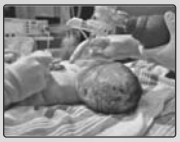
د نیونتالوژي او کوچنیانو د ناروغیو کلینیکي هنډبوک

(انګلیسي)

پوهنوال دوکتور منصور اسلم زی

Afghanic



English PDF
2016

Nangarhar Medical Faculty
نګرهار طب پوهنځی

Funded by
Kinderhilfe-Afghanistan

Clinical Handbook of Neonatology & Pediatrics

(in English)

Prof Dr Mansoor Aslamzai

Download: www.ecampus-afghanistan.org

ننگرهار طب پوهنځی

پوهنوال دوکتور منصور اسلم زی

د نیونتالوژي او کوچنیانو د ناروغیو کلینیکي هنډبوک

(انګلیسي)

سي ډي سره

۱۳۹۵ خرڅول منع دی

د نیونتالوژي او کوچنیانو د ناروغیو کلینیکي هنډبوک (انګلیسي)

Clinical Handbook of Neonatology & Pediatrics (in English)

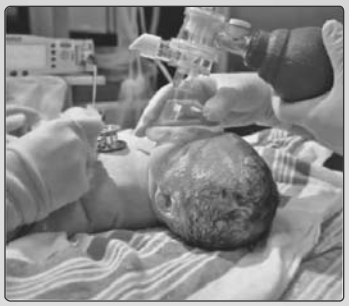
پوهنوال دوکتور منصور اسلم زی ۱۳۹۵

Nangarhar Medical Faculty

Afghanic

Clinical Handbook of Neonatology & Pediatrics

(in English)



Funded by
Kinderhilfe-Afghanistan

With CD

Prof Dr Mansoor Aslamzai

Not For Sale 2016

بسم الله الرحمن الرحيم

Clinical Handbook of Neonatology & Pediatrics

(in English)

There is the PDF file of this book on the CD along with it.

Prof Dr Mansoor Aslamzai

Book Name	Clinical Handbook of Neonatology & Pediatrics (in English)
Author	Prof Dr Mansoor Aslamzai
Publisher	Nangarhar University, Medical Faculty
Website	www.nu.edu.af
Copies	1000
Published	2016, First Edition
Download	www.ecampus-afghanistan.org

This Publication was financed by German Aid for Afghan Children, a private initiative of the Eroes family in Germany.

Administrative and technical support by Afghanic.

If you want to publish your textbooks please contact us:
Dr. Yahya Wardak, Ministry of Higher Education, Kabul
Office 0756014640
Email textbooks@afghanic.org

Printed in Afghanistan 2016

Sahar Printing Press

ISBN 978-9936-620-26-1

Message from the Ministry of Higher Education

In history, books have played a very important role in gaining, keeping and spreading knowledge and science, and they are the fundamental units of educational curriculum which can also play an effective role in improving the quality of higher education. Therefore, keeping in mind the needs of the society and today's requirements and based on educational standards, new learning materials and textbooks should be provided and published for the students.

I appreciate the efforts of the lecturers and authors, and I am very thankful to those who have worked for many years and have written or translated textbooks in their fields. They have offered their national duty, and they have motivated the motor of improvement.

I also warmly welcome more lecturers to prepare and publish textbooks in their respective fields so that, after publication, they should be distributed among the students to take full advantage of them. This will be a good step in the improvement of the quality of higher education and educational process.

The Ministry of Higher Education has the responsibility to make available new and standard learning materials in different fields in order to better educate our students.

Finally I am very grateful to German Aid for Afghan Children and our colleague Dr. Yahya Wardak that have provided opportunities for publishing textbooks of our lecturers and authors.

I am hopeful that this project should be continued and increased in order to have at least one standard textbook for each subject, in the near future.

Sincerely,
Prof. Dr. Farida Momand
Minister of Higher Education
Kabul, 2016

Publishing Textbooks

Honorable lecturers and dear students!
The lack of quality textbooks in the universities of Afghanistan is a serious issue, which is repeatedly challenging students and teachers alike. To tackle this issue, we have initiated the process of providing textbooks to the students of medicine. For this reason, we have published 223 different textbooks of Medicine, Engineering, Science, Economics and Agriculture (96 medical books funded by German Academic Exchange Service, 100 medical with 20 non-medical books funded by German Aid for Afghan Children and 4 non-medical books funded by German-Afghan University Society) from Nangarhar, Khost, Kandahar, Herat, Balkh, Kapisa, Kabul and Kabul Medical universities. It should be mentioned that all these books have been distributed among the medical and non-medical colleges of the country for free. All the published textbooks can be downloaded from www.ecampus-afghanistan.org.

The Afghan National Higher Education Strategy (2010-2014) states:

"Funds will be made available to encourage the writing and publication of textbooks in Dari and Pashto. Especially in priority areas, to improve the quality of teaching and learning and give students access to state–of–the–art information. In the meantime, translation of English language textbooks and journals into Dari and Pashto is a major challenge for curriculum reform. Without this facility it would not be possible for university students and faculty to access modern developments as knowledge in all disciplines accumulates at a rapid and exponential pace, in particular this is a huge obstacle for establishing a research culture. The Ministry of Higher Education together with the universities will examine strategies to overcome this deficit."

The book you are holding in your hands is a sample of a printed textbook. We would like to continue this project and to end the method of manual notes and papers. Based on the request of higher education institutions, there is the need to publish about 100 different textbooks each year.

I would like to ask all the lecturers to write new textbooks, translate or revise their lecture notes or written books and share them with us to be published. We will ensure quality composition, printing and distribution to Afghan universities free of charge. I would like the students to encourage and assist their lecturers in this regard. We welcome any recommendations and suggestions for improvement.

It is worth mentioning that the authors and publishers tried to prepare the books according to the international standards, but if there is any problem in the book, we kindly request the readers to send their comments to us or the authors in order to be corrected for future revised editions.

We are very thankful to **Kinderhilfe-Afghanistan** (German Aid for Afghan Children) and its director Dr Eroes, who has provided fund for this book. We would also like to mention that he has provided funds for 100 medical and 20 non-medical textbooks in the past.

I am especially grateful to **GIZ** (German Society for International Cooperation) and **CIM** (Centre for International Migration & Development) for providing working opportunities for me during the past five years in Afghanistan.

In our ministry, I would like to cordially thank Minister of Higher Education Prof Dr Farida Momand, Academic Deputy Minister Prof M Osman Babury, Deputy Minister for Administrative & Financial Affairs Prof Dr Gul Hassan Walizai, and lecturers for their continuous cooperation and support for this project.

I am also thankful to all those lecturers who encouraged us and gave us all these books to be published and distributed all over Afghanistan. Finally I would like to express my appreciation for the efforts of my colleagues Hekmatullah Aziz, Ahmad Fahim Habibi and Fazel Rahim in the office for publishing books.

Dr Yahya Wardak
CIM-Expert & Advisor at the Ministry of Higher Education
Kabul, Afghanistan, April, 2016
Office: 0756014640
Email: textbooks@afghanic.org

Preface

It is my pleasure to write the ***Clinical Handbook of Neonatology and Pediatrics*** by the help of Allah. Afghanistan has the second highest mortality rate of children in the world, so the best diagnosis and management of the pediatric diseases is highly required. It has been my endeavor to present this subject in a simplified and practical manner to provide adequate clinical guidance to pediatricians so that children derive the benefits of early diagnosis and optimal treatment. This book is more focused on the introduction, essentials of diagnosis and managements of common neonatal and pediatric diseases; necessary for the medical students, trainee doctors of DCH program and other pediatricians.
This clinical handbook includes; Neonatology, Pediatric emergencies, Respiratory diseases, Diarrheal and Infectious diseases, Fluid, Electrolytes and Acid-base disorders, Nutritional disorders, Hematological and Renal disorders. During the writing, I hardly work to describe the diagnosis and management of the mentioned topics in such a way that have up-to-date, enough, easy and step by step information for doctors and medical students.

I would like to thanks from the Ministry of Higher Education and Dr. Yahya Wardak for the publication of this book.

At the end, any suggestions and comments about this book are kindly accepted by me.

Dr.Mansoor (Aslamzai), MD
Professor of Pediatric, Department of Neonatology
Kabul Medical University and Ataturk Hospital

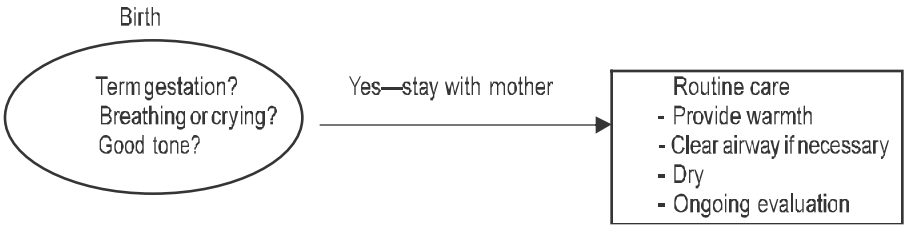
Contents

Part 1
Neonatology
Care of the normal newborn

At the time of birth, you should ask yourself 3 questions about the newborn:

- Is the baby born at term?
- Is the baby breathing or crying? (Identified by observing chest rise, which should be visible and regular)
- Does the baby have good tone? (Identified by noting the posture, which should show generalized flexion at upper and lower limbs).

If the answer to all 3 questions is "Yes," the baby should stay with the mother and routine care should be taken.



Routine care immediately after birth:

- Provide warmth
- Clean mouth & nose
- Dry the baby
- Determine Apgar score at 1min
- Clamping of umbilical cord.
- Placement of identity band.

Care of the Newborn within few hrs after birth:

- Recording of weight
- First examination
- Initiation of breastfeeding within 1hr of birth.
- Vit K 0.5mg for baby less than 1000gr and 1mg for baby more than 1000gr weight should be given intramuscular.
- Eyes care: Application of one drop of Silver nitrate 0.5-1% or Tetracycline ointment in both eyes.
- Communication with family about gender, birth weight and well-being of the baby.

Care of the Newborn beyond few hrs after birth:

- Cord should be kept dry and clean.
- Oil massage: Massage the LBW baby with olive oil.
- Exclusive breast feeding is advised.
- Supine position in sleep is better to prevent SIDS.
- Traditional practice that should be discourages.
- Timing of discharge: At least after 8-24hr and the following criteria should be met:

- Examination has been performed.
- Breastfeeding properly.
- Has received immunization
- No any illness
- Mother is confident and trained to take care of baby.

- Follow up visit at 48-72hr of discharge for primigravida, LBW, preterm, significant jaundice, feeding not established. Normal baby can be seen at 6w of age.

Resuscitation of the Newborn

Needs of resuscitation in newborn babies are assessed by the following questions:

- ***Is the baby born at term?***

If the baby is born preterm, there is a significantly higher likelihood that some degree of resuscitation will be required. For example, preterm babies are more likely to have stiff, underdeveloped lungs, may have insufficient muscle strength to make strong initial respiratory efforts, and have less capacity to maintain body temperature after birth.

- ***Is the baby breathing or crying?***

Breathing is evident by watching the baby's chest. A vigorous cry also indicates breathing. However, do not be misled by a baby who is gasping. Gasping is a series of deep, single or stacked inspirations that occur in the presence of hypoxia and/or ischemia. It is indicative of severe neurologic and respiratory depression.

- ***Is there good muscle tone?***

Healthy term babies should have flexed extremities and be active as opposed to sick or preterm babies, who often will have extended and flaccid extremities.

If the answer to any of the 3 questions is "No," then start ABCD of resuscitation.

Table -1: Equipment and drugs of resuscitation

Equipment
– Resuscitator with overhead warmer and light, and firm, padded, and heated mattress
– Two sources of medical oxygen
– Two sources of medical air
– Two blenders (air/oxygen)
– Clock with timer in seconds
– Warmed towels or other covering
– Polyethylene bag, or wrap, big enough for a baby less than 1,500 g birth weight.
– Stethoscope, neonatal size
– Suction catheters (6 F, 8 F, 10 F, 12 F)
– Oxygen/Air admixture supply (flow rate up to 10 L) with flow meter and tubing
– Face masks (various sizes)
– Oropharyngeal airways (sizes 0 and 00)
– Positive pressure ventilation (Two in case of twin deliveries):
• T-piece device
• Self-inflating bag with an oxygen reservoir and a manometer if available
• Flow-inflating bag with a pressure safety valve and manometer
– Laryngoscopes with straight blade (00, 0, 1), spare bulbs, and batteries
– Endotracheal tubes (sizes 2.5, 3, 3.5, and 4 mm ID)
– Endotracheal stylet or introducer
– Supplies for fixing endotracheal tubes and IVs (e.g., scissors, tape)
– Meconium suction device (to apply suction directly to endotracheal tube)
– Feeding tubes for gastric decompression
– Umbilical vein catheterization set and umbilical catheters (5 F) with suitable skin prep solution
– Syringes with needles (assorted sizes)
– Intravenous cannulae (assorted sizes)
– Pulse oximeter with sensors adequate for different gestational ages

Drugs
– Adrenaline: 1:10 000 concentration (0.1 mg/mL)
– Volume expanders: Normal saline, Ringer lactate, O Rh –ve blood needs to be readily available for a profoundly anemic baby
– Sodium bicarbonate: 0.5 mmol/mL solution (4.2% concentration, or diluted 7.5 or 8.4%)
– Naloxone hydrochloride: 400 mg/mL solution
– Sterile water for injection

ABCDs of Resuscitation

A (Airway Patency): 1st step in first 30 sec

1- **Provide warmth**: Neonates should be kept under radiant warmer. Do not allow the infant to become hyperthermic.

2- **Position**: Keep the baby in supine position with neck slightly extended. Place a rolled towel under the shoulder to elevate 1 inch (figure 1).

Figure-1: Sniffing position of the baby with neck slightly extended

3- **Dry the newborn** to avoid hypothermia.

4- **Suction**: initially mouth then nose, do not insert NGT very deep.

5- **Tactile stimulation**: Flicking or slapping the foot or rubbing the back for a few seconds (figure 2).

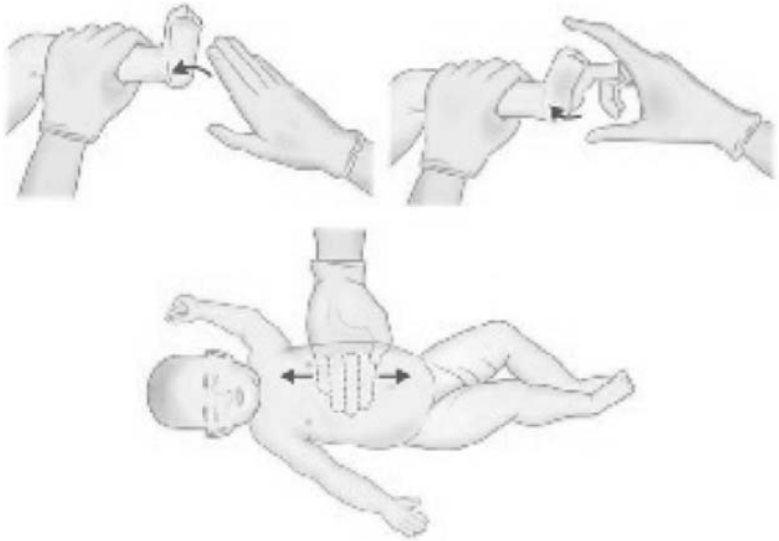


Figure 2: Methods to provide tactile stimulation

Clearing the airway for meconium stained neonates is provided as following:

1- Vigorous infant (good respiratory, heart rate >100 beats/min and good muscle tone): Tracheal intubation to aspirate meconium **should not be attempted**; the mouth and nose may be suctioned with a bulb or suction catheter.

2- Not vigorous infant (depressed respirations, has depressed muscle tone, and/or has a heart rate below 100 beat/min): Direct suctioning of the trachea soon after delivery is indicated before many respirations have occurred to reduce the chances of the baby developing meconium aspiration syndrome.

Evaluate the Respiration, HR and color

1-If the baby *is* breathing and the heart rate is *above* 100 bpm, but the respirations are labored or persistent cyanosis is observed the following actions maybe taken:

- Monitor SpO_2 by attaching a pulse oximeter probe to right wrist and determine the baby's oxygenation. Normal values of SpO_2 are shown in table 2.
- Supplemental oxygen therapy: Give 21-100% oxygen 5L/min.
- Consider continuous positive airway pressure (CPAP) by face mask.

Table 2: Values of preductal oxygen saturation during first 10min of life

Time	SpO2 (%)
1min	60 – 65
2min	65 – 70
3min	70 – 75
4min	75 – 80
5min	80 – 85
10min	85 – 95

2- If the baby is not breathing (apneic) or is gasping, the heart rate is below 100 beats per minute (bpm) even with breathing, and/or the saturation remains below target values despite free-flow supplemental oxygen being increased to 100%, the next step is to provide PPV.

B (Breathing Initiation): 2nd step in second 30 sec.

- Initiated Positive Pressure Ventilation (PPV) or Bag & Mask ventilation with an inspiratory pressure of about 20 cm H_2O, at a rate of 40 to 60 breaths per minute. The mask should cover the tip of the chin, the mouth, and the nose, but not the eyes (figure 3).You will need to position yourself at the baby's side or head to use a resuscitation device effectively.

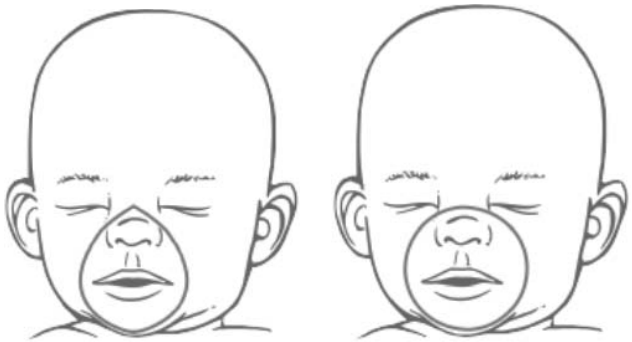


Figure 3: Correct size of masks

If the chest is not moving with each breath and there are poor breath sounds, begin the ventilation corrective sequence. There are 3 possible reasons for ineffective ventilation:

- An inadequate seal between the mask and the baby's face: Use a little more pressure on the rim of the mask and lift the jaw a little more forward.

- The baby's airway is blocked: Reposition the baby's head. Check the mouth, oropharynx, and nose for secretions; suction the mouth and nose if necessary. Try ventilating with the baby's mouth slightly open (especially helpful in extremely small premature babies with very small nares).
- Not enough pressure is being used to inflate the lungs: Increasing the amount of positive pressure to 30 cm H_2O or greater.

- Oxygen therapy: 21% O_2 (room air) is the preferred initial gas for neonatal resuscitation in term infants; if the neonate does not achieve normal oxygen saturation levels within 90 sec, increasing concentrations of oxygen should be blended in (up to 100% oxygen) until normal oxygen saturation levels are achieved. Preterm newborns may achieve normal oxygen saturations more quickly if you start with a somewhat higher oxygen concentration.
- Consider SpO_2 monitoring.

Evaluate the Respiration and HR

1- When the heart rate is above 100 bpm and stable, reduce the rate and pressure of PPV while observing for effective spontaneous respirations and stimulating the baby to breathe effectively. Positive-pressure ventilation may be discontinued when the baby has:

- A heart rate continuously over 100 bpm
- Sustained spontaneous breathing

2- If the heart rate is more than 60 bpm but less than 100 bpm, continue to administer PPV as long as the baby is showing steady improvement.

3- If the baby's condition continues to deteriorate or fails to improve, and the heart rate is below 60 bpm despite 30 seconds of effective PPV (defined by audible bilateral breath sounds and chest movement with ventilation), your next step will be to begin chest compressions (3^{rd} step).

C (Circulation maintenance): 3^{rd} Step in third 30 sec.

Chest compressions are indicated when the heart rate remains below 60 beats per minute, despite 30 seconds of effective positive pressure ventilation.

- Chest compression should be done 90/min. During chest compressions, the breathing rate is 30 breaths per minute and the compression rate is 90 compressions per minute. This equals 120 "events" per minute. One cycle of 3 compressions and 1 breath takes 2 seconds. The ratio of compression and ventilation is 3:1, with tow thumbs (preferred) or tow finger technique on the lower third of the sternum. With your fingers and hands correctly positioned, use enough pressure to depress the sternum to a depth of approximately one-third of the anterior-posterior diameter of the chest (figure 4).
- Positive Pressure Ventilation (PPV) or bag and mask ventilation 30/min should be continuing along with chest compression.

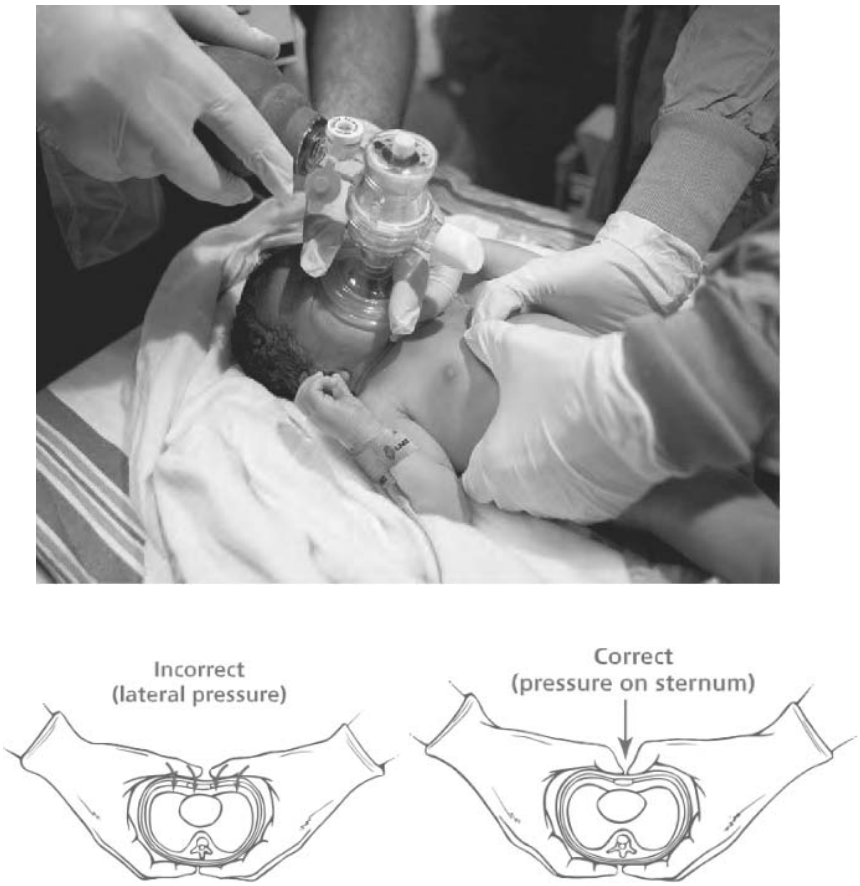


Figure 4: Correct and incorrect application of pressure in thumb technique

Evaluate the HR

After 30 seconds of chest compressions and ventilation, check the heart rate. If the heart rate is:

- Greater than 100 beats per minute discontinue compressions and gradually discontinue ventilation if the newborn is breathing spontaneously.
- Greater than 60 beats per minute discontinue compressions and continue ventilation at 40 to 60 breaths per minute.
- Less than 60 beats per minute, intubate the newborn (if not already done), and give epinephrine as describe in 4th step.

D (Drugs): 4th Step

1. **Epinephrine (Adrenalin)** 0.1 –0.3 ml /kg of 1:10000 solution is administered through umbilical vein. May be repeated every 3-5 min if the response is poor.
2. **NaHCO3** 2 -3 mEq /kg of 4.2% solution without dilution or 7.5% solution diluted with equal volume of distilled water or double volume of 5% glucose solution should be used IV slowly (1 ml /min) for neonate with prolong birth asphyxia (neonate needed bag and mask ventilation even at 5 minute) or suspected metabolic acidosis.
3. **Fluid** (ringer lactate or N/S) 10-20 cc/ kg or 0 – ve blood (in acute hemorrhage).
4. **Naloxan HCL** 0.1mg/kg should be administered via umbilical vein if the mother has history of narcotic analgesic administration within 4hr prior to delivery. It can be repeated after every 2-3 minutes.

Indications for indotrachial intubations

1. For tracheal suctioning in meconium stained.
2. If prolong PPV is required.
3. Ineffective bag and mask ventilation.
4. Diaphragmatic hernia.
5. Extremely preterm baby (< 28 weeks or < 1000gr).
6. Neonates requiring administration of surfactant.

Table 3.Endotracheal tube sizes and depth of insertion from the lips

Weight (g)	Gestation (weeks)	Tube size (mm)	Depth of insertion from lip (cm)
< 1,000	< 28	2.5	6.5–7
1,000–2,000	28–34	3.0	7–8
2,000–3,000	34–38	3.0/3.5	8–9
> 3,000	> 38	3.5/4.0	> 9

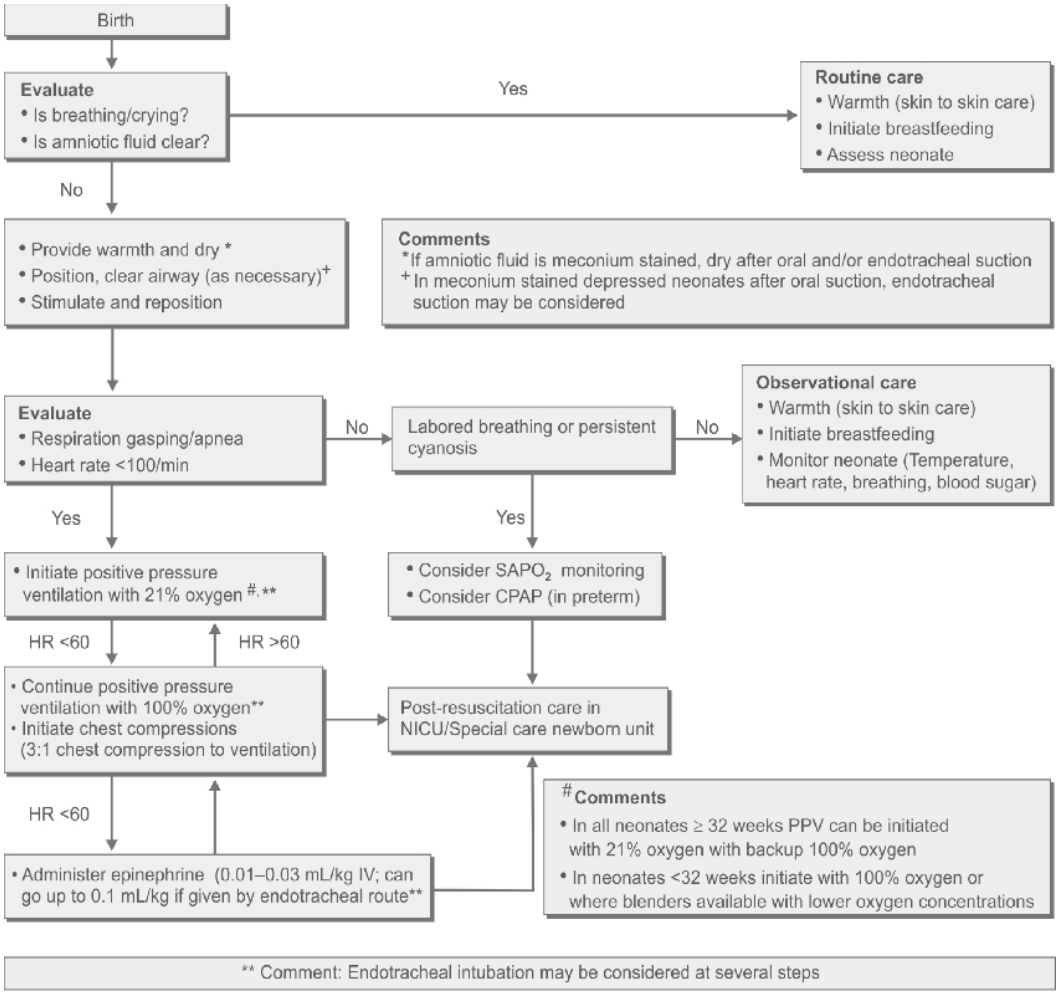


Figure 5: Algorithm of Neonatal resuscitation

When to stop resuscitation

There is no need for resuscitation if:

- Gestational age < 22w or
- BW < 400gr

• Resuscitation is discontinued if:

- After 10 min there is no respiration and heart rate.
-After 20 min there is no respiration and heart rate remain below 60/min.

Perinatal Asphyxia

Definition and Essentials of diagnosis

A gold standard definition of birth asphyxia does not exist. It is probably better to use the term perinatal asphyxia since asphyxia may occur in utero, at birth or in the postnatal period.

WHO has defined perinatal asphyxia as a "failure to initiate and sustain breathing at birth".

According to the definition of AAP (American academy of Pediatrics) and ACOG (American college of Obstetrics and Gynecology), all the following must be present.

(a) Profound metabolic or mixed academia (pH< 7.00) in cord blood. (b) Persistence of Apgar scores 0-3 for longer than 5 minutes. (c) Neonatal neurologic sequelae (eg, seizures, coma, Hypotonia). (d) Multiple organ involvement (eg, of the kidney, lungs, liver, heart, intestine).

Classification:

1-Moderate asphyxia: as slow gasping breathing or an Apgar score of 4-6 at 1 minute of age.

2-Severe asphyxia was defined as no breathing or an Apgar score of 0-3 at 1 minute of age. Apgar score is shown in table.4

Table 4: Apgar score

Table 2.2.1 Apgar score SS			
Parameters	**0**	**1**	**2**
Respiratory effort	Absent	Gasping	Good cry
Heart rate	Zero	< 100/min	> 100/min
Color	Central cyanosis	Peripheral cyanosis	Pink
Tone	Flaccid	Partial flexion of extremities	Complete flexion
Response to nasal catheter	None	Grimace	Sneeze

Management of Perinatal Asphyxia

1- Infants with severe asphyxia (Apgar score 0-3 at 1 minute or need for prolonged bag and mask ventilation >5 minutes) should be transferred to a special care newborn unit for observation and treatment.

2- **Stabilized ABCs** of the resuscitation.(See resuscitation of the newborn)

3- All babies with Apgar scores <4 at 1 minute or <7 at 5minutes of age should be started on intravenous fluids. Maintenance fluid should be reduced to 2/3 of normal.

4- **NBM** (Nothing by Mouth) if the neonate is critical ill, convulsive, has apnea or severe asphyxia.

5- Maintain normal temperature.

6- Consider infusion of volume expander: If the capillary refill time is more than 3 seconds or if there is

metabolic acidosis, volume expansion with normal saline (or Ringer's lactate) 10 ml/kg over 5-10 min should be instituted. This may be repeated, if required.

7- Treat the following conditions if present :

- **Hypoglycemia** (Blood glucose < 50mg/dl): Give 2ml/kg of D/W 10% bolus IV and then 6-8mg/kg/min to maintain normal blood glucose)
- **Hypocalcemia** (Blood calcium < 7mg/dl): For symptomatic (irritability, seizure, apnea, tetany) cases give 1-2ml/kg calcium gluconate 10% IV over 5min.(See management of Neonatal Hypocalcemia)
- **Seizure** (see management of Neonatal Seizure)
- **Acidosis**(see under resuscitation)
- **Shock** (see management of shock)

8- **Antibiotics:** For suspected NNSepsis, history of PROM in mother, foul smelling amniotic fluid and antepartum fever in mother give first line antibiotic as mention under NNSepsis.

9- Prophylactic Vit k 1mg IM within 2hr of birth.

10- **Therapeutic hypothermia:** For hypoxic-ischemic encephalopathy isolated cerebral cooling or more often systemic induced servo controlled hypothermia to a core (rectal) temperature of 33.5°C (92.3°F) within the 1st 6 hr after birth (duration 72 hr) reduces mortality and major neurodevelopmental impairment at 18 mo of age.

Neonatal Seizure

Definition

A seizure is defined clinically as a paroxysmal alteration in neurologic function, i.e. motor, conscious, behavior and/or autonomic function. This definition includes:

1. Epileptic seizures: phenomena associated with corresponding EEG seizure activity e.g. clonic seizures.
2. Non-epileptic seizures: clinical seizures without corresponding EEG correlate e.g. subtle and generalized tonic seizures.
3. EEG seizures: abnormal EEG activity with no clinical correlation.

Classification

Four types of NS have been identified:

A-Subtle seizures: They are called subtle because the clinical manifestations are mild and frequently missed. They are the commonest type and constitute about 50% of all seizures. Common examples of subtle seizures include:

1. *Ocular* - Tonic horizontal deviation of eyes or sustained eye opening with ocular fixation or cycled fluttering
2. *Oral–facial–lingual movements* - Chewing, tongue-thrusting, lip-smacking, etc.
3. *Limb movements* - Cycling, paddling, boxing-jabs, etc.
4. *Autonomic phenomena* - Tachycardia or bradycardia
5. *Apnea* may be a rare manifestation of seizures.

B-Clonic seizures: They are rhythmic movements of muscle groups.

C-Tonic seizures: This type refers to a sustained flexion or extension of axial or

appendicular muscle groups. These seizures may be focal or generalized and may resemble decerebrate (tonic extension of all limbs) or decorticate posturing (flexion of upper limbs and extension of lower limbs).

D-Myoclonic seizures: These manifest as single or multiple lightning fast jerks of the upper or lower limbs and are usually distinguished from clonic movements because of more rapid speed of myoclonic jerks, absence of slow return and predilection for flexor muscle groups.

Investigations

1-Essential investigations: Investigations that should be considered in all neonates with seizures include blood sugar, serum electrolytes (Na, Ca, Mg), cerebrospinal fluid (CSF) examination, cranial ultrasound (US), and electroencephalography (EEG). CSF examination should be done in all cases as seizures may be the first sign of meningitis.

2-**Additional investigations**: These may be considered in neonates who do not respond to a combination of phenobarbitone and phenytoin or earlier in neonates with specific features. These include neuroimaging (CT, MRI), screen for congenital infections (TORCH) and for inborn errors of metabolism.

Management of Neonatal Seizure

1. ABC of resuscitation should be stabilized.
2. O_2 therapy should de started.
3. Open IV rout.

4. A brief relevant history should be obtained and quick clinical examination should be performed. All this should not require more than 2-5 minutes.

5. Correction of hypoglycemia and hypocalcemia: Give the following drugs step by steps:

A: Dextrose: If glucostix shows hypoglycemia or if there is no facility to test blood sugar immediately, 5-10 ml/kg of 10% dextrose should be given as a bolus injection followed by a continuous infusion of 6-8 mg/kg/min.

B: Calcium gluconate 10% 2cc /kg diluted with equal volume of 5% glucose or distilled water and injected slowly in 5-10 minute through IV. If ionized calcium levels are suggestive of hypocalcemia, the newborn should receive calcium gluconate at 8 ml/kg/d for 3 days.

C: Magnesium sulphate: If seizures continue despite correction of hypocalcemia, 0.25 ml/kg of 50% magnesium sulfate should be given intramuscularly (IM) in 2 doses 12hr apart and be followed by maintenance oral dose of 0.2ml/kg of 50% solution once daily for 3 days. It is recommended for unresponsive hypocalcemia, suspected or proved hypomagnesaemia (serum level < 1mEq/l)..

6: Anticonvulsants: The following anticonvulsive drugs should be given step by step if seizures persist even after correction of hypoglycemia/ hypocalcemia.

A: Phenobarbital loading dose 20 mg /kg is administered slowly IV over 20 minutes (not faster than 1 mg/kg/min). If seizures persist after completion of this loading dose, additional doses of phenobarbitone 10 mg/kg may be used every 15 minutes until a total dose of 40 mg/kg has been given. If convulsions are still uncontrolled, add Phenytoin.

Maintenance dose of Phenobarbital is 5mg /kg/day usually in one or 2 divided doses, started 12 hours after the loading dose.

B: Phenytoin or Fosphenytoin is administered intravenously in a loading dose of 15-20 mg /kg. Phenytoin is diluted in normal saline (not in glucose containing solution) and given slowly over 10-20 minutes or at a rate of 1mg/kg/min. Assess control after 30 minute, if seizure persist then repeat 10mg/kg (may repeat totally up to the dose of 30mg/kg). Maintenance dose is 5mg /kg/day in one or 2 divided doses. Only IV route is preferred in neonates and it should preferably be discontinued before discharge.

C: Benzodiazepines: For refractory cases give the following drugs (especially Lorazepam and Midazolam) step by steps

- **Lorazepam** 0.05-0.1mg/kg IV bolus over 2-5 minutes; may be repeated q 8-12hr.
- **Midazolam** 0.05-0.15 mg/kg IV bolus followed by infusion of 0.1 to 0.4 mg/kg/hour.
- **Diazepam** 0.1-0.3mg/kg is given intravenously over 3-5 minutes. Repeat every 15-30 minutes if not respond.

7: **Pyridoxine** 50-100mg IV or IM is useful in refractory cases.

8: Lidocaine, Paraldehyde, Sodium valproate, Vigabatrin and Topiramate are the anticonvulsants in refractory cases.

Duration of Anticonvulsive therapy:

The duration of anticonvulsive therapy is guided by neurological status, cause of the seizure and EEG findings. All anticonvulsants are stopped except Phenobarbitone when seizures are controlled. At discharge, if CNS examination is normal Phenobarbitone may be stopped. Phenobarbitone is

continued if there are any CNS abnormalities at the time of discharge and baby is re-assessed at one month. If there is no recurrence of seizures; normal CNS examination, EEG, CT scan and or MRI, Phenobarbitone is tapered over next 2 weeks. When Phenobarbitone is continued, infant is evaluated at the age of 3 months and treated as a case of epilepsy.

Neonatal Sepsis

Introduction

Neonatal sepsis is a clinical syndrome characterized by signs and symptoms of infection with or without accompanying bacteremia in the first month of life. It encompasses various systemic infections of the newborn such as septicemia, meningitis, pneumonia, arthritis, osteomyelitis, and urinary tract infections.

Classification

Neonatal sepsis can be classified into two major categories depending up on the onset of symptoms.

1-Early onset sepsis (EOS): Presents within the first 72 hours of life.

2-Late onset sepsis (LOS): It usually appears after 72hrs of age.

Etiology

1- Early onset Sepsis pathogens:

- Group B Streptococcus.
- E coli
- Klebsiella pneumoniae

- Listeria monocytogenes
- Enterobacteria
- Others (Viruses and Fungus)

2- Late onset Sepsis pathogens:

- Klebsiella pneumoniae
- E coli
- Group B Streptococcus
- Staphylococcus aureus
- Pseudomonas
- Enterobacteria
- Others (Viruses and Fungus)

Risk factors

1- Risk factors for EOS are:

- Low birth weight (<2500 grams) or Prematurity.
- Febrile illness in the mother with evidence of bacterial infection within 2 weeks prior to delivery.
- Foul smelling and/or meconium stained liquor.
- Rupture of membranes >24 hours.
- Single unclean or > 3 sterile vaginal examination(s) during labor
- Prolonged labor (sum of 1st and 2nd stage of labor > 24 hrs)
- Perinatal asphyxia (Apgar score <4 at 1 minute).

2- Risk Factors of LOS: low birth weight, prematurity, admission in intensive care unit, mechanical ventilation, invasive procedures, administration of parenteral fluids, , and use of stock solutions. Poor hygiene, poor cord care, bottle-feeding, and prelacteal feeds.

Essentials of Diagnosis

Sepsis in the newborn is diagnosed when it meets the following criteria: (1) any two clinical signs and (2) any two laboratory signs, (3) in the presence of or as a result of suspect or proven infection (European Consensus statement 2010).

1. Any two clinical signs:

- Temperature instability

-- Core temperature greater than 38.5°C or less than 36°C

- Cardiovascular instability

-- Tachycardia (heart rate 180 beats/min) in the absence of external stimulus or pain or drugs, i.e. "unexplained" increase in heart rate for 0.5–4 hours

-- Bradycardia (heart rate 100 beats/min) for 0.5 hours in the absence of heart block, external vagal stimulus, beta-blockers

-- Rhythm disturbances

-- Reduced blood pressure (systolic BP less than 65 mm Hg in first week and less than 75 mm Hg between 1 week and 1 month)

-- Mottled skin and impaired peripheral perfusion

-- Decreased urine output (less than 1 mL/kg/hour)

- Respiratory instability

-- Apneic episodes

-- Respiratory rate greater than 2 SD (> 60 beats/ min)

-- Increased oxygen need

--Ventilation for acute process for causes other than neuromuscular or general anesthesia

- Gastrointestinal

-- Feed intolerance, abdominal distension, poor sucking

- Petechial rash or sclerema
- Nonspecific: lethargy, irritability, hypotonia

2. Any two laboratory tests:

- Abnormal leukocyte count (> 20,000 × 10^9/L or less than 5000 × 10^9/L)
- Immature to total neutrophil (I/T) ratio (> 0.2)
- Platelet count less than 100,000 × 10^9/L
- C-reactive protein (CRP) greater than 1mg/dl
- Procalcitonin (PCT) greater than 2 ng/ml
- Metabolic acidosis; base excess (BE) greater than –10
- Blood sugar greater than 180 mg/dl or less than 45mg/dl confirmed at least two times on age appropriate infusions

3. Evidence of infection:

- Proven (positive culture or microscopy or polymerase chain reaction)
- Suspected (clinical syndrome, chest X-ray consistent with pneumonia or white cells in normally sterile fluid).

Investigations

1-Sepsis screen: When two or more indirect markers of table 5 are seen sepsis screen become positive.

2- Blood culture, urine culture, imaging and lumbar puncture are needed.

Table 5: Sepsis screen

Parameters (Indirect markers)	Abnormal Value
Total leukocyte count	< 5000 - > 20000/mm3
Absolute Neutrophil count	< 1800/mm3
Absolute Neutrophil count	> 0.2
Micro-ESR	> 15mm 1st hr
C-reactive protein	> 1mg/dl

Management of Neonatal Sepsis

1. ABCs of resuscitation should be stabilized if needed.
2. The patient should be nursed in a thermo-neutral environment taking care to avoid hypo/hyperthermia.
3. If the infant is hemodynamically unstable, intravenous fluids should be administered.
4. Infuse D/W 10% 2 ml/kg intravenously.
5. **NBM** is indicated for very sick neonates, RR >80/min, convulsion or apnea attacks. In such condition give maintenance IV fluid.
6. **Antibiotics**

Indications for starting antibiotics:

A-The indications for starting antibiotics in neonates at risk of EOS include any one of the following:

(a) Presence of >3 risk factors for early onset sepsis (*see above*)

(b) Presence of foul smelling liquor

(c) Presence of 2 antenatal risk factor(s)

(d) Positive septic screen

(e) Strong clinical suspicion of sepsis.

B-The indications for starting antibiotics in LOS include:

(a) Positive septic screen and/or

(b) Strong clinical suspicion of sepsis.

A: First line Antibiotics: **Ampicillin + Gentamicin.**
If the child is critically ill or do not respond in 48 hours, start second line antibiotics:

B: Second line Antibiotics: **Ampicillin + 3rd generation Cephalosporin.**

C: In resistant cases; **Cloxacillin, Vancomycin, Cefepime, Meropenem** or **imipenem** administration should be evaluated.

Dosages of antibiotics:

- **Ampicillin:** 50mg/kg BD for less than 7 days old or less than 1200g, 50mg/kg TID for 7 days or older and more than 1200g IM or IV. **For meningitis double the dosages.**
- **Amikacin:** 7.5mg/kg BD for less than 7 days old and less than 2000g, 10mg/kg BD for less than 7 days old and more than 2000g , 15mg/kg TID for 8-28days old IM or IV.
- **Gentamicin:** 2.5mg/kg BD for less than 7 days old or less than 1200g, 2.5mg/kg TID for 7 days or older and more than 1200g IM or IV.
- **Cefotaxime:** 50mg/kg BD for less than 7 days old or less than 1200g, 50mg/kg TID for 7 days or older and more than 1200g IM or IV. **For meningitis double the dosages.**
- **Ceftazidime:** 50mg/kg BD for less than 7 days old or less than 1200g, 50mg/kg TID for 7 days or older and more than 1200g IM or IV.

- **Cefepime:** 50mg/kg BD or TID intramuscular or intravenous.
- **Meropenem:** 60 mg/kg/24 hr divided q 8 hr IV. Meningitis: 120 mg/kg/24 hr q 8 hr IV
- **Imipenem:** Postnatal age ≤7 days weight <1200 g: 20mg/kg q 18-24 hr IV or IM; weight >1200 g: 40mg/kgdivided q 12 hr IV or IM; postnatal age >7 days weight 1200-2000 g: 40 mg/kg q 12 hr IV or IM; weight >2000 g: 60 mg/kg q 8 hr IV or IM
- **Cloxacillin**: 25mg /kg BD for less than 7 days old or less than 1200g, 25mg/kg TID for 7 days or older and more than 1200g IM or IV.
- **Vancomycin**: 15mg/kg OD for less than 1200g, 15mg/kg BD for less than 7 days or less than 2000g. 15mg/kg TID for 7 days or older and more than 2000g. Each 5mg is diluted in 1ml of normal saline and the whole dosage infuses intravenously over 30 min.

Duration of treatment: Duration of antibiotic therapy in Neonatal Sepsis is shown in table 6.

Table 6: Duration of treatment

Diagnosis	Duration
Meningitis *(with or without positive blood/CSF culture)*	21 days
Blood ***culture positive*** *but* ***no meningitis***	14 days
Culture ***negative,*** *sepsis* ***screen positive*** *and clinical course* ***consistent with sepsis***	7-10 days
Culture and sepsis screen ***negative,*** *but* ***clinical*** *course* ***compatible with sepsis***	5- 7 days

7. **IVIG** 750mg/kg in critically sick preterm baby (1gr/kg in term baby) for a single dose improved survival.
8. **Granulocyte transfusion:** Indicated $1\text{-}2x10^9$ granulocyte/kg in a volume of 10-15ml/kg every 12-24hr in the followings conditions:
   - Lack of clinical improvement after 48hr of appropriate antibiotic therapy.
   - Neutrophil count < $3000/mm^3$ during first week of life and < $1000/mm^3$ after the age of one week.
   - Peripheral blood having > 70% immature PMN leukocytes or bone marrow having < 10% nucleated neutrophils.
9. **Corticosteroid:** Are indicated in gravely sick neonates with shock, sclerema and adrenal insufficiency.
10. **Exchange Blood Transfusion:** Double volume exchange transfusion maybe perform with cross-matched fresh whole blood as adjunctive therapy in septic neonates with sclerema.

Necrotizing Enterocolitis (NEC)

Introduction

NEC is characterized by various degrees of mucosal or transmural necrosis of the intestine. Although nearly 90% of all cases of NEC occur in preterm infants, the disease can occur in full-term neonates.

Essentials of diagnosis and classification

The modified Bell■s staging criteria classify NEC according to clinical and radiographic presentation:

Stage I: Suspected NEC

- Systemic signs: Nonspecific, including apnea, bradycardia, lethargy and temperature instability.
- Intestinal findings: Feeding intolerance, recurrent gastric residuals, and abdominal distention.
- Radiographic findings: Normal or non specific.

Stage I I: Proven NEC

- Systemic signs: Including stage I signs plus thrombocytopenia.
- Intestinal findings: Prominent abdominal distention, tenderness, bowel wall edema, absent bowel sounds and gross bloody stool .
- Radiographic findings: Peumatosis with or without portal gases.

Stage III: Advanced NEC

- Systemic signs: Respiratory or metabolic acidosis, respiratory failure, hypotension, decreased urine out put, shock, neutropenia and DIC.
- Intestinal findings: Tense discolored abdomen with spreading abdominal wall edema, induration and discoloration.
- Radiographic findings: Pneumoperitoneum.

Investigations

1-Blood: CBC, CRP, blood culture, electrolytes, ABG, coagulation study (PT, PTT, FDP and fibrinogen)

2- Stool culture

3- Imaging

- Abdominal x-ray: Abnormal bowel gas pattern, ileus, fixed sentinel loop of bowel are supportive findings.

Peumatosis intestinalis and portal venous gases are confirmatory findings.

- Abdominal sonography: Ultrasound can detect intermittent gas bubble in the liver parenchyma and portal venous system that are not detected on abdominal x-ray.

Management of Necrotizing Enterocolitis (NEC)

1. Stabilized ABC of resuscitation.
2. The baby should be kept NPO for 7 – 10 days and gastric aspiration by slow continuous suction is advised.
3. Intravenous line must be established to administer fluid, electrolytes and drugs.
4. If there is any umbilical catheter it should be removed.
5. The fluid requirements are markedly increased due to abdominal fluid sequestration, peritonitis and septic shock.
6. TPN is useful if indicated.
7. Antibiotics can be used. (See NNSepsis)
8. Metronidazol 15mg/kg is administered IV as a loading dose followed by 7.5 mg/kg every 12hrly for anaerobic infections.
9. Fresh frozen plasma 10ml/kg is recommended every alternate day to complement, humoral immune factors, coagulation factors, and to improved blood volume to correct shock.
10. EBM is started in small amounts if the following conditions have achieved:

- When abdominal distension disappears.
- Gastric aspirate is negligible.

- Intestinal peristalsis is audible.
- There is no occult blood in stool.

11. Surgery:

NEC should be managed under closed guidance and supervision of pediatric surgeon. Most cases of NEC can be managed conservatively; but surgery is indicated in the following conditions:

- Bowel perforation as evidenced by pneumoperitoneum or portal venous gas on plain abdominal radiography.
- Peritonitis as suggested by ascites, abdominal mass, induration and erythema of abdominal wall and localized abdominal rigidity.
- Full thickness necrosis of bowel wall with impending perforation as evidenced by dilated loop of intestine that remains unchanged in position and shape for more than 24hr on serial radiography.

Neonatal Hyperbilirubinemia (Jaundice)

Introduction

Yellow discoloration of sclera, skin and mucus membrane is called Jaundice which occurs due to the elevation of blood bilirubin level more than 5mg/dl in neonates.

Classification

1-Physiologic Jaundice which has the following criteria:

- General condition of neonate is well.
- Start between 24-72hr of age.
- Rise of serum bilirubin is less than 0.5mg/dl /hr or 5mg/dl/24hr.

- Peaks between 3 days and 5 days in term and 5–7 days in preterm.
- The maximum TSB is less than 95 percentile in hour-specific nomogram. Usually it level is < 15mg/dl.
- Direct bilirubin <2mg/dl
- Jaundice continues less than 10-14days of live in term and preterm respectively.

2-Pathologic Jaundice which has the following criteria:

- General condition of neonate is not usually well
- Occurrence of jaundice within 24hr of age is always pathologic.
- Rise of serum bilirubin over 0.5mg/dl /hr or more than 5mg/dl/24hr
- The maximum TSB is more than 95 percentile in hour-specific nomogram. Usually it level is > 15mg/dl.
- Direct bilirubin >2mg/dl
- Jaundice persists after 10-14days of live in term and preterm respectively.

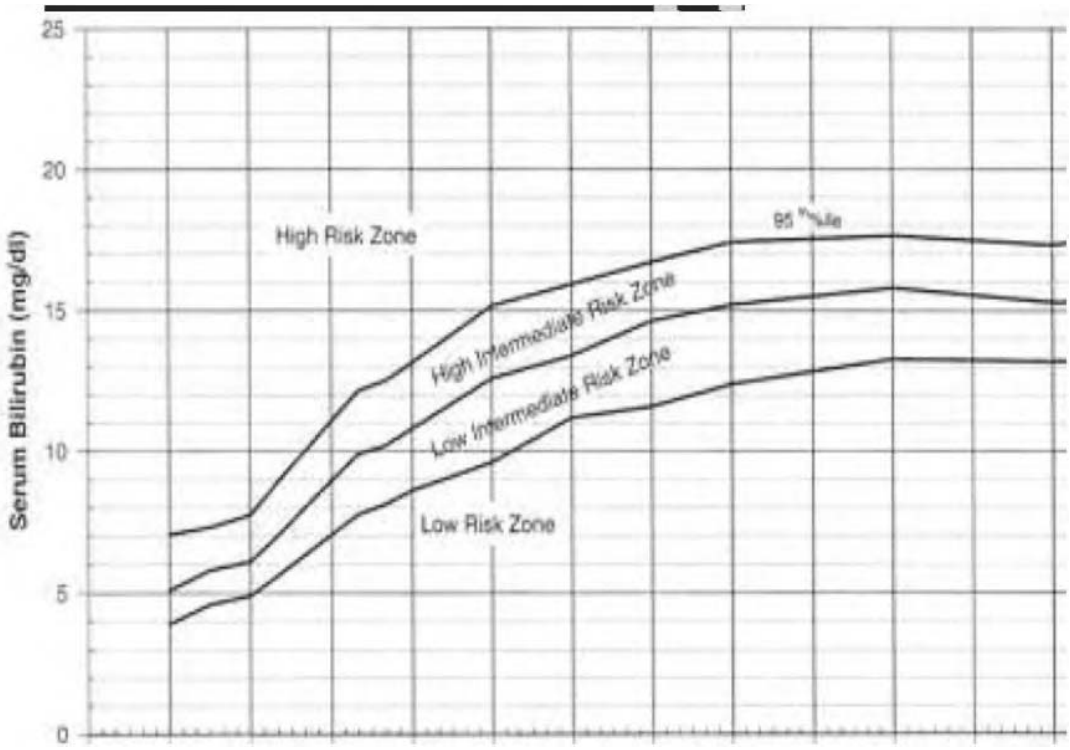


Figure 6: IIour specific nomogram

Investigation

- Total and direct bilirubin
- Mother and baby blood group
- Complete blood count
- Peripheral blood smears (for RBC shape and evidence of hemolysis)
- Reticulocyte count
- Direct Coombs test (if mother is "O" or Rh negative)
- G6PD assay

Sick infant with jaundice or prolonged jaundice (> 3 weeks)

- Complete blood count
- Urine examination and culture

- Evaluate for infection as indicated
- Urine for reducing substances
- Thyroid profile (T4, TSH)
- Evaluate for cholestasis (if direct bilirubin is elevated)

Management of Indirect Hyperbilirubinemia

Management of Physiologic Jaundice:

- Does not need any specific therapy.
- Frequent breast feeding should be continued.
- The baby must be watched closely for the severity of jaundice.
- Any change to the mother feeding should be inhibited, keeping the mothers on a specific diet (like tea and bread) which is a part of our culture; have no scientific basis.

Management of Pathologic Jaundice:

1. Supportive measures:

- Early and frequent breast feeding should be encouraged.
- Maintenance of adequate hydration is useful. If the patient need phototherapy, increase 12-24ml/kg/day to IV fluid.
- Hypoxia, hypothermia, hypoglycemia, acidosis and infection should be managed properly.
- Aspiration of cephalhematoma is indicated if bilirubin level is 18mg/dl or more in association with cephalhematoma.

2. **Phototherapy:** See table-7 and table-9 for indications of phototherapy.
3. **Exchange Blood Transfusion (EBT):** The indications are shown in table-8 and table-10. For this invasive procedure the following points should be undertaken.
   - The equipment and medication that need for resuscitation should be immediately available.
   - Equipment for umbilical vein catheterization (UVC or NGT No 6, 7 or 8) and disposable Exchange Transfusion tray are needed.
   - NGT for evacuating the stomach before beginning the transfusion and should be left in place to maintain gastric decompression, prevent regurgitation and aspiration of gastric contents.
   - The room of procedure should be warmed.
   - An EBT should take 45min-2hr duration.
   - 170ml/kg fresh (<72hrs old) and warmed (37ûC) blood is needed. The blood type are as follow:

   - For Rh incompatibility use O-ve blood or Rh negative with ABO compatible to the baby.
   - For ABO incompatibility use O with Rh compatible to the baby.
   - For Rh and ABO incompatibility O-ve blood should be used.
   - In the absence of incompatibility, ABO and Rh compatible to the baby blood is given.

   - EBT should be performed in Push-Pull method. Blood that used in each aliquots of push or pull is shown in table-11.

- After each 100-200 of EBT, administration of 1-2ml Calcium gluconate 10% maybe useful.

4. **Drugs:** The following drugs can be used.
   - **Phenobarbital** utility in NNJ is prophylactic rather than therapeutic; it takes 3-7 days to become effective. It can be given in a single dose of 10mg/kg intramuscular or 5mg/kg/day in 2 divided doses orally for 3 days in the following conditions:
     - Crigler-Najjar syndrome type 2.
     - Gilbert syndrome.
     - Early onset of jaundice due to any cause.
     - Cord serum Bilirubin > 2.5mg/dl.
     - Difficult or instrumental oxytocine-induced delivery with bruising and cephalhematom.
     - G-6-PD deficiency.
   5. **Albumin infusion** 0.5-1g/kg over 2hr before EBT maybe effective if bilirubin level is more than 20mg/dl and serum albumin level is < 3g/dl.
   6. **Intravenous Immune Globulin (IVIG)** decreased the need for EBT in Rh and ABO incompatibility. It is recommended if the TSB is rising despite intensive phototherapy or a TSB is within 2-3 mg/dl of exchange level. Dose 0.5-1gr/kg over 2hr.

Table-7: Guideline for Phototherapy in Neonates of Normal Birth Weight (≥2.5kg).

Age(hrs)	SBR or TSB (mg/dl)		
	Neonates of 35-37w plus risk factors*	Neonates of ≥38wk plus risk factors* or 35-37w and well	Neonates of ≥38wk and well
Birth	>4	>5	>6
12hrs	>6	>7	>9
24 hrs	>8	>9	>11
48 hrs	>11	>13	>15
72 hrs	>14	>15	>17
96 hrs	>15	>17	>20
≥5days	>15	>18	>23

Table-8: Guideline for Exchange Blood Transfusion in Neonates of Normal Birth Weight (≥2.5kg).

Age(hrs)	SBR or TSB (mg/dl)		
	Neonates of 35-37w plus risk factors*	Neonates of ≥38wk plus risk factors* or 35-37w and well	Neonates of ≥38wk and well
Birth	>12	>14	>16
12hrs	>13	>15	>17
24 hrs	>15	>16	>19
48 hrs	>17	>19	>22
72 hrs	>18	>21	>24
96 hrs	>19	>22	>25
≥5days	>19	>22	>25

* Risk Factors include perinatal asphyxia, significant lethargy, sepsis, acidosis, temperature instability, G6PD deficiency, Rh and ABO groups' incompatibility.

Table-9: Guideline for Phototherapy in Low Birth weight Neonates.

Age(hrs)	SBR or TSB (mg/dl)		
	Weight<1500g	Weight=1500-2000g	Weight >2000g
< 24	>4	>4	>5
25-48	>5	>7	>8
49-72	>7	>9	>12
> 72	>8	>10	>14

Table-10: Guideline for Exchange Blood Transfusion in Low Birth weight Neonates.

Age(hrs)	SBR or TSB(mg/dl)		
	Weight <1500g	Weight =1500 - 2000g	Weight >2000g
< 24	>10	>15	>16
25-48	>10	>15	>16
49-72	>10	>16	>17
> 72	>15	>17	>18

Table-11: Aliquots usually used Neonatal Exchange Transfusion

Neonates weight	Aliquots(ml)
> 3kg	20
2-3kg	15
1-2kg	10
850g-1kg	5
< 850g	1-3

Management of Direct Hyperbilirubinemia (Direct Bilirubin> 20% of TSB)

Direct hyperbilirubinemia has no need to Phototherapy and EBT, just treat the etiologic cause. In the cases of mixed type hyperbilirubinemia, direct bilirubin level should not subtract from TSB until it exceeds 50% of TSB, for the indications of phototherapy and EBT.

Neonatal Hypoglycemia

Definition: Blood glucose value of less than 40 mg/dl is called Neonatal Hypoglycemia.

Essentials of Diagnosis

1- Asymptomatic hypoglycemia: The blood glucose level is below 40mg/dl without clinical symptoms of hypoglycemia.
2- Symptomatic hypoglycemia: The diagnosis made on the basis of whipple's triads:

- Presence of signs and symptoms: Major (apnea, convulsion and coma) and minor (Jitteriness, irritability, tremor, lethargy, cyanotic spells and temperature instability).
- Low blood glucose
- Disappearance of symptoms when blood glucose level is normalized.

Management of Neonatal Hypoglycemia

1. Symptomatic Hypoglycemia:

- In a symptomatic neonate with seizure, give 5-10 ml/kg of 10% dextrose intravenously as a bolus. In the absence of seizure, bolus of 2ml/kg of 10 % dextrose IV is effective.
- After bolus dose, a continuous infusion of 10 % dextrose at a rate of 6-8mg/kg/min is maintained, preferably with the help of infusion pump.
- Blood glucose is monitored hourly till euglycemia and then 6 hourly.

A. If blood glucose is less than 40mg/dl the following steps should be considered.

- Repeat bolus and increase glucose infusion by 2mg/kg/min every 6 hour till a maximum infusion rate of 12mg/kg/min is reached or the blood glucose has crossed the level of 40mg/dl.

- Start the following drugs if hypoglycemia not resolved by day 7 or baby need > 12mg/kg/min glucose infusion:

- **Hydrocortisone sodium succinate** 5mg/kg IV every 12hr or Prednisolone 2mg/kg/day orally.
- **Glucagon** 100-300 mcg/kg/dose upto 3 doses IM and or Epinephrine, in baby with hypoglycemia due to erythroblastosis or maternal diabetes mellitus. These agents are not recommended for malnourished or preterm babies.
- **Diazoxide** 10-25 mg/kg/day in 3-4 divided doses slow IV or orally.

B. If blood glucose is ≥ 40mg/dl:

- Continue 6-8mg/kg/min glucose infusion for 24hr. If blood glucose is stable then wean infusion by 2mg/kg/min every 6hr and start oral feeds.
- Stop glucose infusion when baby is stable at 4mg/kg/min for 12hr.

2. Asymptomatic Hypoglycemia:

A. If blood glucose is < 20 mg/dl:

- Start infusion of 10% glucose at a rate of 6-8 mg/kg/min. Monitor blood glucose hourly till euglycemia and then 6 hourly.
- If not improved, increase glucose infusion by 2mg/kg/min every 6 hour till a maximum infusion rate of 12mg/kg/min is reached or the blood glucose has crossed the level of 40mg/dl.

- Start drugs that mentioned above if hypoglycemia not resolved by day 7 or baby need > 12mg/kg/min glucose infusion.

B. **If blood glucose is 20-40 mg/dl:**

- Sugar fortified oral feeds along with breast feeding.
- Monitor blood sugar.

1- If blood sugar is still < 40mg/dl;

- Start infusion of 10% glucose at a rate of 6-8 mg/kg/min. Monitor blood glucose hourly till euglycemia and then 6 hourly.
- If not improved, increase glucose infusion by 2mg/kg/min every 6 hour till a maximum infusion rate of 12mg/kg/min is reached or the blood glucose has crossed the level of 40mg/dl.
- Start drugs that mentioned above if hypoglycemia not resolved by day 7 or baby need > 12mg/kg/min glucose infusion.

2- If blood sugar is still ≥ 40mg/dl;

- Continue oral feeds. Monitor blood glucose for 48hr.
- Stop complementary feeds if blood glucose > 50mg/dl.

Neonatal Hypocalcemia

Definition: Hypocalcemia is defined as total serum calcium of less than 7 mg/dl (1.75mmol/L) or ionized calcium less than 4 mg/dL (1 mmol/L) in preterm infants and less than 8 mg/dL (2 mmol/L; total) or <1.2 mmol/L (ionic) in term neonates.

Classification

1-Early-onset hypocalcemia < 72hr of age

2-Late-onset hypocalcemia > 72hr of age. Usually in 5-10 days of live.

Essentials of Diagnosis

1-Asymptomatic: The blood calcium level is below 7mg/dl without clinical symptoms of hypocalcemia. Most cases are asymptomatic

2- Symptomatic: Jitteriness, irritability, apnea, tetani convulsion, stridor, cardiac arrhythmia

Management of Neonatal Hypocalcemia

A. Asymptomatic Hypocalcemia:

1. If baby is orally feed, 10% Calcium gluconate solution 2ml/kg (20mg elemental calcium/ml) every 6 hourly for 48hr can be given through oral rout.
2. For babies can't feed orally, give 10% Calcium gluconate solution 2ml/kg (maximum 10ml for full term and 5ml for preterm) intravenously after dilution with equal volume of 5% dextrose or distilled water and administered 1 ml/min (5-10min) every 6 hourly for 48hr or when ECG return to normal.
3. Monitor heart rate during IV administration of Calcium.
4. Calcium solution should never be given through umbilical vein or intramuscularly because of hepatic and tissue necrosis; it should not be added to solution containing sodium bicarbonate due to risk of precipitate of calcium carbonate.

B. Symptomatic (irritability, convulsion, apneic attack, tetany) Hypocalcemia:

1. Calcium gluconate solution 2ml/kg (maximum 10ml for full term and 5ml for preterm) should be given intravenously after dilution with equal volume of 5 % dextrose and administered 1 ml/min (5-10min). It is followed by 8 ml/kg/day as a constant infusion for at least 48hr after ECG had returned back to normal.
2. If hypocalcemia is unresponsive to calcium therapy, Magnesium sulfate should be administered 0.2ml/kg of 50% solution intramuscularly in 2 doses 12hr apart and be followed by maintenance oral dose of 0.2ml/kg of 50% solution once daily for 3 days.

C. Prophylactic management:

Baby at increased risk to developed hypocalcemia (preterm<1500gr, severe birth asphyxia, IDM) should received Calcium gluconate 10% solution 1ml/kg/dose intravenously after dilution with equal volume of 5% dextrose or distilled water should be administered 1 ml/min (5-10min) every 6 hourly for 48-72 or till oral feeds with supplements of calcium are started.

Neonatal Hypomagnesemia

Definition: Serum magnesium level less than 1mEq/l is called Neonatal Hypomagnesemia.

Management of Neonatal Hypomagnesemia

Magnesium sulfate should be administered 0.2ml/kg of 50% solution intramuscularly in 2 doses 12hr apart and be

followed by maintenance oral dose of 0.2ml/kg of 50% solution once daily for 3 days. It is recommended for unresponsive hypocalcemia, suspected or proved hypomagnesaemia (serum level < 1mEq/l).

Prematurity and Low Birth Weight

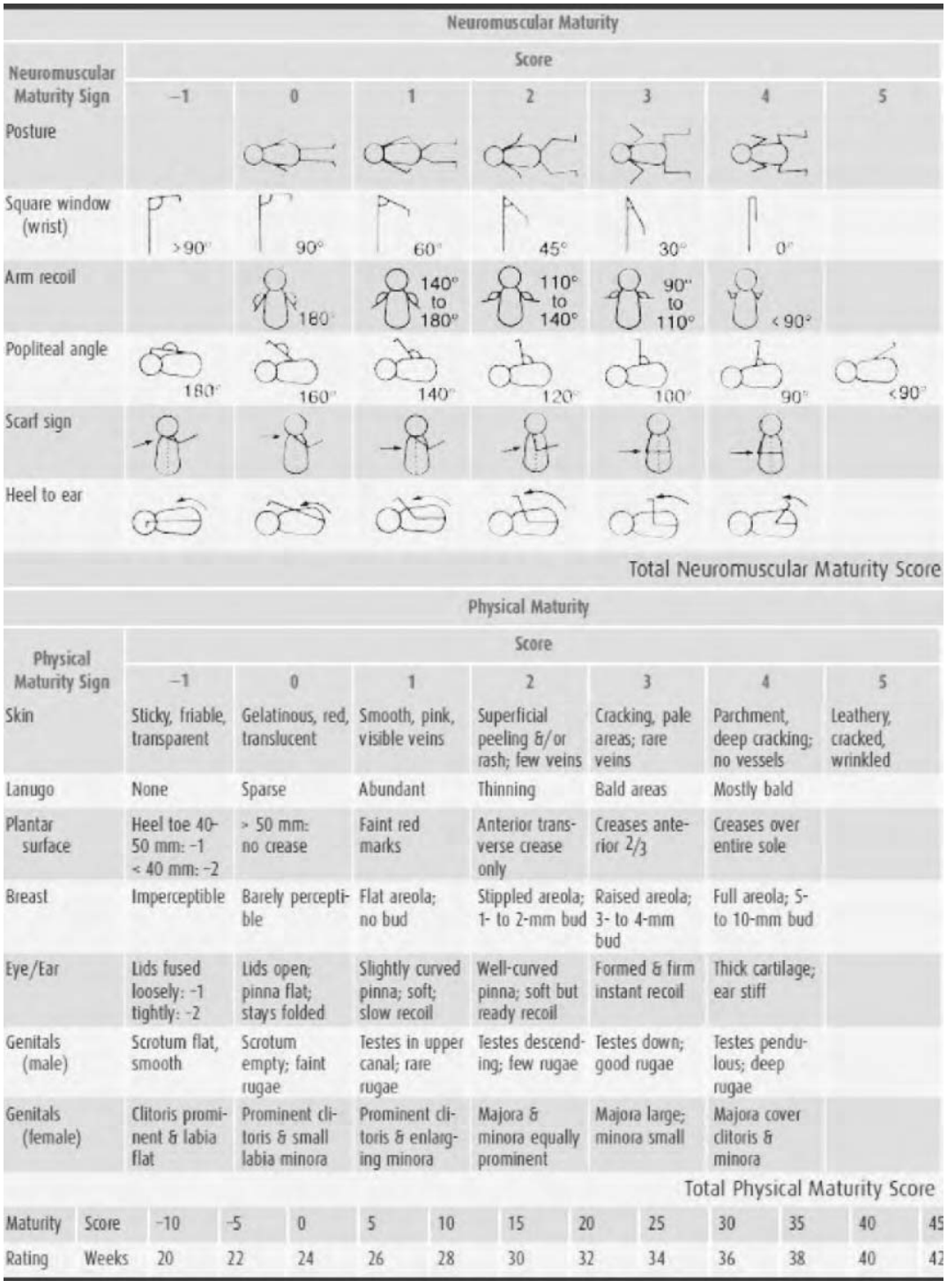
Introduction

Prematurity: Birth of neonate before 37 week of pregnancy or gestational age less than 37 week is called prematurity. Gestational age is assessed by the date of Last Menstrual Period (LMP), maternal ultrasound and New Ballard Score shown in figure 5. Prematurity classified as following:

- Mildly preterm: Gestational age of 34-36 w
- Moderate: Gestational age of 32- 34 w
- Very preterm: Gestational age of 28- 32w
- Extreme preterm: Gestational age less than 28w

Low Birth Weight (LBW): Low birth weight has been defined as a birth weight of less than 2,500 g. Birth weight is the baby weight within 24 hr of life. LBW is classified as following:

- Low birth weight: Birth weight less than 2,500 g.
- Very low birth weight (VLBW): Birth weight less than 1500 g.
- Extremely low birth weight (ELBW): Birth weight less than 1000 g.



Neuromuscular Maturity

Neuromuscular Maturity Sign	Score						
	−1	0	1	2	3	4	5
Posture							
Square window (wrist)	>90°	90°	60°	45°	30°	0°	
Arm recoil		180°	140° to 180°	110° to 140°	90° to 110°	< 90°	
Popliteal angle	180°	160°	140°	120°	100°	90°	<90°
Scarf sign							
Heel to ear							

Total Neuromuscular Maturity Score

Physical Maturity

Physical Maturity Sign	Score						
	−1	0	1	2	3	4	5
Skin	Sticky, friable, transparent	Gelatinous, red, translucent	Smooth, pink, visible veins	Superficial peeling &/or rash; few veins	Cracking, pale areas; rare veins	Parchment, deep cracking; no vessels	Leathery, cracked, wrinkled
Lanugo	None	Sparse	Abundant	Thinning	Bald areas	Mostly bald	
Plantar surface	Heel toe 40-50 mm: −1 < 40 mm: −2	> 50 mm: no crease	Faint red marks	Anterior transverse crease only	Creases anterior 2/3	Creases over entire sole	
Breast	Imperceptible	Barely perceptible	Flat areola; no bud	Stippled areola; 1- to 2-mm bud	Raised areola; 3- to 4-mm bud	Full areola; 5- to 10-mm bud	
Eye/Ear	Lids fused loosely: −1 tightly: −2	Lids open; pinna flat; stays folded	Slightly curved pinna; soft; slow recoil	Well-curved pinna; soft but ready recoil	Formed & firm instant recoil	Thick cartilage; ear stiff	
Genitals (male)	Scrotum flat, smooth	Scrotum empty; faint rugae	Testes in upper canal; rare rugae	Testes descending; few rugae	Testes down; good rugae	Testes pendulous; deep rugae	
Genitals (female)	Clitoris prominent & labia flat	Prominent clitoris & small labia minora	Prominent clitoris & enlarging minora	Majora & minora equally prominent	Majora large; minora small	Majora cover clitoris & minora	

Total Physical Maturity Score

Maturity Rating	Score	−10	−5	0	5	10	15	20	25	30	35	40	45
	Weeks	20	22	24	26	28	30	32	34	36	38	40	42

Figure 7: New Ballard score

Management of Prematurity and LBW

1. Most healthy near term or borderline preterm baby with a birth weight of 1800gr or more and gestational maturity of 35 or more can be managed at home.
2. ABC of resuscitation should be established if needed.
3. Keep the baby warm.
4. Incubation care are indicated if:
   - Birth weight is less than 1.8 kg.
   - Gestational age is less than 34wk.
   - Hypothermia
5. Indications for transferring out of incubator are all of the followings:
   - The baby who is feeding well.
   - Is reasonably active.
   - With a stable body temperature at an environmental temperature of < 30 C° for 12hr.

Table 12. Environmental temperature for the care of newborn

Environmental temperature						
Birth weight (kg)	37°C	36°C	35°C	34°C	33°C	32°C
Less than 1.0	For 1 day	After 1 day	After 2 weeks	After 3 weeks	After 4 weeks	After 6 weeks
1.0–1.49			For 10 days	After 10 days	After 3 weeks	After 5 weeks
1.5–1.99				For 10 days	After 10 days	After 4 weeks
2.0–2.5				For 2 days	After 2 days	After 3 weeks
More than 2.5					For 2 days	After 2 days

When facilities are enough it well be better to reach baby weight to 1600-1800gr otherwise mentioned criteria is irrespectively of body weight.

6. Manage infection properly. See NNSepsis.
7. Feeding:

A. Method of feeding:

- NPO: In the following conditions neonates should be kept NPO and intravenous infusion (10% glucose solution in babies > 1000gr and 5% glucose solution in babies < 1000gr until stabilization, totally for upto 3-4days) should be started :

  Critically ill, suspected or proved NEC, ileus, intestinal obstruction, recurrent seizure, esophageal atresia, RR>80/min, apneic attacks, birth weight less than 1200gr and severe birth asphyxia.
- Total Parenteral Nutrition (TPN) is considered if intravenous infusion exceeding 4-5 days in above conditions.
- Intravenous Glucose Infusion:

  Babies who are NPO, their all maintenance fluid should be administered as intravenous infusion of 10% glucose solution for babies > 1000gr and as 5% glucose solution in babies < 1000gr with Na and K. (see fluid therapy)

  For neonates who feed through NGT the amount of milk is subtracted from the maintenance fluid and the remainder is given as intravenous infusion that mentioned above.
- Feeding via NGT (Gavage feeding): Weak sucking, RR>70/min, stable neonates with gestational age between 30-34 weeks and birth weight less than 1500gr are the indications for gavage feeding.

  There is no need to burp tube feed babies.

Preparation for breast feeding should begin in all neonates irrespective of their gestation, by promoting rooting reflex and by putting them to breast for non-nutritive sucking before expressing the breast milk.

Table 13: Guidelines for the methods of feeding for low birth weight (LBW) neonates

Birth weight (g) **Gestation (week)** **Condition**	**< 1,200** **< 30**	**1,200–1,800** **30–34**	**> 1,800** **> 34**
Initial	Intravenous fluids; Try gavage feeds if not sick	Gavage	Breastfeeding; if unsatisfactory, give spoon or *paladai* feeds
After 1–3 days	Gavage	Spoon or *paladai* feeds	Breastfeeding
Later (1–3 weeks)	Try spoon or *paladai* feeding	Breastfeeding	Breastfeeding
After some more time (4–6 weeks)	Breastfeeding	Breastfeeding	Breastfeeding

B. Amount and frequency of feeding:

- As a general rules start 10-20ml/kg/day of Expressed Breast Milk (EBM) 2-3hrly then increases 10-20ml/kg/day every day, if the patient tolerate. The total daily amount should be gradually reached to 150-170 ml/kg/day. Or give EBM as follow:
  - Birth weight less than 1000gr; start EBM 1ml/2hrly and increase 1ml/2hrly every day.
  - Birth weight less than 2000gr; start EBM 2ml/2hrly and increase 2ml/2hrly every day.
  - Birth weight 2000gr or more; start EBM 2-5ml/kg/2hrly and increase 2-5ml/kg/2hrly every day.

C. Tolerance of feeds:

Increased abdominal girth by 2cm, abdominal distention, emesis and nonbilous gastric aspirate exceeds 50% of the last feeding are the early markers of feed intolerance.

Gastric bilious residuals, occult blood and reducing substances in stool are the signs of NEC, intestinal obstruction, meconium plug, meconium ileus and Hirschsprung disease.

A. Gastric nonbilious residual should be managed as follow:

- If aspirate volume <2-3mL or < 25% of last feed volume:

  Look for local cause, continue feeds and monitor.
- If aspirate volume 25-50% of last feed volume:
  - Reduce next feed volume (equal to the aspirate volume)
  - Or increase feeding interval to 3hrly.
  - Reposition the baby to elevate the head and upper body with right side posture.
  - Metochlopramide and Erythrocin are used to stimulate gastric emptying and decrease gastric residual volume.
  - Enema (1-2ml glycerine) can be given to treat constipation.
  - Monitor.
- If aspirate volume >50% of feed volume:
  - Withhold one or two feeds
  - Take an abdominal X-ray
  - Evaluate the cause and monitor.

B. Gastric bilious residual should be managed as follow:

- Making the neonate NPO.

- Decompress the gut via NGT.
- Evaluate the causes.

8. The maintenance fluid should be considered. (See Fluid Therapy)
9. **Nutritional supplements**
   - **Vit. K** 0.5-1mg intramuscularly or orally to all preterm babies are needed to prevent hemorrhagic disease of the newborn.
   - **Vitamin D (400 IU/day), vitamin B complex and zinc** (about 0.5mg/day) – usually in the form of multivitamin drops are administered after full enteral feeds are established (after 2 weeks of age) and continued till the post conceptional maturity of 40 weeks or body weight of 2000gr.
   - **Calcium** 160mg/kg/day and **Phosphorus** 80mg/kg/day are given by a suitable oral preparation to VLBW and receiving EBM to prevent osteopenia of prematurity. The supplement should be continued till the post conceptional maturity of 38 weeks or body weight of 2000gr. The IV preparation is started after 48-72hr of age.
   - **Iron** 2-4mg/kg/day supplement is provided when full enteral feeds are established and baby is gaining weight to prevent late iron deficiency anemia. Usually it starts on 6-8 weeks of ages and continued upto the age of one year.
   - **Human Milk Fortifier (HMF):** For all preterm (<32 weeks) or VLBW infants. It is started once they reach 150 mL/kg/day of enteral feeds in the dose

recommended by the manufacturer (4g [2 sachets] /100mL of expressed breast milk).

- **Preterm formula:** If HMF is unavailable or parents could not afford it, fortify EBM with preterm formula (0.4g/10 mL). Since calcium, phosphorus, and vitamin D intakes are low even after fortification with formula, supplement these nutrients additionally. Continue fortification till the infant reaches 40 weeks PMA or attains 2kg (whichever is later).

10. Treatment of complications

- Apnea:
  - Keep the baby warmth.
  - Make sure the position of the neonate does not compromise respiration.
  - Avoid triggers such as vigorous suctioning.
  - Olfactory stimulation (pleasant odor like vanillin) in incubator decreased apnea unresponsive to caffeine and Doxapram.
  - Gentle tactile stimulation is often adequate therapy for mild and intermittent episodes.
  - Neonates with recurrent and prolong apnea may require suctioning, positioning, bag and mask ventilation.
  - Oxygen should be administered in a low concentration to treat hypoxia.
  - **Caffeine citrate** 20mg/kg (10mg caffeine base/kg) intravenously over 30min or orally is followed after 24hr by a maintenance dose of 5-10mg/kg IV or orally in single or 2 divided doses.

- **Loading dose of Aminophylline** 5mg/kg IV or **Theophylline** orally should be followed by 2mg/kg every 8hrly through IV or oral routs.
  Therapy can be discontinued by post conceptional age of 35-37 weeks or if the baby is free of apnea for 5-7days.

- Hypoglycemia, Hypocalcemia, Hypothermia, Jaundice and Respiratory distress syndrome must be managed properly.(See related topics)

Respiratory Distress in Newborn

Introduction

Respiratory distress (RD) in newborn is the presence of two or more of the following features:

Respiratory rate greater than or equal to 60/min, chest retractions, nasal flaring and grunt. In a newborn presenting within the first 6 hours of birth with RD, one should consider the possibilities of Respiratory Distress Syndrome (RDS), Transient Tachypnea of the Newborn (TTN), Meconium Aspiration Syndrome (MAS) or congenital pneumonia.

1- Idiopathic Respiratory Distress syndrome Or Hyaline Membrane Disease (HMD)

Introduction

HMD is a clinical syndrome usually developed in preterm infant due to surfactant deficiency and characterized by tachypnea, retractions, grunting, cyanosis and specific radiographic feature.

Essentials of Diagnosis

- Respiratory distress usually within 6hr of birth in preterm neonate.
- Chest X-ray shows fine reticulogranular pattern, reduced lung volume, diffuse haziness (ground glass appearance) and air bronchograms (figure 8).

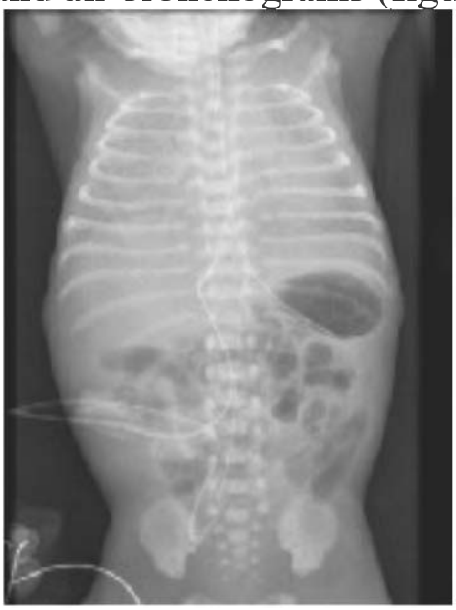


Figure 8. Ground glass appearance

- Shake Test: The test is based on the premise that if there is sufficient amounts of surfactant present in the amniotic fluid (or gastric aspirate taken within 30 min of birth), it would generate a stable foam layer at air-liquid interface when mixed with ethanol. Inadequate foam layer could suggest insufficient surfactant and an indirect support for the diagnosis of RDS.

Management of HMD

1. ABC of resuscitation should be done.
2. It is advisable to keep the baby NPO to prevent aspiration and start intravenous infusion, preferably through prepheral vein.

3. In infant of requiring prolong NPO and ventilation; NGT feeding or total parenteral nutrition is needed to prevent tissue catabolism.
4. **Oxygen** is administered through head box to relieve the cyanosis and keep arterial oxygen saturation between 90-95%. The ambient oxygen saturation should be 5-10 % higher than the cyanotic threshold (usually less than 50% oxygen concentrations is enough to prevent its toxicity).
5. **Warmth and Humidity:** The neonates should be nursed in a thermoneutral environment and with skin temperature around 36.5C°. Humidity must be maintained above 60 %.
6. **Antibiotics** are used routinely. (see NNSepsis)
7. **Vit E** may be recommended 100iu/kg/day intramuscularly for low birth weight baby receiving oxygen therapy to prevent oxygen toxicity (BPD and Retinopathy of Prematurity).
8. **Surfactant** is indicated in premature infant ≥ 28 weeks gestational age with RDS needing CPAP with ≥ 50% oxygen or assisted ventilation. It is administered intratracheally by instillation via endotracheal tube. The dose is 100mg/kg divided into four equal aliquots and is given during four different positions. Adequate oxygenation, ventilation, perfusion and monitoring should be established before starting treatment of HMD with surfactant.
9. **Nonsteroidal Anti-inflammatory Drugs (NSAID)** has been used for pharmacological closure of Patent Ductus Arteriosus. Commonly NSAID are:

- **Indomethacin** is administered in a dose 0.2mg/kg orally or preferably intravenously every 12 hourly for a total of three doses. Or
- **Ibuprofen** 10mg/kg stat followed by 5mg/kg at 24hr and 48hr can be given orally or intravenously.

10. **Continuous Positive Airway Pressure (CPAP)**.
11. **Assisted Ventilation**.

2-Transient Tachypnea of the Newborn (TTN)

Introduction and Essentials of Diagnosis

Transient tachypnea is most common after term cesarean delivery. It is characterized by the early onset of tachypnea, sometimes with retractions, or expiratory grunting and, occasionally, cyanosis that is relieved by minimal oxygen supplementation (<40%). Most infants recover rapidly, usually within 3 days. The chest generally sounds clear without crackles or wheeze.

Management of TTN

- Treatment is supportive.
- Oxygen therapy is useful.
- Inhaled salbutamol enhancing resolution of transient tachypnea of the newborn.

3-Meconium Aspiration Syndrome (MAS)

Introduction

Respiratory distress due to aspiration of meconium-stained amniotic fluid is called MAS.

Essentials of diagnosis

- Respiratory distress within first hrs of live; usually occurs in term or postterm infants.

- Meconium stained neonate: Meconium stained (green or yellow-green) skin, mucus membrane and nails.
- Chest X-ray: The typical chest radiograph is characterized by patchy infiltrates, coarse streaking of both lung fields, increased anteroposterior diameter, and flattening of the diaphragm.

Management of MAS

1-ABCD of resuscitation soon after birth as following:

- **Vigorous infant** (good respiratory, heart rate >100 beats/min and good muscle tone): Tracheal intubation to aspirate meconium **should not be attempted**; the mouth and nose may be suctioned with a bulb or suction catheter.
- **Not vigorous infant** (depressed respirations, has depressed muscle tone, and/or has a heart rate below 100 beat/min): Direct suctioning of the trachea soon after delivery is indicated before many respirations have occurred to reduce the chances of the baby developing meconium aspiration syndrome.

2-Administration of exogenous surfactant and/or iNO to infants with MAS and hypoxemic respiratory failure, or pulmonary hypertension requiring mechanical ventilation, decreases the need for ECMO.

3-Patients with MAS that is refractory to conventional mechanical ventilation may benefit from ECMO.

Hemorrhagic Disease of the Newborn (HDN) or Vitamin K Deficiency Bleeding (VKDB)

Introduction

Acquired coagulation disorder due to vit K deficiency in newborns is called HDN.

Classification

1- Early onset:
- Appears in the first 24hr of life.
- Interference of vit K metabolism due to maternal drugs is the cause.

2- Classic:
- Appears within 2-7 days of life.
- Decreased vit K storage, production and fewer amounts in breast milk is the cause.

3- Late onset:
- Appears after first w (usually in 2-6w).
- Malabsoption of vit K is the cause.

Essentials of Diagnosis

- Bleeding from deferent site of the body is the important manifestation.
- General appearance is usually well.
- Prolong PT and CT.
- Response to IV administration of vit K is enough for the confirmation.

Management of HDN

1-Vit K 1-5mg IV single dose is given for all types. For the treatment of late onset due to malabsoption, 50-100 µg every day orally or 2mg/month IM should be continue until improvement of underlying disease.

2- Fresh frozen plasma 10-20ml/kg is indicated for life-threatening hemorrhage or no improvement of PT despite vit K administration

3-Transfusion of whole blood for the management of shock.

Colic

Introduction and Essentials of diagnosis

Colic is a behavioral sign or symptom that begins in the first few weeks of life and peaks at age 2–3 months. An otherwise healthy infant seems to be in pain, cries for > 3 hours a day, for > 3 days a week, for > 3 weeks ("rule of threes").

Management of Colic

1. Parents may need to be educated about the developmental characteristics of crying behavior and made aware that crying increases normally into the second month and abates by the third to fourth month.

2. Parents may need reassurance, based on a complete history and physical examination, that the infant is not sick. Although these behaviors are stressful,

3. For parents to effectively soothe and comfort the infant, they need to understand the infant's cues. One should encourage a quiet environment without excessive handling. Rhythmic stimulation such as gentle swinging or rocking, soft music, drives in the car, or walks in the stroller may be helpful, especially if the parents are able to anticipate the onset of crying. Another approach is to change the feeding habits so that the infant is not rushed, has ample opportunity to burp, and if necessary can be fed more frequently so as to decrease gastric distention if that seems to be contributing to the problem.

4. Medications such as phenobarbital elixir and dicyclomine have been found to be somewhat helpful
5. A trial of ranitidine hydrochloride or other proton pump inhibitor might be of help if gastroesophageal reflux is contributing to the child's discomfort.
6. For colic that is refractory to behavioral management, a trial of changing the feedings, and eliminating cow's milk from the formula or from the mother's diet if she is nursing, may be indicated. The use of whey hydrolysate formulas for formula-fed infants has been suggested.

Perinatal Infection
TORCH

Introduction

TORCH is acronym of:
T= Toxoplasmosis
O= Other: syphilis, tuberculosis, malaria, HBV, HIV, Varicella , Echovirus , Coxackievirus, Parvovirus
R= Rubella
C= Cytomegalovirus
H= Herpes simplex
The presence of any three of the following alert the possibility of intrauterine infections:

- Maternal H/O infection
- IUGR
- Hepatosplenomegaly
- Jaundice
- Petechiae & purpura
- Meningoencephalitis

- Radiological abnormalities
- Raised IgM in cord blood

Toxoplasmosis

Introduction

Toxoplasma gandii is the etiologic agent which is an intracellular parasite.

Essential of diagnosis

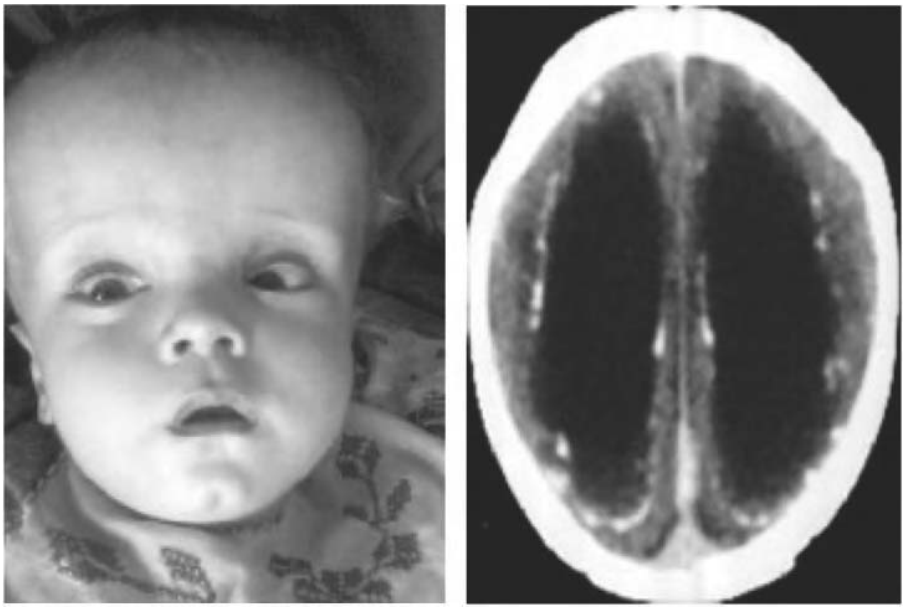
A-Clinical features

About 70-90% infants are asymptomatic. In symptomatic cases the classic tetrads are:

- Hydrocephalus
- Chorioretinitis
- Intracranial calcification
- Epilepsy

Within months or years following s/s maybe developed:

- Hepatosplenomegaly
- Jaundice
- Anemia
- Lymphadenopathy
- Cataract
- Deafness
- Delayed development



A B

Figure 9: Infant with hydrocephalus (A). Brain CT scan show dilated ventricles and calcification

B- Investigations

- Blood and body fluid culture for detection of Toxoplasma gandii
- Pathologic examination of amniotic fluid, placenta, neonates tissue and fluid
- Detection of Toxoplasma antigen in the blood
- PCR of blood, CSF, urine and amniotic fluid.
- Serologic tests: The most common test for the diagnosis.

Management of Toxoplasmosis

All neonates (symptomatic or asymptomatic) cases are treated with the following drugs for one year:

- Pyrimethamine 2mg/kg/day BID for 2day then 1mg/kg/day for 2-6months followed by 1mg/kg on Monday, Wednesday, and Friday.
- Sufadiazine : 100mg/kg/day BID

- Leucovorin 5-10mg/dayon Monday, Wednesday, and Friday.

Rubella

Introduction

Rubella caused by Rubella virus (an RNA virus) belongs to the family of Tagaviridae and genus Rubivirus.

Essential of diagnosis

A- Clinical features

1-Systemic transient manifestation: Low Birth Weight, hepatosplenomegaly, Purpura, meningoencephalitis, thrombocytopenia.

2- Systemic permanent manifestation:

- CHD
- Eyes defect
- CNS problem
- Deafness

3: Late onset anomalies: Deafness, CNS disease, DM, eye defect

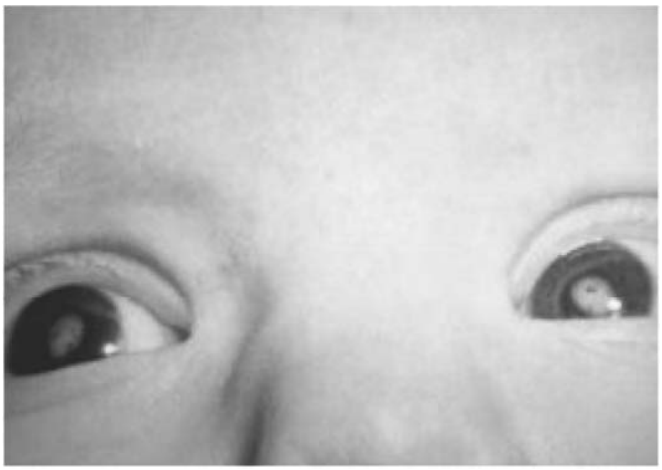


Figure 10: Congenital bilateral cataract

B- Investigations

- Culture of nasopharyngeal secretion, CSF and urine.
- CSF examination show increased WBC and protein.

Serologic tests: Increased rubella specific IgM is the mainstay of rubella diagnosis.

Management of Rubella

No specific treatment.

Cytomegalovirus (CMV)

Introduction

CMV is a DNA virus and a member of herpes virus group.

Essential of diagnosis

A- Clinical features

1-Asymptomatic (90%)

2-Symptomatic (Cytomegalic inclusion disease)

- LBW
- Prematurity
- Jaundice
- Anemia
- Hepatoslenomegaly
- Purpura
- Deafness
- Microcephaly
- Chorioretinitis
- Thrombocytopenia

B- Investigations

1- Laboratory

- Culture of urine or mouth secretion
- CMV PCR: to detect virus DNA in blood

- Serologic test detect IgM in blood but have no diagnostic value.

2- Radiography: Skull x-ray and CT scan can show periventricular calcification.

3-

Management of CMV

Gancyclovir 6mg/kg every 12hr IV for 6w is indicated in the following cases:

- CNS manifestations
- Chorioretinitis
- Seriously ill premature neonate

Herpes Simplex

Introduction

Herpes simplex virus is the etiologic agent which is an DNA virus belong to Herpesvirus family and have the following two form

- HSV-1: Involve face and area above the waist
- HSV-2: Involve genital and area below the waist

Essential of diagnosis

A-Clinical features: HSV has three clinical types.

1- Skin, Eye and mouth (SEM) disease: Vesicle and bull in skin, keratoconjuntivitis and chorioretinitis in eyes

Ulcers in mouth

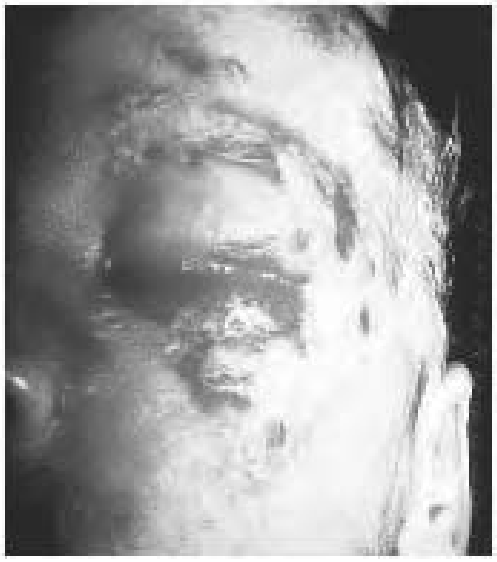


Figure 11: SEM disease

2- Disseminated disease: Fever, lethargy, apnea,shock like state, respiratory failure, lever failure, neutropenia, thrombocytopenia and DIC.

3- CNS disease: Convulsion, lethargy, irritability, bulging fontanela, tremor

B-Investigations

- Culture of skin lesion
- PCR: Detected DNA of HSV in blood and CSF
- Immunologic assay : Detect antigen of HSV in skin lesion
- Serologic test is not diagnostic
- Lumbar puncture
- Radiographic investigation

Management of Herpes Simplex

1- Antepartum: If H/O genital HSV infection of pregnant woman be present the following managements are advised:

- Acyclovir 400mg TID or 200mg five times a day for 7-10 days.
- Cesarean delivery for overt HSV infection

2- Neonatal treatment: Neonates born from mother with genital HSV infection should be treated by Acyclovir 60mg/kg/d IV TID in SEM for 14days and in disseminated and CNS infection for 21days.

Part 2
Pediatric Emergencies
Triage

Triage is the process of rapidly screening sick children soon after their arrival in hospital, in order to identify:

–Those with **emergency signs**, who require immediate emergency treatment;

– Those with **priority signs**, who should be given priority in the queue so that they can be assessed and treated without delay; and

– Non-urgent cases, which have neither emergency nor priority signs.

Emergency signs include:

■ Obstructed or absent breathing

■ Severe respiratory distress

■ Central cyanosis

■ Signs of shock (cold hands, capillary refill time longer than 3 s, high heart rate with weak pulse, and low or unmeasurable blood pressure)

■ Coma (or seriously reduced level of consciousness)

■ Convulsions

■ Signs of severe dehydration in a child with diarrhoea (lethargy, sunken eyes, very slow return after pinching the skin or any two of these).

Children with these signs require **immediate** emergency treatment to avert death.

The **priority signs** identify children who are at higher risk of dying.

These children should be **assessed without unnecessary delay**. If a child has one or more emergency signs, don't spend time looking for priority signs.

Summary of steps in emergency triage assessment and treatment

First check for **emergency signs** in three steps:

• **Step 1.** Check whether there is any airway or breathing problem; start immediate treatment to restore breathing. Manage the airway and give oxygen.

• **Step 2.** Quickly check whether the child is in shock or has diarrhoea with severe dehydration. Give oxygen and start IV fluid resuscitation. In trauma, if there is external bleeding, compress the wound to stop further blood loss.

• **Step 3.** Quickly determine whether the child is unconscious or convulsing.

Give IV glucose for hypoglycaemia and/or an anti convulsant for convulsing.

If emergency signs are found:

• Call for help from an experienced health professional if available, but do not delay starting treatment. Stay calm and work with other health workers who may be required to give the treatment, because a very sick child may need several treatments at once. The most experienced health professional should continue assessing the child to identify all underlying problems and prepare a treatment plan.

• Carry out emergency investigations (blood glucose, blood smear, haemoglobin [Hb]). Send blood for typing and cross-matching if the child is in shock, appears to be severely anaemic or is bleeding significantly.

• After giving emergency treatment, proceed immediately to assessing, diagnosing and treating the underlying problem.

If no emergency signs are found, check for priority signs:

■ **T**iny infant: any sick child aged < 2 months
■ **T**emperature: child is very hot
■ **T**rauma or other urgent surgical condition
■ **P**allor (severe)
■ **P**oisoning (history of)
■ **P**ain (severe)
■ **R**espiratory distress
■ **R**estless, continuously irritable or lethargic
■ **R**eferral (urgent)
■ **M**alnutrition: visible severe wasting
■ **O**edema of both feet
■ **B**urns (major)

The above can be remembered from the mnemonic **3TPR MOB**.

These children need prompt assessment (no waiting in the queue) to determine what further treatment is needed. Move a child with any priority sign to the front of the queue to be assessed next. If a child has trauma or other surgical problems, get surgical help where available.

Assessment of emergency and priority signs

■ **Assess the airway and breathing (A, B)**

Does the child's breathing appear to be obstructed? Look at the chest wall movement, and listen to breath sounds to determine whether there is poor air movement during breathing. Stridor indicates obstruction.

Is there central cyanosis*?* Determine whether there is bluish or purplish discoloration of the tongue and the inside of the mouth.

Is the child breathing? Look and listen to determine whether the child is breathing.

Is there severe respiratory distress? The breathing is very laboured, fast or gasping, with chest indrawing, nasal flaring, grunting or the use of auxiliary muscles for breathing (head nodding). Child is unable to feed because of respiratory distress and tires easily.

■ **Assess circulation (for shock) (C)**

Children in shock who require bolus fluid resuscitation are lethargic and have cold skin, prolonged capillary refill, fast weak pulse and hypotension.

Check whether the child's hand is cold. If so, determine whether the child is in shock.

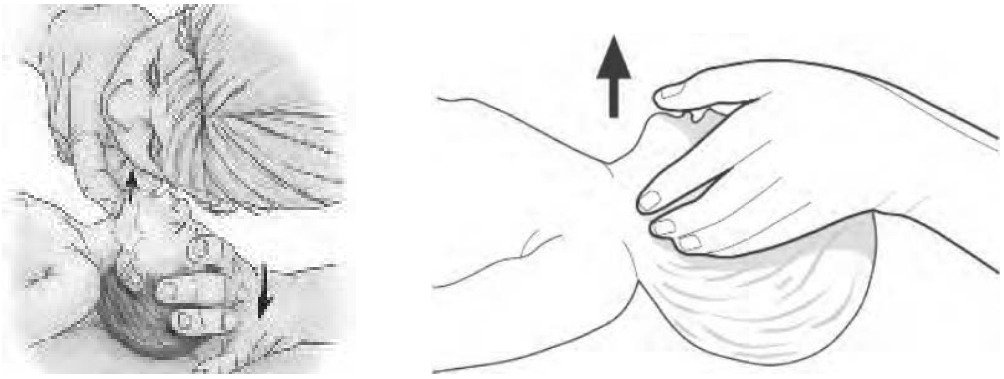
Check whether the capillary refill time is longer than 3 s. Apply pressure to whiten the nail of the thumb or the big toe for 5 s. Determine the time from the moment of release until total recovery of the pink colour.

If capillary refill is longer than 3 s, check the pulse. Is it weak and fast? If the radial pulse is strong and not obviously fast, the child is **not** in shock. If you cannot feel the radial pulse of an infant (< 1 year old), feel the brachial pulse or, if the infant is lying down, the femoral pulse. If you cannot feel the radial pulse of a child, feel the carotid.

Cardiopulmonary Resuscitation (CPR)

A (Airway Patency):

- If the child is unresponsive, he/she should be place on a hard surface in supine position.
- Place the head in the sniffing position. The neck should be slightly flexed and the head gently extended so as to bring the face forward. In infant and children younger than 8 years, the relatively large occipitus causes significant neck flexion and poor airway position. This is relieved by placing a towel rolled under the shoulder.
- If airway obstruction is present, quickly inspect the pharynx. Clear secretion or vomitus by brief suction.
- Maintain the airway with backward head tilt, chin left or forward jaw thrust(figure 6).



A B

Figure 12: A-Head tilt and chin left maneuver. B-Jaw Thrust maneuver

B (Breathing):

- If adequate spontaneous ventilation does not resume, give 1 breath every 3-5 sec by bag and mask or mouth.
- **Oxygen** 100% should be given 6-10 lit/min.

C (Circulation):

Start cardiac compression over the lower sternum if:

- Pulse is not palpable.
- Pulse is less than 60/min with poor perfusion.

Place the patient on a firm surface and depress the lower sternum one third the dept of the chest (4cm in infant and 5cm in children) 80-100/min. Compression-ventilation rate for one rescuer is 30:2 and for two rescuer 15:2. Children ≤ 8 yr get more benefit from the second rate. Cardiac compression performed as follow:

- Newborns or infants less than 1 year: Tow thumb technique in which the hands encircle the chest.
- Children 1-8 y old: The heel of one hand.
- Children more than 8y: The tow- handed technique

D (Drugs):

- **Adrenaline** 0.01mg/kg (0.1ml/kg of 1:10000) is indicated intravenously if there is no pulse. It can be repeated every 3 min or increase to 0.1-0.2mg/kg, if the first dose is ineffective.
- **Atropine** 0.02mg/kg IV can be used for bradycardia. Repeat once if needed.
- **Calcium gluconate 10%** for Hypocalcemia. 1-2ml/kg of Calcium gluconate 10% should be diluted with equal amount of sterile water or 5 % dextrose and administered intravenously over 5-10 min. Parenteral calcium (8ml/kg/day) should be gradually tapered over 2 day and oral calcium be started for 2 days.
- **Glucose 10%** 2ml/kg IV for Hypoglycemia.

- **Sodium bicarbonate** 1-2mEq/kg diluted with double volume of sterile water and administered slowly through intravenous for acidosis.
- **N/S or Ringer lactate** 20ml/kg if shock is developed.
- **Amiodarone, Procainamide, Lidocaine** and **DC** shock are useful for ventricular tachycardia and ventricular fibrillation.

Pediatric Bradycardia

Introduction

By definition, a child is *bradycardic* when the heart rate is slower than the normal range for age. A sinus rate <90 beats/min in neonates and <60 beats/min in older children is considered to be sinus bradycardia. A clinically significant bradycardia occurs when the heart rate is slow and there are signs of systemic hypoperfusion (i.e., pallor, altered mental status, hypotension and acidosis).

Management of Pediatric Bradycardia

Pediatric Bradycardia with a pulse and poor perfusion should be managed as follow:

1. Identify and treat underlying cause.
2. Maintain patent airway; assist breathing as necessary.
3. Oxygen must be given.
4. Cardiac monitor to identify rhythm; monitor blood pressure and oximetry.
5. IV/IO (Intravenous or Intraosseous) access should be established.
6. 12-lead ECG is useful if available; don't delay therapy.

Assess cardiopulmonary compromise (hypotension, acutely altered mental status and signs of shock)

1. If HR is less then 60/min with poor perfusion despite oxygenation and ventilation then do cardiac compression as described under CPR.
2. For persistent bradycardia despite above management the following drugs are given:

   A: Epinephrine (Adrenalin) 0.01mg/kg (0.1ml/kg of 1:10000 concentration) IV or IO. Repeat every 3-5 min. if IV or IO access not available but endotracheal tube (ET) in place, may give ET dose 0.1mg/kg or 0.1ml/kg of 1:1000 solution).

   B: Atropine IV/IO 0.02mg. May be repeated once. Minimum dose is 0.1mg and maximum dose is 0.5mg.

3. If pulseless arrest develops, go to cardiac arrest management.

Pediatric Tachycardia

Introduction

Faster heart rate than the normal range for age is called tachycardia.

Management of Pediatric Tachycardia

Pediatric Tachycardia with a pulse and poor perfusion should be managed as follow:

1. Identify and treat underlying cause.
2. Maintain patent airway; assist breathing as necessary.
3. Oxygen must be given.
4. Cardiac monitor to identify rhythm; monitor blood pressure and oximetry.
5. IV/IO (Intravenous or Intraosseous) access should be established.

6. 12-lead ECG is useful if available; don't delay therapy.

Evaluate QRS duration

A. If QRS > 0.09 sec then Ventricular Tachycardia is possible.

1. For Ventricular Tachycardia with cardiopulmonary compromise (hypotension, acutely altered mental status and signs of shock) Synchronized Cardioversion is effective. Begin with 0.5-1 J/kg; if not respond increase to 2 J/kg. Sedate if needed, but don't delay cardioversion.

2. For Ventricular Tachycardia without cardiopulmonary compromise consider one of the following drugs; if rhythm is regular and QRS monomorphic:

a. Adenosine IV/IO first dose 0.1mg/kg rapid bolus (maximum 6mg) and second dose 0.2mg rapid bolus (maximum 12mg).

b. Amiodarone IV/IO 5mg/kg over 20-60 minute.

c. Procainamide IV/IO 15mg/kg over 30-60 minute.

B. If QRS ≤ 0.09 sec then Sinus Tachycardia or Supraventricular Tachycardia is probable.

1. Sinus Tachycardia has the following features:

- Compatible history consistent with known cause.
- P waves present/normal.
- Variable R-R; constant PR interval.
- Infants; HR usually < 220/min.
- Children; HR usually < 180/min.

For Sinus Tachycardia search and treat the cause.

2. Supraventricular Tachycardia has the following features:

- Compatible history is nonspecific. History of abrupt rate change.

- P waves absent/abnormal.
- HR not Variable.
- Infants; HR usually ≥ 220/min.
- Children; HR usually ≥ 180/min.

For Supraventricular Tachycardia:

- If IV/IO access present give Adenosine.
- If IV/IO access not available or Adenosine is ineffective, do synchronized cardioversion.

Pediatric Cardiac Arrest

1. Start CPR; initial step is to restore ventilation and oxygenation (See A and B parts of resuscitation). If the child is pulseless, chest compression should be initiated (See part C of resuscitation).
2. Give oxygen.
3. Attach monitor/defibrillator.

Evaluate the rhythm

4. If Ventricular Fibrillation (VF) or Ventricular Tachycardia (VT) be observed; managed as follow:

- Shock (Defibrillation) first 2 J/kg, second 4 J/kg and subsequent shocks ≥ 4 J/kg, maximum 10 J/kg.
- After each shock; CPR must be performed for 2 minutes.
- IV/IO access.

Evaluate the rhythm

If VF or VT remains; do the following steps:

- Shock should be done.
- CPR for 2 minutes.

- Epinephrine is administered every 3-5 min with CPR. Defibrillations can be alternated with epinephrine.
- Amiodarone IV/IO 5mg/kg over 20-60 minute.
- Treat reversible causes (hypovolemia, hypoxia, acidosis, hypoglycemia, hypothermia, hypo/hyperkalemia, cardiac temponade, tension pneumothorax etc)

5. Asystole without VF/VT managed with:
   - CPR for 2minute.
   - Epinephrine is administered every 3-5 min followed by CPR in each cycle.
   6. Treat reversible causes (hypovolemia, hypoxia, acidosis, hypoglycemia, hypothermia, hypo/hyperkalemia, cardiac temponade, tension pneumothorax, etc)

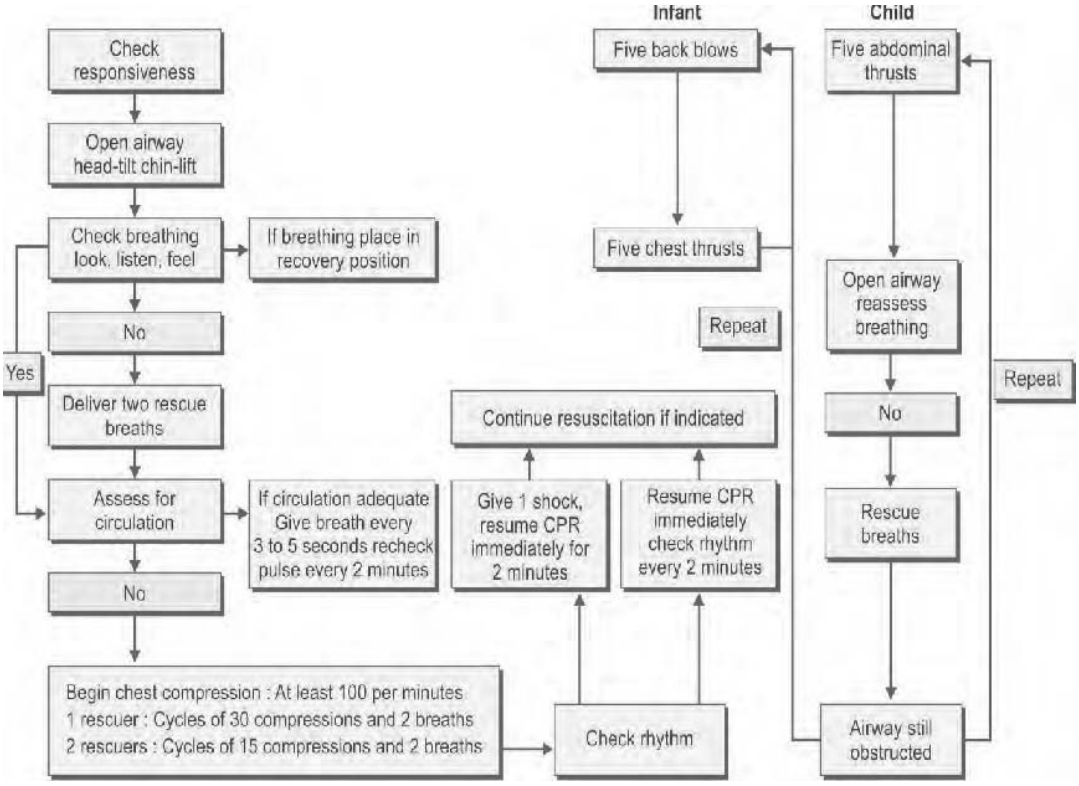


Figure 13: Summary of resuscitation

Shock

Introduction

Shock is an acute process characterized by the body's inability to deliver adequate oxygen to meet the metabolic demands of vital organs and tissues.

Classification: Five major types of shock are as follow;

1-Hypovolemic shock: The most common cause of shock in children worldwide; is most frequently caused by diarrhea, vomiting, or hemorrhage.

2-**Cardiogenic shock** is seen in patients with either congenital heart disease or with congenital or acquired cardiomyopathies, including acute myocarditis.

3-**Obstructive shock** is caused due to tamponade, tension pneumothorax, pulmonary embolism, and ductus-dependent congenital heart lesions.

4-**Distributive shock** is caused by inadequate vasomotor tone, which leads to capillary leak and maldistribution of fluid into the interstitium.

5-Septic shock is often discussed synonymously with distributive shock, but the septic process usually involves a more complex interaction of distributive, hypovolemic, and cardiogenic shock.

Essentials of Diagnosis

Lethargic or unconscious and cold hands plus either:

- Slow capillary refill (longer than 3 sec) or
- Weak fast pulse

Management of Shock

1. **Stabilize** ABC (Air way, Breathing, Circulation) of resuscitation. (See resuscitation)
2. **Give O_2** (O_2 saturation should be keep 95-99 %).
3. **Management of specific types**:

A: Hypovolumic and Septic:

- **I.V Fluid** (N/S or Ringer lactate) 20 ml /kg is administered as a bolus over 5- 20 min. IF pulse, capillary refill, urine output and sensorium not improved repeat fluid for up to 60- 80 ml/ kg in 1- 2 hours.
- **Dopamine or Dobutamine** are effective vasopressor drugs that given in fluid resistant shock. Dosage is 3-10 µg/kg/min by IV infusion over 60 minutes (1cc Dopamine add with 100cc 1/5 N/S + D/W 5% then 1drop /kg /min of this solution should be used until improvement of shock).
- **Epinephrine (Adrenalin)** as IV infusion of 0.05- 0.3 µg /kg /min is administered for Dopamine or Dobutamine resistant cold shock.
- **Norepinephrine** as IV infusion of 0.05- 0.1 µg /kg /min is administered for Dopamine or Dobutamine resistant warmth **shock**.
- **Hydrocortisone** replacement may be beneficial in pediatric shock. In the cases of Adrenaline resistant septic shock (after 60 min) give Hydrocortisone 50mg/kg bolus then 50mg /kg /day IV.
- In catecholamine resistant cold or warmth shock; fluid and Epinephrine or Norepinephrine should be titrated (increased until response) respectively.

B: Cardiogenic shock:

- **Dopamine or/and Dobutamine** are the drugs of choice for cardiogenic shock. Dosage is 3-10 µg/kg/min by IV infusion (1cc Dopamine add with 100cc 1/5 N/S + D/W 5% then 1drop /kg /min of this solution should be used until improvement of pulse, capillary refill, urine output and sensorium).
- **Milrinone** 0.25-0.75 mg/kg/min intravenously is added for Dopamine or Dobutamine resistant cardiogenic shock.
- **Diuretics** can be administered to reduce pulmonary edema.

C: Antibiotics for septic shock: Early administration of antibiotics is a key factor in improving outcome. Antibiotics are given as follow:

- **Infants less than 2month: Ampicillin** (200mg/kg/day) + third generation cephalosporin (Ceftriaxone 100mg/ kg/ day or Cefotaxime 150mg / kg / day). For dosage of neonates see NNSepsis.
- **Infants > 2 month old and children**: third generation cephalosporin as above dosage.

In suspected meningitis or staphylococcal infection add vancomycin (60 mg/kg/day 8-12hrly IV infusion over 30 minutes). Each 5mg of vancomycin is diluted in 1ml of normal saline

D: **Anaphylactic shock:**

- **Adrenalin** 0.01mg/kg (0.01ml of 1:1000 solution) IM is the first line treatment for anaphylactic shock. Repeat every 10 minutes up to the dose of 0.03mg/kg.

- **Fluid:** As mentioned above.
- **Antihistaminics**:

a. **Cetrizine** 0.25mg/kg PO, **Diphenhydramine or phenerami**ne (Avil) 1-2mg/kg IM for 3days.

b. **Ranitidine** 1mg /kg / IV BD for 3days for Anaphylaxis. 10 – 5 mg /kg /day for stress ulcer.

c. **Corticosteroid:** Give **Methylprednisolon** (Solu-Medrol (IV) 1-2 mg/kg up to 125 mg IV Depo-Medrol (IM) 1 mg/kg up to 80 mg IM) or **Hydrocortisone** 5mg /kg / IV every 6 hours for 2- 3 day.

4. Blood Transfusion (Fresh) If Hb is < 10 g/d or bleeding is present.

5. Vit K can be used if bleeding is observed. For infants 1mg, for children 2-3mg, for Adolescents and adults 5-10mg is administered once intravenously.

6. Treat Hypoglycemia and Hypocalcemia if present or suspected:

- **Hypoglycemia** (Blood glucose less than 50mg/dl): Give 2ml/kg of Dextrose 10%, followed by a continuous infusion of glucose at 6-8mg/kg/min (0.06-0.08ml/kg/min of dextrose 10%). If hypoglycemic seizure is observed the loading dose of Dextrose 10% should be 4ml/kg.
- **Hypocalcemia** (Serum calcium less than 7mg/dl)**:** 1-2ml/kg of Calcium gluconate 10% should be diluted with equal amount of sterile water or 5 % dextrose and administered intravenously over 5-10 min. Parenteral calcium (8ml/kg/day) should be gradually tapered over 2 day and oral calcium be started for 2 days.

Coma

Introduction

The state of coma requires that the patient be unresponsive, even to noxious stimuli.

Essentials of Diagnosis

- Reduction or alteration in cognitive and affective mental functioning and in arousability or attentiveness.
- Acute onset.

Table 14: Glasgow coma scale

COMA SCALES			
Glasgow Coma Scale		Modified Coma Scale for Infants	
Activity	Best Response	Activity	Best Response
EYE OPENING			
Spontaneous	4	Spontaneous	4
To speech	3	To speech	3
To pain	2	To pain	2
None	1	None	1
VERBAL			
Oriented	5	Coo/babbles	5
Confused	4	Irritable	4
Inappropriate words	3	Cries to pain	3
Nonspecific sounds	2	Moans to pain	2
None	1	None	1
MOTOR			
Follows commands	6	Normal spontaneous movements	6
Localizes pain	5	Withdraws to touch	5
Withdraws to pain	4	Withdraws to pain	4
Abnormal flexion	3	Abnormal flexion	3
Abnormal extension	2	Abnormal extension	2
None	1	None	1

Data from Jennet B, Teasdale G. Aspects of coma after severe head injury. *Lancet.* 1977;1:878; and James HE. Neurologic evaluation and support in the child with an acute brain insult. *Pediatr Ann.* 1986;15:16.

A score less than 8 usually indicates CNS depression requiring positive-pressure ventilation.

Investigations

Investigations needed for comatose children are shown in table 15.

Table 15: Investigations for comatose child

Sample	Patients	Indications
Blood		
Complete blood count	All	Infection
Blood sugar	All	Hypoglycemia/hyperglycemia
Serum electrolytes	All	Metabolic disturbances
Calcium, phosphate	All	Metabolic disturbances
Liver function tests	All	Hepatic encephalopathy, metabolic, toxic, infections
Renal function tests	All	Uremic encephalopathy, infections, metabolic, toxic
Arterial blood gas	All	Acidosis
Ammonia	All	Metabolic, toxic, infections
Lactate	All	Infections, metabolic, toxic
Culture	Specific	Infections
Peripheral blood film for malaria	Specific	Cerebral malaria
Viral markers	Specific	Viral encephalitis
Leptospira serology	Specific	Leptospirosis
Weil Felix test	Specific	Rickettsial infections
Widal test	Specific	Enteric encephalopathy
Toxicology screen	Specific	Poisoning
Enzyme studies	Specific	Metabolic, neurodegenerative, syndromic
Urine		
Culture	Specific	Infections
Reducing substances	Specific	Metabolic disorders
Toxicology screen	Specific	Poisoning
Ketones	Specific	Metabolic, diabetic ketoacidosis
Cerebrospinal fluid		
Cell count, protein, sugar, culture	Specific	Infections
Viral markers	Specific	Viral encephalitis
Tuberculosis workup	Specific	Tubercular meningitis
Fungal culture, serology, galactomannan enzyme immunoassay	Specific	Infections
Neuroimaging		
CT head (Contrast/plain)	All; except documented uncomplicated metabolic causes such as diabetic ketoacidosis, hypoglycemia	Trauma, infections, bleed, tumor, vascular cause
CT angiography	Specific	Vascular cause
MRI	Specific	Infections, acute disseminated encephalomyelitis, acute necrotizing encephalopathy of childhood, neurometabolic disorders
Electrophysiological studies		
Conventional electroencephalogram	Specific	Seizure, nonconvulsive status, infections
Continuous electroencephalogram	Specific	Seizures, comatose children on paralysis

Management of Coma

1. Stabilize ABC of Resuscitation.(see resuscitation)
2. O_2 Therapy.
3. Position should be turned every 2 hour.
4. Repeated suction of air way is useful.
5. Nutrition: If there is no danger of aspiration due to convulsions and fast breathing; feed through NGT otherwise keep NPO and maintain IV fluid therapy.
6. DO LP if indicated.
7. Find underlying cause and treat it.
8. Determine Glasgow coma scale.
9. Management of unknown coma : All of the followings drugs should be given:
   - **A. D/W 10%** 5ml/kg or D/W 25% 1-2 ml/kg IV.
   - **B. Antibiotic** as for meningitis.
   - **C. Naloxan** 0.1mg/kg or 0.4-2mg IV.
   - **D. Quinine DHC** in high risk area of malaria as describe for severe malaria.
   - **E. Anti viral: Acyclovir** 30-45 mg/kg/day is administered in 3 divided doses for 10 days; if Encephalitis is suspected.

10: For suspected ICH treat as the following:

A. Intubation and Mechanical ventilation to prevent aspiration and induce hyperventilation for the management of cerebral edema.

C. Sedation, Analgesia and elevate head of the bed (15- 30 degree).

D. Manittol 5 cc /kg IV over 30 minutes is useful. It may be repeated 6-8 hourly for 6 doses, if needed. Or/and

E. 3% Saline 0.1-1ml/kg/hr intravenously.

F. Lasix 1 mg/kg/day IV can be added.

G. Phenobarbital with a loading dose of 5-10 mg/kg over 30 minutes followed by 5mg/kg every hour for 3 doses and then infusion of 1mg/kg/hr is useful for refractory cases.

Signs of suspected ICH are:

- Decreased level of conscious.
- Repeated convulsions.
- Vomiting.
- Cardiac arrhythmia.
- Focal Neurological deficit.
- Headache.
- Papilledema (Old child).

Respiratory Failure

Definition

Respiratory failure is defined as inability of the lungs to provide sufficient oxygen (hypoxic respiratory failure) or remove carbon dioxide (ventilatory failure) to meet metabolic demands.

Essentials of Diagnosis

- Clinical manifestations: Nasal flaring, retractions, tachypnea, wheezing, stridor, grunting, paradoxical

respirations, shallow or slow respirations, abnormal respiratory patterns and apnea.

- PaO2 <60 torr with breathing of room air and PaCO2 >50 torr resulting in acidosis.

Management of Respiratory Failure

Oxygen Therapy

A good clinical rule is to administer sufficient oxygen to keep PaO_2 in the range of 60–90 mm Hg and oxygen saturation more than 90%.Oxygen is humidified in a bubble humidifier and delivered via nasal prongs inserted in to the nares.

Airway Adjuncts

Maintenance of a patent airway is a critical step in maintaining adequate oxygenation and ventilation. Artificial oropharyngeal or nasopharyngeal airways are useful devices.

Inhaled Gases

Helium-oxygen mixture (*heliox*) is useful in overcoming airway obstruction and improving ventilation.

Nitric oxide (NO) is a powerful inhaled pulmonary vasodilator. Its use may improve pulmonary blood flow and V_ /Q_ mismatch in patients with diseases that elevate pulmonary vascular resistance.

Positive-Pressure Respiratory Support

Noninvasive positive-pressure respiratory support is useful in treating both hypoxemic and hypoventilatory respiratory failure.

1- Bag & mask ventilation

2- Continuous Positive Airway Pressure (CPAP): A high-flow nasal cannula delivers gas flow at 4-16 L/min, providing significant continuous positive airway pressure (CPAP).

3-Bilevel Positive Airway Pressure (BiPAP) machines provide positive airway pressure during exhalation and additional positive pressure during inspiration.

4- Endotracheal Intubation and Mechanical Ventilation

When hypoxemia or significant hypoventilation persists despite the interventions already described, tracheal intubation and mechanical ventilation are indicated.

Anaphylaxis

Definition

Anaphylaxis is defined as a serious allergic reaction that is rapid in onset and may cause death.

Essentials of Diagnosis

Anaphylaxis is highly likely when any *1* of the following 3 criteria is fulfilled:

1. Acute onset of an illness (minutes to several hours) with involvement of the skin and/or mucosal tissue (e.g., *generalized* hives, pruritus or flushing, swollen lips/tongue/uvula)

AND AT LEAST 1 OF THE FOLLOWING:

a. Respiratory compromise (e.g., dyspnea, wheeze/ bronchospasm, stridor, reduced peak PEF, hypoxemia)

b. Reduced BP or associated symptoms of end-organ dysfunction (e.g., hypotonia, syncope, incontinence)

2. Two or more of the following that occur rapidly after exposure *to a likely allergen for that patient* (minutes to several hours):
a. Involvement of the skin/mucosal tissue (e.g., *generalized* hives, itch/flush, swollen lips/tongue/uvula)
b. Respiratory compromise (e.g., dyspnea, wheeze/ bronchospasm, stridor, reduced PEF, hypoxemia)
c. Reduced BP or associated symptoms (e.g., hypotonia, syncope, incontinence)
d. *Persistent* gastrointestinal symptoms (e.g., crampy abdominal pain, vomiting)
3. Reduced BP following exposure to *known allergen for that patient* (minutes to several hours):
a. Infants and children: low systolic BP (age-specific) or >30% drop in systolic BP
b. Adults: systolic BP <90 mm Hg or >30% drop from patient's baseline.

Management of Anaphylaxis

1. ABC of resuscitation should be stabilized.
2. **Adrenalin** 0.01mg/kg (0.01ml of 1:1000 solution) IM is the first line treatment for anaphylactic shock**.** Repeat every 10 minutes up to the dose of 0.03mg/kg.
3. **Fluid for shock:** As mentioned under shock .
4. **Antihistaminics**:
   - **Cetirizine** 0.25mg/kg PO, **Diphenhydramine or phenerami**ne (Avil) 1-2mg/kg IM for 3days.
   - **Ranitidine** 1mg /kg / IV BD for 3days.

5.**Corticosteroid:** Give **Methylprednisolon** (Solu-Medrol (IV) 1-2 mg/kg up to 125 mg IV Depo-Medrol (IM) 1 mg/kg

up to 80 mg IM) or **Hydrocortisone** 5mg /kg / IV every 6 hours for 2- 3 day.

Seizure

Introduction

Seizure is a sudden and transient brain dysfunction manifest by abnormal motor, sensory, autonomic, behavior and conscious state.

Classification of febrile seizure

(1) Simple febrile seizure: Primary generalized seizure associated with fever in a child 6–60 months of age that is non-focal, lasts <15 minutes and does not recur in a 24-hour period

(2) Complex febrile seizure: Seizure associated with a fever in a child 6–60 months of age that is focal, lasts >15 minutes, or recurs within a 24-hour period

Classification of Epilepsy

INTERNATIONAL CLASSIFICATION OF EPILEPTIC SEIZURES[10]

I. Partial seizures (seizures with focal onset)
  1. Simple partial seizures (consciousness unimpaired)
    a. With motor signs
    b. With somatosensory or special sensory symptoms
    c. With autonomic symptoms or signs
    d. With psychic symptoms (higher cerebral functions)
  2. Complex partial seizures (consciousness impaired)
    a. Starting as simple partial seizures
      (a) Without automatisms
      (b) With automatisms (e.g., lip smacking, drooling, dazed-eyes look)
    b. With impairment of consciousness at onset
      (a) Without automatisms
      (b) With automatisms
  3. Partial seizures evolving into secondarily generalized seizures
II. Generalized seizures
  1. Absence seizures: Brief lapse in awareness without postictal impairment (atypical absence seizures may have mild clonic, atonic, tonic, automatism, or autonomic components)
  2. Myoclonic seizures: Brief, repetitive, symmetric muscle contractions
  3. Clonic seizures: Rhythmic jerking, flexor spasm of extremities
  4. Tonic seizures: Sustained muscle contraction
  5. Tonic-clonic seizures
  6. Atonic seizures: Abrupt loss of muscle tone
III. Unclassified epileptic seizures

Investigations

1- Febrile seizure

a. No further workup is necessary for a simple febrile seizure in a neurologically intact child who appears well, has a normal neurologic exam, is fully immunized, and has no meningeal signs.

b. Neuroimaging, blood work, and EEG are not routinely recommended in previously healthy children who have a simple febrile seizure. Further studies should be directed toward ascertaining the source of the fever.

c. Perform a lumbar puncture in any child with seizures and meningeal signs or symptoms (e.g., nuchal rigidity, Kernig and/or Brudzinski signs, etc.).

d. Consider lumbar puncture in these circumstances:

- Infant 6–12 months of age if incomplete or unknown *Haemophilus influenzae* or *Streptococcus pneumoniae* immunizations.
- Febrile seizure in a child pretreated with antibiotics. Antibiotics can mask signs and symptoms of meningitis.

2- Non febrile seizure

a. If clinically indicated, check glucose, Na, K, Ca, Phos, blood urea nitrogen (BUN), creatinine (Cr) and complete blood cell count (CBC).

b. Blood pressure (BP): Supine and upright.

c. EEG: Recommended in all children with first nonfebrile seizure to evaluate for an epilepsy syndrome.

d. If this is not the first seizure and patient is receiving antiepileptic therapy, a change in seizure pattern should prompt a drug level.

e. Imaging: Although not required for diagnosis, MRI and CT can detect focal brain abnormalities that may predispose to focal seizures.

- Head ultrasound may be used in early infancy and requires open fontanelles.
- Head CT without contrast: Can detect mass lesions, acute hemorrhage, hydrocephalus, and calcifications secondary to congenital disease such as cytomegalovirus infection. Obtain a head CT only when concerned about a mass or bleed or in an emergency situation.
- Brain MRI with contrast: Obtain in infants with epilepsy and children with recurrent partial seizures, focal neurologic deficits, or developmental delay. Not routinely indicated when evaluating a first-time seizure.

Management of seizure

1. ABC of resuscitation should be stabilized.
2. O_2 therapy.
3. Open IV rout.
4. Do LP if indicated.
5. Find the cause and treat it
6. Manage fever if present.
7. **Anticonvulsive and other drugs**:

A. For Newborn : Give the following drugs step by steps:
Correction of hypoglycemia and hypocalcemia:

a. Dextrose: If glucostix shows hypoglycemia or if there is no facility to test blood sugar immediately, 5-10 ml/kg of 10% dextrose should be given as a bolus injection followed by a continuous infusion of 6-8 mg/kg/min.

b. Calcium gluconate 10% 2cc /kg diluted with equal volume of 5% glucose or distilled water and injected slowly in 5-10 minute through IV. If ionized calcium levels are suggestive of hypocalcemia, the newborn should receive calcium gluconate at 8 ml/kg/d for 3 days.

c. Magnesium sulphate: If seizures continue despite correction of hypocalcemia, 0.25 ml/kg of 50% magnesium sulfate should be given intramuscularly (IM) in 2 doses 12hr apart and be followed by maintenance oral dose of 0.2ml/kg of 50% solution once daily for 3 days. It is recommended for unresponsive hypocalcemia, suspected or proved hypomagnesaemia (serum level < 1mEq/l).

d. Anticonvulsants: The following anticonvulsive drugs should be given step by step if seizures persist even after correction of hypoglycemia/ hypocalcemia.

- **Phenobarbital** loading dose 20 mg /kg is administered slowly IV over 20 minutes (not faster than 1 mg/kg/min). If seizures persist after completion of this loading dose, additional doses of phenobarbitone 10 mg/kg may be used every 15 minutes until a total dose of 40 mg/kg has been given. If convulsions are still uncontrolled, add Phenytoin. Maintenance dose of Phenobarbital is 5mg /kg/day usually in one or 2 divided doses, started 12 hours after the loading dose.
- **Phenytoin or Fosphenytoin** is administered intravenously in a loading dose of 15-20 mg /kg. Phenytoin is diluted in normal saline (not in glucose containing solution) and given slowly over 10-20 minutes or at a rate of 1mg/kg/min. Assess control after 30 minute, if seizure persist then repeat 10mg/kg (may repeat totally up to the dose of 30mg/kg). Maintenance dose is 5mg /kg/day in one or 2 divided doses. Only IV route is preferred in neonates and it should preferably be discontinued before discharge.
- **Benzodiazepines:** For refractory cases give the following drugs (especially Lorazepam and Midazolam) step by steps
  - **Lorazepam** 0.05-0.1mg/kg IV bolus over 2-5 minutes; may be repeated q 8-12hr.
  - **Midazolam** 0.05-0.15 mg/kg IV bolus followed by infusion of 0.1 to 0.4 mg/kg/hour.
  - **Diazepam** 0.1-0.3mg/kg is given intravenously over 3-5 minutes. Repeat every 15-30 minutes if not respond.
- **Pyridoxine** 50-100mg IV or IM is useful in refractory cases.

- Lidocaine, Paraldehyde, Sodium valproate, Vigabatrin and Topiramate are the anticonvulsants in refractory cases.

B: For Infant and children: Give the following drugs step by steps:

a: **Diazepam** 0.2- 0.5mg/kg IV over 1- 5 min; if do not respond in 5-20 minute repeat for up to 3 doses.

b: **Phenobarbital** 5 – 20 mg /kg IV loading. In refractory case repeat 10mg/kg. Maintenance dose is 3-5 mg /kg/day

c: **Phenytoin** 10-20 mg / kg IV loading over 5-20 minutes. In refractory case repeat 10mg/kg. Maintenance dose is 10 mg /kg/day

d: **Midazolam** 0.05-0.2 mg/kg IV bolus, followed by continuous infusion of 1-2µg/kg/min for persistent seizures is effective.

8. Antipyretic and **sponging** are indicated for febrile convulsion (See FWF).

9. Suspected ICH: Should be treated as mention under management of coma.

Heart Failure

Introduction

Heart failure (HF) is the clinical condition in which the heart fails to meet the circulatory and metabolic needs of the body. The term *congestive heart failure* is not always accurate, as some patients with significant cardiac dysfunction have symptoms of exercise intolerance and fatigue without evidence of congestion.

Essentials of diagnosis

Physical examination may reveal tachycardia, increased work of breathing, and weak pulses. Pulmonary crackles can be heard upon auscultation and indicate pulmonary edema. Cardiac auscultation may reveal a gallop rhythm (S3 and/or S4) and occasionally a holosystolic murmur, caused by mitral or tricuspid regurgitation. Venous congestion causes hepatomegaly in all age groups and jugular venous distention in older children. Dependent lower extremity edema is mostly seen in older children, while infants may display facial edema.

Ross classification of heart failure in children

Class	Interpretation
I	Asymptomatic
II	Mild tachypnea or diaphoresis with feeding in infants
	Dyspnea on exertion in older children
III	Marked tachypnea or diaphoresis with feeding in infants; prolonged feeding times with growth failure due to heart failure
	Marked dyspnea on exertion in older children
IV	Symptoms such as tachypnea, retractions, grunting, or diaphoresis at rest

Investigations

1-Chest radiography: Chest radiography shows cardiomegaly and prominent vascular markings

2- Electrocardiography: Electrocardiographic changes may reflect the underlying diagnosis.

3- Echocardiography: Echocardiography provides good information regarding the cardiac lesion and allows assessment of left ventricular function.
4- Blood: Electrolytes, Hb, glucose and B-type natriuretic peptide (BNP).

Management of Heart Failure

1. Bed rest and restriction of activities are advised: Strict bed rest is rarely necessary except in extreme cases, but it is important that the child be allowed to rest during the day as needed and sleep adequately at night.
2. Propped up position (about 30 degree) is effective.
3. **O_2** therapy.
4. **PPV:** For patients with pulmonary edema, positive pressure ventilation may be required along with other drug therapy.
5. IV fluid should be restricting to 2/3 of maintenance.
6. Nutrition should be high calorie. If rapid respiration and extreme fatigue is present feed by NGT.
7. **Diuretics :**
    - **Lasix 1-2 mg/kg** IV should be repeated every 12 hour until toleration of oral intake then 2-4 mg/kg /day orally 1-4 divided doses.
    - **Spironolactone** administered 2-3mg/kg/day orally in 2-3 divided doses
8. **Digoxin** : Orally TDD(Total Digitalization Dose) is as follow:

For neonates less than 1 week 0.04mg/kg, for 1week -2 years old 0.06mg/ kg and for < 1 w or > 2 y old 0.0 5mg /kg. ½ TDD is used first, ¼ TDD at 6 hour, 12hour intervals. ¼ TDD is given 24 hours after initial dose. For IV administration give 2/3 of oral dose.

9. **Dopamine:** IF shock is present treat as cardiogenic shock.
10. **Morphine** 0.1-0.2mg/kg/dose sc q 2-4hr for anxious patient and pulmonary edema.
11. **Afterload –Reducing Agents:** If Diuretic and Digoxin do not control the CHF; or cardiomyopathy, severe mitral or aortic insufficiency is the cause give one of the followings should be added :

- **Captopril** 0.1-0.5mg/kg/day for infants 0.1-2mg/kg/day for children in 2 divided doses.
- **Enalapril** 0.08-0.5mg/kg/dose q 24hr.

12. **Antibiotics** to treat infection as mentioned forpneumonia.
13. **Phosphodiestrase inhibitor (Amrinone, Milrinone)** and **β-Blockers (Carvidilol, Metoprolol)** may be used.
14. Find and treat precipitating factors like infection, anemia, and arrhythmia.

Liver Failure

Introduction

Fulminant hepatic failure (acute liver failure) is a clinical syndrome resulting from massive necrosis of hepatocytes or from severe functional impairment of hepatocytes.

Essentials of diagnosis

The currently accepted definition in children includes biochemical evidence of acute liver injury (usually <8 wk duration) with one of the following condition.

- INR > 2 or PT> 20se without encephalopathy. or
- INR > 1.5 or PT> 15sec with encephalopathy.

Investigations

Serum direct and indirect bilirubin levels and serum aminotransferase activities may be markedly elevated. Serum aminotransferase activities do not correlate well with the severity of the illness and can actually decrease as a patient deteriorates. The blood ammonia concentration is usually increased, but hepatic coma can occur in patients with a normal blood ammonia level. PT and the INR are prolonged and often do not improve after parenteral administration of vitamin K. Hypoglycemia can occur, particularly in infants. Hypokalemia, hyponatremia, metabolic acidosis, or respiratory alkalosis can develop.

Management of Liver Failure

A. General Measures:

1. CPR is essential.

2. Intubation and Mechanical ventilation is required for comatose patients to prevent aspiration and induce hyperventilation for the management of cerebral edema.

3. Oxygen therapy is often necessary.

5. Apply NGT and do regular gentle saline lavage to detect upper GI bleeding.

6. Hemoglobin should be maintained above 10gr/dl to provide maximum oxygen delivery to tissue.

7. Maintenance fluid should be 75% of normal.

8. Adequate glucose is used (6-8mg/kg/min).
9. Protein intake should be restricted in patient with more than grade 2 encephalopathy.
10. Manage ICH as following:

A. Intubation and Mechanical ventilation to prevent aspiration and induce hyperventilation for the management of cerebral edema.

C. Sedation, Analgesia and elevate head of the bed (15- 30 degree).

D. Manittol 5 cc /kg IV over 30 minutes is useful. It may be repeated 6-8 hourly for 6 doses, if needed. Or/and

E. 3% Saline 0.1-1ml/kg/hr intravenously.

F. **Lasix** 1 mg/kg/day IV can be added.

11. Shock must be treated properly.
12. Lactulose should be given 1-2ml/kg 6-8hrly orally or via NGT to cause diarrhea. It probably lowers blood ammonia level.
13. Seizures are treated with Phenytoin or Phenobarbital.
14. Antibiotics:

A. Oral antibiotic may be more effective than Lactulose in lowering serum ammonia level. For this purpose Metronidazol or Neomycin is used.

B. Parenteral antibiotics are used empirically to treat sepsis, pneumonia, peritonitis, UTI and other infections. A combination of third generation cephalosporin and cloxacillin are used. Aminoglycosides are administered if renal function are normal. If there is no improvement within 72hr, it is prudent to step up antibiotic to cover Pseudomonas aeruginosa, anaerobic organism and/ or fungi.

15. Vitamin K 5-10mg IV or SC, fresh blood, fresh frozen and platelets transfusion are used to treat coagulopathy (clinically significant bleeding, DIC, PT > 30 sec and INR>2).

16. Prophylactic use of proton pump inhibitors, H_2 blockers or antacid should be considered because of the high risk of GI bleeding.

17. Liver transplantation can be live saving in patient who reaches advanced stage.

B. Specific:

N- acetylcystin is given for Acetaminophen over dosage, corticosteroid for Autoimmune Hepatitis and Acyclovir for Herpes simplex and CMV.

Acute bacterial Meningitis

Introduction

Inflammation of membrane lining CNS is called meningitis.
Bacterial infections of the CNS may present acutely (symptoms evolving rapidly over 1–24 hours), subacutely (symptoms evolving over 1–7 days), or chronically (symptoms evolving over more than 1 week).

Essentials of diagnosis

- Feature of infection: Fever, Associated findings such as a maculopapular or petechial rash in meningococcal infections, otitis media or pneumonia in pneumococcal infections, and pustular skin lesions in staphylococcal infections may be seen.
- Signs of meningeal irritation: On examination, neck rigidly and other signs of meningeal irritation are seen. These include:

- Kernig's sign: With thighs flexed on abdomen, passive extension of knee produces pain in the back

- Brudzinski's sign: Passive flexion of neck produces flexion of both lower limbs

- Tripod sign: In the sitting position, the child supports himself with both arms extended behind the back, which is kept straight

- Knee-kiss sign: The child cannot bend forward to kiss his knees.

- Feature of Intracranial Hypertension: Signs of suspected ICH are:

- Decreased level of conscious.
- Repeated convulsions.

- Vomiting.
- Cardiac arrhythmia.
- Focal Neurological deficit.
- Headache.
- Papilledema (Old child).

• CSF findings:

The diagnosis of acute pyogenic meningitis is confirmed by analysis of the CSF, which typically reveals microorganisms on Gram stain and culture, a neutrophilic pleocytosis, elevated protein, and reduced glucose concentrations.

The CSF leukocyte count in bacterial meningitis usually is elevated to >1,000/mm3 and, typically, there is a neutrophilic predominance (75-95%). Turbid CSF is present when the CSF leukocyte count exceeds 200-400/mm3. Normal healthy neonates may have as many as 30 leukocytes/mm3 (usually <10), but older children without viral or bacterial meningitis have <5 leukocytes/mm3 in the CSF; in both age groups there is a predominance of lymphocytes or monocytes.

Investigations

1- Blood: For CBC and blood culture and CRP.

2- Lumbar puncture: For Gram stain, Culture, PCR, cytological and biochemical examination of CSF.

3- CT scan: CT scan before the LP is not routinely needed; it is indicated in children with focal neurological symptoms or signs, papilledema, critically raised ICP or suspicion of a mass lesion.

Management of Acute bacterial Meningitis

1. Stabilized ABC of resuscitation if need.
2. Open IV line.
3. Lumber puncture should be done. Contraindications for an immediate LP are:
   - Evidences of increased ICH (other than a bulging fontanel) such as papilledema, 3rd or 6th nerve palsy with a depressed level of conscious or hypertension and bradycardia with respiratory abnormalities.
   - Severe cardiopulmonary compromised.
   - Infection of the skin overlying the site of LP.
   - Thrombocytopenia is a relative contraindication.
4. Manage convulsion (see management of seizures).
5. O_2 therapy if needed.
6. Treat ICH as following:

A. Intubation and Mechanical ventilation to prevent aspiration and induce hyperventilation for the management of cerebral edema.

C. Sedation, Analgesia and elevate head of the bed (15- 30 degree).

D. Manittol 5 cc /kg IV over 30 minutes is useful. It may be repeated 6-8 hourly for 6 doses, if needed. Or/and

E. 3% Saline 0.1-1ml/kg/hr intravenously.

F. Lasix 1 mg/kg/day IV can be added.

7. Antibiotic:

A. For neonates and infants up to 2 month:

Ampicillin (200-400mg/kg/day) + **third generation cephalosporin** (**Cefotaxime** 150-200mg/kg/day or **Ceftriaxone** 100mg/kg/day) can be used for 21 days in

neonate and 10 days in older. The dosage for neonate is mentioned under NNSepsis.

B. For older than 2 month :

Third generation cephalosporin (**Cefotaxim**e 150-200mg/kg/day or **Ceftriaxone** 100mg / kg/ day) is administered for 10-14 days.

C. **In resistant case** to above drugs, vancomycin (60mg /kg /day IV infusion over 30-60min) is indicated.

D. If a patient is immunocompromised and Gram-negative bacterial meningitis is suspected, initial therapy might include Ceftazidime and an aminoglycoside or Meropenem.

8. Dexamethazone 0.15mg/kg every 6hr for 2-4 days can be used in infant older than 6 weeks; specially for Meningitis due to H. influenza type b. It appears to have maximum effect if given 1-2hr before initiated antibiotics. It also maybe effective if given concurrently with or soon after the first dose of antibiotics.

Encephalitis

Introduction

Dysfunction of the brain due to inflammation is called encephalitis. In majority cases the cause is viral agent.

Essentials of diagnosis

Typical features include an initial stage of fever, headache and vomiting lasting for less than a week followed by convulsions, coma and neurological deficits with or without signs of meningeal irritation. Severe cases may be associated with life-threatening rise in intracranial tension,

decerebration or flaccid coma. Typically this stage lasts for 7–10 days after which, there is gradual recovery with or without sequelae.

Investigations

1- Blood: In the acute stage, blood counts usually reveal a polymorphonuclear leukocytosis.

2- CSF examination: Cerebrospinal fluid in VE typically shows a pleocytosis of up to 300 cells/mm3, which can be either predominantly lymphocytic or polymorphonuclear, with normal to slightly raised protein and normal sugar level. Cerebrospinal fluid pleocytosis (> 5 cells/mm3) is present in more than 95% cases of acute VE and exceeds 500 cells/mm3 in 10% cases of AVE. The CSF in acute VE is indistinguishable from aseptic or viral meningitis.

3- Samples for Viral Culture**:** Samples for viral culture from respiratory secretions, throat swab, CSF, blood, urine and stool.

4- PCR: Polymerase chain reaction (PCR) is the mainstay of diagnosis. This is widely used for diagnosis of HSE.

5- Serological test: Japanese encephalitis is commonly diagnosed by the antibody capture ELISA for IgM antibody in acute phase serum and CSF.

6- Neuroimaging of Brain: Neuroimaging of brain is now a standard investigation in patients with suspected VE.

Management of Encephalitis

1. Stabilized ABC of resuscitation if need.
2.Open IV line.

3. Lumber puncture should be done. Contraindications for an immediate LP are:

- Evidences of increased ICH (other than a bulging fontanel) such as papilledema, 3rd or 6th nerve palsy with a depressed level of conscious or hypertension and bradycardia with respiratory abnormalities.
- Severe cardiopulmonary compromised.
- Infection of the skin overlying the site of LP.
- Thrombocytopenia is a relative contraindication.

4. Manage convulsion (see management of seizures).
5. O_2 therapy if needed.
6. Treat ICH as following:

A. Intubation and Mechanical ventilation to prevent aspiration and induce hyperventilation for the management of cerebral edema.

C. Sedation, Analgesia and elevate head of the bed (15- 30 degree).

D. Manittol 5 cc /kg IV over 30 minutes is useful. It may be repeated 6-8 hourly for 6 doses, if needed. Or/and

E. 3% Saline 0.1-1ml/kg/hr intravenously.

F. Lasix 1 mg/kg/day IV can be added.

7. Antiviral: Specific therapy is recommended in encephalitis due to herpes group of viruses. Acyclovir in a dose of 15 mg/kg administered as an intravenous infusion over 1 hour every 8 hours for 14 days (21 days in immunocompromised) is indicated in HSE. Success of antiviral therapy depends on early institution of therapy. Acyclovir is also recommended for varicella-zoster encephalitis.
8. Antibiotics:

A. For neonates and infants up to 2 month:

Ampicillin (200-400mg/kg/day) + **third generation cephalosporin** (**Cefotaxime** 150-200mg/kg/day or **Ceftriaxone** 100mg/kg/day) can be used for 21 days in neonate and 10 days in older. The dosage for neonate is mentioned under NNSepsis.

B. For older than 2 month :

- Third generation cephalosporin (**Cefotaxime** 150-200mg/kg/day or **Ceftriaxone** 100mg / kg/ day) is administered for 10-14 days.
- **In resistant case** to above drugs, Vancomycin (60mg /kg /day IV infusion over 30-60min) is indicated.
- If a patient is immunocompromised and Gram-negative bacterial meningitis is suspected, initial therapy might include Ceftazidime and an aminoglycoside or Meropenem.

Sepsis

Introduction

Sepsis is defined as **SIRS** resulting from a suspected or proven infectious etiology.

Essentials of Diagnosis

SIRS diagnosed by two of 4 criteria, 1 of which must be abnormal temperature or abnormal leukocyte count:

1. Core temperature >38.5°C (101.3°F) or <36°C (96.8°F) (rectal, bladder, oral, or central catheter)
2. Tachycardia:

Mean heart rate >2 SD above normal for age in absence of external stimuli, chronic drugs or painful stimuli *or*

Unexplained persistent elevation over 0.5-4 hr *or*

In children <1 yr old, persistent bradycardia over 0.5 hr (mean heart rate <10th percentile for age in absence of vagal stimuli, β-blocker drugs, or congenital heart disease)

3. Respiratory rate >2 SD above normal for age or acute need for mechanical ventilation not related to neuromuscular disease or general anesthesia

4. Leukocyte count elevated or depressed for age (not secondary to chemotherapy) or >10% immature neutrophils

Sepsis diagnosed by SIRS plus a suspected or proven infection.

Severe sepsis diagnosed by sepsis plus 1 of the following:

1. Cardiovascular organ dysfunction, defined as:

• Despite >40 mL/kg of isotonic intravenous fluid in 1 hr:

- Hypotension <5th percentile for age or systolic blood pressure <2 SD below normal for age

or

- Need for vasoactive drug to maintain blood pressure

or

• 2 of the following:

- Unexplained metabolic acidosis: base deficit >5 mEq/L
- Increased arterial lactate: >2 times upper limit of normal
- Oliguria: urine output <0.5 mL/kg/hr
- Prolonged capillary refill: >5 sec
- Core to peripheral temperature gap >3°C (5.4°F)

2. ARDS as defined by the presence of a PaO2/FIO2 ratio ≤300 mm Hg, bilateral infiltrates on chest radiograph, and no evidence of left heart failure

or

Sepsis plus 2 or more organ dysfunctions (respiratory, renal, neurologic, hematologic, or hepatic)

Investigation

1- Blood: CBC, CRP, Glucose and raised inflammatory markers with neutrophilia, or leucopenia or thrombocytopenia..

2- Culture: blood, urine, CSF. PCR can be performed on CSF to aid bacterial identification.

3- Urinalysis (including urine culture)

4- Chest radiography.

Management of Sepsis

1. Stabilized ABC of resuscitation if need.
2. Open IV line.
3. Give Paracetamol for the management of fever.
4. Antibiotics

A. Infants up to 2 month:

Ampicillin (200-400mg/kg/day) + **third generation cephalosporin** (**Ceftriaxone** 100mg/kg/day or **Cefotaxime** 150-200mg/kg/day) can be used for 10 days.

C. For older than 2 month :

- Third generation cephalosporin (**Ceftriaxone** 100mg / kg/ day or **Cefotaxime** 150-200mg/kg/day) is administered for 10-14 days.
- **In resistant case** to above drugs, vancomycin (60mg /kg /day IV infusion over 30-60min) is indicated.
- If a patient is immunocompromised and Gram-negative bacterial meningitis is suspected, initial therapy might include Ceftazidime and an aminoglycoside or Meropenem.

Diabetic Ketoacidosis (DKA)

Introduction

Severe ketoacidosis is a life-threatening complication of diabetes that is present in 20% to 40% of newly diagnosed type 1 (juvenile-onset) diabetic patients; it accounts for 65% of all admissions of diabetic patients younger than 19 years of age.

Essentials of Diagnosis

A-Clinical Features

- Can present with any combination of:

-Vomiting, abdominal pain.
- Polyuria, polydipsia
- Weight loss.
- Kussmaul (deep sighing) respiration.
- Smell of ketones.
- Reduced conscious level (10–20 %).

B-Investigations

- Fasting blood glucose > 250mg/dl or Random blood glucose > 300mg/dl.
- Blood pH< 7.3
- Blood ketone > 3mmol/L
- Ketonuria and glucoseuria

Classification

Table 16: Classification of DKA

Table 589-4	Classification of Diabetic Ketoacidosis			
	NORMAL	MILD	MODERATE	SEVERE*
CO_2 (mEq/L, venous)†	20-28	16-20	10-15	<10
pH (venous)†	7.35-7.45	7.25-7.35	7.15-7.25	<7.15
Clinical	No change	Oriented, alert but fatigued	Kussmaul respirations; oriented but sleepy; arousable	Kussmaul or depressed respirations; sleepy to depressed sensorium to coma

Management of Diabetic Ketoacidosis (DKA)

1- In mild DKA (with blood glucose 200–250 mg/dL, mild dehydration, pH 7.2–7.3 and bicarbonate 10–15 mmol/L), the child can be given oral rehydration with appropriate (sugar-free) fluid and subcutaneous insulin therapy started as below

2- Management of moderate and severe DKA:

- 1st hr: Give 10-20 mL/kg IV bolus 0.9% NaCl or LR and Insulin drip at 0.05 to 0.10 units/kg/hr
- 2nd hr until DKA resolution: Give 0.45% NaCl ($\text{IV rate} = \frac{85\,\text{mL/kg} + \text{maintenance} - \text{bolus}}{23\,\text{hr}}$): plus 20 mEq/L KPhos and 20 mEq/L K Acetate. continue insulin drip.
  Add 5% glucose in IV fluid if blood glucose <250mg/dl.
- Alkali therapy: Sodium bicarbonate may be indicated for symptomatic hyperkalemia or if the blood pH persists at less than 6.9 after the first hour of rehydration, with cardiovascular instability. The dose to be infused is calculated by the formula: (mL of sodium bicarbonate = 0.15 × base deficit × kg body weight). The amount (usually 40 mL) is never given as a bolus, but added to a 0.5 N saline infusion over 2 hours. Alkali therapy should be stopped when pH is greater than 7.0.
- After correction of acidosis, ketonuria and emesis (which may take 24 hours or longer in severe DKA), the child is ready to be started on subcutaneous insulin therapy. An initial dose (0.2–0.4 units/ kg) of regular

insulin is given, half an hour after which the insulin infusion is stopped and the child allowed to eat.

Management of Poisonings

General Managements

1- **ABCs of Resuscitation** should be established.(See Resuscitation)
2- **Shock** and **convulsions** are generally managed as mentioned in related topics.
3- If the level of conscious is depressed and a toxic substances is suspected, **10% Dextrose** 2-4ml/kg, **Naloxan** 0.4 -2mg and **100% oxygen** should be administrated.
4- **Decontamination:**

A. Gastrointestinal decontamination : Most liquid drug products are almost completely absorbed within 30-45min of ingestion and most solid forms are absorbed within 1-2hrs.Complete intestinal absorption of large overdosage of solid form drugs (tablets and capsules) can be delayed as much as 3-6hrs and for drugs or toxins with anticholinergic properties absorption can be delayed by up to 8-12hrs. The following managements can be used to prevent the absorption.

- **Activated Charcoal:** It is more efficacious than emesis or gastric lavage and is currently regarded as a universal antidote. Usually a dose of 1-2g/kg is recommended. Repeated doses may be useful for those agents that have slow passage through gastrointestinal tract

(Phenobarbital, Thiophylline, Quinine, Cyclic antidepressants, Digoxin and Carbamazepin) every 2-4 hrs or 0.25g/kg/hrly. A Cathartic should be used only with the first Charcoal dose. Activated charcoal is more effective when it is administrated within the first 30 min after exposure. Beyond 60min of exposure it is less effective. Charcoal is known to be ineffective against caustic or corrosive agents, hydrocarbon, heavy metals (arsenic, lead, mercury, iron, lithium) glycol and water-insoluble compounds. Patients with paralytic ileus should not receive activated charcoal.

- **Emesis:** Vomiting can be induced by tickling the fauces with a finger, though rarely used in pediatric due to the danger of aspiration. To prevent aspiration in small children, the head should be kept low. This measure is contraindicated in corrosive or hydrocarbon poisonings, comatose and convulsive patients.
- **Gastric lavage:** It has a limited place in management and indicated in serious poisonings. Gastric lavage is done with 15ml/kg of 0.9% saline through large bore NGT. Contraindications are the same as emesis.
- **Cathartics:** One of the followings cathartics have been used in conjunction with the first dose of activated charcoal.
  - Sorbitol 1-2g/kg.
  - Magnesium sulphate 250mg/kg.
  - Magnesium citrate max 250mg/kg.
- **Whole Bowel Irrigation:** This technique has been successfully used to remove slowly absorbed products,

such as iron or sustained-release preparations as well as foreign bodies. WBI is accomplished through rapid and large volume (30ml/kg/hr) administration of polyethylene glycol electrolyte solution into the stomach via NGT.

B- Dermal and Ocular Decontamination: The affected area should be washed with tepid water for 10min in skin and eyes exposure.

C- Blood Decontamination:

- **Urine Alkalinization** with $NaHCO_3$ (2 mEq/kg IV) should be considered for significant Salicylates and Phenobarbital poisonings.
- **Dialysis and Hemoperfusion** Should be undertaken only for toxins that may cause tissue damage. Dialysis (hemodialysis and peritoneal dialysis) may be useful for toxic alcohols, methanol and ethylene glycol as well as large symptomatic ingestion of salicylates, thiophylline or lithium. Hemoperfusion can successfully treat large ingestion of salicylates and thiophylline.

D-Specific poisons and Antidotal therapy:

- **Acetaminophen:** N-acetyl cysteine (NAC) 140mg/kg PO initial dose (diluted in sweet fruit juice), then 70mg/kg PO q 4h × 17 doses. If the patient is encephalopathic , an initial IV loading dose of 150mg/kg in 5ml/kg of glucose 5% is infused over 15-

60min, followed by 50mg/kg over 4hr, then followed by 100mg/kg over next 16hr.

- **Alcohol (Ethanol and Methanol):** For hypoglycemia start D/W 25% 1-2ml/kg IV. Metabolic acidosis is treated with IV sodium bicarbonate at a dose of 1-2 mEq/kg. In the cases of Methanol poisoning give Ethanol loading dose 10ml/kg IV or orally followed by maintenance dose 1-2ml/kg/h IV or orally.
- **Antidepressants (Tricyclic antidepressants and Selective Serotonin-reuptake inhibitors):** Emesis is contraindicated. IV sodium bicarbonate (0.5-1mEq/kg) is one of the most effective therapies in treating and preventing cardiac conduction abnormality. Lidocaine is used to treat dysrhythmias that are unresponsive to serum Alkalinization.
- **Antihistaminic:** Physiostigmin 0.5-2mg IV slowly administrated for anticholinergic effects.
- **Belladonna Alkaloids (Atropine, Scopolamine, Potato leaves Dathura or Jimson weed):** Physiostigmin (0.5-2mg IV, slowly administrated) dramatically reverses the central and peripheral signs of atropinism.
- **Benzodiazepine:** Flumazenil 0.01-0.02mg/kgIV is the antidote.
- **Beta-Blockers:** Atropine, IV fluid, vasopressors, D/W10% and glucagon are the useful agent, for the management of bradycardia, hypotension, shock and hypoglycemia. If symptomatic bradycardia is refractory

to all of these measures, ventricular pacing should be considered.

- **Calcium Channel Blockers:** Atropine is the drug of choice for symptomatic bradycardia, a pacemaker should be considered for refractory cases. Administration of calcium gluconate 10% (100mg/kg) may reveres myocardial depression, impaired conduction and hypotension.
- **Carbon Monoxide:** Give 100% oxygen or keep in fresh air. Dexamethasone (0.1mg/kg IV or IM every 4-6hrs) should be added if cerebral edema develops.
- **Caustics (Acid and Bases):** Activated charcoal, Emesis and gastric lavage are not indicated. The skin and mucous membranes should be cleaned with copious amount of water. Water or milk (<15ml/kg) is used to dilute the Acids. For dilution of Bases water is effective. If symptoms of esophageal burn are present, oral fluids or solids should be withheld. Antacid and H_2 blockers are given for 6-8 week to suppressed acid secretion. Antibiotic is used to prevent infection. The use of corticosteroid and esophageal stents are controversial.
- **Digoxin:** Digoxin-specific Fab antibody fragment (digibind) is useful for life-threatening dysrhythmias; 1vial (40mg) neutralizes 0.6mg Digoxin. In the absence of digibind, ventricular ectopy should be treated with Phenytoin 15mg/kg IV then 2mg/kg q 8hr. Atropine is the standard therapy for symptomatic bradycardia.

- **Hydrocarbons (Benzene, Kerosene, Charcoal, Gasoline, Petroleum distillates):** Emesis and gastric lavage are usually contraindicated. Activated charcoal is not useful. Oxygen and mist are helpful. Antibiotic should be reserved for patient with infection.
- **Iron:** Deferoxamine 15mg/kg/hr in 5% dextrose should be given IV if the patient is symptomatic (shock) and the serum iron determination cannot be obtained. Discontinue Deferoxamine therapy 24hrs after urine loses vin rose (pink to red orange) color. GI bleeding, shock, metabolic acidosis, hypoglycemia and coagulopathy should be corrected.
- **Isoniazid:** Pyridoxine (Vit.B_6) should be used equal to the amount of Isoniazid ingested, up to 250mg/kg.
- **Mushrooms:** Give Atropine 0.02-0,05mg/kg IM and repeat every 30min, when cholinergic effects are present.
- **Opoids** (Codeine, Heroin, Morphine, Methadone, and Propoxyphene): Naloxan is the antidote. For infants younger then 1year, 0.4mg should be given initially, if there is no response, 2mg should be used rapidly. Older children should be given 0.4-0.8mg, followed by 2-4mg if there is no response.
- **Organophosphate insecticides (Malathion, Parathion, clorothion**): Decontamination of skin, nails, hair and clothing with soapy water is extremely important. Atropine plus Pralidoxime is the antidote. An appropriate starting dose of Atropine is 0.05mg/kg slow IV. Repeat the dose every 10-15min until

atropinization (clearing of secretion, dry warm skin and mydriasis) is achieved. Maintain atropinization with repeated dosage of 0.02-0.05mg/kg/IV for 2-12hrs or longer depending on the severity of poisoning. Pralidoxime should also be given immediately in severe case and repeated every 6-12hrs as needed. The dosage is 25-50mg/kg diluted to 5% concentration with N/S and infused over 30min at a rate of no more than 500mg/min.

- **Phenothiazines (Chlorpromazine, Prochloperazine and Trifluoperazine):** Extrapyramidal signs are alleviated within minutes by the slow IV administration of Diphenhydramine, 1-2mg/kg or Benztropine mesylate, 1-2mg/kg IV.

Envenomations

Snake Bite

1. **ABC** of resuscitation should be undertaken.
2. **Immobilize** the bitten extremities with bandage.
3. Always premedicate the patient with **Adrenaline** 0.005-0.01mg/kg (0.05-0.1ml/kg SC of 1:10000 or 0.005-0.01ml/kg of 1:1000 solutions) before Antivenom therapy.
4. **Antivenom:** Antivenom is most effective if given within 4hr of the bite and is of little value if administration is delayed beyond 12hr. Dilute

Antivenom 1:4 with 0.45N/S and administer 20ml/kg/h. The dosage that mentioned below should be repeated every 2hr if symptoms persist.

- **Minimal Envenomation** (swelling, erythema or ecchymosis confined to the site of the bite): Required 5vials (50ml) of reconstituted Antivenom.
- **Moderate Envenomation** (progression of swelling, erythema or ecchymosis beyond the site of the bite; mild systemic symptoms like nausea, vomiting, perioral paresthesias, mild hypotension, mild bleeding and mild laboratory abnormalities): This type require 10vials (100ml) of reconstituted Antivenom.
- **Severe Envenomation**(Rapid swelling, erythema or ecchymosis involving the entire body part; severe S/S like hypotension, altered sensorium, tachycardia, tachypnea, respiratory distress, bleeding and platelet count less than 20000/mm^3): For this type 15vials (150ml) of reconstituted Antivenom is required.

5. **Antibiotics** can be used for anaerobic and Gram negative bacteria.
6. **Tetanus** prophylaxis if indicated.
7. A course of **Prednisolon 1mg/kg/day** orally for 2-5days may prevent serum sickness which may occur after Polyvalent Antivenom.

Scorpion Sting

1. Stabilize ABC of resuscitation if needed.
2. Localized pain can be treated with application of ice and analgesics. Pain usually diminished within 24hr.

3. **Autonomic Storm** (Tachycardia, cool extremities, systolic BP>130mm or <70mm and restlessness) should be treated with all of the following:
   - **Prazosin** 30μg/kg/dose administer orally or by NGT (if vomiting is present). It is repeated at the end of 3hrs according to clinical response and later every 6hrs till extremities are warm. No more than four doses have been required in majority of cases.
   - **Diazepam** 0.5- 2mg/kg/dose orally or IM.
4. **Myocardial dysfunction** (Tachypnea, ice cold extremities, palmoplantar sweating, S_3 gallop and altered conscious) should be managed with all of the following:
   - **Oxygen** 2lit/min.
   - IV fluid.
   - **Prazosin** as mentioned above.
   - **Diazepam** 0.5-2mg/kg IV.

5. Antivenom is reserved for severe cases (cardiopulmonary compromise).

Part 3
Respiratory Diseases

Pneumonia

Introduction

Pneumonia, defined as inflammation of the lung parenchyma, is the leading cause of death globally among children younger than age 5 yr. Although most cases of pneumonia are caused by microorganisms, noninfectious causes include aspiration (of food or gastric acid, foreign bodies, hydrocarbons, and lipoid substances), hypersensitivity reactions, and drug- or radiation-induced pneumonitis. Etiologic agents of pneumonia in different age group is shown in table 17

Table 17: Infectious etiology of pneumonia in different ages

AGE GROUP	FREQUENT PATHOGENS (IN ORDER OF FREQUENCY)
Neonates (<3 wk)	Group B streptococcus, *Escherichia coli*, other Gram-negative bacilli, *Streptococcus pneumoniae, Haemophilus influenzae* (type b,* nontypeable)
3 wk-3 mo	Respiratory syncytial virus, other respiratory viruses (rhinoviruses, parainfluenza viruses, influenza viruses, adenovirus), *S. pneumoniae, H. influenzae* (type b,* nontypeable); if patient is afebrile, consider *Chlamydia trachomatis*
4 mo-4 yr	Respiratory syncytial virus, other respiratory viruses (rhinoviruses, parainfluenza viruses, influenza viruses, adenovirus), *S. pneumoniae, H. influenzae* (type b,* nontypeable), *Mycoplasma pneumoniae*, group A streptococcus
≥5 yr	*M. pneumoniae, S. pneumoniae, Chlamydophila pneumoniae, H. influenzae* (type b,* nontypeable), influenza viruses, adenovirus, other respiratory viruses, *Legionella pneumophila*

Essential of diagnosis

A- Clinical features

Pneumonia is frequently preceded by several days of symptoms of an upper respiratory tract infection, typically rhinitis and cough. In viral pneumonia, fever is usually present but temperatures are generally lower than in bacterial pneumonia. Tachypnea is the most consistent clinical manifestation of pneumonia. Increased work of breathing accompanied by intercostal, subcostal, and suprasternal retractions, nasal flaring, and use of accessory muscles is common. Severe infection may be accompanied by cyanosis and lethargy, especially in infants. Auscultation of the chest may reveal crackles and wheezing.

B- Investigations

1. Chest radiography: An infiltrate on chest radiograph (posteroanterior **and** lateral views) supports the diagnosis of pneumonia; the film may also indicate a complication such as a pleural effusion or empyema. Viral pneumonia is usually characterized by hyperinflation with bilateral interstitial infiltrates.

2. Blood: The peripheral white blood cell (WBC) count and CRP can be useful in differentiating viral from bacterial pneumonia. In viral pneumonia, the WBC count can be normal or elevated but is usually not higher than 20,000/mm3, with a lymphocyte predominance. Bacterial pneumonia is often associated with an elevated WBC count, in the range of 15,000- 40,000/mm3, and a predominance of granulocytes. A large pleural effusion, lobar consolidation, and a high fever at the onset of the illness are also suggestive of a bacterial etiology.

Indications for Admission are:

- Sever Respiratory distress.
- Cyanosis.
- Altered conscious.
- Vomiting / poor intake.
- Multiple lobe involvement.
- Dehydration.
- Age less then 2 month.
- Immunocompromised state
- Complicated pneumonia
- Toxic appearance
- No response to appropriate oral antibiotic therapy

Management of pneumonia

1- Slightly propped up position is effective.

2- Give **humidified 25- 100% oxygen** 4- 6 liter / minute in neonates and 2- 4 lit / mint in infants and older children. Keep O_2 Saturation > 92%.

Indications for humidified oxygen:

1. **Respiration distress**
2. **Cyanosis**
3. **Increased R R** (>70/min in infants less than 2month, >60/min in infants aged 2-12months and >50/min in children older than one year)

3- Nutrition and maintenance fluid should be undertaken.

4- **Zinc:** oral zinc (10 mg/day for <12 mo, 20 mg/day for ≥12 mo) reduces mortality among children with clinically defined severe pneumonia.

5- Antibiotics:

A: **For infant less than 2 m**:

a: First line: Ampicillin (100-200mg/kg/day) + **gentamicin** (5-7.5mg/kg/day).

b: Second line: If the patient has critical condition or above mention antibiotics do not respond in 48 hour, start **Ampicillin** 100-200mg/kg/day **+ 3rd generation cephalosporin** (**Ceftriaxone** 50 -75mg/kg/day or **Cefotaxime** 100mg /kg /day). For dosage of neonates see NNSepsis.

B: **For 2 m -5 y old children:**

a: First line: **Ampicillin** (or other penicillin) as above dosage. For mildly ill children who do not require hospitalization, high doses of amoxicillin (80-90 mg/kg/24 hr) is recommended.

b: **Second line**: **Ceftriaxone or cefotaxime**.

C: **For children older than five year:**

a- First line: macrolids (**Erythrocine** 50mg /kg/day**, clarithromycine** 15mg/kg/day**, Azithromycin** 10mg/kg 1st day then 5mg/kg/day**).** In adolescents, a respiratory fluoroquinolone (levofloxacin, moxifloxacin) may be considered as an alternative.

b- Second line: **Ceftriaxone or cefotaxime**.

D: **Third line antibiotic in all ages:** For suspected cases of staphylococcal pneumonia or cases not improve in 48 hour with above mention antibiotics, add **cloxacillin** (50-

100mg/kg/day) or **vancomycin** (40mg/kg/day 8hrly by IV infusion over 30min).

Duration of antibiotic therapy is 7-10 days. Treatment of suspected staphylococcal pneumonia should be 2-6 weeks till all clinical and radiological evidences of the disease disappear.

Bronchiolitis

Introduction

Bronchiolitis is a disease of small bronchioles with increased mucus production and occasional bronchospasm, sometimes leading to airway obstruction. The disease is more common in children younger than 2 years of age.

Bronchiolitis is usually a result of a viral respiratory tract infection. Respiratory syncytial virus is the most common underlying viral infection. Other viral pathogens such as influenza, parainfluenza, adenovirus, coronaviruses and rhinoviruses can also cause bronchiolitis. Mycoplasma is also implicated occasionally in children with lower respiratory tract infection (LRTI) and wheezing.

Grading of Bronchiolitis

Table 18: show grading of Bronchiolitis

	Mild	Moderate	Severe
Ability to feed	Ability to feed normally	Appear short of breath during feeds	May be reluctant or unable to feed
Respiratory effort	Little or no respiratory distress	Moderate distress with some chest wall retractions and nasal flaring	Severe distress with marked chest wall retractions, nasal flaring and grunting May have frequent or prolonged apneic episodes
Oxygen saturation	Saturations SaO_2 more than 92%	Saturations less than 92%, correctable with oxygen	Saturations less than 92%, may or may not be correctable with oxygen

Essential of diagnosis

A- Clinical Features

- H/O upper respiratory infection
- First attack of wheeze and cough maybe accompany with low grade fever, tachypnea, retraction, cyanosis and nasal flaring.
- Age less than 2year.
- Fall and winter season

B-Investigations

1-Chest radiographs frequently show signs of lung hyperinflation, including increased lung lucency and flattened or depressed diaphragms.

2-It is important to assess oxygenation in severe cases of bronchiolitis. Pulse oximetry is adequate for monitoring oxygen saturation.

3-Antigen tests (usually by immunofluorescence or enzymelinked immunosorbent assay [also referred to as *ELISA*]) of nasopharyngeal secretions for RSV, parainfluenza viruses, influenza viruses, and adenoviruses are sensitive tests to confirm the infection.

4- Rapid viral diagnosis also is performed by polymerase chain reaction.

Indications for hospital admission are:

- Age < 6M.
- Respiration distress.
- Cyanosis.
- Poor oral intake.
- Apnea.
- CHD& CHF.

- Underling lung diseases.

Management of Bronchiolitis

1. Propped up position is effective.
2. Give **humidified and cold oxygen** 25- 50 % 2-4 lit/min. keep O_2 saturation >92%.

Indications for O_2 therapy are:

- **severe respiratory distress**
- **cyanosis**
- **Increased R R** (>70/min in infants less than 2month, >60/min in infants aged 2-12months and >50/min in children older than one year)
- O_2 saturation < 92%.

3. Nutrition and maintenance fluid should be undertaken.
4. Nebulization of ventolin or Adrenalin: 0.5ml of Ventolin or Adrenaline + 2-3ml N/S should be repeated after 20 min for three doses. If responses have been achieved then give 4- 6 hourly until improvement.
5. Nebulized hypertonic saline has been reported to have some benefit, and may shorten hospital length of stay.
6. Corticosteroid: **Hydrocortison** or **Prednisolon** can be used in resistant cases.
7. Antiviral: Ribavirin can be administered as a mist, 12-20hrs daily for 3-5 days. Indications are:

- Age < 2month
- CHD (congenital Heart Disease)
- Bronchopumonary dysplasia
- Hyaline membrane disease

- Severe or complicated illness
- Mechanical ventilated infant

8. Antibiotics: First line antibiotics that mentioned for pneumonia should be used.

Br. Asthma

Introduction

Asthma is a chronic inflammatory condition of the lung airways resulting in episodic airflow obstruction. This chronic inflammation is due to airways hyperresponsiveness.

Classification of Asthma severity

Table 19: Classification of Asthma severity

CLASSIFICATION	steps	Day symptom	Night symptom	FOR ADULTS AND CHILDREN AGE > 5 YEARS WHO CAN USE A SPIROMETER OR PEAK FLOW METER	
				FEV	PEF
Severe persistent	4	Continual	Frequent	≤ 60	>30
Moderate persistent	3	Daily	> 1/wk	>60 -<80	>30
Mild persistent	2	>2/wk, but <1time/day	> 2/m	≥ 80	20 – 30
Mild intermittent	1	≤2/wk	< 2/m	≥ 80	< 20

Essentials of diagnosis

Clinical Diagnosis of Asthma is depends on:

- Recurrent attack of cough and wheeze.
- Improvement of S/S with bronchodilator therapy.
- Family H/O Asthma.
- PEF variation >20% is consistent with asthma.

Investigations

1- Pulmonary function test:

Lung Function Abnormalities in Asthma are:

A-Spirometry (in clinic)

- Airflow limitation

- Low FEV1 (relative to percentage of predicted norms)

The guidelines cutoff criteria of FEV1 <80% and <60% of predicted for moderate and severe asthma, respectively

- FEV1/FVC ratio <0.80

-Bronchodilator response (to inhaled β-agonist) improvement in FEV1 ≥12% or ≥200 mL.

- Exercise challenge worsening in FEV1 ≥15%

B-Daily peak flow or FEV 1 monitoring: day to day and/or AM-to-PM variation ≥20%

Management of Br. Asthma

Management of life threatening attack (Cyanosis, silent chest, poor respiratory effort & altered sensorium)

All of the following should be used

1. **O_2 therapy**: Give humidified 3 - 6 lit / min to keep O_2 saturation more than 92 %.
2. **Adrenalin** (1:1000) 0.0ml/kg SC or IM.
3. **Nebulization of Salbutamol** (0.5%) 0.15mg /kg or 0.5cc + 2-3cc N/S can be repeated after each 20 min for three doses.
4. **Ipratropium bromide** 2 puffs every 20 min for 3 doses.
5. **Methylprednisolon** 1mg/kg/dose 6 hourly **or Hydrocortisom** 10mg/kg/dose IV 6 hourly.

Reassess after one hr

A: If the responses are good, continue all of the following drugs.

a. Nebulization of ventolin every 3- 4 hr for 48 hours.

b. Ipratropium bromide every six hours for 48 hours.

c. Methylprednisolon 1mg/kg/dose **or Hydrocortison** 10mg/kg /dose 6 hourly for 48 hour then Prednisolon 1-2 mg/kg /day for 3-7 days.

B: If no or partial responses are observed, continue to above treatment. And add:

a. Aminophyllin 5-6 mg/kg 10 loading IV infusion over 30 min then 0.5mg/kg/min.

b. Give IV fluid and correct acidosis.

C: If not respond after 2-4 hours then give:

a. Magnesium sulphate 50 mg/kg +50 CC 1/5 saline +D/w 5% over 30 min.

b. Mechanical ventilation, Hiliox and in resistant case general anesthesia.

Management of Moderate to sever attack
(Fast breathe chest, in drawing, wheezing, difficult in speech and feeding, paradoxical pulse)

Give all of the following

1. **O2 therapy**.
2. **Nebulization of ventolin** or **Adrenalin** sc.
3. **Methyl Prednisolon or Hydrocortison or Prednisolon**

Assess after one hour

A: if no responses have been occurred then add.

a. Ipratropium bromide.

b. Aminophyllin.

B: If not respond in 2 -4 hours then add **Magnesium sulfate**.

Management of Mild attack
(Fast breathe, wheezing no chest in drawing).

1. **Nebulization of ventolin**: Should be repeated after each 20 min for three doses if the response is poor. If the symptoms resolved, keep the patient for 4 hours and then discharge.
2. If do not improved after one hour treat **as moderate asthma**.

Antibiotic: First line antibiotic that mentioned for pneumonia are useful to treat infection.

Croup

Introduction

The term croup refers to heterogeneous group of mainly acute and infectious process of larynx that are characterized by a bark-like or brassy cough and maybe associated with hoarseness, inspiratory stridor, and respiratory distress. Croup syndrome including:

- Laryngotracheobronchitis (Viral croup)
- Epiglottitis
- Spasmodic croup
- Laryngitis
- Bacterial tracheitis

A-Laryngotracheobronchitis (Viral croup): caused by Parainfluenza type 1,2,3 (75%), influenza A and B,

adenovirus, respiratory syncytial virus, measle, mycoplasma pneumonia

Essentials of diagnosis

- Diagnosis is usually clinical. URTI 1-3day before appearance of S/S. Barking cough, hoarseness, inspiratory stridor, low grade fever are the manifestations.
- In suspected cases P/A neck x-ray is taken which show steeple sign

Indications for admission are:

- Stridor at rest
- Cyanosis
- Respiratory distress
- Drooling
- High grade fever
- Depressed mental status
- Poor oral intake

Management of Viral Croup

1- **ABCs** of resuscitation should be done.
2- **Nebulized Racemic epinephrine 2.25% 0.25-0.75ml in 3ml N/S or Nebulized Adrenaline** 0.1-0.3ml/kg of 1:1000 solution dilutions to a maximum of 5 mL is useful. In case of persistent stridor the dose can be repeated every 2–4 hours.
3- Give **humidified oxygen** 25- 50 % 2-4 lit/min. Keep O2 saturation above 92%.

4- **Dexamethasone** 0.15-0.6mg/kg IM for single dose. Or oral Dexamethazone 0.15mg/kg appears equally effective.
5- I.V maintenance fluid is given, if needed.
6- **Antibiotic:** Ampicillin 100-200mg /kg/day for 7-10 days to treat secondary bacterial infection.

B- Acute Epiglottitis: Caused by Haemophilus influenza type b, Streptococcus pyogenes, Staphylococcus aureus.

Essentials of diagnosis

- Diagnosis is usually clinical. This dramatic potentially lethal condition characterized by acute rapidly progressive of: High fever, sore throat, dyspnea, progressive respiratory distress, drooling, hyperextended neck, tripod position, air hunger and restless followed by cyanosis and coma, stridor is a late finding and barking cough is rare.
- Lateral neck x-ray is useful for suspected cases.
- Visualization of swollen epiglottis (cherry-red) by direct laryngoscopy is diagnostic if the concern for epiglottitis still exists after radiography.

Management of Acute Epiglottitis

1. Stabilize ABCs of resuscitation.
2. Indotrachial Intubation should be done.
3. Maintenance IV fluid.
4. Antibiotic: Ceftriaxone 100mg/kg/day is given for 10days.

Part 4
Diarrheal and Infectious Diseases

Diarrhea

Introduction

Diarrhea is usually defined as passage of three or more loose or watery stools in a 24-hour period a loose stool being one that would take the shape of a container. However, for practical purposes, it is the recent change in consistency and character of stool and its water content rather than the number of stools that is important.

Acute Watery Diarrhea: It refers to diarrhea that begins acutely, lasts for less than 14 days, with passage of frequent loose or watery stools without visible blood. Vomiting may occur and fever may be present.

Dysentery: It is the term used for diarrhea with visible blood and mucus. It is often associated with fever and tenesmus.

Persistent Diarrhea: The World Health Organization (WHO) has defined persistent diarrhea (PD) as a diarrheal illness with passage of three or more loose stools of presumed infectious etiology, starting acutely and lasting for more than 14 days.The episode may begin either as acute watery diarrhea (AWD) or as dysentery. Marked weight loss is common. When there is some or severe dehydration, persistent diarrhea is classified as 'severe persistent diarrhea'.

Intractable diarrhea of infancy: Often begins before the age of 3 months with more than three liquid stools lasting for

more than 2 weeks under 1 year of age with either weight loss or no weight gain during this period.

Chronic diarrhea: is defined as diarrhea greater than 2 weeks duration, with an insidious onset and usually due to noninfectious cause. Almost all patients need a complete workup for underlying malabsorptive state.

Etiology

In developing countries, the organisms most frequently associated with AWD include enterotoxigenic *Escherichia coli* (ETEC), enteropathogenic *Escherichia coli* (EPEC), *Shigella dysenteriae* and *Campylobacter jejuni.* Rotavirus is a common cause of severe diarrhea, vomiting and fever leading to rapid dehydration. *Vibrio cholerae* is an important organism in endemic areas and during epidemics. Nontyphoidal Salmonella is a common organism in areas where commercially processed foods are widely used and in hospital outbreaks. Most of these organisms produce watery diarrhea. The main cause of acute dysentery is *S. dysenteriae*,

C. jejuni and infrequently enteroinvasive *Escherichia coli* (EIEC) or *Salmonella.* Epidemics of dysentery are usually caused by *S. dysenteriae type 1. Entamoeba histolytica* can cause dysentery in adults but is a less common cause in young children. Diarrhea may also be caused by a number of antibacterial agents like ampicillin, cotrimoxazole, chloramphenicol, amoxicillin, clindamycin, etc. Pseudomembranous colitis is the most severe form of antibiotic associated diarrhea.

Common causes of persistent diarrhea include persistent infection with one or more enteric pathogens, secondary

malabsorption of carbohydrates and fat, intestinal parasitosis and infrequently dietary protein allergy/ intolerance.

Table 20: Assessment of hydration state

Clinical signs			
General condition	Well, alert	Restless, irritable	Lethargic or unconscious
Eyes	Normal	Sunken	Sunken
Thirst*	Drinks normally, not thirsty	Drinks eagerly, thirsty	Drinks poorly, not able to drink
Skin pinch	Goes back quickly	Goes back "slowly"	Goes back "very slowly"
Decide hydration status	The patient has "no signs of	If the patient has two or more signs, there is	If the patient has two or more signs, there is
Treatment plan	Plan A	Plan B	Plan C

* In a young infant less than 2 months of age, thirst is not assessed and decision regarding "some" or "severe dehydration" is made if "two" of the three signs are present.

Indications for hospital admission are:

- Severe dehydration.
- Shock
- Persistent vomiting
- Metabolic Acidosis
- Paralytic ileus.

Management of Acute Diarrhea

A - Fluid therapy: Asses hydration state and follows dehydration treatment. (See management of dehydration)

B - Nutritional management: should be done according to plan A rehydration (See management of dehydration).

C - Zinc: For infants < 6m old 10mg/day and for infants > 6m old 20mg/day should be given for 10-14 days.

D- Probiotic: *Saccharomyces boulardii* is effective in antibiotic-associated and in *C. difficile* diarrhea, and there is some evidence that it might prevent diarrhea in daycare centers. *Lactobacillus rhamnosus GG* is associated with

reduced diarrheal duration and severity, which reduction is more evident in cases of childhood rotavirus diarrhea.

E - Chemotherapy (Antibiotics and Anti parasitic)

1- Antibiotics:

Indications: Bloody diarrhea, suspected cholera, Infant less than 2 months, malnutrition, suspected bacteremia and immunodeficiency. Presence of poor sucking, abdominal distention, fever or hypothermia, fast breathing and lethargy or inactivity in well nourished or well hydrate infant point toward sepsis.

One of the following antibiotics can be used:

- **Cotrimoxazol:** 10mg/kg/day Trimethoprim in 2 divided doses for 5 days.
- **Nalidixic acid:** 50-60mg/kg/day in four divided doses for five days.
- **Ampicillin (parentral):** 100 -200mg /kg/day in four divided doses for five days (In persistent vomiting).
- **Third generation cephalosporin:** If dysentery or suspected bacteremia is present and above antibiotic didn't respond in 48 hours give **Ceftriaxone** 50-75 mg /kg/day or **cefixim** 10 mg/kg/day in single or 2 divided doses for five days.
- **Suspected Cholera** should be treated with one of the below mentioned antibiotics:

  - Tetracycline 50mg/kg/d in four divided doses for 3 days.

  - **Doxycycline** 5mg/kg/d in single dose. These tow antibiotic are used in over 9y old children.

  - Azithromycine 10mg/kg/d in one dose for 1-5 days.

- **Cotrimoxazol:** As above dosage for 3 days.
- **Furazolidone** 6mg/kg/day in four divided doses for 3 days.
- **Erythromycine** 50mg/kg/day in four divided doses for 3 days.
- **Ciprofloxacine** 10 - 20mg/kg/day IV or 20 - 30mg/kg/day orally in 2 divided doses for 3 days.

2- Anti parasitic:

a - For Amebiasis: Give one of the following:

- **Metronidazole :** 35- 50mg/kg/day in three divided doses for 7 - 10 days plus **Iodoquinol** 30-40mg/kg/day in three divided doses for 20 days or plus **Diloxanide furoat** 20 mg/kg/day in three divided doses for 7 days .
- **Tinidazole:** 50mg /kg once daily for three days plus diloxanide furoat or iodoquinol .
- **Secnidazole:** 25mg/kg OD for 3 days.

b- For Giardiasis: Give one of the following.

- **Tinidazole :** 50mg/kg once.
- **Nitazoxanide:**
  1-3 yr: 100 mg (5 mL) bid for 3 days
  4-11 yr: 200 mg (10 mL) bid for 3 days
  >12 yr: 500 mg bid for 3 days
- **Metronidazole:** 15 mg/kg/day in three divided doses for five days.
- **Secnidazole:** 25mg/kg once.

c- For Ascariasis: Give one of the following.

- **Albendazole 400mg** single dose or 200mg in children 1-2 year of age.
- **Mebendazole** 100mg BID for 3 days or 500mg once.
- **Pyrantel Pamoat** 11mg/kg a single dose.

d – For Hookworm : Give one of the following.

- **Albendazole 400mg** single dose or 200mg in children 1-2 year of age.
- **Mebendazole** 100mg BID for 3 days or 500mg once **Pyrantel Pamoat** 11mg/kg OD for 3 days.

c – For Enterobiasis: Give one of the following:

- **Albendazol** 400mg single dose for all ages repeated in 2w.
- **Mebendazole** 100mg single dose repeated in 2w.
- **Pyrantel Pamoat** 11mg/kg single dose repeated in 2w.

d – For Trichuriasis: Give one of the following:

- **Albendazole 400mg** single dose or 200mg in children 1-2 year of age.
- **Mebendazole** 100mg BID for 3 days or 500mg once.
- **Nitazoxanide:**

  1-3 yr: 100 mg (5 mL) bid for 3 days
  4-11 yr: 200 mg (10 mL) bid for 3 days
  >12 yr: 500 mg bid for 3 days

e – Hymenolepiasis(H.nana): Give one of the following:

- **Praziquantel** 25mg/kg once.
- **Niclosamide** 50mg/kg once.
- **Nitazoxanide:**

  1-3 yr: 100 mg (5 mL) bid for 3 days

4-11 yr: 200 mg (10 mL) bid for 3 days
>12 yr: 500 mg bid for 3 days

f- T. saginata : Give one of the following:

- **Praziquantel** 25mg/kg once.
- **Niclosamide** 50mg/kg once.
- **Nitazoxanide:**

1-3 yr: 100 mg (5 mL) bid for 3 days
4-11 yr: 200 mg (10 mL) bid for 3 days
>12 yr: 500 mg bid for 3 days

E – Management of vomiting:

a: Stop ORT for 10 minutes and then restarts cold water or ORS by spoon, slowly.

b: If vomiting is persistent **Metochlopramide** (0.1 – 0.2mg/kg I M) are given.

c: If persistent vomiting isn't stopped with above management give required fluid through I.V.

F- Management of Abdominal distension:

a: If bowel sounds are present and abdominal distension is mild no specific treatment is needed.

b: If bowel sounds are not present and abdominal distension is gross, manage as follow:

- Oral intake should be stopped (NPO).
- Put NGT and aspirate stomach intermittently.
- **KCL** (1-3mEq/kg) or (1-3cc/kg) should be diluted in IV fluid (3-4mEq/100cc) and administered no faster than as rate of 0.5mEq/kg/hr.
- Suspected septicemia should be treated with appropriate antibiotic.

G - Management of suspected symptomatic metabolic acidosis:

a: $NaHCO_3$: 1-3mEq/kg (1-3ml/kg of 7.5% $NaHCO_3$) with I.V fluid over 8-12 hour.

b: In severe cases $NaHCO_3$ 1-2mEq/kg/dose should be diluted 1:1 with sterile water and give over 5-10 min (1ml/min).

c: Don't add **$NaHCO_3$** with **calcium** containing solution (like Ringer lactate).

Management of Persistent Diarrhea

1- Give fluids according to treatment plan B or C, as appropriate.

2- Give micronutrients and vitamins: About twice recommended daily allowance of supplemental multivitamins and minerals are to be given for at least 2–4 weeks (special attention to be given for vitamin A (200,000 units for children > 12 months or 1,00,000 IU for infants elemental zinc 10 mg/day from 2 months and 20 mg/day above 6 months of age for 14 days. Folic acid (1 mg/day), elemental copper (0.3 mg/kg/day) and vitamin D (200–400 U/day) are recommended.

3- Feeding:

A. Infants aged < 6 months

• Encourage exclusive breastfeeding. Help mothers who are not breastfeeding exclusively to do so.

• If the child is not breastfeeding, give a breast milk substitute that is low in lactose, such as yoghurt, or is

lactose-free. Use a spoon or cup; do not use a feeding bottle. Once the child improves, help the mother to re-establish lactation.

B. Children aged ≥ 6 months

Feeding should be restarted as soon as the child can eat. Food should be given six times a day to achieve a total intake of at least 110 calories/kg per day. Many children will eat poorly, however, until any serious infection has been treated for 24–48 h. These children may require nasogastric feeding initially.

Two recommended diets

There are two kinds of diets recommended for children and infants aged > 6 months with severe persistent diarrhoea. If there are signs of dietary failure (see below) or if the child is not improving after 7 days of treatment, the first diet should be stopped and the second diet given for 7 days.

Successful treatment with either diet is characterized by:

- Adequate food intake
- Weight gain
- Fewer diarrhoeal stools
- Absence of fever.

First diet for persistent diarrhoea: a starch-based, reduced-milk (low-lactose) diet

The diet should contain at least 70 calories/100 g, provide milk or yoghurt as a source of animal protein, but no more than 3.7 g lactose/kg per day and should provide at least 10% of calories as protein. The following example provides 83 calories/100 g, 3.7 g lactose/kg per day and 11% of calories as protein:

- Full-fat dried milk (or whole liquid milk: 85 ml) 11 g

- Rice 15 g
- Vegetable oil 3.5 g
- Cane sugar 3.0 g
- Water to make up 200 ml

Second diet for persistent diarrhoea: a reduced-starch (cereal) no-milk (lactose-free) diet

The diet should contain at least 70 calories/100 g and provide at least 10% of calories as protein (egg or chicken). The following example provides 75 calories/100 g:

- Whole egg 64 g
- Rice 3 g
- Vegetable oil 4 g
- Glucose 3 g
- Water to make up 200 ml

Finely ground, cooked chicken (12 g) can be used in place of egg to give a diet providing 70 calories/100 g

4- Management of Infectious Persistent Diarrhea are shown in table

Table 21 .Management of Infectious Persistent Diarrhea

	FACTOR	INDICATIONS	DOSAGE	DURATION
Antibiotics	Trimethoprim-sulfamethoxazole	*Salmonella* spp., *Shigella*	10-50 mg/kg/day in 2 divided doses–daily os	7 days
	Azithromycin	*Shigella*	1° day: 12 mg/kg/day once–daily os 2°-5° days: 6 mg/kg/day once–daily os	5 days
	Ciprofloxacin		20-30 mg/kg/day in 2 divided doses–os or iv	7 days
	Ceftriaxone		50-100 mg/kg/day once–im or iv	7 days
	Erythromycin	*Campylobacter*	50 mg/kg/day in 2-3 divided doses–os	7 days
	Metronidazole	*Giardia, Entamoeba*	20-30 mg/kg/day in 2-3 divided doses–os	7 days Small intestinal bacterial overgrowth
Antiparasitic	Nitazoxanide Albendazole	*Amebiasis, Giardiasis, Cryptosporidiosis* and helminth infections	100 mg every 12 hr for children ages 12-47 mo 200 mg every 12 hr for children ages 4-11 yr 500 mg every 12 hr for children older than 11 yr 400 mg once	3 days
Probiotics	*Lactobacillus* GG		$1\text{-}2 \times 10^{11}$–$1 \times 10^{11}$ CFU/day–os	For a minimum period of 7 days or until diarrhea stopped
	Saccharomyces boulardii		1×10^{10} germs live (500 mg)/day–os	For a minimum period of 7 days or till diarrhea stopped
Human serum immunoglobulin		Severe *Rotavirus* diarrhea	300 mg/kg single oral administration	
Antisecretory	Racecadotril	Secretory diarrhea	1.5 mg/kg every 8 hr–os	For a minimum period of 7 days or till diarrhea stopped
Adsorbents	Diosmectite		3-6 g every 12-24 hr–os	5 days

Malaria

Introduction

Malaria is caused by the protozoan parasite of the genus *Plasmodium* through bite of female anopheles mosquito. The four *Plasmodium* species that infect humans are *P. falciparum*, *P. vivax*, *P. ovale* and *P. malariae* of which the first two are the main causative agents in our country. In 2004 *P. knowlesi* (a primate malaria species) was also shown to cause human malaria, and cases of *P. knowlesi* infection have been documented in Malaysia, Indonesia, Singapore, and the Philippines.

Essential of diagnosis

A. Clinical features

1- Uncomplicated Malaria:

- Residence in or travel to an endemic area.
- Cyclic paroxysms of chills, fever, and intense sweating.
- Headache, backache, cough, abdominal pain, nausea, vomiting, diarrhea.
- Splenomegaly, anemia.

2- Complicated or Severe malaria is characterized by one or more of the following clinical or laboratory features:

- Impaired consciousness or non-arousable coma
- Prostration so that the patient is unable to walk or sit up without assistance
- Failure to feed
- Multiple convulsions, more than two episodes in 24 hours
- Deep breathing, respiratory distress (acidotic breathing)
- Circulatory collapse or shock, systolic blood pressure <50 mm Hg in children
- Clinical jaundice plus evidence of other vital organ dysfunction
- Hemoglobinuria
- Abnormal spontaneous bleeding
- Pulmonary edema (radiological)

The laboratory features include:

- Hypoglycemia (blood glucose <40 mg/dl)
- Metabolic acidosis (plasma bicarbonate <15 mmol/L)

- Severe normocytic anemia (Hemoglobin <5 g/dl, PCV <15%)
- Hyperparasitemia (>2% or 100,000/mL)
- Renal impairment (serum creatinine >3 mg/dl)
- Hyperlactemia (lactate >5 mmol/L)

B. Investigations

1- Microscopic diagnosis: Light microscopy of well stained (Giemsa-stained smears of peripheral blood) thick and thin films by a skilled microscopist has remained the 'gold standard' for diagnosis.

2- Rapid Diagnostic Tests (RDT): These are immunochromatographic tests (ICT) to detect *plasmodium* specific antigens in blood sample.

3- PCR: Polymerase chain reaction is even more sensitive than microscopy but is technically more complex.

Indications for hospital admission are:

- **Altered conscious and behavior.**
- **Convulsion.**
- **Difficulty in breathing.**
- **Sings of shock.**
- **Decreased urine, dark or red color urine.**
- **Sever pallor.**
- **Spontaneous bleeding**
- **vomit every think**

A – Management of Uncomplicated Malaria

1. **P.vivax: Chloroquine phosphate** should be used 10mg base /kg orally, follow by 5mg base/kg at 6 hour, 24hours, 48 hour or 10 mg base /kg on day 1, 10mg base /kg on day 2 and 5mg base /kg on day 3.

Alternatively: Amodiaquine can be used as **Chloroquine.**

2. **P. falciparum** or **Chloroquine resistant** P.vivax one of the following regimes:

A. Atovaquone-proguanil (Malarone)

Adult tab = 250 mg atovaquone/100 mg proguanil
Pediatric (ped) tab = 62.5 mg atovaquone/25 mg proguanil
5-8 kg: 2 ped tabs PO qd . 3 days
9-10 kg: 3 ped tabs PO qd . 3 days
11-20 kg: 1adult tab PO qd . 3 days
21-30 kg: 2 adult tabs PO qd . 3 days
31-40 kg: 3 adult tabs PO qd . 3 days
> 40 kg: 4 adult tabs PO qd . 3 days

B. Artemether-lumefantrine (Coartem)

1 tablet = 20 mg artemether and 120 mg lumefantrine
A 3 day treatment schedule with a total of 6 oral doses is recommended for both adult and pediatric patients based on weight. The patient should receive the initial dose, followed by the second dose 8 hr later, then 1 dose PO bid for the following 2 days
5-<15 kg: 1 tablet per dose
15-<25 kg: 2 tablets per dose
25-<35 kg: 3 tablets per dose

≥35 kg: 4 tablets per dose

C: Quinine sulphate: 10mg/kg TID for 7days **plus** one of the following.

- **Doxycycline:** 4mg/kg /day BID for 7 Days . Indicated in children older than 8 y .
- **Tetracycline**: 25mg /kg /Day QID for 7 Days. Indicated in children older than 8y
- **Clindamycin :** 20mg /kg /day TID for 7 Days.
- **Fansider :** Pyramithamine 1mg/kg once

D: Mefloquine : 15mg /kg orally follow 10mg/kg 12hour later.

c: Arthemeter : 3.2mg/kg /day for one day follow by 1.6mg/kg for 5-6 days.

B-Management of Severe and Complicated Malaria

A.Artesunate 4 mg/kg by intravenous or intramuscular injection, followed by 2.4 mg/kg at 12 hr and 24 hr; continue injection once daily if necessary.

B.Artemether 3.2 mg/kg by immediate intramuscular injection, followed by 1.6 mg/kg daily

C.QUININE DHC: 20mg /kg (loading dose) should be diluted in 10 ml /kg 5% or 10% D/W I.V and given over 4 hour; follow 8 hour after starting the loading dose with 10mg/kg in 10ml/kg 5% or 10% D/W over 4 hour; repeat 8 hourly until the child can swallow oral Quinine sulphate

(10mg /kg TID) to completed 7 days treatment. If coma continues more than 48 hours reduce Quinine DHC dose to 7mg /kg/day. **Plus** one of the drugs which mentioned under **Quinine sulfate**.

Artesunate is the treatment of choice. Artemether should only be used if artesunate is unavailable. Quinine dihydrochloride should be given only when artesunate and artemether are unavailable.

Supportive treatment of Malaria:

For the treatment of fever, convulsion, coma, shock, dehydration and other see related topics.

Enteric Fever (Typhoid Fever)

Introduction

Typhoid fever is caused by *S. enterica* serovar Typhi (*S.* Typhi), a Gramnegative bacterium. A very similar but often less-severe disease is caused by *Salmonella* Paratyphi A and rarely by *S.* Paratyphi B (Schotmulleri) and *S.* Paratyphi C (Hirschfeldii).

Essentials of diagnosis

A.Clinical manifestations

Typhoid fever usually manifests as high-grade fever with a wide variety of associated features, such as generalized myalgia, abdominal pain, hepatosplenomegaly, abdominal pain, and anorexia . In children, diarrhea may occur in the earlier stages of the illness and may be followed by

constipation. The fever may rise gradually, but the classic stepladder rise of fever is relatively rare. In approximately 25% of cases, a macular or maculopapular rash (rose spots) may be visible around the 7th-10th day of the illness, and lesions may appear in crops of 10-15 on the lower chest and abdomen and last 2-3 days

B.Investigation

The mainstay of the diagnosis of typhoid fever is a positive result of culture from the blood, stool or, in some cases, from urine, CSF, or pus from a suppurative lesion.The classic Widal test measures antibodies against O and H antigens of *S.* Typhi but lacks sensitivity and specificity in endemic areas.

Indications for hospitalization and parenteral antibiotic therapy are:

- Persistent vomiting
- Sever diarrhea
- Abdominal distention
- Other complications

Management of Enteric fever

1. Bed rest may be required.
2. Low residue, soft and easy digestible diet should be continued unless the patient has abdominal distention or ileus.
3. Adequate hydration.
4. Management of fever.(See FWF)
5. Antibiotics:

Table 22: Antibiotic therapy for enteric fever

	Optimal Therapy			Alternative Effective Drugs		
SUSCEPTIBILITY	ANTIBIOTIC	DAILY DOSE (mg/kg/day)	DAYS	ANTIBIOTIC	DAILY DOSE (mg/kg/day)	DAYS
UNCOMPLICATED TYPHOID FEVER						
Fully sensitive	Chloramphenicol	50-75	14-21	Fluoroquinolone, e.g., ofloxacin or ciprofloxacin	15	5-7*
	Amoxicillin	75-100	14			
Multidrug-resistant	Fluoroquinolone	15	5-7	Azithromycin	8-10	7
	or					
	Cefixime	15-20	7-14	Cefixime	15-20	7-14
Quinolone-resistant†	Azithromycin	8-10	7	Cefixime	20	7-14
	or					
	Ceftriaxone	75	10-14			
SEVERE TYPHOID FEVER						
Fully sensitive	Fluoroquinolone, e.g., ofloxacin	15	10-14	Chloramphenicol	100	14-21
				Amoxicillin	100	
Multidrug-resistant	Fluoroquinolone	15	10-14	Ceftriaxone	60	10-14
				or		
				Cefotaxime	80	10-14
Quinolone-resistant	Ceftriaxone	60	10-14	Azithromycin	10-20	7
	Cefotaxime	80	10-14	Fluoroquinolone	20	7-14

Tuberculosis

Introduction

Tuberculosis is a granulomatous disease caused by *Mycobacterium tuberculosis*. There are 5 closely related mycobacteria in the *Mycobacterium tuberculosis* complex: *M. tuberculosis, Mycobacterium bovis, Mycobacterium africanum, Mycobacterium microti,* and *Mycobacterium canetti. M. tuberculosis* is the most important cause of tuberculosis disease in humans.

Essentials of diagnosis

A.Clinical features

Pulmonary Primary Complex

Pulmonary primary complex (PPC) is the most commonly encountered presentation in the outpatient setting. Cough

with some constitutional symptoms may or may not be present. Such symptoms with the persistence of a doubtful shadow on chest X-ray even after an appropriate course of antibiotics, in the presence of a contact in the household who is sputum-positive, must not be ignored.

Progressive Primary Disease

Progressive primary disease (PPD) is the result of progression of primary disease. Child may present with moderate to high-grade fever and cough. Expectoration of sputum and hemoptysis are usually associated with advanced disease and development of cavity or ulceration of bronchus. This type is usually present at adolescence but if present in infancy, HIV is to be ruled out.

Endobronchial Tuberculosis

Fever, troublesome cough with or without expectoration, dyspnea, wheezing or cyanosis may be present. Partial compression of the airway can lead to emphysema. Features of collapse may be present if a large airway is completely compressed.

Miliary Tuberculosis

Miliary tuberculosis is an illness characterized by heavy hematogenous spread and progressive development of innumerable small foci throughout the body. The disease is most common in infants and young children.

The onset of illness is often sudden. In the clinical symptoms child may have high-grade fever, dyspnea and cyanosis. The pulmonary findings are minimal in the form of fine crepitations. In addition, there may be hepatosplenomegaly. In another group, the onset may be insidious with the child appearing unwell, febrile and losing weight. Due to

involvement of meninges and brain, neurotuberculosis (TBM) may occur. Such children require a lumbar tap for examination of CSF to rule out TBM.

Pleural Effusion

Pleural effusion may be minimal associated with primary complex in young infants. Massive effusion occurs at a later age.

B. Investigation

1. Identification of M. tuberculosis by smear, culture or PCR.
2. Chest x-ray: Enlarged lymph nodes are usually seen in hilar or right paratracheal region. Adenopathy alone may be the sole manifestation of primary tuberculosis. Consolidation in progressive primary disease (PPD) is usually heterogeneous, poorly marginated with predilection of involvement of apical or posterior segments of upper lobe or superior segment of lower lobe. Collapse may occur if large airway is obstructed.
3. Tuberculin Skin Test (TST): Positive TST is as following.

INDURATION ≥5 MM

Children in close contact with known or suspected contagious people with tuberculosis disease

- Children suspected to have tuberculosis disease:

•Findings on chest radiograph consistent with active or previously tuberculosis disease

• Clinical evidence of tuberculosis disease

- Children receiving immunosuppressive therapy or with immunosuppressive conditions, including HIV infection

INDURATION ≥10 MM

- Children at increased risk of disseminated tuberculosis disease:

• Children younger than 4 yr of age

• Children with other medical conditions, including Hodgkin disease, lymphoma, diabetes mellitus, chronic renal failure, or malnutrition.

- Children with increased exposure to tuberculosis disease:

• Children born in high-prevalence regions of the world

• Children often exposed to adults who are HIV infected, homeless, users of illicit drugs, residents of nursing homes, incarcerated or institutionalized, or migrant farm workers

• Children who travel to high-prevalence regions of the world

INDURATION ≥15 MM

- Children ≥4 yr of age without any risk factors

4- Interferon-γ Release Assays: Two blood tests (T-SPOT.*TB* and QuantiFERON-TB) detect IFN-ã generation by the patient's T cells in response to specific *M. tuberculosis* antigens (ESAT-6, CFP-10, and TB7.7).

1. Latent TB (Positive skin test, no symptoms and normal chest radiography) should be treated with 9 month of Isoniazid 10mg/kg/day orally.

Management of Tuberculosis

2. Pulmonary TB and/ or Hilar lymphadenopathy should be manage by 2 month of Isoniazid 10 - 15mg / kg / day orally, Rifampin 10–20 mg/kg/day orally, Pyrazinamide 20-40 mg/kg/day orally and Ethabutol 20mg/kg/day orally

followed by Isoniazid and Rifampin for another 4 month (2HRZE/4HR).

3. CNS, Disseminated, Bone and Join TB are treated by 2 month of Isoniazid orally 10 - 15mg / kg / day, Rifampin 10–20 mg/kg/day orally, Pyrazinamide 20-40 mg/kg/day, Ethabutol 20mg/kg/day orally and Sreptomycin 20-40 mg/kg/day IM followed by Isoniazid and Rifampin for another 7-9 month (2HRZES/7-9HR).

4. Corticosteroid: Prednisolone 1-2kg/kg/day is administered in 1-2 divided doses orally for 4-6 weeks, followed by gradually tapering. It is indicated in the following conditions:

- TB Meningitis
- Miliary TB
- TB pleural effusion
- Endobroncheal TB
- TB Pericardial effusion

3. TB chemoprophylaxis:

A. Infant born to mother of TB:

If signs/symptoms, skin test and chest film are normal; give Isoniazid 5mg/kg/day for 6month.

B. Children in contact with active adult TB case:

It is suggested that children below 5 years of age in contact with adult sputum positive TB should receive 6 month of Isoniazid 5mg/kg/day.

Pertussis (whooping cough)

Introduction

Pertussis is an acute respiratory tract infection. *Bordetella pertussis* is the cause of epidemic pertussis and the usual cause of sporadic pertussis. *Bordetella parapertussis* is an occasional cause of sporadic pertussis.

Essentials of diagnosis

- Prodromal catarrhal stage (1–3 weeks) characterized by mild cough and coryza, but without fever.
- Persistent staccato, paroxysmal cough ending with a high-pitched inspiratory "whoop."
- Leukocytosis with absolute lymphocytosis.
- Diagnosis confirmed by PCR or culture of nasopharyngeal secretions.

Indications for Hospital admission are:

1. Life threatening paroxysms that has the following features:
    - Duration more than 45 sec.
    - Blue color change.
    - Bradycardia.
    - Low oxygen saturation not resolves spontaneously at the end of paroxysm.
    - No whoop at the end of paroxysm.
    - Post-tussive unresponsiveness.
4. Apnea
5. Cyanosis
6. Seizure
7. Encephalopathy
8. Complications

Management of Pertussis

1. General Measures:

- Providing adequate nutrition and hydration. Small feeding, tube feeding and TPN may be needed.
- Avoid factors aggravating cough.
- Cough suppressants are of little benefits.
- Corticosteroids reduce the severity disease but may mask signs of bacterial superinfection.
- Albuterol 0.3-0.5mg/kg/day in four doses has reduced the severity of illness but tachycardia is common when the drug is given orally, and aerosol or nebulization may precipitate paroxysms.

2. Specific Measures:

An antimicrobial agent is always given when pertusis is suspected or confirmed, primarily to limit the spread of infection and secondarily for possible clinical benefit.

- Azithromycin (for infants less than 6month the dose is 10mg/kg/day in a single dose for 5days; for ≥ 6month 10mg/kg/day on day 1 then 5mg/kg/d on day 2-5) is the preferred agent for most patients particularly neonates.
- Erythromycin 40-50mg/kg/d in 4 divided doses for 7-14days. It is not preferred for neonates.
- Clarithromycin 15mg/kg/day in 2 divided doses for 7 days. It is not recommended for neonates
- TMP-SMZ (Co-trimoxasole)TMP 8mg/kg/day in 2 divided doses for 14 days. Contraindicated at age <2m.

Measles (Rubeola)

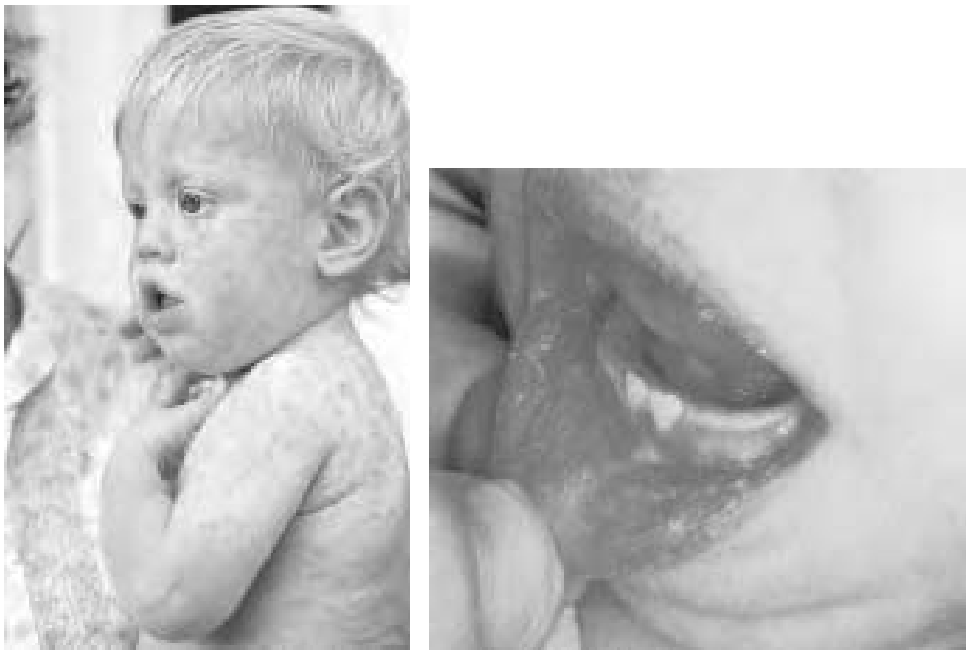
Introduction

Measles is a contagious disease characterized by catarrhal symptoms, followed by appearance of a typical rash.

Measles is caused by a RNA virus, of genus *morbillivirus*, of the family *paramyxoviridae*. Only one serotype of measles is known.

Essentials of diagnosis

- Exposure to measles 9–14 days previously.
- Prodrome of fever, cough, conjunctivitis, and coryza.
- Koplik spots (few to many small white papules on a diffusely red base on the buccal mucosa) 1–2 days prior to and after onset of rash.
- Maculopapular rash spreading down from the face and hairline to the trunk over 3 days and later becoming confluent.
- Leukopenia.



A B

Pictures14: A. Skin rash in measles B. Koplik spots

Management of Measles

Management of measles is supportive. Antiviral therapy is not effective. During the febrile period of illness, activity should be discouraged and fluid status should be maintained by the liberal provision of soft drinks and ice. Fever may be controlled with acetaminophen. Cough frequently is distressing and can be managed by the judicious use of common antitussive agents. Room humidification also is useful in controlling the cough and generally can be expected to make the patient more comfortable. As the fever disappears, a gradual return to normal activity is indicated.

Vaccination prevents the disease in susceptible exposed individuals if given within 72 hours Immune globulin (0.25 mL/kg intramuscularly; 0.5 mL/kg if immunocompromised)

will prevent or modify measles if given within 6 days. Vitamin A therapy is indicated for all patients with measles. Vitamin A should be administered once daily for 2 days at doses of 200,000 IU for children 12 mo of age or older; 100,000 IU for infants 6 mo through 11 mo of age; and 50,000 IU for infants younger than 6 mo of age. In children with signs and symptoms of vitamin A deficiency, a 3rd age-appropriate dose is recommended 2 through 4 wk after the 2nd dose.

Prevention: The current recommendations include a 1st dose at 12-15 mo of age (9mo in developing countries) , followed by a 2nd dose at 4-6 yr of age.

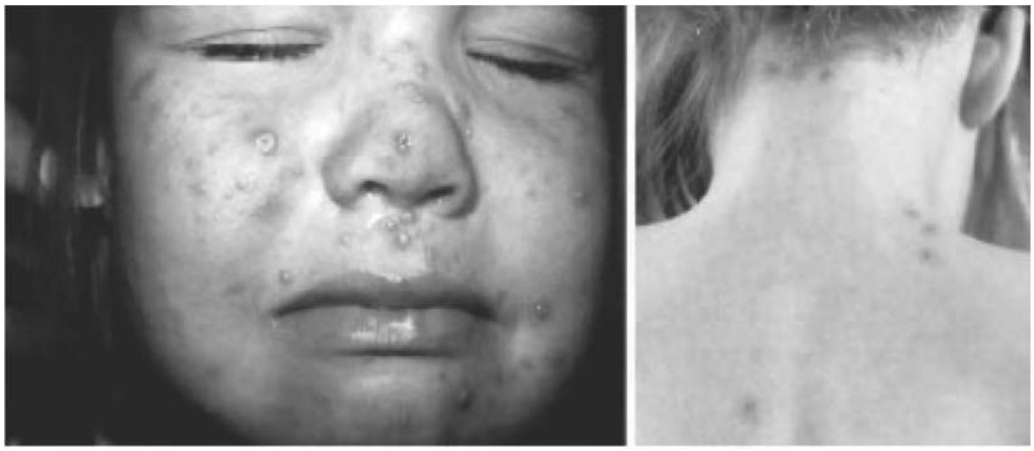
Chickenpox (Varicella)

Introduction

Chickenpox is a common childhood disease, caused by the *Varicella zoster* virus (VZV). Though generally considered as a benign infection, this can cause severe illness especially in newborns, immunocompromised children and elderly patients.

Essential of Diagnosis

- Exposure to varicella or herpes zoster 10–21 days previously; no prior history of varicella.
- Widely scattered red macules and papules concentrated on the face and trunk, rapidly progressing to clear vesicles on an erythematous base, pustules, and then crusts, over 5–6 days.



Picture 15: Skin rash in chickenpox

Management of Chickenpox (Varicella)

1. General Measures:

- Maintenance of hydration.
- Antipyretics to relieve fever and pain. Aspirin is contraindicated due to the risk of Reye syndrome.
- Antihistaminic: Diphinhydramin 1.2mg/kg every 6 hr or Hydroxyzine 0.5mg/kg every 6hr for the management of itching.
- Good hygiene: keep nails trimmed and skin clean.
- Topical or systemic Antibiotic may be needed for bacteria superinfection.
- The child should not attend school until no new lesions appear and all lesions have crusted.

2. Specific measures:

Oral Acyclovir 20mg/kg/dose four times a day for 5days; preferably within 24hr of the onset of exanthemas is recommended in the following conditions:

- Chronic cutaneous or pulmonary disorders.

- Individuals receiving corticosteroid therapy.
- Individuals receiving salicylate therapy.
- Possibly secondary case among household contact.
- Complicated cases.

Acute Otitis Media

Introduction

Otitis media is an infection associated with middle ear effusion (MEE) (a collection of fluid in the middle ear space) or with otorrhea (a discharge from the ear through a perforation in the TM or a ventilating tube).

Essential of diagnosis

A diagnosis of AOM requires (1) a history of acute onset of signs and symptoms, (2) the presence of MEE (Middle ear effusion), and (3) signs and symptoms of middle-ear inflammation. Elements of the definition of AOM are all of the following:

1. Recent, usually abrupt, onset of signs and symptoms of middle-ear inflammation and MEE
2. The presence of MEE that is indicated by any of the following:
   a. Bulging of the tympanic membrane
   b. Limited or absent mobility of the tympanic membrane
   c. Air-fluid level behind the tympanic membrane
   d. Otorrhea
3. Signs or symptoms of middle ear inflammation as indicated by either
   a. Distinct erythema of the tympanic membrane or

b. Distinct otalgia (discomfort clearly referable to the ear[s]) that results in interference with or precludes normal activity or sleep.

Management of Acute Otitis Media

A. Pain Management:

1. **Acetaminophen or Ibuprofen** is effective.
2. **Topical anesthetic drops** should be added if tympanic membrane is not perforated.
3. **Tympanocentesis** should be considered, if severe pain is present

B. Antibiotic Therapy:

First-line therapy:

1. Amoxicillin 90mg/kg/d upto 4gr/day. For children over age 2 year give for 5 day; under age 2y for 10days.
2. If Amoxicillin has caused a rash, give Cefuroxime, Cefdinir or Cefpodoxime.
3. If urticaria or other IgE mediated events have occurred, give Trimethoprim-Sulphamethoxazole or Azithromycin.

Second-line therapy:

1. Amoxicillin-Clavulanate (Augmentin) Amoxicillin at 90mg/kg/day. It can be the first-line drug for patient whose body temperature is $\geq 39C^{o}$ or / and have sever otalgia.
2. If Amoxicillin cause allergic symptoms, see recommendations above.

Third-line therapy:

1. Tympanocentesis is recommended to determine the cause.
2. Ceftriaxone two doses given IM 48hr apart, with option of third dose.

Acute Pharyngitis

Introduction

Pharyngitis refers to inflammation of the pharynx, including erythema, edema, exudates, or an enanthem (ulcers, vesicles).

A. Symptomatic Therapy:

1. **Acetaminophen or Ibuprofen** can relieve fever and sore throat.
2. Gargling with warm salt water is often comforting.
3. Anesthetic spray and lozenges can provide local relieve.

B. Antibiotic Therapy: Give one of the following:

1. Penicillin V 50-75mg/kg/day in 3 divided doses for 10 days.
2. Amoxicillin 50mg/kg/day (750mg fixed dose maximum 1gr) once daily for 10 days.
3. Azithromycin 12mg/kg once daily for 5 days.
4. Cephalexin 25-50mg/kg/day in divided doses for 10 days.
5. Benzathin penicillin 600000 U for children < 27kg, 1.2million U for larger children and adult as a single intramuscular dose.

Fever Without a Focus (F.W.F)

Introduction

Fever of acute onset, with duration of <1 wk and without localizing signs, is a common diagnostic dilemma in children <36 mo of age.

1. Manage fever as follow :

A: **Antipyretic**: If temperature is ≥ 39° C or irritability is observed give:

- **Paracetamol** 15 mg /kg /dose every4- 6 hourly. **or**
- **Ibuprofen** 10mg/kg 6-8hrly.

B: Sponging is recommended for febrile convulsion, febrile delirium and temperature more than 40° C.

C: Cold water immerse is indicated if Temperature ≥ 41°C.

2. **Antibiotics:** Give the following antibiotics according to the patients' ages. The dosages are mentioned under the management of Pneumonia.

A. For Neonates: Ampicillin + Gentamicin(or 3rd generation of cephalosporin) are indicated if the temperature is > 39° C. Acyclovir should be included if HSV infection is suspected.

B. For Infants 1- 3m old: Ampicillin + Gentamicin (or 3rd generation of cephalosporin) are indicated if the infant is ill appearing or the temperature is > 39° C.

C. For infant 3-36m old: Ampicillin or Ceftriaxone is indicated in the following conditions:

a: Toxic appearance .

b: Temperature ≥ 39° C and one of the following :

- TLC $\geq$ 15000 /mm^3.
- WBC >10 HPF in urine.
- Elevated absolute neutrophil count, band count, erythrocyte sedimentation rate (ESR), or C-reactive protein.

3. Antimalarial drugs: According to the protocol of Malaria.

Part 5
Fluid, Electrolytes & Acid-base disorders
Maintenance Fluid Therapy

1- Amount of maintenance fluid for Neonates:

A. Neonates > 1500gr Birth weight:

1st day ---------- 60ml/kg/day
2nd day ---------- 75ml/kg/day
3rd day ---------- 90 ml/kg/day
4th day ---------- 105 ml/kg/day
5th day ---------- 120 ml/kg/day
6th day ---------- 135 ml/kg/day
7th day and more -------- 150 ml/kg/day

B. Neonates < 1500gr Birth weight:

1st day ---------- 80ml/kg/day
2nd day ---------- 100ml/kg/day
3rd day ---------- 120 ml/kg/day
4th day ---------- 130 ml/kg/day
5th day ---------- 140ml/kg/day
6th day ---------- 150 ml/kg/day
7th day and more -------- 160 ml/kg/day

2- Composition of maintenance fluid for Neonates: N/5 + 10% or 5 % D/W is adequate IV fluid. Also we can use Prep solution.

All of the following should be add to prepare a solution named (Prep Solution) and administered every 4hr:

- **0.9% N/S 21ml/kg/day (Na=3mEq/kg/day)**
- **7.46%KCL 2ml/kg/day (K=2mEq/kg/day)**

- **10% D/W for > 1000gr birth weight and 5 % D/W for < 1000gr birth weight should be contained the remainder of fluid.**

Note: Neonates up to 48hr after birth don't need to Na, K and Calcium, so maintenance fluid should be contained of 10% D/W for > 1000gr birth weight and 5% D/W is preferred for < 1000gr birth weight .

3- **Amount of maintenance fluid for older infants and children:** The fluid should be calculated according to the body weight as the following:

- 3-10 kg ---------- 100ml/kg/day
- 10-20kg ---------- 1000ml + 50ml/kg/day for extra kg from 10kg.
- More than 20kg ---------- 1500 + 20ml/kg/day for extra kg from 20kg.

4- **Composition of maintenance fluid for older infants and children:**

- 1/2N/S (0.45% NaCl) + 5%D/W.
- 7.46% KCL 2ml/kg/day or 2ml per 100ml of maintenance fluid.

5. Conditions Affecting Maintenance Fluid Therapy:

A. Increase requirement:

- Phototherapy; fluid must be increased 20-40ml/kg/day.
- Radiant warmer; fluid requirement should be increased 40-80ml/kg/day.

B. Decrease requirement: Maintenance fluid can be decreased to 2/3 of normal in the following conditions:

- Birth asphyxia
- Pneumonia

- HMD
- CHF
- Meningitis

Dehydration

Introduction

Dehydration is net loss of body water.

Classification of Dehydration

1. Mild dehydration (<5% in an infant; <3% in an older child or adult): Normal or increased pulse; decreased urine output; thirsty; normal physical findings

2.Moderate dehydration (5-10% in an infant; 3-6% in an older child or adult): Tachycardia; little or no urine output; irritable/lethargic; sunken eyes and fontanel; decreased tears; dry mucous membranes; mild delay in elasticity (skin turgor); delayed capillary refill (>1.5 sec); cool and pale

3.Severe dehydration (>10% in an infant; >6% in an older child or adult): Peripheral pulses either rapid and weak or absent; decreased blood pressure; no urine output; very sunken eyes and fontanel; no tears; parched mucous membranes; delayed elasticity (poor skin turgor); very delayed capillary refill.

Table 23: Classification of dehydration

Characteristics			
Infants	Mild 1–5%	Moderate 6–9%	Severe ≥ 10% (> 15% shock)
Children	Mild 1–3%	Moderate 4–6%	Severe ≥ 7% (> 9% shock)
Pulse	Normal	Tachycardia	Tachycardia, weak pulse
Systolic BP	Normal	Normal-low	Orthostatic to shock
Urine output	Decreased	Decreased	Oliguria
Buccal mucosa	Slightly dry	Dry	Parched
Anterior fontanel	Normal	Sunken	Markedly sunken
Eyes	Normal	Sunken	Markedly sunken
Skin turgor/capillary refill	Normal	Decreased	Markedly decreased
Skin (< 12 months age)	Normal	Cool	Cool, mottling, acrocyanosis

Note: In a malnourished child, subcutaneous tissue is markedly reduced. Reliance on sunken eyebal, fontanel and loss of skin turgor in these children may lead to overestimation of dehydration. On the other hand, in chubby children dehydration may be underestimated. Thirst, dry mucosa, urine flow, metabolic acidosis, and circulatory status, therefore, are more reliable indicators of dehydration in these children.

Table 24: Assessment of hydration state according to IMNCI

Clinical signs			
General condition	Well, alert	Restless, irritable	Lethargic or unconscious
Eyes	Normal	Sunken	Sunken
Thirst*	Drinks normally, not thirsty	Drinks eagerly, thirsty	Drinks poorly, not able to drink
Skin pinch	Goes back quickly	Goes back "slowly"	Goes back "very slowly"
Decide hydration status	The patient has "no signs of dehydration"	If the patient has two or more signs, there is "some dehydration"	If the patient has two or more signs, there is "severe dehydration"
Treatment plan	Plan A	Plan B	Plan C

* In a young infant less than 2 months of age, thirst is not assessed and decision regarding "some" or "severe dehydration" is made if "two" of the three signs are present.

Indications for admission or IV fluid administration are:

- Sever dehydration
- Shock
- Persistent vomiting
- Paralytic ileus
- Metabolic acidosis

Management of Dehydration

Treat Mild, Moderate and Severe dehydration according to Plan A, B and C:

Plan (A): For the management of Mild dehydration:

Counsel the mother on the 4 rules of home treatment:

Give extra fluid, continue feeding, when to return and give zinc supplement.

1. Give extra fluid (as much the child will take)
   - Breast feed frequently and for longer at each feed.
   - If the child is exclusively breastfed, give ORS or clean water in addition to breast milk.
   - If the child is not exclusively breast fed, give one or more of the following: ORS solution, food based fluids (such as soup, rice water, and yoghurt drinks), or clean water.

It is especially important to give ORS at home when:

- The child has been treated with plan B or plan C during this visit.
- The child cannot return to a clinic if the diarrhea gets worse

➢ **Teach the mother how to mix and give ORS .Give the Mother 2 packets of ORS to use at home.**

➢ **Show the mother how much fluid to give in addition to the usual fluid intake:**

Up to two years..... 50 to 100 ml after each loose stool.

2 years or more .....100 to 200 ml after each loose stool.

Tell the mother to:

- Give frequent small sips from a cup.
- If the child vomits wait 10 minutes then continues but more slowly.
- Continue giving extra fluid until the diarrhea stops.

2. **CONTINUES FEEDING.**
3. **WHEEN TO RETURN.**
4. **Give Zinc Supplementation.**

Plan B: For the management of the Moderate or Some dehydration give ORS or IV fluid as below in 4hrs:

- The approximate amount of ORS or IV fluid is 75ml/kg.
- Use the children age according to the following chart only when you do not know the weight.

DETERMINE AMOUNT OF ORS TO GIVE DURING FIRST FOUR HOURS.

AGE	Up to 4 months	4month up to 2 years	12months up to 2years	2years up to 5 years
Weight	< 6kg	6 - <10kg	10- <12kg	12 – 19 kg
In ml	200 – 400	400 - 700	700 - 900	900 – 1400

- If the child wants more ORS than shown give more.
- For infants under six months who are not breastfed also give 100 – 200 ml can clean water during this period.

➢ SHOW THE MOTHER HOW TO GIVE THE ORS SOLUTION :

- Give frequent small sips from a cup.

- If the child vomits wait 10 minutes then continue but more slowly.
- Continue breastfeeding when ever the child wants.

➢ **AFTER FOUR HOURS :**

- Reassess the child and classify the child for dehydration.
- Select the appropriate plan to continue treatment.
- Begin feeding the child in clinic.

➢ **IF THE MOTHER MUST LEAVE BEFORE COMPLTING TREATMENT:**

- Show her how to prepare ORS solution at home.
- Show her how much ORS to give to finish four hour treatment at home.
- Give her enough ORS packets to complete rehydration also give her two packets as recommended in plan A.
- Explain the four rules of home treatment.

1. GIVE EXTRA FLUID.
2.CONTINUE FEEDING.
3.WHEN TO RETURN.
4.Give Zinc

} See plan A for

Plan C: For the Treatment of Severe dehydration quickly :

➢ Follow the arrows. IF Answer is YES GO ACROSS. IF NO GO DOWN.

START HERE:

Can you give intravenous (IV) Fluid immediately?

▬ Yes ▬▶ Start IV fluid immediately. If the child can drink gives ORS by mouth while the drip is set up give 10 ml/kg ringers lactate solution (or if not available, normal saline), Divided as follows:

AGE	First give 30ml/kg in :	Then give 70ml/kg in :
Infants (under 12months).	One (1) hours.	Five (5) hours.
Children 12months up to five (5) years.	Thirty (30) minutes.	2 ½ hours

Repeat once if radial pulse is still very weak or not detectable.

- Reassess the child every 1- 2 hours. If hydration status is not improving, give the IV drip more rapidly.
- Also give ORS (a bout five (5) ml /kg /hour) as soon as the child can drink: Usually after 3-4 hours (Infants) or 1-2 hours (Children).
- Reassess an infant after six (6) hours and a child after three (3) hours. Classify dehydration. Then choose the appropriate plan (A, B. or C) to continue treatment.

No

IS IV treatment available nearly within thirty30minuts?

▬ YES ▬▶ **Refer urgently to hospital for IV treatment.** If the child can drink provide the mother with ORS solution and show her how to give frequent sips during the tripe.

NO

Are you trained to use a naso gastric (NG) tube for rehydration?

Start rehydration by tube (or mouth) with ORS solution give 20 ml/hour for six hour (total of 120 ml/kg).
Reassess the child every 1-2 hours.
If hydration status is not improving after 3 hours send the child for IV therapy.
After 6 hours reassess the child classify dehydration then choose the appropriate plan (A, B or C) to continue treatment.

▬ Yes ▬▶

Hyponatremia

Introduction

Serum Na concentration less than 130mEq/l is called Hyponatremia.

Essentials of diagnosis

1-Clinical features

- Neurologic symptoms of hyponatremia include anorexia, nausea, emesis, malaise, lethargy, confusion, agitation, headache, seizures, coma, and decreased reflexes.
- Na concentration < 120mEq/l cause irritability, headache, lethargy and confusion.
- Na concentration < 115mEq/L cause seizure.

2- Investigation

- Serum Na concentration less than 130mEq/l

Management of Hyponatremia

1-Hypovolemic hyponatremia:

- Deficit + maintenance fluid from 5% dextrose+ 0.45% saline should be given as following:
- Half of the fluid over 8hr and the remainder over 16hr.
- The rise of serum Na concentration should not exceed 0.5-1mEq/L/hr or 20mEq/L/24hr.

2-Hypervolemic hyponatremia:

- The cornerstone of therapy is water and sodium restriction because these patients are volume-overloaded.
- Diuretics may help by causing excretion of both sodium and water.

3-Iso or Euvolumic hyponatremia:

- Therapy is directed at eliminating the excess water by restricting the amount of fluid.
- In severe hyponatremia (Na <120mEq/l) with symptoms of CNS, 4-6ml/kg of 3% Nacl over one hr is required.

Hypernatremia

Introduction

Hypernatremia is a sodium concentration >145 mEq/L, although it is sometimes defined as >150 mEq/L.

Essentials of diagnosis

1-Clinical features

- Most children with hypernatremia are dehydrated and show the typical clinical signs and symptoms.
- Probably because of intracellular water loss, the pinched abdominal skin of a dehydrated, hypernatremic infant has a "doughy" feel.
- Central nervous system symptoms are irritable, restless, weak, ICH and lethargic. Some infants have a high-pitched cry and hyperpnea.
- Alert patients are very thirsty, even though nausea may be present.

2- Investigation

- Serum Na concentration more than 145mEq/l

Management of Hypernatremia

- Most patients with hypernatremic dehydration do well with a fluid sodium concentration of approximately ½ normal saline (NS), but with a fluid rate that is only 20–30% greater than maintenance. Or using 5% dextrose with 0.2% saline to replace the calculated fluid deficit with maintenance fluid over 48 hours.
- Hypernatremia should not be corrected rapidly. The goal is to decrease the serum sodium by <12 mEq/L every 24 hr, a rate of 0.5 mEq/L/hr.
- If the serum [Na+]is not correcting appropriately, the free water deficit may be estimated as 4 mL/kg of free water for each milliequivalent of serum [Na+] above 145 and provided as 5% dextrose over 48 hours.

Hypokalemia

Introduction

Serum K level less than 3.5mEq/l is called Hypokalemia.

Essentials of diagnosis

1-Clinical features

- Weakness
- Hypotonia
- Abdominal distention
- Decreased bowel sound
- Paralytic ileus with levels <2.5 mEq/L
- Paralysis generally only at levels <2.5 mEq/L
- Hyporeflexia
- Apathy
- Arrhythmia
- Respiratory distress

2- ECG changes

- ST segment depressed
- T wave flattened or inverted
- U wave appearance
- Ventricular fibrillation may occur

3- Investigation

- Serum K concentration less than 3.5mEq/l

Management of Hypokalemia

- Because of the risk of hyperkalemia, intravenous potassium should be used very cautiously.
- Oral potassium 3mEq/kg/24hr is safer, albeit not as rapid in urgent situations.

- The dose of intravenous potassium is 0.5–1 mEq/kg, usually given over 1 hr and the IV preparation of K should not exceed 40mEq/L. .IV potassium indicated if oral intolerance, K<2.5mEq/L or cardiac arrhythmia are observed.
- For patients with excessive urinary losses, potassium-sparing diuretics are effective, but they need to be used cautiously in patients with renal insufficiency.

Hyperkalemia

Introduction

Serum potassium concentration > 5.5mEq/L is called Hyperkalemia. Hyperkalemia, because of the potential for lethal arrhythmias, is 1 of the most alarming electrolyte abnormalities.

Essentials of diagnosis

1-Clinical features

- The cardiac conduction system is usually the dominant concern (Heart block and arrhythmia).
- Some patients have paresthesias, weakness, and tingling, but cardiac toxicity usually precedes these clinical symptoms

2- ECG changes

- Electrocardiographic (ECG) changes begin with peaking of the T waves.
- This is followed, as the potassium level increases, by an increased P-R interval, flattening of the P wave, widening of the QRS complex, heart block and ventricular fibrillation.

3- Investigation

- Serum K concentration more than 5.5mEq/l

Management of Hyperkalemia

The treatment of hyperkalemia has 2 basic goals:

1-To stabilize the heart to prevent life-threatening arrhythmias.

2-To remove potassium from the body.

1-To stabilize the heart to prevent life-threatening arrhythmias. The following measure undertaken:

A- Mild hyperkalemia (Serum K= 5.5-6mEq/l):

- Stopping the intake of potassium
- Discontinue drugs (potassium sparing diuretics, Angiotensin-converting enzyme, inhibitors Angiotensin II blockers

B- Moderate hyperkalemia (Serum K=6-8mEq/l or peaked Twave)

- Glucose insulin infusion 0.5g/kg glucose with 0.3 u regular insulin/g glucose over 2hrs) and / or
- Sodium bicarbonate infusion 2mEq/kg over 5-10 min

C- Severe hyperkalemia (Serum K > 8mEq/l or ECG change)

- Calcium gluconate 10% 0.5ml/kg urgently administrated IV.
- This should be followed up with the measures as for moderate hyperkalemia
- Intravenous or nebulized Salbutamol also rapidly lowers serum potassium.

2- To removed potassium from the body:

- It is critical to begin measures that remove potassium from the body but none of these work quickly, it is important to start them as soon as possible.
- In patients who are not anuric, a loop diuretic increases renal excretion of potassium. A high dose may be required in a patient with significant renal insufficiency.
- Sodium polystyrene sulfonate (Kayexalate) is an exchange resin that is given either rectally or orally. Sodium in the resin is exchanged for body potassium, and the potassium-containing resin is then excreted from the body.
- Some patients require dialysis for acute potassium removal. Dialysis is often necessary if there is severe renal failure or if there is an especially high rate of endogenous potassium release. Hemodialysis rapidly lowers plasma potassium levels. Peritoneal dialysis is not nearly as quick or reliable, but it is usually adequate as long as the acute problem can be managed with medications and if the endogenous release of potassium is not extremely high.

Acid-Base Disturbances

- **Acidemia** is a blood pH below normal (<7.35) and **alkalemia** is a pH above normal (>7.45).
- An *acidosis* is a pathologic process that causes an increase in the hydrogen ion concentration, and an *alkalosis* is a pathologic process that causes a decrease in the hydrogen ion concentration.

Classification

1-Acidosis: pH<7.35

- Metabolic: increased acid or decreased in bicarbonate
- Respiratory: increased PCO2

2-Alkalosis: pH>7.45

- Metabolic: increased bicarbonate or loss of H+
- Respiratory: decreased PCO_2

Table 25. Initial and compensatory changes

Acid-Base disturbances	PH	Initial change	Compensatory change
Metabolic Acidosis	↓	↓ HCO3	↓ PCO2
Respiratory Acidosis	↓	↑ PCO2	↑ HCO3
Metabolic Alkalosis	↑	↑ HCO3	↑PCO2
Respiratory Alkalosis	↑	↓PCO2	↓ HCO3

Metabolic Acidosis

Introduction

This type of acidosis occurs due to excess loses of bicarbonate or excess blood acids.

Metabolic acidosis with normal anion gap is due to excess loses of bicarbonate; and if the anion gap is increased excess blood acid is the cause.

- Anion gap= Na – ($Cl+HCO_3$)
- normal anion gap = 4-11

Essentials of diagnosis

1-Clinical features

- The clinical manifestations of the acidosis are related to the degree of acidemia; patients with appropriate respiratory compensation and less severe acidemia have fewer manifestations than those with a concomitant respiratory acidosis.
- The normal respiratory response to metabolic acidosis—compensatory hyperventilation—may be subtle with mild metabolic acidosis, but it causes discernible increased respiratory effort with worsening acidemia.
- Deep and rapid respiration (Kussmaul respiration) is seen in with severe metabolic acidosis.
- Severe acidosis (serum pH <7.2)may cause confusion, drowsiness, coma, myocardial depression, arrhythmia and shock.

2- Investigation

- Initially low blood pH and decreased bicarbonate level followed by decreased PCO_2.

Management of Metabolic Acidosis

- The most effective therapeutic approach for patients with a metabolic acidosis is repair of the underlying disorder, if possible.
- The administration of insulin in diabetic ketoacidosis and the restoration of adequate perfusion in lactic acidosis eventually result in normalization of the acid-base balance.
- In other diseases, the use of bicarbonate therapy is indicated
- Oral or intravenous base can be used in acute metabolic acidosis; intravenous therapy is generally used when a rapid response is necessary. Sodium bicarbonate may be given as a bolus, usually at a dose of 1 mEq/kg, in an emergency situation.

Respiratory Acidosis

Introduction

A respiratory acidosis characterized by reduced arterial blood pH, elevated PCO2 and an increase in plasma HCO3 concentration. These change occurs due to hypoventilation and inappropriate increase in blood carbon dioxide (PCO2).

Essentials of diagnosis

1-Clinical features

- Patients with a respiratory acidosis are often tachypneic in an effort to correct the inadequate ventilation.
- Exceptions include patients with a respiratory acidosis resulting from central nervous system depression and

respiratory failure secondary to fatigue of the respiratory muscles.

- The potential central nervous system manifestations of respiratory acidosis include anxiety, dizziness, headache, confusion, asterixis, myoclonic jerks, hallucinations, psychosis, coma, and seizures.

2- Investigation

- Initially low blood pH and elevated PCO_2 level followed by increased bicarbonate.

Management of Respiratory Acidosis

- Respiratory acidosis is best managed by treating the underlying etiology.
- Naloxone to a patient with a narcotic overdose. The response is very rapid.
- The child with pneumonia may require a number of days of antibiotic therapy before the respiratory status improves.

Metabolic Alkalosis

Introduction

Metabolic alkalosis is manifested by an elevated arterial pH, an increase in the serum [HCO3], and an increase in PCO2 as a result of compensatory alveolar hypoventilation.

Metabolic alkalosis occurs due to excessive loss of acid or increased blood base. In children it is most commonly secondary to emesis or diuretic use.

Essentials of diagnosis

1-Clinical features

- Children with chloride-responsive causes often have symptoms related to volume depletion, such as thirst and lethargy. In contrast, children with chloride-unresponsive causes may have symptoms related to hypertension.
- Most patients with a metabolic alkalosis have hypokalemia, and symptoms may be related to the hypokalemia.
- During alkalemia, the ionized calcium concentration decreases as a result of increased binding of calcium to albumin. The decrease in the ionized calcium concentration may cause symptoms of tetany (carpopedal spasm).
- Arrhythmias are a potential complication of a metabolic alkalosis

2- Investigation

- Initially increased blood pH and elevated bicarbonate level followed by increased PCO_2.

Management of Metabolic Alkalosis

- In children with a mild metabolic alkalosis ([HCO3-] < 32), intervention is often unnecessary.
- Intervention is usually necessary in children with moderate or severe metabolic alkalosis.
- Administration of sufficient sodium chloride and potassium chloride to correct the volume deficit and the

potassium deficit is necessary to correct the metabolic alkalosis.

- Nasogastric suction may be decreased or discontinued.
- Alternatively, the addition of a gastric proton pump inhibitor reduces gastric secretion and losses of HCl.
- Diuretics are an important cause of metabolic alkalosis and, if tolerated, they should be eliminated or the dose reduced.
- Adequate potassium supplementation or the addition of a potassium-sparing diuretic is also helpful in a child with a metabolic alkalosis caused by diuretics.
- **If chloride deficit:** Replace volume deficit by N/S.
- **If chloride-unresponsive**: Give K replacement or mineralcorticoid antagonist (Aldactone).
- **If volume overload and unresponsive** : give Acetazolamide
- **Prolonged gastric suctioning**: Use H2 blocker or proton pump inhibitor.

Respiratory Alkalosis

Introduction

Respiratory alkalosis is characterized by elevated arterial blood pH, reduced PCO2 and reduced plasma HCO3.

A respiratory alkalosis occurs due to inappropriate reduction in the blood carbon dioxide concentration. This is usually secondary to hyperventilation

Essentials of diagnosis

1-Clinical features

- Acute respiratory alkalosis may cause chest tightness, palpitations, lightheadedness, circumoral numbness, and paresthesias of the extremities.
- Less common manifestations include tetany, seizures, muscle cramps, and syncope.
- The paresthesias, tetany, and seizures may be partially related to the reduction in ionized calcium that occurs because alkalemia causes more calcium to bind to albumin

2- Investigation

- Initially increased blood pH and reduced PCO_2 level followed by decreased bicarbonate.

Management of Respiratory Alkalosis

- Treatment focuses on the underlying disease.
- Along with reassurance, patients with psychogenic hyperventilation may benefit from benzodiazepines.
- During an acute episode of psychogenic hyperventilation, rebreathing into a paper bag increases the patient's PCO_2.

Part 6
Nutritional Disorders

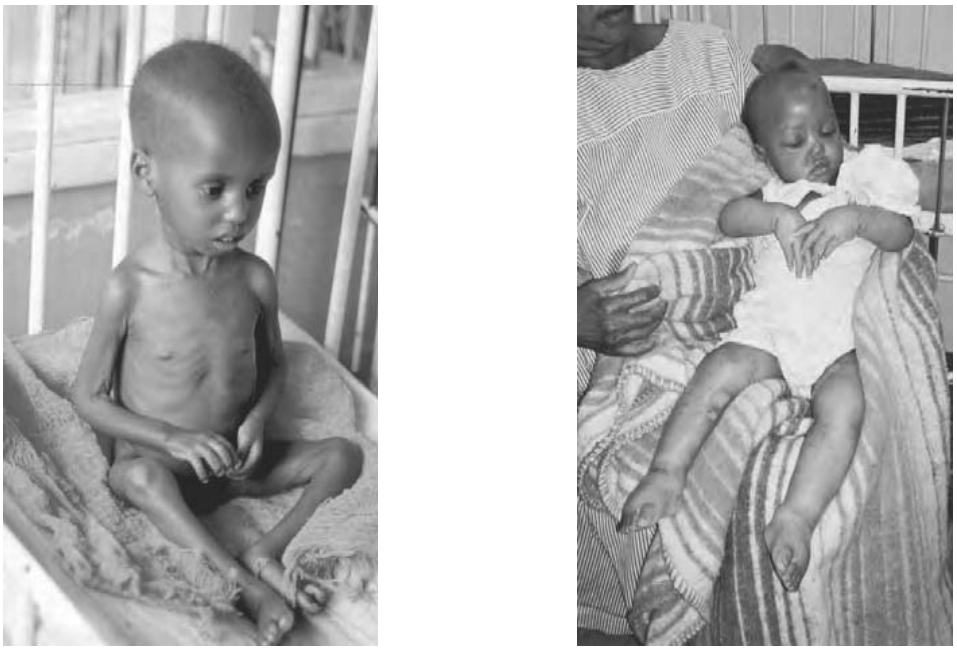
Severe Acute Malnutrition

Introduction

Severe acute malnutrition is defined as severe wasting and/or bilateral edema.

Essentials of Diagnosis

- Weight-for-length/height < -3SD (wasted) or
- Mid-upper arm circumference < 115 mm(in children ages 6-59 mo, or
- Oedema of both feet (kwashiorkor with or without severe wasting).



Pictures 16: Marasmus with severe wasting (left) Kwashiorkor with edema (right)

Table 26. Clinical signs of Malnutrition

SITE	SIGNS
Face	Moon face (kwashiorkor), simian facies (marasmus)
Eye	Dry eyes, pale conjunctiva, Bitot spots (vitamin A), periorbital edema
Mouth	Angular stomatitis, cheilitis, glossitis, spongy bleeding gums (vitamin C), parotid enlargement
Teeth	Enamel mottling, delayed eruption
Hair	Dull, sparse, brittle hair, hypopigmentation, flag sign (alternating bands of light and normal color), broomstick eyelashes, alopecia
Skin	Loose and wrinkled (marasmus), shiny and edematous (kwashiorkor), dry, follicular hyperkeratosis, patchy hyper- and hypopigmentation (crazy paving or flaky paint dermatoses), erosions, poor wound healing
Nails	Koilonychia, thin and soft nail plates, fissures, or ridges
Musculature	Muscle wasting, particularly buttocks and thighs; Chvostek or Trousseau sign (hypocalcemia)
Skeletal	Deformities, usually as a result of calcium, vitamin D, or vitamin C deficiencies
Abdomen	Distended: hepatomegaly with fatty liver; ascites may be present
Cardiovascular	Bradycardia, hypotension, reduced cardiac output, small vessel vasculopathy
Neurologic	Global developmental delay, loss of knee and ankle reflexes, impaired memory
Hematologic	Pallor, petechiae, bleeding diathesis
Behavior	Lethargic, apathetic, irritable on handling

Indications for IPD care

1- Infants less than 6 month of age or < 65cm length: Any of the following:

- Weight-for-length/height < -3SD
- Visible severe wasting
- Bilateral edema
- Weight-for-length/height between -2SD and -3SD and one of the followings :
  - Difficulties of breastfeeding or too weak to suckle
  - Not enough breast milk of mother

2- Children equal to or more than 6 month of age or ≥ 65 cm length:

- Children with Acute Severe Malnutrition plus loses of appetite or complications.
- Bilateral edema (+++)
- Marasmic-Kwashiorkor
- Weight less than 4kg

Complications are:

- Persistent vomiting
- Convulsion
- Lethargy or unconscious
- High fever
- Hypothermia
- Severe dehydration
- Lower RTI
- Shock

- Hypoglycemia
- Sever anemia

Management of Severe Malnutrition

1- Infants aged 6-59 months:

A- Phase – 1(Stabilization Phase)

- **Start feeding :** Give F-75 130ml/kg/24hr by divided feed every 3 hr. NGT should be used when a patient takes less than 3/4 of feed or has pneumonia with rapid respiration , painful lesion of the mouth , cleft palate and disturbances of consciousness.
- **Systemic Antibiotics:** the following antibiotics should be given to severe malnourished child for 10 days.
  - First line antibiotic: Ampicillin or Amoxicillin.
  - Second line antibiotic: Add Chloramphenicole or Gentamicin or Third Generation Cephalosporin.
- Give the following medicines :
  - Vit A: single dose of 50000IU for infants less than 6month, 100000IU for infants 6-11 month and 200000IU for children age 1year or more.
- **Treat Hypoglycemia** (Blood sugar < 56mg/dl) :
  - Patients who are conscious and able to drink should be given 50ml of 10% sugar or F-75 diet.
  - Patients losing consciousness should be given 50ml of 10% sucrose by NGT. They should also be given 5ml of Glucose 10% intravenous.

- Second line antibiotics for bacterial infections.

- **Treat Hypothermia** (Rectal Temperature < 35.5 ˚C and under arm Tem <35˚C) :
  - Warmed the patients using kangaroo technique, warm cloths, blanket or incandescent lamp.
  - Treat Hypoglycemia.
  - Give second line antibiotics.
- **Treat dehydration :**

  In Marasmic patients:
  - ReSoMal 50 – 100 ml /kg over 12 hr: 5ml/kg every 30 min for 2hrs and then 5-10 ml per hr for 10 hrs orally or by NGT.
  - IV fluid (Ringer lactate + D/W 5% or 1/2 N/S + D/W 5%) : for patients who has all of the following:

    Semi-conscious or unconscious state, rapid weak pulse and cold hands and feet.

    Give 15ml / kg IV fluid over the first hr. If there is improvement repeat the 15 ml/ kg over the next hr if there is no improvement then assume that the child has septic shock.

  In Edematous patients:
  - ReSoMal 30 ml per watery stool.
  - Hypovolemia should be manage as septic shock (see below)

- **Treat Septic Shock :**
  - IV fluid (Ringer lactate + D/W 5% or 1/2 N/S + D/W 5%): Give 15 ml / kg over the first hr if the patient is unconscious.
  - Second and first line antibiotics.
  - Be kept warm.
  - Give 10 % sucrose orally or by NGT.
- **Treat Paralytic ileus** (Absent bowel sound , abdominal distention and intestinal splash) :
  - Give a single IM injection of Magnesium sulfate (2ml of 50% solution).
  - Pass an NGT aspirate the contents of stomach and then irrigate it with 50ml isotonic clear fluid repeatedly until the content become clear.
  - For candidiasis give oral Nystatin or Fluconazol.
  - Keep the child warm.
  - If the child's level of consciousness is poor give IV glucose (see Treatment of Hypoglycemia).
    Don't start IV fluid at this stage. Monitor the child for 6 hr.
  - If there is intestinal improvement (decrease in distention, return of bowel sound and visible peristalsis) then give F75 by NGT.
  - If there is no improvement after 6hr then give IV fluid contain KCL 20mmol /L or Ringer lactate with D/W 5% .The amount should not be more than 2-4ml/kg/h.
  - Give second line antibiotic.

- **Treat Congestive Heart Failure :**
  - No food and fluid should be given until the heart failure has improved even if this takes 24- 48hrs.
  - Give a diuretic: Furosemide 1mg/kg IV.
  - Digoxin 5 µg/kg can be given in a single dose. Don't give loading dose.
- **Treat severe anemia (Hb level Less than 4g/dl) :**
  - Blood transfusion
- **Treat Dermatosis of Kwashiorkor :** If Cadidiasis is present manage as follow:
  - Diaper area should be uncovered.
  - Nystatin ointment twice daily for 2 week.
  - Oral Nystatin 100000IU four times daily.

In other affected area Zinc ointment or Paraffin relieve pain and prevent infection.

Criteria to pass the patient from Phase 1 to Transmission Phase:

- The return of appetites
- Visible reduction in the amount of edema that not judged by loss of weight alone.

B - Transmission Phase:

- Give F-100; 130 ml/kg /24hr in 6 divided feed in 1-2 day. Then increase 10ml per feed.

Criteria to pass the patient from Transmission Phase to Phase 2:

- Marasmic patients spend a minimum of 2 days in Transmission Phase and pass to Phase 2 when they are completing the diet with a good appetite.

- Edematous patients should remain in Transmission Phase until they have completely loss all their edema and are completing the diet.

C – Phase 2 (Rehabilitation Phase):

- Give F-100; 130 – 200ml/kg/24hr in 6 divided feed.
- The following medicines should be administered:
  - Iron: Add one crushed tablet of ferrous sulfate (200mg) per F-100 package.
  - Mebendazole: 100mg BID for 3 days (Not used below 1year) or
  - Albendazole a single dose of 400mg, or 200mg for children 1-2 year of age.

Vit A deficiency (Xerophthalmia)

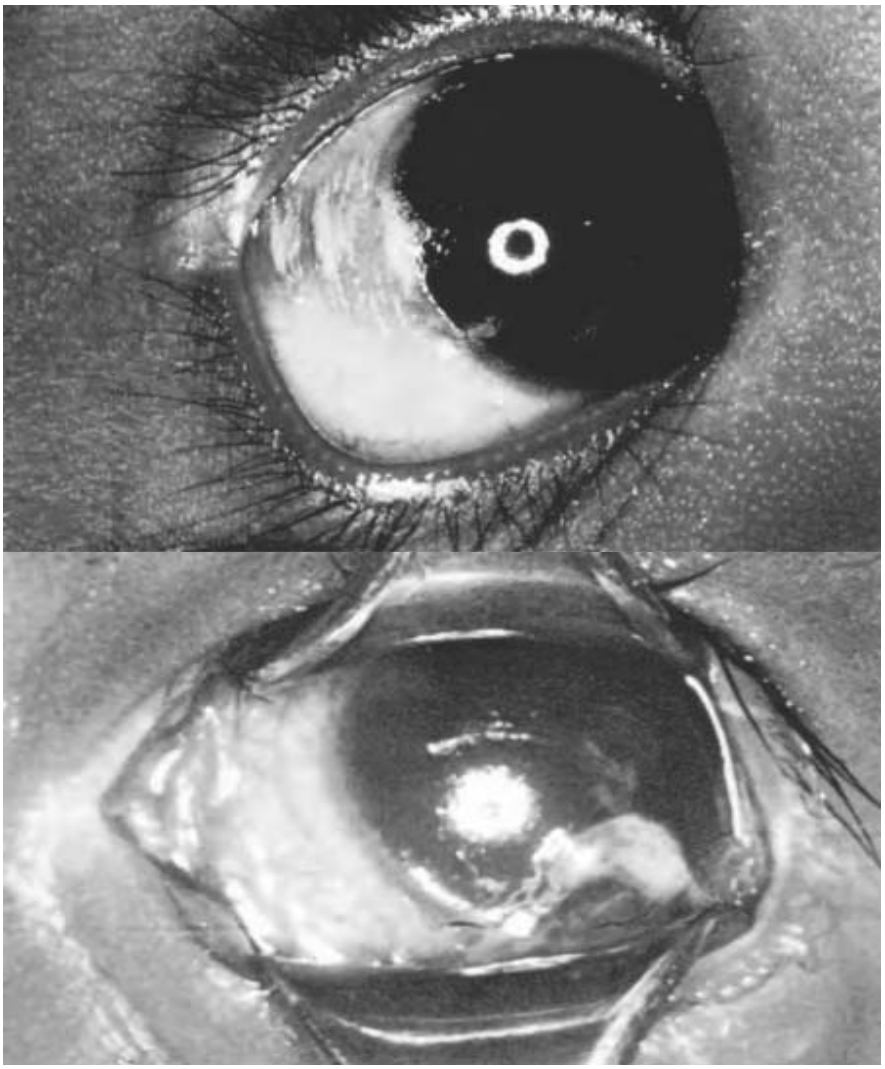
Essentials of diagnosis

1-Ocular Lesions

- Dark adaptation and night blindness.
- Xerosis of conjunctiva. The conjunctiva becomes dry, lusterless, wrinkled and dirty brown in color. Conjunctival xerosis may lead to formation of the so called "Bitot's spot," which consists of almost a triangular area, usually about the temporal aspect of the limbus covered by a fine, white foamy or greasy substance.
- Corneal xerosis reflects more advanced deficiency.
- Keratomalacia is seen in the late stage and consists of softening, necrosis and ulceration of the cornea.

2- Extraocular Lesions

- Dry, scaly skin, especially over the outer aspect of the limbs, called follicular hyperkeratosis.
- Increased susceptibility to infections due to squamous metaplasia of respiratory, urinary and vaginal tract epithelium.



Pictures: Bitot spots (left) opaque dull cornea (right)

Table 27: WHO classification for Xerophthalmia

Classification	Primary signs
X1A	Conjunctival xerosis
X1B	Bitot's spots
X2	Corneal xerosis
X3A	Corneal ulceration
X3B	Keratomalacia
	Secondary signs
XN	Night blindness
XF	Fundal changes
XS	Corneal scarring

Management of Vit A deficiency (Xerophthalmia)

Specific treatment consist of oral vitamin A in a dose of 50000, 100000, 200000 IU in children aged < 6month, 6-12 month and > 1y respectively. The same dose is repeated next day and 4 weeks later.

Vit D deficiency (Rickets)

Introduction

Failure or inadequate mineralization of growing bone or osteoid tissue is called Rickets which occurs in children (peak incidence is between 6m-2y).

Types of Rickets:

1- Vit D deficient (Nutritional) Rickets

2- Vit D resistant Rickets

3- Vit D dependant Rickets
4- Renal Rickets
5- Hepatic Rickets
6- Congenital Rickets

Essentials of diagnosis

A-Clinical features

1- General S/S:

- Failure to thrive
- Irritability
- Profuse sweating
- Weakness
- Bone pain and tenderness
- Abdominal distension
- Fractures

2- Head:

- Craniotabes (ping pong ball sensation)
- due to thinning of outer table of skull.
- Frontal bossing (duc to excess ostcoid)
- Delayed closure of anterior fontanel
- Delayed dentition, caries
- caput quadratum (square like head)

3- Chest:

- Rachitic rosary
- Harisson groove
- Respiratory infection & atelectasis

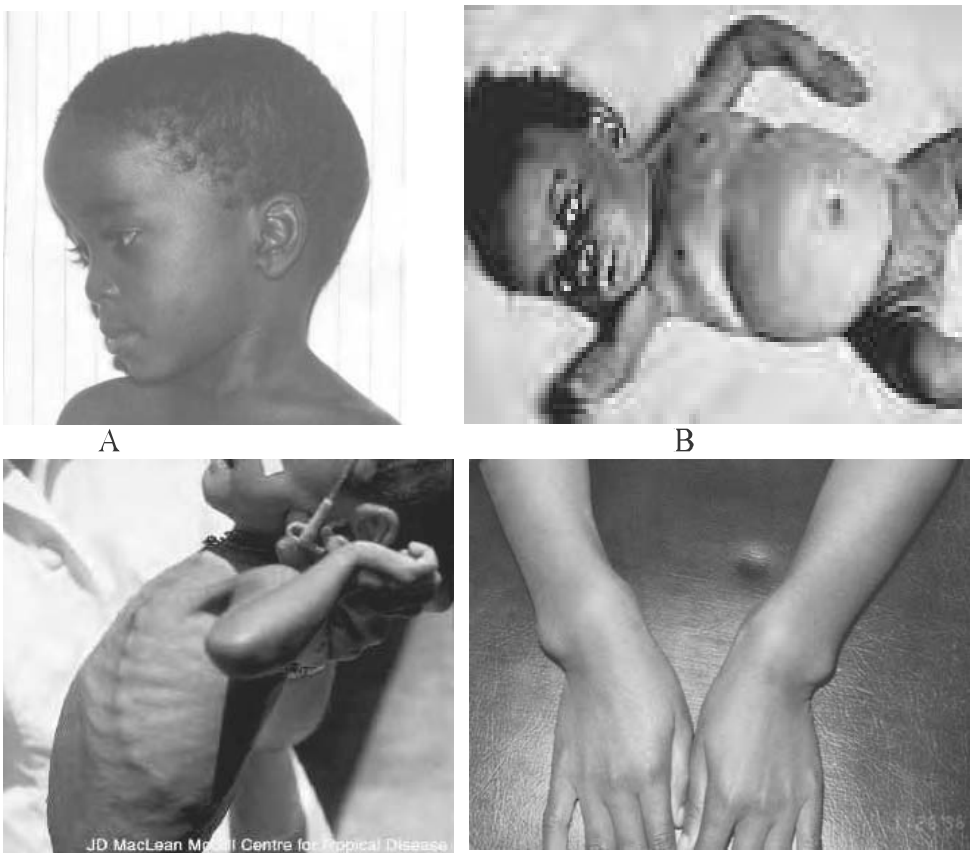
4- Back:

- Scoliosis

- Kyphosis
- Lordosis

5- Extremities:

- Enlargement of wrist and ankle
- Valgus or varus deformities



A B C D

Pictures 17: A. caput quadratum (square like head) B. Harrison grove C. Rachitic rosary. D. Double maleole and wide wrist

B- Investigation

1-X-ray changes:

- Cupping and fraying of long bones, easily seen at the distal end of radius, ulna and fibula.
- Widening and flaring of metaphysis.

- Bone density is decreased.

2-Laboratory tests:

- Scrum Calcium and Phosphorus arc normal or low.
- Serum Alkaline phosphatase and Parathyroid hormone are raised.
- Low level of 25-hydroxycholecaciferol confirmed Vit D deficiency. Its increased level indicates deficient intake of calcium or phosphorus.

Management of Vit D deficiency (Rickets)

There are 3 strategies for the administration of Vit D:

1. 300000 – 600000 IU of vitamin D are administered orally or intramuscularly as a single or 2-4 doses over one day.
2. 600000 IU over 10 days (60000 IU/day for 10 days) is given orally.
3. 2000 – 5000 IU/day over 4-6 weeks.

Patient with vitamin D deficiency rickets show evidence of radiological healing within 4 weeks of therapy. Reduction of blood alkaline phosphatase and resolution of clinical signs occur slowly. If no healing can be demonstrated with 2 mega doses of vitamin D, patient should be evaluated for refractory reckits.

- Either strategy should be followed by daily vitamin D intake of 400 IU/day if patient is < 1y old or 600 IU if age > 1y, typically given as a multivitamin.
- It is important to ensure that children receive adequate dietary calcium and phosphorus; usually provided by milk, formula and other dietary product.

Managements of Iron, vitamin B_{12}, folic acid and vitamin K deficiency are mentioned under the topic of Blood Disorders.

Vitamin C deficiency (Scurvy)

Introduction

Prolong Vit C deficiency results in Scurvy. It usually occurs in those who are deprived of citrus fruits, fresh vegetables or vitamins for some cultural or geographic reasons.

Essentials of diagnosis

A. Clinical features

1- Infant:

- Irritability, loss of appetite, low grade fever and legs tenderness.
- Knee and ankle swelling, pseudoparalysis and frog position.
- Subperiosteal hemorrhage at lower limb (end of femur) sometime can be palpable.
- Scorbutic rosary

2-Children:

- Gum change (bluish purple, spongy swelling)
- Hemorrhagic manifestation

- Petechiae
- Ecchymosis
- Epistaxis
- Gum bleeding
- Perifollicular hemorrhage

B. Investigations

1- Radiographic changes: Typically radiographic change occurs at distal end of long bone (knees) as follow:

- Pencil outlining of diaphysis and epiphysis.
- White line of Frankle at the metaphysis .
- Zone of rarefaction, a linear break parallel to the white line
- Epiphyseal centers of ossification have a ground-glass appearance by a white ring.

2- Low plasma ascorbate concentration (<0.2mg/dl) usually considered deficient but do not reflect the tissue status.

Treatment of Scurvy

Vit C 100-200mg/d orally or parenterally for 3 months ensure rapid and complete cure. Clinical improvement is seen within a week in most cases.

Part 7
Hematological Disorders

Physiologic Anemia of Infancy

Introduction & Essentials of diagnosis

The lowest normal level of Hb in term and preterm infant at the ages of 8-12w and 3-6w are called Physiologic anemia of infancy.

Treatment

- No specific treatment
- Infant feeding should be contained sufficient amount of iron and folic acid
- Supplementary iron for VLBW
- Blood transfusion if Hb > 6gr/dl
- Recombinant Erythropoietin

Iron Deficiency Anemia (Microcytic-Hypochromic Anemia)

Introduction

Iron insufficiency leads to iron depletion, iron deficiency and iron deficiency anemia (IDA) with microcytic hypochromic RBCs with increased red cell distribution width (RDW), reduced physical stamina, lack of concentration and learning ability, pica and koilonychia.

Essentials of diagnosis

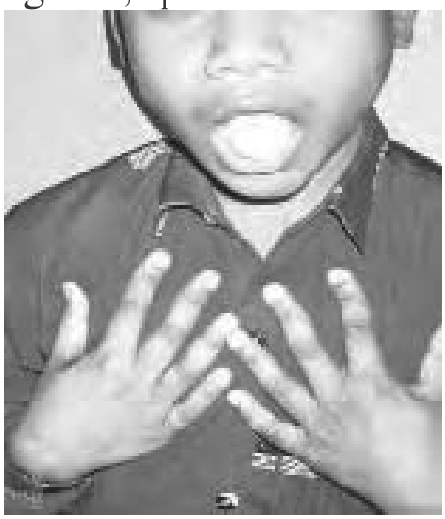
A- Clinical features

- Paleness
- Irritability
- Pica

- Lose of appetite
- Tachycardia
- Tachypnea
- Fatigue
- Palpitation
- Murmur
- CHF

In long standing cases

- Koilonychia
- Delayed growth
- Glossitis
- Stomatitis
- Gastro-intestinal disorder
- Motor, intelligence, speech disorder



Picture 18: Child with IDA

B- Investigations

- Depletion of stainable iron from BM
- Reduction of serum ferritin levels (< 15 ng/mL)
- Increased RDW (> 14.5%)

- Low serum iron (< 75 mg/dL)
- Increased TIBC (> 470 ìg/dL)
- Low transferrin saturation (< 12% and 14% for infants and children)

Management of Iron Deficiency Anemia

1- Orally Elemental iron 2-6 mg/kg /day TID should be continued for 8week after the blood values become normal.

2- **Parenteral Iron** is indicated in the following conditions :

- Intolerance to oral iron.
- Poor compliance by the patient.
- Chronic diarrhea.
- Bleeding from GI tract which is aggravated by oral iron therapy.
- Sever bleeding when hemoglobin level can not be maintained with oral iron.

Iron requirement are determined from the following equation:

$$\text{Iron (mg)} = \text{Wt (kg)} \times \text{Hb deficit (g/dl)} \times 4$$

This amount is given as Iron Dextran or Iron Sorbitol (Jectofer) which daily dose should be limited to 50mg in infant and 100mg in adult intramuscularly.

3- Blood Transfusion: When the Hb level is below 4g/dl packed RBC should be used 2-3 cc/kg slowly along with IV Furosemide 1-2mg/kg.

4- Milk consumption should be limited to 500cc/day or less.

Megaloblastic Anemia

Introduction

Megaloblastic anemia is caused by the deficiency of either folate or Vitamin B12 or both.

Essentials of diagnosis

A. Clinical features

1- Vit B12 & Folic acid

- Paleness
- Anorexia
- Irritability
- Apathy
- Glossitis
- Tremor
- Hyperpegmentation of skin

2- Vit B12

- Paresthesia
- Hypotonia
- Delayed development
- Convulsion

B. Investigations

- Macrocytic RBC on peripheral smear
- Hypersegmented neutrophil
- Neutropenia and thrombocytopenia in severe cases.
- Decreased serum Vit B12 and folic acid level.

Management of Megaloblastic Anemia

Vit B_{12} and Folic acid should be used together in suspected cases or if there is no facility to determine the level of them in the blood, otherwise give separately:

- **Vit B_{12}:** The hematologic symptoms respond promptly to parenteral administration of 250-1,000 μg vitamin B12. Children with severe deficiency and those with neurologic symptoms need repeated doses; daily or alternate days in first week followed by weekly for the first 1-2 m, and then monthly thereafter. Children having only hematologic presentation recover fully within 2-3 m, whereas those with neurologic disease need at least 6 m of therapy. Children with continuing malabsorptive state, and those having inborn errors of vitamin B12 malabsorption need lifelong treatment.
- **Folic acid:** Treatment of folate deficiency needs 1–5 mg daily for 14–21 days for therapeutic response and then needs to be continued for 1–2 months for the replenishment of the body stores..

Aplastic Anemia

Introduction

Pancytopenia caused by bone marrow failure called Aplastic anemia.

Essentials of diagnosis

A. Clinical features

- Thrombocytopenia: Purpura and bleeding
- Neutropenia: Fever and recurrent infections.

- Anemia: Paleness, fatigue weakness.

B. Investigation

- Blood: Pancytopenia with normocytic anemia
- Bone marrow: Hypocellularity or hypoplasia

Management of Aplastic Anemia

- **Supportive**
- **Etiologic**
- **BMT**
- **Immunosuppressive Drugs**
  - **Antilymphocyte globulin**
  - **Cyclosporin**
  - **Corticosteroid**

Thalassemia

Introduction

Thalassemia is an inheritance hemoglobinopathies with decreased production of globin chains that cause hemolytic anemia and ineffective erythropoiesis.

Classification

- Alpha thalassemia
- Beta thalassemia
- Delta thalassemia

Beta Thalassemia is the most common type of thalassemia lead to decrease or no production of globin beta chain. It has the following types:

- β – Thalassemia Minor
- β – Thalassemia Intermedia
- β – Thalassemia Major

Essentials of diagnosis

A. Clinical features

1- β – Thalassemia Major

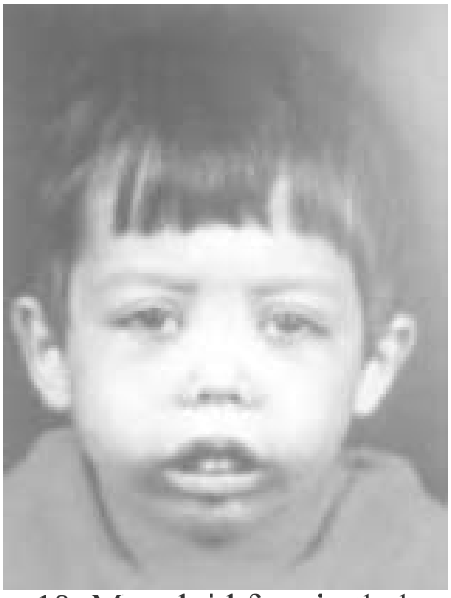
- Severe and progressive anemia
- Hepatosplenomegaly
- Abdominal distension
- Mongoloid face
- Growth retardation
- Bone deformity
- Jaundice

2- β – Thalassemia Intermedia

- Intermediate anemia
- Jaundice
- Hepatosplenomegaly
- Delayed growth
- Bile stone
- Chronic liver disease

3- β – Thalassemia Minor

- Mild anemia
- Jaundice



Picture 19: Mangloid face in thalassemia

B. Investigations

1- β – Thalassemia Major: Anemia (Hb= 2-6gr/dl), HbF= 90- 95%, HbA2 normal or slightly high and Microcytic-hypochromic

2- β – Thalassemia Intermedia: Hb =7-9gr/dl, HbF = 60-80% and Microcytic-hypochromic RBC

3- β – Thalassemia Minor: HbF = 2-10%, HbA2 = 5-7% and Microcytic-hypochromic anemia.

Management of β- Thalassemia Major and intermedia

1- **Blood transfusion:** Packed RBC 10-15 cc/kg should be transfused every 2- 4week to keep the pretransfusion level of Hb more than 9.5g/dl and less than 10.5g/dl.

2- **Folic acid should** be given 1mg /day.

3- **Chelating Therapy:** One of the following chelating agents should be start by 10-15th transfusion.
   - **Deferoxamine:** 25-50mg/kg/day over 8 – 12 hours as continuous subcutaneous infusion at least 5-6 nights per week. Vit C 100mg/day can increase the chelating and excretion of iron.
   - **Deferiprone:** 75-100mg/kg/day orally in 2-3 divided doses.
   - **Deferasirox:** 20-80 mg/kg/day orally before meal.
4- **Splenectomy** is recommended in cases when the transfusion requirement exceed 250ml/kg/ year or Hypersplenism be developed. Splenectomy should be done beyond the age of 6y.
5- **Bone Marrow Transplantation:** If HLA donor is available and the patient has young age, well-chelated, with no hepatomegaly and hepatic fibrosis ; BMT is indicated.

G6PD Deficiency

Introduction

G6PD deficiency is the most common enzymatic defect of RBC which involved more than 400 million people worldwide.

Drugs and condition cause hemolysis in G6PD deficiency

1-Drugs:

- Antibacterial: Sulfonamide, Co-trimoxazole, Nalidixic acid, Chloramphenicol, Nitrofurantoin.
- Antimalarial: Chloroquine, Premaquine, Quinine, Quinidine.

- Other drugs: Aspirin, Paracetamol, Phenacetin, Vit.K, high dose of Vit.C, Probencid, L-dopa, and Methyline blue.

2- Chemical agent: Benzene, Naphthalene.
3- Diseases: DKA, Hepatitis, Sepsis.
4- Fava beans

Essentials of diagnosis

A. Clinical features

The following signs are developed after 24-48hr of taking antioxidant agents.

- Hemolytic anemia
- Paleness
- Jaundice
- Hemoglobinuria

Clinical types

Type-1(Mild disease):

- Present in African blacks.
- Hemolysis occurs after powerful antioxidants.
- Jaundice often not developed in neonatal period.
- Enzyme activity is 60-150%

Type-2 (moderate disease):

- Present in south east Asian, middle east and Mediterranean.
- Hemolysis occurs after a lot of antioxidants.
- Jaundice often developed in neonatal period.
- Enzyme activity is 10-60%

Type-3 (Severe disease):

- Most cases are seen in north American and European.
- Hemolysis occurs even in the absence of antioxidants.

- Jaundice often developed in neonatal period.
- Enzyme activity is 1-10%

B. Investigations

- Low Hb and Hct.
- Increased level of unconjugated bilirubin, reticulocytes and plasma free Hb.
- Heinz body in RBC.
- Hemoglobinuria.
- Low level or activity of RBC G6PD.

Management G6PD Deficiency

- Antioxidants should be stopped.
- Blood transfusion for severe anemia.
- Phototherapy and EBT for the treatment of neonatal hyperbilirubinemia.
- Intravenous Sodium bicarbonate.

Immune Thrombocytopenic Purpura (ITP)

Introduction

ITP is an acquired hemorrhagic disorder due to increased platelet destruction and characterized by thrombocytopenia, purpura and normal bone marrow.

Classification

1- Acute ITP (80-90%):

Thrombocytopenia is continued for up to 6m.

2- Chronic ITP (10-20%):

Thrombocytopenia is continued more than 6m.

Essentials of diagnosis

A. Clinical features

1-Acute ITP

- Most common type(80-90%)
- Incidence 2-8y
- Boys and girls are affected equally
- H/O viral infection in half cases
- Abrupt onset of purpura and bleeding
- Spontaneous remission in 80% over 1-2m and 100% over 6m.

2- Chronic ITP:

- 10-20% cases
- Incidence after 10y
- Girls are more affected
- No H/O Viral illness
- Asymptomatic or gradually onset purpura and bleeding.
- Splenomegaly in 5-15%
- Thrombocytopenia last for more than 6m

B. Investigation

- Thrombocytopenia < 100000/mm3
- Bleeding time increased
- Large thrombocyte on peripheral smear
- Antiplatelet antibody
- Normal TLC, DLC, Hb, prothrombine time, partial thromboplastine time.

Bone marrow:Normal or increased megakaryocyte, Normal myeloid and erythroid

Indications:

- Abnormal TLC
- Abnormal DLC
- Anemia not proportioned to bleeding
- Abnormal signs (Hepatosplenomegaly and lymphadenopathy)

Management of Immune Thrombocytopenic Purpura (ITP)

1- Supportive treatment: Restriction of physical activity, and avoidance of drugs which disturbs platelet function (Aspirin, Heparin etc).

2- Drugs: One of the following drugs should be used if platelet count is less than 20000/mm^3 or bleeding be observed.

- **Prednisolon:** 1-4 mg/kg/day for 2-3 weeks than tapered over the next 1-2 weeks.
- **Intravenous Immune Globulin (IVIG) :** Total dose of 2g/kg is given , either 0.4g/kg for 5days or 1g/kg for 2days .
- **Anti-Rh (Anti-D) Therapy:** For Rh positive patients give 50-75μg/kg Anti-D intravenously.

3- Splenectomy: Recommended for Chronic ITP and whose symptoms are not controlled with above therapy.

Henoch-Schonlein Purpura (HSP)

Introduction

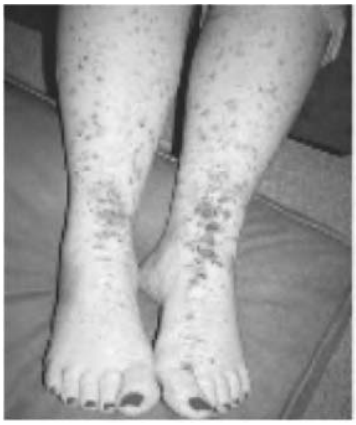
HSP or Anaphylactoid Purpura is an inflammatory disease of the small vessel characterized by palpable purpura, arthritis, abdominal pain, GI bleeding and nephritis.

Essentials of diagnosis

A. Clinical features

Presence of palpable purpura with one of the following is enough for the diagnosis:

- Abdominal pain
- Arthritis or arthralgia
- Kidney involvement (Protienuria and/or hematuria)
- Any biopsy showing the deposition of IgA on the small vessels.



Picture 20: HSP with palpable purpura

B. Investigation

1-Blood:

- Normal platelet count and function
- Normal bleeding time
- High ESR, ASO, IgA and IgM.

- Positive throat culture for Group A β-hemolytic streptococcus

2- Urine:

- Hematuria
- Protienuria

3- Stool:

- Blood in stool
- Melana

Management of HSP

1-General measure

- Bed rest
- Enough fluid
- Liquid food
- Paracetamol for analgesia

2-Penicillin is indicated for 10days if:

- ASO is high
- Positive throat culture

3-Corticosteroid: Steroids are most often used to treat significant gastrointestinal involvement or other life-threatening manifestations. Prednisone (1 mg/kg/day for 1-2 wk, followed by taper) reduces abdominal and joint pain but does not alter overall prognosis nor prevent renal disease. Rapid tapering of corticosteroids may lead to a flare of HSP symptoms.

Disseminated Intravascular Coagulation (DIC)

Introduction

Disseminated intravascular coagulation is a syndrome characterized by massive activation of coagulation that occurs inside the blood vessels, leading to widespread

deposition of fibrin in the small vessels, thus compromising blood supply to major organ systems. At the same time, the ongoing coagulation consumes platelets and coagulation factors, leading to severe bleeding.

Essentials of diagnosis

A. Clinical features

1.Hemorrhagic: Skin and mucous membrane bleeding, hemorrhage from surgical incisions, drains, intravascular catheters, venipuncture sites

2. Thrombotic: Peripheral acrocyanosis, purpura fulminans gangrenous changes in digits, nose, genitalia.

3. Multiorgan dysfunction: Derranged hepatic, renal and cardiac function jaundice, arrhythmias, oliguria, respiratory distress, gastrointestinal ulcerations, adrenal insufficiency, CNS abnormalities.

B. Investigations

Thrombocytopenia, prolong CT, BT, PT, APTT and increased FDP.

Management of Disseminated Intravascular Coagulation (DIC)

1- Treat the cause.

2-Treat the exaggerated factors (Shock, Acidosis, Hypoxia, Hypothermia and Electrolyte Disturbances).

3-If bleeding is observed **Transfuse fresh whole blood, Platelet or Cryoprecipitate.**

4- In the presence of **Fulminant Purpura, Heparin** should be given 10-15 IU/kg/hr by continuous IV infusion or 50-70 IU/kg every 6 hourly.

5- Corticosteroid is effective if Fulminant **Purpura** be developed.

6- Vit K: For infants 1mg, for children 2-3mg, for Adolescents and adults 5-10mg once intravenously.

Blood transfusion

Indications for Transfusion of Whole Blood:

1- Acute massive blood loss (> 17cc/kg or > 25% in less than 24hrs).

2- Exchange Transfusion in neonates (< 72hrs old blood).

3- Cardiovascular bypass surgery.

Indications for Transfusion of Packed RBC:

1- Infants within the first 4 month of life:

- Hb < 13g/dl and sever pulmonary disease.
- Hb < 10g/dl and moderate pulmonary disease.
- Hb < 13g/dl and sever cardiac disease.
- Hb < 10g/dl and major surgery.
- Hb < 8g/dl and symptomatic anemia.

2- Children and adolescents:

- Acute blood loss of > 25% of circulating blood volume.
- Hb < 8g/dl in the preoperative period.
- Hb < 13g/dl and sever cardiopulmonary disease.
- Hb < 8g/dl and symptomatic chronic anemia.
- Hb < 8g/dl and marrow failure.

Amount of the blood: If cardiovascular status is stable give 10-20 ml/kg over 2-4hrs. If unstable use smaller volume.

Patients with iron-deficiency anemia are often treated successfully with oral iron alone, even at Hb level < 5g/dl.

When the Hb level is below 4g/dl packed RBC should be used 2-3 cc/kg slowly along with IV Frusemide 1-2mg/kg.

Management of the Blood Transfusion Complications

1- Hemolytic Reactions:

- Stop Blood transfusion.
- Give IV fluid and Diuretic (Frusemide) to maintain normal renal output.
- $NaHCO_3$ should be used intravenously if hemoglobinuria be developed.
- Shock should be managed as Anaphylactic Shock. (See management of Shock).
- Treat DIC if present.
- For severe case transfuse compatible blood.

2- Allergic Reactions:

- For mild and moderate cases give **Diphenhydramine** 5mg/kg/day TID or **Pheneramine** 0.5-1mg/kg/day TID.
- **Adrenalin** SC and **Hydrocortison** intravenously should be used for severe cases (Anaphylaxis).See Anaphylactic Shock.

3- Febrile reactions:

- **Antihistaminics, Antipyretics** and **Hydrocortison** are effective.

Part 8
Renal Disorders

Acute Kidney Injury (AKI) or Acute Renal Failure

Introduction & Essential of diagnosis

Acute kidney injury (AKI), formerly called acute renal failure, is considered to be present when there is an abrupt (within 48-hour) reduction in kidney function, defined as absolute increase in serum creatinine of either more than or equal to 0.3 mg/dL or a percentage increase of more than or equal to 50% or reduction in urine output (oliguria of < 0.5 mL/kg/hour for > 6-hour).

Etiology: Table show causes of AKI.

Table 28 : Important causes of AKI

Prerenal failure

Hypovolemia (dehydration, blood loss, diabetic ketoacidosis)
Third space losses (septicemia, nephrotic syndrome)
Congestive heart failure
Perinatal asphyxia
Drugs (ACE inhibitors, diuretics)

Intrinsic renal failure

Acute tubular necrosis
Prolonged prerenal insult (see above)
Medications: Aminoglycosides, radiocontrast, NSAIDs
Exogenous toxins: Diethylene glycol, methanol
Intravascular hemolysis, hemoglobinuria
Tumor lysis syndrome
Hemolytic uremic syndrome: Diarrhea associated (D^+) and atypical (D^-) forms
Glomerulonephritis

- Postinfectious glomerulonephritis
- *Systemic disorders:* SLE, Henoch-Schönlein purpura, microscopic polyangiitis
- Membranoproliferative glomerulonephritis

Interstitial nephritis (drug-induced, idiopathic)
Bilateral renal vessel occlusion (arterial, venous)

Postrenal failure

Posterior urethral valves, urethral stricture
Bilateral pelviureteric junction obstruction
Ureteral obstruction (stenosis, stone, ureterocele)
Neurogenic bladder

Abbreviations: ACE, Angiotensin converting enzyme; NSAIDs, Nonsteroidal anti-inflammatory drugs; SLE, Systemic lupus erythematosus

Table 29: Staging of Acute kidney injury

Stage	Serum creatinine criteria	Urine output criteria
1	Increase in serum creatinine of ≥ 0.3 mg/dL or ≥ 150–200% (1.5- to 2-fold) from baseline	Less than 0.5 mL/kg/hour for > 6-hour
2	Increase in serum creatinine to more than 200–300% (> 2- to 3-fold) from baseline	Less than 0.5 mL/kg/hour for > 12-hour
3**	Increase in serum creatinine to more than 300% (> 3-fold) from baseline (or serum creatinine of ≥ 4.0 mg/dL with acute	Less than 0.3 mL/kg/hour for 24-hour, or anuria for 12-hour

*Only one criterion (creatinine or urine output) is required for staging
**Patients receiving renal replacement therapy are considered in stage 3

Management of AKI

1. Determine and treat the causes.
2. Put urine catheter for urine out put determination. In infants and children with urinary tract obstruction, such as in a newborn with suspected posterior ureteral valves, a bladder catheter should be placed immediately to ensure adequate drainage of the urinary tract.
3. Give **N/S 20 ml/kg** in 30 min if there is no evidence of overload or Heart Failure. For dehydration treat as dehydration protocol.
4. **Furosemide** (2-4 mg/kg) and mannitol (0.5 g/kg) may be administered as a single IV dose if urine out put remain low (<0.5 ml/kg/hr) in one hour. Bumetanide (0.1 mg/kg) may be given as an alternative to furosemide.
5. **Dopamine.** To increase renal cortical blood flow, many clinicians administer dopamine (2-3 µg/kg/min) in conjunction with diuretic therapy.
6. Maintenance fluid: **D/W 10 %**(400ml/m^2/24hr or 30-40cc/kg + amount of urine out put).

7. Management of complications: show in the following table.

Table 30: Management of complications in AKI

Complication	Treatment	Remarks
Fluid overload	*Fluid restriction:* Insensible losses (400 mL/m²/d); add urine output, other losses; 5% dextrose for insensible losses; N/5 saline for urine	Monitor other losses and replace as approprgiate, consider dialysis
Pulmonary	Oxygen; frusemide 2–4 mg/kg IV	Monitor using CVP; consider dialysis
Hypertension	*Symptomatic:* Sodium nitroprusside 0.5–8 µg/kg/minute infusion; frusemide 2–4 mg/kg IV; nifedipine 0.3–0.5 mg/kg oral/sublingual *Asymptomatic:* Amlodepine, prazosin, labetalol, clonidine	In emergency, reduce blood pressure by one-third of the desired reduction during first 6–8 hours, one-third over next 12–24 hours and the final one-third slowly over 2–3 days
Metabolic acidosis	Sodium bicarbonate (IV or oral) if bicarbonate levels < 18 mEq/L	Watch for fluid overload, hypernatremia, hypocalcemia; consider dialysis
Hyperkalemia	Calcium gluconate (10%) 0.5–1 mL/kg over 5–10 minutes IV Salbutamol 5–10 mg nebulized Sodium bicarbonate (7.5%) 1–2 mL/kg over 15 minutes Dextrose (10%) 0.5–1 g/kg and insulin 0.1–0.2 U/kg Calcium or sodium resonium (Kayexalate) 1 g/kg/day	Stabilizes cell membranes; prevents arrhythmias Shifts potassium into cells Requires monitoring of blood glucose Given orally or rectally, can be repeated every 4 hours
Hyponatremia	Fluid restriction; if sensorial alteration or seizures give 3% saline 6–12 mL/kg over 30–90 minutes	Hyponatremia is usually dilutional; 12 mL/kg of 3% saline raises sodium by 10 mEq/L
Severe anemia	Packed red cells 3–5 mL/kg; consider exchange transfusion	Monitor blood pressure, fluid overload
High phosphate	Phosphate binders (calcium carbonate; aluminum hydroxide)	Reduce dietary phosphate; avoid milk products; high protein diet

8. Indications for dialysis are:

a. Severe hyperkalemia (>8mg/kg/lit).

b. Volume overload, refractory to diuretic.

c. C.N.S symptoms of uremia.

d. Severe metabolic acidosis unresponsive to treatment.
e. BUN > 100 - 150 mg/dl.

Acute Glomerulonephritis

Introduction

Glomerulonephritis (GN) refers to disorders in which an immunologic insult triggers inflammation and proliferation of glomerular tissue with damage to the GBM, mesangium, or capillary endothelium. Glomerulonephritis may be primary (confined to the kidney) or secondary (part of a systemic disorder).

Acute GN, or the acute nephritic syndrome, is defined as sudden onset of hematuria and proteinuria, accompanied by hypertension, edema and impaired renal function. Postinfectious GN is the most common cause, and nearly 80% cases are poststreptococcal glomerulonephritis (PSGN).

Etiology

Postinfectious

• *Bacteria:* Streptococci (group A, beta-hemolytic), staphylococci,
pneumococci, meningococci, *Treponema pallidum*, *Salmonella typhi,*
leptospira

• *Viruses:* Hepatitis B and C, cytomegalovirus, parvovirus, Ebstein-Barr
virus, coxsackie virus, echovirus, varicella, rubella, rickettsiae and
mumps

• *Parasites: Plasmodium malariae, P. falciparum, Toxoplasma,* filariasis,

Schistosoma mansoni

• *Others:* infection of shunts and prosthesis, infective endocarditis

Noninfectious

• *Primary renal diseases*

– IgA nephropathy

– Membranoproliferative glomerulonephritis

– Mesangial proliferative glomerulonephritis

– Hereditary nephropathy

• *Systemic diseases*

– *Vasculitis:* Henoch-Schönlein purpura, microscopic polyangiitis,

Wegener's granulomatosis

– Collagen vascular disorder: Systemic lupus erythematosus

Essentials of diagnosis

A. Clinical features

- Poststreptococcal glomerulonephritis affects children inthe age group of 5–12 years (rare below 2 years), with male preponderance.
- Asymptomatic disease is 4–5 times morecommon among sporadic PSGN, although less frequentlyduring epidemics. Subclinical cases have microscopic hematuria, and low complement with or without hypertension.
- Symptomatic cases have an abrupt onset with hematuria, proteinuria and variable edema and hypertension. Anuria is rare, though transient oliguria may be noted.

- History of streptococcal infection may precede clinical disease onset by 1–2 weeks for throat infections and 3–4 weeks for skin infections.

B. Investigations

Blood: Although most patients have mild to modest elevations in the serum concentrations of creatinine and urea, the spectrum could range from severe azotemia to even normal levels at presentation. Hyperkalemia, metabolic acidosis and hyponatremia are only present in patients with significant renal function impairment. Antistreptolysin O titers are elevated in up to 80% of cases suggesting preceding streptococcal infection, although antibiotic treatment may attenuate this response.

Urine: urinalysis, gross hematuria is present; dysmorphic red cells, and red cell and hyaline casts may be seen. While proteinuria is often mild, occasionally nephrotic range proteinuria may be observed.

Renal Biopsy: Kidney biopsy is not needed to confirm the diagnosis of PSGN. It is indicated in those with heavy proteinuria, suspected crescentic GN or significant deviation from the natural course of the disease.

Management of Acute Glomerulonephritis

1. Therapy is essentially symptomatic with mild cases of PSGN, requiring home based treatment.
2. Hospital admission is required for those with oligoanuria, moderate to severe edema or hypertension and impaired renal functions.
3. Penicillin for 7 days may be used in those with residual pharyngitis or pyoderma, prevents spread of streptococci to others but does not alter disease course in the index case.

4. Those with oliguria/anuria require strict control of fluid intake to insensible water losses along with replacement of urine output. Prudent use of diuretics (frusemide 1–3 mg/kg) helps to manage fluid overload and circulatory congestion.
5. Salt and fluid intake should be limited in those with significant hypertension, edema or renal failure.
6. Mild hypertension is treated with oral diuretics, beta adrenergic blockers or angiotensin converting-enzyme inhibitors. Severe hypertension requires oral/IV frusemide and oral or sublingual nifedipine. For those presenting with hypertensive emergencies, IV short-acting antihypertensive (sodium nitroprusside, labetalol or nicardipine) are used for controlled reduction in blood pressure.
7. Dialysis is required in patients with refractory or worsening acute renal failure.

Nephrotic Syndrome

Introduction

Nephrotic syndrome is characterized by heavy Proteinuria [defined as proteinuria >3.5 g/24 hr or presence of 3–4+ (300–1000 mg/dL) or a urine protein : creatinine ratio >2], hypoalbuminemia (albumin < 2.5 g/dL), hyperlipidemia (cholesterol > 200 mg/dL) and edema.

Important definitions to clarify the course of Nephrotic Syndrome

Remission: Urine albumin nil or trace (or proteinuria < 4 mg/m2/hour) for three consecutive early morning specimens

Relapse: Urine albumin 3+ or 4+ (or proteinuria > 40 mg/m2/hour) for three consecutive early morning specimens, having been in remission previously

Frequent relapses: Two or more relapses in initial 6 months or more than three relapses in any 12 months

Steroid dependence: Two consecutive relapses when on alternate day steroids or within 14 days of its discontinuation

Steroid resistance: Absence of remission despite therapy with daily prednisolone at a dose of 2 mg/kg/day for 4-8 weeks

Essentials of diagnosis

A. Clinical features

- The idiopathic nephrotic syndrome is more common in boys than in girls (2 : 1) and most commonly appears between the ages of 2 and 6 yr.
- The initial episode of idiopathic nephrotic syndrome, as well as subsequent relapses, usually follows minor infections.
- Children usually present with mild edema, which is initially noted around the eyes and in the lower extremities.
- With time, the edema becomes generalized, with the development of ascites, pleural effusions, and genital edema. Anorexia, irritability, abdominal pain, and diarrhea are common.
- Important features of minimal change idiopathic nephrotic syndrome are the absence of hypertension and gross hematuria (the so-called nephritic features).

A. Investigations

1-Urine: The urinalysis reveals 3+ or 4+ proteinuria, and microscopic hematuria is present in 20% of children. A spot urine protein : creatinine ratio should be >2.0.

2-Blood: The serum creatinine value is usually normal, but it may be abnormally elevated if there is diminished renal perfusion from contraction of the intravascular volume. The serum albumin level is <2.5 g/dL, and serum cholesterol and triglyceride levels are elevated. Serum complement levels are normal.

3-Renal Biopsy:A renal biopsy is not routinely performed if the patient fits the standard clinical picture of MCNS. Indications are: Age at onset less than 1 year or more than 12 years; (ii) gross or persistent microscopic hematuria, or low C3; (iii) renal failure, not attributed to hypovolemia; (iv) suspected secondary cause, (v) sustained hypertension and (vi) steroid resistance.

Indications of admission are:

- Large Pleural Effusion.
- Ascites.
- Sever genital edema.

Management of Initial Episode

1. Children with their first episode of nephrotic syndrome and mild to moderate edema may be managed as outpatients.
2. A high protein and low salt diet is recommended as long as proteinuria is present.

3. **Diuretic** use should be reserved for severe symptomatic edema. For such cases Frusemide (1-4mg/kg/d in 2 divided doses) alone or with Spironolactone (2-3mg/kg/d in 2 divided doses) are administered.
4. **Prednisolon** is started for children with onset of Nephrotic syndrome between one and 8yr of age without renal biopsy. prednisone or prednisolone should be administered as a single daily dose of 60 mg/m2/day or 2 mg/kg/dayto a maximum of 60 mg daily for 4-6 wk followed by alternate-day prednisone (starting at 40 mg/m2 qod or 1.5 mg/kg qod) for a period ranging from 8 wk to 5 mo, with tapering of the dose.
5. When a patient has severe generalized edema with evidence of intravascular volume depletion (e.g., hemoconcentration, hypotension, tachycardia), IV administration of 25% albumin (0.5-1.0 g albumin/kg) as a slow infusion followed by furosemide (1-2 mg/kg/ dose IV) is sometimes necessary.

Management of Relapses

A. Management of Infrequent Relapses:

1. Diet and diuretics indications are the same as initial episode.
2. **Prednisolon:** Relapses are often triggered by minor infections. Symptomatic therapy of infectious illness often results in remission of 1+/2+ proteinuria. However, persistence of 3+/4+ Proteinuria with

infections requires therapy. Prednisolone is given at a dose of 2 mg/kg/day until urine protein is trace or nil for three consecutive days (*remission*), and subsequently as a single morning dose of 1.5 mg/kg on alternate days for 4 weeks. Treatment for a relapse usually lasts for 5–6 weeks.

B. Management of Frequent Relapses and Steroid Dependant Nephrotic Syndrome:

One of the following regimes can be used:

These patients require prolonged treatment in order to maintain disease remission. The strategies are summarized below.

1-Long-term, Alternate Day Steroids

Following treatment of a relapse, prednisolone is tapered to a dose of 0.3–0.7 mg/kg on alternate days, which is given for 9–18 months.

2- Steroid Sparing Agents

Alternative agents are recommended if: (i) prednisolone threshold more than 0.5–0.7 mg/kg on alternate days; (ii) features of corticosteroid toxicity (growth failure, hypertension and cataract) appear. The agents used are listed below:

A.Levamisole

• **Dose and duration:** 2–2.5 mg/kg on alternate days for 12–24 months.

• **Concomitant steroid therapy:** The dose of prednisolone is tapered to 0.25–0.5 mg/kg on alternate days.

While therapy with prednisolone may be discontinued in some cases, many patients require a small dose of prednisolone.

B. Cyclophosphamide

• **Dose and duration:** 2–2.5 mg/kg/day for 8–12 weeks.

• **Concomitant steroid therapy:** Cyclophosphamide has a potent steroid sparing potential, allowing discontinuation of steroids. The dose of prednisolone is maintained at 1mg/kg during cyclophosphamide therapy; subsequently prednisolone is tapered and discontinued.

C.Mycophenolate Mofetil (MMF)

• **Dose and duration:** 600–1000 mg/m2/day; 20–30 mg/kg/day for 12–24 months.

• **Concomitant steroid therapy:** The agent has moderate steroid sparing potential; tapering doses of prednisolone are given for 6–12 months.

D.Calcineurin Inhibitors [Cyclosporine (CsA), Tacrolimus]

These agents are indicated in patients with steroid dependence that fails to benefit with levamisole, cyclophosphamide and/or MMF.

• **Dose and duration:** CsA 4–5 mg/kg/day; Tacrolimus 0.1–0.2 mg/kg/day for 12–24 months.

B. **Management of Steroid Resistant Nephrotic Syndrome:** After renal biopsy; Cyclophosphamide, Cyclosporine or Methylprednisolon

Urinary Tract Infection (UTI)

Introduction

The diagnosis of UTI requires demonstration of significant bacteriuria on urine culture in the presence of symptoms.

Etiology

The most common causative organism in children is *E. coli* (60–80%); *Proteus*, *Klebsiella*, *Staphylococcus saprophyticus*, *Enterococcus* and *Enterobacter* are the other common organisms identified.

Classification

The 3 basic forms of UTI are pyelonephritis, cystitis, and asymptomatic bacteriuria. Focal pyelonephritis ("nephronia") and renal abscesses are less common.

Essentials of diagnosis

A. Clinical features

- Newborns with UTI present with septicemia, jaundice, vomiting and shock.
- In infancy, symptoms are nonspecific, such as unexplained fever, diarrhea, vomiting and failure to thrive.
- Older children have dysuria, frequency and suprapubic pain. Fever, flank pain and toxic appearance suggest renal parenchymal involvement (complicated UTI).

B. Investigations

Urinanalysis: Detection of leukocytes (> 5 WBC/HPF in centrifuged urine) and bacteria on microscopic examination of a carefully collected fresh sample of urine suggests UTI. Enhanced urinalysis using uncentrifuged urine sample for

leukocyturia (> 10 WBC/mm3) in Neubauer counting chamber along with Gram staining of sediment for bacteria is useful. Rapid tests like Greiss test and nitrite and leukocyte esterase tests are popular but may have false positivity and negativity.

Urine culture: On culture of urine collected by a standard midstream clean catch specimen, a colony count of more than 105CFU/mL should be documented.

Indications for admission are:

Dehydration, complicated UTI, unable to drink, vomiting, less than 3 month, toxic appearance and suspected sepsis.

Management

C. OPD management for suspected cystitis: Give one of the following :

1. **Amoxicilline** 50 mg /kg /day.
2. **Cotrimoxazole** 10mg/kg/day of **Trimethoprim.**
3. **Nitrofurantoin** 5-7mg/kg/day.
4. **Cefixime 10mg/kg/day**

Duration of treatment: 7 – 10 days.

B: IPD management for suspected pyelonephritis.

Fist line: **Ampicillin** (100-200mg/kg/day) **+ Gentamicin** (5-7.5 mg/kg/day).

Second line: **Third generation cephalosporin** (Ceftriaxone 50 -75 mg/kg/day).

Duration of treatment: 10 – 14 days.

References

1- Cherry JD, et al (2014): ***Feigen & Cherry's TEXTBOOK OF PEDIATRIC INFECTIOUS DISEASES***. 2nd ed. Elsevier, Philadelphia, USA.

2- Cloherty JP, Eichenwald EC, Stark AR (2004): ***MANUAL OF NEONATAL CARE*** . 5TH ed. Lippincott WW, Philadelphia.

3- Crisp S, Rainbow J (2013): ***EMERGENCY IN PEDIATRIC AND NEONATOLOGY***. 2nd ed. Oxford University, UK.

4- Elzouki AY, et al (2012). ***TEXTBOOK OF CLINICAL PEDIATRICS***. Springer. Philadelphia. USA.

5- Fleisher GR, Ludwig S (2010): ***TEXTBOOK OF PEDIATRIC EMERGENCY MEDECINE***. 6th ed. lippincott wiliams & wilkins. China.

6- Ghai OP, Paul VK, Bagga A (2013): ***Ghai ESSENTIAL PEDIATRICS***. 7th ed. CBC, New Delhi.

7- Gomella TL, Cunningham MD, Eyal FG, Zenk KE (2013): ***NEONATOLOGY*** . 7th ed. Lange Medical book, USA.

8- Gupte S (2004): ***THE SHORT TEXTBOOK OF PEDIATRICS***. 10thed. Jaypee Brothers, New Delhi.

9- Hay WW, Levin MJ, Sondheimer JM et al (2011): ***CURRENT DIAGNOSIS & TREATMENT PEDIATRICS,*** 20th ed. McGrawHill, USA.

10- Hoffbrand AV, Moss PAH, Pettit JE (2006): ***ESSENTIAL HAEMATOLOGY***. 5th ed. Blackwell, USA.

11- Kattwinkel J, et al (2011): TEXTBOOK OF NEONATAL RESUSCITATION. 6th ed. USA.

12- Kliegman RM, Stanton GF, St Jeme JW, et al (2015): ***NELSON TEXTBOOK OF PEDIATRICS.*** 20th ed. Elsevier, Philadelphia.

13- Marcdante KJ, Kliegman RM (2015): ***NELSON ESSENTIALS OF PEDIATRICS***. 7th ed. Elsevier, Philadelphia.

14- Mclntosh N, et al (2008). Fofar & Arneil's TEXTBOOK OF PEDIATRIC. 7th ed. Elsevier. China.

15- Parthasarathy A , Gupte P , Agrawal R , et al (2013): ***IAP TEXTBOOK OF PEDIATRICS*** . 5th ed. Jaypee Brothers, New Delhi.

16- Renni JM (2012): ***Rennie and Robertones TEXTBOOK OF NEONATOLOGY.*** 5th ed . Elsevier. China.

17- Sachdev HPS , Ghugh K , Choudary P , Ramji S , et al (2006) : ***PRINCIPLES OF PEDIATRIC AND NEONATAL EMERGENCIES***. 2nd ed. Jaypee Brothers, New Delhi.

18- Sinha S, Miall L, Jardine L (2012). ***ESSENTIALS NEONATAL MEDICINE***.5th ed. Wiley-Blackwell. Malaysia.

19- Sing M (2010): ***CARE OF THE NEOBORN***. 7th ed, Sagar, New Delhi.

20- Thomas K, Tey D, Marks M (2009): ***PEDIATRIC HANDBOOK***. 8th ed. Wiley-Blackwell, Australia.

21- WHO. ***HOSPITAL CARE FOR CHILDREN (2013).***2nd ed. who press. Geneva. Switzerland.

22- WHO: Integrated Management of Childhood Illness.

Introduction of the Author

Name: Mansoor
Last name: Aslamzai
Academic rank: Professor (Pohanwal)
Date of Birth: 1969
Place of Birth: Bihsood district, Nangarhar Province, Afghanistan

Dr. Masoor Aslamzai graduated in 1986 from Nangarhar High School and admitted in this year to Kabul Medical Faculty. In 1992 he graduated from the faculty and introduced to the Pediatric Department of Ningarhar Public Heath Hospital. In 1997 he got the lecturer position in Nangarhar Medical Faculty; and got promotion to the academic rank of Professor (Pohanwal) in 2012. Now he works at the Department of Neonatology in Kabul Medical University and Ataturk hospital. Beside of the teaching and academic activity in the faculty, Dr.Mansoor has worked as a clinical instructor, facilitator and trainer in deferent courses of IMCI, Neonatal and Severe Malnutrition.

Other publications of the author are:

1. Twelve academic articles and researches which are published in the Kabul Medical University and Nangarhar University Magazines.
2. Pediatric Hematology (Pushto).
3. Therapeutic Guideline of Pediatrics (English)
4. Neonatology (Dari)

د لوړو زده کړو وزارت پیغام

د بشر د تاریخ په مختلفو دورو کې کتاب د علم او پوهې په لاسته راوړلو، ساتلو او خپرولو کې ډیر مهم رول لوبولی دی. درسي کتاب د نصاب اساسي برخه جوړوي چې د زده کړې د کیفیت په لوړولو کې مهم ارزښت لري. له همدې امله د نړیوالو پیژندل شویو معیارونو، د وخت د غوښتنو او د ټولنې د اړتیاوو په نظر کې نیولو سره باید نوي درسي مواد او کتابونه د محصلینو لپاره برابر او چاپ شي.

له ښاغلو استادانو او لیکوالانو څخه د زړه له کومې مننه کوم چې دوامداره زیار یې ایستلی او د کلونو په اوږدو کې یې په خپلو اړوندو څانګو کې درسي کتابونه تألیف او ژباړلي دي، خپل ملي پور یې اداء کړی دی او د پوهې موتور یې په حرکت راوستی دی. له نورو ښاغلو استادانو او پوهانو څخه هم په درنښت غوښتنه کوم تر څو په خپلو اړوندو برخو کې نوي درسي کتابونه او درسي مواد برابر او چاپ کړي، چې له چاپ وروسته د ګرانو محصلینو په واک کې ورکړل شي او د زده کړو د کیفیت په لوړولو او د علمي پروسې په پرمختګ کې یې نېک ګام اخیستی وي.

د لوړو زده کړو وزارت دا خپله دنده بولي چې د ګرانو محصلینو د علمي سطحې د لوړولو لپاره د علومو په مختلفو رشتو کې معیاري او نوي درسي مواد برابر او چاپ کړي.

په پای کې د افغان ماشومانو لپاره د جرمني کمیټې او زموږ همکار ډاکتر یحیی وردک څخه مننه کوم چې د کتابونو د خپرولو لپاره یې زمینه برابره کړېده.

هیله منده یم چې نوموړې ګټوره پروسه دوام وکړي او پراختیا ومومي تر څو په نیږدې راتلونکې کې د هر درسي مضمون لپاره لږ تر لږه یو معیاري درسي کتاب ولرو.

په درنښت

پوهنوال دوکتور فریده مومند

د لوړو زده کړو وزیره

کابل، ۱۳۹۵

د درسي کتابونو چاپول

قدرمنو استادانو او ګرانو محصلينو!

د افغانستان په پوهنتونونو کې د درسي کتابونو کموالی او نشتوالی له لويو ستونزو څخه ګڼل کېږي. يو زيات شمير استادان او محصلين نويو معلوماتو ته لاس رسی نه لري، په زاړه ميتود تدريس کوي او له هغو کتابونو او چپترونو څخه ګټه اخلي چې زاړه دي او په بازار کې په ټيټ کيفيت فوتوکاپي کېږي.

تر اوسه پورې مورِ د ننګرهار، خوست، کندهار، هرات، بلخ، کاپيسا، کابل او کابل طبي پوهنتون لپاره ۲۲۳ عنوانه مختلف درسي کتابونه د طب، ساينس، انجنيري، اقتصاد او زراعت پوهنځيو (۹۶ طبي د آلمان د علمي همکاريو ټولنې DAAD، ۱۰۰ طبي سره له ۲۰ غير طبي د افغان ماشومانو لپاره د جرمني کمپټې Kinderhilfe-Afghanistan او ۴ نور غير طبي د آلماني او افغاني پوهنتونونو ټولنې DAUG) په مالي مرسته چاپ کړي دي.

د يادونې وړ ده، چې نوموړي چاپ شوي کتابونه د هېواد ټولو اړونده پوهنځيو ته په وړيا توګه وېشل شوي دي. ټول چاپ شوي کتابونه له www.afghanistan-ecampus.org ويب پاڼې څخه ډاونلوډ کولای شئ.

دا کړنې په داسې حال کې تر سره کېږي چې د افغانستان د لوړو زده کړو وزارت د (۲۰۱۰-۲۰۱۴) کلونو په ملي ستراتيژيک پلان کې راغلي دي چې:

"*د لوړو زده کړو او د ښوونې د ښه کيفيت او زده کوونکو ته د نويو، کره او علمي معلوماتو د برابرولو لپاره اړينه ده چې په دري او پښتو ژبو د درسي کتابونو د ليکلو فرصت برابر شي د تعليمي نصاب د ريفورم لپاره له انګريزي ژبې څخه دري او پښتو ژبو ته د کتابونو او درسي موادو ژباړل اړين دي. له دې امکاناتو څخه پرته د پوهنتونونو محصلين او استادان نشي کولای عصري، نويو، تازه او کره معلوماتو ته لاس رسی پيدا کړي.*"

مونږ غواړو چې د درسي کتابونو په برابرولو سره د هيواد له پوهنتونونو سره مرسته وکړو او د چپټر او لکچر نوټ دوران ته د پای ټکی کېږدو. د دې لپاره دا اړينه ده چې د لوړو زده کړو د موسساتو لپاره هر کال څه نا څه ۱۰۰ عنوانه درسي کتابونه چاپ شي.

له ټولو محترمو استادانو څخه هیله کوو، چې په خپلو مسلکي برخو کې نوي کتابونه ولیکي، وژباړي او یا هم خپل پخواني لیکل شوي کتابونه، لکچر نوټونه او چپټرونه ایډیټ او د چاپ لپاره تیار کړي، زمونږ په واک کې یې راکړي چې په ښه کیفیت چاپ او وروسته یې د اړوند پوهنځیو، استادانو او محصلینو په واک کې ورکړو. همدارنګه د یاد شویو ټکو په اړوند خپل وړاندیزونه او نظریات له مونږ سره شریک کړي، تر څو په ګډه پدې برخه کې اغیزمن ګامونه پورته کړو.

د مؤلفینو او خپروونکو له خوا پوره زیار ایستل شوی دی، ترڅو د کتابونو محتویات د نړیوالو علمي معیارونو په اساس برابر شي، خو بیا هم کیدای شي د کتاب په محتوی کې ځینې تیروتنې او ستونزې ولیدل شي، نو له درنو لوستونکو څخه هیله مند یو تر څو خپل نظریات او نیوکې مؤلف او یا مونږ ته په لیکلې بڼه راولیږي، تر څو په راتلونکي چاپ کې اصلاح شي.

د افغان ماشومانو لپاره د جرمني کمیټې او د هغې له مشر ډاکتر ایروس څخه ډېره مننه کوو چې د دغه کتاب د چاپ لګښت یې ورکړی دی، دوی په تېرو کلونو کې هم د ننګرهار پوهنتون د ۱۰۰ عنوانه طبي او ۲۰ عنوانه غیرطبي کتابونو د چاپ لګښت پر غاړه درلود.

په ځانګړې توګه د جې آی زیت (GIZ) له دفتر او CIM (Center for International Migration & Development) چې زما لپاره یې په تېرو پنځو کلونو کې په افغانستان کې د کار امکانات برابر کړی دي، هم د زړه له کومې مننه کوم.

د لوړو زده کړو له وزیرې پوهنوال دوکتور فریده مومند، علمي معین پوهنوال محمد عثمان بابري، مالي او اداري معین پوهنوال ډاکتر ګل حسن ولیزي، د ننګرهار پوهنتون د پوهنځیو رییسانو او استادانو څخه مننه کوم چې د کتابونو د چاپ لړۍ یې هڅولې او مرسته یې ورسره کړې ده. د دغه کتاب له مؤلف څخه ډېر مننوی یم او ستاینه یې کوم، چې خپل د کلونو-کلونو زیار یې په وړیا توګه ګرانو محصلینو ته وړاندې کړ.

همدارنګه د دفتر له همکارانو هر یو حکمت الله عزیز، احمد فهیم حبیبي او فضل الرحیم څخه هم مننه کوم چې د کتابونو د چاپ په برخه کې یې نه ستړې کیدونکې هلې ځلې کړې دي.

ډاکتر یحیی وردک، د لوړو زده کړو وزارت سلاکار
کابل، اپریل ۲۰۱۶
د دفتر ټیلیفون: ۰۷۵۶۰۱۴۶۴۰
ایمیل: textbooks@afghanic.org

تقریظ

په وروستیو څو کلونو کې په هېواد کې د پوهې او علم په برخه کې یوځه پرمختګونه شوي دي، چې ورسره د ښونځیو، مسلکي انستیتوتونو، پوهنتونونو او محصلینو په کمیت کې زیاتوالی راغلی دی.

که له یوې خوا پوهنتونونه د کمیت په لحاظ ډېر شوي دي، نو له بل پلوه اړینه ده چې د لوړو زده کړه کیفیت ته زیاته توجه وشي.

زمونږ په وطن کې علمی او نوی آثار لاتر اوسه هم ډېر کم دی، خدای وکړی چې د داسی تدریسي کتابونو لیکل او چاپول به دغه تشه یو څه ډکه کړی. هیله مند یوو چې زمونږ نور استادان هم تشویق شی، تر څو په خپلو مسلکي برخو کې نوي علمي کتابونه ولیکي.

دغه کتاب د ننګرهار د طب پوهنځی لخوا وکتل شو، ښه معیاری او د محصلینو لپاره ګټور کتاب دی.

مونږ د لیکوال دغه زیار ستایو، د لوی خدای ج له دربار څخه نوموړي ته د نورو بریاوو هیله کوو.

د ننګرهار د طب پوهنځی

د کتاب نوم د نیونتالوژي او کوچنیانو د ناروغیو کلینیکي هنډبوک (انګلیسي)
لیکوال پوهنوال دوکتور منصور اسلم زی
خپرندوی ننګرهار پوهنتون، طب پوهنځی
وېب پاڼه www.nu.edu.af
چاپ شمېر ۱۰۰۰
چاپ کال ۱۳۹۵، لومړی چاپ
ډاونلوډ www.ecampus-afghanistan.org
چاپ ځای سهر مطبعه، کابل، افغانستان

د تدریسي کتابونو د چاپولو لپاره له مور سره اړیکه ونیسئ:
ډاکتر یحیی وردک، د لوړو زده کړو وزارت، کابل
تیلیفون ۰۷۵۶۰۱٤٦٤۰
ایمېل textbooks@afghanic.org

ای اس بي ان ۱-۲۶-۶۲۰-۹۹۳۶-۹۷۸ ISBN