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ZAMBIA PAEDIATRIC PROTOCOLS

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ACRONYMS

3TC – Lamivudine
ABG – Arterial Blood Gas
ACEIs – Angiotensin Converting Enzyme Inhibitors
ACS – Acute Chest Syndrome
ACTH- Adrenocorticotropic Hormone
ADD – Acute Diarrhoeal Disease
ADH - Anti-Diuretic Hormone
ADHD – Attention Deficit Hyperactive Disorder
AEDs – Anti-Epileptic Drugs
aEEG – Amplitude-integrated Electroencephalography
AF – Atrial Fibrillation
AG – Anion Gap
AIS – Androgen Insensitivity Syndrome
AKI – Acute Kidney Injury
AKIN - Acute Kidney Injury Network
ANA – Antinuclear Antibodies
anti-dsDNA – Anti-Double-Stranded DNA Antibodies
APLA – Antiphospholipid Antibodies
ARBs – Angiotensin Receptor Blockers
ARF – Acute Rheumatic Fever
ART – Anti-retro viral therapy
ASA – Acetylsalicylic Acid (aspirin)
ASD – Atrial Septal Defect
ASD – Autism Spectrum Disorder
ASOT – Anti-streptolysin O titers
ATV/r – Atazanavir/Ritonavir
AZT – Azidothymindine (also called Zidovudine ZDV)
BD – Twice a Day
BMI – Body Mass Index
BMT Bone Marrow Transplant
BPD – Bronchopulmonary Dysplasia
CAH – Congenital Adrenal Hyperplasia
CCBs – Calcium Channel Blockers
CCF – Congestive Cardiac Failure
EMTCT – Elimination of Mother-To-Child Transmission (of HIV)
CDD – Chronic Diarrhoea
CFU – Colony forming unit
CHD – Congenital Heart Disease
CKD – Chronic Kidney Disease
CNS – Central Nervous System
CO – Cardiac Output
CoA – Coarctation of Aorta
CPAP – Continuous positive airway pressure
CPP – Central precocious puberty
CRP – C Reactive Protein
CRT – Capillary Refill Time
CSA – Child Sexual Assault
CSF – Cerebral Spinal Fluid
CT Scan – Computed Tomography scan
CTG – Cardiotocography
CVA – Cerebral Vascular Accident
CVD - Cardiovascular Disease
CXR – Chest X-Ray
DAT – Direct Antiglobulin
DCM – Dilated Cardiomyopathy
DHTR – Delayed Haemolytic Tranfusion Reaction
DIC – Disseminated intravascular coagulation
Disease
DKA – Diabetic ketoacidosis
DM – Diabetes Mellitus
DMSA – Dimercapto succinic acid
DNS – Dextrose Normal Saline
DRTB - Drug resistance TB
DSD – Disorders of Sexual Differentiation
DST – Drug sensitivity testing
DTG – Dolutegravir
EBM – Expressed Breast Milk
ECG - Electrocardiography
ECHO Echocardiography
EEG - Electroencephalography
ELBW – Extremely low birth weight
IBD – Inflammatory Bowel Disease
IBMFSInherited Bone Marrow Failure Syndrome

EPTB – Extra Pulmonary TB
ESR – Erythrocyte Sedimentation Rate
ESRD – End-stage Renal Disease
EWS – Early Warning Score
FBC Full Blood Count
FFA – Free Fatty Acids
FFP – Fresh Frozen Plasma
FiO2 – Fraction of Inspired Oxygen
FSH – Follicle Stimulating Hormone
FTT – Failure To Thrive
G6PD – Glucose 6 Phosphate Dehydrogenase
GAS - Group A β-Haemolytic Streptococcus
GBV – Gender-Based Violence
GCS – Glasgow Coma Scale
GERD – Gastroesophageal Reflux Disease
GFR – Glomerular Filtration Rate
GGT - Gamma-Glutamyl Transferase
GI – Gastrointestinal
GnRH – Gonadotropin Releasing Hormone
Hb – Haemoglobin
HBsAg – Hepatitis B surface Antigen
HbSS – Homozygous Sickled Haemoglobin
HBV – Hepatitis B Virus
HCO_3^- - Bicarbonate
HDN – Haemolytic Disease of the Newborn
HDU – High Dependency Unit
HEU – HIV Exposed Uninfected
HFNC – High Flow Nasal Cannula
HHV – Human Herpes Virus
HIE – Hypoxic Ischaemic Encephalopathy
HIV – Human Immunodeficiency Virus
HLA Human Leukocyte Antigen
HPS – Hypertrophic Pyloric Stenosis
HPV – Human Papilloma Virus
HSCT Haematopoietic Stem Cell Transplant
HSP – Henoch Schonlein Purpura
HTN – Hypertension
HUS – Haemolytic Uremic Syndrome
MCUG – Micturating Cystourethrogram
MDRTB – Multi-Drug Resistance TB
MPO – Myeloperoxidase
IBS – Inflammatory Bowel Syndrome
ICP – Intracranial Pressure
ICS – Inhaled Corticosteroid
ICU Intensive Care Unit
IE – Infective Endocarditis
IEC – Information, Education and Communication
IEMs – Inborn Errors of Metabolism
IgAN – Ig A Nephropathy
IH – Infantile Haemangioma
ILAE – International League Against Epilepsy
IM – Intramuscular
INR – International Normalization Ratio
InSurE – Intubate-Surfactant-Extubate
IO - Intraosseous
IV – Intravenous
IVH – Intraventricular Hemorrhage
IVIG Intravenous Immunoglobulin
JVD = Jugular Venous Distension
JVP – Jugular Venous Pressure
KDIGO – Kidney Disease Improving Global Outcomes
KMC – Kangaroo Mother Care
KUB – Kidney, Ureter, and Bladder
LABA – Long-Acting Beta Agonist
LAM – Lipoarabinomannan
LBW – Low Birth Weight
LDH Lactate Dehydrogenase
LFTs – Liver Function Tests
LH – Luteinizing Hormone
LISA – Less Invasive Surfactant Administration
LN – Lupus Nephritis
LPA – Line Probe Assay
LPV/r – Lopinavir/Ritonavir
LTRA – Leukotriene Receptor Antagonist
LVH – Left Ventricular Hypertrophy
M/C/S – Microscopy, Culture and Sensitivity
MCA - Middle Cerebral Artery
PTH – Parathyroid Hormone
PTT – Prothomboplastin Time
PUD – Peptic Ulcer Disease

MRA/MRV - Magnetic Resonance Angiography/Magnetic Resonance Venography
MRI – Magnetic Resonance Imaging
MRI/MRA/MRV Magnetic Resonance Imaging/AngiographyVenography
MTB – Mycobacterium Tuberculosis
NAT – Nucleic Acid Test
NE – Neonatal Encephalopathy
NEC – Necrotizing Enterocolitis
NGT – Nasogastric Tube
NPO – Nil Per Oral
NRB – Non Rebreather Mask
NS – Nephrotic Syndrome
NSAIDs – Nonsteroidal Anti-Inflammatory Drugs
NVP – Nevirapine
OD - Once Daily
OGT – Oral Gastric Tube
ORS – Oral Rehydration Salt
OSC – One Stop Centre
PBS - Peripheral Blood Smear
PD - Persistent Diarrhoea
PDA – Patent Ductus Arteriosus
PDD – Persistent Diarrhoeal Disease
PDN - Prednisolone
PEF – Peak Expiratory Flow
PEG – Polyethylene Glycol
PEP – Post Exposure Prophylaxis
PIAGN – Post-infectious Acute Glomerulonephritis
PICU – Paediatric Intensive Care Unit
PO – Per Oral
PPIs – Proton Pump Inhibitors
PrPEP – Pre-Exposure Prophylaxis
TTP – Thrombotic Thrombocytopenic Purpura
ULN – Upper Limit Normal
UTI – Urinary Tract Infection
VCUG – Voiding cystourethrography
VDRL – Venereal disease research laboratory
PUV – Posterior Urethral Valve
QID – Four Times a Day
RAS – Renal Artery Stenosis
RBS – Random Blood Sugar
RDT – Rapid Diagnostic Test
RF – Rheumatic Fever
RHD – Rheumatic Heart Disease
ROM – Rupture of Membranes
RPGN – Rapidly Progressive Glomerulonephritis
RPR – Rapid Plasma Reagent
SABA – Long-Acting Beta Agonist
SAM – Severe Acute Malnutrition
SBE – Subacute Bacterial Endocarditis
SC – Subcutaneous
SCD Sickle Cell Disease
SIADH – Syndrome of Inappropriate Anti-Diuretic Hormone
SLE – Systemic Lupus Erythematosus
SPA – Suprapubic Bladder Aspiration
STAT – Right Now
STIs – Sexually Transmitted Infections
SVR – Systemic Vascular Resistance
TB – Tuberculosis
TBM – Tuberculous Meningitis
TBT – TB Preventive Therapy
TCAs – Tricyclic Antidepressants
TCB – Transcutaneous Bilirubin
TDF – Tenofovir Disoproxil Fumarate
TDS – Three Times a Day
TFT Thyroid Function Test
TIA – Transient Ischaemic Attack
TOF – Tetralogy of Fallot
TP-PA - Treponema Pallidum Particle Agglutination
TSH – Thyroid Stimulating Hormone
TST Tuberculin Skin Test
VLBW – Very Low Birth Weight
VMA – Vanillylmandelic Acid
VOC – vaso occluive crisis
VSD – Ventricular Septal Defect
XFTC – Emtricitabine

FOREWORD

The Ministry of Health prioritises the health of children and is committed to delivering improved quality healthcare services across the country. So far, there has been a significant reduction in the overall under-five mortality rate, from 197 per 1000 live births in 1996 to the current 61 per 1000 live births. However, if the current trajectory continues, Zambia may not attain the 2030 global target of reducing the Under 5 mortality to 25 per 1000 live births.

The Ministry of Health therefore welcomes the 2024 Zambia Paediatric Protocols that have been developed at a very critical stage. They will serve as an added tool in our quest to improve healthcare provided to our children. These protocols focus on triage, assessment and classification of illness severity, criteria for admission, and inpatient management of the major causes of childhood morbidity & mortality such as pneumonia, diarrhoea, malaria, severe acute malnutrition, meningitis, HIV, TB, Sickle Cell and neonatal conditions.

These protocols are the first edition and seek to streamline and standardize paediatric healthcare service delivery across the continuum of care in the country. They are aimed at doctors, clinical officers, nurses, pharmacists, laboratory scientists, and all healthcare workers who are responsible for the care of sick newborns and children. They are also intended to be a useful resource for students in medical schools and other Health training institutions. These protocols are meant to serve as a guide for clinical practice, based on the best available evidence at the time of development. They do not substitute the need to consult as necessary. Ultimately every healthcare worker is responsible for the management of their patient based on the clinical features presented by the patient and the management options available locally.

It is my expectation that the protocols shall provide drive to our mission to significantly reduce the morbidity and mortality in children and provide client and patient satisfaction.

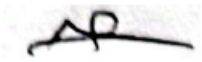


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ACKNOWLEDGEMENTS

The development of this 1st Edition of the National Paediatric Protocols marks a significant landmark in the efforts of the health sector to standardize the care of sick children in Zambia, and an important step towards assuring quality healthcare services to children anytime and anyplace in Zambia.

I wish to thank all those who contributed towards the realization of these protocols. Special gratitude goes to the partners and contributors.



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University Teaching Hospital HIV/AIDS Programme

DEDICATIONS

The following are paediatricians who devoted their lives to the health and well-being of children.

Their unwavering commitment to nurturing the smallest of patients, often during their most vulnerable moments, is nothing short of heroic. They were more than just doctors— they were healers, advocates, and champions for a brighter and healthier future. Their tireless work, compassionate care, and endless patience ensured that children everywhere can grow, thrive, and reach their fullest potential.

Though they are no longer with us, we thank them for their resilience, kindness, and dedication to making the world a better place, one child at a time.



Prof. Chifumbe Chintu: 08.12.1935 – 04.05.2017
Father of Paediatrics & Child Health in Zambia

Prof. Chifumbe Chintu attained his medical degree at the University of Toronto in 1966. He returned to Zambia where he was posted to Kitwe General Hospital. He went back to Canada to pursue his postgraduate studies in paediatrics and in 1973 he became a Fellow of the Royal College of Physicians of Canada and a diplomate of the American Board of Paediatrics. And later, he became a Fellow of the Royal College of Physicians in London.

He returned to Zambia and became a consultant paediatrician in 1973 and later joined the University of Zambia, School of Medicine. He rose from senior lecturer to Associate Professor and became a full professor in 1980. He was Head of Department for Paediatrics and Child Health at the University Teaching Hospital (UTH) where he founded the Haematology and Oncology unit. He was a pioneer in Sickle Cell Disease care in children and adults. He set up the only combined paediatric and adult haematology clinic, living up to his motto of "paediatricians care for patients from the womb to the tomb'"

Prof Chintu later became Dean of the School of Medicine. He was instrumental in founding the Post Graduate Training Programmeme (MMed) that birthed locally trained specialists. He also founded the Research Ethics Committee which is key in regulating research in the country. He lobbied for funding from the Japanese government to build the neonatal and paediatric surgical building (D-block) at UTH. In 1994, Prof Chintu and Alimuddin Zumla founded a collaborative research and training programmeme between the University of Zambia and University College London Medical School (UNZA–UCLMS). UNZA–UCLMS studies have changed global practice and WHO management guidelines for tuberculosis, tuberculosis–HIV co-infection, and respiratory infections.

Prof Chintu served in numerous associations and professional bodies such as the Zambia Paediatric Association, Zambia Medical Association, Zambia Medical Council (now Health Professions Council of Zambia) and others. He received numerous awards, among them, from World Health Organisation in 2006 World Health Day for his contribtions in teaching, training, clinical cre and research over a period of 40 years. He was also given the Grand Order of Distinguished Service First Division on 24th October 2007 by President Levy Mwanawasa. He also received Life Time Achievement Awards and Distinguished Service Awards from the Zambia Medical Association as well as the Zambia Paediatric Association.

Prof Chintu was a great and dedicated clinican who was loved by his patients and their families. He taught and mentored generations of health care workers in Zambia and other countries and inspired many to specialise in Paediatrics and Child Health. The community of practice has fond memories of his teachings as well as the passionate care he had for his patients. His legacy lives on!



Prof. James Chipeta (01.03.1962 – 02.03.2020)

Prof Chipeta studied Medicine at the University of Zambia School of Medicine and graduated in 1990. He then joined the Department of Paediatrics and Child Health at the UTH in 1993. He was awarded the MonBosho Scholarship to study Paediatrics and Clinical Immunology from 1994 to 2000 at Mie University in Japan. He obtained his PhD in Paediatrics and Clinical Immunology in 2000. He joined the University of Zambia as Lecturer and in 2009 he was promoted to Senior Lecturer and in the same year he was appointed inaugural Assistant Dean Research. He was appointed Full Professor of the University of Zambia in 2018. He received numerous awards, amongs them, the National Science and Technology Council Merit Award for Research in 2013 and the meritorious award by the Zambia Medical Association in 2018.



Dr Samuel Phiri (23.12.1943 – 19.07.2023)

Dr Samuel Phiri graduated from UNZA in 1975, with a Bachelor's Degree in Medicine and surgery and had a Distinction in Paediatrics. He worked in Scotland at the Hospital for Sick Children. He was also Chief Medical Officer in Chililabombwe under ZCCM. He became a Consultant Pediatrician at Perirenyatwa and Mpilo Hospital in Zimbabwe. He served as consultant at UTH and lectured medical students as well as postgraduates at University of Zambia, School of Medicine. He also served as Deputy Director at UTH and later become Senior Medical Superintendent for Arthur Davison Children's hospital, from 2003 until he retired in 2015. He had special interest in cardiac conditions. He was awarded for dedicated service to the care of children by the Fellowship of Royal College of Physicians of Edinburg, Scotland and the Zambia Paediatric Association.



Prof. Ganapati Bhat (10.06.1945 – 13.03.2005)

Prof. Ganapati Bhat was a distinguished pediatrician who had a significant career at the University Teaching Hospital where he served as a head of department for Paediatrics and Child Health. He was also a lecturer at the University of Zambia, School of Medicine. His work focused on various aspects of child health, particularly in the context of infectious diseases. Prof. Bhat published extensively on topics such as HIV, human herpesvirus 8 (HHV-8), and anthropometric measurements in children. His research contributed to a deeper understanding of these conditions in the pediatric population, particularly in Zambia and the broader sub-Saharan African region.



Dr Sylvester Sinyangwe (29.11.1950-24.05.2021)

Dr Sylvester Sinyangwe graduated from the University of Zambia School of Medicine and later specialized in Paediatrics and Child Health. He served in the Zambia Army and pursued further training in the USA and Japan. His interests span from clinical care particularly in neonatology as well as administration and public health. He worked in Lesotho, before returning to Zambia where he served as medical director at the Zambia Sugar company. He later joined the University of Zambia, School of Medicine as a lecturer and served as head of the department of Paediatrics and Child Health. He also served as a honorary consultant at the UTH Children's Hospital.

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The process involved health workers from all provinces and representatives from major hospitals. This lengthy process came to fruition through the dedication and hard work of the clinicians who had to divide time between service delivery and gathering the practical information to come up with these protocols. The passion and willingness of these busy health workers has to be recommended as it has shown their dedication to providing quality health care to the children of Zambia. We therefore encourage this spirit in our fraternity to continue as this will help foster improved health care for children. Special thanks to all who have made this edition possible among many others:

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DISCLAIMER

These protocols serve as a guideline for the management of some common childhood illnesses in Zambia. The guideline is not a substitute for clinical judgement.

Variation from the guideline, taking into account individual circumstances may be appropriate, depending on locally available resources and expertise, or with new evidence based research findings.

CHAPTER 1: COMMON PAEDIATRIC EMERGENCIES

Emergencies are common in children and result from a variety of conditions affecting various systems. The clinician should focus on prevention of cardiopulmonary failure through early recognition and management of respiratory distress, respiratory failure, and shock that can lead to cardiac arrest due to progressive hypoxia, acidosis, and ischaemia.

The approach to all critically ill patients or those in cardiopulmonary arrest is the same. The underlying principle is to use Airway, Breathing, Circulation, Disability, Exposure (ABCDE) approach to assess and treat patients.

The ABCDE approach is beneficial in that

- It treats the most life-threatening problem first
- Allows for a systematic approach
- Prevents the clinical team from forgetting something
- Allows a clinical team to work seamlessly together as they all know what needs to be done

Some common emergencies and their treatment have been described in this chapter.

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1.1 CARDIOPULMONARY RESUSCITATION

Cardiac arrest is a condition defined by the absence of pulses. The two types of paediatric cardiac arrest are hypoxic/asphyxia arrest and sudden cardiac arrest. Hypoxic/asphyxial arrest is the most common type of cardiac arrest in infants and children while sudden cardiac arrest is more common in adults.

Infants and children can also present with a respiratory arrest (not breathing normally but presence of pulses). They are much more likely to recover if promptly treated than those with a cardiac arrest.

AETIOLOGY

Hypoxic/ashyxial cardiac arrest
It results from progressive tissue hypoxia and acidosis due to respiratory failure and/or shock.

Causes include

- Septic shock, upper airway obstruction, pneumonia, metabolic derangement (eg hypoglycaemia, inborn errors of metabolism, electrolyte disturbance), neurologic catastrophe (brain herniation or hemorrhage), poisoning, trauma, non-trauma exsanguination, anaphylaxis and sudden infant death syndrome,

Sudden cardiac arrest
This is most often caused by the development of ventricular fibrillation (VF) or pulseless ventricular tachycardia (pVT).

Causes in children include

- Hypertrophic cardiomyopathy, myocarditis, Anomalous coronary artery, Wolff-Parkinson-White syndrome, Long QT syndromes, drug intoxication, commotio cordis.

MANAGEMENT

Rapid recognition of cardiac arrest and prompt performance of cardiopulmonary resuscitation (CPR) are important in order to improve survival.
Cardiopulmonary resuscitation comprises a group of interventions performed to provide oxygenation and circulation to the body during cardiac arrest.

The steps of resuscitation, following the ABC approach are elaborated in the algorithms below.

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PAEDIATRIC BASIC LIFE SUPPORT

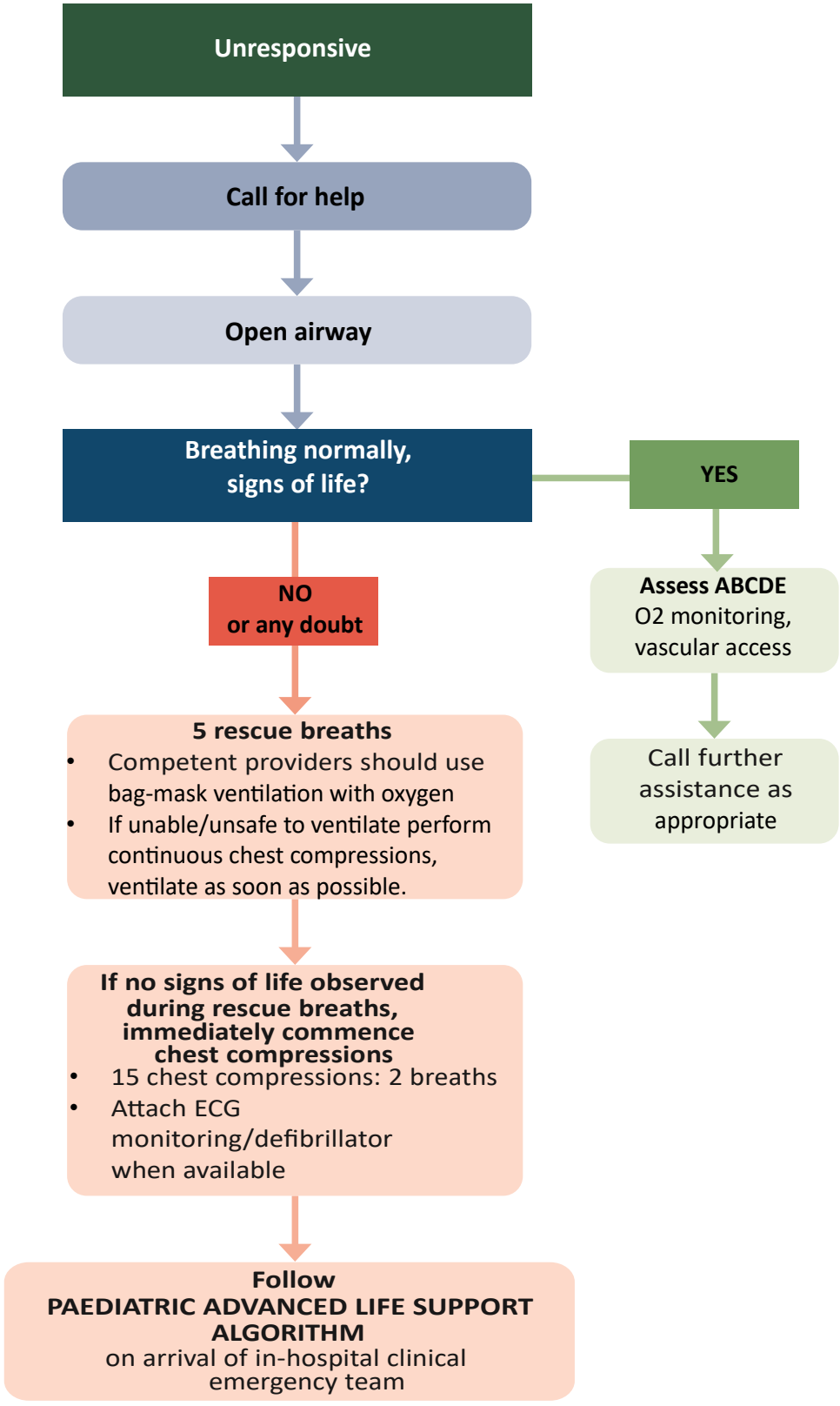
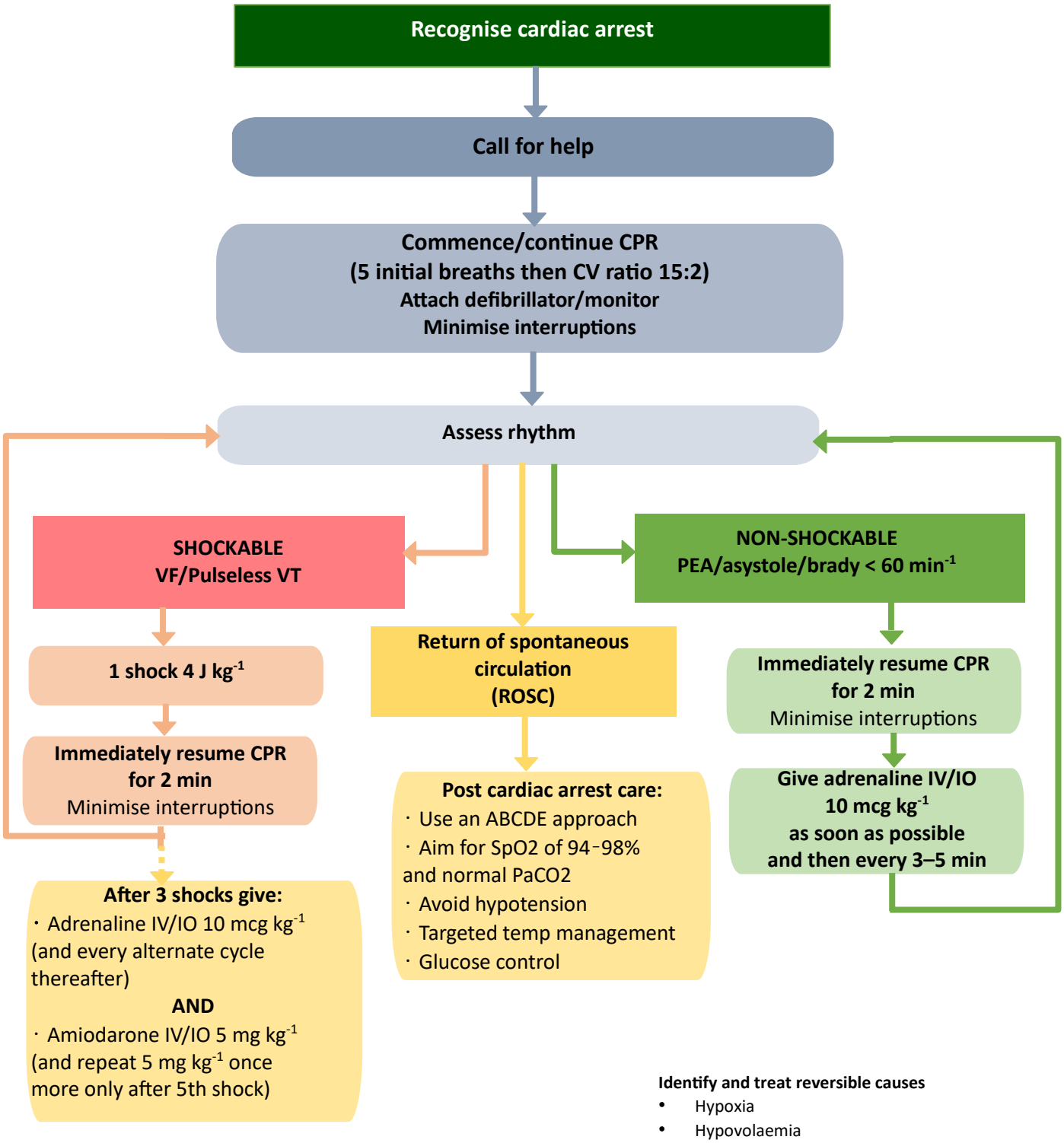


Fig 1. Adapted from resuscitation council UK guideline 2021

PAEDIATRIC ADVANCED LIFE SUPPORT



During CPR

- **Ensure high quality chest compressions are delivered:**

– Correct rate, depth and full recoil

- Provide BMV with 100% oxygen (2-person approach)
- Provide continuous chest compressions when a tracheal tube is in place.
- Competent providers can consider an advanced airway and capnography, and ventilate at a rate (breaths minute-1) of:

Infants: 25	1–8 years: 20	8–12 years: 15	> 12 years: 10–12

- Vascular access IV/IO
- Once started, give Adrenaline every 3-5 min
- Maximum single dose Adrenaline 1 mg
- Maximum single dose Amiodarone 300 mg

Identify and treat reversible causes

- Hypoxia
- Hypovolaemia
- Hyperkalaemia, hypercalcaemia, hypermagnesemia, hypoglycaemia
- Hypo-/hyperthermia
- Thrombosis ‑ coronary or pulmonary
- Tension pneumothorax
- Tamponade ‑ cardiac
- Toxic agents

Adjust algorithm in specific settings (e.g. special circumstances)

Fig 1.1. Adapted from resuscitation council UK guideline 2021

1.2 FOREIGN BODY OBSTRUCTION

Foreign body aspiration (FBA) is a potentially life-threatening event. An aspirated solid or semisolid object may lodge in the larynx or trachea and block respiration thereby impairing oxygenation and ventilation. Asphyxia may rapidly cause death.

Children are usually at high risk due to the tendency of putting small objects in their mouths. The foreign body usually lodges in a bronchus (more often in the right) and can cause collapse or cause consolidation of the portion of lung distal to the site of blockage.

Foreign body aspiration is common in children, especially below the age of 2 years. This is because of their tendency to put everything in their mouths and they chew their food incompletely with incisors. Children with neurological and cognitive disorders are also at risk of foreign body aspiration.

The commonly aspirated objects include seeds, nuts, food particles, coins, popcorn, small toys, pins, pen caps among others. Infants and toddlers commonly aspirate food items while older children aspirate nonfood items

Choking is a frequent initial symptom. Objects such as fish bones can lodge in the larynx, causing stridor or wheeze. Sometimes the chocking may be followed by a symptom-free interval of days or weeks before the child presents with persistent wheeze, chronic cough, or pneumonia, which fails to respond to treatment.

A large object lodged in the larynx can cause death from asphyxia, unless it can be dislodged, or an emergency tracheostomy be done.

Diagnosis

Inhalation of a foreign body should be considered in a child with the following:

- sudden onset of choking, coughing, or wheezing; or
- segmental or lobar pneumonia that fails to respond to antibiotic therapy.

Examine the child for:

- unilateral wheeze
- an area of decreased breath sounds that is either dull or hyper-resonant on percussion
- deviation of the trachea or apex beat

Obtain a chest X-ray at full expiration to detect an area of hyperinflation or collapse, mediastinal shift (away from the affected side) or a foreign body if it is radiopaque.

Treatment

Emergency first aid for the choking child: Attempt to dislodge and expel the foreign body. The management depends on the age of the child.

***For infants*:**

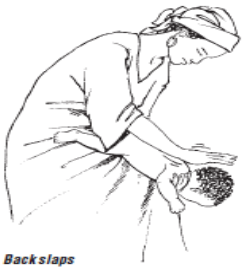
a. Lay the infant in a head-down position on one of your arms or on your thigh.

b. Strike the middle of the infant's back five times with the heel of your hand.

c. If the obstruction persists, turn the infant over and give five firm chest thrusts with two fingers on the lower half of the sternum.

d. If the obstruction persists, check the infant's mouth for any obstruction that can be removed.

e. If necessary, repeat this sequence with back slaps.



Back slaps



Chest thrusts

Adapted from WHO pocketbook of care for children in hospital, 2nd ed, 2013

***For older children*:**

While the child is sitting, kneeling, or lying, strike the child's back five times with the heel of the hand.

If the obstruction persists, go behind the child, and pass your arms around the child's body; form a first with one hand immediately below the sternum; place the other hand over the fist and thrust sharply upwards into the abdomen. Repeat this up to five times. This known as the Heimlich manoeuvre.



Then check the child's mouth for any obstruction that can be removed. If necessary, repeat the sequence with back slaps.

Once this has been done, it is important to check the patency of the airway by:

- looking for chest movements,
- listening for breath sounds and
- feeling for breath.



Heimlich manoeuvre for a chocking older child

If further management of the airway is required after the obstruction is removed, keep the child's airway open and prevent the tongue from falling back to obstruct the pharynx while the child recovers.

Later treatment of suspected foreign body aspiration

If a foreign body is suspected but cannot be confirmed, refer the child to a hospital where diagnosis is possible, and the object can be removed after bronchoscopy.

If there is evidence of pneumonia, begin treatment with ampicillin (or benzylpenicillin) and gentamicin, as for severe pneumonia (see above), before attempting to remove the foreign body.

PAEDIATRIC FOREIGN BODY AIRWAY OBSTRUCTION

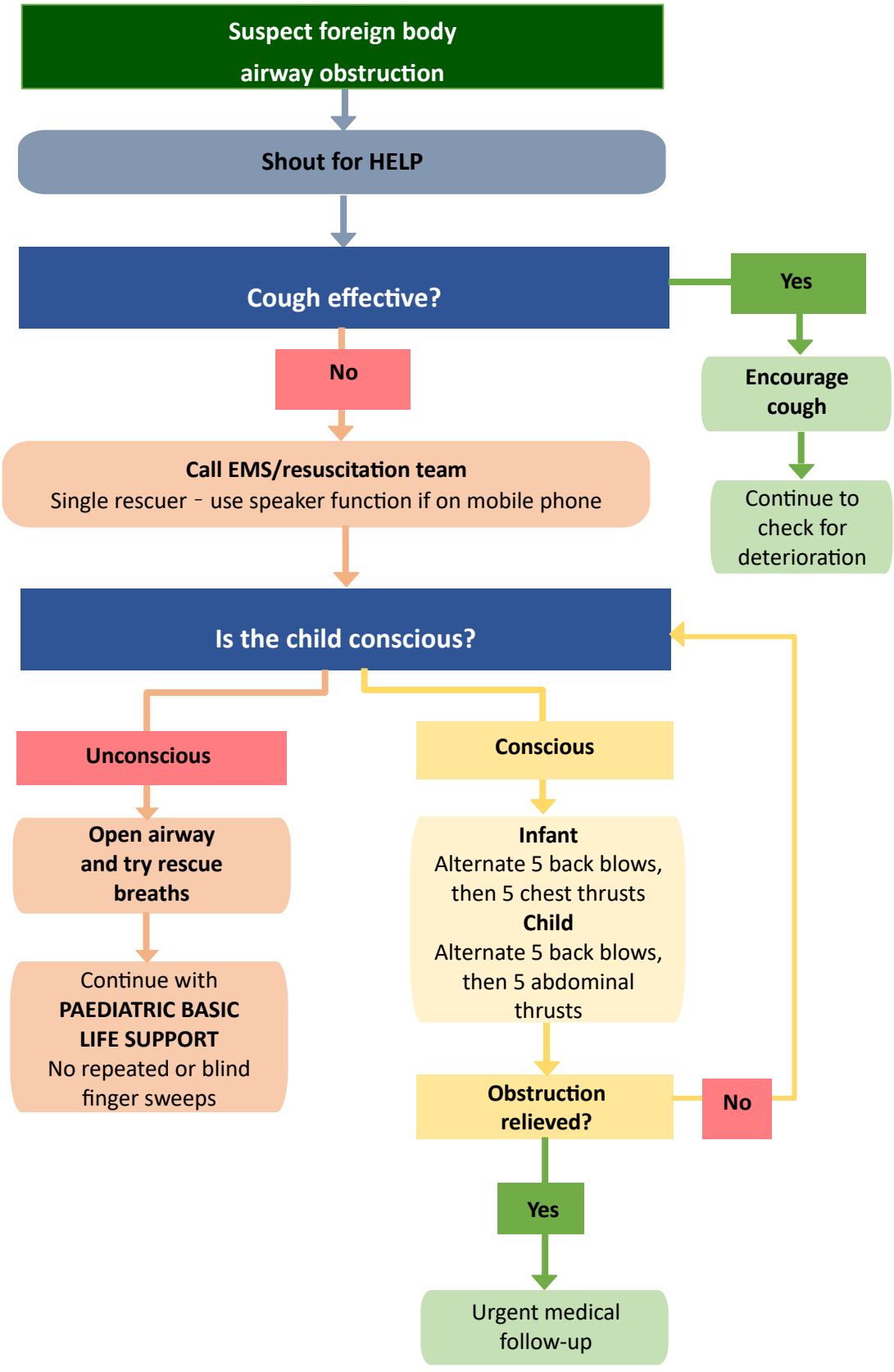


Fig 1.2. Adapted from resuscitation council UK guideline 2021

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1.3 RESPIRATORY SUPPORT (OXYGEN) ESCALATION

Oxygen is a life-saving essential medicine used to treat critically ill patients with acute hypoxic respiratory failure and is necessary to maintain normal cellular metabolism.

Rapid and effective oxygen delivery is an essential component of the care of critically ill children. A variety of systems are available to deliver oxygen to spontaneously breathing patients.
Factors that influence the appropriate choice for any given situation include dose of oxygen required and how well the patient tolerates the device.

General principles regarding oxygen delivery include

- The choice of system will depend on the clinical status of the patient and the desired dose of oxygen, which is a function of the fraction of inspired oxygen (FiO2 or concentration) and the rate of oxygen gas flow
- When oxygen delivery is anticipated to be prolonged, it should be humidified, whenever possible, to prevent dried secretions from obstructing smaller airways
- The effectiveness of oxygen delivery should be monitored with pulse oximetry

Patient considerations include

- Young children in respiratory distress may become frightened or agitated when oxygen is administered, causing their clinical condition to deteriorate. Therefore, they should remain in a position of comfort whenever possible. A parent or caregiver can often hold the oxygen source in proximity to or over the child's face.
- As long as oxygenation is adequately maintained, a nasal cannula may be maintained, as nasal cannula may be preferable to a face mask for delivering oxygen to confused or delirious children.

Important safety issues include

- Although oxygen itself is not flammable, it causes other material, such as hair, skin oils, clothing, and furniture to catch fire at lower temperatures and to burn hotter. Patients receiving oxygen therapy and stored oxygen cylinders should be kept at least 1.5 meters away from open flames, heat sources or electrical devices
- If oxygen cylinders are used, they should be secured to that they cannot fall over, develop a rapid leak and become propelled like a missile. Regular monitoring of exchanged oxygen cylinders is necessary to ensure that all valves are working properly
- Where oxygen supply is limited, the duration of oxygen administration should be considered when selecting the mode of oxygen delivery. Therefore, if prolonged administration is anticipated, use of oxygen-conserving devices or titrating oxygen delivery to match the minimal clinical requirement in non-critically ill patients is prudent.

The algorithm for oxygen administration and its escalation is shown below

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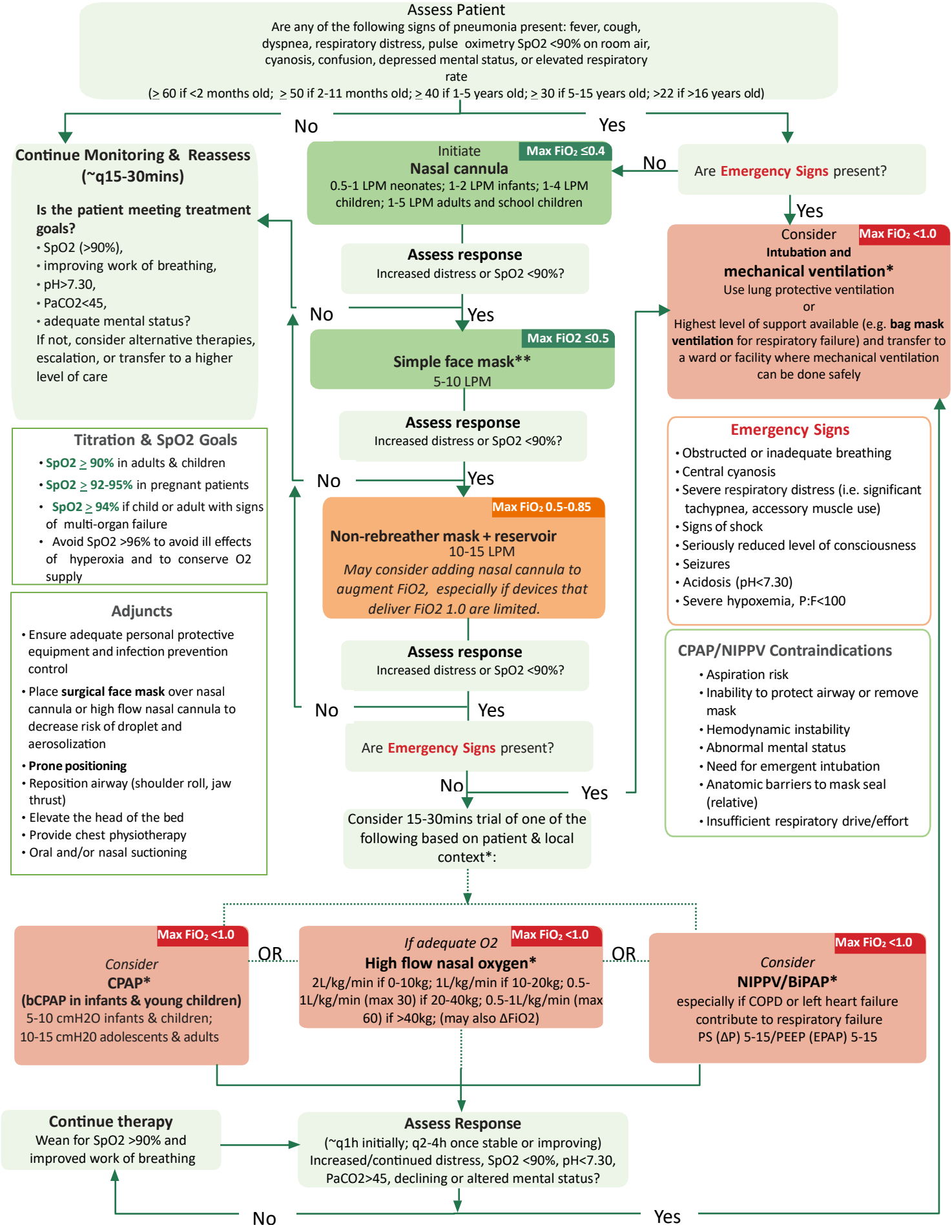


Fig 1.3. Respiratory support escalation

1.4 ACUTE EXACERBATION OF ASTHMA

Asthma is a chronic inflammatory disorder of the airways, associated with airway hyperresponsiveness leading to recurrent wheezy episodes, breathlessness, chest tightness and persistent cough.

Diagnostic criteria

- Episodes of breathlessness
- Chest tightness
- Wheezing
- Persistent cough
- Airflow obstruction, variability and reversibility by peak flow measurement or spirometry

Table 1. Assessment and management of asthma severity

Moderate asthma
Normal mental state
Ability to talk in sentences or vocalise as normal
Some accessory muscle use
PEF ≥ 50% of best or predicted
O2 saturations > 92% in air
Moderate tachycardia
HR ≤ 125 min-1 (> 5 years)
HR ≤ 140 min-1 (2–5 years)
RR ≤ 30 min-1 (> 5 years)
RR ≤ 40 min-1 (2–5 years)
Management
Continuous O2 saturation monitoring
High-flow O2 via NRB mask titrated to achieve O2 saturations 94–98%
ß2 agonist 2–10 puffs via pMDI + spacer
+/-face mask, repeat dose every 20 min reviewing effect; no improvement in 1 h treat as acute severe
Ipratropium bromide given early via pMDI
+ spacer +/- face mask, particularly if poorly responsive to ß2 agonist
Oral steroids: prednisolone 20 mg for children aged 2 to 5 years; 30 to 40 mg for children > 5 years

Acute severe asthma
Agitated, distressed
Can't complete sentences in one breath
Moderate to marked accessory muscle use
PEF 33–50% of best or predicted
O2 saturations > 92% in air
HR ≤ 125 min-1 (> 5 years)
HR ≤ 140 min-1 (2–5 years)
RR ≤ 30 min-1 (> 5 years)
RR ≤ 40 min-1 (2–5 years)
Management
Continuous O2 saturation monitoring
High-flow O2 via NRB mask titrated to achieve O2 saturations 94–98%
ß2 agonist nebulised (salbutamol 2.5–5 mg) every 20 min with Ipratropium bromide (250 mcg) for first 2 h; review frequently
Oral steroids: 20 mg prednisolone for children aged 2 to 5 years; 30 to 40 mg for children > 5 years
Consider intravenous magnesium and aminophylline 5mg/kg over 20mins then infusion at 1mg/kg/hr if the child is unresponsive to maximal doses of bronchodilators and steroids
Consider ABG if poor response to early treatment
Refer to PICU

Life-threatening asthma
Confused, drowsy, exhausted
Unable to talk
Maximal accessory muscle use (poor respiratory effort is **pre-terminal**)
Marked tachycardia (sudden fall in HR is **pre-terminal**)
PEF < 33% of best or predicted
O2 saturations < 92% in air
Silent chest
Cyanosis
Hypotension
Management
Continuous O2 saturation monitoring
High-flow O2 via NRB mask titrated to achieve O2 saturations 94–98%
Refer to PICU
ß2 agonist nebulised (salbutamol 2.5–5 mg) every 20 min with Ipratropium bromide (250 mcg) for first 2 h; review frequently
Oral steroids: 20 mg prednisolone (2–5 years); 30 to 40 mg (> 5 years). Repeat dose if vomiting or consider intravenous steroids (hydrocortisone 4 mg kg-1 every 4 h)
Give bolus of IV 50% magnesium sulphate 40mg/kg over 20mins (Max 2g) Diluted 1:5 0.9% saline.
Consider early single bolus dose of IV salbutamol where child has responded poorly to inhaled therapy followed by an infusion
Consider aminophylline if child unresponsive to maximal doses of bronchodilators and steroids
Consider ABG if poor response to early treatment.

If no pulse oximeter is available, check for cyanosis.

NRB – non-rebreather mask with reservoir
pMDI – pressurised metered-dose inhalers
Note: Evidence is unclear which of intravenous salbutamol, aminophylline or magnesium should be the first line in severe asthma.

Early management of asthma – September 2019. Based on the British Thoracic Society, Scottish Intercollegiate Guidelines
Network, British guideline on the management of asthma revised 2019

NOTE. Aminophylline and adrenaline should never be used as first line treatment for acute exacerbation of asthma, except in situations where no better alternative is available.

Discharge plan:

- Patient can be discharged when stable on 4 hourly salbutamol inhalations.
- To complete 3 days course of prednisolone.
- Patient/parent education to be done on the ward.
- Patients with mild exacerbation of asthma can be reviewed at the local clinic.
- Those with moderate, severe, or life-threatening exacerbations should be booked for review in the asthma clinic.
- **Reliever**: as-needed rather than routinely
- **Controller**: Higher dose for short term (1-2 weeks) or long term (3 months) depending on the background of the exacerbation.
- **Risk factors**: Check and correct modifiable risk factors including inhaler technique.
- Written **action plan**

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1.5 SHOCK

Shock is the inadequate perfusion of the vital organs of the body to meet metabolic demands.

There are 3 stages of shock:

1. Compensated shock: signs of tissue hypoperfusion (cool peripheries, CRT > 3sec, weak pulse) with normal blood pressure
2. Decompensated shock: hypotension, organ dysfunction with signs of impaired perfusion
3. Irreversible shock: multiple organ dysfunction (comatose, DIC, AKI, metabolic acidosis) with poor response to inotropic support

Table 1.1. Types of shock

Type	Mechanism	Causes
Hypovolemic	Fluid loss	Gastroenteritis, osmotic diuresis, haemorrhage, capillary leakage
Distributive shock e.g. septic, neurogenic, anaphylactic	Widespread vasodilatation	Sepsis, spinal cord trauma, anaphylaxis, drugs e.g. barbiturates, antihypertensives
Cardiogenic	Pump failure	CHD, arrhythmias, myocarditis, heart failure, cardiomyopathy
Obstructive	Obstruction to the flow of blood	Cardiac tamponade, tension pneumothorax, pulmonary embolism, duct-dependent cardiac lesions e.g. critical COA, HLHS

Table 1.2. Clinical presentation

History	Physical examination	Investigations
Fluid loss; gastroenteritis, DKA, GI bleeding Trauma; tension pneumothorax, cardiac tamponade, spinal injury Fever, immunocompromised states (HIV, SCD) Allergen exposure Ingestion of toxins Chronic heart disease; acquired or congenital	Vital signs – fever, tachycardia, hypotension Weight and length – evaluate for severe malnutrition Tachypnoea – acidotic breathing Abnormal breath sounds e.g. wheezing, stridor, crepitations Distended neck veins Abnormal heart sounds – gallop rhythm Differential pulse or blood pressure; upper extremities vs lower extremities Hepatomegaly Abnormal skin findings: oedema, purpura (meningococcaemia), urticaria (anaphylaxis)	Random blood sugar Urine dipstick Full blood count and cross match Serum urea, creatinine and electrolytes Blood lactate Arterial blood gas Clotting profile Chest xray, ECHO Point of care ultrasound (POCUS) Blood. Urine and stool cultures, CSF studies

WHO clinical criteria for shock

- Cold extremities, prolonged capillary refill time > 3 seconds, weak or fast pulse (note: in septic shock, peripherals may be warm and flushed, rapid CRT, bounding pulses)

WHO definition of hypotension

- Less than 1 year = SBP < 50 mmHg,
- 1 to 5 years = SBP < 60 mmHg,
- > 5 years = SBP < 70 mmHg.

MANAGEMENT OF SHOCK

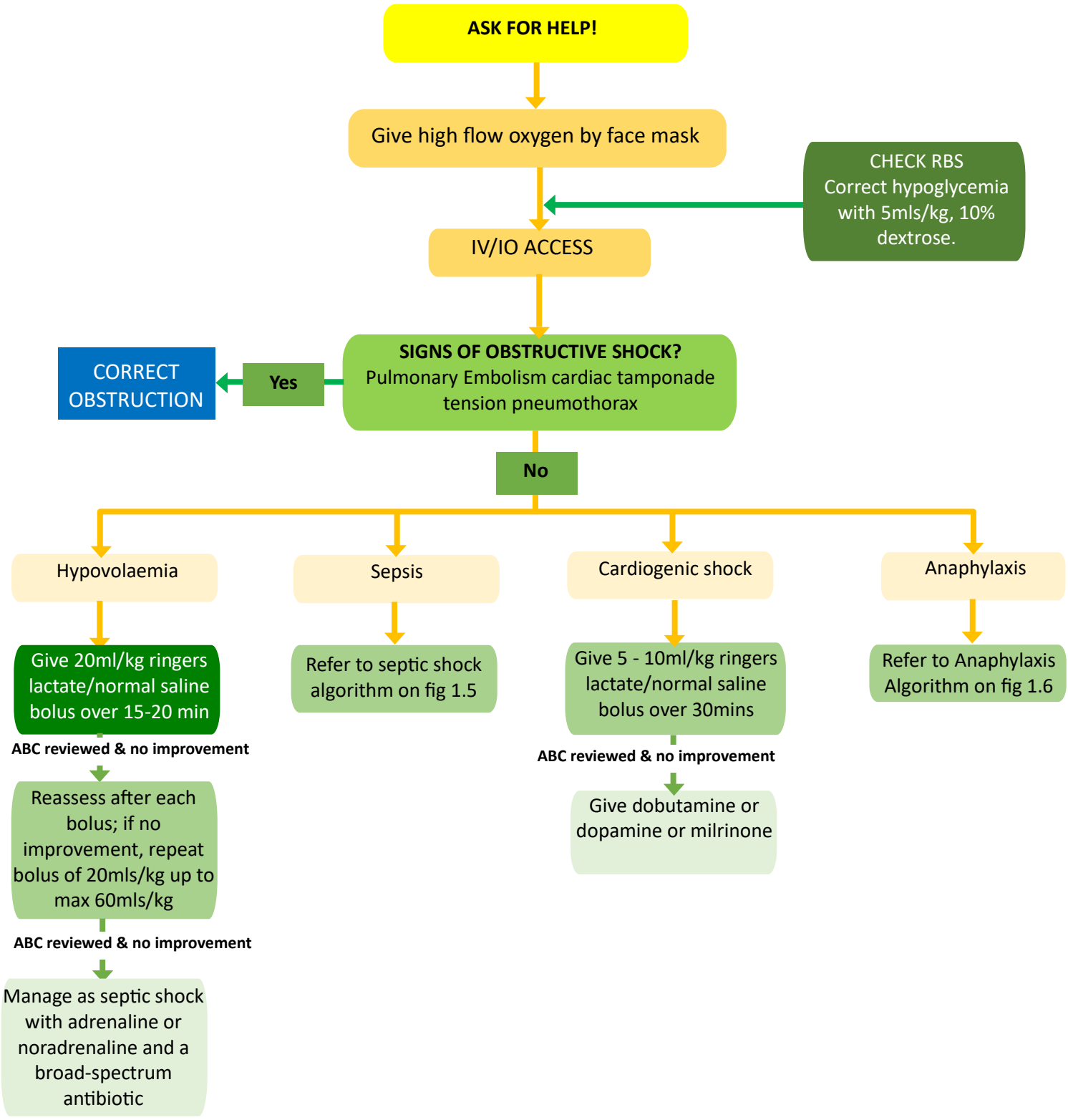


Fig 1.4. Management of shock

Table 1.3. Formulae for administration of vasoactive medications

Drug	Dose ranges	Infusion preparation	Infusion rate
Dopamine Dobutamine	Dopamine (3 to 20 mcg/kg/min) Dobutamine (1 to 20 mcg/kg/min)	Body weight in kg x 6 = Amount of drug (mg) to be added to total volume of 100 ml IV fluid (saline)	1 ml/h = 1 mcg/kg per minute (Example: to deliver 10 mcg/kg per minute, run infusion at 10 ml/h)
Epinephrine Norepinephrine Milrinone	Epinephrine (0.05 to 0.3 µg/kg/min) max 1.0µg/kg/min Norepinephrine (0.05 to 0.3 µg/kg/min) max 1.0µg/kg/min Milrinone 50 µg/kg IV push over 30 min then 0.25 – 0.75 µg/kg/min	Body weight in kg x 0.6 = Amount of drug (mg) to be added to total volume of 100 ml IV fluid	1 ml/h = 0.1 mcg/kg per minute (Example: to deliver 0.3 mcg/kg per minute, run infusion at 3ml/h)

SEPTIC SHOCK AND SEPSIS-ASSOCIATED ORGAN DYSFUNCTION IN CHILDREN

RECOGNITION

Assess with ABCDE approach

A, B assessment
- Airway, RR, work of breathing, oxygen saturations, breath sounds, recognition respiratory distress/failure.
- Open airway and start high-flow oxygen via non-rebreather mask with reservoir or BMV as appropriate.

C assessment
- HR, CRT, BP, peripheral and central perfusion, rhythm recognition; recognition circulatory failure/shock.
- Establish IV/IO access (take blood cultures, full blood count, blood glucose, urea and electrolytes, lactate*, blood gas and other bloods as indicated**) and give fluid resuscitation as below.

D assessment
- AVPU score; recognition of altered mental status secondary to poor perfusion.

E assessment
- Rash, temperature (high or low).

Sepsis is diagnosed if there is evidence of infection as cause of the acute illness (suspected or proven) plus at least two of the following: core temperature < 36°C or > 38.5°C; white cell counts elevated or depressed for age; inappropriate tachycardia; altered mental state; reduced peripheral perfusion.

10–15 MIN

Initial resuscitation

- If no signs fluid overload (hepatomegaly, crackles at lung bases) then give 10 mL kg^{-1} balanced crystalloids*** IV bolus over 5–10 min and re-assess after each bolus up to 40–60 mL kg^{-1} or until perfusion improved.
- Therapeutic end points: CRT < 2 s; normal BP for age; UO > 1 mL kg^{-1} h^{-1}, normal pulses, normal mental state.
- Watch for signs of fluid overload; if present stop bolus therapy and start inotropic support.
- Correct hypoglycaemia and hypocalcaemia.
- Start broad-spectrum antibiotics; seek and aggressively control any infection source.
- Call for more senior help and an anaesthetist urgently; call PICU for bed +/- PICU transfer team.
- If mechanical ventilation is required, then cardiovascular instability during intubation is less likely after appropriate cardiovascular resuscitation.

15–60 MIN

Fluid refractory shock?

Start IV/IO inotrope infusion; central (preferable) or peripheral IV (clinical signs unreliable at differentiating 'warm' and 'cold' shock in children).

Adrenaline 0.05–0.3 mcg kg-1 min-1 (use more dilute infusion if peripheral)

and/or

Noradrenaline via central IV or IO, starting infusion rate 0.05 mcg kg^{-1} min^{-1} Titrate inotropes upwards according to clinical response and haemodynamic effects using haemodynamic monitoring (where possible) ****

Use ketamine +/- atropine IV/IO/IM to gain central access and airway if needed.

Fluid and catecholamine-resistant shock?

Further management as per Paediatric Intensive Care/retrieval service advice.

Warm shock – high cardiac output with low systemic vascular resistance.	**Cold shock** – low cardiac output with high systemic vascular resistance.	**Fluid in mL kg^{-1} should be dosed for ideal body weight (max bolus 500 mL)**

* lactate measurements are useful if available as they have prognostic ability if measured serially.

** Other bloods that may be indicated: coagulation studies, liver function tests, magnesium levels or any others indicated by the child's clinical picture.

*** Balanced (buffered) fluids are used in preference to 0.9% sodium chloride, but if not available, 0.9% sodium chloride should be used.

**** These are starting dose ranges for these inotropes, and increases may be necessary but should be guided by PICU retrieval team/senior clinicians. Choice of inotropes is dictated by clinician preference, response to treatment and monitored parameters, and again decisions should be made in conjunction with PICU teams.

Fig 1.5. Adapted from resuscitation council UK guideline 2021

ANAPHYLAXIS

Anaphylaxis?

A = Airway **B** = Breathing **C** = Circulation **D** = Disability **E** = Exposure

Diagnosis – look for:

- Sudden onset of Airway and/or Breathing and/or Circulation problems[1]
- And usually skin changes (e.g. itchy rash)

Call for HELP
Call resuscitation team or ambulance

- Remove trigger if possible (e.g. stop any infusion)
- Lie patient flat (with or without legs elevated)
  - A sitting position may make breathing easier
  - If pregnant, lie on left side



Give intramuscular (IM) adrenaline[2]

Inject at **anterolateral aspect** – middle third of the thigh



- Establish airway
- Give high flow oxygen
- Apply monitoring: pulse oximetry, ECG, blood pressure

If no response:

- Repeat IM adrenaline after 5 minutes
- IV fluid bolus3

If no improvement in Breathing or Circulation problems[1] despite TWO doses of IM adrenaline:

- Confirm resuscitation team or ambulance has been called
- Follow REFRACTORY ANAPHYLAXIS ALGORITHM

1. Life-threatening problems

Airway
Hoarse voice, stridor

Breathing
↑work of breathing, wheeze, fatigue, cyanosis, SpO2 <94%

Circulation
Low blood pressure, signs of shock, confusion, reduced consciousness

2. Intramuscular (IM) adrenaline
Use adrenaline at 1 mg/mL (1:1000) concentration

Adult and child >12 years:	500 micrograms IM (0.5 mL)
Child 6–12 years:	300 micrograms IM (0.3 mL)
Child 6 months to 6 years:	150 micrograms IM (0.15 mL)
Child <6 months:	100–150 micrograms IM (0.1–0.15 mL)

The above doses are for IM injection **only**.
Intravenous adrenaline for anaphylaxis to be given **only by experienced specialists** in an appropriate setting.

3. IV fluid challenge
Use crystalloid

Adults:	500‑1000 mL
Children:	10 mL/kg

Fig 1.6. Adapted from resuscitation council UK guideline 2021

REFRACTORY ANAPHYLAXIS

No improvement in respiratory or cardiovascular symptoms despite 2 appropriate doses of intramuscular adrenaline

Establish dedicated peripheral IV or IO access

Seek expert1 help early
Critical care support is essential

Give rapid IV fluid bolus
e.g. 0.9% sodium chloride

&

Start adrenaline infusion
Adrenaline is essential for treating all aspects of anaphylaxis

Give IM* adrenaline every 5 minutes until adrenaline infusion has been started
*IV boluses of adrenaline are not recommended, but may be appropriate in some specialist settings (e.g. peri-operative) while an infusion is set up

Give high flow oxygen
Titrate to SpO2 94-98%

Monitor HR, BP, pulse oximetry and ECG for cardiac arrhythmia
Take blood sample for mast cell tryptase

Follow local protocol
OR
Peripheral low-dose IV adrenaline infusion:

- 1 mg (1 mL of 1 mg/mL [1:1000]) adrenaline in 100 mL of 0.9% sodium chloride
- Prime and connect with an infusion pump via a dedicated line

DO NOT 'piggy back' on to another infusion line

DO NOT infuse on the same side as a BP cuff as this will interfere with the infusion and risk extravasation

- In both adults and children, start at 0.5-1.0 mL/kg/hour, and **titrate according to clinical response**
- Continuous monitoring and observation is mandatory
- ↑ ↑ BP is likely to indicate adrenaline overdose

Continue adrenaline infusion and treat ABC symptoms
Titrate according to clinical response

A = Airway

Partial upper airway obstruction/stridor:
Nebulised adrenaline (5mL of 1mg/mL)

Total upper airway obstruction:
Consult a senior. Expert help needed.

B = Breathing

Oxygenation is more important than intubation
If apnoeic:

- Bag mask ventilation
- Consider tracheal intubation

Severe/persistent bronchospasm:

- Nebulised salbutamol and ipratropium with oxygen
- Consider IV bolus and/or infusion of salutamol or aminophylline
- Inhalational anaesthesia

C = Circulation

Give further fluid boluses and titrate to response:
Child 10 mL/kg per bolus
Adult 500-1000 mL per bolus

- Use glucose-free crystalloid (e.g. Hartmann's Solution, Plasma-Lyte®)

Large volumes may be required (e.g. 3-5 L in adults)

Place arterial cannula for continuous BP monitoring
Establish central venous access

IF REFRACTORY TO ADRENALINE INFUSION
Consider adding a second vasopressor in addition to adrenaline infusion:

- Noradrenaline, vasopressin or metaraminol
- In patients on beta-blockers, consider glucagon

Consider extracorporeal life support

Cardiac arrest – follow ALS ALGORITHM

- Start chest compressions early
- Use IV or IO adrenaline bolus (cardiac arrest protocol)
- Aggressive fluid resuscitation
- Consider prolonged resuscitation/extracorporeal CPR

[1]Intravenous adrenaline for anaphylaxis to be given only by experienced specialists in an appropriate setting.

Fig 1.7. Adapted from resuscitation council UK guideline 2021

1.6 STATUS EPILEPTICUS

CONCEPTUAL DEFINITION (ILAE 2015)

Status epilepticus is a condition resulting either from the failure of the mechanisms responsible for seizure termination or from the initiation of mechanisms, which lead to abnormally, prolonged seizures (after time point t1 - 5min) and can have long-term consequences (after time point t2 – 30min), including neuronal death, neuronal injury, and alteration of neuronal networks, depending on the type and duration of seizures.

CLASSICAL DEFINITION

Continuous convulsion lasting longer than 20 to 30 mins or the occurrence of serial convulsions between which there is no return of consciousness.

OPERATIONAL DEFINITION

Seizure lasting more than 5 min or recurrence of two or more seizures, between which there is incomplete recovery of consciousness

Every child who presents for clinical care while having a seizure must be treated as status epilepticus.

Causes

- Febrile seizures
- Known epileptic who is non-compliant to treatment, is on erratic supply of drugs, had sudden withdrawal of anticonvulsants, is sleep deprived or has an inter-current infection
- Initial presentation of epilepsy
- CNS infections
- Congenital malformations of the brain (lissencephaly, schizencephaly)
- IEMs especially in neonates
- Electrolyte abnormalities – hypocalcaemia, hypoglycaemia
- Drug intoxication – amphetamines, cocaine, phenothiazines, theophylline, TCAs
- Others – Reye's syndrome, lead intoxication, extreme hyperpyrexia, brain tumour.

Investigations

- FBC and DC
- Random Blood Sugar
- Electrolytes & creatinine
- LFTs
- Blood-gas analysis
- Lactate
- Toxicology
- CSF studies (after brain CT)

Treatment

There are 4 goals of therapy:

- Ensure adequate vital signs, systemic and cerebral oxygenation
- Terminate seizure activity
- Prevent seizure recurrence
- Establish the diagnosis and treat the underlying cause if possible

1. First steps in management include stabilizing the child- ensure airway is open and circulation is stable. Administer oxygen. Place an IV line if possible.
2. Immediately treat first with diazepam (or if available, Lorazepam).

- If IV access is available, use 0.2-0.3mg/kg of diazepam IV (or 0.1mg/kg of Lorazepam) and there after give glucose (5ml/kg of 10% dextrose)
- If no IV access, administer diazepam 0.5mg/kg rectally. **Diazepam should NOT be administered intramuscularly.**

3. 5 minutes

- Administer a 2nd dose of diazepam. **DO NOT EXCEED 2 DOSES.**
- Alternatively, after the 1st dose of diazepam is administered, and there are limited resources for respiratory support, consider proceeding to step 4

4. 10 minutes

- If the seizures do not stop and Phenytoin is available, administer 20mg/kg IV x 1 (max 1gm), ***(dilute in normal saline- 100mg in 50ml)*** given over 20minutes.
- **This must be administered in a new IV (not in dextrose) using a G22 cannula. Cardiorespiratory monitoring is also indicated due to risk for arrhythmias and hypotension.**
- An additional 10mg/kg can be administered if seizures continue 15 minutes after the first dose

5. If phenytoin is not available

- Stable respiratory status: administer 15-20mg/kg over 20 minutes (maximum dose 1gm) of **phenobarbital** with caution, very closely monitoring respiratory status.
- If respiratory status is not stable, provide available respiratory support. If there is an option for mechanical ventilation, emergently prepare to intubate the child if needed as you administer phenobarbital. If there is no mechanical ventilation available, choose one of the following based upon medication availability:
- Levetiracetam 40mg/kg (max dose 1gm) IV (preferred) or NGT
- Sodium valproate 20mg/kg (max dose 1gm) (only in children over 2 years) IV if no other option consider administration via NGT

- OR Phenobarbital 10mg/kg IV or IM x 1 and repeat the dose as you monitor respiratory status closely.

6. If the seizures continue, use any of the other medication options listed. **Alternatively, if no other options are available and there is no clear infection,** consider a trial of high dose steroids of 30mg/kg daily Methylprednisolone (max 1gm) x 3 days, followed by a one-week oral prednisolone taper.

- If available, other treatment options for refractory status epilepticus include midazolam infusion, ketamine, propofol, pentobarbital coma, thiopental and other inhaled anesthetics.

7. Evaluate (and treat as appropriate) for underlying cause of seizure:

- Consider head CT, and request urgent EEG (if feasible) if there is any concern for non-convulsive status epilepticus.
- Review medication compliance, dose and formulation for all children with prior Epilepsy diagnosis and re-dose home medication if indicated

8. If the child has high risk of seizure recurrence (e.g. CNS infection), maintain on anti-seizure medication until child can be seen in follow up (within 3 months)

9. If no provoking factor for SE can be identified, and the child does not have known epilepsy, proceed with investigations as noted in first unprovoked seizure guidelines

10. Children with known epilepsy and on medication who present in status epilepticus should have re-evaluation of treatment plan and adjustment of medication prior to discharge

11. All children who present in status epilepticus should be given a rescue medication (e.g. rectal diazepam) and instructions on seizure first aid.

MANAGEMENT OF STATUS EPILEPTICUS

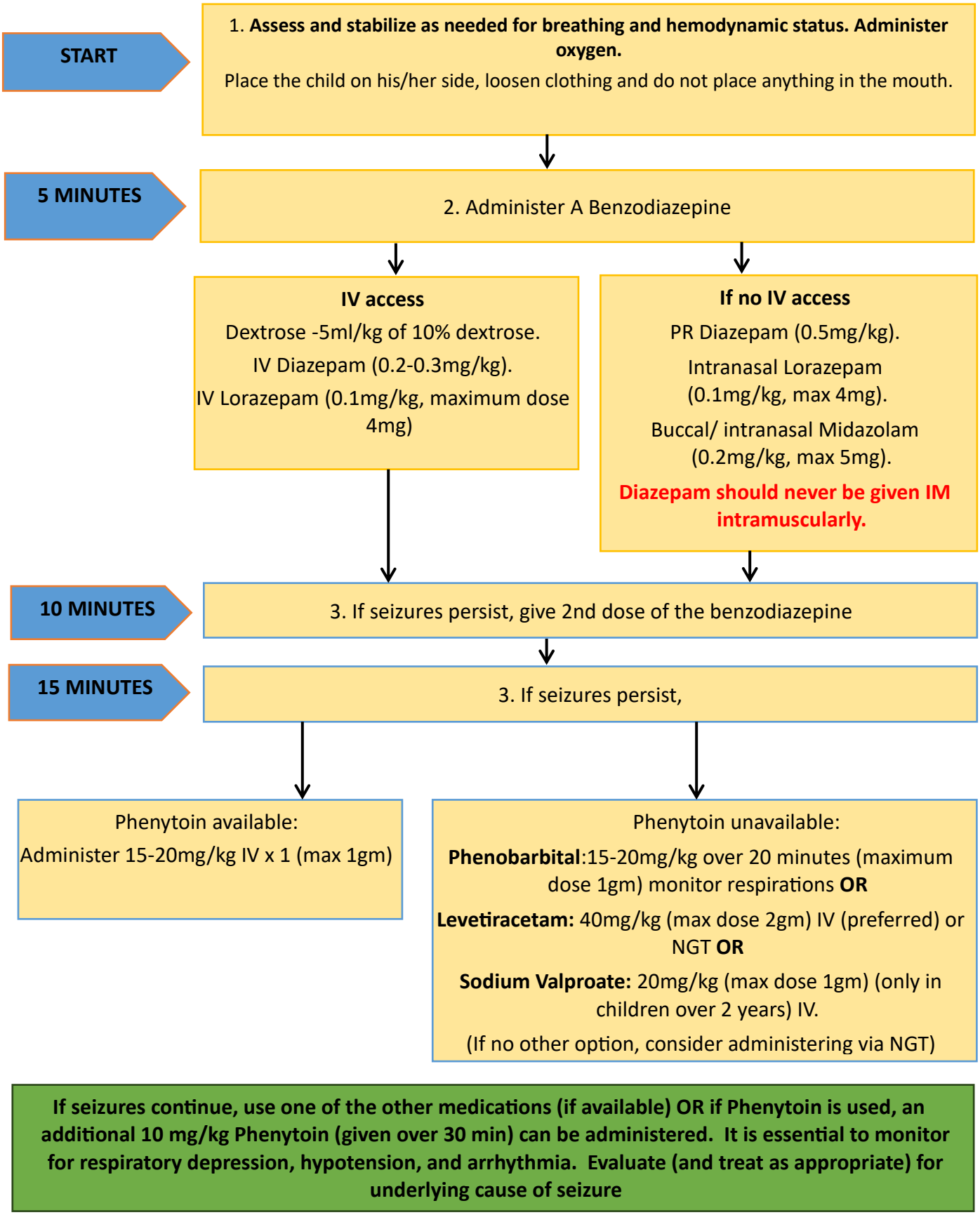


Fig 1.8. Guidelines for first line anti-seizure drug of choice-add..

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1.7 COMA

Coma is a state of deep unarousable unconsciousness with total loss of awareness of self and environment. Consciousness is awareness of oneself and surroundings in a state of wakefulness, altered by disease, poisoning or trauma. The goal of treatment is to limit brain injury.

Table 1.4. Causes of coma

Coma with fever	Coma without fever
• **Meningitis/sepsis** • **Cerebral malaria** • **Viral meningo-encephalitis**	• Metabolic disorders • Hypertensive encephalopathy • Post-ictal state • Non-Convulsive status epilepticus • Stroke • Poisoning/intoxications • Mass brain lesions with raised ICP

Focused clinical history

- Child's health and activity in the last 5 days (e.g. preceding viral illness)
- Any history of fever
- Any chronic condition (DM, SCA, epilepsy)
- Time of last meal
- Pre-existing neurological disability
- Recent trauma

General physical examination

- Check vital signs- for signs of increased intracranial pressure (bradycardia, hypertension, respiratory rate/pattern (Cheyne stokes breathing)- Cushing triad. Check for signs of trauma to rule out traumatic brain injury.
- Head – Full AF
- Eyes – Pupillary size and reaction to light and position (CN III, IV and VI palsies); jaundice
- Neck – Rigidity
- Odour – Metabolic disorders, poisoning
- Abdomen – Enlarged liver associated with hypoglycaemia
- Skin – Rash, trauma, haemorrhage

Table 1.5. Primary Assessment/Resuscitation

A	Airway: Assess patients airway patency Airway maneuvers: Head tilt - chin lift, jaw thrust. Check for obstruction or secretions	
B	Effort of breathing: Recessions, respiratory rate, grunting, flaring, use of accessory muscles. Effect of breathing: heart rate, oxygen saturation, skin colour, mental state, cyanosis. Pattern of breathing: e.g. acidotic, Cheyne- stokes.	
C	Circulation: Pulse volume, pulse rate, capillary refill time, BP	
D	Disability:	
	A **V**	Alert Verbal
	P	Pain
	U	Unresponsive
		Degree of coma (see below), posture (decorticate, decerebrate, hemiparesis), pupils, seizures
E	Exposure: Temperature, rash, evidence of poison	

Table 1.6. Degree of coma

This can be measured using: Glasgow Coma Scale

	> 1 Year		< 1 year	Score
EYE OPENING	Spontaneously		Spontaneously	4
	To verbal command		To shout	3
	To pain		To pain	2
	No response		No response	1
MOTOR RESPONSE	Obeys		Spontaneous	6
	Localizes pain		Localizes pain	5
	Flexion withdrawal		Flexion withdrawal	4
	Flexion-abnormal (decorticate rigidity)		Flexion-abnormal (decorticate rigidity)	3
	Extension (decerebrate rigidity)		Extension (decerebrate rigidity)	2
	No response		No response	1
	> 5 years	**2 – 5 years**	**0 – 23 Months**	
VERBAL RESPONSE	Oriented	Appropriate words / phrases	Smiles/ coos appropriately	5
	Disoriented/confused	Inappropriate words	Cries and is consolable	4
	Inappropriate words	Persistent cries and screams	Persistent inappropriate crying and/or screaming	3
	Incomprehensible sounds	Grunts	Grunts, agitated, and restless	2
	No response	No response	No response	1
		TOTAL PAEDIATRIC GLASGOW COMA SCALE (3 – 15):		

Investigations

- Blood glucose
- Malaria parasite slide/RDT
- FBC, Renal function tests, Electrolytes (calcium, phosphate, Mg), blood culture,
- LFTs
- Blood/
- Urine toxicology
- CSF studies (if not contraindicated)
- EEG (if non convulsive status epilepticus is suspected)
- Imaging studies
    - X-rays in order to localize infection (e.g. Chest x-ray to r/o disseminated TB)
    - Neuro-imaging based on clinical findings and judgement. (CT is quick, MRI if possible)

Management

- Pay meticulous attention to Airway, Breathing, Circulation and Disability
- Intubabte if GCS <8
- IV access, Oxygen support
- Check RBS (if not available, give 5mls/kg of 10% dextrose IV)
- Treat for raised ICP, if present- See next chapter
- Treat any seizure (refere to seizure protocol)
- treat infections
- keep euthermic
- check and treat for possible intoxication
- treat electrolyte imbalnces
- Nursing care (skin care, oral hygiene, eye care, feeds, fluid balance)
- Further treatment depends on the provisional diagnosis

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1.8 RAISED INTRACRANIAL PRESSURE

Intracranial pressure (ICP) is the pressure within the cranium and is measured in mm Hg. Normal values are 5-15 mmHg. Intracranial hypertension (or raised ICP) is defined as sustained ICP levels above 20mmHg.

CAUSES

- Traumatic Brain Injury, with mass effect
- Large vessel ischemic stroke (Carotid or MCA occlusion)
- Cytotoxic cerebral Oedema with mass effect.
- Intracranial haemorrhage with mass effect.
- Hydrocephalus
- Diffuse cerebral Oedema (hepatic failure, cerebritis/encephalitis/meningitis)
- Jugular venous obstruction or elevated right sided cardiac venous pressure
- Brain neoplasms causing mass effect or vasogenic Oedema
- Idiopathic Oedema

CLINICAL PRESENTATION

- The clinical picture varies and may be age dependent.
- Infants may have irritability, vomiting (worse in the early mornings), or lethargy, lack of interests in surrounding and poorly feeding. altered mental state
- Children may present with headache, vomiting (worse in the early mornings), raised blood pressure with bradycardia or tachycardia. The cushing's triad of hypertension, bradycardia and respiratory depression are a late sign of herniation.
- Papilloedema
- visual changes (double vision, reduced visual acuity, new squint).

Focused history

- Pain/vomiting with Valsalva maneuvers
- Headache
- vision
- Nausea
- Vomiting

Examination

- Papilledema in awake patients (may proceed to stupor and coma over time)
- Bradycardia, hypertension and Cheyne-Stokes breathing in comatose patients.
- Papillary dilation, extensor or flexor posturing and Cheyne stokes breathing, as a result of acute stupor or coma
- Nuchal rigidity
- Retinal haemorrhage
- Bulging fontanel and sun-setting eyes in infants
- CN III (often ipsilateral anisocoria), IV, or VI palsies (VI very common!)

Management

In addition, ensure that the temperature, glucose, blood pressure and Hb are within normal limits.

Pain management is also an essential part of treatment therefore, ensure that the patient has adequate analgaesia.

For intubated patients

- head position must be midline
- If ventilated avoid high PEEP and PIP, as this increases the intrathoracic pressure and impedes venous return.
- suction with caution to avoid eliciting the gag reflex as this may increase the ICP
- adequate sedation to allow for adequate ventilation

FLOWCHART FOR THE MANAGEMENT OF RAISED ICP

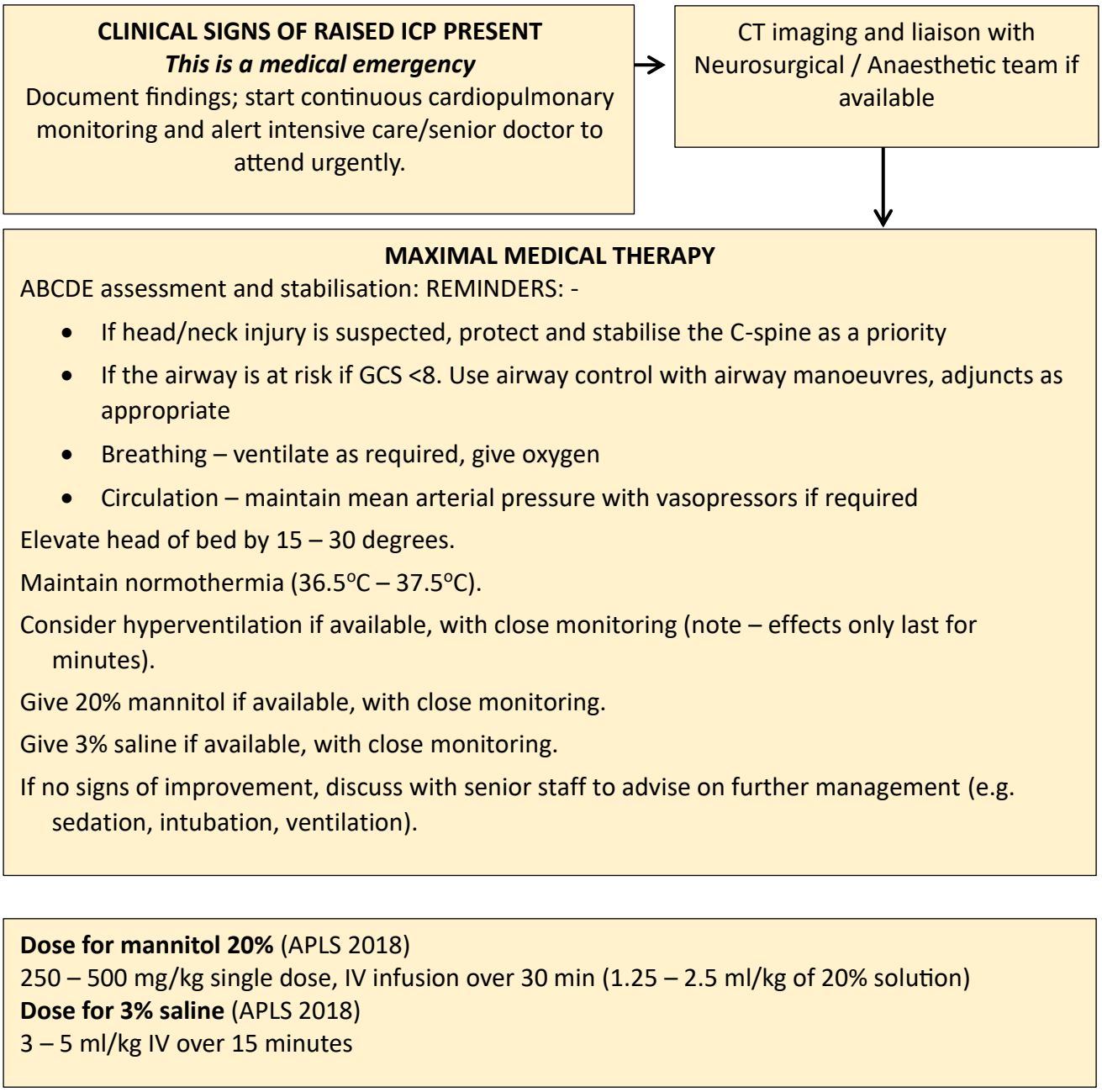


Fig 1.9. Management of raised ICP

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1.9 DIABETIC KETOACIDOSIS (DKA)

Diabetic ketoacidosis (DKA) is the state of uncontrolled catabolism associated with insulin deficiency, resulting in: hyperglycaemia (RBS >11mmol/L), osmotic diuresis, dehydration and ketonaemia (>3mmol/L), acidaemia (blood pH <7.3), significant ketonuria (2+or more or standard urinalysis). It is the commonest endocrine emergency usually encountered in previously diagnosed diabetics.

Causes

It is caused by increased fatty acid metabolism and the accumulation of fatty acids due to reduced insulin activity or deficiency of insulin during an intercurrent illness or in a new presentation of diabetes.

Clinical features

- Dehydration
- Abdominal pain
- Ketone smell on the breath
- Acidosis
- Acidotic breathing/hyperventilation
- Unexplained coma
- Nausea
- Vomiting

In DKA, a child may die from hypokalaemia or cerebral oedema. Cerebral oedema is unpredictable, occurs more frequently in younger children and new diabetics, and has mortality of around 80%.
These guidelines are intended for the management of children who are more than 5% dehydrated and/or vomiting, drowsy or clinically acidotic.

Investigations

- Random glucose
- U/E, Creatinine, LFTs
- FBC
- Malaria parasite slide
- Urinalysis
- Urine m/c/s
- Arterial blood gas

Treatment

General resuscitation: A, B, C.

Airway: Ensure that airway is patent. If comatose, insert an airway. If comatose and has recently vomited, insert a NGT, aspirate and leave on open drainage.

Breathing: Give 100% oxygen ***where indicated***. Bag and mask ventilation if apnoeic.

Circulation: **Insert two large bore IV cannulae** and take blood samples.

If shocked (tachycardia with poor capillary refill time or hypotension) give 20 ml/kg 0.9 saline as quickly as possible, and repeat if necessary up to a max of 60mls/kg.

Confirm the diagnosis:

- History: Polydypsia, polyuria
- Clinical: Acidotic respiration; dehydration; drowsiness; abdominal pain/vomiting
- Biochemical: High glucose, ketones or glycosuria, ketonuria
- Conscious Level
  - GCS or BCS
  - Cerebral oedema: Irritability, slow pulse, high BP. Fundoscopic examination.
- Look for the focus of infection *and* ***treat accordingly.***
- Observation to be carried out:
  - Strict fluid balance chart
  - Hourly blood glucose
  - Daily weights
  - GCS or BCS
  - ECG

Fluids

Requirement = Deficit + Maintenance

- Deficit = % dehydration x body weight
  - Severe dehydration 10% deficit – give 100ml/kg over **48 hours**
  - Some dehyration 7.5% deficit – give 75ml/kg over 48 hours
  - No dehydration <5% deficit – give oral fluids as tolerated by patient.

- Maintenance fluids over **24 hours** (using Holiday-Sager)
  - 100mls/kg-1st 10kgs
  - 50mls/kg- next 10kgs
  - 20mls/kg for every kg thereafter.

Add deficit and maintenance and give over the next 36 – 48 hrs

Example:

For a severely dehydrated (deficit 10%) 25kg diabetic patient, fluid is calculated as follows:

Deficit IVF = 100ml/kg x 25 = 2.5L =2500mls, to be given over 48 hrs, therefore 1250mls to be given over 24hrs

Maintenance IVF = 1000 mls + 500 mls +100mls = 1600mls/24hrs

Therefore, total amount of fluid to be given over 24hrs is:

1600mls+1250mls =2850mls

This may be divided by 3 or 4 to be given at 950 mls 8 hourly or ~710 mls 6 hourly respectively.

Type of fluid is:
- 0.9% normal saline
-Change to DNS once RBS has fallen to around 12 mmol/l.

In the presence of hypernatraemia, then half normal saline must be used (***please consult***).

Potassium

Commence potassium immediately unless anuria is suspected or there are peaked T waves on ECG or the serum potassium is > 7 mmol/l.
Add 10 to 20 mmol KCl to every 500 ml intravenous fluids given for the first 24 hrs.

Insulin

1. Short acting insulin (eg soluble) while in DKA
Short acting insulin at 0.05 to 0.1 IU/kg/hour continuous intravenous infusion.

- Insulin infusion should be run in a different IV line from the one running IVF
- Average rate of fall of blood glucose must not exceed 5mmol/l per hour.
- If the drop in blood sugar is more rapid (>5mmol/l per hour)

Example of how to calculate insulin in a 20kg child

2. Transitioning to subcuteanous (subcut) insulin once out of DKA
Once a patient is out of DKA, is fully conscious, hemodynamically stable and is taking feeds orally, they are transition from iv insulin to subcut insulin.

The insulin dosage in the twice daily dosing regimen is based on the hourly dosage of insulin that was used to get the patient out of DKA.

Table 1.7 Insulin Dosage

STAGE OF MANAGEMENT	INSULIN REGIMEN	NOTES
During acute DKA	IV short acting (soluble) insulin 0.05-0.1 iu/kg/hour	• Insulin should be given in a different infusion line from the one for IVF • Patient should be NPO • Give for at least 24 hours before transitioning to subcut insulin • If a patient has been transitioned to dns and the serum glucose falls to below 7mmol/L, the insulin should not be stopped but a sliding scale should be used and consult the senior
After moving from IV to subcut, while in hospital	Subcut soluble insulin 6 hou	• Transition to this regimen when patient is fully conscious, haemodynamically stable, taking feeds orally
In readiness for discharge	Subcut short acting and intermediate	• The IV insulin should be stopped 30 minutes after the subcut insulin is commenced

Example

A 20kg child that was treated with soluble insulin of 0.05 iu/kg/hr while in DKA and is now being transitioned to subcut insulin will receive insulin as follows:

1. 6-hourly short acting regimen while in hospital

- Insulin dose = 0.05iu x 20kg = 1.0IU/hour
- Total dose of insulin in 24 hours = 1.0IU x 24 hours = 24IU
- Frequency of administration in 24 hrs will be 6 hourly, therefore = 24IU/4 = 6IU (if it is an odd number, round it off to the nearest even number to arrive at the total daily dose)
- Type of insulin to be used: short acting insulin (e.g. soluble)

2. 12 hourly regimens in readiness for discharge (if above patient has achieved normoglycaemic control on 1.0iu/hour)

- insulin dose patient controlled on = 1.0 IU/hour
- total dose of insulin in 24 hours = 24IU
- types on insulin to be used: short acting insulin (e.g. soluble) and intermediate acting insulin (eg Protaphane) or a combination of both (Premixed or Mixtard)
- of the total dose of insulin, 2/3 should be given in the morning and 1/3 in the evening

- of the morning dose, 1/3 should be short acting insulin and 2/3 should be long acting insulin
- of the evening dose, 1/3 should be short acting insulin and 2/3 should be long acting insulin

  NOTE: if it is an odd number, round it off to the nearest even number to arrive at the total daily dose of insulin

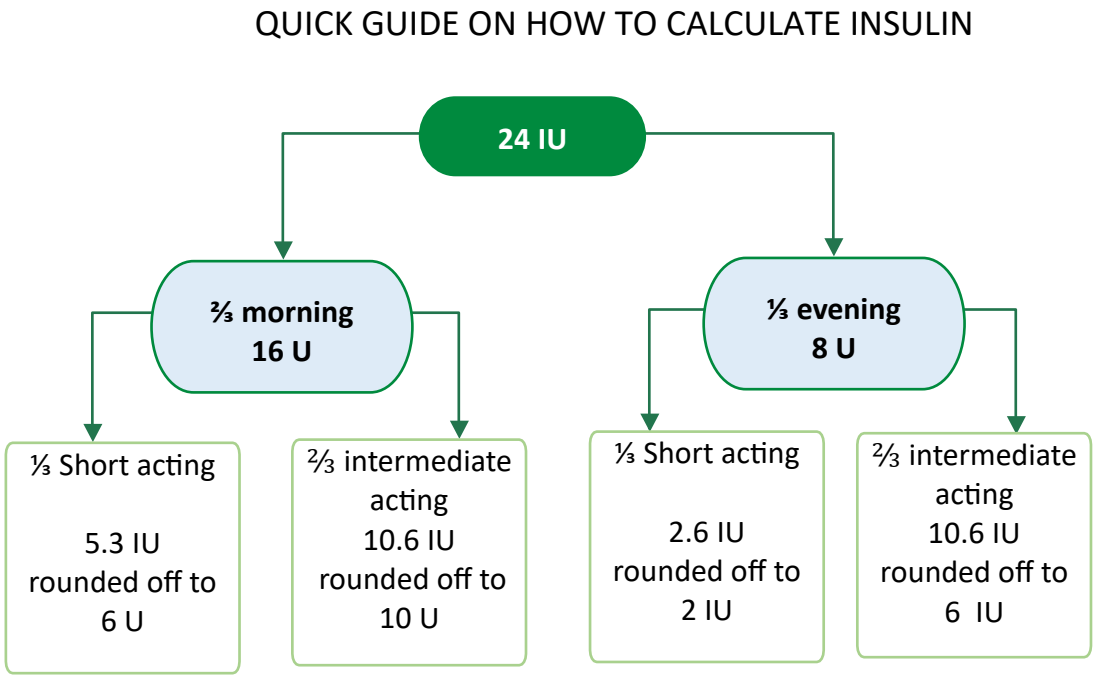


Fig 1.1.1. Guide to insulin calculation

Note:

Information for patients and caregivers before discharge

1. Patient and caregiver should have been taught how to administer insulin
2. Diet counselling should have been given using locally available foods
3. Patient and caregiver should have been taught how to identify and manage complication e.g. hypoglycaemia
4. A follow up plan should have been made
5. Patient should be taught how to store insulin. Patients without a fridge should be taught how to use locally appropriate technology
6. If able, the patient should be advised to buy a glucometer for home use and counselled on how to interpret and respond the readings

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1.1.1 ASSESSMENT AND MANAGEMENT OF ACUTE PAIN IN CHILDREN

Pain assessment is an important component of emergency department care. Children present unique challenges to assessing pain due to their constantly changing developmental ability to understand concepts, communicate verbally, and use common pain severity rating scales. Pain severity in young and noncommunicating cognitively impaired children is rated using behavioural scales. School-aged children use faces scales to self-report pain severity, whereas older preteens and adolescents can use the verbal numeric rating scale.

Children less than 3 years old

These must be assessed through pain behaviours such as facial grimacing and crying.

Children 3 years old and above

You can use the Wong-Baker Faces Pain Rating Scale. The person administering the tool explains to the child that each face represents a different level of pain intensity. He asks the child to choose the face that best describes their own pain. The clinician scores the chosen face with a numerical rating between 0 (no pain) and 10 (extreme pain). He should avoid using words like "happy" and "sad" as the scale measures how the child feels inside, not how their face looks



For the older child, you can use the Verbal Report Scale.

The Verbal Report Scale is a self-reported tool used across units at the University Teaching Hospitals (UTHs)- Children's Hospital.

The person administering this tool says to the patient, "On a scale of 0 to 10, with 0 being 'no pain' and 10 being 'the worst pain ever', how would you rate your pain?"

A child with mild pain:

- rates his/her pain as 1–3 on a Verbal Report Scale (of 0–10).
- may not appear uncomfortable, but complains of pain.
- may or may not be crying, may have a neutral facial expression, and may be at rest or shifting position in chair.

A child with moderate pain:

- Rates his/her pain as 4–6 on the Verbal Report Scale (0–10).
- May show facial grimacing, unhappiness, irritability, and a poor appetite.
- May (according to the family) have been unable to carry out normal daily activities, or been uninterested in social interaction.

A child with severe pain:

- Rates his/her pain as > 7 on the Verbal Report Scale (0–10).
- Show facial grimacing, unhappiness, irritability, and a poor appetite.
- Is generally crying, very uncomfortable to the point of agony.

Management

- Hospital admission is mandatory if pain control with oral analgesics is not adequate.
- Patient should receive analgesia 30 minutes within hospital arrival.
- Assess severity of pain.
- If in mild pain, give paracetamol 15 mg/kg with NSAIDs (E.g., Ibuprofen 10 mg/kg with food given 3 times a day). Maximum 30 mg/kg per day.
- If in moderate pain give or add tramadol 50- 100 mg orally 4- 6 hourly, for children > 12 years of age, adjusted according to response. Usual maximum 400 mg/ 24 hours.
- If in severe pain give or add opioid IV or oral morphine. For IV morphine, give 0.1 mg/ kg 4 hourly adjusted according to response. For oral morphine, give 0.2- 0.3 mg/kg (maximum per dose 10 mg) 4 hourly, adjusted according to response.
- For patients on morphine, give stool softener (e.g. lactulose) unless the child has diarrhoea.

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The body loses fluids through urine, insensible loses (respiration, skin, stools) and intrinsic losses (by-product of metabolism).

2.1 FLUID AND ELECTROLYTE THERAPY-MAINTENANCE

The goal of maintenance therapy is to replace fluid and electrolyte needed by the average **child with normal renal function** over a 24-hour period.
Calculating maintenance fluids: Holliday-Segar method.

Table 2. Fluid and Electrolyte Therapy-Maintenance

	Fluid per day	Rate per hour
1st 10kg	100 mL/kg/day	4 mL/kg/hr
Next 10kg	50 mL/kg/day	2 mL/kg/hr
For every additional kg	20 mL/kg/day	1 mL/kg/hr

Examples

6kg child
6 x 100mL/kg/day
= 600mL per day

27kg child
(10 x100) + (10 x 50) + (7 x 20)
= 1,640mL per day

Maintenance electrolyte requirements in children are:

- 2-3 mmol/kg/day of sodium,
- 1-2 mmol/kg/day of potassium
- 3-5 mmol/kg/day of chloride

Maintenance fluid in critically ill children

Holliday-Segar method assumes 1ml of fluid is required for every kcal of energy consumed (Study was done on healthy children).

- Children in PICU consume significantly less energy due to extreme inactivity as a result of sedation and/or paralysis
- Mechanical ventilation reduces work of breathing and hence, calorie consumption
- Inspiration of humidified air reduces insensible losses
- Positive pressure ventilation, CNS insults and lung pathologies increase secretion of ADH.

Consider restricting maintenance fluid to two thirds (2/3) in such patients

2.2 HYPONATRAEMIA

Hyponatraemia is serum sodium (Na) concentration of less than 135 mmol/L. Many patients presenting with acute neurological symptoms may have unrecognised hyponatraemia.

Severity

- Mild hyponatraemia – Serum concentration between 130 and 134 mEq/L
- Moderate hyponatraemia – Serum concentration between 120 to 129 mEq/L
- Severe hyponatraemia – Serum concentration <120 mEq/L

Table 2.1. Causes of Hyponatraemia

Hypervolaemic	Hypovolaemic	Normovolaemic
Nephrotic syndrome	GI losses: Vomiting, Diarrhoea, Fistula	Syndrome of inappropriate ADH secretion (SIADH)
Acute & Chronic Renal failure	Third spacing: Pancreatitis, Ascites, Effusions, Burns	Hypotonic fluid hydration
Cirrhosis	Drugs: Diuretics	Hypothyroidism
Congestive Heart Failure	Adrenal insufficiency	

Clinical features

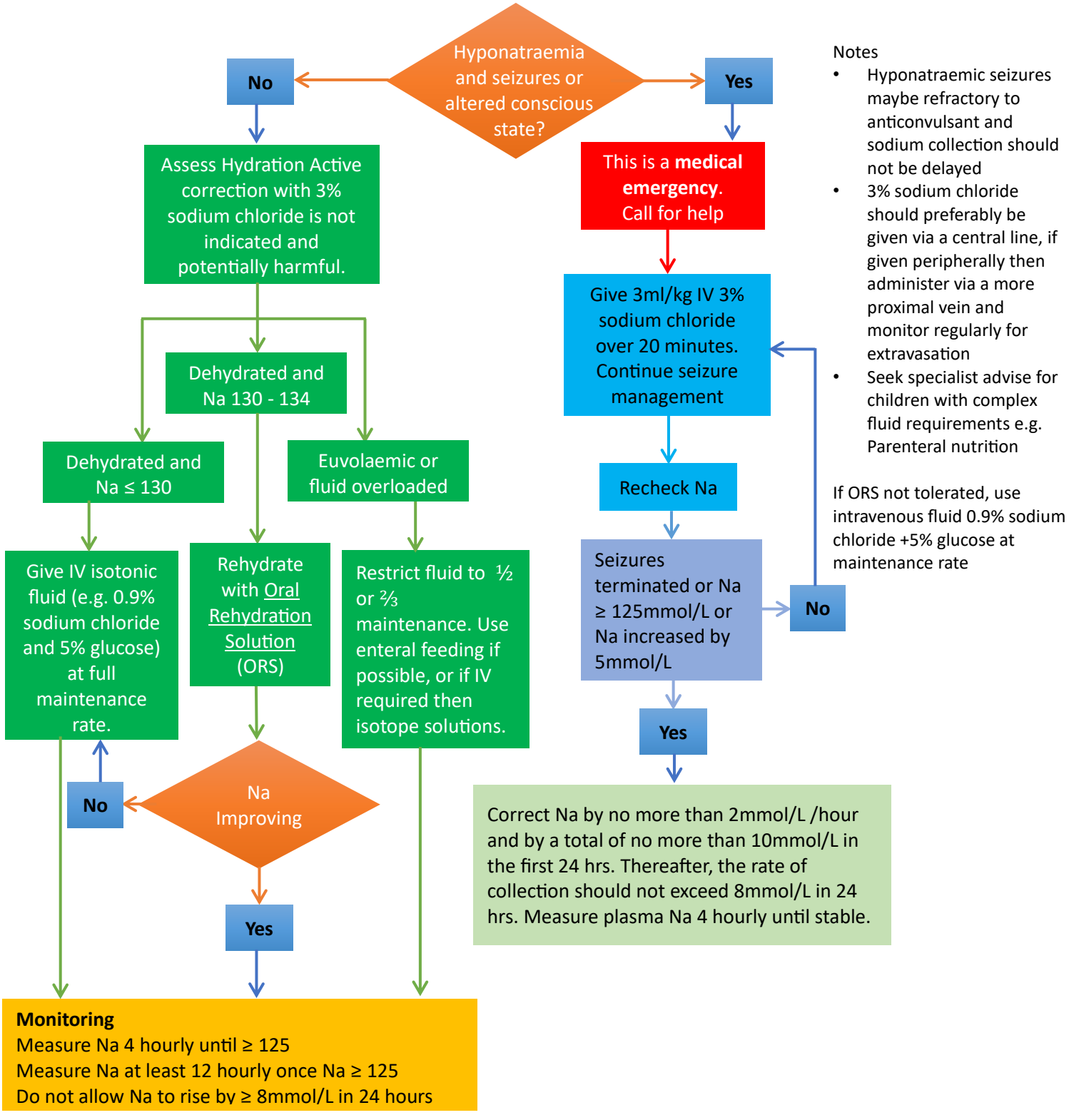
Symptoms occur when serum Na levels fall below 125 mmol/L. If the changes are chronic, the patient may be asymptomatic. These are:

- Early signs: anorexia, headache, nausea & vomiting
- Hypo/hypertension, muscle weakness & cramps,
- Bizarre behaviour, hallucinations, seizures, decorticate/decerebrate posture, coma

Investigations

- Serum Na+; serum osmolality; Urea & creatinine; Urine osmolality & Na+
- Aldosterone, cortisol, free T4 & TSH, ACTH & ADH levels
- Imaging studies for underlying cause – U/S & CT scans (head, abdomen)

ALGORITHM FOR EVALUATION OF HYPONATRAEMIA



Notes

- Hyponatraemic seizures maybe refractory to anticonvulsant and sodium collection should not be delayed
- 3% sodium chloride should preferably be given via a central line, if given peripherally then administer via a more proximal vein and monitor regularly for extravasation
- Seek specialist advise for children with complex fluid requirements e.g. Parenteral nutrition

If ORS not tolerated, use intravenous fluid 0.9% sodium chloride +5% glucose at maintenance rate

NOTE: Osmotic Demyelination Syndrome
Brain injury due to too rapid correction of chronic hyponatraemia is often associated with delayed neurological deterioration (onset of symptoms 1 to 4 days after serum Na is increased by > 12 mmol/L in less than 24 hours).

Fig 2. Approach to hyponatraemia

MANAGEMENT

Correction of hyponatraemia should be slowly, not exceeding 8 -10 mmol/L/day.

Severe Hyponatraemia with neurologic symptoms	Severe hyponatraemia without neurologic symptoms
Raise the serum Na concentration by giving doses of 1-2 ml/Kg of 3% saline until symptoms resolve. Symptoms typically resolve with a rise in sodium of 3-7 mmol/L. Note: 3% NaCl = 513meq/L	Deficit = Body weight (kg) x 0.6 x (125 – serum Na) Replaced using 3% NaCl IV or oral NaCl Tablets

Hypovolaemic hyponatraemia

- Correct volume depletion with normal saline, then restriction of fluids to two-thirds (or less) of the volume needed for maintenance

Normovoalemic hyponatraemia due to SIADH

- Include fluid restriction to two-thirds (or less) of the volume needed for maintenance using normal saline;
- Or the use of 3% NaCl, and IV administration of furosemide

Hypervolaemic hyponatraemia

- Restrict fluids and administer 3% NaCl to stop the symptoms

Remember: Treat the underlying cause

NaCl Preparations available

3% NaCl = 513 meq/L
NaCl Tablets = Check drug leaflet
Table Salt = 58.44 mmol/mg

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2.3 HYPERNATRAEMIA

Hypernatraemia is a serum sodium level greater than **145 mmol/L.**

Table 2.3. Hypernatraemia (Causes)

Excessive Sodium	Water Deficit	Water and Sodium Deficit
Improperly mixed formula	Nephrogenic diabetes insipidus	Diarrhoea
Cow's milk (UHT, Fresh milk)	Central diabetes insipidus	Emesis
Excess sodium bicarbonate	Increased insensible losses	Burns
IV hypertonic saline	Inadequate intake	Chronic kidney disease
		Osmotic diuresis

Suspect Hypernatraemic Dehydration
In Any Neonate Presenting With History Of Poor Breastfeeding And Weight Loss > 10% of Birth Weight

Clinical Features

- Irritability
- Restlessness
- Lethargy
- High pitched cry
- Hyperpnoea
- Cerebral haemorrhage
- Seizures
- Coma
- Stroke

ALGORITHM FOR HYPERNATRAEMIA

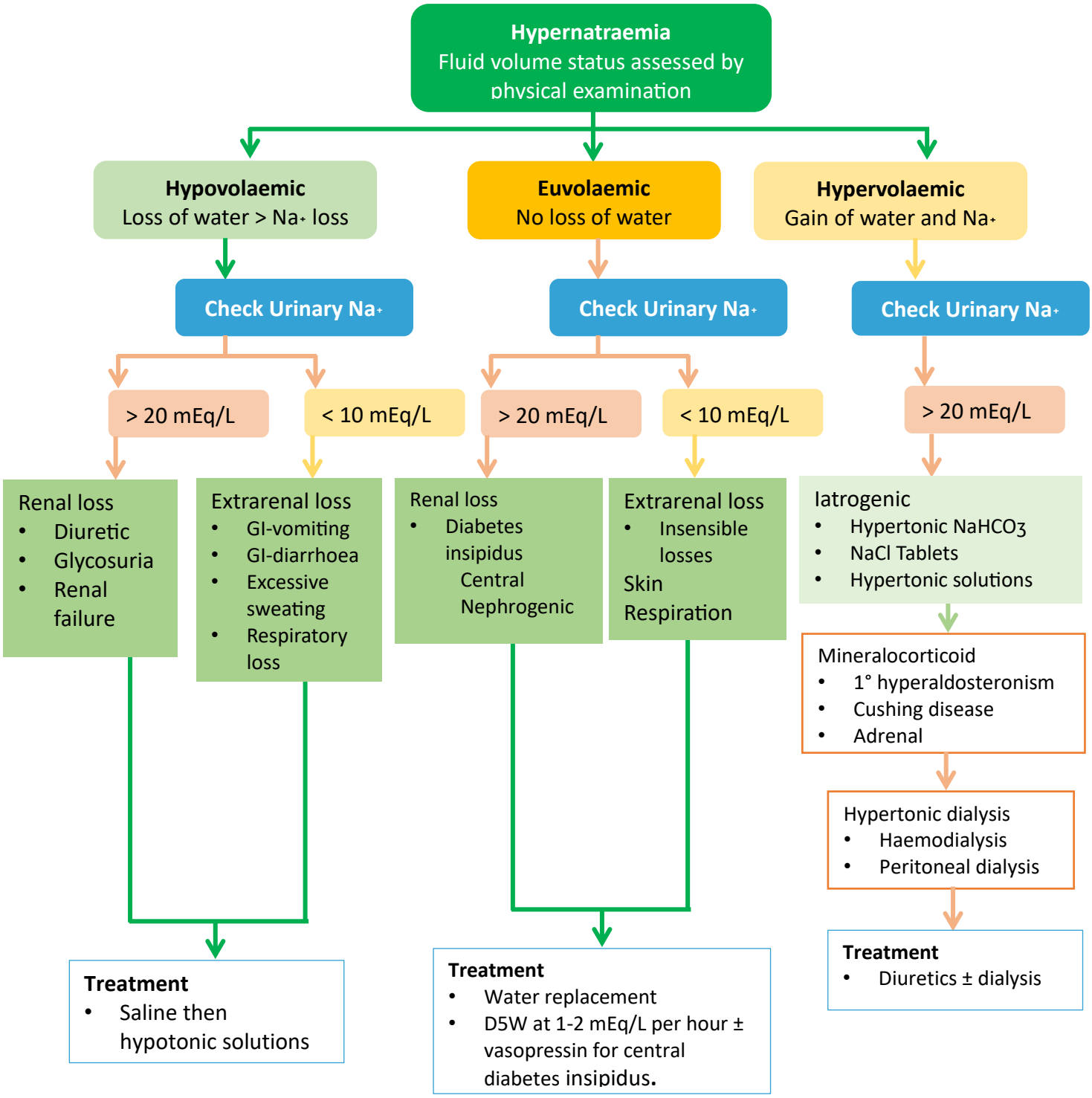


Fig 2.1. Diagnosis and management of Hypernatraemia.

D5W indicates 5% of dextrose in water; GI, gastrointestinal tract; Na, sodium; NaCl, sodium chloride; $NaHCO_3$, sodium bicarbonate.

Complications

- Dural sinus thrombosis
- Peripheral thrombosis
- Renal vein thrombosis

Management

Priority is restoration of intravascular volume with isotonic fluid (slow infusion of normal saline).

Firstly, the water deficit is calculated as follows:

Water deficit (in Litres): = Body weight × 0.6 (1-145/ [current sodium])

Replacement fluid is then calculated as follows:

Replacement volume (in L)
= TBW deficit X [1 ÷ 1 - (Na concentration in replacement fluid in mmol/L ÷ 154 mmol/L)]

The volume calculated must be replaced slowly over 48-72 hours.
For example:

A 10kg child arrives with a history of dehydration. His labs reveal a Sodium 160 mmol/l.
Water Deficit: 10 x 0.6 x (1-145/160) = **0.6 litres**
Fluid available is ½ Normal Saline: Sodium content in Saline = 77 mmol/L.
Therefore:-
replacement volume: 0.6 litres x [1/1- (77/154)] =**1.2 litres**
This volume of fluid should be given over 48-72 hours in addition to the daily maintenance fluid requirement.

Please monitor sodium **six hourly.** Aim at not dropping sodium by more than 0.5 mmol/hour or a total of 12 mol in any 24 hour period.

Other measures

- Severe cases (>200 mmol/L) may require dialysis with a high glucose-low sodium dialysate.
- Hyperglycaemia resulting from hypernatraemia is not usually treated with insulin as rapid lowering of glucose may precipitate cerebral oedema.
- The underlying cause should be treated whenever possible

Central diabetes insipidus should be treated with desmopressin

2.4 HYPERKALAEMIA

Hyperkalaemia is defined as serum potassium (K) level of more than 5mmol/L or 6mmol/L in neonates.

Table 2.4. Classification of Hyperkalaemia

Mild	Moderate	Severe
K+ >5.5 mmol/L	K+ 6-7 mmol/L	K+ >7.0 mmol/L or at risk of increasing and/or
Asymptomatic	Asymptomatic	Patient symptomatic and/or
Normal ECG	ECG may be normal	ECG disturbance

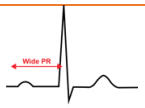
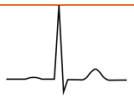
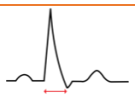
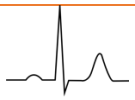
Table 2.5. Causes of Hyperkalaemia

Increased intake	Decreased excretion	Transcellular shift	Drugs
Intravenous or oral	Renal failure	Rhabdomyolysis	Angiotensin Converting Enzyme inhibitors
Blood transfusions	Urinary tract obstruction	Tumor Lysis Syndrome	Angiotensin II Receptor blockers
	Renal tubular disease	Severe Hemolytic processes	Potassium sparing diuretics
	Renal transplant		Non-steroidal anti-inflammatory drugs

Clinical Features

- Paraesthesia
- Weakness
- Tingling
- Symptoms of cardiac conduction abnormality e.g.
    - Palpitations
    - Syncope
    - Cardiac arrest

Table 2.6. ECG Changes

ECG CHANGES (HYPERKALAEMIA)	
Normal ECG	
wide P-R interval	
flattening p wave	
widening QRS complex	
peaked T waves	
Ventricular fibrillation	
Asystole	

Management

Aims

- Stabilise the heart to prevent life threatening arrhythmias
- Remove potassium from the body
- The flow chart (fig 2.2) demonstrates the step by step approach

APPROACH TO HYPERKALAEMIA

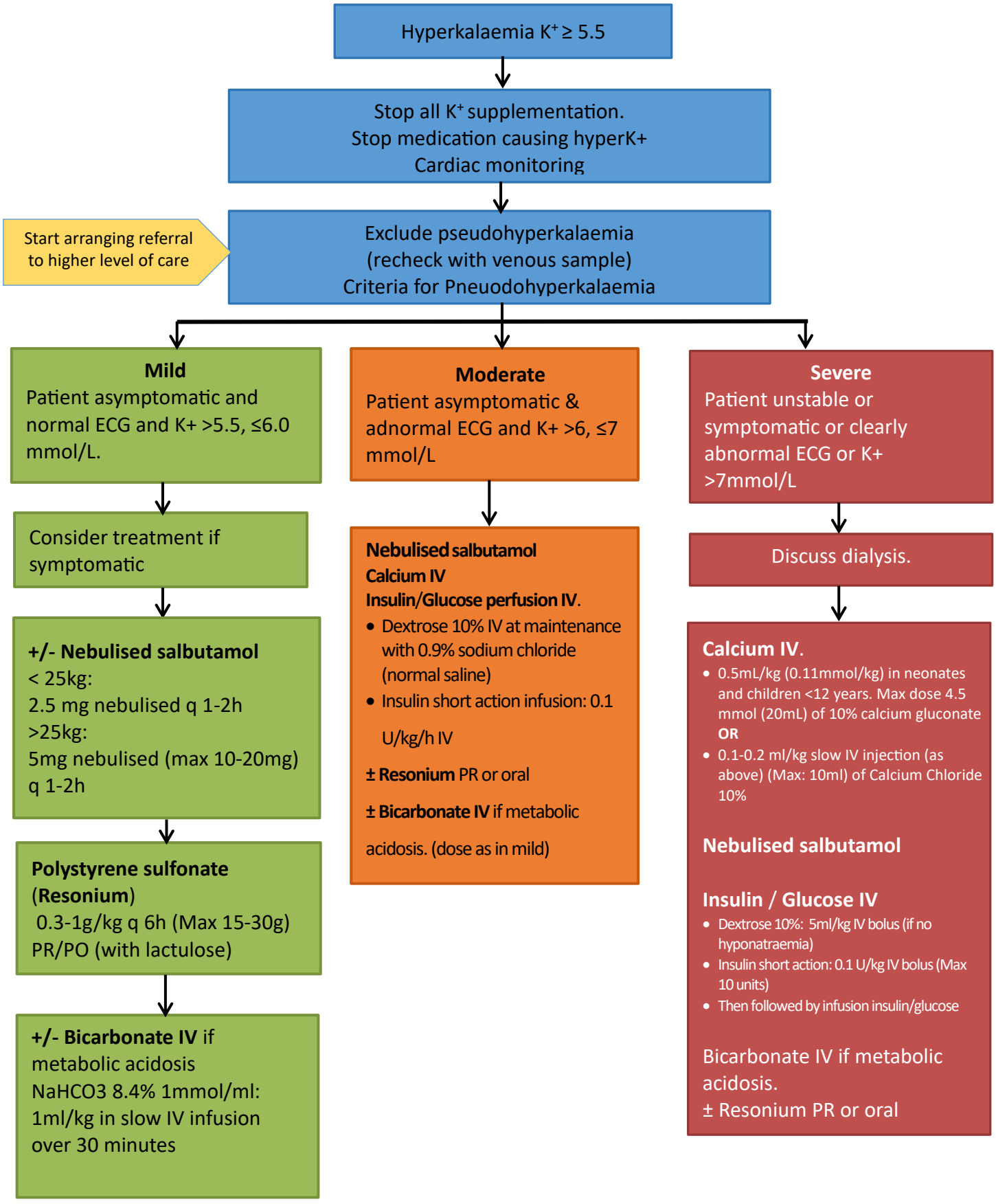


Fig 2.2. Approach to Hyperkalaemia adapted from the Royal Alexandra Children's Hospital

Table 2.7. Treatment of Hyperkalaemia

Drug	Dosage	Onset and expected Actions	Mechanism	Considerations
10% calcium gluconate (~225µ/mL) 10% calcium chloride	Neonates and children <12 years **10% calcium gluconate** 0.5mL/kg (0.11mmol/kg Max dose 4.5mmol (20mL) **OR** **10% calcium chloride** 0.1-0.2ml/kg slow IV injection (as above) (Max: 10ml)	**Onset of Action:** <3 minutes, should see normalization of ECG. If not; repeat dose (twice) **Duration**: peak 60 minutes, 2-3hours	Stabilize the heart	**Not** to be given simultaneously with bicarbonate **Not** to be given if digitalis toxicity
Soluble insulin/glucose	Neonates: 0.3-0.6 IU/kg/hr; Children 1 month & older: 0.05-0.2IU/kg/hr; 5-10ml/kg of D10% If severe hyperkalaemia: • Dextrose 10%: 5ml/kg IV bolus (if no hyponatremia) • Insulin short action: 0.1 U/kg IV bolus (Max 10 units) Then followed by infusion insulin/glucose (see below) If moderate hyperkalaemia: • Dextrose 10% IV at maintenance with 0.9% sodium chloride (normal saline) • Insulin short action infusion: 0.1U/kg/hr IV	**Onset of Action:** 15 minutes **Duration:** peak 60 minutes, 2-3hours	Shifts potasium into the cells	To be given at the same time. **Closely monitor glucose every 30-60 minutes**

Table 2.8. Treatment of Hyperkalaemia

Drug	Dosage	Onset and expected Actions	Mechanism	Considerations
8.4% NaHCO3 (IV)	**Severe hyperkalaemia** and **metabolic acidosis** Sodium bicarbonate 8.4% 1mmol/ml:1-3ml/kg IV over 5 minutes **Mild to moderate hyperkalaemia and metabolic acidosis:** Sodium bicarbonate 8.4% 1mmol/ml:1ml/kg in slow IV infusion over 30 minutes	**Onset of Action:** 30-60 minutes **Duration:** 2-3 hours	Shifts potasium into the cells	In metabolic acidosis, only
Salbutamol (nebulised of IV)	<25kg: 2.5mg nebulised q 1-2h >25kg: 5mg nebulised (max 10-20mg)q 1-2h	**Onset of Action:** 30 minutes **Duration:** 2-3 hours	Shifts potasium into the cells	Reduces intravascular K+ of 0.5-1.5mmol/L
Resonium	Mild effect, multiple doses necessary, may be used as long term agent Polystyrene sulfonate (Resonium) 0.3-1g/kg q 6h (Max 15-30g) Per Rectal (PR) or Oral (with lactulose)	**Onset of action:** 1hour PR, 4-6hours oral, should reduce intravascular K+ of 0.5-1 mmol/L **Duration:** variable	Potassium excretiion through gut	Mild effect, multiple doses necessary, may be used as long term agent. NOT to be used if ileus, recent abdominal surgery, perforation, hypernatraemia
Dialysis	Transfer to tertiary care center	Rapid- haemodialysis slower-peritoned	Removes potassium	Organise with local Paediatric renal or intensive care team.

NOTE

- Do not wait for repeat K+ results to start treatment
- If No ECG and cardiac monitor available, give calcium gluconate or calcium chloride and REFER
- Calcium resonium and dialysis are the only treatments that eliminate K+ from the body
- Never give calcium salts through the same line as sodium bicarbonate

Consider consultation with local paediatric team:

- Any child with moderate or severe hyperkalaemia
- Underlying medical cause – e.g., renal abnormalities

When to consider transfer to tertiary centre:

- Any child with severe hyperkalaemia
- Any child requiring dialysis
- Child requiring care beyond the comfort level of the hospital.

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2.5 HYPOKALAEMIA

This a serum potassium level < 3mmol/L.

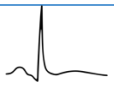
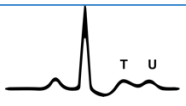
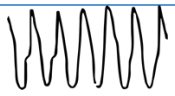
Table 2.9. Causes of Hypokalaemia

Decreased intake	Extra-renal losses	Renal losses	Drugs
Anorexia nervosa	Diarrhoea	Renal tubular acidosis	Loop and thiazide diuretics
	Vomiting or nasogastric drainage	Diabetic ketoacidosis	Amphotericin
	Sweating (cystic fibrosis)	Interstitial nephritis	Cisplatin
	Laxative abuse	Ureterosigmoidostomy	Aminoglycosides
		Alkalosis	Beta-adrenergic agents

Clinical features

- Muscle weakness and cramps
- Paralysis
- Heart block
- Paralytic ileus
- Polyuria
- Polydipsia

Table 2.1.1. ECG Changes

ECG CHANGES (HYPOKALAEMIA)	
Normal ECG	
Flattened T waves	
depressed ST segment	
Prominent U wave	
Supraventricular Tachycardia	
Ventricular tachycardia	

Management

Goals of treatment

- Prevent life threatening complications
- Replenish K+ stores
- Correct the underlying cause

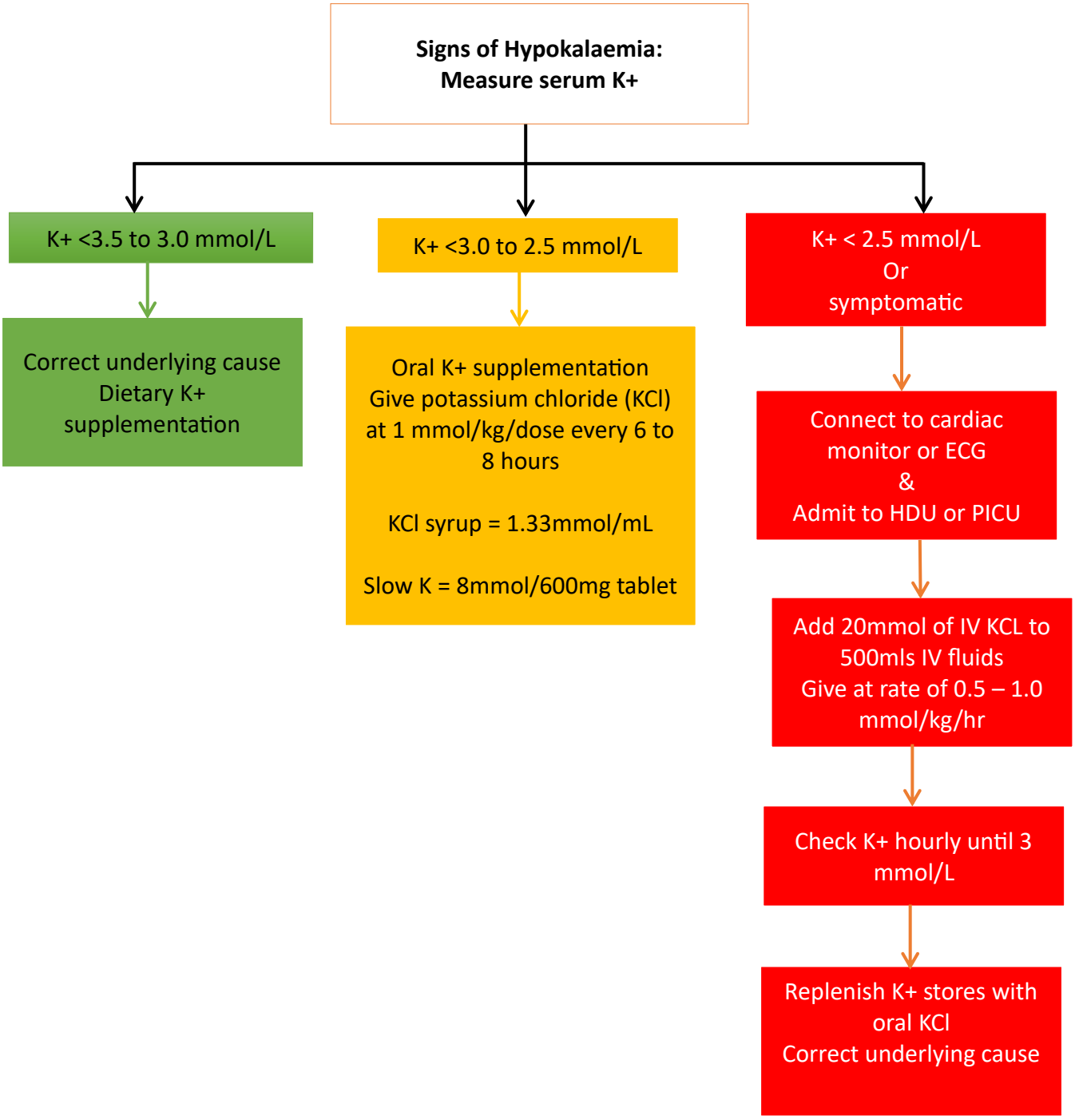


Fig 2.3. Management of hypokalaemia adapted the Royal Children's Hospital, Melbourne.

Note

- Slow release KCl tablet must be swallowed whole. Not broken or compounded. K+ level rises after 4 hours.
- Oral KCl syrup raises K+ level after 2 hours

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2.6 HYPOCALCAEMIA

Hypocalcaemia is a total serum calcium concentration < 8.8 mg/dL (< 2.20 mmol/L) in the presence of normal plasma protein concentrations or a serum ionized calcium concentration < 4.7 mg/dL (< 1.17 mmol/L).

Causes

- Hypoparathyroidism
- Pseudohypoparathyroidism
- Vitamin D deficiency and dependency
- Renal disease
- Magnesium depletion
- Acute pancreatitis
- Hypoproteinaemia (asymptomatic)
- Persistent hypocalcaemia and hypophosphataemia correction of moderate to severe hyperparathyroidism
- Septic shock
- Hyperphosphataemia
- Drugs including anticonvulsants (e.g., phenytoin, phenobarbital) and rifampicin,
- Transfusion of > 10 units of citrate-anticoagulated blood
- Use of radiocontrast agents containing the divalent ion-chelating agent
- Infusion of gadolinium

Clinical features

Tetany characteristically results from severe hypocalcaemia but can result from reduction in the ionized fraction of serum calcium without marked hypocalcaemia, as occurs in severe alkalosis. It is characterized by the following:

- Sensory symptoms consisting of paraesthesia of the lips, tongue, fingers, and feet
- Carpopedal spasm, which may be prolonged and painful
- Generalized muscle aching
- Spasm of facial musculature
- Seizures
- Twitching
- Cramping
- Laryngospasm, a rare initial manifestation

Diagnosis

- Estimation or measurement of ionized calcium
- Measurement of serum magnesium, PTH, phosphate, alkaline phosphatase (ALP), and vitamin D concentrations
- Urine calcium, magnesium, and phosphate.

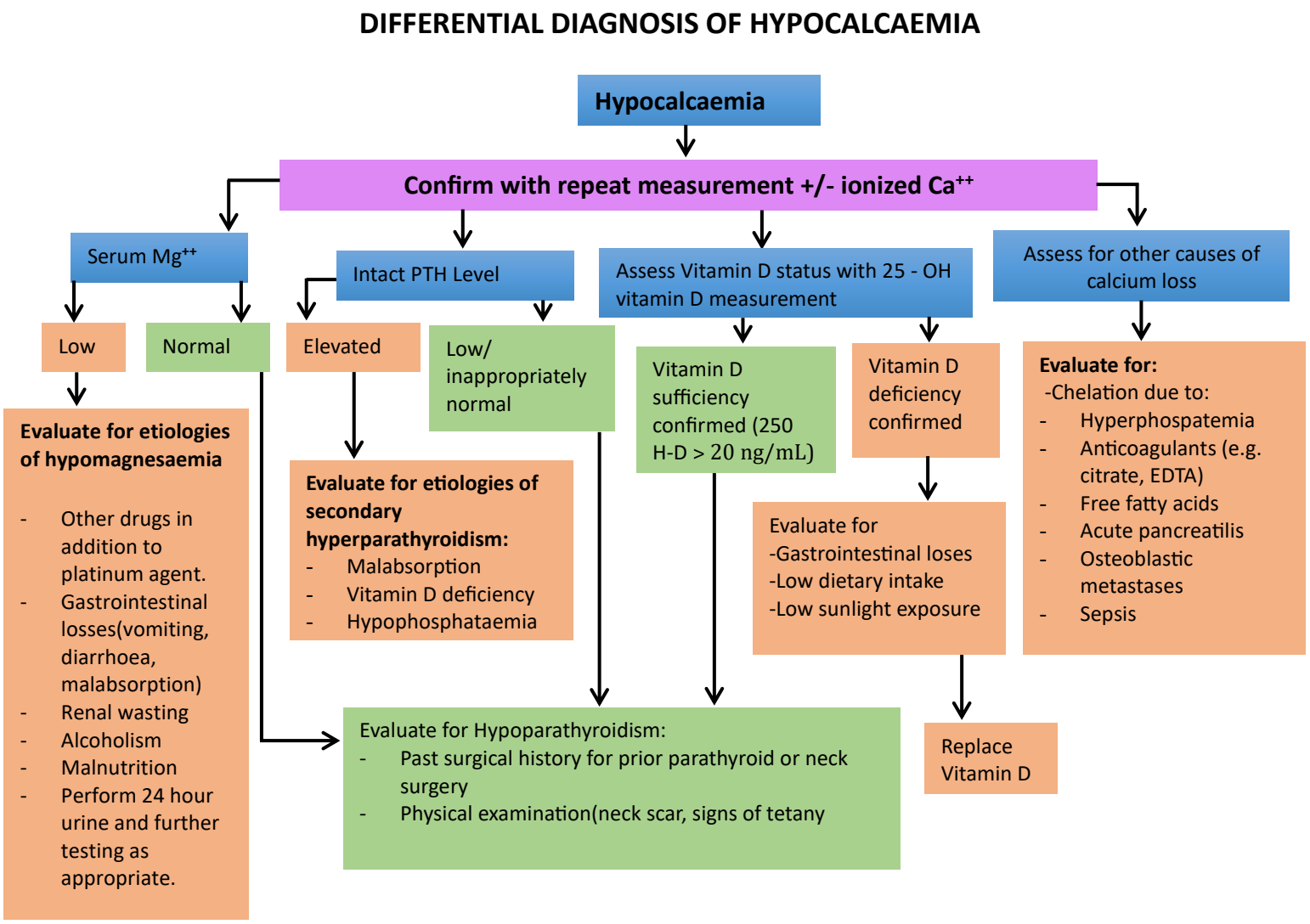


Fig 2.4. Diagnosis of hypocalcaemia.

Treatment

- Oral calcium therapy (calcium gluconate, calcium carbonate) is preferred in asymptomatic patients and as follow-up treatment after intravenous (IV) calcium therapy
- IV calcium gluconate for tetany and patients having seizures, critically ill, and pre-operatives.
- Oral calcium for postoperative hypoparathyroidism
- Oral calcium and vitamin D for chronic hypercalcaemia
- Ensure that the patient is well hydrated and passing urine
- Correction of hypomagnesaemia is essential as hypocalcaemia does not respond until the low magnesium level is corrected.

Tetany

- IV Calcium gluconate should be given as follows:
- Give calcium gluconate 10 mL of 10% solution IV over 10 min; calcium infusion drips should be started at 0.5 mg/kg/hr and increased to 2 mg/kg/hr as needed.
- Repeated boluses or a continuous infusion with 20 to 30 mL of 10% calcium gluconate in 1L of 5% D/W over the next 12 to 24 to may be needed.
- Give bolus of 0.5mL/kg (0.11mmol/kg) of 10% solution in neonates and children <12 years (Max dose 4.5 mmol i.e. 20mL of 10% solution). For IV infusion dilute to at least 45 µmol/mL with D5% or 0.9% sodium chloride.
- Oral supplements (calcium gluconate) may precipitate NEC in neonates.

1. Infusions of calcium are hazardous in patients receiving digoxin and should be given slowly and with continuous ECG monitoring after checking for and correcting hypokalaemia.
2. When tetany is associated with hypomagnesaemia, give a 10% magnesium sulphate solution (1 g/10 mL) IV, followed by oral magnesium salts (eg, magnesium gluconate 500 to 1000 mg po tid).

Transient hypoparathyroidism

Give supplemental oral calcium: 1 to 2 g of elemental calcium/day can give as calcium gluconate.

Chronic hypocalcaemia

- Give oral calcium and/or vitamin D supplements: 1 to 2 g of elemental calcium/day as calcium gluconate or calcium carbonate.
- In patients without renal failure, give vitamin D as a standard oral supplement (e.g., cholecalciferol 800 IU once/day) with adequate dietary or supplemental calcium and phosphate.
- In patients with renal failure, give calcitriol or another $1,25(OH)_2D$.
- In hypoparathyroidism, give calcitriol, 0.5 to 2 mcg po once/day.
- In pseudohypoparathyroidism give calcitriol 1 to 3 mcg/day.
- Monitor serum calcium concentration weekly at then at 1- to 3-mo intervals after calcium concentrations have stabilized as the maintenance dose of calcitriol or its analogy, dihydrocholecalciferol, usually decreases with time.

2.7 APPROACH TO INTERPRETATION OF ARTERIAL BLOOD GASES (ABGS)

ABG interpretation is especially important in critically ill patients.

6-STEP APPROACH BELOW, IS ESSENTIAL DURING THE INTERPRETATION:

***Step 1*:** Assess the internal consistency of the values using the Henderson-Hasselbalch equation:

$[H+] = \frac{24(PaCO_2)}{[HCO^{-3}]}$

If the pH and the $[H_+]$ are inconsistent, the ABG is probably not valid.

***Step 2*:** Is there alkalemia or acidaemia present?

pH < 7.35 acidaemia

pH > 7.45 alkalaemia

This is usually the primary disorder

Remember: an acidosis or alkalosis may be present even if the pH is in the normal range (7.35 – 7.45)

You will need to check the $PaCO_2$, HCO^{-3} and anion gap

Step 3: Is the disturbance respiratory or metabolic? What is the relationship between the direction of change in the pH and the direction of change in the $PaCO_2$?

Table 2.1.2. Approach to Interpretation of Arterial Blood Gases (ABGS)-Step 3

Acidosis	Respiratory	pH ↓	$PaCO_2$ ↑
Acidosis	Metabolic	pH ↓	$PaCO_2$↓
Alkalosis	Respiratory	pH ↑	$PaCO_2$↓
Alkalosis	Metabolic	pH ↑	$PaCO_2$↑

***Step 4*:** Is there appropriate compensation for the primary disturbance? Usually, compensation does not return the pH to normal (7.35 – 7.45).

Table 2.1.3. Approach to Interpretation of Arterial Blood Gases (ABGS)-Step 4

Disorder	Expected compensation	Correction factor
Metabolic acidosis	$PaCO_2$= (1.5 x $[HCO^{-3}]$) +8	± 2
Acute respiratory acidosis	Increase in $[HCO^{-3}]$ = Δ $PaCO_2$/10	± 3
Chronic respiratory acidosis (3-5 days)	Increase in $[HCO^{-3}]$ = 3.5(Δ$PaCO_2$/10)	
Metabolic alkalosis	Increase in PaCO2 = 40 + 0.6(ΔHCO^{-3})	
Acute respiratory alkalosis	Decrease in $[HCO^{-3}]$ = 2(Δ$PaCO_2$/10)	
Chronic respiratory alkalosis	Decrease in $[HCO^{-3}]$ = 5(Δ$PaCO_2$/10) to 7(Δ$PaCO_2$/10)	

Step 5: Calculate the anion gap (if a metabolic acidosis exists): AG= $[Na^+]$+(K^+]-[Cl-] + $[HCO_3^-]$

- A normal anion gap is approximately 12 mmol/L.
- If patient has hypoalbuminaemia, the AG is about 2.5 mmol/L lower for each 1 g/dL decrease in the plasma albumin concentration (for example, a patient with a plasma albumin of 2.0 g/dL would have an AG of approximately 7mmol /L).
- If the anion gap is elevated, consider calculating the osmolar gap in compatible clinical situations.
- Elevation in AG is not explained by an obvious case (DKA, lactic acidosis, renal failure)
- Toxic ingestion is suspected
  - Osmolality (OSM) gap = measured OSM – (2[Na+] - glucose/18 – BUN/2.8
  - The OSM gap should be < 10
  - If the concentration of glucose and BUN are in mmol/L, do not divide by 18 and 2.8 respectively.

Step 6: If an increased anion gap is present, assess the relationship between the increase in the anion gap and the decrease in $[HCO^{-3}]$.

Assess the ratio of the change in the anion gap (ΔAG) to the change in $[HCO^{-3}]$ (Δ$[HCO^{-3}]$): ΔAG/Δ[HCO^{-3}]

This ratio should be between 1.0 and 2.0 if an uncomplicated anion gap metabolic acidosis is present.

If this ratio falls outside of this range, then another metabolic disorder is present:

- If ΔAG/Δ[HCO^{-3}] < 1.0, then a concurrent non-anion gap metabolic acidosis is likely to be present.
- If ΔAG/Δ[HCO^{-3}] > 2.0, then a concurrent metabolic alkalosis is likely to be present.

Table 2.1.4. Hypocalcaemia (Characteristics of acid-base disturbances)

Disorder	pH	Primary problem	Compensation
Metabolic acidosis	↓	↓ in HCO^{-3}	↓ in $PaCO_2$
Metabolic alkalosis	↑	↑ in HCO^{-3}	↑ in $PaCO_2$
Respiratory acidosis	↓	↑ in $PaCO_2$	↑ in [HCO^{-3}]
Respiratory alkalosis	↑	↓ in $PaCO_2$	↓ in [HCO^{-3}]

Selected aetiologies of respiratory acidosis

- Airway obstruction
- CNS depression
- Sleep disordered breathing
- Neuromuscular impairment
- Ventilatory restriction
- Increased CO_2 production: shivering, rigors, seizures, malignant hyperthermia, hypermetabolism, increased intake of carbohydrates
- Incorrect mechanical ventilation settings

Selected aetiologies of respiratory alkalosis

- CNS stimulation: fever, pain, fear, anxiety, CVA, cerebral oedema, brain trauma, brain tumour, CNS infection
- Hypoxemia or hypoxia: lung disease, profound anaemia, low FiO_2
- Stimulation of chest receptors: pulmonary oedema, pleural effusion, pneumonia, pneumothorax, pulmonary embolus
- Drugs, hormones: salicylates, catecholamines, medroxyprogesterone, progestins
- Pregnancy, liver disease, sepsis, hyperthyroidism
- Incorrect mechanical ventilation settings

Selected causes of metabolic alkalosis

- Hypovolaemia with Cl- depletion
- GI loss of H+
  - Vomiting, gastric suction, villous adenoma, diarrhoea with chloride-rich fluid

- Renal loss H+
  - Loop and thiazide diuretics, post-hypercapnia (especially after institution of mechanical ventilation)
- Hypervolaemia, Cl- expansion
  - Renal loss of H+: oedematous states (heart failure, cirrhosis, nephrotic syndrome), hyperaldosteronism, hypercortisolism, excess ACTH, exogenous steroids, hyperreninaemia, severe hypokalaemia, renal artery stenosis, bicarbonate administration

Selected aetiologies of metabolic acidosis

- Elevated anion gap:
  - Methanol intoxication
  - Uraemia
  - Ketoacidosis
  - Paraldehyde toxicity
  - Isoniazid
  - Lactic acidosis
  - Ethanol or ethylene glycol intoxication
  - Salicylate intoxication
- Normal anion gap: will have increase in $[Cl^-]$
  - GI loss of HCO_3^- (diarrhoea, ileostomy, proximal colostomy)
  - Renal loss of HCO_3^- (proximal RTA, acetazolamide), renal tubular disease (ATN, chronic renal disease)

Table 2.1.5. Hypocalcaemia (Selected mixed and complex acid-base disturbances)

Disorder	Characteristics	Selected situations
Respiratory acidosis with metabolic acidosis	↓in pH ↓ in HCO_3^- ↑ in $PaCO_2$	Cardiac arrest Intoxications Multi-organ failure
Respiratory alkalosis with metabolic alkalosis	↑in pH ↑ in HCO_3^- ↓ in $PaCO_2$	Cirrhosis with diuretics Pregnancy with vomiting Over ventilation of COPD
Respiratory acidosis with metabolic alkalosis	pH in normal range ↑ in $PaCO_2$, ↑ in HCO_3^-	COPD with diuretics, vomiting, NG suction Severe hypokalaemia

Respiratory alkalosis with metabolic acidosis	pH in normal range ↓ in $PaCO_2$ ↓ in HCO_3^-	Sepsis Salicylate toxicity Renal failure with CHF or pneumonia Advanced liver disease
Metabolic acidosis with metabolic alkalosis	pH in normal range HCO_3^- normal	Uraemia or ketoacidosis with vomiting, NG suction, diuretics, etc.

Treatment

Treatment is dependent on correctly identifying the acid-base disorder and, whenever possible, repairing the underlying causal process to maintain serum pH greater than 7.20.

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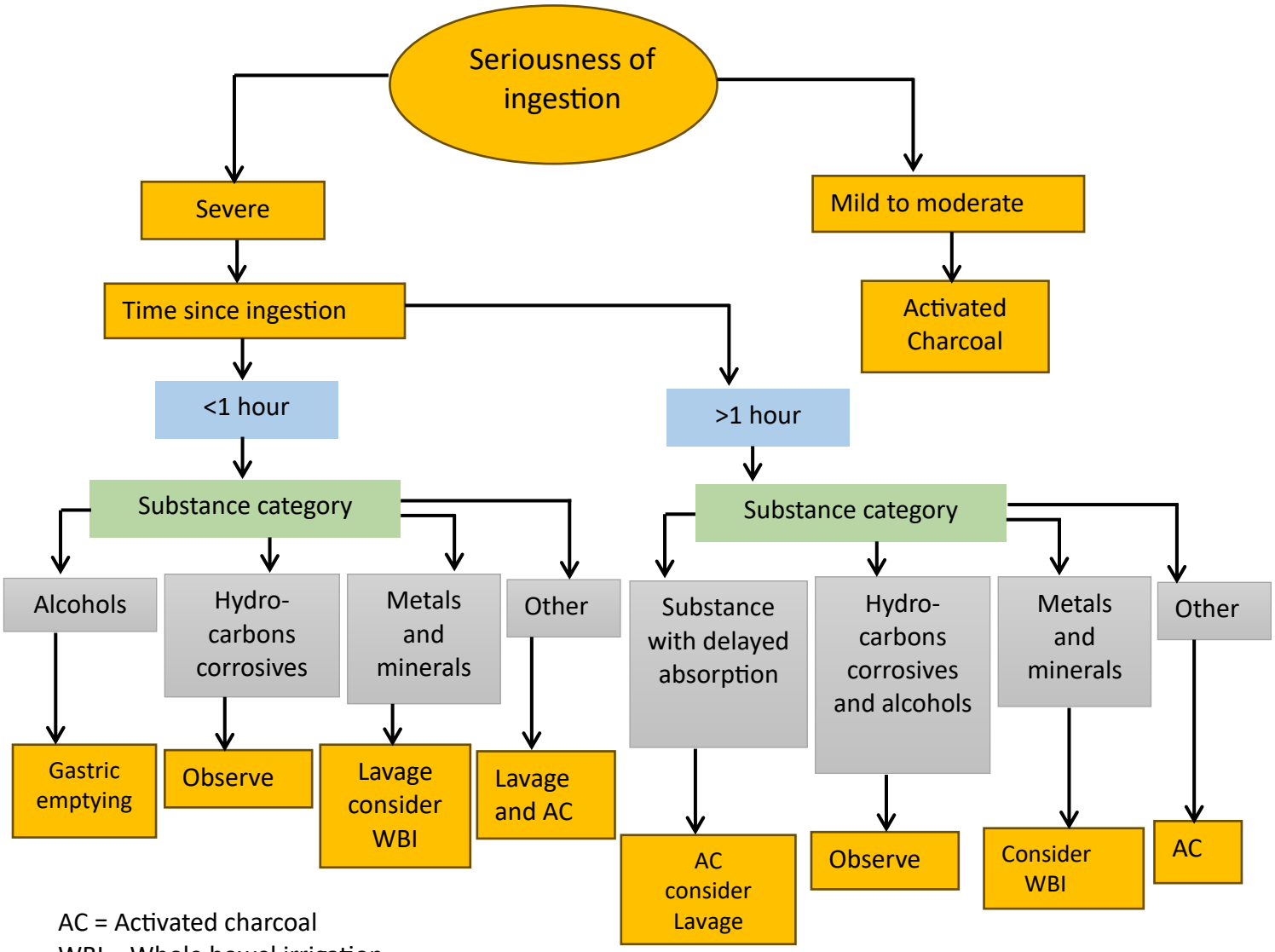
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Most poisoning that occurs in children is accidental. The other causes of poisoning can be due to intentional overdose, drug abuse and iatrogenic poisoning. The most common drugs involved in poisonings in children are organophosphates and paracetamol. The specific types of poisonings and their management are discussed below.

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APPROACH TO PAEDIATRIC POISONING



AC = Activated charcoal
WBI = Whole bowel irrigation

NB:
Lavage may not be beneficial two hours after ingestion.

Fig 3. Approach to paediatric poisoning

Contraindications of activated charcoal:

- Acid and alkalis
- Corrosives
- Cyanide
- Ethanol/methanol/glycols
- Eucalyptus and essential oils
- Fluoride
- Hydrocarbons
- Metals – including lithium, iron, compounds, potassium, lead
- Mineral acids -boric acid

3.1 ASPIRIN POISONING

Aspirin is used as an analgaesic, an antipyretic and an anti-inflammatory agent.

Clinical signs

- Tinnitus, rapid breathing, vomiting, dehydration, fever and double vision are early signs.
- Later signs include drowsiness, confusion, bizarre behaviour, unsteady walking and coma.
- Respiratory alkalosis and metabolic acidosis
- Rhabdomyolysis (dark urine), acute renal failure and respiratory failure may occur.

Management

- Plasma salicylate concentration should be measured in all patients with suspected toxicity where available.
- The acute toxic dose of salicylates is generally considered to be > 150 mg/kg
- Gastrointestinal decontamination: Activated charcoal, 1 g/kg (max: 50 g) within four hours of potential toxic ingestion
- Give IV sodium bicarbonate 8.4 % 1 mmol/kg over 4 hours to correct acidosis and raise urine pH to above 7.5
- Give maintenance IV fluids unless the child shows signs of dehydration
- Give supplemental potassium: 20-40 mEq/L to the maintenance fluids used
- Monitor blood glucose every 6 hours
- Where plasma salicylate concentration is available then treatment can be given according to Table 7 below:

Table 3. Aspirin Poisoning

Poisoning	Mild	Moderate	Severe
Plasma salicylate	< 350 mg/l	> 350 mg/l	> 700 mg/l
Fluids	Encourage oral fluids	Intravenous fluids [two thirds normal requirement]	Intravenous fluids [two thirds normal requirement]
Sodium bicarbonate 1 mEq/kg IV bolus then a continuous infusion of 100 -150 mEq in 1 L of 5% Dextrose to run at 2 times maintenance	No	Yes	Yes
Consider repeated doses of activated charcoal	No	Yes	Yes
Urgent referral for haemodialysis	No	No	Yes

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3.2 ORGANOPHOSPHATE POISONING

Organophosphates are a diverse group of chemicals used in in both domestic, agricultural and industrial settings. They form an initially reversible bond with the enzyme cholinesterase that later becomes irreversible.

SOURCE

Insecticides, rat poison, etc.

HISTORY

- Most symptoms appear within 12-24 hours of exposure
- Exposure may be due to ingestion, dermal exposure or inhalation
- Children often ingest home pesticides they find in unmarked or poorly stored containers
- Children may also be exposed when playing in areas recently treated with organophosphates

DIAGNOSIS

- A history of possible exposure combined with physical signs and symptoms consistent with exposure often leads to diagnosis

PHYSICAL EXAMINATION

Muscarinic findings

- Diaphoresis and diarrhoea, urination, miosis, bradycardia, bronchorrhoea, bronchospasm, emesis, lacrimation and salivation (**dumbels**)

Young children are more likely to present with altered consciousness than with the classic DUMBELS seen in adults

Nicotinic findings

- Muscle fasciculations, fatigue, paralysis, respiratory muscle weakness, diminished respiratory effort, tachycardia, hypertension
- CNS: Anxiety, restlessness, confusion, headache, slurred speech, ataxia, seizures, coma, central respiratory paralysis, altered consciousness or hypotonia

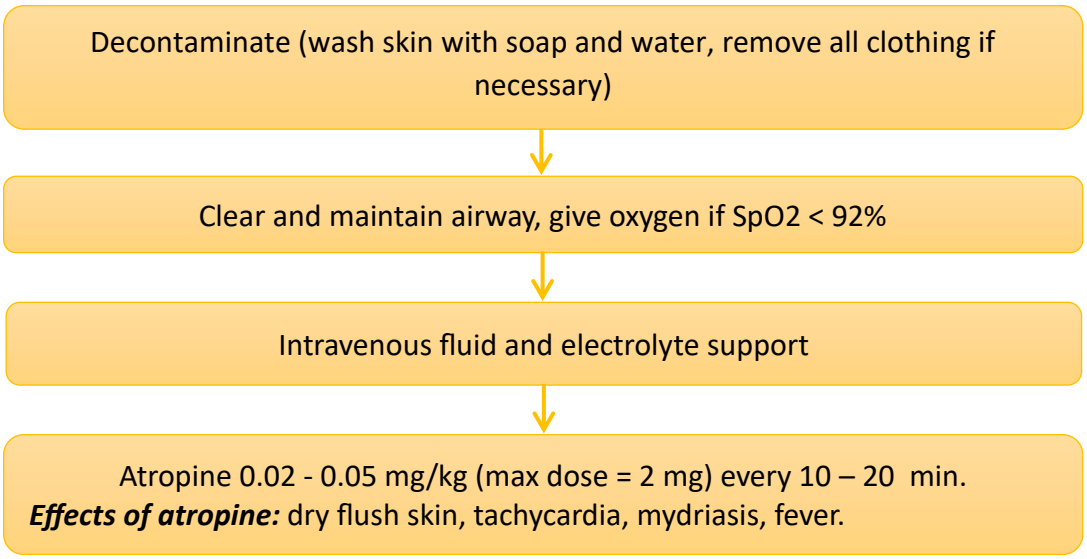


Fig 3.1. Management of organophosphate poisoning

MANAGEMENT

Atropine

- Antogonises the central and muscarinic effects by blocking these receptors
- It does not bind to nicotinic receptors so the signs are not affected
- It should be used for at least 24hours to reverse cholinergic signs while organophophates are metabolised
- Should be given till the signs of atropinisation appear
  - Warm, dry, flushed skin, dilated pupils and increased heart rate

Pralidoxime (2-PAM)

- Is the antidote for organosphosphate poison
- Is a cholinesterase reactivator
- It primarily affects the nicotinic receptors and does not reverse the CNS effects
- Should be administered as soon as possible because it effectiveness decreases with prolonged exposure due to aging of the organophosphate-cholinesterase bond
- Administer as in IV infusion after a loading dose until signs of weakness improve
- Pralidoxime [in combination with atropine] at 20-50 mg/kg/dose. Repeat in 1-2 hours if muscle weakness has not been relieved, then 10 to 12 hours intervals if cholinergic signs recur.

For management of seizures, (refer to seizure management).

PROGNOSIS

- Prognosis is excellent in patients treated early. Most patients recover in 7-10 days
- Patients untreated for more than 24hours may have prolonged and severe course with lasting neurologic complications. Morbidity and mortality results from anoxic injury due to respiratory failure

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3.3 HYDROCARBONS POISONING

Hydrocarbons are a heterogeneous group of organic substances that are primarily composed of carbon and hydrogen molecules. Commonly ingested hydrocarbons include petrol and kerosene. Volatile substances containing hydrocarbons are commonly abused for their euphoric effects e.g. glue, propellants

Examples

Petrol, Kerosene, Lighter Fluid, Paraffin Oil, Diesel Fuel, Lubricating Oil, Furniture Polishes, Essential oils, Mineral Turpentine, dry cleaning solutions, paint, spot remover, solvents

Clinical signs

Transient mild CNS depression, aspiration is characterized by coughing. Respiratory symptoms may remain mild or may rapidly progress to respiratory failure. Fever occurs and may persist for as long as 10 days after aspiration.

Management

- Emesis is contraindicated because of the risk of aspiration.
- Gastric lavage is also contraindicated.
- If hydrocarbon-induced pneumonitis develops, respiratory treatment is supportive.
- Corticosteroids should be avoided, because they are not effective and may be harmful
- Do CXR after 24 hrs if signs of respiratory distress

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3.4 IRON OVERDOSE

Iron is a common cause of poisoning in children. This may be due to the wide availability of iron product and their candylike appearance and taste eg prenatal vitamins containing iron and ferrous sulphate tablets for postnatal mothers.

In the event of poisoning, it is important to estimate the amount of elemental iron ingested as the potential severity of depends on the amount ingested. The calculation of the amount of elemental iron involves estimating the number of tablets ingested and the percentage of elemental iron that the tablets contain.

Clinical features:

- Nausea, vomiting, abdominal pain and diarrhoea. The vomit and stools are often grey or black.
- In severe poisoning, there may be gastrointestinal bleeding, hypotension, drowsiness, convulsions and metabolic acidosis. Gastrointestinal features usually appear within the first 6 hr, and a child who has remained asymptomatic for this time probably does not require an antidote.

Table 3.1. Stages of Iron Toxicity

Stages of Iron Toxicity		
Stage	**Clinical Effect**	**Time Frame**
Stage I	• GI Irritation	**30 min – 6 hrs**
Stage II Latent	• Recovery from GI Symptoms	**6 hrs – 24 hrs**
Stage III Shock and metabolic acidosis	• Metabolic acidosis (anion gap) • Dehydration • Lactic acidosis	**6 hrs – 72 hrs**
Stage IV Hepatotoxicity/Hepatic necrosis	• Fulminant hepatic failure	**12 hrs – 96 hrs**
Stage V Bowel obstruction	• GI mucosa healing leads to scarring	**2 weeks to 8 weeks**

Management

- Consider a gastric lavage if potentially toxic amounts of iron were taken.
- **Do not** give activated charcoal
- Decide whether to give the antidote. As this can have side-effects, it should be given only if there is clinical evidence of poisoning (see above).
- Start fluid boluses of 20ml/kg of normal saline or ringer's lactate as children with iron overload are hypotensive.
- Do a chest x-ray in case of iron tablets as they may be seen
- If clinical signs are present give deferoxamine, preferably by slow IV infusion: initially 15 mg/kg/hour, reduced after 4–6 h so that the total dose does not exceed 80 mg/kg in
- 24 h. Maximum dose, 6 g/day.
- If deferoxamine is given IM: 50 mg/kg every 6 h. Maximum dose, 6 g/day.
- Stop infusion if patient is clinically stable. This is usually within 24 hours

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3.5 PARAQUAT POISONING

Paraquat (1,1'-dimethyl-4,4'-dipyridylium) is a broad-spectrum liquid herbicide associated with both accidental and nonaccidental ingestion, leading to severe and often fatal toxicity. It is rapidly but incompletely absorbed and then largely eliminated unchanged in urine within 12–24 h.

Clinical Features

Clinical features are largely due to intracellular effects. Paraquat generates reactive oxygen species which cause cellular damage via lipid peroxidation, mitochondrial damage and apoptosis in many organs:

- Vomiting,
- Fever,
- Tachycardia
- Tachypnoea,
- Occasional diarrhoea.
- Drowsiness
- Paraquat is actively taken up against a concentration gradient into lung tissue leading to pneumonitis and lung fibrosis.
- It also causes pancreatic, renal and liver injury.

Investigations

- Electrolytes, renal and liver function tests, full blood count, should be done at least daily.
- A chest radiograph (if pneumomediastinum, pneumothorax or lung fibrosis is suspected).
- A CT scan of the chest (useful in detecting early lung fibrosis or assessing long-term damage in survivors).
- Amylase and lipase (acute pancreatitis suspected if patients develop abdominal pain and a raised blood sugar).
- Plasma paraquat concentrations
- Urine and plasma dithionite tests

Management

- Gastric lavage is helpful.
- Activated charcoal (1gm/kg of activated charcoal by gastric tube every 2 hours 3 to 4 doses) routinely given to minimize further absorption.
- **Supplemental oxygen should be withheld** because oxygen may contribute to the pulmonary damage, mediated through lipid peroxidation.

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3.6 PARACETAMOL POISONING

Paracetamol is one of the most commonly used oral analgaesics and antipyretics. It has an excellent safety profile when used in therapeutic doses but hepatotoxicity can occur after overdose.

The minimum toxic dose of a paracetamol for a single ingestion posing significant risk of severe hepatotoxicity is 150mg/kg in children.

The clinical course of toxicity is generally divided into 4 phases with findings dependent on time after ingestion and the degree of hepatotoxicity.

Table 3.2. Paracetamol Poisoning (Clinical Signs)

Phases	Period after ingestion	Clinical presentation
1	Up to 24 hrs	The child may appear well. They may report nausea, anorexia, vomiting and malaise. Physical examination may reveal pallor, diaphoresis, malaise and fatigue.
2	24 – 72 hrs	Anorexia, nausea, vomiting and right upper quadrant abdominal pain may develop. Physical findings may show right upper quadrant tenderness, tachycardia, hypotension and oliguria
3 (Hepatic)	72 – 96 hrs	The child may have continued nausea, vomiting, abdominal pain and a tender hepatic edge. Hepatic necrosis and dysfunction may manifest as jaundice, hypoglycaemia, coagulopathy and encephalopathy. In severe cases, renal failure and multi-organ failure develop. Death most commonly occurs at this stage from multiorgan failure.
4 (Recovery)	4 – 21 days	Those who survive critical illness have full recovery with complete resolution of symptoms and organ failure

The time of ingestion, the quantity and the formulation of paracetamol ingested should be determined by the clinician.

Investigations

The serum paracetamol concentration is the basis of diagnosis and treatment.

- Serum paracetamol concentration
  - Rumack-Matthew monogram interprets paracetamol concentration in relation to time (in hours) after ingestion and is predictive of possible hepatotoxicity after single acute ingestion
- Liver function tests
  - Alanine aminotransferase (ALT); aspartate aminotransferase (AST); total and fractionated bilirubin; alkaline phosphatase
- Prothrombin time (PT) with international normalised ratio (INR)
- Glucose
- Kidney function tests
  - Electrolytes, urea, creatinine
- Lipase and amylase (in patients with abdominal pain)
- Human chorionic gonadotropin (HCG) in females of childbearing age
- Salicylate level (in patients with concern of co-ingestants)
- Arterial blood gas and ammonia (in clinically compromised patients)
- Urinalysis (to check for haematuria and proteinuria)
- ECG (in suspected salicylate co-ingestant)
- Assessment and management of Airway, Breathing and Circulation (ABC)
- Decontamination and antidote therapy

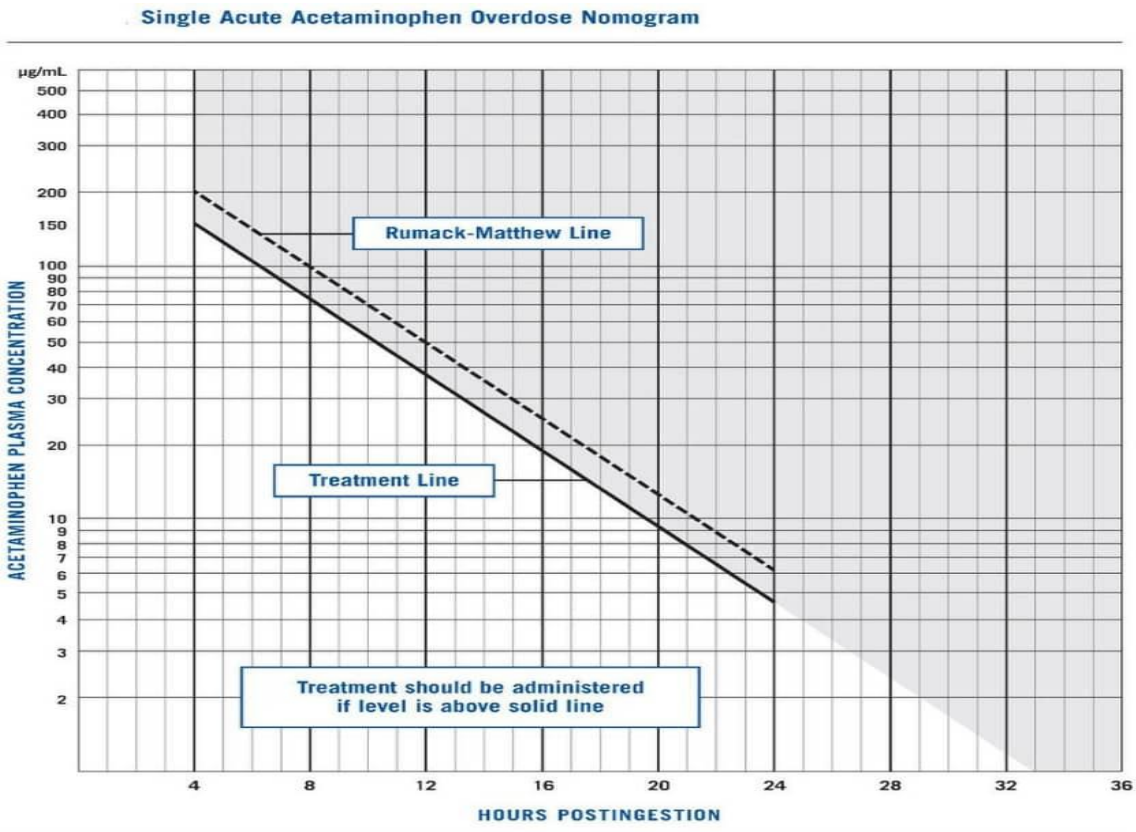
Table 3.3. Paracetamol Poisoning (Management)

Poisoning parameter	Treatment	
Single dose <150mg/kg or cumulative dose <90mg/kg/day	No action needed	
Single dose ≥ 150 mg/kg	Gastric decontamination	If stable mental and clinical state, patent airway and within 4 hr of ingestion • Activated charcoal 1g/kg (max 50g)
Ingestion of ≥ 150 mg/kg or Toxic 4 hours paracetamol level Possible or probable risk of hepatotoxicity on rumack-matthew nomogram	Antidote (to prevent liver damage)	IV n-acetylcysteine (NAC) within 8 hours (or later) of ingestion: IV acetylcysteine: ≤ 20 kg: • 150 mg/kg in 3ml/kg of 5% glucose over 1 hour followed by • 50 mg/kg in 7ml/kg of 5% glucose over 4 hours then • 100 mg/kg in 14 ml/kg of 5 % glucose over 16 hours. >20kg and < 40 kg: • 150 mg/kg in 100 ml of 5% glucose over 1 hour followed by • 50 mg/kg in 250ml of 5% glucose over 4 hours, then • 100mg/kg in 500ml of 5% glucose over 16 hours ≥40kg (as in adults) • 150mg/kg in 200ml 5% glucose over 1 hour followed by • 50mg/kg in 500ml 5% glucose over 4 hours, then • 100mg/kg in 1000ml 5% glucose over 16 hours

- If ingestion occurred with an intent to self-harm, a thorough psychosocial, psychological and/or psychiatric evaluation are indicated

Table 3.4. Rumack-Matthew nomogram

- Used to interpret plasma paracetamol values to assess hepatotoxicity risk after a single, acute ingestion
- Nomogram tracking begins 4 hours after ingestion and ends 24 hours after ingestion



Nomogram: acetaminophen plasma concentration vs time after acetaminophen ingestion (adapted with permission from Rumack and Matthew. *Pediatrics*. 1975;55:871-876). The nomogram has been developed to estimate the probability of whether a plasma acetaminophen concentration in relation to the interval postingestion will result in hepatotoxicity and, therefore, whether acetylcysteine therapy should be administered.

CAUTIONS FOR USE OF THIS CHART:

1. Time coordinates refer to time postingestion.
2. Graph relates only to plasma concentrations following a single, acute overdose ingestion.
3. The Treatment Line is plotted 25% below the Rumack-Matthew Line to allow for potential errors in plasma acetaminophen assays and estimated time from ingestion of an overdose (Rumack et al. *Arch Intern Med*. 1981;141(suppl):380-385).

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3.7 IBUPROFEN POISONING

Ibuprofen is a nonsteroidal anti-inflammatory drug (NSAID) and is often involved in intentional and unintentional overdoses owing to its widespread availability and common use as an analgaesic, antipyretic and anti-inflammatory agent.

Ibuprofen inhibits prostaglandin synthesis by reversibly inhibiting the activity of cyclooxygenase (cox). Side effects with therapeutic use include: GI irritation, reduced renal blood flow and platelet dysfunction.

ETIOLOGY OF TOXICITY

An overdose may be self-injurious, suicidal or accidental.

Symptoms of toxicity are unlikely at doses <100mg/kg in children. Ingestion of >400mg/kg in a child is associated with serious or life-threatening toxicity.

Symptoms usually occur within 4 to 6 hours of ingestion

Table 3.5. Ibuprofen Poisoning (Clinical Manifestations)

For ingestion < 400 mg/kg	For ingestion >400 mg/kg
Nausea and vomiting	Apnoea
Abdominal pain	Bradycardia
Headache and drowsiness	Dysrhythmias and prolonged QT
Blurred vision	Hypothermia
Ataxia and dizziness	Metabolic acidosis
	Polyuria
	Renal failure
	Coma
	Seizures

Investigations

- Blood glucose
- Electrolytes, blood urea and nitrogen and creatinine
- Arterial blood gases
- Full blood count
- Urinalysis
- Electrocardiogram
- Serum paracetamol level in case of concomitant paracetamol poisoning
- Management is primarily supportive
- There is no antidote

Table 3.6. Ibuprofen Poisoning (Management)

Patient/clinical description	Treatment
Most patients have no symptoms or mild GIT symptoms	Supportive treatment Asymptomatic patients with normal vitals and no paracetamol poisoning: - Observe for 4-6 hours
Ingestion of <100mg/kg	Observe from home
Children with ingestion of >400mg/kg Ingestion within 2 hours	Admit for further evaluation Gastrointestinal decontamination: 1 dose of activated charcoal 1-2g/kg/dose (max 50g)
Clinical life-threatening manifestations e.g. respiratory depression, hypotension, seizures, metabolic acidosis	ABCs and appropriate supportive care Monitor life-threatening arrhythmias with ECG which can be due to electrolyte imbalance e.g. hypokalaemia Haemodialysis cannot be used to remove the heavily protein-bound ibuprofen, but can be used to treat hyperkalaemia or refractory metabolic acidosis

- Oral anti-acids like Magnesium Hydroxide or H^2 Receptor antagonists like Cimetidine for epigastric abdominal pain
- Anti-emetics like Ondansetron where vomiting persists
- Sodium bicarbonate for metabolic acidosis
- Benzodiazepines for seizures. Refer to management of seizures for dosing
- Consult mental health counsellor or psychiatrist to assess self-harm risk or potential need for psychiatric admission in intentional or self-injurious exposure

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4.1 CONGESTIVE CARDIAC FAILURE (CCF)

Congestive cardiac failure is a clinical syndrome in which the heart:

- Is unable to pump enough blood to the body to meet its needs,
- Is unable to dispose of venous return adequately,
- **OR** a combination of the two.

Diagnosis of CCF relies mainly on clinical findings and no single test is specific.

AETIOLOGY

CCF may result from congenital or acquired heart diseases with volume and/ or pressure overload, or from myocardial insufficiency.

Table 4. Congestive Cardiac Failure (Aetiology)

Cause	Example/s
1. Cardiac	
A. Congenital	• Ventricular Septal Defect (VSD) • Atrial Septal Defect (ASD) • Patent Ductus Arteriosus (PDA) • Arrhythmias
B. Acquired	• Endocardial/Valvular disease e.g. Rheumatic Heart Disease (RHD) • Myocardial diseases e.g. Viral myocarditis, dilated cardiomyopathies, hypertrophic cardiomyopathy • Pericardial diseases e.g. TB pericarditis
2. Extracardiac	
• Anaemia • Pulmonary diseases e.g. Pulmonary hypertension • Severe pneumonia especially in neonate • Systemic hypertension • Metabolic disorders e.g. Electrolyte imbalances, hypoglycaemia • Endocrine e.g. Thyroid disease • Drugs e.g. antineoplastic	

DIAGNOSIS

The diagnosis is based on:

History

Poor feeding, FTT, Difficulties in breathing, poor weight gain, easy fatiguability in older children.

Examination

Tachypnoea, tachycardia, hepatomegaly, +/- Cardiac murmurs, oedema in older children.

Investigations

FBC, U&Es, Serum Creatinine, Echocardiography, Chest x-ray and ECG.

Aims of treatment

- To relieve symptoms
- Elimination of the underlying/precipitating cause
- Improve survival

General measures

- Propped up position
- Oxygen
- Salt and fluid restriction
- Daily weight in hospitalised patients

Drug therapy

The cornerstones of management are:

- Preload reduction: Diuretics e.g. Furosemide, Spironolactone
- Afterload reduction: ACEIs (Captopril, Enalapril),
- Inotropes: Digoxin (stable patient), Dobutamine/Dopamine (Severe CCF)
- β-blockers e.g. Carvedilol (used in chronic heart failure state typically in dilated cardiomyopathy

Acute Heart Failure

SYMPTOMS
Easy fatiguability, not able to lie flat when sleeping, difficulty breathing, swelling of feet and abdomen

ASSESS
A+B Resp Rate, Work of breathing, SpO_2, +/- Crepitations
C JVP, Pulse rate and volume, Bp, CRT, warm vs cold peripheries
D RBS, GCS
E Temp, oedema
F Input, output, urinalysis
G Hepatomegaly

What does the patient have?

Has normal findings

Warm & Dry
Do nothing

PATIENT HAS
Raised JVP, increased work of breathing, crepitations, oedema, ascites, hepatomegally, warm peripheries and normal volume pulse, normal BP (NOT IN SHOCK)

DIURETICS
Furosemide 2-3mg/kg IV STAT
THEN 3mg/kg/day in 3 doses

Spironolactone 2-3mg/kg in 2-3 divided doses

consider **Moduretic

Warm & Wet
Optimize antifailure medications

***INOTROPES**
Dobutamine 2-20mcg/kg/min
OR
Dopamine 2-20mcg/kg/min

IN ICU
Milrinone 50mcg/kg loading dose
THEN 0.25-0.75mcg/kg/min

Cold and Dry
Inotropic Support

PATIENT HAS
History of fluid loss such as diarrhoea, excessive diuretic use, etc

Normal JVP COOL peripheries and low volume pulse (IN HYPOVOLAEMIC SHOCK)

Cold and Wet
Inotropes and Diuretics

***INOTROPES**
Dobutamine 2-20mcg/kg/min
OR **Dopamine** 2-20mcg/kg/min

IN ICU
Milrinone 50mcg/kg loading dose
THEN 0.25-0.75mcg/kg/min

PATIENT HAS
Raised JVP, increased work of breathing, crepitations, oedema, ascites, hepatomegally, COOL peripheries and LOW volume pulse, LOW BP (IN CARDIOGENIC SHOCK)

DIURETICS
Furosemide 2-3mg/kg IV STAT
THEN 3mg/kg/day in 3 doses

Spironolactone 2-3mg/kg in 2-3 divided doses
**consider moduretic

ENSURE
To Correct ABC issues
Nurse patients in propped position
Patients in respiratory distress and/or in SHOCK are placed on High flow Oxygen

MONITOR
SpO2
RBS
Input/Output (aim for negative balance)
Fluid restrict patients with wet forms of Heart failure
Daily weights and urinalysis

***DILUTION OF INOTROPES**
Milrinone 0.75mg/kg dilute to 50ml using 5% Dextrose. 1ml=0.25mcg/kg/mins

Dobutamine and Dopamine
6mg/kg dilute to with 5% Dextrose
Water to 100mls. 1ml/hr=1mcg/kg/min

**Moduretic (Co-amiloride) 5/50
1/4 tab in discussion with Senior

INVESTIGATIONS
ECG
CXR
ECHO
Urgent U&Es
FBC/DC
Blood Gas

Improvement is shown by:
- Reduced work of breathing, improved saturations
- Reduced JVP and hepatomegaly
- Urine output > 0.5ml/kg/hr with negative balance and reducing weight

Table 4.1. Congestive Cardiac Failure (Dosages)

Drug	Route	Dosage
Furosemide	IV Oral	1-3mg/kg/dose, 2-3 times a day 1-2mg/kg/dose, 1-3 times a day
Spironolactone	Oral	1-2mg/kg/dose, 1-2 times a day
Hydrochlorothiazide	Oral	2-4 mg/kg/day in 2 – 3 divided
Digoxin	Oral	0.02 - 0.05 mg/kg OD PO
Captopril	Oral	0.1-0.5mg/kg Divided 8hourly, max. dose 0.6mg/Kg
Enalapril	Oral	0.1mg/Kg divided OD or BD, Max. dose 0.5mg/kg/day
Carvedilol	Oral	0.08 mg/kg 12 hourly, if tolerated increase by 0.08 mg/kg every 1-2 weeks to a maximum 0.50mg/kg 12 hourly.

NOTE: If in cardiogenic shock and requiring Dopamine, Dobutamine or Milrinone, refer to the shock protocol.

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4.2 ACUTE RHEUMATIC FEVER

Acute Rheumatic Fever is an autoimmune inflammatory condition that develops as a sequela of streptococcal infection.

DIAGNOSIS

Diagnostic criteria for rheumatic fever is done according to the modified 2015 Jones criteria. Has defined high risk population recognizing variability in clinical presentation and had included Echocardiography as a tool to diagnose cardiac involvement (for subclinical carditis). Zambia falls into a high-risk category.

Table 4.2. Acute Rheumatic Fever (Modified 2015 Jones' criteria)

Major	Minor
Carditis (clinical or subclinical)	Monoarthralgia
Arthritis – monoarthritis or polyarthritis	Fever (≥ 38.0ºC)
Polyarthralgia	ESR ≥ 30 mm/hour and/or CRP ≥ 3.0 mg/dl
Chorea	Prolonged PR interval (after considering the differences related to age; if there is no carditis as a major criterion)
Erythema marginatum	
Subcutaneous nodules	

ESR – erythrocyte sedimentation rate; CRP – C-reactive protein (Adapted from ref 13). All patients with ARF should have an Echocardiography done even in the absence of clinical suspicion of valvular disease.

Revised Jones Criteria

WHO guidelines set the international standard for diagnosis of ARF:
Two major manifestations ***plus*** evidence of preceding streptococcal infection **OR one major** and **two minor** manifestations **plus** evidence of preceding streptococcal infection.

MANAGEMENT

Treat the illness

Benzathine penicillin injection stat or oral penicillin for ten days

Relieve symptoms

- Bed rest
- Relief of arthritis, pain and fever
- Treat chorea (if severe)
- Anti- heart failure medication (see table 2 above)

Table 4.3. Management of Arthritis and severe carditis

	Arthritis	Carditis
Prednisolone	Nil	2 – 4 weeks
Aspirin	1 – 2 weeks	Until symptoms subside
*Ibuprofen	Until symptoms subside	Until symptoms subside

Dosages

Prednisolone 2 mg/kg/day in four divided doses

Aspirin 50 to 60 mg/kg/day in four to six divided doses

*Ibuprofen can be given at 30 mg/kg/day in 3 divided doses, where Aspirin not tolerated.

The dose of prednisolone should be 2 mg/kg/day (max 60 mg); then taper by 20–25% per week. Aspirin can be reduced to 25 to 30 mg/kg/day when symptoms improve. The dose of prednisolone should be tapered, and aspirin started during the final week.

Management of Sydenham's chorea

1. Reduce physical and emotional stress and use protective measures as indicated

Benzathine penicillin IM stat (eradicate group a streptococcus), then every 28 days for secondary prophylaxis.

Anti-inflammatory agents not indicated

For severe chorea, any of the following drugs may be used:

- Carbamazepine 7–20 mg/kg/day (7–10 mg/kg day usually sufficient) given TDS PO until chorea is controlled for at least 2 weeks, then trial off medication
- Valproic acid 15–20 mg/kg/day (can increase to 30 mg/kg/day) given TDS PO until chorea is controlled for at least 2 weeks, then trial off medication.
- Phenobarbitone, Haloperidol and Chlorpromazine can be used when above not available.

Further management plan

- Baseline echocardiography (ECHO)
- ARF register (cardiac clinic), issue ARF prophylaxis card
- Education of patient and family
- Dental examination
- Long term secondary prophylaxis plan

Secondary prophylaxis to prevent recurrent ARF is a long term, regular administration of antibiotics to:

- Prevent group A β-Haemolytic Streptococcal (GAS) pharyngitis
- Prevent repeated development of ARF
- Prevent development of rheumatic heart disease (RHD)
- Reduce severity of RHD
- Help reduce the risk of death from severe RHD

Table 4.4. Antibiotic Regimens for Secondary Prophylaxis (*Adapted from ref 14*)

Antibiotic	Dose	Route	Frequency
First line			
Benzathine Penicillin G	1,200,000 U (900 mg) ≥ 30 Kg 600,000 U (450 mg) ≤30 Kg	Deep IM Injection	4-weekly
***Second line* (If IM route is not possible or refused, adherence should be carefully monitored)**			
Phenoxymethylpenicillin (Pen V)	250mg	Oral	Once daily
Following documented penicillin allergy			
Erythromycin	250mg	Oral	Twice daily

Table 4.5. Recommended duration of prophylaxis for rheumatic fever

Category	Duration
Rheumatic fever without carditis	At least for 5 years or until 21 years of age, whichever is longer
Rheumatic fever with carditis but without residual heart disease (no valvular disease)	At least for 10 years or well into adulthood, whichever is longer
Rheumatic fever with carditis and residual heart disease (persistent valvular disease)	At least 10 years since last episode and at least until age 40 years; sometimes lifelong prophylaxis

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4.3 RHEUMATIC HEART DISEASE

Rheumatic heart disease (RHD) includes a spectrum of lesions from pericarditis, myocarditis, and valvulitis during acute rheumatic fever (ARF), to chronic valvular lesions that evolve over years following one or more episodes of ARF.

The common valvular lesions in RHD

- Mitral regurgitation/stenosis
- Aortic regurgitation/stenosis
- Tricuspid regurgitation/stenosis

Best practice for RHD requires

- Access to echocardiography (for diagnosis)
- Secondary prevention with penicillin prophylaxis (see above on ARF)
- Access to oral healthcare
- Access to a specialist physician, paediatrician and/ or cardiologist
- Adequate monitoring of anticoagulation therapy in patients with atrial fibrillation (AF) and/or mechanical prosthetic valves (where possible)
- Access to cardiothoracic and interventional cardiology services (where possible).

RHD patients may present with

- Heart failure (as a result of valvular insufficiency or stenosis) and/or
- Complications such as atrial arrhythmias, pulmonary oedema, recurrent pulmonary emboli, infective endocarditis, intracardiac thrombus formation, and systemic embolism.

TREATMENT OF RHD

Medical

- Prevent ARF (elimination GAS pharyngitis as above)
- Supportive treatment for CCF (as above)
- Prevent recurrent ARF in children with RHD (see secondary prophylaxis above)
- Monitoring for the complications and sequelae of chronic RHD.

Surgery

- Surgery aims to decrease valve insufficiency/regurgitation.
- Indicated in patients with persistent CCF **OR**
- Worsening after aggressive medical therapy for RHD

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1. Kliegman RM, St. Geme III JW. Nelson Textbook of Pediatrics (21st ed). Elsevier; 2019.
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4.4 INFECTIVE ENDOCARDITIS

Infective endocarditis (IE) is defined as an infection of the endocardial surface of the heart (heart valves and mural endocardium) by microorganisms (mainly bacteria) hence also called bacterial endocarditis.

Risk factors

- Congenital heart disease especially cyanotic CHD
- Rheumatic heart disease
- Prosthetic heart valve
- History of endocarditis
- IV drug use or chronic IV access
- Immunocompromised (HIV, diabetes, cancer patients on chemotherapy)

CLASSIFICATION

Acute infective endocarditis

Caused by virulent organisms, like *S. aureus, enterococci and streptococcus,* which are harmful even on healthy endocardium. Onset of disease is stormy with high grade fever and causes destructive lesions on the endocardium like ulcerations, perforation, regurgitation and ring abscesses especially around prosthetic valves.

Subacute infective endocarditis

Caused by relatively low virulent organisms e.g S.*viridans* and HACEK group (Haemophilus, Aggregatibacter, Corynebacterium, Eikanella, Kingelle). It runs a more insidious course:

- Low grade fever, anorexia, weight loss, influenza like syndromes, myalgia, pleuritic pain
- No specific heart pathological features. Characterised by slowly growing chronic inflammation, fibrosis and with tightly held endocardial vegetations. Chronicity of this type of IE causes chronic antigenemia which in turn is prone to immune complex formation.

Diagnosis

A fever with new/or changing murmur is IE until proven otherwise. The diagnosis of infective endocarditis is challenging. The modified Duke's criteria is used in the diagnosis. There are three categories of diagnosis.

Table 4.6. Definition of infective endocarditis according to the modified Duke's criteria

DEFINITE INFECTIVE ENDOCARDITIS
A. Pathological criteria 1. Microorganisms demonstrated by culture or histologic examination of a vegetation, a vegetation that has embolized, or an intracardiac abscess specimen; or 2. Pathologic lesions; vegetation or intracardiac abscess confirmed by histologic examination showing active endocarditis **B. Clinical criteria** 1. 2 major criteria; or 2. 1 major criterion and 3 minor criteria; or 3. 5 minor criteria
PROBABLE INFECTIVE ENDOCARDITIS
1. 1 major criterion and 1 minor criterion; or 2. 3 minor criteria
REJECTED INFECTIVE ENDOCARDITIS
1. Firm alternative diagnosis explaining evidence of IE; or 2. Resolution of IE syndrome with antibiotic therapy for <4 days; or 3. No pathological evidence of IE at surgery or autopsy, with antibiotic therapy for <4 days; or 4. Does not meet criteria for possible IE as above

Table 4.7. Duke's criteria for the diagnosis of IE

Major criteria	Minor criteria
A. Positive blood culture 1. Typical microorganism consistent with IE from ≥2 blood cultures, 2. Microorganisms consistent with IE from persistently positive blood cultures, defined as: • ≥2 Positive cultures of blood samples drawn >12 h apart or • All of 3 or a majority of ≥4 blood cultures, irrespective of the timing • 1 Positive blood culture for Coxiella burnetii or antiphase-I immunoglobulin G antibody titre >1:800 **B. Evidence of endocardial involvement** Positive echocardiogram, defined as: • Oscillating intracardiac mass on valve or supporting structures, in the path of regurgitant jets, or on implanted material in the absence of an alternative anatomic explanation or • Abscess **OR** • New partial dehiscence of prosthetic valve **OR** • New valvular regurgitation (worsening or changing of pre-existing murmur not enough)	1. Predisposition: predisposing heart condition or IV drug use 2. Fever: temperature ≥38.0°C 3. Vascular phenomena: major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial haemorrhage, conjunctival haemorrhages, and Janeway lesions 4. Immunologic phenomena: glomerulonephritis, Osler nodes, Roth's spots, and rheumatoid factor 5. Microbiological evidence: positive blood culture but does not meet a major criterion as noted above or serological evidence of active infection with organism consistent with IE

Diagnosis is made when there are 2 major criteria or 1 major plus 3 minor or 5 minor criteria. Adapted from ref 15

Table 4.8. Treatment

	Drugs	Dosage	Duration	Remarks
1st Line	Ceftriaxone 80-100mg/kg/day 1-2 times daily/day And, Gentamicin 3-5mg/kg/day 2 divided doses/day (For 2 weeks)		4 weeks	Then, Ciprofloxacin 15mg/kg/day in 2 divided doses for 2 weeks
	ONLY IN THE ABSENCE OF CEFTRIAXONE Crystalline Penicillin (X Pen) 50-100,000 IU/kg in 4 divided doses/day And, Gentamicin 3-5mg/kg/day in 2-3 divided doses/day		4 weeks	Then, Ciprofloxacin 15mg/kg/day in 2 divided doses for 2 weeks
2nd Line	Vancomycin 30-40mg/kg/day in 4 divided doses		4 weeks	Then, Ciprofloxacin 15mg/kg/day in 2 divided doses for 2 weeks

Cardiac Conditions for which prophylaxis with dental procedure is recommended

1. Prosthetic cardiac valve
2. Previous infective endocarditis
3. Congenital heart disease (CHD)*:
    a. Unrepaired cyanotic CHD, including palliative shunts and conduits
    b. Completely repaired CHD with prosthetic material or device, whether placed by surgery or by catheter intervention, during the first 6 months after the procedure
    c. Repaired CHD with residual defects at the site or adjacent to the site of a prosthetic patch or prosthetic device (which inhibits endothelialization)
4. Cardiac transplantation recipients who develop cardiac valvulopathy

*Except for conditions listed above, antibiotic prophylaxis is no longer recommended for any other form of CHD.

Table 4.9. Prophylactic Regimens for Dental Procedures

	Single dose 30-60 min before procedure	
SITUATION	**AGENT**	**CHILDREN**
Oral	Amoxicillin	50 mg/kg
Unable to take oral medications	Cefazolin or ceftriaxone	50 mg/kg (IM, IV)
Allergic to penicillin or ampicillin—oral	Cephalexin Clindamycin, Azithromycin	50 mg/kg 20 mg/kg 10 mg/kg
Allergic to penicillin or ampicillin and unable to take oral medication	Cefazolin or ceftriaxone Clindamycin	50 mg/kg (IM, IV) 20 mg/kg

REFERENCES

1. Kliegman RM, St. Geme III JW. Nelson Textbook of Pediatrics (21st ed). Elsevier; 2019.
2. Park MK, Salamat M. Park's Pediatric Cardiology for Practitioners (7th ed). Elsevier; 2020.

4.5 SYSTEMIC HYPERTENSION

Hypertension in children is defined as systolic and/or diastolic pressure levels greater than the 95th percentile for age and gender on at least three occasions.

BP readings of 5mmHg or more above the 99th centile values are considered as severe hypertension.

Pre-hypertension

Average systolic or diastolic blood pressure between the 90th and 95th percentiles for age and gender.

Centiles are used for the 1-13yrs and the **actual BP values for adolescents > 13yrs**

Table 4.1.1. Normal Values

Blood pressure classification in children and adolescents		
	For children aged 1 to 13 years	**For children aged 13-17 years**
Normal blood pressure	<90th centile	<120/80 mmHg
Elevated blood pressure	≥90th centile to <95th centile or 120/80 mmHg to <95th centile (whichever is lower)	120/80 to 129/<80 mmHg
Stage 1 Hypertension	≥95th centile to <95th centile + 5mmHg or 130/80 to 139/89 mmHg (whichever is lower)	130/80 to 139/89 mmHg
Stage 2 Hypertension	≥95th centile + 5mmHg, or ≥140/90 mmHg (whichever is lower)	≥140/90 mmHg
Severe Hypertension		
Hypertensive Urgency	95th centile + 30 mmHg without symptoms/signs of target end organ damage (See Examination)	>180/120 without symptoms/signs of target organ damage (See Examination)
Hypertensive Emergency	>95th centile + 30 mmHg associated with encephalopathy, eg headache vomiting, vision changes and neurological symptoms (facial nerve palsy, lethargy, seizures,	>180/120 associated with encephalopathy, eg headache vomiting, vision changes, neurological symptoms (facial nerve, palsy, lethargy, seizures, coma) +/- target organ damage

	coma) +/- target-end organ damage

Key points to remember

1. All hypertension in children requires monitoring and follow-up
2. Where possible, abnormal BP measurement should be confirmed, preferably with a manual BP, ensuring appropriately sized cuff is used for accurate measurement
3. Blood pressure should be measured annually in children and adolescents ≥ 3 years of age.
4. Blood pressure should be checked in all children and adolescents ≥ 3 years of age at every healthcare encounter if they have obesity, are taking medications known to increase blood pressure, have renal disease, a history of aortic arch obstruction or coarctation, or diabetes.
5. Measure blood pressure at every healthcare encounter in children < 3 years of age if they have an underlying condition that increases their risk for hypertension.
6. Severe hypertension requires urgent consultation and management. Hypertension associated with encephalopathy is a medical emergency
7. Use simplified blood pressure tables to screen for blood pressure values that may require further evaluation by a clinician as shown below:

Blood pressure screening values based on the fifth percentile of height

Age (years)	Blood pressure (mmHg)			
	Boys		Girls	
	Systolic	Diastolic	Systolic	Diastolic
1	98	52	98	54
2	100	55	101	58
3	101	58	102	60
4	102	60	103	62
5	103	63	104	64
6	105	66	105	67
7	106	68	106	68
8	107	69	107	69
9	107	70	108	71
10	108	72	109	72
11	110	74	111	74
12	113	75	114	75
≥13	120	80	120	80

The goal of this simplified table is to improve the recognition of elevated blood pressure in children starting with the frontline care providers.

Risk Factors

- Obesity
- Renal Disease
- Repaired Coarctation of aorta
- Diabetes Mellitus 1 & 2
- Prematurity
- Intrauterine growth restriction

AETIOLOGY

Essential or primary hypertension, in which a specific aetiology cannot be identified

- **Secondary hypertension in which a cause can be identified** (commonest in paediatric population)

More than 90% of the cases of secondary hypertension are caused by chronic renal disease, renovascular disease and coarctation of the aorta.

Table 4.1.2. Commonest causes of secondary hypertension in children

	Examples of lesions/pathology
Renal	• Acute and chronic Glomerulonephritis • Acute and chronic Pyelonephritis • Congenital anomalies [polycystic or dysplastic kidneys] • Obstructive uropathies [hydronephrosis] • Haemolytic-uremic syndrome • Renal damage from nephrotoxic medications, trauma or radiation
Renovascular	• Renal artery disorders (e.g. stenosis, polyarteritis, thrombosis) • Renal vein thrombosis
Cardiovascular	• Coarctation of the aorta • Conditions with large stroke volume [PDA, aortic insufficiency, system A-V fistula, complete heart block]. These conditions cause only systolic hypertension
Endocrine	• Hyperthyroidism (systolic hypertension) • Excessive catecholamine levels (Pheochromocytoma, Neuroblastoma) • Adrenal dysfunction • Cushing's syndrome • Hyperaldosteronism
Neurogenic	• Increased intracranial pressure
Drugs and chemicals	• Steroids • Sympathomimetic drugs [cough medications] • Heavy metal poisoning [mercury, lead] • CNS stimulants: Cocaine [acute or chronic use]

Table 4.1.3. Most common causes of hypertension by age groups

Age	Causes
New born	Renal artery stenosis, renal artery thrombosis, congenital renal malformation coarctation of the aorta, bronchopulmonary dysplasia
< 6 years	Renal parenchymal disease, Coarctation of the aorta, Renal artery stenosis
6 -10 years	Renal artery stenosis, renal parenchymal disease, primary hypertension
>10 years	Primary hypertension, renal parenchymal disease

Diagnosis and work-up

- Mild hypertension is usually asymptomatic and may only be picked up incidentally
- Severe hypertension may be symptomatic (headache, dizziness, nausea and vomiting, irritability, personality changes) occasionally presenting with neurological manifestation, CCF, renal dysfunction, and stroke.
- History, physical findings and laboratory tests usually point to the cause of hypertension.

Table 4.1.4. Routine and special laboratory tests and their significance

Routine Laboratory Tests	Significance of Abnormal Results
Urinalysis, urine culture, urea creatinine, uric acid	Renal parenchymal disease
Serum electrolyte (hypokalaemia)	Hyperaldosteronism 1° or 2°, congenital adrenal hyperplasia, renin producing tumours
ECG, CXR, ECHO	Cardiac causes e.g. coarctation of the aorta
Specialised tests	
IVU, US KIDNEY, CT scan	Renal parenchymal disease, renovascular disease, tumours (neuroblastoma, Wilms)
Plasma renin activity	High-renin hypertension Renovascular disease Renin-producing tumours Some Cushing's syndrome Some essential hypertension Low-renin hypertension Adrenogenital syndrome Primary hyperaldosteronism
24-hr urine collection for 17-ketosteroids and 17-hydroxycorticosteroids	Cushing's syndrome, congenital adrenal hyperplasia
24-hr urine collection for catecholamine levels and VMA	Pheochromocytoma, neuroblastoma
Aldosterone	Hyperaldosteronism, primary or secondary renovascular disease, renin-production tumours
Renal vein plasma renin activity	Unilateral renal parenchymal disease, renovascular hypertension
Abdominal aortogram	Renovascular hypertension, abdominal CoA, unilateral renal parenchymal disease, pheochromocytoma.

MANAGEMENT

As most of the causes of hypertension in children are secondary, the ultimate aim of treatment, in addition to general measures and pharmacological therapy should be to remove the cause of hypertension whenever possible: e.g coarctation repair or renal artery balloon angioplasty or surgery for renovascular disease. The blood pressure treatment goal in children without diabetes or chronic kidney disease treated with nonpharmacologic and/or pharmacologic therapy is a reduction of systolic and diastolic BP below the 90th percentile or <130/80 in adolescents (13 years or older).

Non-pharmacologic/General measures:

- Encourage weight reduction if indicated
- Low salt foods/diet
- Avoidance of smoking/Drugs
- Regular exercise and restriction of sedentary lifestyle

PHARMACOLOGIC MEASURES

Whom to treat

- Symptomatic hypertension and severe hypertension
- Prehypertension in presence of comorbid conditions, such as chronic kidney disease or diabetes mellitus
- Hypertensive children with diabetes mellitus or other risk CVD factors, such as dyslipidaemia
- Hypertensive target-organ damage, most often left ventricular hypertrophy (LVH) seen on echo or ECG.
- For essential hypertension (without any evidence of target-organ damage) that persists despite a trial of four to six months of nonpharmacologic therapy.

Choice of Antihypertensive drugs

Data comparing antihypertensive drugs in children are lacking and hence it is not possible to make an evidence-based choice. Therefore, the recommendations on the choice of agent for initial therapy are based upon the underlying cause of HTN, concurrent disorders, and the preference and experience of the responsible clinician. Data from adult trials and limited paediatric information have provided information regarding the choice of antihypertensive drugs for children with a specific underlying medical condition, such as primary HTN, CKD, and diabetes mellitus (DM).

Primary hypertension:

- Start with an agent that blocks the renin/angiotensin system (i.e., ACE inhibitor or ARB) or a CCB for adolescent females who are sexually active. Monitoring serum creatinine and potassium, particularly after medication initiation or significant dose increases.
- If the target BP goal is not met with the maximum allowable dose of the initial medication add a thiazide diuretic as a second agent, since salt and water retention commonly occur with antihypertensive therapy.
- Other alternative approaches include switching agents between drug classes or the use of a beta blocker.

- Data on the initial use of combination therapy of thiazide diuretics and other classes of antihypertensive medications (beta blockers, ACE inhibitors, and CCBs) are limited in children with primary HTN, and it is not recommended.

Secondary hypertension is the commonest cause of hypertension in children and the treatment of the underlying medical conditions influences the choice of specific class of antihypertensive drugs.

- **Renovascular disease**: Use CCBs rather than an ACE inhibitor or ARB because of the concern that ACE inhibitors and ARBs reduce glomerular filtration rate (GFR) and may cause renal function impairment and hyperkalaemia. However, the use of ACE/ARB is not contraindicated in unilateral renal artery stenosis (RAS) as long as there is appropriate monitoring of renal function and serum potassium and follow-up for adjustment of medication if there is evidence of renal impairment or an increase in potassium level.

ACE inhibitors are contraindicated in obstructive lesions like bilateral renal artery stenosis, aortic stenosis and coarctation of the aorta.

- **Chronic kidney disease**: Use ACE inhibitor or ARB therapy as they appear most beneficial in patients with renal disease associated with proteinuria and should be used as the initial therapy in these patients. (Monitor creatinine and potassium)
- **Diabetes mellitus**: Use ACE inhibitors and ARBs as they delay the progression of diabetic nephropathy and decrease albuminuria in both type 1 and type 2 diabetes.)

The stepped-care approach, using three classes of drugs: diuretics, B-blockers, and vasodilators, is popular.

Step 1

Initiate with a small dose of a single antihypertensive drug (thiazide diuretic or adrenergic inhibitor), adjust upwards as necessary

Step 2

If the first drug is not effective, a second drug may be added to, or substituted for, the first drug, starting with a small dose and proceeding to full dose.

Step 3

If the blood pressure remains elevated, a third drug, such as a vasodilator, may be added to the regimen. At this point, the possibility of secondary hypertension should be reconsidered.

Table 4.1.5. Recommended oral dosage of selected antihypertensive drugs for children

Drugs	Dose	Times/day
Calcium channel blockers **(CCB)**		
• Nifedipine • Amlodipine (≥ 6 years old)	• 0.1 -1 mg/kg/day • 2.5-5mg/day	• 1-2 times a day • Once a day
• ***β-Blockers***		
• Propranolol • Atenolol	• 1-3mg/kg/day • 1-2mg/kg/day	• 2-3 times per daily • Once daily
• ***ACE inhibitors***		
• Captopril • Enalapril • Lisinopril	• 0.3-0.5mg/kg/dose (max 6mg/Kg/day) • 0.08-0.6mg/kg/day • 0.07-0.6mg/kg/day	• 3 times per day • 1-2 times per day • Once daily
• ***ARB***		
• Losartan	• 0.7-1.4mg/kg/day	• Once daily
• ***Diuretics***		
• Hydrochlorothiazide • Furosemide [lasix] • Spironolactone [aldactone]	• 1-2mg/kg/day • 0.5- 2mg/kg/dose • 1-3mg/kg/day	• Once daily • 1-2 times per day • 1-2 times per day

Hypertensive crisis (Chart)

A hypertensive emergency is defined as any of the following features:

- The patient has severe hypertension [>180 mm Hg systolic or 110 mm Hg diastolic] or rapidly increasing blood pressure.
- The patient has neurologic signs [hypertensive encephalopathy] with severe headache, vomiting, irritability, apathy, papilloedema, retinal haemorrhage, or exudates.
- The patient has CHF or pulmonary oedema.

Aggressive administration of antihypertensive drugs is indicated to lower blood pressure – no more than 25% reduction of systolic BP in the first 8 hours; gradually attain normal BP over 24-48 hours:

Table 4.1.6. Hypertensive Crisis

Drug	Dose	Comment
Nifedipine	0.2 - 0.5 mg/kg, every 4 to 6 hours, 12 to 24 hours if retard Nifedipine is used ***maximum 10mg per dose**	If patient is conscious
Hydralazine	0.15 mg/kg IV slow infusion over 20 minutes	The dose may be repeated at 4 to 6 hours interval. The dose may be repeated at 4-6 to 6-hours interval.
Nitroprusside	1-3µg/kg per min as IV drip	
Labetalol	0.2-2mg/kg/hour IV drip	Alpha and beta blocker
Diazoxide	3-5mg/kg as IV bolus	
Furosemide	1mg/kg IV bolus	To initiate diuresis

(*Fluid balance must be controlled carefully, so intake is limited to urine output plus insensible loss. Seizures may be treated with slow intravenous infusion of diazepam 0.2 mg/kg or another anticonvulsant medication. When a hypertensive crisis is under control, oral medications replace the parenteral medications.)

REFERENCES

1. Kliegman RM, St. Geme III JW. Nelson Textbook of Pediatrics (21st ed). Elsevier; 2019.
2. Park MK, Salamat M. Park's Pediatric Cardiology for Practitioners (7th ed). Elsevier; 2020.

4.6 CYANOTIC SPELL

A cyanotic spell, also called hypoxic spell or “Tet” spell occurs in young infants with Tetralogy of Fallot (TOF). It consists of hyperpnoea (i.e. rapid and deep respiration) worsening cyanosis and the disappearance of the heart murmur.

This occasionally, if not treated, results in complication of the central nervous system and even death.

Place the infant who has a hypercyanotic spell in the knee-chest position. This position increases systemic vascular resistance in the lower extremities.

Adapted from Child Health Nursing: Partnering with Children and Families, 2006.



Source: https://www.aboutkidshealth.ca/article?contentid=896&language=english



Source: https://cpictures.homes/knee-chest-position-child

ALGORITHM FOR CYANOTIC SPELL

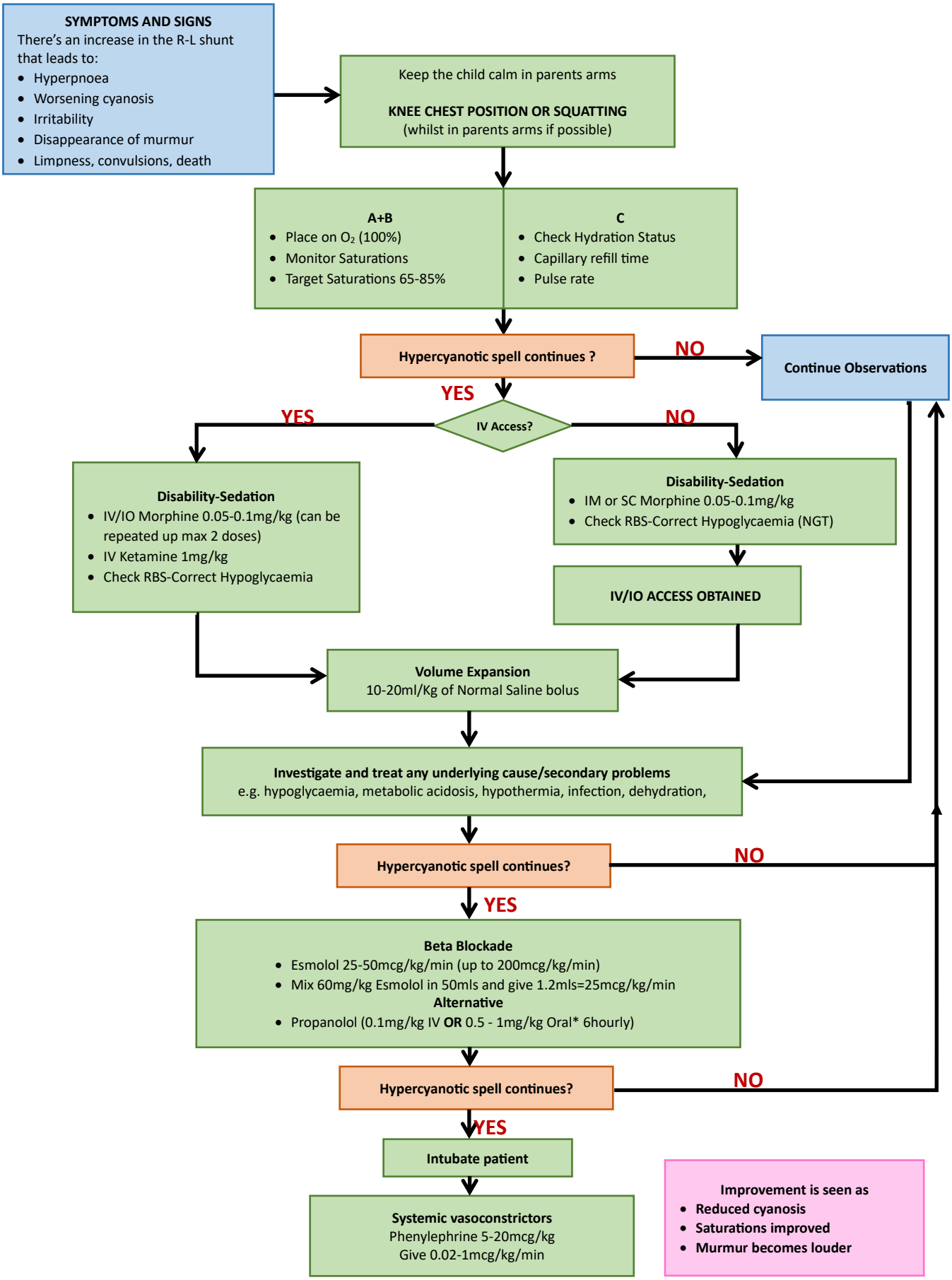


Fig 4. Cyanotic spell

Treatment

- Using the knee-chest position and holding the baby traps systemic venous blood in the leg thereby decreasing the systemic venous return and helping to calm the baby. The knee-chest position may also increase SVR by reducing arterial blood flow through the femoral arteries.
- Morphine sulphate, at 0.2 mg/kg administered SC, IM or IV suppresses the respiratory centre and abolishes hyperpnoea.
- Sodium bicarbonate (NaHCO3) at 1 mmol/L IV Corrects acidosis and eliminates the respiratory centre–stimulating effect of acidosis. The same dose can be repeated in 10 to 15 minutes
- Administration of oxygen may improve arterial oxygen saturation a little.
- Vasoconstrictors such as phenylephrine at 0.02 mg/kg IV raise SVR.
- Ketamine at 1 to 3 mg/kg given over 1 minute is a good drug to use since it simultaneously increases the SVR and sedates the patient. Both effects are known to terminate the spell.
- Propranolol at 0.01 to 0.25 mg IV slowly has been used successfully in some cases both acute or chronic. Its mechanism of action is not entirely clear. When administered for acute cases propranolol may reduce the spasm of the RV outflow tract and slow the heart rate. The successful use of propranolol in the prevention of hypoxic spell is more likely the result of the drugs peripheral action. It may stabilize vascular reactivity of the systemic arteries thereby preventing a sudden decrease in the SVR.
- Oral propranolol therapy at 2 to 6 mg/kg/day in three to four divided doses may be used to prevent recurrence of hypoxic spell and delay corrective surgical procedures in high risk patients.

Physicians should recognize and treat hypoxic spells. It is important to educate parents to recognize the spell and what to do.

Maintenance of good dental hygiene and antibiotic prophylaxis against subacute bacterial endocarditis (SBE) are important

Relative iron-deficiency anaemia should be detected and treated. Anaemic children are more susceptible to CVA. Normal Hb or HCT or decreased RBC indices indicate iron-deficiency anaemia in cyanotic patients.

REFERENCES

1. Kliegman RM, St. Geme III JW. Nelson Textbook of Pediatrics (21st ed). Elsevier; 2019.
2. Park MK, Salamat M. Park's Pediatric Cardiology for Practitioners (7th ed). Elsevier; 2020.

4.7 HYPERVISCOCITY SYNDROME

Hyperviscosity syndrome refers to the clinical sequelae of increased blood viscosity. It can result from increased cellular blood components, typically white or red blood cells, in hyperproliferative states such as leukemia, polycythemia and myeloproliferative disorders. It can also occur as a result of increased serum viscosity due to increased circulating serum immunoglobulins

Indications for Phlebotomy

Hb >21g/dl or HCT>65 % with any of the following signs and symptoms;

- Headache
- Abdominal pain
- Limb pain
- Transient Ischaemic Attack (TIA)
- Cerebral vascular Accident (CVA)
- Convulsions

$$\textit{Blood Volume to exchange} = \frac{Initial\ Haematocrit - Desired\ Haematocrit}{Initial\ Haematocrit} \ x\ 90ml/kg\ x\ Weight$$

- Prehydrate with Normal Saline (NS) at 20ml/ kg over 30 minutes to 1 hour
- The desired Haematocrit is usually set at 60%
- Bleed as per calculation above and replace the amount with NS at 1:1.(remove 10-12% of total blood volume(80-90ml/kg) per session).
- When large volumes are calculated perform multiple sessions (e.g half the volume calculated in 2 sessions)

Example: Patient with Haematocrit of 71 and a weight of 12kgs

$$\textit{Blood Volume to exchange} = \frac{71-60}{71}\ x\ 90ml/kg\ x\ 12kg$$

Blood Volume to be Exchange = 136.9mls

*Therefore, 137mls of blood to be withdrawn and replaced with 137mls of normal saline

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1. Hemingway TJ. Hyperviscosity Syndrome. Medscape. Updated Jul 19, 2023. Accessed Oct 31, 2024
2. Kliegman RM, St. Geme III JW. Nelson Textbook of Pediatrics (21st ed). Elsevier; 2019.
3. Park MK, Salamat M. Park's Pediatric Cardiology for Practitioners (7th ed). Elsevier; 2020.

4.8 ARRHYTHMIAS

Arryhthmias are abnormalities in the rate or rhythm of the heart or both. They may occur due to physiological responses or due to a pathological process (e.g. sepsis, drugs or poisoning) or they may be a result of an underlying heart problem. Arrhythmias are broadly classified into bradyarrhythmias and tachyarrythmias.

Recognizing patients with Arrhythmias

Pulse character and rate remain the key vital signs for every paediatric patient. Remember for younger paediatric patients, there may be no reported symptoms and they rely on the parents agency. Secondly illness in children often results in a change in vitals such as temperature and heart rate due to limited reserve

Presentation of arryhthmias in children[17]

- History of palpitations (verbal child)
- Poor feeding (pre-verbal child)
- Heart failure or shock
- No symptoms

Table 4.1.7. ABC assessment for patients with arrythmias

Airway
Ensure the airway is open – consider airway-opening manoeuvres, airway adjuncts or urgent induction of anaesthesia and intubation to secure the airway.
Breathing
High-flow oxygen through a face mask with reservoir. Hypoventilating or bradycardia: support with a bag–valve–mask device and consider intubation and ventilation.
Circulation
An abnormal pulse rate is defined as one falling outside the normal range Most serious disease or injury states are associated with a sinus tachycardia. In infants this may be as high as up to 220 beats per minute (bpm) and in children up to 180 bpm. Rates over these figures are highly likely to be tachyarrhythmias, but in any case of significant tachycardia, i.e. 200 bpm in an infant and 150 bpm in a child, **an ECG rhythm strip should be examined and, if in doubt, a full 12-lead ECG performed**. Very high rates may be impossible to count manually and the pulse oximeter will often be unreliable. Again, a rhythm strip is advised. An abnormally slow pulse rate is defined as one less than 60bpm or a rapidly falling heart rate associated with poor systemic perfusion. This will almost always be in a child who requires major resuscitation.

How is the ECG?
1. Is the rate: too fast vs too slow? 2. Is the rhythm: regular vs irregular? 3. Are the QRS complexes: narrow vs broad?

Bradyarrhythmias

Bradyarrhythmias manifest as slow heart rate and often an irregular rhythm. Causes of bradyarryhthmias in children include the following but are not limited to:

- Pre-terminal event in hypoxia or shock
- Raised intracranial pressure
- Conduction pathway damage following cardiac surgery
- Congenital heart block (rare)
- Long QT syndrome

Bradycardia is usually a pre-terminal rhythm. It is seen as the final response to profound hypoxia and ischaemia and its presence is ominous. Bradycardia may also be precipitated by vagal stimulation as occurs in tracheal intubation and suctioning and may be found in postoperative cardiac patients. The rhythm is usually irregular. Bradycardia may be seen in patients with raised intracranial pressure. Bradycardia can be a side effect of poisoning with digoxin or β-blockers. Incidental bradycardia in a clinically well child may be seen in athletic and sporty children and does not require any treatment.
The approach to a child with bradycardia is shown in the algorithm below.

APPROACH TO A CHILD WITH BRADYCARDIA

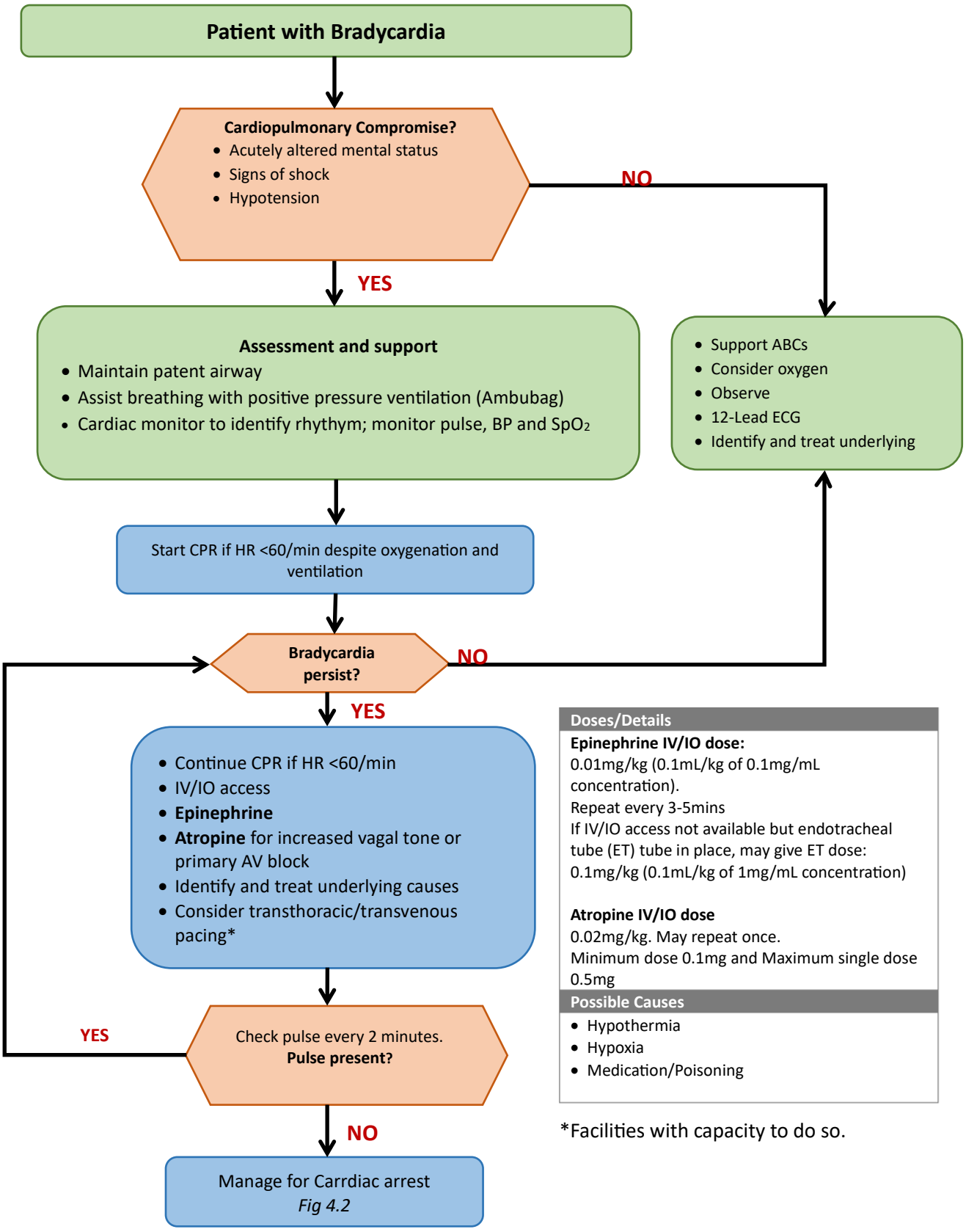


Fig 4.1. Adapted from American Heart Association

Tachyarrhythmias

Tachyarrhythmias manifest as a fast heart rate and often a regular rhythm. Causes of tachyarryhthmias in children include the following but are not limited to:

- Tachyarrhythmia with absent P-waves and a narrow QRS complex on the ECG is supraventricular tachycardia. The rhythm is usually regular.
- Tachyarrhythmia with a wide QRS complex on the ECG is ventricular tachycardia; this can be provoked by:
- Hyperkalaemia, or poisoning with tricyclic antidepressants. Additional details on the management of the poisoned child with ventricular tachycardia can be found on *fig 4.2*

The approach to a child with tachycardia is shown in the algorithm below.

PAEDIATRIC TACHYCARDIA WITH A PULSE ALGORITHM

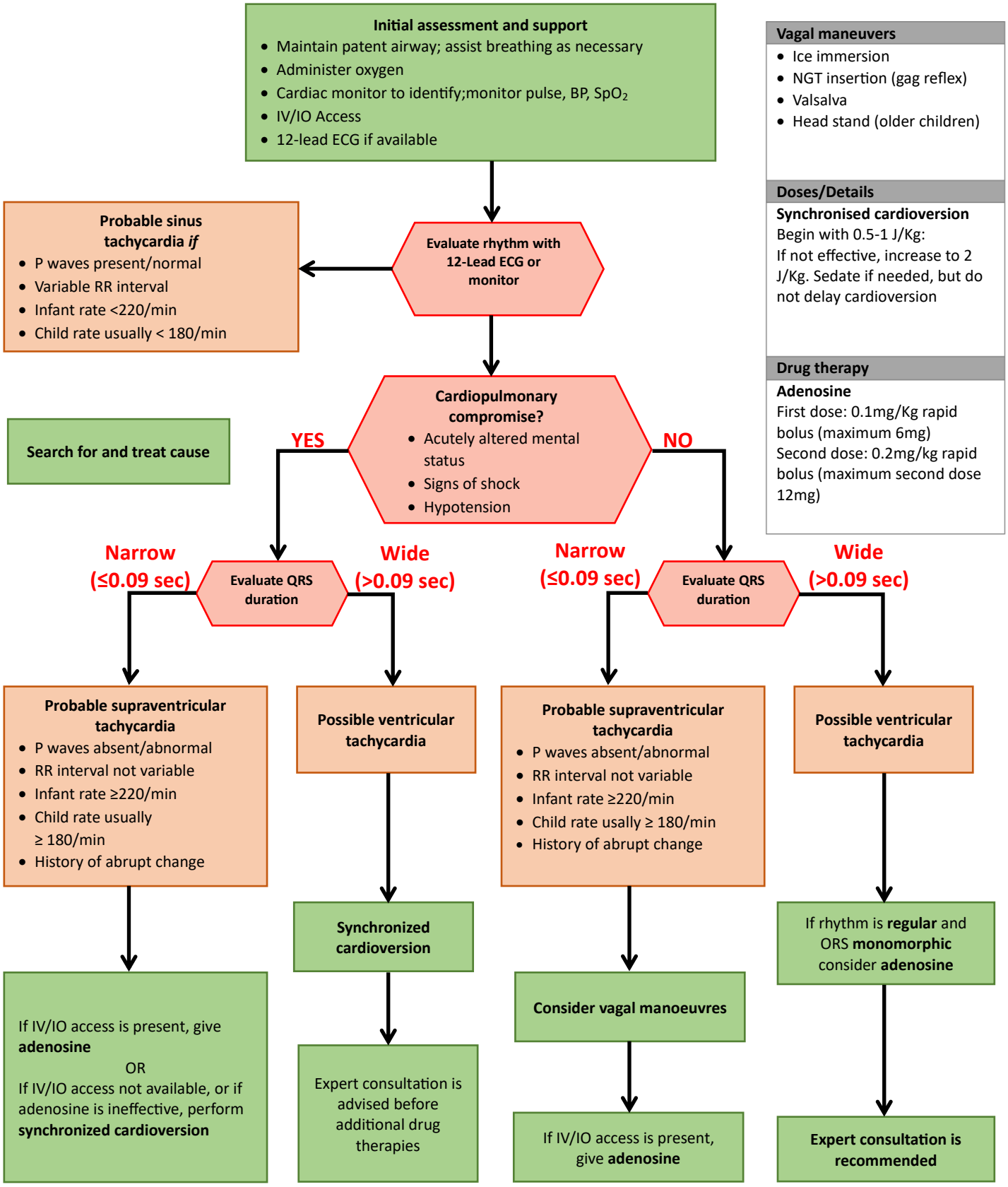


Fig 4.2. Adapted from American Heart Association

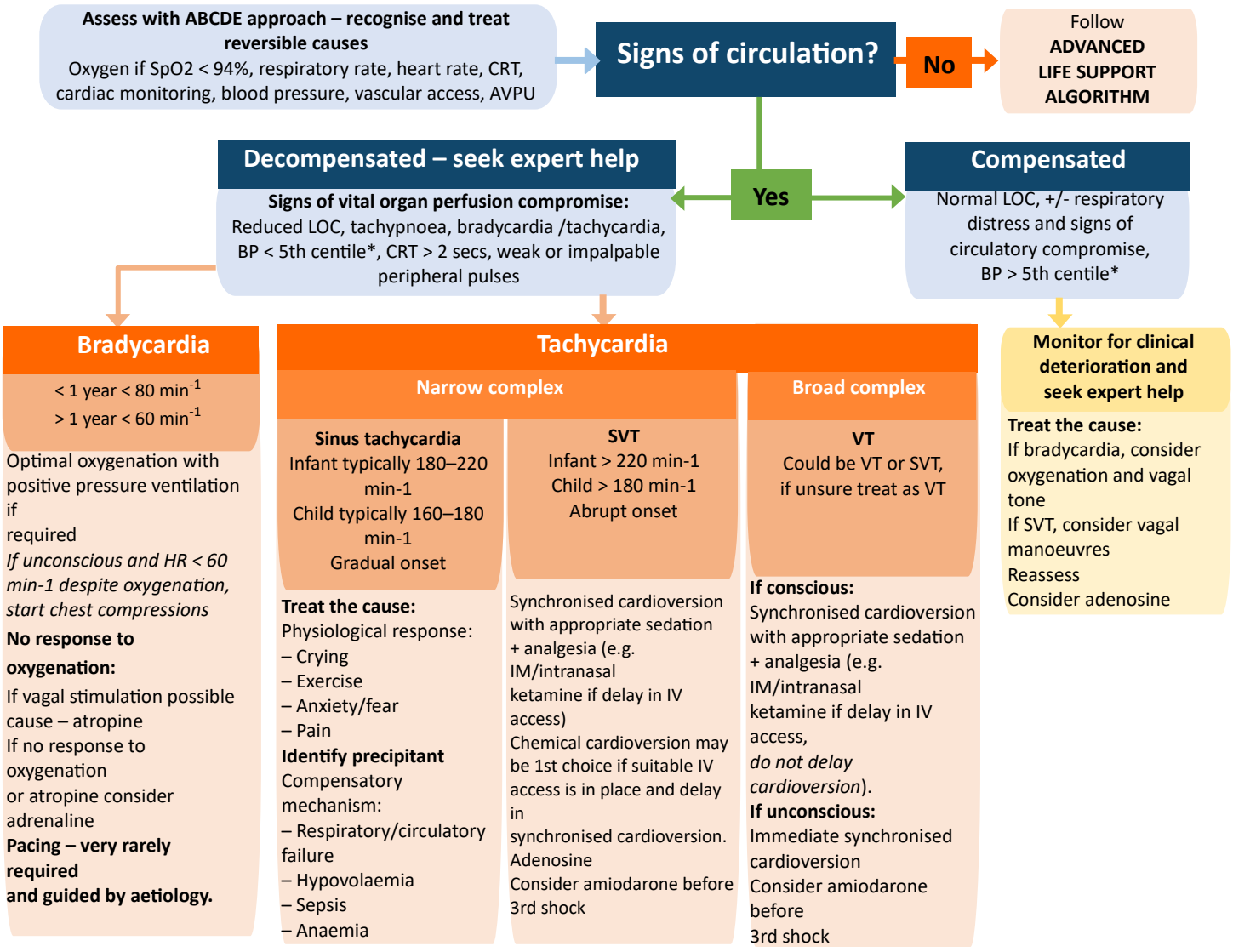
FOLLOW UP CARE OF PATIENTS WITH SVT

Follow up therapy is indicated for patients without spontaneous resolution or who those not responding to vagal manoeuvres. For patients with more frequent or sustained episodes, or those who cannot reliably distinguish symptoms, a daily prophylactic medication is used after cardioversion. Infants, by definition, cannot report their symptoms. For this reason, infants are typically treated with a prophylactic medication regimen to reduce risk of recurrent SVT. Propranolol, flecainide, digoxin, and sotalol are the most common agents for long term prophylaxis of SVT. Amiodarone has also found use in some patients but requires thyroid function monitoring particularly for long term therapy. Patients are followed up regularly for the first 3-4months when the frequency of recurrence is highest and less frequently thereafter. The pill in the pocket regimen practiced in western countries can only be used on a case-by-case basis in discussion with a paediatric cardiologist. Below is a table showing regimens for SVT prophylaxis:

Table 4.1.8. SVT prophylaxis

Drug	Dosage	Remarks
Digoxin	0.01-0.015 mg/kg/day	Dosage is age dependent
Atenolol	1-2 mg/kg/day	Single daily dose
Propranolol	1-3 mg/kg/day	3 daily doses
Nadolol	0.5-2.5 mg/kg/day	Single daily dose
Propafenone	200-600 mg/m^2/day	QRS prolongation
Flecainide	3-12 mg/kg/day	Proarrhythmogenic
Sotalol	2-8 mg/kg/day	Proarrhythmogenic
Amiodarone	Loading dose: 10-20 mg/kg/day (10 days) Maintenance dose: 5-10 mg/kg/day	Systemic adverse effects

PAEDIATRIC CARDIAC ARRHYTHMIAS



Drug	Atropine	Adrenaline	Adenosine	Amiodarone	Synchronised cardioversion	Magnesium
Treatment	**Up to 11 years:** 20 mcg kg^{-1}. **12–17 years:** 300–600 mcg, larger doses may be used in emergency.	**For bradycardia:** 10 mcg kg^{-1} repeat if necessary.	**Up to 1 year:** 150 mcg kg^{-1}, increase 50–100 mcg kg^{-1} every 1–2 min. Maximum single dose: Neonates 300 mcg kg^{-1}, Infants 500 mcg kg^{-1}) **1–11 years:** 100 mcg kg^{-1} increase 50–100 mcg kg^{-1} every 1–2 min. Maximum single dose: 500 mcg kg^{-1} (max. 12 mg) **12–17 years:** 3 mg IV, if required increase to 6 mg after 1–2 min, then 12 mg after 1–2 min	5 mg kg^{-1} – by SLOW IV infusion (> 20 min) before 3rd cardioversion in discussion with paediatric cardiologist/expert	With appropriate sedation + analgesia (e.g. IM/intranasal Ketamine if delay in IV access + airway management) – IV access attempts must not delay cardioversion **1st shock:** 1 J kg^{-1} **2nd shock:** 2 J kg^{-1}, consider up to 4 J kg^{-1}	25–50 mg kg-1 Maximum per dose 2 g to be given over 10–15 min, may be repeated once if necessary, in Torsades de pointes VT

Age	*Systolic BP 5th centile mmHg
1 month	50
1 year	70
5 years	75
10 years	80

Fig 4.3. Adapted from resuscitation council UK guideline 2021

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5.1 CHILD WITH STRIDOR

Stridor is a harsh sound produced during inspiration, which is due to narrowing of the airway from the oropharynx, glottis, and the trachea. It usually occurs in inspiration but may occur in expiration.

Common causes

- Viral croup
- Foreign body
- Epiglottitis
- Congenital anomalies especially in neonates

Others causes

- Anaphylaxis
- Burns
- Retropharyngeal abscess

The different causes of stridor are discussed in detail in the following chapters.

5.2 VIRAL CROUP (LARYNGOTRACHEOBRONCHITIS)

Croup (laryngotracheobronchitis) is a common cause of upper airway obstruction in toddlers (1-3 years old).

It is characterised by varying degrees of inspiratory stridor, barking cough, and hoarseness due to inflammation in the laryngeal region.

Clinical Presentation

- Low grade fever and coryzal symptoms are followed over 12–24 h by a harsh, barking cough
- Stridor is most evident when the child is upset or agitated
- Respiratory distress of varying degrees
- Usually, resolves spontaneously over a 3–4-day period

Diagnosis

Croup is a clinical diagnosis

Neck x-rays are unnecessary unless the diagnosis is in doubt

Important differential diagnoses

- Acute epiglottitis
- Bacterial tracheitis
- Foreign body inhalation
- Anaphylaxis
- Retropharyngeal abscess

Table 5. Assessment of severity

Sign	Mild croup	Moderate croup	Severe croup
Stridor	Only when agitated	At rest	Severe, biphasic
Recession	Mild subcostal	Moderate tracheal tug	Use of accessory muscles
Level of consciousness	Restless when disturbed	Anxious, agitated	Lethargic, drowsy

Management

Keep the child on the mother's lap and handle gently.

Do not attempt to *forcefully* examine the oropharynx.

See flow chart below:

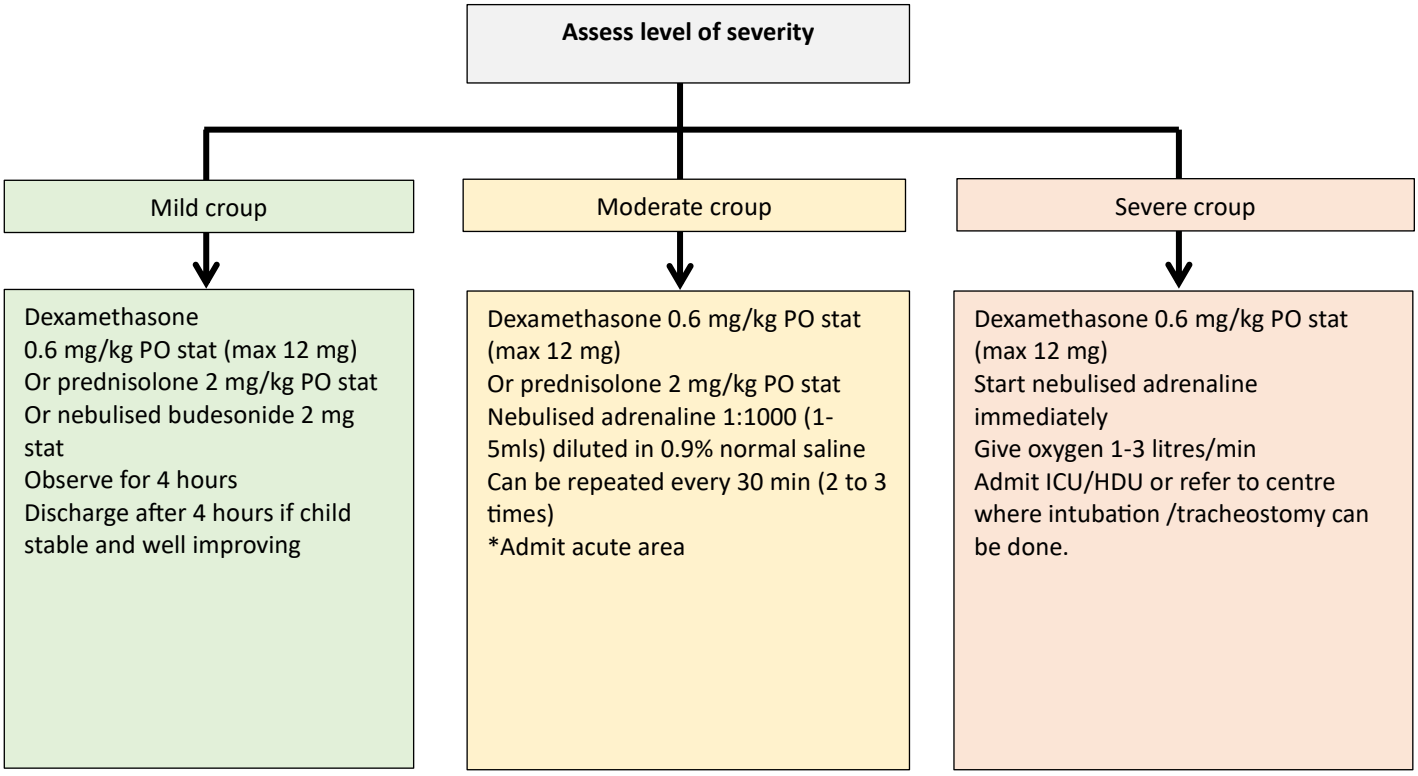


Fig 5. Injectable dexamethasone taken orally can be used if no oral dexamethasone is available.

* As airway obstruction can occur suddenly, ensure that facilities for an emergency intubation and/or tracheostomy are immediately available if required.

Caution

- Avoid using oxygen unless there is impending airway obstruction. Nasal prongs or a nasal or nasopharyngeal catheter can upset the child and precipitate obstruction of the airway.
- Do not use antibiotics or bronchodilators unless diagnosis is in doubt
- Do not give adrenaline nebulisations if baseline heart rate >200/min

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5.3 ACUTE EPIGLOTTITIS

Epiglottitis is inflammation of the epiglottis. The epiglottis closes the airway during swallowing. Epiglottitis is a medical emergency that may result in death if not treated quickly due to rapid airway obstruction.

The incidence of epiglottitis due to Haemophilus influenza (HiB) has reduced due to Hib vaccine in the Extended Programme on Immunisation.

Signs and Symptoms

- sore throat with difficulty in speaking
- difficulty in breathing
- stridor
- fever
- drooling of saliva, difficulty in swallowing or inability to drink.

Management

- Treatment of patients with epiglottitis is directed to relieving the airway obstruction and eradicating the infectious agent.
- Keep the child calm in a seated or leaning forward position, and provide humidified blow-by oxygen, with close monitoring. Avoid laying the child in supine position.
- Avoid examining the throat if the signs are typical, to avoid precipitating obstruction.
- Elective intubation is the best treatment if there is severe obstruction but may be very difficult due to severe swelling; consider the need for surgical intervention to ensure airway patency.
- Give IV antibiotics when the airway is safe: ceftriaxone at 80 mg/kg once daily for 5 days.
- Give Paracetamol 15mg/kg qid or Ibuprofen 10mg/kg tds when airway is secure.

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5.4 THE WHEEZING CHILD

Wheezing is a common presentation in young children. Diagnosis and treatment of these children can be challenging, as arriving at a final diagnosis often requires a process of exclusion.

Without a clear diagnosis, a correct treatment approach is not possible, and a diagnosis may become more certain depending on the treatment response.

The algorithm shown in Fig 13. below considers the most common primary presentations with wheeze and gives one possible approach to this problem. A brief summary of the major wheezing conditions is provided in Table 17.

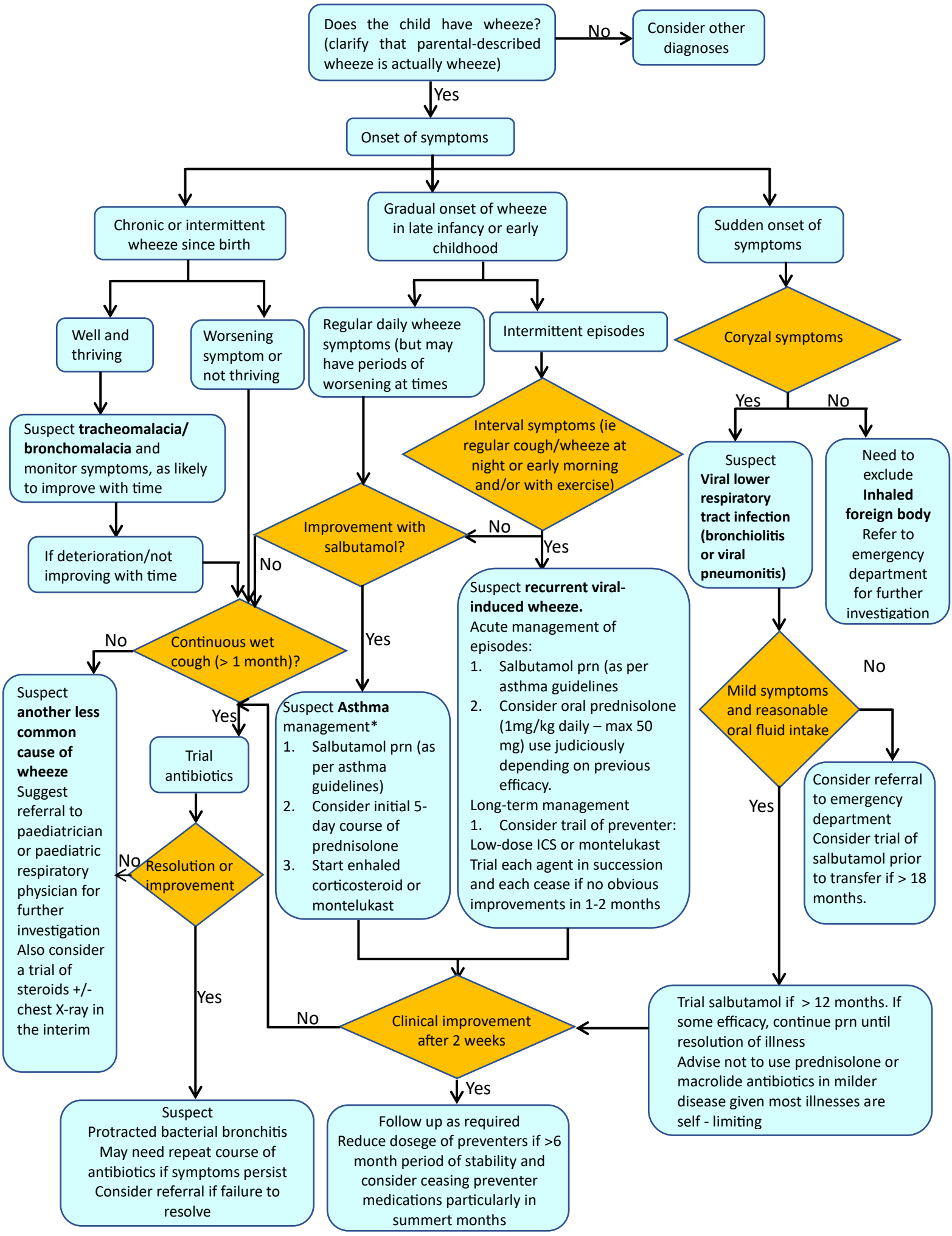


Fig 5.1. Most common primary presentations with wheeze

Table 5.1. Summary of the most common wheezing conditions in young children (continues on next page)

Condition	Estimated incidence in children	Clinical signs	Investigation	Expected clinical course	Management
Viral wheezing (these include a spectrum of viral LRTIs that are not always clearly separated e.g., viral LRTI/recurrent viral-induced wheeze/bronchiolitis – management of episodes is identical, and the distinction is sometimes arbitrary)	Very common, especially in the first 2 years of life 50% of children will have at least one wheezing episode	Wheeze associated with respiratory tract infections May be singular or recurrent Bronchiolitis (usually in children <2 years) manifests with fine crackles +/– wheeze on auscultation	No specific investigations Nasal samples sent for virology usually do not change clinical management but isolation of RSV in infants is highly suggestive of bronchiolitis	60% will outgrow wheeze by 6 years A further 15% acquire wheezing after 6 years After 7–8 years, only 1 in 5 will outgrow it	Trial salbutamol if >1 year of age and continue only if effective Supportive care involving monitoring adequate fluid intake (>50% of usual intake) and for signs of increasing respiratory distress
Asthma	5–8 % of the paediatric population	Wheeze on a regular basis Some will have persistent/interval symptoms between episodes of viral wheeze (cough and/or wheeze at night or with exercise)	Spirometry with bronchodilator response may be possible in children ≥5 years of age in experienced laboratories	Usually expected to be lifelong but clinical courses can vary widely between individuals	Exacerbations: Regular salbutamol (as per asthma guidelines) and consider oral prednisolone for up to 5 days Regular preventer usually indicated
Airway malacia (airway floppiness): either tracheomalacia or bronchomalacia	1 in 2100	Usually present soon after the neonatal period with wheeze, stridor, cough, and rattling; children are	Bronchoscopy usually diagnostic but not necessary in most cases	Majority outgrow it by age 2 years Secondary PBB can occur, presumably from poor	Treatment rarely required If there are worsening symptoms or failure to thrive, specialist

		usually well and often labelled as 'happy wheezers'		cough clearance	referral is indicated
Protracted bacterial bronchitis (PBB)	Probably common, but exact incidence unknown	Chronic wet cough (typically >4 weeks). Concurrent wheeze and/or rattly breathing is common	Bronchoscopy may assist diagnosis, but usually unnecessary Radiological findings usually normal or non-specific	Majority resolve with 1–2 courses of antibiotics	2–6-week course of antibiotics: commonly amoxicillin/clavulanic acid (approximately 20 mg/kg/dose twice daily)

Key points

- Determining the cause of wheeze in young children can be difficult and sometimes is determined only following a trial of treatment.
- Parents' description of wheeze can be inaccurate and often needs elaboration or confirmation with impersonation or video recordings.
- Asthma is very common but other causes are also common and worth considering in the event of poor efficacy of asthma treatment.
- A diagnosis of protracted bacterial bronchitis should be considered for children with >4 weeks of continuous wet cough.

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5.5 BRONCHIOLITIS

Bronchiolitis is a clinical diagnosis, based on history and examination. It typically begins with an acute upper respiratory tract infection followed by onset of respiratory distress and fever. Illness usually resolves without intervention in 7 – 10 days, with peak severity two to three days post onset.

Diagnosis

History

History should include:

- recent respiratory symptoms
- feeding including:
  - duration of feeds (feeding more difficult with more severe illness)
  - breast feeding refusal
- underlying medical conditions including chronic lung disease, congenital heart disease and chronic neurological conditions
- chromosomal abnormalities including Trisomy 21
- prematurity
- post-natal exposure to cigarette smoke

Symptoms and signs

- cough
- tachypnoea
- retractions
- diffuse crackles or wheeze on auscultation

Table 5.2. Examination

Assessment of severity of acute bronchiolitis			
	Mild	**Moderate**	**Severe**
Behaviour	Normal	Some/intermittent irritability	Increasing irritability and/or lethargy, fatigue
Respiratory rate	Normal – mild tachypnoea	Increased	Marked increase or decrease
Use of accessory muscles	Nil to mild chest wall retraction	Moderate chest wall retractions Tracheal tug Nasal flaring	Marked chest wall retractions Marked tracheal tug Marked nasal flaring
Oxygen saturations in room air	SpO2 >92%	SpO2 90-92%	SpO2 <90% May not be corrected by oxygen
Apnoeic episodes	None	May have brief apnoea	May have increasingly frequent or prolonged apnoea
Feeding	Normal	May have difficulty with feeding or reduced feeding	Reluctant or unable to feed

When to admit

When assessing a child in a secondary care setting, admit them to hospital if they have any of the following:

- Apnoea (observed or reported)
- Persistent oxygen saturation of less than 92% when breathing air
- Inadequate oral fluid intake (50–75% of usual volume, taking account of risk factors and using clinical judgement)
- Persisting severe respiratory distress, for example grunting, marked chest recession, or a respiratory rate of over 70 breaths/minute.

When deciding whether to admit a child with bronchiolitis, take account of any known risk factors for more severe bronchiolitis such as:

- Chronic lung disease (including bronchopulmonary dysplasia), haemodynamically significant congenital heart disease
- Age in young infants (under 3 months)
- Premature birth, particularly under 32 weeks
- Neuromuscular disorders

- Immunodeficiency

When deciding whether to admit a child, consider factors that might affect a carer's ability to look after a child with bronchiolitis, for example:

- Social circumstances
- The skill and confidence of the carer in looking after a child with bronchiolitis at home/confidence in being able to spot red flag symptoms.
- Distance to healthcare in case of deterioration.

Consider seeking senior emergency/paediatric advice for infant with moderate disease.

Seek senior advice or refer for a child with severe bronchiolitis.

Risk factors for severe disease

- Gestational age less than 37 weeks
- Chronological age at presentation less than 10 weeks
- Chronic lung disease
- Congenital heart disease
- Chronic neurological conditions
- Failure to thrive.
- Trisomy 21
- Post-natal exposure to cigarette smoke
- Breast fed for less than 2 months.

Differential diagnoses

While bronchiolitis is the most common cause of respiratory distress in infants, less common diagnoses, or dual diagnoses must be considered in all children.

Table 5.3. Differential diagnoses

Less common causes of respiratory distress in infants	
Respiratory	• bacterial pneumonia, including pertussis. • aspiration of milk/formula or foreign body • tracheo/bronchomalacia
Other	• congestive cardiac failure • sepsis • intrathoracic mass • allergic reaction

Table 5.4. Initial management (continues on next page)

INITIAL MANAGEMENT The main treatment of bronchiolitis is supportive. This involves ensuring appropriate oxygenation and fluid intake, and minimal handling			
	Mild	**Moderate**	**Severe**
Likelihood of admission	Suitable for discharge Consider risk factors.	Likely admission may be able to be discharged after a period of observation. Management should be discussed with a local senior clinician.	Requires admission and consider need for transfer to an appropriate children's. facility/PICU Threshold for referral is determined by local capacity but should be early
Observations Vital signs (respiratory rate, heart rate, Oxygen saturations, temperature	Adequate assessment in ED prior to discharge (minimum of two recorded measurements or every four hours)	One to two Hourly (not continuous) Once improving and not requiring oxygen for 2 hours discontinue oxygen saturation monitoring	Hourly with continuous cardiorespiratory (including oximetry) monitoring and close nursing observation

Initial management (continued)

	Mild	Moderate	Severe
Hydration/ nutrition	Small frequent feeds	If not feeding adequately (less than 50% over 12 hours), administer NG hydration	If not feeding adequately (less than 50% over 12 hours), or unable to feed, administer NG hydration
Oxygen saturation/oxygen requirement	Nil requirement	Administer oxygen to maintain saturations greater than or equal to 90% Once improving and not requiring oxygen for 2 hours discontinue oxygen saturation monitoring	Administer oxygen to maintain saturations greater than or equal to 90%
Respiratory support		Begin with Nasal prong oxygen High-flow nasal cannula to be used only if nasal prongs oxygen has failed	Consider High-flow nasal cannula or Continuous Positive Airway Pressure
	Mild	**Moderate**	**Severe**
Disposition escalation	Consider further medical review if early in the illness and any risk factors are present or if child develops increasing severity after discharge	Decision to admit should be supported by clinical assessment (including risk factors), social and geographical factors, and phase of illness	Consider escalation if severity does not improve. Consider ICU review/ admission or transfer to local centre with paediatric HDU/ICU capacity if: • Severity does not improve • Persistent desaturations

			• Significant or recurrent apnoea associated with desaturations • Has risk factors
	Mild	**Moderate**	**Severe**
Parental education	Provide advice on the expected course of illness and when to return (worsening symptoms and inability to feed adequately)	Provide advice on the expected course of illness and when to return (worsening symptoms and inability to feed adequately)	Provide advice on the expected course of illness

Oxygen and respiratory support

Administer oxygen for children with saturations persistently below the target oxygen saturations (SpO2) ≥92%

Table 5.5. Low flow oxygen

Oxygen Administration Device	Flow Rate	Fraction of inspired Oxygen FiO2
Nasal Catheter	Neonates: Not Recommended Infant/child: 1-2 L/min	Delivers 40% to the patient
Nasal Prongs	Standard flow 0-1L/min- Neonate 1-2 L/min – Infant 1-4 /min- Child High flow rate Preterm: 1 L/min Term Neonate: 2 L/min Infants: 4 L/min Child: 4-8 L/min	Delivers 35% to the patient Delivers 50%
Mask (Non-Rebreather)	All groups: 10-15 L/min	Delivers 80- 95% oxygen

High flow nasal cannula oxygen (HFNC) Therapy

Consider HFNC therapy in infants with bronchiolitis who are hypoxic (SpO2 <92%) with moderate to severe work of breathing.

- HFNC therapy is **not** recommend for infants without hypoxia.
- Continuous positive airways pressure (CPAP).
- Nasal CPAP therapy for infants with bronchiolitis can also used.

Monitoring

Continuous pulse oximetry is recommended for hypoxic infants or unstable infants receiving oxygen.

Hydration/nutrition

- Give small frequent feeds for infants with mild bronchiolitis
- Consider nasal saline drops prior to the time of feeding
- Suction of the nares may assist feeding in infants with moderate distress.

  Caution – Deep suctioning of the nasopharynx is not recommended as may cause oedema and irritation of the upper airway resulting in increased length of illness.

- Oral Gastric Tube insertion is highly recommended for infants on HFNC. Advantages include:
  - gastric decompression
  - ability to feed without interrupting HFNC.
  - avoid potential for worsening of respiratory symptoms during feeding.
- Oral Gastric Tube or IV hydration is recommended for infants with moderate -severe bronchiolitis who are feeding inadequately (less than 50% over 12 hours)
- if using IV route, isotonic IV fluids (0.9% sodium chloride with glucose, or similar) are recommended.
- the volume of fluids required to maintain hydration is unclear.

Treatments NOT recommended.

- beta 2 agonists (e.g., Salbutamol) regardless of a personal/family history of atopy
- corticosteroids
- adrenaline (nebulised, IM, or IV) except in peri-arrest or arrest situation
- hypertonic saline
- antibiotics
- antivirals
- deep nasal suction
- chest physiotherapy

When to refer

Immediately refer children with bronchiolitis for emergency hospital care if they have any of the following:

- apnoea (observed or reported)
- child looks seriously unwell.
- severe respiratory distress, for example grunting, marked chest recession, or a respiratory rate of over 70 breaths/minute.
- central cyanosis
- persistent oxygen saturation of less than 92% when breathing air.
- difficulty with breastfeeding or inadequate oral fluid intake (50–75% of usual volume, taking account of risk factors and using clinical judgement)
- clinical dehydration.

When to discharge

Consider discharge for the following infants:

- is clinically stable.
- has maintained oxygen saturation over 92% in air for 4 hours, including a period of sleep.
- feeding adequately
- parent/caregiver can safely manage the infant at home (consider time of day, parent/carer comprehension and compliance, access to transport and distance to the local clinic).

Key safety information for looking after a child at home

Provide key safety information for parents and carers to take away for reference for children who will be looked after at home. This should cover how to recognise developing 'red flag' symptoms:

- Worsening work of breathing (for example grunting, nasal flaring, marked chest recession)
- Fluid intake is 50–75% of normal or no wet nappy for 12 hours.
- Apnoea or cyanosis
- Exhaustion (for example, not responding normally to social cues, wakes only with prolonged stimulation).
- That people should not smoke in the child's home because it increases the risk of more severe symptoms in bronchiolitis.
- How to get immediate help from an appropriate professional if any red flag symptoms develop

- Arrangements for follow-up if necessary.

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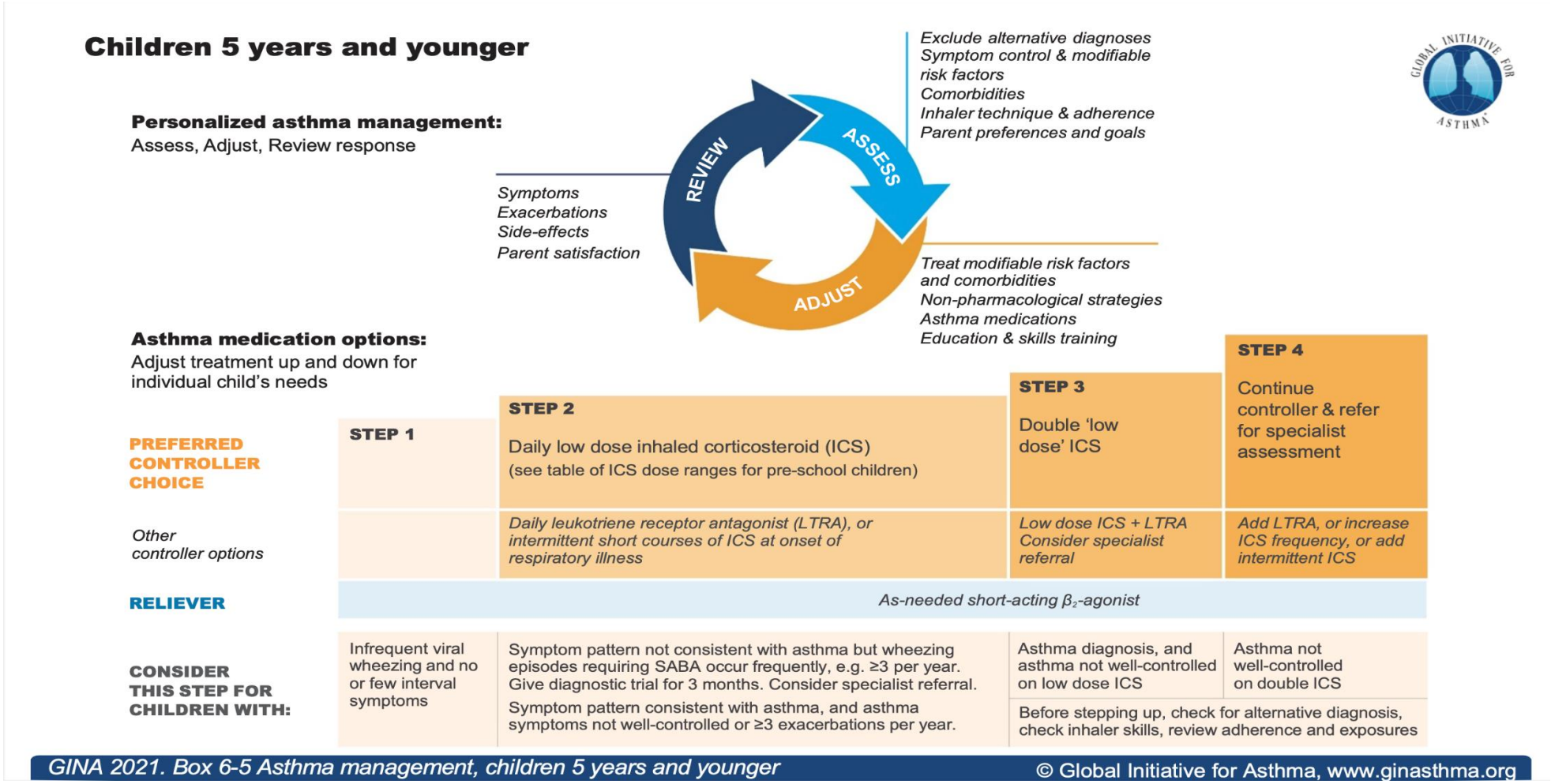
5.6 LONG TERM FOLLOW UP OF CHILDREN WITH ASTHMA

Asthma is a chronic respiratory condition requiring long-term management to maintain control and prevent exacerbations. The latest Global Initiative for Asthma (GINA) guidelines emphasize the importance of regular follow-up in stable asthma patients to assess control, adjust treatment, and monitor for potential complications.

Some definitions:

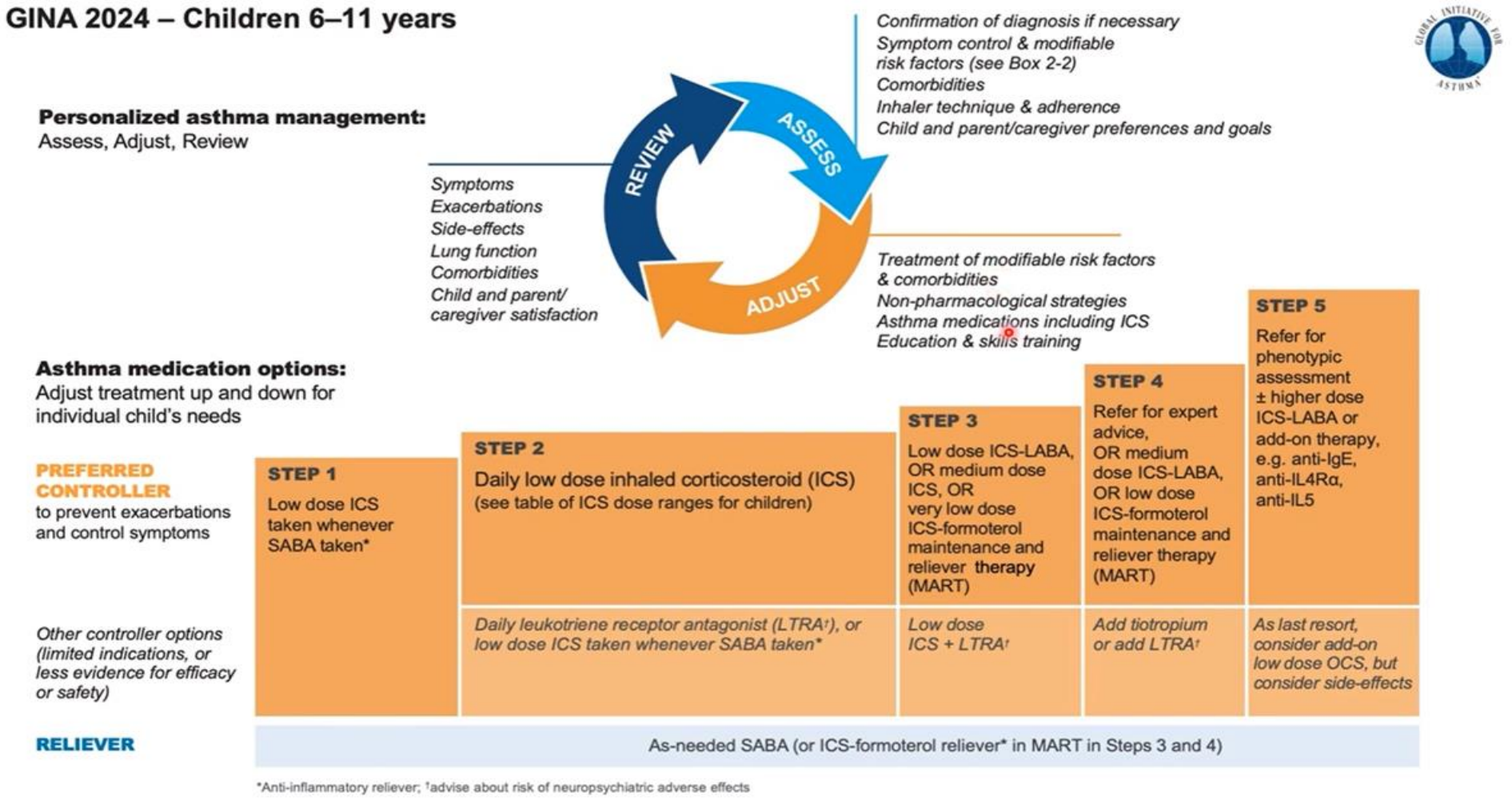
- Maintenance therapy – asthma treatment is that is prescribed for use every day (regularly scheduled)
- Controller- Medication taken to prevent exacerbations of symptoms.
- Reliever- Medication taken as needed for quick relief of symptoms.
- Anti-inflammatory reliever- reliever inhaler that contains both a low dose ICS and a rapid-acting bronchodilator
- Maintenance and reliever therapy- Treatment regimen in which the patient uses an ICS - formoterol inhaler every day (maintenance dose) and also uses the same medication as needed for relief of asthma symptoms (reliever doses)

LONG TERM GINA GUIDELINES



***when suspecting Asthma in a child less than 3 years, please seek advice of Child Specialist**

INITIATION OF TREATMENT

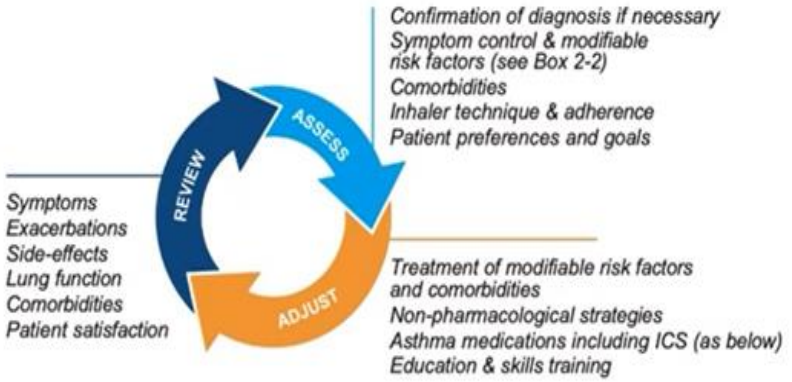


GINA 2024 Box 4-12

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GINA 2024 – Adults & adolescents 12+ years

Personalized asthma management
Assess, Adjust, Review
for individual patient needs



TRACK 1: PREFERRED CONTROLLER and RELIEVER
Using ICS-formoterol as the reliever* reduces the risk of exacerbations compared with using a SABA reliever, and is a simpler regimen

STEPS 1 – 2	STEP 3	STEP 4	STEP 5
As-needed-only low dose ICS-formoterol	Low dose maintenance ICS-formoterol	Medium dose maintenance ICS-formoterol	Add-on LAMA Refer for assessment of phenotype. Consider high dose maintenance ICS-formoterol, ± anti-IgE, anti-IL5/5R, anti-IL4Rα, anti-TSLP

RELIEVER: As-needed low-dose ICS-formoterol*

See GINA severe asthma guide

TRACK 2: Alternative CONTROLLER and RELIEVER
Before considering a regimen with SABA reliever, check if the patient is likely to adhere to daily controller treatment

STEP 1	STEP 2	STEP 3	STEP 4	STEP 5
Take ICS whenever SABA taken*	Low dose maintenance ICS	Low dose maintenance ICS-LABA	Medium/high dose maintenance ICS-LABA	Add-on LAMA Refer for assessment of phenotype. Consider high dose maintenance ICS-LABA, ± anti-IgE, anti-IL5/5R, anti-IL4Rα, anti-TSLP

RELIEVER: As-needed ICS-SABA*, or as-needed SABA

Other controller options (limited indications, or less evidence for efficacy or safety – see text)				
Low dose ICS whenever SABA taken, or daily LTRA†, or add HDM SLIT*	*Medium dose ICS, or add LTRA†, or add HDM SLIT*	*Add LAMA or add LTRA† or add HDM SLIT, or switch to high dose ICS-only*	*Add azithromycin (adults) or add LTRA†. As last resort consider adding low dose OCS but consider side-effects*	

*Anti-inflammatory reliever; †advise about risk of neuropsychiatric adverse effects

GINA 2024 Box 4-6

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5.7 PNEUMONIA

Pneumonia is an important cause of morbidity and mortality in developing countries. Both bacteria and viruses are important causes of pneumonia. Bacteria are the most common cause of pneumonia in HIV-infected children.

It is a clinical diagnosis which includes the following

- Fever above 38 degrees Celsius
- Cough
- Difficulty breathing

The danger signs include;

- Increased respiratory rate of > 60 breaths in an infant and >50 breaths/min in older children
- Severe chest indrawing
- Reduced level of consciousness
- Severe acute malnutrition
- Seizures
- Vomiting and poor feeding

Community-acquired pneumonia

- Pneumonia acquired outside the hospital settings.
- In younger children, viral organisms are the common causative agents.
- Streptococcal pneumoniae is the most likely cause in older children.
- Mycoplasma pneumoniae should be considered in school going children and adolescents.

Clinical presentation

- Fever >38.5^0 C
- Tachypnoea, tachycardia
- Subcostal and intercostal recession
- Crackles
- Abdominal pain (referred pleural pain)

Table 5.6. Clinical Presentation

Age	Cut-offs for fast breathing
< 2 months	60 breaths/min or more
2 – 12 months	50 breaths/min or more
12 Months - 5 Years	40 breaths/min or more

Indications for admission

- Cyanosis, SpO_2 < 92%
- Increased respiratory rate
- Subcostal recession
- Intermittent apnoea, grunting (infants)
- Poor feeding
- Convulsions
- Restlessness or agitation
- Signs of dehydration
- Unconscious or lethargic
- Capillary refill time >3 seconds

Investigations

- Chest x-ray
- FBC, ESR
- Urea, electrolytes (may have hyponatraemia owing to possible SIADH), and creatinine.
- Blood culture
- If able to produce good sputum (send for gene xpert)
- Nasopharyngeal aspirates (send for gene xpert)
- Arterial blood gases (if available)

Management

- General measures
  - Oxygen by nasal cannula or mask
  - IV fluids if required should be < 2/3 of requirements (risk of SIADH in hypoxic children)
  - Antipyretic and analgaesia as indicated.
  - Monitor vital signs.
  - Antibiotics (see table below for disease specific indications)

Table 5.7. Antibiotics therapy

Drugs	Severity
Amoxicillin or Amoxicillin and Clavulanate Amoxicillin 40 -80mg/ kg/Co-Amoxiclav 30-4-mg/kg	Mild /Moderate Pneumonia
0-3 months Benzylpenicillin 50,000 IU/kg/dose every 6 hours + gentamicin 7.5 mg OD or BD (in divided doses **More than 3 months** Benzylpenicillin 50,000 IU/kg/dose every 6 hours + gentamicin 7.5 mg OD or BD (in divided doses If no improvement 24 to 48 hours later, can change to Ceftriaxone (50-80 mg/kg/dose OD) or cefotaxime (50 mg/kg TDS) should be considered Use higher dose in severe infections If no improvement and the infant with HIV infection or exposure, consider adding PCP therapy with high dose IV/PO cotrimoxazole (20 mg/kg/day of trimethoprim) should be included	Severe Pneumonia
In suspected staphylococcus pneumonia, add IV Cloxacillin Macrolides should be considered in school-going children and adolescents who do not improve on 1st line treatment **Erythromycin** **1month – 2 years:** 125 mg QID 2 – 8 years: 250 mg QID 8 – 18 years: 250-500 mg QID **Azithromycin** **6 months-2years:** 10 mg/kg OD 3 – 7 years: 200 mg 8 – 11 Years: 300 mg 12 – 14 years: 400 mg >14 years: 500 mg	

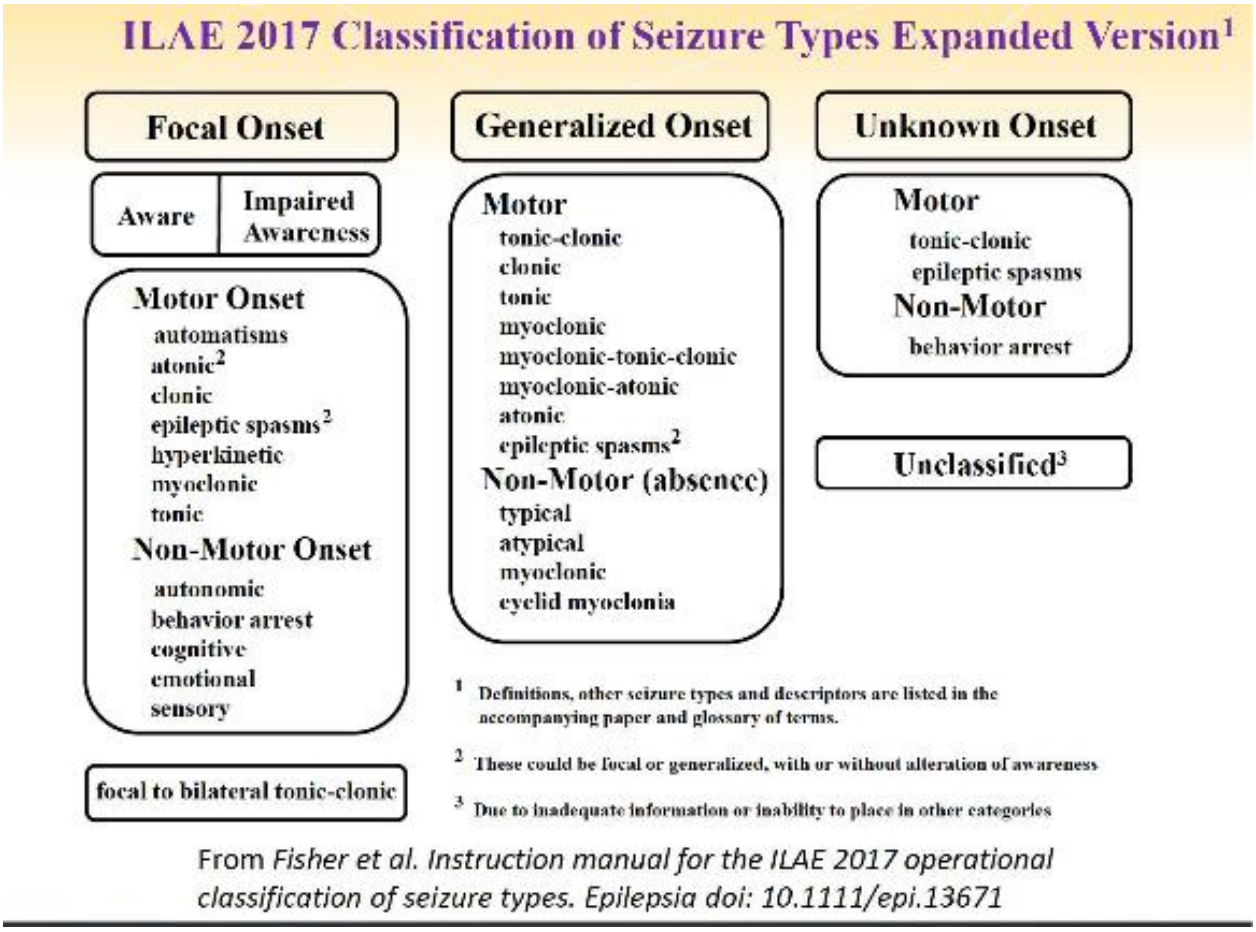
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6.1 SEIZURES

An epileptic seizure is a transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity in the brain.

Types of seizures



From *Fisher et al. Instruction manual for the ILAE 2017 operational classification of seizure types. Epilepsia doi: 10.1111/epi.13671*

FIRST UNPROVOKED SEIZURE

A focal or generalized seizure, including multiple seizures within 24 hours with full recovery of consciousness between seizures, without a clear provoking factor such as head trauma immediately preceding the seizure, CNS infection, or other known acute cause. Also, to be excluded are neonatal seizures (in children <28 days old), status epilepticus, and febrile seizures.

Careful history and Examination

Specifically evaluate for risk factors of epilepsy including:

- Prior seizures
- Family history of epilepsy

- Any concerning historical factors for provoked seizures- including dehydration, vomiting, diarrhoea, toxicology exposure
- abnormality on neurologic exam
- concern for other neurologic disorders

Investigations

- Laboratory testing should be performed based upon individual clinical circumstances.
- Serum glucose, sodium, calcium and magnesium in particular should be considered if concern for electrolyte abnormalities per history
- Toxicology screening should be considered in any paediatric patient if any question of drug exposure or substance abuse
- EEG should be considered in cases of first-time seizure to assess for underlying epileptiform abnormalities and determine risk of future seizures (e.g. diagnosis of epilepsy) **when the study is feasible** to obtain and clinical management is in question. Findings should be interpreted with the clinical history to make a determination if the child has epilepsy. **A normal study does not exclude epilepsy.** EEG testing is **not necessary** to diagnose epilepsy or initiate antiepileptic medication.
- Emergent neuro-imaging should be obtained in any child with
    - persistent unresponsiveness
    - Lack of return to baseline within a few hours
    - Persistent Todd's paresis (transient weakness or paralysis of part or all of the body after a focal-onset seizure) that is not resolving within a few hours.

As timing is essential, the recommended study would be CT unless MRI is feasible within hours.

If the child is back to baseline, a non-urgent MRI/CTI (depending on availability and clinical suspicion- e.g. CT should be performed to rule out neuro-cysticercosis) should be considered for any child who has

- A focal seizure
- Focal EEG findings
- Significant cognitive or motor impairment without known aetiology
- Other unexplained abnormality on neurological exam particularly if unresponsiveness to AED treatment or progressively worsening development is noted.

A diagnosis of epilepsy should be made if:

- A child with 1 unprovoked seizure presents with
    - an abnormal EEG
    - structural abnormality on neuro-imaging

- clear neurologic abnormality on examination suggestive of an underlying neurologic abnormality as the risk of seizure recurrence within a 10-year period is estimated as being higher than 60%.
- A child has 2 or more unprovoked seizures greater than 24 hours apart.

If epilepsy is diagnosed, consideration for antiepileptic treatment should be made based upon the clinical circumstances,

All children should be followed up after the first unprovoked seizure to see if there have been any recurrent events and reassess for risk of epilepsy, even if EEG and other evaluations were normal.

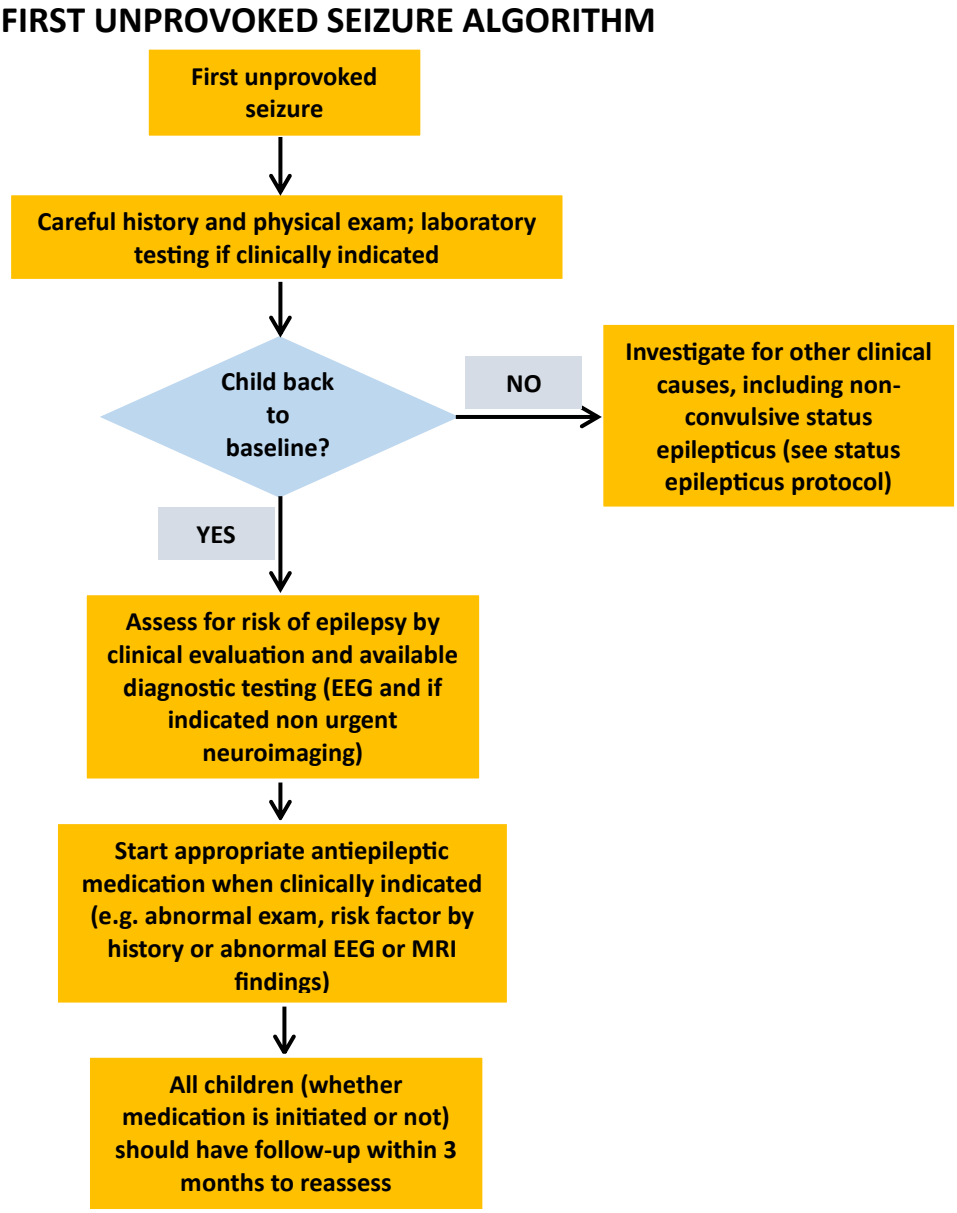


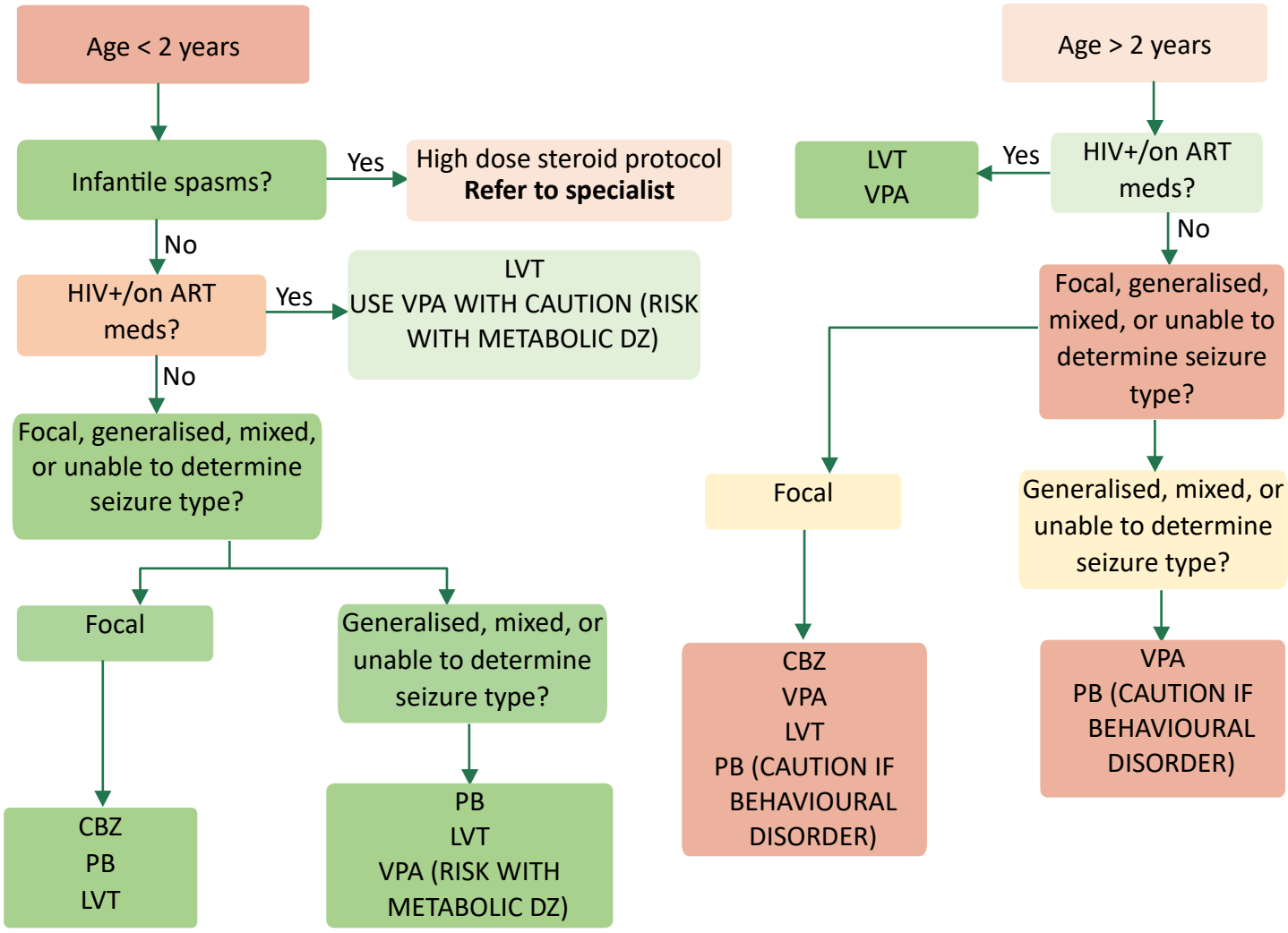
Fig 6. Treatment of unprovoked seizures

EPILEPSY

Epilepsy is defined as a chronic disorder of the brain characterized by an enduring predisposition to generate epileptic seizures, and by the neurobiological, cognitive, psychological, and social consequences of this condition. The definition of epilepsy requires the occurrence of at least one epileptic seizure. The definition of epilepsy is usually practically applied as:

- Having two unprovoked seizures >24 h apart.
- A patient with a first unprovoked seizure after a remote brain insults (such as a stroke, CNS infection, trauma) with a recurrence risk of further seizures comparable to the general recurrence (greater than 60%) risk for further seizures after two unprovoked seizures over the next ten years.
- A diagnosis of an Epilepsy syndrome

MANAGEMENT OF EPILEPSY – GUIDELINES FOR FIRST-LINE ANTISEIZURE MEDICATION



CBZ-CARBAMAZEPINE, PB-PHENOBARBITAL, VPA- VALPROIC ACID, PHT- PHENYTOIN, LVT-LEVETIRACETAM

Fig 6.1. Guidelines for first-line antiseizure medication

Table 6. Anti- seizure medication dosing chart

	CARBAZEPINE	PHENOBARBITAL	PHENYTOIN	SODIUMVALPROATE
STARTING DOSE TITRATION	**5mg/kg/day, in 2-3 divided doses 2 per day, increasing by 2-10mg/kg/day weekly in divided doses to maintenance dose.**	**-3mg/kg/day in 2 doses daily, increase weekly by 1-2mg/kg/day depending on tolerance**	3-4mg/kg/day in 2 divided doses, every 3-4 weeks increase by 5mg/kg/day in two divided doses to goal 3-8mg/kg/day (max300mg/day)	**10-15mg/kg/day, in 2-3 doses per day, increasing weekly by 10-15/kg/day to goal**
MAINTENANCE DOSE	10 – 30mg/kg/day	2-6mg/kg/day	3-8mg/kg/day (max300mg/day)	15-40mg/kg/day
AFFECTS OF OTHERRAEDS	+VPA = ↑↓CBZ levels +PB or PHT = ↓CBZ levels	+CBZ = ↓PB levels +PHT = ↑PB levels	Complicated-CBZ & PB can ↑OR↓CBZ VPA can ↑free PHT	+CBZ, PB or PHT = ↓VPA levels
RARE BUT SERIOUS SIDE EFFECTS	• Severe skin rash (Steven's Johnson Syndrome *; toxic epidermal necrolysis*) • Bone Marrow Depression	• Severe skin rash (Steven's Johnson Syndrome *) • Bone Marrow Depression • Liver Failure	• Anaemia/hematolo gic adnormali \es • Hypersensi \vity react\ons, including severe skin rash (Stevens Johnson*) Hepa \\s	• Drowsiness/confusion (valproate-induced hyperammonemic encephalopathy – sign of toxicity)
COMMON SIDE EFFECTS	• Drowsiness • Trouble walking • Nausea	• Drowsiness • Hyperac\vity in children	• Nausea, vomi\ng, cons\pa\on • Tremor • Drowsiness • Ataxia and slurred speech • Motor twitching • Mental confusion	• Lethargy • Seda\on • Tremor • Nausea, diarrhoea • Weight gain • Transient hair loss (regrowth normally begins in 6months) • Impaired hepa\c func\on
SPECIAL NOTES	- Good for focal seizures - Can worsen some generalized seizures! - Can reduce ART med concentra\ons (efavirenz, nevirapine, & zidovudine)	- Avoid in children with behavioural disorder if possible - Can reduce ART med concentra\ons (efavirenz, & zidovudine)	- Many side effects, least ideal for long term use - Can reduce ART med concentra\ons (efavirenz, nevirapine, & zidovudine)	- If using liquid form dose 3x/day! - First line if on ART meds, fewest interac\ons (may min increase zidovudine)

Psychosocial counselling for epilepsy

- Counselling the family is an important part of managing epilepsy as it
  - encourages medication adherence
  - reduces community stigma
- It is important to provide information to the family on
  - what epilepsy is
  - how medication can help
  - what to expect in life
  - how to stay safe eg from sources of heat that can cause burns
- Instructions on how to manage seizures at home
  - Lay the person down on the side with the head turned to help to breathing
  - DO NOT PUT ANYTHING IN MOUTH OR RESTRAIN THE PERSON
  - Make sure the person is breathing properly
  - Stay with the person until the convulsion stops and they wake up
  - Sometimes people with epilepsy know that a convulsion is coming. They should lie down somewhere safe if they have that feeling.
  - Remind them that epilepsy is not contagious. They will not catch convulsions by helping
  - Remind them to get help if there is concern for breathing such as when seizures last more than 5 minutes, or the child is not waking after the seizure

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6.2 FEBRILE SEIZURES

Febrile seizures are seizures that occur in **febrile** children between the ages of 6 and 60 months who do not have an intracranial infection, metabolic disturbance, or history of afebrile seizures or significant neurological impairment.

Fever may not be detected before the seizure, but it must be present at least in the immediate post-acute period and may be the only symptom of presenting illness.

- Simple febrile seizure
  - Usually generalized tonic-clonic
  - Lasting for a maximum of 15 min
  - Not recurrent within a 24-hour period.
- Complex febrile seizure
  - More prolonged (>15 min)
  - Maybe focal
  - With/without associated post-ictal palsy (Todds paresis)
  - And/or recurs within 24 hours
- Febrile seizures frequently occur in the contest of:
  - Viral URTIs
  - HHV6 infection
  - Acute otitis media
  - Malaria
  - Genetic predisposition, e.g. autosomal inheritance pattern in some families

Clinical features

- Core temperature to ≥ 38^{o}c
- Generalized tonic- clonic, lasting up to 15 min
- Occurs once in 24 hrs. (usually on first day of fever)
- For complex type – see above
- Recurrence is possible
- The vast majority are harmless, prognosis is good

Complete general and neurological examination

- Stabilize the child
- If the child arrives seizing to the facility, treat as status epilepticus (see status epilepticus protocol)
- Check for focus of infections
- Check for possible signs and symptoms of meningitis (see protocol for meningitis).

- **NB:** Lumbar puncture is not required in simple febrile seizures. It should be considered in the following scenarios:
  - Children under 18 months of age (other signs and symptoms of meningitis might not be present or be very subtle).
  - Complex febrile seizures (should only be considered after excluding need for neuro-imaging)
  - Children who have received antimicrobials prior to assessment
  - Children not immunised against Haemophilus and Pneumococcal species

Investigations

- Investigation should be done on individual basis to determine the cause of fever - MPS, FBC, nasopharyngeal swab, urine m/c/s, if no obvious focus is found.
- EEG and Neuroimaging are not routinely required for febrile seizures
- The work-up of children with complex febrile seizures needs to be individualized. This can include EEG and neuro-imaging where there is a clear focality concerning for possible CNS lesion.

Treatment

- Abort seizure - Diazepam IV 0.2 – 0.5 mg/kg slowly (can also be given rectally 0.5 mg/kg) if duration more than 3-5 min.
- Supportive therapy – tepid sponging, nurse in semi-prone position, ensure adequate airway.
- Assess for the cause of fever and treat appropriately when required e.g. antimalarials, antibiotics.
- Anti-seizure medications are not indicated.
- Antipyretic should be used at the same dose and intervals used for lowering fever during a febrile illness at optimal dosing:
  - Paracetamol at 10 to 15 mg/ kg/dose every 4-6 hours (max 5 doses/day)
  - Ibuprofen: 20-30 mg/kg/day in 3 doses.
- Families should be provided counselling on risk or recurrence of febrile seizures (25-40%), risk of epilepsy (10% if complex febrile seizures, similar to general population if simple febrile seizures), and seizure first aid.

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6.3 PAEDIATRIC STROKE

A focal neurological deficit with an underlying vascular pathology is defined as:

Table 6. Paediatric Stroke

Stroke	lasting > 24h
Transient ischaemic attack (TIA)	lasting < 24h
Reversible ischaemic neurological deficit (RIND)	lasting> 24 h but with full recovery
"Stroke-like episode"	Focal neurological deficit lasting >24 hr with no obvious vascular pathology, e.g. Brain tumour, brain abscess. Rare in childhood (1-3 / 100,000 per year)

Paediatric stroke can be **ischaemic** (vessel spasm, stenosis, dissection or vessel occlusion by thrombosis or embolism) or **haemorrhagic** and occur at any age;

- Neonatal stroke:28 weeks gestation- 28 days post-natal life
- Childhood stroke: Greater than 28 days -18 years

Note: SCD is the most common cause of stroke in Zambian children.

Table 6.1. Ischaemia (Arterial or Venous)

Embolism	Cyanotic CHD, Endocarditis
Large vessel stenosis	SCD, Varicella, HIV, Homocystinuria
Vessel spasm	CNS infections
Thrombosis	SCD, Severe dehydration (cerebro-venous-sinus thrombosis), Malignancies (Leukaemia) Thrombocytosis, CNS infections, hyper coagulable states i.e. Protein S or C deficiency, Antithrombin III deficiency, Lupus antibodies, Factor V Leiden, Renal disorders i.e. Nephrotic syndrome
Vessel dissection	Trauma, Congenital Heart Disease
Cerebral Arteriopathy	Moyamoya, Focal/transient arteriopathy, Post varicella
Drugs	i.e. Asparaginase
Syndromes	Williams syndrome, Downs syndrome

Table 6.2. Haemorrhage

Low platelets	Idiopathic thrombocytopenic purpura
Bleeding disorder	Coagulation factor deficiencies i.e. Hemophilia
Vessel disorder	A-V malformation, A-V fistulas, cerebral aneurysm, Cavernous malformations
Trauma	

Clinical features

- Hemiparesis
- Hemi-sensory signs
- Visual field defects
- Seizures (common in neonates)
- Deterioration in level of consciousness (seen in progression of bleed)
- Headache
- Nausea and vomiting

Investigations

- FBC, differential count, ESR, random blood sugar, sickling test, clotting defects (e.g. protein C & S deficiencies, Antithrombin III deficiency)
- Clotting profile
- Electrolytes initially, then daily until stable
- Blood, urine and CSF cultures if febrile (remember to withhold LP if evidence of ↑ICP)
- After stabilization, Neuro-imaging (preferably MRI) and angiography

Table 6.3. Investigations

MRI brain scan	Outline area affected (thrombosis, bleed, abscess, tumour, etc.
CT scan	If MRI unavailable (to exclude haemorrhage)
MRA /MRV	Vascular outline to R/O Arteriopathy, Vessel anomalies and Venous thrombosis
ECG and ECHO	cardiac anomaly or arrhythmia

Investigate for a possible thrombophilia even with an obvious cause like trauma as these two conditions may co-exist.

MANAGEMENT

Ischemic Stroke

- Initial attention to ABCDE
- Consider admission to PICU for 1st 24 hours or until stable
- Monitor vital signs (BP, Pulse, Respiratory rate), input & output, level of consciousness
- Allow permissive hypertension- Recommended BP goal between the 50th-95th percentile for age and height, **with permissive hypertension** up to 20% above the 95th percentile
    - Persistent, significant hypertension should be treated with caution to lower blood pressure by approximately 25% over 24 hours.
- Nurse head flat for 24 hours (unless signs of raised ICP)
- Fluid management- isotonic IVF for at least 24 hours
- Keep nil by mouth
- Maintain normoglycaemia
- Control fever-Maintain normothermia
- O_2 to by face mask if indicated (keep SpO2 greater than 92%)
- Treat acute seizures (no role for AED's for seizure prophylaxis)
- Manage ↑ICP if present (see management of ICP protocol)
- Control systemic hypertension
- If high index of suspicion for CNS infection give Antibiotics (Cefotaxime 100 mg/kg q 6 hr. or Ceftriaxone 100 mg/kg per day)
- Other management is dependent on cause, e.g. exchange transfusion/ blood transfusion in SCD, anticoagulants in prothrombotic coagulopathy
- Anti-viral treatment with Acyclovir has been recommended for varicella zoster virus vasculopathy with **virologic confirmation of active central nervous system VZV infection**

In neonates, treatment for prevention of a 2nd stroke is often not required because the risk of recurrence is low. In children however, the recurrence risk is higher and long-term therapy with low dose aspirin is often needed (1-5mg/kg/day nocte). Aspirin is recommended for a minimum of two years.

Hyper transfusion therapy and hydroxyurea in patients with SCD for secondary prevention of Stroke

Refer to specialist for immunosuppressant therapy for vasculitis.

Haemorrhagic stroke

- Stabilize patient ABCDE
- Admit in the PICU
- Monitor for new or worsening neurological symptoms-extension of haematoma, herniation, hydrocephalous
- Isotonic IVF for at least 24 hours
- Maintain normothermia and normoglycemia

- Maintain normal blood pressure
- Manage raised ICP, if present
- Manage acute seizures
- Manage the cause
    - Replace clotting factors, platelet transfusion for thrombocytopenia
    - MRI/MRA to look for vascular malformation if possible
    - Neurosurgery sometimes can embolize/resect malformation to prevent future strokes

Rehabilitation

A plan for rehabilitation should be discussed with the parents. Speech, occupational and physical therapies, psychological services, special education should be offered.
High risk of future Epilepsy.

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6.4 NEURODEVELOPMENT DISORDERS

Neurodevelopmental disorders are multifaceted conditions characterized by impairments in cognition, communication, behaviour and/or motor skills resulting from abnormal brain development.

They include conditions such as intellectual disability, communication disorders, autism spectrum disorder (ASD), attention deficit/hyperactivity disorders (ADHD) and schizophrenia.

AUTISM SPECTRUM DISORDER

1. Definition

Autism spectrum disorder (ASD) is a biologically based neurodevelopmental disorder characterized by deficits in social communication and social interaction and restricted, repetitive patterns of behavior, interests, and activities.

2. Screening tools

M-CHAT (for children between 16 and 30 months)

DSM V for children over 2 years old.

Table 6.4. Early symptoms and signs of autism

Caregiver concerns about deficits in social skills
Caregiver concerns about deficits in language skills or behavior
Caregiver concerns about frequent tantrums or intolerance to change
Delayed language and social/communication skills: • Lack of orientation to name by age 12 months • Lack of pointing or gesturing to indicate interest (eg, by pointing to an airplane flying over) by age 14 months Lack of pretend play (eg, "feeding" a doll) by age 18 months
Avoiding eye contact or wanting to be alone
Having trouble understanding other people's feelings or talking about their own feelings
Repeating words or phrases over and over (echolalia)
Giving unrelated answers to questions
Getting upset by minor changes
Having obsessive interests
Flapping their hands, rocking their body, or spinning in circles
Having unusual reactions to the way things sound, smell, taste, look, or feel

3. Diagnosis

The diagnosis of Autism is arrived at by history taking and the use of various screening tools which have variability in sensitivity and specificity. The recommended screening tools are the DSM V-TR and ICD 11. The symptoms have to be present currently or in the past to make the diagnosis.

DSMV TR

The criteria in the DSM-5 for diagnosing ASD include **3 listed deficits from A and 2 or more from B.**

A. Social communication and social interactions. Clinicians must be sure that these characteristics are not due to developmental delay alone. **To be diagnosed with ASD, an individual must meet all three of the following criteria:**

1. Difficulties in social emotional reciprocity, including trouble with social approach, back and forth conversation, sharing interests with others, and expressing/understanding emotions.
2. Difficulties in nonverbal communication used for social interaction including abnormal eye-contact and body language and difficulty with understanding the use of nonverbal communication like facial expressions or gestures for communication.
3. Deficits in developing and maintaining relationships with other people (other than with caregivers), including lack of interest in others, difficulties responding to different social contexts, and difficulties in sharing imaginative play with others.

B. The criteria in the DSM-5 also include demonstrating at least 2 of the following 4 **restricted and repetitive behavior, interests, or activities:**

1. Stereotyped speech, repetitive motor movements, echolalia (repeating words or phrases, sometimes from television shows or from other people), and repetitive use of objects or abnormal phrases.
2. Rigid adherence to routines, ritualized patterns of verbal or nonverbal behaviors, and extreme resistance to change (such as insistence on taking the same route to school, eating the same food because of colour or texture, repeating the same questions); the individual may become greatly distressed at small changes in these routines.
3. Highly restricted interests with abnormal intensity or focus, such as a strong attachment to unusual objects (bottle caps, stones, fascination with animals) or obsessions with certain interests, such as moving objects, train schedules.
4. Increased or decreased reactivity to sensory input or unusual interest in sensory aspects of the environment, such as; not reacting to pain, strong dislike to specific sounds excessive touching or smelling objects, fascination with spinning objects.

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6.5 ATTENTION DEFICIT HYPERACTIVE DISORDER

Attention deficit hyperactivity disorder (ADHD) is characterized by a persistent pattern (at least 6 months or more) of inattention (inability to concentrate or finish tasks) and/or hyperactivity-impulsivity that has a direct negative impact on academic, occupational, or social functioning.

2. Screening tools

The diagnosis of ADHD is arrived at by thorough history taking and the use of various screening tools which have variability in sensitivity and specificity. The recommended screening tools are the DSM V-TR and ICD 11. The symptoms have to be present currently or in the past to make the diagnosis. There is evidence of significant inattention and/or hyperactivity-impulsivity symptoms prior to age 12, typically by early to mid-childhood, though some individuals may first come to clinical attention later. The presentation of ADHD in a given patient can change from one to another over time.

2.3. DSMV TR

Depending upon the predominant symptoms, ADHD can be categorized into one of the **three presentations** listed below:

A. Predominantly Inattentive

1. DSM-5-TR criteria require ≥6 symptoms of **inattention** for children <17 years OR ≥5 symptoms for adolescents ≥17 years and adults **and** <6 symptoms of hyperactivity-impulsivity

Inattention

- Makes careless mistakes in schoolwork or activities. Or work is inaccurate.
- Has difficulty sustaining attention in tasks or play activities (e.g., has difficulty remaining focused during lessons, conversations, or lengthy reading)
- Does not seem to listen when spoken to directly (e.g. mind seems elsewhere, even in the absence of any obvious distraction).
- Does not follow through on instructions and fails to finish schoolwork, chores, or duties (e.g. starts tasks but quickly loses focus and is easily sidetracked).
- Has difficulty organizing tasks and activities (e.g. difficulty managing sequential tasks; messy.
- Avoids, dislikes, or is reluctant to engage in tasks that require sustained mental effort (e.g. schoolwork or homework; for older adolescents and adults, preparing reports, completing forms, reviewing lengthy papers).
- Loses things necessary for tasks or activities (e.g., school materials, pencils, books, tools, wallets, keys, paperwork, eyeglasses, mobile telephones).
- Is easily distracted by extraneous stimuli (for older adolescents and adults, may include unrelated thoughts).
- Is forgetful in daily activities (e.g. doing chores, running errands; for older adolescents and adults, returning calls, paying bills, keeping appointments).

B. Predominantly Impulsive/Hyperactive

a. 6 symptoms of for hyperactivity-impulsivity children <17 years

OR

b. ≥5 symptoms for adolescents ≥17 years and adults

and <6 symptoms of inattention

C. Combined A and B

4. Referral

Evaluation by a paediatric specialist (e.g. a psychologist, psychiatrist, neurologist, educational specialist, or developmental-behavioral diagnoses are of concern or complicate the diagnosis of ADHD. The following are possible reasons for referral to specialist.

1. Intellectual disability (characterized by limitations in self-help skills, academics and immature social interaction)
2. Developmental disorder (e.g. speech or motor delay)
3. Learning disorder
4. Visual or hearing impairment
5. History of abuse or exposure to one or more adverse childhood experiences
6. Severe aggression
7. Seizure disorder
8. Coexisting learning and/or emotional problems
9. Chronic illness that requires treatment with a medication that interferes with learning
10. Children who continue to have problems in functioning despite treatment

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2. Centers for Disease Control and Prevention. Signs of autism spectrum disorder. Available from: https://www.cdc.gov/ncbddd/autism/signs.htm.

6.6 HEADACHE

Though children are often implicated as a cause of headache, it is less recognized that there are also real sufferers of headache. Headache is broadly classified into primary (headache without an underlying cause) and secondary (headache due to identifiable cause).

Clinical Features (Primary Headache)

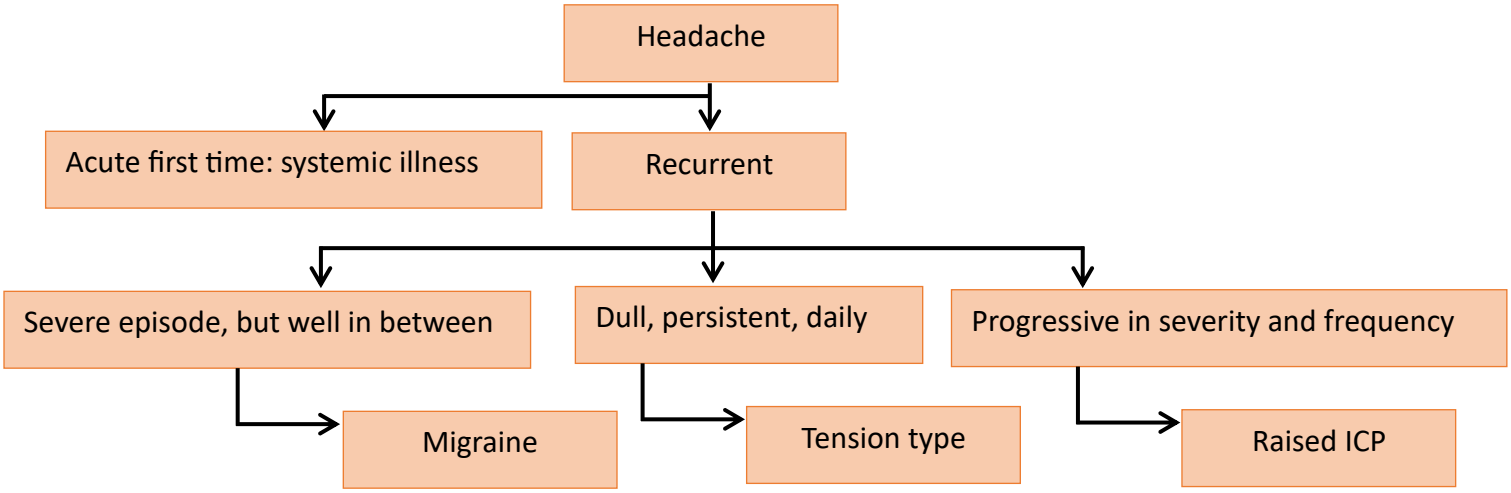


Fig 6.1. Clinical features of headache.

CLINICAL PRIMARY HEADACHE SUMMARIES

1. Migraine Headache

The headache of migraine is severe. It lasts 1-3 hours and sometimes for up to 3 days. It may or may not be unilateral.

Symptoms:

- Nausea and /or vomiting,
- Photophobia, phonophobia, strong smells.
- During episodes prefers dark quiet spaces
- Positive family history of headache
- Perfectly well in between episodes.
- Relieved by sleep.
- May or may not have aura
- It is usually relieved after a deep sleep.

2. Tension Headache

This headache is dull and continuous pain with less severity compared to migraine. Can continue for hours to days. May disappear on Sundays and holidays, it does not interfere with daily activities. It is more common near adolescence. Underlying depression and anxiety are common.

CLINICAL SECONDARY HEADACHE SUMMARIES

1. Raised intracranial pressure

Headaches due to intracranial space occupying lesions, e.g. tumor, hydrocephalus or chronic meningitis increase progressively in severity and frequency. They may wake the child from sleep. Associated symptoms include vomiting, blurred vision, seizures, loss of consciousness and leg weakness.

2. Sinusitis

The headache is localized to forehead, maxilla or periorbital region.
Maybe worse when laying down, and better after standing for a while.

3. Ocular Disorders

Hypermetropia and astigmatism (rarely myopia) may induce a dull aching headache after long use of the eyes in close work. Location is usually periorbital.

Key examination points

1. In an acute onset headache especially with fever:

- Test for meningeal signs.
- Do a careful ear, nose and throat examination.

2. In recurrent headaches;

- concerning in very young children under the age of 5, and must be thoroughly evaluated.
- take the blood pressure. Hypertension can be due to raised intracranial pressure.
- Do a careful fundus examination for papilledema and bleeds or refer /consult the ophthalmologists.
- Look for lateral rectus palsies
- Cerebellar signs which may signal raised intracranial pressure.
- Focal neurological deficit may require neuroimaging.

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1. American Academy of Neurology. Practice parameter: evaluation of children and adolescents with recurrent headaches. Neurology. 2002;59:490-8.
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7.1 URINARY TRACT INFECTION

Urinary tract infections (UTIs) are among the most common bacterial infections in children. UTIs are usually caused when bacteria (usually bowel flora) invade and ascend the urinary tract from the urethra and into the bladder.

Classification

UTIs are classified according to the site, severity, episode, symptoms, and complicating factors. Site and severity are the most important.

CLASSIFICATION ACCORDING TO SITE

i. **Cystitis (Lower UTI)**
is an inflammatory condition of the bladder mucosa. Symptoms include dysuria, frequency, urgency, enuresis, haematuria, suprapubic pain, and malodorous urine.

ii. **Epididymitis**
which is an inflammatory condition of the epididymis can be the presenting symptom of lower urinary tract infection. Symptoms include pain and swelling of the hemi-scrotum.

iii. **Pyelonephritis (Upper urinary tract infection)**
is a diffuse pyogenic infection of the renal pelvis and parenchyma. Symptoms include fever, chills, and flank pain, and could be as severe as septic shock/toxaemia. Pyelonephritis can lead to renal scarring, which can result in loss of kidney function and long-term sequelae such as hypertension and chronic kidney disease.

Classification according to severity

i. A UTI is classified as mild when children are experiencing mild symptoms and can take fluids and oral medication and is usually due to a lower urinary tract infection with the infection localized to the bladder.

ii. Pyelonephritis is an upper UTI in which the bacteria and subsequent inflammatory response further ascend to the ureters and kidneys. Patients usually present with more serious symptoms such as persistent vomiting, dehydration, or fever >39^{o}C and this is classified as a severe UTI.

Causes

Colonic bacteria are the typical causes:

- Escherichia coli (54% to 67% of UTIs in children).
- Klebsiella (6%-7%),
- Proteus (5%-12%),
- Enterococcus (3%-5%)
- Pseudomonas (2%-6%) are other common causative organisms.

Clinical features

Infants and young children usually present late and with nonspecific signs and symptoms such as fever and irritability to express symptoms or localise the pain. Neonates and infants may present with fever, poor feeding, haematuria, and malodorous urine.

Older children can usually localise early symptoms of UTI and therefore present earlier in the clinical course. Symptoms include:

- Fever, Dysuria, Urinary frequency, Suprapubic discomfort, Flank pain, Costovertebral pain, Abdominal pain.

DIAGNOSTIC EVALUATION

i. Medical history and clinical evaluation

History should include the question of first or recurrent infections, congenital abnormalities, possible malformations of the urinary tract, prior operations, family history and the presence of bowel or voiding dysfunction.

ii. Urine sampling

- In neonates, infants and non-toilet trained children there are four main methods to collect urine:

Table 7. Diagnosis Evaluation on Urinary Tract Infections (UTIs)

Method	Risk of Contamination	Test to be done on the urine sample
Plastic bag attached to the cleaned genitalia	50-60%	Dipstick Urinalysis
*Clean-catch urine (CCU) collection	26%	Dipstick Urinalysis
Transurethral bladder catheterisation	10%	Dipstick Urinalysis and Urine M/C/S
Suprapubic bladder aspiration (is invasive but it is the gold standard)	1%	M/C/S

**Careful cleaning of the genitalia, can reduce contamination*

- In toilet-trained children, the use of clean catch urine, especially midstream, after carefully cleaning of the external genitalia, is an acceptable technique for obtaining urine for screening and urine cultures.

iii. Investigations

- Urinalysis
  - A positive nitrite test makes UTI very likely, but the test maybe falsely negative because nitrite needs about 2-4 hours to accumulate in the bladder for it to be detected.

- The leukocyte esterase test is an indirect measure of pyuria.

- Urine microscopy
  - A microscopic urinalysis finding of 10 white blood cell per microliter in uncentrifuged urine specimen is reported to be a more sensitive indicator.
- Urine culture
  - For children who are not toilet trained, only **urethral catheterisation and SPA** are considered to be reliable methods for specimen collection for the purpose of culture.

Table 7.1. Investigation on Urinary Tract Infections (UTIs)

Method of Urine Collection	Significant Growth for single Uropathagen	Significant growth if second uropathogen present	Contamination
Clean-voided sample	≥100,000 CFU/mL	≥100,000 CFU/mL of one uropathogen and <50,000 CFU/mL of a second uropathogen	a second uropathogen with ≥50,000 CFU/mL or growth of >2 organisms
Transurethral Catheterization sample	≥50,000 CFU/mL	≥50,000CFU/mL of one uropathogen and <10,000 CFU/mL of a second uropathogen	A second uropathogen with ≥10,000 CFU/mL or growth of >2 organisms
Suprapubic sample	Any Growth is significant		

- Imaging
  - Ultrasound: Kidney Ureter and bladder (KUB) ultrasound within 24hrs is advised in infants with febrile UTI to exclude obstruction of the upper and lower urinary tract.
  - A pre and post void KUB ultrasound should be performed in toilet trained children with special attention to the post-void residual urine in toilet trained.
  - Other special investigations can be performed at secondary and tertiary level hospitals include VCUG or MCUG, and DMSA.

Treatment

Start empiric treatment with Quinolones but dependent on local susceptibility patterns then change according to culture results.

- Oral antibiotics 10 to 14 days or
- IV antibiotics for 3 days, then 10 days oral antibiotics.

ALGORITHM ON EVALUATION AND MANAGEMENT OF UTIS

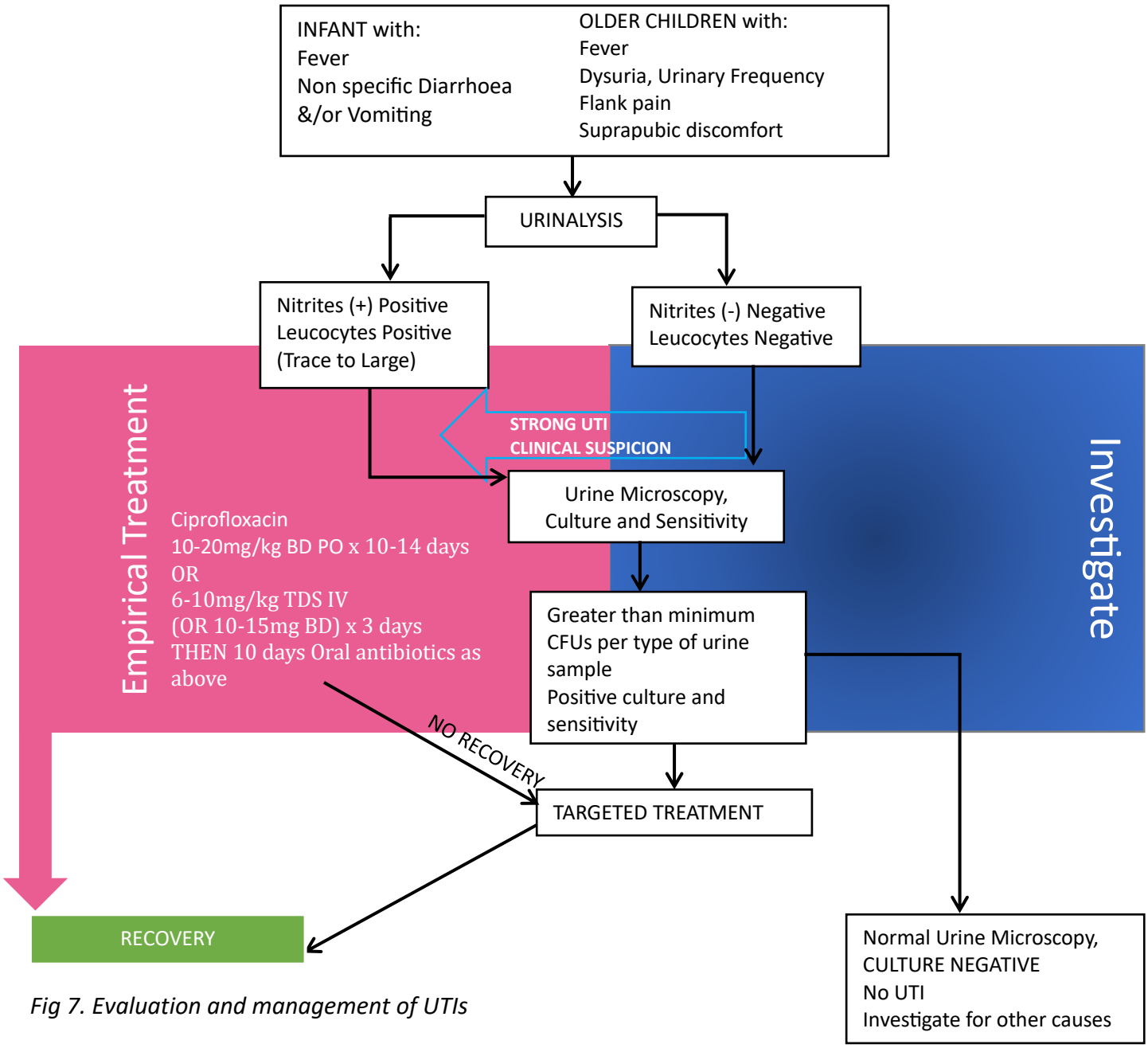


Fig 7. Evaluation and management of UTIs

Table 7.2. Alternative drugs for empirical management of UTIs

Complicated	Uncomplicated
1. Ciprofloxacin 10-15mg/kg BD IV 2. Ceftriaxone 50-80mg/kg 0D IV 3. Cefotaxime 50mg/kg 6-8 hourly IV ***IV drugs to be given for 3 days followed by oral formulations for 10 days***.	1. Ciprofloxacin 10-20mg/kg BD PO 2. Cefixime 8mg/kg OD 3. Nitrofurantoin 5-7 mg/kg/day PO divided q6hr 4. Cefpodoxime 5mg/kg PO 12hourly 5. Nalidixic acid 5-7mg/kg divided 6hourly PO ****Antibiotic therapy to be given for 10-14 days**

Once UTI diagnosis has been confirmed, evaluation for risk factors and complications is warranted and the following algorithm should be followed:

APPROACH TO A FIRST FEBRILE UTI

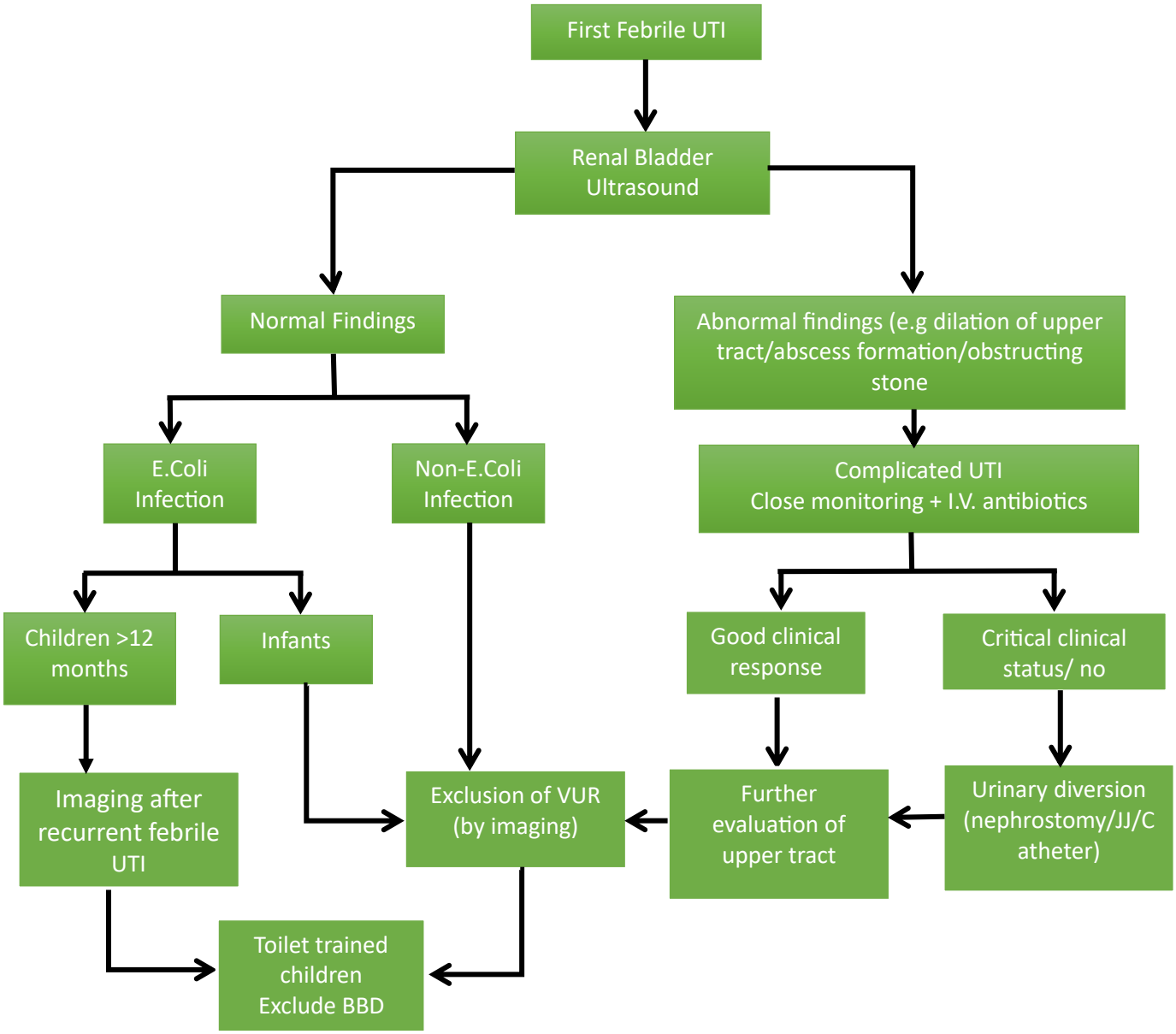


Fig 7.1. Once anatomical anomalies have been detected, refer patient to a Urologist and/or nephrologist for further evaluation and management

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3. Zhu B, Liu Y, Wang H, et al. Clinical guidelines of UTIs in children: quality appraisal with AGREE II and recommendations analysis. BMJ Open. 2022;12:e057736. doi:10.1136/bmjopen-2021-057736.

7.2 HAEMATURIA

Haematuria is defined as the presence of five or more RBCs per high-power (40x) field in three consecutive fresh, centrifuged specimens. Confirmation of haematuria is critical. A positive urine dipstick test may result from myoglobinuria or haemoglobinuria, in which the urine often is discoloured, but no RBCs are noted on microscopic evaluation.

Table 7.2. Haematuria

Glomerular Versus Extraglomerular Haematuria		
Factor	**Glomerular**	**Extraglomerular**
Colour	tea-or cola-coloured, red	Red or pink
RBC Morphology	Dysmorphic	Normal
Casts	RBC, WBC	None
Clots	Absent	Present (+/-)
Proteinuria	≥2+	

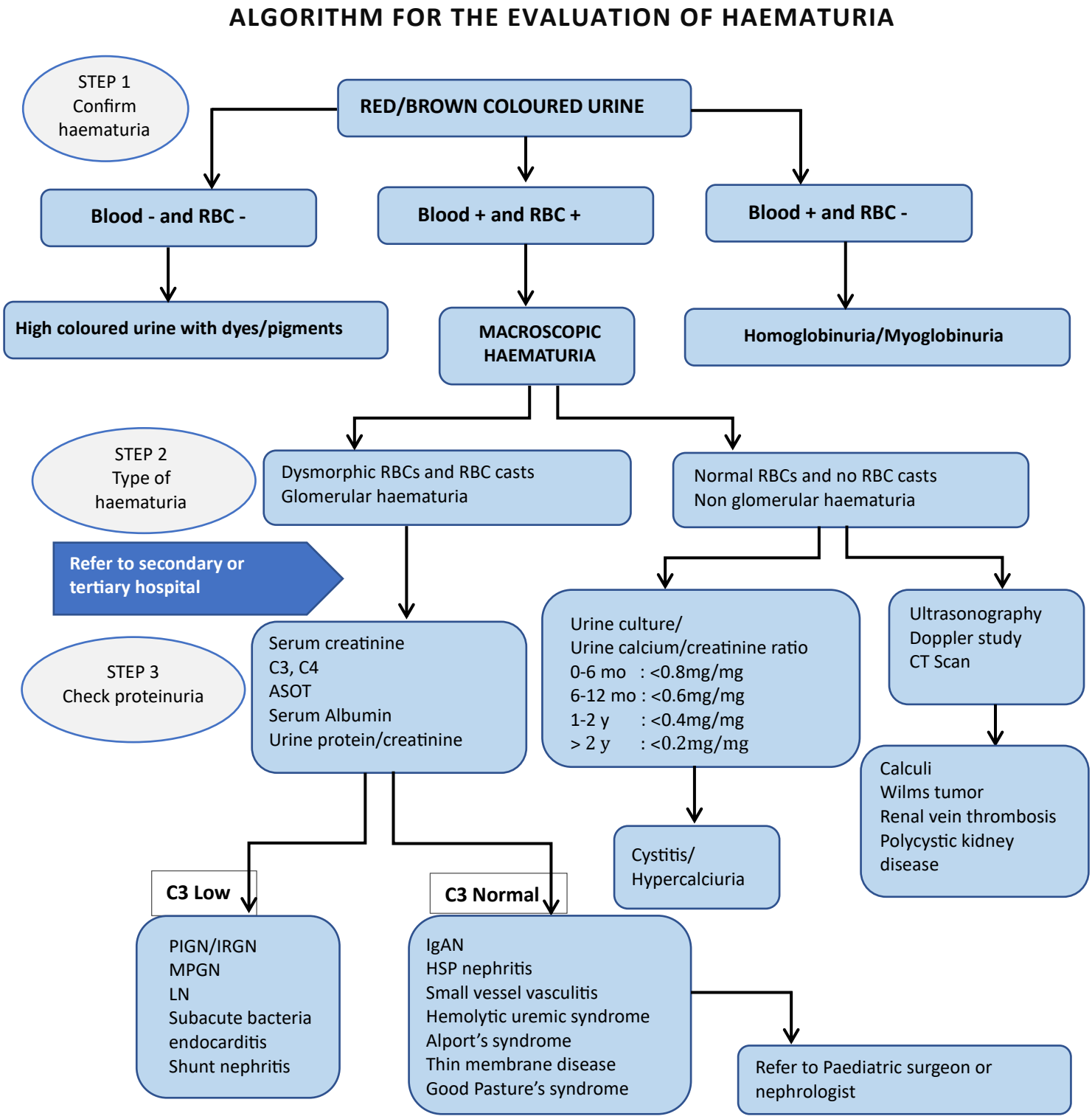


Fig 7.2. Approach to Diagnosis and Management of Haematuria, adopted from Ramya Vedula & Arpana A. Iyengar, The Indian Journal of Pediatrics (August 2020) 87(8):618–624.

Table 7.3. haematuria (History and Possible Diagnosis

History	Possible Diagnosis
Lower tract symptoms (dysuria, urgency, frequency, suprapubic pain)	UTI
Recent illness (pharyngitis, impetigo, viral illness)	Postinfectious glomerulonephritis
Abdominal pain	UTI, HSP, Crystalluria/stone
Arthralgias	HSP, SLE
Diarrhoea(±bloody)	HUS
Cough, haemoptysis	Vasculitis
Sickle cell disease	Glomerulonephritis, papillary necrosis
Swimming in streams	Schistosomiasis
Physical findings	Possible diagnosis
Suprapubic pain	UTI
Flank Pain	IgAN, stones, renal vein thrombosis, pyelonephritis
Oedema	Glomerulonephritis, nephritic syndrome
Abdominal mass	Wilms tumour, hydronephrosis, cystic kidney disease
Meatal stenosis	Infection, trauma
Family History	Possible Haematuria
Sickle cell disease or trait	

Diagnostic evaluation

The first step involves measurement of blood pressure.

1. A dipstick urinalysis evaluates for pyuria, proteinuria, heme positivity, and urinary concentrating defects.
2. Microscopy evaluates for white blood cells and clumps, RBC morphology, crystals, and casts.
3. Crystalluria can be caused by calcium oxalate, calcium phosphate, uric acid, or cystine crystals. Hypercalciuria is by far, the most common cause of crystalluria.
4. Urine culture should be done for suspected UTI.

5. Radiographic studies if indicated.

The second stage of evaluation involves a more thorough search for underlying renal disease, particularly when oedema, hypertension, alterations in urine output, or systemic symptoms are present.

- Evaluation for sickle cell disease or trait by solubility test and electrophoresis were available.
- Suspicion of acute PIAGN should prompt ordering of ASOT, Anti-DNase B and a C3 measurement.
- Secondary causes of renal disease warrant checking for antibodies, complement levels (C3, C4), hepatitis serologies, or HIV serology.
- A full blood count and Peripheral smear is helpful in the setting of petechiae, bruising, fatigue, abdominal masses, history of diarrhoea or suspicion of chronic disease.
- KUB ultrasonography can identify structural abnormality and presence of calculi as well detect whether the patient has chronic kidney disease.
- Abdominal radiographs may identify radiopaque stones.

WHEN TO REFER

1. If cause of gross haematuria is unclear refer to next level
2. Refer early if patient experiencing symptomatic microscopic haematuria**.**
3. Patient who has asymptomatic haematuria needs periodic evaluation every 1 to 2 years to re-evaluate for coexisting symptoms or proteinuria and to revisit the family history with respect to other family members having haematuria or hearing deficits**.**
4. The child who has persistent asymptomatic haematuria and concomitant proteinuria needs additional evaluation, often including a renal biopsy, refer to next level.

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1. Massengill SF. Pediatrics in review. Pediatrics Rev. 2008;29(10):342-4.
2. Vedula R, Iyengar AA. Approach to diagnosis and management of haematuria. Indian J Pediatr. 2020;87(8):618-24.

7.3 NEPHROTIC SYNDROME

Nephrotic syndrome is characterised by:

- Heavy proteinuria
  - Defined as proteinuria of > 40 mg/m²/hr or 1000mg/m²/day or a Urine-Protein: Creatine Ratio (UPCR) ≥200mg/mmol or 2mg/mg or a urine protein dipstick of ≥3+
- Hypoalbuminaemia
  - Defined as serum albumin < 30g/L (0.3g/dL)
- Hyperlipidaemia
- Oedema

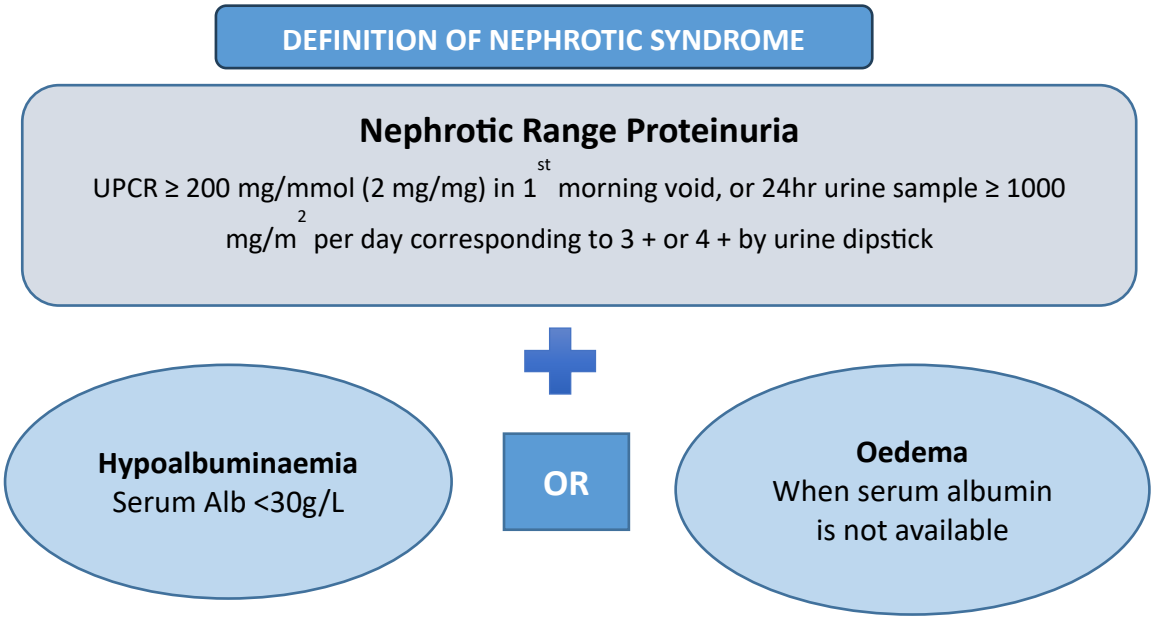


Table 7.4. Definitions in nephrotic syndrome

Nephrotic-range proteinuria	**Urinary protein creatinine ratio (UPCR)≥200 mg/mmol (2 mg/mg) in a spot urine, or proteinuria ≥1000 mg/m² per day in a 24-h urine sample corresponding to 3+(300–1000 mg/ dL) or 4+(≥1000 mg/dL) by urine dipstick**
Nephrotic syndrome	Nephrotic-range proteinuria and either hypoalbuminaemia or oedema where serum albumin is not available
Complete remission	UPCR (based on first morning void or 24 h urine sample) ≤ 20 mg/mmol (0.2 mg/mg) or < 100 mg/m² per day, respectively, or **negative or trace dipstick** on **three or more consecutive days**
Steroid-sensitive nephrotic syndrome (SSNS)	Complete remission within 4 weeks of prednisolone at standard dose (60 mg/m2/day or 2 mg/kg/day, maximum 60 mg/day)
Steroid-resistant nephrotic syndrome (SRNS)	Lack of complete remission within 4 weeks of treatment with prednisolone at standard dose
SSNS late responder	A patient achieving complete remission during the confirmation period (i.e. between 4 and 6 weeks of prednisolone therapy) for new onset NS

Infrequently relapsing Nephrotic Syndrome	< 2 relapses in the 6 months following remission of the initial episode or <3 relapses in any subsequent 12-month period
Frequently relapsing Nephrotic Syndrome (FRNS)	≥ 2 relapses in the first 6-months following remission of the initial episode or ≥ 3 relapses in any 12 months (modified definition)
Steroid-dependent nephrotic syndrome (SDNS)	A patient with SSNS who experiences 2 consecutive relapses during recommended Prednisolone therapy for first presentation or relapse or within 14 days of its discontinuation.
Relapse	Urine dipstick ≥3+ or UPCR ≥ 200 mg/mmol (≥ 2 mg/mg) on a spot urine sample on 3 consecutive days, with or without reappearance of edema in a child who had previously achieved complete remission
Complicated Relapse	A relapse requiring hospitalization due to one or more of the following: severe edema, symptomatic hypovolaemia or AKI requiring IV albumin infusions, thrombosis, or severe infections (e.g., sepsis, peritonitis, pneumonia, cellulitis)

Causes

- Idiopathic (primary) nephrotic syndrome
  - commonest and cause is unknown.
- Secondary nephrotic syndrome
  - Secondary to a known cause e.g. Hepatitis B, Malaria, Syphilis, SLE etc
- Congenital nephrotic syndrome (CNS)
  - Manifests in the first 3 months of life and usually because of genetic causes but can also be due to some TORCH infections.
- Infantile N.S
  - Manifests after 3 months of life but before 12 months of life
  - May be due to genetic causes or infectious causes.

Clinical features

- Sudden onset of dependent pitting Oedema
  - Periorbital
  - scrotal or vulva
  - ankle or leg
- Sudden Weight gain
- Ascites
  - abdominal pain
- Diarrhoea (due to intestinal Oedema)
- Respiratory distress (due to pleural effusions or massive ascites)

- Decreased urine output.
- ±HTN and Haematuria

Investigations

- Urinalysis
  - 3+ to 4+ proteinuria on dip sticks
  - Spot UPC ratio > 2.0mg/mg (>200mg/mmol)
  - Urine Protein Excretion >1000mg/m^2/day
- Blood
  - FBC/ESR
  - U&Es; Creatinine.
  - LFTs including serum albumin (Alb<30g/L)
  - **C3/C4; ANA, ASOT, anti- DNase B*
  - RPR, HCV antibodies, HbSAg
  - Triglycerides and cholesterol levels.
- CXR, KUB ultrasound
- If available: Varicella zoster virus (VZV) IgG blood levels, tuberculin skin test.

Renal biopsy is indicated when:

- Atypical features are present at presentation such as:
  - Macroscopic haematuria
  - Age < 12 months or > 12 years
  - Low C3
  - Vasculitis rash, suspected vasculitis
  - Haematuria and Hypertension
- Steroid Resistance
- Steroid Dependence

NEPHROTIC SYNDROME WORKUP

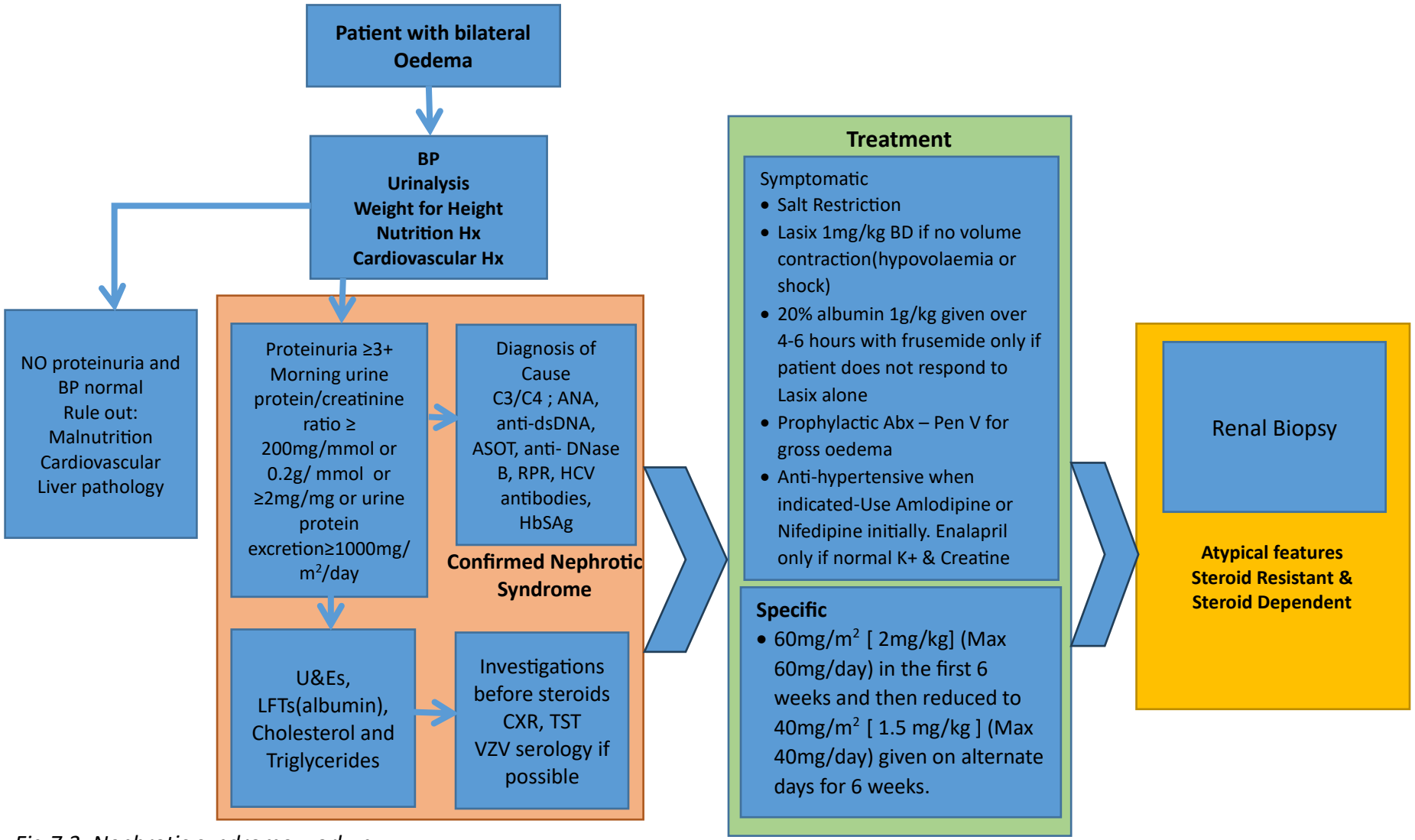


Fig 7.3. Nephrotic syndrome workup

TREATMENT

Symptomatic treatment

- No added salt diets.
- Diuretics can be given but cautiously after assessing that intravascular volume is not contracted, usually Lasix 1mg/kg BD initially- best to discuss with nephrology first.
- 20% or 25% albumin 1g/kg given over 4-6 hours with furosemide at 1-2mg/kg (2 – 3 hours) mid-way through the infusion or at the end. This should be in patients who are severely oedematous or showing signs of intravascular volume contraction. Ensure that patient is passing urine as under these circumstances' albumin can cause pulmonary oedema. Also watch out for hypertension with this treatment. Furthermore, albumin infusions must only be given on weekdays and during regular working hours so that the MO is available to react to any potential complications.
- Prophylactic antibiotic cover (Pen V) as long as patient has gross oedema including ascites. In penicillin sensitive use erythromycin
- Daily weight and daily urinalysis
- By and large children with nephrotic syndrome do not need to be fluid restricted because they have intravascular volume depletion.
- If antihypertensives are required ensure that the patient has a normal creatinine and serum potassium before using ACE- inhibitors. For initial treatment calcium channel blockers such as amlodipine or nifedipine are ideal- discuss with Nephrology.

  In the absence of albumin, fresh frozen plasma could be used in consultation with specialist*[4]*.***

  Specific treatment

  Once the initial diagnostic workup has been completed, it is recommended that infants>3 months and children or adolescents (1–18 years) with their first episode of idiopathic NS should be commenced on Prednisolone and given as follows:

  6 weeks at 60 mg/m^2 or 2 mg/kg (maximum dose 60 mg/day), followed by alternate day at 40 mg/m^2 or 1.5 mg/kg (maximum dose of 40 mg on alternate days) for 6 weeks. Tapering schedule during alternate day dosing is not recommended as it just increases exposure to steroids.

Second episode:

- Prednisone 2mg/kg(60mg/m^2) until protein free for 3 consecutive days then start 1.5mg/kg on alternate day for 4 to 6 weeks.

Adjuvant Therapy during Prednisolone Therapy

- Vit D
- Calcium Carbonate

Ensure adequate calcium intake and normal 25(OH) D serum levels. Supplement

Complications

- Increased susceptibility to bacterial infections.
- Spontaneous bacterial peritonitis
- Hypovolaemia and thromboembolism
- Complications related to chronic steroid administration include hypertension, obesity, and linear growth retardation.

Steroid resistant nephrotic syndrome and steroid dependant nephrotic syndrome:

- These patients will require a renal biopsy and introduction of steroid sparing agents.
- Ideally these patients should be discussed with nephrology and referred if possible.

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7.4 ACUTE GLOMERULONEPHRITIS

Acute glomerulonephritis results from inflammation in the glomerulus. It is a clinical syndrome that is said to be present when a constellation of four features are present in a patient.

- Edema
- Hypertension
- Haematuria
- Renal dysfunction

Causes

Causes of acute glomerulonephritis are divided into five forms of glomerulonephritis based on underlying immune processes.

- Immune Complex GN
- IgA nephropathy, IgA vasculitis (HSP)
- Infection-related GN
  - Streptococcus species (i.e. group A beta-haemolytic) and is the commonest cause in children (80% GN); Syphilis, TB, salmonella, E. coli.
  - Viral: Hepatitis B and C, HIV
  - Parasitic: Malaria
- Systemic Lupus Erythematosus (Lupus Nephritis)
- Pauci-immune GN
- PR3-ANCA GN, MPO-ANCA GN, ANCA negative GN
  - Anti-glomerular basement membrane (anti-GBM) GN
  - Monoclonal IgG GN
  - C3 Glomerulopathy

CLINICAL FEATURES

History

- Sudden development of puffiness of the eyelids and facial oedema in the setting of a post-streptococcal infection.
- The urine may be dark and scanty.
- Non-specific symptoms include weakness, fever, abdominal pain, and malaise.
- Recent upper respiratory tract or skin infection (impetigo)
- Commonest being strep infection.
- Headache and seizures (due to high blood pressure)

Physical examination

- Most frequently patients present with a combination of oedema, hypertension, haematuria, and oliguria.
- The signs of fluid overload may be present.
  - JVP distention
  - Pitting oedema

- Crackles on chest auscultation (pulmonary oedema)
- A tender hepatomegaly

- On cardiovascular examination, a new heart murmur can be auscultated in patients with infective endocarditis.
- Look for signs of scabies or furuncles on skin: common in post-strep.
- Other signs depend on the underlying cause e.g. malar rash and other vasculitic rashes.

Investigations

- Urine microscopy – RBC's (dysmorphic red blood cells), proteinuria, leukocytes
- Blood – FBC, U/E and creatinine, ESR
- Cultures – throat swab
- Other (as indicated) - CXR, ECHO, ASOT, Anti-DNAse B, Antinuclear antibody (ANA), anti-dsDNA antibodies.
- Complement levels (C3, C4), ANCA
- CXR, KUB ultrasound
- +/- renal biopsy

Treatment

- Antibiotic, i.e. penicillin if thought to be post-strep.
- Antihypertensive, i.e. diuretics, calcium channel blockers, beta-blockers
  - **Important note:** Please avoid ACE-inhibitors and ARBs in this group of patients as initial antihypertensive choice until blood results for serum creatinine and potassium are known. And even if normal cautious use of ACE-I is advised unless one can easily monitor renal function of the patient.
- Sodium restriction
- Renally adjust all drug doses based on estimated GFR. This can be calculated using the Schwartz formula in children= 40 X height [cm]/serum creatinine (in micromoles/l)
- Fluid restriction may be necessary depending on extent of renal dysfunction.
- Treat underlying cause
- If patient progresses to acute renal failure, refer to acute renal failure protocol.
- As some patients may end up with rapidly progressive glomerulonephritis early nephrology consultation is highly encouraged
- Admit patient initially for investigation.

Post-strep glomerulonephritis (PSGN)

- Commonest cause in our environment
- Often associated with skin bacterial infection by group A beta haemolytic streptococcus (GAS) with the latent period between GAS infection and PSGN is dependent upon the site of infection: between one- and three-weeks following GAS pharyngitis and between three- and six-weeks following GAS skin infection. Bacterial infection may also be facilitated by infestation with *Sarcoptes* scabei.

- Usually benign in over 90% of patients but has the potential to complicate into rapidly progressive glomerulonephritis.
- Expected to have normal C3 by 8 weeks post onset. If not, then patient requires a biopsy.
- If significant proteinuria present after 6 months patient will need a renal biopsy.
- Patient may continue to have microscopic haematuria for up to 2 years after initial onset.
- Residual hypertension can occur and so patient will need continued monitoring of blood pressure.

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7.5 POSTERIOR URETHRAL VALVE

Posterior urethral valves (PUV) are a problem with the urethra in a boy. This is a remnant of renal embryogenesis at the level of the prostate with valve like leaflets that causes obstructive symptoms of the bladder

When to suspect puv

A male patient with:

- An enlarged bladder that can be palpated suprapubically
- Pain when urinating
- Weak urine stream
- Frequency and urgency
- Bedwetting or pant wetting after toilet training
- Poor weight gain
- Urinary tract infection (rare unless there is a blockage)

How to investigate

- Abdominal/Renal Ultrasound (KUB)
- Voiding Cystourethrogram (VCUG)
- Cystoscopy
- U&Es
- Urine MCS

What to do

- Symptomatic treatment
  - Catheterization
  - Antibiotics for UTI
  - Fluids (oral or IVF)
  - Analgaesia
  - Correct electrolyte imbalances
  - Vesicostomy while awaiting surgery

- Definitive treatment
  - Endoscopic ablation

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7.6 ACUTE KIDNEY INJURY

Acute Kidney injury is an acute, potentially reversible, deterioration in kidney function resulting in failure to maintain normal physiological homeostasis, characterized by an increase in blood urea and serum creatinine values, often accompanied by hyperkalaemia, metabolic acidosis, and hypertension.

The Kidney Disease: Improving Global Outcomes (KDIGO) definition and staging system is the most recent and preferred definition even in paediatric AKI literature. Other classification systems include pRIFLE (paediatric Risk, Injury, Failure, Loss of kidney function, and End-stage kidney disease) and a subsequent modification proposed by the Acute Kidney Injury Network (AKIN). Each definition confers its own set of advantages and disadvantages.

Table 7.5. Acute Kidney Injury

Classification	Staging	Creatinine Criteria	Urine Output Criteria
KDIGO	1	SCr rise ≥ 26.5µmol/L (0.3 mg/dL) within 48 hr or an increase in creatinine of ≥50% within 7 day	>0.5 and ≤ 1 mL/kg/hr
	2	Increase in creatinine of ≥100%	>0.3 and ≤ 0.5 mL/kg/hr
	3	Increase in creatinine of ≥200% or SCr ≥ 353.6µmol/L (4 mg/Dl) or receipt of dialysis or eGFR<35 mL/min/1.73 m2	≤ 0.3 mL/kg/hr
pRIFLE	Risk	eGFR decreased by ≥25%	0.5 mL/kg/hr for 8 hr
	Injury	eGFR decreased by ≥50%	0.5 mL/kg/hr for 16 hr
	Failure	eGFR decreased by ≥75% (or <35 mL/min/1.73 m2)	0.3 mL/kg/hr for 24 hr or anuria for 12 hr
	Loss	Persistent failure >4 week	
	ESRD	Persistent failure >3 months	

AKIN	1	Increase in creatinine of ≥50% or an absolute increase in creatinine of 0.3 mg/dL over 48-hr period	
	2	Increase in creatinine of ≥100%	
	3	Increase in creatinine of ≥200%	
AKIN, Acute Kidney Injury Network; eGFR, estimated glomerular filtration rate; ESRD, end-stage renal disease; KDIGO, Kidney Disease: Improving Global Outcomes; pRIFLE, paediatric Risk, Injury, Failure, Loss of kidney function, and End-stage kidney disease; SCr, serum creatinine.			

Table 7.6. Causes of Acute Kidney Injury

PRE-RENAL	RENAL (INTRINSIC)	POST-RENAL
refers to causes resulting from reduced blood pressure or reduced effective intravascular volume thus resulting in hypoperfusion of the kidneys. This can be due to dehydration, haemorrhage, sepsis, cardiac failure, hypoalbuminaemia (nephrotic syndrome, liver disease).	Renal parenchymal damage, e.g. Acute glomerulonephritis, Intrarenal vascular disease (Haemolytic-uremic syndrome, Vasculiditis), Acute interstitial nephritis, Acute tubular necrosis, Nephrotoxins	**Congenital** - posterior urethral valves, pelvi-ureteric junction if bilateral, vesico-ureteric junction obstruction if bilateral, ureterocoeles, prune belly syndrome, bladder outlet obstruction, neurogenic bladder. **Acquired (rare)** – Urolithiasis, Clots, Tumours if both kidneys involved or if these result in bladder outlet obstruction.

Clinical features

These will reflect the cause and the consequent abnormalities.

- Signs of AKI include signs of fluid overload (hypertension, oedema) and signs of congestive cardiac failure (CCF), such as hepatomegaly, gallop rhythm, and pulmonary oedema.
- Signs of intravascular volume depletion include tachycardia, hypotension, decreased skin turgor, dry mucous membranes, and changes in sensorium.
- Although oliguria is a criterion used to diagnose and stage AKI, AKI can be present without oliguria, especially in patients with nephrotoxic kidney injury, interstitial nephritis, or perinatal asphyxia. Oliguria may be defined as urine output less than 0.5 ml/kg/h in children and < 1ml/kg/hr in neonates.

- Rash and arthritis (e.g. SLE and Henoch-Schoenlein purpura nephritis), flank masses (renal vein thrombosis, tumours, cystic diseases, urinary tract obstruction)

Table 7.7. Assessment and Investigations

Points in the history and examination that assist in the		
• **Pre-renal**	• **Renal**	• **Post-renal**
• Diarrhoea and vomiting	• Bloody Diarrhoea (HUS)	• Antenatally Diagnosed Anomaly
• Cardiac Impairment	• Drugs	• Previous UTIs
• Birth Asphyxia	• Birth Asphyxia	• Poor Urinary stream
• Acute Weight Loss	• Prolonged Convulsions	• Palpable bladder or kidneys
• Umbilical Catheters	• Recent throat Infections	• Calculi
• Poor capillary refill	• Systemically unwell	• Spinal abnormalities

Investigations

- Dipstick urinalysis: leucocytes (UTI), Proteinuria & Haematuria (Glomerulonephritis, nephrotic/nephritic syndrome), specific gravity
- Urine microscopy: Findings consistent with glomerulonephritis such as red blood cell casts, dysmorphic red blood cells, granular casts
- Urine: sodium and urine osmolality
    - UNa<10mmol/L (<20 in neonates), FENa <1% (<2.5% in Neonates) and Urine Osmolality >500mOsm/kg suggests pre-renal AKI
- Serum bicarbonate: if not done by lab routinely can be obtained from blood gas strip.
- Electrolytes: K+, Na+, Ca++, Phosphate, Uric acid and urea, creatinine
- Full blood count: peripheral smear as indicated.
- Other blood tests can be done as dictated by underlying cause.
- CXR: If respiratory or Cardiac signs are present.
- KUB ultrasound (urinary tract obstruction or to rule out Acute on Chronic Kidney Disease)
- Renal biopsy:
    - If kidney function is deteriorating rapidly (as in RPGN) and aetiology is unknown
    - Nephritic/Nephrotic presentation

Table 7.8. Treatment

Initial management

Assessment and Management of Intravascular Volume

Hydration Status	Clinical Features	Initial Management
Dehydrated	-Tachycardia -Cool hands, feet and nose -Prolonged CRT -Low BP (Late sign) -Dry Mucous Membranes, Sunken Eyeballs	-Fluid Resuscitation 10ml/kg normal saline or Ringers Lactate over 30mins, Assess Urine output and repeat if necessary
Euvolaemic		-Fluid Challenge 10-20ml/kg NS over 1hr, with furosemide 2-4mg/kg IV
Intravascular Fluid Overload	-Tachycardia -Gallop Rhythm -Raised JVP and BP -Palpable Liver	-Furosemide 1-2mg/kg IV -Dialysis if no response

Treatment

- Pass urinary catheter e.g. in neonates with suspected posterior valves, or for urine output monitoring in non-ambulatory children/adolescents during AKI. If size 6 Fr foley catheter is unavailable, one can easily use a size 6 Fr feeding tube [NGT] to catheterise the bladder. This can be held in place by strapping to the thigh of the infant so that it does not fall out.
    - NOTE: in cases of PUV please note that the patient may develop post-obstruction polyuria once a urinary catheter is inserted. If not properly managed the infant may become severely fluid depleted and develop a pre-renal AKI or even go into shock and die. Thus, monitor urine output and 6 hourly weight changes meticulously and replace excess urine passed ideally with hypotonic solutions such as half strength Darrow's in older infants and quarter strength Darrow's in the newborn. This is because the post-obstructive kidney often has a poor concentrating ability.
- Fluids – if hypovolaemic will require volume expansion with isotonic saline at 10 ml/kg over 30 min. This may be repeated one or two more times at most depending on the response.
- Those with relatively normal intravascular volume – give insensible losses at 400 ml/m^2/day (30ml/kg/day) plus urine output for the previous 24 hours & extra renal losses as appropriate.
- Daily monitor urine & stool output, fluid intake, body weight, blood chemistries.

- Renally adjust all drug doses. Use the Schwartz formula to calculate the estimated GFR.
- Involve your nearest paediatric nephrology unit earlier rather later in the management of all paediatric AKI patients as per KDIGO AKI guidelines.

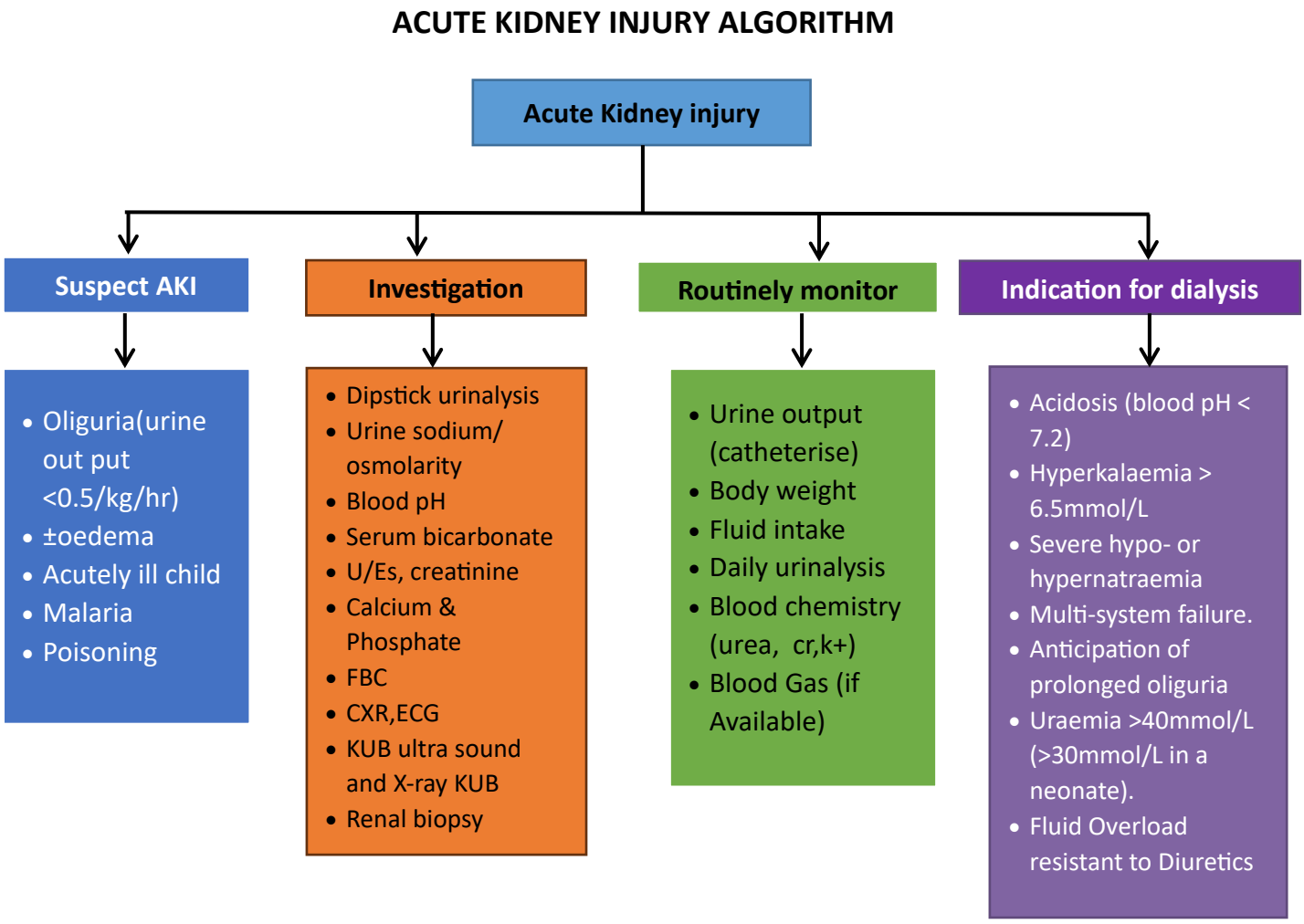


Fig 7.4. Management of paediatric AKI patients as per KDIGO AKI guidelines

Complications

1. Hyperkalaemia

Hyperkalaemia > 6 mmol/l (indicated by the following ECG changes: peaked T-waves, widening QRS complex, ST segment depression, ventricular arrhythmias, and cardiac arrest).

- Eliminate exogenous potassium (IVF, diet) including drugs sources, oral or enema K^+-binding resin (Na polystyrene sulfonate [kayexalate]) at 1g/kg single dose.
- If K^+ >7 mmol/l, + ECG changes, give the following:
  - 10% calcium gluconate (cardio-protective) at 0.5ml/kg IV over 10 min
  - Nebulised salbutamol 2.5 mg if weight < 25kg or 5 mg if weight >25 kg or IV salbutamol 4µg/kg in 10 ml of water over 10 min
  - 8.4% sodium bicarbonate 1-2 mmol/kg IV over 5-10 min
  - Glucose+/- insulin: 10%D 5-10ml/kg/hr
  - If glucose > 15mmol/l add Insulin 0.05IU/kg /hr
  - Lasix at 1mg/kg BD may also be used to drive out potassium.

The above are only temporizing measures. Please discuss with nephrology for arrangement of dialysis ASAP.

2. Hypocalcaemia

Calcium carbonate orally 45 to 65 mg/kg per day. If tetany, IV 10% calcium gluconate 0.5 to1 ml/kg (up to 10 ml) if patient is symptomatic, with low phosphorus diet or phosphorus binders (calcium carbonate given with meals).

3. Hyponatraemia (dilutional usually)

Treat if < 120 mmol/l or symptomatic (seizures, lethargy). Correct to 125 mmol/l using the following formula:

Required NaCl (mmol) = 0.6×weight×deficit (125 mmol/l – serum Na) given over 4-6 hours

- One would need to use 3% hypertonic saline if not available discuss with pharmacist how NaCl tablets or table salt can be given orally in-lieu of hypertonic saline.

4. Metabolic acidosis

Mild acidosis is common and requires no treatment. However, severe acidosis (pH = 7.15, serum bicarbonate < 8 mmol/l) should be corrected partially with IV sodium bicarbonate to pH = 7.20, serum HCO_3^- = 12 mmol/l, then the remainder corrected by oral sodium bicarbonate. *Rapid metabolic acidosis correction with IV $NaHCO_3$ may reduce ionised serum calcium leading to tetany!*

Preferably correct metabolic acidosis by instituting dialysis

5. Hypertension and Anaemia treat as per appropriate protocol.

Nutrition

Nutritional requirements should be addressed accordingly in consultation with the dietician.

Renal replacement therapy

Dialysis (peritoneal dialysis or haemodialysis)

Dialysis indications:

- Fluid overload/hypertension.
- Severe acidosis not responding to medical management.
- Persistent hyperkalaemia or other above uncontrollable electrolyte imbalance.
- Symptomatic uraemia or urea > 35.7 - 53.6 mmol/l
- Dialyzable toxin.
- Established anuria.

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ACUTE KIDNEY INJURY (AKI) RECOMMENDATIONS

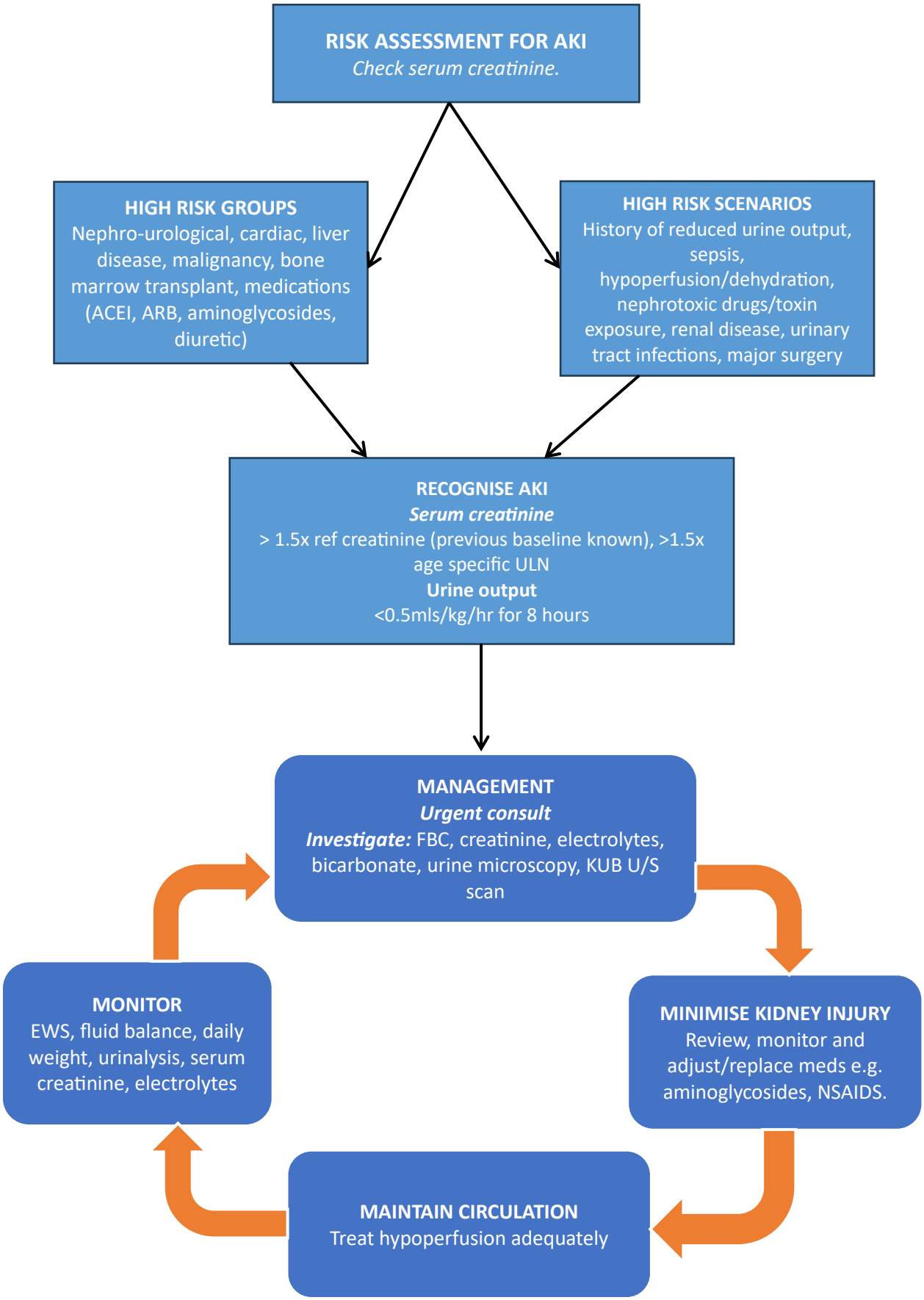


Fig 7.5. Acute Kidney Injury (AKI) Recommendations as per KDIGO guidelines

8.1 ACUTE DIARRHOEAL DISEASE

Passage of three or more loose/watery stool or one voluminous loose/watery stool per day.

CAUSES

Infectious

- Bacterial infections: Commonly, *Escherichia coli (E. coli), Campylobacter jejuni, Salmonella species, Shigella, and, Vibrio cholerae*
- Viral infections: rotavirus, norovirus, adenovirus, Norwalk virus, astrovirus
- Parasites: *Giardia lamblia, Entamoeba histolytica, Cryptosporidium*

Diarrhoea may be watery or bloody (dysentery). The causes of bloody diarrhoea include *Shigella, Enteromoemba histolytica, Campylobacter species and Enteroinvasive E. coli*

***Systemic infections* associated with diarrhoea include:**

- influenza, measles, HIV, malaria, pneumonia, urinary tract infection, meningitis, and sepsis.

Non-infectious causes

- Food poisoning
- Drugs: Antibiotics, anti-hypertensives, cancer drugs, and antacids containing magnesium
- Intestinal diseases: Inflammatory bowel disease and coeliac disease
- Food intolerance: Lactose
- Food allergy: Cow's milk, Soya

NOTE: Some of the above causes may lead to persistent/chronic diarrhoea

Clinical features:

- Watery or loose stools ± bloody stools
- Abdominal cramps
- Tenesmus
- Urgency
- Abdominal pain
- Vomiting
- Fever
- Poor appetite
- Dehydration

Clinical assessment

Divided into four components to guide clinical management:

- Classification of the type of diarrhoeal illness (acute, persistent, chronic)
- Assessment of hydration status
- Assessment of nutritional status
- Assessment of co-morbid conditions

Signs of dehydration

- Dry mucous membranes
- Rapid thread pulse, low blood pressure, capillary refill > 2sec
- No tears when crying
- No wet diapers for 3 hours or more
- Sunken eyes/anterior fontanelle
- High /low temperature
- Listlessness or irritability
- Reduced skin turgor

Table 8. Classification of levels of dehydration; using a combination of two to three physical signs reliably predicts dehydration.

	No dehydration	Some dehydration	Severe dehydration
General Condition	Well, alert	Restless, irritable	Lethargic or unconscious
Eyes	Normal	Sunken	Very sunken
Thirst	Drinks normally, not thirsty	Thirsty, drinks eagerly	Drinks poorly, or not able to drink at all
Skin pinch	Goes back quickly	Goes back slowly (< 2sec)	Goes back very slowly (>2sec)
Treatment	**PLAN A**	**PLAN B**	**PLAN C**
	Give ORS per loose stool: <2 years: 50-100ml 2-5 years: 100-200ml Continue feeding	**Give ORS 75ml/kg over 4 hours** After 4 hours, reassess & determine level of dehydration then treat accordingly. If oral rehydration fails, give ORS via NGT or IV ½ strength darrows in	**Give 100ml/kg ringers lactate or ½ strength Darrow's with 10% dextrose at** <1 year: 30ml/kg over 1 hours, then 70ml/kg over 5 hours ≥1 year: 30ml/kg over 30 minutes, then 70ml/kg over 2 ½ hours Reassess patient every 1-2 hours. If hydration not improving, give IVF more rapidly.

		10% dextrose at 75ml/kg over 4 hours	After IVF, reassess and choose appropriate treatment plan (A, B or C) If IVF not available, give ORS via NGT or orally at 20ml/kg/hour for 6 hours
	Zinc supplementation: give zinc sulphate for 10 to 14 days • <6months of age:10mg daily • ≥ 6months of age: 20mg daily		

Investigations

- FBC, ESR
- Stool m/c/s for bloody and persistent diarrhoea
- U&E, Creatinine
- Abdominal x-ray
- Barium enema/meal

NOTE: Other investigations will depend on the underlying/systemic conditions identified such as RDT/MPS,

Principles of management

- Fluid replacement
- Zinc supplements
- Continued feeding
- Antibiotics (for bloody diarrhoea or persistent diarrhoea with stool results)

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8.2 PERSISTENT DIARRHOEA (PD)

Passage of 3 or more watery stools within 24hrs lasting for 14 days and more.

- Persistent diarrhoea accounts for 1.4 to 28.4% of all cases of diarrhoea. More than one-half of these deaths are attributed to persistent diarrhoea and 30-50% of deaths occur among children younger than five years of age.
- Dehydration, malnutrition and infections are major contributors to PD morbidity.
- The many causes of PD can be divided into four principle pathophysiologic mechanisms: osmotic, secretory, dysmotility associated, and inflammatory.

Clinical features

- Liquid stools often passed after eating, may be explosive
- Occasionally stool may contain visible blood
- Weight loss often evident and signs of malnutrition often present
- Signs of dehydration
- Features of Extra-intestinal infections: e.g. Pneumonia, UTI

Table 8.1. Causes of Persistent Diarrhoea

Cause	Major Clinical Features (In addition to PD)	Laboratory and Imaging Findings
Infectious (e.g. E. *coli*, Cryptosporidium. Giardia, Salmonella, E. *histolytica*)	Possible blood and/or mucus in stool Possible fever and/or abdominal pain	Positive stool culture, ova and parasite examination
Lactose malabsorption	Abdominal discomfort, bloating, flatulence	Elevated breath hydrogen concentration after lactose ingestion
Immunodeficiency state (various diseases)	Recurrent infections Young age, typically in infancy	Abnormal immunoglobulins (eg, low IgG, low IgA, high IgM) Lymphopenia Low antigen titers to previous immunizations
Food Allergy	Most commonly in response to cow or soy milk	May have hypoalbuminaemia and anaemia Electrolyte abnormalities from diarrhoea/ vomiting Serum IgE may be elevated
Hirschsprung disease	Delayed passage of meconium Abnormal barium enema Distended abdomen	Abnormal barium enema Absent ganglion cells on rectal biopsy

	Explosive stool with rectal examination	
Toddlers diarrhoea	Usually thriving toddler. Sweetened juices usually the cause	Normal laboratory and imaging results
Irritable Bowel Syndrome	Alternating constipation with diarrhoea Abdominal pain relieved by defecation Typically diagnosed in adolescence or later	Normal laboratory and imaging results
Celiac disease	FTT, abdominal distention, vomiting Typically, 9-24mo of age	Elevated anti-TTG IgA, antiendomysial IgA Antibodies
Inflammatory Bowel Disease	Bloody stool Stooling urgency, abdominal pain, Fatigue. Weight loss. Arthritis	Elevated ESR & faecal calprotectin Thrombocytosis Iron-deficiency anaemia Hypoalbuminaemia

Treatment Objectives

Treatment of PD consists of:

- Appropriate fluids to prevent and treat dehydration
- A nutritious diet to promote weight gain. **BEWARE** of foods that worsen diarrhoea
- If SAM is present, treat according to SAM protocol (refer to section on SAM)
- Supplementary vitamins and minerals, including zinc for 10-14 days
- Antimicrobials to treat diagnosed infections

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8.3 WASTING AND NUTRITIONAL OEDEMA (ACUTE MALNUTRITION IN INFANTS AND CHILDREN UNDER 5 YEARS)

Malnutrition refers to deficiencies, excesses, or imbalances in a person's intake of energy and/or nutrients.

Acute malnutrition in children under 5 years of age is defined as wasting or having nutritional oedema.

Acute malnutrition may be further sub-classified as follows.

Severe acute malnutrition (SAM):
- nutritional oedema and/or
- WHZ or WLZ < -3 and/or
- MUAC <115mm.

Moderate acute malnutrition (MAM):
- WHZ or WLZ < -2 and/or
- MUAC ≥ 115 and <125mm and
- no nutritional oedema.

DIAGNOSIS OF MALNUTRITION
The diagnosis of malnutrition can be made based on the age of the patient:

A. Infants less than 6 months of age at risk of poor growth and development
- No weight gain or weight loss from one measurement to the next; or downward crossing of weight-for-age centile lines[1]; or Insufficient weight gain
- Weight-for-age z-score (WAZ) <-2 SD; or weight-for-length z-score (WLZ) <-2 SD; or nutritional oedema; or (MUAC) <110mm for infants between 6 weeks to less than 6 months of age.
- Neurodevelopmental concerns; or Infant feeding concerns; or maternal risk (physical or mental health problem(s) affecting caring practices); or history of hospitalization.
- Preterm birth; or Low birth weight; or small for gestational ag

B. Severe wasting and nutritional Oedema in infants and children 6-59 months of age
- WHZ or WLZ <-3 SD or
- MUAC) <115 mm and/or
- nutritional Oedema.

C. Moderate wasting in infants and children 6-59 months of age
- WHZ or WLZ ≥-3 and <-2 SD or
- MUAC ≥115mm to <125mm

MANAGEMENT

A. Infants less than 6 months of age at risk of poor growth and development

Infants who are less than 6 months of age with severe wasting and/or nutritional Oedema who are admitted for inpatient care:

- should be breastfed where possible and the mothers or female caregivers should be supported to breastfeed the infants.
- If an infant is not breastfed, support should be given to the mother or female caregiver to re-lactate. If this is not possible, wet nursing should be encouraged *(refer to MYCN guidelines)*
- for infants with severe wasting but no Oedema, should also be provided a supplementary feed:
  - expressed breast milk (EBM) should be given,
  - where giving EBM is not possible, commercial (generic) infant formula or F-75 or diluted F-100 may be given, either alone or as the supplementary feed together with breast milk
- for infants with Oedema, commercial (generic) infant formula or F-75 should be given as a supplement to breast milk.
- DO NOT give full-strength F-100
- if there is no realistic prospect of being breastfed, infants should be given appropriate and adequate replacement feeds such as commercial (generic) infant formula, with relevant support to enable safe preparation and use, including at home when transferred from inpatient care.

B. Management of infants and children 6-59 months with wasting and/or nutritional oedema

Table 8.2. Principles of management

Steps and actions	Stabilisation (in ITC)		Rehabilitation (in OPC)
	Day 1-2	Day 3-7	Week 2-6
Prevent or treat hypoglycaemia	→		
Prevent or treat hypothermia	→		
Prevent or treat dehydration	→		
Correct electrolyte imbalance	→		
Treat and prevent infection	→		Prevent infection
Correct micronutrient deficiencies	No iron →		With iron →
Start cautious feeding	→		Encourage feeding
Catch-up growth			→
Provide loving care and play, emotional support and sensory stimulation	→		
Prepare for follow-up and discharge*			→

1. Treat/prevent hypoglycaemia

- Hypoglycaemia is serum glucose <3.0mmol/L.
- If the patient is conscious and is able to drink, give 50ml of 10% glucose orally or by nasogastric tube (NGT). Then starter feed F75 every 30 minutes for 2 hours (giving one quarter of the 2 hourly feed each time)
- If the child is unconscious, lethargic or convulsing, give 10% dextrose intravenously at 2.5 ml/kg followed by 50ml of 10% glucose by NGT. Then give starter F75 as above.
- Give antibiotics in case hypoglycaemia is due to an infection.

Give 2 hourly feeds day and night.

2. Treat/prevent hypothermia

- Treat is the axillary temperature is 35.0^{o}C.
- Feed with F75 straight away or start rehydration (if needed)
- Rewarm the child:
  - Either clothe the child, cover the child with a warmed blanket and place a heater or lamp nearby
  - Or put the child on mother's bare chest (skin-to-skin) and cover them
- Give antibiotics in case hypothermia is due to an infection.

3. Treat/prevent dehydration

- Suspect dehydration if the child has 3 or more episodes of watery diarrhoea.
- Clinical signs of dehydration might not be obvious.
- The malnourished child without dehydration is managed entirely by weight changes, clinical signs of improvement and clinical signs of overhydration.

Shock in malnutrition

A dehydrated child can develop shock

- Can be caused by severe dehydration or sepsis
- Shock from dehydration and sepsis are likely to coexist in children with SAM
- In children with shock from dehydration there will often be a clear history of diarrhoea and/or excessive vomiting.

Signs of shock

A patient with SAM is said to be in shock when:

- There is a confirmed history of either diarrhoea or vomiting with dehydration **AND**
- There is decrease in level of consciousness (semi-conscious or cannot be roused) **AND**
- There is a weak or absent radial or femoral pulse, **AND**
- Cold hands and feet
- Prolonged capillary refill time of >3 seconds

It is important to diagnose shock early and institute appropriate treatment

Treatment of shock

- Give oxygen
- Give sterile 10% glucose 5ml/kg by IV
- Keep child warm
- Collect blood for cross match for whole blood
- Begin IV rehydration immediately:
    - Check the starting respiratory and pulse rates and record them on the CCP. Also record starting time
    - Give IV fluids at 15ml/kg over 1 hour (Rehydration needs to be done *slowly* to prevent over-hydration)
    - Fluids to give:
        - ✓ Half strength Darrow's solution with 5% glucose (dextrose) preferably OR
        - ✓ Ringer's lactate with 5% glucose (add 20 mmol/l of potassium chloride)
    - Observe the child and check respiratory and pulse rates every 10 minutes,
    - If the respiratory rate and pulse rate increase, stop IV fluids.
    - If respiratory rate and pulse rate are slower after 1 hour, the child is improving. Repeat the same amount of IV fluids 15ml/kg for another 1 hour.

Monitoring of shock

- Do baseline respiratory rate and pulse rate
- Then monitor respiratory rate, pulse rate and the condition of the patient every 10 minutes
- Rehydration needs to be done slowly and monitored closely to prevent over-hydration.
- After 1 hour, if respiratory rate and pulse rate are slower than the baseline, give another 15ml/kg during the 2nd hour, continue monitoring every 10 minutes
- After 2 hours of IV fluids, if the child has improved, switch to oral or naso-gastric rehydration with ReSomal (as in dehydration protocol).
  Note: Leave IV line in place in case it is needed again.

- If a child fails to improve during the 1st hour of IV fluids, then assume that the child has **septic shock**.
- Give maintenance IV fluids (4ml/kg/hr) while waiting for blood. When blood is available;
  - Stop all oral intake and IV fluids
  - Give a diuretic to make room for the blood
  - Transfuse whole fresh blood at 10ml/kg slowly over 3 hours. If there are signs of heart failure, give packed cells at 7.5ml/kg as these have a smaller volume.

SHOCK IN MALNUTRITION ALGORITHM

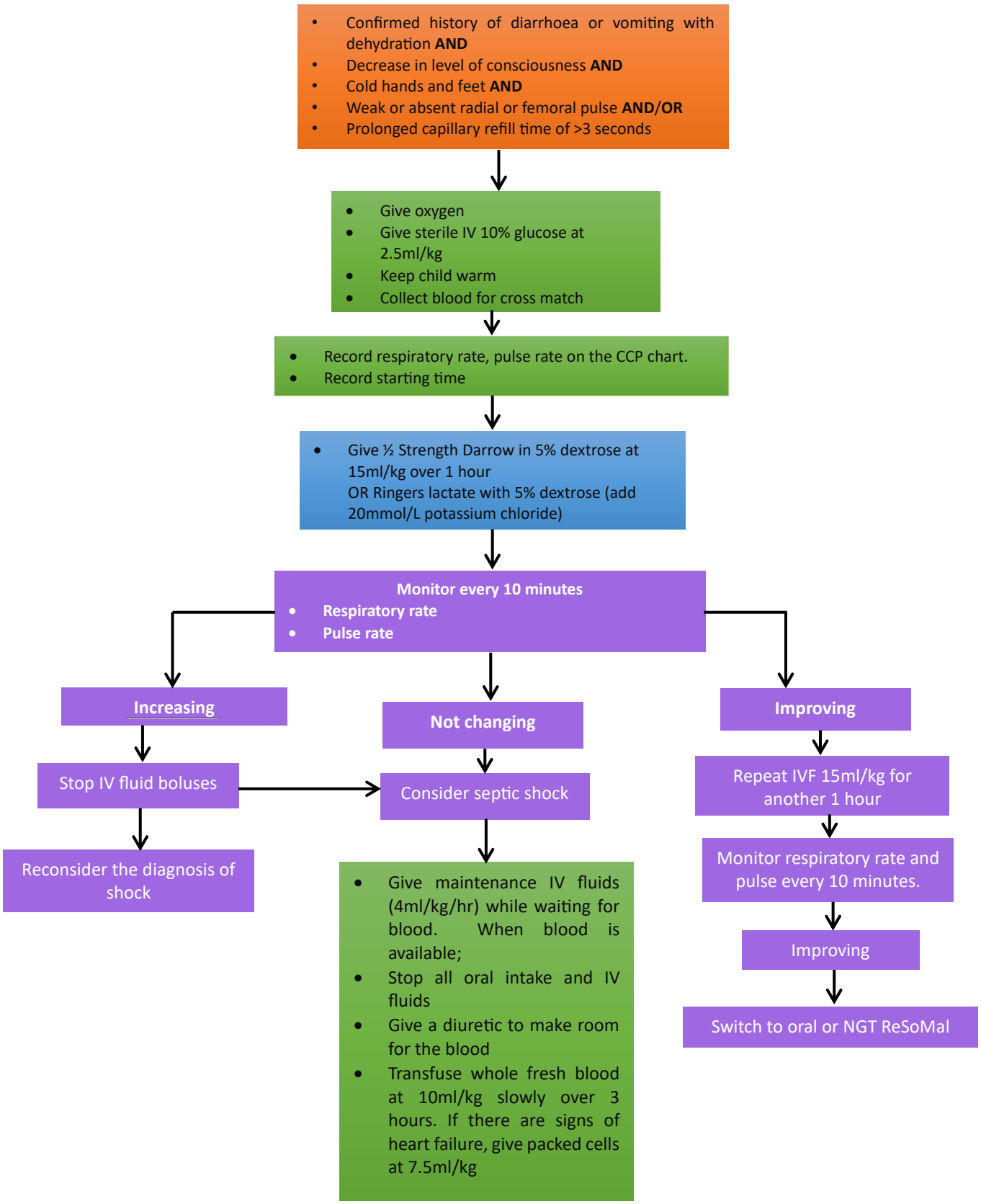


Fig 8. Shock in malnutrition

1. Correct electrolyte imbalance

These are generally corrected in the feeds and fluids specific for treatment of malnutrition.

2. Treat/prevent infections

Note: In severe malnutrition the usual signs of infection such as fever are often absent and infections are often hidden. Therefore, assume that all children have an infection.

- Give broad spectrum antibiotics routinely on admission
  Crystalline penicillin 50,000 IU/kg 6 hourly AND
  Gentamicin 5mg/kg once daily

3. For steps 6-10, refer to the national malnutrition protocol

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8.4 ACUTE UPPER GI BLEEDING

Upper gastrointestinal (GI) bleeding refers to haemorrhage from any level above the ligament of Treitz.

Table 8.3. Causes of Acute Upper GI Bleeding

Age Group	Cause
Neonates	• Haemorrhagic disease of the new-born • Swallowed maternal blood • Stress ulceration • Coagulopathy, A-V malformations
Infants (1 month – 1 year)	• Oesophagitis, • Gastric ulceration
Infants (1 -2 years)	• Peptic ulcer disease • Gastritis
Children older than 2 years	• Oesophageal varices • Gastric varices
Adolescents	• Duodenal ulcers, NSAIDs

Clinical features

Presentation of bleeding depends on the amount and location of haemorrhage.

- Haematemesis
- Coffee ground vomiting
- Melaena
- Haematochezia- if the haemorrhage is severe
- May also present with complications of anaemia/shock

Investigations

- FBC, ESR
- U/E's, Creatinine, LFTs
- Barium swallow/meal
- Clotting profile
- Endoscopy

Treatment

- ABCDE
- Brief history as to possible cause of the bleeding
- Consider gastric wash out
- Consider antidote for bleeding due to poisoning

Iron – Desferrioxamine (Deferoxamine)

Warfarin – Vitamin K

- Monitor vital signs
- Replacement of volume with intravenous solutions and blood products if required
- Endoscopy (electrocautery, clipping or banding) when indicated
- Pharmacotherapy including the following:

Proton pump inhibitors (PPIs) – Omeprazole IV

H_2 receptor inhibitors - Cimetidine IV

Octreotide is a somatostatin analog believed to shunt blood away from the splanchnic circulation

Terlipressin is a vasopressin analog used for variceal upper GI haemorrhage

Propranolol

REFERENCES

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8.5 FUNCTIONAL CONSTIPATION

This is amongst the commonest conditions associated with **chronic** or **recurrent abdominal pain** in children. Usually during weaning, toilet training and beginning of schooling as well as during stressful events.

Features last 2 weeks or more and may include:

- Non-specific chronic/recurrent abdominal pain.
- ≤2 defecations per week.
- At least one episode/week of incontinence after the acquisition of toileting skills
- History of retentive posturing or excessive stool retention
- History of painful or hard bowel movements
- Soiling of underwear
- History of large diameter stools

Accompanying symptoms may include:

- irritability,
- decreased appetite, and/or
- early satiety.

NOTE: Accompanying symptoms disappear immediately following passage of a large stool. Review feeding practices (formula, fibre/water intake).

Red Flags

The presence of the following signs warrants further investigation or referral.

- Fever
- Blood in stool
- Weight loss
- Vomiting
- Anaemia

Diagnosis: Based on ROME IV Criteria (2016) as shown below.

Age	<4years	Developmental age of ≥4years
Rome IV criteria	1. <3 defecations per week 2. History of excessive stool retention 3. History of painful or hard bowel movements 4. History of large-diameter stools 5. Presence of a large faecal mass in the rectum 6. In toilet-trained children, the following additional criteria may be used: ≥1 episode of faecal incontinence per week after the acquisition of toileting skills 7. History of large-diameter stool that may obstruct the toilet Must fulfill ≥2 criteria for ≥1 month prior to diagnosis.	1.<3 defecations in the toilet per week 2. ≥1 episode of faecal incontinence per week 3. History of relative posturing or excessive volitional stool retention 4. History of painful or hard bowel movements 5. Presence of a large faecal mass in the return 6. History of large-diameter stools that may obstruct the toilet Must fulfill ≥2 criteria at least once per week for ≥1 month prior to diagnosis of irritable bowel syndrome with constipation

History and examination are usually sufficient to make a diagnosis. A plain **abdominal X-ray** may help make a diagnosis, but should not routinely be done.

Differential diagnosis

- UTI
- PUD
- Hypothyroidism
- Rarely: IBS and IBD

NOTES: Neonates/Infants: Obtain good history to rule out congenital causes of constipation (Hirschsprung's disease, spine abnormalities, metabolic/endocrine, etc).

Treatment

- Parental/patient education and behaviour interventions are paramount (toilet training coupled with a reward system).
- Diet modification (increase fluids, fibre and fruits).
- **Disimpaction** when there is clinical or radiological evidence of impacted stool with either Polyethelene glycol (PEG) or lactulose +/- enema.

- **Maintenance therapy** with either PEG, lactulose or liquid paraffin. Others include Magnesium hydroxide, magnesium citrate, Senna and sorbitol.
- **Weaning off drugs:** laxatives can be **slowly** weaned of once child is asymptomatic and does not fulfil diagnostic criteria for functional Constipation (ROME IV Criteria of 2016).
- Counsel parents on need for prolonged treatment duration (usually months) to avoid recurrence and overcome fear of pain during defecation in the child.

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5. Rome IV Diagnostic Criteria for Functional Gastrointestinal disorders (FGIDs).2016
6. Tabbers MM et al. Evaluation and Treatment of Functional Constipation in Infants and Children: Evidence -Based Recommendations from ESPGHAN and NASPGHAN. Journal of Paediatric Gastroenterology and Nutrition. 2014;58(2).

8.6 ACUTE HEPATIC FAILURE

Paediatric acute liver failure is a complex rapidly progressive clinical syndrome that is the final common pathway for many known and unknown conditions.

Causes

Approximately 50% of cases are caused by viruses

- Infective
  - Viral - Hepatitis A, B, C and D, HIV, Parvovirus, Herpes simplex virus, Enterovirus, Adenovirus, Varicella, Echovirus, CMV, EBV
  - Bacteria - *Neisseria meningitidis, treponema pallidum*, Leptospira SPP, Brucella SPP, *Coxiella burnetti*
  - Parasites – *Plasmodium falciparum, Entomoeba histolytica*
- Drugs - Paracetamol, antituberculous drugs (Isoniazid), carbamazepine, sodium valproate, halothane, salicylates, methanol, intravenous tetracycline
- Toxins - Mushroom, particularly amanita phalloides, herbs and traditional medicines
- Infiltrative - Leukaemias, lymphomas
- Metabolic - Wilson's disease, galactosemia, tyrosinaemia
- Idiopathic

Clinical Features

The child may present within hours or weeks with

- Protracted vomiting
- Jaundice
- Tender hepatomegaly
- Abdominal distention (ascites)
- Coagulopathy (bruising, petechiae, bleeding)
- Hypoglycaemia
- Electrolyte disturbance
- Encephalopathy (early signs of encephalopathy include alternate periods of irritability, confusion, drowsiness and convulsions. Older children may be aggressive or show unusual behaviour)

Table 8.4. Diagnostic criteria

Acute liver dysfunction is defined by the presence of all three of the following

Criteria	Description
Acute onset	Onset of hepatic failure within 8 weeks of onset to clinical liver disease with no prior evidence of liver disease
Biochemical evidence	Either: Hepatocellular injury -AST or ALT >100 iu/l unless explained by myopathy Or Biliary dysfunction – total bilirubin >85.5µmol/l, direct or conjugate bilirubin >34.2µmol/l and/or GGT >100 iu/l
Coagulopathy	Coagulopathy that persist after vitamin K administration, defined -prothrombin time ≥ 15 seconds OR INR ≥1.5 with evidence of hepatic encephalopathy OR -Prothrombin time ≥ 20 seconds OR INR ≥ 2.0 with or without encephalopathy

Investigations

Liver function tests:

- Prothrombin time (PT)
- International normalised ratio (INR)
- Serum albumin
- Aspartate aminotransferase (AST)
- Alanine aminotransferase (ALT)
- Bilirubin total and direct
- Alkaline Phosphatase (ALP)
- Gamma glutamyl transpeptidase (GGT)
- Blood Glucose levels
- Full Blood Count
- Urea, creatinine and electrolytes
- CRP/ESR
- Imaging
- Investigate for underlying cause

Treatment

- ABCDE
- Oxygen by nasal cannula or face mask
- Vitamin K, stat dose IV or IM
  - 300 micrograms/kg for age 1 month to 12 years and
  - 10 mg if >12 years to attempt correction of prolonged clotting time
- If frank bleeding (GI or other), give fresh frozen plasma at 10 ml/kg IV
- Maintain blood glucose between 4 and 9 mmol/litre using 2/3 of maintenance fluid volume consisting of 10% dextrose IV or orally
- Strictly monitor urine output and fluid balance; aim for a urine output of not less than 0.5 ml/kg/hr
- Correct hypokalaemia if present as it can worsen encephalopathy
- Broad spectrum antibiotics for example cephalosporins of the third generation to treat sepsis
- Systemic fungal infection may require IV amphotericin or oral fluconazole
- Maintain normothermia by environmental measures, **do not give paracetamol**
- Lactulose 5-10mls 2-3 times daily to produce two to four soft stools per day, to be omitted if diarrhoea occurs
- Neomycin 20-30 mg/kg/day 6 hourly orally, maximum dose 2 gm/day to decrease enteric bacteria that produce ammonia
- Treat the underlying cause (e.g. acyclovir for herpesvirus; N-acetylcysteine for paracetamol overdose etc)
- Liver transplantation remains the only effective mode of treatment

Prognosis

It is a potentially devastating disease with a mortality rate of upto 80-90% without liver transplantation.

REFERENCES

1. Kliegman R, Behrman R, et al. Nelson Textbook of Pediatrics. 18th ed. Philadelphia: Saunders Elsevier; 2007.
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8.7 INTESTINAL OBSTRUCTION

It is defined as an interruption of forward flow of contents within the intestinal lumen
It is among common causes of emergency admissions in infants and children all over the world. Most causes result from complications of congenital anomalies or from inflammatory conditions that affect the bowel.

Causes

A. Small bowel obstruction

- Duodenal stenosis or atresia
- Atresia or stenosis of the jejunum or ileum
- Malrotation with volvulus
- Meconium ileus
- Meconium plug

B. Large bowel obstruction

- Hirschsprung's disease
- Rectal atresia

C. Mixed (small or large bowel)

- Inflammatory lesions like tuberculosis and Crohn's disease
- Worm infestation (e.g. Ascaris lumbricoides)

D. Extrinsic causes

- Incarcerated hernia
- Vascular bands
- Intussusception.

Symptoms and Signs

- Vomiting which progresses to become bile-stained
- Cramping abdominal pain with anorexia
- Constipation
- Abdominal distension which is greater the more distal the obstruction
- Tachycardia and signs of dehydration
- Abdominal tenderness
- Hyperactive or absent bowel sounds

Investigations

- Full blood count
- Urea, creatinine and electrolytes
- Group and save
- Abdominal x-rays, supine and erect (≥ 3 air fluid levels)
- Chest x-ray (gas under the diaphragm)
- Contrast x-rays is needed for bilious vomiting and for incomplete obstruction (e.g. malrotation)

The goal of treatment is to relieve the obstruction before ischemic bowel injury occurs.
The patient should be resuscitated in the following way:

- Keep NPO
- Give Oxygen if saturations ≤ 92 %
- IV access and collect blood for investigations
- Rehydrate according to the level of dehydration
- Give maintenance fluids if patient is not dehydrated
- If patient is hypokalaemic, add potassium chloride to fluids
- Nasogastric tube for gastric decompression
- Give IV 10% dextrose if patient is hypoglycaemic
- Fluid input and output balance chart
- Broad spectrum antibiotics: Triple antibiotic therapy:
  - Crystalline Penicillin, 50 000IU/kg/dose QID IV
  - Gentamycin, 7.5mg/kg/day OD IV
  - Metronidazole, 7,5mg/kg/dose TDS IV
- Give adequate intravenous analgaesia

Once the patient is adequately resuscitated and fluid and electrolyte imbalances corrected,

- Consult a surgeon to perform a laparotomy
- Treat the underlying cause where possible

REFERENCES

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2. McIntosh N, Helms JP, Smyth LR, Logan S. Forfar & Arneil's Textbook of Pediatrics. 7th ed. Edinburgh: Churchill Livingstone; 2008.
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8.8 INTUSSUSCEPTION

Intussusception refers to the invagination (telescoping) of a part of the intestine into itself. It is the most common abdominal emergency in early childhood, particularly in children younger than two years of age.
Ileocolic intussusception accounts for 90% of all cases.

Clinical Manifestations

- Typically presents between 6 and 36 months
- Intermittent, severe, crampy, progressive abdominal pain of sudden onset
- Inconsolable crying and drawing up of legs to the abdomen
- Relative calm between paroxysms of pain
- Vomiting, non-bilious initially but later bilious as condition progresses
- Stool with red blood and mucus (currant jelly stool)
- Palpable sausage shaped mass in the right upper quadrant
- Emptiness in the right lower quadrant (Dance sign)
- Progressive weakness and lethargy
- Shock-like state with fever and peritonitis may develop

Diagnosis

- Ultrasonography which shows the classic “target sign”
- Plain abdominal X-ray to rule our perforation

Treatment

- Refer to intestinal obstruction for notes on how to stabilize the patient.
- Reduction of an acute intussusception is an emergency procedure and should be performed immediately after diagnosis in preparation for possible surgery.
- Surgical intervention is advised in patients
  - who are acutely ill
  - have evidence of perforation and
  - where radiographic facilities and expertise to perform non-operative reduction are not readily available.
- **Non-operative** reduction in patients with no evidence of bowel perforation by hydrostatic or pneumatic pressure.

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8.9 INFANTILE COLIC

Infantile colic is commonly described as a behavioural syndrome in neonates and infants that is characterized by excessive, paroxysmal crying.
For clinical purposes it is defined broadly as crying for no apparent reason that lasts for ≥3 hours per day and occurs on ≥3 days per week in an otherwise healthy infant <3 months of age.
It is a poorly understood phenomenon with no difference in incidence between males and females, breastfed and formula-fed or full term and preterm infants.

Clinical Manifestations

- It is paroxysmal and occurs mostly in evening hours
- The crying is louder, higher and more variable in pitch and more turbulent than non-colicky crying
- Associated tensing of the abdomen with drawing up of the legs
- Clenching of the fingers and stiffening of the arms
- Arching of the back
- Difficult to console with relief at times after passage of flatus or stool

On physical examination, the keys to the diagnosis are:

- Normal physical findings
- Weight gain is normal (failure to thrive should raise suspicion of another diagnosis)

Diagnosis

- The diagnosis is confirmed in retrospect after it has run its characteristic course
- Other causes of prolonged crying need to be excluded:
  - Hunger/Inadequate feeding
  - Diaper pin poking the skin/Diaper rash
  - Trauma (abusive/non-abusive)
  - Corneal abrasion or foreign body
  - Otitis media
  - Oral candidiasis
  - UTI
  - Meningitis

Treatment

- The goal of management is to help parents cope with the child's symptoms and prevent long term sequelae in the parent-child relationship
- Drug treatment generally has no place in the management of colic
- Parental support is the mainstay of management
- Parental education and support should include:
  - Reassurance that it is common and usually resolves spontaneously by three to four months of age
  - Reassurance that the infant is not sick.
  - Education that colic is not caused by something they are doing or not doing
  - Providing tips and techniques to soothe baby
  - Reassurance that the infant is difficult to soothe. This prevents the parents feeling like they have failed
  - Encourage parents to access additional caretakers when they are overly tired or stressed
- Parents should experiment with several soothing techniques and continue with those that are helpful. Some techniques include:
  - Rocking the infant
  - Changing the scenery
  - Providing a warm bath
  - Rubbing the infant's abdomen
  - Playing an audiotape of heart beats
  - Providing 'white noise'

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9.1 HYPOXIC ISCHAEMIC ENCEPHALOPATHY (HIE)

Hypoxic Ischaemic Encephalopathy (HIE is an acquired syndrome of acute brain injury characterized by neonatal encephalopathy (NE) and evidence of intrapartum hypoxia.

NE is characterized by an abnormal level of consciousness, abnormal tone and abnormal primitive reflexes. Abnormal breathing and seizures may also occur.

Features suggestive of intrapartum hypoxia

- An acute intrapartum event
- Fetal bradycardia or reduced variability on CTG
- Meconium stained liquor
- Prolonged second stage
- Need for resuscitation at birth for 10 minutes or longer
- a 5-minute Apgar score < 7
- Acidosis on cord or neonatal blood in the first hour of life (defined as pH < 7 or Base Excess < - 10).

NOTE: The presence of features that suggest intrapartum hypoxia does not exclude other causes of encephalopathy.

ALL babies who required bag mask ventilation at birth should be observed for a **minimum of 24-48 hours** even if seemingly well.

Immediate management on admission

Manage A- airway, B- breathing and C- circulation

Check glucose, give glucose at 3mls/kg if < 2.6 mmol/L

Document congenital anomalies

Document the Thompson HIE score – before 5 hours of age, then daily for 7-10 days

If there are signs of encephalopathy:

- Keep nil by mouth
- Commence IV 10% dextrose at 40 ml/kg/day
- Apply the aEEG as soon as possible if available

Three stages of Encephalopathy

Stage 1(mild): Irritability, increased tone, poor sucking and exaggerated Moro reflex

Stage 2 (moderate): Lethargy, decreased tone and primitive reflexes. Often with seizures

Stage 3 (severe): Stupor or Coma, flaccid tone and seizures often clinically less apparent.

Initial investigation and management of infants with moderate – severe HIE should include:

Lab tests

Blood gas

FBC

Electrolytes; Sodium, Potassium, Calcium, Magnesium

Urea, creatinine

Septic screen (blood cultures/ CRP/ lumbar puncture)

Cerebral function monitor, aEEG

If available, apply aEEG to all infants with signs of HIE as soon as possible and monitor for 24 hours

Discuss any abnormal traces with consultant and treat any seizures (see algorithm below)

Cranial ultrasound

Performed at least on Day 1 and again at Day 7-14

MRI

Most reliable guide to prognosis and diagnosis available. It is best done at 7-14 days of age.

Clinical management of infants with moderate – severe HIE

Temperature

- **Avoid overheating** infants at any stage, maintain normothermia
- Septic screen (blood cultures/ CRP/ lumbar puncture) and commence 1st line antibiotics (Benzylpenicillin and Gentamicin)
- Discontinue antibiotics if sepsis excluded with normal CRP/ negative blood cultures at 36-48 hours

Ventilation

- Aim for normal PaCO2 and oxygen saturation (Targets: PaCO2 35-45mmHg; Preductal Sats 90-95%)
- If oxygen is needed and respiratory effort is good, nasal cannula or nasal CPAP (if tone is reasonable) are often adequate
- Ventilate if apnoea or respiratory acidosis with pH < 7.25 and inform senior clinician

Blood pressure

- Monitor blood pressure and keep it in the normal range (Mean arterial pressure [MAP] = 40-55)
- Avoid fluid boluses (unless hypovolaemia is suspected – in which case give one fluid bolus of 0.9% sodium chloride at 10mls/kg IV)
- Metabolic acidosis alone, is not an indication for a fluid bolus
- Treat normovolaemic hypotension with inotropes (central access required)
- An echocardiogram to assess cardiac function can inform fluid and inotrope management

Haemoglobin

- Transfuse if significant anaemia

Transfusion thresholds:

Hb < 10g/dL if on CPAP

Hb < 11g/dL if FiO2 < 0.3 ventilated

Hb < 12 if FiO2 > 0.3 ventilated

Coagulation management

- Check INR/PTT/Fibrinogen in severely anaemic or bleeding infants (e.g. subaponeurotic haemorrhage)
- Treat active bleeding with vitamin K (1-2mg/kg IV) and FFP (15mls/Kg over 30 minutes)
- consider cryoprecipitate if poor response or low fibrinogen

Fluid balance, acidosis and metabolic management

- Initially fluid restrict to 40 ml/kg/24 hours Day 1- 10% dextrose solution and monitor blood glucose

Common electrolyte abnormalities

Hypocalcaemia
Hypomagnesaemia
Hyponatraemia

General Principles

Intrinsic renal injury and SIADH commonly occur

If blood glucose low, increase glucose concentration of IV fluid rather than increasing volume (may require central IV access)

Catheterise if urinary retention or oliguria (<1ml/kg/hr)

Avoid:

- Potassium containing fluids
- Use of sodium bicarbonate

Feeding

- Beware of necrotizing enterocolitis
- Hold feeds for 24 hours, then introduce feeds slowly, preferably breast milk via nasogastric tube
- Increase by 12-24mls/kg/day during the first 3 days and by up to 30ml/kg/day thereafter if abdomen exam normal

When to stop resuscitation or offer withdrawal/redirection of care

- Stop resuscitation after 10 minutes of asystole
- Consider stopping resuscitation if no spontaneous respiration after 20 minutes – first ensure that infant is not hypocarbic and has not been exposed to maternal opiates/benzodiazepines
- Persisting isoelectric or severely abnormal aEEG/ coma/ apnoea
- Consider additional factors such as cranial ultrasound and MRI findings and multiorgan failure.
- Discuss with senior clinician before stopping resuscitation or redirecting care considered

Communication with Parents

- Explain the clinical condition and the potential for other causes
- Explain the management
- Document the parent's version of events
- Give opportunity for questions
- Prepare them for a potential poor outcome if investigations and signs are suggestive

Discharge criteria

Maintaining temperature, saturating well in room air, managing all feeds orally and gaining weight.

Seizures stopped or controlled on maintenance anticonvulsant

Document

- ***admission and discharge weight and head circumference***
- ***highest Thompson score and HIE grade***

Follow-up

- ***Review feeding/ growth/ presence of seizures at 1-2 weeks post discharge***
- ***Review monthly thereafter if growth adequate***
- ***Refer early for physiotherapy/ hearing screening/ nutritional support***
- Arrange neurodevelopmental follow up at 20 (18-22) weeks

Infants with mild encephalopathy can be fed with higher volumes

REFERENCES

1. Zambia Neonatal Protocols and Drug Doses. 2016
2. Zambia Neonatal Protocols for District Hospitals

9.2 PREMATURITY

Prematurity is the delivery of an infant born before 37 completed weeks of gestation. It is categorized by gestation age or birthweight.

Gestation age

- Late preterm infant - infant born between 34 and 36 weeks gestation
- Moderately preterm infant - infant born between 32 and 34 completed weeks of gestation
- Very preterm infant - infant born between 28 and 32 completed weeks of gestation
- Extreme Preterm infant - born before 28 weeks

Birth weight

- Low birthweight (LBW) - 1500g- < 2500g
- Very low birthweight (VLBW) - 1000g- <1500g
- Extremely low birthweight (ELBW) - <1000g

Resuscitation and management at birth

- Most preterm babies do not need extensive resuscitation at birth
- If resuscitation required – follow the Zambian Neonatal Resuscitation Algorithm
- Most babies respond well to assisted transition

<32/40 or <1.5kg – place in a plastic bag WITHOUT drying first

- Ideally practice Immediate Kangaroo Mother Care by placing the baby in the plastic on the mother's skin
- Practice early delivery room CPAP where possible

Thermal Protection

- Kangaroo Mother Care is the ideal method of thermal protection and should be started as close to birth as possible
- If mother is not available, the father, grandmother or other close relative can step in to assist if willing
- Incubators can be used for periods of time where the mother/ relative is resting/ eating
- CPAP/ respiratory support is not a contraindication to KMC
- Temperature should be closely monitored and maintained 36.5C – 37.5C

Respiratory Support

- CPAP should be started early for small babies (<1.5kg) with respiratory distress, or any preterm baby with significant work of breathing
- Mild hypoxia in bigger preterm infants can be treated with low flow nasal prong oxygen with close monitoring and escalation to CPAP
- Caffeine is the preferred respiratory stimulant in preterm babies
- Where caffeine is not available, load with Aminophylline 8mg/kg IV in all babies <1.5kg (give as infusion) then maintenance dose 2.5mg/kg BD
- Consider surfactant replacement (discuss with consultant)
- If CPAP failure and mechanical ventilation is available, discuss with consultant and consider gestational age and weight

Maintain blood sugars, hydration and provide nutrition

- May loose up to 15% of body weight in the early postnatal period
- Maintain blood sugars 2.6-7mmol/l

Table 9. Initiate fluids as below:

Birth Weight(g)	Fluids	Day 1 (ml/kg/day)	Glucose (mg/kg/min)
<1000	5% Dextrose	90	3.2
1000 – 1199	10% Dextrose	80	5.6
1200 – 1499	10% Dextrose	70	4.2
>1500	10% Dextrose	60	4.2

- Commence feeds on day 1 of life with maternal expressed breastmilk (EBM) wherever possible
- Babies <1.5kg – commence feeds at 24ml/kg/day as 2 hourly boluses and increase daily. Provide remaining daily fluid requirements as IVI until 150ml/kg/day reached. Where maternal milk is not available, donor EBM is preferred
- Stable Babies >1.5kg – commence full volume EBM feeds on day 1 of life (60ml/kg/day). Where maternal milk not available, consider preterm formula
- For additional information consult the Zambian Neonatal Protocols

Treat infection

- Preterm babies are at increased risk of neonatal sepsis and poor outcomes as a result of infection
- Babies may be born prematurely due to infections such as syphilis or chorioamnionitis
- Have a high index of suspicion, and commence antibiotics if risk factors are present, even if the baby seems initially well
- First line antibiotics – Benzylpenicillin and Gentamicin

Routine care

- Vitamin K 1 500 micrograms IM once in babies weighing less than 2.5kg
- Eye care
- Cord care
- Developmental care – limit noise, provide skin to skin (KMC) wherever possible and nest when not in KMC
- Monitor for jaundice and treat early (preterm babies are at higher risk of kernicterus at lower serum bilirubin levels as compared to term babies)
- Supplements:
    - Multivitamin syrup – from day 14 or 48 hours after establishing full feeds. Prescribe according to vitamin D content
    - Iron syrup – 2-3mg/kg/day (max 6mg/kg) from day 28 of life upto 2 years of age
    - Consider breastmilk fortification in babies <1.5kg

Complications

- Respiratory Distress Syndrome
- Bronchopulmonary dysplasia
- Intracranial Haemorrhage
- Patent Ductus Arteriosus

- Necrotising enterocolitis
- Anaemia of prematurity
- Apnoea of prematurity
- Retinopathy of prematurity

9.3 RESPIRATORY DISTRESS SYNDROME

Syndrome of surfactant deficiency leading to increased work of breathing, hypoxia, and if untreated is a significant cause of preterm mortality.

Clinical Features

Appear within minutes of birth, however, they may not be seen for several hours. These may include:

- Cyanosis
- Apnoea
- Nasal flaring
- Rapid breathing
- Shallow breathing
- Grunting

Investigations

- **Usually clinically diagnosed**
- Blood gas analysis shows hypoxia and acidosis
- Chest x-ray shows a "ground glass" appearance to the lungs that is typical of the disease. This often develops 6 to 12 hours after birth.
- Lab tests help to rule out infection

Treatment

- Maintain normal temperature and blood sugar
- Early CPAP (or HFNC)
- Consider surfactant replacement (using LISA or INSuRE)
- Aim for oxygen saturations of 90-95% (avoid hyperoxia)
- Caffeine (or Aminophylline if not available)
- Intubate and ventilate for preterm infants who are not coping on CPAP or remain apnoeic

9.4 NECROTISING ENTEROCOLITIS (NEC)

Necrotising Enterocolitis (NEC) is a life-threatening condition that affects the bowel of infants, the majority are preterm

Patients are severely ill, with an acute abdomen.

NEC should be considered in any preterm infant with abdominal distention or term infant with risk factors.

The use of breast milk is associated with a lower incidence of NEC.

Clinical features

- NEC can occur in the first few days of life in term patients, but more commonly from the second week onward in preterm infants.
- Onset can be insidious but patients with a rapid onset deteriorate quickly
- Lethargy, abdominal distension, absent bowel sounds, bloody stools, vomiting and discoloured abdominal wall are the cardinal signs
- Other signs of illness like tachycardia, respiratory distress and poor perfusion may be present.

Preventive measures

- Start small volume feeds early to stimulate GIT mucosal development
- Cautious advancement of feeds in small premature infants and those with risk factors (e.g. neonatal encephalopathy)
- Feeding with breastmilk

APPROACH TO NECROTISING ENTEROCOLITIS (NEC)

Neonate with distended abdomen/ not tolerating feeds
Assess for possible Necrotising Enterocolitis (NEC)
Risk factors – prematurity, infections, PDA, polycythaemia, evidence of intrapartum hypoxia

Abdomen mildly full but not tensely distended or tender, active bowel sounds
All vital signs stable, **clinically well**
Not vomiting
Regular normal looking stool (no bleeding)

Consider other aetiologies including
Feeding intolerance – progress feeds more slowly, give smaller more frequent bolus feeds or continuous feeds
CPAP belly – leave OGT open for 30 minutes pre feed/ gently remove excess air pre-feed
Continue to monitor closely for any developing illness or worsening distension

Stage 1. Suspected NEC: **clinically ill**.
Abdominal X-ray: normal to mild distension of bowel loops

Stage 2. Definite NEC: mild or moderate systemic illness, absent bowel sounds, abdominal tenderness, metabolic acidosis, low platelets.
Abdominal X-ray: pneumatosis intestinalis or portal venous gas.
Stage 3. Advanced NEC: severely ill, marked distension, signs of peritonitis, hypotension metabolic and respiratory acidosis, DIC.
Abdominal X-ray: pneumatosis intestinalis, portal venous gas or pneumoperitoneum

- **Initial Management**
- Resuscitate: Support breathing and intubate if required.
- Assess circulatory status; apply fluid and inotropic support judiciously
- Stop enteral feeds immediately. Insert naso-/orogastric tube, leave on free drainage, monitor gastric output
- Strict input-output monitoring
- Investigations: Abdominal X-ray, FBC, CRP, blood culture. Consider U&E, ABG and INR/PIT.
- Start broad-spectrum antibiotics including adequate gram-negative cover
- Serial abdominal examination is important

Stage 1
- Reassess abdomen in 24-48 hours. Restart small volume feeds cautiously if examination is normal

Stage 2 and 3
- Keep NPO for 5-10 days. Start total parental nutrition
- Patients with advanced disease may require repeat FBC, and clotting profile. Blood products (i.e. packed RBC, platelets or FFP) should be transfused if anaemic and/or DIC present
- Paediatric surgeons should be consulted after initial stabilization and daily as may require surgical intervention/ peritoneal drainage.
- Avoid Indomethacin/ NSAIDS
- Restart feeds slowly (20-30ml/kg/day)
- Continue antibiotics until septic markers are normalized

Fig 9. Approach to necrotising enterocolitis

9.5 SEPSIS IN NEONATES

Neonates are particularly susceptible to bacterial sepsis. The highest incidence being in the very low birth weight infants.

Early-onset sepsis

- < 72 hours after birth
- Results from vertical exposure to bacteria before and during birth

Risk factors

- Preterm infant
- Prolonged rupture of membranes > 18 hours
- Maternal fever in labour (>38 C)
- Chorioamnionitis
- Maternal colonization with Group B Strep

Infant with clinical features of sepsis

- Often non-specific
- Altered behaviour or responsiveness/ Irritability, inactivity, lethargy
- Apnoea and bradycardia/ Cyanosis or poor colour
- Respiratory distress - increase in oxygen requirement/respiratory support
- Poor feeding, poor suck, vomiting, abdominal distension
- Hypoglycaemia or hyperglycaemia
- Fever, hypothermia or temperature instability
- Hypotension / Tachycardia/ Circulatory collapse or shock/ Poor perfusion (CRT>3sec; mottling)
- Seizures/ Hypotonia
- Jaundice
- Rash

Features suggestive of Meningitis

- Tense or bulging fontanelle
- Head retraction (Opisthotonos)
- Seizures/ altered consciousness

Late – onset sepsis

- After 72 hours of birth
- Nosocomial and community acquired organisms

Risk factors

- Preterm infant
- Indwelling catheters or tracheal tube
- Prolonged antibiotics
- Damage to skin
- Community acquired (cord care/ skin care/ hand hygiene)

Investigations

- Full Blood count and differentials
- C-reactive protein
- Blood culture
- Screen for syphilis/TORCH infections
- Lumbar puncture – if blood culture positive or clinical features of meningitis
- CXR – if indicated
- Tracheal aspirate
- Coagulation screen
- Blood gas
- Placental tissue culture and histopathology

Interpretation of laboratory investigations

Features suggestive of sepsis:

- Neutropenia or neutrophilia
- Increased ratio of immature(bands): total neutrophils
- Thrombocytopaenia
- Positive blood culture
- Raised CRP (>10 mg/L)
- CSF ↑ protein, ↓ glucose, ↑ WBC, +gram stain

Treatment

Supportive care -fluids and feeds, respiratory support, manage seizures
Commence antibiotics

Early onset sepsis

1st line antibiotics
Penicillin G and gentamicin

Late onset sepsis

1st line antibiotics
Penicillin G/cloxacillin and gentamicin/amikacin
NB: consider syphilis as cause

Not responding to 1st line antibiotics

- Second line antibiotics: Cefotaxime and cloxacillin
- Third line: Ciprofloxacin/Meropenem
- Meropenem should be used if meningitis or NEC is suspected
- Vancomycin only if the patient has central lines in place and is at risk of Staphylococcus aureus sepsis.

Fig 9.1. Sepsis in Neonates

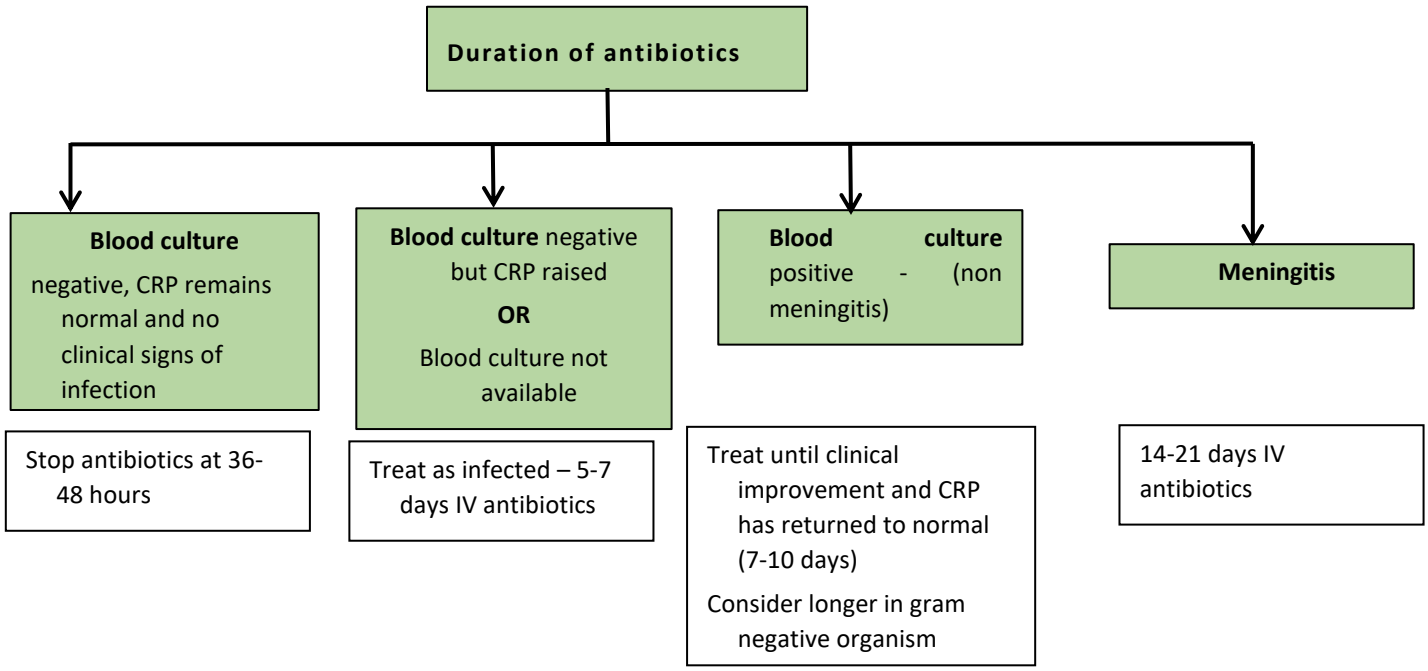


Fig 9.2. Criteria for commencing Empirical antibiotics in a baby with risk of having an early onset infection (who may appear clinically well)

2. *Absolute indication for empiric antibiotic treatment*

- Maternal invasive bacterial infection requiring antibiotics (e.g. septicaemia) – suspected or confirmed (NB: Not prophylaxis)
- Confirmed or suspected infection in twin
- Respiratory distress starting more than 4 hrs after birth
- Mechanical ventilation in a term baby
- Seizures
- Signs of shock
- Home delivery

Start antibiotics if two or more are present of:

Antenatal

- Preterm birth following spontaneous labour <37 weeks or Prelabour ROM
- ROM ≥18 hours
- Maternal fever ≥ 38ºC or chorioamnionitis

Postnatal

- Altered behaviour/tone/responsiveness
- Feeding difficulties (e.g. Poor feeding in a term baby) or intolerance
- Respiratory distress
- Apnoea
- Abnormal heart rate (bradycardia or tachycardia)
- Altered glucose homeostasis (hypo/hyperglycaemia)
- Metabolic acidosis
- Temperature abnormality >38º or < 36º not explained by environmental factors

NOTE: If only one risk factor is present, consider observation for 24 hrs

REFERENCES

1. Zambia Neonatal Protocols and Drug Doses. 2016

9.6 NEONATAL SEIZURES

Neonatal seizures are an abnormal synchronous electrical discharge of a group of neurons in the central nervous system.

Status epilepticus are continuous seizures lasting 30 minutes or recurrent seizures occupying 50% of the EEG recording for at least 60 minutes.

Table 9.1. Important causes

Brain damage:	Hypoxia – ischaemia, bleeding, infarction, oedema
Brain malformations	
Meningitis or encephalitis:	*Acute*: (bacterial or viral) or *Chronic*: (viral, syphilis)
Bilirubin encephalopathy	
Biochemical:	Hypoglycaemia, hypomagnesaemia Hypernatraemia, hyponatraemia Hyperammonaemia, pyridoxine dependency, other
Inborn errors of metabolism	
Drug withdrawal	Maternal opiate or cocaine abuse
Iatrogenic:	Air embolism
Familial:	Fifth day fits (benign)

Clinical manifestations

Clinical manifestation may be:

- Absent
- Subtle: eye deviation, eyelid fluttering, Bucco-lingual movement or pedalling of arms and legs
- Focal: tonic or clonic
- Generalised: multifocal rhythmic jerking, generalized posturing or myoclonic

Treatment

- Commence management using the airway, breathing, circulation approach
- Treat electrolyte and glucose abnormalities and sepsis
- Ensure adequate ventilation and perfusion. Commence CFM/Brain monitor
- Treat seizures according to the algorithm below (clinical and/or electrical) with anticonvulsants if they are recurrent or last more than 3 minutes.

Treatment considerations

1. When the seizures are controlled, keep the newborn on Phenobarbitone maintenance at 3-5 mg/Kg/day.

NOTE:

- In case of excessive sedation do not discontinue Phenobarbitone (probably the drowsiness is secondary to the underlying condition) and evaluate reduction of Phenobarbitone at lower maintenance dose with close clinical monitoring.
- In case of acute symptomatic seizures resolved by correction of the underlying etiology and not associated with increased risk of brain injury, AEDs can be discontinued gradually.
- If the infant is taking medication and seizures recur, increase the dose back to levels at which no seizures occurred and ask for neurological evaluation.
- Generally, patients who had neonatal seizures that required recommencement of
- anticonvulsants/continued AEDs; should be discharged on maintenance phenobarbital and followed up monthly at least until 3 months of age.
- The choice to taper down Phenobarbitone after 1 month should be evaluated case by case considering risk factors for recurrence of seizures.
- Paediatric neurology evaluation and EEG at 1- 3 month for infant at increased risk of epilepsy should be requested where available.

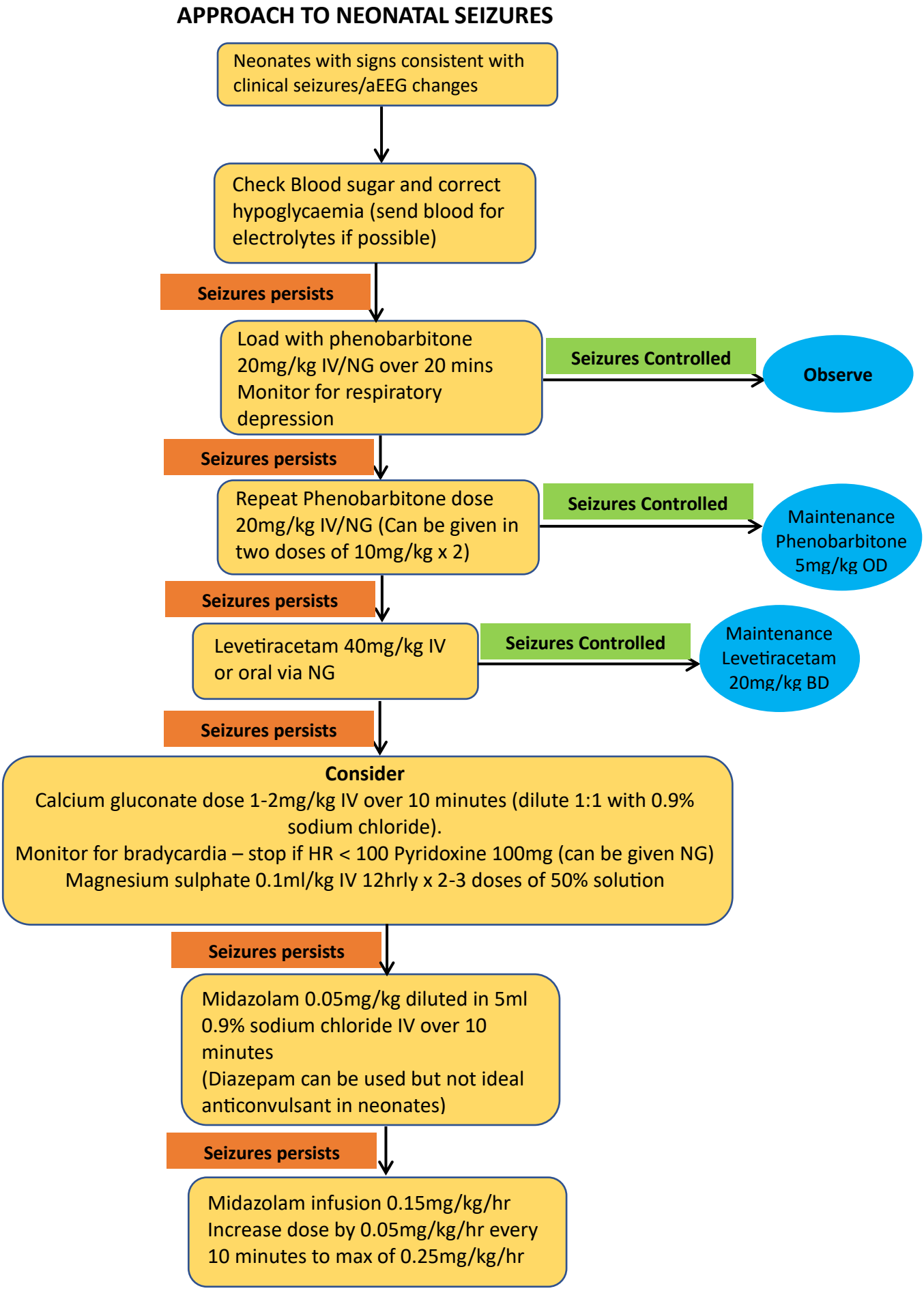


Fig 9.3. Approach to neonatal seizures

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1. Zambian Neonatal Protocols. 2016
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9.7 NEONATAL JAUNDICE

Neonatal jaundice occur in between 50-60% of normal new-borns become clinically jaundiced in the first week of life.

Jaundice may be physiological or pathological

Physiological jaundice is due to:

- Increased production of bilirubin (large erythrocyte mass with shortened life span)
- Decreased hepatic excretion of bilirubin (low hepatocyte ligandin level, low glucuronic transferee activity)
- Increased entero-hepatic circulation of bilirubin (high intestinal B-glucuronidase levels decreased intestinal motility)
- Breast feeding jaundice is thought to be due to feeding problem, which lead to a decreased intake of milk, increased entero-hepatic circulation and sometimes dehydration
- Breast milk jaundice is diagnosed in clinically well breastfed infant who remains jaundiced for several weeks following physiological jaundice. The mechanism is unknown. It is thought that breast milk glucuronidase leads to increased absorption of unconjugated bilirubin via increased entero-hepatic circulation. Diagnosis is by exclusion. Breastfeeding may be continued.

Pathological jaundice

may result from an increased unconjugated or conjugated fraction or both.

Red flags (features of pathological jaundice plus)

- Mother Rh negative
- Mother blood group O. If known check total serum bilirubin (TSB) at 6 hours post-delivery
- Baby Coombs positive
- Anaemia
- Evidence of haemolysis
- Preterm
- Acidosis, hypoglycaemia, hypoxaemia, hypothermia
- Features of kernicterus
- Bilirubin levels not decreasing despite effective phototherapy, (i.e. 17 – 34 µmol/l within 4 – 6 hours)
- Family history of pathological jaundice

Table 9.2. Neonatal Jaundice

Features	Physiological jaundice	Pathological jaundice
Clinical onset of jaundice (after birth)	> 36 hours	≤ 24 hours
Duration of jaundice	Term <10days Preterm <21 days	Term >Day 10-14 Preterm > Day 21
Peak TSB (days after birth)	Term Day 3 Preterm Day 5 – 7	Early or late
Peak TSB	< 275 µmol/l	> 272µmol/l
Rise in TSB per 6 hours		>50 µmol/l
Conjugated serum bilirubin	Only unconjugated fraction increased	>34 µmol/l
Evidence for haemolysis	No	Yes/No
Underlying illness	No	Yes/No
Hepatomegaly	No	Yes/No
Pale stool/dark urine	No	Yes/No

Management

Early onset jaundice (within 24 hours) – caused by haemolytic disease of new born (HDN) (ABO or rhesus):

- Check mother's blood group
- If the mother's blood group is O, ABO likely. Do following investigations:
  - Infants blood group and Coombs
  - FBC and peripheral smear
  - Check TSB 3 hourly
  - Start phototherapy
  - In rare cases look for other causes for haemolysis

Jaundice after 24 hours

- Exclude blood incompatibility
- Exclude obvious infection or extravasated blood/bruising
- Check feeding and infant's weight to exclude breastfeeding jaundice
- Exclude polycythaemia

- Often a cause will not be found; in term infants thought to be an exaggeration of physiological hyperbilirubinemia and in preterm infants due to the immaturity of the conjugation mechanism
- Treatment will depend on the level of the TSB which should be checked daily

Other modes of assessment

Transcutaneous bilirubin (TCB) screening

- TCB is a screening tool to select infants who require formal total serum bilirubin (TSB)levels
- TCB screening decreases the number of heel pricks and readmissions for phototherapy
- Do not rely on TCB monitoring in severe hyperbilirubinemia
- TCB should not be used within 24 hours of phototherapy or an exchange transfusion
- Localised oedema and decreased tissue perfusion affect accuracy of TCB measurements
- Technique**:**
  - Ensure all staff are properly trained on the TCB device
  - Calibrate the TCB device daily in accordance with the manufacturer's specification
  - Preferred sites are inter-scapular (for infants < 35 weeks), forehead or sternum
  - Measurements should not be made over bruised skin, areas covered by hair or birthmarks
  - Measure TCB by gently pressing the tip of the device over the examination site then press the trigger button until the device indicates the measurement is complete
  - Take 3 - 5 measurements (device dependent) from the examination site, ensuring consistent placement of the tip and amount of pressure applied
  - If the TCB reading is within 20 µmol/L of the phototherapy line do TSB

Kramer's rule

In a resource limited country like Zambia, laboratory diagnostics may not be available in a timely manner. Clinical examination (e.g. Kramer's rule) can be used in making the decision to start phototherapy **(It should be known that clinic estimation of jaundice is very unreliable, especially in dark skinned infants)**

Kramer's rule entails visual inspection in natural light. Depth of jaundice progresses from head to toe as the level of bilirubin rises a follow:

Table 9.3. Kramer's Rule Entails Visual Inspection in Natural Light

Zone	Baby's body area	Approximate bilirubin level	Diagram
1	Head and neck	6 mg/dl (100 µmol/L)	
2	Upper body (chest)	9 mg/dl (150 µmol/L)	
3	Lower body, below the belly button and upper thighs and arms	12 mg/dl (200 µmol/L)	
4	Lower legs and forearms	15 mg/dl (250 µmol/L)	
5	Hands and feet	>15 mg/dl (>250 µmol/L)	

Initiate phototherapy for any jaundice from the chest and below.

PHOTOTHERAPY

In presence of risk factors use one line lower (the gestation below) until <1000g.

If gestational age is accurate, rather use gestational age (weeks) instead of body weight

Infants > 12 hours old with TSB level below threshold, repeat TSB level as follows:

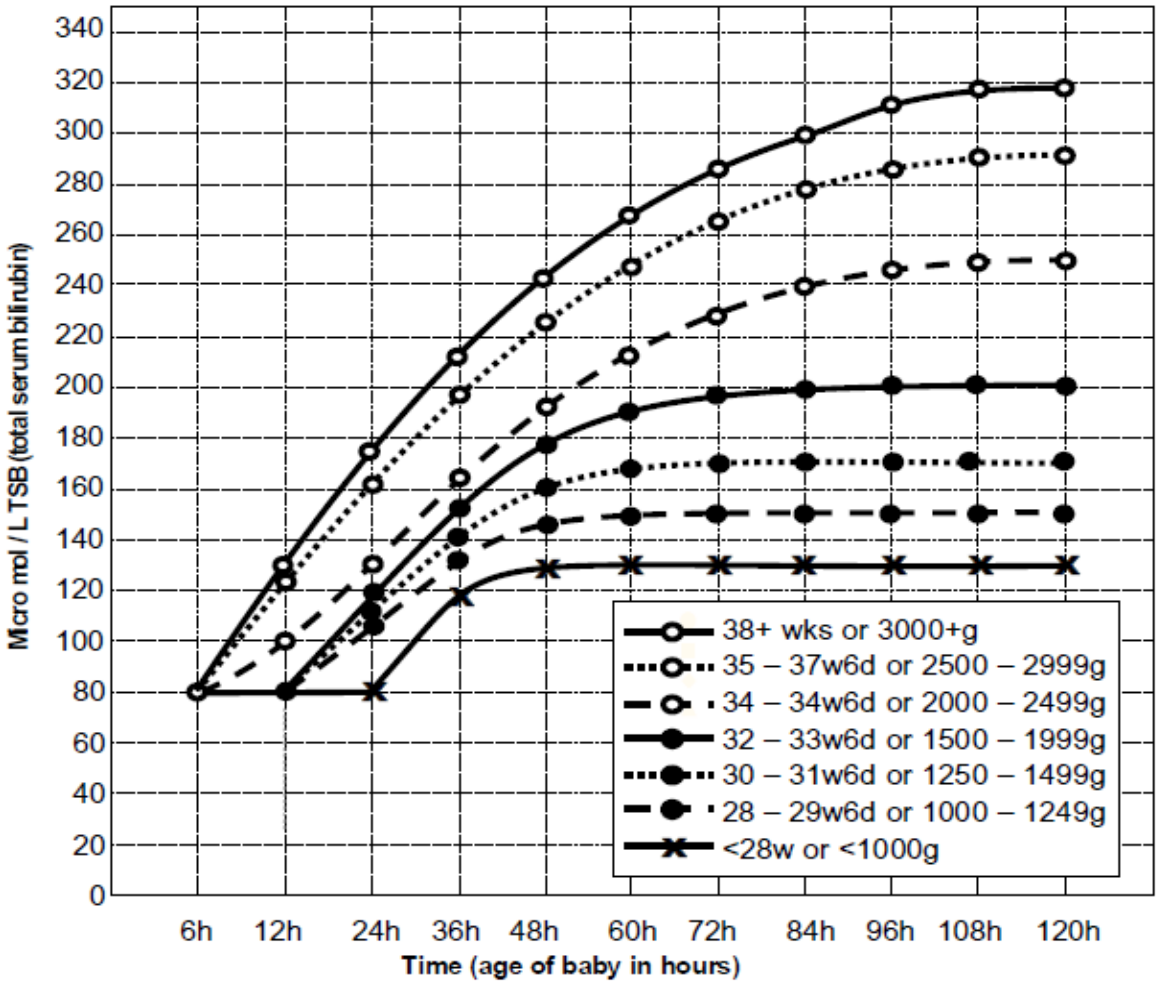
1- 20µmol/L below line:repeat TSB in 6hrs or start phototherapy and rept TSB in 12- 24hrs,
21 - 50 µmol/L below line: repeat TSB in 12 – 24hrs,
>50 µmol/L below line: rept TSB until it is falling and/or until jaundice is clinically resolving

Infants under phototherapy :

Check the TSB 12 – 24 hly but if TSB >30 µmol/L above the line , check TSB 4 – 6hly.

STOP phototherapy :

If TSB > 50 µmol/L below the line. Recheck TSB in 12 – 24hr.



Start Intensive phototherapy when the TSB is ≥ the line according to gestation or weight.

TREATMENT

Phototherapy

High intensity phototherapy (PT) reduces total serum bilirubin (TSB) and decreases exchange transfusions.

Indications

- See phototherapy guideline charts
- Indicated for unconjugated hyperbilirubinemia
- In the absence of TCB to help in decision making, DO NOT wait for TSB results before starting phototherapy (Kramer classification can be used if possible)
- A low threshold to start phototherapy in high risk infants (e.g. with sepsis, HIE etc)

Contraindications

- Congenital porphyria or a family history of porphyria
- Concurrent therapy with metalloporphyrin haem oxygenase inhibitors
- Concurrent use of drugs or agents that are photosensitisers

Technique

- Intensive PT is defined as light wavelengths between 430-490nm, delivered spectral irradiance of 30µW/cm$^{2/}$nm or higher to the greatest exposed body surface
- Position the PT unit approximately 40cm from the infant
- Measure the irradiance of phototherapy units periodically with the use of a photoradiometer
- PT units require adequate ventilation. Do not cover with blankets

Care of the infant receiving phototherapy

- Monitor temp and ensure adequate fluid intake
- Cover the eyes with gauze pads and place infant naked under lights (nappy un tied)
- Remove the eye pads during feeds and observe for conjunctivitis
- Turn infant every 2-3 hours
- In severe jaundice check the TSB 3 hourly
- Visual assessment of jaundice is unreliable once the infant is under phototherapy

Efficacy of PT

- Successful PT should produce a decline in TSB of 17-34 µmol/L within 4-6 hours and TSB should continue to fall
- Stop PT if TSB ≥ 50µmol/L below the PT line

Complications

Rashes, “bronzing”, loose stools, dehydration, hypothermia, hyperthermia and separation from mother.

REFERENCES

1. Zambian Neonatal Protocols. 2016

9.8 PROLONGED NEONATAL JAUNDICE

Defined as jaundice lasting >14 days in term infant >21 days in a preterm infant

Determine whether it is unconjugated or conjugated hyperbilirubinemia.

Unconjugated hyperbilirubinemia

- Determine whether or not the baby is breastfed
- Collect urine for MCS and reducing substances to exclude galactosaemia
- Check liver enzymes
- Exclude hypothyroidism
- Exclude haemolysis, check reticulocytes and Hb
- Hereditary enzyme defects such as Gilbert's and Crigler – Najjar syndromes are rare

Conjugated hyperbilirubinemia

- History and examination
- Liver function tests and cholesterol
- Examine stools daily. Acholic (white) stools require urgent referral to exclude biliary atresia
- Exclude infective causes
- Exclude metabolic causes
- Exclude genetic conditions

Table 9.4. Causes of conjugated hyperbilirubinemia

Causes	
Infective	***Viral:*** Hepatitis A, B, CMV, HIV, rubella, herpes simplex
	Bacterial: Syphillis, septicaemia, UTI
	Protozoal: Toxoplasmosis gondii
Biliary	Biliary atresia, choledochal cyst, Alagille's syndrome, bile plugs, cystic fibrosis
Metabolic/ genetic	Alpha 1-antitrypsin, tyrosinaemia type 1, galactosaemia, Wilson's disease, hypothyroidism, hypopituitarism, familial intrahepatic cholestasis, rotor and Dubin-Johnson syndrome
Drug/toxins	TPN
Autoimmune	Autoimmune hepatitis, sclerosing cholangitis

Table 9.5. Diagnostic workup for conjugated hyperbilirubinemia

Investigation	Test	Date Taken	Results
Haematology	FBC		Hb, WCC, platelets, reticulocytes
	Coagulation		INR, PTT, fibrinogen
	Blood group		
	Coombs		
Biochemistry	U&E		Na, K,urea, Creatinine
	TSB, conjugated bilirubin		TSB, conjugated bilirubin
	Liver functions		AST/ALT, ALP, GGT
	CMP		Ca, Mg, PO_4, Albumin
	Cholesterol*		
	Glucose		
Microbiology	Blood culture		
	Urine culture		
Virology	IgM		Rubella, HSV
	Urine CMV		
	Hepatitis A, B, C		
	HIV		
Metabolic	Urine reducing substances		
	GAL-1-PUT*		
	alpha 1-antitrypsin*		
	Plasma amino acids*		
	Urine amino acids*		
	Urine organic acids*		
	Sweat test*		
Endocrine	Thyroid functions		
Radiology	Abdominal U/S		
	CXR/spinal X-ray*		
	Radio-isotope scan*		
Histology	Liver biopsy*		
Other	Stool		
	Ophthalmology*		

*Second line investigations and drugs discuss with senior clinician

REFERENCES

1. Zambian Neonatal Protocols. 2016

9.9 SYPHILIS IN PAEDIATRICS

Syphilis is an infectious disease caused by *Treponema pallidum*.

Children experience two forms of the disease

1. Congenital syphilis which is transmitted transplacentally from the mother to the baby
2. Acquired, which is transmitted sexually

Consider Syphilis testing in the following situations

In babies:

- Failure to thrive
- Low birth weight
- Unexplained anaemia or thrombocytopenia (low platelets)
- Rashes
- Bone abnormalities
- Rhinitis (heavy nasal congestion, sometimes blood stained) – "snuffles"
- Hepatosplenomegaly
- Fever
- Not able to move an arm or leg (pseudoparalysis)
- Pneumonia
- Jaundice
- Convulsions
- Any sick baby where mum was not tested at delivery

In older babies and children: as for babies plus

- Failure to thrive
- Abnormal/ notch-shaped teeth
- Bone pain
- Blindness/ deafness
- Clouding of the cornea
- Saddle nose (flattened nasal bridge)
- Grey patches around anus and vagina
- Joint swelling
- Saber shins
- Scarring of the skin around the mouth/ anus/ genitalia
- Neurological and behavioural issues

TEST – both mother and child (NB: if maternal test positive treat the symptomatic child regardless of child's test result)

Management

Collect full blood count and pay attention to the Hb and platelet count (severely affected sick infants may require transfusion)

Consider other investigations as indicated (according to availability) – long bone x-rays, lumbar puncture, cranial ultrasound

In babies and children over 4 weeks of age

- Benzylpenicillin 50,000 units/kg every 6 hours (QDS) for 10-14 days
    - **NB: Where suspicion of neurological involvement or severe illness - 14 days**

Where the infant is presenting with sepsis like illness/ possibility of other illness/sepsis, combine with Gentamicin

- Provide nutritional support
- Screen for HIV
- Follow up syphilis tests results and ongoing symptoms with repeat at 3 months post treatment
- Refer for audiology testing on discharge

REFERENCES

1. Waseem M. Pediatric Syphilis. Medscape. Updated Dec 12, 2022. Accessed Nov 06, 2024

10.1 ADRENAL CRISIS

Severe adrenocortical insufficiency resulting in peripheral shutdown, cyanosis, tachycardia, tachypnoea, hypotension, drowsiness and coma. It can be fatal if not quickly recognised and urgently treated.

Causes

It may be due to several processes e.g.

Congenital

- Congenital adrenal hyperplasia
- Congenital adrenal hypoplasia
- Adrenoleukodystrophy

Acquired

- Acute haemorrhagic destruction of both adrenal glands in previously well children due to sepsis especially meningococcaemia
- Rapid withdrawal of steroids
- Autoimmune process (Addison's disease)
- Tuberculosis
- Birth Asphyxia
- Hypopituitarism

Clinical Features

- Vomiting/diarrhoea
- Weight loss
- Nausea
- Dehydration
- Hypotension
- Signs of shock
- Acidosis
- Convulsions
- Meningococcaemia-high fever, rash (petechiae, ecchymoses, purpura, dermal gangrene, neck stiffness
- TB-weight loss, fever, cough, night sweats, reduced appetite with positive history of TB contact

Investigations

- Random glucose (hypoglycaemia)
- Urinalysis (ketosis)
- ECG (hyperkalaemia)

- Serum Cortisol - <3µg/dL in the morning (09.00) defines low cortisol
- ACTH- often high
- U/Es (hyponatraemia, hyperkalaemia), Creatinine – (GFR decreases)
- Aldosterone - levels are often normal
- Urinary Na and Cl increased, K is decreased
- Abdominal U/S, CT scan, MRI - size of adrenal glands
- CXR- miliary picture or opacities suggestive of TB
- Gene Xpert

Treatment

- Airway
- Breathing
- Circulation – correct shock with normal saline at 20 mls/kg, then correct dehydration
- Treat hypoglycaemia
- Normal saline to correct salt deficit.
- Hydrocortisone 8mg/kg stat then
- Hydrocortisone 4mg/kg 6 hourly daily maintenance
- Treat underlying cause

Note: Dexamethasone has no mineralocorticoid activity and so should not be used to treat adrenal crisis

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10.2 DISORDER OF SEXUAL DEVELOPMENT (DSD)

Definition

- DSD is a term that encompasses a broad spectrum of conditions with atypical development of chromosomal, gonadal and phenotypic(genital) characteristics that define sexual differentiation.
- Most often present in the newborn and the adolescent.
- Newborns with DSD present with genital ambiguity or discordance between genotypic and phenotypic sex.
- Adolescents present with atypical sexual development and maturation during the pubertal years such as delayed puberty, amenorrhea or insufficient or excessive virilization.

CLASSIFICATION OF DSD

Sex chromosome DSD

- 47,XXY: Klinefelter syndrome and Variants.
- 45,X0 and 45,X0/46,XX mosaicism: Turner syndrome and variants.
- 45,X0/46,XY: Mixed gonadal dysgenesis
- 46,XX/46,XY: chimeric/ovotesticular DSD
- 47,XY.

46,XY DSD

- 46,XY gonadal dysgenesis
- 46,XY ovotesticular DSD
- Testicular regression syndrome(includes anorchidism and vanishing testis syndrome)
- LH receptor defects(Leydig cell hypoplasia or aplasia)
    - smith-lemli-opitz syndrome(7-dehydrocholesterol reductase deficiency)
- Testosterone biosynthesis defects
- 3β hydroxysteroid dehydrogenase deficiency
- 17α-hydroxylase/17,20 lyase deficiency
- Cytochrome b5 reductase deficiency
- 17β-hydroxysteroid dehydrogenase deficiency
- Androgen insensitivity syndrome.

46,XX DSD

- 46,XX gonadal dysgenesis
- 46,XX ovotesticular DSD
- Disorders of gonadal development due to androgen excess
  - 21α hydroxylase deficiency
  - 11β hydroxylase deficiency
  - Cytochrome P450 oxidoreductase
  - 3β-hydroxysteroid dehydrogenase deficiency
  - Placental and fetal aromatase deficiency
  - Fetal and maternal androgen- secreting tumors.
  - Maternal hyper-androgen exposure

Table 10. Clinical features

In addition to genital ambiguity in the neonatal period, other clinical manifestations suggestive of the presence of a DSD are:

Male genitalia with:	Female genitalia with:
• Proximal(scrotal) hypospadias, • Micropenis, • Bilateral cryptorchidism, • Testicular atrophy • Distal or medial hypospadias associated with unliteral cryptorchidism.	• Inguinal or labial mass • Enlarged clitoris with posterior labial fusion.

DIAGNOSTIC APPROACH TO A PATIENT WITH DSD

History

- Parental consanguinity
- Potential prenatal exposure to androgens, androgen antagonists
- Maternal virilization during pregnancy
- Family history of hypospadias, infertility, amenorrhea or menopause, salt-losing or unexplained infant deaths.
- Delayed puberty
- Atypical puberty

Physical examination

- Careful inspection with palpation of genitalia must be performed:
- Asses the degree of virilization/masculinization using two scales that can be used interchangeably, female use the Prader staging system and males use the external masculinization score.
- Palpation of gonads along the inguinal canal (from the labial scrotal fold to the abdomen)
- Assessment of hydration and blood pressure, blood sugar.
- Look out for any dysmorphic features. DSD Can be associated with some syndromes.

Diagnostic investigations- first line

- Karyotype analysis
- Hormone evaluation
  - 17-hydroxyprogesterone
  - Dehydroepiandrosterone (DHEA), progesterone,17-hydroxypregnenolone, 11-deoxycortisol
  - Testosterone, follicle- stimulating hormone (FSH) and luteinizing hormone (LH)
  - Basal cortisol, ACTH
- Imaging
  - Abdominal ultrasound-important to assess the presence of gonads, uterus and vagina

Diagnostic investigations- second line.

- These are used when the etiology remains unclear or need further assessment.
  - Beta-human chorionic gonadotropins, β- hcg stimulation test, assessment of testicular function based on the response to Leydig cells, measuring the levels of synthesized testosterone and its precursors and metabolites.
  - additional imaging test, recommended by surgeons or urologist such as genitogram, retrograde urethrogram or cystoscopy/vaginoscopy.
  - Laparoscopic examination with genital biopsy may be needed for assessment of potential dysgenesis.

Treatment:

Treatment of a patient with DSD aims to;

1. Confirm the diagnosis
2. Promptly rule out life threatening emergencies (CAH or hypopituitarism)

- Management of a patient with DSD should be family/patient centered with a multi-disciplinary approach:
- Rule out CAH in every child with genital ambiguity (if patient in adrenal crisis treat the shock, treat with glucocorticoids and mineralocorticoids)
- Sex assignment is a multi-step process and should be done by a multidisciplinary team with the involvement of the patient/parents
- A multi-disciplinary team should comprise of paediatrician, paediatric endocrinologist, paediatric surgeon, urologist, psychologist and social worker
- Continuous counselling and psychological care should be offered to the family throughout the process of care.

Important notes:

- **DO NOT** assign sex at birth to a child with atypical genitalia. If the child has to be named advise the parents to assign a sex neutral name and DO NOT call the child, he or she until gender is appropriately assigned
- The parents should be reassured in an empathetic manner that the child's sex shall be determined collectively after appropriate investigations are conducted

A child with atypical genitalia must be referred to a centre with a paediatrician for appropriate management.

ALGORITHM FOR EVALUATION OF DSD.

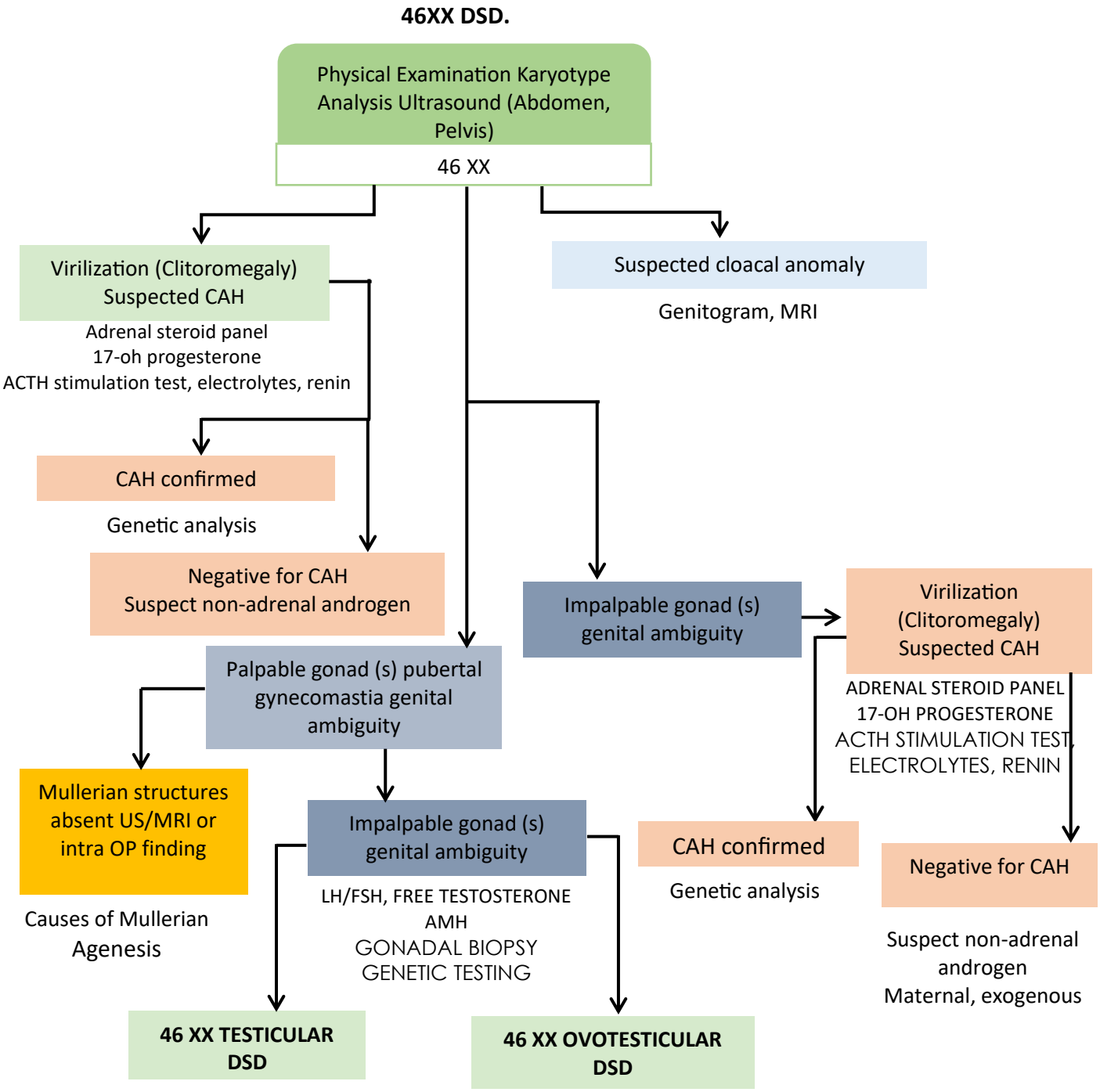


Fig 10. flow chart showing evaluation of 46XX DSD, adapted from (Rawal AY and Austin P F 2015)

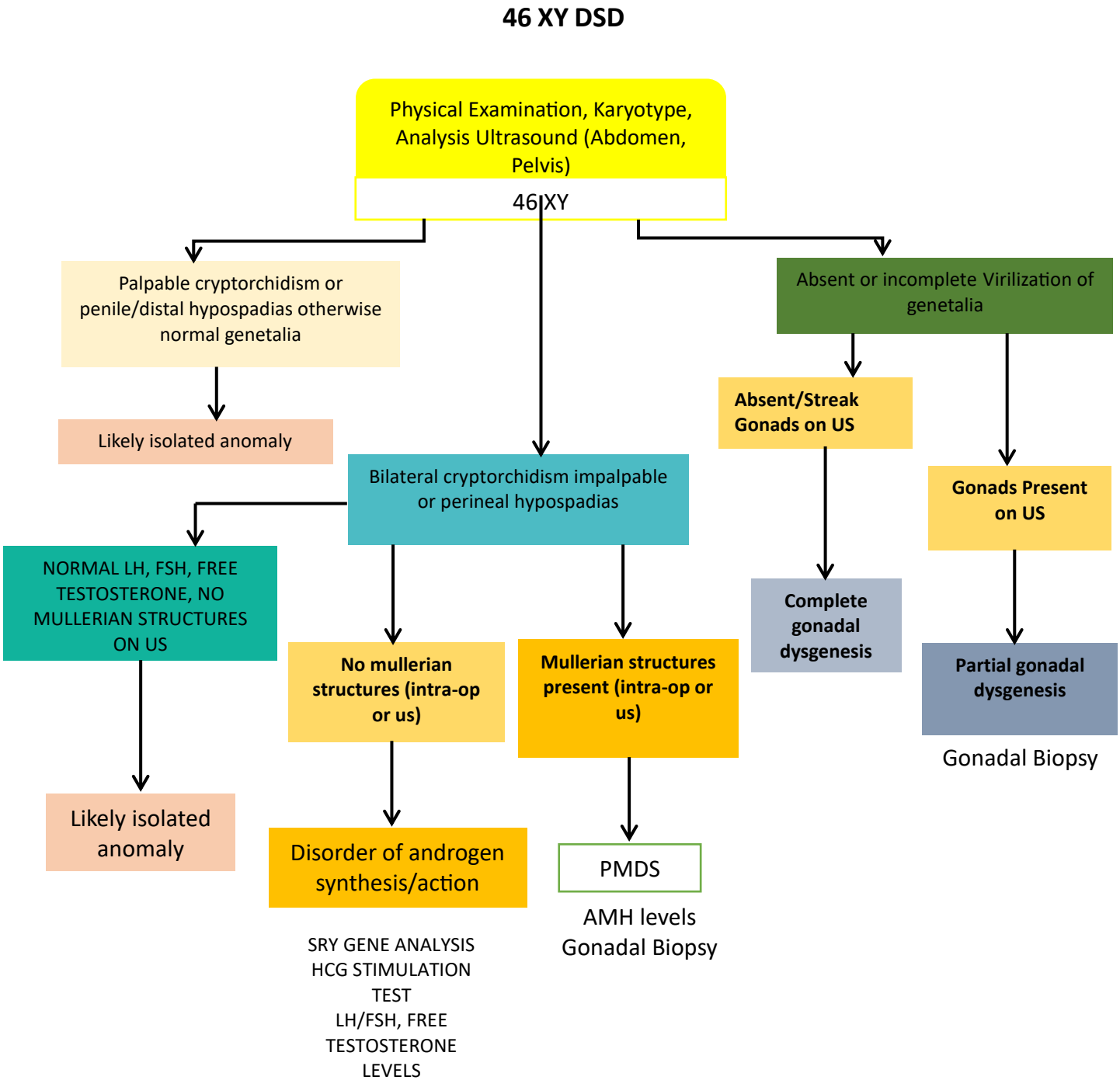


Fig 10.1. flow chart showing evaluation of 46XY DSD, adapted from (Rawal AY and Austin P F 2015)

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10.3 RICKETS

Rickets is a heterogenous group of acquired and inherited diseases resulting in disturbances in calcium and/or phosphate homeostasis and thereby, affecting the growing skeleton.

The abnormal serum calcium and phosphate levels may cause abnormalities in differentiation and maturation of chondrocytes resulting in defective mineralization of the growth plate.

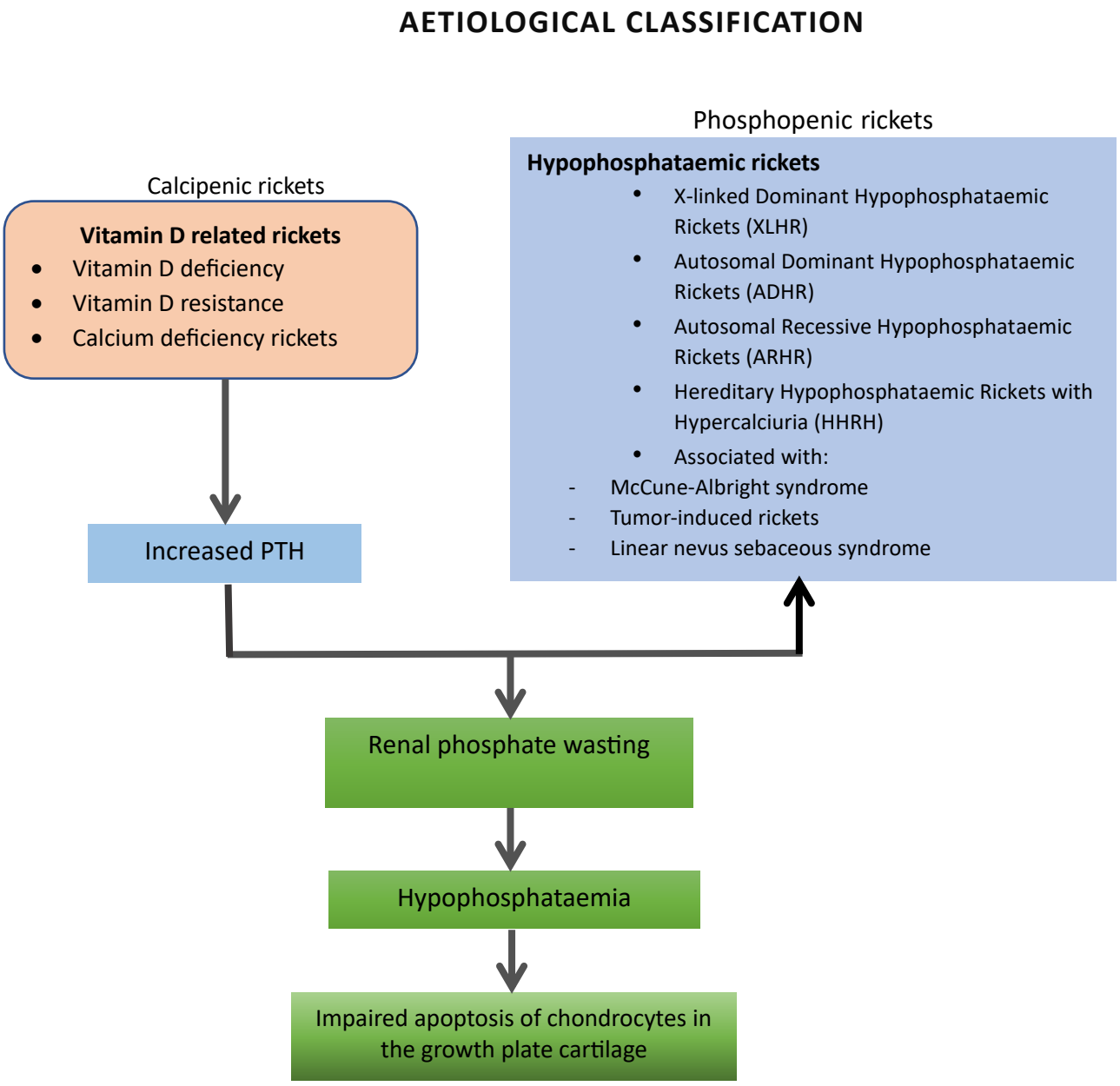


Fig 10.2. showing aetiological classification of Rickets, adapted from(Gentile C and chiarelli F 2015)

Table 10.1. Symptoms and signs

Age category	Signs and symptoms
During infancy	• Delayed milestones • Delayed dentition • Craniotabes • Irritability • Sweating • Delayed closure of anterior fontanelle • Bossing of the skull
Older children	• Waddling gait, • Harrison sulcus, • Rachitic rosary, • Genu valgum, • Genu varum (intercondylar distance more than 5cm) or windswept deformity are known to occur
Adolescents	• Seizures and bone pain are the major features • Short stature may be a feature in hypophosphataemic rickets.

INVESTIGATIONS

Biochemical investigations

- U&Es, Creatinine
- FBC
- Bone profile (Ca, Mg, phosphate, alkaline phosphatase)
- 25-OH Vitamin D levels (combined vitamin D2 and D3), $1,25(OH)_2$ vitamin D
- Urine dipstick- glucose, protein, pH (to rule out proximal renal tubular acidosis)
- Additional testing may be required depending on context

Radiological investigation

X- rays of wrist and knee (cupping and fraying of metaphysis, widening of epiphyseal plate)

Classification for serum 25(OH)D;

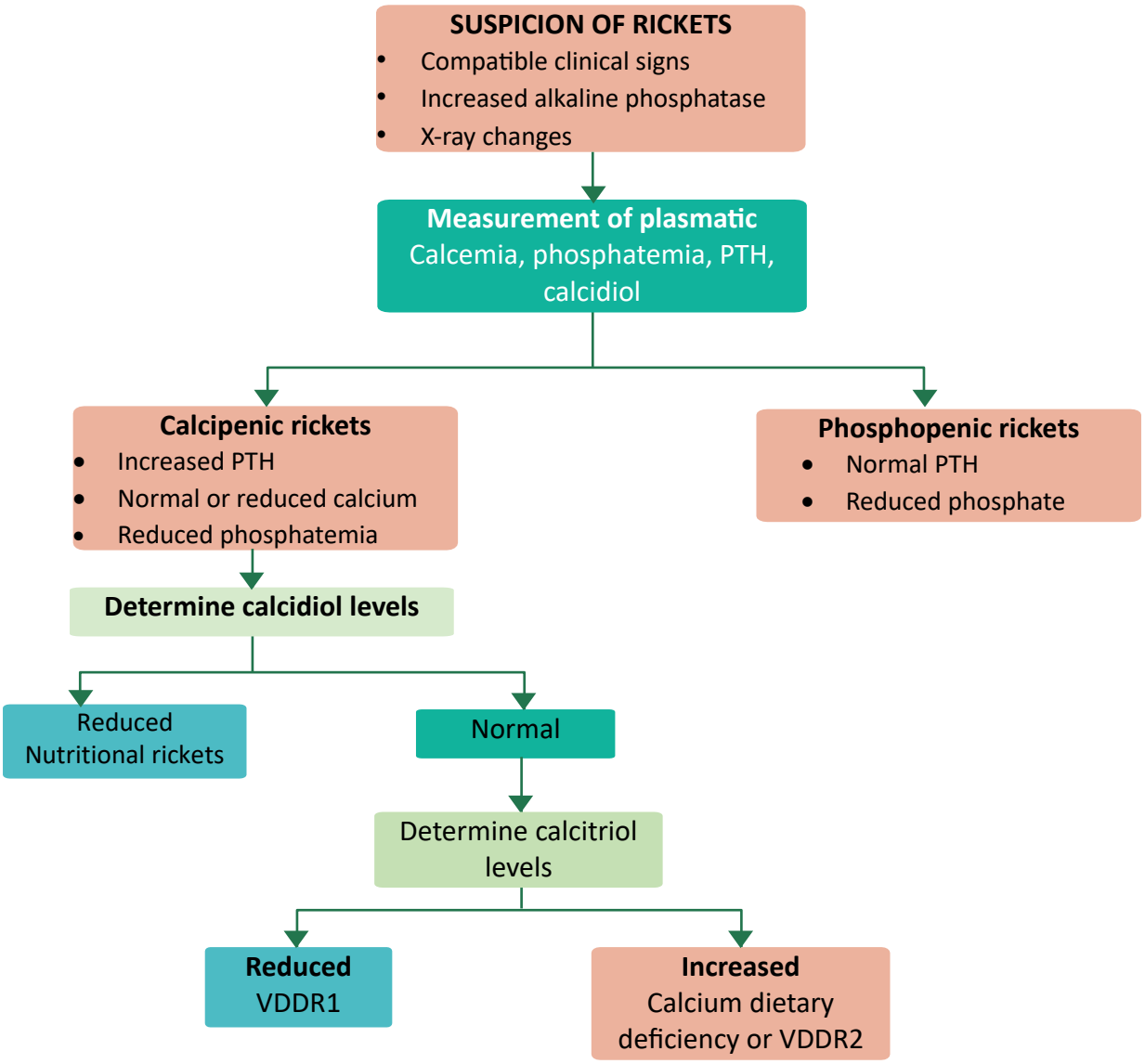
- Sufficiency: >50 nmol/L (>20 ng/mL);
- Insufficiency: 30–50 nmol/L (12–20 ng/mL);
- Deficiency: <30 nmol/L (<12 ng/mL).

Table 10.2. Approach to diagnosis of Rickets

Type	calcium	phosphate	Alkaline phosphatase	PTH	25(OH)D	$1,25(OH)_2D$
Calcipenic Rickets						
Vitamin D Variable Deficiency	L or N	L or N	H or VH	H	L	
VDDR1	L	L or N	VH	H	N	L
VDDR2	L	L or N	VH	H	N	H
Phosphopenic Rickets						
XLHR	N	L	H	N or H	N	L or N
ADHR	N	L	H	N	N	L
ARHR	N	L	H	N	N	L
ARHR	N	L	H	L or N	N	H

VDDR1: Vitamin D-dependent Rickets type 1; VDDR2: Vitamin D-dependent Rickets type 2; XLHR: X-linked Hypophosphataemic Rickets; ADHR: Autosomal Dominant Hypophosphataemic Rickets; ARHR: Autosomal Recessive Hypophosphataemic Rickets; HHRH: Hereditary Hypophosphataemic Rickets with Hypercalcuria,25(OH)D: 25-hydroxy vitamin D; $1,25(OH)_2$ D: 1,25-dihydroxy vitamin D, L: Low; N: Normal; H: High; VH: Very High.

APPROACH TO DIAGNOSIS OF RICKETS- FLOW CHART



VDDR1: Vitamin D Dependent Rickets type 1; VDDR2: Vitamin D Dependent Rickets type 2.

Fig 10.3. approach to diagnosis of Rickets, adapted from(Gentile C and chiarelli F 2015)

Table 10.3. Treatment

Age	Vit D daily dose for 12 weeks	Vit D single dose	Vit D maintenance dose (IU)	Calcium dose mg/day
<3 mo	2000 IU/day	N/A	400	500
3 -12 mo	2000 IU/day	50,000	400	500
>12 mo – 12 yr	3000–6000 IU/day	150,000	600	1000
>12 yr	6000 IU/day	300,000	600	1000

Important Notes

- Maintenance dose to be given until resolution of symptoms.
- Patients require monitoring of nutritional rickets after the onset of treatment until resolution of symptoms.
- Patients with non-nutritional rickets should be managed at a tertiary centre.
- In children with tetany or convulsions, treat with intravenous calcium gluconate at a rate of 10–20 mg/kg in 5–10 minutes, repeated as needed. (for calcium treatment in severe hypocalcaemia refers to hypocalcaemia protocol).

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10.4 PRECOCIOUS PUBERTY (PP)

Puberty is the period of physical, hormonal and psychological transition from childhood to adulthood, with accelerated linear growth and achievement of reproductive function. Puberty starts at age 8 in females and 9 in males.

Definition

Precocious puberty is defined as onset of puberty before age 8 in girls and before age 9 in boys.

Classification of Precocious puberty

Precocious puberty is classified according to the underlying physiopathology mechanisms as;

1. **Central precocious puberty**- also known as true precocious puberty and is gonadotropin-dependent, premature activation of the hypothalamic-pituitary-gonadal (HPG) axis occurs. Causes include

- Idiopathic (sporadic or familial). No cause is found in 80% of girls and 40% of boys.
- Abnormalities of the central nervous:
  - Tumors-gliomas, astrocytoma's, hcg-secreting germ cell tumors.
  - CNS trauma or injury (causes include infection, radiation, surgery)
  - Hamartomas of hypothalamus
  - Congenital disorders such as hydrocephalus and arachnoid cysts

2. **Precocious pseudo puberty or peripheral precocious puberty** is independent of gonadotrophin release and it accounts for about 20% of cases of precocious puberty, appearance of secondary sexual characteristics is independent of the of the maturation of the HPG axis. The causes include:

- Congenital adrenal hyperplasia
- Tumors: HCG producing tumors, adrenal tumors, ovarian tumors, testicular Leydig-cell tumors.
- McCune-Albright syndrome (MAS)
- Silver-Russel syndrome
- Severe hypothyroidism
- Exogenous estrogen or androgen exposure

Causes of benign variants of precocious pubertal development:

- Non-progressive precocious puberty, following early signs of puberty, the situation stabilizes or regresses rather than progressing.
- Isolated precocious thelarche: early breast development without other features.

- Isolated precocious pubarche: early pubic hair development (with or without axillary hair) without other features of puberty, this is due to adrenal androgen secretion in middle childhood.
- Isolated precocious menarche. Isolated early vaginal bleeding in the absence of other causes or features.

Symptoms and signs

The clinical manifestation of puberty includes the following;

Girls; breast development (thelarche), pubic hair (pubarche), axillary hair, apocrine odour, menarche, heterogenization of vaginal epithelium

Boys; testicular enlargement, penile enlargement, pubic hair development, axillary hair, breaking of voice
Accelerated growth in both sexes

Assessment for precocious puberty

A thorough history and examination is important.

History

- Age and rate of pubertal changes.
- Central nervous system (CNS) symptoms: headache, visual changes, seizures
- Growth history plotted on growth chart e.g. under-five card.

Examination

- Height, height velocity, target height and weight plotted on growth charts
- Assess the turner's stage of pubertal development
- Measure testicular volume
- Increase as with normal puberty in CPP and with testicular disorders
- Volume remains prepubertal in many causes of peripheral precocious puberty such as adrenal disorders.
- CNS examination- fundoscopy, cranial nerves
- Testicular and pelvic examination for masses.
- Examination for specific causes- signs of hypothyroidism, skin lesion (MAS, neurofibromatosis)

Diagnostic testing

Investigation are used selectively after a thorough clinical assessment. Tests available to refine the diagnosis further are:

Laboratory investigations

- Level of sex steroids
- Early morning testosterone in boys is higher in early puberty

- Estradiol levels are a less reliable measure of stage of puberty in girls as they are highly variable, very high levels suggest ovarian pathology.
- Gonadotrophin (LH and FSH)
- A random LH is a useful initial test for CPP. Random FSH will not distinguish prepuberty and puberty. Low or prepubertal levels with high sex steroid level are found in gonadotrophin-independent precocious puberty
- Thyroid function test
- Adrenal steroid precursors if CAH is suspected
- HCG when hcg secreting tumors are suspected

DIAGNOSTIC IMAGING:

- **Ultrasound**
  Pelvic ultrasound is essential in gonadotrophin-independent precocious puberty to detect ovarian tumors or cysts.
- **Other ultrasound**: testicular and adrenal ultrasound can help to establish diagnosis of tumors.
- **Hand and wrist X-rays for bone age**: If bone age is within one year of chronological age, either puberty has not started or has only just started. If the bone age is two years advanced then puberty has probably been present for at least a year or is progressing rapidly.
- **Brain MRI** to exclude CNS abnormalities should be performed in all cases of progressive CPP.. Pelvic MRI can be useful in girls to assess the uterus and ovaries.
- **Other tests**:
  GnRH stimulation test, LH and FSH levels are measured sequentially after GnRH is given. There is a flat response in gonadotrophin-independent precocious puberty.

PRECOCIOUS PUBERTY EVALUATION FLOW CHART.

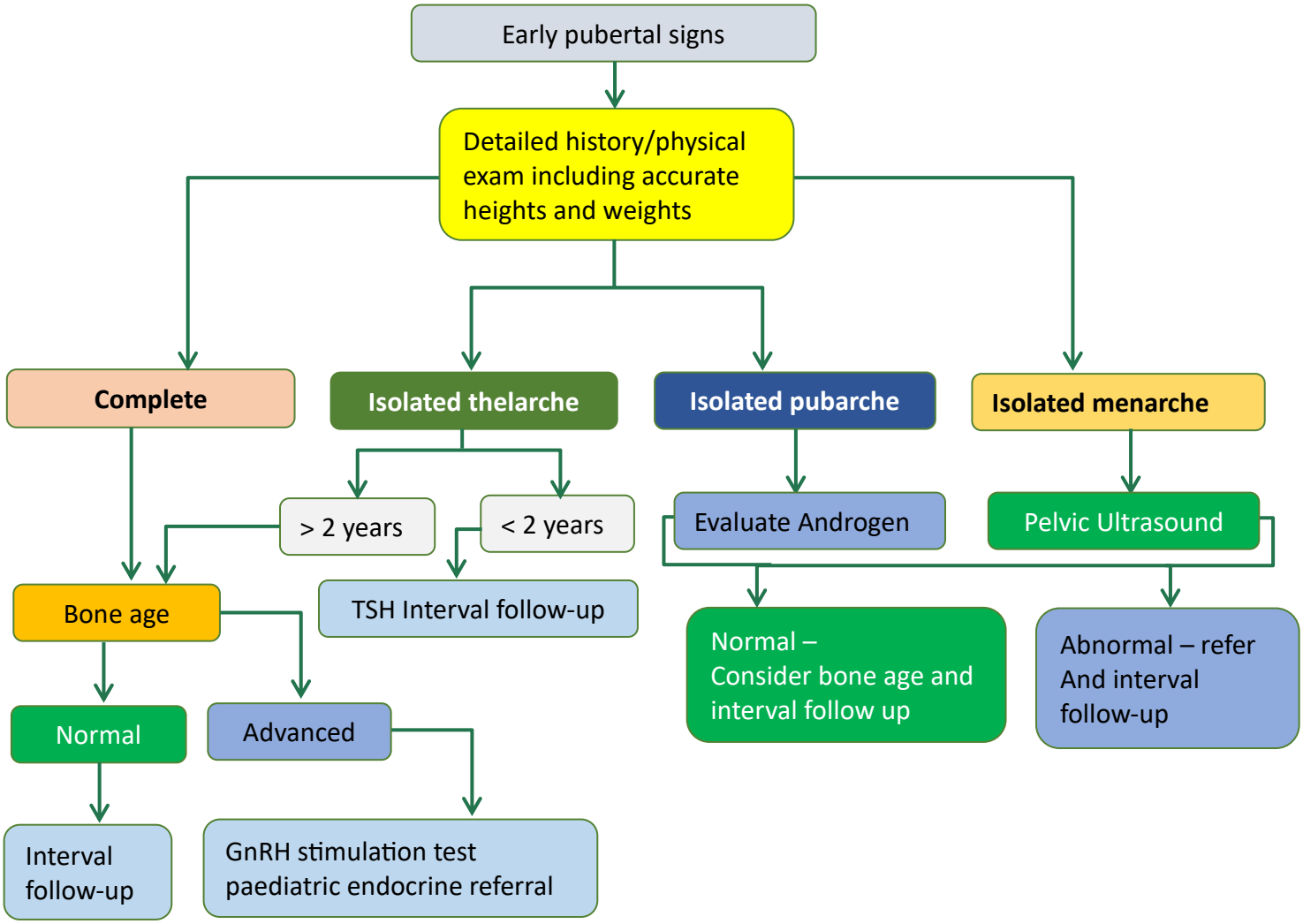


Fig 10.4. precocious puberty evaluation flow chart, adapted from clinical gynaecology, section 5, chapter 34, by Rowan P S and Murray J P.

IMAGING ALGORITHM FOR PRECOCIOUS PUBERTY

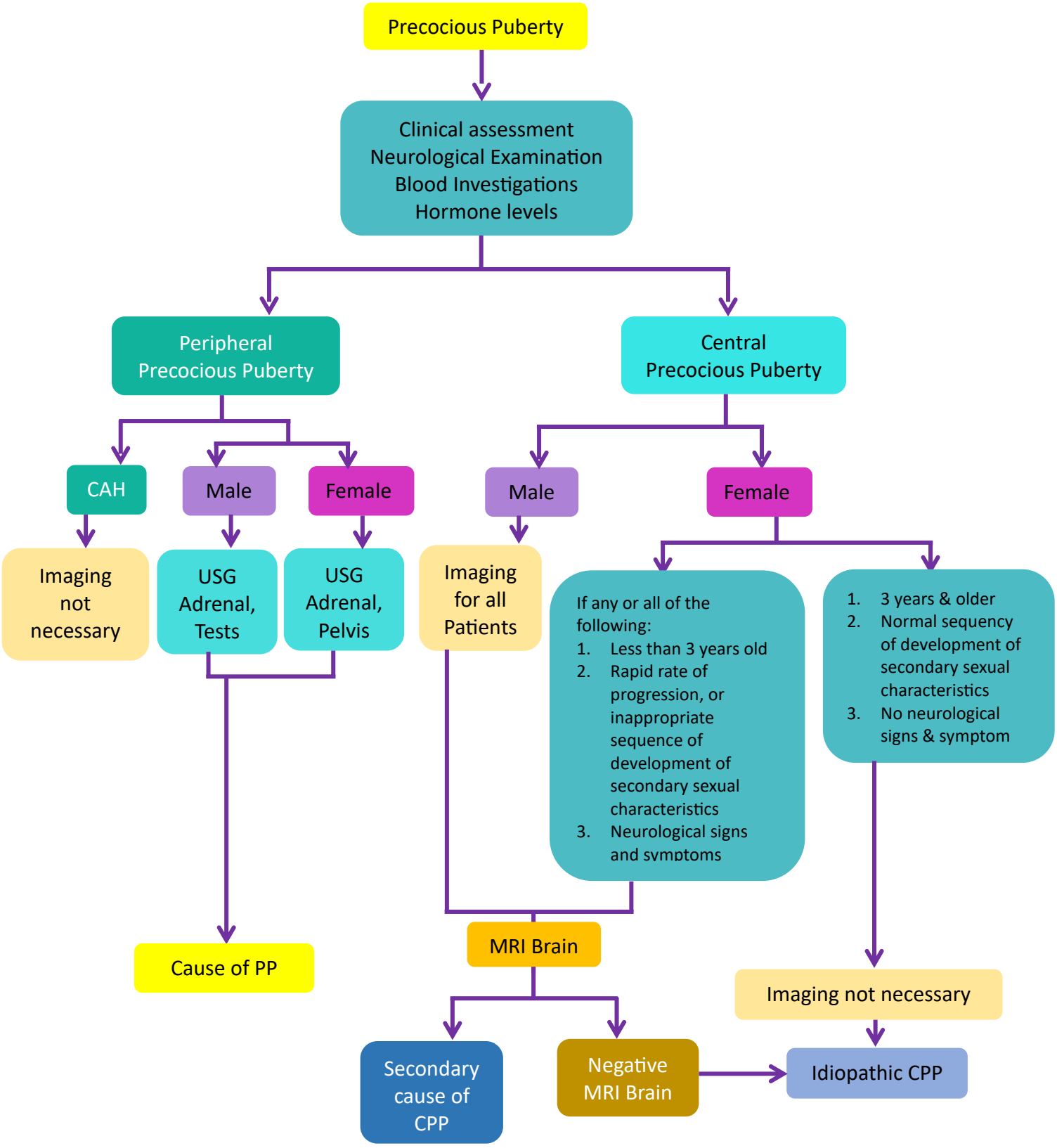


Fig 10.5. Imaging precocious puberty adopted from (Faizah Mz et al 2012)

Treatment

- Treatment of precocious puberty depends on the type and cause.
- Isolated benign variant of puberty; Premature thelarche, vaginal bleeding, premature pubarche, lipomastia without any other sign of puberty usually just requires surveillance every three to six months
- Parents need adequate counselling to alley anxiety

Peripheral precocious puberty

- Treatment is based on the cause (e.g. MAS, exogenous steroids, gonadal tumours etc)

Central precocious puberty

- The goal of therapy is preservation of adult height potential and manage psychological difficulties that come with early puberty
- The main stay of treatment is GnRHa e.g. leuprolide acetate.
  - Administered every 4 weeks
  - Monitor height velocity, breast and testicular size
  - Dosage is age dependant

Table 10.4. Central Precocious Puberty

Body Weight	Recommended Dose
≤ 25 kg	7.5 mg
> 25-37.5 kg	11.25 mg
> 37.5 kg	15 mg

Referral criteria for a patient with PP

- Every patient with suspicion of PP should be referred to a second or third level hospital with a presence of a paediatrician.
- Patients with PP should be managed by a paediatrician or paediatric endocrinologist.

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10.5 DIABETES MELLITUS

Diabetes mellitus is a metabolic disorder characterized by chronic hyperglycemia resulting from defects in insulin secretion, insulin action or both.
Types of diabetes mellitus:

- Type 1 (insulin dependent diabetes mellitus)
- Type 2 (Non-insulin dependent diabetes mellitus)
- Gestational diabetes mellitus
- Others (monogenic diabetes, maturity onset diabetes of the young, Secondary diabetes e.g. cystic fibrosis related diabetes)

The presenting clinical features of a patient with diabetes mellitus may include:

- Polyuria
- Polydipsia
- Weight loss
- Polyphagia
- Blurred vision
- Fatigue
- Tingling or numbness in hands or feet

Any one of the following with the clinical features of diabetes is diagnostic of diabetes mellitus

1. Fasting plasma glucose ≥ 7mmol/l
2. 2-hour Oral Glucose Tolerance Test ≥11.1mmol/l
3. Hemoglobin A1c ≥6.5%
4. Random plasma glucose ≥11.1mmol/l

The following readings are considered Pre-diabetes. This is a stage where the blood glucose levels are higher than normal but not high enough to be classified as diabetes.

- Impaired fasting glucose of 5.6-6.9 mmol/l
- Impaired glucose tolerance of 7.8-11.0mmol/l

Investigations

- Fasting or random glucose
- 2-hour Oral Glucose Tolerance Test
- Hemoglobin A1c
- Urine glucose and ketones
- FBC/DC
- Electrolytes
- Kidney function test
- Liver function test

- Lipid profile
- Thyroid function test
- Blood pressure measurement
- C-peptide levels
- Auto-antibody panel (anti-GAD, Zinc T8A, insulin autoantibodies)
- Screen for infections e.g., malaria, UTIs etc.

Management of hyperglycemia in a stable diabetes mellitus patient

Management of a stable diabetes mellitus has short term and long term goals which include the following.

- **Short-term goals**: Correct hyperglycemia, prevent diabetic ketoacidosis and restoring normal fluid and electrolytes balance.
- **Long-term goals**: Achieve optimal glycemic control, prevent microvascular and macrovascular complications, and improve quality of life.

Several factors can cause hyperglycemia in a diabetic patient on insulin therapy and will need to be investigated, explored, and managed.

- **Inadequate insulin dose**: If the patient isn't taking the correct dose of insulin, the blood sugar will not be regulated effectively.
- **Improper, incorrectly stored, or expired** insulin injection.
- **Dietary factors**: Consuming too many carbohydrates or not following a diabetes-friendly eating plan.
- **Lack of physical activity**.
- **Illness or infection** e.g., UTIs, URTI, etc.
- **Medications**, such as steroids or immunosuppressants, can increase blood sugar levels.
- **Stress:** Emotional stress, such as family problems, school issues, or workplace issues, can cause poor blood sugar control levels.
- **Physical trauma or surgical procedures** can lead to temporary increases in blood sugar levels

APPROACH TO HYPERGLYCAEMIA IN A KNOWN DIABETIC PATIENT

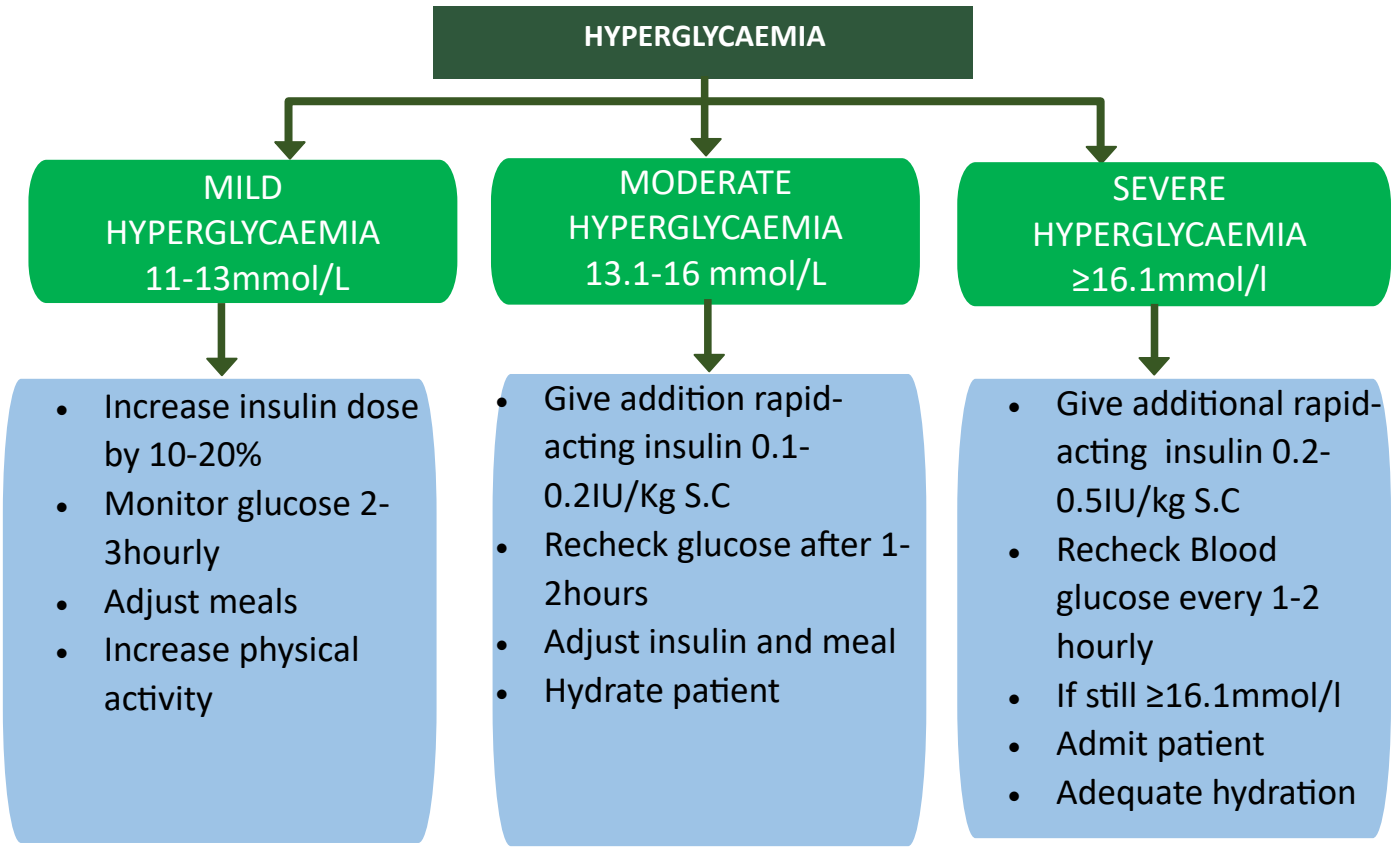


Fig 10.6. Approach to hyperglycaemia in a known diabetic patient

APPROACH TO HYPOGLYCAEMIA IN A KNOWN DIABETIC PATIENT

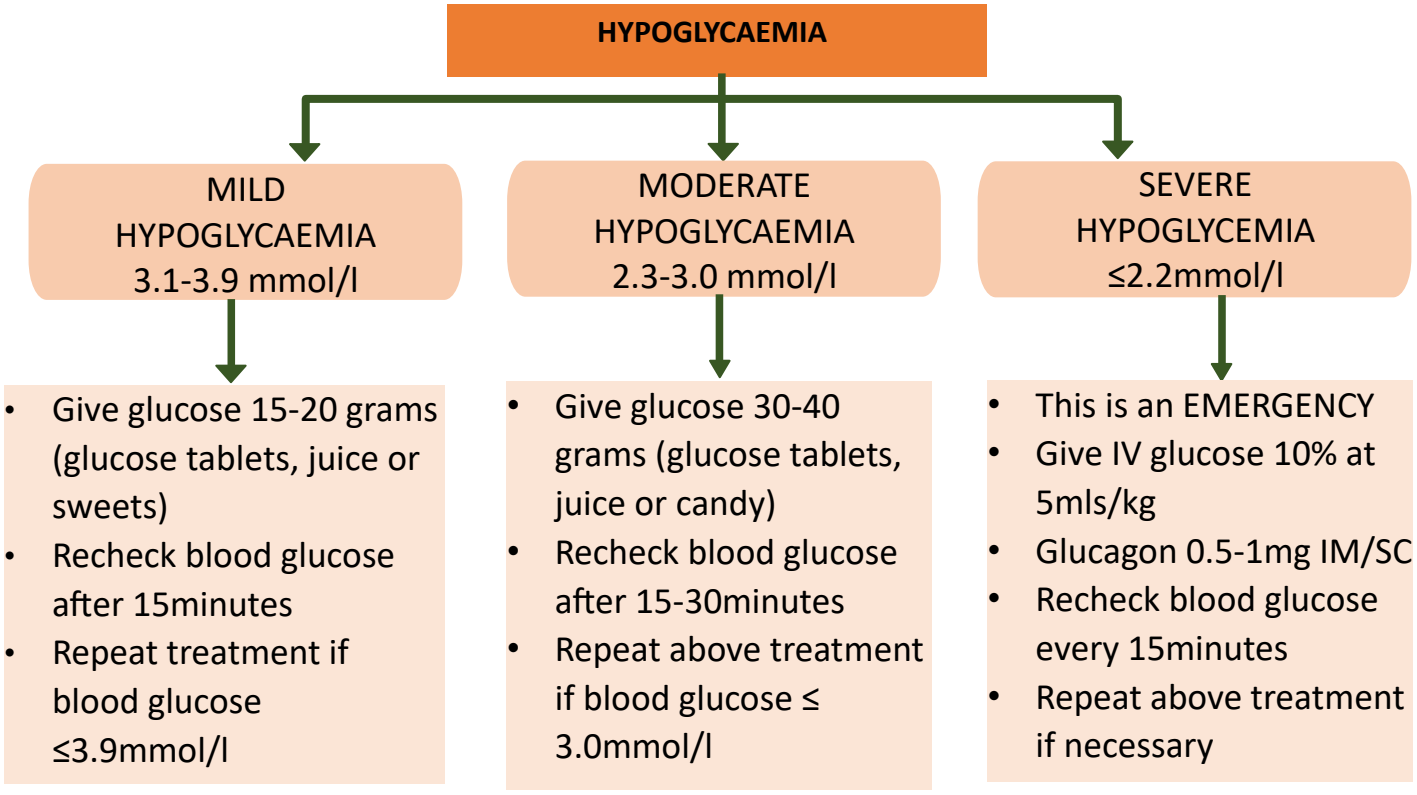


Fig 10.7. Approach to hypoglycaemia in a known diabetic patient

Guide on how to use an Insulin pump

Insulin pump is a device used for continuous subcutaneous delivery of insulin. Some of the indications for the use of an insulin pump are very young children, recurrent hypoglycemia or hyperglycemia, ketosis prone patient and wide fluctuation of blood glucose.

Clinicians should always consult the manufacturer for instructions on a particular device. The following are important steps to note:

- To use an insulin pump, start by Setting date, time and units (mg/dl or mmol). Ensure the set and tube are primed and insulin reservoir is filled.
- Next, set the insulin rate profile according to patient's needs such as daytime, nighttime or during exercise, while monitoring and adjusting the rate based on blood glucose.
- When delivering an insulin bolus, calculate dose based on carbohydrate intake and current blood glucose. Choose from different bolus types such as standard, extended or dual-wave.
- It's important to change the infusion set every 2-3 days and rotate the insertion sites to prevent skin irritation and maintain proper insulin absorption.
- Finally, be prepared to troubleshoot the pump by addressing alarms such as low battery or occlusion.

Investigations for long-term follow-up of Diabetes Mellitus patients

- Annual (yearly) investigations

1. Eye examination
2. Urine micro-albumin to creatinine ratio
3. Lipid profile
4. Electrocardiogram
5. Foot examination
6. Dental examination

- 6-monthly investigations

1. Glycated hemoglobin HbA1c
2. Renal function test

Long-term complications

Microvascular complications refer to damage to the small blood vessels e.g. (capillaries, arterioles and venules)

- Diabetic retinopathy: Damage to the retinal blood vessels, leading to vision loss and blindness.
- Diabetic nephropathy: Damage to the kidney blood vessels, leading to chronic kidney disease and end-stage renal disease
- Diabetic neuropathy: Damage to nerve blood vessels leading to nerve damage, pain, and numbness.

Macrovascular complications refer to damage to large blood vessels e.g. (arteries and veins)

- Coronary artery disease: Damage to heart arteries, leading to myocardial ischemia.
- Cerebral vascular disease: Damage to the brain arteries, leading to stroke
- Peripheral arterial disease: Damage to leg arteries, leading to claudication, gangrene and amputation.
- Atherosclerosis: Hardening and narrowing of arteries.

10.6 HYPOGLYCAEMIA IN CHILDREN AND ADOLESCENTS

Hypoglycaemia in children is a blood glucose value of less than 40mg/dl(2.2mmol/L). A plasma glucose level of less than 30mg/dl(1.65mmol/L) in first 24 hours of life and less than 45mg/dL(2.5mmol/L) thereafter constitutes hypoglycaemia in the new born.

Causes:

Aetiological Classification

Insulin-mediated disorders – Characterized by suppressed ketones and free fatty acids and a positive glycemic response to glucagon. This category includes congenital hyperinsulinism (the most common cause of hypoglycaemia in children), insulinoma, and factitious hypoglycaemia.

Fatty acid oxidation disorders – Characterized by suppressed ketones with elevated free fatty acids. The most common of these disorders is medium-chain acyl CoA dehydrogenase deficiency (MCADD).

Ketotic hypoglycaemic disorders – Characterized by elevated ketones. This category includes glycogen storage disorders (GSDs), cortisol and growth hormone deficiencies, and idiopathic Ketotic hypoglycaemia.

Disorders of gluconeogenesis – Characterized by elevated lactate. Examples of these disorders are GSD type I and fructose-1,6-bisphosphonate deficiency.

Table 10.5. Classification according to age

Age	Causes of hypoglycaemia
Neonate<48-72hrs	Prematurity, IUGR, perinatal hypothermia, sepsis, resp distress, diabetic mother, macrosomia syndrome (e.g. beckwith-Weidemann), pancreatic dysfunction.
Neonate-2yrs	Congenital hyperinsulinism (most common cause of persistent hypoglycaemia<2 years) inborn errors of metabolism, congenital hormone deficiencies (e.g. growth hormone deficiency).
Child	Accelerated starvation (previously known as Ketotic hypoglycaemia) hypopituitarism, growth hormone deficiency.
Adolescent	Insulinoma, adrenal insufficiency, eating disorder.

Signs and symptoms: Hypoglycaemia are often accompanied by signs and symptoms of autonomic (adrenergic) activation and/ or neurological dysfunction (neuroglycopenia)

Table 10.6. Signs and symptoms

Infants	Older children and adolescents	
• Irritability • Lethargy • Jitteriness • Feeding problems • Hypothermia • Hypotonia • Tachypnoea • Cyanosis • Apnoea • Seizures	Neurogenic (autonomic response) • Sweeting • Tachycardia • Palpitations • Tremor • Nervousness • Hunger • Pallor • paraesthesia's	Neuroglycopenic response • irritability • confusion • uncharacteristic behaviour. • Lethargy • Loss of consciousness • Seizures • Coma • Occasionally, transient focal neurologic deficits.

Approach to hypoglycaemia in infant and children

History

The history of a child with hypoglycaemia should include following.

- Age at presentation, this will give a clue on the possible causes.
- **Symptoms** – It is important to inquire about any symptoms of hypoglycaemia prior to the acute event.
- **Triggers** – Should include feeding history, concurrent illness, and medication exposure.
- **Duration of fasting** – How long the child fasted prior to the acute event, as well as how long the child fasts on a routine basis.
- **Specific foods** – Determine whether specific foods and nutrients may have triggered the hypoglycaemic episode(s). Symptoms after ingestion of milk products or fructose may indicate
  galactosemia or hereditary fructose intolerance, respectively.
- **Concurrent illness** – In children with unrecognized hypoglycaemic disorders, the episodes are often triggered by illnesses that interrupt normal feeding.
- **Ingestion** – inquire about possible exposure to substances that cause hypoglycaemia, such as oral hypoglycaemic agents (sulfonylureas or meglitinides), ethanol, or beta blockers
- **Perinatal history** – A thorough perinatal history is crucial and should include the birth weight, gestational age, and whether the child had hypoglycaemia at birth or in the neonatal period, including what type of treatment was necessary.
- **Prior events** – It is important to explore the child's past medical history and to review available medical records to determine whether the child had other episodes suggestive of hypoglycaemia that may have been missed or diagnosed as another condition (eg, seizure disorder).

- **Family history** – Family members with a history of hypoglycaemia or a monogenic form of diabetes suggests the possibility of a familial hyperinsulinemia disorder. unexplained infant deaths, or unexplained hypoglycaemic episodes.

Physical examination

- A thorough examination can provide important diagnostic clues.
- Anthropometrics – The child's weight and length or height should be measured and plotted on an appropriate growth chart, and the child's growth trajectory should be evaluated.
- Midline defects (eg, a single central incisor, optic nerve hypoplasia, cleft lip or palate, umbilical hernia and micro phallus or undescended testicles in boys may indicate hypopituitarism and/or growth hormone deficiency.
- Hepatomegaly is common feature of the glycogen storage disorders.
- Macroglossia, abdominal wall defects, or hemihypertrophy may indicate Beckwith-Wiedemann syndrome.
- Hyperventilation may be a clue to metabolic acidosis from an inborn error of metabolism or ingestion.
- Hyperpigmentation suggests primary adrenal insufficiency

Investigation of hypoglycaemia

Critical blood sample: Blood taken for a diagnostic screen is only useful if taken when the patient is hypoglycaemic (glucose < 2.6 mmol) and should include the following:

- Glucose
- Insulin
- c-peptide
- Cortisol
- Growth hormone
- Lactate
- Free fatty acids
- Amino acids
- Ketone bodies (hydroxyl butyrate and acetoacetate)
- Urine The critical urine sample should be obtained at the same time and tested for organic acids. The sample should also be tested for ketones if blood ketone testing is not available.
- Organic acids
- **Important**: collecting of a critical sample should not delay management of patient, and centres that are unable to do above investigations should aim at just treatment of hypoglycaemia and refer to next level facility if indicated.

NOTE: In hypoglycaemic states and in the absence of ketones, it is important to look at free fatty acids (FFA). Normal FFA suggests hyperinsulinism and raised FFA suggests a fatty acid oxidation defect. Hypoglycaemia in the presence of urinary ketones suggests either a counter regulatory hormone deficiency or an enzyme defect in the glycogenolysis or gluconeogenesis pathways.

Treatment

- Hypoglycaemia should be treated promptly after obtaining the critical intravenous samples by intravenous infusion of 5 ml/kg of 10% dextrose followed by adequate glucose infusion to maintain euglycaemia.
- Severe hypoglycaemia can be reversed by the injection of glucagon: glucagon 0.5 mg < 12yr, 1.0 mg for ages >12yr or 10-30 mcg/kg body weight. Glucagon is given intramuscularly or subcutaneously.
- Treatment should also be aimed at the underlying cause of hypoglycaemia. Fig 35 shows flow chart for the emergency management of hypoglycaemia

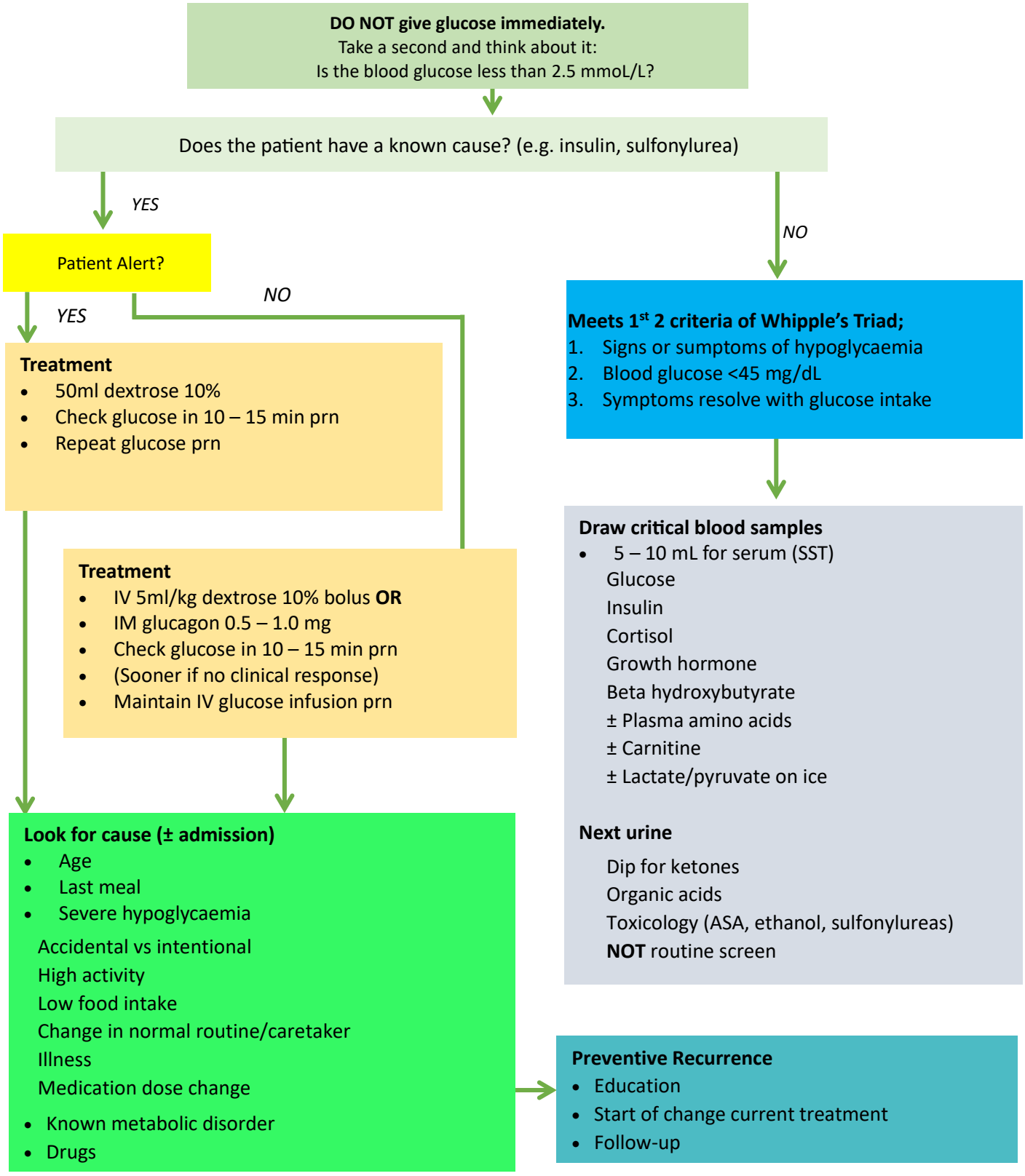


Fig 10.8. Emergency management of hypoglycaemia

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10.7 GRAVE'S DISEASE

Hyperthyroidism: is a pathological state characterized by increased synthesis and secretion of thyroid hormones. Thyroxin(T4) and triiodothyronine(T3) by thyroid gland.

Thyrotoxicosis: refers to biochemical and clinical picture of thyroid hormone excess.

Graves' disease: is an autoimmune disorder characterized by the presence of anti-TSH receptor antibody and anti-thyroperoxidase antibodies (TSHRAb and anti-TPO) resulting in an overactive thyroid gland resulting in hyperthyroidism, ocular abnormalities (graves orbitopathy) and very rarely localised dermopathy (pretibial myxoedema).

GD may be associated with other autoimmune diseases such as type 1 diabetes mellitus, vitiligo, coeliac rheumatoid arthritis etc.

Table 10.7. Grave's Disease

Symptoms	Signs
Common Symptoms of GD include: Heat intolerance, sweating Palpitation Exophthalmos Nervousness Irritability Emotional instability Tremor Weight loss in spite of increased appetite, Deterioration of attention, Restless sleep, Fatigue Nocturia, frequent micturition, Irregular menstrual periods	Tachycardia, atrial fibrillation Goiter Ophthalmopathy Diffuse goiter Wide pulse pressure, Uncoordinated movements, Pretibial myxoedema Anxiety Muscle wasting Signs of heart failure

Laboratory test Investigations

Table 10.8. Thyroid function tests.

Type of test	Values
TSH	0 – 1uU/ml in severe disease (1 -3 uU/ml in mild disease
FT4	
FT3	

** Note that an elevated FT3 is a more sensitive marker of GD than elevated FT4.

2. Antithyroid antibodies; the presence of antibodies provides supporting evidence for Graves' disease.

More than 95% of patients with GD are positive for TPO (thioperoxides antibody) About 50% have positive anti-thyroglobulin antibody titres. Thyroid stimulating-thyrotropin receptor antibodies (TRab, TSI) present in about 90%

3. Thyroid ultrasound scan; is useful to confirm or exclude the presence of a nodule particularly if a colour doppler if performed

4. Radioisotope scan is of little value in the diagnosis of graves' disease

EVALUATION OF PATIENT WITH HYPERTHYROIDISM.

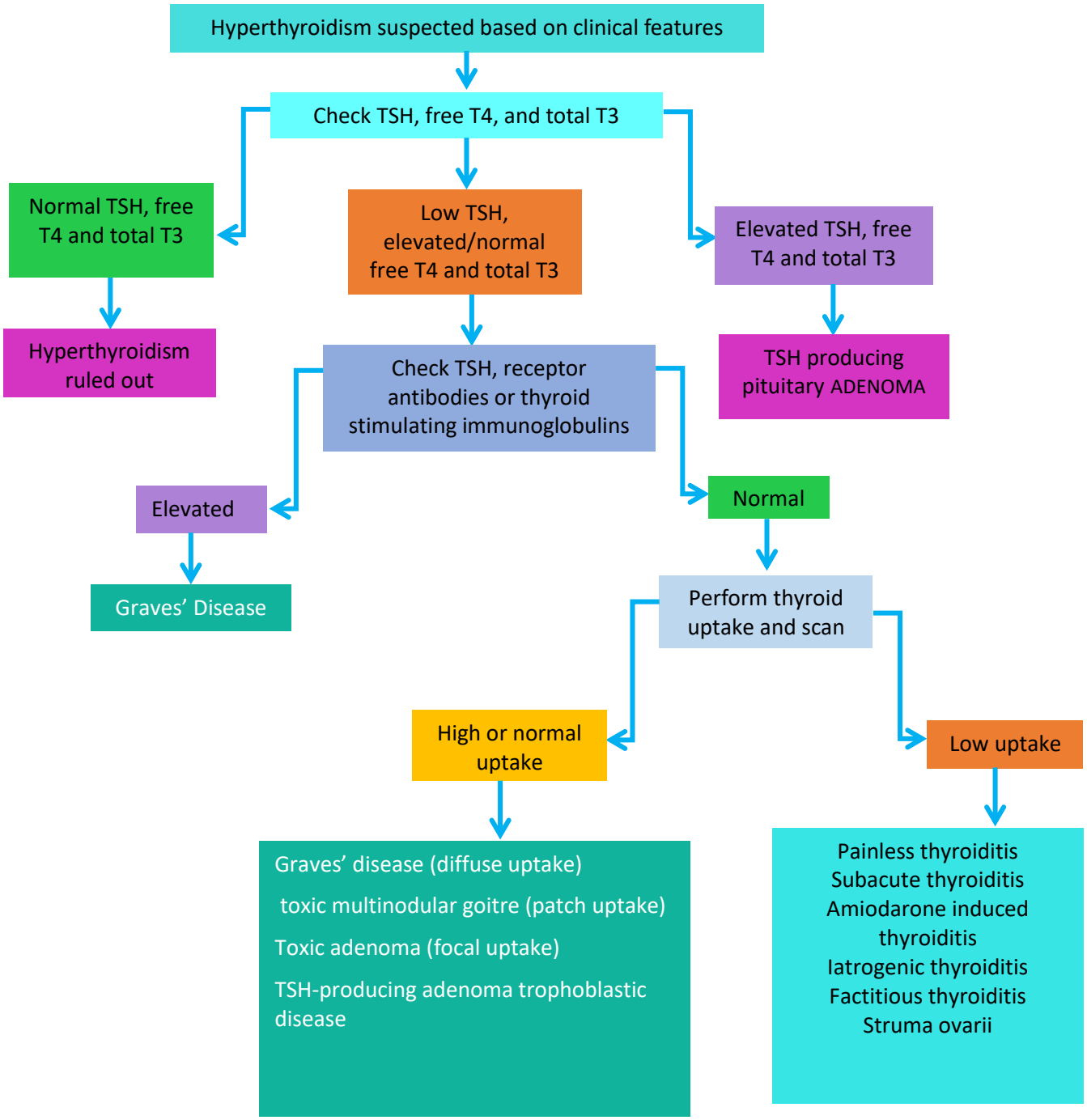


Fig 10.9. Evaluation of patients presenting with hyperthyroidism, adapted from (Mathew p, Kaur J, Rawla P 2023)

TREATMENT

Antithyroid drug therapy

Carbimazole 0.5-1 mg/kg

Propylthiouracil (PTU) 5-10 mg/kg

Iodine-131 therapy

Radioiodine ablation of the thyroid gland is quick, easy, moderately expensive, avoids surgery, and is without significant risk in adults and probably teenagers.

Surgery

Thyroidectomy was the main therapy, has been to a large extent replaced by 131-I treatment.

Thyroid Storm

Patients with GD can present with a thyroid crisis or storm which can arise in someone with undiagnosed or poorly controlled GD and, can be precipitated by infection, surgery and occur very occasionally following RAI therapy.

Clinical picture includes: Tachycardia, heart failure, hyperthermia, extreme anxiety, altered mental status and gastrointestinal upset.

Treatment include: administration of iodine (potassium iodide solution), glucocorticoid, Anti thyroid drug, beta-blocker.

Note: Patients with a thyroid storm should be managed in an intensive care setting.

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10.8 HYPOTHYROIDISM

- Deficiency in the production or the activity of thyroid-hormones leads to hypothyroidism.
- Hypothyroidism is caused by failure of the hypothalamic-pituitary-thyroid axis, which results in decreased production of thyroid hormones. The hypothyroidism may be primary (at the level of thyroid gland), secondary (at the level of pituitary gland), or tertiary (at the level of hypothalamus).

Congenital hypothyroidism (CH)

Congenital hypothyroidism can be defined as dysfunction of the hypothalamic-pituitary-thyroid axis present at birth, resulting in insufficient thyroid hormone production.

Classification of the causes of hypothyroidism is as follows:

1. Dysgenesis
- Agenesis
- Hypo-genesis
- Ectopia

2. Dyshormonogenesis
- Absences of the TSH response
- Defect in iodide transport or uptake
- Defect in organification
- Defect in thyroglobulin synthesis
- Defect in iodotryrosinase deiodinase

3. Hypothalamic-pituitary anomaly
- Hypothalamic-pituitary anomaly
- Pan-hypopituitarism
- TSH deficiency alone
- Resistance to TH

4. Transitory hypothyroidism.
- Drug-induced
- Induced by maternal antibodies
- Pregnant women treated with antithyroid or irradiation drugs
- Idiopathic

Symptoms and Signs

- More than 95% of newborns who have congenital hypothyroidism have little if any symptoms or signs at birth. This is why the performance of the thyroid function test in the nursery is so important.
- **Early signs include**: prolonged or recurrent jaundice, delay in umbilical cord separation and umbilical hernia, crying is hoarse and sounds emitted are low.
- **First few months of life other signs become presen**t: feeding difficulty, insufficient weight gain, noisy breathing, nasal congestion, respiratory disorders, obstipation, lethargy, dry, cold and pale skin
- Newborns who have congenital thyroid Dyshormonogenesis may have a palpable goitre, but the goitre typically develops later in untreated patients.

Acquired Hypothyroidism.

- The most common cause of acquired hypothyroidism autoimmune thyroiditis (Hashimotos thyroiditis), occurs most commonly during adolescence, but it also occurs in younger children, typically after first few years of life.
- The other causes that less commonly include hypothyroidism that occurs after radiation of the head and neck for certain cancers, after total body irradiation in preparation for bone marrow transplant and secondary to certain drugs e.g. antiepileptic drugs, lithium, amiodarone, tyrosine kinase inhibitors.
- Iodine deficiency remains the most common worldwide cause of hypothyroidism in children

Symptoms and Signs

The onset of acquired childhood hypothyroidism often is very subtle; in retrospect, it may be evident that signs and symptoms were present for a longer time, sometimes for 2 to 3 or more years.

- Decline in linear growth; if previous height measurements are available, a decline in linear growth from the onset of hypothyroidism will be evident
- Fatigue
- Constipation
- Cold intolerance
- Poor school performance
- Weight gain
- Irregular menstrual periods, and somnolence
- Other clinical features of acquired hypothyroidism include bradycardia
- Dry skin
- Increased body hair
- Pallor
- Myxoedema of the face

- An enlarged thyroid gland
- Proximal muscle weakness
- Delayed relaxation phase of the ankle reflex, and
- Delayed puberty
- Occasionally, acquired childhood hypothyroidism presents with precocious puberty.

The enlarged thyroid gland usually is diffuse and non-tender; Sometimes the gland may be firm.
Children afflicted with Hashimoto thyroiditis also may have other autoimmune disorders and a family history of thyroid and other autoimmune disorders to support the diagnosis.

LABORATORY TESTS

For new-born screening:

Initial T4 testing with follow-up TSH testing

- Between 1 and 4 days of age, the normal range for serum total T4 concentrations is 10 to 22mcg/dL (128.7 to 283.2 nmol/L).
- Between 1 and 4 weeks of age, the normal range for serum total T4 concentrations is 7 to 16mcg/dL (90.1 to 205.9 nmol/L).
- Subclinical hypothyroidism is defined as a normal serum total or free T4 concentration and a high serum TSH concentration.
- Infants who have subclinical hypothyroidism should be treated during the first 3 years after birth due to the critical dependence of the CNS myelinating on thyroid hormone.

CONGENITAL HYPOTHYROIDISM: DIAGNOSTIC FLOWCHART

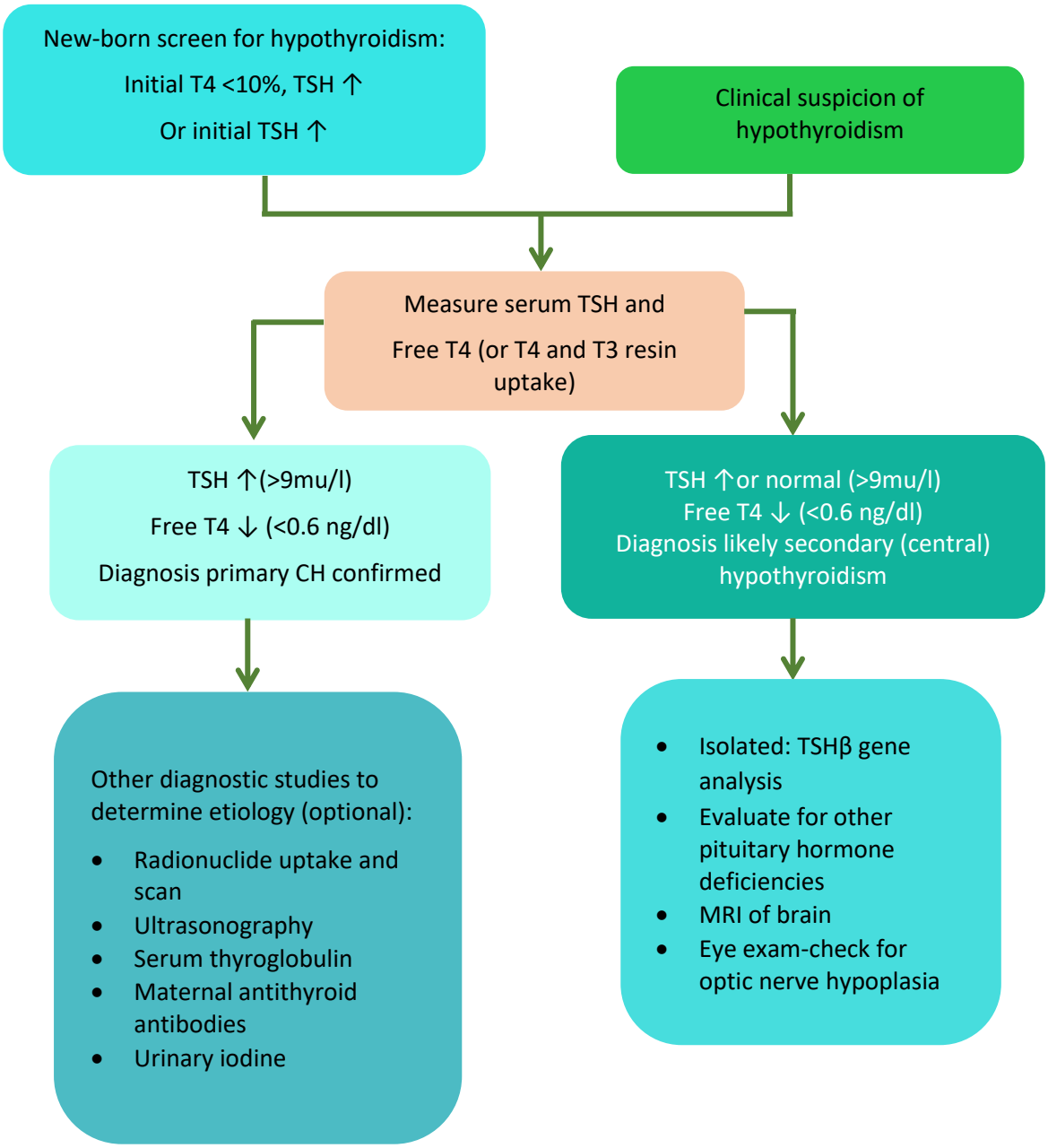


Fig 10.1.1. congenital hypothyroidism: diagnostic flow chart adopted from (Rastogi and LA Franchi 2010)

MANAGEMENT

Congenital

- The overall goals of treatment are normal growth and good cognitive outcome.
- Serum T4 concentrations should be restored rapidly to the normal range, followed by continued maintenance of euthyroidism.
- The aim of treatment is to keep the serum T4 or free T4 concentration in the upper half of the normal range adjusted for age.
- Of note, many commercial laboratories do not provide age-adjusted normal ranges in their reports. In the first postnatal year, serum T4 should be 10 to 16 mcg/dL and serum freeT4 should be 1.4 to 2.3ng/dL. The serum TSH concentration should be less than 5 mU/L.
- Oral T4 (levothyroxine) is the treatment of choice. Initial dose of 10 to 15 mcg/kg per day, usually 50 mcg/day.
- In preterm and other low-birthweight infants, the thyroid replacement dose should be calculated by using10 to15mcg/kg per day, with the higher end administered to babies who have low T4 concentrations.

Only T4 tablets should be used because thyroid suspensions prepared by individual pharmacists may result in unreliable dosing.

Parents should be instructed to crush the T4 tablet and mix It with a small volume of human milk, formula, or water. **Soy formulas or any preparation containing concentrated iron or calcium should not be used because they reduce the absorption of T4**.

The more rapidly T4 concentrations are corrected, the better the neurologic outcome. Aim to normalise the T4 concentrations within two weeks

Patient follow up

The recommended follow up schedule is as follows;

- At 2 and 4 weeks after the initiation of T4 treatment
- Every 1 to 2 months during the first 6 postnatal months
- Every 3 to 4 months between 6 months and 3 years of age
- Every 6 to12months thereafter until growth is complete weeks after any change in dose
- At more frequent intervals when compliance is questioned or abnormal results are obtained

Acquired

- Childhood hypothyroidism is treated with levothyroxine. (refer to table below for dosage)
- Treatment should be individualized because the absorption of T4 and metabolism vary among individuals.
- Serum free T4 and TSH concentrations should be monitored periodically, preferably at 3- to 6-month intervals.
- The goal is to keep the serum free T4 concentration at the mid-normal range and the TSH concentration in the normal range.
- Once the patient is euthyroid, many of the symptoms disappear.

Table 10.9. Congenital hypothyroidism (CH)

Dosage of levothyroxine				
Age band	6 to 12 months	1 to 3 years	3 to 10 years	10 to 18 years
Dosage	5 to 8 mcg/kg	4 to 6 mcg/kg	3 to 5 mcg/kg	2 to 4 mcg/kg

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11.1 APPROACH TO DIAGNOSIS AND INVESTIGATION OF COMMON MALIGNANCIES

The most common types of cancer among children and adolescents vary by age group. The common types include leukemias, brain cancers, lymphomas and solid tumours such as neuroblastoma and Wilms tumour. Childhood cancers cannot generally be prevented or identified through screening

Causes of malignancy

Risk factors for cancer can be broadly classified as:

Genetic causes

Environmental causes

1. Pesticide exposure
2. Ionising Radiation exposure
3. Drugs

Clinical features

In the first two years of life, embryonic tumours occur more commonly. Examples include Nephroblastoma, Retinoblastoma, Neuroblastoma, Teratoma, Rhabdomyosarcoma, Medulloblastoma.

From 2-5 years of age, more cancers tend to occur in addition to the embryonic tumours. These later occurring cancers include Acute Lymphoblastic Leukaemia, Non-Hodgkin's Lymphoma, and Gliomas.

In adolescents Hodgkin's disease, Bone, Gonadal and Connective tissue tumours predominate

Common signs and symptoms of cancer in children.

- Fever
- Malaise/weakness
- Bone pain/tenderness
- Abnormal growths (e.g. Abdominal mass, eye masses)
- Persistent generalized or regional lymphadenopathy
- More than one abnormal hematopoietic lineage
- Specific neurologic deficit
- Increased intracranial pressure
- Proptosis
- White pupillary reflex

- Vaginal bleeding
- Pelvic/testicular mass
- Joint or bone swelling or pain

Uncommon symptoms and signs of cancer in children

- Superior vena cava syndrome
- Subcutaneous nodules
- Chronic diarrhoea
- Failure to thrive
- Skin lesions

History

General: a patient may seek medical attention for any of the following: Pain, mass, Bleeding, recurrent fevers, weakness

Table 11. Specific symptoms to look for in the history

CNS	R/S	MSS
Headache	Cough	Swelling
Fits	Difficulties breathing	Wasting
Blindness	Haemoptysis	Bone/joint pain
Vomiting	Neck	Non healing ulcer
Stiff neck	Swelling	Skin lesion
Confusion	Engorgement	Bleeding spots

Table 11.1. symptoms to look for in the history

GIT	GUT	OTHERS
Abdominal mass	Pelvic/suprapubic mass	Malaise
Diarrhoea	Dysuria	Anorexia
Easy satiety	Haematuria	Nights sweats
Dysphagia	Urinary retention	Fever
Poor appetite		
Melaena		

PHYSICAL EXAMINATION		
CNS	**R/S**	**MSS**
Slow Mentation	Tachypnoea	Skin lesion
Cranial palsy	Reduced breath sounds	Wasting
Paraplegia/Paraparesis	Stridor	Oedema
Meningism	Haemoptysis	

Vomiting Confusion		
GIT	**GUT**	**NECK**
Abdominal mass Hepatomegaly/splenomegaly Visible distension of vessels	Proteinuria Haematuria	Lymphadenopathy Engorged vessels

Common cancers referred to the Haematology-Oncology Unit

- Nephroblastoma
- Retinoblastoma
- Leukaemia
- Hodgkin's lymphoma

Investigations

- Blood Tests
- Complete Blood count
- Peripheral Smear
- Erythrocyte sedimentation rate
- Liver and Kidney Test
- Retroviral test
- Cancer specific tests e.g- FISH, Philadelphia Chromosome 9 and 22, Flow cytometry etc

IMAGING STUDIES

Chest radiography: Postero-anterior and lateral views to

- Assess mediastinal masses
- Evaluate the airway
- Assesses presence/absence of pulmonary parenchymal lesions

Ultrasonography: Abdomen, Pelvis, Eye, Cranial to identify site and disease extent

MRI/Computed tomography

- MRI/CT scans of the chest, abdomen, and pelvis can be used to stage lesions
- Chest CT scan is indicated to assess for the degree of tracheal compression
- Head CT scans assist in excluding mass lesions and possible meningeal involvement among individuals with CNS disease.

Bone scanning and skeletal surveys

When additional symptoms are present, these tests help in identifying additional sites of disease.

Echocardiography
Can be obtained as baseline findings before patients are given chemotherapy with anthracyclines, which can cause cardiomyopathy.

Procedures
Bone marrow aspiration/biopsy.
Biopsy is necessary to assess for evidence of bone marrow involvement in patients with lymphomas. Sites for BMA include the anterior iliac crest, posterior iliac crest, sternum and tibia (in infants).

Biopsy
For patients with a mass, tissue is generally available from resection or intra-operative biopsy. As an alternative, a diagnosis may be made by using pleural fluid or ascetic fluid.

Lumbar puncture
To determine the CSF cell count and differential: This test is done to assess CNS involvement, the presence of which alters therapy.

Management
Once diagnosis is made either histologically or clinically, stage the patient according to the staging used for that particular tumour. Consult the protocol to determine treatment modality which could be either one or a combination of two or all three:

- Surgery
- Chemotherapy
- Radiation

Treatment should aim at either to cure or palliate.
Patient preparation is critical for treatment initiation.

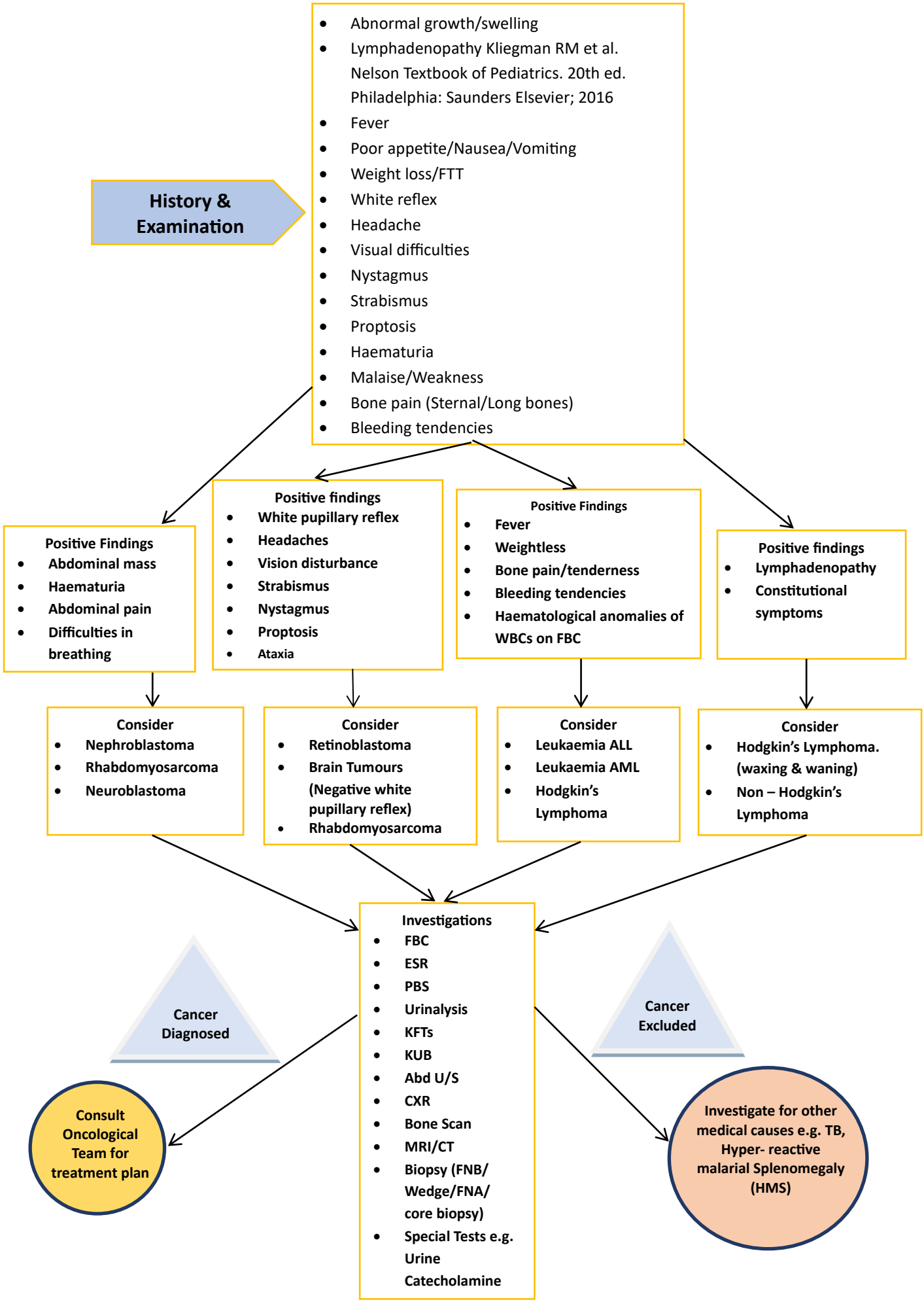


Fig 11. Approach to diagnosis and investigation of common malignancies

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12.1 APLASTIC ANAEMIA

Aplastic anaemia is defined as haemoglobin 10 g/dL, platelet count 50 x 10^9/L, granulocytes 1.5 x 10^9/L, and a bone marrow biopsy demonstrating a decrease in cellularity and the absence of significant fibrosis or neoplastic infiltration.

Evaluation

- Any evaluation for new onset severe aplastic anaemia (SAA) must start with a thorough history.
  - A detailed history may identify factors directly causing SAA or may lead to suspicion that the cytopenia reflects another underlying disease such as an Inherited Bone Marrow Failure Syndrome (IBMFS).
- A thorough physical examination is critical.
  - While some findings, such as pallor or bruising, may reflect the presence of cytopenias, other specific exam findings may suggest a diagnosis other than SAA.
  - A non-exhaustive list of examples includes lymphadenopathy, hepatosplenomegaly, joint swelling, non-petechial rash, icterus, café au lait spots, short stature, weight discordance for height, abnormal limbs, nails or digits, or other visible congenital anomalies.

  ❖ To make a proper diagnosis, these evaluations need to be interpreted together with the clinical presentation.
  ❖ Depending upon the patient characteristics and provider's clinical judgement, additional evaluations may also be indicated.
  ❖ Patients with presentations not meeting criteria for idiopathic SAA may warrant a genetic evaluation.
  ❖ Once a diagnosis is made, the patient must be referred to a tertiary hospital for further management.

Table 12. Differential diagnosis for aplastic anaemia

Acquired	Inherited
Infection • Hepatitis A, B, C. • CMV • HIV	Bone marrow failure secondary to inherited syndromes • Fanconi anaemia • Dyskeratosis congenita/telomere biology disorders • Shwachman-Diamond syndrome
Medications/ Toxins • Benzenes, chloramphenicol • Cytotoxic medications • Ionizing radiation	MDS secondary to haematopoietic malignancy predisposition syndrome
Nutritional deficiencies • Zinc • Copper, • Vitamin B12 • Folate	Pancytopenia associated with primary immunodeficiency
Myelodysplastic Syndrome (MDS)	
Rheumatologic disorders	
Paroxysmal nocturnal Haemoglobinuria (PNH)	

Newly diagnosed children should be referred promptly to a tertiary institution/ paediatric haematology unit for urgent work- up and treatment. Those admitted will begin their complete work- up as follows:

Work- up

Blood work related to acquired aplastic anaemia:

1. FBC, differential, peripheral smear, reticulocyte count
2. Hb electrophoresis
3. Bone marrow aspirate and trephine biopsy (BMAT), including cytogenetics. Note: BMAT is done under anaesthesia in theatre. Liaise with paediatric anaesthesia for pre- operative assessment.
4. Liver enzymes and function tests- ALT, AST, alkaline phosphatase, bilirubin (conjugated & unconjugated), GGT, albumin, total protein, INR/PTT/fibrinogen.
5. Electrolytes (Na, K, Cl), calcium, phosphate, magnesium.
6. Renal function test- urea, creatinine

7. Direct Antiglobulin Test (DAT), haptoglobin, LDH, ESR.
8. Iron studies- serum iron, ferritin, transferrin
9. Viral studies- Hepatitis A, B and C, HIV, and CMV.
10. Anti- nuclear antibody (ANA), anti- double stranded DNA, rheumatoid factor (RF).
11. Complement C3 & C4, CRP, CH50, immunoglobulins (IgG, IgA, IgM)
12. Serum vitamin B12, and serum and red cell folate
13. Referral for HLA typing
14. Group and cross match

Blood work related to inherited aplastic anaemia:

1. Peripheral blood for chromosomal breakage analysis to exclude Fanconi Anaemia.
2. Flow cytometry for CD55/CD59 to exclude Paroxysmal Nocturnal Haemoglobinuria
3. Peripheral blood to test exon 2 of SBDS gene to exclude Shwachman- Diamond Syndrome.
4. Telomere length testing to exclude Dyskeratosis Congenita.
5. Abdominal ultrasound as screen for congenital anomalies that may related to an inherited bone marrow failure syndrome.
6. Referral for HLA typing.

Treatment

Supportive measures:

1. Transfusion support.

- Transfuse packed red blood cells as needed for clinical symptoms related to anaemia and/or to maintain **haemoglobin levels > 7 g/dL**.
- Transfuse platelets as needed for clinical indications of bleeding and/or to maintain **platelet counts > 10 x 10^9/L** when patient is well and **> 20 x 10^9/L when febrile**.

2. Management of fever.

- In the event of fever and neutropenia (**neutrophil counts 0.5 x 10^9/L)** for more than 3- 5 days despite antibiotic therapy, then initiation of neupogen should be considered. [3] Neupogen dose is 5 mcg/kg/dose subcutaneously daily. Discontinue if there is no significant increase in neutrophil count within one week.
- In case of severe infection, or recurrent bacterial or fungal infection, or clinical instability the neupogen should be increased by 5- 10 mcg/kg/dose every 3- 5 days, up to a maximum of 60 mcg/kg/dose or 1,500 mcg/day whichever is less.

3. Infection Prevention

- **Pneumocystis Jirovecii Pneumonia (PJP) Prophylaxis**: Prophylaxis should be started on initiation of immunosuppressive therapy, and discontinued once patient has responded to immunosuppressive therapy and has been discontinued.
  Cotrimoxazole is not an option for PJP prophylaxis due to bone marrow suppression with its use.

Dapsone 4 mg/kg/day (maximum 200 mg/dose) PO once/week. Contraindicated in patients with G6PD deficiency. Obtain baseline methaemoglobin level prior to starting the drug.

- **Fungal Prophylaxis:** Fluconazole prophylaxis for patients with severe neutropenia (neutrophil count < 0.2 x 10^9/L). Fluconazole dose is 3 mg/kg/day IV/PO once daily (maximum 400 mg/day). Due to the interaction with cyclosporine, discuss potential need to adjust doses with pharmacist.

Treatment of patients with a matched sibling donor for HSCT:

- A matched sibling donor for Hematopoietic Stem Cell Transplant (HSCT) is the first line of therapy for patients with acquired severe and very severe aplastic anaemia and IBMFS resulting in approximately 90% cure rate.
- HLA-typing of the patient and all immediately related family members (parents and siblings) is carried out as part of the initial work-up.
- Arrangements must be made through the Ad-hoc committee at the University Teaching Hospital (UTHs)- Children's Hospital for HSCT abroad.

Treatment of patients without a matched sibling donor for (HSCT)- Immunosuppressive therapy:

- Immunosuppressive therapy (IST) should be initiated as soon as possible. This requires admission to the paediatric haematology inpatient unit.
- Baseline blood work to be done within 24 hours of admission:

1. FBC, differential, retic count, and PBS
2. Liver enzymes- ALT, AST, Alk Phosphatase, bilirubin (Unconjugated & conjugated), GGT
3. Liver Function Tests
4. Renal function tests- urea and creatinine
5. Na, K, Cl, Mg, Ca, Phos, LDH, and urate

Table 12.1. Medications for immunosuppressive therapy

IST Regime	Dose	Notes
Anti-thymocyte Globulin (ATG) (ATGAM® = equine ATG)	40 mg/kg/dose IV over 6 hours daily x 4 days	To be infused over a minimum of 6 hours; infusion may be prolonged up to a maximum of 12 hours
Prednisolone	1 mg/kg/dose (Max 40 mg/dose) PO two times daily x 7 days	• Start 3 hours before ATG • Taper slowly over 3 weeks once ATG therapy completed.
Cyclosporine (CsA)	5 mg/kg/dose (max 250 mg/dose) PO q12h for 6 months after maximal response is achieved, then taper over 6 months.	• Trough CsA concentration to be drawn on day 3 of therapy • Adjust dose to maintain whole blood serum concentration between 150 -200 mcg/L
Pre- medication	**Dose**	**Notes**
Paracetamol	15 mg/kg (max 1000mg/dose) PO pre- ATG and then q6h PRN	Dose limit of 75 mg/kg/day or 4 g/day, whichever is less.
Diphenhydramine	1 mg/kg/dose (max 50 mg/dose) PO/IV pre-ATG and then q6h PRN	
Pethidine	1 mg/kg/dose (max 50 mg/dose) IM q6h PRN for rigors	

If there is no response at 3-6 months' post-initial IST, then consideration should be made for a BMT from an unrelated HLA matched hematopoietic stem cell donor. A thorough discussion must occur with the parents/guardian and BMT team regarding potential donors and the approach to therapy that will yield the best possible outcome.

Table 12.2. Management of possible complications secondary to medications

Medication	Complication	Treatment/Management
ATG	Serum sickness	Serum sickness post ATG is a type III hypersensitivity (immune complex- mediated) reaction that results due to the injection of foreign protein into a patient. Symptoms & signs may include: fever, malaise, cutaneous eruptions (especially urticaria), arthralgias/arthritis, GI complaints (abdominal pain, nausea, vomiting, diarrhoea), headache, myalgia, blurred vision, dyspnoea/wheezing and lymphadenopathy starting usually 1-2 weeks post-ATG administration. Clinical recovery can take between 1-4 weeks. Treatment: Continue prednisolone dose for 1 week, and Diphenhydramine 1mg/kg/dose (max 50 mg/ dose) PO/IV q6h PRN.
	Anaphylaxis	Stop ATG infusion immediately. See anaphylaxis treatment protocol
Cyclosporine	Hypomagnesaemia	Supplement with oral magnesium. Provide patient/family with magnesium diet.
	Hypertension	If SBP and/or DBP >95th %ile for age and height on 2 separate occasions 15 minutes apart, & on > 2 consecutive days, start amlodipine dosed as per Paediatric dosing schedule.
	Nephrotoxicity	If serum creatinine is elevated to 1.5 x baseline, increase fluid intake to 1.5 x maintenance, and consider reducing cyclosporine target trough concentration after discussion with pharmacist.
Prednisolone	Gastritis/ Heartburn	Initiate omeprazole if symptomatic while receiving prednisolone.
	Hypertension	See cyclosporine above

Follow- up Post Immunosuppressive Therapy

Once the patient has completed cyclosporine taper, close monitoring during the first two (2) years post- therapy is important to assess for symptoms and signs of relapse. Should the patient begin to exhibit clinical and/or haematological changes during post- therapy follow- up, a BMA and biopsy may be necessary.

Table 12.3. Follow- up schedule

	Frequency of visits	**Tests/Bloodwork**
Year 1 – Year 2	• 1 month after stopping cyclosporine and then • Every 2-4 months*	Every visit: • Vital signs, height, weight, • FBC, differential, retic count, smear Every 6 months*: • Urea, creatinine • LFTs (ALT, AST, alk phosph, bili – unconjugated & conjugated)
Year 2 – Year 5	• Every 6 months*	Every visit: • Vital signs, height, weight CBC, differential, retic count, smear • Annually*: • Urea, creatinine • LFTs (ALT, AST, alk phosph, bili – unconjugated & conjugated)
Year 5 +	• Annually*	• Vital signs, height, weight, • FBC, differential, retic count, smear • urea, creatinine • LFTs (ALT, AST, alk phosph, bili – unconjugated & conjugated)

*May be completed more frequently if clinically indicated.

Immunizations

Patients can continue to receive recommended scheduled inactivated virus immunizations. There is a possibility that patients may not mount an appropriate immune response to immunization, thus re-immunization may be necessary once they are no longer immunosuppressed.

Live attenuated viral vaccines, such as the MMR, are contraindicated while patients are on immunosuppressive therapy due to the increased risk of developing disease caused by the vaccine.

REFERENCES

1. Davies JK, Guinan EC. An update on the management of severe idiopathic aplastic anaemia in children Br J Haematol. 2007;136:549-64.
2. International Agranulocytosis and Aplastic Anemia Study (IAAAS). 1980-1986.
3. Marsh JCW, Ball SE, Cavenagh, Darbyshire P, Doak I, Gordon-Smith, EC, et al. Guidelines for the diagnosis and management of aplastic anaemia. Br J Haematol. 2009;147;43-70.
4. Young NS, Kaufman DW. The epidemiology of acquired aplastic anaemia. Haematologica. 2008;93(4):489-92.

12.2 HAEMOPHILIA

Haemophilia refers to inherited bleeding disorders caused by deficiency of specific coagulation factors.

Haemophilia A is caused by coagulation factor VIII (FVIII) deficiency, haemophilia B by deficiency of coagulation factor IX (FIX), and haemophilia C by deficiency of coagulation factor XI.

These clotting factor deficiencies are caused by recessive mutations of the respective clotting factor genes. As the recessive mutant FVIII and FIX genes are located on the X chromosome, both haemophilia A and haemophilia B are inherited in an X-linked pattern. Consequently, in these conditions, males are affected and females are carriers of haemophilia. Both diseases have the same clinical presentation, so their specific diagnosis must be established by factor assay.

The combined prevalence of both haemophilia A and B has been estimated as approximately 1 in 5 000 live male births. Haemophilia C is an autosomally inherited condition, uncommon in the general population of Zambia, and will therefore not be discussed further in this guideline.

Clinical Features

- Many have spontaneous or provoked bleeding
- Large or deep bruises
- Joint pain and swelling due to bleeding within them
- Excessive bleeding following minor injury or surgery
- Easy bruising
- Menorrhagia
- Nose bleeds
- Spontaneous nose bleeds
- Unexplained bleeding and haematuria or haematochezia
- Intracranial haemorrhage

Diagnostic Tests

- Prolonged APTT with a normal PT and normal platelet count is suggestive
- Confirm with a Factor VIII or Factor IX assay:

Table 12.4. Classification of haemophilia

Severity	Factor level
Severe	< 1%
Moderate	1- 4 %
Mild	5- 40 %

NOTE: Normal factor level ranges from 50- 200%

- Recommended weekly factor prophylaxis to prevent bleeds is 25- 40IU/ kg or more depending on clinical situation.

Treatment of bleeding in haemophilia

- Manage people with haemophilia at or under the supervision of a haemophilia comprehensive care centre.
- Patients with haemophilia can have spontaneous bleeding and/or excessive bleeding with mild trauma. It is imperative to treat haemophilia patients with factor within 30 minutes of presentation first and then consider diagnostic testing later.

Table 12.5. Sites of bleeding in Haemophilia

Non- life or non- limb- threatening bleeds	Life- or organ- threatening bleeds
Joints	Intracranial
Muscles	Muscle compartment
Easy bruising	Neck/throat
Mucosal bleeding (epistaxis, gingival)	Massive gastrointestinal
Gastrointestinal	Genitourinary tract

Factor replacement

- Factor VIII deficiency (Haemophilia A)
  - 1 unit/kg increases Factor VIII activity by 2%
  - Give 30 units/kg of Factor VIII for an acute minor bleed
  - Give 50 units/kg of Factor VIII for an acute major bleed
  - After initial dose, discuss treatment plan with paediatric haematology unit. Half- life of Factor VIII is 8- 12 hours. Major bleed will likely need 8- 12-hour dosing.

- Factor IX deficiency (Haemophilia B)
  - 1 unit/kg increases Factor IX activity by 1%
  - Give 50 units/kg of Factor IX for an acute minor bleed
  - Give 100 units/kg of Factor IX for an acute major bleed
  - After initial dose, discuss treatment plan with paediatric haematology unit. Half- life of Factor IX is 18- 24 hours. Major bleed will likely need at least 24-hour dosing.
  - It is important to round up both Factor VIII and IX to a unit vial dose whenever possible to avoid wastage.

Important: Where Factor VIII or Factor IX is unavailable or not immediately available in emergency situations, Call blood bank immediately and order Fresh Frozen Plasma (FFP) at 10-20 mL/kg IV to run over 1 hour.

Supportive care (PRICE)

- Protection
- Rest/Replacement
- Ice/Immobilization

- Compression
- Elevation

Add Tranexamic acid (antifibrinolytic- prevents clot breakdown) 10mg /kg 8 hourly IV (or 15-25 mg/kg orally) for mucocutaneous (mouth, nose) bleeding. Contraindicated in urinary tract bleeds.

Pain management

- Pain in people with haemophilia has multiple causes, and these include:
  - Joint capsular stretching as a result of haemarthrosis.
  - Haemophilic arthropathy
  - Compartment syndrome
- The goals of pain management include:
  - To remove pain completely without increasing the risk of bleeding
  - To improve the patient's quality of life.
- COX- 2 inhibitors are favoured largely owing to their favourable side- effect profile, analgaesic effects, anti- inflammatory effects and anti- angiogenic effects.
- Aspirin and other antiplatelet agents should be avoided.
- Analgaesia requiring intramuscular injection should also be avoided.

- For patients with a confirmed diagnosis of haemophilia, offer to enroll in the World Bleeding Disorder Registry (WBDR).

- **Surgery**
  - For patients with haemophilia requiring elective or emergency surgery, discuss this with paediatric haematologist.

- **Vaccinations**
  - Vaccinate all individuals with haemophilia with appropriate vaccines. Standard vaccines for children can be given without factor concentrate prophylaxis.
  - An ice pack can be applied to the injection area for 5 minutes before injection.
  - The smallest gauge needle available (usually 25- 27 gauge) should be used.
  - Application of ice and prolonged pressure for 10 minutes is recommended after administration of the vaccine.
  - Prophylactic Clotting Factor Concentrate (CFC) cover for irritant vaccines such as tetanus should be given prior to vaccination.

Table 12.6. For routine clinic follow-up, see the table below.

AGE	VISIT FREQUENCY	LAB TESTS	ADDITIONAL CHECKS

0-6 years	3 months	• Blood group at first visit. • FBC, diff, retic count every visit. • Serum ferritin annually. • Hepatitis B/Hepatitis C/ HIV serology annually. • HJHS annually.	• Demonstrate • Emergency preparedness. • Vaccination check • Counselling about Haemophilia.
6 - 16 years	6 months	• Blood group at first visit. • FBC, diff, retic count every visit • Hepatitis B/HIV serology annually. • Serum ferritin annually. • HJHS annually.	• Assess readiness for self-infusion. • Emergency preparedness. • At 12 years of age, begin transition assessments.

REFERENCES

1. Evidence- based management of sickle cell disease, Expert Panel Report, 2014. Guidelines for the Management of Haemophilia 2nd ed. World Federation of haemophilia. Blackwell Publishing; 2014.
2. Zambian National Guidelines for the Management of Haemophilia. Ministry of Health; 2020.

12.3 SICKLE CELL DISEASE

Sickle cell disease is an inherited (genetic) disease that affects the haemoglobin in the red blood cells. Haemoglobin is the protein in red blood cells that carries oxygen to every part of the body. SCD causes red blood cells to "sickle" (change from a normal doughnut shape to a crescent moon shape).

ROUTINE MEDICATIONS

Folic acid supplementation

- Patients with chronic haemolysis as with SCD benefit from folic acid supplementation.
- Give 5mg OD PO for all ages.
- For infants, the tablet can be crushed into a suspension for ease of administration.

Hydroxyurea

- Mechanism: Hydroxyurea is an antineoplastic that may cause inhibition of DNA synthesis by acting as a ribonucleotide reductase inhibitor. It increases fetal haemoglobin, reduces neutrophils, alters adhesion of red blood cells to endothelium, increasing water content of red blood cells and increases deformability of sickled cells.
- Indication: All HbSS or HbS beta^0 thalassaemia patients from 9 months of age should be counselled on the potential benefits and risks of chronic hydroxyurea therapy, and encouraged to begin taking it.
- Exclusions: acute liver disease, history of severe hydroxyurea toxicity or hypersensitivity, pregnancy or sexually active and unwillingness to use contraception.
- Baseline investigations prior to initiating hydroxyurea treatment: FBC with differential count, reticulocyte count, Hb electrophoresis or High Performance Liquid Chromatography (HPLC), (with quantitative HbF%, & HbS%), Chemistry profile, Liver Function Tests (AST, ALT), renal function tests (urea, creatinine).
- Patients on hydroxyurea shall have Hb electrophoresis done every 6 months for monitoring.
- Consult with paediatric haematologist for appropriate work- up before initiating a patient on hydroxyurea.
- Dosing:
    - Starting dosage for infants and children is 20 mg/kg/day given as a single daily dose.
    - Monitor FBC with differential and reticulocyte count at least every 4 weeks when adjusting dosage.
    - If dose escalation is warranted based on clinical and laboratory findings, proceed as follows- increase dose by 5 mg/kg/day every 8- 12 weeks.
    - Increase dose until absolute neutrophil count is 2, 000- 4, 000/L (mild myelosuppression) is achieved or up to a maximum tolerated dose (MTD) of 35mg/kg/day dose achieved.
    - Once a stable dose is established, laboratory safety monitoring should include FBC with differential, reticulocyte count, and platelet count every 2- 3 months.

- People should be reminded that the effectiveness of hydroxyurea depends on their adherence to daily dosing. They should be counselled not to double up doses if a dose is missed.
- A clinical response to treatment with hydroxyurea may take 3- 6 months. Monitor RBC MCV and Hb F levels for evidence of consistent or progressive laboratory response.
- If neutrophil count < 2.0 x 10^9/L, or retic count < 80 x 10^{12}/L, or platelets < 80 x 10^9/L or Haemoglobin < 5 g/dL, discontinue hydroxyurea until counts recover (usually for 5- 7 days) and repeat FBC. If FBC returns to baseline, restart at same dose.
- Liquid formulation can be prepared by compounding pharmacy using published guideline. The liquid formulation expires after 2 weeks.
- Hydroxyurea therapy should be continued during hospitalizations or illness.

Malaria prophylaxis

- Deltaprim to all patients with sickle cell anaemia. A tablet contains pyrimethamine at 12.5 mg and dapsone at 100 mg.

- Dosing:
  - Infancy up to 3 years of age ¼ tablet once weekly PO,
  - 3- 10 years old ½ tablet once weekly PO and
  - Over 10 years old 1 tablet once weekly PO.

In event of allergy to deltaprim, use dapsone once weekly PO.

Penicillin prophylaxis

- Patients should be receiving penicillin prophylaxis
  - Penicillin V 125 mg BD PO daily for 2 months- 3 years
  - Penicillin V 250mg BD PO daily for > 3 years.
  - Substitute with erythromycin if penicillin hypersensitivity present.
- Discontinuing penicillin prophylaxis can be considered for patients age 5 years and older who meet BOTH criteria:
  - Patients without prior history of documented pneumococcal infection
  - Patients who have received 1 dose of PPV (pneumococcal 23) and appropriate PCV 13 doses.
  - Never had a splenectomy.

Diagnostics

- Ideally, haemoglobin (Hb) electrophoresis or High Performance Liquid Chromatography (HPLC) should be sent after 6- 12 months of age as long as there has not been a red blood cell transfusion in the previous 3 months.
- For infants 0- 3 months of age born in Lusaka or Ndola, they can be referred to the University Teaching Hospitals (UTHs)- Children's Hospital or Arthur Davison Children's Hospital respectively for newborn screening for SCD.

Table 12.7. For routine clinic follow up, see the schedule below.

AGE	VISIT FREQUENCY	LAB TESTS	ADDITIONAL INFORAMATION
0-2 years	3 months	• Hb electrophoresis • Blood group • FBC, diff, retic count every visit	• Vaccination check • Antibiotic prophylaxis • Demonstrate palpation of spleen • Hydroxyurea (start after age 9 months) • Vitals: SpO_2, weight, height, every visit
2- 5 years	6 months	• FBC, retic count every visit • Bili, RFTs, LFTs annually • HIV, Hepatitis B • TCD annually	• Vaccination check. • Pneumococcal polysaccharide vaccine (PPV23) should be administered as soon as 24 months of age for supplemental protection. • Antibiotic check • Hydroxyurea • Vitals: SpO_2, weight, height, every visit
5- 12 years	6 months	• FBC, retic count every visit • Bili, urinalysis, RFTs, LFTs annually • HIV, Hepatitis B annually • Brain MRI/MRA/MRV (baseline scan) at age 8. • TCD annually	• Vitals, SpO_2, weight, height every visit • Hydroxyurea • Ophthalmology screen at age 10. • Echocardiogram with PHTN screen: Only when symptomatic • PFTs at age 10, if normal, again when symptomatic. • Linkages
12- 16 years	6 months	• FBC, retic count every visit • Bili, urinalysis, RFTs, LFTs annually • HIV, Hepatitis B annually	• Vitals, SpO_2, weight, height every visit • Hydroxyurea • Ophthalmology screen annually • Echocardiogram: Only when symptomatic • PFTs • Linkages • Transition assessment

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1. Kliegman RM et al. Nelson Textbook of Pediatrics. 20th ed. Philadelphia: Saunders Elsevier; 2016
2. Guidelines for the Management of Sickle Cell Disease in Zambia. 2020
3. Ware RE. How I use hydroxyurea to treat young patients with sickle cell anaemia. Blood. 2010;115(26):5300-11.
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12.4 PAIN CRISIS

Pain crisis is caused by ischaemic tissue injury from the obstruction of blood flow by sickled red blood cells. Reduced blood flow causes hypoxia and acidosis. Infection, fever, acidosis, hypoxia, stress, dehydration, sleep apnoea, and exposure to extremes of temperature can precipitate crisis.

Clinical features

Common sites of pain include bone (extremities, hand/foot syn
drome, back) and the abdomen. Bone pain, the most common type of VOC, may or may not be accompanied by swelling, low-grade fever, redness, and warmth. It may be symmetrical, asymmetrical, or migratory. Dactylitis is a common presentation in infants and toddlers; back and abdominal pain are more common in older children. Abdominal pain in children with sickle cell disease is usually a simple VOC

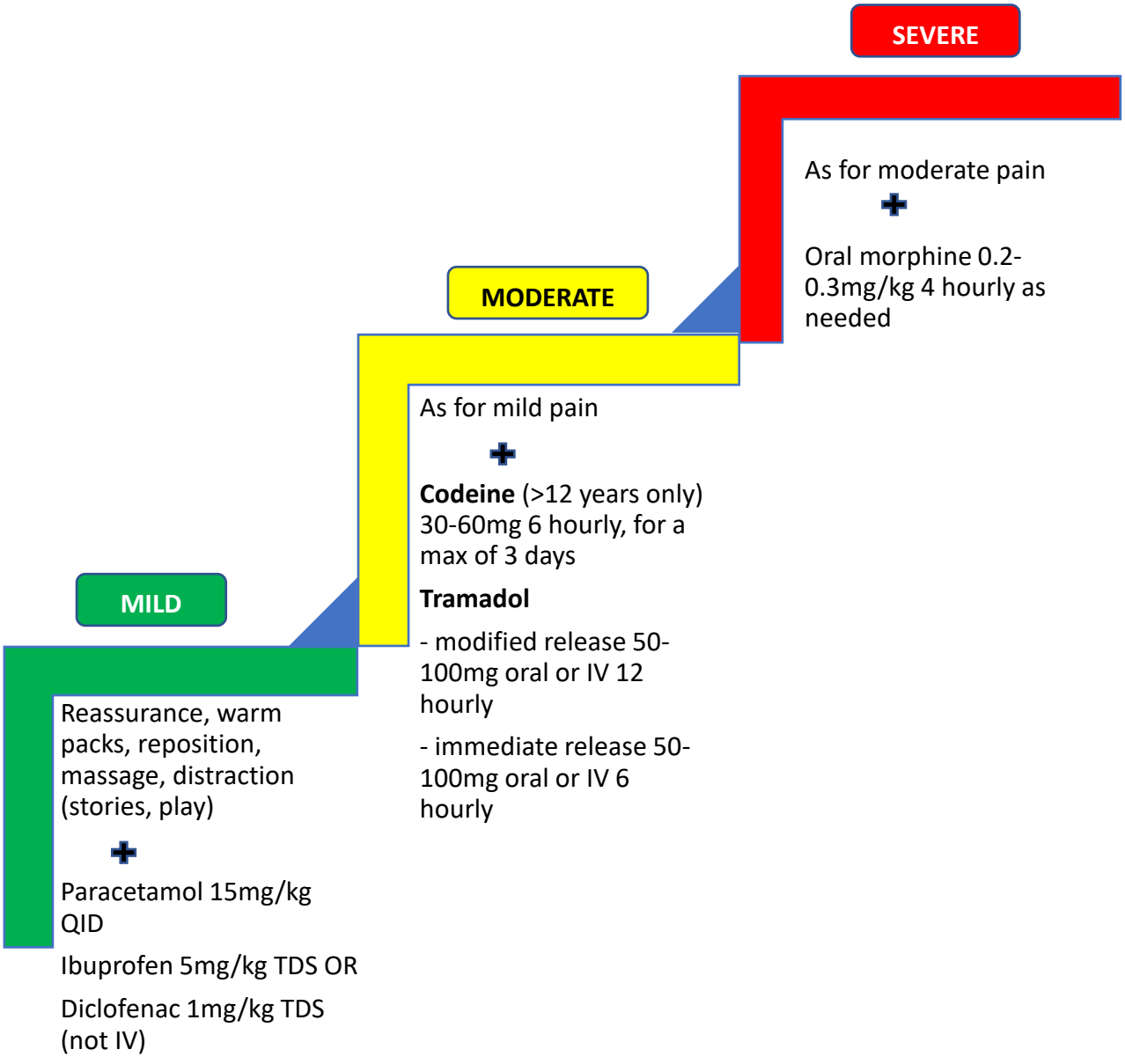
Investigations

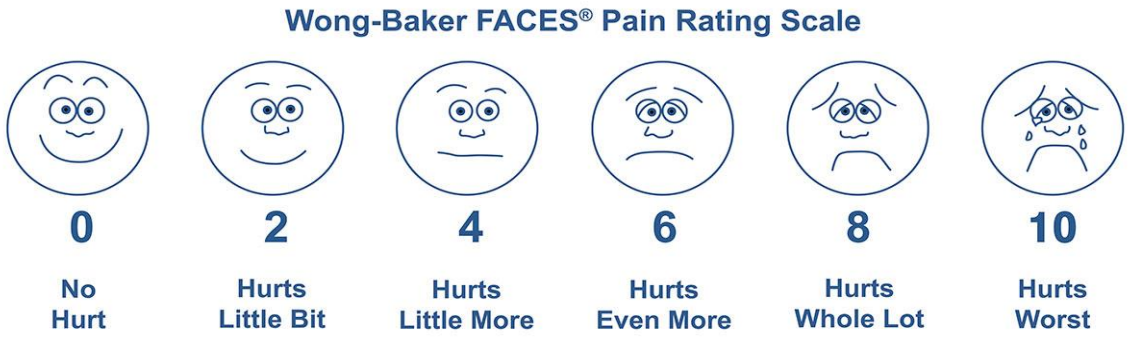
- FBC with differential and reticulocyte count (anaemia and reticulocytosis typical).
- Peripheral smear: sickled red blood cells, red blood cell fragments, Howell- Jolly bodies (splenic infarction).
- Total bilirubin and LDH (hyperbilirubinaemia and elevated LDH due to haemolysis).

Management

- Hospital admission is mandatory if pain control with oral analgaesics is not adequate, or if other problems (such as fever or dehydration) exist.
- Patient should receive analgaesia 30 minutes within hospital arrival.
- Assess severity of pain.
- If in mild pain, give paracetamol 15 mg/kg with NSAIDs (E.g. Ibuprofen 10 mg/kg with food given 3 times a day. Maximum 30 mg/kg per day.)
- If in moderate pain give or add tramadol 50- 100 mg orally 4- 6 hourly, for children > 12 years of age, adjusted according to response. Usual maximum 400 mg/ 24 hours.
- If in severe pain give or add opioid IV or oral morphine. For IV morphine, give 0.1 mg/ kg 4 hourly adjusted according to response. For oral morphine, give 0.2- 0.3 mg/kg (maximum per dose 10 mg) 4 hourly, adjusted according to response.

PAIN MANAGEMENT LADDER FOR PAIN N SICKLE CELL DISEASE





WHO step ladder management of pain

- For patients on morphine, give stool softener (e.g. lactulose) unless the child has diarrhoea.
- Hydration: 1.5 x daily maintenance IV fluid in 24 hours, if no cardiopulmonary compromise.
- The fluid of choice is 5% dextrose in normal saline (DNS).
- Do not give much fluid in patients with ACS, severe acute anaemia, stroke or acute splenic sequestration- There is a risk of cerebral or pulmonary oedema. In these cases, use 2/3 of maintenance fluid.
- Monitors vitals, and assess for improvement of symptoms.
- Rest and heat to the area of pain; avoid cold as this can worsen pain.

Discharge criteria

- Patient is tolerating fluids and medications by month, pain control is adequate with no medications, and concurrent problems are resolved.

REFERENCES

1. Guidelines for the Management of Sickle Cell Disease in Zambia. 2020
2. Kliegman RM et al. Nelson Textbook of Pediatrics. 20th ed. Philadelphia: Saunders Elsevier; 2016

12.5 PRIAPISM

Priapism is a sustained, unwanted painful erection unrelated to sexual stimulation and unrelieved by ejaculation. It affects ~35% of boys & men but is frequently understated. Prompt recognition and immediate initiation of conservative medical management may lead to penis returning to flaccid state & limit need for aggressive & invasive intervention.

Clinical features

- Document time of onset. Onset usually occurs during sleep, when relative oxygenation decreases.
- Erection lasting more than four hours unrelated to sexual interest or stimulation.
- Rigid penile shaft, but the tip of penis (glans) is soft.
- Progressively worsening penile pain
- No evidence of trauma.

Investigations

- FBC with differential and reticulocyte count
- Hb Electrophoresis
- Group and cross match
- Urinalysis and culture
- Blood culture if febrile

Management

- Rapid triage- place immediately into examination room & establish intravenous access.
- Hydration: 1.5 x maintenance in 24 hours, if no cardiopulmonary compromise. The fluid of choice is 5% dextrose in normal saline (DNS). If DNS is not available, refer to the fluid guidelines.
- Pain control: Paracetamol + NSAIDs. Escalate to opioids if severe (See Pain Control section).
- Benefit in voiding or having a warm shower or bath to improve blood circulation.
- Maintain oxygen saturation above 95% (if the patient is hypoxic).
- There is no Randomised Controlled Trial (RCT) for either simple or exchange transfusion in acute priapism and variable responses are described in the literature. Patients who require shunt procedures under general anaesthesia may require simple transfusion prior to anaesthesia if they have Hb below 9g/dL. Blood transfusion should be considered when acute priapism does not settle with conservative management and prior to surgery.
- The recommended treatment for priapism that does not settle with conservative treatment presenting at < 48 hours is penile aspiration of the stagnant ischaemic blood to decompress the corpora cavernosum.
- This is best performed with a 19-gauge needle through the glans or laterally from the penile shaft. It can be performed under local anaesthetic in the emergency department after a penile block by a urologist.
- Discharge if priapism is resolving, child is taking fluids & medications orally, is afebrile, and pain relief is adequate.

- Commence hydroxyurea as per protocol.
- Consider chronic phosphodiesterase type 5 (PDE5) inhibitor therapy such as Sildenafil in patients with recurrent priapism.
- Patients with recurrent episodes of acute priapism will usually employ self- management techniques at home including exercise, hydration and urination and they will usually be prescribed an alpha adrenergic agent.
- Joint urology/haematology follow up.

REFERENCES

1. Guidelines for the Management of Sickle Cell Disease in Zambia. 2020
2. Kliegman RM et al. Nelson Textbook of Pediatrics. 20th ed. Philadelphia: Saunders Elsevier; 2016

12.6 ACUTE SPLENIC SEQUESTRATION

Sudden onset enlargement of the spleen and reduction in Hb concentration by at least 2 g/dL below the baseline value. Splenic pooling of a large amount of blood and platelets typically occurs in children with HbSS between the age of 6 months and 5 years.

Clinical Features

- Sudden onset asthenia
- Increased fussiness
- Thirstiness
- Decreased activity (lethargy)
- Acute anaemia (pallor, tachycardia, tachypnoea, frank cardiovascular collapse)
- Splenomegaly (pain in the left upper quadrant and abdominal distension)

Investigations

- FBC with differential and reticulocyte count
- Group and cross match
- Note: Imaging is not essential

Management

- Transfuse with pRBC urgently with goal back to baseline Hb. (Notify blood bank).
- Generally, transfuse with 10mL/kg packed red blood cells as patient is at risk of auto-transfusion (sequestered blood leaving the spleen).
- If in hypovolaemic shock, manage for shock with guidance of senior present. Refer to management of hypovolaemic shock.
- Avoid aggressive splenic palpation to prevent splenic rupture.
- Usually resolves within 2- 5 days.
- In consultation with haematologist, address the performance and timing of splenectomy to prevent recurrence.

REFERENCES

1. Guidelines for the Management of Sickle Cell Disease in Zambia. 2020
2. Kliegman RM et al. Nelson Textbook of Pediatrics. 20th ed. Philadelphia: Saunders Elsevier; 2016

12.7 ACUTE CHEST SYNDROME (ACS)/PNEUMONIA

Acute Chest Syndrome presents with a new infiltrate on CXR plus any one of the following:

- Temp > 38.5
- Cough
- Wheezing
- Tachypnoea
- Chest pain

It commonly occurs in children aged 2 to 4-years. It causes life threatening lung infarction. It occurs in 10-20% of hospitalized patients with SCD, usually 1-3 days after admission for severe VOC.

Clinical Features

- Fever
- Cough
- Excruciating chest pain
- Shortness of breath
- Low oxygen saturation
- Often precipitated by a lung infection
- Low O_2 leads to further sickling of red cells, thus exacerbating pulmonary and systemic hypoxaemia, sickling & VOC.

Investigations

- FBC with differential and reticulocyte count
- C- reactive protein
- Arterial blood gases
- Group and cross match
- Hb electrophoresis
- LFTs
- Chemistries
- Blood culture
- Total bilirubin and/or Lactate dehydrogenase (LDH).
- CXR- posterior anterior (PA) and lateral views (NOTE: 60% of children with ACS will have an infiltrate on CXR but a normal pulmonary exam.

Management

- Hydration: IVF at 2/3 maintenance maximum depending on oral intake. Can decrease or discontinue IVF if taking enough PO fluids to meet daily maintenance goal.
- Antibiotics for severe pneumonia: Cefotaxime or ceftriaxone + Erythromycin or azithromycin.
- Incentive spirometry is indicated for older children with chest or back pain
- Over- hydration can lead to pulmonary oedema and worsen acute chest syndrome.

- Pain management: be cautions with narcotics but do not withhold. Balance between pain control and sedation as either may lead to respiratory distress. Narcotics can lead to hypoventilation and worsen acute chest syndrome.
- Respiratory care: oxygen therapy with pulse oximetry monitoring.
- Blood transfusion: 10- 15mL/kg pRBC if significant anaemia (Hb <5g/dL or if Hb drops >2 g/dL from baseline), significant hypoxia, or worsening respiratory status.

Discharge criteria

The child does not require supplemental oxygen, has been afebrile for at least 24 hours, the child is taking fluids and medications by mouth, pain control is adequate with PO medications, and concurrent problems are resolved.

REFERENCES

1. Guidelines for the Management of Sickle Cell Disease in Zambia. 2020
2. Kliegman RM et al. Nelson Textbook of Pediatrics. 20th ed. Philadelphia: Saunders Elsevier; 2016

12.8 STROKE

Stroke presents as rapidly developing clinical signs of focal (or global) disturbance of cerebral function, lasting > 24 hours or leading to death, of vascular origin. It occurs in 10- 15% of children with sickle cell disease. The peak age of onset is 2- 5 years.

Clinical features

- Hemiparesis
- Facial weakness
- Severe headache (w/without associated vomiting)
- Visual and language disturbances
- Seizures (especially focal seizure)
- Altered sensation, behaviour and mental status
- Coma

Investigations

- FBC with differential and reticulocyte count
- Blood glucose
- RDT for malaria/MPS
- Serum electrolytes
- Hb electrophoresis
- Group and cross match
- Emergent CT scan or MRI/MRA/MRV (where available).
- **NOTE** contrast may exacerbate sickling. Discuss using contrast with paediatric haematologist on call.

Management

- High flow oxygen
- Monitor Glasgow Coma Scale (GCS) and vitals regularly
- Control seizures if present
- Look for signs of raised ICP and refer to neurology section on stroke.
- Urgent blood transfusion at 10mL/kg is recommended (NOTE exchange transfusion is preferred management where available) to target Hb S < 30%.
- Patients presenting with suspected transient ischaemic attack (TIA) or stroke should have urgent neuroimaging.
- Commence on hydroxyurea.
- Enroll in chronic monthly blood transfusion protocol (see National SCD management guidelines).
- For patients on chronic monthly transfusion, monitor for iron overload.
- Never exceed a haematocrit of 30% due to a risk of hyperviscosity.

REFERENCES

1. Guidelines for the Management of Sickle Cell Disease in Zambia. 2020
2. Kliegman RM et al. Nelson Textbook of Pediatrics. 20th ed. Philadelphia: Saunders Elsevier; 2016

12.9 SICKLE CELL DISEASE WITH INFECTION/FEVER

Infection is a major complication of SCD, causing morbidity and mortality. Many children with sickle cell anaemia have significant haemolytic anaemia and impaired splenic function. The spleen's phagocytic function is markedly reduced, putting them at risk of overwhelming sepsis.

Common pathogens (encapsulated bacteria)

- Streptococcus pneumoniae
- Haemophilus influenzae type b
- Escherichia coli
- Salmonella
- Mycoplasma pneumoniae
- Chlamydia pneumoniae
- Streptococcus pyogenes
- Staphylococcus aureus
- Neisseria meningitidis

Investigations

- FBC with differential and reticulocyte count
- RDT for malaria or Malaria Parasite Slide (MPS)
- Group and save
- Total bilirubin and/or LDH
- Urinalysis
- Blood culture
- CXR
- When clinically indicated- blood chemistries, urine culture, stool culture, lumbar puncture and evaluation for osteomyelitis.

Treatment

- Admit for intravenous antibiotic if any of the following is found: age < 1yr, temperature >38°C, toxic or ill- appearing patient, history of pneumonia, pulse oximetry reading <90%, on room air, Hb <6 g/dL, WBC <5, 000/mm^3 or > 30, 000/mm^3, platelet count < 100, 000/mm^3.
- Oxygen if indicated.
- Blood transfusion if Hb < 5 g/dL.
- Hydration
- Start gram- negative rod coverage with intravenous cephalosporin such as ceftriaxone OR cefotaxime for 7- 10 days.
- Consider Ciprofloxacin for suspected Salmonella osteomyelitis.
- Patient with ACS/severe pneumonia, add erythromycin 50mg/kg/day 6 hourly/ or azithromycin 10 mg/kg daily.
- Anti-pyretic such as paracetamol.

REFERENCES

1. Guidelines for the Management of Sickle Cell Disease in Zambia. 2020

12.1.1 SICKLE CELL CARDIOMYOPATHY

Sickle cell cardiopathy develops due to the hyperdynamic status of a child living with SCD. They are at increased risk of developing restrictive cardiomyopathy characterized by diastolic dysfunction, left atrial dilation and normal systolic function. This results in mild, secondary pulmonary venous hypertension and elevated tricuspid regurgitant jet velocity. Systolic dysfunction is very uncommon.

Clinical features

- Asymptomatic
- Tachycardia
- Galloping Heart sounds
- Loud P2 due to pulmonary hypertension

Management

Anti- failure medication is not prescribed for cardiomyopathy, unless or otherwise guided by physical assessment, Echo and chest X- ray. In the presence of high output failure due to severe anaemia, Red Cell blood transfusion is used to treat the cardiac failure. Prolonged use of loop diuretics is not advised as it may lead to dehydration of the patient, which may trigger a VOC. If the patient is otherwise asymptomatic for cardiac failure, the patient may have Echo screening done annually.

REFERENCES

1. Guidelines for the Management of Sickle Cell Disease in Zambia. 2020
2. Kliegman RM et al. Nelson Textbook of Pediatrics. 20th ed. Philadelphia: Saunders Elsevier; 2016

12.1.2 OSTEOMYELITIS

Osteomyelitis is a infection affecting the metaphysis of bone. The commonest site of osteomyelitis is the lower limbs, especially the tibia. It is important not to miss the acute stage of osteomyelitis as this helps prevent serious long-term orthopaedic sequelae caused by chronic osteomyelitis. Also note that it is very difficult to distinguish between acute osteomyelitis and bone infarctions caused by VOCs. A high index of suspicion from history, and thorough workup is important.

Classification

Osteomyelitis can be classified, depending on the duration and presentation, into:

- Acute
- Subacute
- Chronic

Subacute osteomyelitis should be distinguished from post- acute osteomyelitis as this is a type of osteomyelitis that is caused by less virulent organisms.

Micro-organisms implicated in osteomyelitis include:

- Staphylococcus aureus (commonest)
- Salmonella typhi (common)
- non-Salmonella typhi spp (common)
- Escherichia coli
- Pseudomonas aeruginosa
- Enterobacter spp
- Hemophilus influenza type B
- Klebsiella spp
- Proteus spp

Bacteraemia caused due to the gram-negative microbes is believed to result from sickling within the mesenteric vessels and subsequent gastrointestinal infarction and translocation of the GI microorganisms into the bloodstream.

Clinical features

Acute osteomyelitis:

- Fever or chills
- Irritability or lethargy in young children
- Pain in the area of the infection
- Swelling
- Warmth and redness over the area of the infection
- Loss of movement
- Sometimes no signs and symptoms or
- Have signs and symptoms that are difficult to distinguish from other problems such as VOC.

Sub-acute osteomyelitis:

- Uncommon infection with bone pain
- Radiographic changes without systemic symptoms
- It is usually caused by indolent bacteria
- Characterised by a Brodie's abscess on imaging.

Post-acute osteomyelitis:

- Same as acute stage with increased bone pain
- Possible pyomyositis (abscess).

Chronic osteomyelitis:

- Pus discharge from sinus
- Pathological fractures presenting as acute pain
- Deformity or
- Inability to bear weight on the affected limb.

Differential diagnosis

- VOC (Bone infarctions)
- Septic arthritis
- Local skin infection/ pyomyositis (abscess)
- Tumour

Investigations

- Gold standard tools for the diagnosis is bone biopsy test with histopathological examination and tissue culture, (both aerobic and anaerobic bacteria culture)
- FBC,
- Blood cultures, pus swab and culture
- CRP/ESR
- X- ray- changes in acute osteomyelitis do not appear until about 10 days after the onset of symptoms which coincides with the post-acute stage.
- Ultrasound/MRI in acute episodes are difficult to assess**.**
- Joint aspiration for culture of septic arthritis (never aspirate an affected joint without prior consultation with a consultant).

Management

Acute osteomyelitis should be managed medically with antibiotics and no surgical intervention is required.

- Cloxacillin for *S. aureus* osteomyelitis (commonest)
- If Gram stain shows gram-negative bacilli, add a third-generation cephalosporin
- Ciprofloxacin can also be used especially when we suspect Salmonella species.

Note!! The total duration for antibiotic

- Administration should be for at least 4 – 6 weeks which should include 1-2 weeks of intravenous administration.
- Serial infection markers should be done to monitor antibiotic response. ESR is less reliable in neonates and sickle cell patients.

Other supportive management includes:

- Hydration
- Adequate pain management
- Rest of limb

Orthopaedics consultation:

For post-acute and chronic osteomyelitis

- Bone drilling if acute (to relieve the exudate and hence pressure in the medullary cavity to prevent bone death; sequestrum formation) and post-acute osteomyelitis (to reduce the pressure from the accumulated pus within the medullary cavity of the bone).
- Splintage of affected limb with Plaster of Paris (POP) or traction for pathological fractures prior to referral.
- Co-management of patient by medical and surgical teams is needed.

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12.1.3 PERIOPERATIVE MANAGEMENT OF CHILDREN WITH SCD

Children with SCD are predisposed to complication requiring surgical procedures, such as cholecystectomy and splenectomy. Peri-operative complications are higher in patients with SCD compared with the general population and may be sickle or non-sickle-related. Complications may be reduced by meticulous peri-operative care and blood transfusion.

Research showed that for the majority of minor elective procedures (hernia repair, circumcision, and dental rehabilitation) in sickle cell patients, pre-operative transfusions are unnecessary, as these patients usually have uncomplicated courses. On the other hand, patients undergoing thoracotomy, laparotomy, or tonsillectomy/adenoidectomy are at higher risk of developing post-operative complications (the rate is 50%) and may benefit from pre-operative transfusion regimens. These procedures are characterized by longer intra-operative duration and by compromised chest wall and pulmonary mechanics.

If transfusion is undertaken, the choice includes simple or exchange (automated or manual) transfusion. A simple transfusion may be of benefit when the patient's haemoglobin is below baseline. But by raising Hb without significantly lowering HbS, a simple transfusion may dramatically increase blood viscosity and thereby increase the risk of sickle-related complications. Red cell exchange transfusion allows dilution of HbS without increasing haematocrit. Transfuse RBCs in children with SCD to bring the haemoglobin level to 10 g/dL.

Pre-operative assessment and preparation

- Senior member of the anaesthetic pre- assessment team should review elective case to assess arrangements for pre- operative transfusion, requirement for an ICU bed, and pain management plan.
- FBC, urea, electrolytes, creatinine, Hb electrophoresis, antibody screen (if possible) and group and cross match must be done before surgery.
- Measure baseline oxygen saturation.
- Patient should receive either a simple blood transfusion or exchange transfusion to bring the haemoglobin level to 10 g/dL prior to undergoing surgical procedure involving general anaesthesia.
- Echocardiogram should be considered for in children with symptoms suggestive of pulmonary hypertension (SpO_2 < 94%, reduced exercise capacity).
- For children < 16 years of age, the results of transcranial Doppler (TCD) assessment should be available and documented from the previous 12 months. If abnormal, there should be a multidisciplinary discussion as to how the child should be managed.
- Starvation instruction should be documented clearly and reinforced with the patient and their family.
- All patients should be given warmed DNS IV for a minimum of 12 hours at maintenance flow rate during starvation for the operation.

Perioperative management

- The operating room should be pre- warmed, with assurance that temperature can be well monitored and properly maintained, because hypothermia can trigger sickling.
- The patient should be well oxygenated pre- operatively; induction and intubation should be undertaken with little or no hypoxic insult.
- The temperature should be monitored regularly postoperatively. Note that a spike in temperature may be an early sign of sickling.
- Monitor oxygen saturation and Arterial Blood Gases (ABG) closely.

Post- operative management

- This begins in the operating room, with assurance that the patient does not become hypothermic, hypoxic, or hypotensive during emergence from anaesthesia. Before extubation, the patient should be well awake, ventilating and oxygenating well.
- In the recovery room, assess the patient carefully before transferring the child to a paediatric ward. Request a CXR, if there is any concern about respiratory function.
- Patients should be monitored for O_2 saturation (aim to keep O_2 sat >96%); children 4 years or older should be on a pulmonary clearing programme (incentive spirometry). If atelectasis or oxygen desaturation develops, seriously consider admission to the ICU with intensified efforts at pulmonary clearing, including re-intubation if necessary.
- Maintain hydration to prevent vasoconstriction, hypoperfusion, and microvascular occlusion, which ultimately lead to sickling. Avoid over-hydration, however, because pulmonary interstitial oedema can lead to hypoxia and a sickling crisis.
- Warmed intravenous fluids should be continued until the patient is able to tolerate an adequate intake of oral fluids. Fluid balance should be monitored.
- Provide appropriate analgaesia so that the patient is co-operative with ambulation and pulmonary clearing, but not so much that he/she is constantly narcotized and asleep.
- Patients with SCD are more susceptible to infection than the general population, including respiratory and postoperative wound infections. Infection in the peri- operative period may precipitate sickle complications such as painful crisis or ACS. Antibiotic prophylaxis should be administered as dictated by the surgical procedure.
- If operative complications develop or the patient becomes toxic or septic, consider early transfer to the ICU (in consultation with paediatric haematology).

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12.1.4 MANAGEMENT OF HYPERHAEMOLYSIS IN SICKLE CELL DISEASE

Hyperhaemolysis is a rare life-threatening complication in SCD with rapidly dropping haemoglobin, raised lactate dehydrogenase, intravascular haemolysis and haemoglobinuria leading to multi organ failure and death.

It is characterised by rapid haemolysis following a blood transfusion, and the post- transfusion haemoglobin will often be lower than the pre- transfusion haemoglobin, implying the destruction of recipient as well donor red blood cells (RBCs).

Blood transfusion is an important treatment in the management of patients with SCD. However, about one third of transfused SCD patients develop antibodies to red blood cell antigens, becoming alloimmunized, and around 10% develop the most serious consequence of this alloimmunization which is a delayed haemolytic transfusion reaction (DHTR).

The rapid haemolysis and may be associated with fever and with pain. The direct antiglobulin test (DAT) may be either negative or positive and new red cell allo-antibodies are not usually identified but may be present. There may be a reticulocytopenia. Hyperhaemolysis can recur in such patients following blood transfusions several months or years after the initial episode.

Erythropoietin, iron replacement, B12 and folate replacement is usually required and should be considered.

Hyperhaemolysis can recur many years after the first episode and transfusion is generally avoided if possible in all patients who have had an episode. However, should a blood transfusion be clinically indicated in such a patient, this must be discussed and agreed with a haematology consultant, and the patients should be retreated with IVIG prior to transfusion. Patients with hyperhaemolysis should be treated with intravenous immunoglobulin (IVIG) and IV methylprednisolone.

Diagnosis

Hyperhaemolysis should be considered in any patient with SCD who presents with increasing haemolysis after a blood transfusion, typically, 1-week post transfusion, but may occur sooner than this if the patient is re-challenged with transfusion.

Clinical features

Increasing jaundice, dark urine (‘coca-cola’ coloured), anaemia. They may also have a fever, back, leg or abdominal pain, hepatomegaly or hepatic discomfort.

Investigations

- **FBC**: Worsening anaemia – Hb may fall to below the pre-transfusion level.
- **Reticulocytes:** May be raised (in keeping with haemolysis) or decreased, due to suppression of red cell production.
- **Direct Antiglobulin Test (DAT):** Usually negative but can be positive, blood bank should send for an eluate if DAT is positive.
- **Group and screen:** New allo-antibodies may be found but are usually absent.
- **Haemoglobin electrophoresis to measure Hb S% and Hb A%:** This is useful to quantify how much, if any, Hb A (transfused blood) remains.
- **Other markers of haemolysis**:
  - Raised Bilirubin
  - Raised LDH
  - Hyperferritinaemia may also be seen as a marker of macrophage activation

Treatment

- All SCD patients with suspected hyperhaemolysis must be discussed with a Haematology Consultant.
- Supportive management should continue with analgaesia, hydration and oxygen therapy as required.
- Prescribe folic acid 5mg.
- Consider treatment with erythropoietin and providing IV iron replacement if not iron replete (i.e. if ferritin <100 ng/ml).
- Consider B12 replacement.
- Blood transfusion may be necessary if clinically indicated (profound symptomatic anaemia), but should only be given after discussion with the Haematology consultant.
- Phenotyped blood should be given (C,D,E and Kell matched). Notify blood bank immediately.
- Primary treatment is with IV methylprednisolone and IVIG.

Dosages of drugs

- **Intravenous immunoglobulin (IVIG)**

Dose 1g/kg once daily for 2 days. Round the dose to nearest vial size (5 g, 10 g and 20 g vials available).

- **Methylprednisolone**

Dose 1 mg/kg daily in 2 divided doses IV for 2 days. Review dose after 2 days.

- **Erythropoietin**

Dose 300 IU/kg once daily for 5 days. Then 300 IU/kg once daily on alternate days (i.e. 3 times per week).

Hyperhaemolysis can be severe and there are documented cases that have had fatal outcomes. If there is ongoing haemolysis despite actions above this must be discussed urgently with the haematology consultant for SCD. There are case reports of successful outcomes using eciluzimab and rituximab. These may be considered though not licenced yet.

Monitoring of treatment

- Haemoglobin: Target is return of haemoglobin to baseline.
- Stop erythropoietin if the haemoglobin returns to baseline, or if lack of response after full treatment dose.

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12.1.5 IMMUNE THROMBOCYTOPENIA

Immune thrombocytopenia is defined by a platelet count of < 100, 000 x 10^9/L (usually < 20 x 10^9/L).

It is a self- limiting disease with shortened platelet survival and increased megakaryocytes. It resolves in 80% of patients within 6 weeks to 6 months and has a good prognosis. The mechanism of action is shortened platelet survival due to platelet autoantibody production.

It is classified as

- Acute 0- 3 months
- Persistent 3- 12 months
- Chronic > 12 months.

Clinical presentation

- Acute onset bruising, purpura and petechiae.
- History/evidence of mucocutaneous bleeding. petechiae, and bruising but otherwise well appearing.
- History of previous viral infection in the last 2- 3 months (often, but not always) or recent live virus vaccination administration.
- Absence of hepatosplenomegaly, lymphadenopathy, and evidence of serious cause/chronic underlying illness.
- If headache and/or neurological signs, urgent CT scan of the head.

Investigation

1. Bone Marrow aspiration unnecessary unless:

- Neutropenia
- Hepatosplenomegaly
- Lymphadenopathy
- Pain limb/abdomen/back
- Limp.

2. Isolated thrombocytopenia.

- Typically, < 20, 000/mm3.
- Smear results consistent with ITP
    - Platelet size normal or large in size.
    - Occasional giant platelets seen.
    - Normal WBC and RBC morphology
    - Microcytic, hypochromic red cells due to underlying iron deficiency is common.
    - Occasional activated lymphocytes can also be seen.
    - Absence of tumour lysis (normal potassium, phosphorus, uric acid, LDH)

ITP is a diagnosis of exclusion so a thorough history and physical examination and work up needs to be taken. You must rule out leukaemia.

If the following features are present, then you need to think about a diagnosis other than ITP.

- Family history of low platelets
- Medication exposure including antibiotics, anticonvulsants, heparin, anti- arrhythmia medication, sulfa drugs, aspirin.
- Haemarthroses or significant bleeding
- Evidence of active infection or fever.
- Arthralgia
- Weight loss, bone pain
- hepatomegaly, splenomegaly, lymphadenopathy.
- Dysmorphic features, skeletal abnormalities, growth delay, failure to thrive.
- Abdominal pain, bloody diarrhoea, renal failure, haemolytic anaemia in additional to thrombocytopenia then think HUS/TTP.
- HIV risk- exposure of HV + status (although ITP is common with HIV).

Immediate Treatment

- None regardless of platelet count, unless life- threatening owing to significant bleeding. Monitor platelet counts and educate the family about potential bleeding symptoms.
- If significant bleeding (e.g. uncontrolled epistaxis, GI haemorrhage, intracranial bleeding), give platelets along with immunoglobulin (IVIG) at 0.8- 1 g/kg by IV infusion for 2 days.
- If moderate bleeding, prolonged mucosal bleeds, give prednisolone 2 mg/kg/day (Maximum 120 mg daily) for 5-7 days only. There is no benefit with longer duration of corticosteroids.
- Consider Tranexamic acid for mucosal bleeds.
- Avoid NSAIDs e.g. ibuprofen.
- Reassure parents. About 80% resolve in 6 months. Avoid contact sports.
- Continue monitoring FBC and PBS monthly until diagnosis is clear or recovery.

Chronic ITP

- Avoid NSAIDs
- Avoid contact sports
- Investigate for autoimmune diseases (ANA antinuclear antibody; APLA antiphospholipid antibodies; ACA, anticardiolipin antibody; and LAC, lupus anticoagulant) and immune deficiency (HIV, IgG, IgA, IgM).
- Treat only:
  - Profound thrombocytopenia (<10 x 109/L) with repeated mucosal bleeding.
  - Older girls with menorrhagia.
  - Diminished health related quality of life
  - Trauma
  - Acute neurological signs

- If treatment indicated, give dexamethasone 0.6 mg/kg/day (maximum 40 mg).
- If unresponsive, discuss with paediatric haematologist about treatment with thrombopoietin receptor agonist (eltrombopag) or rituximab.
- Splenectomy reserved for those with persistent/ significant bleeding non- responsive or intolerant of other therapies.

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12.1.6 INFANTILE HAEMANGIOMA

Infantile haemangiomas (IH) are the most common vascular tumours, affecting around 4% of infants. IH typically appear in the first few weeks of life and grow rapidly for the first few months. Thereafter, slow proliferation can continue for up to 6- 12 months. Majority do not require treatment due to their spontaneous involution. However, about 15% of IH result in complications such as obstruction, ulceration, or disfigurement, and then require therapeutic intervention.

Propranolol has become the first- line therapeutic agent in the management of complex IH. Studies have shown that propranolol is a safe and effective treatment for IH in most patients.

Indications for initiating treatment with oral Propranolol

1. Vision compromise
2. Airway IH with actual or potential airway compromise.
3. Nasal IH with actual or potential obstruction
4. Lip IH causing actual or potential functional impairment and/or disfigurement.
5. Involvement of the auditory canal causing recurrent infection
6. Ulcerated IH, especially where topical treatment would not be appropriate or has not been effective.
7. Risk of permanent disfigurement
8. Spinal cord cord compression by IH
9. Liver IH in conjunction with cutaneous IH in selected cases in collaboration with other specialists.

Absolute Contraindications

1. Recent or ongoing hypoglycaemic episodes.
2. Second and third degree heart block
3. Hypersensitivity to propranolol hydrochloride.

Relative Contraindication

1. Frequent wheezing
2. Blood pressure outside normal range for age. Treatment in conjunction with paediatrician is advised.
3. Heart rate outside normal range for age. Treatment in conjunction with paediatrician is advised.

Baseline observations and investigations

- FBC
- Blood glucose
- Urea and electrolytes.
- Thyroid Function Tests (TFTs)
- Liver Function Tests (LFTs)
- ECG
- Echo
- Liver ultrasound should be requested for infants with more than 5 cutaneous haemangiomas to look for liver haemangiomas.
- MRI scan

Starting dose: A preparation of 1 mg/ml solution is preferable.

- Week 1: Propranolol 1 mg/kg day in 3 divided doses
- Week 2: Propranolol 2 mg/kg/day in 3 divided doses
- Dose escalation: If response is unsatisfactory after 4 weeks (and no adverse effects have occurred) the dose may be increased to: Propranolol 3 mg/kg/day in 3 divided doses.

Follow- up:
Should be arranged 4 weeks after treatment commences.

Note: Medication should be given with or shortly after food. If an inter- current illness causes significant vomiting, propranolol should be temporarily withheld to reduce the likelihood of hypoglycaemia.

Potential side effects:

- Bradycardia
- Hypoglycaemia
- Heart failure
- Hypotension
- Cardiac conduction disorders
- Bronchospasms
- Peripheral vasoconstriction
- Weakness and fatigue
- Sleep disturbances

Administration:
Use of the medication should be explained to the parents and information leaflet provided. Advice on potential side effects should be explained to parents prior to starting therapy.

Stopping Propranolol

Propranolol should be temporarily discontinued in the setting of significant reduced oral intake or of the patient has wheezing that requires treatment.

Treatment of IH should extend beyond the proliferative period of IH to avoid rebound growth and the decision when to stop treatment will have to be guided by clinical features. There is no uniform cut off age that determines the risk of rebound growth, although a European study suggested that children aged 17 months or older had a significantly lower risk of rebound growth compared to younger age groups.

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13.1 MALARIA

Malaria is one of the top five diseases causing morbidity and mortality in Zambian children. It is the 3rd leading cause of under-5 mortality following pneumonia and diarrhoea. Malaria is a febrile illness caused by *plasmodium falciparum, a* parasite which is transmitted from person to person by mosquitoes. Malaria is diagnosed by examining a patient's stained blood slide through a microscope (MPS). A malaria rapid diagnostic test (RDT) which detects the malaria antigen can be done in the examination room as a point of care test (POC) and results are available within 15 minutes. It is important to also do a full blood count (FBC). Left untreated, Malaria can be fatal hence prompt diagnosis and treatment is critical.
Uncomplicated malaria symptoms and signs are as follows:

- Fever
- Headache
- Abdominal pain
- Nausea
- Diarrhoea and Vomiting
- General body pains
- Weakness

Simple malaria is treated with Artemether 20 mg and Lumefantrine 120 mg) given with food as follows:

- stat,
- after 8 hrs,
- then 12 hourly doses on day 2 and day 3.

If the drug is spat out or vomited within 30 minutes, the dose should be repeated. If more than two consecutive episodes of vomiting occur, parenteral Artesunate should be considered when available.

Table 13. Malaria treatment

WEIGHT (Kg)	AGE (approx. in yrs.)	DOSE (number of tabs of AL, Artemether 20, Lumefantrine 120 mg)
5-<15	2months -3yrs	1 tab
15-25	3-8yrs	2 tab
25-35	9-12yrs	3 tab
>35	>12yrs	4 tab

Severe Malaria

Severe forms of malaria, may affect the

- Brain (cerebral malaria),
- Kidney (black water fever, acute kidney injury),
- Lungs (pulmonary oedema),
- Blood (severe anaemia)
- Cardiovascular system (shock).

The symptoms and signs of severe malaria include

- Convulsions
- Changes in behaviour
- Reduced level of consciousness
- Coma
- Severe pallor
- Respiratory distress
- Jaundice
- Shock
- Dark (Coca Cola) coloured urine
- Reduced urine output
- Bleeding tendency
- Generalized weakness

If a blood slide or RDT is negative then only children with severe disease or those with severe anaemia should get presumptive treatment immediately then to send a sample to the National Malaria Elimination Centre for further analysis.

Investigations for severe malaria

- Thick and thin malaria parasite smear or RDT
- FBC
- Dextrostix (RBS)
- U&Es and Creatinine
- LFTs
- Blood gases
- CXR
- Urinalysis
- Consider LP to exclude meningitis if indicated and if no signs of raised ICP

Severe malaria can include fever with any of the following

- Impaired level of consciousness (AVPU= V, P, U or low GCS <11, Blantyre Coma Scale <3).
- Unable to drink/feed.
- Respiratory distress and/or acidotic breathing.
- Severe anaemia Hb < 5g/dl.
- Hypoglycaemia blood glucose < 3 mmol/L.
- 2 or more convulsions within 24 hours or a single episode of status epilepticus.
- Plasma bicarbonate of < 15mmol/l 0r PH<7.35.
- Serum creatinine >3 times from baseline for age.
- Bleeding tendency- recurrent or prolonged bleeding from the nose, gums, melaena, haematemesis, venepuncture sites.
- Shock – impaired perfusion (Capillary refill time of >3 seconds, cool peripheries).

TREATMENT

Artesunate

- If any of the above features are present, those >20kg should be given Artesunate 2.4mg/kg of body weight IV or IM given on admission (time=0), then at 12hrs and 24hrs, then re-assess the patient and repeat MPS.
- For patients with weight <20kg give Artesunate at 3mg/kg IV or IM
- After initial parenteral treatment for a minimum of 24hrs, once the patient regains consciousness and can take medications orally, discontinue parenteral therapy and commence full course of Artemether Lumefantrine
- After initial parenteral treatment for a minimum of 24 hrs, and the patient has not regained consciousness or cannot take medications orally, continue parenteral therapy once a day for a maximum of 6 days and commence full course of Artemether Lumefantrine .
- Repeat the blood slide every 24 hours in patients with unsatisfactory clinical response until there is zero parasitaemia.
- If MPS remains positive despite full course of treatment, to consult the National Malaria Elimination centres and to send DBS on filter paper for further analysis.
- There should be an interval of at least 8hrs between the last dose of artesunate and the first dose of artemether lumefantrine.

In the absence of IV Artesunate, artemether may be used at 3.2mg/kg IM loading dose, then maintain with 1.6mg/kg OD for 5days **OR** Alternative 2nd line drug for severe malaria is

QUININE IV

- Loading dose of 20mg/kg body weight diluted in 10ml/kg of 5% or 10% dextrose by IV infusion over 4 hours. After eight hours, give a maintenance dose at 10mg/kg body weight over 4 hours, and repeat every eight hours until patient can swallow.

- Then use oral quinine at 10mg/kg body weight every eight hours to complete a seven-day course of treatment
- Treat hypoglycaemia with 5 ml/kg 10% dextrose IV, followed by a continuous infusion of either 5% or 10% dextrose
- Maintenance fluids/feeds and supportive oxygen by mask
- If weak pulse and CRT > 3 sec, give 20 mls/kg normal saline, up to 40mls/kg/day. Use blood for resuscitation, if Hb < 5g/dl at 20 ml/kg whole blood).
- Use inotropes when there`s poor response to fluid boluses
- Treat convulsions as indicated in the algorithm for treatment of convulsions.
- Children with cerebral malaria can receive concurrent treatment for possible bacterial infections in our setting.

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13.2 TYPHOID (ENTERIC FEVER)

Typhoid fever is a multisystemic infection caused primarily by Salmonella enterica subtype typhi.

CAUSES

Salmonella typhi, Salmonella paratyphi

Clinical features

High grade fever, coated tongue, anorexia, vomiting, hepatomegaly, diarrhoea, toxeamia, abdominal pain, pallor, splenomegaly, constipation, headache, jaundice, obtundation, ileus, intestinal perforation.

DIAGNOSIS

Culture

Ideally 1st week of symptoms - Blood culture
2nd week of symptoms - Urine culture
3rd week of symptoms - Stool culture
NOTE: As most patients present late, all three cultures should be taken on admission.

Widal test

A single Widal test may be positive in only 50% cases in endemic areas
Serial tests may be required
Not a preferred test in endemic settings

Diagnosis is usually clinical due to limited diagnostics

Any high fever of >72 hours duration (with aforementioned features), especially with no alternative diagnosis (l no signs of upper respiratory tract infection, meningitis or malaria) must be suspected of typhoid and managed accordingly.

Full Blood count

Leucopoenia (WCC < 4 x10^9/litre) with a left shift in neutrophils may be seen in a third of children; young infants may also commonly present with leukocytosis.
Anaemia – is a relatively common feature

Other serological tests (expensive)

1. Enzyme limmunoassay (Dot-Elisa)
2. Latex agglutination
3. Tubex^R

Management

- Early diagnosis and instituting appropriate supportive measures and specific antibiotic therapy are the key to appropriate management
- Adequate rest, hydration, correction of fluid-electrolytes and nutrition
- Anti-pyretic therapy (paracetamol) as required if fever > 39°C
- Antibiotic therapy

1st line therapy- **ciprofloxacin** (while awaiting culture results). Alternative → Azithromycin

2nd line (drug resistant) – **imipenem** **Alternative - Ceftriaxone**

- Monitor vital signs and thoroughly examine to recognise **surgical emergencies**

  Intestinal haemorrhage (<1%) and perforation (0.5-1%)

  Features:
  - sudden and/or worsening abdominal pain
  - Pulse Rate -a sudden rise in pulse rate,
  - BP- hypotension,
  - Temperature
  - Respiratory rate
  - Plain –X-ray abdomen should be done

Intestinal perforation and peritonitis may be accompanied by:

- a sudden rise in pulse rate,
- hypotension,
- marked abdominal tenderness and guarding, and
- subsequent abdominal rigidity.
- A rising white blood cell count with a left shift and free air on abdominal radiographs may be seen in such cases.

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13.3 SCHISTOSOMIASIS

Schistosomiasis is a parasitic disease caused by blood flukes (trematode worms) of the genus *Schistosoma*.

Transmission/infection

- People become infected when larval forms of the parasite released by freshwater snails penetrate the skin during contact with infested water
- There are 2 major clinical forms of schistosomiasis that are caused by 5 main species of blood fluke:

I. *Intestinal*

Schistosoma mansoni,
Schistosoma japonicum
Schistosoma mekongi (uncommon)
Schistosoma intercalatum (uncommon)

II. *Urogenital*

Schistosoma haematobium

Clinical Features

In acute infection, mild, maculopapular skin lesions may develop within hours after exposure. Depending on which specy is responsible for the infection, the clinical features may be distinct between intestinal, urogenital or both:

- ***Intestinal schistosomiasis***

Acute: Abdominal pain, diarrhoea, blood in the stool, fatigue.
Chronic: hepatomegaly early on, then Liver atrophy due to periportal fibrosis, splenomegaly ascites, portal and pulmonary hypertension.

- **Urogenital Schistosomiasis**

Acute: Haematuria, dysuria, urinary frequency.
Chronic: Fibrosis of the bladder and ureter, kidney dysfunction, bladder cancer (later stages).
Adolescent girls: genital lesions, vaginal bleeding, pain during sexual intercourse, nodules in the vulva.
Adolescent boys: pathology of the seminal vesicles, prostate and other organs.

Investigations

- Stool/Urine M/C/S for blood (including occult) and Schistosoma ova.
- FBC, DC (usually shows eosinophilia and microcytic anaemia).
- Urea, Electrolytes, LFTs.
- Chest x-ray
- Plain abdominal x rays

- Abdominal ultrasound
- Blood culture (if possible concomitant disease i.e. salmonellosis).
- Rectal Snip (especially for S. mansonii

Treatment

- Praziquantal is the treatment of choice for all forms of Schistosomiasis (children >2 years and adults: 40mg/kg as a single dose). Consider steroid therapy if very severe disease or features of high worm burden.
- Adverse effects of praziquantel include dizziness, headache, nausea, vomiting, diarrhoea, abdominal discomfort, bloody stool, urticaria, and fever following initiation of treatment. These are usually mild and last about 24 hours.

Prevention

Routine deworming of risk populations with Praziquantel 40/mg/kg as a single dose.
Sanitary disposal of human waste,
water sanitation programmes,
Health education about the source of infection

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13.4 TUBERCULOSIS IN CHILDREN

TB is chronic infection cause by mycobacterium tuberculosis complex. It is capable of causing multisytemic disease but pulmonary disease is the commonest. TB in children is an indicator of recent and ongoing transmission of M. tuberculosis in the community, as majority of children will develop tuberculosis disease within 1 year after infection. (Unlike tuberculosis in adults who may develop TB disease when their immunity goes down).

Pulmonary TB is the commonest type of TB in children but extrapulmonary disease is also common estimated to be around 30-40% of cases.

Most immunocompetent children with TB disease present with nonspecific symptoms of a chronic disease. In infants the presentation may be more acute and can present as acute severe, recurrent or persistent pneumonia. TB should be suspected when there are features of infection with a poor response to appropriate conventional antibiotics.

Key risk factors for TB disease in children

- Household contact with a newly diagnosed smear positive case.
- Age < 5 years
- HIV infection
- Severe malnutrition

Symptoms of tuberculosis

TB should be suspected in children presenting with following symptoms especially if they persist for more than 2 weeks or show no response to (appropriate) antibiotic therapy for the initial diagnoses.

- Cough
- Fever
- Loss of appetite
- Weight loss or failure to thrive
- Decreased activity.

Other symptoms will depend on the anatomical site of the disease.

Clinical signs suggestive of pulmonary TB.

There are no specific features on clinical examination that can confirm that the presenting illness is due to TB hence a high index of suspicion in an endemic setting.

Clinical signs/features suggestive of extrapulmonary TB.

- Gibbus- Spinal TB, especially of recent onset.
- Non-painful enlarged cervical lymphadenopathy, with or without fistula formation- TB of lymph nodes.
- Pleural effusion
- Pericardial effusion

- Distended abdomen with ascites- TB abdomen.
- Non painful enlarged joints- Osteoarticular TB.
- Meningitis not responding to antibiotic treatment - TB meningitis.

Guidance on approach to diagnosis of TB in children

- Careful history (including history of TB contact and symptoms consistent with TB).
- Clinical examination (including growth assessment).
- Bacteriological confirmation with Xpert MTB/RIF or culture or smear microscopy).
- Chest X-ray
- Tuberculin skin testing (>2years)
- Urinary lateral flow lipoarabinomannan (LAM) for HIV infected children) disease.
- Other appropriate Investigations relevant for suspected extra-pulmonary TB.
- HIV testing

Appropriate specimens should be obtained for Xpert MTB/RIF testing, staining and microscopy, culture (and histopathological examination in extrapulmonary TB).

For pulmonary tuberculosis

Samples for Xpert MTB/RIF or culture testing should be obtained using any the following methods depending on the age of the child;

- Gastric lavage
- Sputum induction
- Nasopharyngeal aspiration
- Expectorated sputum

For extrapulmonary TB

- Pleural fluid: Xpert MTB/RIF, biochemistry, cell count and culture.
- Pericardial fluid: Xpert MTB/RIF, biochemistry, cell count and culture.
- Lymph node biopsy or Fine-needle aspiration of enlarged lymph glands: Xpert MTB/RIF, ZN staining, culture and histology.
- CSF: Biochemistry, cell count, Xpert MTB/RIF, TB culture.
- In addition, appropriate imaging studies such as abdominal ultrasound, Echocardiogram, CT and MRI should be used depending of the site of disease of extrapulmonary disease.

Table 13.1. Recommended TB treatment regimens in children

TB disease category	Recommended regimen	
	Intensive phase	Continuation phase
	2 (HRZE)	4 (HR)
Severe forms - TB meningitis, Osteo-articular TB, Spinal TB, Miliary TB, other severe forms of TB	2 (HRZE)	10 (HR)

Table 13.2. TB drug dosing table

Weight bands	Number of Tablets		
	Intensive Phase		Continuation Phase
	RHZ (75/ 50/150 mg)	E* (100 mg)	RH (75/50 mg)
4-7 kg	1	1	1
8-11kg	2	2	2
12 – 15 kg	3	3	3
16- 24 kg	4	4	4
25 and above			

Rifampicin (R) – 15 mg/kg (range 10 – 20 mg/kg); maximum dose 600 mg/kg, Isoniazid (H) – 10 mg/kg (range 7 – 15 mg/kg); maximum dose 300 mg/kg, Pyrazinamide (Z) – 35 mg/kg (30 – 40 mg/kg) and Ethambutol (E) – 20 mg/kg (15 – 25 mg/kg)

Use of corticosteroids

Corticosteroids are indicated in the management of some complicated forms of TB such as:

- TB meningitis.
- Complications of airway obstruction by TB lymph glands.
- Pericardial TB.

Prednisolone is recommended at a dose of 2mg/kg daily, increased to 4 mg/kg daily in the case of the most seriously ill children, with a maximum dosage of 60 mg/day for 4 weeks. The dose should then be gradually tapered over 2–4 weeks before stopping.

Pyridoxine supplementation.

Isoniazid may cause symptomatic pyridoxine deficiency, which presents as neuropathy, particularly in severely malnourished children and HIV-positive children on antiretroviral therapy (ART). Supplemental pyridoxine (5–10 mg/day) is recommended in HIV-positive or malnourished children being treated for TB.

Drug resistant tuberculosis in children

Drug-resistant TB (DR TB) should be suspected under the following conditions:

- Close contact with a person known to have DR-TB including household and other contacts.
- Close contact with patient that died from TB, failed or is not adherent to TB treatment.
- History of previous TB treatment (in the past 6-12 months).
- Not improving after 2-3 months of first line TB treatment, including persistence of positive smear or culture, persistence of symptoms and failure to gain weight (radiological improvement is frequently delayed).
- A child who develops active TB while on Isoniazid (INH) prophylaxis.

- Appropriate samples should be obtained in any suspected case of DR-TB. These should include the following:
- Xpert MTB-RIF
- Mycobacterial culture
- drug susceptibility testing (DST)
- line-probe assays (LPA)

Indications to initiate MDR TB regimen in children;

- Confirmed MDR TB by DST or LPA.
- RR on Xpert MTB/RIF.
- Smear positive case with confirmed MDR contact.
- Child with TB and unconfirmed DST resulting who is not responding to standard TB therapy and is a known contact of an MDR TB case.

For treatment of DR TB refer to the Guidelines for the Programmematic Management of Drug Resistant Tuberculosis and discuss with the TB coordinator at the district/province or clinician with experience in treating drug resistant TB.

Indications for Tuberculosis preventive therapy (TBT) in children

TB preventive therapy is recommended for the following at risk groups of children

- Asymptomatic HIV negative children who are household contacts of bacteriologically confirmed tuberculosis in whom active TB disease has been excluded.
- All HIV infected children, regardless of TB contact status, who are considered unlikely to have active TB disease. For HIV infected infants' preventive therapy is provided only when child has established TB contact.

Table 13.3. Recommended and dosing options for TBT in children

Drug regimen	Dose per kg body weight	Maximum dose
Isoniazid daily for 6 – 9 months	H 10 mg (range, 7 – 15 mg)	300 mg
isoniazid plus rifampicin daily for 3 – 4 months	H 10 mg (range, 7 – 15 mg) R 15 mg (range, 10 – 20 mg)	H – 300 mg R – 600 mg
Rifapentine plus isoniazid Weekly for 3 months (12 doses)	H: Age ≥ 12 years: 15 mg/kg, H: Age 2 – 11 years: H 25 mg/kg Rifapentine 10.0 – 14.0 kg = 300 mg 14.1 – 25.0 kg = 450 mg 25.1 – 32.0 kg = 600 mg 32.1 – 49.9 kg = 750 mg ≥ 50.0 kg = 900mg	H 900 mg Rifapentine, 900 mg

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13.5 PAEDIATRIC HIV

Zambia is part of Global Alliance to End AIDS in Children by 2030 with focus on eliminating mother -to child- transmission of HIV, Syphilis and Viral hepatitis B.

In 2021, Zambia made significant progress towards achieving the UNAIDS 95-95-95 targets. According to the results of the most recent representative sample survey on HIV prevalence conducted in 2018, HIV prevalence was 14.2 percent among women aged 15 to 49. According to the Zambia Population HIV Impact Assessment (ZAMPHIA 2016), HIV prevalence among children aged 0-14 years is 1.3 percent. In Zambia, the overall HIV incidence (new infections) is estimated to be 3.6 persons per 1000.

The expansion of the prevention of mother-to-child transmission of HIV (PMTCT) programme has resulted in a decrease in infant HIV infection rates. PMTCT services are available to 93% of HIV-positive pregnant women according to programme data and in 2021, 98% of women who tested positive for HIV were on ART.

13.6 ELIMINATION OF MOTHER TO CHILD HIV TRANSMISSION - EMTCT

Human immunodeficiency virus (HIV) can be transmitted from a mother to her child. The risk during pregnancy, delivery and breastfeeding is a high as 25-30% in the absence of treatment. The risk can be reduced to less than 2% through various interventions
Triple EMTCT-Elimination of Mother-To-Child Transmission of HIV, Syphilis and Hepatitis B Virus.

Modes of Transmission of HIV in Children:

- 95% of Pediatric HIV infections are due to Mother-To-Child Transmission (MTCT)
- 5% are due to:
  - sexual transmission (adolescents/abuse)
  - unsterile injection procedure
  - scarification and other traditional practices

Risk of Transmission of HIV without PMTCT interventions

Overall, an HIV exposed infant has 30-40% risk of being infected:

- During pregnancy: 5%
- During labor and delivery: 10-20%
- Breast feeding: 10-20%

The Four (4) Pillars /prongs of the Elimination of Mother to Child transmission of HIV (EMTCT) are:

1. Primary prevention of HIV among women of child bearing age

IEC- on STIs and HIV, provision of condoms, PrEP for Pregnant and Breast-feeding Wo men, ART for positive partners.

2. Prevention of unintended pregnancies among women living with HIV. Counselling and provision of Family planning services.
3. Prevention of HIV transmission from a woman living with HIV to her infant. Testing and ART initiation in all HIV positive pregnant and breastfeeding mothers.
4. Provision of appropriate treatment, care and support to women and children living with HIV and their families

VL monitoring should be done every 3 months in Pregnant and Breastfeeding women.
All HIV positive pregnant women should have viral load testing within 4 weeks before delivery:

Laboratory Diagnosis of Pediatric HIV Infection

Early Infant Diagnosis using Nucleic Acid Test (NAT)

HIV infection is confirmed by positive NAT test in children ≤ 24 months

a) If NAT 1 is positive, initiate ART and collect NAT 2 to verify the HIV positive status
b) If NAT 2 is positive, continue with lifelong ART and no need for an extra NAT
c) If 2nd NAT is negative, we should continue with ART and collect the 3rd NAT. If the 3rd NAT is positive, the child is HIV positive and should continue with lifelong ART (no need of any extra HIV test)

If 3rd NAT is negative, this is considered as HIV discordant result, we should stop ART. However, we should continue close monitoring of this child for EID at 4 weeks, 4 months, and 8 months after ART interruption with NAT and viral load test.

Serological or Antibody tests- is confirmatory for children aged >24 months

Determine HIV 1/2 test has been used as a screening test and SD Bioline HIV-1/2 3.0 test as a confirmatory test for routine HIV antibody tests.

Know Your Child's HIV Status+ (KYCHS+)

A strategy to ensure all biological children of HIV positive women who are <19 yrs and non-biological children <19 yrs old staying in the same household are fully indexed together

Table 13.4. Timing of HIV testing services for specific populations

Specific populations	Whom to test	When to test	Type of HIV testing
Pregnant women, breast- feeding women (and their sexual partners)	All	During antenatal care (ANC): at first ANC visit and retest every 3 months if negative In labour and delivery (L&D): test if last test > 6 weeks ago During postnatal care (PNC): test at first contact if unknown status. Serological test at 6 weeks if negative. If breast-feeding: retest every 3 months if negative until cessation of breast-feeding Partner testing: same time points	Serological test
(0 to < 10 years old)	Well, never-breastfed HIV Exposed Infant (HEI)	At birth/first week of life or at first contact	NAT*
		6 weeks old	
		24 months old	Serological test
	Well, breastfed HEI	At birth/first week of life or at first contact	NAT*
		6 weeks old	
		6 months old	
		9 months old	NAT
		12 months old	Serological test, if positive, follow up with NAT. If negative, follow up with serological test at 18 months
		18 months old	Serological test; if positive, follow up with NAT. If negative, follow up with serological test at 24 months
		24 months old	Serological test; if positive, follow up with NAT
	Infant or child who has completely stopped breast-feeding	≥ 6 weeks after breast-feeding cessation in children < 24months old	Serological test; if positive, follow up with NAT
		> 24 months old	Serological test

	Asymptomatic infant with unknown HIV exposure	At first contact	Maternal serological test and/or infant serological test; follow up with NAT for positive serological child ≤ 24 months old
	Infant or child symptomatic for HIV infection	Immediately regardless of age	Serological test; follow up with NAT for positive serological child ≤24 months old
	Positive serological child < 24 months old	At first contact	NAT
	All infants and children with unknown HIV status admitted for inpatient care, attending malnutrition clinic, outpatient care or immunization clinics	Administer Paediatric Screening Tool and test appropriately	Age-appropriate tests
Adolescents (10 – 19 years) and adults	All sexually active persons with their partners and any person of unknown HIV status	Administer the HIV Screening Tool at first contact, if negative repeat test at 3 months and appropriate intervals depending on risk assessment	Serological test

* **Where there is no Point of Care (POC) NAT, a DBS should be sent for HIV DNA PCR.** Where NAT is positive, a repeat test should be done to rule out false-positive results. ART should be initiated without waiting for the receipt of the second test result because of the substantial risk of mortality with in- utero infection.

Management of an HIV-Exposed Infant (HEI) and Extended Prophylaxis

- All HEI should receive prophylaxis for at least 12 weeks with AZT + 3TC plus NVP to be stopped when there is a documented suppressed Viral Load in the mother at 3 months postnatal
- In a situation where the Viral Load of the mother is unsuppressed (or the mother not on ART). the prophylaxis should be extended until the mother is suppressed or one-week post-breast-feeding cessation
- Where the mother refuses to be on treatment, continued counselling should be done, and ART initiated as soon as possible
- Never breast-fed babies are given prophylaxis only for 6 weeks

Table 13.5. Simplified Infant Prophylaxis Dosing

Weight Band	NVP 10mg/mL	AZT/3TC 60/30mg tablet dissolved in 6mL of water (10mg/5mg/mL)
Birth weight < 2kg	0.8mL OD	0.8mL BD
Birth weight 2 – 2.499kg	1mL OD	1mL BD
Birth weight > 2.5kg	1.5mL OD	1.5mL BD
6 weeks to < 6 months old	2mL OD	Use treatment dose based on infant's weight (Table 8)
6 months to 9 months old	3mL OD	Use treatment dose based on infant's weight (Table 8)
9 months old to 1 week after cessation of breast-feeding	4mL OD	Use treatment dose based on infant's weight (Table 8)

Table 13.6. Simplified Dosing Chart for AZT/3TC Prophylaxis

Weight Band	Strength of AZT/3TC Tablet	AZT/3TC Dosage for Prophylaxis
3 – 5.9kg	60/30mg	1 tablet BD
6 – 9.9kg	60/30mg	1.5 tablets BD
10 – 13.9kg	60/30mg	2 tablets BD
14 – 19.9kg	60/30mg	2.5 tablets BD

NOTE: AZT/3TC(60/30mg) is a dispersible tablet containing AZT = 60mg and 3TC = 30mg:

- Dissolve one dispersible tablet into 6mL of water; 1mL of the suspension will contain 10mg of AZT and 5mg of 3TC
- Take NOTE that the suspension made should be kept in a cool place! Daily reconstitution is recommended to assure stability of the suspension. Shake the suspension before use
  - Care givers should be educated by the pharmacists and clinicians on how to reconstitute the AZT/3TC dispersible tablets
  - Infants weighing < 2kg should receive 2mg/kg NVP once daily and 2mg/kg once daily calculated based on the AZT in the AZT/3TC
  - Babies on AZT based prophylaxis should be monitored for toxicity with FBC
  - For complicated cases e.g., severe AZT induced anemia, consult a medical officer with appropriate training or **call 7040**:

Disclosure

Disclosure is an ongoing process of

1. informing the adolescent that she or he is HIV infected and helping him or her understand what that means and
2. helping the Adolescents Living with HIV (ALHIV) to disclose her or his status to others. It is understood that this process may take place gradually over a period to ensure that the adolescent has an eventual full understanding of his/her diagnosis and feels empowered to take responsibility of his/her health.

Disclosure can be:

1. **Partial disclosure** (usually done at age 5-10) done incrementally in age/developmentally appropriate language
2. **Full disclosure** (usually done at 10-14) resulting in full knowledge of HIV status, transmission and treatment and integration into a peer/adolescent model.

The disclosure process can either be Care giver initiated, HealthCare Worker assisted, or Adolescent initiated

Benefits to disclosure: include prevention of onward transmission and support (psychosocial, financial, etc.), improved adherence and care giver relationships, and empowered adolescents.

Risks to disclosure: include violence from sexual partners, stigmatization, discrimination, abandonment, and or other legal consequences**.**

Antiretroviral Drugs

The main classes of antiretroviral drugs currently available in Zambia are:

1. Nucleoside analogue reverse transcriptase inhibitors (NRTIs):
   - Zidovudine – AZT
   - Lamivudine – 3TC
   - Emtricitabine – FTC
   - Abacavir – ABC
   - Tenofovir Disoproxil Fumerate – TDF
   - Tenofovir Alafenamide – TAF

2. Non-nucleoside reverse transcriptase inhibitors (NNRTIs)
   - Nevirapine – NVP (only recommended for infant prophylaxis and for treatment in 0-4weeks old babies)
   - Etravirine - ETR
   - Delavirdine – DLV
   - Rilpivirine – RPV
   - Doravirine – DOR
   - Dapivirine - DPV

3. Integrase strand transfer inhibitor (INSTI)
   - Dolutegravir – DTG
   - Cabotegravir-CAB
   - Elvitegravir -EVG
   - Bictegravir – BIC

4. Protease inhibitors (PIs).
   - Ritonavir RTV
   - Lopinavir/Ritonavir – LPV-r
   - Darunavir - DRV
   - Darunavir/Ritonavir

At least three fully active drugs, usually from two different classes, are the recommended minimum standard for all settings.
WHO currently recommends that first-line regimens should be based upon two nucleoside analogue reverse transcriptase inhibitors (NRTI) plus one integrase strand transfer inhibitor (INSTI) or a protease inhibitor (PI).

Antiretroviral Therapy for Infants and Children
ART should be initiated immediately or within seven days after adequate preparation of the child and family
Provide optimized, fixed-dose ART regimens

Preinitiation Laboratory monitoring
- Do second NAT (≤24 months) but start ART without waiting for the results
- FBC/DC
- Liver function test (ALT/AST)

- Renal function test (Creatinine/Urinalysis)
- CD4 count and or CD4%
- Baseline viral load
- Pregnancy test and HPV (Human Papilloma Virus) test or visual inspection with acetic acid (VIA) in sexually active adolescents
- RPR in adolescents
- Cholesterol and triglycerides (if starting on PI)
- Serum CrAg for adolescents with cd4 <200 cells/ mm3 or WCS 111 or IV
- Urine LAM for children 5yrs and adults with cd4 <200 cells / mm3 WCS 111 or IV

Combination Anti-Retroviral Therapy

New recommendations:

- Weight-based regimen- as children grow, their medication doses must be adjusted accordingly
- No NNRTIs except the first 4 weeks of life
- DTG based regimen for all children (≥ 4 weeks and ≥3kg) and adolescents

First-Line ART

Table 13.7. Preferred First-Line ART and Alternative Regimens by Specific Populations

Specific Populations	Description	Preferred 1st line ART	Alternative Regimen
Children (0-4 weeks)	All	AZT + 3TC + NVP	Get Expert Opinion or call 7040 for advice
Children (≥ 4weeks), adolescents and adults	3 – 24.9kg	ABC + 3TC + pDTG*	ABC + 3TC + LPV-r
	≥ 25kg	TAF + FTC + DTG	ABC + 3TC + DTG
	≥ 30kg	TAF + FTC + DTG**	TDF + XTC + DRV-r*** ABC + 3TC + DTG
		TDF + XTC + DTG	TDF + XTC + LPV-r
Children, adolescents, and adults Co-infected with TB	3 – 29.9kg	ABC + 3TC + DTG	ABC + 3TC + AZT ABC + 3TC + LPV-r
	≥ 30kg	TDF + XTC + DTG Increase the frequency of DTG to 50mg twice daily if on Rifampicin-based ATT (continue DTG bd for extra 2 weeks after ATT completion)	ABC + 3TC + DTG TDF + XTC + DRV-r***

* pDTG 10mg is available for children weighing 3 – 19.9kg. Children and adults weighing ≥ 20kg, DTG 50mg is available and should be given once daily
CurrentlyABC60/3TC30/DTG5mgFDC formulation is available
** For children and adolescents who have their weight ≥ 30kg, the preferred 1st line regimen should be TafED
*** DRV-r should be given to children aged ≥ 12 years and ≥ 40kg, otherwise consult Expert opinion or call 7040. Avoid if on Rifampicin-based ATT

Clinical Reviews

- Every 2 weeks for the first 4 weeks
- Then every 4 weeks until week 12

Thereafter:

- For children < 24 months – monthly
- For children ≥ 24 months – < 10 years every 3 months once stable on therapy
- For adolescents ≥10 years every 6 months once stable on therapy

Follow up Laboratory Monitoring

Includes conducting labs routinely and as needed

- Viral load
- CD4 + T cell count -those 5 yrs and above with CD4 count > 350 cells/mm3 at baseline and at 6 months of ART with Suppressed viral load should NOT have subsequent repeat CD4 count monitoring as long as the viral load remains suppressed
- Creatinine/ Urinalysis (TDF and TAF based)
- Cholesterol and triglycerides (PI based)
- RPR annually for adolescents
- Serum CrAg for adolescents with cd4 <200 cells/ mm3 or WCS 111 or IV
- Urine LAM for children 5yrs and adults with cd4 <200 cells/ mm3 or WCS 111 or IV

Viral Load Monitoring

- At 6 months
- 12 months
- Then every 12 months if suppressed.

Management of Treatment failure

There are 3 types of failure -

1. Virologic
2. Immunologic
3. Clinical

- Routine VL monitoring is done to diagnose failure early as virologic failure is the first one to develop
- Virologic failure is defined as persistent viraemia after 24 weeks (6 months) of ART in an adherent patient: i.e., a VL of >1000 copies/ml after at least 24 weeks ART in a treatment-adherent child. Enrol all those who are diagnosed with treatment failure, for Enhanced Adherence Counselling (EAC) .
- After EAC, those with VL <1,000cp/ml should continue on the same ARV drug regimen and
- if VL still remain >1,000cp/ml, do genotype resistance test.
- Recipients of Care failing First-Line treatment must be switched to the standard Second Line according to the guidelines without waiting for the genotype results.
- The Second-Line regimen can be modified once the genotype results are available

Note: Younger children may have very high viral loads at commencement of ART. Therefore, they may take longer to attain viral suppression, and viral loads may be detectable at 6-9

months after initiation. This does not necessarily mean treatment failure. The VL should be repeated after 3 months and if still persistently detectable after 12 months of good adherence and adequate dosing consider virologic failure

Enhanced Adherence Counselling (EAC)

Enhanced Adherence Counselling (EAC) is a structured counselling intervention conducted in unsuppressed recipients of care (ROCS) with VL ≥ 1000 cp/mL**.** Studies show that EAC leads to viral re-suppression in over **70% of unsuppressed ROCS**. At a minimum, **4 EAC sessions** should be conducted for a period of **at least 3 months**. The retest viral load should be done at the end of a successful EAC.

Table 13.8. Recommended Second-Line ART Regimens by Specific Populations

Specific populations	Description	1st line ART used	Preferred 2nd line ART	Alternative 2nd line ART
Children (0 – 4 weeks)	All	AZT + 3TC + NVP		
Children (≥ 4 weeks), Adolescents and Adults	3 – 24.9kg	ABC + 3TC + DTG	AZT + 3TC + LPV-r	Consult Expert Opinion or call 7040
		ABC + 3TC + LPV-r	AZT + 3TC + DTG	
	≥ 25kg	TAF + FTC + DTG[2]	AZT + 3TC + LPV-r	
	≥ 30kg	TDF + 3TC + DTG	AZT + 3TC + DRV-r[4]	AZT + 3TC + LPV-r TDF + XTC + DRV-r[3] AZT + 3TC + ATV-r
		TAF[2] + FTC + DTG		
		TDF + XTC + EFV	TDF + 3TC + DTG[1]	AZT + 3TC + DRV-r[3] AZT + 3TC + DTG AZT + 3TC + LPV-r TDF + XTC + DRV-r[3] AZT + 3TC + ATV-r
			TAF + FTC + DTG[2]	

1. Emerging evidence indicates the backbone of TDF + XTC can be maintained or recycled even if used in First-Line
2. Note that evidence not sufficient/conclusive for use of TAF in patients co-infected with TB on Rifampicin-based ATT, therefore avoid use in such patients
3. DRV-r 400/50mg should be used in those who are ≥ 12 years and ≥ 40kg, otherwise consult Expert opinion or call 7040. Avoid if on Rifampicin-based ATT

Table 13.9. Summary of preferred second-line art regimens for adults and adolescents with tb and hepatitis b co-infection

Specific Conditions	Description	1st line ART used	Preferred 2nd line ART	Alternative 2nd line ART
HIV and HBV Co-infection in Adolescents and Adults	All	TDF + XTC + EFV	TDF + 3TC + DTG	Consult expert opinion
		TDF + 3TC + DTG	AZT + 3TC + LPV-r + TDF*	
		TAF + FTC + DTG		
Children, Adolescents, and Adults Co-infected with TB	3 – 29.9kg	ABC + 3TC + DTG	AZT + 3TC + LPV-r (*+TDF if ≥ 30kg)	
	≥ 30kg	TDF + 3TC + DTG		

*TDF + XTC should always be part of the combination in HBV/HIV co-infections. Since stand-alone TDF is currently not available, use TDF + XTC in consultation with expert or call 7040

As a part of comprehensive care Cotrimoxazole (CTX) prophylaxis is given to all under five children staring from 6 weeks of age provided there is no contra indication and children aged 5yrs and above are given CTX with cd4 count <350cells/mm3 and WHO stage 3 and 4
TB Preventive Therapy (TPT) is given to all children ≥ 1 yr after ruling out active TB provided no contra indication, in less than 1 yr TPT is given only if there is history of TB contact.
TPT regimens include INH, Rifapentine with INH as 3HP and 1HP

Table 13.1.1. Dosage for isoniazid preventative therapy, co-trimoxazole prophylaxis, B6 and combination INH/CTX/VIT B6 drugs

Drug	Child tablet or oral suspension	Number of scoops or tablets by weight band					Adult tablet
		3to<6kg	6to<10 kg	10to<14 kg	14to<20kg	20to<25kg	≥25kg
Isoniazid (INH)	100mg	0.5	1	1.5	2	2.5	300mg (1tablet)
Co-trimoxazole (CTX)	Suspension 200/40mg per 5mL	2.5mL	5mL	5mL	10mL	10mL	–
	Tablet 100/20mg	1	2	2	4	4	–
	Tablet 400/80mg	NA*	1⁄2	1⁄2	1	1	400/80mg (2tablets)
	Tablet 800/160mg	NA	NA	NA	1⁄2	1⁄2	800/160mg (1tablet)
Pyridoxine (VitaminB6)	Tablet 25mg	1/2	1/2	1/2	1⁄2	1⁄2	25mg (1tablet)
INH/CTX/VitB6	Tablet 300/960/25mg	NA	NA	NA	1⁄2	1⁄2	300/960/25mg(1tablet)

13.7 ADVANCED HIV DISEASE (AHD)

Definition of Advanced HIV Disease

For adults and adolescents, and children older than five years, AHD is defined as CD4 count < 200 cells/mm^3 or WHO stage 3 or 4 event. All children younger than five years old with HIV are considered as having AHD.

Although children younger than five years are defined as having advanced disease at presentation, those who have been receiving antiretroviral therapy for more than one year and who are stable should not be considered to have advanced disease

Screening for Advanced HIV Disease

To reduce HIV associated mortality, it is recommended that the following categories of HIV+ patients are reflexively screened for advanced HIV disease using a CD4 count test or WHO Clinical Staging where the CD4 count test is not available. These include:

- Those newly diagnosed with HIV
- Those reinitiating in care following treatment interruption
- Those with a high viral load
- Those hospitalized with a serious illness

The Advanced HIV Disease Package of Care

All ROCS that screen positive for advanced HIV disease must be offered the advanced HIV disease package of care. For individuals with AHD, the Ministry of Health recommends a package of care that includes:

- Screening for opportunistic infections especially Tuberculosis and Cryptococcal infection
- Prophylactic services against Tuberculosis, Cryptococcal infection and severe bacterial infections
- Rapid initiation of ART (within 7 days)
- Adherence Counselling

Table 13.1.2. Package of care for people with ahd

	Intervention	CD4 Cell Count	Adults	Adolescents	Children
Screening & Diagnosis	Sputum Xpert MTB/RIF as the first test for TB diagnosis among symptomatic people	Any	Yes	Yes	Yes
	LF-LAM for TB diagnosis among people with symptoms and signs of TB	≤ 200 cells/mm^3 or at any CD4 count if seriously ill	Yes	Yes	Yes (limited data for children)
	Cryptococcal antigen screening	≤ 200 cells/mm^3	Yes	Yes	No
Prophylaxis and Pre-emptive	Co-trimoxazole prophylaxis	≤ 350 cells/mm^3 or clinical stage 3 or 4	Yes	Yes	Yes (regardless of CD4 for under 5)
	TB preventive treatment	Any	Yes	Yes	Yes

	Fluconazole pre-emptive therapy for cryptococcal antigen–positive people without evidence of meningitis	< 200 cells/mm^3	Yes	Yes	Not applicable (Screening not advised)
ART initiation	Rapid ART initiation	Any	Yes	Yes	Yes
	Defer initiation if clinical symptoms suggest TB or Cryptococcal meningitis	Any	Yes	Yes	Yes
Individualised Adherence Support	Tailored counselling to ensure optimal adherence to the AHD package, including home visits if feasible	< 200 cells/mm^3	Yes	Yes	Yes

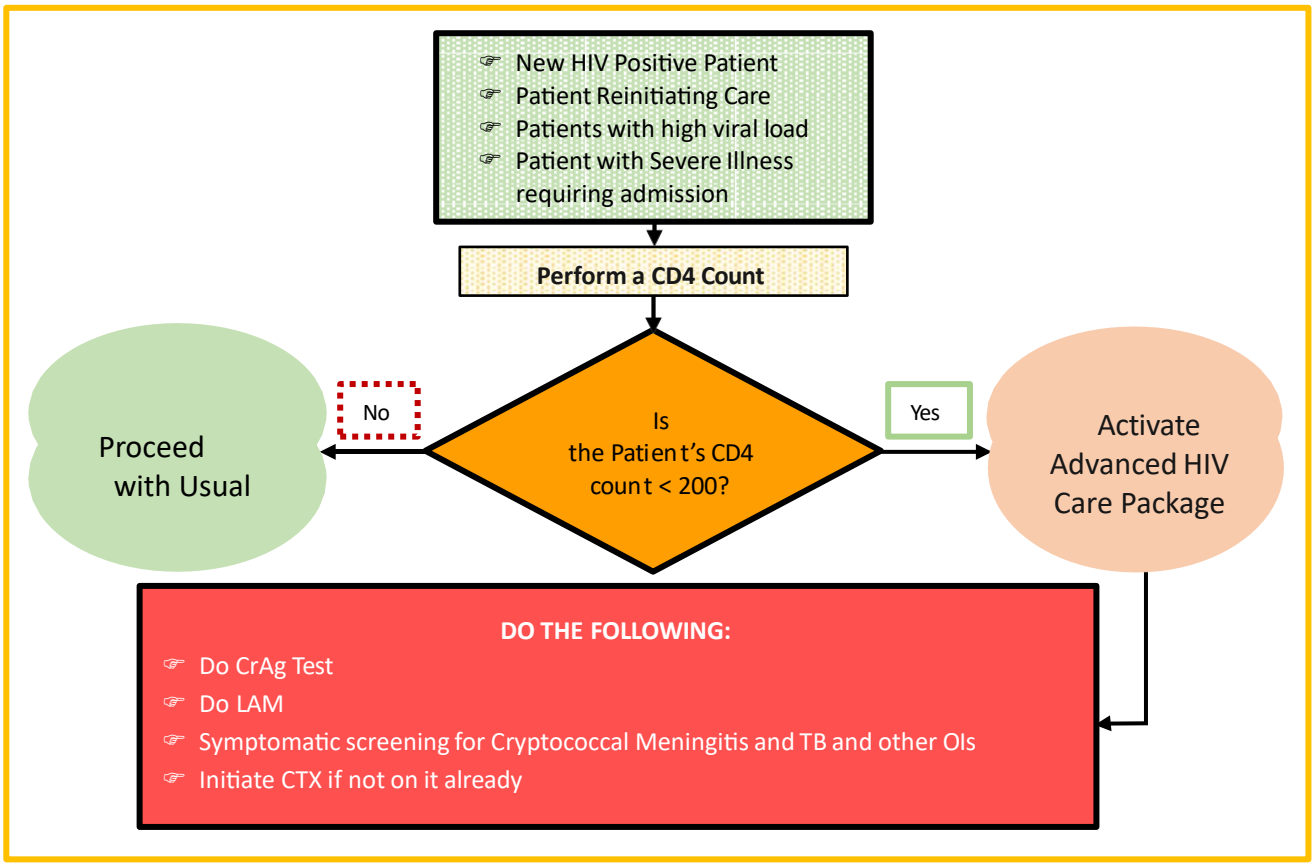


Fig 13. Advanced HIV Disease Package of Care

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2. Zambia Consolidated Guidelines for Treatment and Prevention of HIV Infection,2022
3. Peterson AT. HIV in Pregnancy. Medscape. Updated May 10, 2024. Accessed Nov 03, 2024.

13.8 CHILD SEXUAL ASSAULT (CSA)

Child Abuse: Abuse of children which includes mistreatment, exploitation, neglect and/or violation of a child's rights by any other person(s). It may be a single or repeated act(s), intentional or unintentional.
Child Sexual Abuse: Forcing or enticing a child to take part in sexual activities that he or she does not fully understand and has little choice in consenting to. This may include, but is not limited to, rape, oral sex, penetration, or non-penetrative acts such as masturbation, kissing, rubbing, and touching. It may also include involving children in looking at, or producing sexual images, watching sexual activities, and encouraging children to behave in sexually inappropriate ways.

One Stop Centre (OSC)

OSC is a model that brings multi-sectorial stakeholders together to provide all essential GBV services in one place. The team includes:

- Doctors and clinicians
  History, Examination, Investigations and Treatment
- Counsellors, Social Workers and Nurses
  Provide crisis counselling and psychosocial support
  Provide emergency shelter
- Police Officers and Paralegal
  Open dockets and/or take statements
  Provide legal services

Medical Approach

- History
- Examination
- Investigations
- Treatment

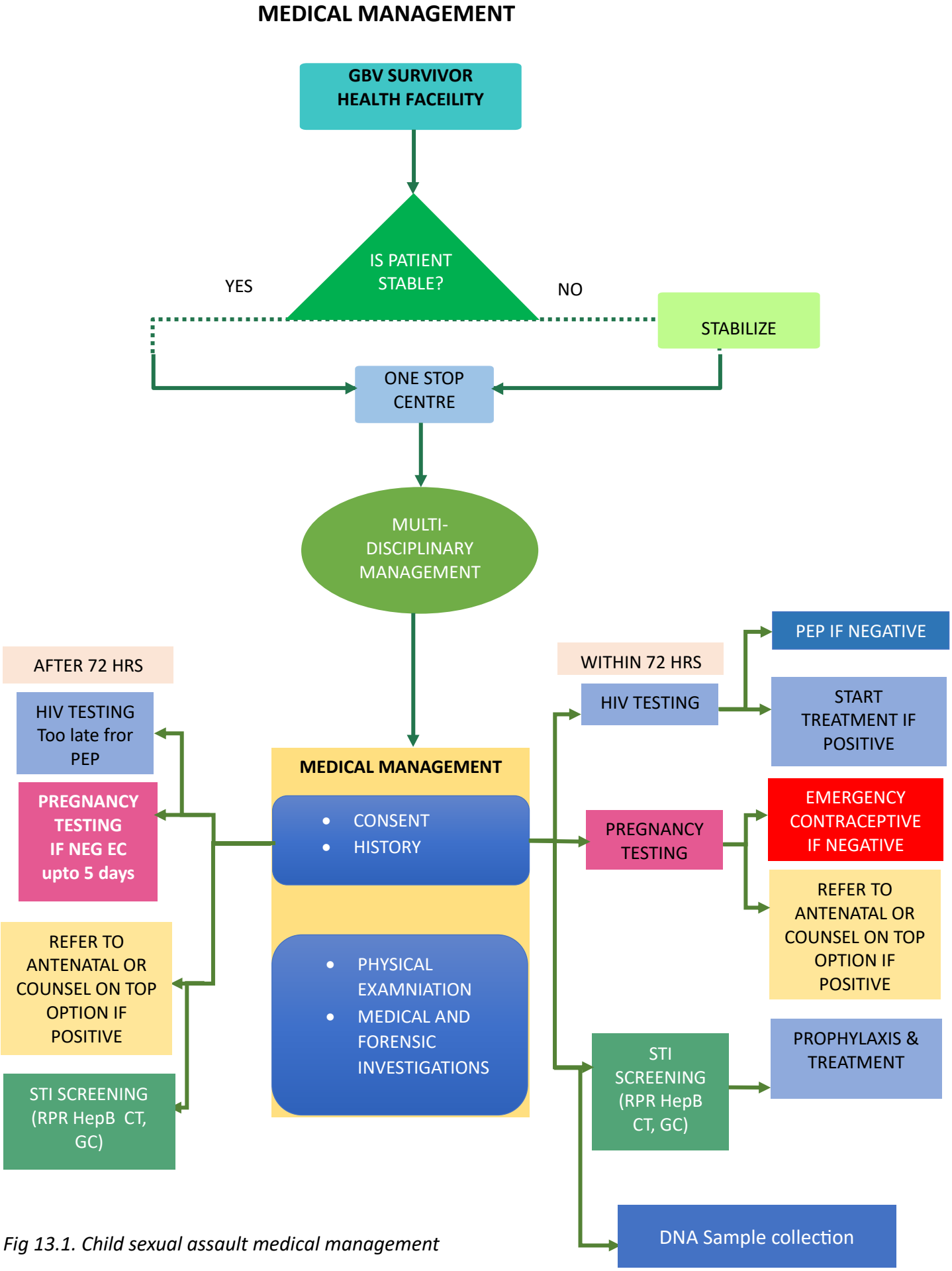


Fig 13.1. Child sexual assault medical management

History Taking (Ref to Appendix on MEDICAL FORM FOR GBV**)**

- What happened – Before – During - After?
- Dates (Day, Month, Year – approximate time)
- Where events took place – Address or general location
- How many people –any distinguishing features or dress – known people and how are they known – identification – names – witness(es) to events
- How the event happened – details, threats either physical or mental – was anything said – Any witness(es) to the events and how or where they can be contacted
- Why did the event happen – Did anyone say anything before the event – Were there any threats, either physical or mental – What was said or done after the event?
- Who was an eye witness or who can corroborate your interview/statement?
- What did you do after the incident occurred – who did you tell of the incident?
- What happened after?

Table 13.1.3. Examination (Important to know your Genital Anatomy)

Step	**Action to be taken**
Step 1	• General appearance and demeanour • Vital signs • Quick mental state
Step 2	• Examine the scalp for hematomas, hair loss and other injuries • Inspect the face, eyes, nose and mouth (petechiae on the hard/ soft palate may indicate penetration) • Inspect the ears using an otoscope to inspect the eardrum • Collect an oral swab, if indicated
Step 3	The neck area is of great forensic interest
Step 4	Inspect the forearms for defense injuries, bruising, abrasions, lacerations or incised wounds. The inner surfaces of the upper arms and the armpit or axilla need to be carefully observed for signs of bruising.
Step 5	Examine the breasts, respiratory and cardiovascular systems
Step 6	Abdomen: Inspect for bruising, abrasions, lacerations Palpate to exclude any internal trauma or to detect pregnancy.
Step 7	Lower extremities: commence with the front of the legs, inner thighs, ankles (and wrists), and feet Inspect each buttock.
Step 8	**Genital examination** • Examine Supine position and Knee-chest positions

	• For Anal examination Left lateral position may be used **The external genitalia** Labia majora and minora, clitoris, vestibule, posterior commissure/fourchette, fossa navicularis and perineum need to be assessed for signs of acute trauma. **The internal genitalia** The hymen and vagina canal need to be examined for signs of acute trauma as well as for any structural changes suggesting penetration. • *In children, it is not recommended to insert anything in the vagina eg fingers, speculum, swabs. If there is an imperative need for vaginal examination eg bleeding, an examination under anaesthesia (EUA) should be performed* **NOTE:** There is NO such thing as an "Absent Hymen"or "perforated Hymen" **Anal Examination:** Inspect and examine the anal area, look for any bruises or cuts. Note if anal spinchter is incompetent with stool incontinence M**ale Genitalia** Positions: standing or supine Inspect for scarification around the genitalia and gently palpate the penis and scrotum

Investigations

- Blood
  - HIV, RPR, HBV
  - Hb, (Grouping and X-match if needed)
  - Toxicology
- Radiological (If indicated): X-ray-Limbs, Cranial, Chest, Abdomen and Ultrasound, CT scan, MRI.
- Urine: Pregnancy test, Urinalysis, MCS, toxicology
- Swab: Anal, Oral, Vaginal/urethral (make a glass slide for spermatozoa as well)

Management

- Ongoing counselling
- PEP - Refer to the national guidelines
  - If the HIV test is Negative: offer HIV prophylaxis (PEP) if present within 72 hours of exposure for a total of 28 days.
  - If the HIV test is positive: post-test counselling, refer for treatment, care & support.

- Refer to national guidelines for Regimen
- Emergency Contraception
  - Emergency contraception is administered within 120 hours (5 days) of exposure.
- Manage sexually transmitted infections
- In women and girls STI's are often silent so offer STI prophylaxis
- Pain management and administration of tetanus toxoids where necessary
- Appropriate care and referral for other complications
- Ensure to complete the Zambia Police Medical Form ZP32B form (attached below)
- Schedule review at 2 weeks, 6 weeks and 3 months and on-going if needed.
- Linkage with social welfare for safe home custody or admission at the hospital.

Table 13.1.4. PEP Regimen (Otherwise refer to current HIV National Guidelines)

Weight Band	Number of tablets	Dose in mg	
3-5.9kg	AZT/1 TAB pDTG 0.5 TAB OD	AZT 60 mg BD 3TC 30mg pDTG 5mg OD	pDTG 10mg-1/2 TAB
6-9.9	AZT/3TC 1.5 TAB pDTG 1.5 TABS OD	AZT 90 mg BD 3TC 45 mg pDTG 15mg OD	pDTG 10mg 1 and half TABS
10-13KG	AZT/3TC 2TABS pDTG 2 TABLETS OD	AZT 120 mg BD 3TC 60 mg pDTG 20mg OD	pDTG 10mg 2 TABS
14-19kg	AZT /3TC 2.5 pDTG 2.5 TAB	AZT 150mg 3TC 75 BD pDTG 25mg OD	pDTG 10mg 2 and half TABS
20-24	AZT /3TC 1 TAB DTG 50mg 1 TAB	AZT 300mg BD 3TC 150mg DTG 50mg OD	Adult DTG 50mg TAB
25kg 30kg	TAF /3TC./DTG TDF/3TC/DTG	One Tab 25/300/50 One Tab 300/300/50	Fixed Dose Combinations

Table 13.1.5. STI (Refer to BNF for dosages)

Organism	Sensitivity pattern
Gonorrhoea	Ceftriaxone, Ciprofloxacin
Chlamydia	Erythromycin, Azithromycin (< 12yrs), Doxycycline (Over 12yrs and Non-pregnant client)
Syphilis	Benzathine Penicillin
Anaerobes	Metronidazole

Doxycycline should not be given to children < 12 years and during pregnancy

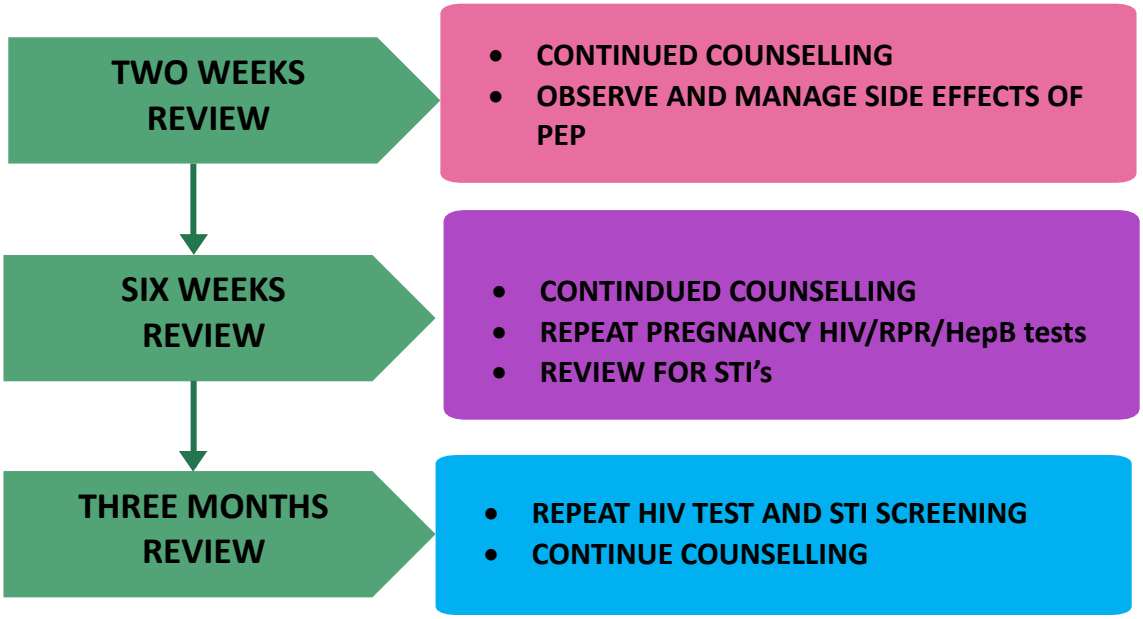
Table 13.9. Hepatitis B

Has she or he been vaccinated against Hepatitis B?	
No, never vaccinated	1st dose: at first visit 2nd dose: 1 – 2 months after the first dose 3rd does: 4 – 6 months after the first dose
Started but not yet completed a series	Complete the series as scheduled
Yes, completed series	No need to re-vaccinate

Emergency Contraception

- Levonorgestrel 1.5 mg PO STAT

FOLLOW UP SCHEDULE



Insert ZP32B Form

Stocked by QM Stores:

ZP Form 32

Police-Medical Report Form

A. Details of Complainant.				
Name (first, middle and last name):				
Address:				
Age:		Occupation:		Sex:
Marital Status:	Married/	Single/	Divorced/	Separated/ Other
Next of Kin:				
Name (first, middle and last name):	Relationship:	Address:		Phone #:
B. **Details of Allegations/Complaint:**				
C. **Details of Police Officer:**				
Name:	Rank:	Station:		
Service Number: Signature:				
Date:		Date Stamp		
D. **Details of Examiner:**				
Name:	Title/Designation:	Health Centre/Hospital		
	Signature:	Date Stamp		
Details of Person(s) Present during Exam. (Refers to parent, guardian, support person or any person the victim is comfortable with).				
Name:	Address:	Contact:		
Relationship to Victim:	Signature:	Date:		
Details of Examination: (*Please provide full details on the extent of the injuries, discharge, or deposits of semen* or any other findings).				
E. **General Examinations.**				

E. General Examinations:
Condition and Appearance:
Garments:
Body Injuries:
Identification Marks:
F. Ano-Genital Examinations:
Genital Examination:

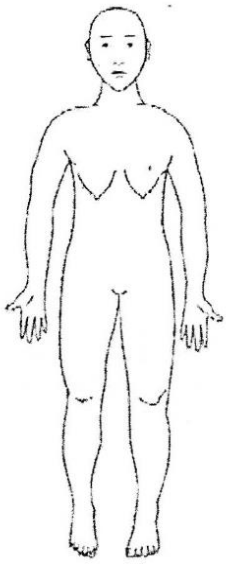
Anal examination:
G. Investigations:

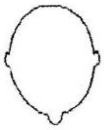
Swabs:	**Date Done**	**Not Done (Tick)**	**Results**	**Comments**
Oral				
Vaginal				
Urethral				
Rectal				
Other				
Tests:				
Pregnancy				
HIV				
Syphilis				
Other				

H. Indicate injuries on the sketches below:[1]

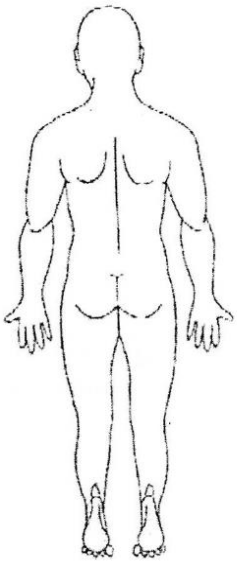
Sketch of person

Anterior View





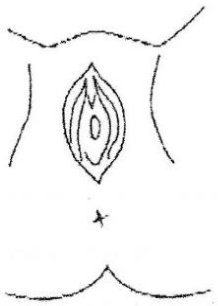
Posterior View

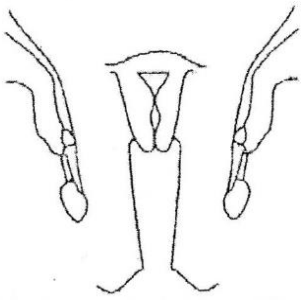


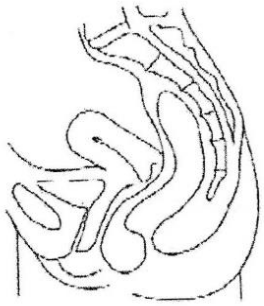
Comments



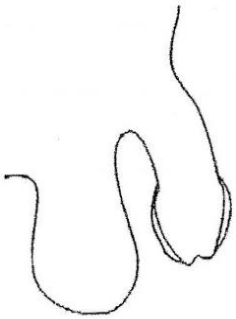
Female Genitalia

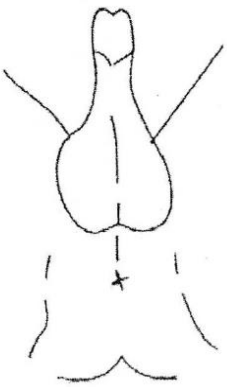


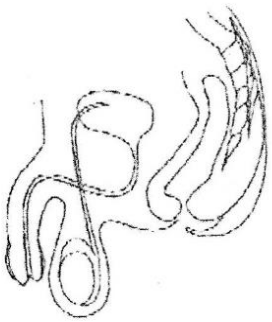




Male Genitalia







I. Findings (clearly Indicate the findings):
J. Opinion in relation to the allegation/complaint in Part (B) (after conclusive examinations, state whether they are consistent or not consistent with the allegations/complaint):
FOR USE BY THE EXAMINER

Name:	Official Date Stamp:
Title/Designation:	
Signature:	
Date:	

REFERENCES

1. Anti-Gender Based Violence Act No. 1 of 2011. Lusaka: Government of Zambia; 2011
2. Government of the Republic of Zambia (GRZ), United Nations (UN). Joint Programmes on Gender Based Violence. 2012.
3. McCann JJ, Kerns DL. Step by Step Guide for the Management of Sexually Abused Children. 2nd ed. Population Council; Department of Health, KwaZulu Natal; 2010
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11. Zambia Demographic Health Survey. 2014. CSO, Lusaka, Zambia.
12. Zambia Demographic Health Survey. 2018. ZAMSTATS, Lusaka, Zambia.

13.9 POST EXPOSURE PROPHYLAXIS - OCCUPATIONAL HIV EXPOSURE

Post exposure prophylaxis is the use of ART to prevent HIV transmission. In adult's exposure occurs commonly from occupational injuries.

The risk of infection after occupational exposure to HIV infected blood is low and estimated at:

- 1: 300 in percutaneous exposure
- 1: 1000 in mucocutaneous exposure

There is no risk of transmission if the skin is intact but there are factors that can increase the risk of infection e.g.

- Deep injury
- Visible blood on device that caused injury
- Injury with a large bore needle
- Non-suppressed viral load in source patient

Other body fluids that can lead to HIV transmission include: amniotic fluid, CSF, breast milk, pericardial, pleural, peritoneal fluids, synovial fluid, unfixed human tissue, vaginal secretions, semen, any other visibly blood-stained fluid, fluid from burns or skin lesions.

Management of occupational exposure

Immediately after exposure to infection:

- Clean site: wash skin injuries with soap and running water. Do not squeeze but allow wound to bleed freely.
- If the exposed area is an eye or mucous membrane, rinse with copious amounts of clean water.
- Establish HIV status of the patient and of the injured provider/victim – HIV testing
- Contact the person responsible for PEP or medical staff on duty.
- The person in charge of PEP should assess the need for PEP and the benefits of taking ART prophylaxis.

Table 13.1.6. PEP Treatment Regimen

Risk Category	ART prophylaxis	Duration
Skin intact	No PEP	
Medium risk - invasive injury, no visible blood on needle/device	Preferred: TDF +XFTC + DTG Alternative: TDF + XTC + ATV/r Children below 10 yrs: AZT +3TC+ LPV/r	28 days
High risk - large volume of blood/fluid, deep extensive injury, known HIV infected patient, large bore needle		

REFERENCES

1. Ministry of Health, Zambia. Zambia Consolidated Guidelines for Treatment and Prevention of HIV Infection. 2022.

14.1 ACR GUIDELINES- SEIZURES

Table 14. Neonatal seizures, age 0 to 29 days. Initial imaging

American College of Radiology
ACR Appropriateness Criteria®
Seizures-Child

Variant 1: **Neonatal seizures, age 0 to 29 days. Initial imaging.**

Procedure	Appropriateness Category	Relative Radiation Level
MRI head without IV contrast	Usually Appropriate	O
US head	May Be Appropriate	O
MRI head without and with IV contrast	May Be Appropriate	O
CT head without IV contrast	May Be Appropriate	☢☢☢
CT head with IV contrast	Usually Not Appropriate	☢☢☢
CT head without and with IV contrast	Usually Not Appropriate	☢☢☢☢
HMPAO SPECT or SPECT/CT brain	Usually Not Appropriate	☢☢☢☢
FDG-PET/CT brain	Usually Not Appropriate	☢☢☢☢

Variant 2: **Children 6 months to 5 years of age. Simple febrile seizures. Initial imaging.**

Procedure	Appropriateness Category	Relative Radiation Level
US head	Usually Not Appropriate	O
MRI head without and with IV contrast	Usually Not Appropriate	O
MRI head without IV contrast	Usually Not Appropriate	O
CT head with IV contrast	Usually Not Appropriate	☢☢☢
CT head without and with IV contrast	Usually Not Appropriate	☢☢☢☢
CT head without IV contrast	Usually Not Appropriate	☢☢☢
HMPAO SPECT or SPECT/CT brain	Usually Not Appropriate	☢☢☢☢
FDG-PET/CT brain	Usually Not Appropriate	☢☢☢☢

Table 14.1. Appropriateness Category Names and Definitions

Appropriateness Category Names and Definitions

Appropriateness Category Name	Appropriateness Rating	Appropriateness Category Definition
Usually Appropriate	7, 8, or 9	The imaging procedure or treatment is indicated in the specified clinical scenarios at a favorable risk-benefit ratio for patients.
May Be Appropriate	4, 5, or 6	The imaging procedure or treatment may be indicated in the specified clinical scenarios as an alternative to imaging procedures or treatments with a more favorable risk-benefit ratio, or the risk-benefit ratio for patients is equivocal.
May Be Appropriate (Disagreement)	5	The individual ratings are too dispersed from the panel median. The different label provides transparency regarding the panel's recommendation. "May be appropriate" is the rating category and a rating of 5 is assigned.
Usually Not Appropriate	1, 2, or 3	The imaging procedure or treatment is unlikely to be indicated in the specified clinical scenarios, or the risk-benefit ratio for patients is likely to be unfavorable.

Table 15. Normal ranges for respiratory rate and heart rate

Normal Ranges for respiratory rate and heart rate				
Age	Guide weigh (KG)		RR at rest breaths per minute 5^{th} to 95^{th} Centile	Heart Rate Beats per Minute 5^{th} to 95^{th} Centile
	Boys	Girls		
Birth	3.5	3.5	25 - 50	120 - 170
1 month	4.5	4.5		
3 months	6.5	6	25 - 40	115 - 160
6 months	8	7	20 - 40	110 - 160
12 months	9.5	9		
18 months	11	10	20 - 35	100 – 155
2 years	12	12	20 - 30	100 – 150
3 years	14	14		90-140
4 years	16	16		80 - 135
5 years	18	18		
6 years	21	20		80 - 130
7 years	23	22		
8 years	25	25	15 - 25	70 - 120
9 years	28	28		
10 years	31	32		
11 years	35	35		
12 years	43	43	12 - 24	65 – 115
14 years	50	50		60 – 110
Adult	70	70		

Table 15.1. Blood pressure (bp) levels in girls for age and height percentile

Blood Pressure (BP) levels in Girls for age and Height Percentile							
Age (year)	BP Percentile	SBP (mmHg)			DBP (mmHg)		
		Height Percentile or CM			Height Percentile or cm		
	Height Percentile	5%	50%	95%	5%	50%	95%
1	Height (cm)	75.4	80.8	86.1	75.4	80.8	86.1
	50%	84	86	88	41	43	46
	90%	98	100	102	54	56	58
	95%	101	103	105	59	60	62
2	Height (cm)	84.9	91.1	97.4	84.9	91.1	97.4
	50%	87	89	91	45	48	51
	90%	101	103	106	58	60	62
	95%	104	106	109	62	64	66
3	Height (cm)	91	97.6	104.6	91	97.6	104.6
	50%	88	90	93	48	50	53
	90%	102	104	107	60	62	65
	95%	106	108	110	64	66	69
4	Height (cm)	97.2	104.5	112.2	97.2	104.5	112.2
	50%	89	92	94	50	53	55
	90%	103	106	108	62	65	67
	95%	107	109	112	66	69	71
5	Height (cm)	103.6	111.5	120	103.6	111.5	120
	50%	92	93	96	52	55	57
	90%	105	107	110	64	67	70
	95%	109	110	113	68	71	73
6	Height (cm)	110	118.4	127.7	110	118.4	127.7
	50%	92	94	97	54	56	59
	90%	105	108	111	67	69	71
	95%	109	111	114	70	72	74
7	Height (cm)	115.9	124.9	134.7	115.9	124.9	134.7
	50%	92	95	99	55	57	60
	90%	106	109	112	68	70	72
	95%	109	112	115	72	73	75
8	Height (cm)	121	130.6	140.9	121	130.6	140.9
	50%	93	97	100	56	59	61
	90%	107	110	113	69	72	73
	95%	110	113	117	72	74	73
9	Height (cm)	125.3	135.6	146.6	125.3	135.6	146.6
	50%	95	98	101	57	60	61
	90%	108	111	114	71	73	73
	95%	112	114	118	74	75	75

Blood Pressure (BP) levels in Girls for age and Height Percentile							
Age (year)	BP Percentile	SBP (mmHg)			DBP (mmHg)		
		Height Percentile or cm			Height Percentile or cm		
	Height Percentile	5%	50%	95%	5%	50%	95%
11	Height (cm)	135.6	147.8	160	135.6	147.8	160
	50%	98	102	106	60	61	64
	90%	111	114	120	74	74	75
	95%	115	118	124	76	77	77
12	Height (cm)	142.8	154.8	166.4	142.8	154.8	166.4
	50%	102	105	108	61	62	65
	90%	114	118	122	75	75	76
	95%	118	122	126	78	78	79
13	Height (cm)	148.1	159.2	170.2	148.1	159.2	170.2
	50%	104	107	109	62	64	66
	90%	116	121	123	75	76	76
	95%	121	124	127	79	79	81
These normative blood pressure figures were extracted from the guidelines “Clinical Practice Guideline for Screening and Management of High Blood Pressure in children and adolescents.” Flynn JT et al. Paediatrics. (2017)							
10	Height (cm)	129.7	141	152.8	129.7	141	152.8
	50%	96	99	103	58	60	62
	90%	109	112	116	72	73	73
	95%	113	116	120	75	76	76

Blood Pressure (BP) levels in Boys for age and Height Percentile							
Age (year)	BP Percentile	SBP (mmHg)			DBP (mmHg)		
		Height Percentile or CM			Height Percentile or cm		
	Height Percentile	5%	50%	95%	5%	50%	95%
1	Height (cm)	77.2	82.4	87.9	77.2	82.4	87.9
	50%	85	86	88	40	41	42
	90%	98	100	101	52	53	54
	95%	102	103	105	54	55	57
2	Height (cm)	86.1	92.1	98.5	86.1	92.1	98.5
	50%	87	89	91	43	44	46
	90%	100	102	104	55	56	58
	95%	104	106	108	57	59	61
3	Height (cm)	92.5	99	105.8	92.5	99	105.8
	50%	88	90	92	45	47	49
	90%	101	103	105	58	59	61
	95%	106	107	109	60	62	64
4	Height (cm)	98.5	105.9	113.2	98.5	105.9	113.2
	50%	90	92	94	48	50	52
	90%	102	105	107	60	62	64
	95%	107	108	110	63	66	68
5	Height (cm)	104.4	112.4	120.3	104.4	112.4	120.3
	50%	91	94	96	51	53	55
	90%	103	106	108	63	65	67
	95%	107	109	112	66	69	71
6	Height (cm)	110.3	118.9	127.5	110.3	118.9	127.5
	50%	93	95	98	54	56	58
	90%	105	107	110	66	68	69
	95%	108	111	114	69	71	73
7	Height (cm)	116.1	125.1	134.5	116.1	125.1	134.5
	50%	94	97	99	56	58	59
	90%	106	109	111	68	70	71
	95%	110	112	116	71	73	74
8	Height (cm)	121.4	131	141	121.4	131	141
	50%	95	98	100	57	59	60
	90%	107	110	112	69	71	73
	95%	111	114	117	72	74	75
9	Height (cm)	126	136.3	147.1	126	136.3	147.1
	50%	96	99	101	57	60	62
	90%	107	110	114	70	73	74

	95%	112	115	119	74	76	77
10	Height (cm)	130.2	141.3	152.7	130.2	141.3	152.7
	50%	97	100	103	59	62	63
	90%	108	112	116	72	74	76
	95%	112	116	121	76	77	78

Blood Pressure (BP) levels in Boys for age and Height Percentile							
Age (year)	BP Percentile	SBP (mmHg)			DBP (mmHg)		
		Height Percentile or CM			Height Percentile or cm		
	Height Percentile	5%	50%	95%	5%	50%	95%
11	Height (cm)	134.7	146.4	158.6	134.7	146.4	158.6
	50%	99	102	106	61	63	63
	90%	110	114	118	74	75	76
	95%	114	118	124	77	78	78
12	Height (cm)	140.3	152.7	165.5	140.3	152.7	165.5
	50%	101	104	109	61	62	63
	90%	113	117	122	75	75	76
	95%	116	121	128	78	78	79
13	Height (cm)	147	160.3	173/4	147	160.3	173.4
	50%	103	108	112	61	62	65
	90%	115	121	126	74	75	77
	95%	119	125	131	78	78	81
These normative blood pressure figures were extracted from the guidelines “Clinical Practice Guideline for Screening and Management of High Blood Pressure in children and adolescents.” Flynn JT et al. Paediatrics. (2017)							

Table 15.2. 5th centile blood pressures for age

Age	5th Centile Blood Pressure
< 1 year	65 – 75
1 – 2 years	70 – 75
2 – 5 years	70 – 80
5 – 12 years	80 – 90
> 12 years	90 – 105
The calculation for expected systolic pressure is: 65 + (2 x age in years) mmHg for 5th centile	
Reference: Advanced Paediatric Life Support: The Practical Approach to Emergencies, 6th Edition 2016	

Table 15.3. Anthropometric measurements by age

Age	Weight (kg)	Height (cm)	Head size (cm)
Birth	3.5	50	35
6 months	7	68	42
1 year	10	75	47
2 years	12	85	49
3 years	14	95	49.5
4 years	16	100	50
5 – 12 years		5 cm/year	0.33 cm/year

Points to Note

Weight

- In the first 7 - 10 days of life, a healthy baby can lose up to 10% of their birth weight.
- In the first 3 months of life, the rate of weight gain is 25 gm/day
- Babies regain their birth weight by the 2nd week, double this by 5 months of age, and
- triple the birth weight by 1 year of age
- Weight estimation for children (in Kg):

Infants: (Age in months X 0.5) + 4

Children 1 – 10 years: (Age in years + 4) X 2

Head circumference

• Rate of growth in preterm infants is 1 cm/week, but reduces with age.

Head growth follows that of term infants when chronological age reaches term

• Head circumference increases by 12 cm in the 1st year of life (6 cm in first 3

months, then 3 cm in second 3 months, and 3 cm in last 6 months).

REFERENCES

1. Advanced Paediatric Life Support: The Practical Approach Textbook,
5th Edition 2011
2. Nelson Textbook of Pediatrics, 18th Edition.

16.1 LABORATORY PROTOCOLS

Safety Practices

Safety practices in the Laboratory are very fundamental, here are a few essential practices to follow:

- Treat all microorganisms as potential pathogens
- No foods or drinks
- Wear PPE and proper lab attire
- Good Hygiene and disinfect regularly
- Label work space and bottles clearly
- Don't work alone
- Stay focused and aware of surroundings

Specimen Collection

Specimen collection is the process of acquiring tissue or body fluids for laboratory analyzing.

Competency of Sample collectors:

The all-health facilities are responsible for ensuring that the persons collecting specimens are competent.

Table 16. Types of Specimen Collection

Type	Sites	Transport, Storage and other considerations
Phlebotomy	Arterial, veins, capillary	Clearly indicate site of blood collection. **Do Not REFRIGERATE Blood Cultures**
Skin and soft tissue	Skin, Wounds, Surgical sites, Burns.	Indicate site of collection. **Do Not put in formalin container for surgical infections**
Aspiration/ Puncture	Body mass, Spine,	CSF sample should be sent the lab immediately after collection and should NOT be Refrigerated.
Swabs	Urethral discharge, rectal swab, genital lesions, Pelvic abscess, High Vaginal discharge, conjunctival swab, Ear swab	**Do Not Refrigerate.** Transport in transport media within an hour.

Body fluids	Urina, Saliva, sputum	Sterile leak proof container Preferably Mid- stream urine at noon Sputum should preferably in the morning. Catheter urine should be collected from a sterile catheter.
Stool	stool sample	Sterile, clean, leak proof container. Preferably freshly collected, take to the lab immediately if not store under refrigeration (2 – 8c)

Transporting Specimen:
specimen must never be transported directly by hand, specimens should be transported in plastic sealable biohazard bags, for multiple specimens or other potentially infectious materials transport container or cooler box must be used. Ensure it is labeled BIOHAZRD.

Requesting Examination:
All requests for the laboratory tests should be made on the approved examination forms or should at minimum contain the following.
The table below summarizes the Minimum information required when requesting a laboratory investigation

Patient data item	Request Form	Specimen Container
Surname	*	*
Forename (not initials)	*	*
Investigations required	*	
Date of birth or Age	*	*
File Number	*	
Gender	*	
Location (ward or Clinic)	*	
Report Destination	*	
Report Destination	*	
Requesting doctors name	*	
Clinical details	*	
Time of collection	*	

Specimen type and site	*	* Especially for Histopathology and Bacteriology
Date of collection	*	*

Time of collection is extremely important as it allows the laboratory to:

1. Assess the suitability of sample e.g. K+ leakage may interfere with or give wrong results.
2. Properly perform tests that are part of a timed series e.g., glucose, tolerance tests).
3. Audit specimen turnaround time.

Handling Rejected Specimen:

Rejected specimen and request forms are still received and registered, the reason of rejection is indicted in the system, the specimen will be disposed of by the laboratory.

Specimen Rejection Criteria:

In general, the following conditions will lead to rejection of specimen:

- Clotted specimens
- Hemolyzed specimens
- Insufficient specimens
- Over filled tube
- Wrong specimen container
- Unlabeled specimen container
- Soiled Specimen container
- Soiled request forms
- Mismatched request forms and specimens
- Inadequately filled in request forms

Table 16.1. Sample collection containers

Container Type	Tests	Precautions to take
Paediatric Blood Culture Bottle 	Blood Culture	Invert gently to mix. When a blood culture is ordered along any other blood work the blood culture MUST be drawn first.
EDTA/ Purple top 	FBC, ESR, PBS, BMA, Coagulation, Solubility, Hb Electrophoresis, Blood Grouping, Hormonal tests	Mix blood adequately immediately after sample collection to avoid clotting and check for clots before sending
Plain/ Red container 	All Serum tests and sometimes Chemistry tests. CSF samples	Mixing not required.
Sodium Citrate/ Blue top 	Coagulation studies	Mix gently 3-4 times. Check on tube for correct volume of blood required.
Sodium Citrate/ Blue top Sodium Fluoride and potassium oxalate /Grey top 	Plasma test and chemistry tests Blood Glucose	Mix adequately 8-10 times to avoid clotting Delayed transport may cause low sugar results.

Lithium Heparin/ Green top 	Biochemistry	Mix blood adequately immediately after sample collection to avoid clotting and check for clots before sending
SST/Yellow top 	Biochemistry	Check for clots before sending
Glass Slide	Malaria and BMA	Smear should not be too thick or too thin

In the case of point of care testing (POCT) all relevant principles of quality assurance, Patient care, Training, audit trail and specimen integrity apply. Should there be any doubt or need for training and advisory service please contact the laboratory.

Table 16.2. CSA – Laboratory

Test	Specimen Type	Container and Volume	Transport, Storage, Precautions
HIV, Syphilis, Hepatitis A, B and C, Herpes Simplex types 2 (HSV) Chlamydia, Gonorrhea	Blood	Red Top / Plain container 4mls – 6mls	Can store at 6 – 8 C failure to send to the lab Tested using rapid diagnostic Kit
Chlamydia Trachomatis, Neisseria Gonorrhea	Endocervical swab Urethral swab Rectal swab	Swab with Transport Media	Transport within 1 hour of collection Tested using rapid diagnostic Kit
	Urethral discharge Vaginal discharge	Swab with Transport Media Smear on slide directly	Do not refrigerate

	Genital Lesion • Suspected Chancroid	Swab with transport media	Do not refrigerate
	Genital lesion • Suspected syphilis	Serous exudates on clean glass slide or capillary tube or swab of lesion base directly on slide	Slide preparation is for dark field microscope examination Transport to the lab immediately at room temperature
Gravindex Test	Urine	Sterile leak proof container	Repeat test after 2 weeks
Microscopy	Virginal swab	Swab with media	Take immediately to the lab

SPECIMEN REQUIREMENTS BY LABORATORY UNIT

Table 16.3. Bacteriology Laboratory

Specimen type	Collection Container	Transport, Storage and precaution
Urinary Tract Infection		
Mid- stream Urine • Clean catch in small Children • Catheter urine • Urine bag for infants	Sterile leak proof container	Patient to wash skin around genital area before collection If unable to send to the lab immediately store at 2-8c Do Not refrigerate for more than 24 hours
Urine • Mycobacteria	2mls urine	First void urine Refrigerate during transportation in cooler box
Gastrointestinal Tract		
Stool	Clean leak proof container	Freshly collected specimen preferred Transport within 1 hour or store at 2-8c

Rectal / faecal swab	Visible portion of stool on swab in transport media	When transport delay is foreseen • Swab in transport media should be kept at room temp to within 24 hours • Notify the laboratory whenever there is a suspicion of unusual pathogens, outbreaks clostridium, food poisoning etc.
Stool for H. pylori antigen test	Sterile leak proof container	Transport within 24 hours
Anthrax	Stool, Blood, swab	
Blood		
Culture	Blood culture bottles	Collect 3 blood samples within 3 hours Do not refrigerate
Fluids and Aspirates		
Open abscess – aspirate or swab Closed abscess – needle and syringe	Sterile leak proof container Sterile plain vacutainers	Tissue or fluid is always better to a swab Do not refrigerate
Meningitis		
Cerebral Spinal Fluid	Sterile leak proof container Sterile plain vacutainers	Send to the lab immediately Do not refrigerate
Reparatory Tract		
Nasopharyngeal swab	Flexible wire calcium alginate tipped swab Transport media swab	
Sinus aspirate	Anaerobic broth	Transport to lab immediately
Throat swab	Transport in media	Notify lab if suspected Diphtheria pertussis or gonococcal pharyngitis.
Sputum for culture	Sterile leak proof container 2-5 ml	Collect fresh sample resulting from deep cough No saliva or postnasal discharge If delayed more than 24 hours store at 2-8c

Lung abscess	Anerobic broth Add second specimen in sterile plain container for staining	Transport to lab immediately
Skin and soft tissue		
Wound specimens	Swab of purulent drainage placed in media	Transport to lab immediately If delayed more than 24 hours store at 2-8c Indicate site of collection
Surgical site infections • Optimum specimen, tissue/ biopsy/ aspirate	Sterile leak proof container	Keep tissue moist Aerobic transport Transport at room temp Indicate site of collection Do Not Put in Formalin
Skin and Subcutaneous tissue Dermatophytes Fungal skin lesions	Sterile leak proof container	Collect skin scrapings/ hair follicles/ nail scrapings Transport within 4 hours
EYE		
Coneal lesion scraping	Inoculated directly onto appropriate media Apply directly to 3 clean slides for staining and microscopy	Transport to lab immediately Place second sample into anaerobic broth (at bedside)
Conjunctival swab	Transport media	Transport to lab within 2 hours
Vitreous aspirate or biopsy	Inoculate plates and make smear slides	Transport to lab within 2 hours
EAR		
Fluid Swab from middle ear	Sterile leak proof container	Transport to lab immediately
Specimen should be collected prior to administration of antibiotics Don't contaminate sample with Normal Flora		

Table 16.4. Clinical Chemistry Laboratory

TEST	Specimen type	Transport/ Storage/ Precautions
Blood glucose • FBS • GTT • RBS	Plasma or serum	Delayed transport may cause low sugar results
Urine Glucose • RBS	Urine	Delayed transport may cause low sugar results
• Cholesterol • HDL • LDL • Trig	Heparinized plasma or serum	FASTING Specimen Trig is over estimated with random specimens
Lactate	Serum	Do not use SERUM (red top)
HbA1c	Whole blood	
Lithium	Serum	ACE inhibitors and NSAIDS should be stopped 48 hours before sample collection
Total protein Albumin Urea and electrolytes Creatinine TBIL and DBIL Liver enzymes • AST • ALT • ALP • GGT • LDH HBDH ADA Amylase Uric acid CK-NAC PO4 Lipase	Heparinized plasma or serum	Hemolysis affects results Protect from heat / light (may degrade bilirubin)

CSF biochemistry	CSF	Deliver to the laboratory immediately after collection
C3 C4 IgA IgG	Whole blood, serum, heparinized plasma	Deliver to the lab immediately
Total Iron	Heparinized plasma and serum	Iron therapy overestimates iron
Urine Biochemistry		
Urine protein Urine Urea, and electrolytes Uric acid Urine creatinine	24-hour urine Sterile leak proof container	Calcium is stable in urine for 4 days if stored at 2-8c and 2 days when stored at 15- 25c.
Dynamic function tests such as glucose tolerance test (GTT) can be set up on request. Kindly arrange with lab at least 12- 24 hours prior to undertaking those tests.		

Table 16.5. Hematology

Test	Specimen	Storage/ Transport / Precautions
FBC/DIFF ESR Solubility test HB electrophoresis Peripheral blood smear	Whole Blood	Transport at room temp within 4 hours of collection. All of these tests can be performed in one container as long as blood volume is more than 6mls
Bone Marrow	Bone marrow aspirate	Aspirate must not exceed 0.5ml Poorly made slides will be rejected
CSF/ fluid body cytology	CSF, pleural fluids, ascetic fluids	Transport within room temp within 1 hour
Coagulation Test		
PT/INR		
APTT Thrombin Time Factor VIII Factor IX	Whole Blood	Please check on the tube for correct volume blood required. Over filled under filled will be rejected Do Not refrigerate

FIBRINOGEN D-Dimer		If patient is on anticoagulants, indicate on request form Inadequate mixing of blood can trigger a degree of coagulation activation and result in erroneous lab results
Please avoid requesting ESR test unless it is absolutely necessary because this causes undue delays in laboratory process and releasing of results		

Table 16.6. Tuberculosis

Test	Specimen	Container/ Volume	Storage/ Transportation/Precaution
GeneXpert/ RIF	sputum	Screw capped sterile leak proof 2-5ml	Morning specimen
Line probe assay (LPA)	Sputum	Screw capped sterile leak proof 2-5ml	For diagnosis Patient must submit 2 specimens collected at spot-morning For follow-up a patient must submit 2 specimens collected as spot-morning Specimens should be packed separate from the request form to prevent soiling Triple packing must be used for specimen being transported
Microscopy Culture DST	Sputum	Screw capped sterile leak proof 2-5ml	
	Lymph node aspirate	Smeared slide	
	Cerebral spinal fluid, Pleural effusion, synovial fluid	Plain vacutainer bottle 2-5ml	
	Gastric lavage urine, bronchial lavage		
	Tissue Biopsy		
	Pus	Swab smeared	
TB LAM	Urine	sterile leak proof 2-5ml	
If there is a delay in delivering to the lab specimen must be refrigerated at 2-8c for NOT more than 5 days. **DO NOT FREEZE**			

Table 16.7. Virology

TEST	Container/ Specimen Volume	Transport / Precaution
HIV rapid plasma Rheumatoid Factor Hepatitis A, B and C	Whole Blood	Sample should be transported at room temp
CD-4 lymphocyte counts	Whole Blood	Sample older than 24 hours will be REJECTED NOT REFRIGERATED
HIV Viral load test	Whole Blood	At room temp within 24 hours If not separate plasma by centrifuging and freeze and transported to the lab frozen

Special Test		
Measles IgM Rubella IgM Yellow fever IgM	Blood	Samples must be stored and transported at 2-8 c
Polio Virus isolation from acute flaccid paralysis (ATP)cases	Stool	Two samples collected at least 24 hours apart
Rota virus antigen assay	Stool	Collect within 2 days of admission and 7 days of onset of diarrhoea
Influenzas	Oropharyngeal and nasopharyngeal swab	Transport directly to the lab at 2 – 25 c immediately
SARs – COV-2 PCR		

Table 16.8. Sample collection and storage conditions

Type	Sites	Transport, Storage and other considerations
Phlebotomy	Arterial, veins, capillarity	Clearly indicate site of blood collection. **Do Not REFRIGERATE Blood Cultures**
Skin and soft tissue	Skin, Wounds, Surgical sites, Burns.	Indicate site of collection. **Do Not put in formalin container for surgical infections**
Aspiration/ Puncture	Body mass, Spine,	CSF sample should be sent the lab immediately after collection and should NOT be Refrigerated.
Swabs	Urethral discharge, rectal swab, genital lesions, Pelvic abscess, High Vaginal discharge, conjunctival swab, Ear swab	**Do Not Refrigerate.** Transport in transport media within an hour.
Body fluids	Urina, Saliva, sputum	Sterile leak proof container Preferably Mid- stream urine at noon Sputum should preferably in the morning. Catheter urine should be collected from a sterile catheter.
Stool	**stool sample**	**Sterile, clean, leak proof container.** **Preferably freshly collected, take to the lab immediately if not store under refrigeration (2 – 8c)**

Specimen type	Collection Container	Transport, Storage and precaution
Urinary Tract Infection		
Mid- stream Urine • Clean catch in small Children • Catheter urine • Urine bag for infants	Sterile leak proof container	Patient to wash skin around genital area before collection If unable to send to the lab immediately store at 2-8c Do Not refrigerate for more than 24 hours
Urine • Mycobacteria	2mls urine	First void urine Refrigerate during transportation in cooler box
Gastrointestinal Tract		
Stool	Clean leak proof container	Freshly collected specimen preferred Transport within 1 hour or store at 2-8c
Rectal / faecal swab	Visible portion of stool on swab in transport media	When transport delay is foreseen • Swab in transport media should be kept at room temp to within 24 hours • Notify the laboratory whenever there is a suspicion of unusual pathogens, outbreaks clostridium, food poisoning etc.
Stool for H. pylori antigen test	Sterile leak proof container	Transport within 24 hours
Anthrax	Stool, Blood, swab	
Blood		
Culture	Blood culture bottles	Collect 3 blood samples within 3 hours Do not refrigerate
Fluids and Aspirates		
Open abscess – aspirate or swab Closed abscess – needle and syringe	Sterile leak proof container Sterile plain vacutainers	Tissue or fluid is always better to a swab Do not refrigerate

Meningitis		
Cerebral Spinal Fluid	Sterile leak proof container Sterile plain vacutainers	Send to the lab immediately Do not refrigerate
Reparatory Tract		
Nasopharyngeal swab	Flexible wire calcium alginate tipped swab Transport media swab	
Sinus aspirate	Anaerobic broth	Transport to lab immediately
Throat swab	Transport in media	Notify lab if suspected Diphtheria pertussis or gonococcal pharyngitis.
Sputum for culture	Sterile leak proof container 2-5 ml	Collect fresh sample resulting from deep cough No saliva or postnasal discharge If delayed more than 24 hours store at 2-8c
Lung abscess	Anerobic broth Add second specimen in sterile plain container for staining	Transport to lab immediately
Skin and soft tissue		
Wound specimens	Swab of purulent drainage placed in media	Transport to lab immediately If delayed more than 24 hours store at 2-8c Indicate site of collection
Surgical site infections • Optimum specimen, tissue/ biopsy/ aspirate	Sterile leak proof container	Keep tissue moist Aerobic transport Transport at room temp Indicate site of collection Do Not Put in Formalin
Skin and Subcutaneous tissue Dermatophytes Fungal skin lesions	Sterile leak proof container	Collect skin scrapings/ hair follicles/ nail scrapings Transport within 4 hours

EYE		
Coneal lesion scraping	Inoculated directly onto appropriate media Apply directly to 3 clean slides for staining and microscopy	Transport to lab immediately Place second sample into anaerobic broth (at bedside)
Conjunctival swab	Transport media	Transport to lab within 2 hours
Vitreous aspirate or biopsy	Inoculate plates and make smear slides	Transport to lab within 2 hours
EAR		
Fluid Swab from middle ear	Sterile leak proof container	Transport to lab immediately
Specimen should be collected prior to administration of antibiotics Don't contaminate sample with Normal Flora		

17.1 PHARMACY LIST DRUGS

DRUG NAME	INDICATIONS	DOSE	SIDE EFFECTS
ADENOSINE 6MG/2ML IV	SVT	**NEONATES** INITIAL: 0.15mg/kg then increased in steps of 0.05-0.1mg/kg every 1-2mins to a max dose of 0.3mg/kg **CHILD** 1-11m - 0.15 - 0.5mg/kg (max) 1yr-11yrs - 0.1 - 0.5mg/kg (max. 12mg) *Increase by 0.05-0.1mg/kg every 1-2mins* >12yrs - initially 3mg then 6mg after 1-2 minutes if required followed by 12mg after 1-2 minutes if required	Flushing, dyspnea, chest pain, paresthesia.
ADRENALINE 1:1000 (1MG/ML) IM/IV	Anaphylaxis	**CHILD** **IM** <6m - 0.1mg to 0.15mg 6m – 5 yrs - 0.15mg 6 – 11yrs - 0.3mg 12 – 17 yrs - 0.5mg *Repeat dose after 5mins.*	Muscle necrosis, peripheral ischemia, angina, anxiety, arrhythmias, dizziness, hypertension, dyspnea
ADRENALINE 1MG/10ML 1:10,000 IV	Cardiac arrest Acute Hypotension Severe Croup	**NEONATES & CHILDREN** **Cardiac arrest:** 0.01mg/kg Max: 1mg/dose Every 3-5mins as required. ***SLOW IV INJECTION*** **Acute hypotension:** 0.0001- 0.0015mg/kg/min *CONTINUOUS INTRAVENOUS INFUSION* **Nebulization (croup)** 1m- 11yrs - 0.4mg/kg/dose for 1 dose. Max- 5mg, dose to be repeated after 30 minutes if necessary. *INHALATION OF NEBULISED SOLUTION*	Hemiplegia, muscle rigidity, Angina, anxiety, arrhythmias, dizziness, hypertension, Dyspnea

ALBUMIN 5% 50MG/ML IV (CONSULT PRODUCT LITERATURE DUE TO DIFFERENCES IN PRODUCTS)	Hypovolaemia Ascites with Hypoalbuminaemia.	0.5-1g/kg/dose or Consult product literature.	Fever, Flushing, Nausea, Shock, Urticaria.
ALBUMIN 25% 250MG/ML IV (CONSULT PRODUCT LITERATRUE DUE TO DIFFERENCES IN PRODUCTS)	Hemolytic diseases of the new born, Severe hypoalbuminemia, acute loss of plasma volume, Adjunct in hyperbilirubinemia by exchange transfusion in the new born	CONSULT PRODUCT LITERATURE DUE TO DIFFERENCES IN PRODUCTS	Fever, flushing, nausea, shock, urticaria

DRUG NAME	INDICATIONS	DOSE	SIDE EFFECTS
AMIODARONE 150MG/3ML IV	Supraventricular and Ventricular, arrhythmias, Ventricular fibrillation (VF), Pulseless VT	**ORAL** **Neonate – 11 Years** 5-10mg/kg 12hrly for 7-10days thereafter reduced to 5-10mg/kg OD (max 200mg per day). **Child** 12- 17 years 200mg TDS for 1 week, then 200mg BD for 1 week, then 200mg daily adjusted according to response **IV INFUSION** **Neonate** 5mg/kg every 12-24 hours (given over 30min) **Child** *INITIAL*: 5-10mg/kg over 20mins-2hrs. *THEN BY CONTINUOUS IV INFUSION*: 0.3-1.5mg/kg/hr **max**: 1.2g/day **VF and pulseless VT** 5mg/kg over 3mins; repeat ONCE if required. Max:300mg/dose	Arrhythmias, hepatic disorders, hyperthyroidism, respiratory disorders.
ATROPINE 1MG/5ML 1MG/10ML 600MCG/ML IV	Treatment of Bradycardia, Organophosphate poisoning, Adjunct during rapid sequence intubation (RSI)	**Bradycardia:** **Child** 0.02mg/kg (max 1.2mg per dose) Repeat doses may be necessary **Organophosphate poisoning:** **CHILD** 0.02mg/kg every 5-10 mins (max 2mg per dose) until atropinisation **Adjunct during RSI:** 0.02mg/kg	Anhidrosis anxiety, arrhythmias, decreased dysphagia, gastrointestinal disorders, hyperthermia, mydriasis, taste loss, thirst
CALCIUM GLUCONATE 1G/10ML = 10% 2.2 MMOL/10ML IV	Hypocalcemia, Hyperkalemia, Magnesium toxicity	**Acute Hypocalcemia (correction) / Hyperkalemia:** Slow intravenous injection **Neonates** 0.5mL/kg of 10% solution IV over 5-10 minutes. **Child**	**Common:** Local irritation, mild hypotension. **Serious:** Hypercalcemia, arrhythmias, tissue necrosis if extravasation occurs.

		0.5mL/kg of 10% solution IV over 5-10 minutes, maximum 20 mL/dose. **Acute hypocalcemia** **(maintenance)** **Neonate** 2.3 ml/kg to be given over 24hrs. **Child** 1 month - 1 yr: 4.5ml/kg to be given over 24hrs 2 - 17 yrs: 40ml dose given over 24 hrs. Revert to oral route ASAP **Mild asymptomatic** **Hypocalcemia** **Neonate – 4 years:** 0.25mmol/kg QID **Child** 5 – 11 yrs: 0.2mmol/kg QID 12 – 17 yrs: 10mmol QID **Magnesium toxicity**: **Child** 0.5 mL/kg of 10% solution IV over 5-10 minutes, maximum 20 mL/dose.	
DEXAMETHASONE 4MG/ML IV	Cerebral oedema, Severe croup, Anti-inflammatory and immunosuppressant, Adrenal insufficiency	SUPPRESSION OF INFLAMMATORY AND ALLERGIC DISORDERS ORAL Child: 0.01-0.1mg/kg daily in 1–2 divided doses (0.3mg/kg daily may be required in emergency situations) **IM, slow IV INJ or IV infusion** Child 1 month–11 years: 0.083–0.333mg/kg daily in 1–2 divided doses. (Max 20 mg per day)	**Common:** Weight gain, mood swings, hyperglycemia. **Serious:** Immunosuppression, adrenal suppression, potential for growth retardation with long-term use.

		12–17 years: Initially 0.4–20 mg daily **Cerebral oedema** **IV inj** Child (body-weight up to 35 kg): Initially 16.6 mg, then 3.3 mg every 3 hours for 3 days, repeat dose QID for 1 day, then 1.7 mg QID for 4 days, begin to taper down in steps of 0.83 mg daily and discontinue after 7–10 days or change to oral dexamethasone maintenance if required Child (body-weight 35 kg and above): Initially 20.8 mg, then 3.3 mg every 2 hours for 3 days, repeat dose every 4 hours for 1 day, then QID for 4 days, begin to taper down in steps of 1.7 mg daily and discontinue after 7–10 days or change to oral dexamethasone. Maintenance if required **Adjunctive treatment of suspected bacterial meningitis (starting before or with first dose of antibacterial)** **IV Injection** Child 3m–15 yrs: 0.15 mg/kg QID for 4 days (max. per dose 10 mg) Child 16–17 years: 10 mg QID continued for 4 days in patients with pneumococcal meningitis. *Discontinue treatment if another cause of meningitis is suspected or confirmed* Mild croup and Severe croup **ORAL** **Child** 0.15mg/kg STAT	

DIAZEPAM 10MG/2ML IV	Muscle spasms Status epilepticus, Febrile convulsions, convulsions due to poisoning Life threatening acute drug – induced dystonic reactions.	**Muscle spasms:** **ORAL** **Child** 1 – 11m: 0.25mg/kg BD 1 – 4 yrs: 2.5mg BD 5 – 11 yrs: 5mg BD 12 – 17 yrs: 10mg BD (Max 40mg/day) **Status epilepticus, Febrile convulsions, convulsions due to poisoning** **IV** **Neonate – 11 yrs** 0.3 -0.4mg /kg (Max 10mg/dose) **Child** 12-17yrs: 10mg STAT *Repeat dose after 10 minutes if required (over 3 – 5 minutes)* **RECTAL** **Neonate:** 1.25-2.5mg **Child** 1 month – 1 year: 5mg STAT 2- 11 yrs: 5-10mg STAT 12- 17 yrs: 10 -20mg STAT *Repeat dose after 10 minutes if required* **Life threatening acute drug – induced dystonic reactions.** **IV** **CHILD** 1m-11yrs: 0.1mg/kg 12-17yrs: 5-10mg *Repeat dose if required (over 3 – 5 minutes)*	Appetite abnormal, concentration impaired, movement disorders muscle spasms. palpitations, sensory disorders. vomiting, Uncommon constipation diarrhea, hyper salivation, skin reactions, speech slurred

DRUG NAME	INDICATIONS	DOSE	SIDE EFFECTS
ESOMEPRAZOLE IV/PO	Gastro-oesophageal reflux disease (GERD- in the absence of oesophagitis).	**ORAL** 1yr-11yrs (10kg and above): 10mg OD x 8weeks 12yrs-17yrs: 20mg OD for up to 4 weeks (in the presence of oesophegitis dose may be increased to 40mg OD for 4 weeks, maintenance 20mg OD for 4 weeks) **IV** 1-11yrs: 10mg OD (body weight up to 20kg) 12-17yrs: 20mg OD (in the presence of oesophegitis dose may be increased to 40mg OD) *Until oral administration possible* Infusion given over at least 3 minutes	Hypomagnesaemia, abdominal pain, dizziness, constipation, Electrolyte imbalance.
ENOXAPARIN SC	DVT prophylaxis and treatment.	**Prophylaxis** **SC Inj** Neonates - 1 month: 0.75 mg/kg BD Child 2 months – 17 years: 0.5mg /kg BD (max per. Dose 20mg) **Treatment** Neonate – 1month: 1.5 – 2mg/kg BD Child 2 months – 17 years: 1mg/kg BD	Hemorrhage, Injection site ulcer (after deep SC injection), Increased liver amino transferase, Anaphylaxis, Osteoporosis (long-term use)
FUROSEMIDE 10MG/ML IV push/IM/IV infusion	Oedema, renal failure, hepatic disease, hypertension, chronic congestive heart failure, Hyperkalaemia.	**Oedema in heart failure|Renal disease|hepatic disease|Pulmonary oedema** **ORAL** **Neonate**: 0.5–2 mg/kg every 12–24 hours Child 1 month–11 years: 0.5–2 mg/kg 2–3 times a day, *higher doses may be required in resistant oedema Max dose:12 mg/kg per day or 80mg per day*	Dizziness, headache, electrolyte imbalance, Hyponatremia, hypokalemia, ototoxicity, hypomagnesemia

		12–17 years: 20–40 mg daily; increased to 80–120 mg daily, in resistant oedema **SLOW INTRAVENOUS INJECTION** **Neonate**: 0.5–1 mg/kg every 12–2 4 hours **Child** 1 month–11 years: 0.5–1 mg/kg every 8 hours (max. per dose 40 mg) increased if necessary upto 2 mg/kg every 8 hours (max. per dose 40 mg) 12–17 years: 20–40 mg every 8 hours as required, higher doses may be required in resistant cases **BY CONTINUOUS INTRAVENOUS INFUSION** **Child**: 0.1–2 mg/kg/hour **Oedema in HF, Renal disease& hepatic disease, Pulmonary oedema post-surgery** **BY CONTINUOUS INTRAVENOUS INFUSION** **Child**: 0.1 mg/kg/hour, dose to be doubled 2hrly until urine output exceeds 1 mL/kg/hour **Oliguria due to acute or chronic renal insufficiency [GFR below 20 mL/minute]** **ORAL** **Child 12–17 years:** Initial: 250 mg daily, then Increase in steps of 250 mg every 4–6 hours (max dose 2g) if required **BY INTRAVENOUS INFUSION** **Child** 1m–11yrs: 2–5 mg/kg up to 4 times a day; max: 1g per day	

		12–17 years: Initially 250 mg given over 1 hr, then 500 mg over 2hrs if required; then 1g over 4hrs if required. These increased doses are given if satisfactory urine output is not obtained. effective dose of up to 1g given at a maximum rate of 4 mg/minute can be repeated every 24 hours	

COMMONLY USED ANTIBIOTICS

DRUG NAME	INDICATIONS	DOSE	SIDE EFFECTS
AMPICILLIN 500MG POWDER IV/PO	Susceptible infections e.g. Bronchitis, uncomplicated CAP, Enterococcal Endocarditis, listerial Meningitis, sinusitis, otitis media, UTIs, Salmonellosis	**ORAL ROUTE** **Neonates** 7 to 20 days: 30mg/kg TDS 21 to 28 days: 30mg/kg QID (max 125mg) **Child** 1 to 11m: 125mg QID 1yr-4yrs: 250mg QID (max: 30mg/kg QID) 5yrs-17yrs: 500mg QID (max: 1g per dose) **IV ROUTE** **Neonates** Up to 7days: 30-60mg/kg BD 7-20days: 30-60mg/kg TDS 21-28days: 30-60mg/kg QID **Child** 25-50mg/kg QID **IV ROUTE** 50mg/kg in Endocarditis and 100mg/kg in Listerial Meningitis (frequency aged based as above)	Black hairy tongue, Pseudomembranous colitis, interstitial nephritis, hemolytic anaemia, oral candidiasis.
ACICLOVIR IV/PO	Viral encephalitis, shingles, chicken pox, Herpes Simplex.	**HERPES SIMPLEX** **Suppression of Herpes (PO)** 12-17yrs: 400mg BD; can be increased to 400mg TDS. **Herpes simplex treatment (PO)**	With oral use; agitation, anemia, ataxia, angioedema, coma, confusion, drowsiness, dysarthria, dyspnea, hallucination, abdominal pain, diarrhea, dizziness,

		1-23months: 100mg 5times daily for 5 days (double dose in Immunocompromised patients) 2-17yrs: 200mg 5times daily for 5 days (double dose in immunocompromised patients) **IV INFUSION** **Neonate – 2 months** 20mg/kg TDS for 14 days (21 days if CNS involvement) **Child 3 months – 11 years** 250mg/m² TDS for 5 days **Child 12 - 17 years** 5mg/kg TDS for 5 days **VARICELLA ZOSTER (chickenpox) / HERPES ZOSTER (shingles) treatment (PO)** 1-23m: 200mg QID x 5days 2-5yrs: 400mg QID x 5days 6-11yrs: 800mg QID x 5days 12-17yrs: 800mg 5 times daily x 7days **IV INFUSION** **Neonate - 2m** 10-20mg/kg TDS x 7days **3m-11yrs** 250mg/m² TDS x 5days (double dose in immunocompromised) **12-17yrs** 5mg/kg TDS (double dose in immunocompromised) **ENCEPHALITIS (Herpes and Varicella zoster) treatment** **IV INFUSION** **Neonates - 2m** 10-20mg/kg TDS x 10-14days **3m-11yrs** 500mg/m² TDS x 10-14days **12-17yrs** 10mg/kg TDS x 10-14days	fatigue, fever, headache With intravenous use; Nausea, photosensitivity reaction, skin reaction, vomiting, anemia, leucopenia.

DRUG NAME	INDICATIONS	DOSE	SIDE EFFECTS
BENZYLPENICILLIN G SODIUM 5MU (3G) IM, Slow IV inj, Infusion	Throat infections, otitis media, Pneumonia, Cellulitis, Endocarditis, Meningitis, sepsis, Congenital Syphilis.	**Mild to moderate susceptible infections, Throat infections, Otitis media, Pneumonia, Cellulitis** **IM/ SLOW IV, IV INFUSION** **Neonate up to 7 days** 25 mg/kg BD increased if necessary, to 25 mg/kg every TDS **Neonate 7 days to 28 days** 25 mg/kg TDS increased if necessary, to 50 mg/kg TDS in severe infection **Child** 25 mg/kg QID increased if necessary, to 50 mg/kg every 4–6 hours (max. per dose 2.4 g every 4 hours) in severe infection *IV route recommended in neonates and children.* **Endocarditis (in combination with another antibacterial agent if necessary)** **SLOW IV, IV INFUSION** Child: 25 mg/kg every 4 hours; increased if necessary, to 50 mg/kg every 4 hours (max. per dose 2.4 g every 4 hours) **Meningitis, Meningococcal disease** **IV INFUSION** Neonate up to 7 days: 50 mg/kg BD. Neonate 7 days to 28 days: 50 mg/kg TDS. Child: 50 mg/kg every 4–6 hours (max. per dose 2.4 g every 4 hours) **Neonatal sepsis** **IM/ SLOW IV, IV INFUSION** **Neonate up to 7 days** 25 mg/kg BD increased if necessary, to 25 mg/kg TDS *Intravenous route recommended in*	hypersensitivity, Antibiotic-associated colitis, Jarisch-Herxheimer reaction (JHR), Intravenous route preferred for neonates and infants.

		neonates. **Neonate 7 days to 28 days** 25 mg/kg TDS increased if necessary, to 50 mg/kg TDS in severe infection, IV recommended in neonates.	
BENZATHINE BENZYLPENICILLIN IM	Syphilis, Erysipelas, prophylaxis of Rheumatic fever.	**Erysipelas, Prophylaxis of rheumatic fever [without cardiac involvement/transient/persistant], Prophylaxis of poststreptococcal glomerulonephritis [without cardiac involvement/ transient/persistant], Prophylaxis of erysipelas [without cardiac involvement/transient/persistant]** **Deep IM** **Child** body-weight up to 30 kg: 600, 000 units STAT body-weight 30 kg and above: 1.2 million units STAT *mid-lateral thigh muscle is the preferred site of injection* **Primary and secondary stage Syphilis** **DEEP IM INJ** **Child** 1m–11yrs: 50 000 units/kg (max. per dose 2.4 million units) STAT 12–17 years: 2.4 million units STAT *Outer quadrant of the gluteus maximus or Hochstetter's ventrogluteal field is the preferred site of Injection* **Syphilis [late-stage (latent seropositive)]** **DEEP IM INJ** **Child** 1 m–11 yrs: 50 000 units/kg once weekly (max. per dose 2.4 million units) for 3 weeks *Mid-lateral thigh muscle is the preferred site of injection* 12–17 yrs: 2.4 million units once weekly for 3 weeks	Hypersensitivity, anaphylactic reactions, increased risk of infection, JHR, Nausea, vomiting

		Outer quadrant of the gluteus maximus or Hochstetter's ventrogluteal field is the preferred site of injection **Congenital syphilis [without neurological involvement]** **DEEP IM INJECTION** **Neonate**: 50 000 units/kg STAT **Child** 1–24 months: 50 000 units/kg STAT *mid-lateral thigh muscle is the preferred site of injection.*	
CEFOTAXIME INJ IV/IM	Gonorrhoea, Haemophilus influenzae, congenital gonococcal conjunctivitis, meningitis, meningococcal disease, severe infections due to gram negative and positive bacteria.	**Uncomplicated gonorrhoea** **IM INJECTION** **Child** 12–17 years: 500 mg stat Disseminated gonococcal infection **IV INJ OR INFUSION** **Child** 16–17 years: 1 g TDS for 7 days, may be switched 24–48 hours after symptoms improve to a suitable oral antibacterial **Severe exacerbations of Haemophilus influenzae infection in cystic fibrosis** **BY IV INJ OR INFUSION** **Child**: 50 mg/kg every 6–8 hours; maximum 12 g per day **Congenital gonococcal conjunctivitis** **BY IM INJ** **Neonate**: 100 mg/kg (max. per dose 1 g) stat. **Infections due to sensitive Gram-positive and Gram negative bacteria | Surgical prophylaxis | Haemophilus epiglottitis** **BY IM OR IV INFUSION** **Neonate** up to 7 days: 25 mg/kg BD Neonate 7 days to 20 days: 25 mg/kg TDS	Drug fever, renal impairment, neutropenia, Pseudomembranous enterocolitis

		Neonate 21 days to 28 days: 25 mg/kg TDS - QID Child: 50 mg/kg BD-TDS **Severe susceptible infections due to sensitive Gram positive and Gram-negative bacteria, Meningitis** **IM, IV, OR BY IV INFUSION** Neonate up to 7 days: 50 mg/kg BD Neonate 7 days to 20 days: 50 mg/kg TDS Neonate 21 days to 28 days: 50 mg/kg TDS/QID Child: 50 mg/kg QID (MAX 12g/ day)	

DRUG NAME	INDICATIONS	DOSE	SIDE EFFECTS
CEFTRIAXONE 1g IM/IV	Susceptible gram negative and positive bacteria e.g. Pneumonia, intra-abdominal infections, skin and soft tissue infections, meningitis, Endocarditis, PID, Gonorrhea, Lyme disease, congenital gonococcal conjunctivitis	**Community-acquired pneumonia, Hospital-acquired Pneumonia, Intra-abdominal infections, Complicated UTI** **BY IV INFUSION** Neonate up to 15 days: 20–50 mg/kg OD Neonate 15 days to 11years 50–80 mg/kg OD (MAX 4g per day for child 1m-11yrs) Child 9–17 years (body-weight 50 kg and above): 1–2g OD, *2g dose to be used for hospital-acquired pneumonia and severe cases* IV INJECTION 9-17yrs: 1-2g OD IM INJECTION 1month-11yrs(<50kg): 50-80mg/kg OD Max: 4g 9-17yrs(>50kg): 1-2g OD **Complicated skin and soft tissue infections, Infections of**	Kernicterus, antibiotic associated colitis, cholelithiasis, hypersensitivity, pancreatitis, nephrolithiasis, neutropenia Precipitates of Calcium-Ceftriaxone can occur in the gall bladder and urine especially in the young, dehydrated or immobilized. Discontinue treatment if symptomatic

		bones and joints **IV INFUSION** Neonate up to 15 days: 20–50 mg/kg OD **Neonate 15 days to 11years(<50kg):** 50–100 mg/kg OD (maximum 4 g per day for child 1 m -11 yrs) **Child 9–17 yrs(>50kg):** 2 g OD **IM INJECTION** **1month-11yrs(<50kg**): 50-100mg/kg OD Max: 4g per day **9-17yrs(>50mg):** 2g OD **Bacterial meningitis, Bacterial endocarditis** **IV INFUSION** **Neonate up to 15 days**: 50 mg/kg OD **Neonate 15 days to 11 yrs**: 80–100 mg/kg OD (100 mg/kg OD dose should be used for bacterial endocarditis.) max: 4 g per day in child 1m - 11 yrs Child 9–1 7years: 2–4 g OD **IV INJECTION** **9-17yrs(>50kg)**: 2-4g OD **IM INJECTION** 1month-11yrs(<50kg): 80-100mg/kg(100mg/kg should be used for Bacterial Endocarditis). 9-17yrs(>50kg): 2-4g OD **PID** **IM INJECTION** 1month-11yrs(<45kg): 125mg STAT 9-11yrs(>45kg): 250mg STAT 12-17yrs: 500mg STAT	

		SYPHILIS **Neonate** upto 15days: 50/mg OD x 10-14days Neonate 15days-11yrs(<50kg): 75-100mg OD x 10-14days Max: 4g for 1month-11yrs 9-17yrs(>50kg): 0.5-1g OD x 10-14days **IV INJECTION** 9-17yrs(>50kg): 0.5-1g OD x 10-14days **IM INJECTION** 1month-11yrs(<50kg): 75-100mg/kg OD x 10-14days Max: 4g per day 9-17yrs(>50kg): 0.5-1g OD x 10-14days	
CEFEPIME 1g IV/IM	Infections due to sensitive gram negative and positive bacteria.	**1 month**: 30mg/kg 8-12hrly **2 months – 17 yrs:** 50mg/kg 8-12hrly **Max per dose**: 2g.	Anemia, GIT disorders, increased risk of infection, genital pruritus, paresthesia, vasodilation
CEFTAZIDIME 1g IV/IM	Pseudomonal lung infection, Meningitis Febrile neutropenia, Infections due to sensitive gram positive and gram-negative bacteria	**NEONATES** Up to 7days: 25-50mg/kg 24hrly 7-20days: 25-50mg/kg 8-12hrly 21-28days: 25-50mg/kg 8hrly **CHILD** 25-50mg/kg 8hrly (max 6g/days)	Give over 20-30mins Thrombocytosis, Thrombophlebitis, Antibiotic associated colitis, fungal infection, Acute kidney injury (rare) neurological effects, tremor, altered taste
CIPROFLOXACIN PO/IV	Chron's disease, GIT and respiratory tract infections, Gonococcal infection, bronchiectasis, pseudomonal lower respiratory tract infection in cystic fibrosis, complicated UTIs, Anthrax, acute pyelonephritis, prevention of secondary case of meningococcal meningitis	**Fistulating Crohn's disease** **ORAL** **Child**: 5 mg/kg BD **Severe respiratory-tract infections, GIT infection** **ORAL** **Neonate**: 15 mg/kg BD. **Child**: 20 mg/kg BD(max. per dose 750 mg) **IV INFUSION** **Neonate**: 10 mg/kg BD **Child**: 10 mg/kg TDS (max. per dose 400 mg), *to be given over 60 minutes*	skin reactions, taste alterations, arrhythmias, fungal infection, constipation, cough

		Acute exacerbation of bronchiectasis, Pseudomonal lower respiratory-tract infection in cystic fibrosis **ORAL** **Child** 1–17 years: 20 mg/kg BD (max. per dose 750 mg) for 7–14 days **IV INFUSION** **Child** 1–17 years: 10 mg/kg TDS (max. per dose 400 mg) *to be given over 60 minutes* **Complicated urinary-tract infections** **ORAL** **Neonate**: 10 mg/kg BD. **Child**: 10 mg/kg BD, dose to be doubled in severe infection (max. 750mg BD) **IV INFUSION** **Neonate**: 6 mg/kg BD *to be given over 60 minutes.* **Child**: 6 mg/kg TDS; increased to 10 mg/kg every TDS (max. per dose 400 mg), in severe infection **Uncomplicated gonorrhoea [when sensitivity confirmed given in combination with azithromycin], Uncomplicated gonorrhoea [anogenital and pharyngeal infection, when sensitivity confirmed]** **ORAL** **Child** 13–15 years: 500 mg STAT **Disseminated gonococcal infection** **IV INFUSION** **Child** 16–17 years: 500 mg BD for 7 days, may	

		be switched 24–48 hours after symptoms improve to a suitable oral antibacterial agent **ORAL** **Child** 16–17 years: 500 mg BD, following IV antibacterial treatment, starting 24–48 hours after symptoms improve, treatment given for 7 days **Anthrax (treatment and PEP)** **ORAL** **Child**: 15 mg/kg BD (max. per dose 500 mg) **IV INFUSION** **Child**: 10 mg/kg BD (max. per dose 400 mg) **Prevention of secondary case of meningococcal meningitis** **ORAL** **Neonate**: 30 mg/kg (max. per dose 125 mg) STAT. **Child** 1m–4 yrs: 30 mg/kg (max. per dose 125 mg) STAT 5–11 years: 250 mg STAT 12–17 years: 500 mg STAT **Acute pyelonephritis, Urinary tract infection (catheter associated)** **ORAL** **Child** 16–17 years: 500 mg BD for 7 days **IV INFUSION** **Child** 16–17 years: 400 mg every 8–12 hours	
CO-TRIMOXAZOLE PO/IV	Prophylaxis and treatment of PJP and Toxoplasmosis, Nocardiosis.	**ORAL** **6 weeks-5m:** 120mg x 12hrly **6m- 5 yrs:** 240mg x 12hrly **6yrs-11yrs:** 480mg x 12hrly **12yrs-17yrs:** 960mg x 12hrly (24mg/kg 12hrly) **IV** **> 6 weeks:** 18-27mg/kg x 12hrly Max: 1.44g per dose **PJP treatment**	Electrolyte imbalance, aplastic anemia, angioedema. Discontinue use when blood disorders such as leucopenia, megaloblastic anemia, thrombocytopenia,

		ORAL / IV 120mg/kg in 2-4divided doses for 14 - 21days **Prophylaxis of PJP** **ORAL** Child: 450 mg/m2 BD (max. per dose 960 mg BD) 3 x week	eosinophilia or SJS or Toxic Epidermal Necrolysis occur
DAPSONE TABLETS PO	Prophylaxis treatment of PJP/PCP	**1m-11yrs:** 2mg/kg OD (max dose 100mg). **>12yrs-17yrs:** 100mg OD.	Hemolytic anemia, hepatic disorders, hypoalbuminemia, lepra reaction, methemoglobinemia, motor loss nausea peripheral neuropathy, photosensitivity reaction, psychosis. Severe Cutaneous adverse reactions (SCARs). If dapsone syndrome occurs (rash with fever and eosinophilia)—discontinue immediately
DRUG NAME	**INDICATIONS**	**DOSE**	**SIDE EFFECTS**
FLUCONAZOLE IV/PO	Candidal balanitis, Vaginal candidiasis, Prevention of fungal infection in immunocompromised patients, Tinea capitis, Tinea pedis, corporis, cruris, pityriasis versicolor, dermal candidiasis, Vulvovaginal candidiasis (recurrent), Mucosal candidiasis (except genital), Invasive candidal infections and cryptococcal infections	**Candidal balanitis** **ORAL** **Child 16-17 years:** 150mg STAT **Vaginal candidiasis** **Child** 150mg STAT, for use in patients that are post puberty **Prevention of fungal infection in immunocompromised patients** **ORAL or by IV infusion** Neonate up to 14 days: 3-12 mg/kg every 72 hours Neonate 14days to 28 days: 3-12 mg/kg every 48hours dose given according to extent and duration of neutropenia Child: 3-12mg/kg daily (max, per dose 400mg)	GIT discomfort, skin reactions, diarrhea, vomiting, hepatic disorders, flatulence, alopecia, seizure

		Tinea capitis **ORAL:** Child 1- 17years: 6mg/kg daily (max per dose 300mg) for 2-4 weeks **Tinea pedis, corporis, cruris, pityriasis versicolor, dermal candidiasis** **ORAL:** CHILD: 3mg/kg daily (max. per dose 50mg) for 2-4 weeks (for up to 6 weeks in tinea pedis). Max duration of treatment 6 weeks **Vulvovaginal candidiasis (recurrent)** **ORAL:** CHILD: initially 150mg every 72 hours for 3 doses, then 150mg once weekly for 6 months, for use in patients that are post puberty **Mucosal candidiasis (except genital)** Neonate up to 14 days: 3-6mg/kg, dose to be given on first day, then 3mg/kg every 72 hours 14 days – 28days: 3-6mg/kg, dose to be given on first day, then 3mg/kg every 48 hours Child 1 month to 11 years: 3-6mg/kg, dose to be given on first day, then 3mg/kg daily (max per dose 100mg) for 7-14 days in oropharyngeal candidiasis. Child 12-17 years: 50mg daily for 7-14 days in oropharyngeal candidiasis **Invasive candidal infections and cryptococcal infections** Neonate up to 14 days: 6-12mg/kg every 72 hours, treatment continued according to response (atleast 8 weeks for cryptococcal meningitis) Neonate 14days to 28 days: 6-12mg/kg every 48hours, treatment continued according to response (atleast 8 weeks for cryptococcal meningitis)	

		Child: 6-12mg/kg daily (max per dose 800mg), treatment continued according to response (atleast 8 weeks for cryptococcal meningitis)	
GENTAMYCIN IV	Septicemia, Meningitis, Biliary-tract infection, Endocarditis, Pneumonia, Listeria Meningitis, Sepsis, Pseudomonal lung infection, Bacterial Ventriculitis, Acute Pyelonephritis, uncomplicated Gonorrhea	**NEONATAL SEPSIS** **SLOW IV INJ, OR IV INFUSION** **Neonates to 7days:** 5mg/kg every 36hrs **Neonates 7-28 days**: 5mg/kg every 24hrs. **Pseudomonal lung infection in cystic fibrosis** **SLOW IV INJ, OR IV INFUSION** **Child:** 3mg/kg TDS *intravenous injection to be administered over at least 3 minutes* **Septicemia/Meningitis/CNS infection/biliary tract infection/Endocarditis/Pneumonia/ listerial Meningitis** **IM / slow IV** **Child 1m-11yrs:** 2.5mg/kg TDS **12-17yrs:** 2mg/kg TDS *intravenous injection administered over at least 3 minutes* **IV INFUSION** **Child:** 7mg/kg once daily initially and then adjusted according to serum concentrations. (NOT SUITABLE FOR ENDOCARDITIS OR MENINGITIS)	Antibiotic-associated colitis, Blood disorders, depression, neurotoxicity, Vestibular damage, Peripheral neuropathy
IMIPENEM+ CILASTATIN IV	Anaerobic and aerobic gram positive and negative bacterial infections, cystic fibrosis, Pseudomonal infections, hospital Acquired septicemia.	**IV INFUSION** **NEONATES** Up to 7days: 20mg/kg BD 7-20days: 20mg/kg TDS 21days-2m: 20mg/kg QID 3m-17yrs:15-25mg/kg QID Max per dose: 1g.	bone marrow disorders, confusion, thrombophlebitis, eosinophilia.

MEROPENEM IV	Anaerobic and aerobic gram positive and negative bacterial infections, Exacerbations of chronic lower respiratory-tract infection in cystic fibrosis, hospital Acquired septicemia, meningitis	**IV INFUSION, IV INJ** **NEONATES** **Up to 7days:** 20-40mg/kg BD **7-28days:** 20-40mg/kg TDS **CHILD** **1m-11yrs(<50kg):** 10-40mg/kg TDS **1m-11yrs(>50kg):** 0.5g-2g x TDS **12-17yrs:** 0.5g-2g TDS	Abdominal pain, diarrhoea, inflammation, thrombocytosis, haemolytic anaemia, antibiotic associated colitis, neutropenia.

TOXIN	ANTIDOTE	DOSE
PARACETAMOL	N-ACETYLCYSTEINE	**First Infusion (over 1 hour)** 150 mg/kg (0.75 mL/kg) of acetylcysteine dose to be administered in 3 mL/kg glucose 5% (child body weight 20-39kg use 100ml glucose 5% and child 40kg and above use 200mls of glucose 5%) *Up to a maximum dose of 15 grams.* **Second Infusion (over 4 hours)** 50 mg/kg (0.25 mL/kg) of acetylcysteine dose to be administered in 7 mL/kg glucose 5%, (child body weight 20-39kg use 250mls glucose 5% and child 40kg and above use 500mls of glucose 5%) *Up to a maximum dose of 5 grams.* **Third Infusion (over 16 hours)** 100 mg/kg (0.5 mL/kg) of acetylcysteine Dose to be administered in 14 mL/kg glucose 5%. (child body weight 20-39kg use 500mls glucose 5% and child 40kg and above use 1L of glucose 5%) *Up to a maximum dose of 10 grams.*
OPIODS	NALOXONE	IV, IM, SC: 0.1 mg initially; repeat (if necessary) at 1 minute intervals (max 2mg)
ORGANOPHOSPHATE POISONING	PRALIDOXIME	Adjunct to atropine in the treatment **IV INFUSION** **Child**: Initially 30 mg/kg, to be given over 20 minutes, followed by 8 mg/kg/hour (Max 12 g per day)
BENZODIAZEPINES	FLUMAZENIL	**Initial dose: (IV)** 0.01 mg/kg (up to maximum of 0.2 mg) every 1 minute if required, administered over 15 seconds (for a child max dose is 0.2mg/ 0.05mg/kg)

		Reversal of sedative effects of benzodiazepines **IV INFUSION** 0.002–0.01 mg/kg/hour, adjusted according to response. (max. per dose 0.4mg/hour), adjusted according to response
CYANIDE	SODIUM THIOSULPHATE	Inhale 0.3 ml amyl nitrite, emptied onto gauze swab. Followed by 10 ml of sodium nitrite administered IV over 3-4 minutes. After 5 minutes this should be followed by 50 ml of 50% sodium thiosulphate solution IV over 10 minutes. A repeat dose of both sodium nitrite and thiosulphate may be given after 1 hour if necessary

TOXIN	ANTIDOTE	DOSE
ISONIAZID	PYRIDOXINE	Pyridoxine-dependent seizures IV INJ Neonate: Test dose 50–100 mg, repeated, if necessary, if responsive, followed by an oral maintenance dose 50–100 mg OD, dose to be adjusted as necessary. Child 1m–11 yrs: Test dose 50–100 mg daily, if responsive, followed by an oral maintenance dose, maintenance 20–50 mg 1–2 times a day, dose to be adjusted as necessary, increased if necessary up to 30 mg/kg daily, alternatively increased if necessary, up to 1 g daily
WARFARIN	VITAMIN K	Reversal of Warfarin Effects Omit 1-2 doses, or hold warfarin; monitor INR and adjust warfarin dose accordingly INR 4.5-10, no bleeding: vitamin K1 (phytonadione) 1-2.5 mg PO STAT INR >10, no bleeding: Vitamin K1 PO: 2.5-5 mg PO STAT; INR reduction observed within 24-48 hr, monitor INR and give additional vitamin K if needed
METHOTREXATE	FOLINIC ACID (LEUCOVORIN)	**Infants, Children, and Adolescents** **ORAL, IM, IV** 10mg/m2/dose every 6 hours until the methotrexate level is < 0.01 micromolar *begin as soon as possible after overdose*. If serum creatinine is increased more than 50% above baseline 24 hours after methotrexate administration, or if methotrexate level is > 5 micromolar, or if 48 – hour methotrexate level is > 0.9 micromolar, increase leucovorin dose to 100mg/m2 IV every 3 hours until the methotrexate level is < 0.01 micromolar. Hydration (3L/day) and urinary alkalinization with sodium bicarbonate solution should be employed concomitantly, bicarbonate dose should be adjusted to maintain urine pH at 7.0 or greater.
DIGOXIN	IMMUNOTHERAPY WITH DIGOXIN FAB FRAGMENTS (DIGIBIND)	**Acute Ingestion of Unknown Quantity** POWDER FOR INJECTION

		40mg/vial Administer 20 vials (40mg /vial) Larger dose has faster onset, but may cause febrile reaction; may give 10 vials initially, then at physician's discretion, administer 10 more vials Each vial of DigiFab (40 mg of Fab) binds 0.5 mg digoxin *Equimolar dose to ingested digoxin (76/80 mg FAB = 1 mg digoxin)* **Acute Ingestion of Known Amount** Total digoxin load (mg) divided by 0.5 mg digoxin bound per vial = number of vials to use **Toxicity During Chronic Therapy** Acute distress or for whom a serum digoxin concentration is not available: Administer 6 vials Administer by IV infusion over 15-30 minutes May be given IVP for life-threatening dysrhythmias
FERROUS SULPHATE	DESFERRIOXAMINE	**IV INFUSION:** 15 mg/kg/hour (Max 80mg/kg in 24 hours, dose to be reduced after 4- 6 hours) as continuous infusion until lab values and clinical status are normalized and urine is of normal color.
HEPARIN	PROTAMINE SULPHATE	**Overdosage with intravenous injection or infusion of unfractionated heparin (less than 30 minutes lapsed since overdose)** **IV** **Child**: 1 mg (max. per dose 50 mg), to be administered at a rate not exceeding 5 mg/minute, to neutralize each 100 units of unfractionated heparin **Overdosage with subcutaneous injection of low molecular weight heparin** **CONTINUOUS INTRAVENOUS INFUSION** **Child:** (max. per dose 50 mg), 50 – 100 % of the total dose to be administered by intermittent intravenous injection at a rate not exceeding 5 mg/minute thereafter remaining dose given by continuous intravenous infusion over 8-16 hrs, 1mg neutralizes approx. 100 units of low molecular weight heparin, consult product literature of low molecular weight heparin for details

TOXIN	ANTIDOTE	DOSE
CARBAMAZEPINE, DAPSONE, QUININE, PHENOBARBITONE, THEOPHYLLINE.	ACTIVATED CHARCOAL	**Reduction of absorption of poisons in the gastro-intestinal system** **ORAL** Neonate: 1 g/kg. 1m–11 years: 1 g/kg (max. per dose 50 g) Child 12–17 years: 50 g **Active elimination of poisons** **ORAL** Neonate – 11yrs: 1 g/kg every 4 hours, dose may be reduced and the frequency increased if not tolerated, reduced dose may compromise efficacy. Child 12–17 years: Initially 50 g, then 50 g every 4 hours, reduced if not tolerated to 25 g every 2 hours,
ANTICHOLINERGIC TOXICITY	PHYSYOSTIGMINE	0.02 mg/kg/dose (maximum 0.5 mg/min) slow **IV** Repeat dose as needed every 5- 10 minute until desired clinical effect (not to exceed maximum cumulative dose of 2 mg) Additional doses may be required for relapse after initial clinical improvement due to short duration of action of physostigmine
REVERSAL OF CHOLINERGIC TOXIDROME DUE TO ORGANOPHOSPHATE AND CARBAMATE PESTICIDES	ATROPINE	Children less than 12 years: 0.02 - 0.05 mg/kg **IV** every 10 - 20 minutes until atropine effect is seen, then every 1 - 4 hours for at least 24 hours. Max single dose for neonate (0 -4 weeks): 0.03 mg/kg. Max single dose for children: 0.5 mg. Children 12 years of age or older: 1 - 2 mg IV initially. If no response, the dose is doubled every 5 - 10 minutes until tracheobronchial secretions are dry and patient can be oxygenated. Continue treatment until gradual reduction* in dose does not cause reappearance of cholinergic symptoms. In severe organophosphate poisoning, very large doses and treatment for days to weeks has been

		necessary (total of 9 to 11 g over 30 to 40 days). Administration by infusion in severe cases may be considered. Initial infusion rates of 0.02 - 0.08 mg/kg/hour have been recommended (up to 2.4 mg/kg/hour has been required). *After prolonged use of high doses of atropine it must be tapered gradually and reinstated if cholinergic symptoms recur.
LEAD	DIMERCAPROL	IM Dosing 2.5 mg/kg QID x 2 days (days 1 & 2) then bd for 1 day (day 3) thereafter QID (days 4-10)
CALCIUM CHANNEL BLOCKER/BETA BLOCKER OVERDOSE AND HYPOCALCAEMIA	CALCIUM GLUCONATE	**Acute Hypocalcemia (urgent correction)** **Slow IV INJ** Neonates:0.5mL/kg of 10% solution IV over 5-10 minutes. Child: 0.5mL/kg of 10% solution IV over 5-10 minutes, maximum 20 mL/dose. *MONITOR SERUM CALCIUM LEVELS AND REASSESS.* **Hypotension** Intravenous fluid resuscitation Atropine for bradycardia 0.02mg/kg IV injection; Repeat doses may be necessary. Maximum per dose is 1.2mg NOTE: Only if overdose is known to include a Beta Blocker, give Glucagon as follows: **Glucagon IV injection:** Child: 0.05-0.15 mg/kg administered over 1-2 mins, may repeat in 20 minutes Followed by 0.05mg/kg/hr infusion titrated according to response. **Vasopressors** ADRENALINE 1:10,000 (CONTINUOUS INTRAVENOUS INFUSION) Adrenaline 0.0001- 0.0015mg/kg/min, titrate as required If NO response, add Dopamine OR Dobutamine (And start high dose insulin euglycemia therapy)

CHAPTER 18: TELEPHONE DIRECTORY

Province	Hospital	Contact
Lusaka	**UTHs – Children's Hospital** ***National Paediatric Hotline available 24/7 for referrals and consultations***	+260 978 498 020 +260 950 000 266
	UTH – Women and Newborn Hospital	+260 950 303 884
	UTH – Adult Hospital	+260 971 463 589
	UTH – Cancer Disease Hospital	+260 211 257 706
	UTH - Eye Hospital	+260 954 673 317
	UTH – Blood Bank	+260 776 450 578
	National Heart Hospital	+260 954 973 872 +260 957 661 399
	Levy Mwanawasa University Teaching Hospital	+260 979 694 675
	Kafue General Hospital	+260 770 876 663
	Kanyama Level 1 Hospital	+260 972 210 895 +260 772 240 293
	Chawama Level 1 Hospital	+260 770 450 114 +260 977 610 285
	Chilenje Level 1 Hospital	+260 750 430 900 +260 773 311 852
	Chelstone Level 1 Hospital	+260 773 026 558
	Matero Level 1 Hospital	+260 969 274 465
	Chongwe District Hospital	+260 975 910 830
	St Luke's Mission Hospital	+260 969 827 775
	Katondwe Mission Hospital	+260 972 068 319
Copperbelt	Ndola Teaching Hospital	+260 212 611 589
	Kitwe Teaching Hospital	+260 966 928 617 +260 974 677 046
	Arthur Davison Hospital	+260 212 640 216 +260 955 810 545
	Roan General Hospital	+260 974 931 102
	Nchanga North Hospital	+260 966 946 956
	Kalulushi General Hospital	+260 966 782 717
	Ronald Ross Hospital	+260 961 948 105
	Thompson General Hospital	+260 961 893 374
	Chililabombwe District Hospital	+260 975 443 377
	St Theresa Mission Hospital	+260 965 747 665
	Mpongwe Mission Hospital	+260 966 540 470
	Chifubu Level 1 Hospital	+260 972 319 118
	Chamboli Level 1 Hospital	+260 775 881 863

Central Province	Kabwe General Hospital	+260 976 776 001
	Kabwe Women And New Born Hospital	+260 977 477 861
	Mumbwa District Hospital	+260 960 533 750
	St. Thomas Mission Hospital	+260 968 385 440
	Kapiri Mposhi District Hospital	+260 956 291 328
	Serenje General Hospital	+260 976 497 505 +260 960 959 456
	Mkushi District Hospital	+260 978 580 060
Northern Province	Kasama General Hospital	+260 953 231 030
	Mbala General Hospital	+260 975 217 770
	Mpulungu District Hospital	+260 779 508 678
	Kaputa District Hospital	+260 777 596 027
	Mporokoso District Hospital	+260 973 997 270 +260 958 887 118
	Chilubi District Hospital	+260 972 767 997
Muchinga	Chinsali General Hospital	+260 976 993 202
	Chilonga Mission General Hospital	+260 979 742 838
	Micheal Chilufya Sata Hospital	+260 977 451 289
	Nakonde District Hospital	+260 970 865 203
	Isoka District Hospital	+260 968 196 619
	Mafinga District Hospital	+260 776 223 598
	Muyombe District Hospital	+260 972 230 500
	Lavushi Manda District Hospital	+260 764 723 205
Eastern Province	Chipata Central Hospital	+260 972 660 767 +260 961 517 604
	St Francis Mission Hospital	+260 976 503 061
	Kalindawalo General Hospital	+260 965 894 701
	Mwami Mission Hospital	+260 964 235 601
	Mambwe District Hospital	+260 775 478 605
	Nyimba District Hospital	+260 972 827 853
	Vubwi District Mission	+260 975 169 884
Southern Province	Livingstone University Teaching Hospital	+260 977 312 204
	Choma General Hospital	+260 977 750 289
	Monze Mission Hospital	+260 977 381 314
	Mazabuka General Hospital	+260 773 026 558
	Macha Mission Hospital	+260 975 099 177
	Chikankata Mission Hospital	+260 979 335 805
	Chirundu District Hospital	+260 963 358 007
	Namianga Mission Hospital	+260 770 168 864
	Lewanika General Hospital	+260 977 418 258

Western Province	Senanga General Hospital	+260 976 016 162
	Kaoma District Hospital	+260 962 657 496
	Kalabo District Hospital	+260 978 068 711
	Yeta District Hospital	+260 976 169 609
	Lukulu District Hospital	+260 766 822 466
	Shang'ombo District Hospital	+260 779 379 662
	Mwandi Mission Hospital	+260 772 464 821
	Mulobezi District Hospital	+260 765 184 790
	Nkeyama District Hospital	+260 977 655 933
	Munkuye Hospital	+260 768 379 004
North-Western Province	Solwezi General Hospital	+260 969 108 621
	Mukinge Mission Hospital	+260 965 647 761 +260 979 305 675 +260 971 555 878
	Kabompo District Hospital	+260 965 277 595 +260 952 942 544 +260 973 401 023
	Zambezi District Hospital	+260 966 458 064
	Kaleni Mission Hospital	+260 978 409 662
	Chavuma District Hospital	+260 977 661 360
	Mufumbwe District Hospital	+260 968 674 830
	Mwinilunga District Hospital	+260 777 643 307
Luapula Province	Mansa General Hospital	+260 978 475 446
	Kawambwa District Hospital	+260 772 019 259
	Samfya District Hospital	+260 970 975 474

Ministry of Health

Zambia Paediatric Association

World Health Organization

United Nations Children's Fund

Programme for Appropriate Technology in Health

Centre for Infectious Disease Research in Zambia

University of Zambia – School of Medicine

Clinton Health Access Initiative

Catholic Relief Services

University Teaching Hospital HIV/AIDS Programme

Ministry of Health
Ndeke House
P.O Box 30205
Haile Selassie Avenue, Longacres
Lusaka, Zambia.