



*Sudan Association of Paediatricians*

# ***Management Protocols for Paediatric Emergencies***

*Second edition*

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**Dr . Layla Ali Abd Al Rahman**  
Head of Advisory Council Ministry  
of Health

**Prof . Zein El Abdeen Karrar**  
President of Medical Council

**Prof . Mohammed Ahmed Abdullah**  
University of Khartoum

**Prof . Salah Ahmed Ibrahim**  
University of Khartoum

**Prof Ali Haboor**  
Dean Faculty of Gazeira University

**Prof . Mabyou Mustafa**  
African International University

**Prof .Hassan Mohammed Ahmed**  
Cancellor, University of Al Zaim Al  
Azhari

**Dr . Soad El Tigani El Mahi**  
Head of Protocols Committee

## *Editors*

**Prof. Zein El Abdeen Karrar**

*Acute liver failure*

**Prof . Mohammed Ahmed Abdulla**

*Head of endocrinology committee*

**Prof . Salah Ahmed Ibrahim**

*Head of respiratory committee*

**Prof . Ali Haboor**

*Head of the tropical diseases committee*

**Prof . Mabyou Mustafa**

*Malnutrition*

**Prof . Hassan Mohammed Ahmed**

*Severe dehydration*

**Dr . El Tigani Mohammed Ahmed**

*Head of nephrology committee*

**Dr . Mohammed Osman Mutwakil**

*Head of poisoning committee*

**Dr . Mohammed Khaleel**

*Head of neonatology committee*

**Dr . Fath Alrahaman El Aawad**

*Head of haematology committee*

**Dr . Abdul Moneim Ahmed Hamid**

*Head of PICU committee*

**Dr . Sulafa Khalid**

*Head of cardiology committee*

**Dr .Yassmin Mahgoub Obeid**

*Management of coma*

**Dr . Safaa Abdul Hameed**

*Hypertension crises*

**Dr . Maha Abdul Moneim El Seed**

*Management of neurological emergencies*

**Dr . Ahlam Abd Al Rahman**

*Management of neurological emergencies*

**Dr . Zeinab Gaily**

*Heat stroke*

## ***Acknowledgment***

Special thanks go to the Paediatric Advisory Committee

Chaired by ***Dr . Layla Ali Abd Rahaman***

And the Protocol Committee chaired by ***Dr . Soad El Tigani El Mahi***

## **Preface**

It is a great honour and pleasure to introduce this manual on protocols of management of paediatric emergencies on behalf of the Advisory Council of Paediatrician. This manual hopefully will build on the success of the first edition of management protocols for paediatric emergencies 2005 published by previous Sudan association of paediatricians & FMOH.

The high mortality & morbidity among children in Sudan make the Advisory Council of Paediatrician face real challenges and have a major role to play in consultation, planning , provision and implementation of health services , in order to provide better quality of care and to ensure an equitable standardized strategy on management of paediatric emergencies . This was first initiated by paediatrician from different parts of Sudan (general paediatrician & subspecialities ) in an attempt to standardize paediatric care in different health facilities and to provide a guidance for doctors of different levels without continuous need for senior consultation in remote areas . Standardization of care provided will also help in medication & drug policy. This uniformity in practice will hopefully translate in improvement of medical outcome.

Regular updating will enhance using new concepts and recent advances in management of emergencies in an attempt to close the gap between vision and reality so that we can proceed towards better quality in management of childhood diseases and regular updating will follow in future .

Lastly thanks are due to all who supported the effort behind these protocols, the genuine contribution and collaboration of all colleagues from different universities and MOH and from different parts of the country is greatly appreciated.

*Dr . Layla Ali Abd Rahaman*

*Chairman of Paediatric Advisory Council*

## ***Introduction***

Paediatric medical problems are one of the major serious health problems in Sudan causing significant hardship morbidity and mortality in children. Data obtained from Sudan house hold survey in 2006 showed the following: Under five mortality rate is 112/1000 live birth and infant mortality rate is 71/1000 live birth, most of them in the first day of life.

This second edition of the protocol has taken a year to complete and has harnessed the efforts of many of our prominent paediatricians and emerging new talents. Three workshops were held, where consultant paediatricians from the capital and the provinces attended, discussed and agreed on the topics included in this edition.

Each topic included has been written by a committee of consultant paediatricians, and the final version was revised, discussed and agreed upon by the editorial board. In this new edition treatment protocols have been updated where required. For quick and easy guidance, protocols have been summarised in easy flow Charts with some texts for further explanation.

The sections on basic and advanced paediatric life support have been expanded which a vital life saving procedure for first line health workers to be able to perform. The fact that even the most basic medical equipments and drugs may not be available at times cannot be ignored , and provision for these eventualities are included in some protocols .

This manual is also an attempt at standardization of paediatric care across treatment centres. Be it a teaching hospital or a rural health centre. It will also provide a guidance for the use of medications, dose calculation and route of administration which help in reducing drug prescription errors. Standardization of care provided will then hopefully translate in improved medical outcome and a reduction in patient morbidity and mortality. Uniformity of practice will also benefit medical students and doctors in training who move between different treatment sites and hospitals .

Our expectation for the success of the protocols is ambitious and we hope it will be widely disseminated and used to achieve the intended goals .

It has been a great pleasure to work with the contributing paediatricians, Quality Control Department and M.O.H and all contributing colleagues and staff of the M.O.H in this worthwhile venture, and I thank them sincerely for their time and effort.

*Dr . Soad El Tigani El Mahi*

*Chairman of Protocol Committee*

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### **Referral**

## **Cardiopulmonary Resuscitation (CPR)**

This guideline is based on the International Consensus on Science published by the American Heart Association in collaboration with the International Liaison Committee on Resuscitation (ILCOR) , evaluations of the science of resuscitation which culminated in the publication of the Guidelines 2000 for Cardiopulmonary Resuscitation , revised 2005, and European Resuscitation Council (ERC) recommendations 2005.

### **Management of cardiopulmonary arrest**

#### **A) Basic Life Support (BLS)**

##### **1. Ensure the safety of rescuer and child.**

##### **2. Check the child's responsiveness.**

- Gently stimulate the child and ask loudly: 'Are you all right?'
- Do not shake infants or children with suspected cervical spinal injuries.

##### **3a if the child responds by answering or moving**

- leave the child in the position in which you find him (provided he is not in further danger)
- Check his condition and get help if needed.
- Reassess him regularly.

##### **3b if the child does not respond - shout for help;**

- Open the child's airway by tilting the head and lifting the chin, as follows:
  - Initially with the child in the position in which you find him, place your hand on his forehead and gently tilt his head back; at the same time, with your fingertip(s) under the point of the child's chin, lift the chin.
  - Do not push on the soft tissues under the chin as this may block the airway; or if you still have difficulty in opening the airway, try the jaw thrust method. Place the first two fingers of each hand behind each side of the child's mandible and push the jaw forward;
    - Both methods may be easier if the child is turned carefully onto his back.

- If you suspect that there may have been an injury to the neck, try to open the airway using chin lift or jaw thrust alone. If this is unsuccessful, add head tilt a small amount at a time until the airway is open.

#### **4. Keeping the airway open, look, listen and feel for normal breathing by putting your face close to the child's face and looking along the chest.**

- Look for chest movements.
- Listen at the child's nose and mouth for breath sounds.
- Feel for air movement on your cheek.

Look, listen and feel for no more than 10 s before deciding.

#### **5a if the child is breathing normally**

- Turn the child on his side into the recovery position (see below).
- Check for continued breathing.

#### **5b if the child is *not* breathing or is making agonal gasps (infrequent, irregular breaths)**

- Carefully remove any obvious airway obstruction;
- Give five initial rescue breaths;
- While performing the rescue breaths note any gag or cough response to your action. These responses or their absence will form part of your assessment of signs of circulation.

#### **Rescue breaths for a child over 1 year are performed as follows.**

- Ensure head tilt and chin lift. Pinch the soft part of the nose closed with the index finger and thumb of your hand on his forehead.
- Open his mouth a little, but maintain the chin upwards.
- Take a breath and place your lips around the mouth, making sure that you have a good seal.
- Blow steadily into the mouth over about 1—1.5 s, watching for chest rise.
- Maintain head tilt and chin lift, take your mouth away from the victim and watch for his chest to fall as air is expelled.
- Take another breath and repeat this sequence five times. Identify effectiveness by seeing that the child's chest has risen and fallen in a similar fashion to the movement produced by a normal breath.

### **Rescue breaths for an infant are performed as follows.**

- Ensure a neutral position of the head and a chin lift.
- Take a breath and cover the mouth and nasal apertures of the infant with your mouth, making sure you have a good seal. If the nose and mouth cannot be covered in the older infant, the rescuer may attempt to seal only the infant's nose or mouth with his mouth (if the nose is used, close the lips to prevent air escape).
- Blow steadily into the infant's mouth and nose over 1—1.5 s, sufficient to make the chest visibly rise.
- Maintain head tilt and chin lift, take your mouth away from the victim and watch for his chest to fall as air is expelled.
- Take another breath and repeat this sequence five times. If you have difficulty achieving an effective breath, the airway may be obstructed.
- Open the child's mouth and remove any visible obstruction. Do not perform blind finger sweep.
- Ensure there is adequate head tilt and chin lift, but that the neck is not over-extended.
- If head tilt and chin lift have not opened the airway, try the jaw thrust method.
- Make up to five attempts to achieve effective breaths; if still unsuccessful, move on to chest compressions.

### **6. Assess the child's circulation. Take no more than 10 s to**

- look for signs of a circulation. This includes any movement, coughing or normal breathing (not agonal gasps, which are infrequent, irregular breaths);
  - Check the pulse but ensure you take no more than 10 s.
- 
- **If the child is aged over 1 year, feel for the carotid pulse in the neck.**
  - **In an infant, feel for the brachial pulse on the inner aspect of the upper arm.**

### **7a if you are confident that you can detect signs of a circulation within 10 s**

- Continue rescue breathing, if necessary, until the child starts breathing effectively on his own.
- Turn the child onto his side (into the recovery position) if he remains unconscious
- Re-assess the child frequently.

**7b If there are no signs of a circulation, or no pulse or a slow pulse (less than 60 /min with poor perfusion), or you are not sure**

- Start chest compressions.
- Combine rescue breathing and chest compressions.

**Chest compressions are performed as follows:**

- For all children, compress the lower third of the sternum. To avoid compressing the upper abdomen, locate the xiphisternum by finding the angle where the lowest ribs join in the middle. Compress the sternum one finger's breadth above this;
- The compression should be sufficient to depress the sternum by approximately one third of the depth of the chest. Release the pressure and repeat at a rate of 100/ min.
- After 15 compressions, tilt the head, lift the chin, and give two effective breaths.
- Continue compressions and breaths in a ratio of 15:2 Lone rescuers may use a ratio of 30:2.
- The best method for compression varies slightly between infants and children.

**To perform chest compression in infants:**

- The lone rescuer compresses the sternum with the tips of two fingers. If there are two or more rescuers, use the encircling technique. Place both thumbs flat side by side on the lower third of the sternum with the tips pointing towards the infant's head. Spread the rest of both hands with the fingers together to encircle the lower part of the infant's rib cage with the tips of the fingers supporting the infant's back. Press down on the lower sternum with the two thumbs to depress it approximately one third of the depth of the infant's chest.

**To perform chest compression in children over 1 year of age:**

- Place the heel of one hand over the lower third of the sternum.
- Lift the fingers to ensure that pressure is not applied over the child's ribs. Position yourself vertically above the victim's chest and, with your arm straight, compress the sternum to depress it by approximately one third of the depth of the chest. In larger children or for small rescuers, this is achieved most easily by using both hands with the fingers interlocked.

**8. Continue resuscitation until**

- The child shows signs of life (spontaneous respiration, pulse, movement).
- Qualified help arrives.
- You become exhausted.

## **When to call for assistance**

It is vital for rescuers to get help as quickly as possible when a child collapses.

- When more than one rescuer is available, one starts resuscitation while another rescuer goes for assistance.
- If only one rescuer is present, undertake resuscitation for 1 min before going for assistance.

To minimise interruption in CPR, it may be possible to carry an infant or small child while summoning help.

- The only exception to performing 1 min of CPR before going for help is in the case of a child with a witnessed, sudden collapse when the rescuer is alone. In this case cardiac arrest is likely to be arrhythmogenic in origin and the child will need defibrillation. Seek help immediately if there is no one to go for you.

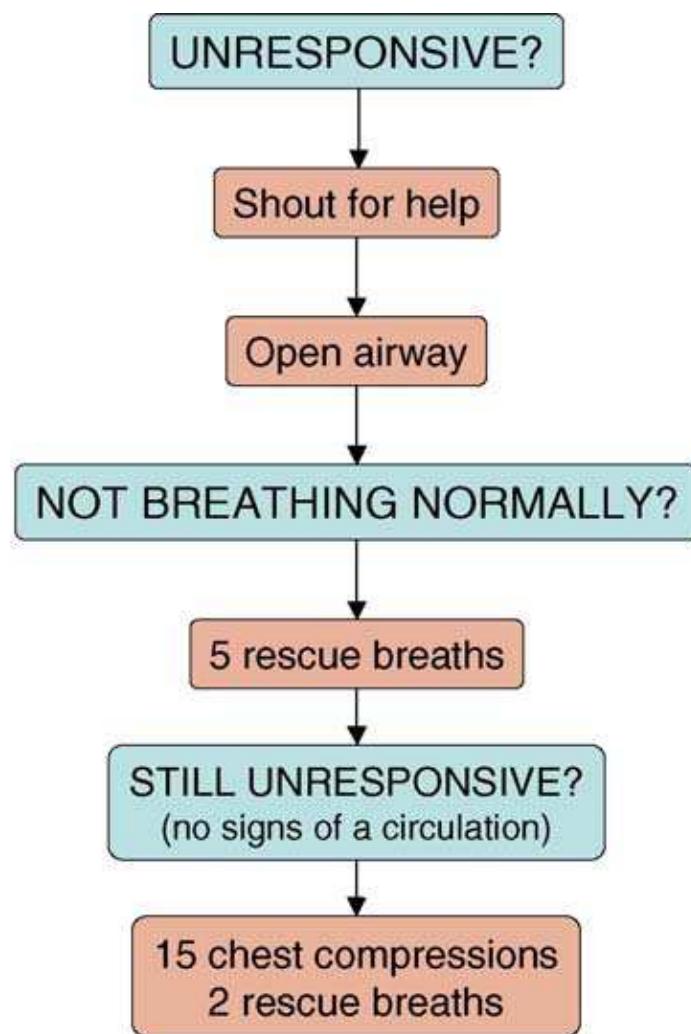
## **Recovery position**

An unconscious child whose airway is clear, and who is breathing spontaneously, should be turned on his side into the recovery position. There are several recovery positions.

- Place the child in as near true lateral position as possible, with his mouth dependent to enable free drainage of fluid.
- The position should be stable. In an infant this may require the support of a small pillow or a rolled-up blanket placed behind the back to maintain the position.
- Avoid any pressure on the chest that impairs breathing.
- It should be possible to turn the child onto his side and to return him back easily and safely, taking into consideration the possibility of cervical spine injury.
- Ensure the airway can be observed and accessed easily.

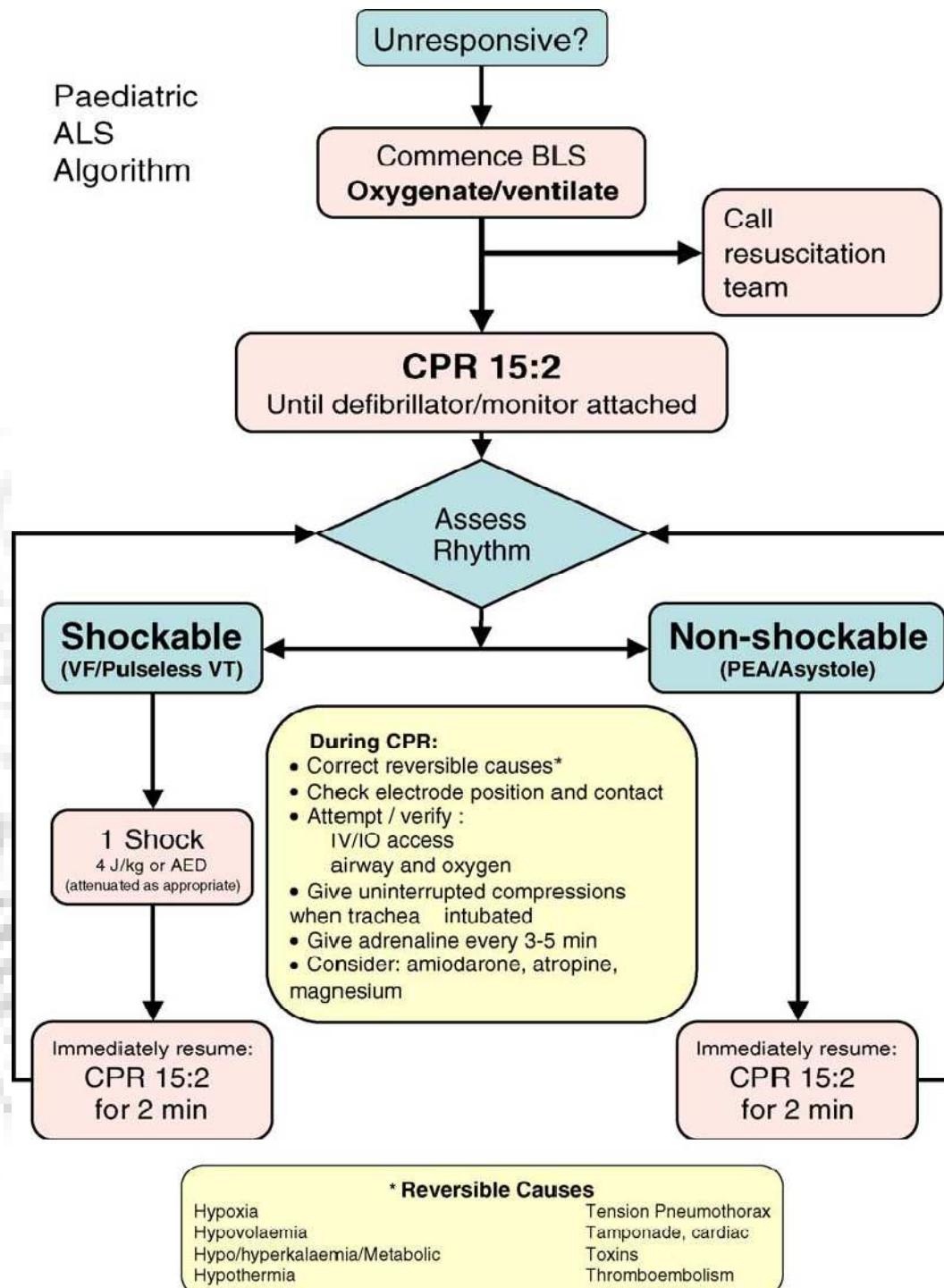
**Fig 1**

**Paediatric Basic  
Life Support  
(Healthcare  
professionals  
with a duty to  
respond)**



After 1 minute call resuscitation team then continue CPR

*Fig2*



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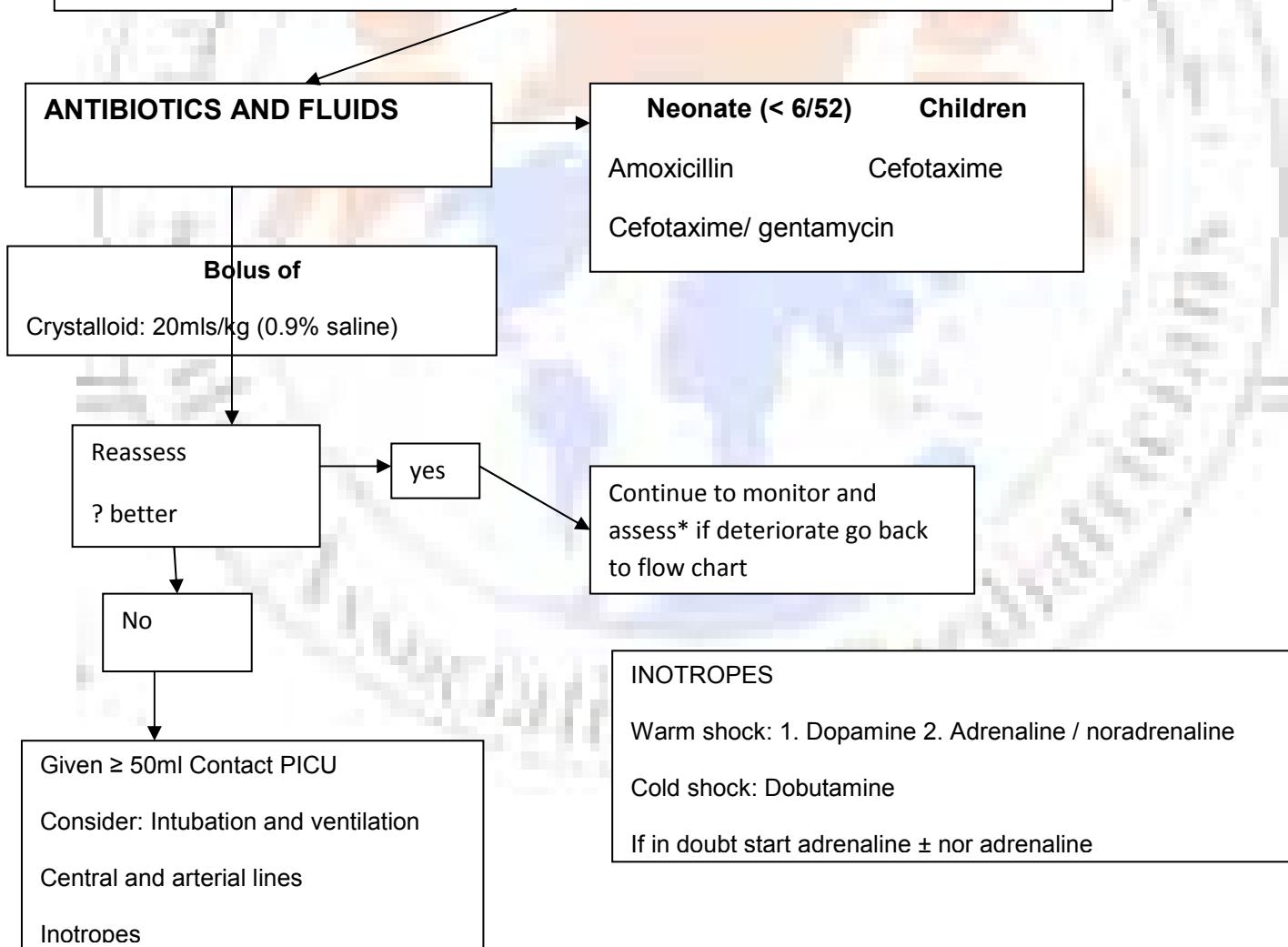
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# shock

- Clear airway
- 100% oxygen
- I.V. access
  - large bore peripheral/saphenous cut-down
  - Intraosseous?
  - Central(femoral/high internal jugular)
- Blood tests
  - ABG/electrolytes/urea/creatinine/blood glucose
  - CBC & differential
  - Coagulation screen(PT,PTT,fibrinogen)
  - Group/ cross match
  - Blood culture

Apply monitors – ECG/oximeter/non-invasive blood pressure, monitor level of consciousness/pupils/hourly urine(catheterise)



## **Antibiotics:**

Neonates (less than 6/52): amoxicillin (to cover Listeria) and cefotaxime or Gentamycin.

- Cefotaxime: 50 mg/kg: < 7/7 old 12 hrly; 7-21/7 old 8 hrly; 21-28/7 old 6-8 hourly.

- Amoxicillin: < 7/7 old 100 mg/kg 6 hrly; 7-28/7 old 100 mg/kg 6 hrly; > 1/12 50 mg/kg 4-6 hrly (max 2 g /24)

- Gentamycin: 2.5 mg/kg: for premature babies: 29/40 - per 24hrs, 29-35/40 - per 18hrs, >35/40 12hrly  
>1/12 – 12yr: 2.5 mg/kg 8hrly.

## **Inotropes:**

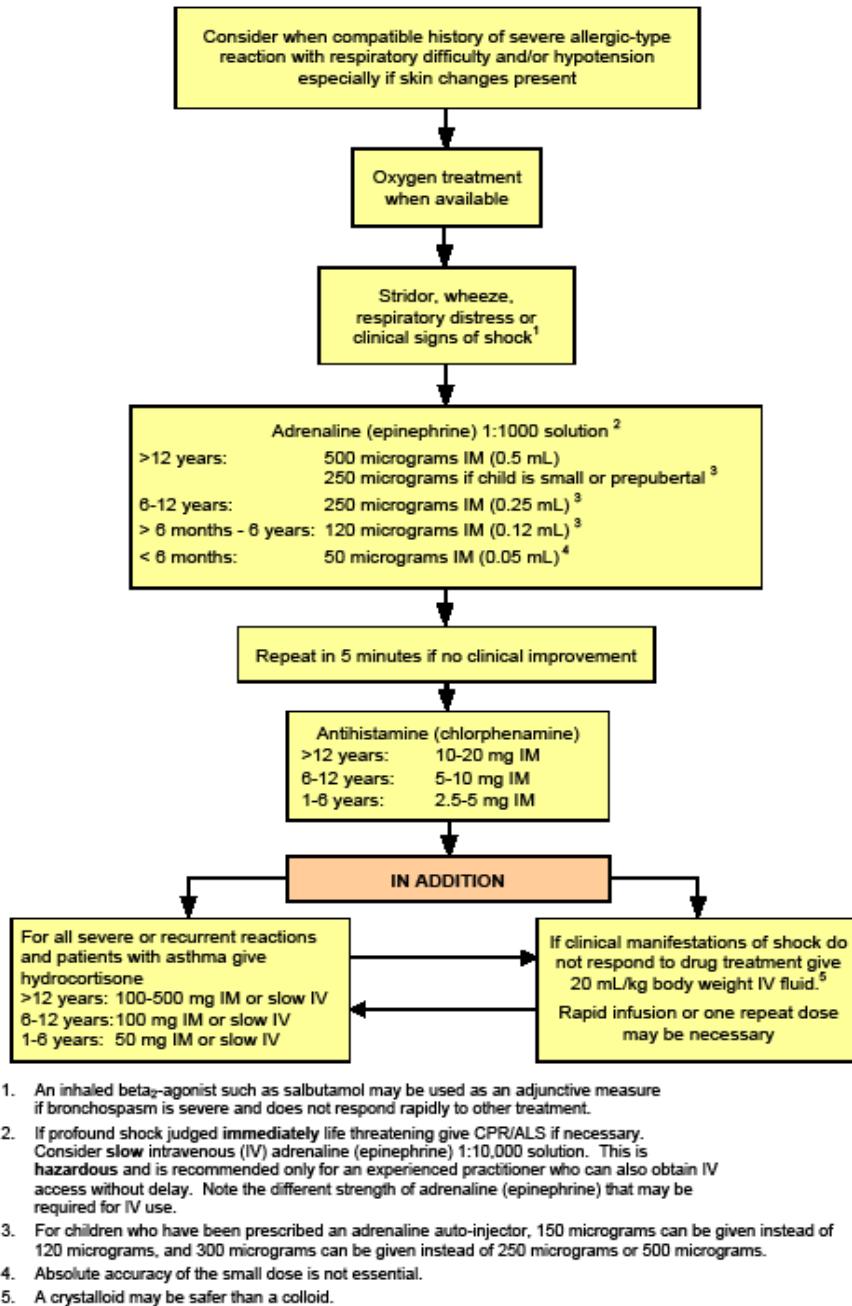
Adrenaline 0.05-1 mcg /kg /min (0.3 X wt in kg = no of mg to be added to 5 or 10 % dextrose to make 50 mls), 1 ml /hr = 0.1 mcg /kg .min.

Nor adrenaline 0.05 - 1 mcg /kg/min (made up as adrenaline) .

Dopamine 2 – 20 mcg / kg / min (30 X wt in kg = no of mg to be added to 5 or 10 % dextrose to make up to 50 mls), 1 ml = 10 mcg / kg / min.

Dobutamine 2.5 – 20 mcg / kg / min (made as dopamine).

**Figure 2 Anaphylactic Reactions: Treatment Algorithm for Children by First Medical Responders**



January 2002  
updated May 2005

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## Management of a comatosed child

Coma is a symptom, not a diagnosis.

The aim of immediate management is to minimise any ongoing neurological damage whilst making a definitive diagnosis. Elements of the history, examination, investigation and treatment will therefore occur simultaneously.

This guideline can be applied to any child with a Glasgow coma score less than 15 or responding only to Voice, Pain or being Unresponsive on the AVPU score.

Glasgow coma scale with modification for children			
<b>Best eye response</b>			
1. No eye opening 2. Eye opening to pain 3. Eye opening to verbal command 4. Eyes open spontaneously			
<b>Best verbal response (use one of the following)</b>		<i>Adult version (aged 5 +)</i>	<i>Children's modification</i>
1. No verbal response 2. Incomprehensible sounds 3. Inappropriate words 4. Confused 5. Orientated	No vocal response Occasionally whimpers and/or moans Cries inappropriately Less than usual ability and/or spontaneous irritable cry Alert, babbles, coos, words or sentences to usual ability	<i>Grimace response for preverbal or intubated patients</i>	No response to pain Mild grimace to pain  Vigorous grimace to pain Less than usual spontaneous ability or only response to touch stimuli Spontaneous normal facial / oromotor activity
<b>Best motor response</b>			
1. No motor response to pain 2. Abnormal extension to pain 3. Abnormal flexion to pain 4. Withdrawal to painful stimuli 5. Localises to painful stimuli or withdraws to touch 6. Obeys commands or performs normal spontaneous movements	<b>AVPU Scale</b> Record the condition which best describes the patient		
	Alert responds to Voice responds to Pain Unresponsive		

### Problem list:

- shock
- Sepsis
- Trauma
- intracranial infections,
- raised ICP,
- hypertension,
- metabolic illness
- prolonged convulsions, post convulsive states

## Immediate management

- Attend to airway, breathing and circulation.
- If trauma cause is possible immobilise cervical spine and arrange urgent neurosurgery involvement.
- Insert i.v. line.
- Perform blood glucose; if glucometer < 2.5 mmol/l in a non-diabetic, send specific bloods tests, administer i.v. dextrose. (See hypoglycaemia guidelines.)
- Consider naloxone 0.1 mg/kg (max. 2 mg) i.v. ± repeat.
- Assess and monitor pulse, respiratory rate, BP, temperature, oximetry ± ECG monitoring and conscious state.
- Look carefully for subtle signs of a continuing convulsion. (See convulsions guidelines)

## History and examination

### Onset and duration of symptoms.

Past history – seizures, diabetes, adrenal insufficiency, infection, cardiac, previous similar episodes (metabolic conditions).

In the presence of	Consider
Scalp bruising or haematoma	Head injury
Inconsistent history, retinal haemorrhage	Non-accidental injury
Fever, seizures	Meningitis, Encephalitis
Focal neurological signs Focal seizures Papilloedema Asymmetric pupils	Focal intracerebral pathology, eg. Tumor
Shunted hydrocephalus	Blocked shunt
Renal disease	Hypertensive encephopathy

## Investigations

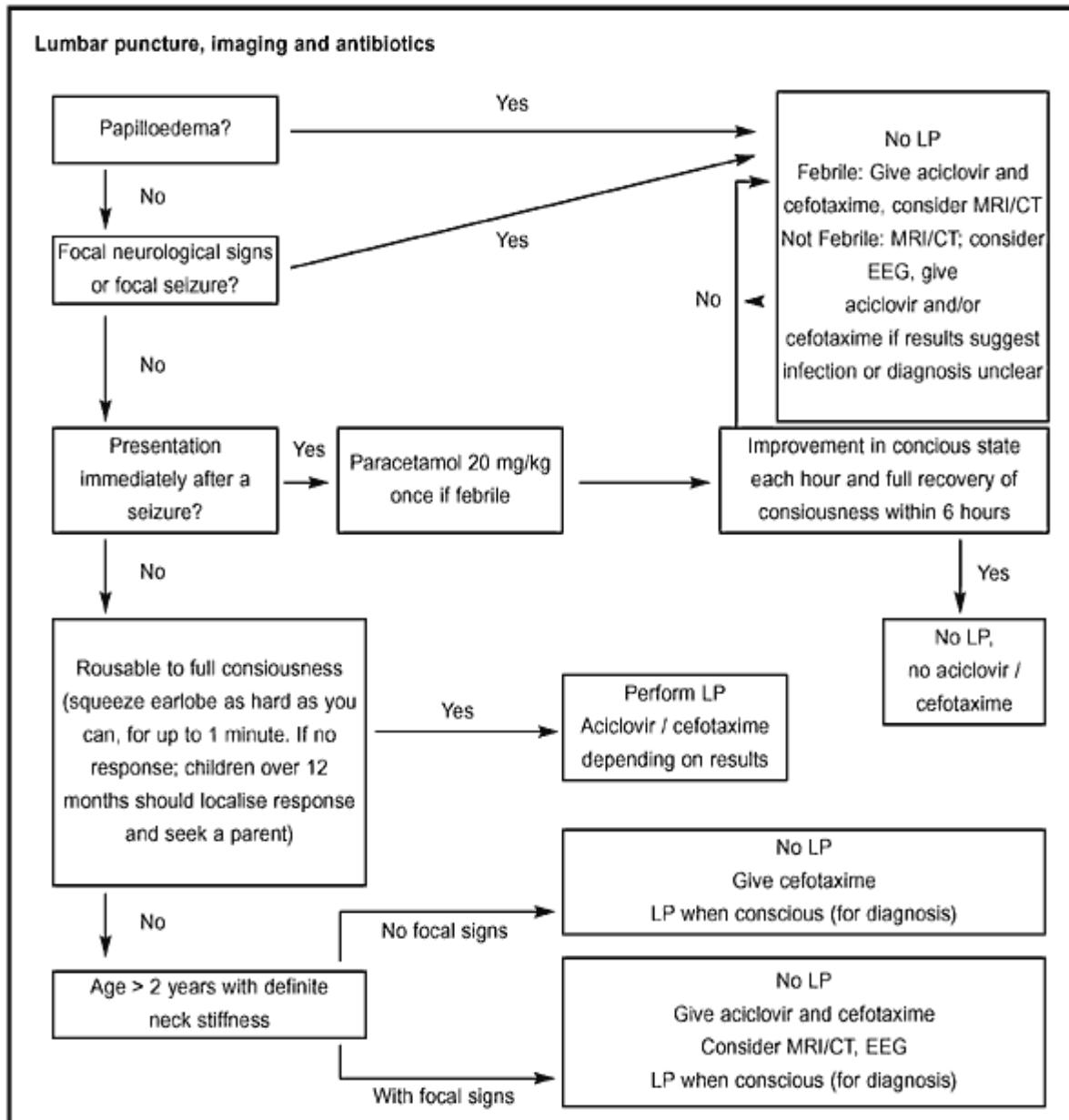
In the light of the possible diagnosis consider these investigations:

- full blood examination
- urea and electrolytes
- glucose
- liver function test
- arterial blood gas
- urine drug ± metabolic screen
- culture of blood and urine
- ammonia
- cortisol
- coagulation screen
- ECG

## Ongoing care

- Will be determined by the diagnosis, level of consciousness and degree of ventilatory and circulatory support needed.

## • Coma Flow Chart



Clinical guidelines RCH (Melbourn)

# **Neurological emergencies**

## **Contents**

- Approach to a convulsing child.
- Management of simple febrile convulsions.
- Status epilepticus protocol.

## **Aim**

- To provide a reasonable guideline for management of simple febrile convulsions based on current scientific evidence.

### **Definition of febrile convolution:**

Convulsion occurring in a child who:

- Is 6 months to 5 years of age and febrile.
- Has no evidence of intracranial infection.
- Has no other defined metabolic disease.
- Is otherwise neurologically normal.
- Has no Past history of afebrile seizures.

Simple febrile convolution:

- Primary generalized convolution.
- Lasts less than 15 minutes.
- Is not repeated within 24 hours.
- All the above mentioned criteria of a febrile convolution definition apply.

This guideline is not about focal or prolonged seizures or a seizure that recurs within 24 hrs. and it is not about febrile status epilepticus

### **Emergency management of seizures:**

A-B-C-D-E.

Maintain vital functions.

Control the convolution.

Identify precipitating factors.

## Approach to convulsing child

If he presents to the health facility convulsing:

Airway- Ensure airway patency; if not consider airway manoeuvre /adjunct .

Look for chest and/or abdominal movements.

Listen for breath sounds-check symmetry

Feel for breath sounds

Gentle suction of the oropharynx.

## Approach to convulsing child

Breathing- RR, recession, accessory muscle use, grunting, chest expansion.

Oxygen saturation if available

High flow oxygen (2-4L/min)-via face mask with reservoir- support breathing with bag-valve-mask device and consider intubation if needed.

Circulation:-Monitor heart rate, pulse volume, capillary refill time, blood pressure, skin temperature and colour.

Shocked ➤ refer to shock management in status epilepticus protocol (will need saline bolus and IV cephalosporin).

## Approach to convulsing child

Get diazepam ready.

Ask for Help.

Calculate the dose-0.5mg/kg PR. (Buccal midazolam can be used at the same dose.).

If weight is not known, use the formula:-

Weight (in kg) = 2(age in years+4) - used for children aged 1-10 years.

Administer diazepam and observe (for 5 mins) if seizure abates place in recovery position if breathing is satisfactory; (**continuous oxygen supply**) if seizure continues refer to status epilepticus protocol.

## Approach to convulsing child

Do not ever forget **blood glucose** – do a bedside glucometer test as a guide,if  $\leq 3$  mmol/l give 5ml/kg of 10% dextrose as soon as iv access is established.

Exposure: - rash, fever (measure the temperature and refer to the fever management protocol).

## Approach to convulsing child

- If the child starts convulsing at the health facility:
- Start your clock.
- A B C D E
- If seizure continues for > 5 min give diazepam (0.5mg/kg PR) or buccal midazolam at the same dose.
- Observe for another 5 mins, if the seizure abates put in recovery position-provided breathing is satisfactory; otherwise refer to the status epilepticus protocol.
- The available midazolam is the IV preparation however it can be used buccally.

## Management of fever

Remove excess clothing.

Put the fan on.

### Antipyretics:-

Paracetamol 10-15mg/kg PO or PR 4-6 hrly

Non-steroidal anti-inflammatory drugs such as Ibuprofen (5mg/kg) 8 hrly.

Ensure adequate fluid intake and correct dehydration

When fully conscious, offer a cold drink / ice-lolly.

**Do not give empirical antibiotics if you are confident about the diagnosis of Simple febrile seizures and there is no obvious bacterial focus.**

**Always** admit after a first febrile convolution.

Subsequent febrile seizures warrant admission if:-

1. The child is ≤ 18 months; meningeal signs are subtle in this group.
2. Seizures are focal and /or last ≥ 15 minutes and/or recur within 24 hours i.e. complex febrile seizure.
3. At any age if there is any suspicion of meningitis/encephalitis
4. Social reasons-anxious parents/inadequate observation at home/ residence far from healthcare facilities.

Once seizure abates, an active search for a focus is advised.

- Good history and thorough clinical examination in pursuit of a cause including an ENT assessment.
- Frequent reassessment of the child is vital!
- Random blood sugar-true lab result
- BFFM

- Urine general and culture
- Consider urea and electrolytes, toxicology screen, throat swab, ASOT as deemed appropriate.
- Lumbar puncture

This should be considered in children with:

- First febrile convulsion  $\leq$  18 month.
- If there is any suspicion of meningitis.
- In infants and children who received recent antibiotic courses.

**Do not do a lumbar puncture in a child with an impaired level of consciousness (SLEEPY/DROWSY) and or has papilloedema and/or focal neurological signs –Do CT brain first!-seek senior opinion.**

### **EEG**

There is no role for EEG in the management of simple febrile seizures as slow wave activities persist for up to 2 weeks following an attack.

Simple febrile seizures have an excellent prognosis.

### **Long term prophylaxis**

Some evidence exists that long term prophylaxis and/or intermittent diazepam therapy can reduce the recurrence of febrile seizures; however the risks outweigh the benefits and it is not recommended at the moment.

### **Immunization**

None of the current standard vaccinations are contraindicated.

### **Parental counseling**

- Reassure and educate.
- Written management plan of the attack should be handed to the parents.
- Fever management at home.
- Emergency management of convulsions; positioning, nothing in the mouth.

### **When to seek help:-**

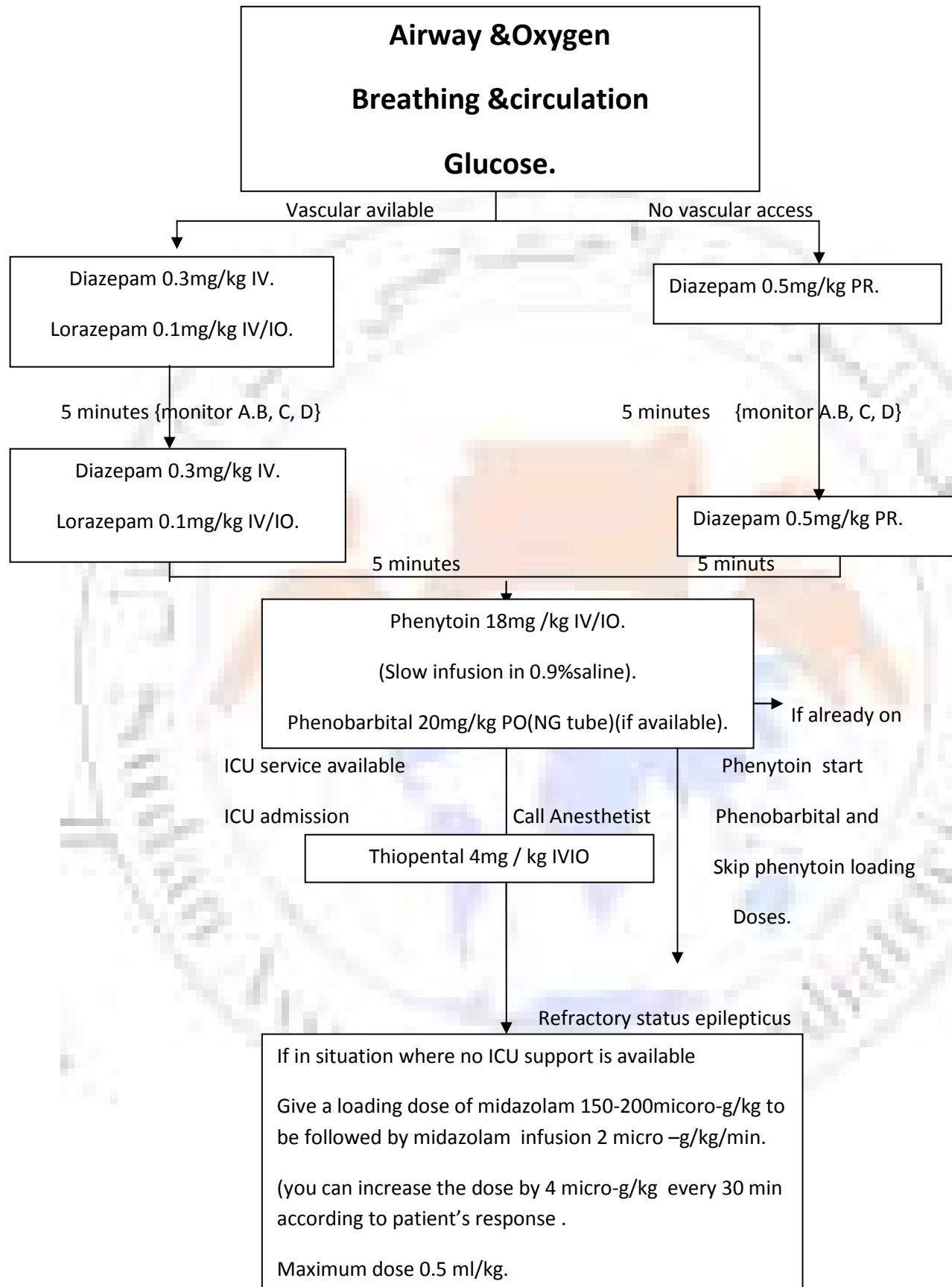
1. Seizures lasting  $\geq$  5 mins.
  2. Lack of normal alertness
  3. Dehydration following diarrhea / vomiting
  4. Non-blanching rash
  5. Fever  $\geq$  5 days
  6. Parental concern
- Beware-recurrent febrile status (seek senior opinion)

## **References:**

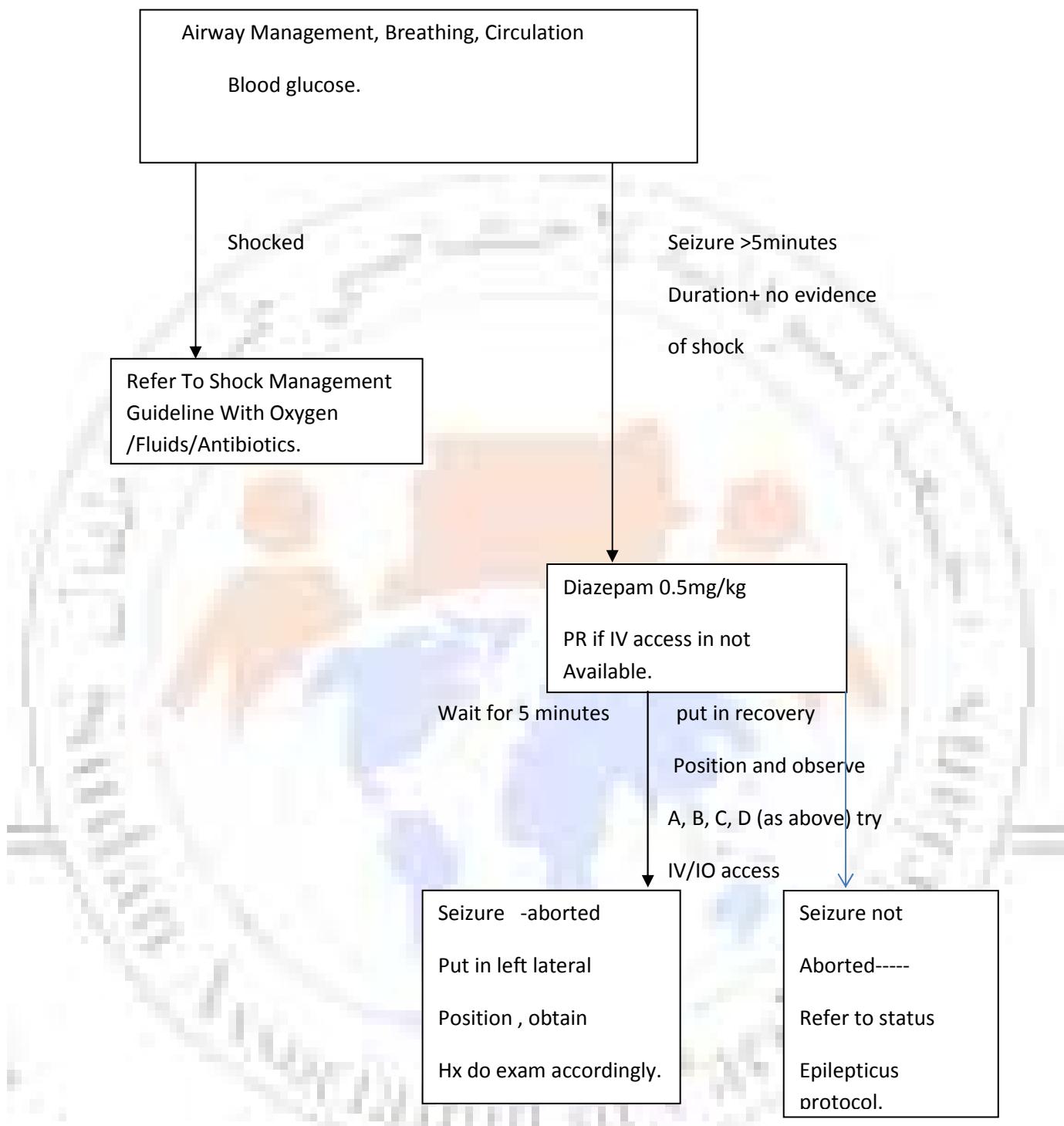
- Advanced Paediatric Life Support Manual-4<sup>th</sup> edition
- Oxford handbook of paediatric neurology by Rob Forsyth and Richard Newton
- American Academy of Paediatrics website



## Status Epilepticus Protocol



## Approach to a child with Febrile Convulsions :-



# **Upper airway obstruction ( stridor )**

## **1. Main causes:**

- Acute laryngeotracheobronchiolitis(croup).
- Acute epiglottitis.
- Retropharyngeal abscess.
- Acute allergic edema (e.g. hair-dye poisoning).
- Diphtheria.
- Foreign body.

## **2. Clinical features:**

**Croup:** barking cough, hoarse voice, stridor developing over several days, congested pharynx.

**Epiglottitis:** fever, drooling of saliva, dysphagia, muffled breath sounds, absence of cough; stridor develops over hours to a day; cherry red epiglottis.

**Retropharyngeal abscess:** fever, neck pain/stiffness, drooling of saliva, dysphagia, stridor, congested tonsils, peritonsillar abscess / bulging posterior nasopharynx.

**Allergic edema (hair-dye poisoning):** angioedema, stridor, dysphagia, drooling, woody tongue, wheezing developing rapidly and shock; develops within minutes to hours.

**Diphtheria:** low grade fever, bull-neck, stridor, dysphagia, drooling saliva, nasal voice, dirty grey membrane over the tonsils. There is usually history of contact and vaccination.

**Foreign body aspiration:** sudden choking followed by stridor and decreased breathing sounds.

## **3. Investigations:**

Chest XR, lateral nasopharynx XR, blood culture, blood gases, urine chromatography.

## **4. Management:**

- Humidified oxygen 6-8 L/min via face mask or nasal prongs.
- Supportive: lowering temperature, adequate hydration and feeding (oral).
- Nebulized epinephrine racemic 0.05 ml/kg to a maximum of 0.5 ml of 2.25% in 2 ml saline nebulized or I-epinephrine (1:1000) 0.5 ml /kg maximum 5ml/dose nebulized can be repeated every 20-30 mins.
- Steroids (dexamethasone 0.15 mg /kg 6 hourly P.O).
- Continuous monitoring.

**If epiglottitis is suspected** (do blood culture).

- a) Do not attempt indirect visualization of the epiglottis in the emergency room.
- b) Take patient to ICU.
- c) IV chloramphenicol 100mg/kg/d or ceftriaxone 100mg/kg/d.
- d) Consider tracheostomy.
- e) Consider intubation and mechanical ventilation if tracheostomy fails or the patient develops respiratory failure.

**If retropharyngeal abscess suspected:**

- a) IV benzyl penicillin 100,000 – 150,000 IU / kg/d.
- b) Consider surgical consultation.

**If diphtheria is suspected:**

(do swab for microbiology "stain and culture").

- a) IV benzyl penicillin 100,000-150,000 IU/kg/d.
- b) IV/IM diphtheria anti-toxin 20,000-120,000 IU single dose after skin test.
- c) Consider tracheostomy.
- d) Consider intubation and mechanical ventilation if tracheostomy fails

**If foreign body is suspected:**

Responsive patients: call for help, meanwhile: for:

- i. Infants (< 1 year): give 5 back blows followed by 5 chest thrust (with head down).
  - ii. Child (1 year to puberty) abdominal thrust (Heimlich's manoeuvre).
- a) Unresponsive patients, call for urgent advanced care and begin CPR and each time you open the airway, deliver 2 breaths and look inside the mouth, if you can see FB remove it, if you can't see it do not blindly try to remove the FB.

**If hair dye poisoning is suspected:**

Call ambulance and refer patient to ENT department immediately, meanwhile

- i. Ensure ABC.
- ii. Gastric lavage.
- iii. Give epinephrine, dexamethasone (see dose above).
- iv. Consider cricotomy or tracheotomy if patient distressed and delay is anticipated.

## **Management of Pneumonia in Children**

### **A. Clinical features :**

Cough, difficulty of breathing, tachypnea, and grunting, intercostal recession, inability to feed, crackles and wheeze.

(Danger signs: cyanosis, apnoea, convulsions, impaired consciousness).

### **B. Indications for hospitalization**

1. All patients with danger signs.
2. Toxic appearance.
3. Hypoxemia (Oxygen Saturation < 90%).
4. Severe respiratory distress (Apnoea, grunting, chest indrawing, head nodding).
5. Dehydration with Vomiting or poor oral intake.
6. Immunocompromised patients.
7. Pneumonia refractory to oral antibiotics.
8. Unreliable home environment.

### **C. Diagnostic studies :**

Chest XR, CBC.

### **D. General management :**

- Supportive: lowering temperature, adequate hydration and feeding (oral).
- Humidified oxygen 6-8L / min via face mask or nasal prongs.
- Continuous monitoring.

#### **D1. Management of infants under one year**

(Bacteria: Escherichia coli, Group B streptococci, Listeria monocytogenes, Haemophilus influenza type b, Staph aureus).

- Admit all newborns / infants with danger signs.
- Antibiotic regimen (consider antibiotic combinations).  
Ampicillin 50-200 mg/kg divided q12 hours.  
Gentamycin 2.5 mg/kg repeated q8-12 hours.  
Cefotaxime 100-150 mg/kg divided q8 hours.

- **Organisms requiring additional antibiotic coverage**

Methicillin Resistant Staphylococcus Aureus (MRSA) Vancomycin.

- **Outpatient ( if a febrile without respiratory distress )**

Amoxicillin 50-90mg/kg/day.  
Amoxicillin – Clavulanic Acid 50-90 mg / kg / day.  
Erythromycin 30 – 40 mg / kg / day PO divided q6 hours ×10d.  
Azithromycin 10 mg / kg day.

**D2. Management of children aged > 1 year**  
(Bacterial: S.pneumonia, Chlamydia pneumonia)

- **Inpatient** ( if febrile or hypoxic )  
Benzyl penicillin 100. 000 -150. 000 IU/kg /d.  
Cefotaxime 100 mg / kg / day IV or  
Ceftriaxone 100 mg / kg / day IV divided q8 hours or
- **Outpatient** ( if febrile without respiratory distress )  
Amoxicillin 50 – 90 mg / kg / day.  
Amoxicillin – Clavulanic Acid 50 – 90 mg / kg / day.  
Erythromycin 30 – 40 mg / kg / day.  
Azithromycin 10 mg / kg day.  
Clarithromycin 15mg /kg/day

## **Acute bronchiolitis**

**Definition** infection caused by Respiratory Syncytial Virus.

### **Clinical features**

Cough, fever, runny nose, tachypnoea, tachycardia, intercostal recession, crackles and wheeze.

### **Diagnostic studies**

Rapid antigen detection for RSV from nasopharyngeal secretions.

### **Management**

- Humidified oxygen 6 -8 L / min via face mask or nasal prongs.
- Hydration.
- Continuous monitoring.
- Consider mechanical ventilation

## **Acute asthma**

### **Definition:**

A Clinical syndrome of recurrent cough, wheeze, tachypnoea and chest tightness.

### **Clinical features:**

- Tachycardia.
- Restlessness.
- Tachypnoea and prolonged expiration.
- Wheeze (could be audible) and mainly expiratory.
- Chest deformity: pigeon chest, barrel chest, Harrison Sulcus.
- Hyper resonant chest.

### **Diagnostic studies:**

- Lung function test :
- Reduced FEV<sub>1</sub> by 20%.
- Reduced PEFR by 20%.
- Response (FEV<sub>1</sub>, PEFR) to bronchodilator by > 15%.
- Blood gases (Sat O<sub>2</sub> > 90%).

### **Management:**

- a. Humidified oxygen 6 – 8 L/ min via face mask or nasal prongs.
- b. Hydration.
- c. Nebulized Salbutamol 2.5 mg ( for children < 5 years ) & 5.0 mg ( for children > 5 years ) in 3ml saline to be nebulized over 5 minutes using face mask OR
- d. Salbutamol by MDI 6 – 8 puffs via spacer OR
- e. Epinephrine 1: 10,000 subcutaneously 0.1 mg / kg.
- f. Reassess for: restlessness, wheeze, RR, PR and air entry.
- g. If no response, repeat (c) after 1 \ 2 hour.
- h. Reassess (f) after another 1 \ 2 hour.
- i. If no response: repeat (c) and start steroids (Hydrocortisone 100 – 300 mg IV), start Prednisolone. 2 – 4 mg / kg stat; continue Prednisolone 2 mg / kg / day for three days.
- j. A child who does not respond to 3 doses of nebulized salbutamol should be considered as acute severe asthma (status asthmaticus).
- k. Continuous monitoring.

## **Acute severe asthma / life threatening asthma**

### **Definition:**

Is an acute asthma that does not respond to the (usual) outpatient treatment of the child or did not respond to three doses of nebulized Salbutamol within two hours.

Life – threatening asthma is asthma that endangers life (cyanosis, drowsiness, and silent chest)

### **Clinical features:**

- Severe respiratory distress.
- Inability to talk or drink.
- Tachypnoea and Severe tachycardia.
- Impaired consciousness.
- Pulsus paradoxus.
- Exhaustion.

### **Diagnostic tests:**

- Lung function test.
- Reduced FEV<sub>1</sub> by > 20%.
- Reduced PEFR by > 20%.
- Blood gases (Sat O<sub>2</sub> < 90%).

### **Management:**

- Admit to I.C.U or high care area (continuous monitoring).
- Humidified Oxygen at 6 – 10 L / min.
- Continuous nebulization of Salbutamol nebulized solution 0.25 mg / kg / hr.
- I.V Hydrocortisone (2 – 4 mg / kg / dose 4 hourly).
- Nebulized ipratropium hydrochloride (15 mcg in 3 ml saline over 5 – 7 minutes), 4 – 6 hrly.
- Subcutaneous adrenaline (0.5 ml (1: 10000) half – to – one hourly (three doses)).
- I.V magnesium sulphate (50 – 100 mg / kg).
- Consider Isoprenaline infusion.
- Reassess half – hourly.
- Consider transfer to the ICU.
- Consider mechanical ventilation.

# **Respiratory failure**

## **Definition:**

Is the inability of body to adequately oxygenate and / or ventilate.

## **Common Causes :**

- **Upper airway obstruction**
  - **Croup syndrome**
    - Laryngeotracheobronchiolitis, retropharyngeal abscess,
    - Diphtheria, epiglottitis.
    - Foreign – body aspiration.
    - hair – dye poisoning
- **Lung disease**
  - Acute severe asthma.
  - Bronchiolitis.
  - Severe pneumonia.
  - Pulmonary edema.
  - Near drowning.
- **Sepsis.**
- **Central.**
  - CNS infection.
  - Drug overdose.
  - Stroke.
  - Traumatic brain injury.

## **Clinical features:**

Type 1 respiratory failure (hypoxemia):

- Anxiety, severe tachypnoea, tachycardia, and pallor.

Type II respiratory failure (hypoxemia and hypercarbia),

- Cyanosis, bradycardia, disturbed level of consciousness, and cardiac arrest.

## **Diagnostic studies:**

- **Type I**

ABG (pa CO<sub>2</sub> <40 mm Hg, pa O<sub>2</sub><80-90 mm Hg, or arterial oxygen saturation less than 90% %)

- **Type II**

ABG (paCO<sub>2</sub> >50 mm Hg, paO<sub>2</sub> <60 mm Hg, or arterial oxygen saturation less than 90%).

**Management:**

- Admission to the ICU.
- Ensure ABC.
- Intubation and mechanical ventilation.
- Ensure adequate oxygenation.
- Determine and treat the underlying cause.



## **Oxygen therapy**

### **Indication:**

- Respiratory problems.
- Shock, seriously ill or injured patients with respiratory insufficiency.

### **Oxygen delivery; device , flow, and concentration :**

Device	O2 flow RATE	FiO2
Nasal cannula (low flow device)	1.litre/min	21-24%
	2. litre/min	25-28%
	3. litre/min	29-32%
	4. litre/min	33-36%
Standard face mask (low flow device)	6-10liters/min	25-60%
Partial rebreathing mask	10-12 liters/min	30-60%
Non-rebreathing mask	6 litre/min	60%
	7 litre/min	70%
	8 litre/min	80%
	9 litre/min	90%
	10-15 litre/min	95-100%
Face tent	10-15 litre/min	40%
Oxygen hood	10-15 litre/min	80-90%
Oxygen tent		50%
Venture mask		24-56%

NB: Nasal cannula: 4L/min the maximum flow rate for children (6L/min in adults).

### **Reference:**

1. McIntosh ( 2002 ) N Engl J Med 346:429.
2. Nelson ( 2000 ) Pediatr Infect Dis 19:251.
3. Ostapchuk ( 2004 ) Am Fam Physician 70(5):899

# Management of Paediatric Patients with Heart Failure

Congestive heart failure (CHF) is a clinical diagnosis

Respiratory distress, tachycardia, hepatomegaly and cardiomegaly (in infants), oedema, raised jugular venous pressure and basal crepitation (in older patients).

- depends on underlying condition, skin perfusion and temperature, urine output, oedema.

Always check the femoral pulse for coarctation of the aorta.

- Chest x ray: helps confirm diagnosis and assess severity.
- Echo to know the cause of HF rather than diagnose it.  
DO NOT DELAY MANAGEMENT TILL AFTER ECHO.

## 2. Management, monitoring and follow up:

### Supportive management:

- O<sub>2</sub>.
- Bed-rest in cardiac position.
- Fluids: 2/3 of maintenance NGT/IV.
- Blood/PRBC transfusion: patients with HF and HB below 8gm% small volumes over 4 hours with monitoring.
- Antibiotics in patients with suspicion of infection.

### Specific Management:

#### Mild-moderate:

- Diuretics: Start with Furosemide 1-3 mg/kg/day orally /IV.
- Use angiotensin converting enzyme inhibitor as second line after diuretics (Captopril/enalopril) captopril dose: 0.2 mg/kg/dose BD/TDS increase gradually to 4 mg/kg/day.

Monitor blood pressure esp after the first few doses.

- **Do not use ACE** inhibitors in patients with obstructive lesions like AS and HOCM.
- If the patient is still significantly symptomatic or tachycardic Digoxin can be added, dose: 3-5 micrograms/kg/dose orally 12-24 hourly.
- Potassium supplement is not needed with the above combination (Lasix, captopril) unless the serum potassium is low.
- Add K-sparing diuretic (e.g. spironolactone) in cases of refractory heart failure/oedema.

### Heart Failure in Cardiomyopathies/Myocarditis:

- Same treatment as above.

### Consult cardiologist before further treatment

- Use of IVIG for acute myocarditis: IVIG still debatable but can give benefit of doubt if history suggests acute disease. Dose 2gm/kg/dose over 12 hours.
- Add beta blockers (carvedolol dose 0.2mg/kg/dose) increase gradually according to the response.
- Add aspirin (3-5 mg/kg/d) for patients with EF<30.
- Add warfarin in patients with H/O cerebrovascular accident or left ventricle clot seen on echo, monitor INR.

### Heart Failure in Acute Rheumatic Carditis:

- Diagnosis is clinical applying the **modified Jones' Criteria** plus lab evidence of streptococcal infection.
- In mild-moderate carditis: Bed rest, penicillin, aspirin (75-100mg/kg/d) 6 hourly and anti-heart failure medications.

### Indications for steroids:

- In severe or refractory carditis.
- Moderate- large pericardial effusion.

## **Dose:**

PO Prednisone 2 mg/kg/d for 2 weeks then aspirin 60mg/kg/d is added.  
Steroids tapered over a week and aspirin alone continued.

- Aspirin is tapered gradually guided by ESR.
- Penicillin prophylaxis 3 weekly continued for life in case of carditis with residual valve lesion and for 18-25 years if there is no cardiac involvement.

SBE prophylaxis on indications.

## **Heart Failure in Patients with established Rheumatic Valvular Disease:**

**Is it a new episode of rheumatic fever??**

**(ESR, ASO, ECG, repeat echo).**

**You need to have 2 minor criteria plus evidence of strep infection to diagnose ARF in this category.**

- Diuretics +/- captopril combination in patients with MR,AR.
- Assess the ventricular dimensions and EF periodically.
- Add Propranolol or digoxin in patients with atrial fibrillation.

## **Management of Supraventricular and Ventricular Tachycardia**

### **Supraventricular Tachycardia:**

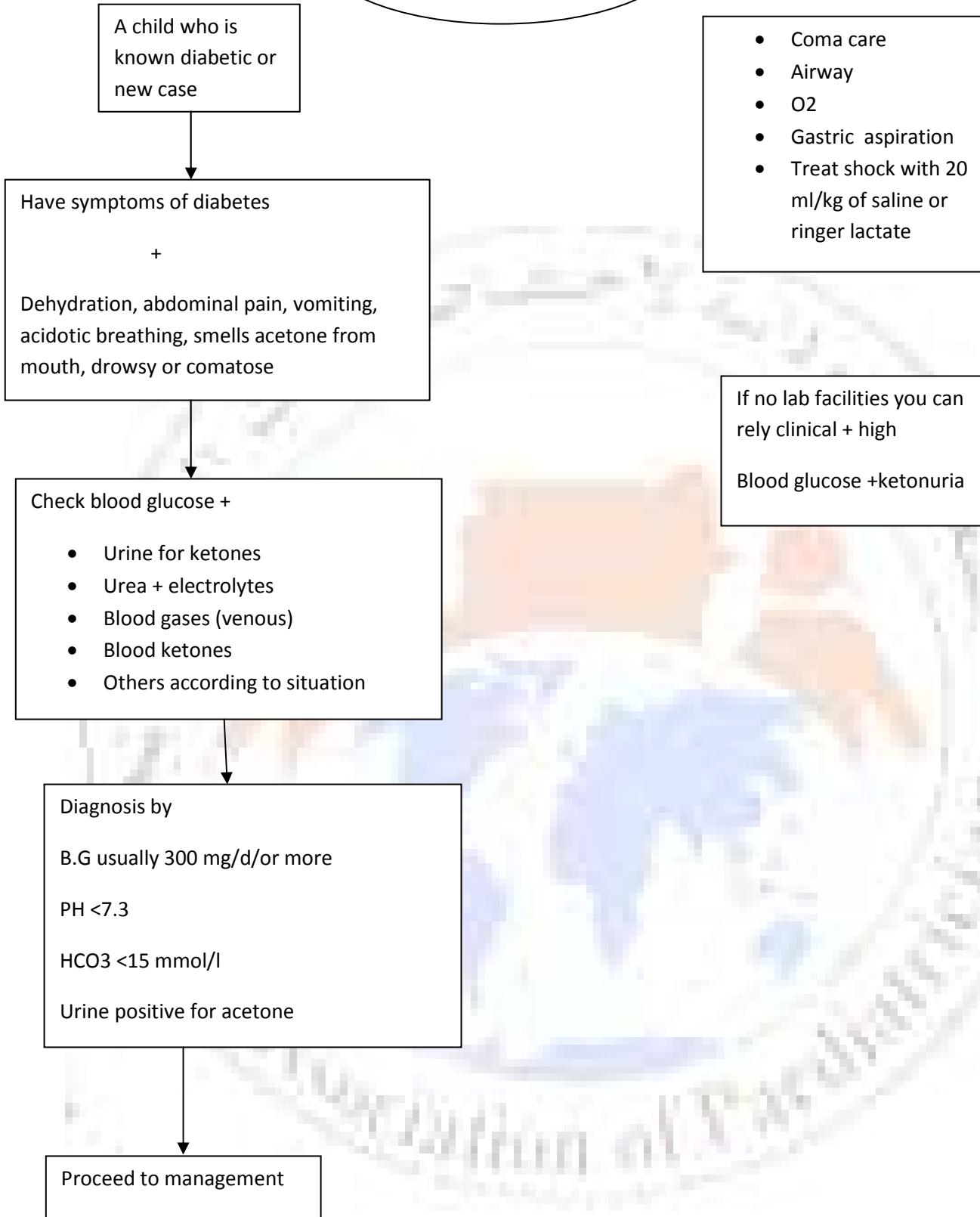
**(Narrow QRS, fast and regular)**

Most common is atrioventricular re-entry tachycardia due to accessory pathways. 2 peaks one early in infancy and one in older children, where it can be due to AV node re-entry tachycardia.

- **MANAGEMENT:**

1. Obtain an ECG strip to document the arrhythmia.
2. If hemodynamically unstable: resuscitation, (airway, breathing, circulation).  
PLUS a synchronized DC shock (see chart).
3. If hemodynamically stable: start with vagal manoeuvres (unilateral carotid sinus message / ice-cold water immersion, for older children Valsalva manoeuvre) while inserting an IV line.
4. Use beta blocker: Propranolol, or Amiodarone can be used, Digoxin if not known WPW.
5. Verapamil (caution: not to be used for under one year of age).

# DKA in children



**DKA fluid Therapy per 24 hours (Maintenance + ½ deficit)**

Body wt	Maintenance ml/24 hours	Deficit ml/24 hours	m/hour (Approx)
4	400	200	25
5	500	250	30
6	600	300	35
7	700	350	40
8	800	400	50
9	900	450	55
10	1000	500	60
11	1050	550	65
12	1100	600	70
13	1150	650	75
14	1200	700	80
15	1250	750	85
16	1300	800	85
17	1350	850	90
18	1400	900	95
19	1450	950	100
20	1500	1000	105
22	1540	1100	110
24	1580	1200	115
26	1620	1300	120
28	1660	1400	125
30	1700	1500	130
32	1740	1600	140

34	1780	1700	145
36	1820	1800	150
38	1860	1900	155
40	1900	2000	160
45	2000	2250	175
50	2100	2500	190
55	2200	2750	200
60	2300	3000	220
66	2400	3250	235
70	2500	3500	250
75	2600	3750	265
80	2700	4000	280

# Management of DKA

1. Establish diagnosis.
2. ABC + coma care / shock management.
3. Fluids:

For **volumes** and rate see table.

Start with normal saline +potassium then change to 5%dextrose with 1 / 2 normal saline with potassium when B.G. is about 250-300 mg/dl.

**Potassium:** add 30-40 mmol/l of kcl after patient starts urinating or if K+ low.

**Bicarbonate:** not recommended except rarely (see text).

4. If no I.V fluids available give ORS 5ml/kg /hours orally or through N/G.

**5. Insulin:**

- Use regular insulin 0.1 unit/kg/hour as continuous infusion if you have pump till patient recovers from DKA.
    - If no pump give 0.3 units/kg S/C then 0.1 unit/kg /hour SC or can start 0.1 unit /kg/ hour SC .after 2 hours of I.V.fluids.
    - Continue hourly insulin till patient recovers from DKA i.e. (clinical PH>7.3, HCO<sub>3</sub> >15, urine no or trace of ketone. OR clinical +no urine ketone in afresh sample.
- 6. Monitoring (see flow sheet )**
- 6.3 blood sugar hourly (by meter) and urine ketones till acidosis is cleared.
  - 6.4 vital signs 1-2 hourly.
  - 6.5 fluid intake and output.
  - 6.6 watch for brain oedema (for management see text).

7. For further management after recovery from DKA (see text).

## Diagnosis and management of diabetic ketoacidosis in children

### Definition:

DKA is defined as significant hyperglycemia (blood glucose > 17 mmol/L or 300 mg/dl), ketonemia, and metabolic acidosis (pH < 7.3, HCO<sub>3</sub> < 15 mmol/L) , coupled with severe disturbance in fluid and electrolytes balance. (Severe DKA = PH < 7.1 HCO<sub>3</sub> < 5 mmol/L), in absence of these lab facilities consider the patient to have DKA if he is symptomatic for diabetes and is dehydrated with hyperglycemia and glycosuria with ketonuria.

**1. Diagnosis and Assessment (see appendix I)**

- Think of DKA in a known diabetic child or any child who presents with either of or a combination of the following.
- Classical symptoms of diabetes.

- Acute abdomen.
- Dehydration.
- Acidotic breathing.
- Disturbed level of consciousness.

Important Relevant Points:

1<sup>st</sup> history:

- Precipitating factors including insulin omission, accuracy of dose, stressful conditions including infections, trauma or home conflict.
- History of recent weight and weight loss.

2<sup>nd</sup> examination:

- Signs and complications of fluid, electrolyte and acid base imbalance including shock, hypotension with acidosis, and CNS status. Establish degree of dehydration (mild, moderate and severe).
- Look for signs of hidden infection and trauma.
- Obtain accurate weight before starting treatment if possible.

## **II. Principles of Management:**

- Treatment of shock.
- Correction of dehydration and replacement of losses with provision of maintenance.
- Correction of electrolyte deficit.
- Correction of hyperglycemia.
- Correction of acidosis.
- Treatment of precipitating factors including sepsis.
- Observation for and treatment of cerebral oedema.
- Prevention of further attacks.

## **III. Management**

### **A. Immediate:**

1. Coma care if child is comatose (gastric tube if abdomen distended).
2. Assess and control breathing and circulation (including oxygen therapy and shock management).
3. Start two IV lines. Line one is for fluid and electrolyte replacement and line 2 for insulin infusion.
4. Lab:
  - a. Blood for: glucose, urea, creatinine, electrolytes, gases, CBC + diff, (culture: if indicated), others if needed e.g. BF for malaria.
  - b. Urine: urinalysis + culture (if indicated).

Notes:

- If a child is comatosed, urine can be obtained by catheter. In infants you can squeeze the napkin urine.
  - Both hyperglycemia (using glucometer) and glycosuria and ketonuria (with strip) should be performed by the doctor in the ER without waiting for the laboratory results to take action.
5. Fluids:
    - If patient is shocked give 10 ml/kg of normal saline (or ringer lactate) as quickly as possible (20 – 30 minutes). Repeat these doses till circulation is restored in the emergency room. This should not later be subtracted from fluid therapy.
    - If not shocked or once circulation is restored, start IV fluids as mentioned below.
    - No need to give bolus saline if the patient is not shocked or hypotensive.
  6. Insulin: no need to give IV insulin bolus but start I.V. insulin infusion 0.1 unit/kg/hour or 0.3 units/kg/s/c.  $\frac{1}{2}$  - 1 hour after starting of fluids.
  7. Disposition:
    - PICU: Those with coma, young infants, cardiovascular instability, and those who deteriorate in the ward.
    - Ward: other cases.
    - Home: Some known cases with mild ketoacidosis or ketonuria can be managed at home (see later →)

B. Further Management during Ketoacidosis (see Appendix 2)

B.I Fluid Replacement

- Fluid repair should extend over 48 hours to achieve a slower correction of serum hyperosmolality to prevent cerebral oedema. Therefore deficit should be given over 48 hours (i.e. give  $\frac{1}{2}$  the deficit over 24 hours).
  - Maintenance needed for 24 hours.
  - 100 ml/kg for first 10 kg.
  - 50 ml/kg for second 10 kg.
  - 20 ml/kg for every kg thereafter.
  - To obtain total maintenance needed for 48 hours multiply the above-calculated volume by 2.

Deficit:

For practical purposes, the usual deficit in most DKA patients is 10%.

Rate of Infusion:

- Add 24 hours maintenance to half of the calculated deficit and divide by 24 to obtain the hourly rate.

Fluid used:

- Use normal saline till blood glucose reaches 14-17 mmol/L (250-300 mg/dl) then change to 5% dextrose with 0.45 normal saline.

Potassium:

- Commenced when the child starts to pass urine (practically after the first hour) or if he is already passing urine and or K is below 5 mmol/L.
- Add 40 mmol/L of potassium chloride (i.e. 20 mmol/bottle of 500 ml).
- Monitor by ECG, clinically & biochemically (if available).
- If serum potassium is > 6 mmol/L withhold potassium temporarily till potassium is < 6 mmol/L.

Bicarbonate:

- Give only if pH is less than 7 and there is circulatory un stability generally it is preferable to avoid it completely.
- Dose: 1-2 mmol/kg; given over 60 minutes.
- Check blood gas (venous or capillary sample in non-shocked patient) every 6 hours.
- Unless child is critically ill avoid giving bicarbonate during first hour or two of resuscitation then repeat blood gas if PH is still  $\leq$  7 offer bicarbonate (if necessary).

Phosphorous:

We don't use it as a routine. Consider using it in comatose patients or if phosphate level < 0.5 mmol/L, give  $\frac{1}{2}$  the dose as potassium phosphate and half as potassium chloride.

Monitor calcium levels every 4-6 hours as patient might develop hypocalcaemia. Always check serum calcium level before phosphate is infused.

## B.2 Insulin Therapy:

- Preparation: use regular (or rapid acting) insulin only. Infuse into a separate IV line using a syringe pump (see above). Add 100 units in 100 ml (or 50 units in 50 ml) (or smaller volumes for young children) of normal saline in a syringe pump (or burette of the normal infusion pump) each ml will contain 1 unit/ml. This solution should be changed every 6 hours.
- Infusion rate: 0.1 unit/kg/hour i.e. 0.1 ml/kg/hour of the above mentioned preparation. Aiming to reduce blood glucose at rate of 4-5 mmol/hr (80 – 90 mg/hour). Usually there is a rapid drop after one hour of starting i.v. fluids.
- Continue this insulin infusion till acidosis is cleared i.e. either pH > 7.3,  $\text{HCO}_3 > 15 \text{ mmol}$  or normal anion gap ( $\text{Na}^+ - (\text{Cl} + \text{HCO}_3)$  normal =  $12 + 2 \text{ mmol/L}$ ).

Note: Discontinuation of insulin infusion is not dictated by blood sugar level, but by clearance of acidosis.

- If there is no facility to monitor blood gasses or serum bicarbonate continue infusion till the patient is clinically stable (fully conscious, well hydrated, doesn't look acidotic and taking orally well) and urine is containing no or trace of ketone.
- If there is no facility for a pump initially give regular insulin 0.3 units/kg s/c as start dose then 0.1 units/kg subcutaneously hourly or 0.15 – 0.2 units s/c 2 hourly till acidosis is cleared.

#### B.3 **Monitoring:** (Appendix 3)

- Blood glucose with a meter hourly during insulin infusion (at least hourly for 1<sup>st</sup> 4-6 hours then 2 hourly if needed) then every 6 hours thereafter.
- Blood gases, blood glucose, urea and electrolytes 4 - 6 hourly (if available) and urine for ketone is 2 hourly or on each voided urine.
- Vital signs (ECG monitor if available) and neuro observation (initially hourly till stable then every 4-6 hours). Also watch for headache, vomiting or behavior change and other signs of cerebral oedema.
- Flow sheet (Appendix 2) to record: blood workup, intake and output, doses of insulin, and urinalysis for glucose and ketone. Adequate urine output =  $\geq 1.5 \text{ ml/kg/hr}$ .

#### Problem Solving During Monitoring:

- After resuscitation, the typical aim of rate of blood glucose fall is 4 - 5 mmol/hour. (80 – 90 mg/dl).
- When blood glucose falls to 14-17 mmol/L (250 – 300 mg) change fluid to 5% dextrose with 0.45 saline to maintain blood glucose in the desired range of 120-200 mg/dl.
- If blood glucose rises again above 17 mmol/L (300 mg/dl) increase the insulin infusion by 25%.
- If blood glucose falls below 100mg/dl or falls too rapidly increase the concentration of glucose to 7.5% (or more).
- The insulin infusion rate should only be decreased if blood glucose levels remains below the target range despite glucose supplementation.
- Don't stop insulin infusion or hourly s/c if the patient is still acidotic.

### C. Management Following Clearance of Acidosis:

#### C.1 Fluids / Diet:

- If the child is alert, conscious, hasn't vomited for 4-6 hours, fluids including juices could be introduced gradually and IV fluid volume reduced gradually till child is able to eat and drink well.

#### C.2 Insulin:

Once the patient has recovered from DKA i.e. (PH more than 7.3, HCO<sub>3</sub> more than 15mmol/l or no ketonemia/ or if no lab facilities the patient is fully conscious, drinking and eating well, well hydrated not clinically acidotic and freshly voided urine contains no or one cross of acetone then do the following:

- If the patient is still sleepy, not drinking or eating well as often happens at night insulin infusion at a lower rates e.g. 0.05 unit/kg/hour could be continued and infusion rate adjusted according to blood sugar levels which is done 2-4 hourly.
- If the patient is drinking well and ready to eat (e.g. during the day), start subcutaneous insulin ½ hour before discontinuing infusion of insulin. However, if the patient is on hourly SC insulin, then he can be started immediately on subcutaneous insulin as follows:
  - a) If known diabetic: Give his usual dose of insulin if insulin time is due or else give regular insulin as explained in the sliding scale mentioned below.
  - b) New patient: Start with empiric dose of 0.5 – 0.7 units/kg/day of NPH + Regular or premixed (one-fifth to one third as regular and the rest as NPH).
  - c) Infants below 3 years can be managed with BID NPH 0.5 u.kg/day without regular. In either case small additive dose of regular insulin can be given S/C between the main insulin doses i.e. lunch time and midnight according to these guidelines:

Blood sugar (mg)	Urine ketone	Insulin dose Units/kg
240 or more	++ or more	0.2
240 or more	+ or negative	0.1
< 240	Negative	none

### C.3 Monitoring:

- Blood sugar, freshly voided urinary ketones before main meals, and midnight.  
Please note that ketonuria alone in an otherwise well child doesn't mean that he has DKA.
- Electrolyte once daily (if necessary).
- Vital sign and neuro observation 4 - 6 hourly at least for the first 24 hours.

### D. Other Problems and Complications:

- Cerebral Oedema:
- Warning signs and symptoms:
- Headache and decreasing of heart rate (not necessarily bradycardia).
- Vomiting.

- Change in neurological status (restlessness, irritability increased drowsiness, incontinence) or specific neurological signs (e.g. cranial nerve palsies).
- Rising blood pressure and decreased oxygen saturation. More dramatic changes such as convulsions, papilloedema and respiratory arrest are late signs and are associated with extremely poor prognosis. Diagnosis of cerebral oedema can be made clinically in presence of one diagnostic criteria, or two major criteria or one major and two minor criteria as follows: (see appendix 4).

Diagnostic criteria:

- Abnormal motor or verbal response to pain.
- Decorticate or decerebrate positive.
- Cranial nerve palsy (especially 3, 4, and 6).
- Abnormal respiration (chyne stokes, apnoea).

Major criteria:

- Abnormal mentation/function of level of consciousness.
- Sustained heart rate deceleration (decrease more than 20 beats/minute) not attributable to sleep or improved intravascular volume.
- Age inappropriate incontinence.

Minor criteria:

- Vomiting.
- Headache.
- Lethargy or not easily arousable.
- Diastolic B.P. > 90 mm.
- Age < 5 yrs.

 Action:

- Exclude hypoglycemia. Give immediate mannitol 1 gm/kg over 20 minutes (i.e. 0.5 ml/kg of 20% solution) (3% saline 5-10 ml/kg I.V over 30 minutes can be used if no mannitol is available).
- Halve rehydration infusion rate until situation improves.
- Nurse child head elevated.
- Move to PICU (or even earlier if possible).
- Call your senior.
- If assisted ventilation required maintain PCO at 23.5 K pa (25 – 30 mmHg).
- Consider continuation of mannitol at 0.25 gm/kg every 6 hours to prevent rebound increase in ICP or repeat bolus every 4 - 6 hours.

- Cranial imaging should only be considered after child is stabilized as other intracranial events as thrombosis hemorrhage and infarcts may occur. Note: a normal CT doesn't exclude cerebral oedema. It is a clinical diagnosis.

Look and treat all precipitating factors of DKA particularly infections.

Patients with ketonuria, Hyperglycemia not Satisfying Criteria of DKA:

These patients if they are otherwise okay and drinking well don't need to be admitted to the hospital. Just give a dose of regular insulin 0.1 – 0.2 units/kg or a dose equal to 10-20% of their usual daily dose as regular insulin subcutaneously. In addition to their usual dose (if it's dose time) or alone (e.g. lunchtime), encourage them to have fluids (small volumes) frequently of any fluid or juice, and to repeat blood sugar and fresh urinalysis after 4 hours and to continue this till blood sugar is normalized and one plus or no ketone in urine. However ALL NEW CASES and those with social problems or uncertainty about diagnosis on assessment should be admitted and not sent home before discussing the case with the senior. These cases can be started on NPH + regular or premixed insulin with extra doses of regular insulin as in the sliding scale above and they don't need to be put on IV fluids.

## **PROTOCOL FOR EMERGENCY MANAGEMENT OF**

### **HYPOGLYCEMIA IN CHILDREN**

#### **1. Definition:**

- Serum or plasma glucose below 2.6 mmol/L (46 mg/dl).
- Whole blood glucose below 2.2 mmol/L (40 mg/dl).
- In severely malnourished children blood glucose below 3 mmol/L (55 mg/dl).

#### **2. Aetiology :**

- For details refer to textbooks. Commonest causes include infections, starvations, malnutrition, diabetic children, metabolic disorders, and endocrinopathies.

#### **3. Symptoms:**

- 3.1 Newborns: Irritability, Jitteriness, respiratory distress, cyanosis, apnoea, hypotonia or seizures.
- 3.2 children: irritability, palpitations, sweating, tremors, confusion, pallor, seizures, coma.

#### **4. Diagnostic Methods:**

- Quick assessment with a glucose meter.
- But try to collect blood sample for plasma glucose, and other samples if cause is not clear from history this is particularly so far newborns and small children: this critical sample include:
- Urine for ketones.
- Serum insulin, growth hormone, cortisol, urine for organic acids, ammonia blood for tandem MS in a filter paper (if possible).
- For hormones just take the blood sample separate serum and freeze it for further decision later.
- If available collect blood for lactate & toxicology.

#### **5. Treatment:**

##### **A. *Unconscious or significantly symptomatic:***

- 1. Newborn:
  - Intravenous bolus of 2-3 mL/kg of 10% dextrose followed by continuous infusion of 3-5 ml/kg/hour (5-8 mg/kg/minute).
- 2. Children:

1 mL/kg of 25% dextrose or 2-3 ml/kg of 10% dextrose followed by infusion of 2-3 ml/kg/hour of 10% dextrose (3-5 mg/kg/minute).

3. Stabilizations:

- After 30 minutes & .... Check blood glucose.
- Then monitor 1-2 hourly till patient is stable.
- Oral intake should be commenced as soon as child can drink and eat well.

B. *Conscious children:*

If they can take orally give oral sugar 2-3 teaspoonfuls in water, sugar drink, honey or jam then feed after a feed.

6. Disposition:

- Children with unknown cause of hypoglycemia should be admitted to the hospital for further evaluation.
- Others can be sent home once stable.

7. Key points:

- When possible draw blood and urine for investigations prior to treatment if cause is not clear from history & examinations.
- Don't wait for laboratory results before starting treatment to avoid brain damage.
- Discuss undiagnosed cases with metabolic or relevant unit.

## Management of hypoglycemia in malnourished child

Definition: blood glucose (3mmol/L (55mg/d))

### Treatment:

- If unconscious use I.V. 4-5 ml/kg 10% dextrose then I.V 10% dextrose as above till recover consciousness.
- Otherwise give 50 ml of 10%dextrose or sucrose solution rounded teaspoonful of sugar in 3 ½ table spoonful of water (i.e. 5 gram in 50 ml of water) orally or by nasogastric tube followed by first feed as soon as possible-  
Divide the first feed into 4 equal amounts and give at ½ hourly intervals; then continues with 2 hourly feeds.
- If first feed is quickly available and in given then omit the glucose & sucrose solution and feed again after 2 hours
- Treat infections.

# FLUIDS AND ELECTROLYTES

## A. GOALS of management:

1. Estimate fluid and electrolyte deficits, maintenance requirements, and ongoing losses.
2. Select and administer appropriate fluids.
3. Monitor the management.
4. Treat the specific cause.

## B. Maintenance requirements:

Table 1: fluid &energy requirement:

Body wt	H2O ml/kg/day	Energy\kg\day
First 10kg	100	110
Second 10kg	50	75
Each additional kg	20	30

Table 2: Electrolyte requirement:

Electrolyte	mmol\kg\day
Sodium	2-4
Potassium	2-3
Chloride	2

## C. Ongoing losses:

The maintenance fluid volume given above includes the total fluid requirement under normal conditions including the insensible losses, plus essential urine output and moderate state of diuresis. Under certain pathological conditions, you might need to calculate the exact water and electrolyte losses. see table 3

**Table 3: Electrolyte composition of various body fluids:**

Fluid	Na <sup>+</sup> mmol/l	K <sup>+</sup> mmol/l	Cl <sup>-</sup> mmol/l
Gastric	20-80	5-20	100-150
Small bowel	100-140	5-15	90-130
Diarrhoea	10-90	10-80	10-110
Sweat	10-30	3-10	10-35

-For management of specific disorders including acute renal failure see their sections.

#### C. Deficits:

The most precise method of assessing fluid deficits (dehydration) is based on pre-illness weight. If this is not available, clinical observation as shown in table 4 is used.

**Table 4: Assessing degree & dehydration:**

	>2yrs: 3%(30ml/kg)	6% 60ml/kg	9% 90ml/kg
Examination	<2yrs: 5% (50 ml/kg)	10% (100 ml/kg)	15% (150 ml/kg)
dehydration	mild	moderate	Severe
Skin turgor	normal	tenting	None
Buccal mucosa	moist	dry	Cracked
Eyes	normal	deep set	Sunken
Tear	Present	reduced	None
Fontanel	flat	soft	Sunken

CNS	fully conscious	irritable	Lethargic obtunded
Pulse rate	normal	slightly increased	Increased
Pulse volume	normal	weak	Feeble
Capillary refill	normal	2seconds	>3 seconds
Urine output	normal	decreased	Anuric

### Fluid Management of Dehydration:

- **Restore intravenous volume (Patient In shock):** normal saline 20 mL/kg over 20 minutes (repeat until intravascular volume is restored)
- Rapid volume repletion: 20 mL/kg normal saline or ringer lactate (maximum = 1 L) over 2 hr
- Calculate 24 hr fluid needs: maintenance + deficit volume
- Subtract isotonic fluid already administered from 24 hr needs
- Administer remaining volume (as calculated) over 24 hr using D5% ½ NS + 20 mEq/L KCL
- Replace ongoing losses as they occur
- For mild, moderate and severe dehydration see table (4)

### E. Fluids available for rehydration

Table5: Shows composition & commonly used I.V crystalloid fluids:

Fluid	Na <sup>+</sup>	K <sup>+</sup> mmol\L	Cl <sup>-</sup> mmol\L	HCO <sub>3</sub> lactate mmol\L	Energy kcal	Mosmol/L

	mmol\L			L		
Normal saline (0.9%)	154	-	154	-	-	308
½ NS	77	-	77	-	-	154
½ NS+D5%	77	-	77	-	200	406
D5%+water		-		-	200	252
D5%+ 1/4 NS(0.225)	34	-	34	-	170	329
D5% +1/5NS	30	-	30	-		346
Ringer lactate (Hartman)	131	5	111	28	273	-
Glucose 10%	0	0	0	0	400	
Albumin 25%	100-160	-	(120)	1000\L		300

For composition & commonly used oral fluids see table (6).

Table(6):

Fluid	CHO g\d	Na <sup>+</sup> mmol\L	K <sup>+</sup> mmol\L	Cl <sup>-</sup> mmol\L	Base mmol\L	Mosm\kg
WHO ORS	2	90	20	80	30	310
*Resomal		45	40		15	300

For formation of Resomal from WHO/ORS or home-made fluids, see gastroenteritis and malnutrition section.

For composition of some commonly used oral fluids at home, see table(7)

**Table(7) Electrolyte composition & some common oral drinks:**

	CHO g\dl	Na <sup>+</sup> mmol\L	K <sup>+</sup> mmol\L	Cl <sup>-</sup> mmol\L	HCO <sub>3</sub> mmol\L	mosm\k g H <sub>2</sub> O
Coca cola – pepsi	11	4.3	0.1		13.4	656
Apple Juice	12	0.4	26			700
Orange Juice	10.5	0.2	49		50	654
Milk	5	22	36	28	30	260

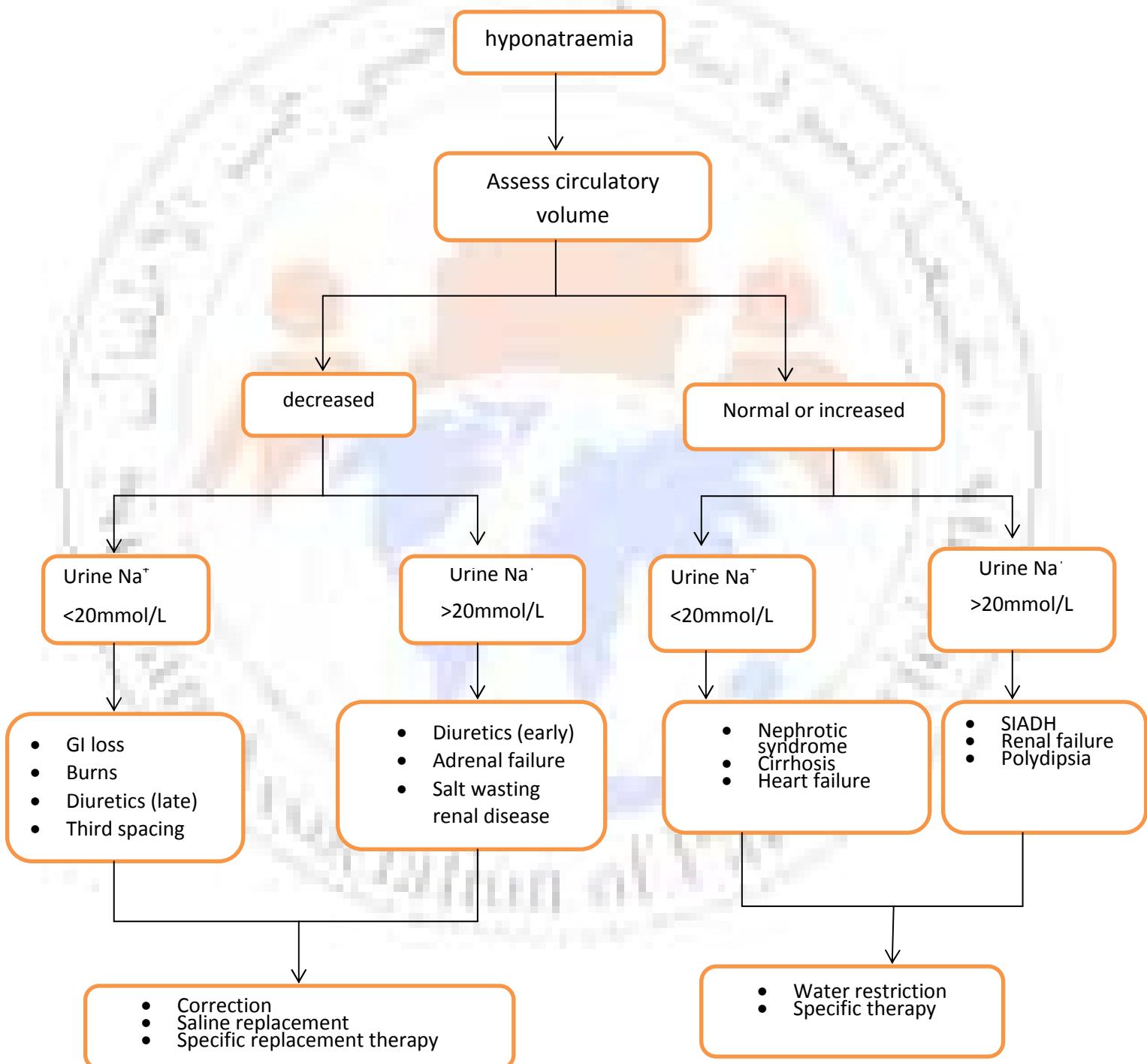
# ELECTROLYTE DISTURBANCES

## F. 1 - Hyponatremia:

1-Definition: serum  $\text{Na}^+$  less than 130 mmol/L

2-Causes: for causes of Hyponatremia see Fig 1:

- Specific replacement therapy



- An important cause of factitious hyponatraemia is hyperglycemia:

$\text{Na}^+$  decreased 1.6mmol/L for each 100 mg (5.5mmol) rising blood glucose.

-Formula for correction of hyponatremia (Deficit)

$$(\text{Desired Na}^+ - \text{actual Na}^+) * \text{body wt}/\text{kg} * 0.6$$

\*If  $\text{Na}^+$  is  $\geq 105 \text{ mmol/L}$  correct to  $125-130 \text{ mmol/L}$

If  $\text{Na}^+$  is  $< 105 \text{ mmol/L}$  correct by  $20 \text{ mmol/L}$  maximum

Rate of rising  $\text{Na}^+$  should not exceed  $2-4 \text{ mmol/L}$ , 4 hourly or  $20 \text{ mmol/L}$  every 24 hours.

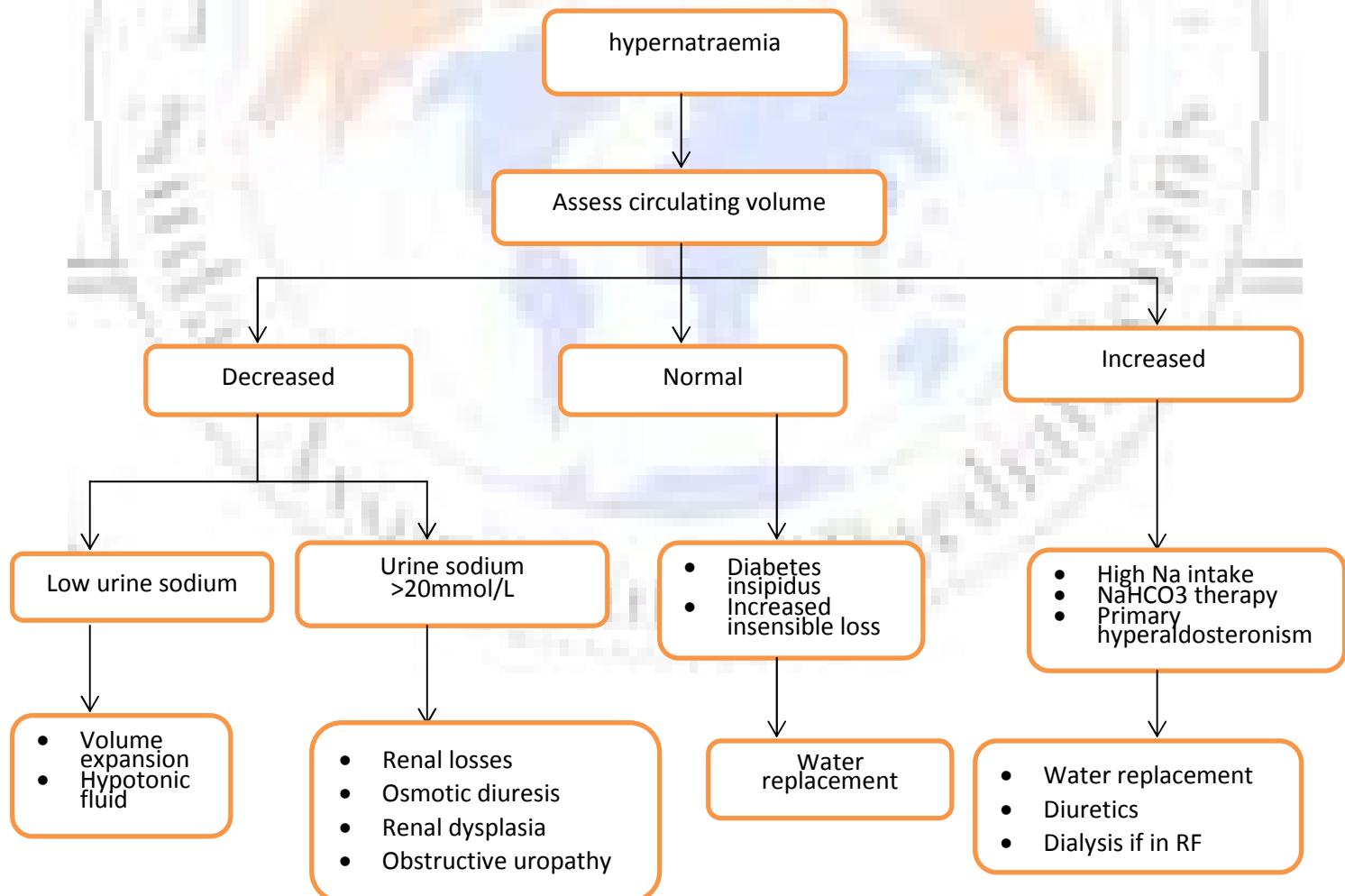
-for symptomatic hyponatremia (seizures) correct serum  $\text{Na}^+$  to  $125 \text{ mmol/L}$  over 2 hours.

Use hypertonic saline 3% each ml will contain  $0.5 \text{ mmol}$  or use normal saline each 1ml will contain  $0.154 \text{ mmol}$

## F.2 HYPERNATREMIA:

Definition: serum  $\text{Na}^+$  more than  $150 \text{ mmol/L}$ .

For classification and diagnosis of causes of hypernatremia see fig(2).



-Clinical evaluation can be difficult, pulse might be normal initially, skin is doughy, and a CNS manifestation such as irritability is common.

#### **Management guidelines:**

-Give maintenance plus 1\2 deficit (or 75% maintenance + deficit over 24 hours) (Subtract boluses)

-Use 1\5<sup>th</sup> or 1\2 NS (Not water & Normal Saline)

-Lower serum Na<sup>+</sup> by 10 – 15 mmol\day or (0.5 –0.75 mmol\hour)

- Normal hydration should be achieved over 36 – 48 hours and perhaps 72 hours if the initial plasma Na is > 170 mmol/L

-Monitor electrolytes 4-6 hourly & adjust fluid accordingly

-Treat hypoglycaemia & hypocalcaemia.

- Persistent oliguria when circulatory impairment has been corrected indicates:

- ARF (due to tubular necrosis)

- Renal vascular thrombosis)

#### **Complication of treatment:**

-Cerebral oedema & seizure: 3% NaCl 4 ml\kg or manitol.

-Pulmonary oedema: give diuretics

-Hypocalcaemia: add calcium gluconate 10 ml 10%

-Renal tubular injury & uremia.

#### **F.3 HPOKALAEAMIA:**

1- Definition: serum K<sup>+</sup> below 3.5 mmol\L.

2-Aetiology :( See table 8)

	Hypertension	Normal BP	exhausted	Normal stone
Disorder	Reno vascular disease: <ul style="list-style-type: none"> <li>• Excess rennin</li> <li>• Excess mineralocorticoid</li> <li>• Liddle's syndrome</li> <li>• Cushing syndrome</li> </ul>	RTA Fanconi Barter DKA Diuretics	Skin burn G.I loss Malnutrition	-Metabolic alkalosis -Insulin -Others
LAB	High urine K <sup>+</sup>	High urine K <sup>+</sup>	Low urine K <sup>+</sup>	High urine K <sup>+</sup>

### 3- Clinical:

- Muscle weakness (paralysis), Smooth muscle (intestinal ileus, ureteric dilatation), Cardiac (arrhythmia and ECG changes: prolonged QRS, flat T-wave, ST depression, U - wave in ECG)

### 4-Treatment:

- a) In acute emergencies (e.g. cardiac arrhythmia) give 0.5mmol/kg/hour in 20 mL of 5% dextrose over 30 min -1hour. Concentration should not exceed 80 mmol/L
- b) Otherwise put 30-40mmol/L in I.V fluids
- c) For oral therapy dose is 2-4 mmol/kg/24hours Bib or Qib

### F.4 HYPERKALAEMIA:

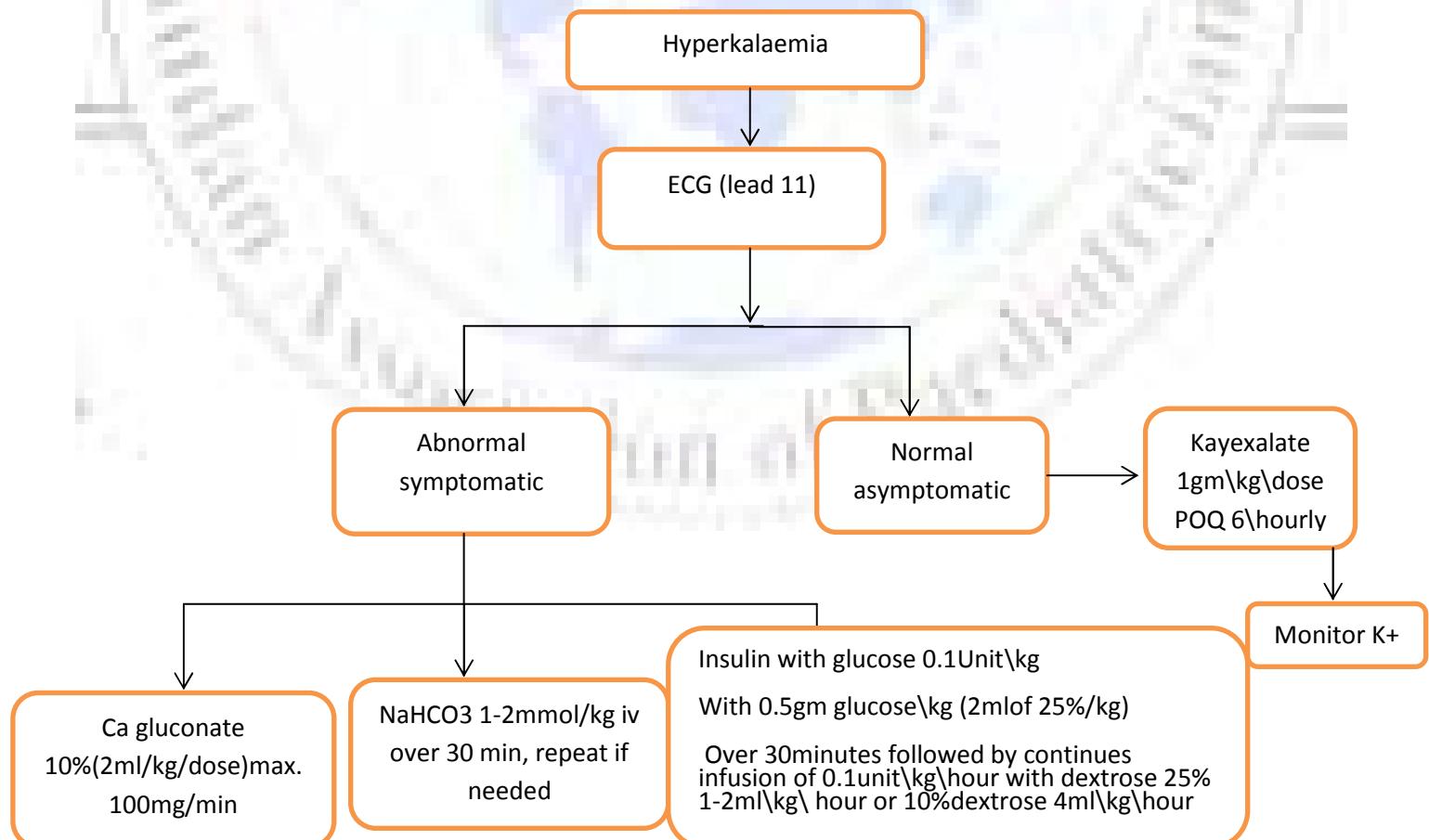
1- Definition: serum k<sup>+</sup> >5.5mmol/L in non-haemolyzed sample:

(A) Aetiology of hyperkalemia

Potassium overload	Intracellular uptake	Pseudohyperkalaemia
- Oral \ I.V (Load)	<ul style="list-style-type: none"> <li>• acidosis</li> <li>• Shock</li> <li>• DKA</li> <li>• B-blockers</li> </ul>	-
- Transfusion of old blood		
- Tumor lysis syndrome		
- poisoning		
Decreased Renal excretion		
-Renal failure		
- Adrenal failure		
-Hypoaldosteronism		

2- ECG changes: peaked T wave (tenting), wide QRS, ST depression

3- B Management guidelines (see algorithm)



# EMERGENCY MANAGEMENT OF HYPOCALCAEMIA

## **DEFINITION**

Total serum calcium below 2 mmol/l = 8 mg/dl in term newborns and older children or ionized calcium below 1 mmol/l

IN PRETERM; total below 1.75 mmol/l

## **SYMPTOMS & SIGNS**

SYMPTOMATIC (usually level below 7 mg/dl)

ASYMPTOMATIC

### **SYMPTOMS & SIGNS**

- CONVULSIONS
- TETANY
- HYPER OR HYPOTONIA
- IRRITABILITY
- ICP
- LARYNGOSPASM
- CARDIAC : Brady/failure/oedema
- Signs of aetiology

## **AETIOLOGY**

### **■ NEONATAL EARLY (0-72 HRS)**

preterm

LBW

RDS

asphyxia

acidosis

infants of diabetic mothers

exchange

### **■ NEONATAL LATE (3-7 DAYS)**

High phosphate milk

VD deficiency

Hypopara

Maternal hyperpara

Mg deficiency

### **■ AETIOLOGY LATER IN CHILDHOOD**

VD DEFICIENCY  
VD METABOLISM PROBLEMS  
HYPOPARTHYROIDISM  
CALCIUM DEFICIENCY  
HYPOMAGNESEAEMIA  
HIGH PHOSPHATE eg enemas/tumor lysis

### **HISTORY**

- ✓ Perinatal
- ✓ Nutrition
- ✓ VD supplement
- ✓ Housing and sun exposure
- ✓ Growth
- ✓ GI/Renal
- ✓ Drugs
- ✓ FH
- ✓ Others

### **EXAMINATION**

- Signs of hypocalcemia
- Signs of aetiology

### **INVESTIGATIONS**

- TO DIAGNOSE HYPOCALCEMIA
- TO FIND AETIOLOGY
  - DRAW BEFORE TREATMENT
  - Based on history & exam
    - Ca.phosphate,alkaline phosphatase
    - Radiology
    - PTH(WITH SAME Ca sample)
    - VD metabolites(draw separate and freeze)till senior or endo consult
    - Others

### **TREATMENT**

- SYMPTOMATIC
  - 10%Calcium gluconate 2ml/kg iv
  - Dilute 1:4 with 5%dextrose or water
  - Give over 30 minutes
  - ECG monitor(if possible) or clinically for bradycardia or even arrest

- Then start infusion of 1mmol/kg /24hours in 5%dex+1/5 saline or give the total daily dose divided as 6hourly infusions
- Monitor pt and calcium 6hourly

#### IF SYMPTOMS CONTINUE

- Give another infusion bolus of 1-2 ml/kg
- OR increase infusion rate to 1.25-1.5 mmol/kg/day
- ASYMPTOMATIC
- After infusion or from the start
  - Oral calcium 1mmol/kg /day
  - 1mmol calcium=40mg of elemental calcium
  - Give 6hourly
  - Check the concentration of preparations in your hospital (eg osteocare 5ml=150 mg)
- Treat The aetiology

# **DIAGNOSIS & MANAGEMENT OF ACUTE ADRENAL INSUFFICIENCY**

## **PATIENTS AT RISK**

- ▶ Known adrenal cases
- ▶ Newborns with ambiguous genitalia or hyper pigmentation
- ▶ Meningococcemia cases
- ▶ Clinical features of Addison's or associated syndromes
- ▶ Chronic steroid therapy+stress or withdrawal
- ▶ Brain insults that lead to hypopituitarism
- ▶ Dehydration with hyponatremia & hyperkalemia

## **CLINICAL FEATURES**

- ▶ Diarrhoea
- ▶ Vomiting
- ▶ Abdominal pain
- ▶ Dehydration
- ▶ Hypotension/Shock
- ▶ Hypoglycemia
- ▶ Acidosis
- ▶ Hyponatremia/hyperkalemia(not always)
- ▶ Fatigability/weakness/wt loss
- ▶ Features of aetiology

## **URGENT INVESTIGATIONS**

- ▶ Lytes, urea , creatinine
- ▶ Glucose
- ▶ CBC
- ▶ Blood gases(if available)
- ▶ Work up for precipitating factors and or suspected primary cause

## **EXPECTED RESULTS**

- ▶ Hyponatremia
- ▶ Hyperkalemia
- ▶ Hypoglycemia
- ▶ Metabolic acidosis
- ▶ Eosinophilia
- ▶ In a clinically highly suspicious case their absence doesn't exclude the diagnosis

## **OTHER INVESTIGATIONS**

- ▶ SERUM CORTISOL
- ▶ ACTH

- ▶ ADRENAL STEROIDS
- ▶ URINE ELECTROLYTES
- ▶ Consult Senior or Endocrinology service
- ▶ Draw blood separate and freeze before treatment if possible or time allows

### **URGENT TREATMENT**

- ▶ IF SUSPECTED DON'T WAIT FOR LAB RESULTS
- ▶ ABC
- ▶ IV HYDROCORTISONE PUSH
  - 25mg for newborns and infants below 3yrs
  - 50mg for young children(3-12)
  - 100mg for older children
- ▶ IV FLUIDS
  - M+D
  - Normal saline + 5-10% dextrose
  - Treat symptomatic hypoglycemia
  - Treat hyperkalemia if significant

### **FURTHER MANAGEMENT**

- ▶ Admit to ICU or high dependency area
- ▶ Close monitor
  - Vital signs
  - Electrolytes 6hourly(if possible)
  - Glucose 6 hourly
  - Treat metabolic acidosis with bicarbonate if significant
  - Hydrocortisone 50mg/M<sup>2</sup> /day (or the same bolus dose)as continuous infusion or Q 6hourly during stress
  - Once stable change to oral dose of 15-20mg/m<sup>2</sup>/day BID or double this dose orally if still stressed
  - Fludrocortisone 0.1mg PO OD
  - Oral salt 0.5-1gm/kg/day for newborns Q 4-6 hourly
  - Alert card/IM hydrocortisone at home
  - Refer to Endo or consult by phone

# **Severe complicated malaria**

## **Definition**

Severe malaria is malaria due to *P. falciparum* that sufficiently serious to be immediate threat to life. It is a medical emergency which requires hospitalization.

A patient is regarded as having severe malaria if he or she has one or more (mostly seen in combination) of the following conditions:

- 1- Prostration.
- 2- Respiratory distress.
- 3- Repeated convulsions within 24 hours.
- 4- Severe anaemia ± congestive heart failure.
- 5- Pulmonary oedema.

If severe malaria is suspected, the following key aspects of assessment should be followed:

- 1- Assess level of consciousness follow Glasgow scale or Blantyre coma scale.
- 2- Vital signs:

- Pulse rate.
- Respiratory rate (look for acidotic breathing deep and rapid).
- Blood pressure.
- Temperature.
- Pallor.
- Assess hydration status.

## **Immediate management:**

- Start resuscitation particularly maintenance of a patent airway.
- Abort convulsion by giving diazepam (see protocol of fits) 0.5 mg/kg PR.
- Establish IV line.

## **Manage fever by the following actions:**

- Remove excess clothing.
- Put fan on if available.
- Paracetamol orally or PR 15 mg /kg 4-6 hourly.
- Correct dehydration.

## **Correct hypoglycaemia**

Do bedside glucometer test (if available) if blood glucose is less than 3 mmol/L give 5ml/kg of 10% dextrose as soon as an IV access is established.

**If level of consciousness is disturbed:**

Insert nasogastric tube if the patient is unconscious or in coma and fix indwelling catheter.

**Immediate tests should include the following:**

- 1- Thick and thin blood film for malaria.
- 2- PCV.
- 3- Hg.
- 4- Blood glucose.
- 5- Lumbar puncture if indicated.

**Specific management:**

**Quinine:**

Is the drug of choice and should be given initially by intravenous infusion, preferably in 5% glucose. The dose is 10 mg salt/kg body weight administered 8 hourly until the patient can tolerate orally, then continue the same dose to complete the course duration for 7 days. If IV not possible, quinine (the same dose) can be given intramuscularly diluted with normal saline or distilled water to a concentration of 60 mg/ml into both anterior upper thighs.

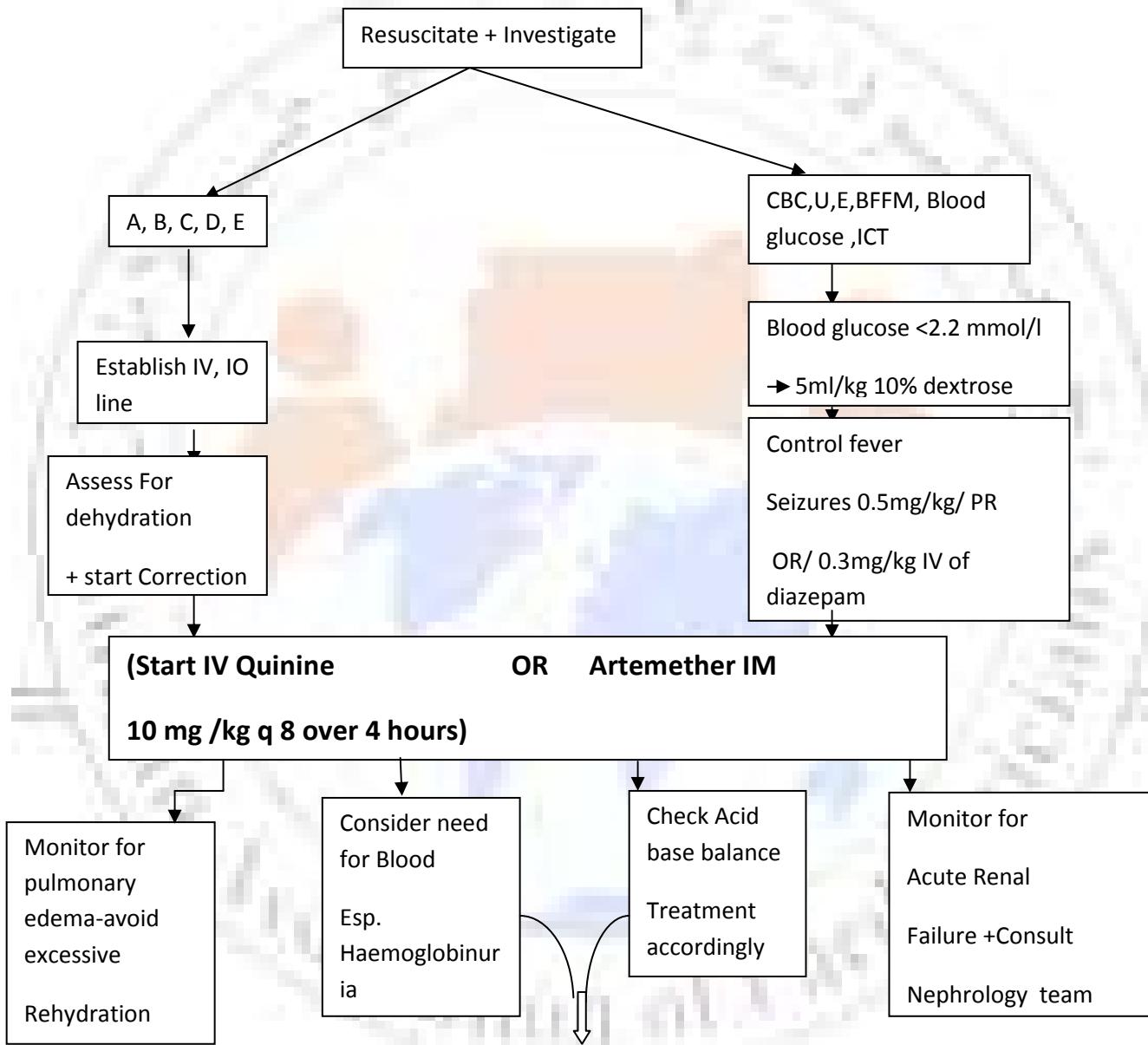
**Artemether:**

Artemether injection is another alternative. The dose for children is 1.6mg/kg twice in the 1<sup>st</sup> 24 hour (12 hours apart). Followed by 1.6 mg/kg daily for 6 days ( 8 days in total ) .

## Severe Malaria

### Suspect if

- Impaired Consciousness.
- Acidotic breathing.
- Repetitive Convulsion.
- Pulmonary oedema.
- Haemoglobinuria.
- Jaundice.



Total duration of treatment is 7 days. Shift to oral Quinine when patient can tolerate orally, usually after 48 hours of parenteral quinine.

# Suspicion of meningitis

## Clinical presentation of meningitis in children

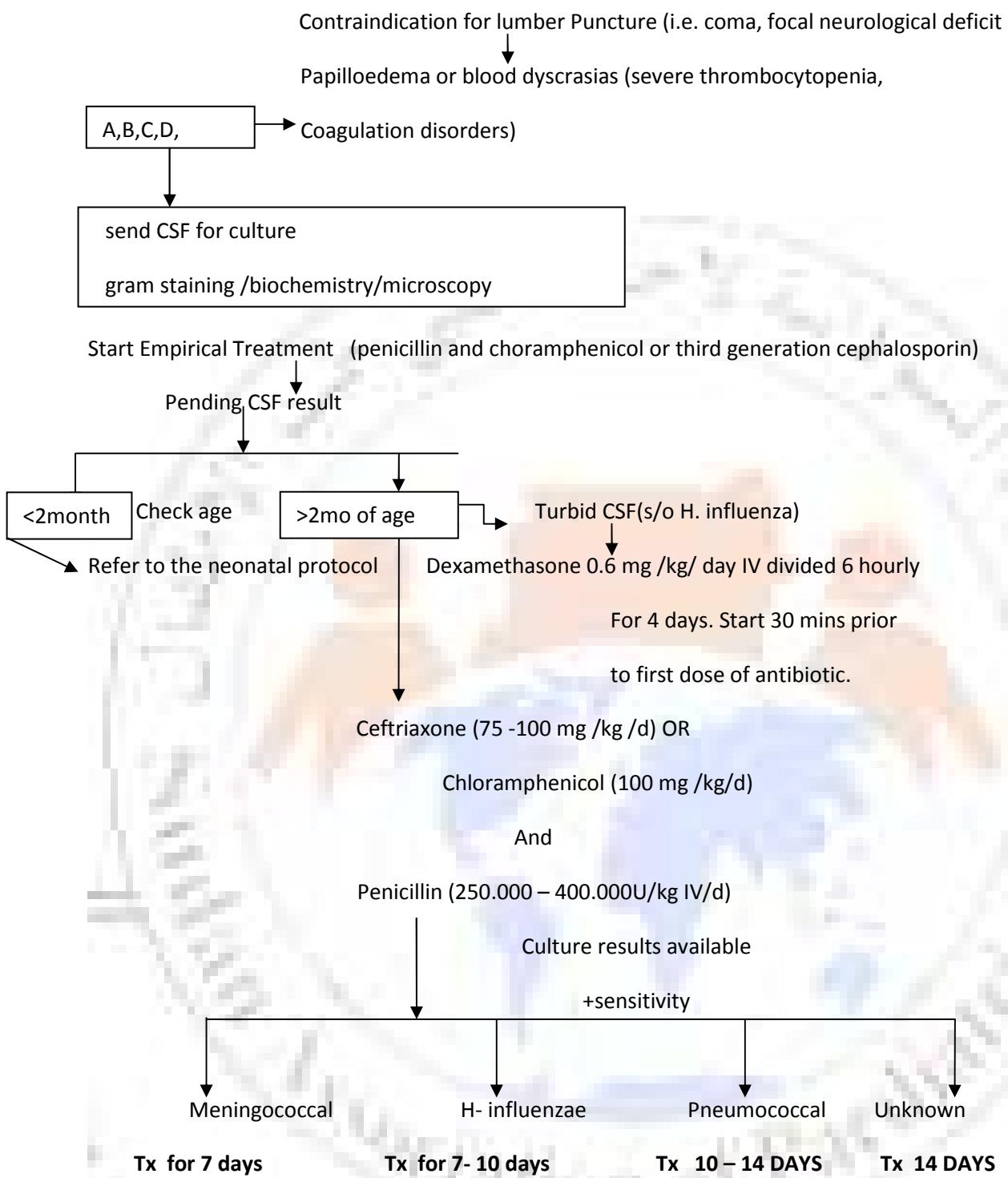
### In infants

- Fever and convulsion.
- Refusal of feeding.
- Bulging anterior fontanelle.
- Irritability.
- Impairment of level of consciousness.
- Petechiae (meningococcaemia).

### In older children

- Neck stiffness.
- Brudzinski' s sign.
- Kernig 's sign.
- Fever with or without convulsion.
- Impairment of level of consciousness.

## LP



### Note:-

In some situations oily choramphenicol can be used as single dose 50mg / kg IM . Repeat after 24 hours if no response.

### The management also includes:

- Supportive management ( ABC) .
- Maintenance fluids (2/3) .
- Anticonvulsants . (if needed ) .

# **Management of tetanus**

## **Principles of management:**

- Eradication of clostridium tetani.
- Neutralization of tetanus toxin.
- Control of seizures and respiration.
- Palliation and provision of supportive care.
- Prevention of recurrences.

## **Management:**

1. Surgical wound excision and debridement to remove the foreign body after administration of human globulin (HTIG) and antibiotics. Removal of umbilical stump in neonate is not recommended.
2. Single injection of 500 U of TIG I.M to neutralize the toxin. But doses as high as 3000-6000 U are also recommended. If TIG is not available I.V human immunoglobulin or tetanus antitoxin (TAT) in dose of 50.000 – 100.000 U I.M (check for sensitivity is needed).
3. Penicillin G 100.000 u/k 6 hourly for 10 days + Metronidazole + Gentamycin in neonate.
4. Diazepam for both seizure and relaxation 0.1- 0.2 mg/kg every 3 - 6 hour I.V for 2 - 5 weeks + chlorpromazine.
5. Keep the Pt. in dark quiet room, maintenance of fluid and electrolyte needs. Careful nursing to mouth, skin, bladder, and bowel function is needed to avoid ulceration and infection.

NB: don't insert nasogastric tube unless the patient is fully sedated.

## **Prevention:**

1. Active immunization (DPT) 6 weeks, 10 weeks and 14 weeks. Booster at 4 years every 10 years (DP).
2. Immunization of women with tetanus toxoid prevents neonatal tetanus with at least 2 doses.

# **Acute liver failure**

## **Definition-1:**

**Definition:**

Fulminant hepatic failure is a clinical syndrome resulting from :

- Massive necrosis of hepatocytes .
- Or from severe functional impairment of hepatocyte's synthetic, excretory, and detoxifying functions of the liver .

## **Definition-2**

Accepted definition in children:

- Biochemical evidence of acute liver injury ( usually less than 8 weeks duration).
- No evidence of chronic liver disease.
- Hepatic-based coagulopathy defined as PT >15 seconds or INR >1.5 not corrected by vitamin K in the presence of clinical hepatic encephalopathy or:
- PT > 20 seconds or INR > 2 regardless of the presence of clinical hepatic encephalopathy.

## **Aetiology**

- Viral : Hepatitis (A,B,D,E) and others.
- Drugs & chemicals ( acetaminophen overdose, sodium valproate, anti-tuberculous drugs..etc).
- Metabolic e.g. ( Wilson's disease, tyrosinemia, Galactosemia,...) .
- Ischemia & hypoxia.
- Herbal supplements.
- Idiopathic.

## **Symptoms and signs**

- Anorexia, vomiting, abdominal pain,
- Fever,
- Progressive jaundice,
- Fetur hepaticus,
- Hemorrhagic diathesis,
- Ascites,
- Hepatic, encephalopathy (disturbances of consciousness, and :

- In infants irritability- poor feeding-change in sleep rhythm,
- In older children, somnolence, asterixis, confusion, combativeness on arousal, and ultimate coma,

## **Investigations**

- Bilirubin level (direct and indirect) markedly increased.
- Aminotransferase levels markedly elevated but may become normal or even decreased as patient deteriorates.
- Prolonged PT, which is often not improved by administration of vitamin K.
- Hypoglycemia.
- Hypokalemia , hyponatremia.
- Metabolic acidosis, or respiratory alkalosis.
- Increased blood ammonia level but may be normal.

## **Principles of management**

- Give supportive care.
- Maintain fluid balance.
- Correct electrolyte imbalance.
- Manage coagulation disturbances and bleeding.
- Manage hepatic coma complications.
- Treat and prevent infection.
- Manage hepatic encephalopathy.
- Manage ascites .
- Nutrition and vitamins supplements.

## **Give supportive care**

- Care for airway and vitals.
- Give appropriate nursing care according to condition and state of consciousness.
- Monitoring: vitals, neuro, fluid balance, renal etc.
- Avoid aggravating factors: infections, drugs, sedatives...etc.
- Avoid hypovolaemia and use infusions of fluids and blood products cautiously.
- Avoid hypoglycemia: monitor and treat with IV glucose .
- Avoid and treat electrolyte imbalances.

## **Correct and maintain fluid balance**

- For the ill patients who are unable to take orally or the comatose patient, maintenance fluids should be provided IV ( may be at less than maintenance rate if critically ill or unconscious).
- Appropriate fluids are 5% D ½ NS for children >10 kg .
- D 5% 1/5 NS for children <10 kg.
- Daily fluids should contain Potassium chloride 1-2 mmol/kg/24 hr .
- If patient has to continue on maintenance IV fluids for > 3 days, consider nasogastric feeding, enteral or parenteral nutrition.

## **Correct and maintain electrolytes**

- Hypokalemia: 1-2mmol/kg over 2-3 hrs at a rate not > 0.2 mmol/kg/hr.
- The conc. Of potassium should not exceed 40 mmol/l.
- Do serial plasma measurements +ECG monitoring if available.
- Hypocalcaemia:
- Hypophosphatemia:
- Hypomagnesaemia:

## **Manage coagulation disturbances and bleeding-1**

- Treat coagulopathy with: IV vitamin K (not IM) : 0.2 mg /kg daily for 3 days , then every other day .
- FFP: 10-15 ml/kg or cryoppt 1 bag / 5 kg.
- Platelets transfusion in cases of clinically significant bleeding with prolonged BT.
- Desmopressin : 0.3 microgram/kg may correct BT in these patients.
- Plasmapheresis for temporary correction of bleeding diathesis without resulting in fluid overload.

## **Manage coagulation disturbances and bleeding-2**

- Recombinant factor VII for transient correction of coagulopathy refractory to FFP especially when performing invasive procedures.
- Maintain PT at 20-25 sec (if no active bleeding) and at less than 20 sec if there is active bleeding.
- Maintain platelet count at more than 50000.
- Maintain haematocrit at more than 30.

## **Monitor and manage complications of hepatic coma**

- Cerebral edema: -

\*Monitor ICP to prevent severe cerebral edema and monitor CPP.

\*Use osmotic diuretic e.g. mannitol :

0.5-1.5 g/kg (2.5-7.5 ml/kg of 20 % solution), repeated if necessary 1-2 times after 4-8 hrs .

- Convulsions:

\*Best is to give phenytoin at a reduced dose.

## Monitor and manage complications of hepatic coma

- Upper GIT bleeding:

\*Prophylactic use of antacids, H2-receptor blockers or both because of the high risk of GIT bleeding.

- Renal failure:

\*Monitoring and conservative management.

\* with fluid overload consider continuous haemofiltration .

Treat and prevent infection.

- Take necessary preventive measures to avoid iatrogenic and hospital cross-infection.
- Protect yourself, the medical team and contacts.
- Do necessary screening work up for infection.
- Use prophylactic antibiotics: I.V ampicillin/cloxacillin + cephalosporins.
- Treat infections according to aetiology and results of microbiology work up.

## **Manage hepatic encephalopathy**

- Treatment of hepatic encephalopathy:
- Restrict or minimal protein intake depending on degree of encephalopathy.
- Avoid and treat constipation with:

\*Enemas (which can be done several times).

\*Lactulose: orally or by NG tube in doses 10-50 ml every 2-4 hrs sufficient to cause diarrhea, then dose adjusted to produce several acidic loose stools daily.

\*lactulose can also be given as retention enema every 6 hrs (dilute with 1-3 volumes of water).

Manage hepatic encephalopathy-2:

Give a non-absorbable antibiotic orally or rectally e.g.. Neomycin (or if not available you can use oral streptomycin).

\*Neomycin dose:

Infant & child 50-100 mg /kg/24 orally 6-8 hrs for 5-6 days (max 12g/24hr).

- Flumazenil,a benzodiazepine antagonist, can reverse early signs of encephalopathy.
- Manage GIT bleed and do frequent gastric suction.

Manage ascites:

- Treatment of massive edema and ascites: --give diuretics in maximum doses (frusemide + spironolactone).

\*Monitor potassium and sodium frequently.

- 25% IV albumin solution 0.5-1g/kg over 30-120 minutes daily for 3 days, then every other day. in cases of associated hypovolemia give by rapid infusion. Contraindicated in cases of congestive heart failure or severe anemia.
- Restrict sodium intake (1-2 mEq/Kg).

### **Nutrition and vitamins supplements-1**

- Oral or nasogastric feeding.
- Provide adequate calories.
- MCT.
- Protein restriction?.
- Vitamin supplements: fat soluble + others.

### **Nutrition and vitamins supplements-2**

- Give supplementation of the fat-soluble vitamins :
- Vitamin K: give oral water-soluble preparation (menadiol sodium phosphate): \*\*C.I in neonates & infants 1-5 \*1-12 yrs: 5-10 mg daily. \*12-18 yrs: 10-20 mg daily.  
Mg daily.
- Vitamin A: oral water-soluble preparation.
- Vitamin D: orally as Ergocalciferol (vit. D2) or cholecalciferol (vit D3):

\*1-12 yrs: 10000-25000 units daily.

\*12-18 yrs: 10000-40000.

- Vitamin E: oral water-soluble preparation (d-alpha-tocopherol polyethylene glycol).

\*Neonates: 25-50 iu/day. \* Children: 1 iu/kg/day.

Enhance bile flow/Treat pruritis.

- Give oral ursodeoxycholic acid:

As a choleretic (increase bile flow into the intestine). This may improve absorption of fat-soluble vitamins.

10-15 mg/kg twice daily, available as tabs. Or caps.

- Cholestyramine for pruritus:

orally, once daily or in 2-4 divided doses, mixed with water or any suitable liquid.

\*1month-1yr: 1g max 9 g

\*1-6 yrs: 2g max 18g

\*6-12 yrs: 4 g max 24 g

- Other drugs to be taken 1 hr before or 4-6 hrs after cholestyramine to reduce possible interference with drug absorption.

Other modalities of treatment

- Cleansing devices:

\*Charcoal Hemoperfusion .

\*Plasmapheresis .

\*Biologic-DT.

\*Molecular adsorbent recirculating system(MARS).

- Bioartificial liver support systems:

\*Bioartificial liver.

\*Extracorporeal liver assist device.

- Liver Transplant:

\*Total transplant.

\*Auxiliary Liver Transplant.

\* Hepatocyte Transplant.

# Management of acute diarrhoea and Severe dehydration

Assessment of dehydration (table 1):

Signs/Symptoms	Classification	Treatment
<p>If any two of the following signs are present:</p> <ul style="list-style-type: none"> <li>• Lethargic or unconscious</li> <li>• Deeply sunken eyes</li> <li>• Not able to drink or drinking poorly</li> <li>• Skin pinch goes back very slowly &gt; 2 seconds)</li> </ul>	Severe dehydration	<p>A-child with shock 20 ml/kg NS or Ringer's lactate, repeat if needed.</p> <p>B-child with no shock 100ml/kg ringers lactate or glucose 5%+1/2 saline:</p> <p>If the child is &lt;1yr 30ml/kg over 1hr then 70ml/kg over 5 hr</p> <p>If &gt;1yr 30 ml/kg over ½ hr then 70 ml/kg over 2 ½ hrs</p> <p>Give Zinc after rehydration</p>
<p>If any two of the following signs are present:</p> <p>Restlessness , irritability , Sunken eyes, Drink eagerly, Skin recoil in &lt; 2 seconds</p>	Some dehydration	<p>ORS 75 ml/kg over 4 hrs., unless there is severe vomiting or patient is unable to drink give i.v fluids glucose 5% with 1/2 saline or Ringer's lactate. Give Zinc after rehydration ORS (7 – 10 ml/kg) or homemade fluid after motion Give Zinc.</p>

## **Management of Severe Dehydration:**

- If the child is having very weak pulse or in shock give rapid bolus of normal saline or Ringer's lactate 20ml/kg/hr.
- The bolus can be repeated until pulse, perfusion, and mental status return to normal.
- The child should be observed closely during this period, and vital signs should be monitored on a regular basis.
- If venous access is not available use intraosseous infusion or do vein section.

2- Child with severe dehydration not in shock Give 100ml/kg Ringer's lactate or 5%D + 1/2 NS divided as follows: (table 3)

<b>Age</b>	<b>First give 30ml/kg in</b>	<b>Then give 70ml/kg in</b>
Infants (under 12 months)	1hour	5 hours
Children (12 months up to 5 years)	minutes30	hours $\frac{1}{2}$

NB: be sure to add ongoing losses to maintenance + deficit fluids and electrolytes:

- The patient should be reevaluated every 1-2 hours by checking vital signs, clinical signs, ongoing losses and urine output.
- Reassess the hydration status after 6hrs to choose the appropriate plan for management.
- If IV line is not available start the rehydration with ORS by NGT: give 20ml/kg for 6hrs. Reassess the child every 1-2hrs.
- An NG tube can be helpful for patients with normal mental status but who are too weak to drink adequately.

Special situations:

1. After rehydration if the child is still lethargic or very ill, start management with anti septicemia treatment.
2. Children with abdominal distention or sluggish bowel sound and if the child is passing urine, start potassium management maximum 3mmol/kg (not exceed 20mmol/500ml of I.V fluids and should not be given in less than 2- 4 hours).
3. In children with C.N.S manifestation e.g. convulsion: exclude hypernatremic dehydration, cerebral malaria, meningitis, if the child is ill cannot tolerate lumbar puncture (L.P) manage as meningitis.

**Child with hypernatremic dehydration** ( $\text{Na} > 150 \text{ mmol/l}$ ). Start slow rehydration, give fluids over 48hours:

- Day 1: 1/2deficit + maintenance for the first 24 hrs.
- Day 2: 1/2deficit + maintenance for the second 24 hrs.
- Usual replacement fluid is D5 1/5 NS or D5 ½ NS.
- If  $\text{Na} > 180 \text{ mmol/l}$ , may need dialysis.

In child **with some dehydration**:

- Rehydrate under observation in the clinic or ORT corner.
- Give 75ml/kg ORS over 4 hrs . Then reassess the child and classify the child's dehydration.
- Nasogastric (NG) feeding allows continuous administration of ORS at a slow, steady rate for patients with persistent vomiting, oral ulcers or who refused to take ORS.
- Check blood film for malaria if the child is febrile.

**Management of child with persistent diarrhoea:**

Assess and classify dehydration and manage accordingly.

1. Assess and classify nutritional status and manage accordingly.
2. Check for lactose malabsorption and other causes of malabsorption.

**Use of antibiotic:**

Antibiotic should be used only for:

- Dysentery : First line antibiotic for Shigellosis is 3rd generation Cephalosporin, Cefixime .If not available use Nalidixic acid or Co-trimoxazole .
- Giardiasis and Amoebiasis: Give Metronidazol.
- Cholera: first-line antibiotic is Tetracycline ( children > 8years) or Erythromycin (children < 8years) .

**Antimotility, antiemetic and anti diarrhoea** drugs should not be used.

## Guidelines of management Of severe malnutrition

### Criteria for admission

1. Wt/ ht less than 3rd percentile ( < 70 % and/or oedema of the both feet).
1. Malnourished children presented with:-
  - ✓ Diarrhoea..
  - ✓ Respiratory infection.
  - ✓ Septicaemia.
  - ✓ Dehydration and shocked child. .
  - ✓

### Hypoglycaemia

- **Treatment:**

- ❖ If the child is conscious give 50 ml of 10 % glucose or 10 % sucrose solution orally or by NG tube.
  - ❖ Then start F-75 every 30 minutes for 2 hours.
  - ❖ Feeds two hourly day and night.
  - ❖ Give antibiotics.
- If the child is unconscious lethargic or convulsing, give IV sterile glucose 10 % 5 ml / kg. Followed by 50ml of 10% glucose or sucrose by NG tube .
- Then start feeding F-75.
  - Two hourly feeds day and night.
  - Give antibiotics.

- **Monitoring**

1. Check blood glucose after 2 hours.
2. If it is < 3 mmol/L, give further 50 ml bolus of 10% glucose or dextrose.
3. Continue feeding every two hours till blood glucose become 3 mmol/L.
4. If rectal temp is < 35.5 c, repeat the bolus.
5. If level of consciousness is deteriorated, repeat the bolus.

- **Prevention**

- Feed every two hours.
- Always give feeds through the day and night.

## 2. Hypothermia

- If the axillary temperature is < 35 c or rectal temp is < 35.5 c:
  1. Feed straight away (or start rehydration).
  2. Rewarm the child (clothes, blankets, heater, or lamp nearby, bottles are dangerous).
  3. Kangaroo technique: placing the child on the mother's bare chest & abdomen and covering both of them.
  4. Give antibiotics.

- **Monitor :**

1. Body temp, rectally every 2 hrs. till rises above 36.5 c.
2. Child must be covered all time esp. at night.
3. Blood glucose level whenever there is hypothermia.

- **Prevention :**

1. Feed 2 hrly throughout day and night.
2. Keep the child dry.
3. Avoid exposure (bathing or prolonged medical examination).
4. Let child sleep beside his mother esp. at **night**.

## 3. Dehydration

- Dehydration progresses from “some” to “severe”, reflecting 5 – 10% and > 10% wt. loss, respectively, whereas septic shock progresses from “incipient” to “developed”, as blood flow to the vital organs decreases.
- Don't use IV line for rehydration except in shock, carefully and slowly to avoid fluid overload.

## Classification of Dehydration

Clinical signs	Some dehydration	Severe dehydration
Watery diarrhoea	Yes	yes
Thirst	Drinks eagerly	Drinks poorly
Hypothermia	NO	NO
Sunken eyes	Yes	yes
Weak radial pulse	No	yes
Cold hands & feet	No	yes
Urine flow	Yes	No
Mental state	Restless, irritable	Lethargic, comatosed

- **Treatment :**

1. Use Resomal ( rehydration solution for malnutrition).
2. Give 5ml/kg every 30 min for first 2 hrs orally or by NG tube.
3. Then 5 – 10ml/kg hrly in the next 4 – 10 hrs.
4. Start feeding F- 75.

- **Monitoring :**

1. Observe the vital signs, urine and stool frequency& vomiting, every  $\frac{1}{2}$  hrly for 2 hrs then hrly for 6 – 12 hrs.
2. Stop to give fluid if continuous rapid breathing and pulse (infection or over hydration) or oedema and puffiness of eyes.

- **Prevention :**

1. Keep feeding with F – 75.
2. Replace the volume of stool losses with Resomal (50 – 100ml after each watery stool).
3. Encourage breast feeding.

#### **4. Electrolyte imbalance**

- Oedema is a result of electrolyte imbalance, that's why we don't treat it with diuretics.
- Give:-
  1. Extra potassium ( 3 – 4 mmol/kg/day).
  2. Extra magnesium ( 0.4 – 0.6 mmol/L/kg/day).
  3. Give low sodium rehydration (Resomal).
  4. Prepare food without salt.

\* 20 ml of combined electrolyte/ mineral solution or Resomal to 1 litre of feed will supply the requirement of K and Mg.

#### **5. infection**

- **Give:**
  1. Broad spectrum antibiotics.
  2. Measles vaccine if the child above 6 month of age and not immunized (delayed in shocked pt).
  3. May give metronidazole ( 7.5 mg/kg/8hrly for 7 days) routinely in addition to antibiotics for:
    - ❖ Hasten repair of intestinal mucosa.
    - ❖ Reduce risk of oxidative damage.
    - ❖ Reduce systemic infection arising of anaerobic bacterial infection in small intestine.

- **Drugs of choice:-**

1. If no complications : cotrimoxazole (5ml = 40 mg TMP + 200 mg SMX).

< 6month: 2.5 ml / BD / for 5 days.

> 6 month : 5 ml / BD / for 5 days.

2. If the child is severely ill and has complications (hypoglycaemia, hypothermia, UTI..etc): ampicillin (50 mg/kg IM or IV / 6 hrly for 2 days) then orally amoxycillin (15 mg /kg/ 8hrly for next 5 days),

And : Gentamycin (7.5 mg/kg IM or IV once daily for 7 days).

3. After 48 hrs if child fail to improve : add chloramphenicol 25 mg/kg IM or IV 8 hrly for 5 days.

- If specific infections are identified add the specific antibiotics.
- If anorexia persist after 5 days of antibiotics: treat a full complete 10 days course.
- If anorexia still persist :Reassess the child fully :
  1. Site of infection.
  2. If it is resistant organism.
  3. Ensure vitamin and minerals supplements.

# HEAT STROKE

## Definition :

- Is a symptom complex produced by excessive body heat , or body temp higher than 41,1°C (106°F).

## Clinical picture:

### Onset :

- The onset is usually acute in 80% of patients with few patients (20%) having prodromal symptoms and signs lasting for minutes to hours. These include : Dizziness , weakness, confusion , drowsiness , nausea, and anorexia, anxiety and headache , disorientation , disassociation, a staring and apprehensive expression , apathy , irritability , aggressiveness , irrationality , mania or psychosis , tremors , twitching , convulsion, ataxia and cerebellar dysfunction.

- Vital signs:

**Temperature:** Typically, the patient's temperature exceeds 41°C, but, in the presence of sweating, evaporating mechanisms, and the initiation of cooling methods, body temperatures can be lower than 41°C .

- **Pulse:** Tachycardia to rates exceeding 130 beats per minute is common.
- **Blood pressure:** Patients commonly are normotensive, with a wide pulse pressure; however, hypotension can occur.
- Symptoms of CNS dysfunction are present universally in persons with heatstroke. Symptoms may range from irritability to coma. Patients also may exhibit decerebrate posturing, decorticate posturing, or they may be limp.
- Examination of the eyes may reveal nystagmus and oculogyric episodes due to cerebellar injury. The pupils may be fixed, dilated, pinpoint or normal.
- Patients commonly exhibit a hyperdynamic state, with tachycardia tachypnoea and hyperventilation.
- Acute renal failure (ARF) is a common complication of heatstroke.

## **Management :**

1. Take investigations including CBC, urea and electrolytes, BF for malaria, blood culture and these based on history and physical examination.
2. Maintain cooling by fine spray of tap water and fanning & don't use ice.
3. No role for anti pyretics.
4. Take the necessary and appropriate measures required for treatment of shock using normal saline.
5. Give IV fluids (deficit+maintenance).

# Hypertensive crises in children

**Definitions:** ((APLS) - pediatric emergency medicine resource)

- Check blood pressure percentile chart.
- Hypertensive emergency: end-organ damage: neuro changes, pulmonary oedema, myocardial ischemia and severe proteinuria.
- Hypertensive encephalopathy: vomiting, vision problems, seizures and stroke.

Other definition to HPT urgency is BP more than 99<sup>th</sup> percentile by sex, age and height without organ damage.

## Management:

- The goal is to lower the BP not below the 95<sup>th</sup> percentile until 24 to 48 hours after presentation and initially.  
The goal is to lower the blood pressure 10%-20% over hours.
  - If there had been evidence of an intracranial bleed, the blood pressure should have been lowered over minutes.
  - The drugs used are all given by IV. It is best to use those that can be given by infusion so that they can be titrated to clinical response, yet avoid hypotension.
  - These include nitroprusside (0.3-0.5 µg/kg/min IV)(if **available**) max. dose 8 microgram /kg/min, in continuous infusion.
  - Hydralazine (0.1-0.2 mg/kg IV), max.dose 3.5mg/kg/day.
  - Esmolol (load of 100-500 µg/kg IV followed by 25-100 µg/kg/min IV) (**if available**) load over 1 to 2 mints.
  - Labetalol (0.2-1mg/kg bolus push over 2 minutes followed by 0.4-1 mg/kg/h IV) (**if available**) max. dose 40mg.
  - Phentolamine (0.1 mg/kg IV).(catecholamine production tumor).
- 
- β-Blockers are **contraindicated** in patients with decreased cardiac output and signs of congestive heart failure.
  - Oral nifedipine is **contraindicated** in patients with signs of end-organ damage (**intracranial bleed**).

- With administration of these medications, it is essential to follow symptoms and blood pressure , patient symptoms and BP were monitored in the **PICU**.

### E-medicine 2008

- Labetalol 0.2-1 mg/kg/dose up to 40 mg/dose as an intravenous (IV) bolus or 0.25-3 mg/kg/h IV infusion.
- Nicardipine 1-3 mcg/kg/min IV infusion.
- Sodium nitroprusside 0.53-10 mcg/kg/min IV infusion to start.
- Sublingual Nifedipine is no longer recommended\*

### Recommended medications for:

**Hypertensive encephalopathy** is nitroprusside or labetalol

**Sudden and severe HPT** is nitroprusside or labetalol or esmolol.

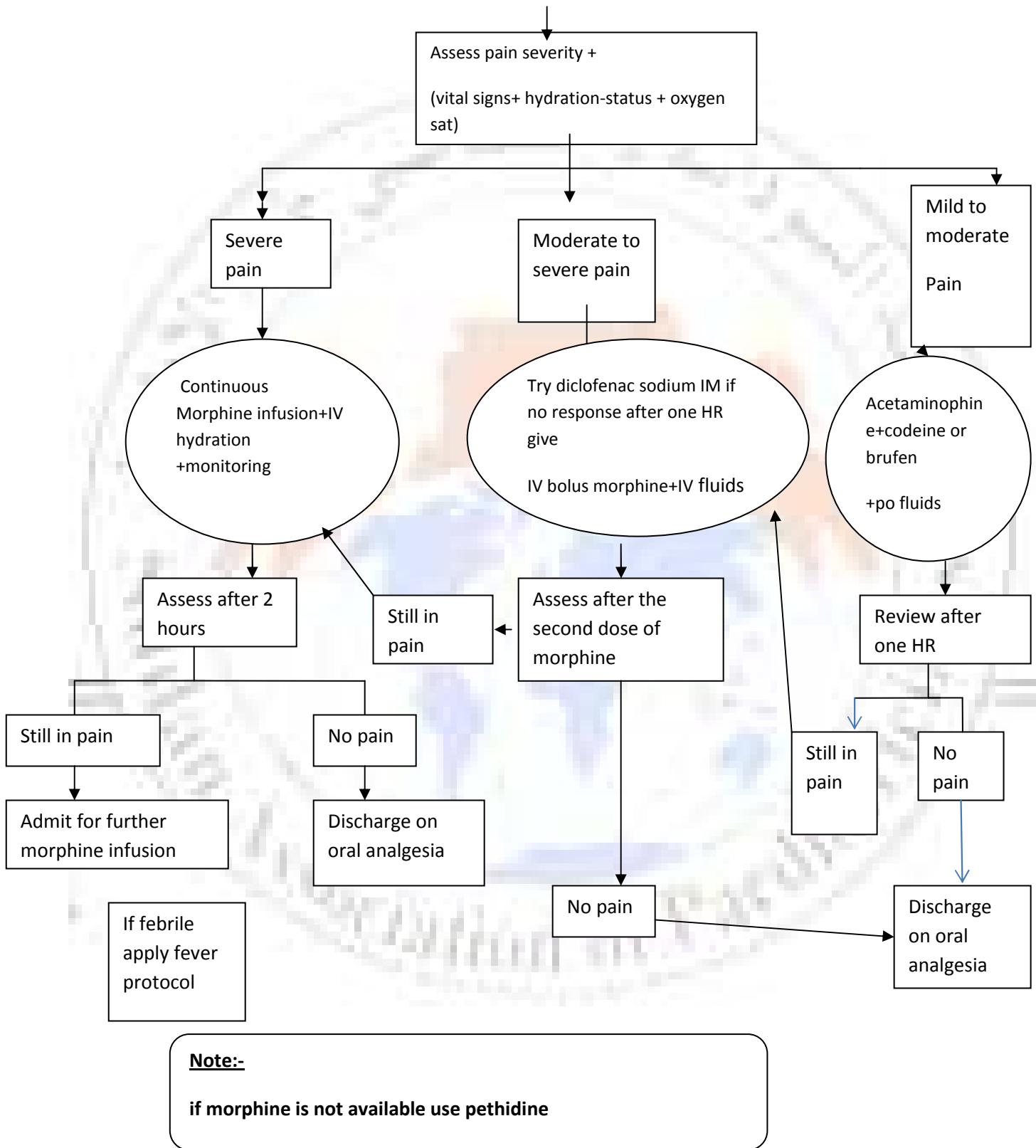
**HPT with intracranial hemorrhage** nitroprusside or labetalol (**don't** use **hydralazine**).

**Catecholamine production tumor** phentolamine (eg pheochromocytoma)

# Sickle cell anaemia

## Sickle cell crises

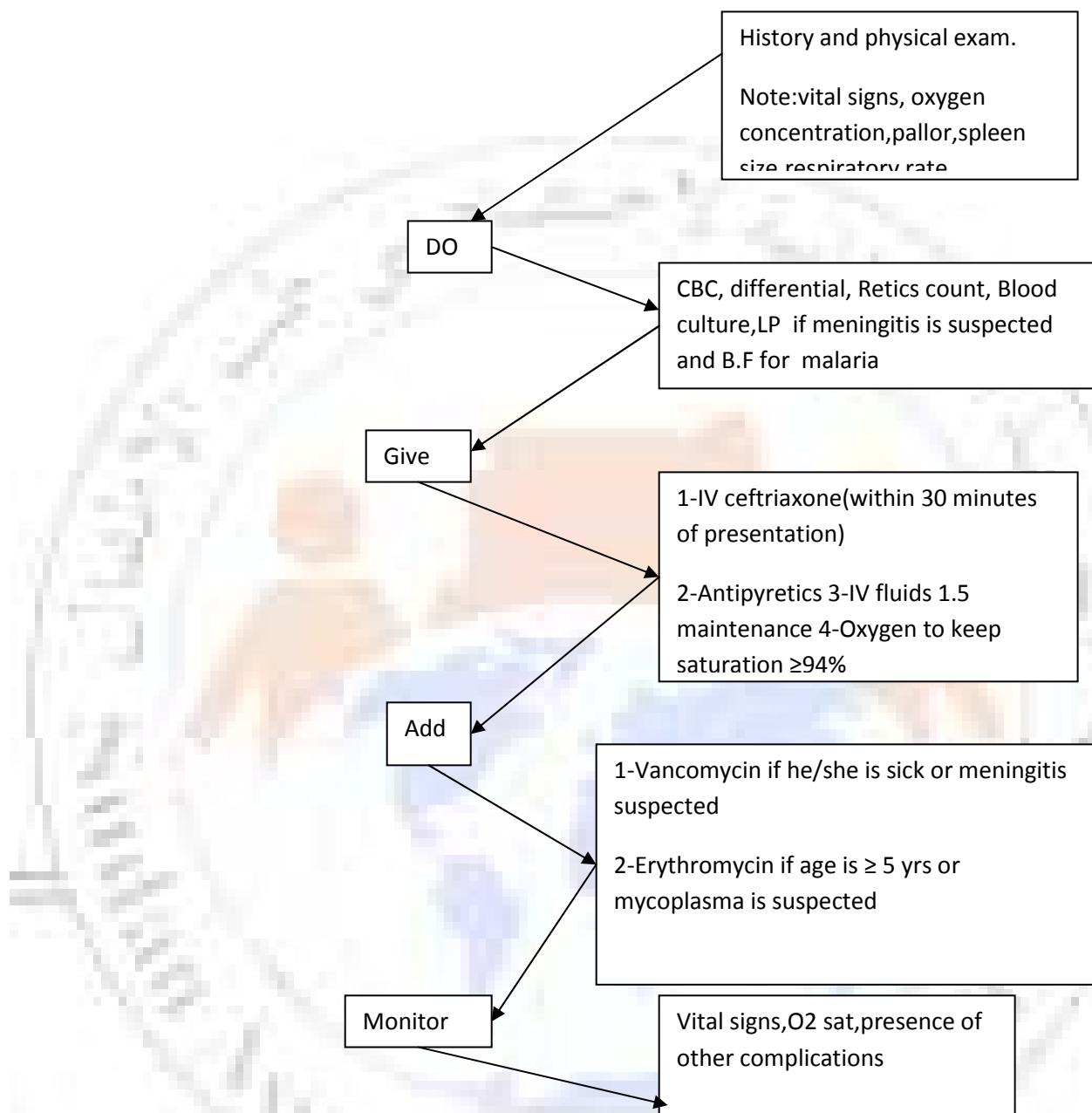
### Vaso - occlusive (painful crises)



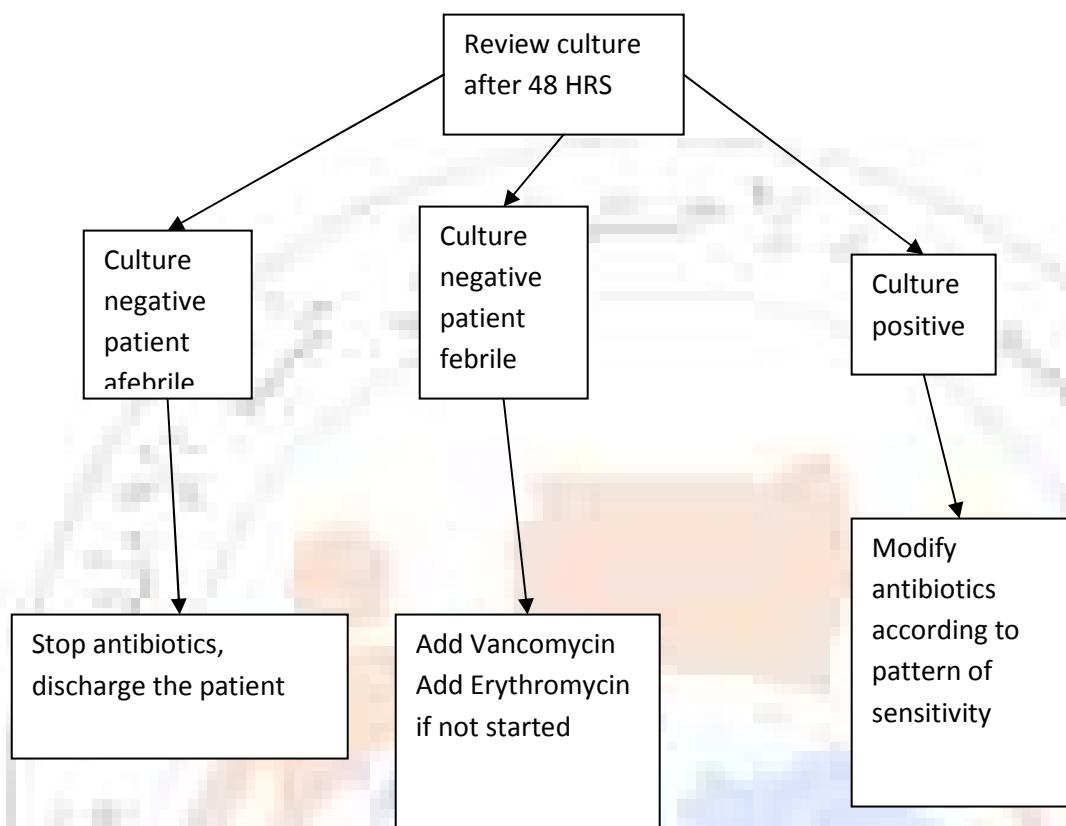
### Sickle cell anemia with fever

Admit all febrile sicklers to hospital

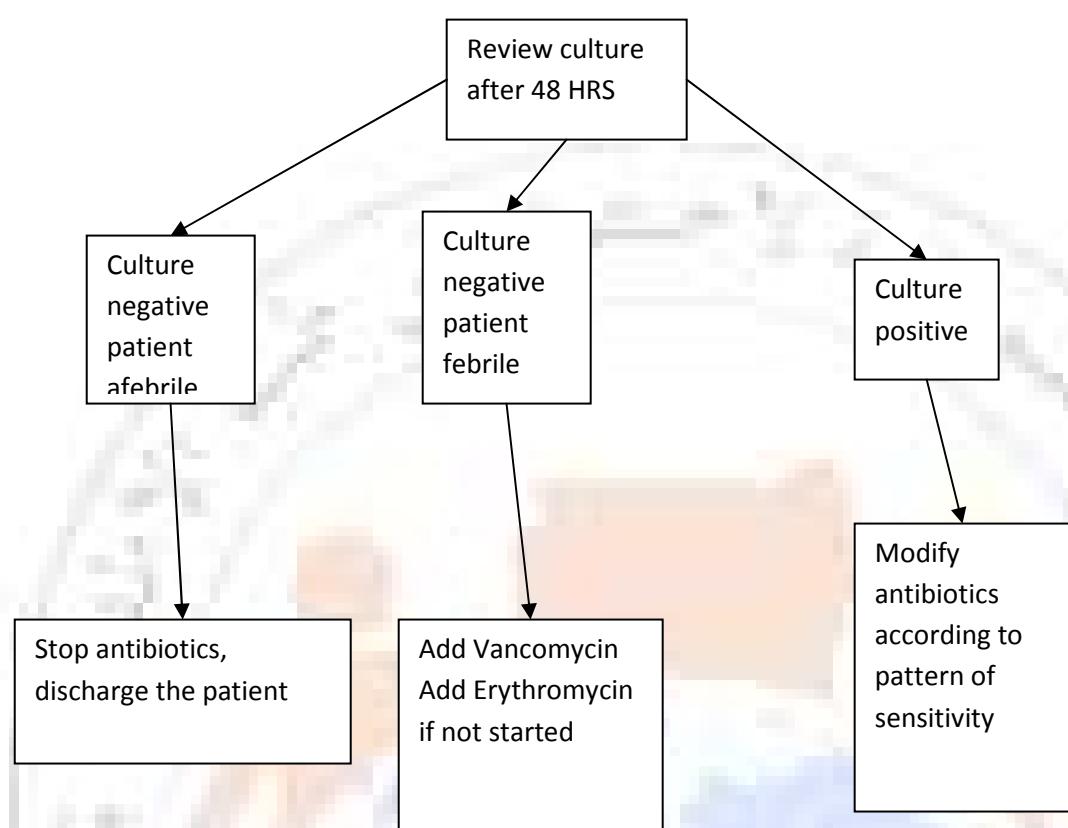
Febrile (temp 38.3 oral or 37.8 axillary)



## The febrile sickler protocol



## The febrile sickler protocol



## Sickle cell anaemia / acute chest syndrome

History and physical note:Vital signs,respiratory distress,hydration status,oxygen saturation

Do:CBC,blood culture,chest x-ray,blood type and cross match (if in severe distress)

Give:IV fluids(maintenance)-IV ceftriaxone-Oxygen to keep saturation  $\geq 94\%$ -Analgesics

Add:1-Vancomycin if sick.2-Diuretics if fluid overload is suspected.3-Bronchodilators if wheezing or has history of asthma

Give blood transfusion if: in moderate distress and hemoglobin  $\leq 6.0$  gm/dl or HB dropped by 1.5gm/dl from baseline HB

Exchange transfusion if:1-Has extensive infiltrate on x-ray .2-Needs  $\geq 40\%$  oxygen.3-Needs admission to ICU

# Sickle cell anaemia / Stroke

Stroke includes

Hemiplegia, hemiparesis, aphasia, convulsions,

Transient ischemic attacks

1-Stabilize vital signs and provide life support as necessary.

2-Treat seizure and increased intracranial

1-Do: MRI brain and MRA, if not available do CT scan brain to rule-out hemorrhage if not available proceed next.

2-CBC, retics, electrolytes, blood typing and cross

Treatment:

1-cefotaxime IV if febrile

2-Exchange transfusion

3-post-exchange HB electrophoresis(aim at HB S <30%)

Monitor vital signs

# Sickle cell anaemia / Priapism

## Management :

At home:

Exercise, Urination, Analgesia, Fluid intake, Warm bath.

Report to hospital if not resolved after three hours



At hospital:

1-IV hydration(1.5 maintenance).

2-Analgesia

3-Catheterization

If no response in 6 hours

Simple blood transfusion: 10 ml/kg of packed RBCS

If no response in 24 Hours

Exchange transfusion

If no response in 24 Hours: surgery

## **Drugs :**

1. **Acetaminophen:** 10 - 15 mg/kg/dose 4 hourly p.o (maximum dose 60 mg /kg/day) if the patient has not taken it before.

Rectal acetaminophen dose 25 – 40 mg / kg / dose 4 hourly.

2. **Ibuprofen sup:** 10 mg /kg/dose 6 hourly (maximum dose 40 mg/ kg/ day).

3. **Tramadol :** ( not to be used below age of one year )

Dose: 1-2 mg /kg/ dose every 4-6 hours.

NB: Excessive doses cause seizures.

4. **Intravenous Morphine:** Indicated in all patients admitted.

A-Starting dose: 0.1 0.15 mg / kg / dose IV.

B-Reassess for pain relief after 30 minutes if:

- No relief and patient not sedated, give 50 % of starting dose.
- Mildly sedated, pain is present but less than before, give 25% of starting dose.

Continue reassessment of pain hourly – repeat bolus doses as above until pain is tolerable.

Then maintain analgesia by morphine infusion; 0.05 mg /kg/hour, offer 25% of hourly infusion as bolus p.r.n 2 hourly if there is intermittent pain .

5. **Pethidine:** Use if morphine is not available or patients develop pruritus and vomiting which are not responsive to symptomatic treatment. Dose 0.05 -2 mg/kg /dose 4 hourly IV – start with small dose and titrate.

### **6. Other medications for patients on IV opioids:**

1. Lactulose syrup: 0.5 ml / kg /dose 12 hourly.

2. Diphenhydramine HCL (for itching) or equivalent, 1-2 mg kg /dose 6-8 hourly p.o.

3. Oxygen: To maintain SPO<sub>2</sub> ≥ 96%.

4. Amphetamines: Used in patients who develop severe sedations with opioids (opioids – induced narcolepsy).

Dexamphetamine: 0.2 mg/ kg p.o daily with breakfast (maximum 10 mg) increase up to 0.6 mg/kg/day (maximum 30 mg).

**7. Cefotaxime:** (200 mg /kg/ day IV ÷ q6 – 8h: max 8g / day)

**8. Clindamycin:** ( 40 mg /kg/day IV ÷ q6 – 8h : max 2.7 g / day )

**9. Erythromycin** (40 mg /kg/ day IV÷ q6 – 8h: max 4 g / day )

**10. Vancomycin :** ( 60 mg / kg / day IV ÷ q6h : max 4g / day )

**11. Penicillin:** (250.000 units /kg/day IV÷ q 4-6 h: max 20 million

Units/ day).

**12. Ceftriaxone:** (80 mg / kg / dose IV, given q 12 h)

**13. Cefixime:** (8 mg /kg/day, given once daily: max 400 mg / day)

**14. Cefaclor:** (40 mg /kg /day ÷ TID: max 1.5 g/ kg day)

or

**Cefuroxime axetil 30 mg / kg / day p.o ÷ bid: max 1 g / day**

As suspension or 250 mg p.o bid as tablets.

### **15. Formula for volume of red cells for transfusion:**

- Whole blood donor unit average PCV = 35%

- Packed red cell unit average PCV = 70%

**Transfusion volume = (total blood volume X (PCV target – PCV**

**Pre transfusion) / PCV of donor unit.**

**Example:** For 30 kg child with pre – transfusion PCV 25 %, goal PCV

32 % average PRBC unit PCV 70 %.

$$\{(75\text{ml/kg} \times 30) (0.32- 0.25)\} / 0.70 = 225 \text{ ml (7.5 ml/kg)}$$

\*The Hb should never be raised acutely to > 10g/dl or haematocrit to > 0.35 since this is likely to incase the blood viscosity.

\*Frusemide is not given with transfusions in SCD because of the increase in viscosity that may result.

\*Do not use a sickle positive blood.

\*Packed red blood cells should be used except when blood volume expansion is needed.

# Transfusion therapy

Transfusion therapy is not without risks. The most common are iron overload, allo immunization, and transmission of infections such as hepatitis or HIV. Since patients with sickle cell disease may require multiple transfusions over the course of their lives ,the risk are multiplied, and except in acute emergencies, transfusions should only be given in consultation with a haematologist . Transfusion is not indicated in chronic, steady-state anaemia, uncomplicated acute painful crisis, infection, uncomplicated pregnancy, or avascular necrosis.

Initial characterization of a patient's red blood cell antigen (phenotyping) is advisable at an early age or when first red cell antibodies have developed. Select donor blood to closely match the patient's phenotype. Extended cross matching for minor antigens is desirable. The patient transfusion history should be checked for previous evidence of allo immunization, as antibody titers may be undetectable months to years after the antibody challenge.

Leukocyte-depleted PRBCs, preferably prefiltered blood and blood product should be used.

Fresh blood less than 5-7 days old must be used.

Use sickle cell negative blood to facilitate proper monitoring of post transfusion level of haemoglobin S through haemoglobin electrophoresis.

Monitor outcome of transfusion therapy by measuring haemoglobin/hematocrit.

## Types of transfusion:

**\*Simple transfusion:** for severe anemia (<5g/dl); a plastic crisis, sequestration crisis, though the decision depends on the patient's clinical status, haemoglobin and hematocrit and the reticulocyte count, or prior to surgery when general anaesthesia is anticipated.

Volume of PRBC to be transfused should be adjusted to the pre-transfusion haemoglobin level to avoid cardiac overload.

**\*: Chronic transfusion:**for stroke prevention, to achieve an S haemoglobin level < 30%: chronic leg ulcers when local measures are unsuccessful; frequent priapism (for prolonged

cases lasting more than 6 – 12 hours , consider a single volume exchanges ).

**\*Exchange transfusion or partial exchange transfusion** in specific cases, and when appropriate, for a acute chest syndrome and cerebral vascular accident.

## Post transfusion (simple, chronic or exchange ) haemoglobin level should never

**Exceed 11 grams.**

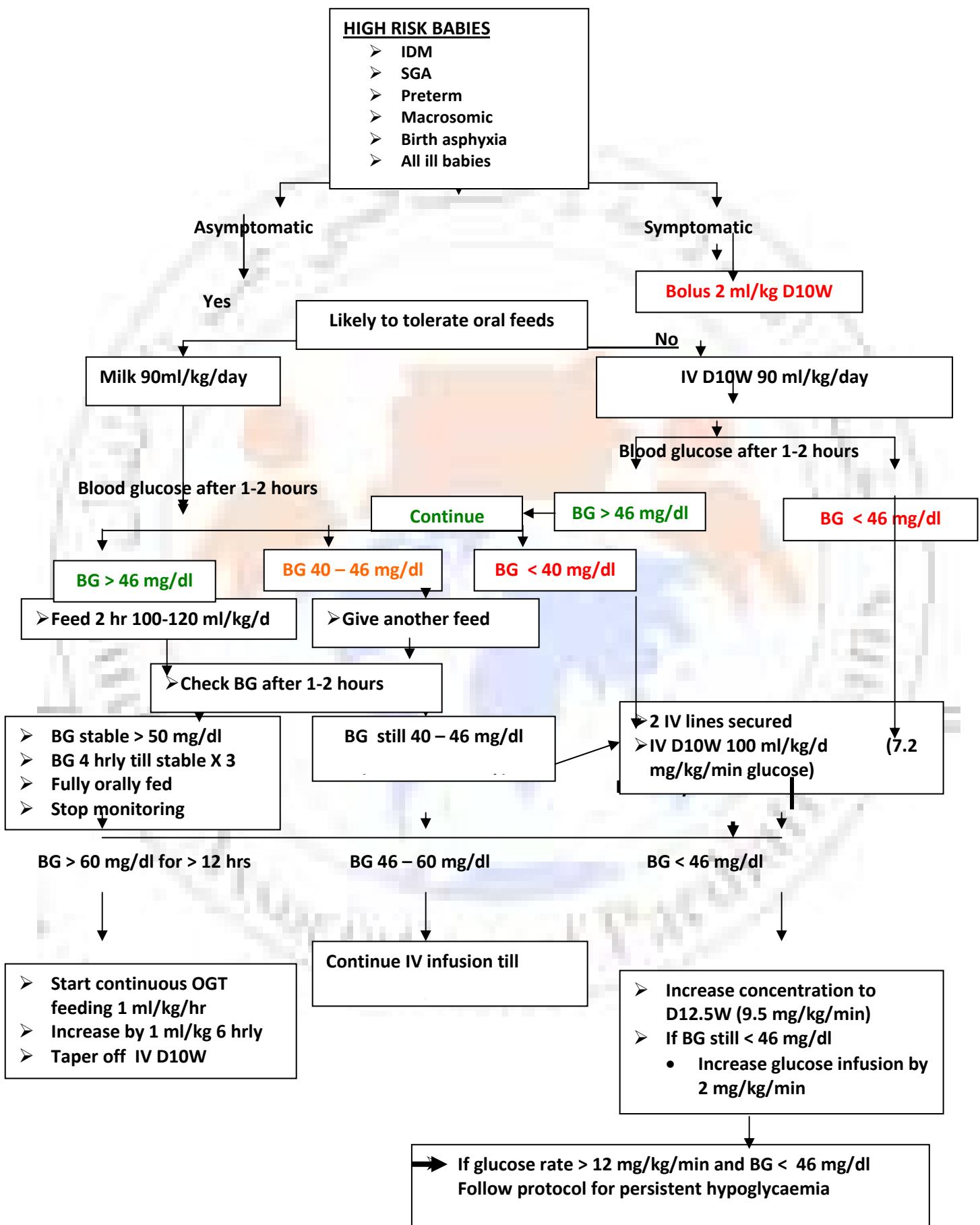
Periodic assessment of iron stores and prompt attention to any abnormalities noted are imperative. Most of these patients require chelation therapy.

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## Management of neonatal hypoglycaemia

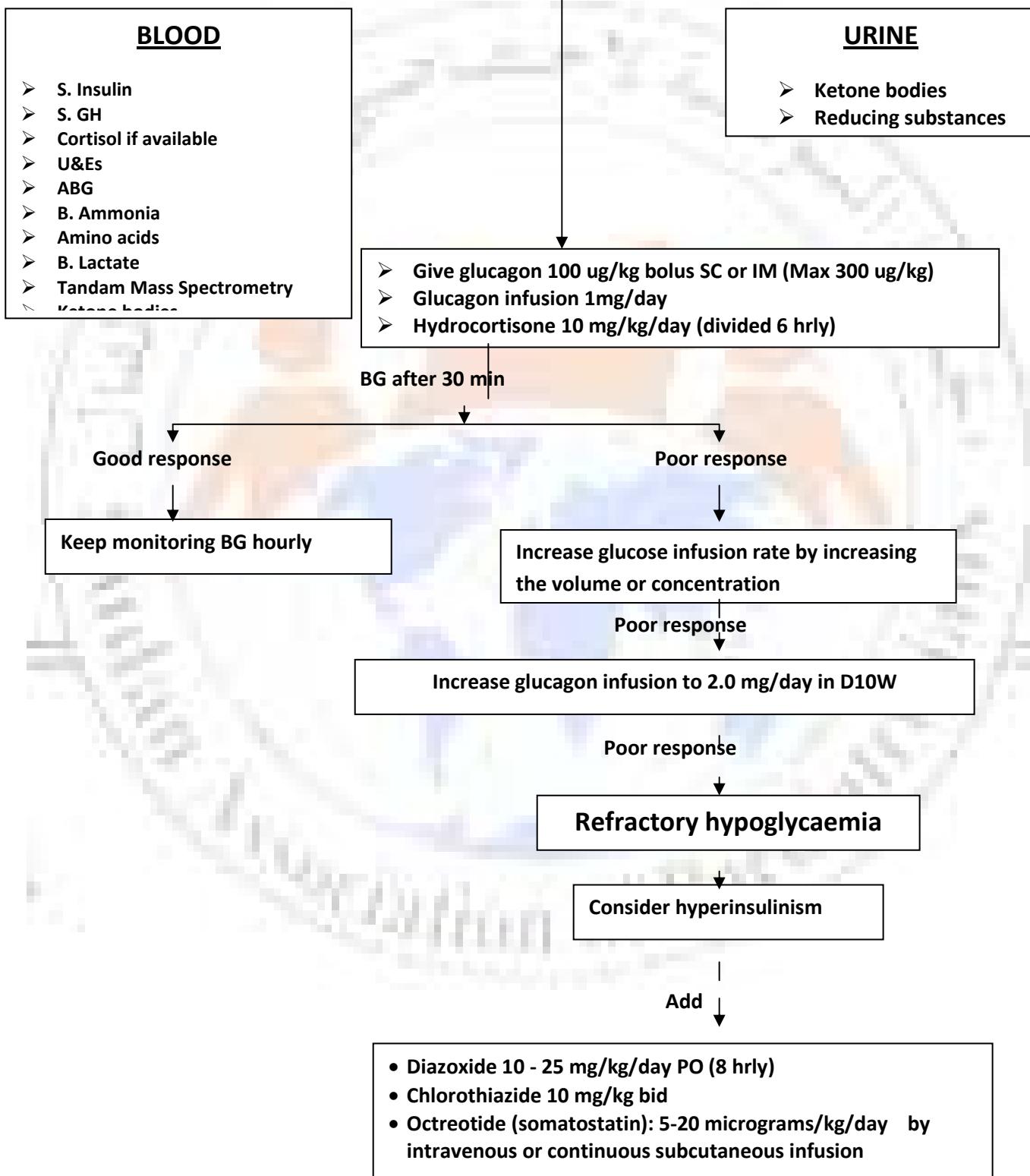
**Definition:** blood glucose less than 2.6 mmol/l (46mg/dl)



## Protocol for persistent hypoglycaemia

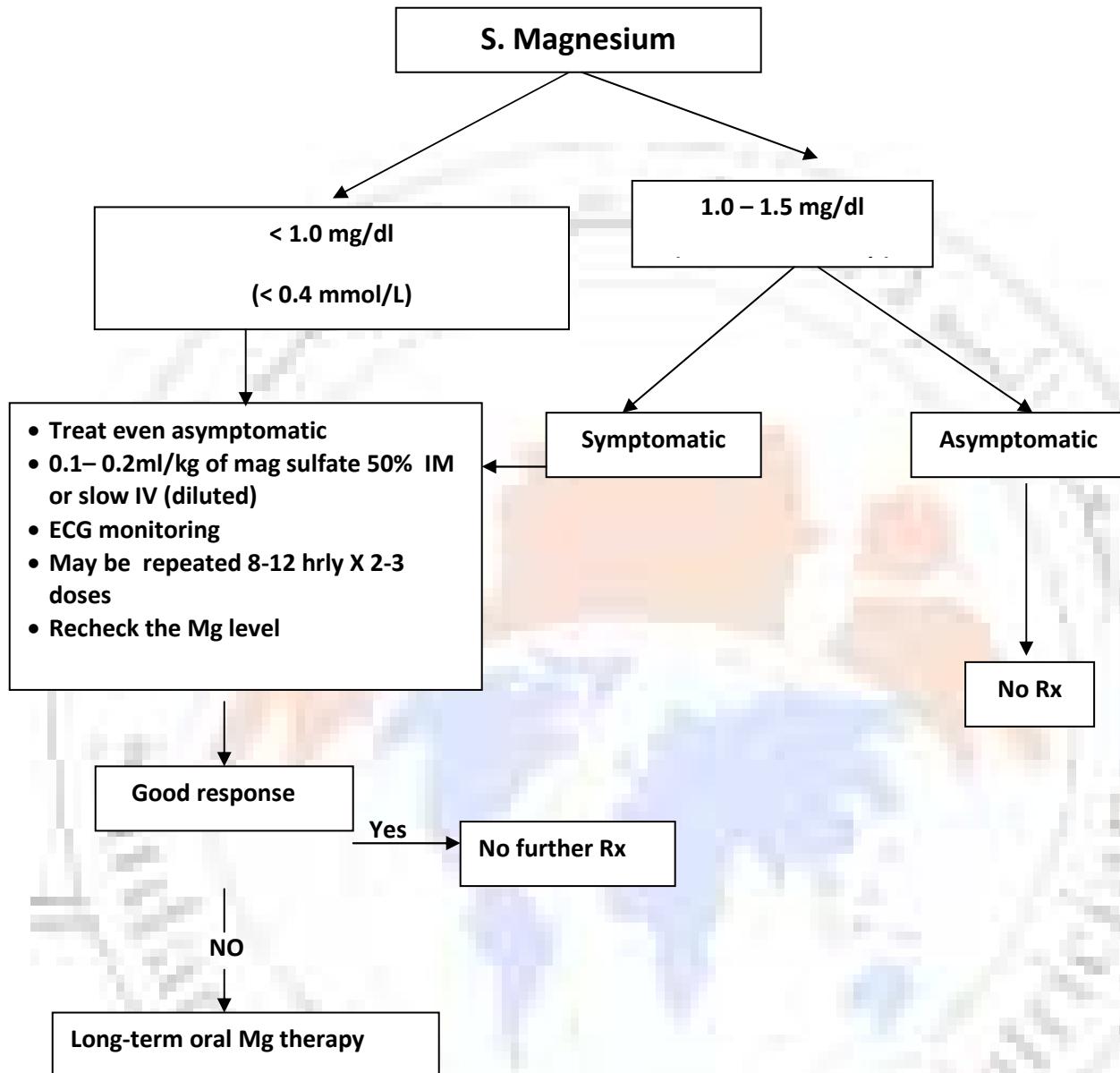
BG < 46 mg/dl (< 2.6 mmol/l) on glucose infusion rate > 12 mg/kg/min

- Discuss with the pathologist (Lab)
- Take blood during hypoglycaemia
- Discuss with the consultant neonatologist on call

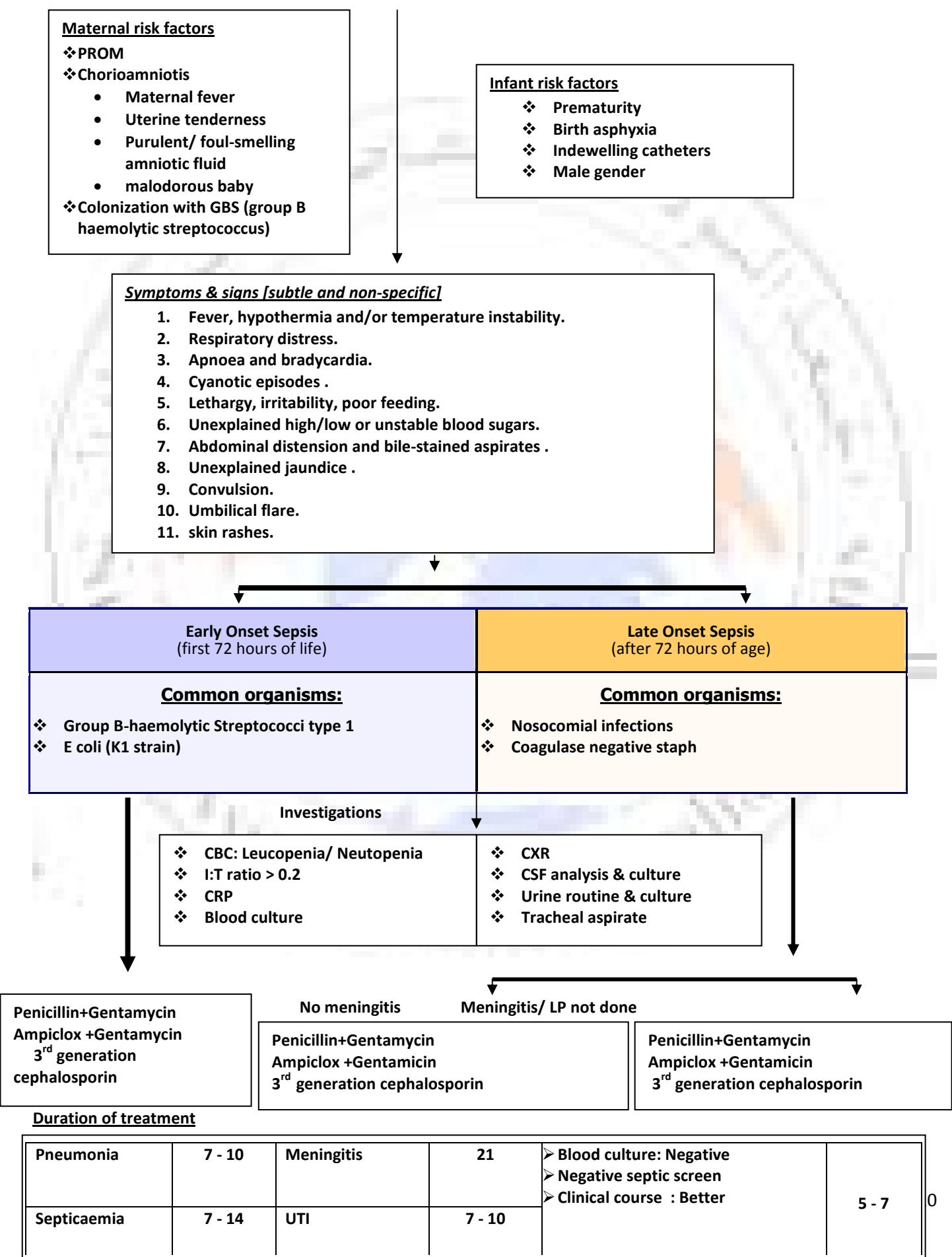


## Serum magnesium <1.5 mg/dl (0.6 mmol/L)

Consider hypomagnesaemia if hypocalcaemia persists despite conventional treatment



## Neonatal sepsis



## Components of septic screen

Component	Abnormal value
Total leucocyte count	< 5000/ mm <sup>3</sup>
Absolute neutrophil count	< 1800/ mm <sup>3</sup>
Immature / total neutrophil	> 0.20
Micro-ESR	> 15 mm in 1 <sup>st</sup> hour
CRP	> 1 mg/ dl

## Normal CSF examination in neonates

CSF Components	Normal range
Cells/ mm <sup>3</sup>	8 (0-30 cells)
PMN (%)	60%
CSF proteins (mg/dl)	90 (20 - 170)
CSF glucose (mg/dl)	52 (34 – 119)
CSF/ Blood glucose (%)	51% (44 – 248%)

## Drugs , route of administration and doses of common antibiotics used in NICU

Drug	Route	BW < 2000 g		BW > 2000 g	
		0 – 7 days	> 7 days	0 – 7 days	> 7 days
Amikacin	IV/ IM	7.5 mg q 12 hrs	7.5 mg q 8 hrs	10 mg q 12 hrs	10 mg q 8 hrs
Ampicillin <i>Meningitis</i> <i>Others</i>	IV IV/IM	100 mg/kg q 12 hrs 25 mg/kg q 12 hrs	100 mg/kg q 8 hrs 25 mg/kg q 8 hrs	100 mg/kg q 8 hrs 25 mg/kg q 8 hrs	100 mg/kg q 6 hrs 25 mg/kg q 6 hrs
Cefotaxime <i>Meningitis</i> <i>Others</i>	IV IV/IM	50 mg/kg q 6 hrs 50 mg/kg q 12 hrs	50 mg/kg q 6 hrs 50 mg/kg q 8 hrs	50 mg/kg q 6 hrs 50 mg/kg q 12 hrs	50 mg/kg q 6 hrs 50 mg/kg q 8 hrs
Piperacillin + Tazbactam	IV	50 –100 mg/kg q 12 h	50 – 100 mg q 8 hrs	50 –100 mg/kg q 12 hrs	50 – 100 mg q 12 hrs
Ceftriaxone	IV/IM	50 mg/kg q 24 hrs	50 mg/kg q 24 hrs	50 mg/kg q 24 hrs	75 mg/kg q 24 hrs
Ciprofloxacin	IV/ PO	10-20 mg/kg q 24 h	10-20 mg/kg q 24 h	10-20 mg/kg q 12 hrs	10-20 mg/kg q 12 h
Cloxacillin <i>Meningitis</i> <i>Others</i>	IV IV	50 mg/kg q 12 hrs 25 mg/kg q12 hrs	50 mg/kg q 8 hrs 25 mg/kg q 8 hrs	50 mg/kg q 8 hrs 25 mg/kg q 8 hrs	50 mg/kg q 6 hrs 25 mg/kg q 6 hrs
Gentamycin <i>Conventional</i> <i>Single dose</i>	IV IV/IM	2.5 mg/kg q 12 hrs 4 mg/kg q 24 hrs	2.5 mg/kg q 8 hrs 4 mg/kg q 24 hrs	2.5 mg/kg q 12 hrs 5 mg/kg q 24 hrs	2.5 mg/kg q 8 hrs 5 mg/kg q 24 hrs
Netilmicin	IV/IM	2.5 mg/kg q 12 hrs	2.5 mg/kg q 8 hrs	2.5 mg/kg q 12 hrs	2.5 mg/kg q 8 hrs
Penicillin G <i>Meningitis</i> <i>Others</i>	IV IV/IM	75000 -100000 U/kg q 12h 25000 units/kg q 12 hrs	75000-100000 U/kg 12h 25000U/kg q 8 hrs	75000 -100000/kg q 8 h 25000 units/kg q 8 hrs	75000-100000 U/kg q 6 h 25000U/kg q 6 hrs
Vancomycin	IV	15 mg/kg q 12 hrs	15 mg/kg q 8 hrs	15 mg/kg q 12 hrs	15 mg/kg q 8 hrs

## Bleeding in neonates

### Causes

Haemorrhagic disease of the newborn.

Neonatal sepsis + DIC

Congenital coagulation defects

Maternal drugs

Trauma

### History

Mode of delivery

Neonatal sepsis

F.H of bleeding tendency

Maternal drugs like anticonvulsants

( phenytoin , phenobarbitone)

### Examination

Sites of bleeding:

- Umbilicus
- Skin ( Petechiae , purpura , echymosis)

Signs of shock and anaemic heart failure

### Investigation

CBC

Bleeding profile

Blood grouping and cross

matching

### Management

Assessment of the vital signs

Manage sites of bleeding

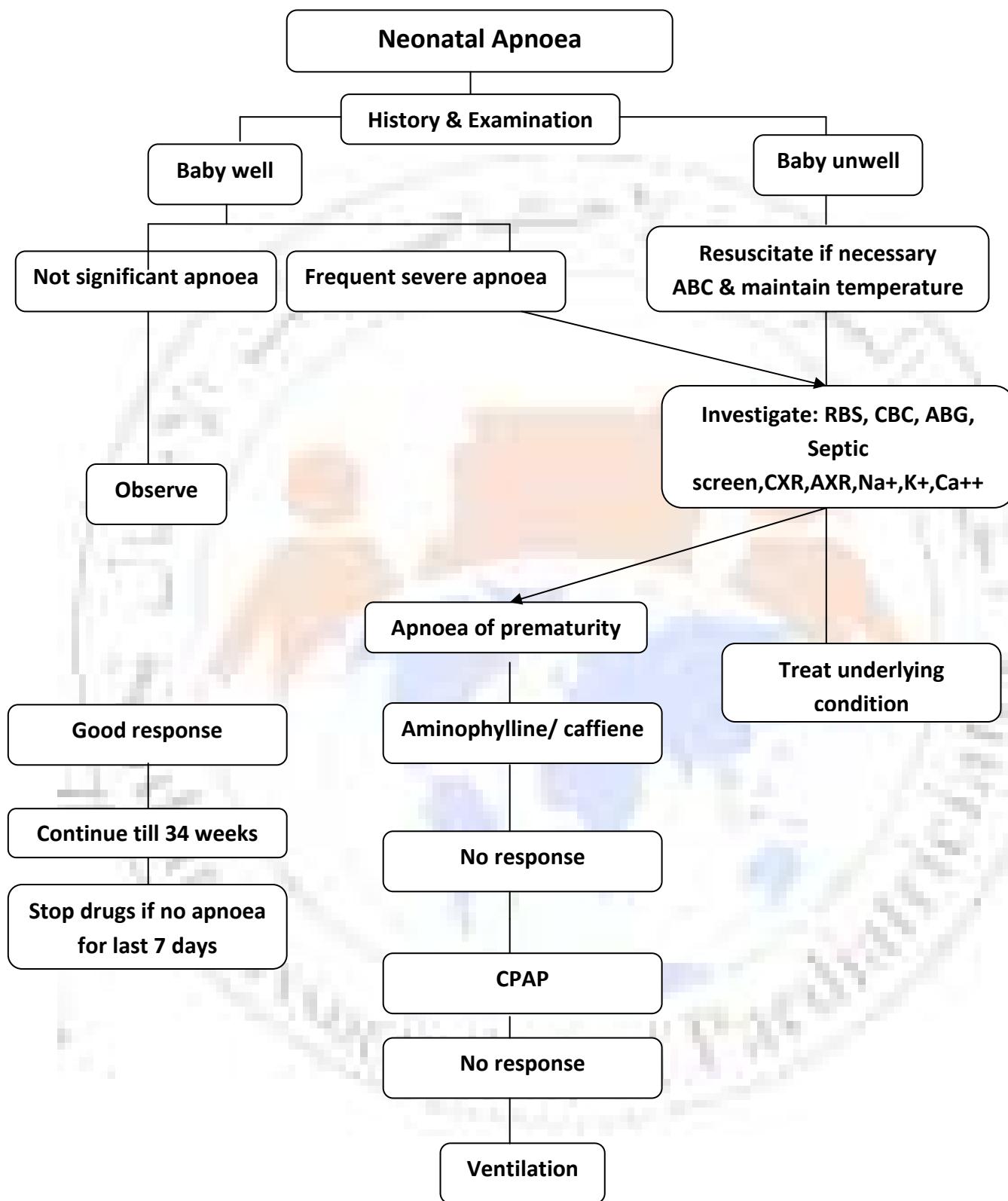
Insure intravenous line

Vit K 1-5 mg IV

Fresh frozen plasma or whole blood 10-15 ml/kg .

IV antibiotic and treatment of the cause

## Flow diagram for treatment of apnoea



# **Neonatal Apnoea**

## **Definition:**

Apnoea is defined as cessation of respiration for >20 sec or cessation of respiration of any duration accompanied by bradycardia (HR <100/min) and/or cyanosis.

All newborns less than 34 weeks gestational age, or less than 1800 grams birth weight, should be monitored for both apnoea and bradycardia.

## **I. Sequelae:**

1. Apnoea in premature infants may lead to a reduction in the cerebral blood flow, resulting in ischemia and eventually leukomalacia.
2. During apnoeic episodes, in an attempt to protect cerebral blood flow, cardiac output is diverted away from the mesenteric arteries resulting in intestinal ischemia and possibly necrotizing enterocolitis (NEC).

## **II. Etiology:**

1. Apnoea of prematurity is the most common cause.
2. Infection: Sepsis, especially in the first day of life, and nosocomial infections and/or NEC in the first weeks of life.
3. Neurological: Intraventricular hemorrhage, intracranial hemorrhage, neonatal seizures, perinatal asphyxia, or other pathology which could lead to increased intracranial pressure.
4. Cardiovascular: congestive heart failure and pulmonary oedema (PDA, coarctation, etc.), cyanotic congenital heart disease.
5. Pulmonary: surfactant deficiency disease, pneumonia, transient tachypnoea of the newborn, meconium aspiration, etc.
6. Metabolic: Hypocalcaemia, hypoglycaemia, hyponatremia or acidosis.
7. Haematological: Anaemia.
8. Gastrointestinal: NEC or gastroesophageal reflux.
9. Temperature Regulation: Hypothermia or hyperthermia.
10. Drugs: Prenatal exposure with transplacental transfer to the neonate of various drugs (narcotics, beta-blockers). Postnatal exposure to sedatives, hypnotics or narcotics.

## **III. Pathophysiology:**

### **Mechanisms of apnoea of prematurity:**

#### **a. Central Apnoea:**

In other words, there is no signal to breathe being transmitted from the CNS to the respiratory muscles. This is due to immaturity of brain stem control of central respiratory drive.

#### **b. Obstructive Apnoea:**

- The pharynx collapses from negative pressure generated during inspiration. Neck flexion will worsen this form of apnoea.
- Excessive secretions in the nasopharynx and hypo pharynx may also cause obstructive apnoea.

#### **c. Mixed Apnoea:**

A combination of both types of apnoea representing as much as 50% of all episodes.

## **Management:**

- a. **Acute:** When the alarm sounds, the infant should immediately be observed for signs of breathing and skin colour. If apnoeic, pale, cyanotic or bradycardic, then tactile stimulation needs to be given. If the infant does not respond, bag and mask ventilation, along with suctioning and airway positioning, may be needed.

## **b. Chronic:**

- The commonest cause is apnoea of prematurity.
- Always diagnose and correct other potential etiologies, before attributing a specific neonate's apnoea to prematurity alone.
- The decision to initiate chronic therapy is based on clinical judgment. Factors to be considered include:
  - The frequency and duration of the episodes.
  - The level of hypoxia.
  - The degree of stimulation needed.
- Chronic management of apnoea of prematurity involves three major therapies:
  - i. **Pharmacologic Therapy** - The most common drugs used to treat apnoea are the methylxanthines: Caffeine (1,3,7-trimethylxanthine) and Theophylline (1,3-dimethylxanthine).
    1. Mechanism of Action - Methylxanthines block adenosine receptors. Adenosine inhibits the respiratory drive, thus by blocking inhibition, the methylxanthines stimulate respiratory neurons resulting in an enhancement of minute ventilation.
    2. Dosages - The following is a guide to the initiation of medical therapy. Further dosing should be based on drug levels and clinical response.
  - **Caffeine Citrate** - 20mg/ml containing the equivalent of 10 mg/ml of caffeine is available for either IV/PO use.
    - a. Loading Dose - 20 mg/kg/dose of caffeine citrate IV/PO.
    - b. Maintenance Dose - 5 mg/kg/day of caffeine citrate given once daily.
    - c. Plasma Half Life - 37-231 hrs.
    - d. Therapeutic Level - 8-20 ug/ml.
    - e. Toxic Level - >30 ug/ml.
  - **Theophylline:**
    - a. Loading Dose - 6 mg/kg/dose IV/PO.
    - b. Maintenance Dose - 6 mg/kg/day divided Q6H/Q8H/Q12H IV/PO.
    - c. Plasma Half Life - 12-64 hrs.
    - d. Therapeutic Level - 6-12 ug/ml.
    - e. Toxic Level - >20 ug/ml.
    - f. Administration - ALWAYS INFUSE SLOWLY over a minimum of 20 minutes. Rapid IV pushes have been associated with SUDDEN DEATH from CARDIAC ARRHYTHMIAS.
  3. Major side effects - tachycardia, vomiting, feeding intolerance, jitteriness and seizures.
  4. Choice of Methylxanthines - This decision depends on the clinical situation and should take into account the following factors. Caffeine has a longer half life (once daily) and is less toxic. At UIHC, caffeine is preferred for the routine management of apnoea of prematurity. Theophylline is a bronchodilator and in neonates with BPD it offers the advantage of treating both apnoea and bronchospasm.
- ii. **Continuous Positive Airway Pressure (CPAP)** - CPAP is effective in treating both

obstructive and mixed apnoea, but not central apnoea. CPAP is most commonly delivered by nasal prongs or by an endotracheal tube placed in the nasopharynx (see also separate section on CPAP).

1. Mechanism of Action - Proposed mechanisms include alteration of the Hering-Breuer reflex (leading to higher lung volumes which minimize inspiratory duration and thus decrease the potential for airway collapse by prolonging expiratory time). Furthermore, CPAP increases stabilization of the chest wall musculature and decreases activity of the intercostal inspiratory inhibitory reflex. However, the most likely explanation is that CPAP splints the upper airway with positive pressure during both inspiration and expiration, thereby preventing pharyngeal collapse.
  2. Initial Settings - Use either nasal prongs or a nasopharyngeal tube to deliver a CPAP of 5 cm H<sub>2</sub>O. Further adjustments should be based on clinical response.
  3. Side Effects - Barotrauma, nasal irritation, abdominal distention and feeding intolerance. Feeding difficulties can be minimized by switching the patient to continuous drip feeds.
- iii. **Intermittent Mandatory Ventilation (IMV)** - If significant apnoea persists despite using both pharmacotherapy and CPAP, the infant should be intubated and ventilated. Initial settings need to be clinically adjusted to prevent episodes of desaturation or cyanosis. In order to minimize barotrauma short inspiratory times should be used along with minimal peak inspiratory and expiratory pressures. The infant may need to remain on a minimal rate for a few weeks while the respiratory control system matures.

## **CONCLUSION:**

Apnoea of prematurity is one of the most common and frustrating conditions that nurses, physicians and neonates face in the intensive care unit. A calm, rational team approach to this problem is beneficial for all involved.

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## **Neonatal jaundice**

- Jaundice is an extremely common problem occurring during the newborn period.
- The etiology of the jaundice is quite varied; although most causes are benign.
- Criteria of non-physiologic jaundice are:

- Visible jaundice on the first day of life,
- A total serum bilirubin level increasing by more than 5 mg/dl per day,
- A direct serum bilirubin level exceeding 1.5 mg/dl, and
- Clinical jaundice persisting for more than 2 weeks in term babies and 3 weeks in a preterm,

- Following the identification of an icteric infant;

- The maternal and preceding neonatal histories are reviewed.
  - Complete physical examination.
  - The following is the minimal work up necessary in each infant:
    - Serum bilirubin level (both direct and indirect),
    - CBC with smear,
    - Infant's blood type and Coombs' tests;
    - Mother blood group and Coombs' tests,
  - A urinalysis, culture and urine testing for reducing substances should be done only if sepsis, urinary tract infection, or galactosemia is suspected.
- Infants with ABO incompatibility may have extremely rapid increases in their serum bilirubin values, so their bilirubin levels may need to be done more frequent.

- Guide to dermal staining with level of bilirubin

Area of body	Level of bilirubin
Face	4-6 mg/dl
Chest, upper abdomen	8-10 mg/dl
Lower abdomen, thighs	12-14 mg/dl
Arms, lower legs	15-18 mg/dl
Palms, soles	15-20 mg/dl

**Suggested guidelines for frequency of monitoring serum bilirubin in healthy term infants are as follows:**

Serum Bilirubin mg/dl <i>[If Direct Bilirubin &lt; 1.5 mg/dl, use the total level]</i>	Days of Age		
	1	2	3
5-10	repeat in 3-5 hr	repeat x 1 in 8-12 hr	Repeat x1 in 24 hr
10-15	repeat in 3-4 hr;	Repeat in 4-6 hr	Repeat in 6-8 hr
15-20	repeat in 2-3 hr	repeat in 2-4 hr;	repeat in 4-6 hr
>20	discuss exchange transfusion with senior staff	repeat in 2-3 hr;	repeat in 3-4 hr;

Shaded area = consider institution of phototherapy

### **Management of Hyperbilirubinemia in the Healthy Term Newborn**

Total Serum Bilirubin (TSB) Level, mg/dL ( $\mu$ mol/L)

<b>Age, hours</b>	<b>Phototherapy</b>	<b>Exchange Transfusion if Intensive Phototherapy Fails †</b>	<b>Exchange Transfusion and Intensive Phototherapy</b>
$\leq 24$ ‡	<b><i>Pathological and requires further evaluation</i></b>		
25-48	$\geq 15$ (260)	$\geq 20$ (340)	$\geq 25$ (430)
49-72	$\geq 18$ (310)	$\geq 25$ (430)	$\geq 30$ (510)
>72	$\geq 20$ (340)	$\geq 25$ (430)	$\geq 30$ (510)

**†** Intensive phototherapy should produce a decline of TSB of 1-2 mg/dL within 4-6 hours and the TSB level should continue to fall and remain below the threshold for exchange transfusion. If this does not occur, it is considered a failure of phototherapy.

## **Phototherapy**

- Infant receiving phototherapy should be left unclothed except for eye protection.
- The insensible water loss is increased with the use of overhead phototherapy, so monitor the weight, fluid intake and urine output on daily basis.
- 10-20% increase in fluids can be considered if the baby is dehydrated.
- The phototherapy unit should be placed 40 cm above the infant and have a Plexiglas shield between the light bulbs and the infant.
- Phototherapy should be given continuously although the baby may come out for breastfeeds if the bilirubin levels are not very high.
- Phototherapy has its greatest effect in the first 24-48 hours of treatment. If bilirubin levels have not dropped by 25-50% by then, think about compliance, haemolysis, sepsis or conjugated hyperbilirubinaemia.
- Babies must have 2 bilirubin measurements 'below the line' **prior to stopping phototherapy** and 2 'rebound' readings still below the line **prior to sending home**.

## Exchange transfusion

### **Indications for exchange transfusion:**

- When phototherapy fails to prevent the rise in bilirubin to a toxic level .
- To correct anaemia & improve heart failure in hydropic infant .
- Stop haemolysis & bilirubin production by removing antibodies.
- In haemolytic disease , immediate exchange is indicated in:
  - The cord bilirubin level is over 5 mg/dL & the cord haemoglobin level is under 10 gm/dl .
  - The bilirubin level is rising over 1 mg/dL per hour despite phototherapy.
  - The haemoglobin level is between 11 & 13 gm/dL & the bilirubin level is rising over 0.5 mg/dL per hour despite phototherapy.
  - The bilirubin level is 20 mg/dL, or it appears that it will reach 20 mg/dL at the rate it is rising .
  - There is progression of anemia in the face of adequate control of bilirubin by other methods (e.g., phototherapy) .
- **Subsequent exchange transfusions are indicated if:**
  - Bilirubin >10 mg/dl within 24 hours of age.
  - Bilirubin>15 mg/dl between 25-48 hours of age.
  - Bilirubin >20 mg/dl after 48 hours of age.
  - Rate of rise of bilirubin is >0.5 mg/dl/hr.

### **Blood for exchange transfusion:**

Red Blood Cells for Exchange Transfusion.

This red cell product has the following specifications:

- Group O
  - CMV Negative.
  - If available, Fresh (< 5 days).
  - Known haematocrit (<0.6).
  - RhD negative.
  - Kell negative.

Commence transfusion within 30 minutes of product receipt and complete transfusion within 4 hours of spiking pack.

- In nonimmune hyperbilirubinemia, the blood is typed and cross-matched against the plasma & red cells of the infant.
- Exchange transfusion usually involves double the volume of the infant's blood & is known as a two-volume exchange (160 mL/kg).

### **Techniques of exchange transfusion:**

- Exchange is done with the infant under a radiant warmer.
- Vital signs recorded.
- Equipment & personnel for resuscitation must be readily available.
- IV line should be in place for the administration of glucose & medication.
- The infant's legs should be properly restrained.
- An assistant should be assigned to the infant to record volumes of blood, observe the infant, & check vital signs.
- The blood should be warmed to 37°C.
- Sterile techniques should be used.
- Old, dried umbilical cords can be softened with saline-soaked gauze.

- If a dirty cord was entered or there was a break in sterile technique, treat with cloxacillin & gentamycin for 2-3 days.
- Do most exchanges by the push-pull technique:
  - Two Catheter Push-pull Technique.

Blood is removed from the artery while infusing fresh blood through a vein at the same rate.

	In	Out
	Umbilical vein	Peripheral artery
or	Umbilical vein	Umbilical artery <sup>2</sup>
or	Peripheral vein	Peripheral artery <sup>1</sup>
or	Peripheral vein	Umbilical artery

#### ➤ One Catheter Push-pull Technique

1. This can be done through an umbilical venous catheter. <sup>3</sup> Exceptionally, an umbilical artery catheter can be used.
  2. Ideally, the tip of the UVC should be in the IVC/right atrium (at or just above the diaphragm) but can be used if it is in the portal sinus. For 'high' UVC placement, position should be checked by an X-ray. This is not always necessary for a low position. A low positioned catheter is usually removed after each exchange.
  3. Withdraw blood over 2 minutes, infuse slightly faster.
- If it is not possible to insert a catheter in the umbilical vein, exchange transfusion can be accomplished through a central venous line placed through the antecubital fossa or into the femoral vein via the saphenous vein.
  - Volume: Usually use two blood volumes (180 ml/kg).
    1. One blood volume removes 65% of baby's red cells.
    2. Two blood volume removes 88%.
    3. Thereafter the gain is small. <sup>3</sup>

<1500 gms	Use 5ml aliquots
1500-2500 gm	10ml
2500-3500 gm <sup>3</sup>	15ml
>3500 gm	20 ml

- The recommended time for the exchange transfusion is 1 hour.
- After exchange transfusion, phototherapy is continued & bilirubin levels are measured every 4 hrs.

### **Conjugated hyperbilirubinaemia:**

This becomes significant if the direct bilirubin is >25 micromol/L in the first few days of life or  $\geq$  20% of the total bilirubin.

#### **First Line investigations:**

- |   |  |
|---|--|
| <ul style="list-style-type: none"> <li>Pre-feed blood glucose</li> <li>Hb, Reticulocytes count</li> <li>ALT, AST, Gamma GT, ALP, Albumin, Total bilirubin, Direct bilirubin, INR</li> <li>Urea, Na+, K+, Creatinine, Ca++, PO4</li> </ul> | <ul style="list-style-type: none"> <li>One EDTA saved</li> <li>One clotted saved</li> <li>Blood culture, Urine culture</li> <li>Urine for reducing substances</li> <li>Group and save</li> </ul> |
|---|--|

#### **Second Line investigations:**

- |   |   |
|---|---|
| <ul style="list-style-type: none"> <li>Liver ultrasound</li> <li>Examine stools for pigmentation</li> <li>Chromosomes for karyotype</li> <li>Cortisol</li> <li>GAL-1- PUT (galactosaemia)</li> <li>Alpha-1 antitrypsin phenotype</li> <li>TSH, T4</li> <li>Sweat test</li> <li>Cystic fibrosis genotype</li> <li>Cholesterol, triglycerides (abnormal in Alagille's)</li> </ul> | <ul style="list-style-type: none"> <li>Urinary succinyl acetone (tyrosinaemia)</li> <li>Serum amino acids</li> <li>Urinary organic acids</li> <li>Hep A IgM, Hep B surface antigen, Hep C antibody</li> <li>EBV, Parvovirus, Throat swab and stool for adenovirus</li> <li>Urine for CMV</li> <li>Herpes, Toxoplasmosis, Rubella, Syphilis</li> <li>Liver biopsy</li> </ul> |
|---|---|

#### **Third Line investigations:**

- |  |  |
|--|--|
| <ul style="list-style-type: none"> <li>Lactate</li> <li>Pyruvate</li> <li>Ammonia (urea cycle defects)</li> <li>White cell enzymes</li> <li>Very long chain fatty acids (peroxisomal disorders)</li> <li>Alpha-fetoprotein</li> <li>Mitochondrial deletions</li> </ul> | <ul style="list-style-type: none"> <li>Acyl carnitine</li> <li>Free carnitine</li> <li>Total bile acids</li> <li>X-ray spine</li> <li>Posterior embryotoxon (Alagille's)</li> <li>MRI head</li> <li>Muscle biopsy (mitochondrial and respiratory chain disorders)</li> </ul> |
|--|--|

### **Prolonged jaundice:**

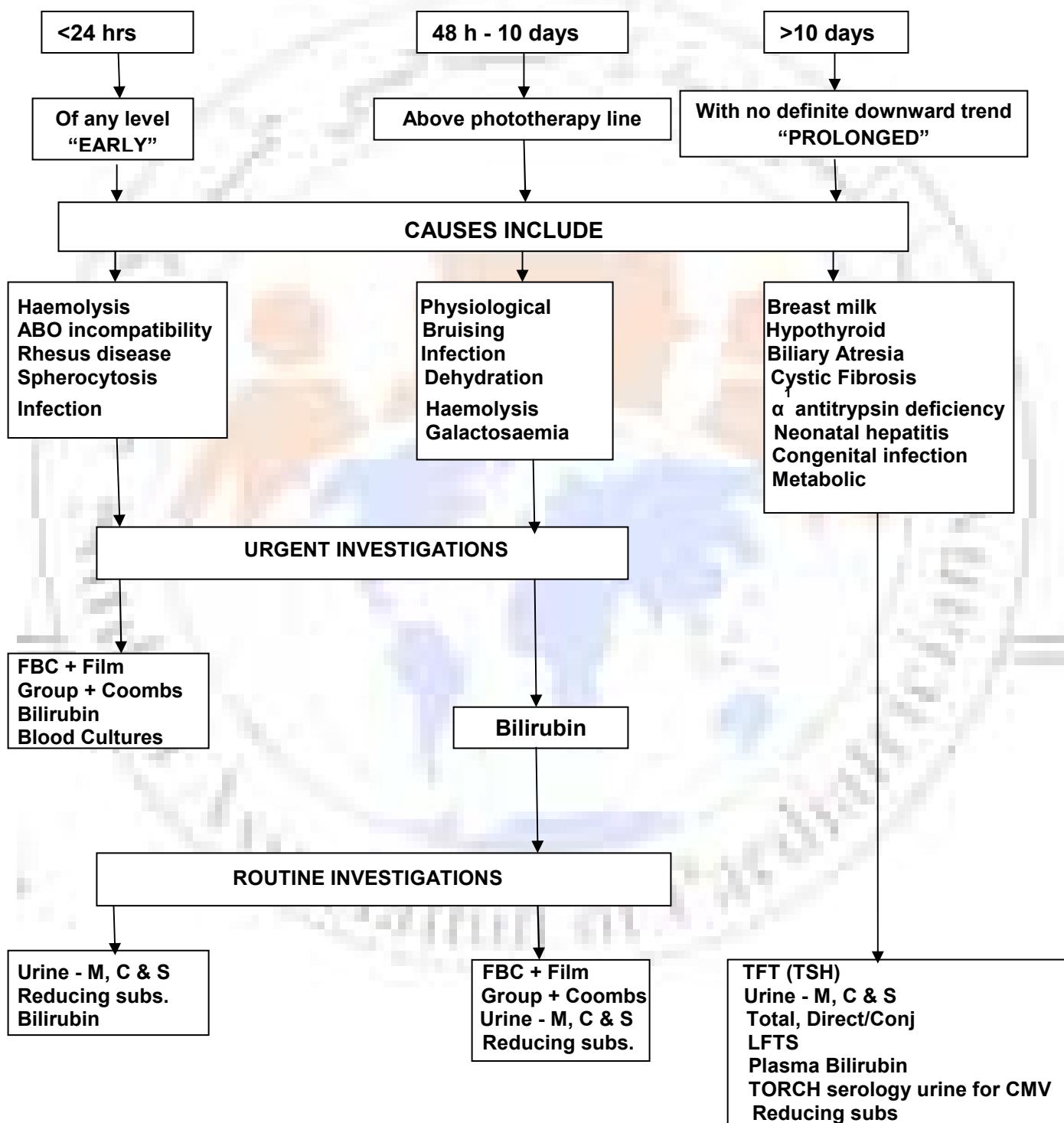
Definition: Significant Jaundice at 2 weeks of age in full term babies, and at 3 weeks for preterm babies. The following screen should be performed:

- Serum Bilirubin total and direct (if the direct bilirubin  $\geq$  20% of the total bilirubin then please see [conjugated hyperbilirubinaemia](#))
- TSH, T4,
- FBC, reticulocytes, Blood film
- Group, DCT
- Urine routine, culture and reducing substances
- G6PD

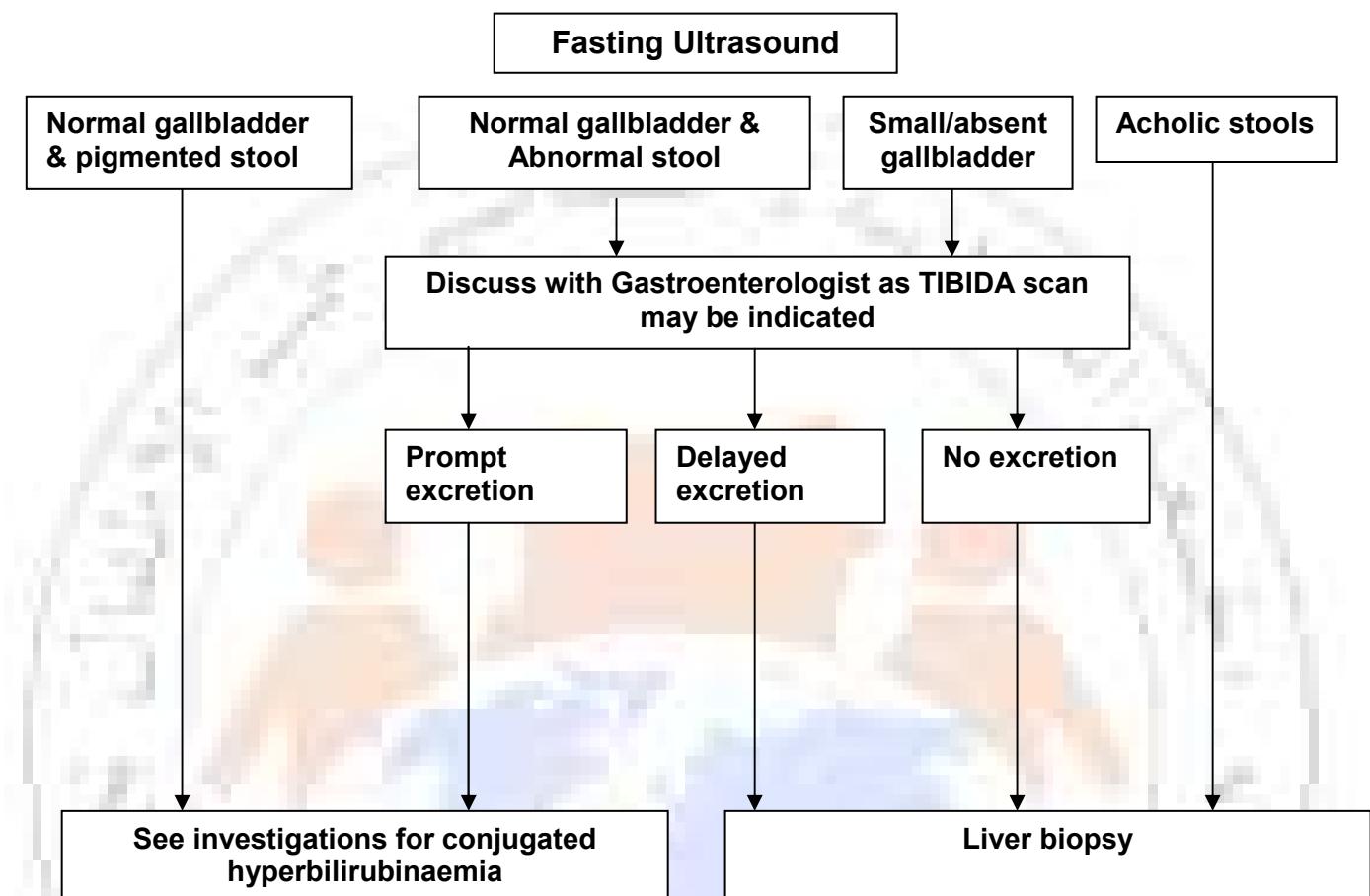
**Attachment:**

Bilirubin Charts

## Flow Chart for Clinically Jaundiced, Well, Term Baby



## Conjugated hyperbilirubinaemia



## **REFERENCES:**

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12. *Hyperbilirubinemia. In: Guidelines for Perinatal Care, Frigoletto FD and Little GA (eds). 1992, American Academy of Pediatrics, Elk Grove, IL, pp 208-210.*

# **Neonatal seizures**

## **Definition:**

Paroxysmal alteration in neurologic function (i.e. behavioural, motor , autonomic function).

## **Background:**

It is important to recognize the presence of seizures in the neonatal period since they are often related to significant underlying illness. In addition, seizures may be sustained for considerable periods of time , interfering with essential supportive care.

## **Causes of neonatal seizures:**

### **1. Perinatal asphyxia :**

There is usually an interval of time between the event and the onset of seizures, but this interval is quite available (1-36 hr).

### **2. Intracranial haemorrhage :**

- Subarachnoid haemorrhage .
- Periventricular or intraventricular haemorrhage.
- Subdural haemorrhage.

### **3. Metabolic disturbances :**

- Hypoglycaemia.
- Hypocalcaemia.
- Hyponatremia.
- Hypernatremia.
- Pyridoxine dependency.
- Amino acid disorders.
- Disorders of subcellular organelles.

### **4. infection:**

- bacterial infection .

- non bacterial infection.
5. **drug withdrawal** : heroin methadone.
  6. **structural defects of the central nervous system.**

**Types of seizures:** there are 4 major types of seizures in neonates:

**1. subtle seizures :**

- More common in preterm than full term infants.
- More commonly associated with EEG activities in preterm than term infants.
- Such seizures include oral- buccal – lingual movements (smacking), certain ocular phenomena, peculiar limb movements (cycling), autonomic alteration and apnoea.

**Manifestations:**

- Tonic horizontal deviation of the eyes, without jerking.
- Eyelid blinking or fluttering.
- Sucking, smacking or drooling.
- Swimming, rowing or pedaling movements.
- Apnoeic spells (convulsive apnoea).

**2. Clonic seizures:**

- More common in full term.
- Commonly associated with EEG activities.

**Types:**

- **Focal:**
  - Well localized, rhythmic, slow, jerking movements.
  - Involving the face, upper and lower extremities, neck or trunk on one side of the body.
  - Usually not unconscious during or after the seizures.
- **Multifocal :**
  - Several body parts seize in a sequential non jacksonian e.g left arm jerking followed by right leg jerking.

**3. Tonic : Mainly in premature infants.**

**Types:**

- **Focal:** Commonly associated with EEG activities.
  - Sustained posturing of limb.
  - Asymmetrical posturing of the trunk or neck or both.
- **Generalized:**
  - EEG changes are uncommon.

- Decerbrate posturing: tonic extension of both upper and lower extremities.
- Decorticate posturing: tonic flexion of the upper extremities with extension of the lower extremities.

#### **4. Myoclonic seizures:**

- Occur in both premature and full term infants.
- Characterized by single or multiple synchronous jerks.

##### **Types:**

- **Focal**
  - Involve flexor muscles of the upper limbs.
  - Not commonly associated with EEG changes.
- **Multifocal:**
  - Asynchronous twitching of several parts of the body.
  - Not commonly associated with EEG seizure activities.
- **Generalized :**
  - Bilateral jerks of flexors of the upper and sometimes the lower extremities.
  - Commonly associated with EEG seizure activities.

DD: jitteriness: myoclonic seizures must be differentiated from jitteriness:

- Jitteriness is not associated with abnormal eye movements.
- Movements in jitteriness cease on application of passive flexion.
- Movements in jitteriness are stimulus sensitive.
- Movements are not jerky.

##### **Diagnosis:**

- **History:**
  - i. **Family history** of previous neonatal seizures.
  - ii. **Maternal history:**
    - Diabetes.
    - Hyperparathyroidism.
    - Drugs during pregnancy.
    - Infection during pregnancy.
  - iii. **Delivery:**
    - Maternal analgesic.
    - Mode and nature of delivery.
    - Fetal intrapartum status.
    - Resuscitative measures used.

### **Physical examination:**

i. **Thorough general physical examination including:**

- Gestational age.
- Blood presence of skin lesions.
- Presence of hepatosplenomegaly.

ii. **Neurologic evaluation :**

- Level of alertness.
- Cranial nerves.
- Motor function.
- Primary neonatal reflexes.
- Sensory function.
- Anterior fontanel: size and 'feel'.
- Tone.
- Eyes: retinal haemorrhage , chorioretinitis , pupillary size , reaction to light , extra ocular movements and cataract.

iii. **Notation of seizures:**

- Site of onset.
- Spread.
- Nature.
- Duration.
- Level of consciousness.

### **Laboratory studies:**

Guided by the information obtained from the history and examination.

1. Serum chemistry:glucose , calcium , sodium , urea , magnesium , phosphate and blood gases.
2. Full septic screen , including L.P (CSF examination includes checking for xanthochromia , lactic & pyruvate-for evidence of mitochondria cytopathies-,PCR,glucose concentration - persistently low in the absence of bacterial meningitis may suggest a glucose transport defect).
3. TORCH screen.
4. Metabolic screen :in the presence of F.H of neonatal convulsion , peculiar odor about the infant , milk intolerance , acidosis , alkalosis, or seizure not responding to anticonvulsant:
  - Blood ammonia level.
  - Urine for reducing substances.
  - Urine and plasma amino acids.

- Urine for 2,4- dinitrophenylhydrazine (2,4-DNPH);fluffy yellow precipitate will be seen in cases of maple syrup urine disease.

#### **Radiologic studies:-**

1. Cranial ultrasound.
2. CT scan of the head.
3. MRI.

#### **EEG:-**

#### **Management:**

##### **1. Hypoglycaemia:**

See hypoglycaemia protocol.

##### **2. Hypocalcaemia:**

See Hypocalcaemia protocol.

##### **3. Hypomagnesaemia or refractory hypocalcaemia:**

See hypomagnesaemia protocol.

##### **4. Anticonvulsants:**

[If facilities available, drug levels should be monitored]

**Phenobarbitone:** 77% of cases are controlled by phenobarbitone.

##### **Dose:**

- Loading dose: 20-30 mg/kg IV or IM over 15-30 min.
- Maintenance doses: 2.5 - 4 mg/kg /day od or in 2 divided doses .

[For neonates <30 weeks 1-3 mg/kg/day]

**phenytoin:** fosphenyton is preferred –available only as IV/IM.

##### **Dose:**

- Intravenous:

Loading dose: 15-20 mg/kg/at a rate not>0.5 mg/kg/min.

Maintenance dose: 5-8mg/kg/day divided q 12-24h.

- Po :highly variable :-5-8 mg/kg/day to 8 mg/kg q 12h

[NB: 75 mg fosphenyton is equivalent to 50 mg.

phenytoin. The dose of fosphenyton is expressed as

phenytoin equivalent (PE) e. g the loading dose from the fosphenytoin is 15-20 mg PE1.

### lorazepam (ativan):

- Initial dose: 0.05 mg/kg/dose. If no response after 15 minutes repeat the dose.
- Dilute with equal volume of sterile water, normal saline, D10 and infuse over 2-3 min.

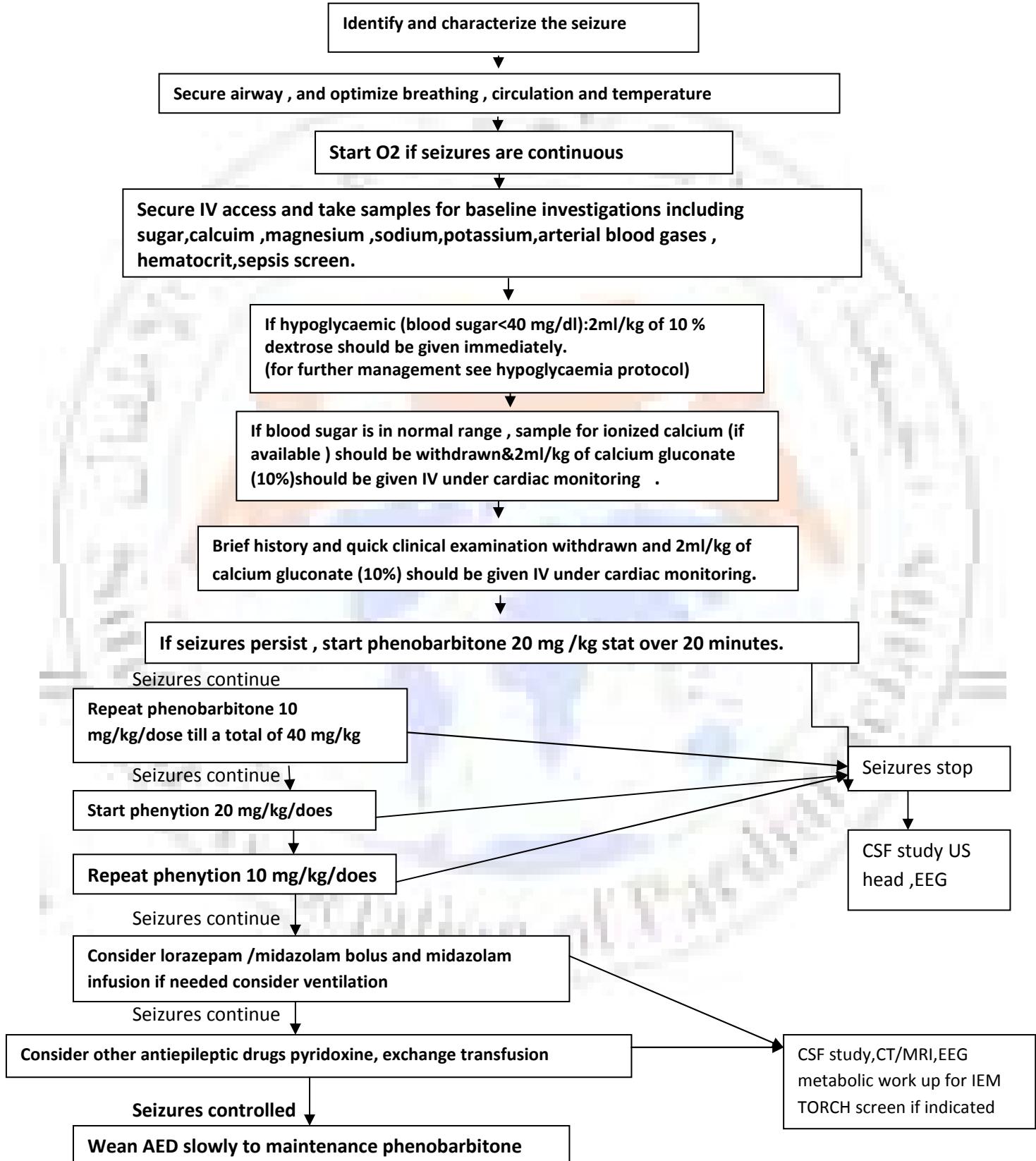
### midazolam:

- **Dose;** 0.05-0.2 mg/kg/dose IV 2-4 hourly PRN.
- Continuous infusion:
  - Loading dose; 0.2mg/kg.
  - Maintenance: 0.4 – 0.6 mg/kg/min (max 6 mg /kg/min).

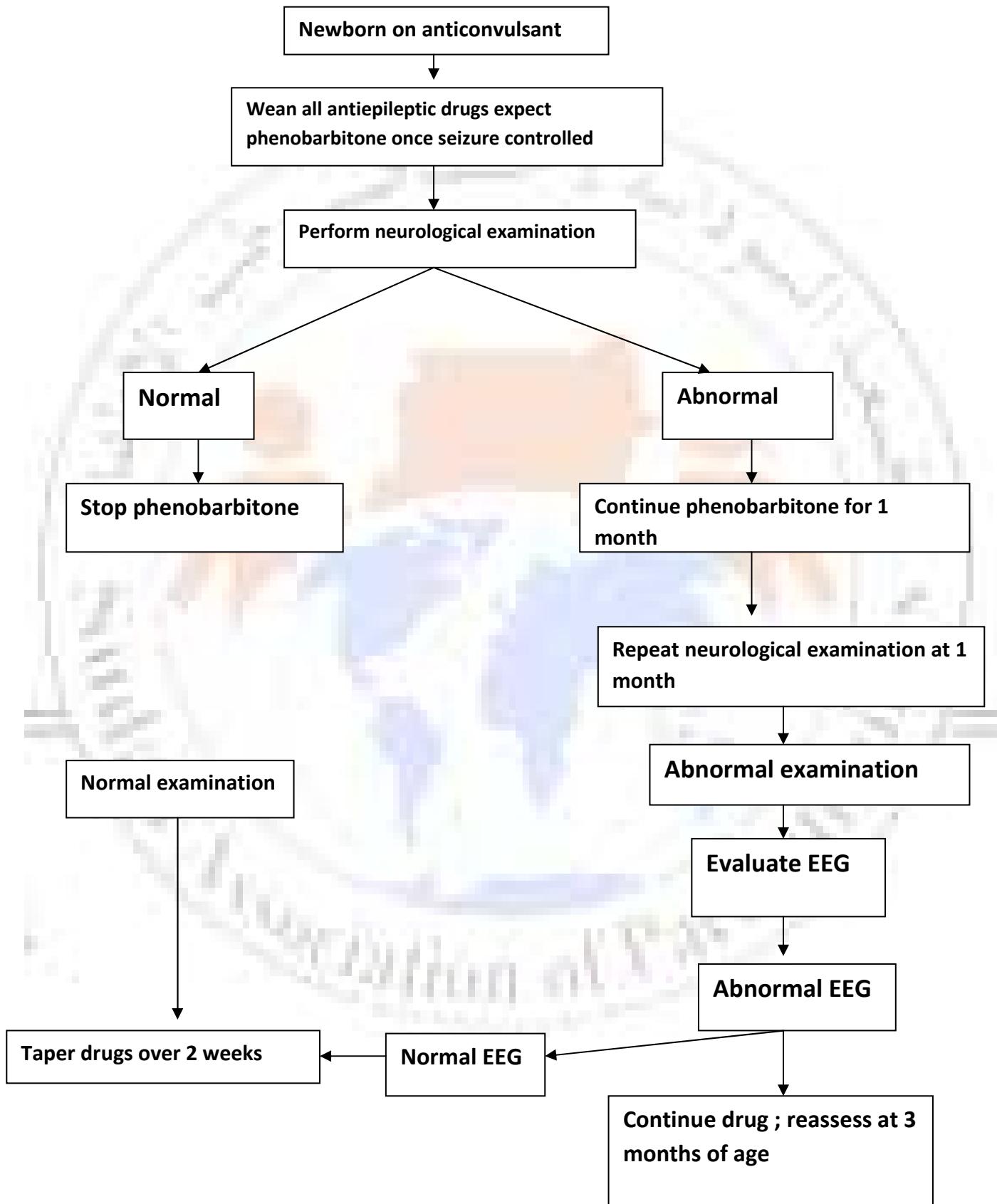
### pyridoxine:

- **Dose:** 100-200 mg IV under EEG control. The seizure will abruptly stop and the EEG will normalize within few hours.
- For rectal use dilute in an equal volume of olive or mineral oil. For oral use ,dilute in infant formula
- Maintenance; 50 – 100 mg PO daily.
- If a trial of withdrawal at the age of 6 months fails, treatment should be continued for life.

## Flow diagram on management of neonatal seizure



## Flow diagram on weaning and duration of anticonvulsant therapy



# Cyanosis at birth

(BLUISH TINGE OF THE SKIN AND MUCOUS MEMBRANES)

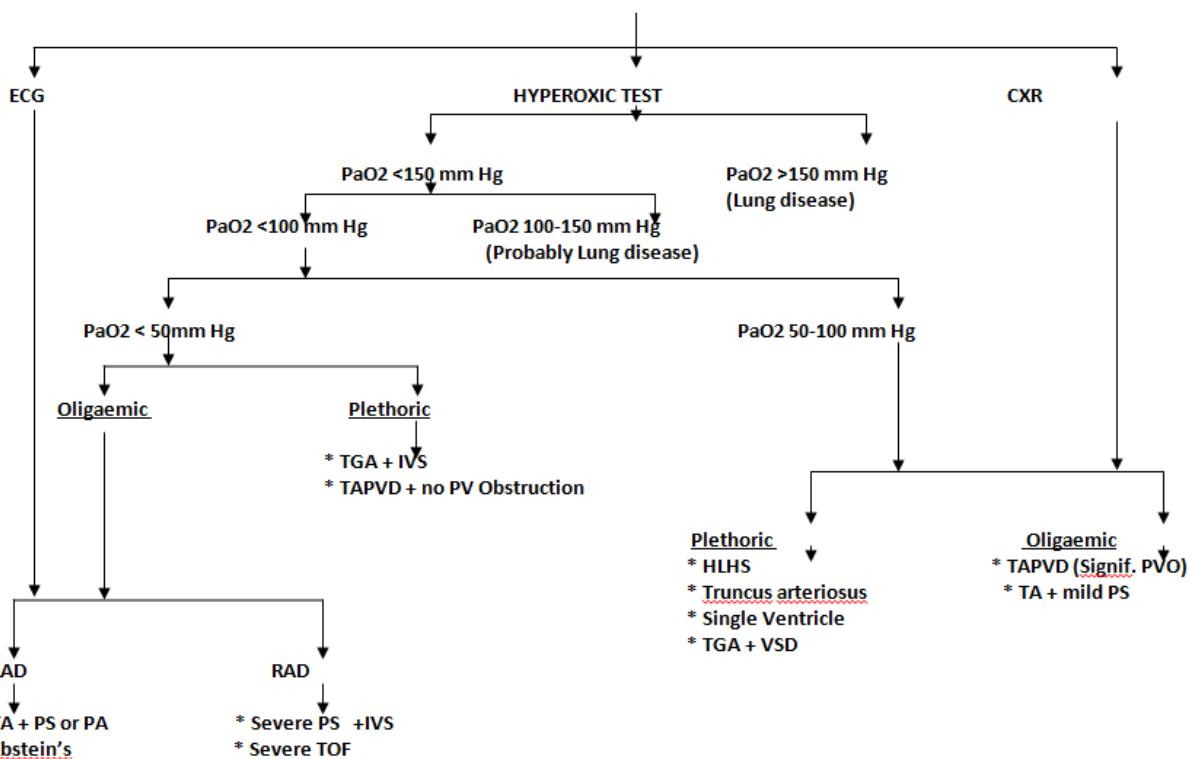
## CAUSES OF CYANOSIS:

<u>RESPIRATORY</u>	<u>CARDIAC DISEASES</u>	<u>CNS</u>	<u>OTHERS</u>
<ul style="list-style-type: none"> <li>a) <u>LUNG DISEASES:</u> <ul style="list-style-type: none"> <li>• HMD (hyaline membrane disease)</li> </ul> </li> <li>• TTN (transient tachypnoea of the newborn)           <ul style="list-style-type: none"> <li>• Meconium Aspiration               <ul style="list-style-type: none"> <li>• Pneumonia</li> </ul> </li> </ul> </li> <li>b) <u>AIR LEAK SYNDROME</u></li> <li>c) <u>Airway obstruction:</u> <ul style="list-style-type: none"> <li>• Choanal atresia or Stenosis</li> <li>• Pierre Robin syndrome</li> </ul> </li> <li>d) <u>Congenital resp. defect:-</u> <ul style="list-style-type: none"> <li>• Diaphragmatic hernia</li> <li>• Hypoplastic lungs</li> <li>• Lobar emphysema</li> </ul> </li> <li>• Intrapulmonary AV malformation</li> </ul>	<ul style="list-style-type: none"> <li>a) <u>Congenital Heart Diseases:</u> <ul style="list-style-type: none"> <li>• TGA</li> <li>• TAPVD</li> <li>• TOF</li> <li>• tricuspid atresia</li> <li>• Pulmonary atresia</li> <li>• Hypoplastic left heart</li> </ul> </li> <li>b) <u>PPHN</u></li> <li>c) <u>Severe CHF</u></li> </ul>	<ul style="list-style-type: none"> <li>• Birth asphyxia</li> <li>• IVH</li> <li>• Neonatal convulsion           <ul style="list-style-type: none"> <li>• Meningitis</li> <li>• Neuromuscular disorders</li> </ul> </li> <li>• <i>Werdnig-Hoffman Disease</i></li> <li>• <i>Congenital myotonic Dystrophy</i></li> </ul>	<ul style="list-style-type: none"> <li>• Methaemoglobinemia</li> <li>• Polycythaemia</li> <li>• Sepsis</li> <li>• Shock</li> </ul>

## DIAGNOSIS:

<u>HISTORY</u>	<u>CLINICAL EXAMINATION</u>	<u>LABORATORY STUDIES</u>
<ul style="list-style-type: none"> <li>a) <u>Prenatal History:</u> <ul style="list-style-type: none"> <li>• Maternal DM: IDM                   <ul style="list-style-type: none"> <li>• Infection;</li> </ul> </li> <li>➢ TORCH: CHD, CNS</li> <li>➢ PROM: Sepsis</li> </ul> </li> <li>• Amniotic Fluid Abnormalities;       <ul style="list-style-type: none"> <li>➢ Oligohyd; Hypoplastic lung</li> <li>➢ Polyhydramnious; TOF</li> </ul> </li> <li>b) <u>Perinatal History cont.:</u> <ul style="list-style-type: none"> <li>• Caesarean section; TTN</li> <li>• Drug; during delivery// abuse</li> </ul> </li> <li>c) <u>Timing of cyanosis</u></li> <li>d) <u>Increased respiratory effort</u></li> <li>e) <u>Is cyanosis:</u> <ul style="list-style-type: none"> <li>• Continuous: lung &amp; heart</li> <li>• Intermittent: CNS</li> <li>• Sudden onset: Air Leak</li> <li>• During feeding: TOF</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li>• Dysmorphism</li> <li>• Date / Size discrepancy       <ul style="list-style-type: none"> <li>• Hyperinflation</li> </ul> </li> <li>• Tachypnoea / Recession / AE       <ul style="list-style-type: none"> <li>• HR /Cardiac murmur</li> <li>• Peripheral pulses</li> <li>• Hepatosplenomegaly</li> <li>• Scaphoid abdomen</li> </ul> </li> <li>• SZ / Apnoeic spells/ OFC / AF       <ul style="list-style-type: none"> <li>• Hypotonia / paucity of movements</li> <li>• Skin rash/ perfusion</li> <li>• Transillumination</li> </ul> </li> </ul>	<ul style="list-style-type: none"> <li>• Oxygen saturation (So2)       <ul style="list-style-type: none"> <li>• ABG</li> <li>• Chest X-ray</li> <li>• ECG</li> </ul> </li> <li>• Hyperoxic test</li> <li>• CBC, Band count       <ul style="list-style-type: none"> <li>• CRP</li> </ul> </li> <li>• Partial / full septic screen</li> <li>• DX / RBS, U&amp;Es, Ca++, Mg ++       <ul style="list-style-type: none"> <li>• TORCH screen</li> <li>• Cranial U/S</li> <li>• Echocardiogram</li> <li>• Methaemoglobin (spectrophotometric)</li> </ul> </li> </ul>

## Cyanotic infant



### Ref:

- Neonatology, Management, Procedures, On-call Problems, Diseases and Drugs, Tricy Lacy Gomella, 6<sup>th</sup> Edition
- Current Diagnosis and Treatment in Pediatrics, 18<sup>th</sup> Edition

# **Management of respiratory distress syndrome ( RDS )**

## **Aetiology:**

- Prematurity
- Perinatal asphyxia
- Caesarean section
- Maternal diabetes

## **Clinical Signs:**

RDS presents within four hours of birth:

- Sternal retraction, intercostal & sub costal recessions.
- An expiratory grunt.
- Tachypnoea above 60/min.

So common to all definitions of the disease is that the signs should present before four hours of age, should still be there at four hours of age and should persist for some period beyond four hours of age.

## **Diagnosis:**

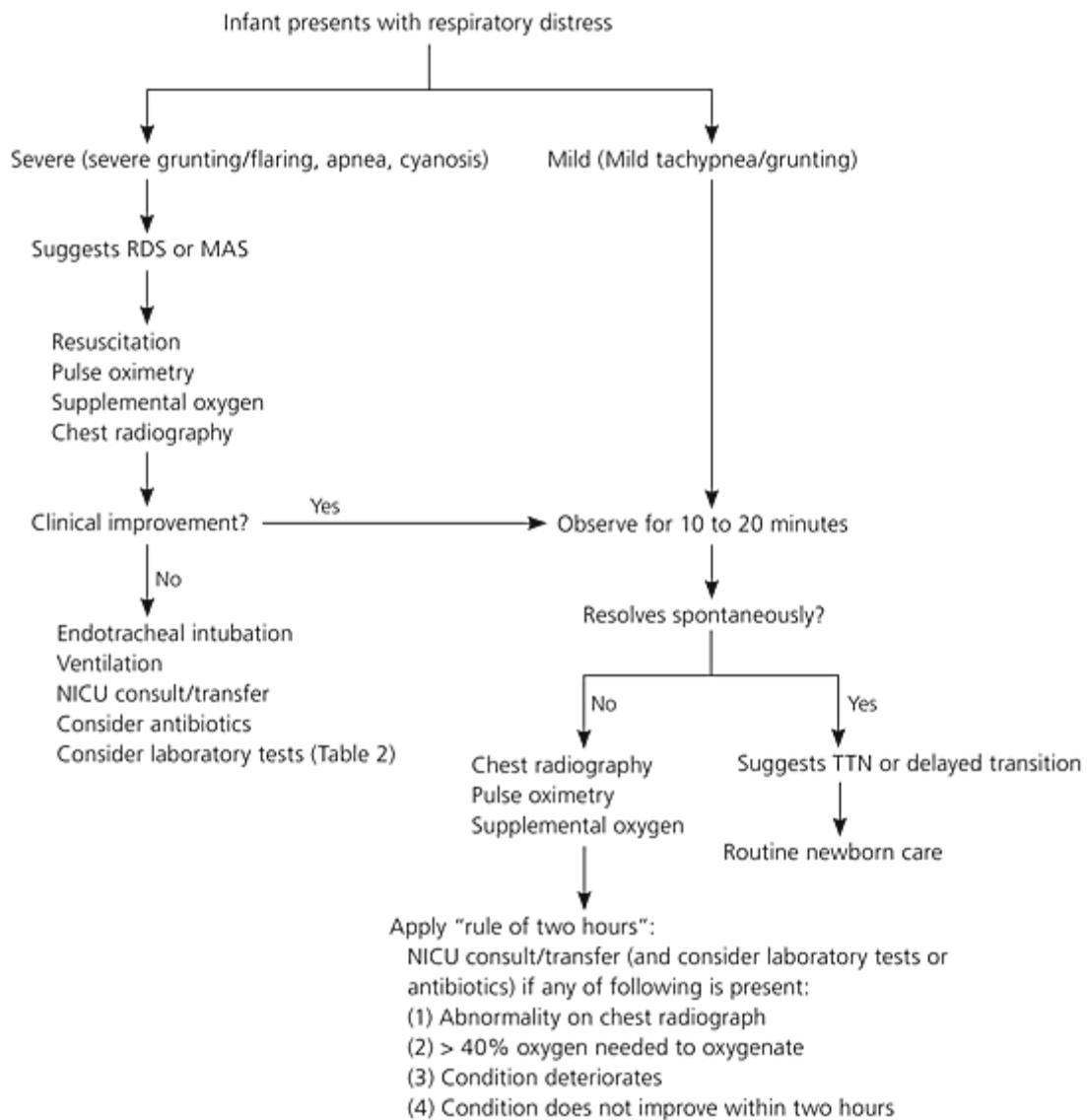
1. History
2. Clinical signs
3. Investigations:
  - a. Radiology:
    - Atelectasis: documented by the ground-glass pattern.
    - Air bronchogram
    - In severe causes the lungs cannot be separated from the cardiac border.
  - b. Hb%, packed cell volume (PCV), WBC & PLATELETS COUNT.
  - c. Electrolytes, Creatinine, calcium (establishing base line).
  - d. Blood gases (initiating treatment).
  - e. Blood group and cross-match.
  - f. Serum albumin.

## **Treatment:**

The aim of treatment is to keep the newborn alive and in good condition until he starts to synthesize his own surfactant 36-48 hours after birth. This means avoiding:

- Hypoxia.
- Acidemia.
- Hypothermia, which inhibits surfactant synthesis

## **Management of Neonatal Respiratory Distress**



## Distinguishing Features of TTN, RDS, and MAS

<b>Cause</b>	<b>Etiology</b>	<b>Timing of delivery</b>	<b>Risk factors</b>	<b>Clinical features</b>	<b>Chest radiography findings</b>	<b>Treatment</b>	<b>Prevention</b>
<b>TTN</b>	Persistent lung fluid	Any	Cesarean delivery <sup>4</sup>  Macrosomia Male sex Maternal asthma <sup>2</sup> Maternal diabetes <sup>3</sup>	Tachypnoea  Often no hypoxia or cyanosis	Parenchymal infiltrates <sup>5*</sup>  Wet silhouette* around the heart <sup>5</sup>  Intralobar fluid accumulation	Supportive, oxygen if hypoxic	Prenatal corticosteroids before cesarean delivery if 37 to 39 weeks' estimated gestation (not accepted U.S. practice) <sup>19</sup>
<b>RDS</b>	Surfactant deficiency Lung under-development	Preterm	Male sex <sup>7</sup>  Maternal diabetes <sup>8</sup> Preterm delivery <sup>6</sup>	Tachypnoea Hypoxia  Cyanosis	Homogenous infiltrates <sup>5</sup> Air bronchograms <sup>5</sup> Decreased lung volumes	Resuscitation, oxygen, ventilation, surfactant	Prenatal corticosteroids if risk of preterm delivery (24 to 34 weeks' estimated gestation) <sup>20</sup> (accepted U.S. practice)
<b>MAS</b>	Lung irritation and obstruction	Term or post-term	Meconium-stained amniotic fluid  Post-term delivery	Tachypnoea Hypoxia	Patchy atelectasis <sup>5</sup>  Consolidation <sup>5</sup>	Resuscitation, oxygen, ventilation, surfactant	Do not impede delivery for suctioning <sup>23</sup> ; amnioinfusion of no benefit <sup>27</sup>

TTN = transient tachypnea of the newborn; RDS = respiratory distress syndrome; MAS = meconium aspiration syndrome.

### Practical points:

- 1- Monitoring of Temperature, Pulse & Respiratory rate.
- 2- Control baby body temperature: incubator, heat radiator.
- 3- Stop enteral feeding for the first 2-3 days of life.
- 4- Give glucose 10% I.V.
- 5- Oxygen :head box, face mask .nasal Cannula
- 6- Continuous Positive Airway Pressure (CPAP).
- 7- Intubation.
- 8- Surfactant.
- 9- Broad spectrum antibiotics.

# **General management of poisoning**

1-The clinician should maintain a high index of suspicion to be able to arrive at the often difficult diagnosis of poisoning. Strongly consider ingestion in any patient with an unexplained loss of consciousness, especially if it is of a sudden onset in a previously healthy child.

2- Management priorities:

Emergency stabilization of the patient comes first.

3- Start by treating the patient, not the poison.

4- A.B.Cs of resuscitation then add "D" for drugs used for relief of other symptoms like convulsions.

5- Perform a brief neurological exam, establish the level of consciousness (Glasgow coma scale), and determine pupillary size and reactivity.

6- Institute an IV line, fluid therapy, drug therapy, oxygen, dextrose , as indicated.

7 -Consider decontamination: ocular, dermal, GI, etc.

## **History:**

-Symptoms complexes (toxicomes) may give clues to an unknown poisoning.

History aims to:

-Identify substance or substances including ingredients made in house.

-Identify the maximum possible amount (number in bottle originally, number left).

-Estimate ingestion, usually grossly under-estimated.

-Estimated time of ingestion.

-Symptoms.

-Home treatment.

- Glue exposure, recurrent episodes, etc.

## **Examinations:**

- Vital signs.

- Level of consciousness (GCS), motor function.

  - Eyes (pupils, EOM, fundi).

  - Mouth (lesions, odor).

- Heart (rate, rhythm).

- Lungs (rate, pattern).

- Skin odors (breath, clothing).

- Can the patient maintain the airway? Does the patient have a gag?

- Major modes of presentation are coma, disturbances, and seizures.

## **Investigations:**

- Blood glucose.
- LFT.
- CBC.
- Blood urea, creatinine & electrolytes. (including calcium).
- Urine and blood for TOX screen and drug levels if intoxicant is known.
- Serum osmolarity.

## **Management:**

### **1-Elimination of the poison:**

#### **A- Inhaled poisons:**

If smoke, gas or fumes have been inhaled carry the victim to fresh air.

#### **B- Poisons on the skins:**

Remove the clothing and flood the involved parts with water. Wash with soapy water and rinse thoroughly.

#### **C- Poisons on the eye:**

Rinse out the eyes with plain tap water for 15-20 minutes Do not try to neutralize acids or alkalies.

#### **D-Swallowed poisons:**

Eliminate poison by either:-

### **2- induction of emesis:-**

Don't induce vomiting if the child is:

- Comatose.
- Convulsing.
- Has lost his gag reflex.
- Ingested strong acid or alkali or hydrocarbons like kerosene.

The best method of vomiting induction is the use of ipecac syrup: 15 ml which might be repeated in 20 minutes if necessary. Be aware that the use of sodium chloride may lead to lethal hypernatremia and apomorphine is contraindicated because it is a centrally acting antiemetic leading to potentiation of the effects of the poisons affecting the CNS.

### **3- Gastric lavage:**

With the contraindications of induction of emesis, gastric lavage may be performed after the introduction of a cuffed endotracheal tube.

Gastric lavage is useful if it is performed within 1-2 hours of ingestion. Use a large 28-36 F. nasogastric tube is recommended, since smaller tubes are less effective. Lavage is best done with warm isotonic saline to avoid hypothermia especially in infants. The amount instilled should approximate the amount removed.

Emesis and lavage will remove about 30% of the amount of poison ingested.

## **2-Prevention of absorption of poisons:**

This is best done by giving activated charcoal:

Dose is 1-2 gm/kg (maximum 100 gm), it might be repeated every 2-6 hours until charcoal is passed per rectum. Prepare charcoal as slurry of a ratio of 1 : 4 charcoal to water.

Consider for all significant toxic ingestions . It is poorly bound to iron and lithium ,so it is not recommended for them . Do not use with caustic ingestion since it is poorly bound to them and it renders endoscopy difficult.

## **3- Enhancement of excretion:**

This is achieved by:

### **A -Forced diuresis**

- Used with pH modifications and it needs close monitoring for fear of toxicity.

1- 1/2 -2 X maintenance (3000 cc/m<sup>2</sup>/day) of fluids is to be given .

-Urine Output should approach 3-6 cc/kg/hr.

1-Alkalization of urine is used with ingestions of Phenobarbital and salicylate

Use 0.5 -2 mg/kg/hour of IV NaHCO<sub>3</sub> titrate to keep urine pH 7.5-8.0.

2-Acidification of urine :

and - Used for ingestions of amphetamine, chloroquine, lidocaine quinidine.

- Use Ammonium chloride 75 mg/kg/day q 4-6 p.O (contraindication, hepatic insufficiency).

Keep urine PH between 5.5 and 6.

### **B-Dialysis (consult nephrology)**

-Dialysis has been used for many substances, some of which are:

Ammonia, amphetamines, anilines, antibiotics, barbiturates, boric acid, bromides, calcium, chloral hydrate, ethylene glycol, fluorides, iodides, isoniazid, meprobamate, methanol, paraldehyde, potassium, quinidine, quinine ,salicylates, strychnine, thiocyanates.

Dialysis is preferably by hemodialysis or peritoneal dialysis if hemodialysis is not available. It is part of the supportive care if the child is having any of the following criteria:-

### **1-Clinical criteria:**

- a- Potentially life threatening toxicity caused by a dialyzable drug that cannot be dealt with conservatively.
- b- Severe hypotension which is not correctable by adjusting circulatory volume.
- c- Marked hyperosmolarity or electrolyte or acid base disturbances not responding to therapy.
- d- Marked hypothermia or hyperthermia not responding to therapy.

e- Dialysis is also indicated when: coma is deeper than level 3

## **2-Immediate dialysis**

Is to be considered in ethanol and methanol poisoning if acidosis is refractory or blood ethanol level is constantly above 100mg/dl.

### **4-Antidotes (see annex1)**

- Use of specific antidotes is invaluable, unfortunately few poisons have antidotes.
- Contact poison control for specific antidotes and doses.

### **5-Disposition:**

- May involve medical and/or psychiatric follow-up (psychiatric treatment may be necessary in certain patients, especially those with suicidal attempts).
- Consider social service involvement.

## **Annex 1**

<b><u>SUMMARY OF ANTIDOTES #</u></b>	
<u>POISON</u>	<u>ANTIDOTE(S)</u>
Acetaminophen	N-Acetylcysteine (Mucomyst) initial dose of 140 mg/kg PO in water, cola, juice or soda: then, 70 mg/kg q 4 hr for 68 hrs (17 doses, 18 total doses), see chapter
Anticholinergics	Physostigmine (adult, 2 mg; child, 0.5 mg) IV; may repeat in 15 min. until desired effect is achieved; subsequent doses q 2 - 3 hrs. prn. ( <b>CAUTION:</b> may cause seizures, asystole, cholinergic crisis)
Anticholinesterases	Atropine 2-5 mg (adults); 0.05-0.1 mg/kg (in children) IM or IV, repeated q 10-15 min until atropinization is evident;
Organophosphates	Pralidoxime chloride 1-2 grams (adults); 25-50 mg/kg (in children) IV; repeat dose in 1 hr if required, then q 6-8 hrs for 24-48 hrs. Consider constant infusion.
Carbamates	Atropine as above; pralidoxime for severe cases
Benzodiazepines	Flumazenil 0.01 mg/kg IV, max. dose 3 mg (estimated pediatric dose)
Beta-adrenergic blockers	Glucagon 50 micrograms/kg IV
Calcium channel blockers	Calcium chloride 10%, 10 ml (adult); 0.2 ml/kg (pediatric) IV or Calcium gluconate 10%, 30 ml (adult); 0.6 ml/kg (pediatric) IV Glucagon 50 micrograms/kg IV
Carbon monoxide	Oxygen 100% inhalation, consider hyperbaric for severe cases

Cyanide	<p>Adult: Amyl nitrate inhalation (inhale for 15-30 sec every 60 sec) pending administration of 300 mg sodium nitrite (10 ml of a 3% solution) IV slowly over 2-4 min., follow immediately with 12.5 grams sodium thiosulfate (2.5-5 ml/min of 25 % solution) IV</p> <p>Children: (Na+ nitrite should not exceed recommended dose because fatal methemoglobinemia may result, see below)</p> <table border="1"> <thead> <tr> <th>Hemoglobin</th><th>Initial dose 3% Na+ nitrite IV</th><th>Initial dose 25% Na+ thiosulfate IV</th></tr> </thead> <tbody> <tr> <td>8 g</td><td>0.22 ml (6.6 mg)/kg</td><td>1.10 ml/kg</td></tr> <tr> <td>10 g</td><td>0.27 ml (8.7 mg)/kg</td><td>1.35 ml/kg</td></tr> <tr> <td>12 g</td><td>0.33 ml (10 mg)/kg</td><td>1.65 ml/kg</td></tr> <tr> <td>14 g</td><td>0.39 ml (11.6 mg)/kg</td><td>1.95 ml/kg</td></tr> </tbody> </table>	Hemoglobin	Initial dose 3% Na+ nitrite IV	Initial dose 25% Na+ thiosulfate IV	8 g	0.22 ml (6.6 mg)/kg	1.10 ml/kg	10 g	0.27 ml (8.7 mg)/kg	1.35 ml/kg	12 g	0.33 ml (10 mg)/kg	1.65 ml/kg	14 g	0.39 ml (11.6 mg)/kg	1.95 ml/kg
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Digitalis	Fab antibodies (Digibind): dose based on amount ingested and/or digoxin level (see pkg. insert)															
Ethylene glycol	(see methanol)															
Fluoride	Calcium gluconate 10%, 0.6 ml/kg IV slowly until symptoms abate, serum calcium normalizes, repeat prn															
<b>Heavy metals/usual chelators</b>																
Arsenic / BAL	BAL (dimercaprol): 3-5 mg/kg/dose deep IM q 4 hours for 2 days, every 4-6 hours for an additional 2 days, then every 4-12 hours for up to 7 additional days															
Lead / BAL, EDTA,(± penicillamine), DMSA, (see chapter)	EDTA 50-75 mg/kg/24 hours deep IM or slow IV infusion given in 3-6 divided doses for up to 5 days;; may be repeated for a second course after a minimum of 2 days; each course should not exceed a total of 500 mg/kg body weight															
Mercury / BAL, DMSA	Penicillamine 100 mg/kg/day (max. 1 gram) PO in divided doses for up to 5 days; for long term therapy do not exceed 40 mg/kg/day  DMSA (succimer) 350 mg/M <sup>2</sup> (10 mg/kg) PO every 8 hours for 5 days, followed by 350 mg/M <sup>2</sup> (10 mg/kg) PO every 12 hours for 14 days															
Iron	Desferoxamine: 5-15 mg/kg/hr IV; use higher dose for severe symptoms and decrease as patient recovers (see chapter)															
Isoniazid	Pyridoxine 5-10%, 1 gram per gram of INH ingested IV slowly over 30-60 min.															

Methanol and Ethylene Glycol	<p>Ethanol, loading dose to achieve blood level of 100 mg/dl</p> <p>Adult: 0.6 grams/kg + 7-10 grams to be infused IV over 1 hour</p> <p>Children: 0.6 grams/kg to be infused over 1 hour</p> <p>Maintenance doses should approximate 10 grams/hour in adults and 100 mg/kg/hour in children, to be adjusted according to measured blood ethanol levels</p> <p><b>ALSO:</b></p> <p>Folate 50 -100 mg IV every 6 hours (methanol) ,Thiamine 0.5 mg/kg and pyridoxine 2 mg/kg for ethylene glycol</p>
Methemoglobinemic agents	Methylene blue 1%, 1-2 mg/kg (0.1-0.2 ml/kg) IV slowly over 5-10 min if cyanosis is severe or methemoglobin level is > 40%
Opioids	Naloxone 0.1 mg/kg IV, IM sublingual or via ETT
Phenothiazines (dystonic reaction)	Diphenhydramine 1-2 mg/kg slow IV or IM Max. dose 300 mg/day
Warfarin (and superwarfarin rat poisons)	Vit. K adult: 10 mg; children: 1-5 mg, slow IV, IM, SQ, or PO

## Annex 11

### TOXIC SYNDROMES

SYNDROME	MANIFESTATIONS	TYPES
Anticholinergic	<p>"mad as a hatter, red as a beet, blind as a bat, hot as a hare, dry as a bone"</p> <p><b>Parasympatholytic:</b> dry skin/mucous membranes, thirst, dysphagia, blurred vision (near objects), fixed, dilated pupils, tachycardia, hypertension, flushing, scarlatiniform rash, hyperthermia, abdominal distention, urinary urgency and retention</p> <p><b>Central:</b> lethargy, confusion, delirium, hallucinations, delusions, ataxia, respiratory failure, cardiovascular collapse, extrapyramidal movements</p>	<p>Belladonna alkaloids, atropine, scopolamine, plants (jimson weed, nightshade, mushrooms, Jerusalem cherries), phenothiazines</p> <p>Synthetic: Glycopyrrolate</p> <p>Others: Antihistamines, cyclic antidepressants</p>
Anticholinesterase	<p><b>Muscarinic:</b> sweating, constricted pupils, lacrimation, wheezing, cramps, vomiting, diarrhoea, tenesmus, bradycardia, hypotension, blurred vision, urinary incontinence, excessive salivation</p> <p><b>Nicotinic:</b> Striated muscle: fasciculations, cramps, weakness, twitching, paralysis, respiratory</p>	Organophosphates, carbamate insecticides

	<p>compromise, cyanosis, cardiac arrest</p> <p><b>Sympathetic ganglia:</b> tachycardia, hypertension</p> <p><b>Central:</b> anxiety, restlessness, ataxia, convulsions, insomnia, coma, absent reflexes, Cheyne-Stokes breathing, respiratory/ circulatory depression</p>	
Cholinergic	see anticholinesterases; nicotinic and muscarinic	Acetylcholine, betel nuts, bethanecol, muscarine, pilocarpine
Extrapyramidal	<b>Parkinsonian:</b> dysphonia, dysphagia, oculogyric crisis, rigidity, tremor, torticollis, opisthotonus, shrieking, trismus	Chlorpromazine, haloperidol, perphenazine, promazine, thiordiazine, trifluoperazine
Hemoglobinopathy	disorientation, headache, coma, dyspnoea, cyanosis, cutaneous bullae, gastroenteritis	Carboxyhemoglobin (carbon monoxide), methemoglobin, sulfhemoglobin
Metal fume fever	chills, fever, nausea, vomiting, muscular pain, throat dryness, headache, fatigue, weakness, leukocytosis, respiratory distress	<b>Fumes of oxides:</b> brass, cadmium, copper, iron, magnesium, mercury, nickel, titanium, tungsten, zinc
Narcotics	CNS depression, pinpoint pupils, slowed respirations, hypotension  Response to naloxone: pupils may dilate and excitement may predominate	Codeine diphenoxylate (Lomotil), fentanyl, heroin, morphine, opium, oxycodone
Narcotic <u>withdrawl</u>	diarrhoea, mydriasis, goose bumps (piloerection), hypertension, tachycardia, insomnia, lacrimation, muscle cramps, restlessness, yawning, hallucination	Cessation of: alcohol, barbiturates, benzodiazepines, chloral hydrate, glutethimide, meprobamate, methaqualone, narcotics, opioids, paraldehyde

Sympathomimetic	CNS excitation, convulsions, hypertension, tachycardia	Aminophylline, amphetamines, caffeine, cocaine, dopamine, ephedrine, epinephrine, fenfluramine, levarterenol, methylphenidate, pemoline, phencyclidine
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## Symptoms of organophosphorus poisoning

### Cardiovascular:

- Bradycardia .
- Hypotension.

### Respiratory:

- Rhinorrhoea .
- Bronchorrhoea .
- Bronchospasm.
- Cough.
- Severe respiratory distress.

### Gastrointestinal:

- Hypersalivation.
- Nausea, vomiting.
- Abdominal pain.
- Diarrhoea.
- Fecal incontinence.

### Genitourinary:

Incontinence.

### Ocular:

Blurred vision , mucosis .

### Glands:

Increase lacrimation ,diaphoresis.

### Nicotinic signs:

Muscle fasciculation, cramps, weakness, diaphoretic fever.

## **Autonomic nicotinic effect:**

Hypertension, tachycardia ,mydriasis, pallor.

## **Diagnosis:**

It is a clinical diagnosis with good history and physical examination .

## **Treatment procedure:**

- E.T intubation.
- I.V line.
- Continuous monitoring of O<sub>2</sub> with pulse oximetry should be established .
- ECG should be performed .
- Remove all clothing and gently cleans patients suspected of organophosphorus exposure.
- Treatment:- person should avoid contamination .
- Irrigate the eyes of the patients who had ocular exposure using isotonic solution and ringer solution.

## **Medications:**

- Mainstay treatment is atropine , benzodiazepine e.g (diazepam).

Route is: intraosseus.

## **Examples of organophosphorus:**

- a) Insecticides.
- b) Nerve gases.
- c) Anti-helminthic.
- d) Herbicides.

# **Paraphenylenediamine (Hair dye) Poisoning**

## **I. Introduction**

- A. Paraphenylenediamine (PPD) has traditionally been used as a dark-coloured hair dye. PPD has been used worldwide as a key ingredient in various hair dye formulations to produce a variety of colours, depending on its concentration. In Sudan, women use PPD to colour their hair and, when added to henna (*Lawsonia alba*), as a dye to decorate the palms of the hands and soles of the feet.
- B. Toxicity occurs through skin absorption. It is well known that PPD is an allergen that may cause contact dermatitis, erythematous urticarial papules and eczema in susceptible individuals. However, the major systemic problem occurs when it is ingested accidentally, for purposes of self-harm or during attempted murder.

## **II. Pathophysiology:**

- 1. PPD induces one of the most severe edema in humans. The edema appears to be grossly specific and selectively localized in the head and neck .
- 2. It is suggested that the toxic effect of the PPD might be produced by the conversion of the PPD on mucus surfaces to its oxidation product quinodamine , which is responsible for intense local irritation .
- 3. It is believed that PPD toxicity is due to some effects either on the blood colloids or on vascular permeability and involvement of the parasympathetic nervous system .
- 4. Deamination and formation of analine is claimed to be responsible

In part for the toxic symptoms .

- 5. PPD induces skeletal muscle lesions in the form of rhabdomyolysis with infiltration of inflammatory cells and necrosis.
- 6. At high concentrations and after a long period of exposure PPD produces cell death.
- 7. The lethal dose for humans is estimated to be 10 grams of pure PPD while 2-3 grams can cause severe toxic effects .

## **III. Clinical Effects:**

- A. The onset of symptoms usually occurs within hours of ingestion or contact with the dye.
- B. Patients with acute poisoning have a characteristic presentation of painless swelling of the face and neck often requiring urgent tracheostomy, with bulging eyes, a swollen dry hard protruding tongue and chocolate brown colour of the urine.
- C. PPD intoxication is a multisystem poison and can cause severe muscular pain due to rhabdomyolysis. It can also cause acute renal failure (ARF), flaccid paralysis, severe gastro-intestinal manifestations, cardiotoxicity and arrhythmias.
- D. This form of severe intoxication is fatal if not treated aggressively.
- E. Cardiac arrest is the main cause of death and it is attributed to arrhythmia.

## **IV. Evaluation and Treatment**

- A. ABC's FIRST!! , Perform urgent tracheostomy if needed.
- B. Perform complete history and physical exam next. (N.B. neurological manifestations and ARF may develop later).
- C. Do not induce vomiting or gastric lavage.

- D. Give steroids (hydrocortisone) 4 – 8 mg/kg/dose initially then 2 -4 mg/kg/dose 6hourly for 48-72 hours for the intense hypersensitivity reaction, the angio-oedema and as anti-inflammatory .
- E. Give Chlorpheneramine maleate 0.25 – 5 mg/kg/dose in children less than 5 years and 5 – 10 mg/kg/dose in children more than 5 years. Dose can be repeated 4hourly for up to 24 hours if needed.
- F. REFER the patient to the ENT hospital if facial oedema is increasing .
- G. All patients with respiratory distress, disturbed level of consciousness and/or irregular pulse should be admitted to the PICU.
- H. TOTALLY asymptomatic children with normal vital signs need close observations and monitoring for at least 72 hours.
- I. Acute renal failure (ARF) was found to be the second life threatening effect. Contact nephrology unit for hemodialysis, peritoneal dialysis and/or haemoperfusion.
- J. Lab. studies: CBC with differential, liver function tests and renal function tests with electrolytes should be drawn at baseline and for follow up. Importantly urine should be tested for myoglobin and for PPD using thin layer chromatography immediately at central laboratories for confirmation of intoxication and medico-legal purposes.
- K. Intubation: Consider if:
  - 1. Moderate to severe respiratory distress.
  - 2. ABG abnormalities; paO<sub>2</sub> <60 on 6 liters O<sub>2</sub> or paCO<sub>2</sub> >50mmHg.
  - 3. Deteriorating mental status .
  - 4. Absent breath sounds .
  - 5. Cyanosis on 40% FiO<sub>2</sub> .
  - 6. Exhausted patient leading to decreased respiratory effort.
- L. When the patient is stable contact authorities (Police, psychologist, social worker).
- M. Prophylactic antibiotics are not routinely prescribed.
  - 1. Antibiotics may be necessary later in the course in the face of persistent fever (> 36 hours), leukocytosis (> 36 hours), clinical deterioration or a positive tracheal gram stain or culture.
  - 2. Antibiotics should cover for mouth and GI flora: H. influenza, Staph aureus, Strep pneumoniae etc. Usual choices are Cefuroxime, Ceftriaxone, Clindamycin or Penicillin G.

#### V. Prognosis

- A. Majority recover fully if they survived the life threatening asphyxia .

# **Scorpion Sting**

## **I. Introduction**

- A. The sociocultural disposition and geographical features of Sudan expose its inhabitants to the risk of contact with a range of venomous scorpions.
- B. Scorpion sting is responsible for a number of deaths each year in many countries. Out of 1500 scorpion species, 50 are dangerous to humans .
- C. Most deaths occur during the first 24 hours after the sting and are secondary to respiratory and/or cardiovascular failure.

## **II. Pathophysiology**

- A. Scorpion venom may contain multiple toxins and other compounds.
- B. The venom is composed of varying concentrations of neurotoxin, cardiotoxin, nephrotoxin, hemolytic toxin, phosphodiesterases, phospholipases, hyaluronidases, glycosaminoglycans, histamine, serotonin, tryptophan, and cytokine releasers.
- C. Venom toxins alter sodium channels, leading to prolonged neuronal activity.
- D. Many end-organ effects are secondary to this excessive excitation. Autonomic excitation leads to cardiopulmonary effects observed after some scorpion envenomations. Somatic and cranial nerve hyperactivity results from neuromuscular overstimulation.
- E. Serotonin may be found in scorpion venom and is thought to contribute to the pain associated with scorpion envenomations.

## **III. Clinical Effects**

- A. Severe local skin manifestations :
  - 1. Pain at the sting site is present in almost all cases and may persist for up to 72 hours in some cases.
  - 2. Mild swelling around sting site.
  - 3. Numbness in the area and Sensitivity to touch.
- B. Systemic manifestations of envenomations in a much lower proportion include: Vomiting, sweating, restlessness, tachycardia and hypertension or hypotension
- C. Shock is marked among those under five years of age, and mild hypertension is common among adolescents and adults.
- D. Serious reaction include: Muscle spasms, Hyperventilation, reduced level of consciousness, anaphylactic shock, dysrhythmias or heart block.
- E. The mode of death is usually via respiratory failure secondary to anaphylaxis, bronchoconstriction, bronchorrhea, pharyngeal secretions, and/or diaphragmatic paralysis, even though venom-induced multiorgan failure plays a large role.

## **IV. Evaluation and Treatment**

### **Local treatment**

- A. ABC's FIRST! To provide adequate airway, ventilation, and perfusion.
- B. Use ice bags to reduce pain and to slow the absorption of venom via vasoconstriction. This is most effective during the first 2 hours following the sting.
- C. Immobilize the affected part in a functional position below the level of the heart to delay venom absorption.
- D. Calm the patient to lower the heart rate and blood pressure, thus limiting the spread of the venom.
- E. Apply a topical or local anesthetic agent to the wound to decrease paresthesia; this tends to be more effective than opiates .
- F. Administer local wound care and topical antibiotics to the wound .

- G. Administer tetanus prophylaxis booster dose if he or she has not had one within 5 years.
- H. Administer muscle relaxants for severe muscle spasm (ie , benzodiazepines) .

### **Systemic treatment**

- I. Monitor vital signs (eg, pulse oximetry; heart rate, blood pressure, and respiratory rate monitor).
- J. Administer oxygen if needed.
- K. Antivenin (polyvalent anti scorpion serum) is the treatment of choice after supportive care is established. The quantity to be used is determined by the clinical severity of patients and by their evolution over time.
  - 1. If the patient is asymptomatic do not give it and observe them for 6 hours
  - 2. If the patient came after 6 hours do not give anything and send home.
  - 3. If symptomatic give IV anti-scorpion and you can repeat every 1-2 hours according to clinical features of envenomation- up to 10-20 ml.
- L. For hypertension give short acting Nifedipine (Adalat). The dose can be repeated
- M. Keep the patient underhydrated and maintain only on one third requirement
- N. If the patient is not improving refer to PICU.

# Snake Bite

## I. Introduction

- A. More than 400 different species of snakes occurring in the African continent, only 90 of these have venomous bites, of them only 30 different species are known to have caused death.
- B. Snake bites can be deadly. It's important to react quickly to bites .
- C. It is worth knowing that different snakes have different systemic effects and the cause of death is mainly respiratory depression .

## II. Pathophysiology

- A. Venom is mostly water. Enzymatic proteins in venom impart its destructive properties. Proteases, collagenase, and arginine ester hydrolase have been identified in pit viper venom.
- B. Neurotoxins comprise the majority of snake venom. Specific details are known for several enzymes as follows:
  1. Hyaluronidase allows rapid spread of venom through subcutaneous tissues by disrupting mucopolysaccharides.
  2. Phospholipase A2 plays a major role in hemolysis secondary to the esterolytic effect on red cell membranes and promotes muscle necrosis.
  3. Thrombogenic enzymes promote the formation of a weak fibrin clot, which, in turn, activates plasmin and results in a consumptive coagulopathy and its hemorrhagic consequences.

## III. Laboratory tests

- A. Base line and serial laboratory tests are critical.
- B. Group and cross match of blood; Complete blood and platelet counts; Prothrombin and partial thromboplastin times; fibrinogen and fibrin degradation products; blood urea, creatinine, electrolytes and creatinine phosphokinase.

## IV. Clinical Effects

- A. Clinical manifestations depend on many variables including victim (age, general health and size), snake (species, glands and fangs) and bite (number, location, depth and amount injected poison).
- B. Envenomation grading determines the need for antivenin in victims of snake envenomations. Grades are defined as mild, moderate, or severe.
  1. Mild envenomation is characterized by local pain, edema, no signs of systemic toxicity, and normal laboratory values.
  2. Moderate envenomation is characterized by severe local pain; edema larger than 12 inches surrounding the wound; and systemic toxicity including nausea, vomiting, and alterations in laboratory values (eg, decreased hematocrit or platelet count).
  3. Severe envenomation is characterized by generalized petechiae, ecchymosis, blood-tinged sputum, hypotension, hypoperfusion, renal dysfunction, changes in prothrombin time and activated partial thromboplastin time, and other abnormal test results defining consumptive coagulopathy. **The crude clotting time is helpful and practical.**
- C. In most severe cases there is generalized oedema, shock, cardiac arrhythmias and death.

## V. Evaluation and Treatment

- A. ABC's FIRST!! and evaluating the patient for signs of shock (eg, tachypnoea, tachycardia, dry pale skin, mental status changes, hypotension).
- B. Important warnings :
  - 1. Do not try to **suck** out the venom.
  - 2. Do not attempt to **cut open the area around the bite**.
  - 3. Do not apply **ice** to the bite area.
  - 4. Do not **rub any substances** into the bite.
  - 5. Do not give anything **orally** to the victim.
  - 6. Do not **inject** anything, including antivenom unless you are qualified to do so. Anyone prone to allergies and asthma may go into **anaphylactic shock**.
- C. First aid manoeuvres should attempt to impede local lymphatic flow, the patient should be:
  - 1. Placed at rest with local pressure and immobilization of the extremity.
  - 2. close monitoring and large-bore IV access should be established
  - 3. Start a broad spectrum antibiotic.
- D. **Antivenin:** the decision to use antivenin should be based on the severity or rapid progression of the symptoms. It is most effective when given within 4 hours of the bite.
- E. Surgical assessment is essential as it focuses on the injury site and concern for the development of compartment syndrome which may need fasciotomy, monitoring of compartment pressure and follow up of the necrotic tissue.

## **Kerosene poisoning**

Kerosene and other hydrocarbons are widely used in Sudan for domestic purposes especially in poor areas when there is no electricity . Kerosene is prepared in bottles used for soft drinks , so children usually mistake it for drink during hot seasons . The main effect is aspiration pneumonitis A small quantity < 1 ml need to exert significant lung injury.

### **Clinical presentation :**

- Smell of kerosene .
  - Nausea and vomiting .
  - Signs of respiratory distress ( cough , tachypnoea , cyanosis )
- respiratory failure .
- Large amount ingestion may cause diarrhoea.
  - CNS depression, hyperthermia and encephalopathy.

### **Investigations:**

- ABG .
- CXR may be normal 8 – 12 hours. Abnormality may persist for long time.  
Pneumatoceles may develop 2 – 3 weeks later.

### **Treatment:**

- General supportive care.
- O<sub>2</sub>.
- Emesis is contraindicated.
- Steroids avoided.
- Antibiotics are not routine.
- Artificial ventilation for respiratory failure.

## **Salicylate poisoning**

Salicylates are found in many pharmaceutical preparations and a common analgesic ( aspirin ) found in commercial preparation . Toxicity is multi- organ , toxic dose > 150 mg / kg .

### **Clinical presentation:**

- 1.Hyperventilation &respiratory alkalosis initially , then dehydration, hypokalaemia and metabolic acidosis
- 2.Nausea , vomiting and tinnitus .
- 3.Hyperthermia , sweating ,delirium , vertigo , irritability and Hallucination are early symptoms .
- 4.Hypo or hyperglycaemia may develop .

### **Investigations :**

- Ferric chloride test for urine → brown red colour .
- Arterial blood gases (ABG) .
- Serum salicylate level plotted on anemogram to determine toxicity .
- Urine general , RFT, electrolytes .
- LFT.
- Blood glucose .
- Coagulation screen .

### **Treatment :**

1. General supportive care.
2. Gastric lavage & activated charcoal.
3. Rehydration & correction of electrolytes, potassium and bicarbonate.
4. Urine alkalinization.
5. Dialysis in severe cases.

### **Complications :**

- 1.Acid – base or electrolyte disturbances .
- 2.Seizures , cerebral oedema & coma .
- 3.liver toxicity & Reye syndrome .
- 4.Bleeding .

**Telephones : Drug Information Center**

1. **4141**
2. **0183 - 793200**
3. **0183 - 793201**
4. **0155 – 100040**
5. **0155 – 100044**

**E . Mail : khmic and live . com**

**Web site : www . khmic . org**

## Referral

**Child with a suspected surgical problem**

- Admit
- IV access
- If necessary nothing by mouth
- Take laboratory samples , blood grouping and cross matching
- Stabilize the child
- IV fluids and antibiotics ( if needed )
- Monitor vitals : blood pressure and others according to diagnosis
- Transfer accompanied by a trained nurse or doctor
- Make sure that an adequate referral note is delivered to the receiving unit

**Paediatric ICU or NICU**

- Stabilize the child and monitor vitals
- Insert a canula and keep IV line
- Give oxygen and suction
- Keep warm and avoid hypoglycaemia
- Make necessary arrangements
- Transport in ambulance with : sucker, oxygen source , ambubag , equipments and drugs for CPR in case of apnoea or cardiac arrest
- To be accompanied by a doctor or a trained nurse
- Make sure that a detailed referral note is delivered to the receiving unit



***Suggestions for improvements and additional guidelines would be most welcome by the Paediatric advisory committee.***

***Email: ytaha@doctors.org.uk***