Emergency Triage Assessment and Treatment

(ETAT)

BASIC LIFE SUPPORT TABLE OF CONTENTS

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The American Heart Association (AHA) Basic Life Support (BLS) course has changed dramatically over the years to make it more accessible to the general public and more effective for the victim of cardiac arrest. Cardiac arrest is the leading cause of death in the world. Providers of BLS can intervene early and possibly prevent a death associated with sudden cardiac arrest.

- Initiate the chain of survival as soon as a possible problem is identified
- Initiate immediate high-quality chest compressions for any victim
- Provide early defibrillation with an Automated External Defibrillator (AED) when one is available
- Initiate rescue breathing when respiration is inadequate
- Perform BLS as a team
- Relieve a choking episode

INITIATING THE CHAIN OF SURVIVAL

Research shows that BLS can increase the rate of survival for certain victims of cardiac arrest. Typically, pediatric victims begin the collapse process after suffering dehydration or respiratory problems. This population will rarely have a primary cardiac arrest. If respiratory events and dehydration can be prevented, cardiac arrest can often be avoided. Therefore, it is critical to prevent the need for resuscitation in infants and children.

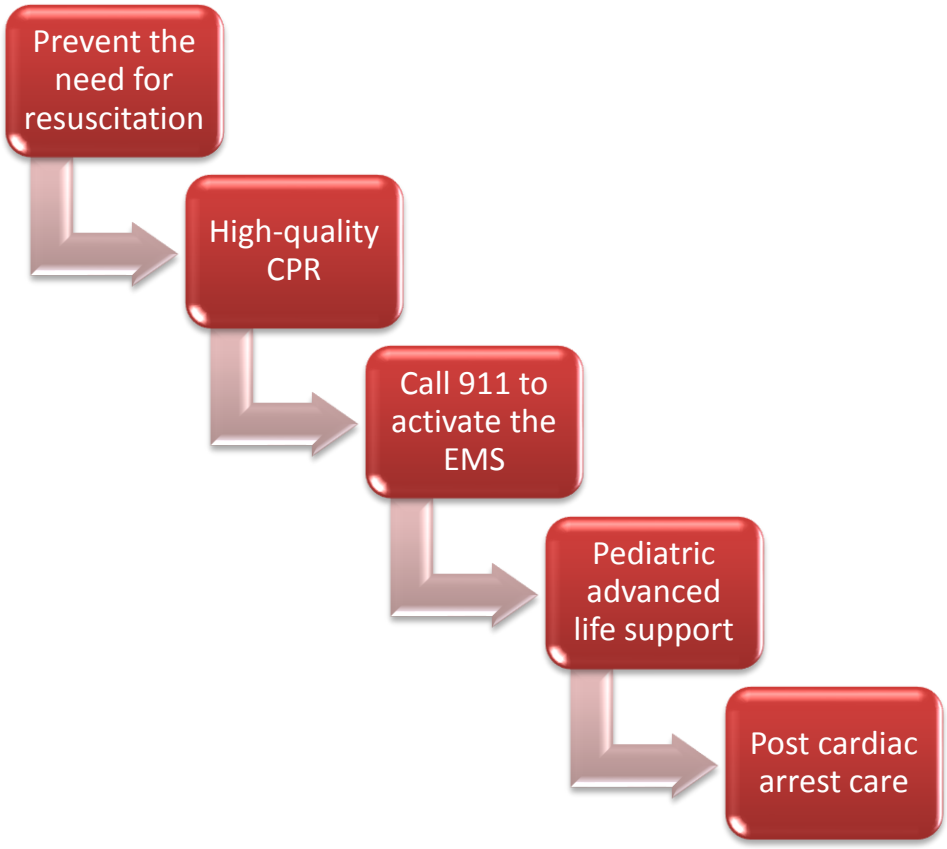

FIGURE 1: PEDIATRIC CHAIN OF SURVIVAL

For adult victims it is critical that the Adult Cardiac Arrest Chain of Survival is initiated quickly and performed at a high level of quality. The Adult Cardiac Arrest Chain of Survival has been updated to include a different response whether the cardiac arrest takes place inside or outside of the hospital.

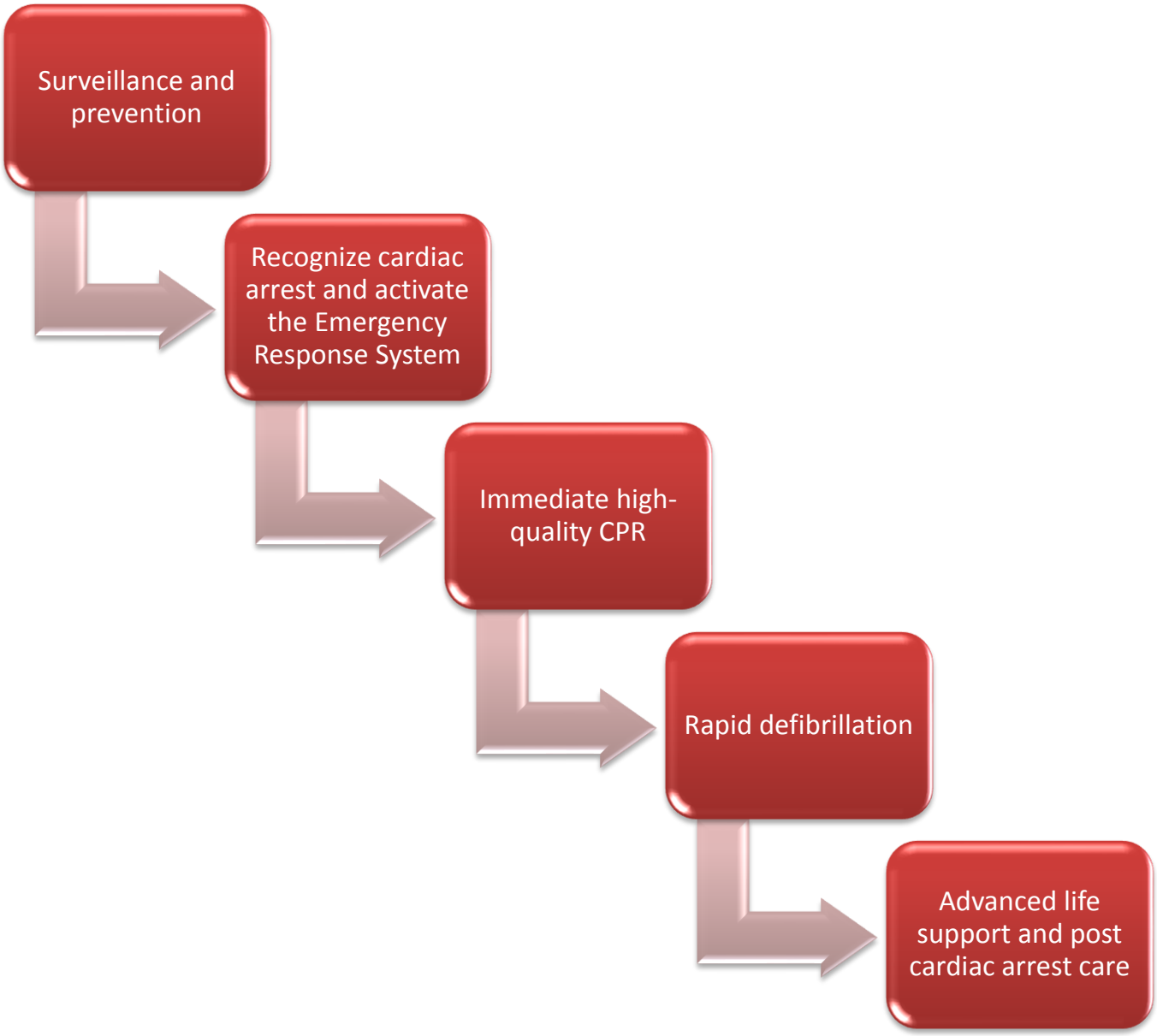

FIGURE 2: IN-HOSPITAL ADULT CARDIAC ARREST CHAIN OF SURVIVAL

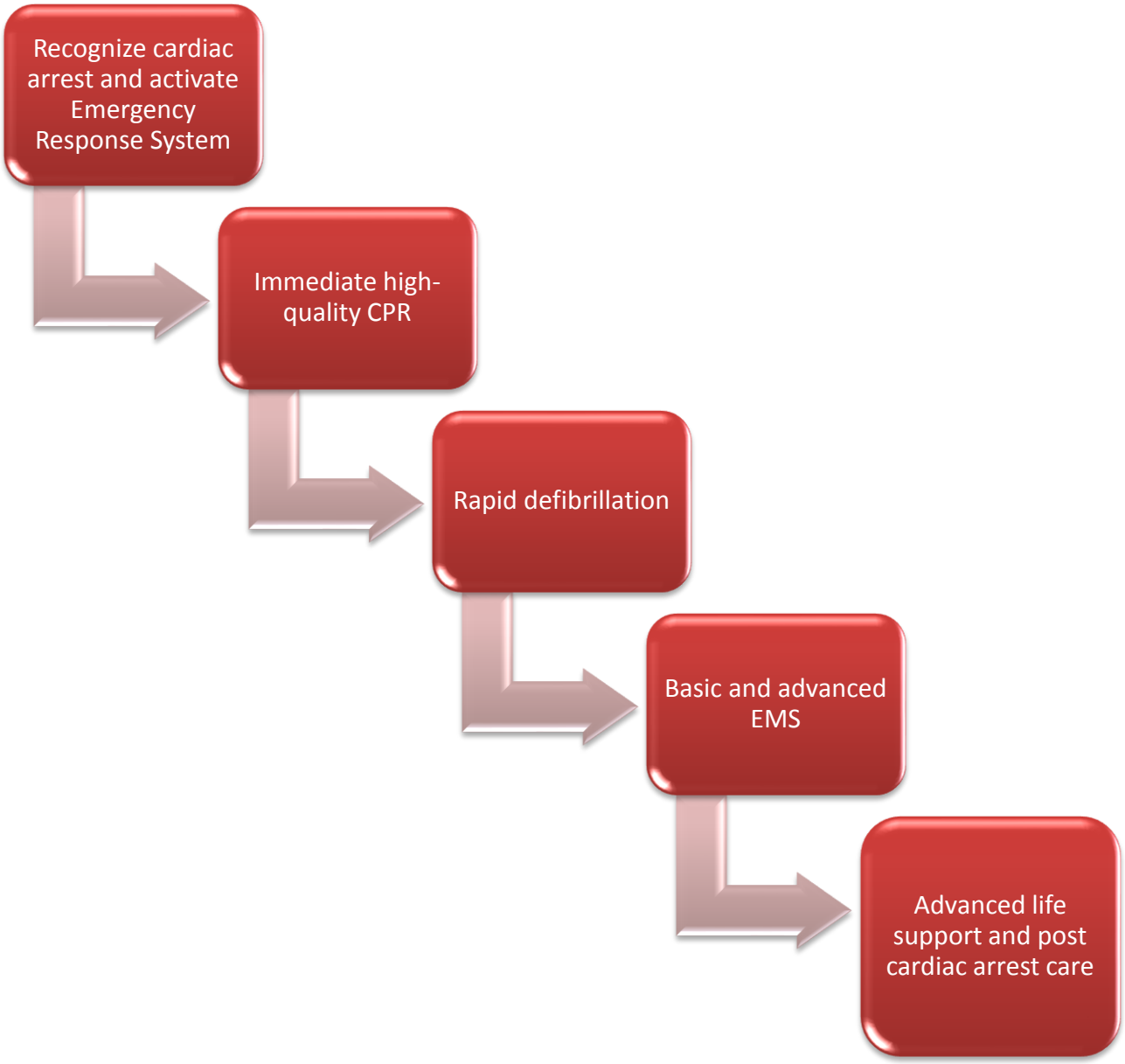

FIGURE 3: OUTSIDE-OF-HOSPITAL ADULT CARDIAC ARREST CHAIN OF SURVIVAL

In 2015, the AHA made some important changes to the BLS Guidelines.

Guideline	Old Guideline	2015 Guideline
EMS Activation	~~Provider should check for a response before activating EMS~~	Call for help immediately, preferably while assessing the victim (pulse and breathing)
EMS Activation		Alone with no cell phone: Leave victim to activate EMS and get AED before CPR UNLESS an unwitnessed collapse of an infant or child. Give 2 minutes of CPR then activate EMS/get AED Alone with cell phone: Activate EMS first Not alone: Split duties; 1-2 people start CPR while 1-2 people activate EMS and get AED
Sequence	CAB (compressions, airway, breathing)	Confirmed in the 2015 Guidelines: Do not delay the first 30 chest compressions
Compression Depth	~~Used "at least" without a maximum depth~~	Infants to children up to puberty: Compress the chest up to 1/3 of the chest diameter Puberty, adolescence, adult: Compression depth between 2 and 2.4 inches (5 to 6 cm)
Compression Frequency	~~At least 100 compressions per minute~~	No less than 100, no more than 120
Chest Recoil	Allow the chest to fully recoil between compressions	Confirmed in the 2015 Guidelines: Do not lean on the chest between compressions; allow the heart to fully fill with blood
Compression-Only CPR	Compression-only CPR emerged since the 2010 update. It has been formalized in the 2015 guidelines for untrained rescuers.	Untrained rescuers should provide chest compressions until EMS arrives or a trained provider arrives (or the victim starts to move). Rescue breathing should only be done if it can be done competently.
Naloxone	New recommendation for 2015	Trained lay providers and EMS should provide intramuscular or intranasal naloxone in the case of known or suspected opioid overdose (abnormal or no breathing, no response, has a pulse)
Shock or CPR First?	~~Highly complex recommendations that changed based on various circumstances~~	Use AED in a witnessed cardiac arrest if immediately available; administer chest compressions until AED arrives and is on the victim, ready for use
Chain of Survival	~~Same Chain of Survival for in-hospital and out-of-hospital cardiac arrest~~	In-hospital and out-of-hospital Adult Cardiac Arrest Chain of Survival are different; Primary providers and lay rescuers provide immediate care and then transfer care to the code team or EMS crew, respectively.

TABLE 1: 2015 BLS CHANGES

- Research shows that beginning compressions early increases the chance of survival so the “CAB” (Chest Compressions, Airway, Breathing) sequence is still recommended.
- Immediate, high-quality CPR is of the utmost importance.
- High-quality CPR includes:
    - Perform compressions at a rate between 100 and 120 per minute, regardless of the age of the victim
    - Chest compressions should be between 2 and 2.4 inches (5 to 6 cm) for adults and adolescents
    - Chest compressions should be given to a depth of 1/3 the diameter of the chest in infants and children up to puberty. This is about 1.5 inches (4 cm) in infants and 2 inches (5 cm) in children
    - Allow the chest to completely recoil between compressions so that the heart can completely refill
    - Do not interrupt compressions except to use an AED or change providers
    - Interruptions should be limited to no more than 10 seconds at a time
    - Chest compressions should make up at least 60% of the rescue time
    - Deliver each breath over 1 second, regardless of victim age
    - Prevent over-inflation of the lungs by avoiding rapid ventilations
    - Perform CPR as a team to perform activities more quickly and efficiently
    - When an advanced airway is in place, ventilate at 1 breath every 6 seconds (10 breaths per minute).
- Feel for a pulse for no longer than 10 seconds:
    - Carotid artery (neck) is preferred for adults and adolescents
    - Femoral artery (inner thigh) is preferred for children
    - Brachial artery (upper arm) is preferred for infants.
- A manual defibrillator is preferred for infants, but if one is not available than an AED should be used. If a pediatric dose attenuator is available on the AED, use it. If the dose attenuator is not available, use an adult AED for a victim of any age.

The BLS process for adults teaches one-rescuer CPR but also recognizes that there may be more rescuers available to help. In the BLS course, students learn both one- and two-rescuer CPR.

ONE-RESCUER ADULT BLS

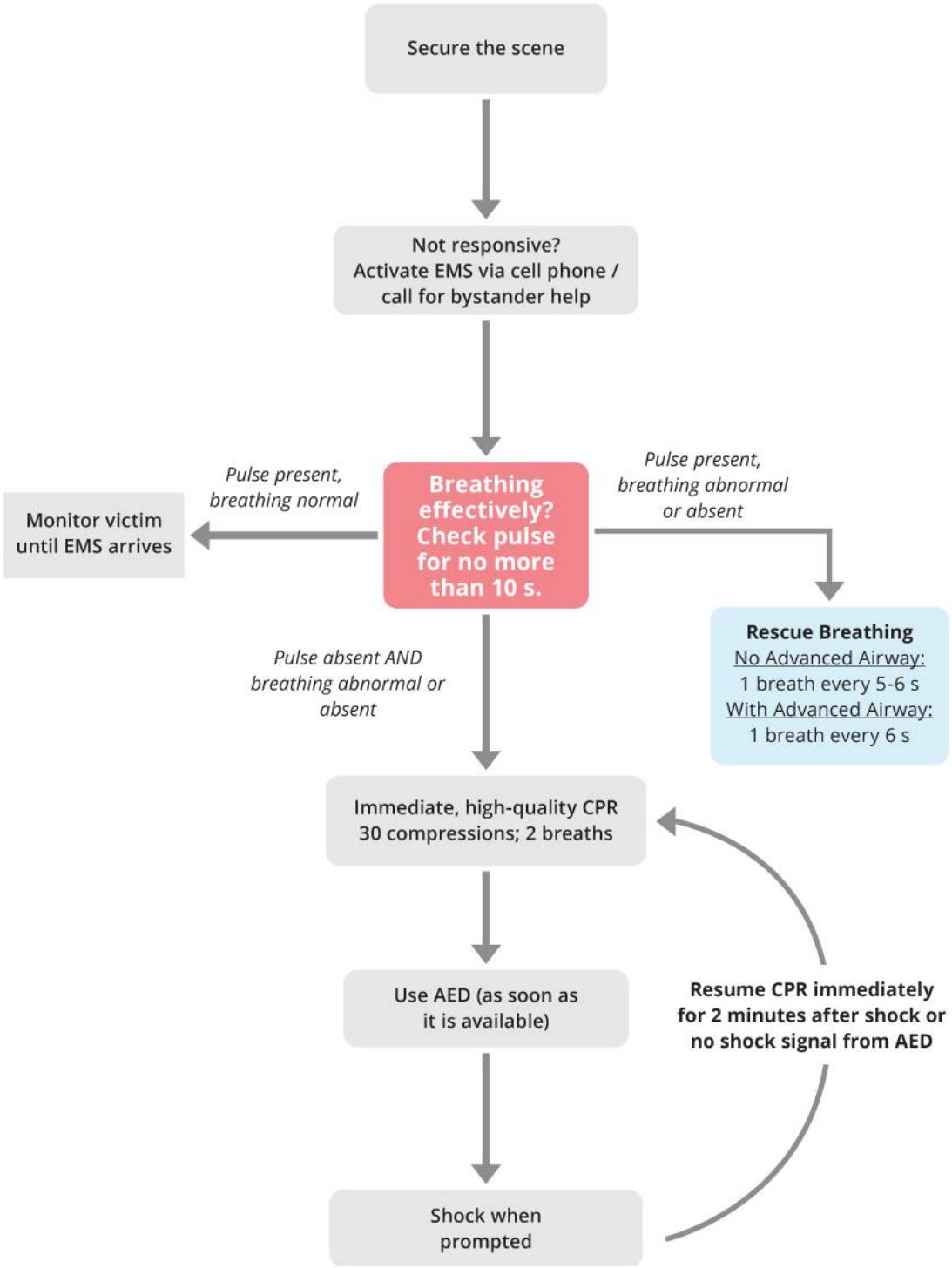

FIGURE 4: BLS ADULT ALGORITHM

Once the assessment is complete and you have determined that the victim is not responsive, does not have a pulse, and is not breathing, it is important to start CPR.

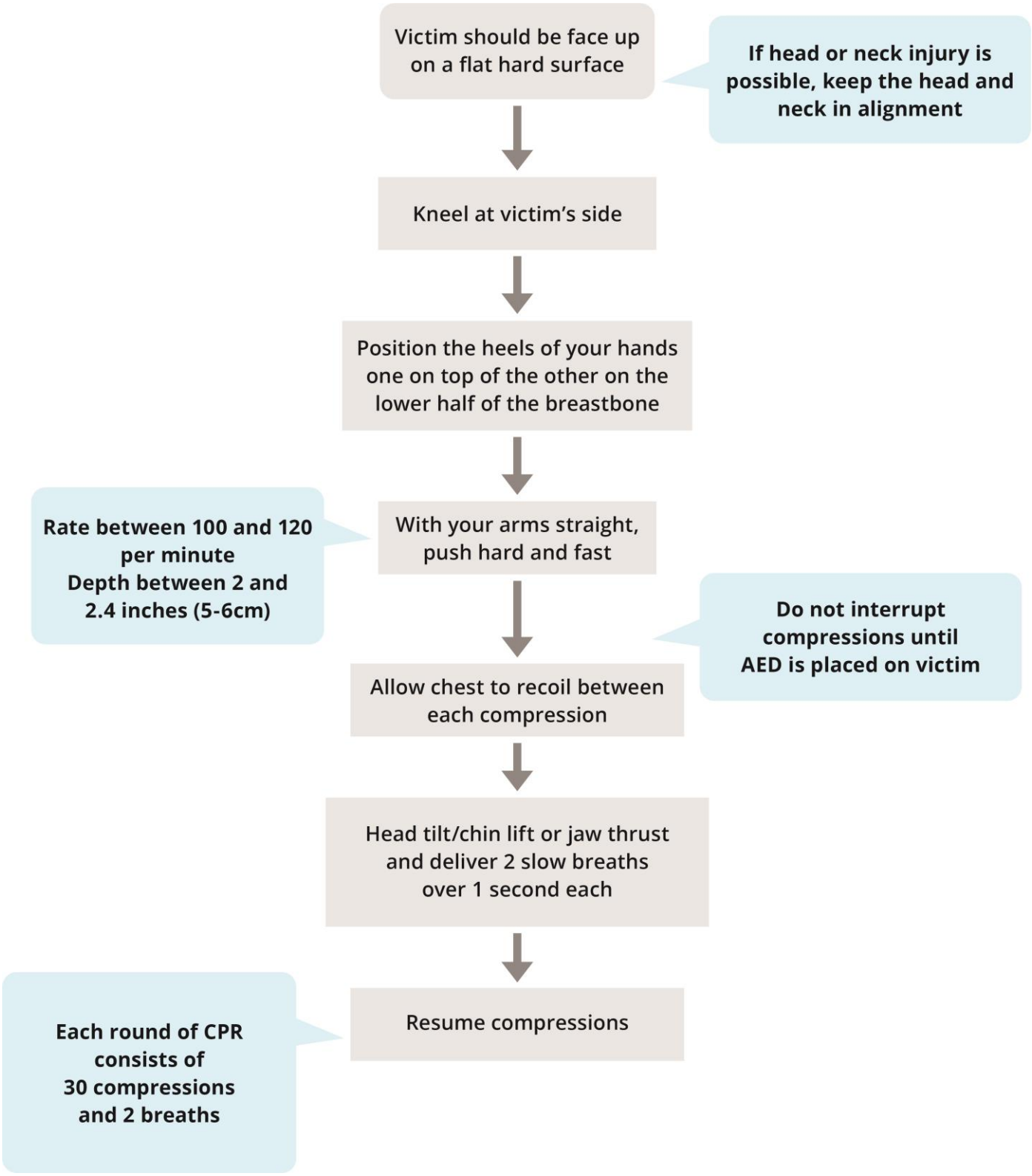

FIGURE 5: BLS CPR ALGORITHM

TWO-RESCUER ADULT BLS/CPR

Sometimes there will be more than one person available to perform CPR. If a second person is available, the steps of CPR do not change, but the tasks can be shared by the team:

- Send the second person to activate EMS and retrieve an AED if one is readily available. At the same time, the first rescuer begins CPR.
- When the second rescuer returns, have them prepare the AED for use. The first person continues CPR counting compressions aloud.
- When the AED is open and ready, continue CPR while applying the pads.
- Stop CPR to allow the AED device to analyze the victim's cardiac rhythm and to provide shocks, if needed.
- The second rescuer ensures that the victim's airway is open then gives 2 rescue breaths, each lasting 1 second.
- The rescuers should switch positions every 2 minutes (5 cycles of 30 compressions and 2 breaths).

ADULT MOUTH-TO-MASK VENTILATION

When performing one-rescuer CPR, rescue breaths should be supplied using a mask if available.

1. Perform 30 chest compressions between 2 and 2.4 inches (5 to 6 cm) deep at a rate between 100 and 120 per minute.
2. Seal the mask against the victim's face by forming your hand in a "C-E" shape and pressing down on the top and bottom edges of the mask. The thumb and index fingers form the "C" while the other three fingers form the "E."
3. Unless you think the victim may have a neck injury, open the airway using the head tilt/chin lift.
4. If you suspect the victim may have a neck injury, open the airway using a jaw thrust.
5. Give two slow deep breaths over 1 second each and watch for the rise of the victim's chest.

COMPRESSION-ONLY CPR

Many rescuers feel uncomfortable or unable to provide mouth-to-mouth ventilations. Because of this they hesitate to perform CPR. Simply providing chest compressions to someone in cardiac arrest is better than not helping at all. In fact, if ventilations cannot be delivered properly and/or in a timely manner, rescue time is better spent delivering chest compressions only. Compression-only CPR is recommended for rescuers who cannot or will not deliver ventilations.

The large majority of cases of pediatric arrest are due to pulmonary issues rather than cardiac problems. When possible and appropriate, it is better to deliver ventilations than compressions only. Nevertheless, if the rescuer of a pediatric victim cannot or will not give ventilations, compression-only CPR is better than not helping at all.

ADULT BAG-MASK VENTILATION IN TWO-RESCUER CPR

If two or more rescuers are available with a bag-mask device, one rescuer should continue compressions while the second rescuer seals the mask over the victim's face and delivers two rescue breaths after every 30 compressions. Ventilations should be delivered over 1 second, regardless of age.

ONE-RESCUER BLS FOR INFANTS AND CHILDREN

If you are alone and an infant (0-12 months) or a child (1 year to puberty) is unresponsive, follow the BLS Infant and Child Algorithm:

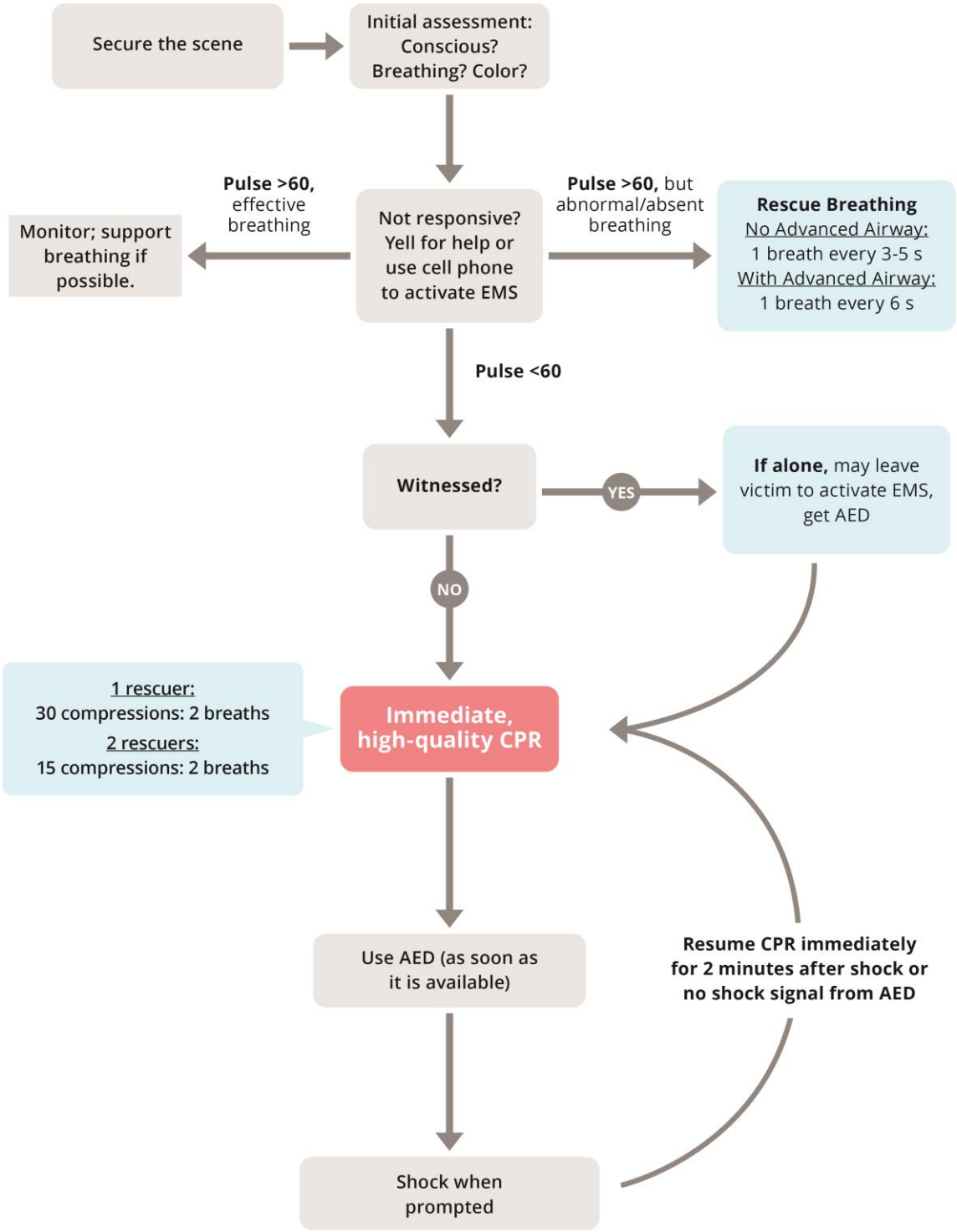

FIGURE 6: BLS INFANT AND CHILD ALGORITHM

BLS for children and adults are very similar, but there are differences. The differences are:

Guideline	Adult and Adolescents	Child (1 year to puberty)
Compression to breath ratio for 2 rescuers	30 compressions : 2 breaths	15 compressions : 2 breaths
Compression to breath ratio for 1 rescuer	30 compressions : 2 breaths	30 compressions : 2 breaths
Chest compressions	Always use 2 hands	For small children, may use 1 hand
Compression depth	Between 2 and 2.4 inches	1/3 of the depth of the chest (about 1.5 to 2 inches)
Unwitnessed arrest (alone, no cell phone)	Leave victim to activate EMS	Perform 2 minutes of CPR then leave victim to activate EMS/get AED
Unwitnessed arrest (alone, with cell phone)	Activate EMS immediately	Activate EMS immediately
Witnessed arrest (alone, no cell phone)	Leave victim to activate EMS	Leave victim to activate EMS
Witnessed arrest (alone, with cell phone)	Activate EMS immediately	Activate EMS immediately

TABLE 2: DIFFERENCES IN BLS FOR ADULTS AND CHILDREN

Likewise, there are differences between BLS for children and infants. The differences are:

Guideline	Child (1 year to puberty)	Infant (0 to 12 months)
Checking the pulse	Carotid or femoral artery	Brachial artery on inside of upper arm
CPR	For small children, may use 1 hand for compressions	May use 2 fingers or 2 thumbs by the encircling hands technique if your hands are big enough to circle the infant's chest; 2-thumb technique is preferred
Compression depth	1/3 of the depth of the chest (1.5 to 2 inches)	1/3 of the depth of the chest (about 1.5 inches)

TABLE 3: DIFFERENCES IN BLS FOR CHILDREN AND INFANTS

TWO-RESCUER BLS FOR INFANTS AND CHILDREN

If two rescuers are available:

- As soon as it is determined that the child is not breathing and responsive, the second rescuer should immediately activate the EMS and find an AED.
- As soon as another rescuer arrives, change the compression to ventilation ratio from 30:2 to 15:2 (i.e., give 2 breaths after every 15 compressions).

CHILD VENTILATION

Adult masks should not be used for small children. If the mask covers the eyes or chin of the child, it is too big and ventilations will not be optimal. Breaths for a child will typically not be as deep as for adults, but still should be administered over 1 second and should result in a visible rise of the child's chest. Unless a neck injury is suspected, open the airway using the head tilt/chin lift technique. If a neck injury is suspected, open the airway using a jaw thrust.

RESCUE BREATHING

Early recognition of and intervention for respiratory distress may prevent deterioration into cardiac arrest. During assessment, if the victim has a strong pulse but has ineffective breathing, open the airway using the head tilt/chin lift technique and begin rescue breathing.

Victim Age	Breathing Rate	# Breaths/Minute	Length of Breath	Evaluation
Adult	Every 5-6 seconds	10-12 per minute	Each breath should be given over 1 second	Check for chest rise and breathing. Check pulse, begin CPR if victim pulseless
Child or Infant	Every 3-5 seconds	12-20 per minute		

TABLE 4: RESCUE BREATHING

CPR WITH AN ADVANCED AIRWAY

An advanced airway includes supraglottic airways, laryngeal mask airways, or endotracheal tubes. These airways should be initiated as soon as available since they offer a better way of providing ventilations for any age. If these advanced airways are not available, continue to use mouth-to-mouth, mouth-to-mask, or bag-mask for breathing in an arrest situation. If an advanced airway is in place, the compression/breath ratio should be as described below.

Guideline	No Advanced Airway	With Advanced Airway
Adult Compression to Breath Ratio	30 compressions : 2 breaths	Deliver 1 breath every 6 seconds (10 breaths per minute)
Child/Infant Compression to Breath Ratio	15 compressions : 2 breaths	

TABLE 5: COMPRESSION TO BREATH RATIOS WITH/WITHOUT ADVANCED AIRWAY

VENTILATION

If a mask or advanced airway is not available, be ready to provide mouth-to-mouth rescue breathing during CPR. Avoid over-ventilation, which can fill the stomach with air and prevent proper lung expansion.

ADULT AND OLDER CHILDREN MOUTH-TO-MOUTH

Do not give breaths too rapidly or too forcefully; doing this may cause air to be forced into the stomach resulting in distention and less room for lung expansion.

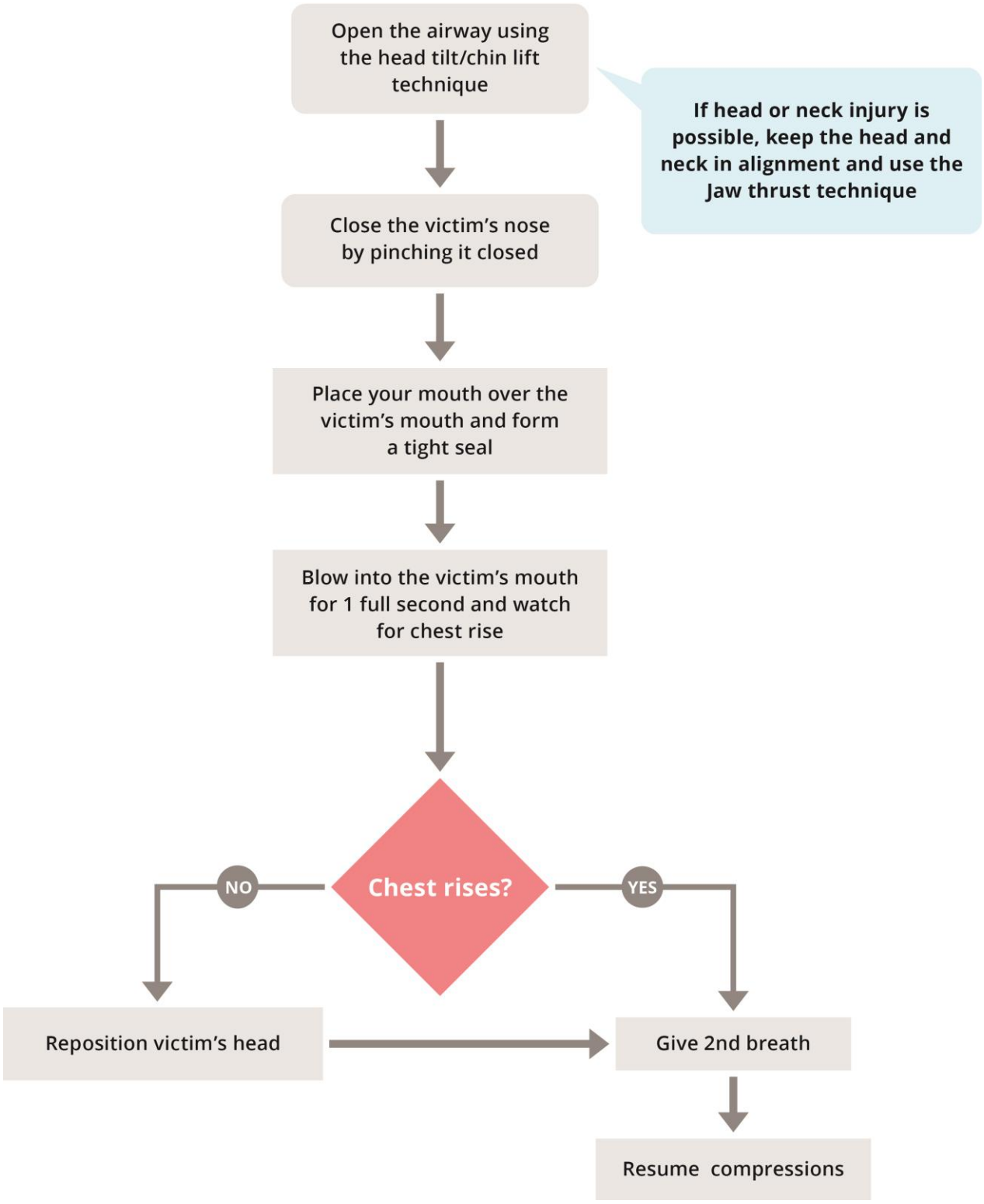

FIGURE 7: BLS RESCUE BREATHING ADULT OR CHILD ALGORITHM

INFANT MOUTH-TO-MOUTH OR MOUTH-TO-NOSE

Ventilation techniques for an infant are the same as for children and adults with the following exceptions:

- If the infant is small enough the rescuer can cover the infant's nose and mouth with their mouth and create a good seal. It is not necessary to pinch the infant's nose.
- Be aware that an infant's lungs are very small so a smaller volume of air will be necessary to inflate the infant's lungs. Every breath should still be given over 1 second, but with less volume.

AUTOMATED EXTERNAL DEFIBRILLATOR USE

One of the most common causes of cardiac arrest is ventricular fibrillation. The Automated External Defibrillator (AED) is the most effective treatment for this disorder. The AED analyzes the heart rhythm and advises a shock only when it is appropriate. The AED is safe for anyone to use since it will talk you through the process and will not allow you to make a mistake. Research indicates that an AED should be used as early as possible in any arrest situation for any age. In fact, survival is increased if an AED can be used (in appropriate cases) within 3 to 5 minutes of collapse outside of a hospital.

If the AED has pediatric pads and a pediatric attenuator, use them for an infant or child less than 8 years old. If pediatric pads are not available, adult pads can be used as long as they are applied so that they do not touch each other. Typically, you will see an adult/pediatric attenuator switch. If this switch is not available, deliver an "adult" shock.

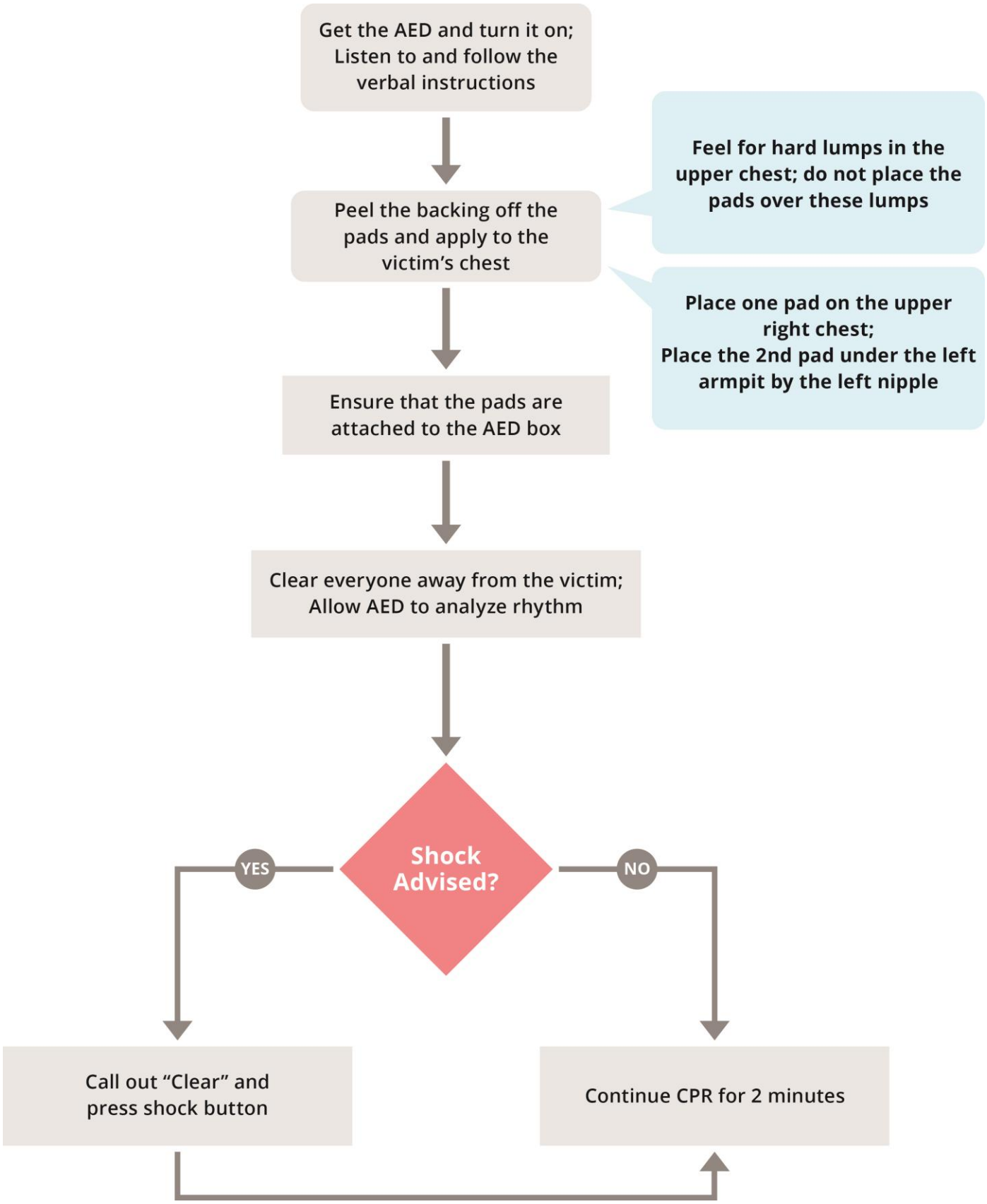

FIGURE 8: BLS AED ALGORITHM

UNIT FIVE: RELIEF OF CHOKING

If a victim is choking, the condition may deteriorate into respiratory arrest and cardiac arrest. Early and proper intervention can prevent this series of events. Proper intervention depends on the age of the victim and the amount of obstruction of the airway.

CHOKING IN AN ADULT OR CHILD OLDER THAN ONE YEAR

Amount of Airway Obstruction	Symptoms	Recommended Actions
Mild	• Breathing may be accompanied by wheezing • Coughing and making noise	• Remain with victim and continue to monitor • Encourage the victim to cough • Call EMS if choking gets worse
Severe	• Exhibiting universal sign of choking (holding neck and throat) • Weak or absent cough • May be making high-pitched noise but unable to talk • Ineffective or no breathing • Skin may be blue around lips and finger tips	• Attempt abdominal thrusts to relieve obstruction • If you see the obstruction in the victim's mouth and can remove it, do so. Do not perform blind sweeps of the mouth • Call EMS • Begin CPR if victim is unresponsive and pulseless

TABLE 6: ADULT AND CHILD AIRWAY OBSTRUCTION

If the choking victim is older than one year and is responsive, perform abdominal thrusts in an attempt to relieve choking.

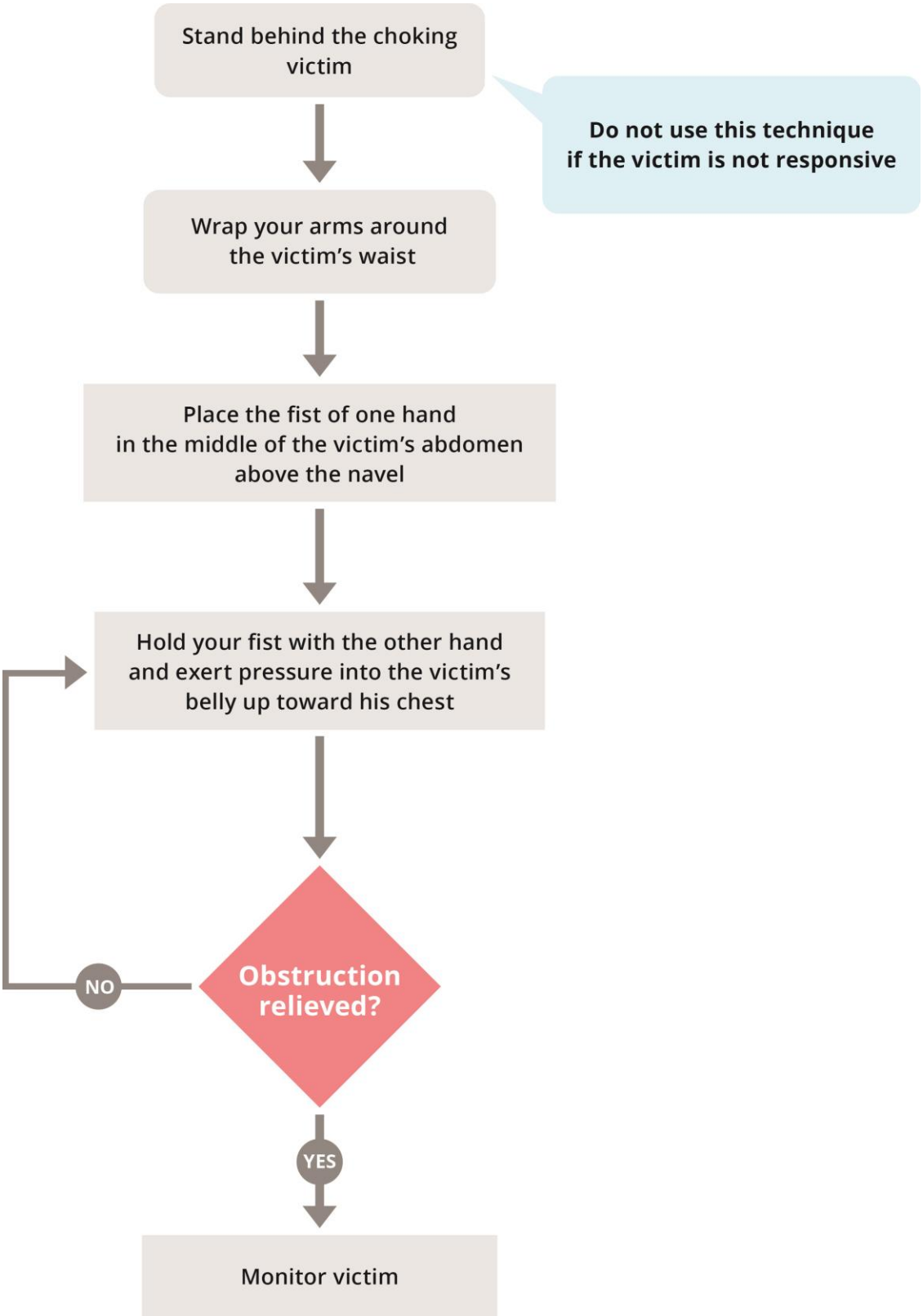

FIGURE 9: BLS CHOKING ADULT OR CHILD ALGORITHM

Amount of Airway Obstruction	Symptoms	Recommended Actions
Mild	• Breathing may be accompanied by wheezing • Coughing and making noise	• Remain with infant and continue to monitor • Do not do a blind finger sweep in an attempt to remove an obstruction • Call EMS if infant begins to deteriorate
Severe	• Holding neck and throat • Weak or absent cough • May be making high-pitched noise but unable to talk • Ineffective or no breathing • Skin may be blue around lips and finger tips	• Attempt back blows/chest thrusts to relieve obstruction • If you see the obstruction in the victim's mouth and can remove it, do so. Do not perform blind sweeps of the mouth • Call EMS • Begin CPR if infant becomes unresponsive and pulseless

TABLE 7: INFANT AIRWAY OBSTRUCTION

In an infant less than 12 months old is choking but responsive, attempt to use back blows and chest thrusts to relieve an obstruction.

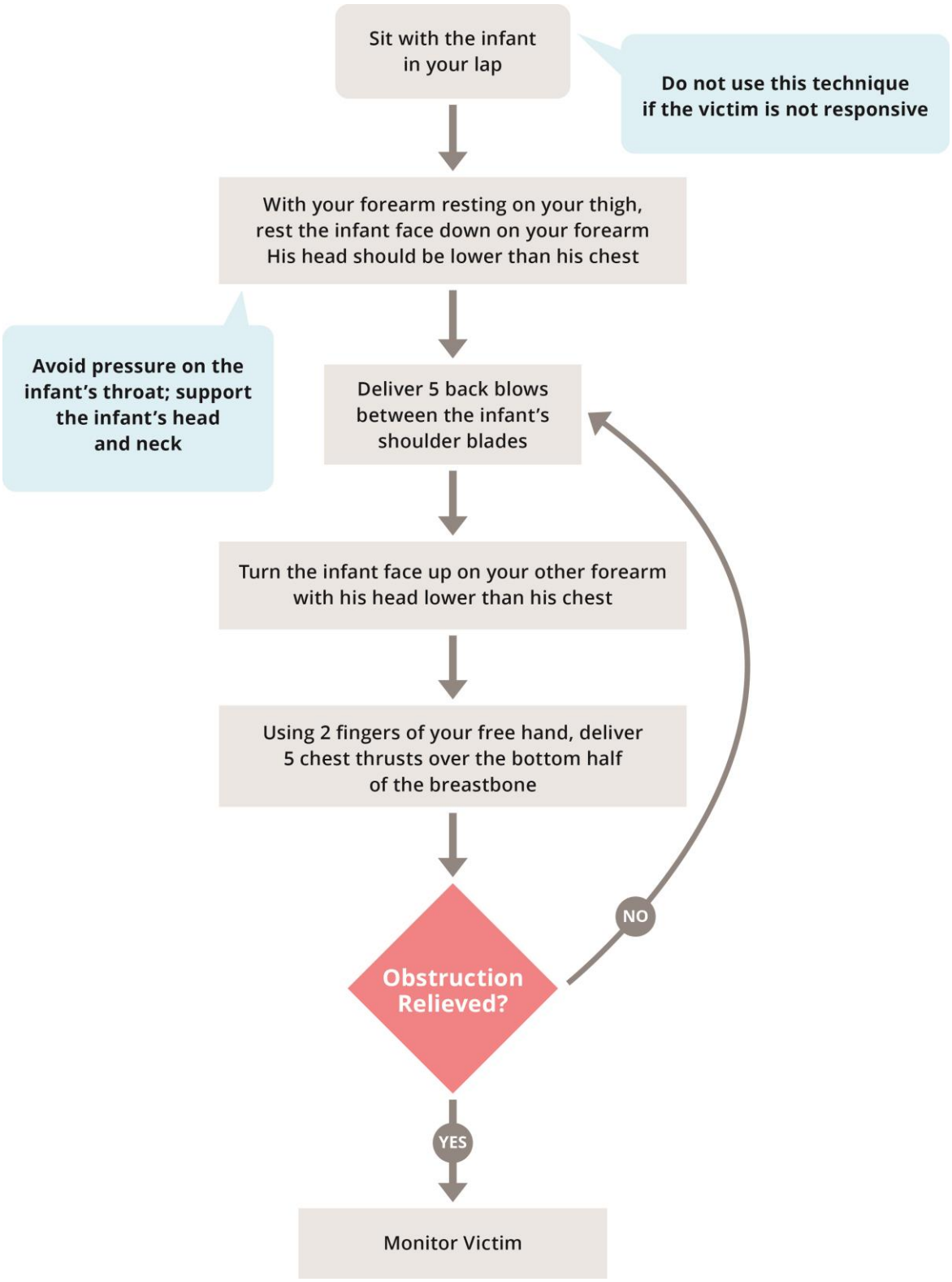

FIGURE 10: BLS CHOKING INFANT ALGORITHM

New in 2015, bystanders may administer naloxone to victims who are apparently suffering from a opioid overdose. Unresponsive opioid users can benefit from timely administration of naloxone (2 mg intranasal or 0.4 mg intramuscular).

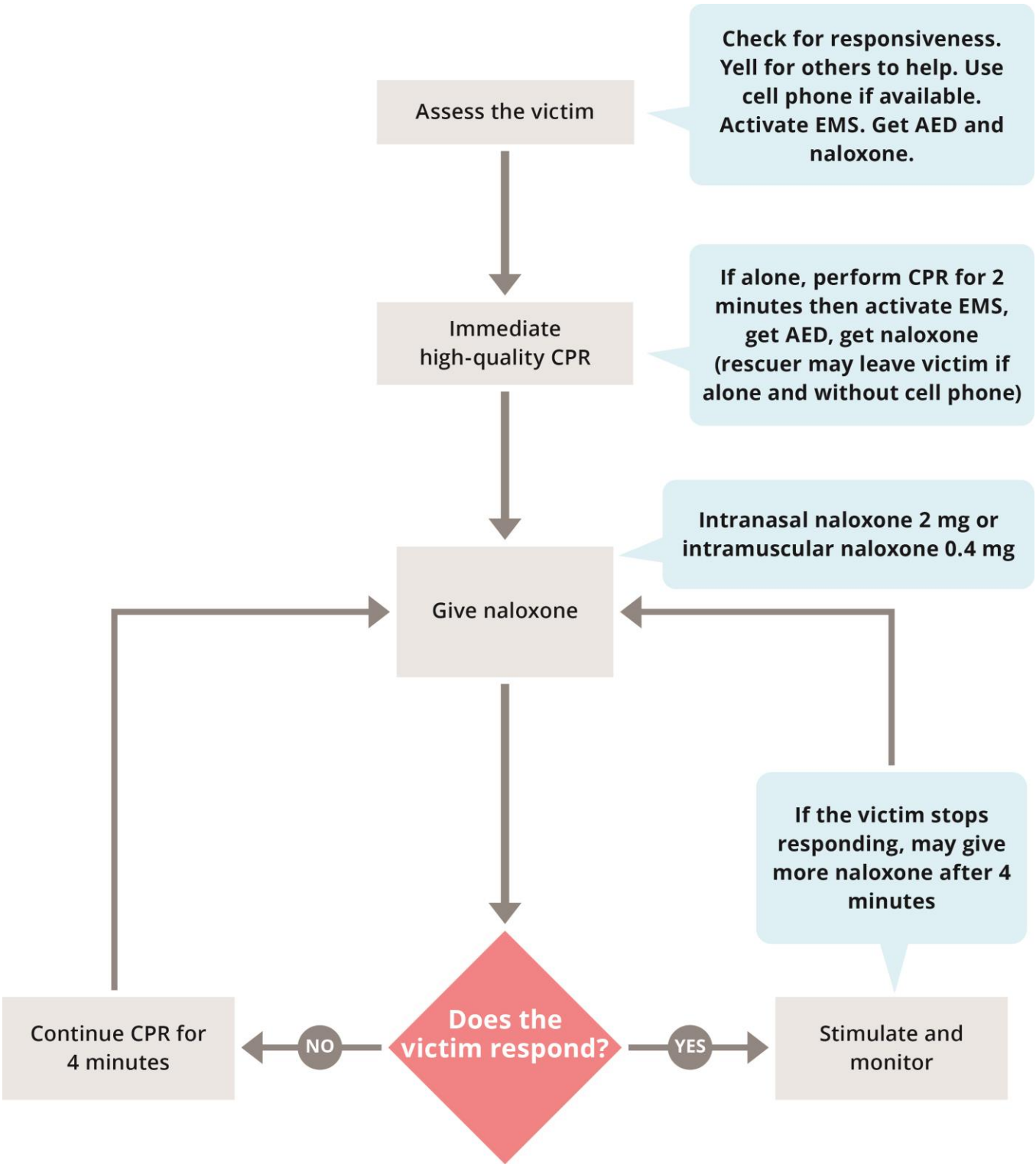

FIGURE 11: BLS SUSPECTED OPIOID OVERDOSE ALGORITHM

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In the pediatric population, cardiac arrest usually results from one of three problems:

- Progressive respiratory distress and failure (the most common cause)
- Progressive shock (second most common)
- Sudden cardiac death from ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) (5-15% of all pediatric cardiac arrest cases).

The Pediatric Advanced Life Support (PALS) course stresses identification and early intervention in each of these problems.

PALS PREPARATION

If attending a PALS course, the student must know the key concepts that will be used during the course:

- ECG rhythm recognition
- Infant and child basic life support (BLS)
- Pediatric pharmacology
- PALS algorithms and treatments.

ORGANIZATION OF THE PALS COURSE

In the PALS course, the student will demonstrate competency in four key skills stations that include simulations that stress the role of the team in the pediatric resuscitation process:

- One- and two-rescuer BLS for both infants and children
- Management of respiratory emergencies
- Rhythm disturbances and electrical therapies
- Vascular access.

The student will be asked to participate as team leader and team member in the skills stations. After successful completion of the skills, the student must successfully complete and pass a written exam testing the cognitive skills associated with pediatric resuscitation.

Guideline	Old Guideline	2015 Guideline
Sequence	CAB (compressions, airway, breathing)	Confirmed in the 2015 guidelines
Compression depth	Used "at least" without a maximum depth	Infants to children up to puberty: compress the chest up to 1/3 of the chest diameter; Puberty and adolescence: use adult compression depth between 2 and 2.4 inches (5 to 6 cm)
Frequency	At least 100 compressions per minute	Between 100 and 120 compressions per minute
Compression-only CPR	Infants and children require compressions and respirations, but compressions are better than nothing	Infants and children still require *compressions and respirations* for optimal CPR since most pediatric emergencies affect respiration primarily; Compression-only CPR is useful in infants/children in cardiac arrest
Fluid resuscitation	Aggressive fluids	Treat septic shock with isotonic IV fluids at a dose of 20 mL/kg, though use with caution in resource-limited settings (i.e. no critical care)
Atropine premedication	Use a minimum atropine dose of 0.1 mg to prevent paradoxical bradycardia	Do not routinely use atropine as premedication for endotracheal intubation; atropine can be used in patients at increased risk of bradycardia
Vasopressors	Use epinephrine for cardiac arrest	*Consider* using epinephrine during cardiac arrest
Extracorporeal CPR	Consider extracorporeal CPR only for children in standard resuscitation-refractory cardiac arrest	Extracorporeal CPR may be considered in at-risk children who have a cardiac arrest within a hospital with proper protocols, personnel, and equipment available
Amiodarone and lidocaine	Amiodarone preferred to lidocaine for pulseless VT/VF unresponsive to shock	Amiodarone and lidocaine equally effective for pulseless VT or VF unresponsive to shock
Post-cardiac arrest	Comatose patients should be cooled to between 32°C and 34°C for 12-24 hours	Comatose patients with cardiac arrest outside of the hospital should be cooled to 32°C to 34°C for 2 days followed by 3 days of normothermia or a total 5 days of normothermia; no recs for in-hospital cardiac arrest; treat fever aggressively
Post-cardiac arrest	New recommendation for 2015	Maintain systolic BP above the fifth percentile by age, use intra-arterial pressure monitoring
Post-cardiac arrest	No recommendations about $PaCO_2$	Titrate oxygen to achieve PaO_2 between 94% and 99%; keep $PaCO_2$ within normal range

TABLE 1: COMPARISON OF PALS GUIDELINES

CHANGES TO PEDIATRIC BLS IN 2015

Pediatric BLS was changed in 2015 to incorporate the use of cell phones into the algorithm. In an out-of-hospital arrest, the lone rescuer may call 911 before providing CPR if that rescuer has a cell phone is available. When others are nearby, they should be instructed to call 911 using an available cell phone, then get an AED.

When a **lone rescuer** finds an **infant or child up to the age of puberty** who is the victim of an **unwitnessed** collapse, the rescuer should give 2 minutes of CPR before leaving the victim to go get help/call 911/get AED. If the lone rescuer has a working cell phone, 911 should be called first.

When a **lone rescuer** finds an **infant or child up to the age of puberty** who is the victim of a **witnessed** collapse, the rescuer should leave the victim to go get help/call 911/get AED. If the lone rescuer has a cell phone, call 911 first.

When a **lone rescuer** finds an **adolescent (puberty and older)** who is the victim of a **witnessed** or **unwitnessed** collapse, the rescuer should leave the victim to go get help/call 911/get AED. If the lone rescuer has a cell phone, call 911 first.

PEDIATRIC CHAIN OF SURVIVAL

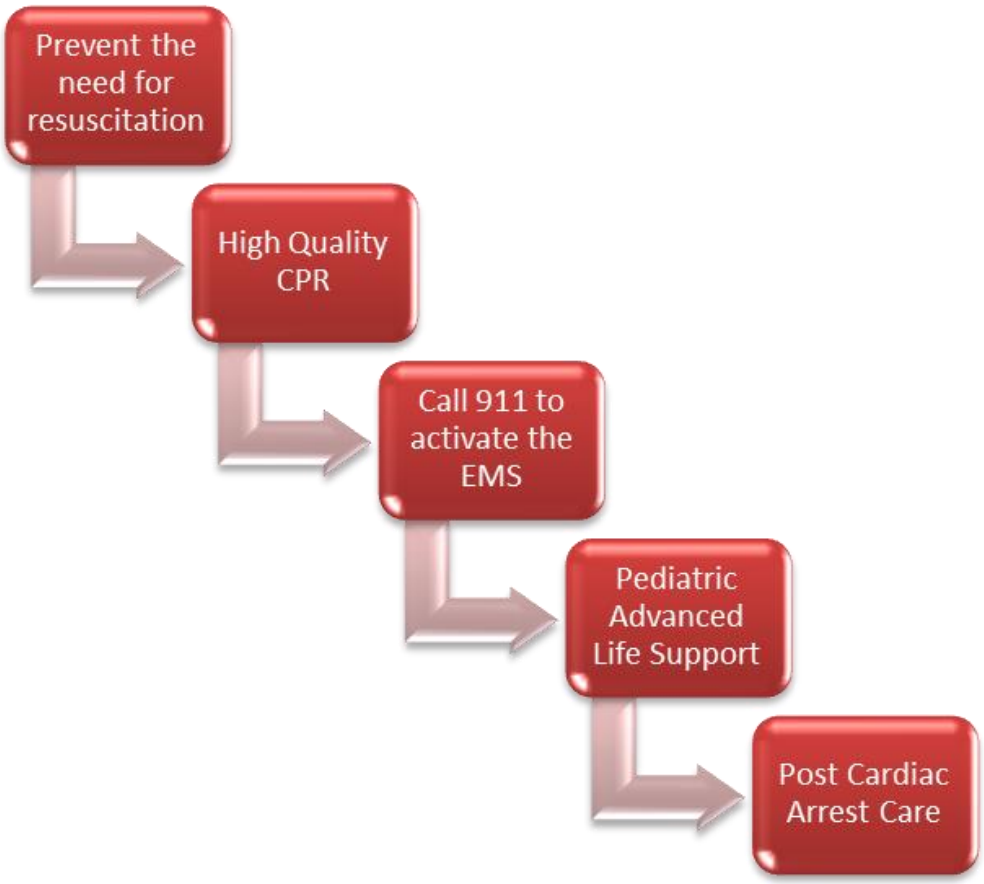


FIGURE 1: PEDIATRIC CHAIN OF SURVIVAL

In infants and children, it is better to monitor for signs of respiratory distress before overt respiratory failure occurs, requiring resuscitation. If resuscitation is required, it should include high-quality CPR and activation of EMS (e.g., call 911). Qualified providers should perform PALS followed by post-arrest care following return of spontaneous circulation (ROSC).

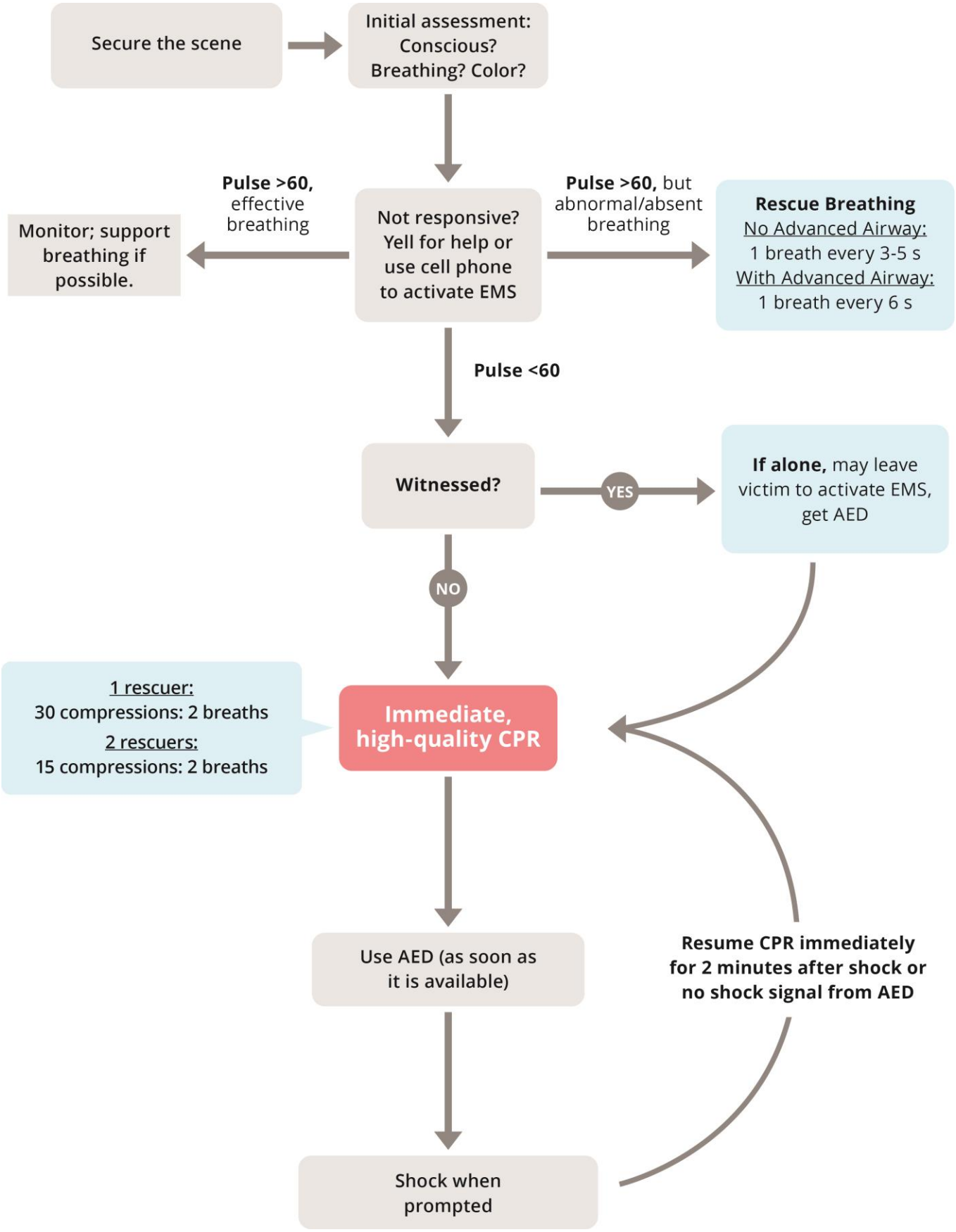


FIGURE 2: BLS INFANT AND CHILD ALGORITHM

In the pediatric population, respiratory and circulatory problems lead to cardiopulmonary failure while arrhythmias can lead directly to cardiac arrest. Rapidly evaluate and intervene using the PALS Sequence Algorithm (Figure 3):

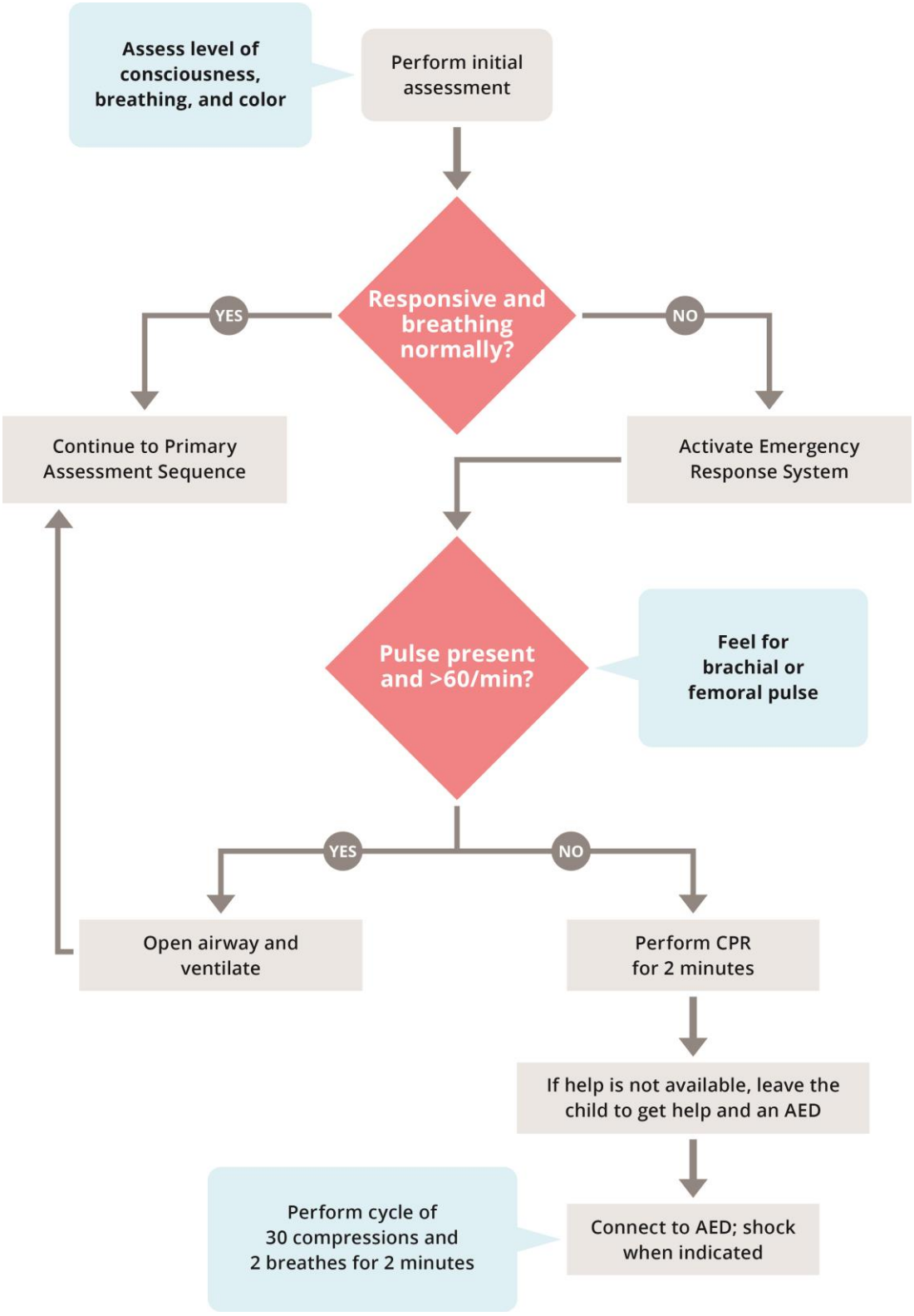


FIGURE 3: PALS SEQUENCE ALGORITHM

When evaluating the child's level of consciousness, breathing and color, note:

- **Level of consciousness**: Is the child awake and alert, irritable and crying, or unresponsive?
- **Breathing**: Is the child's respiratory pattern normal for his age, diminished or absent, or extremely labored?
- **Color**: Is the child's color mottled, cyanotic or pale, and/or normal for his ethnicity?

This initial assessment will guide the next steps of the PALS sequence. Begin the primary and secondary assessments using the Evaluate-Identify-Intervene sequence if the child is breathing, has a good pulse and color, and is responsive.

EVALUATE-IDENTIFY-INTERVENE

The Evaluate-Identify-Intervene sequence will enable identification of the most appropriate intervention. Use this sequence until the child is stabilized or until the child's condition deteriorates and other treatment is indicated.

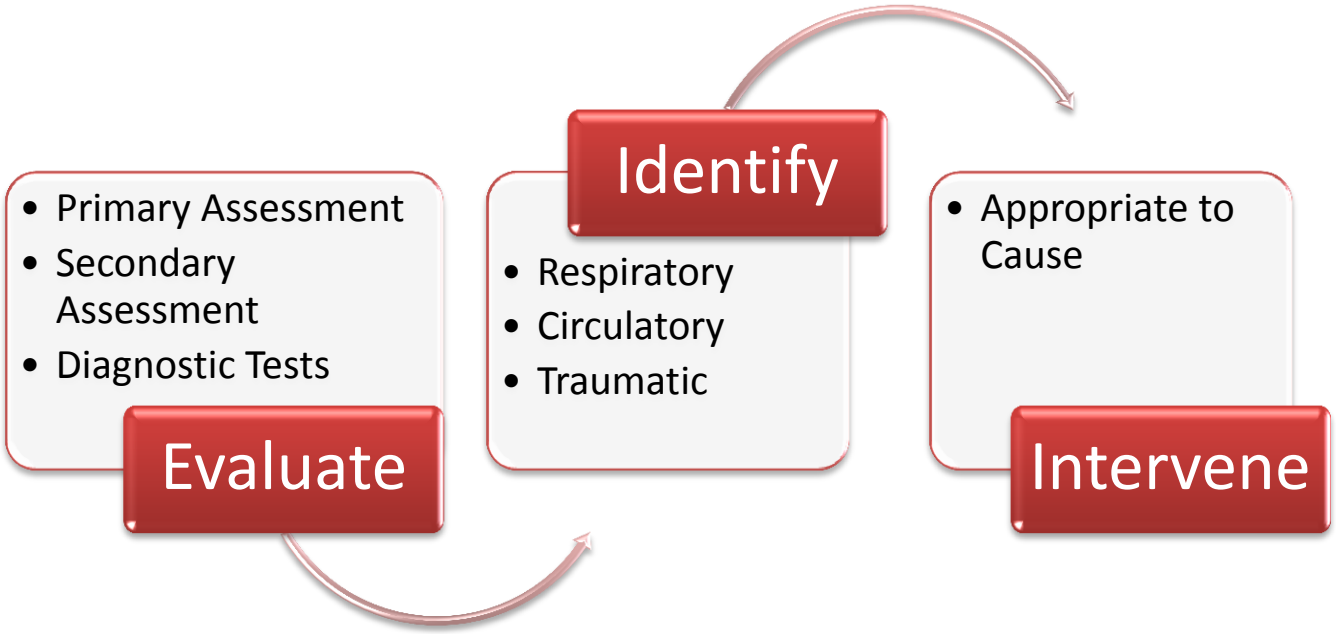


FIGURE 4: EVALUATE-IDENTIFY-INTERVENE SEQUENCE

During evaluation, conduct the primary assessment, secondary assessment, and diagnostic tests. At all times, be aware of whether or not a condition is life-threatening and be ready to move to the appropriate intervention immediately.

Primary Assessment			
Assessment	**Assessment Techniques**	**Abnormal Findings**	**Interventions**
A → Airway	Observe for movement of the chest or abdomen	Obstructed but maintainable	Keep airway open by head tilt/chin lift
	Listen to the chest for breath sounds	Obstructed and cannot be opened with simple interventions	Keep airway open using advanced interventions
B → Breathing	Rate	<10 or >60 = Abnormal (Apnea, bradypnea, tachypnea)	Immediate respiratory intervention required
	Effort	Nasal flaring, head bobbing, seesaw respirations, retractions	Immediate respiratory intervention required
	Chest or abdominal expansion	Asymmetrical or no chest movement	Immediate respiratory intervention required
	Breath sounds	Stridor, grunting, wheezing, rales, rhonchi	Immediate respiratory intervention required
	Oxygen saturation (O_2 sat)	<94% on room air <90% at any time	Supplemental oxygen Advanced airway
C → Circulation	Heart rate	Bradycardia Tachycardia Absent	Bradycardia Algorithm Tachycardia Algorithm Cardiac Arrest Algorithm
	Peripheral pulses (radial, posterior tibial, dorsalis pedis)	Diminished or absent	Close monitoring
	Central pulses (femoral, brachial, carotid, and axillary)	Diminished or absent	Management of Pediatric Shock
	Capillary refill	>2 seconds	Management of Pediatric Shock
	Skin color/temperature	Pale mucous membranes	Management of Pediatric Shock
		Central cyanosis	Immediate respiratory intervention required
		Peripheral cyanosis	Management of Pediatric Shock
	Blood pressure	Outside of the normal range for age	Management of Pediatric Shock
D → Disability	AVPU Scale	***A***lert – Awake, active, responsive to parents (normal) ***V***oice – Responds only to voice ***P***ain – Responds only to pain ***U***nresponsive – Not responsive	Monitor and consult neurologist
	Glasgow Coma Scale	Pediatric Glasgow Coma Scale (see Table 6)	
	Pupils	Unequal or non-reactive	
E → Exposure	General evaluation	Signs of bleeding, burns, trauma, petechiae, and purpura	Management of Pediatric Shock

TABLE 2: PRIMARY ASSESSMENT MODEL

Use the Primary Assessment to evaluate the child using vital signs and an ABCDE model:

A → Airway

Advanced interventions for keeping the airway open may include:

- Laryngeal mask airway
- Endotracheal (ET) intubation
- Continuous positive airway pressure (CPAP)
- Foreign body removal if one can be visualized
- Cricothyrotomy in which a surgical opening is made into the trachea.

B → Breathing

The child's respiratory rate is an important assessment that should be made early in the primary assessment process. The clinician must be aware of normal respiratory ranges by age:

Age Category	Age Range	Normal Respiratory Rate
Infant	0-12 months	30-60 per minute
Toddler	1-3 years	24-40 per minute
Preschooler	4-5 years	22-34 per minute
School age	6-12 years	18-30 per minute
Adolescent	13-18 years	12-16 per minute

TABLE 3: NORMAL RESPIRATORY RATES

C → Circulation

The child's heart rate is another important assessment that should be made in the primary assessment. The normal heart rates by age are:

Age Category	Age Range	Normal Heart Rate
Newborn	0-3 months	80-205 per minute
Infant/young child	4 months to 2 years	75-190 per minute
Child/school age	2-10 years	60-140 per minute
Older child/ adolescent	Over 10 years	50-100 per minute

TABLE 4: NORMAL HEART RATES

The child's blood pressure should be another part of the primary assessment. Normal blood pressures by age range are:

Age Category	**Age Range**	**Systolic Blood Pressure**	**Diastolic Blood Pressure**	**Abnormally Low Systolic Pressure**
Neonate	1 Day	60-76	30-45	<60
Neonate	4 Days	67-84	35-53	<60
Infant	To 1 month	73-94	36-56	<70
Infant	1-3 months	78-103	44-65	<70
Infant	4-6 months	82-105	46-68	<70
Infant	7-12 months	67-104	20-60	<70 + (age in years x 2)
Preschool	2-6 years	70-106	25-65	<70 + (age in years x 2)
School Age	7-14 years	79-115	38-78	<70 + (age in years x 2)
Adolescent	15-18 years	93-131	45-85	<90

TABLE 5: NORMAL BLOOD PRESSURE

D → Disability

One of the assessments of level of consciousness in a child is the Glasgow Coma Scale.

Response	Score	Verbal Child	Preverbal Child
Eye opening	4	Spontaneously	Spontaneously
	3	To verbal command	To speech
	2	To pain	To pain
	1	None	None
Verbal response	5	Oriented and talking	Cooing and babbling
	4	Confused but talking	Crying and irritable
	3	Inappropriate words	Crying with pain only
	2	Sounds only	Moaning with pain only
	1	None	None
Motor response	6	Obeys commands	Spontaneous movement
	5	Localizes with pain	Withdraws when touched
	4	Flexion and withdrawal	Withdraws with pain
	3	Abnormal flexion	Abnormal flexion
	2	Abnormal extension	Abnormal extension
	1	None	None
Total Possible Score	3-15		

TABLE 6: PEDIATRIC GLASGOW COMA SCALE

E → Exposure

If the provider finds any abnormal symptoms in this category they should assess and treat the child for shock (see Unit Seven: Management of Pediatric Shock, particularly Interventions for Initial Management of Shock).

During the primary assessment, if the child is stable and does not have a potentially life-threatening problem, continue with the secondary assessment.

SECONDARY ASSESSMENT

During the secondary assessment, complete a more in-depth history and physical exam in an attempt to identify the cause of the child's circulatory or respiratory symptoms. The physical exam should be focused on the specific symptoms the child is having. When conducting the history, remember the acronym 'SAMPLE'.

History Focus Areas	Information to Gather
Signs and symptoms	Changes in level of consciousness, respiratory problems, vomiting, diarrhea, fever, episodes of bleeding
Allergies	Environmental, food, medication and usual reactions
Medications	Any recent medications including dose and time any recent changes or additions of medications
Past medical/surgical history	Birth history Immunization history Respiratory, cardiac, neurological or surgical history
Last Meal	Time of meal and foods eaten
Events	Time of onset Description of the events leading to this episode Treatments already performed

TABLE 7: SECONDARY ASSESSMENT HISTORY

DIAGNOSTIC TESTS

Lab and other diagnostic tests may help to identify specific problems and the severity of the problems.

Test	Abnormal Result	Why Important	Interventions
Arterial blood gas	Hypoxemia pH abnormalities	Indicates the adequacy of ventilation	Increase or decrease ventilation efforts
Oxygen saturation	Any venous result outside of normal 70-75% range	Indicates the adequacy of oxygenation	Increase oxygen and ventilation efforts
Arterial lactate concentration	Increased lactate	An elevated lactate indicates an ill child Falling lactate levels can indicate success of treatment	See Post Cardiac Arrest Shock Management Algorithm
Arterial and CVP monitoring	Increased or decreased pressures	Can indicate results of fluid resuscitation	See Post Cardiac Arrest Shock Management Algorithm
Chest x-ray	Signs of obstruction or disease	May indicate respiratory issues Verifies position of ET tube	See Management of Pediatric Respiratory Distress or Failure
ECG	Cardiac arrhythmia	Indicates cardiac issues	Dependent on arrhythmia
Peak expiratory flow rate	Diminished PEFR	If the child can understand how to do the test, may indicate respiratory issues	See Lower Airway Obstruction Interventions
Echocardiogram	Valve problems Congenital abnormalities	May indicate problems with size and contractility of the heart	Dependent on diagnosis

TABLE 8: DIAGNOSTIC TESTS IN PALS

IDENTIFY

As the child is assessed, the goal is to identify the underlying problem causing the symptoms. Trauma, respiratory compromise, or circulatory problems can lead to respiratory arrest so it is important to identify the causal issue(s).

Problem	Type
Circulatory	Cardiogenic shock Distributive shock Hypovolemic shock Obstructive shock
Respiratory	Upper or lower airway obstruction Lung disease Disordered control of breathing
Trauma	May lead to shock or respiratory issues

TABLE 9: IDENTIFY CAUSE OF CONDITION

INTERVENE

Finally, the intervention step of the sequence will be based on the assessment and identification of any issues identified. PALS interventions to consider should include:

- Airway maintenance and ventilation support
- Activation of the emergency response system
- CPR
- Use of AED, defibrillator or monitor
- Oxygen delivery
- Fluid and medication administration

Each member on the PALS team must understand his role and how it relates to those of the other team members. A physician usually leads the team. The team leader must be able to:

- Organize the team
- Monitor the performance of each role
- Perform any skills if necessary
- Model appropriate behaviors
- Coach other members of the team as necessary
- Focus on provision of exceptional care
- Mentor the group by providing a critique of team and individual performance when the resuscitation is over.

Team members must only do tasks as identified by their scope of practice. Each team member must be able to:

- Understand his role in the pediatric resuscitation
- Perform the tasks assigned to him
- Understand the PALS protocols and sequences
- Act as a member of the team.

There are certain expectations and actions associated with them for members of the team:

Expectation	Team Leader Actions	Team Member Actions
Roles	Knows the abilities of each of the team members	Team member will let the team leader know if a task is beyond his skill level Asks for help if unable to complete a task
Communication	Clearly defines each task and verifies that assignments are understood Confirms performance of task	Informs the leader that task is understood Informs the leader when each task is completed
Messages	Speaks clearly and in a normal tone of voice when giving assignments and orders	Speaks clearly and in a normal tone of voice when acknowledging assignments and orders Feels comfortable questioning unclear orders
Knowledge Sharing	Asks for suggestions from team members for alternative actions when needed	Shares information with team Helps to identify actions that may be inhibiting the resuscitation effort
Intervention	Intervenes quickly but gently if a team member is about to perform an incorrect action or if a task is taking too long	Asks the leader to repeat an order if the member thinks an error will occur Feels comfortable suggesting alternative courses of action
Evaluation and Summary	Asks for suggestions for alternative actions from team members Is constantly aware of patient's responses Keeps team members informed of patient's current status and plans for change in actions Provides positive and corrective feedback as needed	Draws attention to changes in the patient's status or response to treatments

TABLE 10: TEAM EXPECTATIONS IN PALS

Respiratory distress is the most common cause of respiratory failure and cardiac arrest. If the intervention is not done early and aggressively, the outcome for the child is usually very poor.

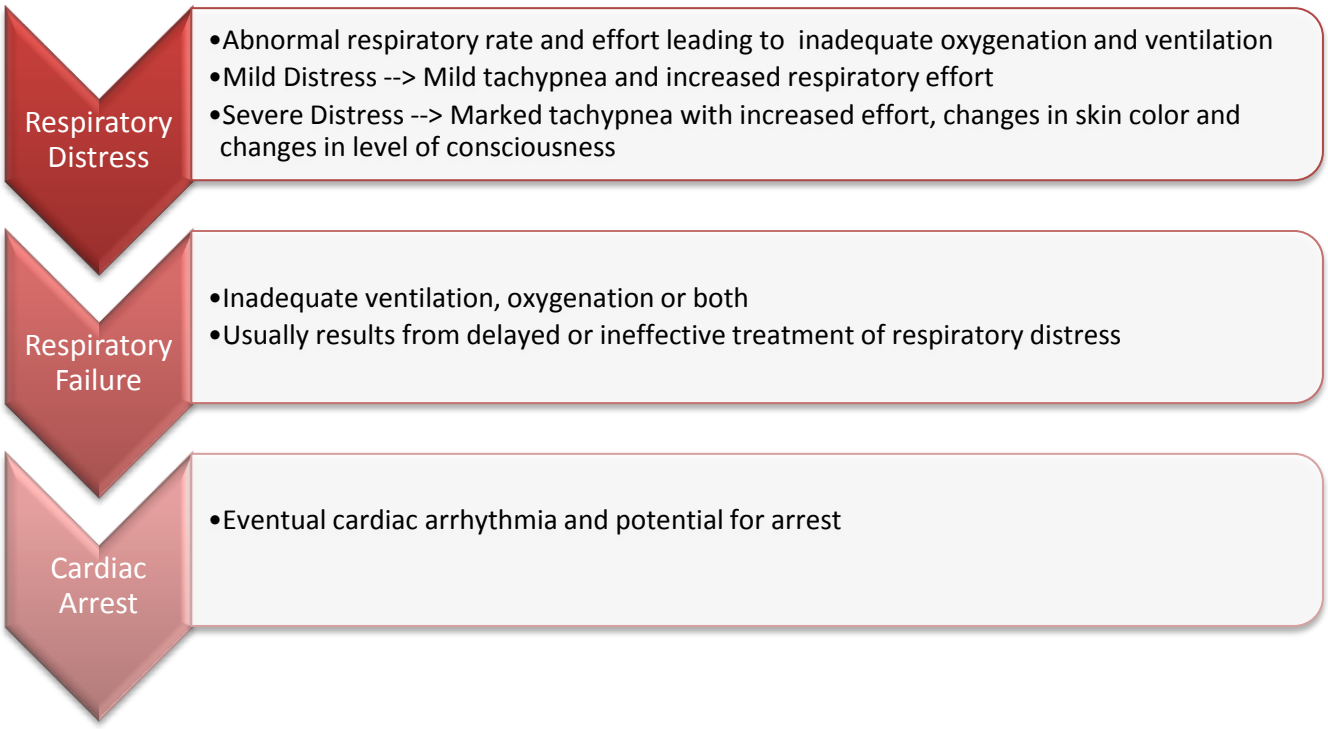


FIGURE 5: RESPIRATORY DISTRESS TO CARDIAC ARREST

Normal respirations are quiet and unlabored. As a respiratory problem develops due to disease, the child may begin to have labored breathing. This may be evidenced by:

- Increased airway resistance due to constriction, obstruction or inflammation
- Decreased lung compliance or stiffness resulting in increased effort to breathe
- Increased use of accessory muscles in the chest and neck during expiration
- Decreased CNS control of breathing resulting in hypoventilation and diminished oxygenation.

HYPOXEMIA

Hypoxemia is low tissue oxygenation resulting from inadequate oxygenation.

Signs and Symptoms	Treatment
Arterial oxygen <94%	Increase amount of oxygen delivered and rate of delivery Treat underlying cause
Tachycardia - fast heart rate	
Tachypnea - fast respiratory rate	
Nasal flaring, retractions	
Skin pallor or cyanosis (late sign)	Increase amount of oxygen delivered Assess for need for PALS Cardiac Arrest Algorithm
Bradypnea - slow respiratory rate (late sign)	
Bradycardia - slow heart rate (late sign)	
Diminished level of consciousness (late sign)	

TABLE 11: TISSUE HYPOXIA SIGNS AND TREATMENT

There are certain conditions that can cause hypoxemia. Some of these conditions can be treated:

- High altitude: Provide oxygen
- Overdose, spinal cord injury, or neuromuscular disease: Treat the underlying cause as appropriate and restore normal ventilation while increasing the delivery of oxygen
- Any of a number of respiratory diseases (e.g., pulmonary edema, pneumonia, collapsed lung, asthma, and foreign body obstruction): Increase oxygen and consider an advanced airway with PEEP;
- Congenital defects: Correct the defect.

HYPERCARBIA

Hypercarbia is increased arterial carbon dioxide caused by inadequate ventilation and carbon dioxide retention.

Signs and Symptoms	Treatment
Increased arterial carbon dioxide	Increase ventilatory rate Assess for need for PALS Cardiac Arrest Algorithm
Diminished level of consciousness (late sign)	
Tachypnea - fast respiratory rate	
Nasal flaring, retractions	
Bradypnea - slow respiratory rate (late sign)	

TABLE 12: HYPERCARBIA SIGNS AND TREATMENT

SIGNS OF RESPIRATORY PROBLEMS

Respiratory problems leading to distress can be classified into four categories: Upper airway obstruction, lower airway obstruction, lung disease and disordered control of breathing. In the Evaluate-Identify-Intervene sequence, the PALS team must quickly determine the underlying problem based on the clinical sign encountered during the assessment process.

Clinical Signs	Upper Airway Obstruction	Lower Airway Obstruction	Lung Disease	Disordered Control of Breathing
Airway patency	Maintainable in respiratory distress, not maintainable in respiratory failure			
Respiratory rate and effort	Initially increased in respiratory distress but will quickly decompensate to bradypnea and apnea in respiratory failure			Variable
Breath sounds	Stridor, cough and hoarseness	Prolonged expiratory wheezing	Grunting, diminished breath sounds, rales	Normal
Air movement	Diminished			Variable
Heart rate	Initially presents as tachycardia early in respiratory distress but will quickly decompensate to bradycardia and arrest as pediatric patient moves toward respiratory failure			
Skin color and temperature	Pale, cool, clammy skin in respiratory distress progressing rapidly to cyanosis in respiratory failure			Normal early
Level of consciousness	Increased agitation in respiratory distress progressing to lethargy and unresponsiveness in respiratory failure			
Core temperature	Variable			

TABLE 13: CLINICAL SIGNS BY RESPIRATORY PROBLEM

To prevent avoidable cases of cardiac arrest, the team must identify and intervene quickly when a respiratory problem is identified. Use the Evaluate-Identify-Intervene cycle to determine the cause of the respiratory distress, and then focus the interventions on the cause(s) that have been identified. Respiratory problems can be categorized as upper airway obstruction, lower airway obstruction, lung tissue disease, and disordered control of breathing.

UPPER AIRWAY OBSTRUCTION MANAGEMENT

Common causes of upper airway obstruction are croup, anaphylaxis, and foreign body obstruction.

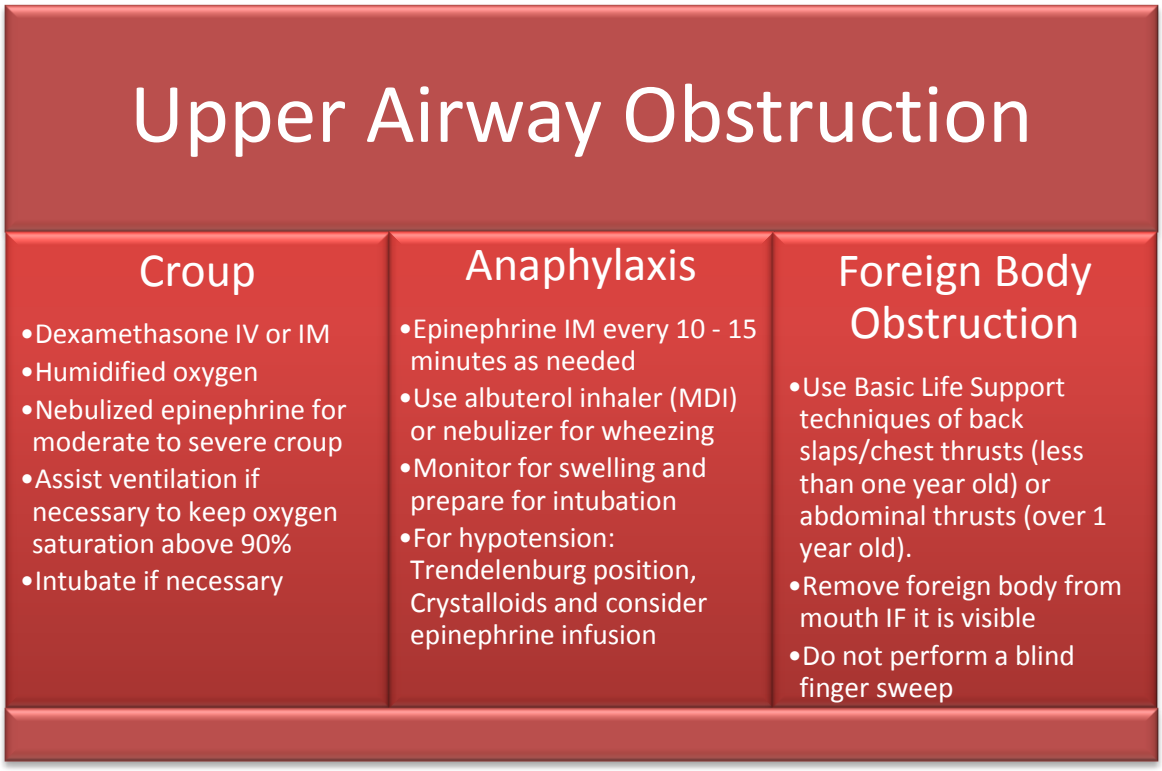


FIGURE 6: UPPER AIRWAY OBSTRUCTION INTERVENTIONS

Common causes of lower airway obstruction include bronchiolitis and asthma:

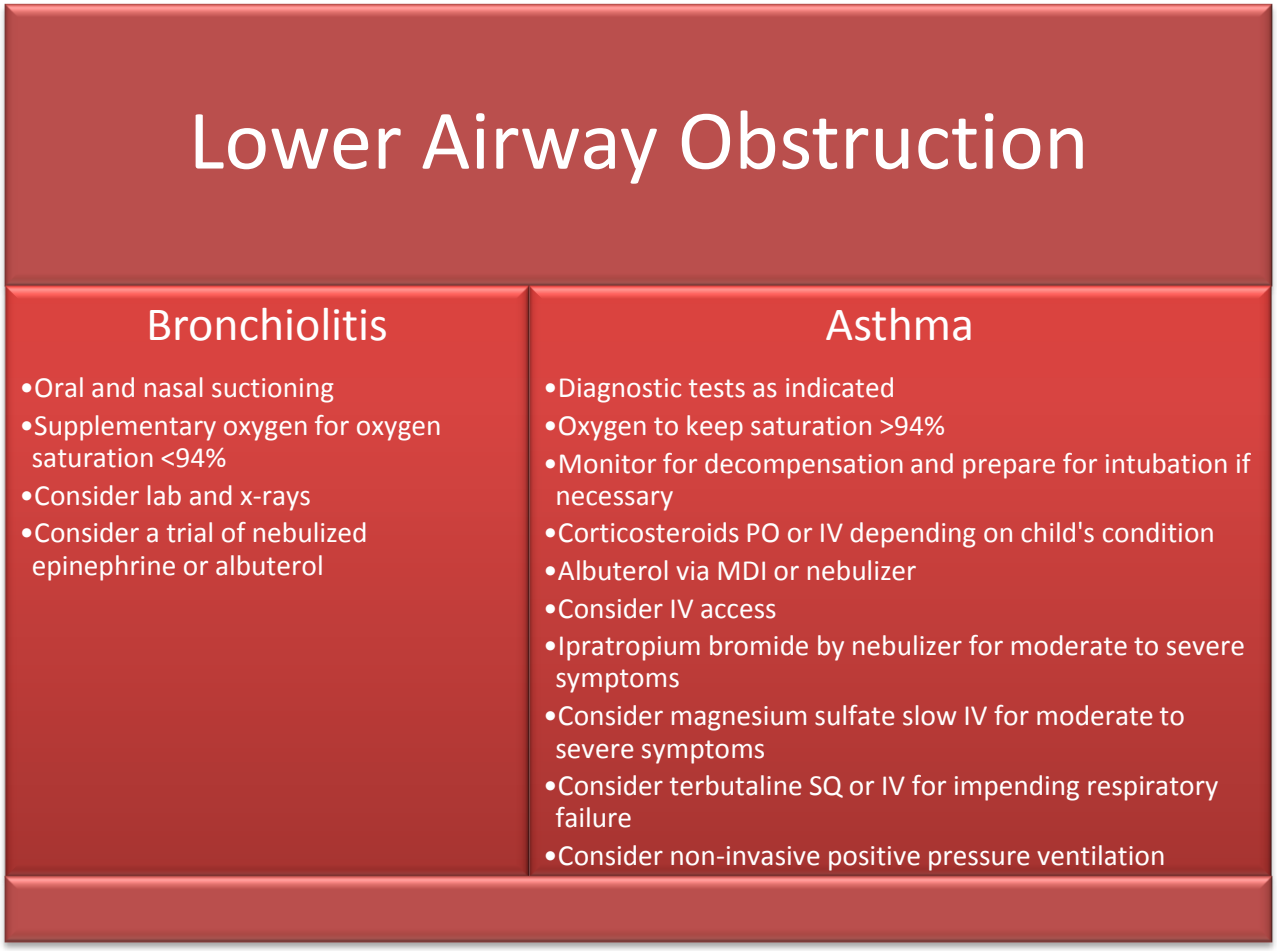


FIGURE 7: LOWER AIRWAY OBSTRUCTION INTERVENTIONS

Asthma is typically classified as mild, moderate, severe, or respiratory arrest imminent based on the evaluation of several parameters as developed by the World Health Organization and the National Heart, Lung and Blood Institute:

Parameter*	Mild	Moderate	Severe	Respiratory Arrest Imminent
Breathless	Walking Can lie down	Talking (Infant will have softer, shorter cry; difficulty feeding) Prefers sitting	At rest (Infant will stop feeding) Hunched forward	
Talks in	Sentences	Phrases	Words	
Alertness	May be agitated	Usually agitated	Usually agitated	Drowsy or confused
Respiratory rate	Increased	Increased	Often >30/min	
	Age — **Normal rate** <2 months — <60/min 2-12 months — <50/min 1-5 years — <40/min 6-8 years — <30/min			
Accessory muscles and suprasternal retractions	Usually not	Usually	Usually	Paradoxical thoraco-abdominal movement
Wheeze	Moderate, often only end-expiration	Loud	Usually loud	Absence of wheeze
Pulse/min	<100	100-120	>120	Bradycardia
	Guide to limits of normal pulse rate in children: **Age** — **Normal rate** Infants (2-12 months) — <160/min Toddler (1-2 years) — <120/min Preschool/school age (2-8 years) — <110/min			
Pulsus paradoxus	Absent <10 mm Hg	May be present 10-25 mm Hg	Often present >25 mm Hg (adult) 25-40 mm Hg (child)	Absence suggests respiratory muscle fatigue
PEF after initial bronchodilator % predicted or % personal best	>80%	Approximately 60%-80%	<60% predicted or personal best (<100 L/min adults) or response lasts <2 hours	
Pao_2 (on air) and/or $Paco_2$	Normal, test usually not necessary <45 mm Hg†	>60 mm Hg <45 mm Hg†	<60 mm Hg Possible cyanosis >45 mm Hg; possible respiratory failure	
Sao_2 %	>95%	91%-95%	<90%	

*The presence of several parameters, but not necessarily all, indicates the general classification of the attack.

†Hypercapnia (hypoventilation) develops more readily in young children than in adults and adolescents.

Reproduced from National Heart, Lung, and Blood Institute and World Health Organization. *Global Strategy for Asthma Management and Prevention NHLBI/WHO Workshop Report.* Bethesda, MD: US Department of Health and Human Services: 1997. Publication 97-4051.

FIGURE 8: CLASSIFICATION OF ASTHMA

LUNG TISSUE DISEASE MANAGEMENT

Common causes of lung tissue disease are pneumonia/pneumonitis and pulmonary edema.

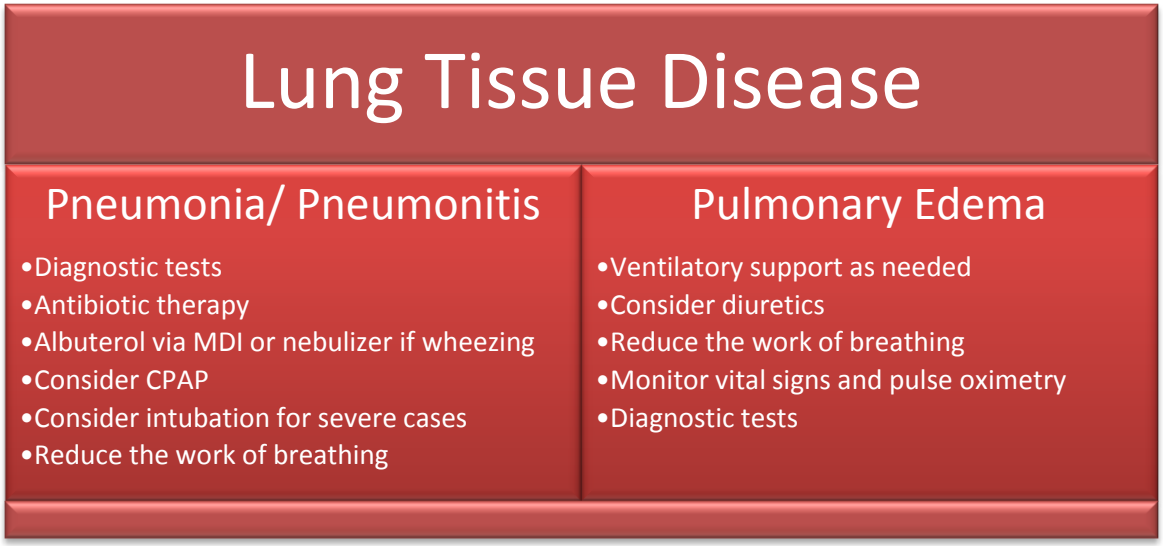


FIGURE 9: LUNG TISSUE DISEASE INTERVENTIONS

DISORDERED CONTROL OF BREATHING MANAGEMENT

Common causes of disordered control of breathing are increased intracranial pressure, toxic poisoning and neuromuscular disease.

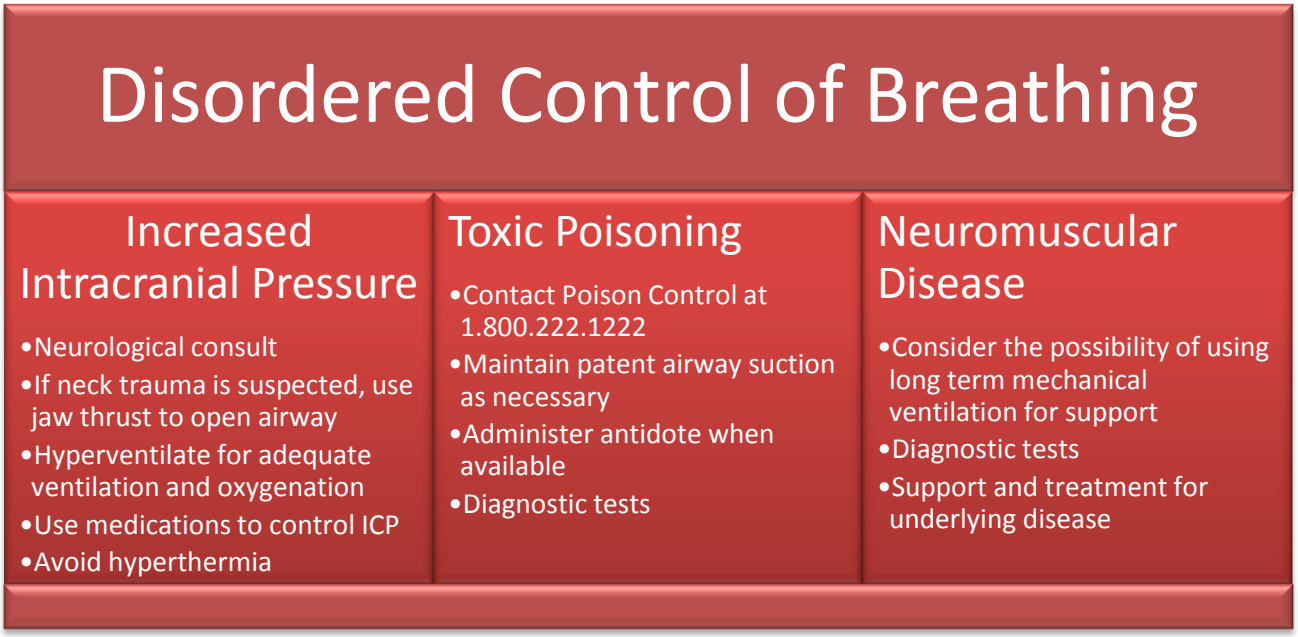


FIGURE 10: DISORDERED CONTROL OF BREATHING INTERVENTIONS

EQUIPMENT FOR RESPIRATORY MANAGEMENT

All treatment in a respiratory emergency must start with a patent airway and adequate oxygenation to maintain the child's oxygen saturation at 94% or higher on room air. Although intubation is often considered the best treatment for a compromised child, studies have shown that ventilation with a bag-mask device was effective when used properly. The mask should cover the child's nose and mouth without covering the eyes or hanging over the chin. Oxygen should be running to the bag at all times. If the bag has a pop-off valve, be sure it is closed. Open the airway and provide ventilations according to BLS standards.

Too much ventilation can cause the abdomen to distend leading to compromised lung filling. Avoid excessive volumes using a bag use just enough to make the child's chest rise. Monitor the child's condition and vital signs (including oxygen saturation) during ventilation. If the child's condition deteriorates and intubation becomes necessary, the following equipment should be at hand:

- Gloves, mask, eye protection
- Monitors: Cardiac, blood pressure, pulse oximetry, carbon dioxide detector (if available)
- IV/IO equipment
- Suction equipment
- Airways of all sizes oxygen supply and bag masks
- Various sizes of endotracheal tubes and laryngoscopes
- Large syringes
- Adhesive tape to secure tube
- Pediatric length-based resuscitation tape (see next section)

PEDIATRIC LENGTH BASED RESUSCITATION TAPE

During resuscitation of a pediatric patient, it is important to know correct sizes of tubes, masks, airways, and catheters based on the child's size. A weight-based system should be available to facilitate accurate selections in a code situation. Many of these systems are organized so that a rescuer can pull a packet based on the child's size that will contain appropriately sized equipment.

FIGURE 11: EXAMPLE OF A WEIGHT BASED SYSTEM

UNIT SIX: RECOGNITION OF PEDIATRIC SHOCK

Shock develops when the body can no longer deliver oxygen and other nutrients to the cells. Therefore, the goal of treatment for shock is to increase oxygen to the cells. Be aware that shock CAN be present even when the blood pressure is normal. In order to ensure adequate oxygen delivery, there must be enough blood and oxygen, appropriate cardiac output, and appropriate distribution of flow. The types of shock are defined by disruptions in these required elements:

- Inadequate blood volume (hypovolemic shock)
- Inappropriate distribution of blood volume (distributive shock)
- Disruptions in cardiac output (cardiogenic shock)
- Obstructed blood flow (obstructive shock).

PATHOPHYSIOLOGY IN SHOCK

The amount of blood pumped by the heart per minute is defined as:

Stroke volume (based on preload, afterload and contractility) × Heart rate = Cardiac output.

Pediatric patients have very small stroke volume; therefore, pediatric cardiac output is primarily dependent on heart rate. Inadequate preload results in hypovolemic shock, whereas poor contractility and increased afterload can result in cardiogenic shock.

COMPENSATORY MECHANISMS IN SHOCK

When the body senses that a shock state is imminent, it attempts to compensate. The child with a normal blood pressure but poor perfusion is in compensated shock. In compensated shock, blood is shunted from the periphery and non-vital organs to the heart and brain. There are several compensatory mechanisms that include:

Mechanism	What You will See in Shock
Elevated heart rate	Tachycardia; reduction in cardiac output
Increased vasoconstriction	Cool, clammy, pale or mottled skin Delayed capillary refill in the extremities Weak peripheral pulses Decreased pulse pressure
Redistribution of blood flow from non-vital organs	Decreased urine output; vomiting may occur

TABLE 14: COMPENSATORY MECHANISMS IN SHOCK

When the body can no longer compensate, hypotensive shock will develop signaling impending cardiac arrest. As the child decompensates, ominous signs will include: Decreasing level of consciousness, bradycardia, and weakening central pulses.

Clinical Sign	**Hypovolemic** (Volume loss)	**Distributive** (Inappropriate volume distribution)	**Cardiogenic** (Myocardial dysfunction)	**Obstructive** (Physical obstruction)
Potential Causes	Vomiting or diarrhea, bleeding, DKA, fluid leaking from the cells into tissues, burns, decreased fluid intake	Sepsis, head injury, spinal injury, anaphylaxis	Congenital heart disease, poisoning, myocarditis, cardiomyopathy, arrhythmias	Cardiac tamponade, tension pneumothorax, congenital heart defects, pulmonary embolism
Preload	Decreased	Normal or decreased	Variable	Variable
Contractility	Normal or increased	Normal or decreased	Decreased	Normal
Afterload	Increased	Variable	Increased	Increased
Airway patency	Airway open and maintainable early, not maintainable in later stages			
Respiratory rate and effort	Increased rate but no increased effort	Increased rate possible with increased effort	Increased effort with retractions and nasal flaring	Increased and labored
Breath sounds	Normal	Rales may be present	Rales and grunting	Rales and grunting, breath sounds may be diminished or absent with tension pneumothorax
Systolic BP	May be normal in compensated shock but will eventually lead to hypotensive shock			
Pulse pressure (difference between systolic and diastolic)	Narrow	Variable	Narrow	Narrow
Heart rate	Increased	Increased	Increased	Increased with muffled heart sounds
Peripheral pulses	Weak	Bounding or weak	Weak or absent, jugular venous distention	Weak
Capillary refill	Delayed	Variable	Delayed	Delayed
Skin color and temperature	Pale, clammy, cool, mottled	Warm, flushed, pale, mottled In septic shock may have petechial or purpuric rash, hives in anaphylactic shock	Pale, cool extremities	Pale, cool, possible cyanosis in pulmonary embolism
Urine output	Decreased			
Level of consciousness	Irritable and anxious Decreased level of consciousness is a late and ominous sign			
Core temperature	Variable			

TABLE 15: SIGNS OF SHOCK BY TYPE

When the resuscitation team is presented with a child in shock the goals for treatment are to:

- Improve tissue oxygenation
- Decrease oxygen demand
- Treat causes of shock
- Repair lost organ function
- Prevent cardiac arrest.

As the pediatric patient's condition begins to worsen, there are certain signs the team should be aware of:

- Rapid heart rate
- Diminished peripheral pulses
- Possibly weakened central pulses
- Narrowing pulse pressure
- Cool pale extremities
- Prolonged capillary refill
- Deteriorating level of consciousness
- Low blood pressure (late sign).

INITIAL MANAGEMENT

Initial management of shock must include:

Intervention	Specific Actions
Position	Allow the child to assume a comfortable position (consider Trendelenburg)
Oxygen	High oxygen concentration with possible mechanical ventilation (PEEP) If anemia is present, consider blood transfusion
Vascular access	IV or IO as soon as possible
Fluids	Crystalloid bolus (consider packed cells for blood loss) 20 mL/kg over 5-20 minutes Monitor for cardiac issues during fluid resuscitation
Assessment	Frequent secondary assessments Monitor vital signs including oxygen saturation, urine output, and mental status
Labs	As indicated for type of shock
Medications	Administer appropriate medication in appropriate doses (see Unit Twelve): • Dopamine to increase heart rate and contractility • Dobutamine to increase heart rate and contractility • Epinephrine to increase systemic vascular resistance, heart rate & contractility • Inamrinone to decrease systemic vascular resistance and increase contractility • Milrinone to decrease systemic vascular resistance and increase contractility • Nitroglycerine to decrease systemic vascular resistance • Nitroprusside to decrease systemic vascular resistance • Norepinephrine to increase systemic vascular resistance and contractility • Vasopressin to increase systemic vascular resistance and contractility

TABLE 16: INTERVENTIONS FOR INITIAL MANAGEMENT OF SHOCK

Signs that shock is resolving include:

- Heart rate and blood pressure within normal range for child's age
- Strong peripheral pulses with warm, pink extremities and brisk capillary refill
- Improved mental status
- Urine output >1 mL/kg/hour (or >30 mL/hour for adolescents)
- Normal or improving oxygen saturation
- Decreased serum lactate.

SHOCK: FLUID AND MEDICATIONS

The golden hour for shock is the first hour. During this hour, the pediatric patient must receive the appropriate treatment in order to enhance his chance of survival. The first step in this process is to recognize the type of shock and appropriate treatment:

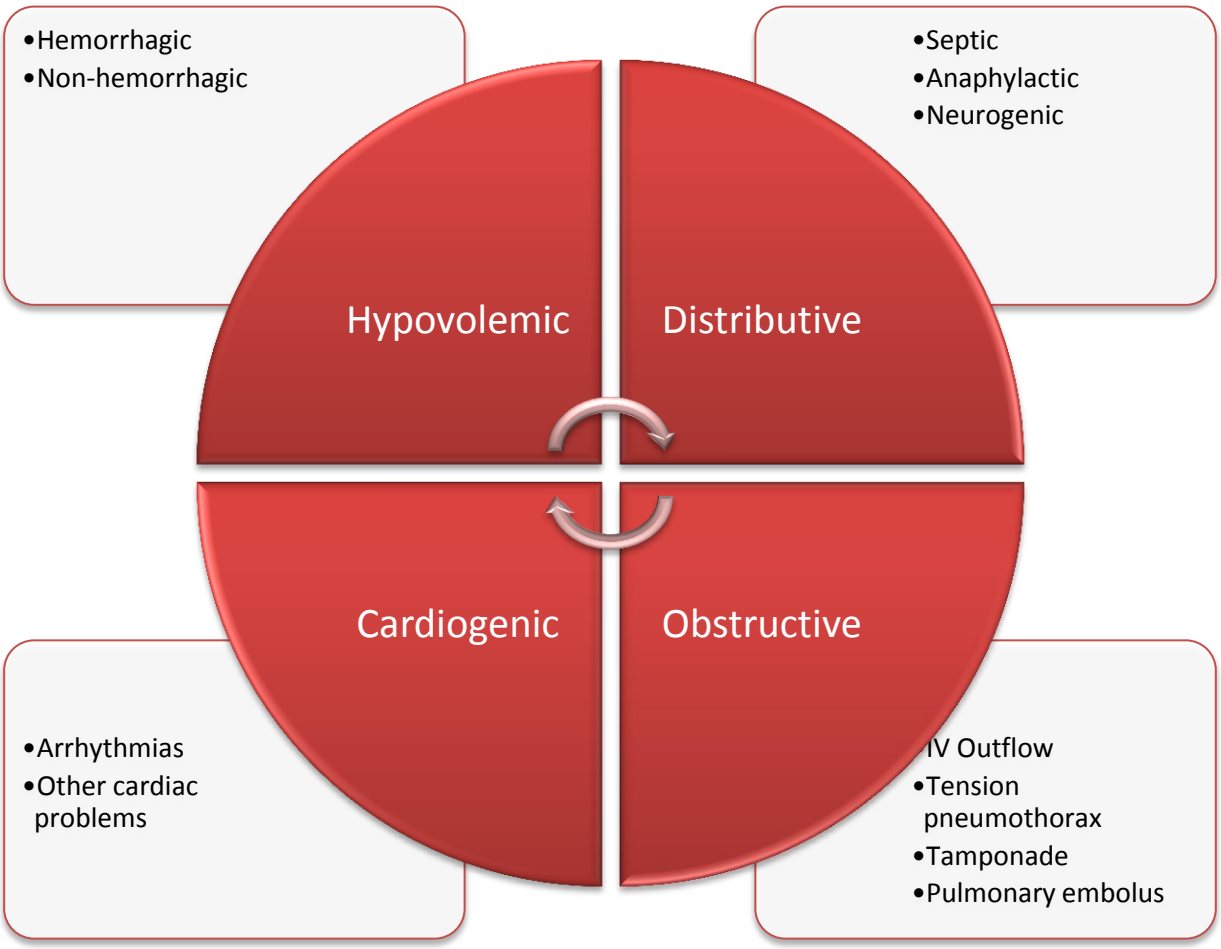


FIGURE 12: TYPES OF SHOCK

It is important to administer fluid resuscitation in shock with extreme caution. Patients should be monitored for signs of fluid overload or cardiovascular deterioration. Using these treatments presumes it is done within facilities that have the equipment and personnel available to treat any secondary effects of IV fluid boluses. The institution should have age- and size-appropriate equipment in an intensive care setting, such as ventilators, arterial pressure monitoring systems, and vasopressors.

Hypovolemic Shock Treatment:

- Hemorrhagic: Stop external bleeding. Administer fluid 20 mL/kg crystalloid bolus and repeat until vital signs and oxygenation restored. Administer packed red blood cells for extreme blood loss.
- Non-hemorrhagic: Administer fluid 20 mL/kg crystalloid bolus and repeat until vital signs and oxygenation restored. Consider colloid infusion if crystalloids are not effective.

Distributive Shock Treatment:

- Septic: Administer fluid 20 mL/kg crystalloid bolus and repeat until vital signs and oxygenation are restored. Administer dopamine, epinephrine or norepinephrine OR consider milrinone or nitroprusside OR dobutamine.
- Anaphylactic: Epinephrine bolus followed by infusion. Administer fluid 20 mL/kg crystalloid bolus and repeat until vital signs and oxygenation are restored. Consider albuterol or antihistamines.
- Neurogenic: Administer fluid 20 mL/kg crystalloid bolus and repeat until vital signs and oxygenation are restored. Consider a vasopressor.

Cardiogenic Shock Treatment:

- Bradyarrhythmias or tachyarrhythmias: Follow the appropriate “Poor Perfusion” algorithm based on heart rate (bradycardia or tachycardia).
- Other conditions leading to cardiogenic shock: Administer fluid 5-10 mL/kg crystalloid bolus and repeat until vital signs and oxygenation restored. Observe for fluid overload. Infuse pressors. Consult cardiology.

Obstructive Shock Treatment:

- Pulmonary embolus: Administer fluid 20 mL/kg crystalloid bolus and repeat until vital signs and oxygenation are restored. Consider anticoagulants or thrombolytics. Consult pediatric cardiologist/pulmonologist.
- Cardiac tamponade: Administer fluid 20 mL/kg crystalloid bolus and repeat until vital signs and oxygenation restored. Pericardiocentesis.
- IV outflow: Administer prostaglandin E.
- Tension Pneumothorax: Needle decompression or thoracostomy.

Intraosseous (IO) access is a viable alternative to an IV when starting an IV is difficult or when CPR must be interrupted for establishment of an IV.

Possible sites for an IO port include the proximal or distal tibia, distal femur, or anterior superior iliac spine.

Do not establish IO access in a bone that is fractured, if there appears to be infection near the site, or if IO attempts have been made in the same bone.

After inserting the IO catheter, be sure to protect the site and replace it with an IV as soon as possible. If a person fails to achieve IO access after breaking the skin, the duty should be passed to someone with more experience.

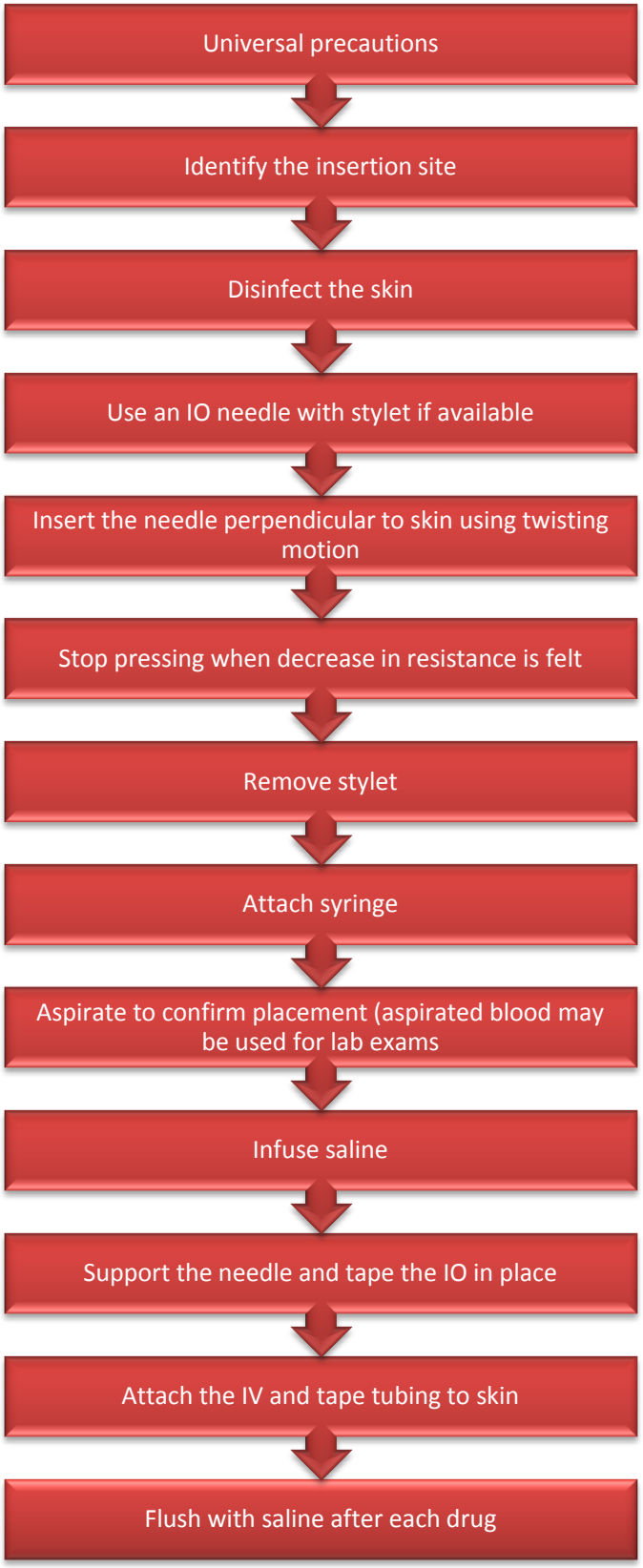


FIGURE 13: INTRAOSSEOUS ACCESS

UNIT EIGHT: RECOGNITION AND MANAGEMENT OF PEDIATRIC BRADYCARDIA

Bradycardia is a heart rate slower than normal for the child's age and activity level. See Unit 2 for normal heart rate ranges for pediatric patients. In the pediatric population, a heart rate less than 60 beats per minute is an ominous sign and CPR should be initiated immediately. Bradycardia is often the cause of hypoxemia and respiratory failure in infants and children.

The exception to this is when a child has primary bradycardia caused by congenital or structural conditions such as congenital abnormalities, cardiomyopathy, myocarditis, or surgical injury. These children must be evaluated by a pediatric cardiologist. If CPR is indicated, do not delay while waiting for the pediatric cardiologist. Secondary bradycardia results from non-cardiac issues including low blood pressure, hypoxia, hypothermia, and some drugs.

SIGNS AND SYMPTOMS OF BRADYCARDIA

System	Sign or Symptom
Airway patency	Usually not affected
Respiratory rate and effort	Respiratory distress or failure
Systolic BP	Hypotensive
Heart rate	Slower than normal for age/activity
ECG characteristics	P wave may/may not be visible QRS complex narrow or wide P wave and QRS complex → AV dissociation
Peripheral pulses	Decreased or absent
Capillary refill	Increased time to pink after blanching
Skin color and temperature	Cool and pale
Level of consciousness	Decreased level of consciousness, fatigue, dizziness

TABLE 17: SIGNS OF BRADYCARDIA BY SYSTEM

UNDERLYING CAUSES OF BRADYCARDIA

Underlying causes of bradycardia may include some of the H's and T's:

- Hypoxia: Administer oxygen
- Acidosis: Treated with increased ventilation and use sodium bicarbonate carefully if needed
- Hyperkalemia: Restore normal potassium level
- Hypothermia: Re-warm slowly to avoid over-heating
- Heart block: Consult pediatric cardiologist for possible administration of atropine, chronotropic drugs, and external pacemaker
- Toxins/overdoses: Supportive care (administer antidote if one is available)
- Trauma: Increase oxygen and ventilation and avoid increased intracranial pressure by treating bradycardia aggressively in cases of head trauma.

When a pediatric patient is bradycardic with poor perfusion, follow the PALS Bradycardia Algorithm (Figure 14):

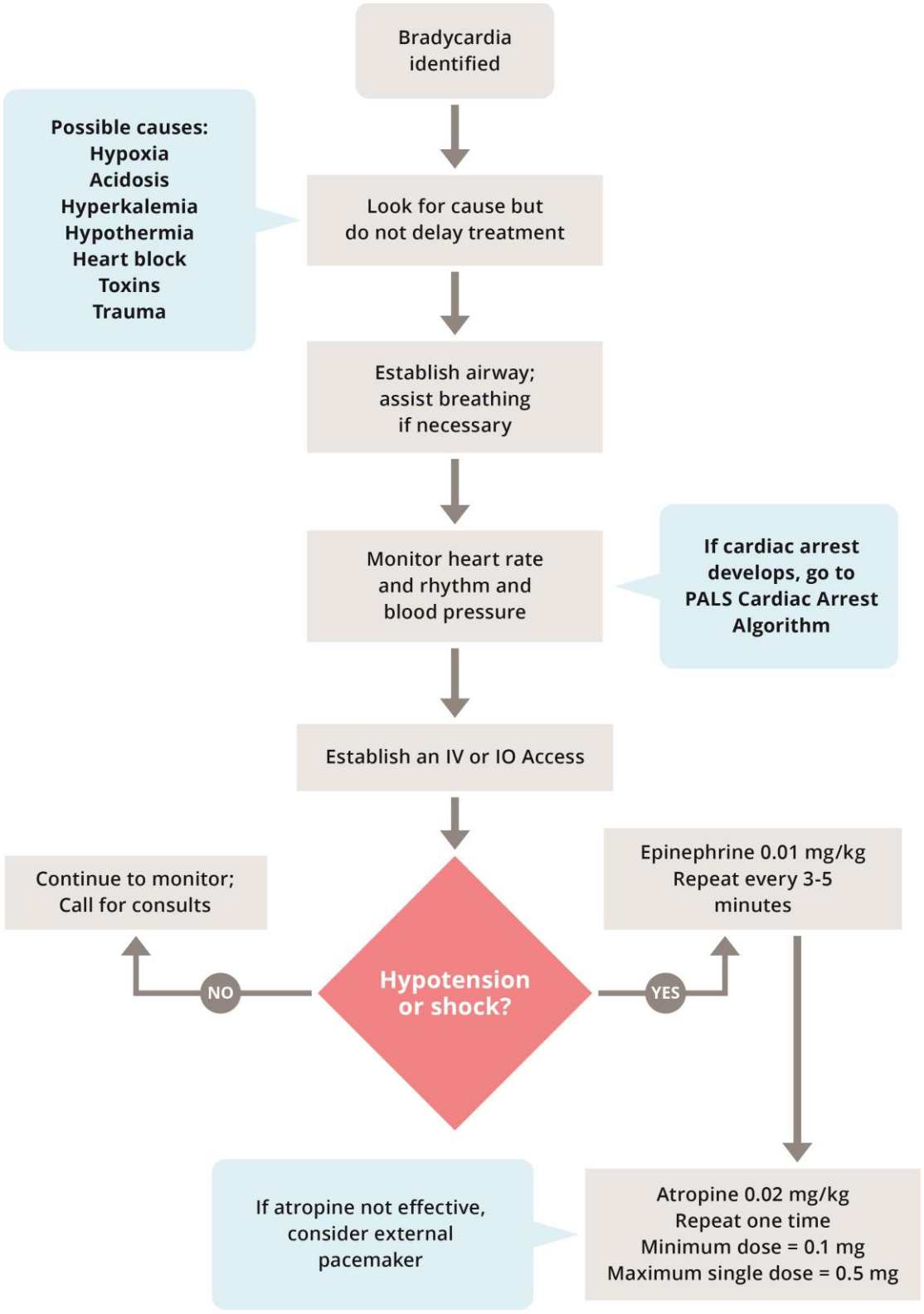


FIGURE 14: PALS BRADYCARDIA ALGORITHM

Tachycardia is a faster than normal heart rate for the child's age and activity level. Typically, a tachycardia is classified as narrow QRS complex (QRS <0.09 seconds) or wide QRS complex (QRS >0.09 seconds).

Sinus tachycardia (ST) is a narrow complex tachycardia that is not a dysrhythmia. Supraventricular tachycardia (SVT) may be wide or narrow complex, originates above the ventricles, and is the most common tachycardic rhythm in the pediatric population. As in adults, ventricular tachycardia (VT) is a wide complex tachycardia that, if left untreated, can deteriorate to ventricular fibrillation (VF), cardiac arrest and death. Fortunately, VT is not very common in children and infants.

SIGNS AND SYMPTOMS OF TACHYCARDIA

Signs and symptoms of tachycardia will depend on the type of tachycardia that is present:

System	Sinus Tachycardia	SVT	VT
Onset	Commonly associated with pain, fever, hemorrhage or dehydration; ST is gradual in onset	Sudden often with palpitations	Sudden but uncommon in children unless associated with an underlying condition
Airway patency	Not affected	Not affected	Not affected
Respiratory rate and effort	Faster than normal	Faster than normal often with rales and wheezes; increased work of breathing	Faster than normal
Systolic BP	Variable	Usually lower than normal	Variable
Heart rate	Infant <220/minute Child <180/minute Rate typically increases with activity or severity of illness	Infant >220/minute Child >180/minute Rate not affected by activity	Greater than 120 beats per minute and regular
ECG characteristics	Narrow QRS complex; P waves normal; PR interval constant; R-R interval may be variable	Narrow or wide QRS complex; P waves absent or abnormal; R-R interval may be constant	Wide QRS complex; P waves may not be present or seen; QRS complexes may be uniform or variable
Peripheral pulses	Normal	Weak	Weak
Capillary refill	Normal	Increased time to pink	Increased time to pink
Skin color and temperature	Cool and pale	Cool, clammy, pale, mottled, gray or cyanotic	Cool and pale
Level of consciousness	May be light-headed or dizzy	Diminished level of consciousness; dizzy, light-headed	Diminished level of consciousness; dizzy, light-headed

TABLE 18: SIGNS OF TACHYCARDIA BY SYSTEM

Pulseless tachycardia is treated as cardiac arrest. Providers should follow the PALS Cardiac Arrest Algorithm in cases of pulseless tachycardia (see Figure 21). Patients who have tachycardia and a palpable pulse should be initially managed as follows:

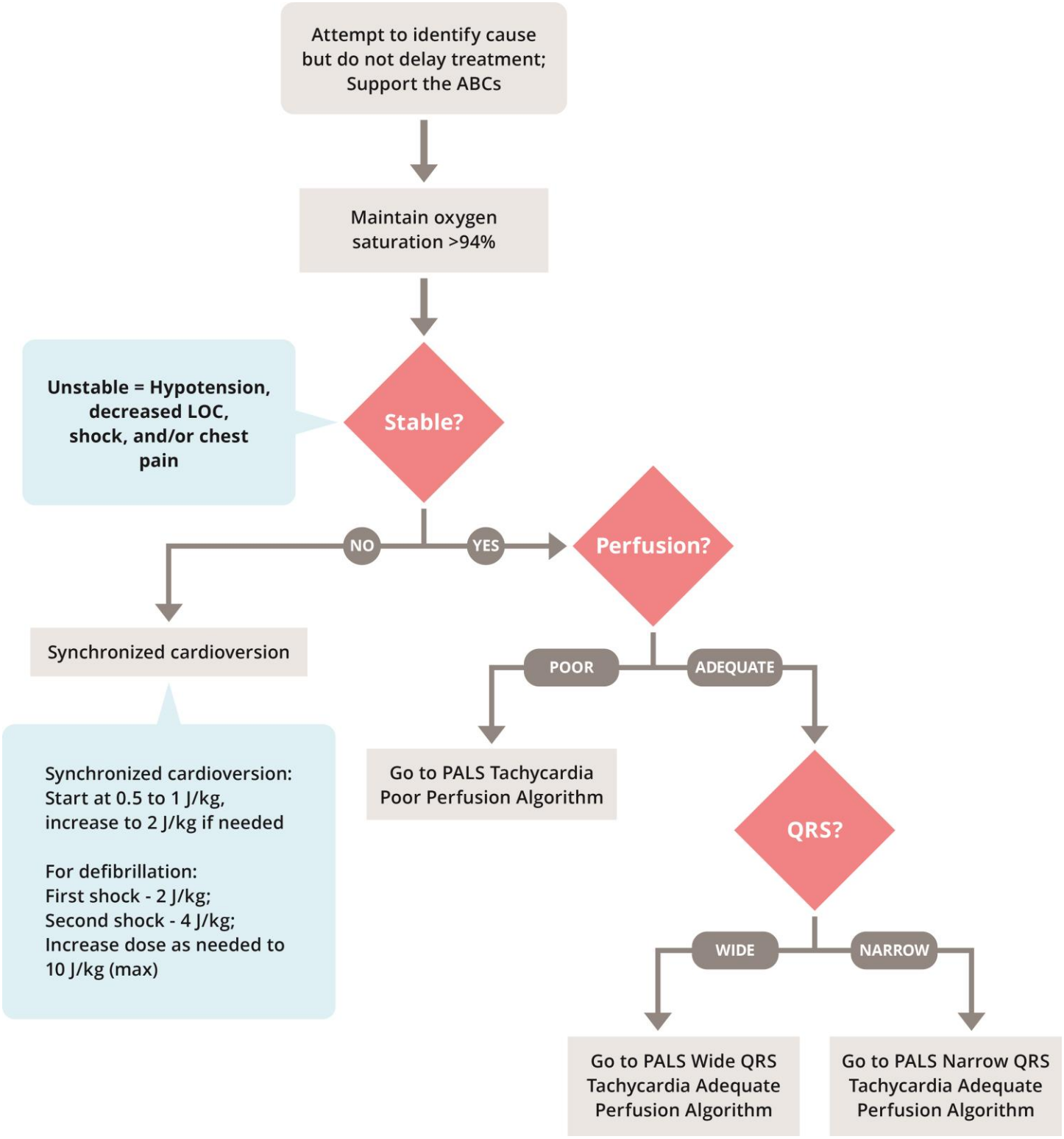


FIGURE 15: PALS TACHYCARDIA INITIAL MANAGEMENT ALGORITHM

Interventions designed specifically for emergency management of tachycardia include:

- Vagal maneuvers: If the child is old enough to understand instructions, have him blow through a straw that is partially pinched shut. For an infant, try an ice bag to the top half of his face for 15 seconds. Carotid massage may be done on older children.
- Synchronized cardioversion: Unstable patients may require synchronized cardioversion. If the cardiac monitor has a synchronization mode, synchronized cardioversion may help to slow the heart rate.
- For synchronized cardioversion, begin with an electrical dose of 0.5 to 1 J/kg of the child's body weight. If ineffective, increase the energy level to 2 J/kg.
- For defibrillation (cardiac arrest with a shockable rhythm), first shock should be given at 2 J/kg and the second shock should be given at 4 J/kg. Subsequent shocks may be higher, up to the adult maximum of 10 J/kg body weight.

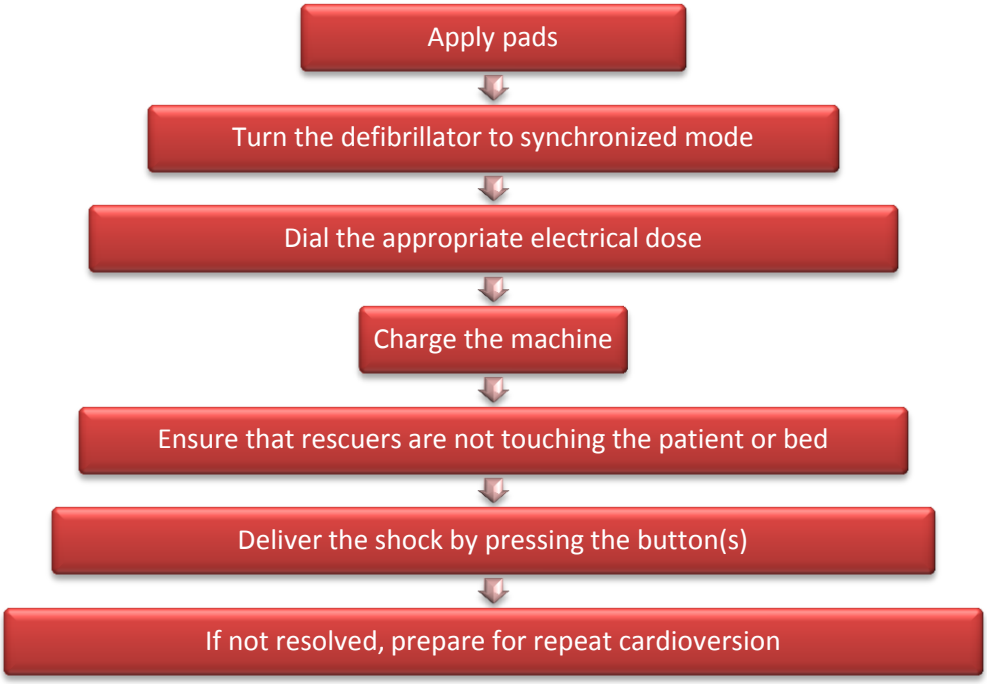


FIGURE 16: SYNCHRONIZED CARDIOVERSION

Definitive and ongoing treatment for tachycardia will depend on:

- Whether or not there is adequate perfusion with the rhythm
- The width of the QRS complex
- The identification of the specific tachycardia

Relatively stable patients (conscious, normotensive, and without chest pain) who have poor perfusion should be managed using the PALS Tachycardia Poor Perfusion Algorithm (see Figure 17).

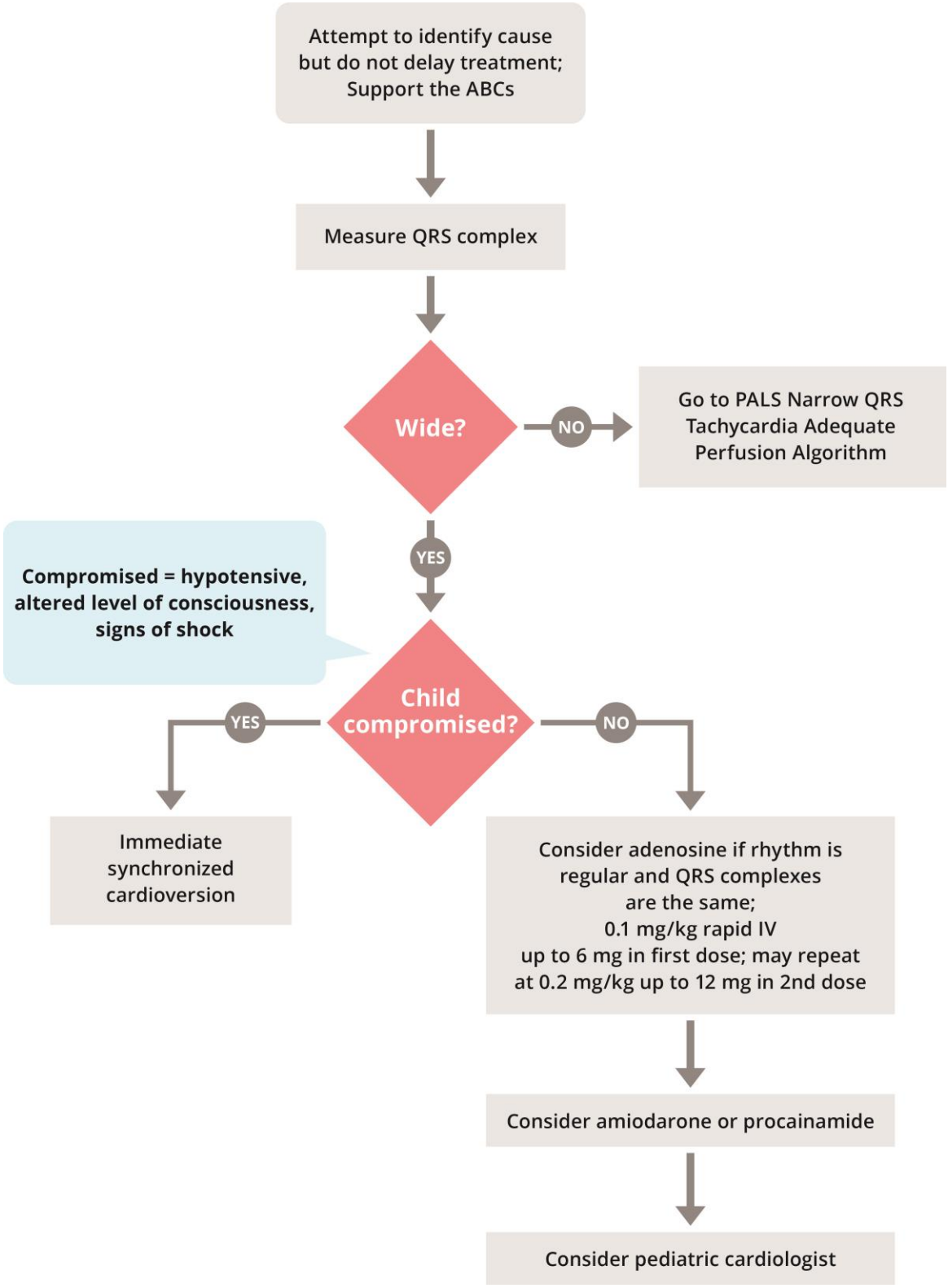


FIGURE 17: PALS TACHYCARDIA POOR PERFUSION ALGORITHM

Children with tachycardia, a palpable pulse, and adequate perfusion are stable enough to tolerate additional investigation into the cause of the tachycardia. The main distinction in this case is whether the QRS complex is narrow (≤0.09 sec) or wide (> 0.09 sec). Narrow and wide QRS algorithms are shown in Figures 18 and 19 respectively.

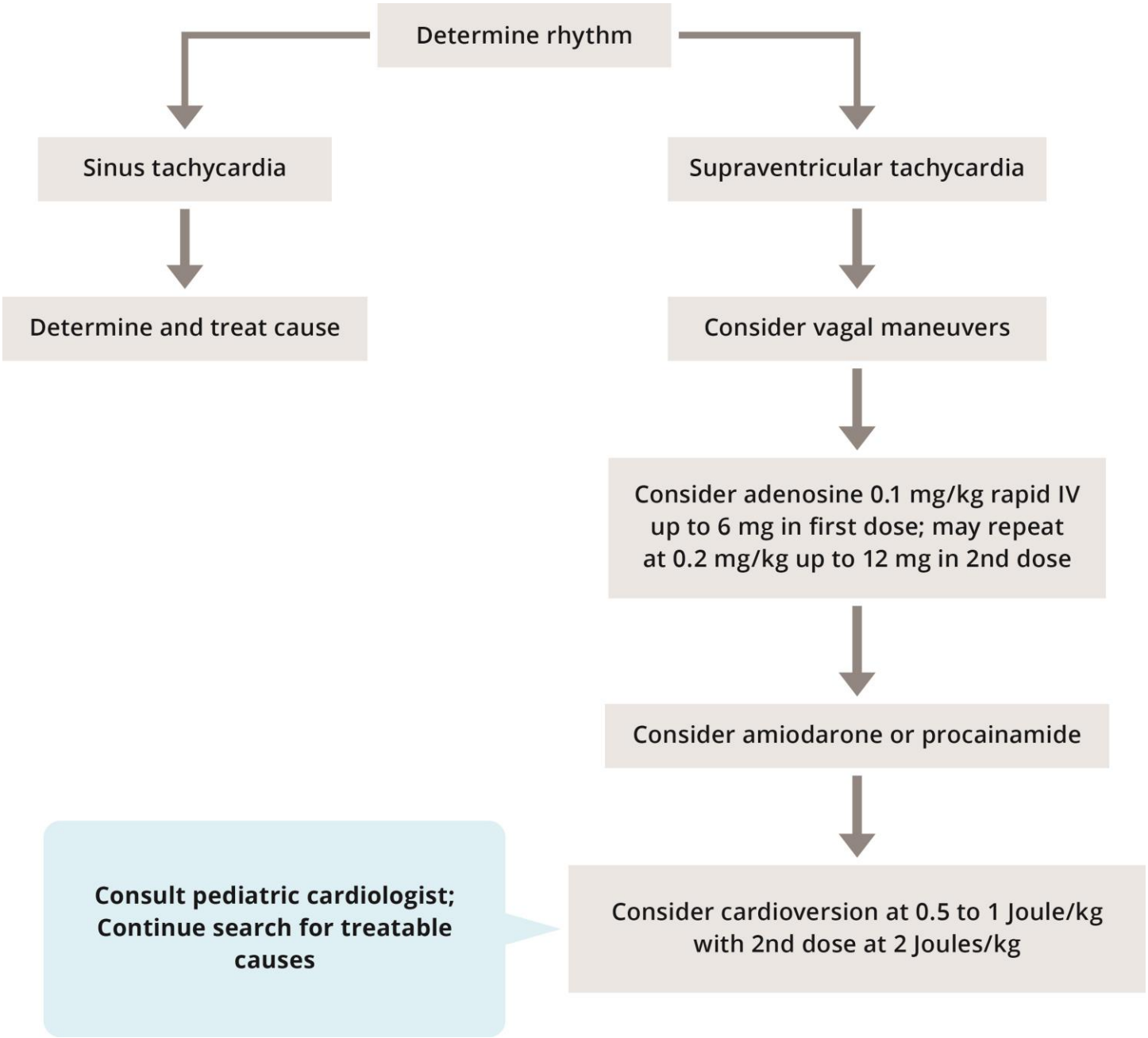


FIGURE 18: PALS NARROW QRS TACHYCARDIA ADEQUATE PERFUSION ALGORITHM

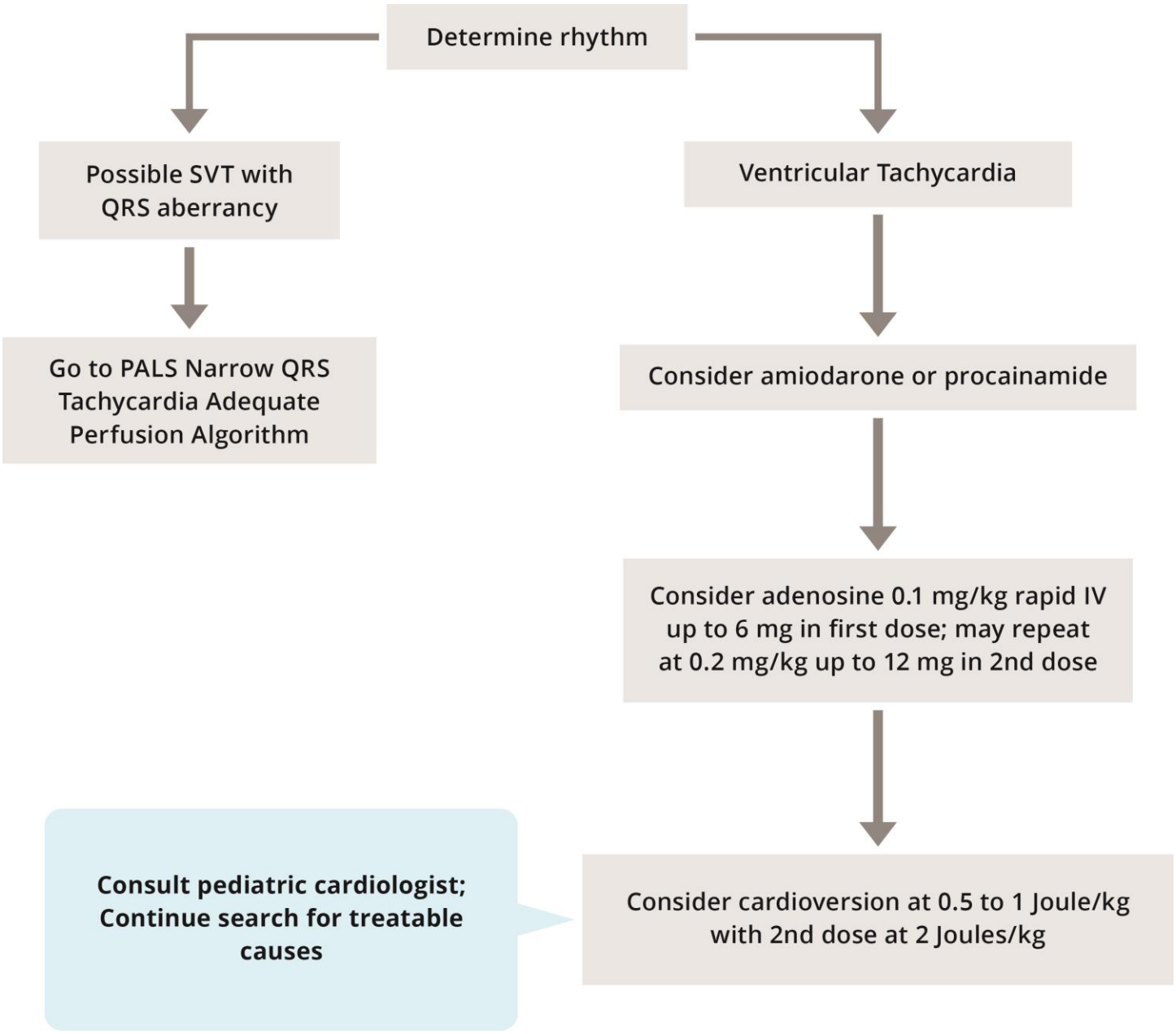


FIGURE 19: PALS WIDE QRS TACHYCARDIA ADEQUATE PERFUSION ALGORITHM

UNIT TEN: RECOGNITION AND MANAGEMENT OF PEDIATRIC CARDIAC ARREST

Cardiac arrest is the absence of circulation and pulses caused by ineffective or absent cardiac activity. In cardiac arrest, the child is pulseless and unresponsive and breathing is absent or gasping. Cardiac arrest in children is typically hypoxic or asphyxial arrest as a result of respiratory distress or shock. Sudden cardiac arrest (SCA) is less common in children and is typically caused by VF or pulseless VT.

Remember that cardiac arrest in the pediatric population is generally preceded by respiratory distress, respiratory failure, or shock so it is critical to intervene before those conditions progress to cardiac arrest. The highest rate of survival is when there is bradycardia with immediate CPR because once a child is in asystole, the prognosis and outcomes are very poor. VF and VT in children are reversible if the underlying cause is determined and treated quickly.

Treatable causes of cardiac arrest are known as the H's and T's (see Unit Eleven: Management of Shock Post-Resuscitation) for a listing of the H's and T's.

CARDIAC ARREST RHYTHMS

Cardiac arrest is typically identified as one of the arrest rhythms:

- Asystole (cardiac standstill or flat line) is the absence of any electrical activity on the ECG.
- Pulseless electrical activity (PEA) is defined as any rhythm with electrical activity on the ECG without palpable pulses in the patient.
- Ventricular fibrillation (VF) is seen as unorganized, chaotic electrical activity on the cardiac monitor with no palpable pulses in the patient. VF is one of the “shockable” rhythms.
- Pulseless ventricular tachycardia (VT) is seen on the monitor as an organized rhythm with wide QRS complexes and no pulses in the patient. The danger of pulseless VT is that it will deteriorate into VF. Typically, VT is more readily converted than VF so it is critical to treat pulseless VT quickly.

BLS COMPONENTS FOR MANAGEMENT OF CARDIAC ARREST

Management of cardiac arrest in the pediatric population must begin with high-quality CPR following the CAB (Circulation, Airway, Breathing) sequence. BLS components for children and infants include:

BLS Component	Children	Infants
Compression rate	100-120/minute	
Compression depth	About 2 inches (1/3 diameter of chest)	About 1.5 inches (1/3 diameter of chest)
Compression to ventilation ratio	30:2 for single rescuer 15:2 for team rescue	
Ventilations	1 breath every 6-8 seconds with visible chest rise if advanced airway is in place	
Defibrillation	Use AED when available	

TABLE 19: BLS COMPONENTS IN CARDIAC ARREST

In cardiac arrest in children, the ultimate goal is return of spontaneous circulation (ROSC). To accomplish this goal, certain principles of ALS must be accomplished:

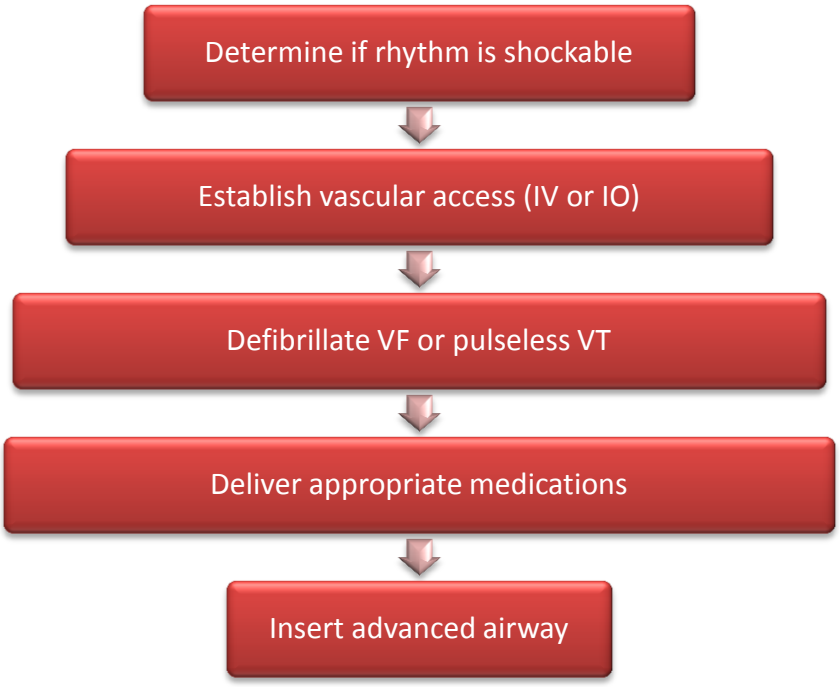


FIGURE 20: ALS INTERVENTIONS IN CARDIAC ARREST

- All medications can be safely administered via the Intravenous (IV) or Intraosseous (IO) routes. The LEAN drugs (lidocaine, epinephrine, atropine, and naloxone) and vasopressin also can be administered via an endotracheal (ET) tube, sometimes abbreviated as ETT. This route is less desirable than IV or IO since drug absorption is less predictable and an ET tube may not be in place. The IV route is best when available, but the IO route can be used when an IV is not established (refer to Unit Seven for specifics on the IO route).
- The ET tube is the preferred advanced airway during CPR but bag/mask ventilations can be as effective as an ET tube for short-term use.

PEDIATRIC CARDIAC ARREST

The purpose of the PALS Cardiac Arrest Algorithm (see Figure 21) is to provide high-quality CPR and electrical and drug intervention as appropriate. In cardiac arrest, the most critical components of the algorithm are the 2-minute periods of CPR. In a team setting, the rest of the team should be preparing drugs or the defibrillator during these periods of CPR. Successful cardiac arrest efforts will be influenced by:

- The period of time between collapse and CPR: Better outcomes will be realized if there is a shorter interval between collapse and CPR.
- The provision of high-quality CPR: Hard and fast is the most effective.
- The duration of CPR efforts: In general, the longer CPR continues, the worse the outcome.
- Underlying causes: Early intervention for reversible causes of arrest can improve outcomes.

If a child is responding to treatment, is hypothermic, or has drug poisoning, resuscitation efforts should continue. For a child without a pulse, the resuscitation team must follow the PALS Cardiac Arrest Algorithm:

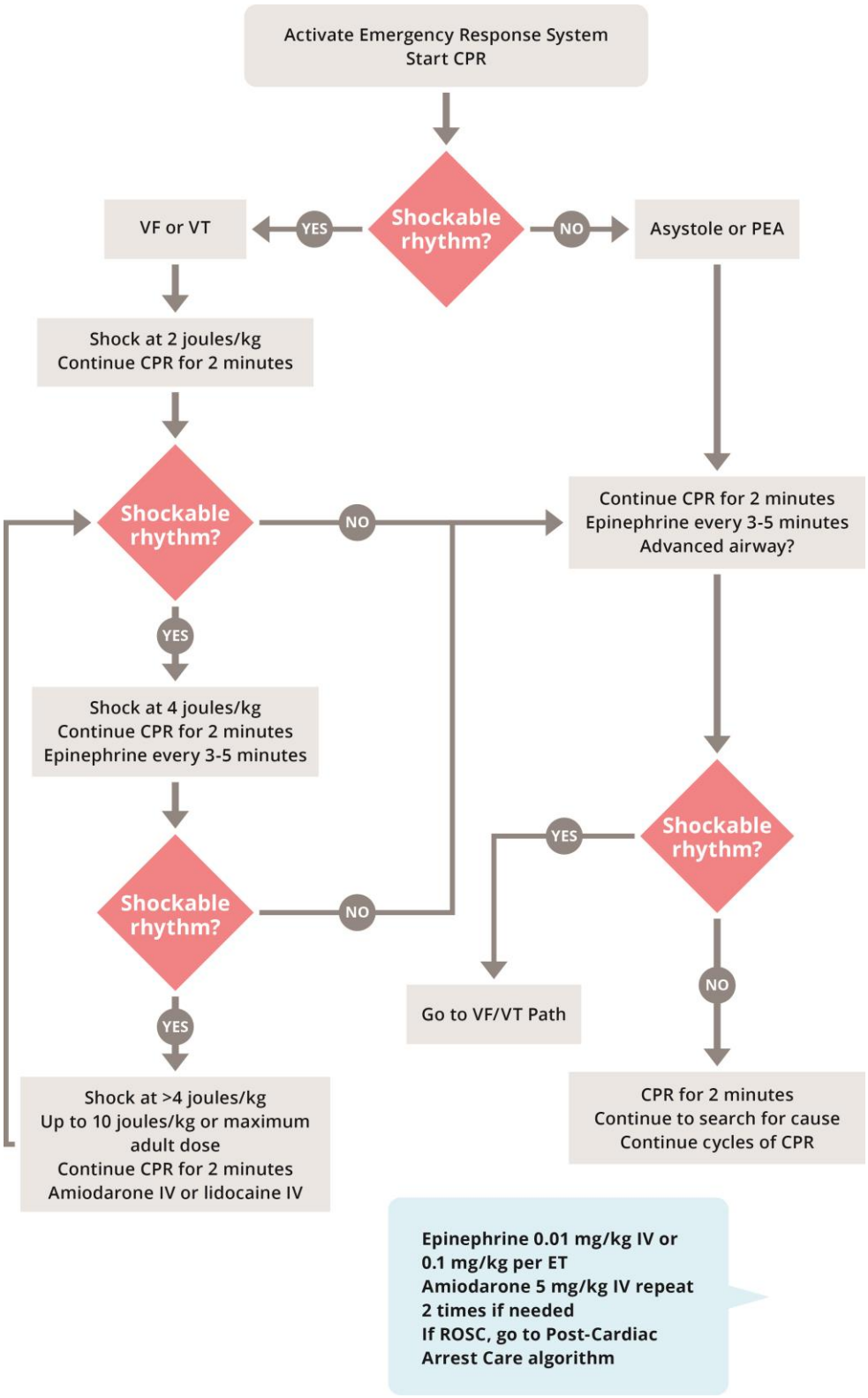


FIGURE 21: PALS CARDIAC ARREST ALGORITHM

MANUAL DEFIBRILLATION FOR VF OR PULSELESS VT

When a shockable rhythm (VF or pulseless VT) is identified, the team should prepare to cardiovert while continuing high-quality CPR:

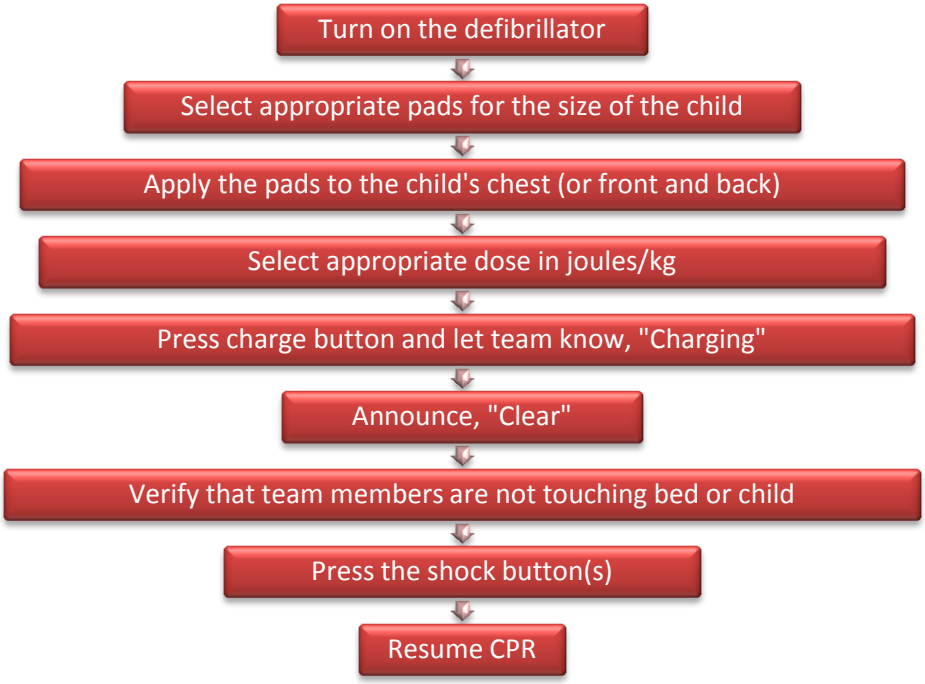


FIGURE 22: MANUAL DEFIBRILLATION IN PEDIATRIC CARDIAC ARREST

Minimize the interruption of CPR. Insert advanced airways and IVs during pulse checks. Give medications during CPR so that they enter circulation with compressions.

SPECIAL CIRCUMSTANCES

If a child is in cardiac arrest because of trauma, drowning, anaphylaxis, poisoning, congenital heart disease or pulmonary hypertension the team should be aware that additional interventions may be needed:

- **Trauma**: If a neck injury may have occurred, keep the neck in line with the spine. Stop any external bleeding and check for a pneumothorax. Be prepared to transfer the child to a specialized pediatric trauma center.
- **Drowning**: If a neck injury may have occurred, keep the neck in line with the spine. Treat hypothermia by slowly rewarming the child.
- **Anaphylaxis**: Fluids and epinephrine should be first line treatments. Antihistamines and steroids should be considered if the child is responding.
- **Poisoning**: If the poison is known and an antidote is available, administer it. Give the antidote time to work before stopping resuscitation efforts.
- **Congenital heart disease**: Consult the child's pediatrician and consider administering heparin if shunt occlusion is a possibility.
- **Pulmonary hypertension**: Increase ventilations to decrease carbon dioxide. Consider isotonic saline. Consider the use of extracorporeal membrane oxygenation (ECMO) if available.

For optimal outcomes in the pediatric patient, the team must concentrate on successful resuscitation AND advanced post-resuscitation support. After successful resuscitation, the team must continue to manage the airway, ventilation, and circulation including performing diagnostic testing. The goals of post-resuscitation support include:

- Treatment of the underlying causes of the initial injury or illness
- Prevention of further injury
- Optimization of body functions
- Transport to the next level of care.

Secondary assessments should continue during this post-resuscitation period and management priorities and actions should be:

System	Priority	Treatments
Respiratory	Maintain adequate oxygen levels	Titrate oxygen to maintain O_2 saturation 94% - 99%
	Maintain adequate ventilation	Keep airway patent; use intubation and mechanical ventilation if necessary
	Monitoring of all vital signs	Monitor oxygen saturation and heart rate; position of ET tube (as applicable); respiratory rate, rhythm and breath sounds; if the intubated child suddenly deteriorates, use the DOPE mnemonic (see below) to identify the possible cause
	Monitor tests	ABGs
	Control pain and anxiety	Titrate fentanyl or morphine to control pain and anxiety monitor hemodynamics
Cardiovascular	Monitoring of all vital signs	Heart rate, blood pressure, oxygen saturation, urine output, CVP and cardiac output (if available)
	Monitor labs	Blood gases, hemoglobin/hematocrit, blood glucose, electrolytes, BUN, calcium, creatinine, CXR, EKG
	Maintain fluid volume	Use the Shock Management Algorithm and administer maintenance fluids as appropriate
	Maintain blood pressure	Maintain blood pressure above fifth percentile by age using fluids and vasopressors as needed
	Maintain oxygenation	Titrate oxygen to keep O_2 saturation 94% - 99% and consider intubation and ventilatory support based on the child's respiratory status
	Control pain	Titrate fentanyl or morphine to control pain and anxiety; control fever
	Control arrhythmias	Monitor cardiac rhythm and use drugs or electrical therapy (see Bradycardia or Tachycardia Algorithm)
Neurologic	Monitoring of all vital signs	Avoid fever; do not re-warm a hypothermic patient unless the hypothermia is suspected to be causing the child's instability; consider therapeutic hypothermia if child remains comatose after successful resuscitation; heart rate, blood pressure, neurologic exams, pupils
	Monitor tests	Blood glucose, electrolytes, calcium, lumbar puncture only if child is stable and CNS infection is suspected

System	Priority	Treatments
Neurologic (cont'd)	Control intracranial pressure	Support oxygenation, ventilation and cardiac output; elevate head of bed unless blood pressure is low; consider IV mannitol for acute brain stem herniation
	Watch for seizures	Treat seizures immediately; search for treatable metabolic cause or toxins
Renal	Monitor urine output	Urine output should be >1 mL/kg/hour for infants and children and >30 mL each hour for adolescents
	Monitor urinary catheter	Insert urinary catheter to monitor output; ensure that catheter is draining
	Monitor lab tests	Urine glucose, lactate level, BUN, creatinine, electrolytes, urinalysis
	Monitor kidney function	Provide adequate fluid volume; avoid medications that effect kidneys when possible
	Monitor acid-base balance	Correct acidosis; sodium bicarbonate is not recommended
Gastrointestinal	Monitor bleeding/excess NG tube drainage	Maintain NG to low suction
	Monitor lab tests	Liver function tests, amylase, lipase
Hematologic	Monitor lab tests	Hemoglobin/Hematocrit/Platelets, PT, PTT, INR, fibrinogen and fibrin split products
	Monitor blood therapy	If hemorrhagic shock does not correct with crystalloid therapy, transfuse with packed red blood cells; transfuse with platelets if active bleeding and a platelet count <50-100,000; transfuse fresh frozen plasma if active bleeding and abnormal coagulation

TABLE 20: POST-RESUSCITATION PRIORITIES AND TREATMENTS

DOPE

When the intubated child suddenly deteriorates, use the DOPE mnemonic to identify the possible cause:

Displacement of ET tube: Assess respirations by checking chest expansion, listening to breath sounds and obtaining a chest x-ray to verify tube placement

Obstruction of ET tube: Suction the ET tube to remove secretions, foreign bodies, or blood

Pneumothorax: Listen to breath sounds and obtain chest x-ray

Equipment failure: Check all machinery to ensure all equipment is operating correctly

MAINTENANCE FLUIDS

Maintain circulating volume with an isotonic crystalloid such as saline or lactated Ringer's solution. Add dextrose or potassium chloride based on the child's condition and lab values. Do not give hypotonic fluids. Give maintenance fluids based on the child's weight:

Child's Weight	Estimated Hourly Maintenance Fluids
<10 kg	4 mL/kg/hour
10-20 kg	40mL/hour + 2 mL/kg/hour for each kg between 10 and 20
>20 kg	60mL/hour + 1 mL/kg/hour for each kg above 20

TABLE 21: CALCULATION OF MAINTENANCE FLUID

After cardiac arrest, manage shock using the following algorithm:

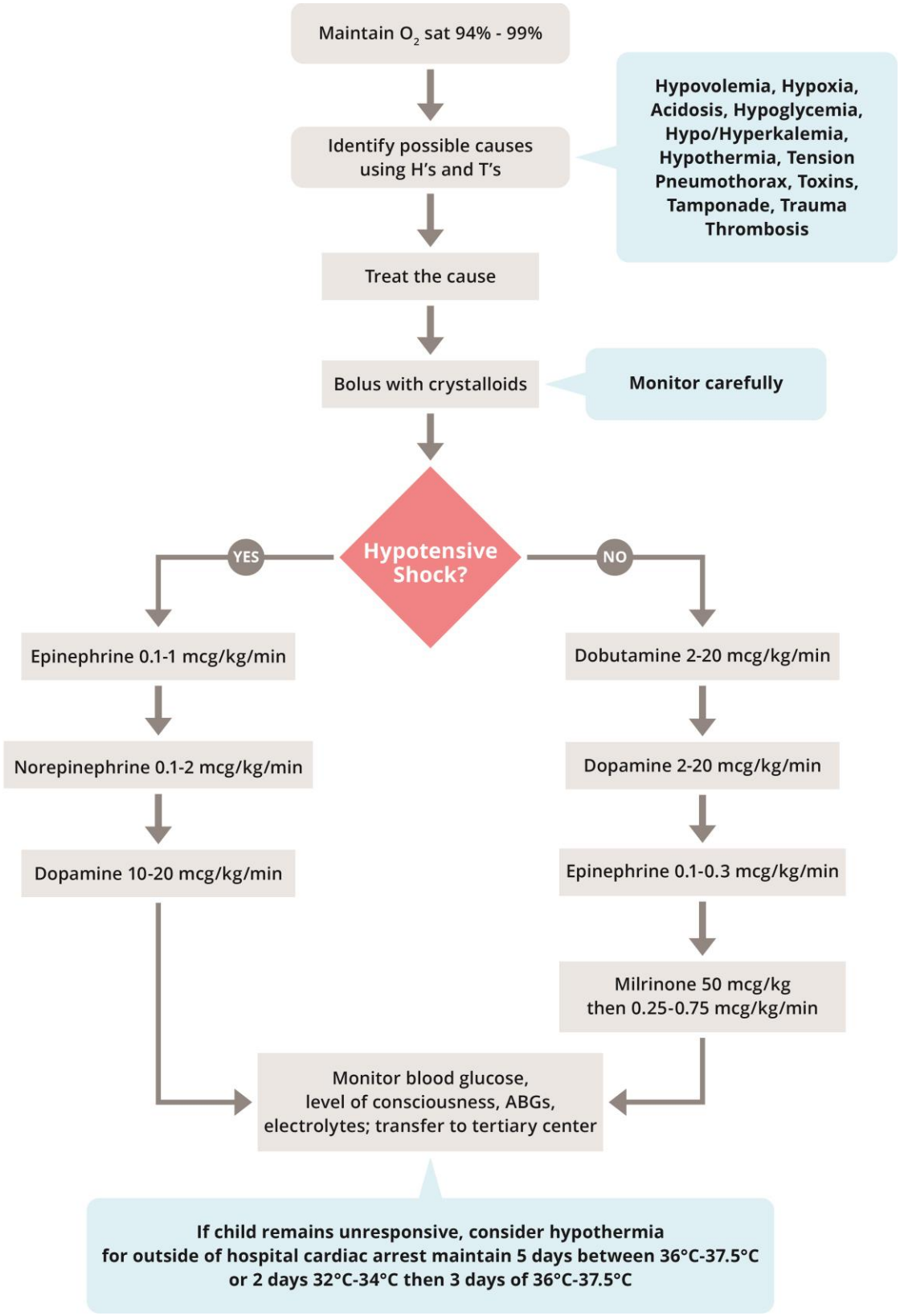


FIGURE 23: PALS POST ARREST SHOCK MANAGEMENT ALGORITHM

Use the following guide to arrange the safe transport of the pediatric patient to definitive care.

- Prepare for transport:
  - Find the nearest tertiary pediatric facility
  - Know and follow the protocol for inter-hospital transport
  - Anticipate medications, fluids or blood products that might be needed during transport
  - Prepare them to go with the team.
- Coordinate with the receiving facility:
  - Identify the specific receiving provider in the facility
  - The resuscitation team leader should communicate directly with the receiving provider
  - Communicate any changes in the child's condition during transport.
- Use all appropriate precautions:
  - Universal precautions
  - Obtain cultures if infection suspected
  - Do not delay antibiotics if infection suspected
  - Use appropriate isolation techniques if needed.
- Prepare the child and family:
  - Communicate all implemented treatments to the family
  - Communicate information about where the child will go and additional capabilities at that facility
  - Obtain consent for transport
  - Answer questions and provide comfort to the child and family.
- Prepare the documentation:
  - Send a copy of chart with the child
  - Send copies of all lab tests and x-rays with the child
  - Send laboratory contact information for all pending tests with the child.
- Determine the most appropriate mode of transportation:
  - Ground ambulance: Inexpensive and available in most weather conditions but may involve increased time for distant transports
  - Helicopter: Faster than ground ambulance for long-distance transports, but more expensive than ground ambulance and may not be able to fly in all weather
  - Fixed wing aircraft: The best mode of transport for long distances or for very unstable child, but expensive and typically requires interim ground ambulance to reach the aircraft.
- Select the transport team:
  - Trained in pediatric care and the pediatric transport equipment
  - Trained in the selected mode of transportation.

Medications change quickly. It is highly recommended that a pharmacist be included on the resuscitation team to manage all drugs and dosages for pediatric patients.

Drug	Classification	Indications	Dose/Administration	Possible Side effects	Considerations
Adenosine	Antiarrhythmic	SVT	1st dose = 0.1 mg/kg rapid IV push to max of 6 mg. 2nd dose = 0.2 mg/kg rapid IV push to max of 12 mg	Dizziness, headache, metallic taste, shortness of breath, hypotension, slow or fast heart rate, nausea, flushing, sweating	Cardiac monitoring during administration; administer through central line if available; flush with saline following administration
Amiodarone	Antiarrhythmic	SVT, VT with pulses, VF, VT without pulse	SVT or VT with pulse 5mg/kg load over 20-60 minutes to 300 mg max VF or VT without pulse: 5mg/kg rapid bolus to 300 mg max	Headache, dizziness, tremors, syncope, hypotension, bradycardia, CHF, nausea, vomiting, diarrhea, rash, skin discoloration, hair loss, flushing, coagulation problems	Monitor ECG and BP; use with caution in patients with a perfusing rhythm, hepatic failure; contraindicated for 2nd or 3rd degree heart block
Atropine	Anticholinergic	Symptomatic bradycardia, toxins and overdoses	Bradycardia: 0.02 mg/kg IV with 0.5 mg max dose may repeat one time By ET tube: 0.04-0.06 mg/kg Toxins/overdose: 0.02-0.05 mg/kg repeated every 20-30 minutes until symptoms reverse	Headache, dizziness, confusion, anxiety, flushing, visual difficulties, pupil dilation, dry mouth, tachycardia, high or low blood pressure, nausea, vomiting, constipation, urinary retention, painful urination, rash, dry skin	Monitor ECG, oxygen, and BP; administer before intubation if bradycardic; contraindicated in glaucoma and tachyarrhythmias
Epinephrine	Catecholamine vasopressor, Inotrope	Anaphylaxis, asthma, symptomatic bradycardia, croup, shock, cardiac arrest, toxins or overdose	Anaphylaxis: 0.01 mg/kg every 15 minutes to max of 0.3 mg Asthma: (1:1000) 0.01 mg/kg subcutaneous every 15 minutes to max 0.3 mg Symptomatic bradycardia: 0.01 mg/kg IV every 3-5 minutes to max dose of 1 mg Croup: 0.25 ml Racemic epi solution via nebulizer Cardiac arrest: 0.01 mg/kg (1:10000) IV or 0.1 mg/kg (1:1000) per ET tube every 3-5 minutes Shock: 0.1-1 mcg/kg/minute IV infusion Toxins/ODs: 0.01 mg/kg (1:10000) IV to max dose of 1 mg	Tremors, anxiety, headaches, dizziness, confusion, hallucinations, dyspnea, SVT, VT, palpitations, hypertension, nausea, vomiting, hyperglycemia, hypokalemia, vasoconstriction	Available in 1:1000 and 1:10000 concentrations so the team must be aware of which concentration is being used; monitor BP, oxygen, and ECG; give via central line if possible; do not give in cocaine induced ventricular tachycardia

Drug	Classification	Indications	Dose/Administration	Possible Side effects	Considerations
Oxygen	Elemental gas	Hypoxia, respiratory distress or failure, shock, trauma, cardiac arrest	In resuscitation, administer at 100% via high flow system and titrate to response to maintain oxygen saturation >94%	Headache, dry nose/ mouth, airway obstruction if secretions become dry	Monitor oxygen saturation; insufficient flow rates may cause carbon dioxide retention
Albumin	Plasma volume expander	Shock, trauma, burns	0.5-1 g/kg by rapid infusion	Fluid overload, increased respiratory rate, flushing, rash, hypocalcemia	Use within 4 hours of opening vial
Albuterol	Bronchodilator	Asthma, bronchospasm, hyperkalemia	2.5 mg if weight <20 kg 5 mg if weight >20 kg	Tremors, anxiety, headaches, bad taste, dry nose/throat, dyspnea, wheezing, tachycardia, hypotension, nausea, vomiting, flushing	Should not be used with tachyarrhythmias
Alprostadil	Prostaglandin vasodilator	Maintain patency of ductus arteriosus in congenital heart disease	Initial: 0.05-0.1 mcg/kg/ minute Maintenance: 0.01-0.05 mcg/kg/ minute	Seizures, apnea, vasodilation, hypotension, bradycardia, cardiac arrest, diarrhea, renal failure, flushing, hypoglycemia, DIC, hypocalcemia, fever	May cause tissue sloughing should be refrigerated until administered
Calcium chloride	Electrolyte	Hypocalcemia, hyperkalemia; consider for calcium channel blocker overdose	In cardiac arrest: 20 mg/kg IV bolus into central line In non-arrest: infuse over 30-60 minutes	Hypotension, cardiac arrhythmias, cardiac arrest, burn or sclerosis of peripheral veins, hypercalcemia	Monitor ECG and BP; contraindicated in digitoxicity or hypercalcemia; flush IV tubing before and after administration
Dexamethasone	Corticosteroid	Croup asthma	0.6 mg/kg for one dose (max dose 16 mg)	Headache, insomnia, seizures, psychosis, visual difficulties, hypertension, edema, tachycardia, osteoporosis, diarrhea, nausea ,GI bleeding, flushing, sweating, poor wound healing, hyperglycemia, sodium and fluid retention, hemorrhage, hypokalemia	Can be given PO, IM or IV
Dextrose	Carbohydrate	Hypoglycemia	0.5-1 g/kg	Sclerosis of veins, hyperglycemia	Do not administer during resuscitation unless hypoglycemia is documented; use point of care glucose monitoring

Drug	Classification	Indications	Dose/Administration	Possible Side effects	Considerations
Diphenhydramine	Antihistamine	Anaphylaxis after epinephrine	1 -2 mg/kg every 4 to 6 hours to a max dose of 50 mg	Dizziness, drowsiness, CNS symptoms, blurred vision, pupil dilation, dry nose/mouth/throat, hypotension, tachycardia, nausea, vomiting, urinary retention or frequency photosensitivity	Monitor oxygen saturations and BP; use with caution in presence of glaucoma, ulcer, hyperthyroidism
Dobutamine	Beta adrenergic	Ventricular dysfunction	2-20 mcg/kg/ minute infusion	Headache, dizziness, hypotension, palpitations, angina, nausea, vomiting thrombocytopenia	Monitor ECG and BP; do not mix with sodium bicarbonate or alkaline solutions
Dopamine	Catecholamine vasopressor, inotrope	Ventricular dysfunction, cardiogenic or distributive shock	2-20 mcg/kg per minute infusion titrated to response	Headache, dyspnea, palpitations, PVCs, SVT, VT, nausea, vomiting, acute renal failure	Monitor ECG and BP; avoid high infusion rates; do not mix in alkaline solutions or with sodium bicarbonate
Etomidate	Short acting sedative with no analgesic properties	Sedation for intubation or for patients with hypotension or multiple trauma	0.2-0.4 mg/kg IV over 30 to 60 seconds with max dose of 20 mg	Fast or slow respiratory rate, high or low blood pressure, tachycardia nausea, vomiting, cough	Sedation will last 10-15 minutes; monitor oxygen, BP and respiratory function; avoid use in septic shock
Furosemide	Loop diuretic	Pulmonary edema, fluid overload	1 mg/kg IV or IM to max dose of 20 mg	Headache, weakness, vertigo, hearing and vision problems, dry mouth, ECG changes, nausea, vomiting, diarrhea, abdominal cramping, polyuria, glycosuria, muscle cramps, sweating, hives, hyperglycemia, anemia, hypokalemia, hyponatremia, metabolic alkalosis	Monitor BP, BUN, serum creatinine and electrolytes (especially potassium)
Hydrocortisone	Corticosteroid	Adrenal insufficiency associated with septic shock	2 mg/kg IV bolus to max dose of 100 mg	Psychological signs, infections, blurred vision, hypertension, diarrhea, nausea, vomiting, osteoporosis, flushing, sweating, slow wound healing, hyperglycemia	Watch for signs of infection
Inamrinone	Inodilator	Myocardial dysfunction, cardiogenic shock, CHF	Loading dose 0.75-1 mg/kg bolus over 5-10 minutes may repeat twice to max dose of 3mg/kg Infusion at 5-10 mcg/kg/minute	Hypoxemia, hypotension, angina, arrhythmias nausea, vomiting, abdominal pain, jaundice, allergic reactions, thrombocytopenia	Monitor ECG, oxygen, and BP

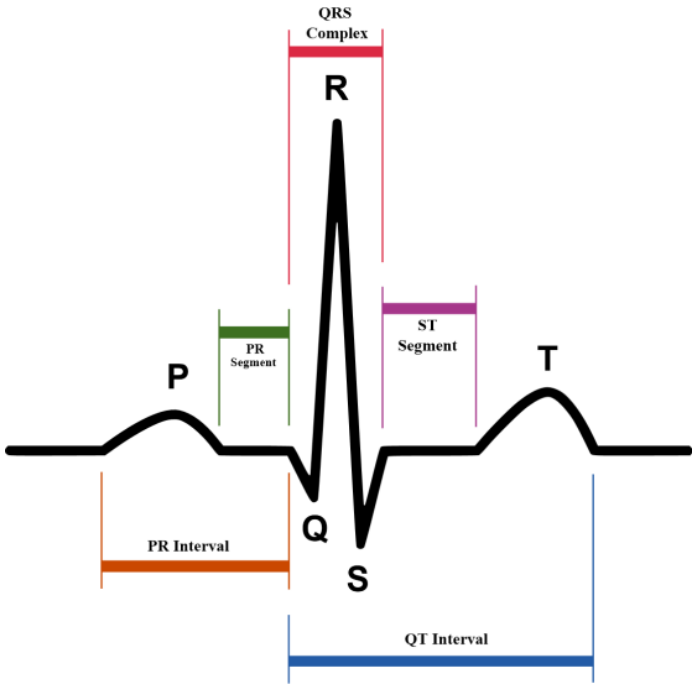
Drug	Classification	Indications	Dose/Administration	Possible Side effects	Considerations
Ipratropium	Anticholinergic bronchodilator	Asthma	250-500 mcg every 20 minutes via nebulizer for 3 doses	Anxiety, dizziness, headache, dry mouth, blurred vision, cough, bronchospasm, palpitations, nausea, vomiting, rash	Monitor oxygen; if medication gets in eyes, will cause pupil dilation
Lidocaine	Antiarrhythmic	VF, pulseless VT, wide complex tachycardia, RSI	Tachyarrhythmias and VF: 1 mg/kg IV bolus followed by infusion of 20-50 mcg/kg/ minute infusion RSI: 1-2 mg/kg IV	CNS symptoms, tinnitus, blurred vision, hypotension, heart block, bradycardia, cardiac arrest, dyspnea, respiratory depression, nausea, vomiting, rash	Monitor ECG and BP; May cause seizures; contraindicated for wide complex bradycardia
Magnesium sulfate	Electrolyte bronchodilator	Asthma; torsades de pointes; hypo-magnesemia	Asthma: 25-50 mg/kg over 15-30 minutes IV Pulseless torsades: 25-50 mg/kg bolus VT with pulses and torsades: 25-50 mg/kg over 10-20 minutes	Confusion, sedation, weakness, respiratory depression, hypotension, heart block, bradycardia, cardiac arrest, nausea, vomiting, muscle cramps, flushing, sweating	Monitor ECG, oxygen and BP; rapid bolus may cause hypotension and bradycardia; calcium chloride can be used if needed to reverse hypermagnesemia
Methylprednisolone	Corticosteroid	Asthma, anaphylactic shock	2 mg/kg to max of 60 mg IV as load dose; 0.5 mg/kg every 6 hours as maintenance dose	Depression, headache, weakness, hypertension, diarrhea, nausea, pancreatitis, ulcer, osteoporosis, hyperglycemia	Watch for rare anaphylaxis
Milrinone	Inodilator	Cardiogenic shock or post-surgery CHF	50 mcg/kg IV over 10-60 minutes as loading dose 0.25-0.75 mcg/kg/ minute IV infusion as maintenance dose	Headache, tremor, hypotension, ventricular arrhythmias, angina, nausea, vomiting, jaundice, hypokalemia	Monitor ECG, BP and platelet count; hypovolemia may make hypotension worse; use longer infusion time
Naloxone	Opioid antagonist	Narcotic reversal	For total reversal: 0.1 mg/kg IV bolus every 2 minutes to max dose of 2 mg Total reversal not needed: 1-5 mcg/kg IV (titrate to response required)	Seizures, drowsiness, rapid respiratory rate, pulmonary edema, VF, VT, tachycardia, asystole, hypertension nausea, vomiting	Monitor ECG, oxygen and BP; repeat doses often needed; establish assisted ventilation before administration; monitor newborn of addicted mother
Nitroglycerine	Vasodilator antihypertensive	CHF, cardiogenic shock	Begin infusion at 0.25-0.5 mcg/kg/ minute and titrate every 15-20 minutes to max dose of 10 mcg/kg/minute	Headache, dizziness, hypoxemia, hypotension, cardiac arrest, tachycardia, flushing, pallor	Monitor ECG and BP; watch for hypotension in hypovolemic children
Nitroprusside	Vasodilator antihypertensive	Cardiogenic shock Hypertension	0.3-1 mcg/kg/minute for initial dose then titrate to max 8 mcg/kg/minute	Seizures, dizziness, headache, agitation, hypotension, slow or fast heart rate, nausea, vomiting	Monitor ECG and BP If used for prolonged times; thiocyanate and cyanide levels should be monitored.

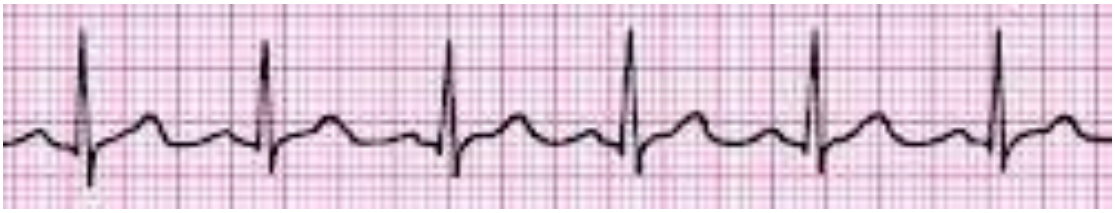
Drug	Classification	Indications	Dose/Administration	Possible Side effects	Considerations
Norepinephrine	Inotrope vasopressor	Hypotensive shock	0.1-2 mcg/kg/m titrated to desired BP	Headache respiratory distress hypertension arrhythmias renal failure	Monitor ECG and BP; IV infiltration may lead to tissue necrosis; should be administered via central line; do not mix in alkaline solution
Procainamide	Antiarrhythmic	SVT, atrial flutter, VT with pulse	15 mg/kg as loading dose over 30-60 minutes	Headache, dizziness, confusion, weakness, hypotension, prolonged QT interval, heart blocks and cardiac arrest, nausea, vomiting, diarrhea, rash, edema, anemia, neutropenia	Monitor ECG (particularly QT interval) and BP; expert consultation should be called before administration
Sodium bicarbonate	Electrolyte to produce alkalinity	Severe metabolic acidosis, hyperkalemia, tricyclic overdose	1 mEq/kg slow IV bolus to max of 50 mEq For overdose 1-2 mEq/kg bolus repeating until pH >7.45 follow with infusion of sodium bicarb solution to maintain alkalosis	CNS symptoms, arrhythmia, hypotension, cardiac arrest, renal calculi, cyanosis, edema, metabolic alkalosis and other derangements, water retention	Monitor ECG, oxygen and ABGs; ensure adequate ventilatory support to reduce the chance of carbon dioxide accumulation; not recommended in cardiac arrest
Terbutaline	Bronchodilator, beta adrenergic agonist	Asthma, hyperkalemia	0.1-10 mcg/kg/ minute IV Infusion 10 mcg/kg SQ every 10-15 minutes until IV is established	CNS symptoms, palpitations, tachycardia, nausea, vomiting, arrhythmias, hypotension	Monitor ECG, oxygen and BP; use cautiously in children with hypokalemia
Vasopressin	Antidiuretic hormone analogue	Cardiac arrest, septic shock	0.4-1 unit/kg bolus to max of 40 units	Fever, vertigo, dysrhythmias, hypertension, nausea, vomiting, abdominal cramps, urticaria	Monitor BP and distal pulses; watch for signs of water intoxication; tissue necrosis may develop from IV extravasation

TABLE 22: RESUSCITATION MEDICATIONS

SINUS RHYTHM

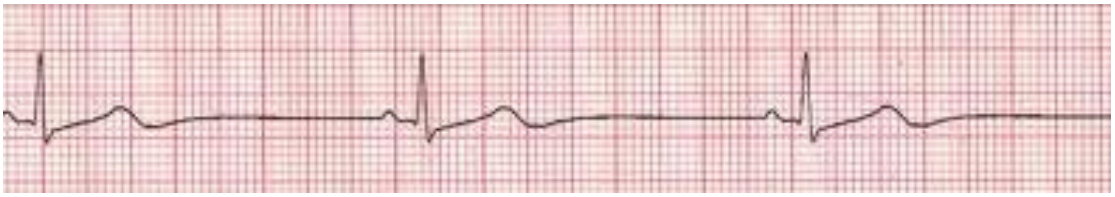
A sinus rhythm is regular with normal P, Q-R-S, T deflections and intervals. Rate = 60-100 at rest.





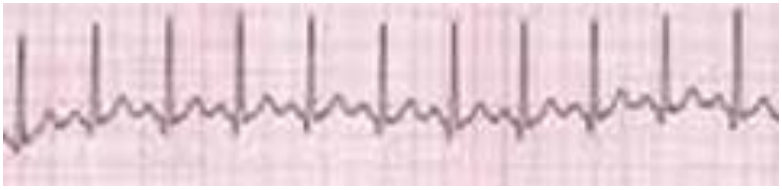
SINUS BRADYCARDIA

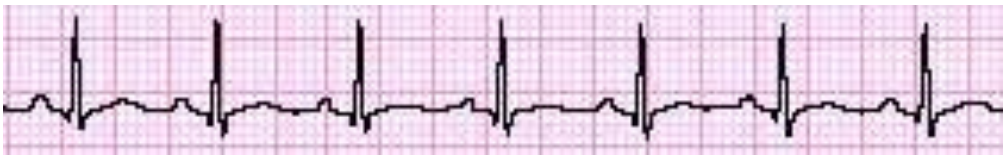
Sinus bradycardia is a sinus rhythm with a rate lower than normal for a child's age.



SINUS TACHYCARDIA

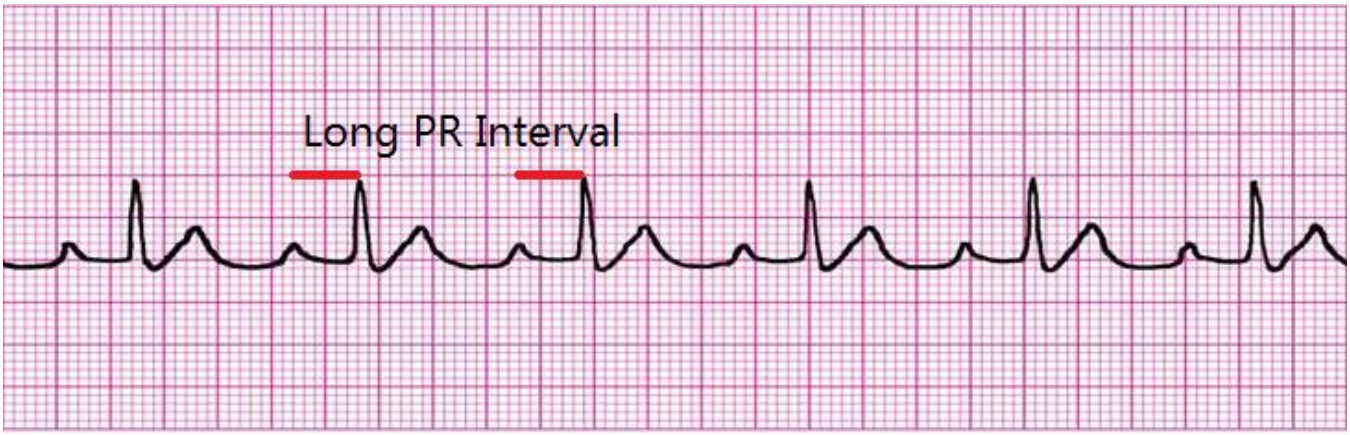
Sinus tachycardia is a sinus rhythm with a rate greater than normal for a child's age. Note that the p waves are still present.





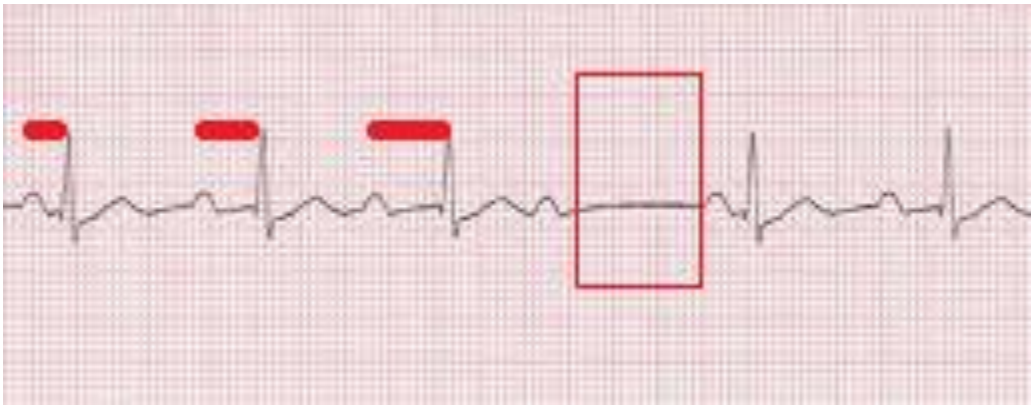
SINUS RHYTHM WITH 1ST DEGREE HEART BLOCK

Sinus rhythm with 1st degree heart block is a sinus rhythm with a prolonged PR interval >0.20 seconds due to a delay in transmission from the atria to the ventricles.

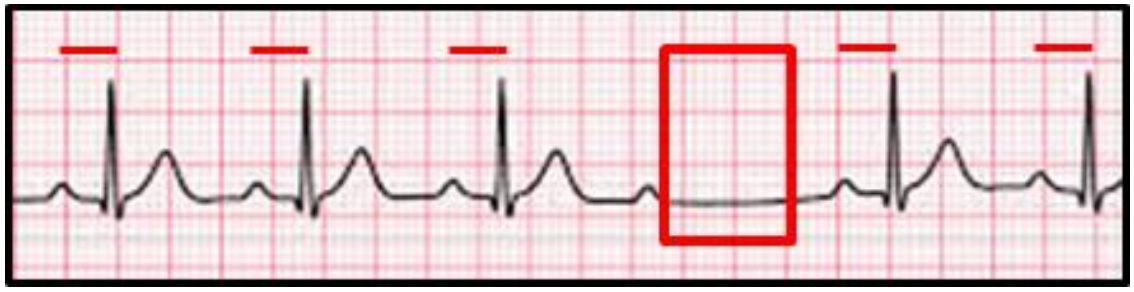


2ND DEGREE AV HEART BLOCK

A 2nd degree AV block is usually classified as Mobitz Type I (Wenckebach) or Mobitz Type II. A Mobitz Type I heart block is characterized by progressive lengthening of the PR interval until a QRS complex is dropped.

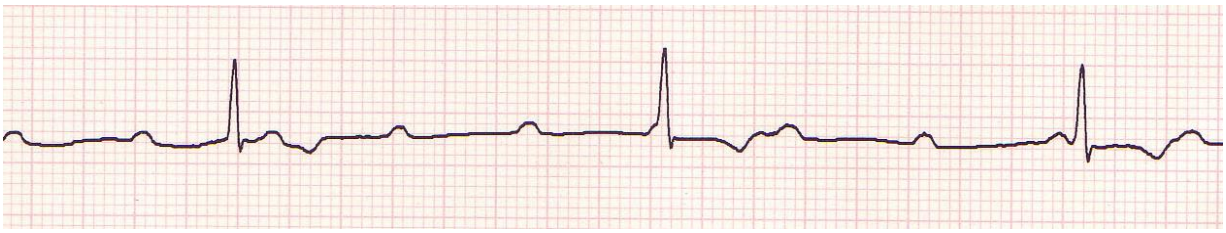


A Mobitz Type II heart block is characterized by an intermittent dropped QRS that is not in a Mobitz Type I pattern. The Mobitz Type II block must be evaluated since it can rapidly progress to a complete heart block.



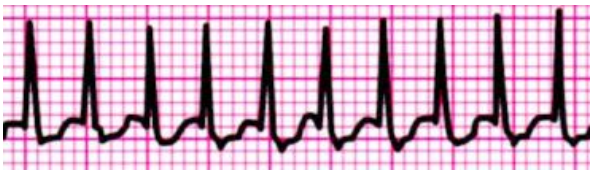
3RD DEGREE HEART BLOCK

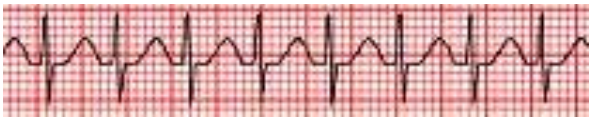
A 3rd degree heart block (sometimes called a complete heart block) is a rhythm in which there is no relationship between the P and QRS waves. In this case, the P to P intervals are regular but have no relationship to the QRS complexes on the ECG.



SUPRAVENTRICULAR TACHYCARDIA (SVT)

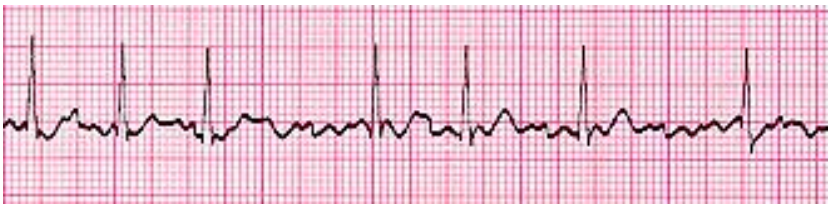
Supraventricular tachycardia (SVT) is an extremely fast atrial rhythm with narrow QRS complexes when the impulse originates above the bundle branches (above the ventricles).





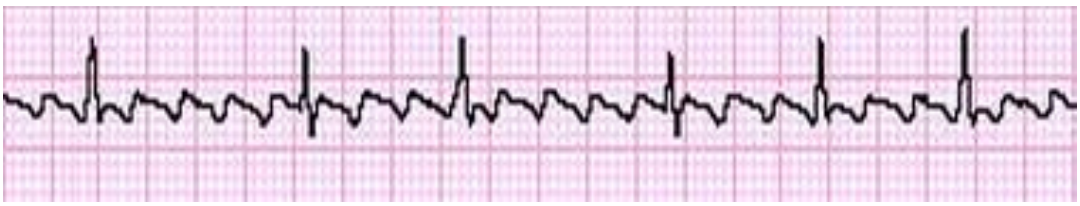
ATRIAL FIBRILLATION (AF)

Atrial fibrillation (AF) is a very common arrhythmia. This rhythm is characterized by no waves before the QRS complex and a very irregular heart rate.



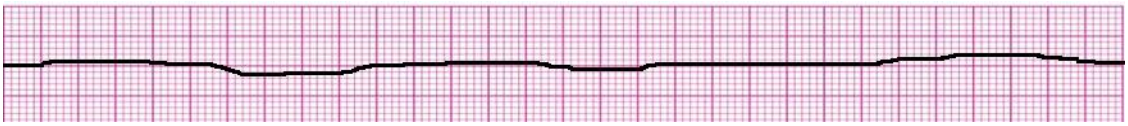
ATRIAL FLUTTER

Atrial flutter is a supraventricular arrhythmia that is characterized by a “saw-toothed” flutter appearance on the ECG that represent multiple P waves for each QRS complex.



ASYSTOLE

Asystole is also commonly known as a "flat line" where there is no electrical activity seen on the cardiac monitor. Not responsive to electrical defibrillation.

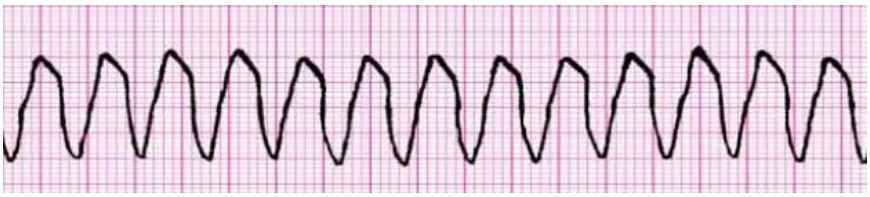


PULSELESS ELECTRICAL ACTIVITY

Can be virtually any organized ECG rhythm in a patient who is unresponsive and lacks a palpable pulse. Thus, one cannot learn a PEA rhythm. It should not be confused, however, with specific pulseless scenarios listed previously.

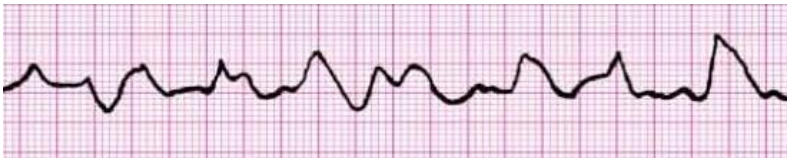
VENTRICULAR TACHYCARDIA (VT)

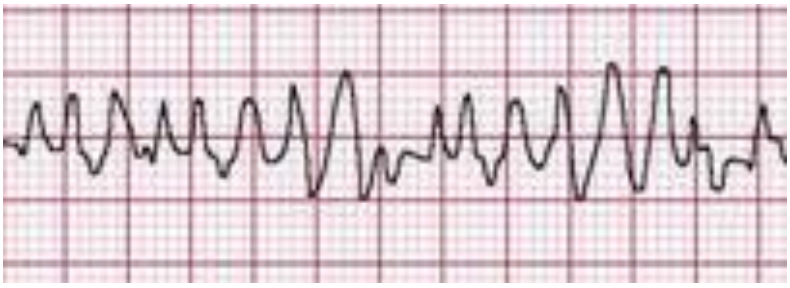
Ventricular tachycardia (VT) is characterized by bizarre widened QRS complexes, no P waves and a heart rate that usually exceeds 100 beats per minute. VT may quickly degenerate to Ventricular fibrillation and death. VT may be responsive to electrical defibrillation.



VENTRICULAR FIBRILLATION (VF)

Ventricular fibrillation (VF) is characterized by a chaotic wave pattern and no pulse. VF may be responsive to electrical defibrillation.





1. SHOCK

Learning objectives
After completionof this Section,the participants should be able to
• Identifyshock based on clinical parameters
• Differentiate between compensated rind decompensatedshock
• Classify based on peripheral perfusion and etiology.
• Initiate fluids andvasoactive therapy as per underlying etiology
• Monitor therapeutic end points
• Look for causes of non-response in refractory shock
• Know when and how to refer?

1.2 What is shock?
• Shock is a clinical syndrome arising out of mismatch between oxygen supply anddemand of the tissues

1.3 How to classifyshock?
- Shock is classified based on both severity (Box l.1)and etiology(Box 1.2)

Box 1.1: Classification of shock based on severity

Compensated	Decompensated / Hypotensive
Tachycardia Impaired perfusion (cold or warm) Decreased urine output Normal blood pressure (BP>5th centile for age) 5thcentile systolic BP is given as Neonates = 65 mmHg Infants (<1 year) = 70 mmHg 1-10 years =70 + (age x 2) mmHg • >10 years =90 mmHg	Tachycardia impaired perfusion (Cold and warm) • Decreased urine output Hypotension (BP< 5thcentile for age) 5th centile systolic BP is given as • Neonates = 65 mmHg • Infants (<I year) = 70 mmHg • 1·10 years = 70 + (age x 2) mmHg • >10 years = 90 mmHg.

Box 1.2: Classification of shock based on etiology

Type	Pathophysiology	Disease states
Hypovolemic	Decrease in effective Circulating blood volume (Preload)	Actual intravascular volume loss (diarrhea dehydration, haemorrhage) apparent intravascular volume loss (capillary leak, third space loss; redistribution
Cardiogenic	Myocardial pump failure	Impaired cardiac contractility (myocarditis, cardiamvopathy)
Distributive	Loss of vascular tone and vasdilation	Sepsis (increased capillary leak with loss of intravascular volume)

		Neurogenic shock (acute spinal injury, CNS catastrophe)
Obstructive	Obstruction to cardiac output	RV outflow obstruction (pulmonaryembolism, lung hyperinflation, pericardial effusion) LV outflow obstruction due to critical aortic stenosis

1.4 How to recognize shock?

The recognition of shock is always clinical. The ABCDE approach during primary assessment helps identify shock (Refer Box 1.3)

Box 1.3: Clinical recognition of shock

Airway	Patency	Airway open and maintainable not maintainable	
Breathing	Respiratory rate	Increased	
	Respiratory effort	Increased/labored	
	Breath sounds	Normal or crepts	
Circulation		Warm Shock	Cold Shock
	Skin	Warm suffused skin	Pale, cool skin
	Heart rate	Tachycardia	Tachycardia
	Peripheral pulse	Bounding pulses Flashed CFT	Weak pulse delayed CFT (>2 sec) Maintained
	Systolic BP	Maintained in compensated, decreased in hypotensive shock	In compensated, Decreased in hypotensive Shock
	Pulse pressure	Increased	Decreased
	Urine output	Decreased	Decreased
Disability	Consciousness	Imitable early/Lethargic late	
Exposure	Temperature	Variable	
	Blood glucose Rash Jaundice		

*Adopted from IAP-ALS handbook

1.5 What are the steps in management of shock?

• Airway: Suction secretions
if patient is obtunded and unable to maintain airway,startbag and mask ventilation,followed by intubation

• **Breathing**: Start oxygen(any delivery device that is available),preferably 100% Fi02.
o Inspontaneously breathing patient start with non- rebreathing mask at flow rateof 10-15 L/min
o In apneic or bradypneic patient start bag and mask ventilation followed by intubation.
• **Circulation:**
o Secure immediate vascular access: Select a large vein close to the heart
o Avoid delay in getting a vascular access;establishintraosseous access if there isinability to secure a peripheral venous accessin 3 attempts or 90 seconds (whichever is earlier)(see appendix)

A) Fluids:
• Fluids are given to augment the preload and optimization of preload is the first consideration in therapy of shock, irrespective of etiology.
• Fluids of choice are always crystalloid either Normal Saline/Ringer's Lactate (whichever is available readily).

• **Rate of infusion:**
- If hypotensive: Administer fluids rapidly through push and pull method
- If in compensated shock: Administer fluids over a period of 15 to 20 mins by gravity method
- Be very cautious with fluids in severe malnutrition, anemia and underlying cardiac disease (refer box 1.4)

Volume:
- 20 ml/kg in hypovolemic and septic shock (may require upto 40-60 ml/kg)

Smaller aliquots (5ml/kg) in suspected myocardial dysfunction

• Watch for signs of fluid overload (Increase in tachycardia and liver span new onset crepitations or worsening tissue perfusion)
• Response to fluids is monitored by looking for therapeutic end points **(refer box 1.5)**

Box 1.4 Fluid therapy in specific conditions

- Severe Malnutrition:
- 15 ml/kg over 1 hour
- RL with 5% D, 0.45% Nacl with 5% D

- Cardiogenic Shock
- 5-10 ml/kg slow boluses
- Start on early ionotropic support
- Accept just 5th centile blood pressure

Box 1.5: Therapeutic end points of shock
- Decreased HR, improved tissue perfusion
- Increasing urine output> 1ml/kg/hr

- Normal capillary refill ≤ 2 sec
- Normal peripheral pulses
- Warm peripheries
- Normal level of consciousness
- Reversing hypotension

B) Optimizing cardiac output: Vasoactives are required to augment cardiac output (inotrope) or improve Systemic vascular resistance (SVR). Any child requiring >60 ml/kg fluids (fluids refractory shock) or develops signs of fluids overload during resuscitation requires vasoactive therapy. Children with septic shock require vasoactive therapy of underlying myocardial dysfunction and vasodilation(refer box 1.6).

1.6 How to prepare infusions of vasoactive drugs?(refer appendix section)
BOX 1.6 : Choice of vasoactive therapy

Cold shock		**Warm shock with hypotension**
With normal BP	With hypotension	
• Low dose epinephrine (<0.3 µ/kg/min) • Dobutamine (5-20 µ/kg/min) • Milrinone (50 µ/kg loading over 10-16 min followed by 3-0.75 µ/kg/min)	• Dopamine (5-20 µ/kg/min) • Epinephrine (0.13 -1.0 µ/kg/min) • Norepinephrine (0.01 – 0.1 µ/kg/min) + Milrinone (0.3 – 0.75 µ/kg/min)	• Norepinephrine (0.1 - 20µ/kg/min • Vasopressin (0.0001) – 0.000251 IU/kg/min)

1. 7 How to monitor?
a) Child should be monitored by a nurse atleast every 2 hourly and by a doctor atleast 6hourly
b) What to assess? (Look for all therapeutic end points Box 1.5)

• Heart rate
• Pulses central and peripheral)
• Capillary refill time
• Blood pressure
• Temperature
• Leyel of consciousness
• Urine output

1.8 What is the checklist for non- response to vasoactive therapy (refer box 1.7)

*Box*1.7 : Checklist for non response
- Uncorrected preload (volume)
- Recheck: dose, infusion, rate

- Correct dyselectrolytemia : Ca++, Mg+, Glucose
  - Calcium (if ionic value not available them do ECG): 2ml/kg of calcium gluconate in equal dilution with D5 over 20 mins under heart rate monitoring (ceiling dose of 10 ml)
  - Glucose: Remember product of volume and concentration of dextrose should be 50
- 2ml/kg of 25 % dextrose
- 5ml/kg of 10% dextrose
- 10ml/kg of 5% dextrose

1.9 Specific treatment of shock (refer box 1.8)

BOX 1.8: Specific treatment of shock

Hypovolemic	a) Hemorrhagic • Control external bleeding 20ml/kg NS/RL bolus, repeat as needed • Transfuse PRBC • Look for hidden bleeding source if shock is uncontrolled b) Non hemorrhagic (e.g. diarrhoea, dehydration) • 20mllkg NS/RL bolus, repeat as needed
Cardiogenic	• NS/RL bolus 5-10mllkg over 10-20min (small bolus) • Assess need for positive pressure ventilation Start early inotropes Dopamine/ dobutamine/epinephrine (can be safely started throughperipheral line)
Obstructive	• Tension pneumothorax: Needle thoracentesis (2nd intercostal space) • Cardiac tamponade: Pericardiocentesis Pulmonary embolism: Thrombolytics Duct dependent circulation: Prostaglandin E1 infusion (0.01 – 0.2µ/kg/min)
Distributive	a) Septic: • As per algorithm b) Anaphylactic: • 0.01 mL/kg of intramuscular epinephrine (1:1000), repeat after 10 – 15 min in severe cases. • Fluid bolus as required • Anthihistamines, steroids

	• If hypotension persists, start epinephrine infusion (0.05 mcg/kg/min) c) Neurogenic: Fluid bolus Vasopressor (norepinephrine/epinephrine)

1.10 What are the causes of refractory shock? (refer Box 1.9)

Box 1.9 Unrecognized morbidities in refractory shock

- Ongoing blood loss and hypovolemia
- Hypoxia
- Acidosis
- Hypoglycemia
- Electrolyte disturbances
- Hypocalcemia
- Pericardial effusion
- Pneumothorax
- Hypoadrenalism
- Inadequate source control (loculated pus, empyema etc)
- Intrabdominal catastrophe (surgical abdomen, abdominal compartment syndrome)

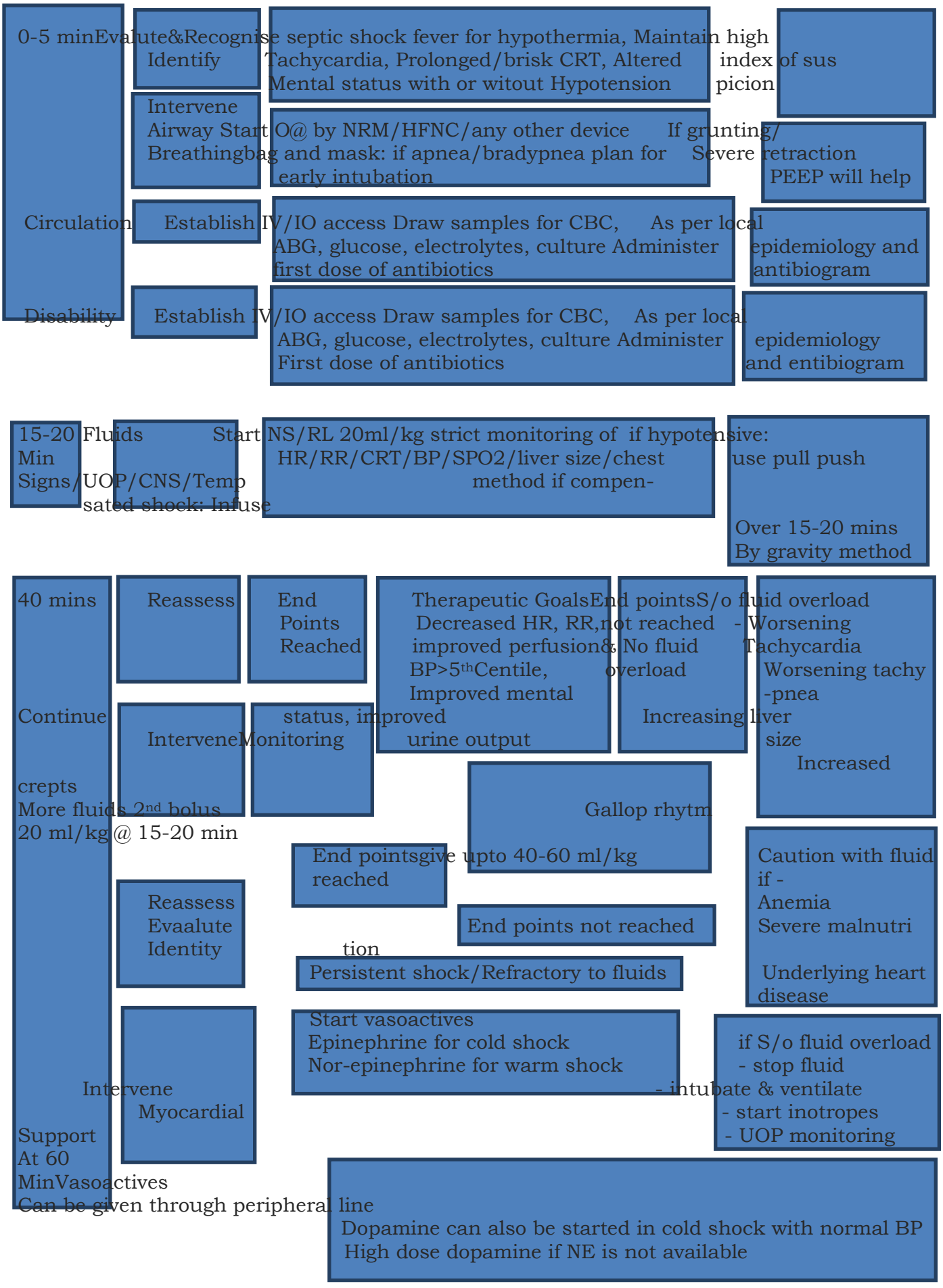
1.11 When to refer?

• Need for mechanical ventilation
• Fluid refractory catecholamine resistant shock - if PICU not available
• Cardiogenic shock which requires advanced support and diagnostics

1.12 How to refer?

• Refer only after stabilization, ensuring adequate airway, breathing and circulation
• Proper documentation regarding the presentation of the child, details of resuscitative measures taken, interventions done during resuscitation and reasons for referral including name and contact number of referring physician
• A doctor/paramedic trained in Pediatric Advanced Life Support/Basic Life Support to accompany the patient
• Transporting ambulance should have sufficient 02 supply and Resuscitation equipment
• Inform the referral Centre prior to sending the patient regarding the diagnosis of the child, indication for referral, current status and approximate time to arrival
• Counsel the family of the child regarding the need for transfer and risks during transfer. Obtain written, signed consent for the same

Protocol for management of septic shock



2. ACUTE GASTROENTERITIS

2.1 Learning objective~

After completionof this section, the participant should be able to

- Identify caseor acute diarrhoea
- Grade, severity of dehydration
- Understand principles of fluid therapy
- Counsel parents regarding home management in non- severe cases
- Understand diarrhoea can be a manifestation of systemic illness
- Know when and how to refer?

2.2 What is diarrhoea?

Diarrhoea: Passage of unusually loose or watery stools with an increase in frequencyConsistencyof stools is more important than frequency

Dysentery: Diarrhoea with blood in stools associated with abdominal cramps and fever

2.3 What is not diarrhoea?

- Passage of frequent wellformed stools
- Passage of pasty stools in breast fed infants
- Passage of stool during or immediately after feeding due to gastrocolic-reflex
- Passage of frequent loose greenish yellow stools on the 3rd and 4th day of life called as transitional stools

2.4 What are the steps in management?

a) Grade the severity of dehydration: The severity of dehydration can be assessed clinically as given in Box 2.1

BOX 2. Grades of dehydration

Characteristics	No dehydration	Some dehydration	Severe dehydration
1. Condition	Well, alert	Restless & irritable	Lethargic
2. Eyes	Normal	Sunken	Very sunken & dry
3. Tears			
4. Tongue	Present	Absent	Absent
5. Thirst	Moist	Dry	Very dry
6. Skin turgor	Drinks normally	Thirst & drinks	Unable to drink
7. Extrmities		eagerly	
8. Pulse quality			
9. Urine output	Normal	Delayed	Very Delayed
10. Estimated	Normal	Prolonged	Prolonged
Weight loss %	Normal	Cold	Weak & thread
ml/kg			Anuria for 6 hr
11. Treatment plan	Normal	Decreased	
	<5%		
	<50 ml/kg	5-10%	>10%
	Plan A	50-100 ml/kg	>100 ml/kg
		Plan B	Plan C

Remember there is an alternate classification of mild, moderate and severe dehydration to assess the severity of dehydration

b) Fluid therapy in diarrhoea has 4 components
a. Resuscitate (Intravascular volume expansion)
b. Rehydrate (Deficit correction)
c. Replace (Ongoing losses)
d. Maintain (Maintenance therapy)

c) Severe dehydration: Children, With severe dehydration and shock need resuscitationfollowed by replacement. Immediate fluid resuscitation. (For more details refersection B, chapter: 1, point 1.8)

20ml/kg of Ringer's Lactate (RL) or Normal Saline (0.9%) IV or intraosseous (IO) till perfusion improves (quality of central and peripheral pulses, CFT and *blood* pressure) In children with no signs of shock or in those with severe dehydration after adequate resuscitation start Plan C: 100ml/kg RL or NS. This needs to be given over a period of 6 hrs in <12months and 3hrs in >12 month old child as shown below

	30ml/kg	70ml/kg
<12 mon	1hour	5 hrs
1- 5 yr	1/2 hour	2.5hrs

Replace losses:
For <2 years: 50 to 100 ml ORS per loose motion
For >2years- 10 years: 100 to 200 ORS per loose motion
If unable to take orally then give by nasogastric tube or as IV 0.45% NS in 5 % dextrose in same dose

d) Some dehydration: Children with some dehydration need rehydration followed by replacement, Plan B. ORS 75ml/kg over 4 hrs is used as rehydrating fluid.
Replace losses:
For <2 years: 50 to 100 ml ORS per loose motion
For >2years- 10 years: 100 to 200 ORS per loose motion
e) No dehydration: Children just need replacement of losses Plan A. Most can be treated on ambulatory basis at home while some may need a brief period of observation. The indication for observation in Emergency department are listed in Box 2.2

Box 2.2 Indications of observation in Emergency department
Stable newborns and infants with diarrhea without features of toxicity and dehydration
Moderate dehydration but accepting well orally
Malnourished children with mil dehydration
Patient with diarrhea without dehydration but with decreased oral mistake.

Box 2.3 Indications for hospitalization
- Severe dehydration
- New born and infants <3 months of age with dehydration

- Malnourished children with moderate/severe dehydration
- Toxic appearance, changing mental status (GCS<11) or seizures
- High output diarrhea (>10 large volume stool/day)
- Persistent vomiting, or decreased/no oral intake
- Suspected surgical cause: localizing findings and enterocolitis need surgical consult
- Suboptimal or no response to oral rehydration therapy (ORT) or further deterioration
- Inability of caregivers to administers ORS replacement

g) Adjuvant therapy: Zinc decreases the severity and duration of dehydration
o Dose:< 6 months- 10mg /d for 14 days
o Dose: >6months - 20mg/d for 14 days

h) Nutrition: Continue breast feeding, home based fluids (rice water, soup, yoghurt drink).Avoid drinks sweetened with sugar (e.g. commercial carbonated beverages, fruit juices, sweetened tea,coffee)as they can cause osmotic diarrhoea and hypernatremia

i) Antibiotics are not routinely recommended, unless indications as enumerated in box 2.4 are there.
Box 2.4 indications for antibiotics
Clinical signs of sepsis (toxic look, leukocytosis, fever>38.5 C, septic shock)
Severe malnutrition
Neonates and very young infants (<3 mo) with fever
Dysentery (bloody stools)

2.5 How to monitor?
• Child needs to be monitored by nurse atleast every 4 hourly
• What to monitor?
o Hemodynamic status
o Sensorium
o Hydration status
o Number of stools passed
o Urine output
o Oral intake
o Adequacy of care by mother /caretaker

2.6 When to discharge patient?
• Once rehydration therapy is initiated, assess patient after 2 hours
• Discharge only if -
o Clinical status improving
o Accepting orally
o Normal sensorium
o Adequate urine output
o No indication for observation or admission
2.7 When should mother/caretaker return to health care facility?
• Stool frequency> 10 times/day
• Has sunken eyes, fever
• Not accepting orally

• Not passed urine for 6 hrs
• Abnormal sensorium

2.8 What is parental diarrhoea?
Few systemic illnesses can have diarrhoeaas the presenting symptom (refer box 2.5)

Box 2.5 Causes of parenteral diarrhea
- Otitis media
- Bacterial Pharyngitis
- Urinary tract infection
- Pneumonia
- Meningitis
- Bacterial sepsis
- Toxic shock syndrome
- Acute surgical abdomen: Colicky abdominal pain vomiting, lethargy, bloody
- Diarrhea, abdominal distention, and palpable mass suggests intussusception, or malrotation

2.9 When to refer?
• Diarrhoea with fluid refractory shock
• Diarrhoea associated with decreased urine output, abdominal distension, encephalopathy, seizures or any other complications
• Diarrhoea as presenting symptom of systemic illness (Box 2.5)

2.10 How to refer?
- Refer only after stabilization, ensuring adequate breathing and circulation
- Proper documentation regarding the presentation of the child, details of resuscitative measures taken, interventions done during resuscitation and reasons for referral including name and contact number of referring physician.
- A doctor/paramedic trained in Pediatrics Advanced Life Support/Basic life Support to accompany the patient
- Transporting ambulance should have sufficient O2 supply and Resuscitation equipment.
- Inform the referral centre prior to sending the patient regarding the diagnosis of the child, indications for referral, current status and approximate time to arrival
- Counsel the family of the child regarding the need for transfer and risks during

Remember
- Fluid therapy in diarrhea has 4 domains
  - Resuscitate (intravascular volume expansion
  - Rehydrate (Deficit correction)
  - Replace (ongoing losses)
  - Maintain (Maintenance therapy)
- Use oral route when available
- In a child not responding to fluids first re-check

- Quantity of fluids
- Compliance to ORS
- Ongoing losses
- Associated sepsis

Transfer. Obtain written, signed consent for the same.

Protocol for management of Acute Diarrhoea

Child with diarrhea

Grade severity of dehydration

No dehydration

Replace losses orally
<2 years: 50 – 100 ml
ORS per loose
Motion > 2 years – 10 years:
100 to 200 ORS per loos
Motion

Supportive treatment:
Nutrition
Ensure adequate calorie intake
Continue breast feeding
Age appropriate normal diet
Zinc supplementation:
Dosage: < 6 months – 10 mg/d, >6 months-20mg/d
Duration 14 days

Some dehydration

Rehydration
75 ml/kg ORS over 4hrs
Replace losses

Replace losses
If unable to take orally then give by nasogastric tube or give IV 5% Dextrose in ½ NS in
Same dose

Severe dehydration

Resuscitation:
20 ml/kg IV/IO RL/NS if in shock till perfusion improves
If no signs of shock then give 100 ml/kg RL
Divided as below

	30ml/kg	70 ml/kg
<12 mon	1 hour	5hrs
1-5 yr	½ hour	2.5 hrs

3. CARDIAC ARREST

3.Learning Objectives
After completion of this section, the participants should be able to
• Understand the sequence of events leading to a cardiac arrest
• Early identification of pre-arrest risk factors
• Timely intervention to prevent cardiac arrest
• Immediate identification of cardiac arrest and the contributory rhythms
• Components of high quality CPR
• Algorithmic management of cardiac arrest

3.2 What are the causes of cardiac arrest?
• Most common cause of arrest in infants, children and adolescent is hypoxia resulting from respiratory failure or shock
    - o These usually occur with prior warning signs (such as signs of respiratory distress and/or shock).
    - o Initially the child will compensate for hypoxia by increasing respiratory rate, work of breathing ,heart rate and cardiac output
    - o Once these compensatory mechanism get exhausted, thereis a rapid downhill progression to cardiopulmonary failure and cardiac arrest
    - o Therefore it is important to recognize and treat children in respiratory distress and shock before they progress to cardiopulmonary failure and cardiac arrest

• Rarely cardiac arrest may occur without prior warning signs as in cardiac arrhythmias *(VF/*pulseless VT). The risk of arrhythmias increases in the following conditions
    - o Congenital heart defects
    - o Myocarditis and coronary artery abnormalities
    - o Electrolyte abnormalities
    - o Long QT syndrome

3.3 How to indentify conditions that cause cardiac arrest ? (refer box 3.1)
a) Certain finding in the assessment need to be identified and corrected immediately so that cardiac arrest is prevented.

Box 3.1

Parameters	**Finding**
Airway	Not maintainable, complete or severe airway obstruction
Breathing	Bradypnea, tachypnea, gasping, irregular breathing, seesaw breathing Increased respiratory effort (severe chest retractions, head bobbing) Stridor, grunting SPo2 <94% in room air, SPO2 <90% on 100 % O2
Circulation	Bradycardia, tachycardia, irregular rhythm Weak/absent peripheral or central pulses CRT>3 sec Cool, pale, dusky, mottled extremities Hypotension
Disability	Decreased level of consciousness (GCS), hypotonia, generalized seizures, abnormal pupillary response
Exposure	Trauma, significant bleeding manifestation

b) Similarly some reversible causes (refer box 3.2) always need to be looked at and corrected for prevention as well as during treatment of cardiac arrest

Box 3.2 : Reversible causes of cardiac arrest

H's	T's
Hypovolemia Hypoxia Hydrogen ions (acidosis) Hypoglycemia Hyperkalemia/Hypokalemia Hypothermia Hyperthermia	Tension pneumothorax Tamponade Toxins (drugs, poisons, anaphylaxis) Thrombosis in pulmonary artery Trauma Prolonged QT interval

3.4 How to indentify a child in cardia arrest?
Unresponsiveness
Not breathing or gasping and
No central pulses (should not take more than 10 seconds to assess the pulse)

3.5 How to manage cardiac arrest?

Goal is to achieve return of spontaneous circulation (ROSC) which is described as resumption of organized cardiac electrical rhythm with palpable central pulses

This can be achieved by

High quality CPR which includes chest compressions and assisted breaths (refer box 3.3)

- Advanced life support measures (refer box 3.4)
- Advanced airway management
- Establishment of vascular access
- Defibrillation
- Medications
- Continuous monitoring

Box 3.3 Components of high quality CPR

- Push hard – at least 1/3rd of anterior – posterior chest diameter, about 1.5 inches (4cm) in infants and 2 inches (5cm) in children
- Push fast – at least 100 compressions/min
- Allow complete recoil of chest after each compression
- Minimize interruptions - <10 seconds for ventilation
- Avoid hyperventilation

To identify rhythms those have caused cardiac arrest. There are four rhythms which can be cause pulseless arrest

Asystole

Pulseless Electrical Activity (PEA)
Pulseless ventricular Tachycardia (pVT)
Ventricular Fibrillation (VF)

Of these the first two are treated with effective CPR and epinephrine and hence known as non shockable rhythms whereas the last two require effective CPR and defibrillation (unsynchronized shock) and known as shockable rhythms

Advanced airway and breathing management	After opeining the airway provide effective ventilation using bag and mask and consider using advanced airway i.e. endotracheal tube or Laryngeal mask (refer appendix airway adjuncts)
Establishing vascular access	To deliver drug directly into circulation Routes used in order of preference are Intravenous Intraosseous (refer appendix) Endotracheal (ET): Used only if any of the above access is unavailable Durgs given by ET route; Vasopressin, Lidocaine, Epinephrine, Atropine and Naloxone (V-LEAN) Deliver the drug when chest compressions are being performed followed by 5 ml normal saline flush every time. Withhold the compressions during ET administration of the drug followed by 5 ml normal saline flush and provide 5 rapid manual breaths.
Defibrillation	Refer appendix
Medications	Drugs used in treating cardiac arrest: Epinephrine Amiodarone, Lidocaine and Magnesium sulfate Calcium, atropine and sodium bicarbonate are not recommended in cardiac arrest as they do not improve survival
Continuous monitoring	At least heart rate, respiratory rate, SpO2, blood pressure, rhythm

3.6 How to manage different rhythms in Cardiac arrest?
Every patient in cardiac arrest, must be connected to a monitor to identify rhythm. TheEEG will show one of the four rhythms as described above. The Management of cardiacarrest follows an algorithm as shown in Fig E.

A. Asystole:
• It is defined as complete absenceof electrical activity of *heart* and is reflected as a flat line on ECG (Fig *A)*. It should be always corroborated by an absent central pulse.
• It is managed with CPR and epinephrine

A:Asystole

B. Pulseless electrical activity:

• PEA is defined as some electrical activity seen on the ECG/ monitor (Fig B) other than VF/VT but without a palpable central pulse
• In other words, there is some electrical activity but no myocardial contractions and cardiac output; hence no centralpulse is felt
• The ECG rhythm may be
o Normal or wide QRS complex (with lower than normal rate)
o Prolonged PR or QT interval or
o Low or high amplitude T waves
o Complete heart block

• This condition is always secondary to underlying reversible causes
• Management includes effective CPR,advanced airway, epinephrine and identifying and treating the underlying cause

Fig B: Pulseless electrical activity

C. Ventricular Fibrillation:
• It is a life-threatening cardiac arrhythmia characterized by disorganized myocardial contractions that results in absent cardiac output and central pulse.

• ECG does not have identifiable QRS complex. Instead small or coarse fibrillatory waves are seen (Fig C)
• This rhythm is uncommon in children and more common in adults with myocardial infarction
• Management includes effective CPR and prompt defibrillation (unsynchronized shock)

Fig C: Ventricular Fibrillation

D. Pulseless Ventricular Tachycardia:
• Ventricular Tachycardia(VT) is a wide QRS arrhythmia and may nor may not be associated with a central pulse
• Management includes effective CPR and prompt defibrillation. .'
(unsynchronized shock

3.7 When to terminate CPR?

- It can be discontinued if there is no ROSC at any time during 30 minutes f cumulative advanced life support.
- Resuscitation efforts can be extended in the following conditions
- Drug overdose
- Sever pre-arrest hypothermia (e.g. near -drowning in cold water)
- Toxin exposure
- Electrolyte abnormalities

Remember
• Most common cause of arrest in infants, children and adolescent is hypoxia resulting from respiratory failure or shock
• Early identification and effective treatment of respiratory failureandshock can prevent cardiac arrest
• Once cardiac arrest is identified, start high quality CPR
• Always look for and correct reversible factors

- Cardiac arrest does not always mean asystole., there are other rhythms associated with cardiac arrest e.g VF, pVT and PEA
- Asystole & PEA require effective CPR & epinephrine (non-shockable rhythms)
- Pulseless VT & VF require effective CPR & defibrillation (Shockable rhythms)

Algorithm For management of cardiac arrest

1. Verify scene safety. Assess responsive & look for evidence of etiology e.g. drug, poisoning or other
2. If unresponsive, shout for help (Activate emergency response system if witnessed collapse)
3. Look for normal breathing and check carotid/brachial pulse simultaneously within 10 second
4. Start CPR if cardiac arrest (unresponsiveness, not breathing or only gasping and no central pulses felt
5. Attach and power on AED or manual defibrillator. Check rhythm

6. VF/pVT
8. Give shock

9. Resume CPR immediately Without pulse check. Give CPR for 2 min. Give epinephrine every 3 to 5 min interval.
10. Check rhythm
11. Give shock if VF/pVT

13. Resume CPR immediately without pulse Check. Give CPR for 2 min Give Amidarone

7. Asystole/PEA

14. Resume CPR immediately. Give CPR for 2 min give Epinephrine every 3 to 5 min interval.

12. Organized rhythm: Pulse present :ROSC Give post cardiac arrest care

Medications

Epinephrine:

IV/IO	Endotracheal
1:10,000 Strength 0.1 ml/kg. Repeat every 3 to 5 min.	1:1000 strength 0.1 ml/kg

Amiodarone:

IV/IO	Endotracheal
5 mg/kg bolus. May be repeated up to 2 times for refractory VF/pVT	Not recommended

Lidocaine:

IV/IO	Endotracheal

Important Instructions:

- During CPR, Bag and mask ventilation should
- Preferably by given using 100 % oxygen.
- Take vascular access – IV/IO
- Prepare and consider for advanced airway if multiple rescuers available.
- Identify the reversible cause. 8H's and 8T's
- Talk to relatives if additional staff available

3. Cardiac Arrest

3.8 Learning Objectives

- After completion of this section, the participant should be able to
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o These usually occur with prior warning signs (such as signs of respiratory distress and/or shock).

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o Therefore it is important to recognize and treat children in respiratory distress and shock before they progress to cardiopulmonary failure and cardiac arrest

• Rarely cardiac arrest may occur without prior warning signs as in cardiac arrhythmias *(YFI* pulseless YT). The risk of arrhythmias increases in the following conditions

o Congenital heart defects

o Myocarditis and coronary artery abnormalities

o Electrolyte abnormalities

o Long QT syndrome

3.10 How to identify conditions that cause cardiac arrest? (refer box 3.1)

c) Certain finding in the assessment need to be identified and correct immediately so that cardiac arrest is prevented.

BOX 3.1

Parameters	Findings
Airway	Not maintainable, complete or severe airway obstruction
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Exposure	Trauma, significant bleeding manifestation

d) Similarly some reversible causes (refer box 3.2) always need to be looked at and corrected for prevention as well as during treatment of cardiac arrest

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Identify a child in cardiac arrest?
Unresponsiveness
Not breathing or gasping and
No central pulses (Should not take more than 10 second to assess the pulse)

3.12 How to manage cardia arrest?

- Goal is to achieve return of spontaneous circulation (ROSC) which is described as resumption of organized cardiac electrical rhythm with palpable central pulses
- This can be achieved by
- High quality CPR which includes chest compression and assisted breaths (refer box 3.3)

Advanced life support measures (refer box 3.4)

- Advanced airway management
- Establishment of vascular access
- Defibrillation
- Medications
- Continuous monitoring

Box 3.3 components of high quality CPR

- Push hard – at least 1/3rd of anterior – posterior chest diameter, about 1.5 inches (4cm) in infants and 2 inches (5cm) in children
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Pulseless Electrical Activity (PEA)
Pulseless Ventricular Tachycardia (pVT)
Ventricular fibrillation (VF)

Of these, the first two are treated with effective CPR and epinephrine and hence know as non-shockable rhythms whereas the last two require effective CPR and defibrillation (unsynchronized shock) and know as shockable rhythms

Box 3.4: Advanced life support measures

Advanced airway and breathing management	After opening the airway provide effective ventilation using bag and mask and consider using advanced airway i.e. Endotracheal tube or Laryngeal mask (refer appendix airway adjuncts)
Establishing vascular access	To deliver drug directly into circulation Routes used in order of preference are Intravenous Intraosseous (refer appendix) Endotracheal (ET); Used only if any of the above access is unavailable Drugs given by ET route; Vasopressin, Lidocaine Eoinphrine, Atropine and Naloxone (V-Lean_. Deliver the drug when chest compressions are being performed followed by 5 ml normal saline flush every time. Withhold the compression during ET administration of the drug followed by 5 ml normal saline flush and provide 5 rapid manual breaths.
Defibrillation	Refer appendix
Medications	Drugs used in treating cardiac arrest: Epinephrine Amiodarone, Lidocaine and magnesium sulfate Calcium, atropine and sodium bicarbonate are not recommended in cardiac arrest as they do not improve survival
Continuous monitoring	At least heart rate, respiratory rate, SpO2, blood pressure, rhythm

3.13 How to manage different rhythms in Cardiac arrest?
Every patient in cardiac arrest, must be connected to a monitor to identify rhythm. The ECG will show one of the four rhythms as described above. The management of cardiac arrest follows an algorithm as shown in Fig. E.

E. Asystole:

- It is defined as complete absence of electrical activity of heart and is reflected as a flat line on ECG (Fig A). It should be always corroborated by an absent central pulse.

- It is managed with CPR and epinephrine

F. Pulseless electrical activity:
PEA is defined as some electrical activity seen on the ECG/monitor (Fig B) other than VF/VT but without a palpable central pulse
In other words, there is some electrical activity but no myocardial contractions and cardiac output: hence no central pulse is felt

The ECG rhythms may be

- Normal or wide QRS complex (with lower than normal rate)
- Prolonged PR or QT interval or
- Low or high amplitude T waves
- Complete heart block

This condition is always secondary to underlying reversible causes
Management includes effective CPR, advanced airway, epinephrine and identifying and treating the underlying cause.

G. Ventricular Fibrillation:

- It is a life-threatening cardiac arrhythmia characterized by disorganized myocardial contractions that results in absent cardiac output and central pulse.
- ECG does not have identifiable QRS complex. Instead small or coarse fibrillatory waves are seen (Fig C)
- This rhythm is uncommon in children and more common in adults with myocardial infarction
- Management includes effective CPR and prompt defibrillation (unsynchronized shock)

H. Pulseless ventricular Tachycardia:

- Ventricular Tachycardia (VT) is a wide QRS arrhythmia and may nor may not be associated with a central pulse.
- Management includes effective CPR and prompt defibrillation
- (Unsynchronized shock)

3.14 When to terminate CPR?

- In can be discontinued if there is no ROSC at any time during 30 minutes of cumulative advanced life support.
- Resuscitation efforts can be extended in the following conditions
  - Drug overdose
  - Severe pre-arrest hypothermia (e.g. near-drowning in cold water)
  - Toxin exposure
  - Electrolyte abnormalities

Remember

- Most common cause of arrest in infants, children and adolescent is hypoxia resulting from respiratory failure or shock.
- Early identifications and effective treatment of respiratory failure and shock can prevent cardiac arrest
- Once cardiac arrest is identified, start high quality CPR
- Always look for and correct reversible factors
- Cardiac arrest does not always mean asystole., there are other rhythms associated with cardiac arrest e.g. VF, pVT and PEA
- Asystole & PEA require CPR & epinephrine (non-shockable rhythms)
- Pulseless VT & VF require effective CPR & Defibrillation (Shockable rhythms)

Algorithm For management of cardiac arrest

7. Verify scene safety. Assess responsive & look for evidence of etiology e.g. drug, poisoning or other
8. If unresponsive, shout for help (Activate emergency response system if witnessed collapse)
9. Look for normal breathing and check carotid/brachial pulse simultaneously within 10 second
10. Start CPR if cardiac arrest (unresponsiveness, not breathing or only gasping and no central pulses felt
11. Attach and power on AED or manual defibrillator. Check rhythm

12. VF/pVT

8. Give shock

9. Resume CPR immediately Without pulse check. Give CPR for 2 min. Give epinephrine every 3 to 5 min interval.

10. Check rhythm

11. Give shock if VF/pVT

13. Resume CPR immediately without pulse Check. Give CPR for 2 min Give Amidarone

7. Asystole/PEA

14. Resume CPR immediately. Give CPR for 2 min give Epinephrine every 3 to 5 min interval.

12. Organized rhythm: Pulse present :ROSC Give post cardiac arrest care

Medications

Epinephrine:

IV/IO	Endotracheal
1:10,000 Strength 0.1 ml/kg. Repeat every 3 to 5 min.	1:1000 strength 0.1 ml/kg

Amiodarone:

IV/IO	Endotracheal
5 mg/kg bolus. May be repeated up to 2 times for refractory VF/pVT	Not recommended

Lidocaine:

IV/IO	Endotracheal

Important Instructions:

- During CPR, Bag and mask ventilation should
- Preferably by given using 100 % oxygen.
- Take vascular access – IV/IO
- Prepare and consider for advanced airway if multiple rescuers available.
- Identify the reversible cause. 8H's and 8T's
- Talk to relatives if additional staff available

APPROACH TO A CASE OF TROPICAL FEVER

1.1 Learning objectives:

After completion of this section, participants should be able to

- Assess and identify a case of tropical fever
- Classify them into Syndromic patterns
- Identify the physiological impairment and MODS
- Initiate stabilisation and broad empirical cover
- Know the point of care testing that should be ordered
- Initiate specific therapy based on etiological diagnosis
- Counsel parents regarding home management in non severe cases
- Know when and how to refer?

1.2 What are tropical fevers?

- Tropical fevers are defined as infections prevalent in, or unique to tropical and subtropical regions
- Most of these infections cluster during monsoon and post monsoon period
- Most are vector borne and transmitted by an insect bite
- There is a significant overlap in clinical presentation of these infections making it difficult to arrive at a specific etiological diagnosis
- Hence simplified Syndromic approach in an emergency setting is better than and individual disease approach
- This strategy helps in timely stabilisation and management.

1.3 How to identify a case with tropical fever?

- Any febrile child presenting to emergency department with the following
  - Duration of fever <2weeks
  - Most of them will cluster around rainy and post rainy season
  - Non-specific symptoms: headache, myalgia, arthralgia, retro-orbital pain, photophobia
  - Skin rash
  - Multiorgan involvement including encephalopathy, seizures, jaundice hepatosplenomegaly, respiratory distress and renal failure.
  - No specific localisation to a single organ
- Common tropical infections include
  - Malaria
  - Dengue

- Scrub typhus
- Leptospirosis
- Typhoid

1.4 How to approach a case of suspected tropical fever?

- The primary focus should be on identification of physiological impairments and stabilizing them
- Identify the presence of associated symptoms along with fever
- Classify the constellation into a syndrome to guide initial empiric therapy in a critically ill child (refer box 1.1)
- Remember children with MODS like encephalopathy, rash, thrombocytopenia, ARDS, jaundice, bleeding and acute kidney injury need urgent treatment
- Proceed with first line point of care investigation (refer box…….)
- Initiate a broad empirical cover while awaiting laboratory results.
- Depending on the result proceed with further management.

Box 1: Syndromic approach to tropical fevers in emergency room

Fever with rash/thrombocytopenia	Dengue Malaria Rickettsial infections Typhoid Leptospirosis
Fever with encephalopathy	Scrub typhus Cerebral malaria Typhoid encephalopathy Meningitis Encephalitis
Fever with ARDS	Scrub typhus P. Faliciparum malaria Leptospirosis Influenza (H1N1)
Fever with jaundice	Malaria Leptospirosis Dengue
Fever with multiorgan dysfunction	Falciparum malaria Leptospirosis Scrub typhus Dengue Bacterial sepsis

Box 2: Point of care test in suspected tropical fevers

Box 1.2: Baseline investigations

- Complete blood count with peripheral smear
- Serum electrolytes with renal function test
- Liver function test
- Urine analysis
- Rapid diagnostic test
  - Malaria card test
  - Dengue card test
  - Dengue NSI antigen

Malaria

- Suspect malaria in any case of tropical fever spectrum with pallor, hepatosplenomegaly, multiorgan involvement
- Confirm the diagnosis with either microscopy or rapid diagnostic test
- Rule out malaria if two negative RDT
- Identify any signs of severe malaria (refer box 3)
- Patient with no features of severe malaria is defined as having uncomplicated malaria
- Treatment of uncomplicated malaria is ACT for 3 days
  - Artemether + Lumefanthrine
  - Artemether +amodiaquine
  - Artemether +mefloquine
  - Artemether +sulfadoxine – pyrimethamine: is safe and effective for uncomplicated P. Falciparum malaria.
  - Chloroquine is safe and an effective for uncomplicated P. vivax malaria.

Box 3. Features of severe malaria

Features of severe malaria

- Multiple convulsions>2 in 24 hours
- Impaired consciousness
- Severe anemia, Hb<5g/dl
- Hypoglycemia, RBS <60mg/dl
- Metabolic acidosis, HCO3<15mmol/l or base deficit>8 meq/l
- Acute renal failure (serum creatinine>3mg/dl)
- Jaundice (serum bilirubin>3mg/dl)
- ARDS
- Shock (“algid malaria)
- DIC
- Hemoglobinuria
- Hyperparasitemia(>5%)

Treatment of severe malaria

1. Stabilize ABCDE:

- Start supplemental oxygen for children with sever pallor, tachypnea and/or shock
- Those with impaired (GCS≤8) on, ALI/ARDS, shock, pulmonary edema, and require endotracheal intubation and mechanical ventilation
- Management of septic shock follows standard guidelines using fluids and vasoactive drugs. Fluid boluses should be used with extreme caution (due to severe anemia) and only if there is frank hypotension.
- Adequate seizure control and management of raised intracranial pressure in cerebral malaria.
- Rapid detection and correction of hypoglycaemia is extremely important. A bolus (5ml/kg) of 10% dextrose solution be given by a rapid IV push. RBS should be rechecked after 30 minutes. It is important to give 10% dextrose in normal saline or Ringer's lactate for maintenance infusion to prevent hypoglycaemia. Routine blood glucose monitoring is mandatory.
- Urgent blood transfusion is indicated if haemoglobin is below 7g%.
- Children presenting with high grade fever and shock should receive IV broad- spectrum antibiotics to cover for coexistent bacterial sepsis.

2. Specific treatment:

- Two important groups of antimalarial drugs are available – artemisinin derivatives (artesunate, artemether), and cinchona alkaloids, (quinine, quinidine, chloroquine).
- Artemisinin derivatives are more effective, simpler, and safer than cinchona alkaloids.
- For pre-referral administration, intramuscular (IM) artesunate is more effective than rectal artesunateIn children > 6 years, rectal artesunate is not recommended.
- The combination drugs available in artemisinin combination therapy (ACT) are given in box 4.
- The specific therapy of malaria is summarized in box 5.

Box 4: Combination drugs used in Artemisinin combination therapy (ACT)

- Artesunate + sulfadoxine – pyrimethamine
- Artesunate +amodiaquine
- Artesunate +lumefantrine
- Dihydroartemisinin + piperaquine
- Artesunate + mefloquine (avoided in cerebral malaria)
- Artesunate + doxycycline or clindamycin

- Drug of choice : Artesunate
  Dose: 3 mg/kg/dose in children < 20 kg : 2.4 mg/kg/dose in children> 20 kg
  ACT must be given parenteral for at least 24 hours\
  Once patient is able to take orally complete treatment with 03 days of ACT

Box 5:	Uncomplicated	Severe
P vivax	Areas With Chloroquine Or ACT Areas With Chloroquine Resistance – ACT Primaquine 0.25 -0.5 mg/kg/d for 14 d Except in < 6m & know G6PD deficiency G6PD deficiency: 0.75 mg/kg/w for 8 w G6pd status unknown 7 unavailable – individualize	ACT + Primaquine
P Falciparum	ACT + single dose of primaquine in low transmission areas (0.25 mg/kg – no G6PD testing required	ACT + single dose of primaquine in low transmission areas (0.25 mg/kg – no G6PD testing required
Mixed	ACT + Primaquine (as for vivax)	ACT + Primaquine (as for vivax)
Not know	As for uncomplicated P falciparum	As for complicate P falciparum

3. Management of complications

- Cerebral edema: Management is largely supportive and includes treatment of raised ICP, and maintenance of euthermia and euglycemia. Short term hyperventilation to achieve PCO2 ~ 30mm Hg can be done if signs of impending herniation are present.
- Hyperparasitemia and role of exchange transfusion: Exchange transfusion (ET) rapidly reduces the parasitic index (PI) and mortality. It may be used in complicated malaria with Hyperparasitemia or multiorgan dysfunction which fail to respond to chemotherapy alone, preferably in and intensive care setting.
- Acute kidney injury: Loop diuretics can convert an oliguric to non-oliguric renal failure and reduce the risk of volume overload. Presence of AKI or severe acidosis unresponsive to fluids is and indication fore renal replacement therapy.

SCRUB TYPHUS

- Suspect scrub typhus in any case of tropical fever spectrum with rash, hepatosplenomegaly, encephalopathy or multiorgan involvement.
- An eschar, when present is a valuable clue to the diagnosis and should be meticulously looked for in every child.
- As it is a potentially fatal but relatively easy to treat, keep a low threshold for empirical treatment.
- Confirm the diagnosis with IgM ELISA.
- Identify organ involvement and need for organ support
- Drug of choice is doxycycline.

1. Stabilize ABCDE:

- Start supplemental oxygen for children with tachypnea and/or shock
- Those with persistent hypoxemia and/or severe encephalopathy (GCS≤8) require endotracheal intubation and mechanical ventilation
- Management of septic shock follows standard guidelines using fluids and vasoactive drugs
- Adequate seizure control and management of raised intracranial pressure is crucial

2. Specific therapy:

- Currently, doxycycline remains the first choice even for children younger than 8 years

- Early treatment with doxycycline (5mg/kg/day in 2 divided doses, IV or oral shortens the disease course and reduces mortality
- Duration of therapy
    - 3-5 days after defervescence in uncomplicated cases
    - 7-14 days ins severe scrub typhus with organ failure
- Azithromycin (10mg/kg/day for 07 days) is an effective alternative

3. Management of complications

- Complications include ARDS, septic shock, myocardotos. Amd encephalopathy and raised intracranial pressure.
- Acute respiratory failure due to ARDS responds well to non-invasive ventilation (NIV) if started early. In delayed presentation, intubation and ventilation will be required.
- Myocardial dsyfuntion should be actively looked for (if possible) in all children with fluid –refractory shock. Addition of an inodilator like dobutamine or milrinone may be required to improve cardiac output. Continuous EKG monitoring is desirable to detect arrhythmias.
- Children with acute encephalitis syndrome require seizure control, sedation and analgesia and management of raised intracranial pressure (ICP_
- Renal replacement for Acute Kidney Injury (AKI) may be required in small proportion of patients.
- Platelet transfusion is required in symptomatic thrombocytopenia. Platelets recover with antiobiotics and defervescence.

4. Acute encephalitis

- Acute encephalitis is a medical emergency where stabilization, management of comlications, investigations and specific treatment all need to be performed simultaneously.
- Airway stabilization and hemodynamic support are the initial priorities.
- Raised ICP and seizures/status epilepticus are the 2 main complications that need aggressive management.
- Lumbar puncture is essential and should be performed after initial stabilization unless contraindicated.
- Neuroimaging is not required in all cases.
- Dengue fever, scrub typhus and cerebral malaria can present like an acute encephalitis's syndrome.

- Identifying treatable causes and empirically start therapy is essential.

1. Stabilize ABCDE:

- Assessment of airway and stabilization
- Hemodynam assessment and resuscitation if needed
- Assessment of level of consciousness using a quantitative scale like GCS. GCS below 8 and clinical signs of raised ICP are indications for rapid sequence intubation and ventilation.
- Management of complications like seizures and raised intracranial pressure (ICP).
- Correct reversible factors like hypoglycaemia, hypo or hypernatremia and hypcalcemia

2. Specific therapy:

- Empiric treatment pending neuroimaging and/or lumbar puncture includes antibiotics for bacterial meningitis and acyclovir for herpes simplex viral (HSV) ncephalitis.
- Remember that other tropical infections like dengue, malaria and scrub typhus can also present like an "encephalitis syndrome" and may need empiric treatment (Box.…)
- Hsv is the most common cause for sporadic encephalitis and one which is potentially treatable. When to give and when not to give acyclovir are enumerated in Box 6.

Box 6. When to give and when not to give acyclovir

Indications for Acyclovir

- Acyclovir should be initiated in all patients with suspected encephalitis, pending results of diagnostic studies.
- Fever with progressive deterioration of consciousness, focal seizures or focl neurological abnormalities in the absence of any other cause
- Dose: 20mg/kg/dose TDS as 1 hour infusion

When not to give acyclovir

- Child with simple febrile convulsions
- Seizures without documented fever or history of fever (unless immunocompromised)
- Other obvious cause for symptoms, e.g. blocked VP shunt, epilepsy (exacerbation with febrile illness)
- Acute head injury, drug overdose
- CSF and clinical picture are highly suggestive of bacterial

meningitis

Box 7: Empirical therapy in acute encephalitis syndrome

Antibiotics	Ceftriaxone
Antivirals	Acyclovir
Antimalarials	Artesunate
Others	Doxycycline Azithromycin

Management of complications:

The most important neurological complications of encephalitis are raised ICP and status epilepticus, therefore supportive management is needed for both. Patient positioning prevention of bed sores, nutrition and physiotherapy should be meticulously taken care off.

Dengue Fever

- Suspect dengue fever in all tropical fevers with rash, features of capillary leak or multiorgan dysfunction.
- Rapid WHO approved caerd tests using a combination of NS-1 antigen and Igm antibody have food sensitivity and specificity.
- Fluids are the mainstay of therapy and normal saline is the fluid of choice.
- Colloids may be needed in some cases.
- Therapeutic endpoints ofr dengue shock are different from those of septic shock
- Titrate fluids with close monitoring of hemodynamic parameters and haematocrit.
- Identifying occult bleeding is important and blood transfusion may be life saving
- All other transfusions and procedures may be more harmful than beneficial.
- Titrate therapy based on natural course of illness; overdoing in terms of fluids, drugs and transfusions can be fatal.

1. Stablize ABCDE:
   - Start supplemental oxygen for children with tachypnea and/or shock
   - Circulation should be classified as hemodynamically stable with danger signs, compensated shock (hypoperfusion with normal

blood pressure), and hypotensive shock as this will decide further management.
- Supportive therapy with supplemental oxygen and initial evaluation with a chest X-ray should be done.

Fluid resuscitation

(i) hemodynamically stable with danger signs:

- Start IV crystalloids, normal saline (NS) or Ringer's lactate (RL) at 5-7 ml/kg/hr for 1-2 hours.
- Titrate fluids according to clinical improvement and HCT change.
- Clinical improvements and decreasing HCT, fluid rate should be decreased to 3-5 ml/hr for 2-4 hours and then 2-3 ml/hr
- Clinical worsening and increasing haematocrit, fluid rate should be increased to 5-10 ml/hr
- Clinical worsening and falling haematocrit, suspect bleeding, which can be a occult gastrointestinal haemorrhage. Urgent blood product transfusion will be required in such cases.

(ii) Compensated Shock:

- Start on NS or RL at 5-10 ml/kg over one hour.
- If no improvements and static or increasing haematocrit, start on IV bolus of RL/NS – 10-20 ml/kg over 1 hour.
- If there is no improvement, give another bolus of crystalloid of colloid.
- Non response to 40 ml/kg of NS/RL should warrant check for haematocrit and occult bleed. Blood transfusion is indicated if there is fall in haematocrit without clinical improvement.
- Fluids should be tapered as soon as child is clinically improving and shock is passive.

(iii) Hypotensive shock:

- Rapid crystalloid 20 ml/kg over 15 minutes under close monitoring
- If no improvement give second bolus preferably colloid 10-20 ml/kg over 30 minutes to 1 hours which can be repeated if need be
- Bleeding should be rules out
- In fluid non-responders, suspect underlying myocardial dysfunction (diastolic or systolic). Initiate inotropic support and manage shock guided by serial HCT and echocardiography (if available).
- In persistent fluid refractory shock, actively look for occult severe bleeding, metabolic derangements (acidosis hypoglycaemia, hypocalcemia), myocarditis and cardiac dysfunction, abdominal

compartment syndrome, obstructive shock due to massive pleural/pericardial effusion and coexistent bacterial sepsis.

(iv) Indications for blood transfusion:

- Shock non-responsive to 40-60 ml/kg fluids
- Fall in haematocrit associated with hemodynamic instability
- Hypotensive shock with low/normal HCT
- Persistent or worsening metabolic acidosis along with abdominal tendereness or distension.
- In the above situations, 5-10 ml/kg of fresh packed red cell or 10-20 ml/kg of fresh whole blood can be given. Current literature does not support prophylactic platelet transfusions for thrombocytopenica; platelet transfuisons may be considered for children with severe bleeding. FFP may be considered in children with significant coagulopathy.

2. Specific treatement:

There is no specific treatment for dengue fever. Supportive therapy is the mainstay of management.

Management of complications

- Fluid overload: Children with dengue are predisposed to fluid overload due to excessive fluid administration during leaky phaseuse of hypotonic fluids during resuscitation resorption of fluid from extravascular to intravascular space at end of critical pahse fluid rate should be decreased timely once hemodynamic stability is achieved to prevent overload.

Treatment of established fluid overload includes fluid restriction, respiratory support in form of continuous positive pressure and removal of excess fluids by diuretics or continuous renal replacement therapy (CRRT), hemodialysis (HD) or peritoneal dialysis. CRRT is preferred because it causes mininmal hemodynamic instability. One has to be very cautious with diuretics during active capillary leakage as it can further worsen intravascular hypovolemia.

- Neurological complications Seizures and encephalopathy are commonest neurological manifestation and their treatment is primarily supportive
- Renal Failure: Renal replacement therapy modes useful during acute infection are CRRT, PD and HD. Bleeding diathesis complicate utilization of anticoagulation in CRRT and HD.

- Abdominal compartment syndrome: Excessive third spacing, fluid overload, intra-abdominal bleed can contribute to intra-abdominal hypertension (more than 10 mm Hg in children) and abdominal compartment syndrome (ACS). This can lead to worsening renal function, shock and respiratory distress. Management includes treatment of fluid overload with diuretics or renal replacement, gastrointestinal decompression through nasogastric tube and flatus tube and in sever cases peritoneal drainage.
- Respiratory failure: Early positive pressure support though non invasive modes will be helpful.

ENTERIC FEVER

- Suspect enteric fever in any case of tropical fever spectrum with hepatosplenomegaly, PUo or multiorgan involvement
- Confirm the diagnosis with blood culture
- Common complications are gastrointestinal andneurological.
- Decide on ambulatory vs. hospital based management.
- Accordingly, oral cefixime or IV 3rd generation cephalosporin is the drug of choice.

1. Stabilize ABCDE:
   - Children with persistent vomiting, abdominal distention, and poor oral acceptance or severe diarrhoea or any complications must be hospitalized
   - Start supplemental oxygen for children with tachypnea and/or shock
   - Those with persistent hypoxemia and/or severe encephalopathy (GCS≤8) require endotracheal intubation and mechanical ventilation
   - Management of septic shock follows standard guidelines using fluids and vasoactive drugs

2. Specific therapy

- Most patients with uncomplicated disease improve with home-based treatment comprising appropriate oral antibiotics, hydration, and supportive care.
- Parenteral antibiotics are indicated in children with persistent gastrointestinal symptoms, poor oral acceptance and those with systemic complications.
- Third –generation cephalosporins are the first line antibiotics of choice for hospitalized patients. Parenteral antibiotics should be

given for a period of at least 5 days after defervescence or for a total duration of 14 days.

- Oral cefixime (20mg/kg/d) is recommended for ambulatory patients.
- For uncomplicated quinolone sensitive strains, defixime or fluoroquinolones are the initial antibiotics of choice
- Alternatively, azithromycin can be used in cases of strains resistant to cephalosporins.
- Multi drug resistant strains should be suspected in children presenting with short duration of illness, serious complications, failure to respond to first line antibiotics or a known household contact or during an apidemic of MDR typhoid fever.
- Children with enteric fever have severe anorexia along with spiking fever, necessitating adequate hydration, liberal use of antipyretics and early resumption of nutrition.

3. Treatment of complication:

- Indications for PICU admission include:
- Hemodynamic monitoring for shock
- Neurological monitoring for encephalopathy
- Monitoring for abdominal complications
- Fluid and electrolyte balance
- Bleeding diathesis die to thrombocytopenia
- Disseminated intravascular coagulation and multiorgan failure

Organ supportive therapy

- Children presenting with shock or enteric encephalopathy should be treated with dexamethasone, initial dose of 3mg/kg followed by 1 mg/kg every 6 hours for total 8 doses.
- Intestinal haemorrhage requires intensive monitoring, volume resuscitation and blood transfusion.
- Patients with suspected intestinal perforation should be stabilized first followed by urgent surgical intervention.
- Secondary peritonitis with E.coli and Klebsiella is common after perforation and may lead to secondary organ dysfunction.
- Acute respiratory distress syndrome requires mechanical ventilator support.
- Myocarditis presenting as cardiogenic pulmonary edema and/or shock with ST segment, and T wave changes requires supportive care including ventilation, fluid restriction and inotropic support.

When to refer a child with suspected tropical fever?

- When child appears sick or toxic and there are signs of hemodynamic instability.
- When additional assistance for diagnosis and management for any of the diseases is required.

How to refer?

- Child's peripheral venous access should be secured
- Child's details history, examination findings, vitals, events occurred and the treatment given should be legibly written in the referral letter.

1. APPROACH TO A CHILD WITH BREATHING DIFFICULTY

1.1 Learning objectives

- After completion of this section, the participants should be able to
- Identify a child with breathing difficulty
- Differentiate between respiratory distress and respiratory failure
- Localise level of respiratory pathology based on clinical signs
- Resuscitate and initiate appropriate stabilisation measures
- Escalate respiratory support as indicated
- Identify likely etiology and initiate specific therapy
- Know when and how to refer?

1.2 How to differentiate respiratory distress and respiratory failure?

- Any pathology in the respiratory system will first cause respiratory distress (increased rate and retractions)
- Clinically the severity of respiratory impairment is classified as respiratory distress or failure
- By increasing respiratory rate and efforts, the child tries to maintain adequate gas exchange. This is body's compensatory mechanismto allow the child to maintain oxygenation within normal limits (SpO2>94%)
- Hypoxemia is defined as SpO2 ≤ 94%
- Respiratory failure ensues when child gets tired and compensatory mechanisms fail
  - This result in inadequate oxygenation (SpO2≤94%) and/or inability to eliminate carbon dioxide (PCO2>50mm Hg)
  - Respiratory failure can be acute (Pneumonia, pulmonary oedema) or chronic (chronic lung disease, heart disease or neuromuscular disorders)
- Based on the inciting mechanism and arterial blood gases, acute respiratory failure can be categorised into two broad categories (refer box 1.1)

Box 1.1: Types of respiratory failure

- Type I Respiratory failure: Occurs due to any condition that leads to hypoxemia, SpO2≤94% or PaO2 <60mm Hg eg. Pneumonia
- Type II Respiratory failure: Occurs due to any condition that causes hypoventilation resulting in hypercarbia (PCO2>50mm Hg) eg. Neuroparalytic snake envenomation

1.3 What is the stepwise approach to a child with breathing difficulty?

- Step 1 : Assessment: Begins with a quick visual and auditory evaluation using Pediatric Assessment Triangle followed by Primary assessment pentagon ABCDE (Refer section A)
- Step 2 : Classify severity and localize level of respiratory pathology
- Step 3 : Initiate stabilization measures
- Step 4: the etiology of respiratory distress by meticulous history and focused physical examination

History should include SAMPLE: signs and symptoms, Allergies, Medications, Past medical history, Last meal, and Events leading to the present illness

- Onset (Acute, recurrent, chronic) and nature of progression
- Associated symptoms: cough, fever, rash, chest pain
- Preceding events: choking, foreign body inhalation, trauma, exposure to chemical or environmental irritants.
- Family history: exposure to infections, tuberculosis, atopy

Focused physical examination should include

Clubbing, lymphadenopathy

Tracheal position and adequacy of chest movements

Adventitious sounds: stridor, wheeze, grunt (suggest alveolar disease causing loss of functional residual capacity), crepts

Detailed assessment of other systems like cardiovascular and neurological

- Step 5: Appropriate investigations
- Step 6: Initiate specific and adjuvant therapy (e.g antibiotics, fluids etc)
- Step 7: Reassessment and monitoring to decide need for escalation or de-escalation of therapy

1.4 How to localise organ system involved based on breathing pattern and understand the pathology by the pattern of breathing? (refer box 1.2 and 1.3)

Box 1.2

System	Characteristics	Examples
Respiratory	Tachypnea with increased efforts: subcostal, intercostal, subxiphoid retractions Abnormal sounds: stridor, grunting, wheezing, crepts	Croup, diphtheria, bronchiolitis, asthma pneumonia, pulmonary oedema
Cardiovascular	Disproportionate tachycardia, soft muffled heart sounds, cardiac murmurs, cardiomegaly, hepatomegaly, raised JVP, basal crepitations	Congestive cardiac failure, myocarditis, arrhythmia, pericarditis

Metabolic	Acidotic breathing: tachypnea without retractions, deep and sighing breathing and clear chest	Diabetic ketoacidosis, acute renal failure, inborn error of metabolism
Neurogenic hyperventilation	Increased rate and depth without retractions, and clear chest. Associated altered senosorium and features of raised intracranial pressur	Acute encephalitic syndrome, traumatic brain injury
Neurogenic	See-saw or paradoxic breathing (chest moving inwards with inspirations)	Neurotoxic snake envenomation, GuillianBarre syndrome, CNS depression

BOX 1.3: How to localise pathology and level within respiratory system?

Retractions + stridor	Upper airway obstruction	Croup, diphtheria, foreign body
Retractions + wheeze	Lower airway obstruction or small airway obstruction	Bronchiolitis, asthma
Retraction + grunt+ crepitations	Parenchymal disease	Pneumonia, pulmonary oedema, hemorrhage

1.5 Steps in stabilisation

- Goals of therapy are
  a. To relieve hypoxemia
  b. To give rest to overworked respiratory muscles
  c. To enhance CO2 wash out
  d. To support other organ system
  e. To treat underlying etiology
- Ensure and maintain an open and patient airway by
  - Proper position (gentle extension of the neck, rolled towel under shoulder)
  - Suction oropharyngeal secretions
  - Consider oropharyngeal airway if patient is unconscious, to prevent tongue falling back over airway (refer appendix)
  - Endotracheal intubation/tracheostomy in emergent conditions

  (refer box 1.4,1.5)

Deliver supplemental oxygen without increasing agitation

- By and delivery device that is available

- Choice between low flow or high flow oxygen delivery device depends on severity of hypoxemia, patient's flow demand and tolerability, availability and familiarity with device (refer appendix)
- Monitor for target SpO2 and escalate respiratory support accordingly
- Patients not responding to increasing FiO2 (SPO2≤94%) can be put on non invasive respiratory support, e.g. Bubble CPAP, Ventilator CPAP, or HFNC (refer appendix)

- If spontaneous breathing is absent or inadequate then initiate
- Assisted ventilation with bag and mask ventilation
- Endotracheal intubation as soon as adequate expertise and equipment are available

- Optimize all circulatory parameters
- Establish intravenous or intraosseous access (if venous access is not feasible)
- If patient is in shock initiate shock protocol (refer section C, Chapter 1)

- Once stabilization measures have been instituted, proceed to established the etiological diagnosis so as to initiate specific therapy (e.g. antibiotics for bacterial pneumonia or diuretics for congestive cardiac failure)

BOX 1.4 Indications of emergent endotracheal intubation

- Children with marked respiratory distress despite adequate FiO2
- Gasping, bradypneic or apneic
- Cyanosis with decreased respiratory efforts
- CNS signs of hypoxia (lethargic, unresponsive, obtunded, seizures, coma)
- Cardiovascular signs of hypoxia (marked tachycardia, bradycardia, or hypotension)
- Children who worsen on CPAP or other non invasiverespiratory support

Box 1.5 Indications of emergent tracheostomy

- Diphtheria
- Upper airway obstruction
- Facial trauma
- Unable to intubate

1.6 What is the likely etiology? How to investigate?

- Chest X ray PA view (once the child is stabilized)
- Arterial blood gas analysis (if available)

- Sepsis Work-up
  - Blood counts (TLC, DLC)
  - C-Reactive protein
  - Blood culture if available

1.7 How to monitor for response?

Monitoring is required to assess response to therapy and detect worsening/ complications

- What is monitor?
  - Temperature
  - Vitals: HR,RR,SPO2, perfusion, BP
  - Work of breathing and use of accessory muscles
  - Cyanosis
  - Signs of exhaustion such as somnolence, confusion, and seizure

1.1 When to refer?

- Persistent hypoxemia/respiratory failure requiring ventilation
- Presence of shock/multiorgan dysfunction

1.2 How to refer?

- Secure airway, breathing and circulation
- Ensure resuscitation kit and a BLS trained provider during transport
- Child's peripheral venous access should be secured and patent
- Child's details, history, examination findings, vitals, events which occurred and treatment given should be legibly written in the referral letter
- The parents should be counselled regarding the child's condition and need for referral
- Written, informed consent should be taken from the parents/guardian's prior to referral

2. COMMUNITY ACQUIRED PNEUMONIA

2.1 Learning objectives

After completion of this section, the participants should be able to

- Identify a case of community acquired pneumonia (CAP)
- Grade severity and stabilize
- Assess the etiology and risk factors
- Specific management of all cases of pneumonia
- Identify causes of non response
- Know when and how to refer?

2.2 What is CAP? How to identify?

- Any child presenting with fever, cough and difficult or rapid breathing should be considered as a case of pneumonia (WHO definition)
- Tachypnea is defined with age specific cut offs (refers box 2.1)
- CAP should be differentiated from other common respiratory illnesses such as

BOX 2.1: WHO Age specific criteria for tachypnea		
Age	Approximate normal respiratory rate (breaths/min)	Tachypnea threshold (breaths/min)

Onchiolitis and upper airway infections.

2-12 Months	25-40	50
1-5 Years	20-30	40
>5 Years	15-30	30

BOX: - 2.2 Risk factors for community acquired pneumonia

- Malnourished state
- Age< 6 months
- Post-measles state
- Absence (or inadequate) breastfeeding
- Solid fuel use

2.3 Clinical features and etiology

The symptomatology and etiology is determined by the age of the patient (refer box 2.3)

BOX 2.3: Age-wise clinical features and etiology of pneumonia

	Neonates	Infants	Preschool Children
Symptoms	Hypothermia Poor feeding Irritability Fever and cough may be absent	History of antecedent URI Cough prominent	History of antecedent URI Cough Post-tussive vomiting Chest pain, if

			pleural involvement Abdominal pain, if lower lobe involved
Signs	Tachypnea, grunting, nasal flaring, retractions	Tachypnea, grunting, and retractions	Tachypnea, grunting, retractions and chest pain
Etiology	Group Streptococcus Escherichia coli Klebsiella Cytomegalovirus	**Febril:** Respiratory syncytial virus S. Pneumoniae H. influenza (type b) Staphylococcus aureus, Mycoplasma Pneumonia **Afebrile:** Chlamydia Trachomatis Mycoplasma hominis Cytomegalovirus	Respiratory viruses, S. Pneumoniae H. Influenzae (type b) Staphylococcus aureus Mycoplasma Pneumoniae Chlamydia pneumonia, Group A Streptococcus

2.4 How to assess severity?

The management of pneumonia is guided by severity of disease (Box No. 2.4)

BOX 2.4 WHO severity criteria (6 months – 5 years)	
Cough and cold (No pneumonia)	No fast breathing No chest in drawings
Pnuemonia (Not severe)	Fast breathing (>age specific threshold) No chest is drawings
Severe pneumonia	Tachypnea + chest indrawing + danger signs (lethargy, refusal to feed, convulsion, central cyanosis, severe malnutrition, grunting)

2.5 How to investigate?

Hemogram with total and differential leukocyte count

CRP – bacterial pneumonia> viral pneumonia

Blood culture: Positive in 10%-20% of children with pneumonia, may be done if available

CSF (if feasible) in case of

Newborn

Infants presenting with altered sensorium

Seizures

Irritability out of proportion to illness

Chest X ray (Refer Box 2.4)

BOX 2.5: Indication for chest X-ray

- When the diagnosis is in doubt (bronchiolitis, asthma, developmental malformation, foreign body inhalation, aspiration pneumonia)
- Asymmetrical findings on chest examination
- Suspected complications of pneumonia (pleural effusion, empyema, lung abscess)
- Know case of recurrent respiratory illness (asthma, cystic fibrosis, immunodeficiency disorders)
- Severe pneumonia

Arterial blood gas: Not routinely indicated. If available in a given set up then can be done for following indications

Box 2.6: Indications of ABG

- Sever pneumonia
- Hypoxemia on pulse oximentry (SpO2 <94% on 40% FiO20
- Cyanosis

2.6 Steps in the management of pneumonia
Grade the seventy and classify (Refer Box 2.4)
Resuscitate and stabilize ABC

- Airway: Maintain open and stable airway; suction if secretions are present. Indications of intubation (Refer box 2.7)
- Breathing: start oxygen by any delivery device, depending on availability free flow oxygen/nasal prongs, at flow rate 1-5L/min (depending on age) if child has lower chest wall, indrawing or SpO2≤94%.
- Circulation: Maintain normal circulatory parameters – correct dehydration; use vasoactives if there is shock despite adequate fluids; think of septic shock

Supportive care

Hydration (intravenous or nasogastric tube feed)

If oral acceptance is good – allow orally

Oral acceptance poor (but feeding not contraindicated) – nasogastric or orogastric feed

Severe distress (feed contraindicated): Start fluids 0.45 Saline in 5% dextrose as 2/3rd to 3/4thmaintainance

Check blood glucose by dextrostix. Correct hypoglycaemia and maintain euglycemia (blood glucose between >60mg/dl – 100mg/dl

Treat electrolyte imbalance (refer section E, Chapter 3,4)

Treat fever with oral paracetamol 15mg/kg/dose, can be given 6 hourly if need be

Box 2.7 Indications of endotracheal intubation

- Children with marked respiratory distress despite adequate FiO2
- Gasping, bradypneic or apneic
- Cyanosis with decreased respiratory efforts
- CNS signs of hypoxia (lethargic, unresponsive, obtunded, seizures, coma)
- Cardiovascular signs of hypoxia (marked tachycardia, bradycardia, or hypotension
- Children who worsen on CPAP or other non invasive respiratory support

2.7 Antibiotics

Choice of Antibiotic therapy is guided by multiple factors (Refer Box 2.6)

BOX 2.8 Guidelines for antibiotic choice

- Age of the child (refer to box 2.3)
- Severity of pneumonia (refer to box 2.4)
- Associated clinical features suggesting specific eitology e.g. pustules suggesting staphylococcal infection
- Immune-suppressed or immunocompromised state (such as PEM, post-measles state, chronic steroid therapy)
- Underlying chronic lung disease e.g. cystic fibrosis
- Radiographic pointers towards a specific etiology
- Presence of complications such as pneumothorax/empyema

- Pneumonia (non severe) can be treated at home with oral antibiotics in most cases
  - Amoxicillin (40mg/kg/dose) in 2 divided doses for 3-5 days
  - Follow-up after 48 hours to reassess for improvement (general well being, respiratory rate, retractions, appetite)

Severe Pneumonia

  - IV ampicillin (50mg/kg/dose,max 12gm/day) 6 hourly + IV gentamicin 7.5 mg/kg/dose 24 hourly
  - Add cloxacillin (100-200mg/kg/day in 4 four divided doses) if clinical & radiographic features suggest staphylococcal infection (pustules, post- measles

state, severe malnutrition, empyema, pneumatoceles, necrotizing pneumonia or air leaks)
  - Add azithromycin if CXR suggests atypical pneumonia
  - Reasess after 48 hours

2.8 How to proceed after reassessment at 48 hours of therapy?

  - In case of (non severe) pneumonia hospitalise urgently if danger signs appear (inability to suck/drink, impaired sensorium, convulsions, grunting, cyanosis)
  - If no danger signs but persistence of difficulty in breathing then
  - Change to amoxicillin-clavulanic acid (80-90 mg/kg of amoxicillin) in 2 divided doses for 5 days or
  - Add azithromycin 10 mg/kg for 5 days if features suggest atypical pneumonia
- In severe pneumonia
  - If improved and patient is able to take orally then shift to oral amoxicillin and continue for 5 more days
  - If not improved or deteriorated: go over a check list of non- response (refer box 2.9)
  - If above are tackled then upgrade antibiotic to IV ceftriaxone (100 mg/kg/day, max 4gm) 12 hrly
  - Change antibiotics as per cultures

2.9 How to monitor?

- Child should be monitored by nurses every hourly and by doctors at least twice a day
- Look for heart rate, respiratory rate, retractions sensorium, SPO2 and circulatory parameters

2.10 What is non response and how do we identify?

- Persistently raised respiratory rate at 48 hours
- Danger signs at any time during the illness, such as inability to suck/drink, impaired sensorium, conculsions, grunting, cyanosis.
- Causes of non response are enumerated in Box 2.7

BOX 2.9 : Causes of Non response

- Asthma/reactive airway disease/ wheeze associated LRTI
- Underlying cardiac disease
- Underlying obstructed bronchus or collapse
- Foreign body
- Pulmonary malformation
- Thrombophlebitis
- Inadequately drained source e.g. empyema
- Incorrect choice and dosing of antibiotics e.g non-staphylococcal cover for post measles pneumonia

2.11 When to refer?

- Severe pneumonia
- Hypoxemia/respiratory failure requiring ventilation
- Presence of shock/multiorgan dysfunction

2.12 How to refer?

- Secure airway, breathing and circulation
- Ensure resuscitation kit and a BLS trained provider during transport
- Child's peripheral venous access should be secured and patent
- Child's details, history, examination findings, vitals, events which occurred and treatment given should be legibly written in the referral letter
- The parents should be counselled regarding the child's condition and need for referral
- Written, informed consent should be taken from the parents/guardian's prior to referral

Protocol for management of CPAP

Identify case of pneumonia (symptoms & signs)
Rule out other causes

Assess severity
WHO classification

Pneumonia

Treat at home with oral antibiotics
Amoxicillin (40 mg/kg/dose0 in 2 divided doses for 3-5 days
Reassess after 48 hrs for General danger sings (inability to suck/drink, impaired sensorium, convulsions grunting, cyanosis) if present hospitalise urgently
If child has persistent tachypnea but no indication for admission

- Change to amoxicillin-clavulanic acid (80-90 mg/kg of amoxicillin) in 2 divided doses for 5 days if features suggest atypical pneumonia.
- Advise to return immediately if the child develops any danger signs

Sever Pneumonia

Resuscitation and stabilisation
A: Maintain open and stable airway; Suction secretions
B: Start oxygen by any delivery device, depending on availability, patient's severity, demand and familiarility of providers. Maintain target SpO2> 94%
C. Maintain normal circulatory parameter – correct dehydration; use vasoactives if there is shock despite adequate fluids; think of septic shock

** Antibiotics in severe pneumonia

- IV ampicillin (50 mg/kg/dose) 6 hourly + IV ge

Supportive treatment:
First dose of antibiotics ** (as early as possible; preferably after obtaining a sample for blood culture)
Hydration (intravenous or nasogastric tube feed)
If oral acceptance is good – allow orally
Oral acceptance poor (but feeding not contraindicated) – Nasogastric or orogastric feed
Severe distress (feed contraindicated); Start fluids 0.45 saline in 5 % dextrose as 2/3rd to 3/4th maintenance

- Maintain euglycemia (blood glucose)
- Treat electrolyte imbalance

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features suggest staphylococcal infection (pustules, post–measles state, severe malnutrition, empyema, pneumatocles, necroti

zing pneumonia or air leaks)
- Add azithromycin if CXR suggests atypical pneumonia
- Monitor and assess after 48 hours

If improved and

patient able to take orally then shift to oral amoxicillin for 5 more days
If not improved in 48 hours or deteriorated, check list of non response (box 2.9)
If above are tackled, upgrade to IV ceftriaxone
Change antibiotic as per cultures

- Monitor and assess after 48 hours

If improved: complete 10-14 days of antibiotics

3. ACUTE EXACERBATION OF ASTHMA

3.1 Learning objectives

After completion of this section the participants should be able to

- Identify a case of acute exacerbation of asthma
- Assess the severity based on clinical evaluation
- Initiate appropriate bronchodilator therapy to relieve airway obstruction
- Identify red flag signs of acute severe asthma
- Initiate appropriate respiratory support
- Monitor therapeutic response
- Know when, how to refer and precautions before referral

3.2 What is acute exacerbation of asthma?

- Acute exacerbation of asthma is defined as episodes of coughing (particularly in the night/early morning), wheezing, and/or breathlessness (with or without fever) due to diffuse inflammation and airflow obstruction of the lower airways. This is reversible either spontaneously or with treatment.
- Acute severe asthma is defined as a severe asthma exacerbation that does not respond to repetitive or continuous administration of inhaled short-acting β2 – adrenergic receptor agonists (SABAs) in an emergency setting

3.3 How to identify an acute attack of asthma?

- Family and past history of reactive airway disease/atopy/eczema
- Correlation with triggers: dust, mold, specific food, animal hair
- History of recurrent symptoms, seasonal predilection
- History of cough, fever, respiratory distress, noisy breathing, whistling sounds
- Therapeutic response to bronochodilators
- Differential diagnosis as given in Box 3.1

Box 3.1 Differential diagnosis

- Wheeze associated lower respiratory tract infection

Bacterial pneumonia: usually a sick looking child, with moderate to high grade fever, bronchial breathing and crepitations

Viral Pneumonia: associated with an upper respiratory prodrome

- Congestive cardiac failure (secondary to myocarditis or congenital heart disease): associated with suck-rest-suck cycle, sweating, tachycardia, hepatomegaly, basal crepitations, murmurs and cardiomegaly on chest radiography.
- Bronchiolitis: As described in section B, Chapter 4
- Anatomic and functional abnormalities : will cause recurrent episodes of wheezing

Extrinsic airway anomalies: vascular ring/sling

Intrinsic airway anomalies: congenital lobar emphysema, lung sequestration,

cystic adenomatoid malformation
Gastroesophageal reflux

- Foreign body: Sudden respiratory worsening, unilateral findings with or without history of choking
- Mucociliary clearance disorders: Recurrent episodes of wheezing with failure to thrive
  - Cystic fibrosis
  - Primary ciliary dyskinesia

3.4 How to grade severity?

- Severity of asthma can be graded by the clinical Assessment severity Score (CASS) (refer box 3.2). This score should be used for initial assessment and to monitor response to therapy

Box 3.2 Clinical Asthma Severity Score (CASS)

Score	RR	Room air SpO2	Asuscultation (wheeze)	Retractions	Dyspnea
0	<30	97-100	None	None	None
1	31-45	94-96	End expiration	+/-	Full sentences
2	46-60	91-93	All expiration	++	Partial sentences
3	>60	<91	Inspiration & Expiration	+++	Single words/ grunts

Maximum score is 15; mild <4, moderate 4-7, severe >7

3.5 Steps in management?
Grade severity (Refer Box 3.2)
Start oxygen
Maintain SpO2 >94%
Use humidified oxygen via free flow, nasal prongs or through face mask @6L/min
Supportive care
Encourage oral fluids in mild distress
Give restricted fluids (80%) in moderate and severe cases
Treat pyrexia with oral paracetamol 15 mg/kg/dose
Start initial nebulizations
Salbutamol (<5yrs – 2.5mg, >5yrs – 5mg) every 20 min, 3 times OR MDI salbutamol (100µg/puff) 6 puffs with spacer with/without mask (if the child can use the MDI)
Budesonide (800µg/dose) 3 times every 20 mins
Ipratropium 250µg/dose or 2 puffs (80µg/puff) 3 times every 20 mins
Mix all three in the nubulizations chamber and dilute in normal saline of required to make a volume of 3-4 ml and give it within one hour

Steroids: indications of steroids are enumerated in Box 3.3
Oral: prednisolone 02 mg/kg/day x 5 days OR
IV hydrocortisone 10 mg/kg followed by 5 mg/kg 6 hrly x 5 days

Box 3.3 Indications of steroids
Acute severe asthma
Previous history of life-threatening attack or severe attacks requiring System steroids
Child on regular oral steroids or high dose inhaled steroids

3.6 How to monitor?
Child should be monitored by a nurse atleast every 1 hourly and by a doctor atleast 4 hourly
Monitor CASS score (Refer Box 3.2)

3.7 How to define adequate or no response? (Refer Box 3.4)
Box 3.4: Response to treatment

Adequate response (improving)	No response (worsening)
No distress/dyspnea Response sustained 60 mins after the last treatment No wheeze SPO2 > 94 % No pulses paradoxus	HR/RR – increase Persistent dyspnea Decreased air entry Accessory muscle usage severe SPO2 < 94 % Pulses paradoxus > 15mmHg

3.8 How to escalate therapy in non-responders to initial nebuliations?
In case of severe asthma not responding to initial therapy alternative drugs may be required
Magnesium sulphate: Causes bronchodilation and decreases neutrophilic burst associated with inflammation
Dose: Intravenous infusion: 50 mg/kg/dose in 30 ml NS with 5 % dextrose
Over 30 min, can be given 6 hrly (maximum 4 doses)
Monitor adequate urine output
Aminophylline: increases endogenous catecholamine release an has anti-inflammatory action
Dose: 5 mg/kg IV loading f/b 0.9mg/kg/hr infusion
Side effects: Tachycardia, hypokalemia
Terbutaline: Beta agonist
Dose: 10 mcg/kg loading dose followed by 0.1 mcg/kg/min continuous infusion (can be hiked every 30 min 0.1-0.2 mcg/kg/min max 10 mcg/kg/min
Side effects: Hypokalemia, arrhythmia, hypotension and myocardial ischemia.

3.9 How to investigayte?
Laboratory studies are generally not indicated in a routine acute exacerbation, unless child is unusually ill with suspicion of infection

Chest radiographs are nor also routinely indicated except as outlined in box 3.5
Arterial blood gases (if available)

Box 3.5 indications of CXR
If clinical examination suggests the possibility of pneumothorax or pneumonia
Suspecting other causes of wheeze such as airway foreign body, anatomic abnormality etc.

3.10 When to give antibiotics?
Not routinely indicated except in following conditions
High grade fever without any signs of viral prodrome
Chest radiograph showing a patch of consolidation
Which antibiotics to be given? (Refer section on pneumonia)

3.11 What is the check list in case of non-responders?
Think of alternative diagnosis (Box 3.6)

Box 3.6: Non responders
Foreign body inhalation
Anatomic abnormalities
Underlying cardiac disease
Secondary bacterial infection
Wheeze associated lower respiratory tract infection
Gastro oesophageal reflux disease
Interstitial lung disease

3.12 Whom to admit? (Refer box 3.6)

Box 3.7 Indications of admission
Severe distress as per CASS (refer Box 3.2)
Inadequate or no response to supportive first hour treatment in mild or moderate cases
Requiring > 10 puffs of salbutamol
Suspicion of alternative diagnosis

3.13 When to refer?

Any child with signs of life-threatening attack
Poor or no response to therapy in emergency department
Development of signs of respiratory fatigue

3.14 How to refer?

Refer only after stabilization, ensuring adequate airway, breathing and circulation
Proper documentation regarding the presentation of the child, details of resuscitative measures taken, interventions done during resuscitation and reasons for referral including name and contact number of referring physician.

A doctor/paramedic trained in Pediatric Advanced Life Support/Basic Life Support to accompany the patient

Transporting ambulance should have sufficient O2 supply and Resuscitation equipment

Inform the referral centre prior to sending the patient regarding the diagnosis of the child, indication for referral, current status and approximate time to arrival

Counsel the family of the child regarding the need for transfer and risks during transfer. Obtain written, signed consent for the same.

Remember:

Red flags:

An anxious asthmatic child indicates severe obstruction and probably hypoxia

Silent chest indicates inadequate air exchange and severe airway Obstruction

Unilateral diminished breath sounds (in a case of asthma) indicates severe obstructive atelectasis or a pneumothorax, and needs urgent attention.

A calm patient usually denotes, at the most, mild distress

Do not interrupt oxygen supply during nebulisation

CXR is not routinely indicated

Intubation is seldom required in asthma. Positive pressure ventilation can worsen hemodynamic and respiratory parameters in lower airway obstruction. Hence decision should be taken cautiously

4. ACUTE BRONOCHIOLITIS

4.1 Learning objectives:

After completion of this section, the participants should be able to

Identify a case of acute bronochiolitis

Categories severity

Rule out other causes of wheezing in an infant

Initial stabilization and other therapies

Familiarize with non-invasive respiratory support modalities like bubble CPAP and high flow Nasal cannula (HFNC)

Monitor for response

Know when and how to refer and what precautionary measure to taken before referral?

4.2 What is Bronchiolitis?

- It refers to inflammation of peripheral small airways
- It is the most common cause of wheezing in young infants
- Smaller children are more prone to wheezing due to following reasons:
- In children <5 yr old, small-calibre peripheral airways contribute up to 50% of the total airway resistance hence even a marginal (slight) additional narrowing causes further flow limitation and wheeze.
- With the very complaint newborn chest wall, the inward pressure produced in expiration subjects the intrathoracic airways to collapse.
- Differences in tracheal cartilage composition and airway smooth muscle tone, further increase airway resistance in comparison to older children.
- Infant who presents with mild to moderate grade fever, upper respiratory symptoms, with wheeze and respiratory distress, without previous such episodes, bronchiolitis is the most likely diagnosis.

Box 4.1 Differential diagnosis:
First episode of asthma: family history or past history of atopy (nasal allergy, atopic dermatitis, eczema or asthma) and good response to bronchodilators
Wheeze associated lower respiratory tract infection:
Bacterial Pneumonia : Usually a sick looking child, with moderate to high grade fever, bronchial breathing and crepitations
Viral Pneumonia: Associated with an upper respiratory prodrome.
Congestive cardiac failure (secondary to myocarditis or congenital heart disease): Associated with suck-rest-suck cycle, sweating, tachycardia, hepatomegaly, basal crepitations, murmurs and cardiomegaly on chest radiography.

4.3 How to assess severity?

The severity can be assessed by the modified Respiratory Distress Assessment (RDAI) Score.

Box 4.2 Modified Respiratory Distress Assessment (RDAI) Score

Mild: 0-4, Moderate: 5-8, Severe: 9-12

Clinical parameter	Score 0	Score 1	Score 2	Score 3
Respiratory rate (per min0	< 40	40-60	60-70	>70
Use of accessory muscles	None	1 accessory muscle used	2 accessory muscles used	3 or more accessory muscle used
Colour/Cyanosis	No cyanosis in room air/pink in room air	Cyanosed when crying	Cyanosed in room air/Pink with oxygen	Cyanosed with oxygen or cardio-respiratory arrest
Auscultatory findings	Normal	Decreased air entry, no rhonchi	Decreased air entry, rhonchi heard	Silent chest

4.4 Treatment of Bronchiolitis:

- Grade severity (refer box 4.2)
- Start oxygen

  - Maintain Spo2>94%. Continue oxygen until no signs of hypoxemia are present (lower chest retractions, tachypnea for age)
  - Use humidified oxygen: Free flow/Nasal prongs/oxygen hood/non rebreathing mask depending on severity, availability and patient's tolerance
  - In moderate and severe distress: Preferable to five some PEEP by using:
  - Indigenous bubble CPAP/high flow nasal cannula. PEEP helps to splint the small airways and keep them patent (refer appendix)

- Supportive Care:
  - Encourage breastfeeding and oral fluids in mild distress
  - Give resitricted fluids (80%) in moderate and severe cases
  - Treat fever with oral paracetomal 15mg/kg/dose
  - Ensure regular and gentle suctioning if any thick secretions present in nose and mouth

- Nebulizations

  - Hypertonic (3%) saline decreases airway inflammation, mucus plugging thus improving the mucus clearance.
  - Dose : 4 ml of hypertonic saline (3%) without dilution
  - Nebulized epinephrine 3-5 ml (1:1000) without dilution has also been shown to improve symptoms
  - Hence a trial of both drugs should be considered but there is nor role of round the clock nebulisations

- Continue oxygen during nebulisation at a flow rate of 6-8 L/min.
- Monitoring: (Refer box 4.3)
- Child should be monitored by a nurse at least every 3 hourly and by a doctor at least twice a day
- Look for respiratory rate, any retractions, sensorium SPO2

Box 4.3 How to judge response:
Decrease in respiratory reate/work of breating
Decrease in heart rate
SpO2 ≥94%

Non responders: (Refer Box 4.4)

Inadequate oxygen delivery; Device malfunction or improper oxygen supply via cylinder or central source
Inadequate drug delivery: Inappropriate dose/inadequate delivery
Associated pathology
Multiple small atelectasis (collapse) on CXR
Secondary bacterial infection: sick child
First episode of asthma: family history of atopy, may require a trial of bronchodilators
Underlying heart disease and congestive cardiac failure

When to intubate a case of bronchoiolitis?
Indications for intubation in bronchiolitis are
Severe respiratory distress not maintaining saturation on CPAP or HFNC
Patient progressing to respiratory failure.

When to five antibiotics to a case of bronchiolitis?
Prolonged illness of >7 days (usually bronchiolitis improves within 3-4 days)
Persistent high grade fever > 48 hrs.
Inadequate response to therapy requiring escalation of respiratory support
Chest x-ray suggestive of lobar consolidation, necrotizing lesions

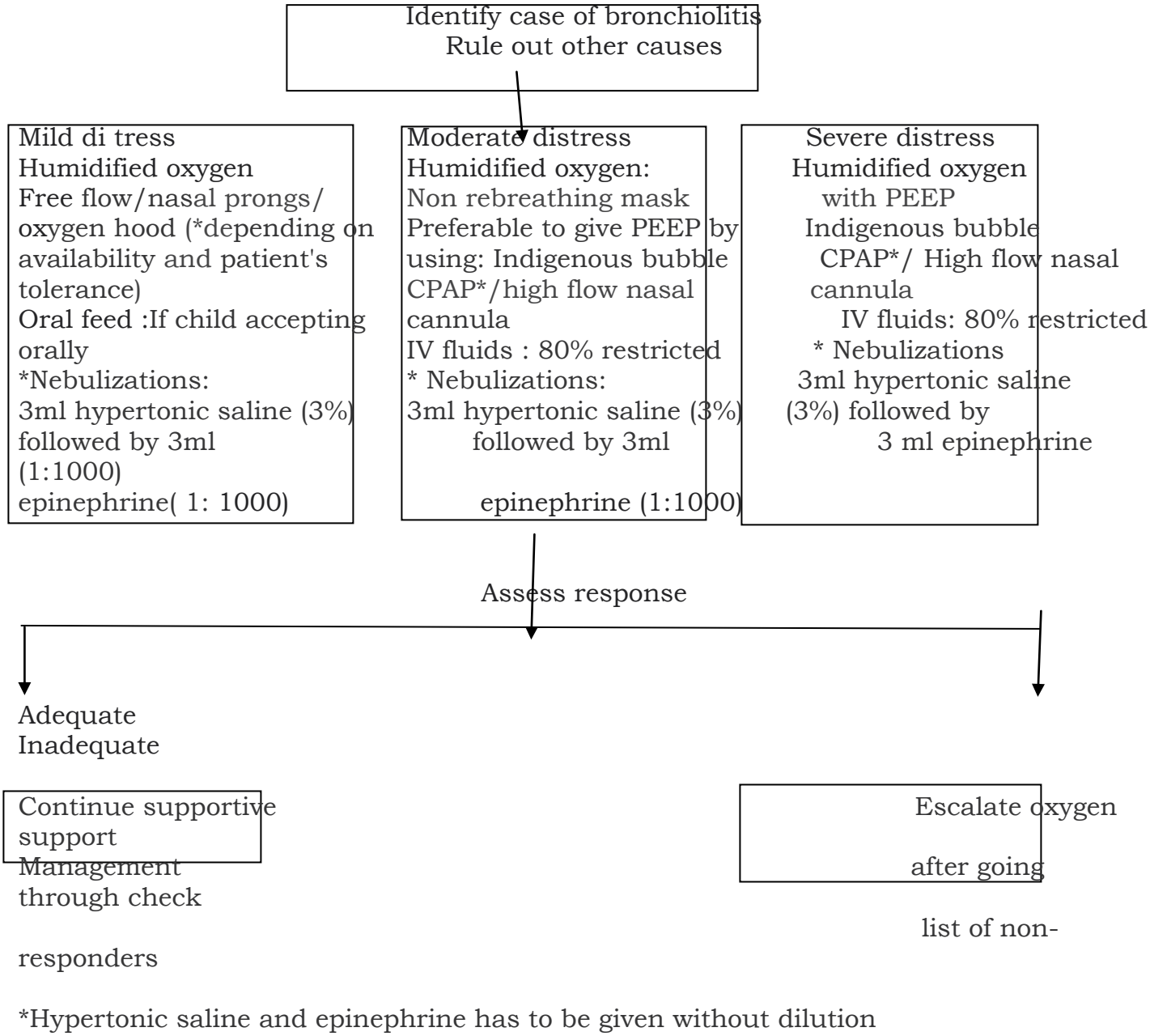
Which antibiotics to give?
Refer section B, Chapter 2
4.5 When to refer?
• No improvement with supportive treatment
• Ventilator support required

4.6 How to refer?
• Child' peripheral venous access should be secured.
• Child's details, history, examination findings, vitals, events occurred and the treatment given should be legibly written in the referral letter.
• Parents should be counseled regarding patient's condition
• A person trained in BLS should accompany the child during referral.
• If child is being referred for ventilator support, then it is preferable to intubate and transport

Remember:
• Anxiety/ tachycardia may be due to hypoxemia; Do not sedate
• Humidification gasesis critical
• Nebulization with epinephrine can be given maximum twice" should not be given round the clock '
• Do not interrupt oxygen supply while nebulizing
• No role of antibiotics in Managementof simple bronchiolitis
• Non invasivemodes of respiratory support like CPAP or HFNC are very useful and help avert intubation

Positive pressure ventilation can worsen hemodynamic and respiratory parameter in lower airway obstruction. Hence, the decision to intubate should be taken cautiously.

Protocol for management of acute bronchiolitis



*Hypertonic saline and epinephrine has to be given without dilution
Continue oxygen at flow rate of 6-8I/min during nebulization.

PROTOCOL FOR MANAGEMENT OF A CASE OF ACUTE EXACERBATION OF ASTHMA

Identify case of acute exacerbation of asthma & grade severity (refer box 3.2)

Mild Case

Humidified oxygen: Free flow or through face Mask 6L/min
*Nebulization:
Salbutamol (0.15 mg/kg) diluted in 3 mL NS, every 20 min, 3 times or MDI salbutamol (100 µg/dose) every 20
2 times every 20 mins mins, 3 times withing 1 hr OR MDI
Steriods:
Oral: Prednisolone 2 mg/kg/day in 2 divided doses x 5 days
times

mg/kg/day in 2

f/b 5mg

Moderate or severe case

Admit the patient
Humidified oxygen: Free flow or through face mask 6 L/min
*Nebulization:
Salbutamol (0.15mg/kg) I
Salbutamol 6 puffs with spacer and mask (if child can use MDI)
Budesonide (800 µg/dose) 3
every 20 mins
Iptatropium 250 µg/dose 3 times every 20 mins
Steroids:
Oral: Prednisolone 2
divided doses x 5 days
OR
IV hydrocortisone 10mg/kg
Per kg 6 hrly x 5 doses

Assess response at 1hour : RR, SpO2 , pulses paradoxus, CASS score

Improving

Mild

- Discharge the patient
- Continue steroids, if salbutamol
- Initiated (5 days)hrly
- Use salbutamol as
- Needed
- sulphate
- Patient education

Moderate

Repeat salbutamol 1 dose & give 2 doses 2

Observe for 6 hrs & discharge if improved

~~If not improved then~~ treat like

Worsening

Continue oxygen
Continue inhaled

Continue systemic steroids
Monitor parameters
IV magnesium

(50%) 50 mg/kg/dose in 30

- Follow up after 48 hr

Severe

ml NS with 5% dextrose

Over 30 min, can be given 6 hrly (maximum 4 doses)

Monitor adequate urine output

worsening
Indications for intubation:

- Severe hypoxemia
- Respiratory fatigue
- Rapid deterioration in the
- Child's mental state
- Cardiopulmonary arrest

Shift to PICU
Continue inhaled Salbutamol continuously
Continue Magnesium sulphate 6 hrly max 4 doses
Consider IV aminophylline 5 mg/kg IV loading f/b 0.9/kg /hr infusion OR
IV ternutaline 10 mcg/kg loading dose f/b 0.1 mcg/kg/min

Continuous infusion (can be hiked every 30 min 0.1 –0.2

Mcg/kg/min max 10 mcg/kg/min
If no response then consider intubation and ventilation

*mix all three in the nebulization chamber and dilute in normal saline if required to make a volume of 3-4 ml and give it within one hour

5. ACUTE UPPER AIRWAY OBSTURCTION

5.1 Learning objective

After completion of this section, the participants should be able to

- Identify a case of acute upper airway obstruction (UAO)
- Initial assessment and severity classification
- Immediate stabilization measures
- Etiology and differential diagnosis of UAO
- Specific management of coup
- Management of other causes of acute UAO
- Know when and how to refer and what precautionary measure to take?

5.2 **How to identify a case of acute upper airway obstruction in ER?**

- Upper airway obstruction is defined as obstruction in the respiratory tract above the level of vacal words.
- Clinical clues to upper airway obstruction include
- Stridor
- Suprasternal supraclavicular retractions
- Drooling of salive
- Dysphagia
- Change in voice
- Extension of neck in very small infants

5.3 **How to score severity of UAO?**

The severity of UAO can be assessed by using scoring systems. Though the croup score was devised originally for patients with croup, it guides in the monitoring and management of other causes of UAO also (refer box 5.1)

Box 5.1 Clinical score for croup severity

Features	**Mild**	**Moderate**	**Severe**	**Impending respiratory failure**
Barky cough	Occasional	Frequent	Frequent	Often not prominent due to fatigue
Stridor	None of minimal at rest	Easily audible to rest	Prominent inspiratory and occasional expiratory	Audible at rest but may be quiet or hard to hear
In drawing suprasternal and/or intercostal	None to mild	Visible to mild	Visible at rest	May not be marked
Distress/agitation /lethargy (CNS hypoxia)	None	None to limited	Substantial lethargy may be present	Lethargy or decreased level of consciousness
Cyanosis	None	None	None	Dusky or

				cyanotic without supplemental oxygen

** Chan A, Langley J, Leblanc J. Interobserver variability of croup scoring in clinical practice. Pediatric Child Health. 2001:6 (6) : 347-51.

5.4 Management

- Initial stabilization: ABCD approach
- Keep the child in a position of maximum comfort (mother's/caregiver's lap in infant or sitting position in older child)
- Administer oxygen without causing agitation preferably by blow by method (a plastic tube held by the mother within a few centimeters of the baby's nose and mouth
- If child is unable to maintain a patent airway then intubate (refer box 5.2)
- Maintain normal hemodynamics by fluids and vasopressors
- Monitor sensorium to identify signs of hypoxia
- Identify the cause and manage accordingly (points 5.5 and 5.6)

Box 5.2 Indication of intubation

- Marked tachypnea
- Cyanosis with decreased respiratory efforts
- Decreased mental status (lethargic, unresponsive)
- Bradypnea to apnea, in severe cases poor or absent air entry
- Cardiovascular signs of hypoxia (marked tachycardia, bradycardia, or hypotension)

Box 5.3 Remember

- Perform immediate intubation in cases of impending airway obstruction as delay may lead to complete obstruction
- When in doubt it is always better intubate.

5.5 DON'T'S of upper airway obstruction

- Insert tongue depressor
- Attempt intravenous access till airway is secure
- Prone position
- Agitate child with nasal prongs or mask
- Sedate and anxious child
- Imaging for establishing diagnosis

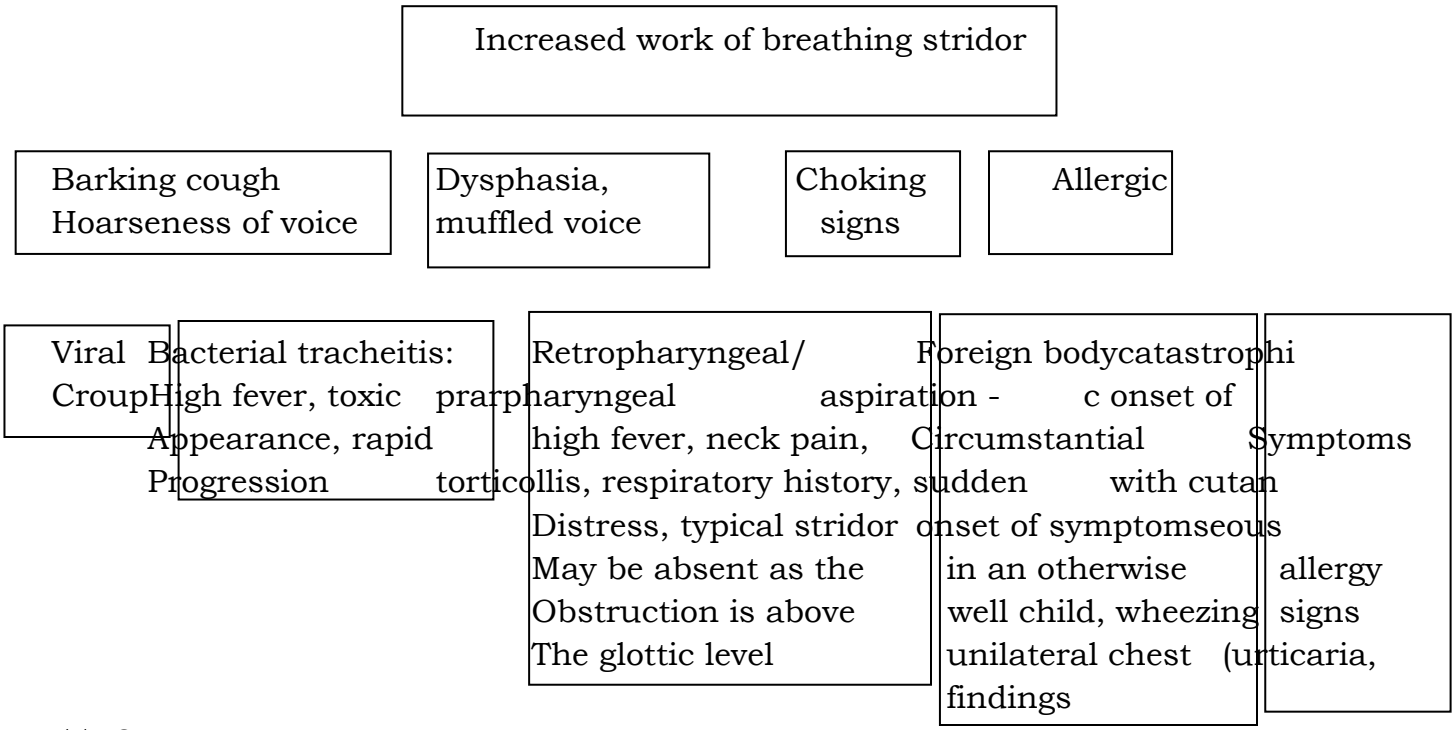
- Transport for any investigations before airway is secure

5.6 Etiology and differential diagnosis

- Causes of acute upper airway obstruction
- Viral croup
- Bacterial Tracheitis
- Epiglottitis
- Retropharyngeal/parapharyngeal abscess
- Foreign body aspiration
- Acute anaphylaxis

- Differential diagnosis of acute upper airway obstruction in enumerated in fig. 1

Figure 1 – Causes of acute upper airway obstruction



5.7 Croup

After initial stabilization, assess the severity (refer box 5.1)

Specific management includes corticosteroids and nebulized epinephrine

Corticosteroids

Dexamethasone should be given to all cases of croup (mild to severe)

Dose: 0.6mg/kg/oral/im/iv single dose (maximum 8 mg), dose may e repeated once more in severe cases

Improvement generally begins within 2 to 3 hours after a single oral dose of dexamethasone and persists for 24 to 48 hours

Nebulished Epinephrine

Recommended for moderate – severe croup

Dose ; 3-5 ml of 1:1000 adrenaline without dilution

Clinical response is sustained for at least 1 h and wanes off by 2 h of administration

Repeat dose is indicated if respiratory distress persists

- Whom to admit?
- Impeding respiratory failure
- Moderate and severe croup
- When to discharge?
- Observation of 3-4 hours is required to see for recurrence of symptoms
- During observation child should not have stridor at rest or chest wall indrawing

5.8 Specific treatment of other causes of UAO

Diagnostic tests should not precede initial stabilization

Bacterial tracheitis	Antibiotics are mandatory IV Ceftriaxone + IV Cloxacillin for 10-14 days Bronchoscopy may be needed in severe cases
Deep neck abscess	Antibiotics Ceftriaxone + Cloxacillin + Clindamycin Referral to higher centre for imaging and source control
Foreign body	Referral to higher centre for immediate bronchoscopic removal Remember do and don't during referral
Acute anaphylaxis	Correct hypotension Place in Trendelenburg position Give IM epinephrine 0.1 ml/kg (1:1000 dilution) immediately And repeat 2-3 times every 5-15 minutes if needed Nebulise with salbutamol for bronchospasm Cosider Hydrocortisone 10mg/kg (max 100 mg)

5.9 When to refer?

- Patients requiring mechanical ventilation
- In croup, if no improvement after two doses of steroid consider alternative diagnosis
- For imaging and bronchoscopy

5.10 How to refer?

- Secure airway, breathing and circulation
- Ensure resuscitation kit and a BLS trained provider during transport
- Child's peripheral venous access should be secured and patent
- Child's details, history, examination findings, vitals, events which occurred and treatment given should be legibly written in the referral letter

- The parents should be counselled regarding the child's condition and need for referral
- Written, informed consent should be taken from the parents/guardian

- **Remember DON'T'S in UAO**

Protocol for management of acute croup

Identify a case of Croup

- Initial stabilization
- Place child on parent's lap
- Provide position of comfort
- Provide free flow oxygen if required

Assessment of severity (refer box 5.1)

- Immediate airway management in impending respiratory failure

Mild

Give oral Dexamethasone 0.6 mg/kg

Send home
Educate parents
About danger signs

Moderate

Nebulized adrenaline (1:1000) 3-5 mL once
Five oral/im Dexamethasone (0.6 mg/kg)

Improved
Observe for 4-6 hrs
Discharge if stable

Severe

No improvement/ worsening
Repeat neb adrenaline (1:1000) 5 ml once
Repeat Dexamethasone single Dose

If still no improvement think of Alternative cause

Pediatrics trauma at primary care level

1.1 Learning objectives

After completion of this section, the participants should be able to

- Perform primary survey of trauma patients
- Initiate resuscitation and stabilization
- Identify the system involved by focused secondary assessment
- Initiate systematic targeted approach in management
- Identify patients who can be managed at primary health care level
- Identify patients who need early referral
- Know how and when to refer?

1.2 How to do initial assessment in the emergency room?

Primary survey: ABCDE approach

Box 1.1 Initial assessment

Airway	Assess the following parameter • Position of the tongue • Stridor yes or no • Trauma to the airway/facial trauma • Secretions/blood/foreign body/dislodged teeth
Breathing	Assess the following parameters • Respiratory rate • Pattern of breathing • Retractions and work of breathing • Auscultate for differential air entry and added sounds like wheeze, stridor, grunt • SPO2 • Chest wall injuries/contusion/rib fracture/flail chest
Circulation	Assess the following parameter • Heart rate • Central and peripheral pulses • Blood pressure

	• Temperature of peripheries • Any active bleeding
Disability	Assess sensorium using Glasgow coma scale Look for focal neurological deficit (refer appendix)
Exposure	Look for any external bleeding, pallor, visible fractures, spinal and neck injury

1.3 What are the steps in initial stabilization?

Airway	• Position patient on a firm surface • Assume cervical injury in every case of trauma and stabilize-spine (refer box 1.2) • Do not use head tilt, chin lift to open airway, use jaw thrust maneuver instead • Suction secretions if present • Insert oropharyngeal airway to prevent tongue fall • Remove visible foreign bodies/dislodged teeth • If above measures fail then intubate (refer box 1.3)
Breathing	• Provide adequate oxygen support (preferable non-rebreathing mask) • Identify tension pneumothorax and perform needle decompression (refer box 1.4)
Circulation	• Apply direct pressure to external bleeding site • Obtain 2 wide bore venous accesses/intraosseous access if IV not possible • Initiate IV fluid therapy (20ml/kg bolus) with warmed crystalloid solution (NS/RL) • If no improvement after first bolus suspect ongoing hemorrhage due to internal organ injury • Give second and if required third fluid bolus but suspicion of internal bleed becomes stronger • Consider blood transfusion if child worsens or is non responder to fluid resuscitation • Transfuse 10mL/kg of type-specific or O-negative warmed pRBCs • Maintain urine output

	• Prevent hypothermia

Box 1.2: Stabilization of cervical spine

Better to assume cervical spine injury in every case of trauma

- Steps of stabilize
  - Immobilization of cervical spine is of utmost importance
  - Move the head and body together
  - Use cervical collar
  - Place pad under shoulder and back

Box 1.3 Indications of intubation

- Unconscious patient of GCS<8
- All manoeuvres to maintain open airway are unsuccessful
- Significant hypovolemia with depressed sensorium
- Perform rapid sequence intubation:
  - Pre-oxygenation
  - Atropine (only in infants) 0.01mg/kg
  - Sedation: Midazolam – 0.1mg/kg
  - Paralysis: Succinylcholine [2mg/kg(<10kg), 1mg/kg (>10kg] or
  - Vecuronium 0.1mg/kg

Box 1.4 Tension pneumothorax

Clinical clues

- Signs of respiratory failure
- Tracheal deviation away from the side of injury
- Hyper-resonant note on percussion
- Unilateral absence of breath sounds
- Cyanosis (late manifestation)
- Obstructive shock

How to treat?

Under water needle decompression: Insert needle just over the top of the third rib in the midclavicular line using a 14-18 gauge needle catheter in infants and small children

1.4 Secondary survey:

- The secondary survey begins once the primary survey (ABCDEs) is completed and normalization of vital functions has been demonstrated with resuscitation.
- Involves two components:
  Appropriate history taking from patient or family or any prehospital personnel detailed head to toe assessment (refer box)

Box 1.5: Components of Secondary Survey

Parameters to assess	**Methods**	**Interpretation**
Levels of consciousness	GCS	<8 : indication for intubation
Pupils	Size, symmetry and reaction to light	Unequal with no reaction to light : CNS injury, bleed (herniation due to mass effect)
Head	Any laceration, depressed fracture	-
Maxillofacial	Any plapable crepitation, mal – occlusion	Facial fracture Soft tissue injury
Neck	Inspect and palpate for any crepitation Auscultate for bruit Palpate cervical spine	Subcutaneous emphysema secondary to airway injury Hematoma-injury to major vein Brut-injury to carotid artery Pain and tenderness of cervical spine – suspect injury and stabilize cervical spine

Thorax	Respiratory and cardiovascular examination	Chest wall tenderness and palpable crepitation – subcutaneous emphysema Paradoxical chest movement – Flail chest due to rib fracture Decreased air entry – Pneumothorax Muffled heart sounds with raised JVP – Cardiac tamponade
Abdomen	Per abdomen examination	Generalized tenderness – peritonitis secondary to perforation Visceral injury – splenic hematoma
Pelvis	Symphysis pubis examination Palpate pelvic bone Inspect perineum	Rectal, vaginal or vulvae injury Hematuria – urethral injury
Spinal cord	Neurological examination for tone and power	Parenthesis/hyperasthesia Paraplegia/quadriplegia
Vertebral Column	Palpate for tenderness, lateralizing signs	Fracture or dislocation
Extremities	Visual inspection and palpation	Swelling, bruising Crepitation Absent and diminished pulses

1.5 Which cases can be treated in primary health care facility?

- Trauma with minimal abrasions
- Fracture of single bone which can be managed conservatively and the child is hemodynamically stable.

- Trauma in which the child is hemodynamically stable and has normal sensorium.

1.6 When to refer immediately?

- Impaired sensorium
- Need for mechanical ventilation
- Fractures requiring immediate fixation
- Disproportionate pallor suggestive of concealed hemorrhage
- Refractory shock not responding to fluid boluses suggestive of ongoing losses
- Fractures and deep penetrating wounds to an extremity complicated by neurovascular or compartment injury
- Anticipated need for surgical intervention.

1.7 How to refer?

- Ensure secured airway
- Provide adequate oxygen support
- Ensure hemodynamic stability
- Immobilize fractures
- With proper written consent obtained from relatives and explained about the sickness level
- In an ambulance with life savings equipment, drugs and monitors
- With a health care personnel who has been trained in basic life support management.
- Provide appropriate analgesia
- Ensure adequate continuous monitoring facility

1. ACUTE FEBRILE ENCEPHALOPATHY

1.1 Learning objectives

After completion of this section the participants should be able to

- Identify a case of acute febrile encephalopathy
- Identify patients with intracranial hypertension
- Emergency resuscitation and stabilization
- Managing intracranial hypertension
- When to give empirical treatment in acute febrile encephalopathy
- Know when and how to refer?

1.2 What is acute febrile encephalopathy'!

Acute onset of fever and a change in mental status (confusion, disorientation, coma, or inability to talk) AND/OR new onset of seizures (excluding simple febrile seizure: refer box 1.1) is labelled as acute febrile encephalopathy (AFE)

BOX 2.1 Features of simple febrile seizure

Fever in a child aged 6 months to 5 years with single generalized seizure lasting less than 15 minutes

The child is otherwise neurologically healthy and without neurologic abnormality by examination or by developmental history

It is a medical emergency with both diagnostic and therapeutic challenge
Various causes of AFE are enumerated in Box 1.2

Box 1.2: Etiology of acute febrile encephalopathy

- Pogenic meningitis
- Viral meningoencephalitis
- Cerebral malaria
- Tubercular meningitis
- HSV encephalitis
- Scrub encephalitis
- Japanese encephalitis
- Dengue encephalitis

1.3 what are the neurocritical problems anticipated or seen in patient with AFE?

Raised intracranial pressure (ICP) or hypertension
Seizures or status epilepticus (refer section D, Chapter 2)

1.4 Intracranial hypertension

Monroe Kelley doctrine states

There is a pressure – volume relationship between volume of CSF, blood, and brain tissue in our non-complaint skull. Any increase in one component is compensated by decrease in the other, until a particular limit is reached, following which raised intracranial pressure manifests.

ICP varies with age. Normal reange of ICP is 5 -15 mmHg

Pediatric literature has shown as association between ICP of >20 mm Hg and poor outcome. Hence, treatment of ICP is recommended at a ICP threshold of 20 mm Hg or when clinical sings and symptoms of ICP are present.

1.5 How to identify a child with raised ICP? (Refer box 1.3)

BOX 1.3: clinical features or raised ICP

Symptoms	Signs
Acute raised ICP: Comatose/obtunded Decerebrate posturing Unexplained irritability, persistent shrill cry in infants Chronic raised ICP: Headache (worse in morning) Projectile vomiting Gradual deterioration in sensorium	Low GCS with or without posturing Neurogenic hyperventilation Bulging anterior fontanel in infants Anisocoria, Papilloedema Cushing's triad: Hypertension, bradycardia, irregular, respiration

1.6 How to manage?

Step I. Initial stabilization (The ABCDE approach)

A. Airway
    - Children are at risk for airway instability due to :
    - Depressed sensorium
    - Loss of tone in oropharyngeal muscles
    - Falling back of tongue which blocks airway
    - Pooling of secretions & risk of aspiration
    - Action

- Positon _ head-tilt-chin- lift, jaw thrust (refer appendix)
- Suction
- Insertion of nasopharyngeal/oral airway (refer appendix0
- Intubation if required (refer box 1.4)

Box 1.4 indications of intubation

Encephalopathy i.e. GCS<8

Unable to protect airway/aspiration

Impaired respiratory drive
Seizures
Raised ICP
Neuromuscular weakness – Hypoventilation – Respiratory failure
Inadequate ventilation/oxygenation

B. Breathing

Monitor SpO2 and breathing
Start supplemental oxygen by delivery device that is available
Start Bag & Mask Ventilation (BMV) in case of inadequate chest movements efforts.
Intubate If – severe hypoxemia, raised ICP, refractory status epilepticus failure of or prolonged BMV

C. Circulation

Adequate systemic perfusion is crucial to maintain adequate cerebral perfusion
Secure IV access
Monitor pulse, perfusion, blood pressure and urine output. Maintain adequate hemodynamic parameters using fluids or vasoactive drugs

D. Desability

Monitor neurological status by Glasgow coma scale (refer appendix)
Treat seizures (refer status epilepticus)
Identify signs of raised ICP (refer Box 1.3)
Correct hypoglycemia, hypocalcemia, hyponatremia

E. Exposure

Look for fever or hypothermia, any rash

Step 2. First tier management of raised ICP

Head in midline with 15-30 elevation
Ensure normoxia SpO2 > 94%
Hypercarbia causes cerebral vasodilatiation thus causing raised ICP
Mild hyperventilation for few minutes (Target PCO2 = 30-35 mm Hg) canc be used to manage acute ICP spikes (refer box 1.5)
Prolonged and /or aggressive hyperventilation should be avoided
Hyperventilation is not useful to achieve long lasting ICP reduction

Ensure normovolemia by assessing hydration status (heart rate, pulse pressure, pulse volume, CFT, blood pressure and urine output)
Maintain blood pressure at 50th centile.
Prevent events that increase ICP

Fever : Ensure normothermia by using round the clock antipyretics
Use sedoanalgesia for pain
Sedation: Midazolam 0.05 – 0.2 mg/kg over 2-3 min followed by 1-2 ug/kg/min (max 6 ug/kg/min) as infusion
Analgesia: Fentany 1-3 ug/kg/hr or Morphine 0.1 mg/kg/dose 4-6 hrly as needed
Paralysis; Use neuromuscular blocker, cecureonium 0.1 mg/kg/dose as required
Avoid noxious stimulation, premedicate with lignocaine prior to ET suctioning (nebulized (4% lidocaine mixed in 0.9% saline) or intravenous (1-2 mg/kg as 1% solution) given 90sec prior to suctioning
Start antiepileptics in case symptomatic seizures and post trauma patient
Maintain normoglycemia: RBS 80-120 mg/dl
Haemoglobin: Maintain around 10 gm/dl

Box 1.5: indication of acute mild hyperventilation
Mild short-term hyperventilation (Target a PaCO2 ˜mmHg)
Should be undertaken if danger of herniation are present
Unequal pupils
Posturing
New onset deterioration in sensorim

Step 3. Pharmacologic therapy of intracranial hypertension
Osmotherapy: Causes movement of water from parenchyma to circulation and also reduces blood visocity and cerebral blood volume to reduce ICP
Agents used are 3% NaCl (hypertonic saline) or mannitol
3% NaCl is preferred osmotherapy indicated in all cases of raised ICP especially in children with hypotension
3% NaCl: Dose: 5-10 ml/kg bolus followed by 0.1-1ml/kg/hr to maintain serum sodium between 145-155 mEq/L
Sodium monitoring has to be done every 6 hourly till infusion is ongoing followed by 12 hourly
Side effects: acute kidney injury

Mannitol: Indicated if signs of impending herniation are there.
Patient should be Normotensive as mannitol can precipitate hypotension.
Initial bolus – 0.25-1g/kg followed by 0.25-0.5 g/kg every 2-6 hourly as per requirement.

Side effects: Hypotension, rebound rise in ICP, hypokalemia, hemolysis and renal failure

Steroids: Dexamethasone (0.15mg/kg/dose 6 hrly) is indicated in
Tubercular meningitis
Intracranial tumours with perilesionaloedema

Acetazolamide (20-100 mg/kg/day 8 hrly, max 2 g/day) is used in hydrocephalus (in infants with hydrocephalus ventricular tap can be attempted if anterior fontanel is open)

Barbiturate coma is reserved for refractory raised ICP. It causes decrease in cerebral blood flow and cerebral metabolic rate thus decreasing ICP. Thiopentone: 3-5mg/kg loading dose followed by 1-5mg/kg/hr is used for barbiturate coma. This drug however needs ICP and EEG monitoring. The most dreaded side effects is hypotension.

Step 5: Refractory intracranial hypertension (second tier therapy)
Defined as no improvement despite osmotherapy and acute mild hyperventilation

Modalities use are

Barbiturate coma
Mild hypothermia (core temperature 32-34 C)
Need to begin within 8 h of injury, and maintained for 48 h
Done by surface cooling
Speed of rewarming should be less than 1 C every 4-6 hr

Decompressivecranectomy: On rare occasions when all other measures fail, decompressivecraniectomy may be valuable.

1.7 Specific therapy
Choice of empirical therapy depends on the geographical location, season and the local microbiological data

Box 1.6

Etiology	Clinical clues	Treatment
Bacterial meningitis	Acute febrile encephalopathy with or without seizure	Ceftriaxone (100 mg/kg/day in 2 divided doses)
HSV encephalitis	Fever with progressive deterioration of consciousness, focal seizures or focal neurological abnormalities in the absence of any other cause	Acyclovir (30 mg/kg/day in 3 divided doses)
Cerebral malaria	AFE with pallor, icterus hepatospleonomegaly	IV Artesunate (3 mg/kg/dose in < 20 kg; 2.4 mg/kg/dose in

		>20 kg
Scrub typhus	AFE with organomegaly, capillary leak, eschar	IV doxycycline (4.5mg/kg/day 12 hourly)
Tuberculous meningitis	Subacute/chronic history of encephalopathy with h/o possible koch's contact	SATT after appropreiate investigations
Enteroviral encephalitis	Preceding loose stools and rash- summer/monsoon	Supportive treatment
Japanese encephalitis	Altered sensorium, changing neurological signs, extrapyramidal movements	Supportive treatment
Vaircella encephalitis	Vesicular rash, cerebellar signs	Supportive treatment
Rabies encephalitis	H/o dog-bite, bulbar signs	Supportive treatment

1.8 How to monitor?

Child should be monitored by a nurse at least every 2 hourly and by a doctor at least 4 hourly

What to look for?
GCS, pupillary size and reaction, seizures, new signs
Any other new signs of worsening
Hemodynamic and respiratory parameters

1.9 How to investigate?

- Complete blood count
- Serum electrolytes, renal function test, liver function test
- Peripheral smear for malaria and/or rapid malaria antigen test: In endemic areas with symptoms & signs: pallor, organomegaly
- Dengue serology: In endemic areas with signs & symptoms: capillary leak with bleeding manifestations
- Rickettsial serology: Endemic areas with eschar, organomegaly, capillary leak
- Lumber puncture: Only in hemodynamically stable patients with no signs of raised ICP
- Neuroimaging : CT brain is indicated in
  - Focal neurological deficit
  - Aferbrile cause of encephalopathy

- History of trauma
- Unexplained pallor
- If lumbar puncture is contraindicated

1.10 When to refer?

- Cases with persistent raised ICP
- Non improvement in sensorium within 3-5 days
- Need for neurosurgical procedures like ventriculo-peritoneal shunt, evacuation of bleed or decompressive craniotomy
- Refractory status epilepticus

1.11How to refer

Secure airway, breathing and circulation
Ensure 2 patent IV lines and fluids
Premedicate with lignocaine, midazolam, morphine before transfer to prevent excessive movement of patient
Ensure resuscitation kit and a BLS trained provider through transport
Monitor: Vitals, sensorium and pupils
Have anti seizure mediations also readily available

Remember:

- In a child with acute onset sltered encephalopathy with or without pupillary changes or brisk reflexes, assume raised ICP until proven otherwise
- Target normothermia, normoxia, normovolemia, normoglycemia
- No role of prophylactic hyperventilation.
- Mild short-term hyperventilation (target a $PaCO_2$ ≈30-35 mmHg) is indicated if signs of herniation are present
  - Unequal pupils
  - Posturing
  - New onset deterioration in sensorium

Protocol for Acute Febrile Encephalopathy

Identify case of acute febrile encephalopathy
Recgnise signs and symptoms of raised ICP

Indications of acute mild hyperventilation

Mild short – term hyperventilation (Target PaCo2≈ 30-35 mmHg) should be under
Taken if danger of herniation are present
Unequal pupils
Posturing
New onset deterioration in sensorium

Immediate measures

A. Maintain Airway
B. Assisted breathing in a child with apnea, bradypnea or irregular brething
C. Adequate circulation to maintain cerebral perfusion with the help of fluid or vasoactive drugs
D. Dextrose check & correction of hypoglycemia
E. Look for fever/hypothermia

First tier Measures

Supportive measures

Head in midline with elevation 15-30
Ensure normoxia Spo2 > 94 %
Ensure normovolemia by assessing hydration Status (heart rate, pulse pressure)
Maintain BP at 50th centile
Prevents events that increase ICP; fever, pain, noxiousstimulation,seizures,
Maintain euglycemia: RBS 80-120 mg/dl
HB: Maintain around 10 gm/dl
If any surgical cause of encephalopathy then refer to higher centre where neurosurgical facilities are available
Specific therapy (refer box 1.6)

Pharmocotherapy

- 3% NaCl is preferred osmotherapy indicated in all cases of raised ICP especially with hyptotension
- Dose: 5-10 ml/kg bolus followed by 0.1-1 ml/kg/hr
- Side effects: AKI
- Mannitol: Used in case of impending herniation if 3% Nacl is unavailable provided patient is normotensive
- Intial bolus – 0.25 – 1 g/kg followed by 0.25-0.5 g/kg every 2-6 h as per requirement side effect:
- Hypotension, rebound rise in ICP, hypokalemia, hemolysis and renal failure
- Steroids: Dexamethasone (0.15mg/kg 6hrly)
- Indicated in TBM,m Intracranial lesions with surrounding oedema
- Acetazolamide (20-100 mg/kg/day 8 hrly) in hydrocephalus
- In infants with hydrocephalus ventricular tap can be attempted in anterior fontanelle open

Second tier measure
Possible only with ICP monitoring

Barbiturate coma:
Thiopental or pentobarbital
Moderate Hypothermia (32-34 C)
Decompressivecranectomy

2. STATUS EPILEPTICUS

2.1 Learning objectives

After completion of this section, the participants should be able to

- Identify a case of status epilepticus
- Provide adequate first line antiepileptics to abort seizures
- Stabilise and manage ABC's
- Send appropriate investigations to detect the cause of seizures
- Look for reversible causes and treat them
- Escalate anti epileptic therapy based on need
- Know when and how to refer

2.2 What is status epilepticus?

- A seizure lasting for more than 30 minutes or recurrent seizures for more tham 30 minutes during which time the patient does not regain consciousness.
- Operational Definition:
  - Prolonged seizure activity 9>5mins) or persistent, repetitive, seizure activity without recovery of consciousness in between episodes.
  - Any child who is brought seizing to the emergency room should be treated as status epilepticus.
  - A child with epilepsy should be considered in status if the seizure persists for more than twice the usual duration of the previous seizures.

2.3 Steps in management

Start immediate stabilization (ABCD approach)

A. Airway

Position the child's head to one side and suction secretions (oral followed by nasal)

Place an oropharyngeal airway if required, to be avoided in conscious patients

Endotracheal intubation (Refer box 2.3)

Box 2.3 Indications of intubation

- Severe hypoventilation and hypoxia
- Failure of bag and mask ventilation
- Prolonged requirement of bag and mask ventilation
- Raised ICP

B. Breathing

- Monitor SpO2 and breathing.
- Start supplemental oxygen by delivery device that is readily available.
- Start bag & mask ventilation (BMV) in case of tachypnea, inadequate chest movements, poor air entry.

C. Circulation

- Secure IV access, monitor pulse, blood pressure and perfusion
- Correct reversible causes like hypoglycemia, Hypocalcemia, hyponatremia (Refer box 2.4)
- Begin continuous cardio – respiratory monitoring
- Abort seizure with escalating antiepileptic drugs and doses (Refer to algorithm)

Box 2.4 Correction of reversible factors

Calcium (if ionic value not available then do ECG): 2 ml of calcium gluconate in equal dilution with D5 over 20 mins under heart rate monitoring (ceiling dose of 10 ml)
Sodium: 5 ml/kg hypertonic saline (3%) bolus
Glucose: Remember product of volume and concentration of destrose should be 50

- 2 ml/kg of 25% dextrose
- 5ml/kg of 10% dextrose
- 10ml/kg of 5% dextrose

2.4 What are the causes of status epilepticus?

Box 2.1: Etiology of status epilepticus

- Acute:
  - CNS infections (meningitis, meningoencephalitis)
  - Febrile convulsions
  - Vascular episodes
  - Trauma
  - Metabolic
  - Poisonings

- With or without and underlying neurologic disorder
  - Non – compliance or withdrawal of anti-epileptic therapy

2.5 What are the conditions that mimic seizures?

Box 2.2 Non epileptic events
Syncope
Usually preceded by blurred vision, dizziness and pallor
Gastroesophageal reflux
Result in an arched back position with crying
No loss of consciousness & events associated with feeding
Cyanoticsbreath holding spells
Day dreaming
Pseudoseizure

Tetanus (patient will be conscious)

2.6 Investigations

Blood Glucose
Serum electroluytes (serum sodium, potassium, chloride)
Serum calcium (If ionic calcium not available them do ECG and calculate QTc)
If febrile: hemogram, lumber puncture
Neuroimaging: CT scan (refer section D, Chapter 1)

2.7 Monitoring

- Child should be monitored by a nurse at least every 4 hourly and by a doctor atleast 6 hourly
- What to look for?
  - Recurrence of seizure
  - Neurological status 2 hourly
  - Hemodynamic status 2 hourly

2.8 When to refer?

- Refractory status Epilepticus: Seizures that persist even after the adequate treatment with benzodiazepine, phenytoin or any second line agent.
- History of trauma, developmental delay, prolonged fever
- Examination suggestive of meningeal irritation, neurocutaneous marker

2.9 How to refer?

Refer only after stabilization, ensuring adequate airway, breathing and circulation
Proper documentation regarding the presentation of the child, details of resusciatative measure taken, interventions done during resuscitation and reasons for referral including name and contact number of referring physician
A doctor/paramedic trained in Pediatrics Advanced life Support/ Basic Life Support to accompany the patient
Transporting ambulance should have sufficient O2 supply and Resusciatation equipment
Inform the referral centre prior to sending the patient regarding the diagnosis of the child, indication for referral, current status and approximate time to arrival
Counsel the family of the child regarding the need for transfer and risks during transfer . Obtain written, signed consent for the same

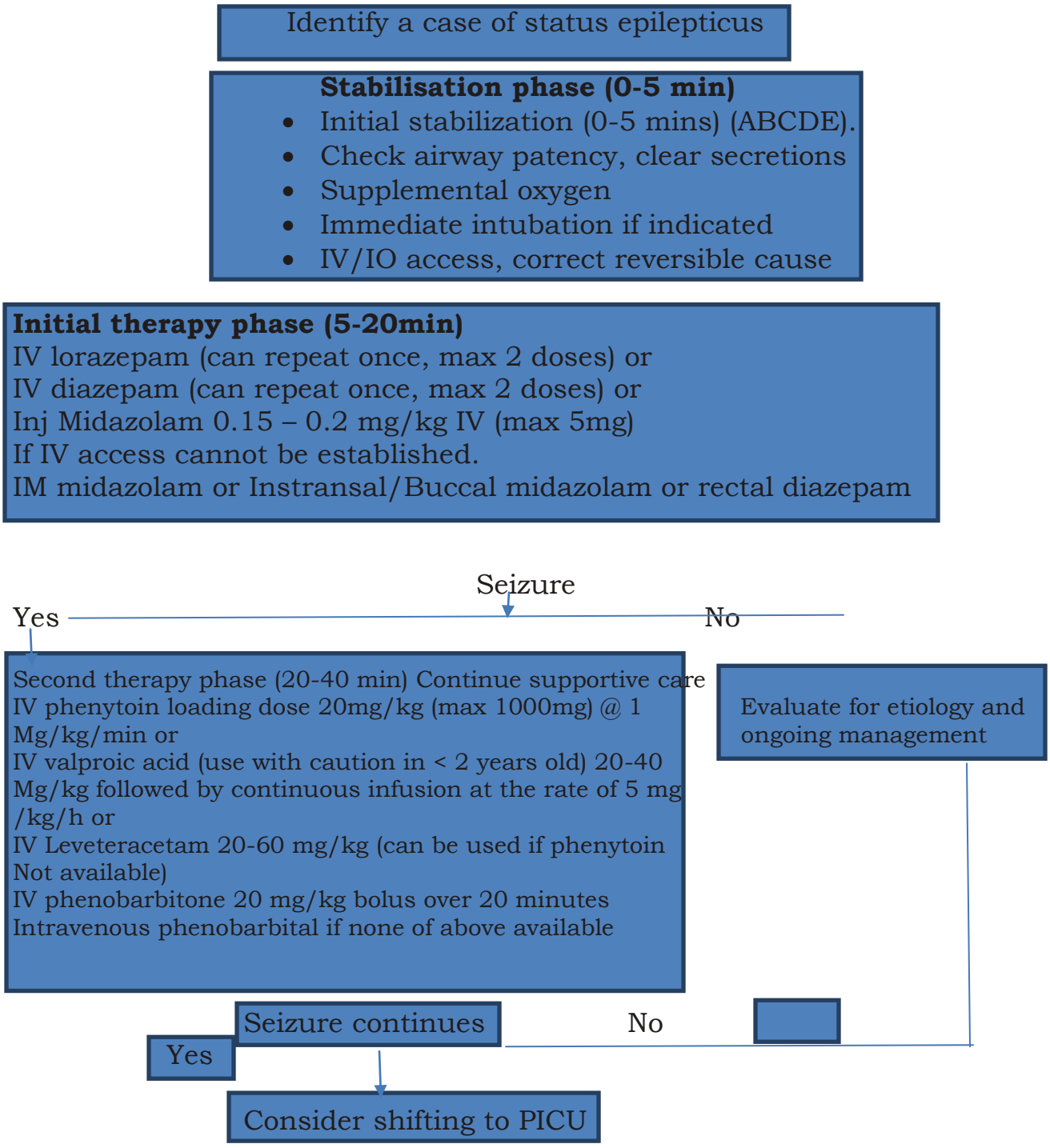
Remember:

- If IV access not available
- Buccal/nasal midazolam 0.2-0.3 mg/kg (max 5mg) or
- Per rectal Diazepam 0.5 mg/kg (max 10mg) or
- IM Midazoloam 0.2 mg/kg (max 5 mg)

- If the patient is already on phenytoin, then administer mini loading 10 mg/kg of phyenytoin s the initial anticonvulsant
- If the patient is already on valproate, the administer 10 mg/kg as the loadind dose. The infusion is continued until seizure free for 6 h, then tapered off @ of 1 mg/kg/h every 2 hourly
- Midazolam infusion is preferred over valproate in children less then 2 years of patients with liver failure
- Calcium infusion should be given only after hypocalcemia is confirmed or clinical examination
- How to calculate QTc interval?

Normal QTc – 0.44 sec, borderline QTc -0.44-0.46 sec, prolonged QTc -0.46 sec

- Thiamine 100 mg IV push: pyriodoxine 100 mg IV push can be tried in refractory in children < 3 years



Third therapy phase

Repeat second line therapy: Use drugs not used in second line

If not controlled then start midazolam 0.2mg/kg bolus followed by 0.05-02 mg/kg/hr

If not controlled then consider pentobarbital, or propofol (all with continuous EEG monitoring).

In children below 2 years of age, pyridoxine (100 mg intravenously) may be tried

**Midazolam infusion needs to be considered if seizure not controlled in 60 min.

PEDIATRIC HIGHER CENTER TRANSFER GUIDELINES

1.1 Learning objectives.

After completion of this section the participants should be able to

- Recognize clinical conditions that require referral to higher centre for further care
- Identify the nearby higher center which is appropriate for management of such a child
- Outline need for referral
- Enlist perquisites for referral
- Maintain transport checklist
- Pre-transport stabilization of ABCD

1.2 Why is transport to higher center required?

- Sometimes critically ill patients cannot be managed at resource limited setting due to inadequacy of available resources
- To overcome this gap in management transfer to higher centre is required
- The outcome is best when this transfer is done by adequate planning and careful risk benefit analysis

1.3 Points to considered while deciding transfer to higher centre

- Urgency of transfer
- Benefit of transfer
- Risk involved in transfer
- Child's clinical status before transfer and his/her ability to sustain transfer
- Child should be adequately assessed, resuscitated and optimal stabilized before transfer

1.4 What are the perquisites for transfer?

- Communication and coordination between the referring and referred team (centre where patient will be transferred)
- Appropriate equipment for monitoring and intervention if required
- Trained personnel to deliver life sustaining treatment during transport

1.5 How to ensure proper communication and co-ordination?

- Contact the referred facility doctor and confirm availability of resource (e.g. ventilator, CT) for which patients needs to be the transferred
- Confirm availability of bed for the patient
- Brief the doctor regarding patient history and present clinical status
- Summarize the treatment given along with all the relevant laboratory and radiological reports (attach copy with transport form)

1.6 What are the conditions requiring higher centre transfer? (refer box 1.1)

Box 1.1 indications for Pediatric Transfer/Referrals

• Medical	Trauma	Burns (Thermal or chemical)
• Depressed or deteriorating neurologic status • Status epilepticus • Severe respiratory distress responding inadequately to treatment and accompanied by any one of the following: • Grunting or gasping respirations • Cyanosis • Apnea • Retractions (moderate to severe) • Stridor (moderate	• Fractures and deep penetrating wounds to and extremity complicated by neurovascular or compartment injury • Fracture to two or more major long bones (i.e. femur, humerus0 • Fracture of the axial skeleton • Spinal cord or column injuries • Traumatic amputation of an extremity with potential for replantation	• and 3 burns>10% of the body surface area in children <10 years • and 3 burns >20% of the body surface area in children >10 yrs • burns > 5% of the body surface area for any age group • Signs or symptoms of inhalation injury • Burns involving face, mouth, throat and • Ears (serious full thickness burns or

to severe0 • Children requiring endotracheal intubation and/or ventilator support • Serious cardiac rhythm disturbance • Post cardiopulmonary resuscitation care • End organ failure (Cardiac, renal, hepatic) Shock responding inadequately to treatment Severe electrolyte imbalances Severe metabolic disturbances Severe dehydration Severe hypothermia or hyperthermia Need for invasive monitoring Arterial pressure Monitoring Central venous pressure Intracranial pressure monitoring	• Head injury when accompanied by any of the following: • Cerebrospinal fluid leaks • Open head injuries • (excluding simple scalp injuries) • Depressed skull fracutures • Significant penetrating wounds to the head, neck, thorax, abdomen or pelvis • Major pelvic fractures • Significant blunt injury to the chest or abdomen	burns involving the ear canal or drums), Deep burns of the hands, feet, genitalia, major joints, or perineum ○ Electrical injury or burns (including lightning)

1.7 How to stabilize the patient before transportation? (ABCDE approach)

- Airway
  - Maintain the airway patent
  - If child's airway is unstable it is safer to intubate
  - Indications of intubations before transfer (refer box 1.2)
  - Fix the endotracheal tube properly since any change in position can cause displacement in tube and accidental extubation

- Breathing
  - Maintain adequate oxygenation
  - Continuous SPO2 monitoring
  - Insert nasogastric tube in children with ileus and on bag and tube ventilation
- Circulation
  - Insert two large bore IV cannula; if IV access is difficult the secure IO access
  - Assess the need for fluid boluses (refer box 1.3)
  - Label the infusions properly
  - Catheterize every child with hemodynamic instability for strict fluid management.

- Disability
  - Assess the sensorium, pupillary size and its reaction to light
  - Control seizures and raised ICP
  - Adequate sedation and analgesia in intubated patients

- Exposure: Maintain temperature and glucose

Box 1.2 Indications for intubation before transfer

- GCS <8
- Respiratory failure (Spo2<94% despite oxygen therapy with any oxygen delivery devices)
- Unequal pupils, decerebrate posturing
- Fluid refractory or dopamine refractory shock
- Unstable airway (pooling of secretions, severe stridor)

Box 1.3 Indications for fluid bolus

Unexplained tachycardia with no features of CCF
Prolonged CFT, cold peripheries or poor peripheral pulses
Tachycardia with flash CFT, wide pulse pressure with bounding pulse (warm septic shock)
Hypotension: systolic BP<70 + (age x 2)

1.8 Accompanying personnel

Minimum of one doctor and one nurse along with the vehicle operator must accompany a critically ill child

The doctor should be trained in Advanced Life Support skill and should be well versed with emergency procedures like establishment of IV/IO access, endotracheal intubation, suctioning.
He/she should be able to manage any complication during transport like seizures, hypoglycaemia
Similarly and advanced life support trained nurse well versed with emergency management should accompany the doctor

1.9 Monitoring during transport

- Continuous SPO2 and ECG monitoring
- Vitals monitoring at regular intervals
- In intubated patients note the endotracheal tube position and identify any signs of displacement

1.10 Transport Check list

This is required to ensure compliance during transfers

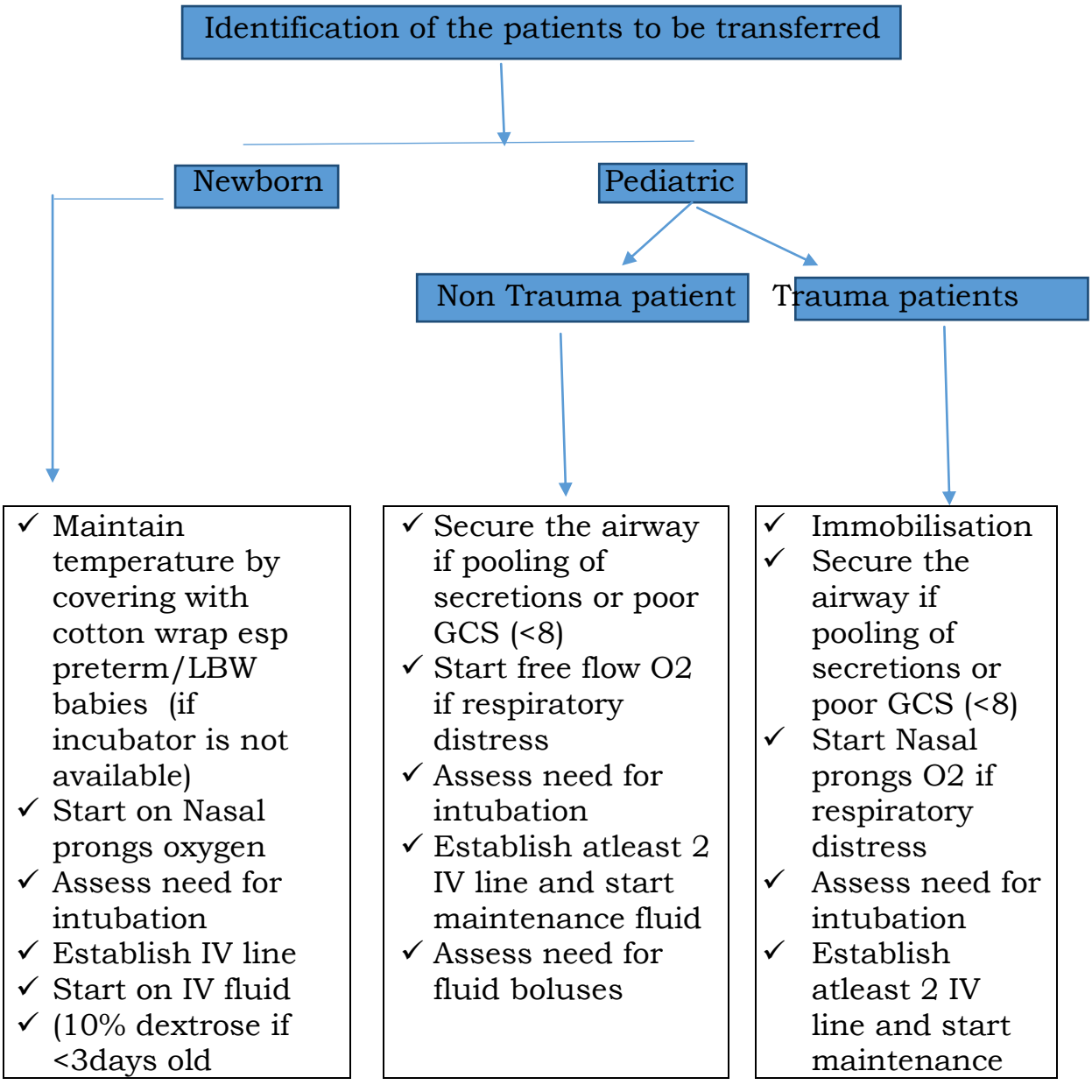
- Document initial evaluation and stabilization
- Indication for transfer
- Informed consent
- Name of the referred hospital
- Appropriate staff
- Monitoring equipment
- Pulse oxymeter
- Glucometer

- Essential equipment
  - Airway bag: Endotracheal tube, laryngoscope, mask, AMBU, tape
  - Fluids for boluses
  - Oxygen tubing
  - Suction
  - Defibrillator
  - Catheter/IO needles/IV cannula/NG tubes

- Medications
  - Adenosine
  - Atropine
  - Calcium
  - Dextrose
  - Dopamine
  - Dobutamine

- Dobutamine
- Epinephrine
- Lidocaine
- Mannitol
- Dextrose
- Dopamine
- Dobutamine
- Epinephrine
- Lidocaine
- Mannitol/3% NaCl
- Magnesium sulfate

Algorithm for transfer of critically ill Pediatric patient



✓ ISOLYTE P if >3 days old if available ✓ Assess need for fluid boluses		fluid ✓ Assess need for fluid boluses

1. DIABETIC KETOACIDOSIS

1.1 Learning objectives

After completion of this section the participant should be able to

- Identify a case of diabetic ketoacidosis (DKA)
- Assess the severity of DKA
- Initiate golden hour management of DKA
- Know when,how to refer and precautionary measures to be taken before referral?

1.2 What is DKA?

- DKA is an acute life threatening complication of diabetes characterized by features enumerated in Box 1.1

1.3 Why does DKA happen?

- It is a complex disordered metabolic state occurring as a consequence of absolute or relative insulin deficiency accompanied by an increase in counter-regulaltory hormones(glucagon, cortisol, growth hormone,epinephrine)
- This hormonal imbalance causes hepatic gluconeogenesis, glycogenolysis resulting in hyperglycemia and lipolysis that causes ketogenesis

Box 1.1: Features of DKA

Clinical	• Dehydration • Acidotic breathing (deep, sighing, no recessions) • Lethargy + drowsiness • Abdominal pain + vomiting • Polyuria and polydipsia
Laboratory	• Hyperglycemia (>200 mg/dl) • Ketosis (Blood > 3 mmol/ and/or Urine > 2+ with ketostix • Metabolic acidosis (pH <7.3) or bicarbonate <15 mmol/L (Note: If blood gas is not available,acidotic breathing can be taken as acidosis)

1.4 When to suspect diabetic ketoacidosis (DKA)?

In new onset diabetes, (diabetes is diagnosed during the first episode of DKA) suspectDKA in case of following presentation

- Unexplained drowsiness
- Unexplained vomiting & abdominal pain
- Unexplained dehydration but preserved urine output
- History of polyuria and polydipsia (history needs to be taken carefully as parents quite often ignore these symptoms)
- Deep sighing respiration
- All the above may or may not be associated with fever

In a known case of diabetes on insulin therapy,who presents with DKA, the following risk factors need to be assessed:

- Omission of insulin, infection, psychological stress as they can precipitate DKA
- Poor metabolic control
- Puberty and adolescence

1.5 Approach to a child with DKA

- Assess the severity (refer box 1.2)

Box 1.2 : Severity of DKA

Parameter	**Mild**	**Moderate**	**Severe**
Arterial pH	7.3- 7.2	7.2 - 7.1	< 7.1
Serum bicarbonate (mmol/l)	10- 15	5- 10	< 5
Level of consciousness	Alert	Alert/drowsy	Stupor/coma

- Goals of management

i) Correct fluid loss (dehydration):

- Calculate fluid requirements: Deficit (A) + Maintenance over 36-48 hours(B)
- **Deficit:** 5% in mild & moderate; 8.5% in severe cases
- A+ B has to be given as hourly infusion
- Normal saline (0.9%) is the initial fluid of choice for first hour management. Subsequently the fluid can be changed to half normal saline (0.45%) and continued till BG<250 mg/dl
- If plain 0.45% NS is not available, then normal saline can be continued
- The fluid is changed to 0.45% NS with 5%Dextrose once the BG is <250 mg/dl

ii) Correct hyperglycemia by starting low dose insulin infusion : 0.05 to 0.1 u/kg/hr (refer box1.3)

iii) Correct electrolyte disturbances

- Hyperkalemia can occur at presentation due to acidosis (refer to management section E, Chapter 4)
- Hypokalemia is a known complication of therapy in DKA. Once fluids and insulin are instituted, hypokalemia occurs due to intracellular shift of potassium. Add potassium at 40mmol/l of KCl after assessing adequate urine output

iv) Correct precipitating factors like infection. Start antibiotic if the child has clinical features of infection like UTI, sepsis etc

Box 1.3 : Insulin administration

- How to prepare insulin infusion?
  - Regular insulin(1ml= 40 U)1ml of regular insulin in 39ml of NS (1ml =1 U)
- How to administer:
  - Dilute this further in 10 ml of NS and infuse @ 1ml/hrwill give 0.1U/hour
  - If infusion pump is not availablethen use dial flow. In this case dilute in 100ml and infuse @10ml/hr
- Never mix other drugs along with insulin in the infusion set
- It is preferable to use a separate IV line for insulin

1.6 Investigations:

- Blood Glucose (capillary glucose with glucometer or venous blood glucose)
- ECG for potassium, if serum electrolytes are not available
- Blood gas & serum electrolytes
- Blood ketones and/orurine ketones

1.7 Complications

- Cerebral edema: it is the most dreaded complication of DKA and carries a high mortality if not detected and treated early
    - Features: Clinical signs of neurological deterioration (headache, irritability, drowsiness, seizures, coma, hyperventilation, pupillary inequality, bradycardia and bradypnea)
    - Treatment
        - Reduce fluids by 1/3rd to half
        - Immediate hyperosmolar therapy – 0.5 gm/kg Mannitol or 5ml/kg 3% saline
        - Intubation, and initiate anti-raised ICP measures(refer section D, chapter 1)
- Hypokalemia :
    - Potassium is mainly an intracellular ion, and there is always massive depletion of total body potassium although initial plasma levels may be low, normal or even high. Levels in the blood will fall once insulin is commenced
    - Treatment
        - Early potassium replacement, after ruling out AKI
        - Rapid correction (0.3-0.5mEq/kg/hour) for hypokalemia which presents with ECG changes(refer section E, chapter 4)
        - Maintenance potassium at 40-60mEq/L to keep serumpotassium between 3.5-5mEq/L
- Hypoglycemia :
    - Malnutrition is an important risk factor for hypoglycemia
    - Aggressive monitoring of blood glucose is required during insulin therapy
    - Early introduction of dextrose containing fluids

1.8 Non responders (refer box 1.4)

Box 1.4: Checklist for non-response

- Check patency of IV lines
- Improper insulin dose, dilution and rate of infusion
- Incorrect administration; flush the IV line completely before starting insulin to saturate insulin binding sites
- Underlying infection
- Uncorrected dehydration (check fluid charting)
- Rarely, there may be associated lactic acidosis/ or renal compromise

1.9 When to refer?

- Neurological deterioration: seizures, encephalopathy.
- Acidosis not improving
- Persisting hyperglycemia
- Decreased urine output

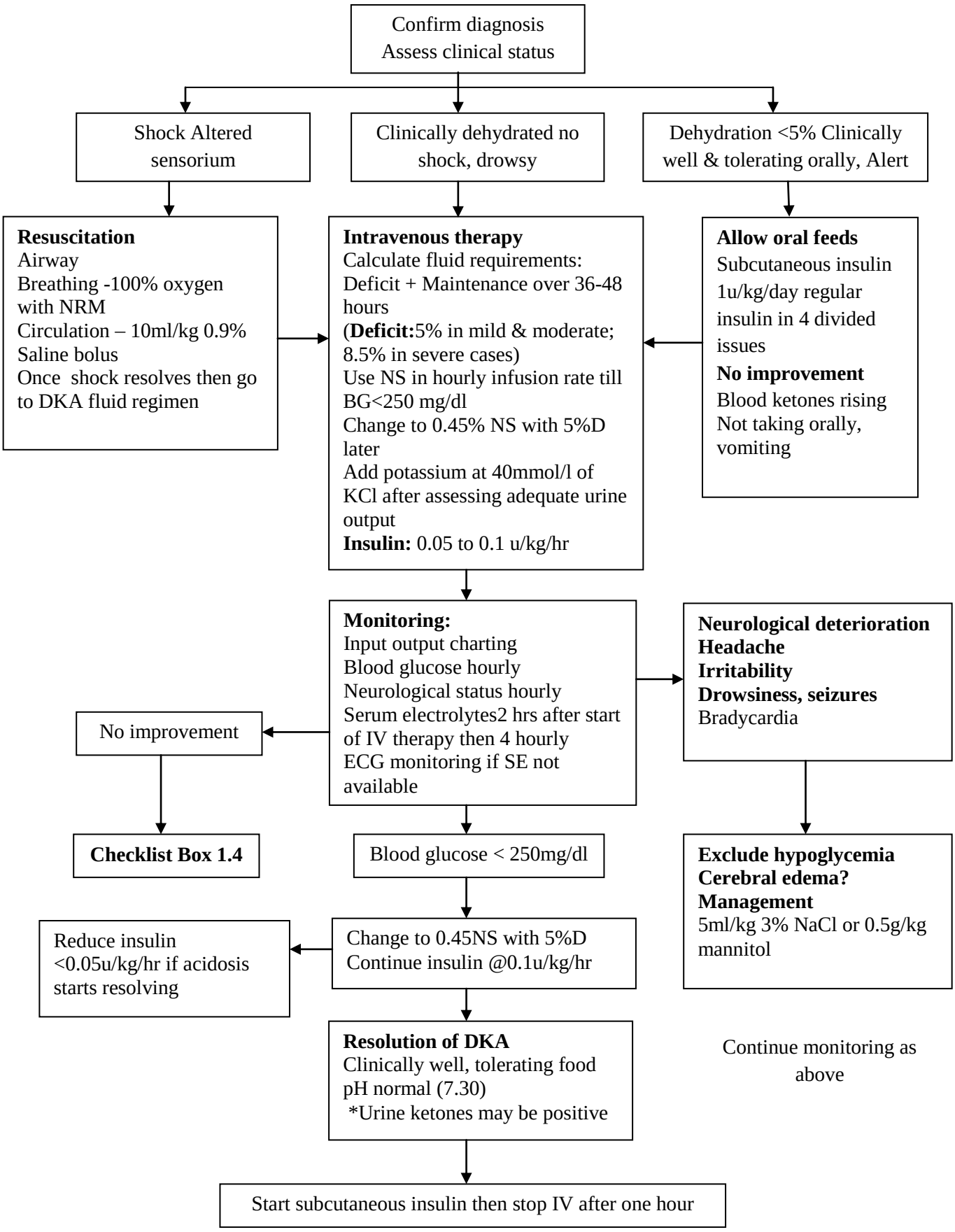
1.10 How to refer?

- Child's peripheral venous access should be secured.
- Child's details, history, examination findings, vitals, events occurred and the treatment given should be legibly written in the referral letter.
- Parents should be counseled regarding the patient's clinical status
- A person trained in BLS should accompany the child during referral

Remember:

- Acidosis not getting corrected , think of following possibilities
  - Insufficient insulin to switch off ketones
  - Inadequate resuscitation
  - Sepsis
  - Hyperchloremic acidosis
  - Salicylate or other prescription or recreational drugs
- Think about sepsis in a child with DKA who has any of the following:
  - fever or hypothermia
  - hypotension
  - refractory acidosis
  - lactic acidosis
- Ensure that all fluids (except any initial bolus) contain 40 mmol/l potassium chloride, unless there is evidence of renal failure.

Approach to DKA



2. Acute kidney injury

2.1 Learning Objectives:

After completion of this section, the participant should be able to

- Define acute kidney injury
- Identify cases at risk for AKI
- Identify clinically children with AKI
- Classify based on severity
- Know the common etiology of AKI
- Steps in management of AKI
- Know when and how t**o** refer?

2.2 Acute kidney injury (AKI)

- It is a spectrum of acutely compromised renal function that result in impaired fluid balance ,dyselectrolytemia ,and raised urea and creatinine.

2.3 How to define AKI?

In 2012, KDIGO Kidney Disease Improving Global Outcomes (KDIGO) developed a harmonized child-adult definition of AKI based on serum creatinine, eGFR, and urine output.

Remember in most situations AKI will be diagnosed based on urine output criteria only

- Urine volume ≤0.5 mL/kg/hour for six hours **OR**
- Increase in serum creatinine by ≥0.3 mg/dL from baseline within 48 hours; **OR**
- Increase in serum creatinine to ≥1.5 times baseline within the prior seven days

2.4 Which patients are at high risk for developing AKI ?

BOX 1.1 High risk states

In community setting

- History of kidney disease
- Hypotension
- Dehydration
- Gastrointestinal losses
- Dark, concentrated urine
- Ongoing sepsis
- Exposure to potential nephrotoxins

In hospital setting

- Multiple organ failure
- Nephrotoxic medication exposure
- Post operative state

2.5 How to identify patients with AKI?

- Identify high risk cohort (refer box 1.1)
- Monitor urine output to quantify oliguria /anuria
- Other clinical features
    - Odema
    - Gross or microscopic hematuria
    - Hypertension
    - Anemia
    - Laboratory parameters (refer box 1.2)

2.6 Investigations

BOX 1.2

To confirm diagnosis	Associated metabolic problems	Ascertain etiology
Urea Creatinine	Sodium , Potassium Acidosis Calcium, phosphorus	Urine routine examination Complete blood count Peripheral smear C3 Reticulocyte count LDH C3 USG (KUB)

Caveats of serum creatinine

- Change in creatinine is a delayed phenomenon. Therefore serum creatinine may be normal in early stages of renal failure
- Serum levels depend on muscle mass
    - Young children, malnourished children with less muscle mass will have lower creatinine for the same degree of AKI
- Fluid overload may falsely lower creatinine levels due to dilutional effect

2.7 Identify the common causes

Etiology of AKI can be divided into three categories as summarized in box 1.3

BOX 1.3 : Etiology of AKI

Etiology	Mechanism
Pre-Renal	A. Low intravascular volume • Fluid responsive o Dehydration-GI losses, Renal losses (e.g.: diuresis, diabetes insipidus) o Hemorrhage/bleeding • Non fluid responsive o Third space Losses-Nephrotic syndrome, liver disease B. Decreased effective circulatory volume • Cardiac dysfunction • Sepsis C. Vascular insult • Renal vein or artery thrombosis
Renal (Intrinsic)	A. Glomerular • Glomerulonephritis B. Vascular • Hemolytic uremic syndrome • Disseminated intravascular coagulation • Malignant hypertension C. Interstitial • Acute interstitial nephritis-infection, nephrotoxic medications, immune mediated D. Tubular • Acute tubular necrosis – hypoxic/ischemic, rhabdomyolysis, hemolysis
Post Renal (obstruction of urinary tract)	A. Unilateral • Mass, stones B. Bilateral • Posterior urethral valves

2.8 How to grade the severity of AKI ? (refer box 1.3)

BOX 1.3 : Staging of AKI

Stage (Severity)	Serum creatinine(S Cr) criteria	Urine output(UO)criteria
1	Rise of SCr to 1.5 to <2 time baseline within a7 day period OR ≥0.3 mg/dl SCr increase from baseline within a 48 hour period	<0.5 ml/kg/h for 6–12 h
2	Rise of SCr 2 to <3 times from baseline	<0.5 ml/kg/h for ≥ 12 h
3	Rise of SCr≥ 3 times baseline OR Increase to ≥ 4mg/dl OR Initiation for renal replacement therapy for AKI OR Decrease in estimated glomerular filtration rate to <35 ml/min/1.73 m2	<0.3 ml/kg/h for ≥ 24 h Or Anuria for ≥ 12 h

2.9 Management of AKI

- Initial stabilization and resuscitation of ABCDE
- Management can be divided in three categories
    - Fluid management
    - Electrolyte management
    - Pharmacological treatment

A. Fluid management

- Assessment of fluid status : This can be done by using clinical variables
    - Baseline body weight
    - Peripheral pulses, central pulses, capillary refill time, blood pressure
    - Urine output
    - History of recent fluid loss
    - Cumulative fluid balance
- This assessment is required to decide whether patient is hypovolemic/ euvolemic / hypervolemic
    - Hypovolemic - Correct fluid deficit (refer diarrhea with dehydration protocol, if fluid refractory refer sepsis protocol)
    - Hypervolemic /fluid overload – furosemide single dose (1-2 mg/kg) can be tried provided hemodynamic parameters are maintained
- Once the child becomes euvolemic replace with following fluid
    - 400ml/m^2 + replacement of losses (Urine output and other losses)
    - Preferred replacement fluid -1/2 NS (0.45%) with or without dextrose depending on patients intake and blood glucose
- How frequently monitoring needs to be done?
    - Intake and output 4-6 hourly
    - Hemodynamic status 4-6 hourly
    - Daily weight
    - Fluid charting should be reviewed every 6^{th} hourly based on intake/output chart

B. Electrolyte management

- Only maintenance sodium of 2-3meq/kg/day is allowed
- Patients with oligo-anuric kidney failure or severe ATN should not receive potassium or phosphorus
- Management of severe acute hyperkalemia (refer to hyperkalemia protocol)
- AKI secondary to interstitial or tubular involvement may have polyuria resulting in loss of electrolytes .In that case higher administration may be required

C. Pharmacological Therapy

- Avoid all nephrotoxins- stop NSAIDs, angiotensin receptor blockers, ACE inhibitors
- Renal dose adjustment based on eGFR
- Management of hypertension as per protocol
- Diuretics single dose can be considered in fluid overload

2.10 When to consider renal replacement therapy (RRT)e.g Dialysis ?

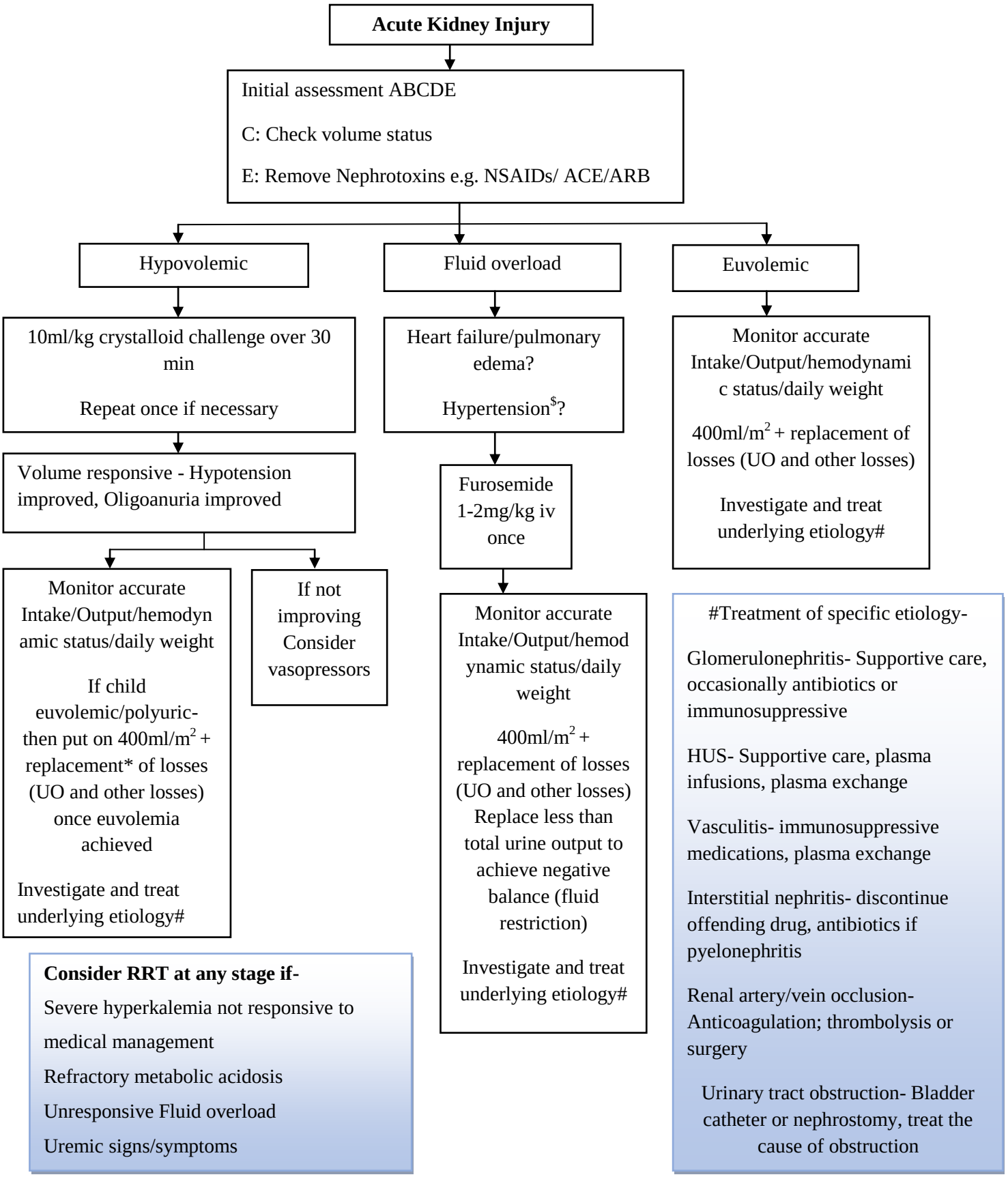
- Hyperkalemia refractory to medical therapy
- Refractory metabolic acidosis
- Fluid overload not responding to medical therapy
- Uremic encephalopathy /pericarditis
- Rising serum urea and creatinine
- Persistent oligoanuria

2.11 When to refer?

- Stage 1 and 2 AKI which is persisting or worsening
- Stage 3 AKI
- Need for RRT
- Imaging or investigation required to confirm diagnosis e.g. obstruction of urinary tract, vascular thrombosis

2.12 How to refer ?

- Secure airway, breathing and circulation
- Ensure resuscitation kit and a BLS trained provider during transport
- Child's peripheral venous access should be secured and patent
- Child's details, history, examination findings, vitals, events which occurred and treatment given should be legibly written in the referral letter
- The parents should be counseled regarding the child's condition and need for referral
- Written, informed consent should be taken from the parents/guardian's prior to referral



*Replacement of urine output with N/5 saline and insensible losses with 5%Dextrose

$ Hypertension- treat as per hypertension management pathway

Electroyte imbalances – refer section

3. Sodium disorders

3.1 Learning objectives

After completion of this section, the participant should be able to

- Recognize clinical states at high risk for hyponatremia
- Identify signs and symptoms of hyponatremia
- Steps in the emergency management
- Know when and how to refer?

3.2 Sodium homeostasis

- Disorders of sodium should always be interpreted from context of water balance.
- Osmolality refers to the total concentration of solutes in water. Plasma osmolality is maintained by strict regulation of the ADH system and thirst.
    - If plasma osmolality increases e.g. diarrhoea, ADH is secreted and water is retained by the kidneys, thus decreasing serum osmolality.
    - If plasma osmolality decreases e.g. fluid overload, ADH decreases resulting in diuresis of free water and a return to homeostasis.
- Usually, hypernatremia (sodium excess) is always associated with free water loss and hyperosmolality
- Hyponatremia (Low sodium) can be associated with low, normal or high serum osmolality. Hyponatremia generally denotes free water excess, though it can be seen due to sodium losses also
- Among the two, hyponatremia is more common and frequently seen electrolyte disorder in critically ill child.

3.3 Definition of hyponatremia:

- < 135 mEq/l in critically ill child
- <130 mEq/l is symptomatic hyponatremia

3.4 Recognize clinical states at high risk and classify them according to water imbalance (refer box 3.1)

BOX 3.1: Causes of hyponatremia

Hypovolemic hyponatremia	Euvolemic hyponatremia	Hypervolemic hyponatremia
• Diarrhoea, vomiting • Diuretics • Cerebral salt wasting • Adrenal insufficiency • Renal tubular acidosis • Osmotic diuresis (mannitol, hyperglycemia) • Burns	• SIADH (all critically ill patients) • Hypothyroidism • Drugs (thiazides, desmopressin)	• Congestive heart failure • Cirrhosis • Nephrotic syndrome • Renal failure

3.5 How to identify symptomatic hyponatremia ?

BOX 3.2 : Clinical features of hyponatremia

Symptomatic	Asymptomatic
• Headache, lethargy • Nausea, vomiting • Cramps • CNS : Confusion, Ataxia, Seizures Obtundation, respiratory depression	• Wait and watch • Monitor carefully

3.6 Treatment

- Identify clinical states at high risk for hyponatremia
- Asymptomatic patients with sodium >125mEq/L do not need any correction. They need to be monitored closely.

- Symptomatic patients or sodium level <125mEq/l need sodium correction. Determine the volume status of patient using vital signs, BP, skin turgor, mucous membranes, urine output, peripheral edema, blood urea/ creatinine
- Calculate the sodium deficit by using the formula **(refer box 3.3)**
    - 0.6 X weight X Deficit (desired sodium –patient's sodium)
    - Correct using 3% saline or by constituting the required fluid
- Following the correction assess the patient clinically to determine the etiology (refer box 3.1)
- Treatment varies as per the etiology
    - Hypovolemic hyponatremia : treat with normal saline
    - Euvolemic hyponatremia: fluid restriction is required
    - Hypervolemic hyponatremia: fluid restriction, diuretics

BOX 3.3 Correction of sodium deficit

- Ask the following questions while managing a child with hyponatremia
    - Is it true hyponatremia ?
    - What is the volume status?
    - Is patient symptomatic or asymptomatic ? If yes then what is the treatment ?
    - What sodium level should I target ?
- Calculate the deficit and correct it by using 3% saline or by constituting the required fluid
- Example : 10kg child with sodium of 124mEq
    - Calculation: 0.6 X weight X deficit(desired sodium – patient's sodium)
    - 0.6 X 10 X (132-124) = 48 (bring sodium upto 130 or 132)
    - It is advisable to avoid correcting the serum sodium concentration by >12 mEq/L/24 hr or >18 mEq/L/48 hr
    - 3% saline required: 1ml of 3%saline increases Na by 0.5 mEq
    - Hence this child needs 96 ml of 3% saline
    - If 3% saline not available then prepare fluid as below
        - DNS: 154 mEq/l, ½ DNS : 77mEq/l, 1/3 DNS : 51 mEq/l
        - Hence this patient needs 1/3 DNS
        - NS- 33ml +10% Dextrose -50 ml + 25 % Dextrose -7 ml

Algorithm for hyponatremia

Hyponatremia (serum Na <130 mEq/l)

Serum Na < 125 mEq/L with severe symptoms (e.g., seizures, ataxia, coma, respiratory depression**)**

- Calculate deficit
- 0.6 X wt X deficit
- Goal of increasing serum sodium level by 6 to 8 mEq /L (not to exceed 10 to 12 mEq/ L in the first 24 hours)
- Can use 3% saline or prepare fluid accordingly (**Refer to box 3.3)**

Serum Na >125 mEq/l Asymptomatic)

Monitor closely for any symptoms

Assess volume status-

Vital signs, BP, skin turgor, mucous membranes, urine output, peripheral edema, blood urea/ creatinine

Hypovolemic hyponatremia

(Decreased TBW and low Na)

Euvolemic hyponatremia

(Increased TBW and normal Na)

Hypervolemic hyponatremia

(Increased TBW)

1. Extra renal losses:

Diarrhea, vomiting, intestinal obstruction, third space losses (dengue, scrub typhus)

Treatment: Normal Saline

2. Renal losses:

Diuretics, aldosterone deficiency, Cerebral salt wasting, RTA

Treatment: Normal Saline,

SIADH

Treatment : Fluid restriction

Diagnostic criteria of SIADH

- Urine osmolality > 100mosm/kg
- Serum osmolality < 280mosm/kg and serum sodium <135
- Urine sodium >30 meq/L
- Absence of
  - Renal , adrenal , thyroid insufficiency
  - Heart failure, nephrotic syndrome or cirrhosis
  - Diuretic ingestion
  - Dehydration
- Correction with water restriction

CCF, cirrhosis, nephrosis, hypoalbuminemia

Treatment : Fluid restriction, Na restriction, diuretics , ACE inhibitors

Hypernatremia

3.7 Learning objectives

After completion of this section, the participant should be able to

- Recognize clinical cases at high risk of hypernatremia
- Identify the signs and symptoms
- Steps in emergency management
- Know when and how to refer?

3.8 Definition of hypernatremia

- Serum sodium >145 mEq/L

3.9 Recognise clinical states at high risk for hypernatremia

- Hypernatremia is usually due to loss of free water from the body (skin, kidney, GIT losses).
- In all cases, access to water intake is lost (intubated patients, altered sensorium) or there is an impaired/ immature thirst mechanism as in neonates/infants
- At risk population is
    - Neonates
    - Infants
    - Improperly reconstituted /concentrated ORS
    - Hyperglycemia
    - Altered sensorium interfering with thirst & water intake

3.10 Classify as per volume status (refer BOX 3.4)

BOX 3.4 Causes of hypernatremia

Hypovolemic Hypernatremia	**Euvolemic hypernatremia**	**Hypervolemic hypernatremia**
Decreased TBW>> Decreased Na Water loss more than sodium loss	Decreased TBW & normal Na Water loss but normal sodium	Increased TBW Water & sodium both increased
Extra renal losses: Diarrhoea, vomiting, intestinal obstruction, losses through NG tube Renal losses: Loop diuretics, osmotic diuresis	**Renal loss:** diabetes insipidus	Saline infusions, saline enemas, Soda bicarb, Salt tablets

3.11 How to identify clinical signs and symptoms which suggest high probability of hypernatremia (refer box 3.5)

- In hypernatremia there is better preservation of intra vascular volume because water shifts from intracellular to extracellular space
- This intracellular water loss leads to a **doughy** feel of the skin
- Conventional signs of dehydration viz. delayed skin turgor, sunken eyes are absent
- Hemodynamic parameters like pulse volume, CFT, BP will be relatively well preserved
- CNS signs and symptoms will be out of proportion to degree of dehydration or hemodynamic status

Box 3.5 Clinical features of hypernatremia

- Infants: hyperpnea, high pitched cry, restlessness, muscle weakness, lethargy, coma

Tearing of intra cerebral and bridging vessels can cause sub dural , Sub arachnoid , parenchymal hemorrhage

3.12 Treatment

- Identify clinical states with hypernatremia
- Classify as per the volume status
- Patients with hypovolemic hypernatremia have decreased total body water and sodium but need correction by replacing the free water as water loss is more the sodium
- Calculate free water deficit **(refer Box 3.6)**

  A. Free water deficit in litres= 0.6 X weight in kgX{1- 145/ Current Na}

  B. Calculate maintenance fluid for the duration of correction

  C. Solute fluid deficit = Total fluid deficit (severity of dehydration) - Free water deficit
- Total fluid required by the child for proposed duration : A+ B
- Decide the duration of correction (24hrsvs.36hrs vs.48hrs)
    - Duration of symptoms(if longer then better to correct slowly)
    - Sodium level (severe hypernatremia >165 , correct slowly)
    - Osmolarity level (>320mosm/kg ,correct slowly)
- Fluid has to be administered as hourly infusion
- Type of fluid
    - No sodium for deficit correction
    - Sodium only for maintenance fraction (3meq/kg) and for amount of solute fluid deficit
- Goal is to decrease serum sodium by <12 mEq/l every 24 hours ; rate of 0.5 meEq/l

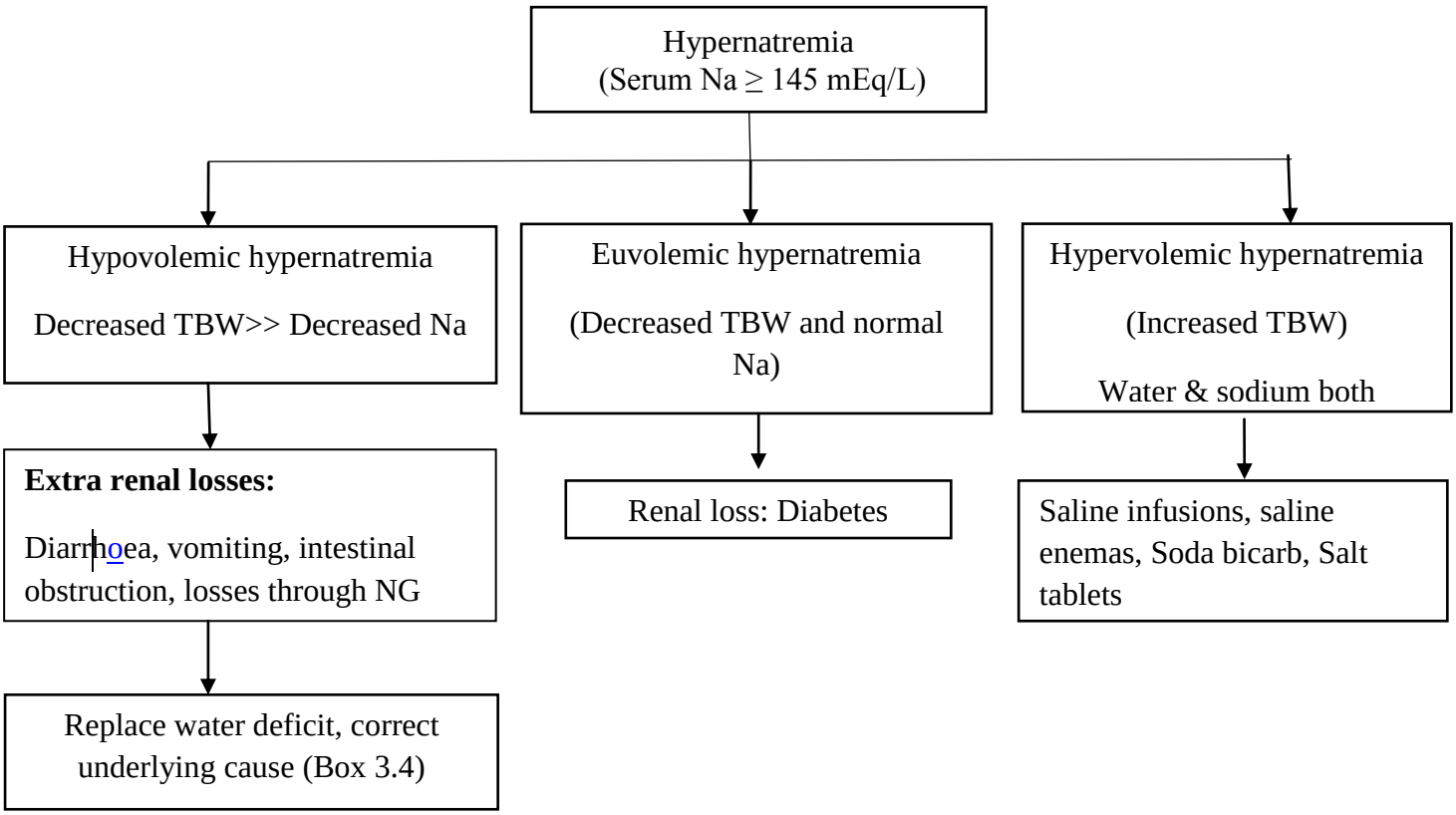
3.13 When to refer?

- As such sodium disorders can be managed at resource limited settings may need referral for investigation of the cause

3.14 How to refer ?

- Secure airway, breathing and circulation
- Ensure resuscitation kit and a BLS trained provider during transport
- Child's peripheral venous access should be secured and patent
- Child's details, history, examination findings, vitals, events which occurred and treatment given should be legibly written in the referral letter
- The parents should be counseled regarding the child's condition and need for referral
- Written, informed consent should be taken from the parents/guardian's prior to referral

Algorithm for hypernatremia



BOX 3.6

Example : 10 kg child with acute history of diarrhoea. On assessing the severity of dehydration ,he has some dehydration (10%) (refer section C, box 2.1). Investigations reveal Na of 160.Cause being concentrated ORS intake. Calculation of fluid

A. Free water deficit : 0.6 x 10 X {1- 145/ 160} = 0.6 litres= 600 ml
B. Maintenance fluid = 1000 ml for 24 hrs
C. Solute fluid deficit
    a. Total fluid deficit (severity of dehydration)- free water deficit
    b. 100ml/kg(10%) – 600
    c. 1000-600= 400
D. Total fluid = A+B+= 600+ 1000= 1600 ml
E. Hourly fluid : 1600/24= 66ml /hr
F. Type of fluid :
    a. Sodium for maintenance + sodium for solute fluid deficit
    b. 3meq/kg(30mEq) +(0.4 X 0.6 X 145)= 30+34.8= 65meq

We need 65meq in 1600 ml : 1/5 NS is the fluid required.

4. Potassium disorders

4.1 Learning objectives

After completion of this section, the participant should be able to

- Recognize clinical states at high risk for hypokalemia
- Identify the signs and symptoms of hypokalemia
- Know the steps in emergency stabilisation
- Know the steps in management of severe symptomatic hypokalemia
- Know when and how to refer?

4.2 Potassium homeostasis

- Potassium is a predominant intracellular cation
- The distribution of potassium levels across cellular membranes helps determine the resting membrane potential as well as timing of membrane depolarization
- Derangements of potassium lead to neuromuscular, gastrointestinal and cardiac conduction abnormalities
- Normal serum potassium levels: 3.5 to 5.5mEq/L

4.3 Recognize clinical states at high risk for hypokalemia (refer Box 4.1)

BOX 4.1: Clinical states at risk for hypokalemia

Inadequate intake	**Abnormal losses**	**Extracellular to intracellular shifts**

• Protein energy malnutrition
• Prolonged starvation
• Total parenteral nutrition
• Critically ill child
	• GI losses: Diarrhoea& vomiting
	• Renal losses:
	  ○ Osmotic diuresis
	  ○ Renal tubular disorders
	  ○ Diuretic therapy
		• Alkalosis
		• Insulin therapy
		• Use of beta 2 adrenergic agonists
		• Hypothermia

4.4 Identify clinical signs of hypokalemia:

- Muscle weakness: floppy neck & limbs, respiratory muscle weakness
- Cardiovascular: Bradycardia, irregular rhythm, ventricular tachycardia or cardiovascular collapse in severe hypokalemia
- Abdominal distension, paralytic ileus

4.5 Confirm hypokalemia

- Electrocardiographic (ECG) changes (Fig A)
  - Prolonged QT interval
  - ST segment depression
  - T wave flattening
  - Appearance of U waves
- Laboratory : Serum potassium < 3.5meq/L

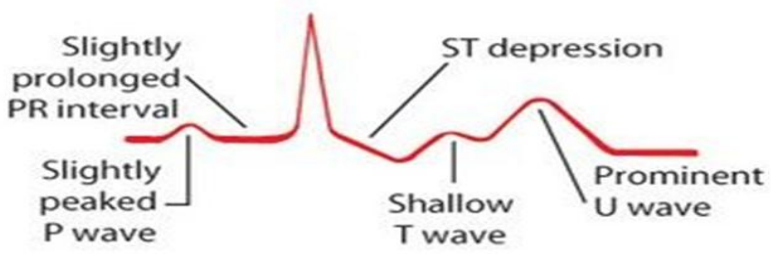


Fig A : ECG changes in hypokalemia

4.6 Approach to a child with hypokalemia :

- Identify the high risk states for hypokalemia (refer box 4.1)
- Remember that serum potassium levels may not be accurately reflective of total body potassium stores
  - Example : In DKA, the initial serum potassium levels are high (extracellular shifts due to acidosis)even though total body stores are severely depleted. Therefore correction of acidosis can result in a precipitous drop in potassium levels
- Decision to treat depends on severity and presence or absence of symptoms

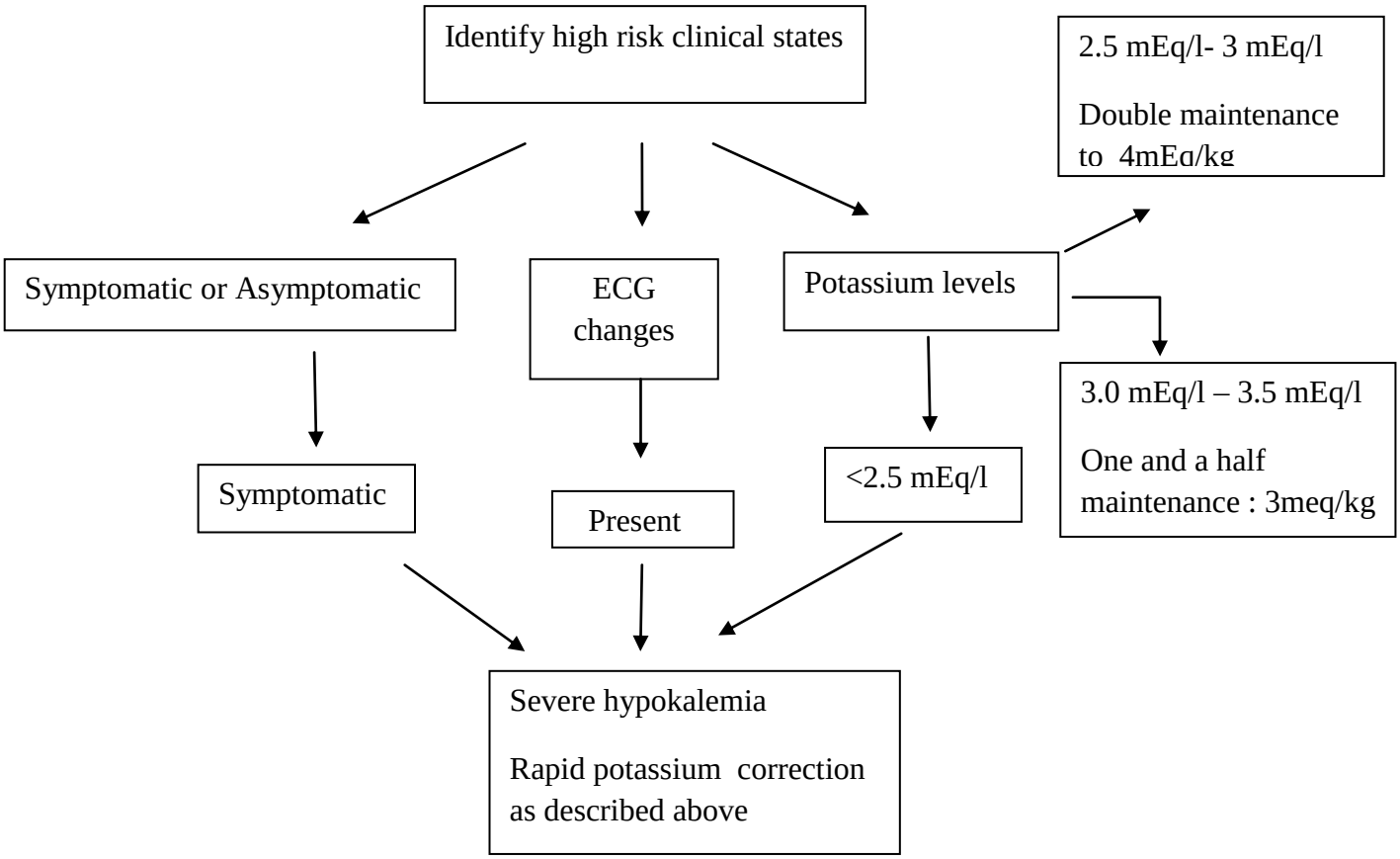
- Rapid IV correction is mandatory for severe and symptomatic hypokalemia (<2.5meq/l) in a dose of 0.3meq/kg/hr infusion (refer box 4.2)
- Non severe hypokalemia can be treated by increasing the maintenance dose
    - Serum potassium 2.5 mEq/l- 3 mEq/l: Double the maintenance dose to 4meq/kg
    - Serum potassium 3.0 mEq/l – 3.5 mEq/l : Increase maintenanceby one and a half times i.e. 3meq/kg
- End point of correction is to increase potassium levels >3 mEq/l and /or until appearance of T waves

BOX 4.2: How to give rapid correction?

Steps of administration

1. Prepare stock solution: 5ml KCl + 45 ml NS to make a total of 50 ml
2. This solution when given @ 1.5ml/kg/hr would provide 0.3mEq/kg/hrpotassium correction
3. To be given under strict cardiac monitoring with continuous ECG recording
4. Duration of correction is based on severity of hypokalemia
5. End point of correction: Raise serum potassium >3 mEq/land /or until appearance of T waves

Protocol for management of hypokalemia



Hyperkalemia

4.7 Learning objectives

After completion of this section, the participant should be able to

- Recognize clinical states at high risk for hyperkalemia
- Identify the signs and symptoms of hyperkalemia
- Know the steps in emergency stabilisation
- Know the steps in the management of severe symptomatic hyperkalemia
- Know when and how to refer?

4.8 Recognize clinical states at high risk for hyperkalemia (refer box 4.3)

BOX 4.3: High risk states for hyperkalemia

Increased intake	Decreased excretion	Transcellular shifts

• Increased oral or parenteral intake
• Intravenous supplementation
• Packed RBC transfusion
	• Oliguric renal failure
	• Primary adrenal disease
	• Drugs :
	  ○ ACE inhibitors
	  ○ Spironolactone
		• Acidosis
		• Cellular injury :
		  ○ Tumour lysis syndrome
		  ○ Crush injury/Rhabdomyolysis
		  ○ Hemolysis
		• Insulin deficiency
		• Strenuous or prolonged exercise
		• Malignant hyperthermia

4.9 Identify clinical symptoms and signs of hyperkalemia

- Nausea , vomiting , paraesthesia
- High levels cause abnormal heart function and skeletal muscle paralysis by preventing repolarization
- Bradycardia/ Cardiac conduction defects
- Respiratory paralysis

4.10 Confirm hyperkalemia

- Serum potassium >> 5.5 mEq/l is defined as hyperkalemia
- Some patients can be asymptomatic and present only with ECG changes (refer fig A)
    - Serum potassium 5.5-6.5mEq/l: Tall peaked T wave + decreased QT interval **(A)**
    - Serum potassium 6.5- 8mEq/l: Tall peaked T wave + increased PR interval **(B)**
    - This can progress to disappearance of P waves and increased QRS intervals **(C)**
    - Serum potassium > 8mEq/l → QRS and T wave merge to form sinusoidal wave**(D)** followed by asystole

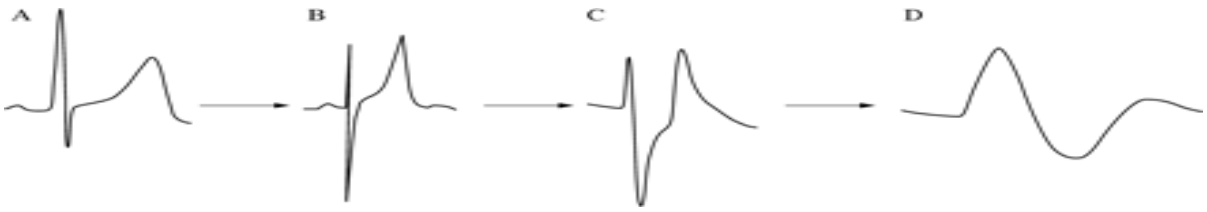


Figure A: Progressive ECG changes in hyperkalemia

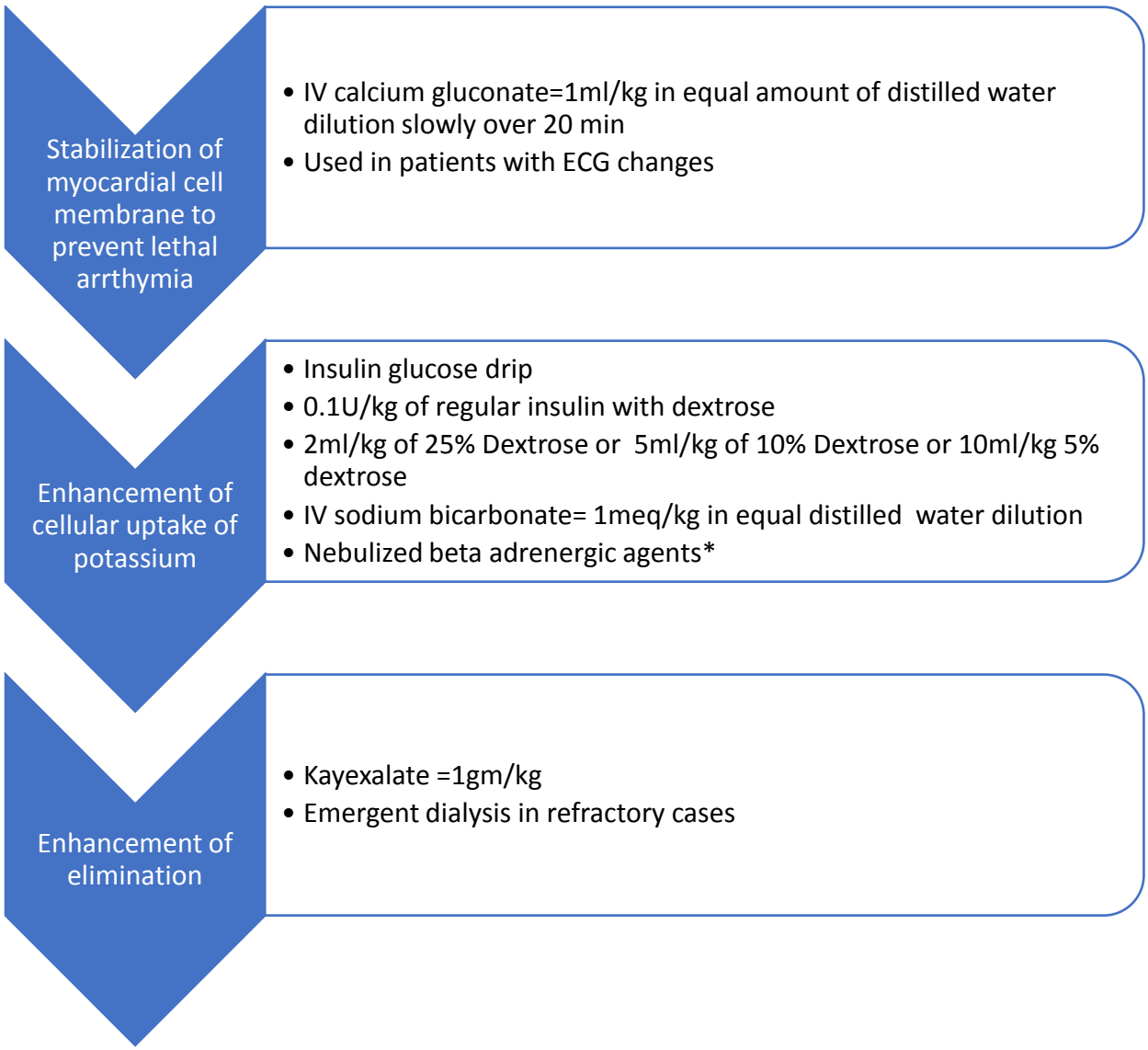
4.11 Approach to a child with hyperkalemia

- Identify clinical states at high risk for hyperkalemia
- In unexplained cases rule out fictitious causes of hyperkalemia (refer box 5.2)
- Confirm hyperkalemia by laboratory levels, if not easily available do ECG and see for progressive changes as described above. The ECG may give a clue to the likely serum level
- Treatment aims at the following domains (refer box 5.3)
    - Stabilizing myocardial cell membrane to prevent arrhythmia
        - IV calcium gluconate
    - Enhancing cellular uptake of potassium
        - IV sodium bicarbonate
        - Insulin glucose infusion
        - Nebulised beta adrenergic agonists
    - Enhancing elimination
        - Potassium binding resins (Kayxelate)
        - Dialysis in refractory cases

BOX 4.4: What is fictitious hyperkalemia ?

- Milking of extremities during phlebotomy or tourniquet application
- Blood sampled upstream of an IV line with potassium rich fluid
- Thrombocytosis (with every 1,00,000/ml elevation in platelets,serum potassium increases by 0.15mEq/L
- Leukocytosis

BOX 4.5 Pharmacologic treatment of hyperkalemia



4.12When to refer?

- Severe hyperkalemia requiring dialysis
- Evaluate required for underlying cause of hyperkalemia

4.13How to refer:

- Always correct the imbalance before referring the child
- Document the treatment given clearly
- Transfer preferably with an ECG monitor

Remember:

- All exogenous sources of hyperkalemia should be immediately discontinued
- Nebulized salbutamol has not been established as first line therapy but can be used as a bridge therapy
- Calcium does not lower serum potassium levels, it primarily protects myocardium from arrthymia. Hence administration of calcium should always be accompanied with drugs that decrease potassium
- Insulin glucose drip
    - 0.1U/kg of regular insulin with dextrose
    - 2ml/kg of 25% Dextrose or 5ml/kg of 10% Dextrose or 10ml/kg 5% dextrose (Remember the rule of 50)

Chart 2.1: Triage of all sick children

ASSESS

ABCD

TREAT

Do not move neck if cervical spine injury possible.
Keep the child warm.

AIRWAY AND BREATHING

- **Obstructed breathing**
  or
- **Central cyanosis,**
  or
- **Severe respiratory distress**

ANY SIGN POSITIVE →

- **IF NOT BREATHING OR GASPING**
  - Manage airway
  - Start life support
- **IF FOREIGN BODY ASPIRATION**
  - Manage airway in choking child
- **IF NO FOREIGN BODY ASPIRATION**
  - Manage airway
  - Give oxygen
  - Make sure child is warm

CIRCULATION

Cold hands with:

- Capillary refill longer than 3 seconds, **and**
- Weak and fast pulse

IF POSITIVE →

Check for severe acute malnutrition

- If the child has any bleeding, apply pressure to stop the bleeding. Do not use a tourniquet.
- Give oxygen
- Make sure child is warm

- **IF NO SEVERE ACUTE MALNUTRITION:**
  - Insert IV line* and begin giving fluids rapidly

- **IF SEVERE ACUTE MALNUTRITION**
  - Give IV glucose
  - Insert IV line and give fluids

COMA/ CONVULSING

- **Coma or**
- **Convulsing (now)**

IF COMA OR CONVULSING →

- Manage airway
- Position the unconscious child (if head or neck trauma is suspected, stabilize the neck first)
- Give oxygen
- Check and correct hypoglycemia
- Give IV calcium if infant <3 months
- If convulsion continue, give anti-convulsant

SEVERE DEHYDRATION (ONLY IN CHILD WITH DIARRHOEA)

Diarrhoea plus any two of these:

- Lethargy
- Sunken eyes
- Very slow skin pinch

DIARRHOEA plus TWO SIGNS POSITIVE →

Check for severe acute malnutrition

- Make sure child is warm

- **IF NO SEVERE ACUTE MALNUTRITION:**
  - Insert IV line and begin giving fluids (NS/RL) rapidly

- **IF SEVERE ACUTE MALNUTRITION:**
  - Do not give IV fluids, give ORS#
  - Proceed immediately to full assessment and treatment

IF THERE ARE NO EMERGENCY SIGNS LOOK FOR PRIORITY SIGNS:
These children need prompt assessment and treatment

PRIORITY SIGNS

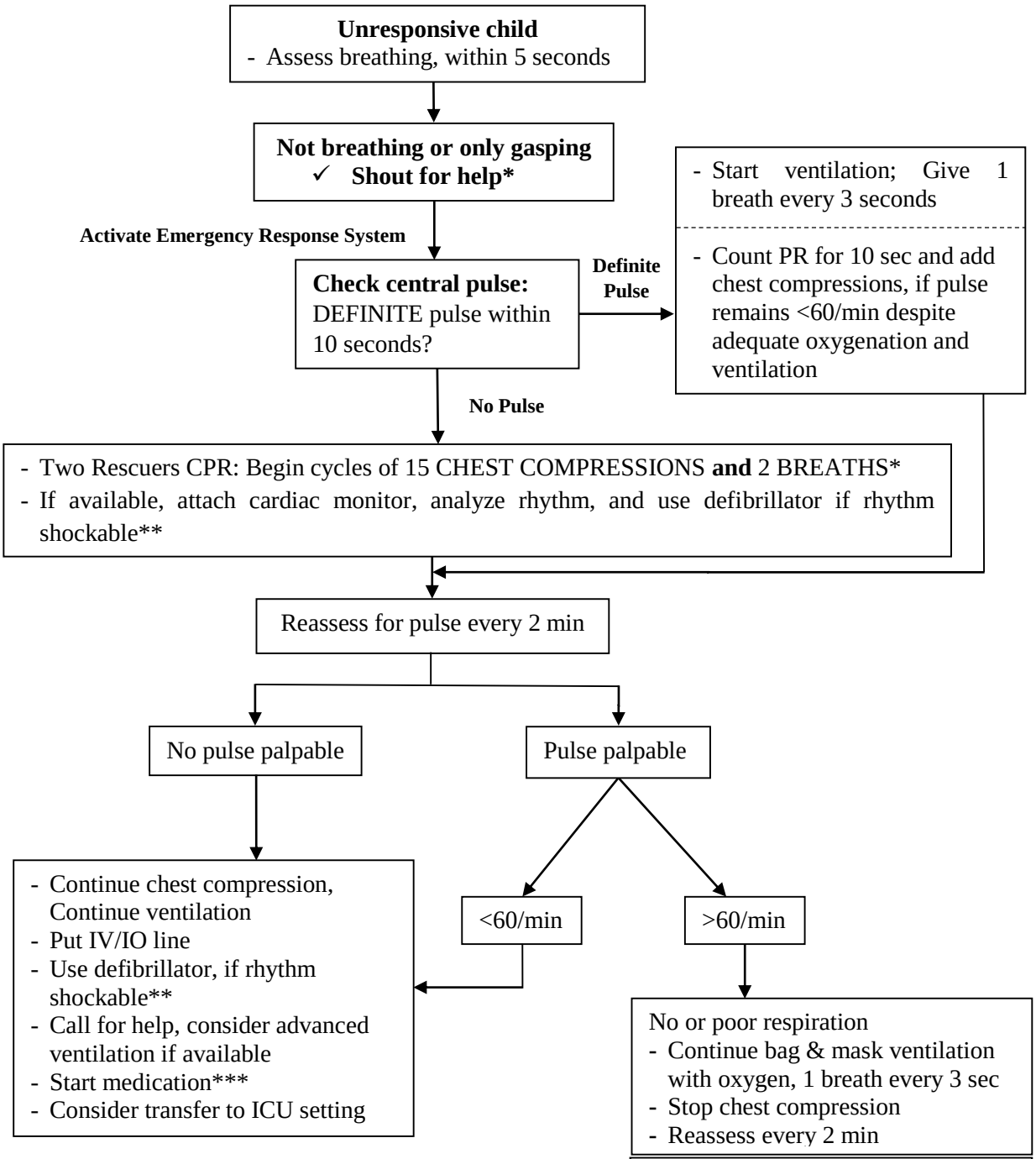
- Tiny baby (<2 months)
- Temperature very high
- Trauma or other urgent surgical condition
- Pallor (severe)
- Poisoning
- Pain (severe)
- Respiratory distress
- Restless, continuously irritable, or lethargic
- Referral (urgent)
- Malnutrition: Visible severe wasting
- Oedema of both feet
- Burns (major)

**If not able to insert peripheral IV, insert an external jugular or intraosseous line.*
 # 5 ml/kg every 30 min for 2 hours

Note: If a child has trauma or other surgical problems, get surgical help or follow surgical guidelines.

NON-URGENT: Proceed with assessment and further treatment according to the child's priority.

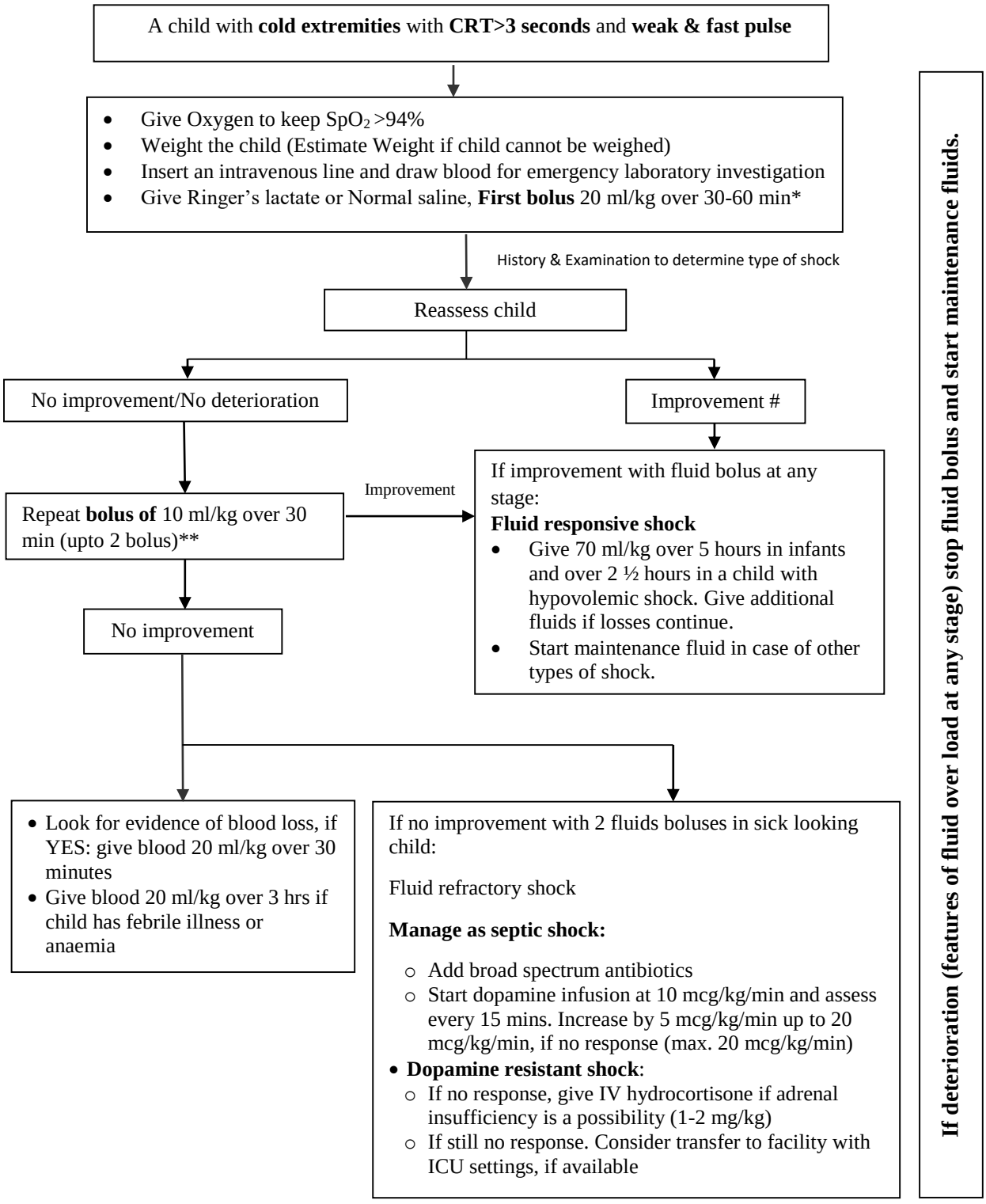
Providing Life Support



**If only one rescuer available give 30 compression and 2 breaths per cycle*
***1st dose 2 J/ Kg; subsequent dose 4 J/Kg*
****Give epinephrine every 3-5 minutes; 0.01 mg/kg (0.1 ml/kg of 1:10000 concentration); max. of 1 mg (10 ml)*

This refers to whether a particular class of **cardiac** dysrhythmia is treatable using defibrillation. The two "shockable" rhythms are **ventricular fibrillation** and **pulseless ventricular tachycardia** while the two "non–shockable" rhythms are **asystole** and **pulseless** electrical activity.

Chart 2.3: How to Give IV Fluids Rapidly for Shock in a Child without Severe Acute Malnutrition



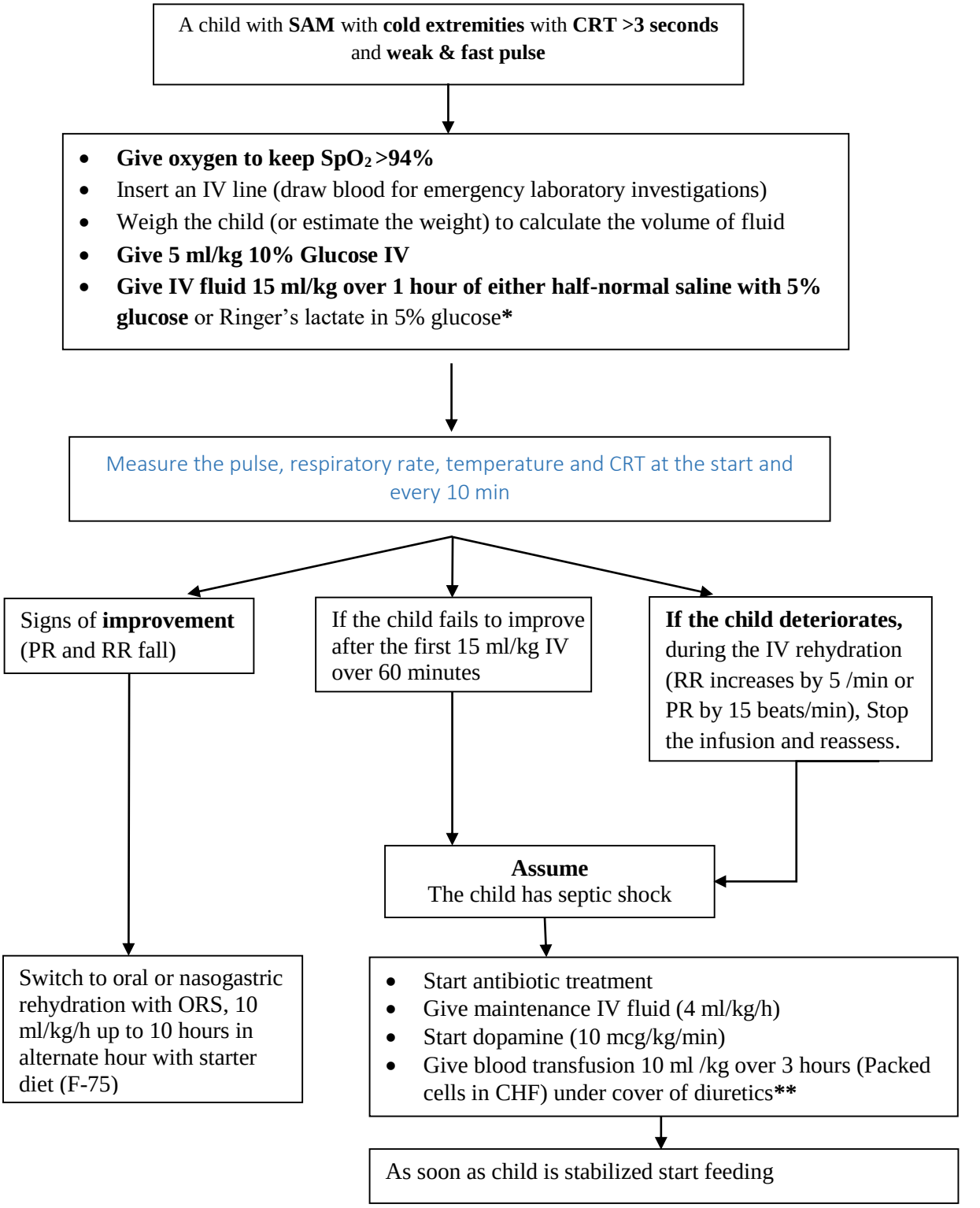
**** Give fast over 15-30 minutes in hypovolemic shock , slow over 60 min if the child has moderate malnutrition or severe pallor or fever***

*****Give 20 ml /kg IV fluid bolus in case of hypovolemic shock***

 # Signs of improvement: Good volume and slowing pulse rate and faster capillary refill.

***** If deterioration (increase in RR > 5 and HR > 15) stop fluid, consider cardiogenic or septic shock.***

Chart 2.4: How to Give IV Fluids for Shock in a Child with Severe Acute Malnutrition



If profuse diarrhoea (more than 10 loose watery stools in last 24 hours), repeat 15 ml/kg of fluid over 1 hour

****The purpose of giving a diuretic during a blood transfusion is to prevent congestive heart failure from overloading the circulation with the transfusion.***