
| Reflexes | Increased | Dry | Normal/decreased |
| Bowel sounds | Increased | Normal | Increased |
| Urination | Increased | Decreased | Increased |
| Other | Possible seizures | Decreased Possible seizures | Muscle fasciculations/ seizures |

**Clinical features of Organo-phosphorus pesticide poisoning**

**i) Features due to overstimulation of muscarinic acetylcholine receptors in the parasympathetic system**

- Bronchospasm
- Bronchorrhoea
- Miosis
- Lachrymation
- Urination
- Diarrhea
- Hypotension
- Bradycardia
- Vomiting
- Salivation

**ii) Features due to overstimulation of nicotinic acetylcholine receptors in the sympathetic system**

- Tachycardia
- Mydriasis
- Hypertension
- Sweating

**iii) Features due to overstimulation of nicotinic and muscarinic acetylcholine receptors in the CNS**

- Confusion
- Agitation
- Coma
- Respiratory failure

**iv) Features due to overstimulation of nicotinic acetylcholine receptors at the neuromuscular junction**

- Muscle weakness
- Paralysis
- Fasciculation's

**Principles of therapy**

Treatment includes resuscitation of patients and supplemental oxygen, a muscarinic antagonist (usually atropine), fluids and an acetylcholinesterase re-activator (an oximes that reactivates acetylcholinesterase by removal of the phosphate group).Respiratory support is given as and when necessary. Gastric decontamination should be considered once the patient has been fully resuscitated and stabilized. Patients must be carefully observed after stabilization for change in atropine needs and worsening respiratory function because of intermediate syndrome and recurrent cholinergic features occurring with fat-soluble organo-phosphorus.

**Initial stabilisation**

Severe acute organophosphorus pesticide poisoning is a medical emergency. Treatment must ensure that the patient has a patent airway and adequate breathing and circulation. Ideally, oxygen should be provided at the first opportunity. However, little evidence supports the common advice that atropine must not be given until oxygen is available. In hospitals that have no access to oxygen, atropine should be given early to patients with pesticide poisoning to reduce secretions and improve respiratory function. The patient should be placed in the left lateral position, with the neck extended. This position reduces risk of aspiration, helps keep the air way patent, and could decrease pyloric emptying and absorption of poison. Supportive care should include giving fluids and control of blood glucose.

Gastrointestinal decontamination

Gastric lavage is often the first intervention poisoned patients receive on presentation to hospital, sometimes at the expense of resuscitation and giving antidote. No evidence shows any form of gastric decontamination to benefit patients poisoned with organophosphorus. Gastric decontamination should only be done after the patient has been stabilised and treated with oxygen, atropine, and an oxime.

Gastric lavage is the most common form of decontamination for organophosphorus poisoning despite the absence of randomised controlled trials to confirm benefit. The rate of absorption of organophosphorus from the human bowel is not known. However, with some pesticides, the rapid onset of poisoning in animals suggests that absorption is rapid, occurring within minutes of ingestion. The time window for effective lavage is therefore probably short. Guidelines for treatment of drug self-poisoning suggest that lavage should be considered only if the patient arrives within 1

hour of ingestion of poison. The relevance of these guidelines to organophosphorus poisoning is unclear but lavage should probably only be considered for patients who present soon after ingestion of a substantial amount of toxic pesticide who are intubated, or conscious and willing to cooperate.

Ipecacuanha-induced emesis should not be used in organo phosphorus pesticide poisoning.Patients poisoned with organophosphorus can rapidly become unconscious, risking aspiration if ipecacuanha has been given. Mechanically-induced emesis with large quantities of water risks pushing fluid through the pylorus and into the small bowel, probably increasing the rate of absorption.

A randomised controlled trial of single and multiple doses of super-activated charcoal in Sri Lanka failed to find a significant benefit of either regimen over placebo in more than 1000 patients poisoned with pesticides. Because activated charcoal binds organo-phosphorus in vitro, the absence of effect in patients might be due to rapid absorption of pesticide into the blood. Alternatively, the ingested dose in fatal cases could be too large for the amount of charcoal given, the charcoal might be given too late, or the solvent might interfere with binding. No evidence suggests that patients with pesticide poisoning benefit from treatment with activated charcoal.

Muscarinic antagonist drugs

Although atropine remains the mainstay of therapy worldwide, other muscarinic antagonists have been studied in animals. An important difference between such drugs is their penetration into the CNS.The main adverse-effect of atropine is anticholinergic delirium in patients who receive too high a dose. Some physicians therefore prefer glycopyrronium to treat the peripheral effects of organo phosphorus without causing confusion. However, its poor CNS penetration suggests that it is ineffective at countering coma and reduced respiration seen in patients with the cholinergic syndrome.

Oximes

Oximes reactivate acetylcholinesterase inhibited by organophosphorus. Pralidoxime was discovered in the mid-1950s by Wilson and colleagues, and was soon successfully introduced into clinical practice for patients with parathion poisoning. Other oximes, such as obidoxime and trimedoxime, have been developed but pralidoxime remains the most widely used.

WHO recommends that oximes be given to all symptomatic patients who need atropine. To ensure a therapeutic concentration, a loading dose of pralidoxime chloride or obidoxime is given, then a continuous infusion. The loading dose of oximes should not be given rapidly as a bolus because this method causes vomiting (risking aspiration), tachycardia and diastolic hypertension. Oximes are effective against diethyl compounds such as parathion and malathion.

**Bed side management of OP poisoning patient**

- Check airway, breathing, and circulation. Place patient in the left lateral position, preferably with head lower than the feet, to reduce risk of aspiration of stomach contents. Provide high fl ow oxygen, if available. Intubate the patient if their airway or breathing is compromised
- Obtain intravenous access and give 1–3 mg of atropine as a bolus, depending on severity. Set up an infusion of 0.9% normal saline; aim to keep the systolic blood pressure above 80 mm Hg and urine output above 0.5 mL/kg/h
- Record pulse rate, blood pressure, pupil size, presence of sweat and auscultatory findings at the time of first atropine dose
- Give pralidoxime chloride 2 g intravenously over 20–30 min into a second cannula; followed by an infusion of pralidoxime 0.5–1 g/h in 0.9% normal saline
- 5 minutes after giving atropine, check for pulse, blood pressure, pupil size, sweat and chest sounds. If no improvement has taken place, give double the original dose of atropine
- Continue to review after every 5 minutes; give doubling doses of atropine if response is still absent. Once parameters have begun to improve, cease dose doubling. Similar or smaller doses can be used

- Give atropine boluses until the heart rate is more than 80 beats per minute, the systolic blood pressure is more than 80 mm Hg, and the chest is clear (appreciating that atropine will not clear focal areas of aspiration). Sweating stops in most cases.

  A tachycardia is not a contraindication to atropine since it can be caused by many. The pupils will commonly dilate; however, this sign is not a useful endpoint for initial atropine treatment because a delay exists before maximum effect.

- Clinical judgment is needed about additional doses of atropine if the heart rate and blood pressure are slightly below their targets but the chest is clear. More atropine at this point might not be needed. Severe hypotension might benefit from vasopressors.

- Once the patient is stable, start an infusion of atropine giving every hour about 10–20% of the total dose needed to stabilise the patient. Check the patient often to see if too much or too little atropine is being given. If too little is given, cholinergic features will re-emerge after some time. If too much is given, patients will become agitated and pyrexial and develop absent bowel sounds and urinary retention. If this happens, stop the infusion and wait 30–60 min for these features to settle before starting again at a lower infusion rate

- Continue the oxime infusion until atropine has not been needed for 12–24 h and the patient has been extubated

- Continue to review respiratory function. Intubate and ventilate patients if tidal volume is below 5 mL/kg or vital capacity is below 15 mL/kg, or if they have apneic spells or PaO2 is less than 8 kPa (60 mm Hg) on FIO2 of more than 60%

- Assess flexor neck strength regularly in conscious patients by asking them to lift their head off the bed and hold it in that position while pressure is applied to their forehead. Any sign of weakness is a sign that the patient is at risk of developing peripheral respiratory failure (intermediate syndrome). Tidal volume should be checked every 4 h in such patients. Values less than 5 mL/kg suggest a need for intubation and ventilation

- Treat agitation by reviewing the dose of atropine being given and providing adequate sedation with benzodiazepines. Physical restraint of agitated patients in warm conditions risks severe hyperthermia which is exacerbated greatly by atropine because it inhibits normal thermoregulatory responses, including sweating. Adequate sedation is therefore important

- Monitor frequently for recurring cholinergic crises due to release of fat soluble organo-phosphorus from fat stores. Such crises can occur for several days to weeks after ingestion of some organo-phosphorus. Patients with recurring cholinergic features will need retreatment with atropine and oxime.

# CHAPTER 13 SNAKE BITE

## Who is at risk ?

A patient who presents with a history of a snake bite, may have witnessed the organism (bite being either venomous or non-venomous) or might not have witnessed the organism (history of a bite suspected to be that of a snake). In some cases where bites are painless and occur during sleep, presenting symptoms suggest a snake-bite, while no history is available.

Vipers and kraits are more common in Central and South India. Cobra (Elapids) are more common in North India.

While species distinction is often difficult based on description, but may be done if patients bring the snake alongwith. The three common venomous snakes are as follows:

It is not possible to identify a poisonous from a non-poisonous snake by appearance, as many non-poisonous species appear similar to those above. Most patients of snake bite do not notice these features due to anxiety, it bite in dark, or the organism quickly slipping away after the bite.

**Situations where snake-bite usually occurs include:**

- Farmers (during ploughing, preparing ground, or in paddy fields)

| Viper | Cobra | Krait |
|---|---|---|
|  |  |  |
| Short, thick, colored with a pattern on the back | Long, thin uniformly colored. May stand on the ground and flatted their head to form a hood. | Short, thin, white under belly, spotted or striped at the top |

Figure : Types of poisonous snakes and their characteristics

- Walking in the dark (accidentally stepping on a snake)
- Rural areas or urban slums (especially if sleeping on floor)
- Garbage handlers (while picking up foliage with hands)
- Herdsmen, hunters, snake handlers, snake-cathers

**Clinical presentation of a snake-bite :**

Initial assessment begins with the following questions:

a) Where were you bitten ?

a. Evaluate for local symptoms (if present)

- Pain, swelling, redness, heat
- Fang marks
- Local bleeding, bruising, blistering, necrosis
- Lymphangitis or lymph node enlargement

b) How were you bitten ?

- Evaluate for situation
- Nocturnal bite, while sleeping (Krait)
- While working in a wet area, paddy fields (Viper)

c) How are you feeling now ?

- Bleeding manifestations
- Epistaxis, Hematuria, Hemptysis or Hematemesis
- Hemoglobinuria
- Conjunctival bleed
- Suggestive of Acute Kidney Injury (AKI) (usually late)
- Anasarca, oliguria, pulmonary edema
- Neurological Symptoms
- Ptosis, diplopia, opthalmoplegia
- Poor neck holding, Respiratory paralysis, general flaccid paralysis

**20-minute whole blood clotting test (20WBCT)**

This is an extension of clinical examination to detect bleeding manifestation. Delayed 20WBCT has been shown to correlate with serum Fibrinogen levels. It is performed in the following steps:

- Place 2 mls of freshly sampled venous blood in a small, new or heat cleaned, dry, glass vessel.
- Leave undisturbed for 20 minutes at ambient temperature.
- At 20 minutes if the blood is still liquid (unclotted) and runs out, the patient has hypofibrinogenaemia ("incoagulable blood") as result of venom-induced consumption coagulopathy.
- This test is diagnostic of a viper bite.
- Please ensure that the test-tube is dry, and has not been cleaned with a detergent.
- If there is any doubt, repeat the test in duplicate, including a "control" (blood from a healthy person such as a relative)

One or more of these symptoms occur with different species of snakes, as different species have different venom characteristics:

- Syndrome 1 (Vipers)
  - Local envenoming
  - Bleeding/clotting disturbances
- Syndrome 2 (Russel's viper)
  - Local envenoming
  - Bleeding/clotting disturbances
  - Acute kidney injury, Hemoglobinuria
  - Ptosis, opthalmoplegia, paralysis
- Syndrome 3(cobra or king cobra)
  - Local envenoming

- Paralysis

- Syndrome 4 (Kraits)
  - No local envenoming
  - Paralysis
- Syndrome 5 (Some Kraits / Vipers)
  - No local envenoming
  - Paralysis
  - Acute kidney injury, Hemoglobinuria

In addition many patients present with effects of anxiety (frightened, hysterical, or even in delirium) with tachycardia, perspiration and fainting. This is usually due to exaggerated beliefs about the potency and speed of action of snake venoms. Some patients may have cut marks, or swelling caused by effects of first-aid (incision or tourniquet application).

## Management of snake-bite

Management of Snake bite is described in the following steps:

**1. First aid treatment**

a. Reassure the patient who may be very anxious

b. Make the person comfortably lie down

c. Immobilize the bitten limb with a splint or sling.

d. If elastic bandage is available, may use a pressure-immobilization or pressure pad (application of a pressure bandage, from proximal to site of bite to one joint distal.

e. Avoid any interference with the bite wound. Do not incise, rub, vigorous cleaning, massage, or application of herbs or chemicals.

f. Above methods fastens the absorbtion of venom into the circulation.

g. Do not apply any tourniquets proximal to the site of the bite. This is because when released, it will result in a faster systemic response. Tourniquets may also lead to gangrene.

**2. Transport to hospital**

a. Transport the person to a nearest hospital / health facility with the fastest available means of transport.

b. Ensure that there is minimal muscular contractions / movements during transportation.

**3. Rapid clinical assessment**

a. Perform a rapid assessment of the patient

i. Airway

ii. Breathing

iii. Circulation

iv. Level of consciousness

b. Initiate emergency treatment protocols for airway compromise (respiratory paralysis, flaccid paralysis) which may lead to pooling of secretions. Need to secure the airway.

c. If hypoxia is present, initiate bag-mask-ventilation, oxygen, and invasive ventilator support if airway has been secured.

d. Look for hypotension, initiate fluid therapy.

e. Evaluate level of consciousness. Often with flaccid paralysis, patients appear unresponsive, but are conscious.

**4. Identification if the bite is venomous**

a. Early clues of a severe envenomation

- Snake identified as a very dangerous one.
- Rapid early extension of local swelling from the site of the bite beyond the proximal joint.

- Early tender enlargement of local lymph nodes, indicating spread of venom in the lymphatic system.
- Early systemic symptoms: collapse (hypotension, shock), nausea, vomiting, diarrhoea, severe headache, "heaviness" of the eyelids, inappropriate (pathological) drowsiness or early ptosis/ophthalmoplegia.
- Early spontaneous systemic bleeding.
- Delayed 20WBCT
- Passage of dark brown/black urine.

b. Identification of a bite as non-venomous

- Absence of any local reaction, and observation of the patient for upto 4-8 hours after a snake-bite does not show any evidence of either a coagulopathy or a neurological manifestation.
- A minimal local reaction (such as swelling, which may be due to effects of first-aid such as incision or tourniquet) which does not extend beyond a single joint space, and absence of any systemic features (bleeding or neurological features)

When in doubt, consider a bite to be venomous and treat accordingly

**5. Antivenom treatment**

- Polyvalent anti-snake venom serum" (ASV) is raised in horses using the venoms of the four most important venomous snakes in India (Indian cobra, *Naja naja*; Indian krait, *Bungarus caeruleus* ; Russell's viper, *Daboia russelii*; saw-scaled viper, *Echis carinatus*)
- Antivenom treatment is recommended if and when a patient with proven or suspected snake-bite develops one or more of the following signs:
- Systemic envenomation.
- Haemostatic abnormalities: Spontaneous systemic bleeding (clinical coagulopathy evidenced by 20WBCT)
- Neurotoxic signs: ptosis, external ophthalmoplegia, paralysis etc
- Cardiovascular abnormalities: hypotension, shock, cardiac arrhythmia (clinical), abnormal ECG.
- Acute kidney injury (renal failure): oliguria/anuria (clinical), rising blood creatinine/ urea (laboratory). (Haemoglobin-/myoglobin-uria:) dark brown urine (clinical), urine dipsticks,
- Local envenomation
- Local swelling involving more than half of the bitten limb (in the absence of a tourniquet) within 48 hours of the bite.
- Swelling after bites on the digits (toes and especially fingers).
- Rapid extension of swelling (for example, beyond the wrist or ankle within a few hours of bite on the hands or feet).
- Development of an enlarged tender lymph node draining the bitten limb

Do not administer ASV to patients who do not have evidence of envenomation. Even small dosages can lead to anaphylaxis, which is frequent with ASV administration.

- There are no contraindications to ASV. It can be given in pregnancy and even to small children.
- Test dose for sensitivity is not required, however pre-loaded adrenaline, and diphenhydramine should be available in case anaphylaxis occurs.
- Obtain a IV access, with free flowing fluid (Normal Saline) before ASV is dissolved. Obtain IV access on a limb which has not been bitten or is swollen.

- Snakes inject the same dose of venom into children and adults. Children must therefore be given exactly the same dose of antivenom as adults.
- Reconstitute 10 vials of ASV with normal saline (total of 100mL). Give 100mL of ASV diluted in 400mL of normal saline. This is to be infused over one hour.
- Donot infilterate any ASV over the wound.
- Donot give ASV as intramuscular injection (unless no other way is possible). If it has to be given as IM, divide the dose into various sites (not more than 10ml at each site, and massage each site vigorously after drug is administered).
- Patients must be closely observed for at least one hour after starting intravenous antivenom administration, so that early anaphylactic antivenom reactions can be detected and treated.
- Usually within 10-180 minutes of starting antivenom, the patient begins to itch (often over the scalp) and develops urticaria, dry cough, fever, nausea, vomiting, abdominal colic, diarrhoea and tachycardia. A minority of these patients may develop severe life-threatening anaphylaxis such as hypotension, bronchospasm and angio-oedema.
- If anaphylaxis occurs:
    - Antivenom administration must be temporarily suspended.
    - Epinephrine (adrenaline) (0.1% solution, 1 in 1,000, 1 mg/ml) SC/IV is the effective treatment for early anaphylactic and pyrogenic antivenom reactions
    - Diphenhydramine (adults 10 mg, children 0.2 mg/kg by intravenous injection over a few minutes) should be given
    - Intravenous hydrocortisone (adults 100 mg, children 2 mg/kg body weight) is also given. It is unlikely to act for several hours, but may prevent recurrent anaphylaxis.
    - Restart ASV infusion at a slower rate after giving loading dosages.

**6. Observing the response to initial antivenom dose.**

a. General : The patient feels better. Nausea, headache and generalized aches and pains may disappear very quickly. This may be partly attributable to a placebo effect.

b. Spontaneous systemic bleeding (e.g. from the gums): This usually stops within 15-30 minutes.

c. Blood coagulability (as measured by 20WBCT): This is usually restored in 3-9 hours. Bleeding from new and partly healed wounds usually stops much sooner than this.

d. In shocked patients: Blood pressure may increase within the first 30-60 minutes and arrhythmias such as sinus bradycardia may resolve.

e. Neurotoxic envenoming of the post-synaptic type (cobra bites) may begin to improve as early as 30 minutes after ASV, but usually takes several hours. Envenoming with presynaptic toxins (kraits and sea snakes) will not respond in this way.

f. Active haemolysis and rhabdomyolysis may cease within a few hours and the urine returns to its normal colour.

**7. Deciding whether further dose(s) of antivenom are needed**

a. Duration of ASV treatment remains undefined. However patients need to be monitored for 24-48 hrs after the initial dose

for signs of envenomation. Further dosages of 100ml each are further advised in following situations:

i. If blood remains un-coaguable (WBCT20) after 6 hrs of initial dose

ii. If neurological features recur after 1-2 hrs

iii. If patient continues to bleed

iv. Worsening of cardiovascular parameters.

**8. Supportive/ancillary treatment**

a. Management of neurotoxicity

i. Assess for respiratory paralysis (tachypnea, shallow breathing, hypoxia, respiratory acidosis)

ii. Secure an airway

1. Airway with Bag-mask-ventilation at the site

2. Endotracheal intubation / supra-glottic device at a facility

iii. Assisted ventilation

1. Ambu bag at the site

2. Transport ventilators during journey to hospital

3. Mechanical ventilation for respiratory paralysis

iv. Perform a neuromuscular drug test

1. Give 0.6mg of Atropine (1ml) IV stat, followed by

2. 10mg of edrophonium (or 0.25mg/kg) and look for improvement in ptosis / neurological signs over next 10-20 minutes

3. If edophonium is not available (as is usual case) you ay use Inj neostigmine 0.02mg/kg IV (1mg for a 50kg person). Look for response in 30-60 minutes.

4. If responsive, continue to give Inj neostigmine 0.5 to 2.5mg IV after every hour (maximum of 10mg/day) to maintain response, along-with Inj Atropine 0.6mg IV after every four dosages.

5. Continue neostigmine in responsive patients, till features of neurotoxicity persist.

b. Management of bleeding manifestations

i. ASV remains mainstay of therapy for bleeding manifestations.

ii. Use of fresh frozen plasma/ platelet transfusion if bleeding is severe.

iii. No arterial punctures

iv. No heparin /anti-fibrinolytics

c. Management of acute kidney injury

i. Recognize acute kidney injury

1. Oliguria (Urine output of <400ml/day or less than 20ml/hr)

2. Proteinuria

3. Hematuria / Hemoglobinuria

4. Rise in serum Creatinine levels (more than 25% of baseline)

ii. IV fluid challenge and diuretics

1. Provide IV fluids (Preferred 0.9% Saline), about 2 liters over 60-90 minutes. See for rise in CVP / resolution of oliguria.

2. If oliguria persists give 100mg of Frusemide stat, and observe over 30-60 minutes for resolution of oliguria. If it persists give a second dose of 200mg. If oliguria still persists, donot give additional dosages of frusemide.

iii. If oliguria is unresponsive to fluids and diuretics

1. Fluid restriction to less than

previous days urine output + 500mL.

2. If hyperkalemia (serum K+ >5.5meq/L)
   a. 10ml of 10% Calcium gluconate IV over 5 minutes followed by
   b. 15U regular insulin in 100ml of 25% dextrose IV over 30 minutes. Repeat as required.
3. Plan for renal replacement therapy
   a. Indications
      i. Severe acidosis (pH <7.2)
      ii. Volume overload
      iii. Persistent hyperkalemia
      iv. Oliguria persists
   b. Modalities
      i. Hemodialysis
      ii. Peritoneal dialysis

**9. Treatment of the bitten part**

i. Clean and dress the wound

ii. Donot puncture the blisters

iii. Watch for swelling / compartment syndrome

iv. The appearance of an immobile, tensely swollen, cold and apparently pulseless snake-bitten limb may suggest the possibility of increased intracompartmental pressure, especially if the digital pulp spaces or the anterior tibial compartment are involved. Swelling of envenomed musclewithin such tight fascial compartments could result in an increase in tissue pressure above the venous pressure, resulting in ischaemia.

v. Fasciotomy is indicated if there is a definite compartment syndrome, and there are no ongoing bleeding abnormalities.

**How to prevent ?**

Snake-bites are preventable by some of the following measures:

- In the house: Be specially vigilant about snake bites after rains, during flooding, at harvest time and at night.
- Snakes may enter the house in search of food or to find a hiding place for a while. Donot keep uncovered food, as it allows rats to breed, and snakes may come in pursuit.
- Do not keep livestock, especially chickens, in the house, as snakes may come to hunt them.
- Cover all cracks and crevices in a house. These may be hiding places for a snake.
- If possible, try to avoid sleeping on the ground. If you have to sleep on the ground use an insecticide-impregnated mosquito net that is well tucked in under the mattress or sleeping mat. This will protect against mosquitoes and other biting insects, centipedes, scorpions and snakes.
- Around the house : Clear termite mounds, heaps of rubbish, building materials etc. Do not have tree branches touching the house.
- Keep grass short or clear the ground around your house and clear low bushes in the vicinity so that snakes cannot hide close to the house.
- Use a light when you walk outside the house or visit the latrine at night. especially after heavy rains.
- In rainy season walk with proper shoes or boots and long dress especially when walking in the dark or in undergrowth.
- Do not put hands into holes or nests or any hidden places where snakes might rest.
- On the road: Drivers or cyclists should never intentionally run over snakes on the road. The snake may not be instantly killed and may lie injured and pose a risk to pedestrians. The snake may also be injured and trapped under the vehicle, from where it will crawl out once the vehicle has stopped or has been parked in the house compound or garage.

(Source: WHO Guidelines for management of Snake bites 2010)

Note

Note

Note

अखिल भारतीय आयुर्विज्ञान संस्थान भोपाल
**All India Institute of Medical Sciences Bhopal**
Saket Nagar, Bhopal
www.aiimsbhopal.edu.in

Anupam Mudran # 9425025574