Standard Treatment Guidelines

A Manual for Medical Practitioners

STANDARD TREATMENT GUIDELINES

MESSAGE

The Tami Nadu Health System Project (TNHSP) is undertaking several initiatives to improve the quality of service in all the secondary care hospitals in the state. Improved quality of care in all the Government Medical Institutions will help significantly in improving the health outcome in the state. Following a standard treatment protocol in treating the patients, is one way to improve the quality of care.

This edition of the Standard Treatment Guidelines for various diseases will enable the Medical Officers to follow an established standard in treating the patients in the Government health facilities. This will ensure that the treatment is effective and done at least cost, too.

The Health and Family Welfare Department of the Government of Tamil Nadu is a front-runner in the provision of health care. It has taken many innovations to enable the best health outcome for the people in the state. The Standard Treatment Guidelines is another indication of the innovation by the state, to sustain the momentum of improving the health outcomes.

I congratulate the experts, for their valuable contribution and the officers of the TNHSP in bringing out the guidelines.

FOREWORD

The management of quality in health care provision and assuring the quality in service provision in public health institutions are critical in ensuring the health outcome of the people.

The Tamil Nadu Health System Project (TNHSP), launched in March 2005, is enabling an architectural correction in the health care delivery in the state. It has taken many steps to improve the health care provision in the state.

The publication of the Standard Treatment Guidelines has been prepared with contributions from several experts. This guideline will assist Medical Officers and other health care providers to provide appropriate and standard health care for clients who visit the public health facilities. This will help us to move towards ensuring and sustaining a good standard in provision of health care thus helping us to realise the best health outcome for the people in the state.

The Health and Family Welfare Department appreciates this initiative of TNHSP in formulating this Standard Treatment Guidelines for implementation in the public health facilities in the state. I recommend this document to all the Medical Practitioners and the health experts in the state and I suggest they utilise this guideline to ensure quality health care in the state.

Principal Secretary to Government
Department of Health & Family Welfare
Government of Tamil Nadu

PREFACE

Guidelines enable the setting of standards for service provision, help in assessing services, improve them, reduce costs and importantly they enable client satisfaction. These are goals that health systems strive towards.

I am happy to note that the Tamil Nadu Health System Project (TNHSP) has published a Standard Treatment Guideline for provision of health care in public health facilities in the state. This is the first initiative of its kind in the state. This guideline will help doctors to provide a standard treatment to all clients. This will ensure that any client, with similar episode of illness, will obtain the same standard of treatment in any public health facility, across the state.

I congratulate the Editorial Committee, the contributors, the reviewers, the publisher and staff of TNHSP for their efforts, in publishing this guideline. I request feedback from the health personnel, which will significantly help us to improve this, further.

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TAMIL NADU HEALTH SYSTEM PROJECT

STANDARD TREATMENT GUIDELINES

INTRODUCTION

The Tamil Nadu Health System Project (TNHSP), Health and Family Welfare Department, Government of Tamil Nadu has taken many steps to improve the quality of care in public health facilities in the state.

The Standard Treatment Guidelines (STG), is a profile of thematic presentation of various medical conditions and existing treatment modalities practiced worldwide. It is a systematically developed method to assist Medical Practitioners and other health care providers in making decisions for specific clinical episodes.
Its use contributes significantly in attaining Total Quality Management (TQM) in health care. It also helps the Medical Practitioners to be apprised of recent advancements in the provision of care.

This initiative by TNHSP is an important component of its efforts at improving quality in health care. A team of experts from renowned Medical Colleges of Tamil Nadu have contributed significantly in compiling this document, especially the various anecdotes, clinical methodology, diagnostic tools, cross references and current practices. An Algorithimic approach to the management of the common clinical conditions will help significantly to save precious time in decision making. The aim of this document is to enable uniformity and rational prescriptions and an attitudinal change to tackle emergencies, epidemics and other health related issues across the state in a better manner.

The guidelines have been peer-reviewed by the faculty at Christian Medical College, Vellore and suggestions were incorporated with the concurrence of the STG Committee.

STG is the first step. Although it is intended to be comprehensive, the users are requested to refer the publications mentioned in the guideline for a comprehensive knowledge. A CD version of the guidelines will be made available through the HMIS (Health Management Information System) of TNHSP. The feedback form in enclosed and the comments and suggestions from the users will help to improve the future editions of this guideline.

It gives me immense satisfaction and joy in introducing the Standard Treatment Guidelines for the benefit of the Medical Practitioners and health care providers in Tamil Nadu.

I also take this opportunity to thank the experts and the staff of TNHSP in enabling the preparation of this guideline.

Project Director
TNHSP

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The Tamilnadu Health Systems Project, Health and Family Welfare Department, Government of Tamilnadu has a taken a major initiative to improve the quality of care in all Government institutions in the state. Standard Treatment Guidelines (STG), a manual for Medical Practitioners to be used at different points of the therapeutic process is one among the components of the Project in its Quality enhancement strategy.

This manual was mode possible with the encouragement of the Secretary, Health and Family Welfare Department, Government of Tamilnadu, Project Director,Tamilnadu Health Systems Project and Chairman of the Standard Treatment Guidelines committee Dr.S.Vijayakumar I.A.S., for his valuable guidance. We are indebted to all our former Project Directors of Tamilnadu Health Systems Project for their timely contributions towards STG.

We express our gratitude to all the contributors, Reviewers, Clinical and Managerial experts in the making of this guidelines.

We are grateful to our Chief Coordinator and Expert Advisor (Capt).Dr.Kamatchi and Prof. Thirumalaikolundu Subramanian, formerly Director and Head, Institute of Internal Medicine, Madras Medical College / Government General Hospital, Chennai who have been the main guiding force behind the STG committee.

We express our thanks to the printers M/s Ikon Press, Chennai for their expertise and special word of thanks to Mr.Karthi of Hybrid screens, Chennai for computer processing.

Statements

- This guidelines is designed to provide concise information and not intended to provide comprehensive scientific information
- For detailed and up to date information as well as to know the current developments, users are requested to go through textbooks, monograms, original articles, review papers, case reports, websites, etc.,
- For administration of each drug, users are requested to go through the latest product information leaflets provided by the manufactures. Moreover dosage schedule are being constantly revised and new side effects are recognized. Hence, users have been reminded to recall the indications dosage, side effects consider the contraindications and interaction before using any drug
- The Editors, the Coordinators, the Contributors, the Reviewers , Publishers and the Funding agency do not assume any liability for any injury and / or damage to person or property arising out of this publication
- Every effort was taken to print the version with appropriate information. However, it is possible that errors might have crept in. Hence users are requested to offer their remarks and suggestions to the following e- mail address < mail@tnhsp.net> tnhsproject@gmail.com for revising the future edition.

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Common Conditions and General Topics

Standard Treatment Guidelines
Tamil Nadu Health Systems Project

Chapter 1

- Fever
- Management of Trauma
- Cardiothoracic Trauma
  - Blunt Injuries
  - Penetrating Injuries
  - Chest Wall Injuries
  - Pleuropulmonary Injuries
  - Mediastinal Injuries
- Principles of Surgery
- Examination of Surgical Patients
- Pre Operative Assessment and Preparation
- Anaesthesia in Surgical Practice
- Post Operative Care
- Wound Care
- Sutures and Wound Dehiscence

Fever

Definition

Fever

An elevation of normal body temperature that exceeds the normal daily variation and occurs in conjunction with an increase in the hypothalamic set point - for example, from 37°C to 39°C.

Hyperthermia

- An unchanged setting of the hypothalamic set point in conjunction with an uncontrolled increase in body temperature that exceeds the body's ability to lose heat
- Heat stroke
  - Thermoregulatory failure in association with a warm environment
- Malignant hyperthermia
  - Hyperthermic and systemic response to halothane and other inhalational anesthetics in patients with genetic abnormality
- Neuroleptic malignant syndrome
  - Syndrome of hyperthermia, autonomic dysregulation, and extrapyramidal side effects caused by neuroleptic agents (e.g., haloperidol)

Hyperpyrexia

- Temperature >41.5°C (>106.7°F) 1
- Can occur with severe infections, but more commonly occurs with central nervous system (CNS) hemorrhages or hyperthermia

Symptoms

Temperature

- The mean normal oral temperature is 36.8° ± 0.4°C (98.2° ± 0.7°F), with low levels at 6 a.m. and high levels at 4–6 p.m.
- The normal daily temperature variation is typically 0.5°C (0.9°F). (However, in some individuals recovering from a febrile illness, daily variation can be as great as 1.0°C.)
  - During a febrile illness, diurnal variations are usually maintained, but at higher levels.
  - Daily temperature swings do not occur in patients with hyperthermia.
  - Rectal temperatures are generally 0.4°C (0.7°F) higher than oral readings. (Lower oral readings are probably attributable to mouth breathing, a particularly important factor in patients with respiratory infections and rapid breathing.)
  - Lower-esophageal temperatures closely reflect core temperature.
  - Tympanic thermometer measurements, although convenient, may be more variable than directly determined oral or rectal values.

Some febrile diseases have characteristic patterns.

- With relapsing fevers, febrile episodes are separated by intervals of normal temperature.
- Tertian fevers are associated with paroxysms on the first and third days. Plasmodium vivax causes tertian fevers.
- Quartan fevers are associated with paroxysms on the first and fourth days. P. malariae causes quartan fevers.
- Other relapsing fevers are related to Borrelia infections and rat-bite fever, which are both associated with days of fever followed by a several-day afebrile period and then a relapse.
- Pel-Ebstein fever, with fevers lasting 3–10 days separated by afebrile periods of 3–10 days, is classic for Hodgkin's disease and other lymphomas.
- In cyclic neutropenia, fevers occur every 21 days and accompany the neutropenia.
- There is no periodicity of fever in patients with familial Mediterranean fever.

Signs

Signs of hyperthermia

- Hallucinations
- Delirium
- Dry skin
- Pupil dilation
- Muscle rigidity

Differential Diagnosis

Fever vs hyperthermia

- It is important to distinguish between fever and hyperthermia.
  - Hyperthermia can be rapidly fatal and characteristically does not respond to antipyretics.
- There is no rapid way to make this distinction.
- Hyperthermia is often diagnosed on the basis of events immediately preceding elevation of core temperature.
  - Heat exposure
  - Treatment with drugs that interfere with thermoregulation
- In addition to clinical history, physical aspects of some forms of hyperthermia may alert the clinician.
  - In heat-stroke syndromes and in the setting of drugs that block sweating, the skin is hot but dry.
  - Antipyretics do not reduce elevated temperature in hyperthermia.
    - In fever and hyperpyrexia, adequate doses of aspirin or acetaminophen usually result in some decrease in body temperature.

Causes of hyperthermia syndromes

- Heat stroke: thermoregulatory failure in association with a warm environment
  - Exertional: caused by exercise in high heat or humidity
    - Even in healthy individuals, dehydration or common medications (e.g., over-the-counter antihistamines with anticholinergic side effects) may help to precipitate exertional heat stroke.
  - Nonexertional: occurs in high heat or humidity
    - Typically affects very young, elderly, or bedridden individuals, particularly during heat waves
    - Also affects patients taking anticholinergic agents (e.g., phenothiazines), antiparkinsonian drugs, diuretics
- Drugs
  - Monoamine oxidase inhibitors (MAOIs)
  - Tricyclic antidepressants
  - Amphetamines
  - Cocaine
  - Phencyclidine
  - “Ectasy” (methylenedioxymethamphetamine)
  - Lysergic acid
  - Diethylamide
  - Salicylates
  - Lithium
  - Anticholinergic agents
- Malignant hyperthermia
  - Elevated temperature, increased muscle metabolism, muscle rigidity, rhabdomyolysis, acidosis, and cardiovascular instability develop rapidly.
  - Occurs with use of inhalational anesthetics or succinylcholine
  - Often fatal
- Neuroleptic malignant syndrome
  - Characterized by “lead-pipe” muscle rigidity, extrapyramidal side effects, autonomic dysregulation, and hyperthermia
  - Occurs in the setting of:
    - Neuroleptic agent use
    - Phenothiazines
    - Butyrophenones, including haloperidol and bromperidol
    - Fluoxetine
    - Loxapine
    - Tricyclic benzodiazepines
    - Metoclopramide
    - Domperidone
    - Thiothixene
    - Molindone
    - Withdrawal of dopaminergic agents
- Serotonin syndrome
  - Seen with selective serotonin uptake inhibitors (SSRIs), MAOIs, tricyclic antidepressants, and other serotonergic medications
  - Has many overlapping features, including hyperthermia, but is distinguished by diarrhea, tremor, and myoclonus
- Endocrinopathy
  - Thyroxicosis

    * Pheochromocytoma
- CNS damage
    * Cerebral hemorrhage
    * Status epilepticus
    * Hypothalamic injury

Diagnostic Approach

- Few signs and symptoms in medicine have as many possible diagnoses as fever.
- The tempo and complexity of the workup will depend on the pace of the illness, diagnostic considerations, and the patient's immune status.
- If findings are focal or if the history, epidemiologic setting, or physical examination suggests certain diagnoses, the laboratory examination can be focused.
- If fever is undifferentiated, the diagnostic net must be cast further.
- A meticulous history is most important.
- Attention must be paid to :
    * Prescription and nonprescription drugs (including supplements and herbs)
    * Surgical or dental procedures
    * Exact nature of prosthetic materials and/or implanted devices
    * Occupational information concerning exposure to:
        » Animals
        » Toxic fumes
        » Potentially infectious agents
        » Possible antigens
        » Febrile or infectious individuals in the home, workplace, or school
    * Geographic area in which the patient has lived
    * Travel history (including military service)
    * Information on:
        » Unusual hobbies
        » Dietary proclivities (e.g., raw or poorly cooked meat, raw fish, unpasteurized milk or cheeses)
        » Household pets
        » Sexual orientation, including precautions taken or omitted
        » Use of tobacco, alcohol, and marijuana or other illicit drugs
        » Trauma
        » Animal bites
        » Tick or other insect bites
        » Transfusions
        » Immunizations
        » Drug allergies or sensitivities
        » Ethnic origin
        » Blacks are most likely to have hemo globulinopathies.
        » Turks, Arabs, Armenians, and Sephardic Jews are especially likely to have familial Mediterranean fever.
    * Information on family members with:
        » Tuberculosis
        » Other febrile or infectious diseases
        » Arthritis or collagen vascular disease
        » Unusual family symptomatology, such as deafness, urticaria, fevers and polyserositis, bone pain, or anemia
- Physical examination should include:
    * Determination of oral or rectal temperature
    * Look for Focus of Infection
        » Drain Pus, if present
        » Check for Intravenous canulae and catheters and remove them and if they cannot be removed, replace them. Treat as per Culture / Sensitivity Report
        » Check for Nosocomial Infection and treat accordingly
    * Examination of:
        » Skin
        » Lymph nodes
        » Eyes
        » Nail beds
        » Cardiovascular system
        » Chest
        » Abdomen
        » Musculoskeletal system
        » Nervous system

» Rectum

* In men: examination of penis, prostate, scrotum, and testes
  » The foreskin, if present, should be retracted.
* In women: pelvic examination, looking for causes of fever such as pelvic inflammatory disease and tubo-ovarian abscess

Investigations

Laboratory Tests

- If history, epidemiologic situation, or physical examination suggests more than a simple viral infection, the following tests may be indicated:
  * Complete blood count
  * Differential count
    » Perform manually or with an instrument sensitive to the identification of eosinophils, juvenile or band forms, toxic granulations, and Döhle bodies
  * Blood smear
    » Appropriate if there is a history of exposure or possible exposure to a variety of pathogens, including: Malaria parasites, Babesia Ehrlichia, Borrelia, Trypanosomes
  * Erythrocyte sedimentation rate
    » Extremely high values (> 100 mm/h) may suggest a primary rheumatologic disorder, vasculitis, or malignancy.
  * Urinalysis, with examination of urinary sediment
  * Chemistries
    » Electrolytes
    » Glucose
    » Blood urea nitrogen
    » Creatinine
    » Liver function
    » Creatine phosphokinase (elevated in hyperthermia) or amylase
  * Microbiologic tests
    » Rapid streptococcal test or throat culture if there is pharyngitis
    » Cultures of blood and urine
    » Stain, fluid analysis, and culture of samples from specific sites of concern identified by history and examination
    » Sputum analysis in patients with suspected pneumonia
    » Joint fluid analysis in patients with arthritis
    » Cerebrospinal fluid analysis in patients with suspected meningitis
    » HIV test in patients at epidemiologic risk

Imaging

- Chest x-ray : Part of the evaluation of any significant febrile illness
- Other imaging studies: guided by symptoms and signs

Diagnostic Procedures

- Lumbar puncture : Indicated in patients with possible bacterial meningitis
- Aspiration and drainage of possibly infected collections or abscesses : Often done with radiologic guidance
- Bone marrow biopsy (not simple aspiration) for histopathologic studies (as well as culture) : Indicated in febrile syndromes when marrow infiltration by pathogens or tumor cells is possible

Treatment Approach

1. If there is Cardio Respiratory Compromise à Shift Patient to Higher Centre with ICU Facilities / Shift Patient to ICU
2. If the Core Body Temperature is More than 41 deg Celsius (105 deg F) à Treat as Hyperthermia and Refer the Patient to a Higher Centre with ICU Facilities / Shift Patient to ICU.
3. Take Smear for Malaria

- Based on whether the Area is High Risk (refer to the chapter on Malaria for more details about High Risk Areas) or Low Risk
  » In High Risk Area, 25 mg/kg of chloroquine base for 3 days (10mg/kg on day 1 and day 2 and 5 mg/kg on day 3) with a single dose of Primaquine 0.75mg/kg on the first day.
  » In Low Risk Area, presumptive treatment with Chloroquine 10 mg/kg single dose

d. Further Management as per Results of Blood Smear Examination

5. Tab Ciprofloxacin 10 mg/kg in 2 divided doses upto a maximum of 750 mg twice daily for 10 – 14 days

6. If associated with Cough / Evening Rise of Temperature / Loss of Appetite / Loss of Weight, Check Sputum for AFB

Treatment

Treatment of fever

- Objectives
  - To reduce the elevated hypothalamic set point
  - To facilitate heat loss
- Treatment to reduce fever is recommended for:
  - Patients with cardiac, cerebrovascular, or pulmonary insufficiency
  - Patients with organic brain disease
  - Children with a history of febrile or nonfebrile seizures
  - There is no correlation between absolute temperature elevation and onset of a febrile seizure in susceptible children.
- Antipyretic treatment should be given on a regular schedule rather than intermittently.
  - Intermittent therapy aggravates chills and sweats.
  - Chronic high-dose therapy with antipyretics (such as aspirin or nonsteroidal anti-inflammatory drugs [NSAIDs] used in arthritis) does not reduce normal core body temperature.

Treatment of hyperthermia

- Objectives
  - To facilitate heat loss
  - To reduce heat production in endogenous hyperthermia

Specific Treatment of fever

- Antipyretic treatment
  - Aspirin, NSAIDs, and glucocorticoids are effective antipyretics.
  - Acetaminophen is preferred because it:
    - Does not mask signs of inflammation
    - Does not impair platelet function
    - Does not adversely affect the GI tract
    - Is not associated with Reye's syndrome
  - Treating fever and its symptoms does no harm and does not slow the resolution of common viral and bacterial infections.
  - Reducing fever with antipyretics also reduces systemic symptoms of headache, myalgias, and arthralgias.
- In hyperpyrexia, the use of cooling blankets facilitates the reduction of temperature.
  - However, cooling blankets should not be used without oral antipyretics.

Specific Treatment of Hyperthermia

- Antipyretics are of no use in hyperthermia.
- Physical cooling should be initiated immediately.
  - A sponge bath with cool water, coupled with the use of fans, is often sufficient.
  - Cooling blankets and ice baths are effective but not well tolerated.
  - Intravenous fluid administration
  - Internal cooling by gastric or peritoneal lavage with iced saline in severe cases
  - In extreme cases, hemodialysis or cardiopulmonary bypass
- Malignant hyperthermia
  - Cessation of anesthesia
  - Administration of dantrolene (1–2.5 mg/kg q6h for at least 24–48 hours) plus
  - Procainamide administration because of risk of ventricular fibrillation
- Neuroleptic malignant syndrome
  - Discontinuation of offending agents
  - Pharmacotherapy not well studied
    - Efficacy has been questioned.
    - Potential agents include:
    - Dantrolene (0.25–2 mg/kg q6–12h IV)
    - Bromocriptine (2.5–10 mg PO or via na sogastric tube q6–8h)
    - Amantadine (200 mg PO or via nasogas tric tube q12h)

Monitoring

- Monitoring of patients with fever depends on the underlying cause.
- Patients with hyperthermia generally require admission to a monitored-care setting until cooling measures have restored normothermia.

Complications

- Complications are related to the underlying cause of fever.
- Hyperthermia is often fatal.

Prognosis

- Fever
  - In most cases, either the patient recovers spontaneously or the history, physical examination, and initial screening laboratory studies lead to a diagnosis.
  - When fever continues for 2–3 weeks and repeat examinations and laboratory tests are unrevealing, the patient is provisionally diagnosed as having fever of unknown origin.
- Hyperthermia
  - The prognosis for hyperthermia depends on the rapidity of cooling.

Prevention

- Fever
  - No common preventive measures
- Hyperthermia
  - Avoid excessive activity in hot or humid environments.
  - Maintain adequate intake of fluids before, during, and after strenuous activity or exposure to extreme heat.
  - Maintain proper ventilation to promote cooling from sweat evaporation.

ICD-10

R50 Fever of other and unknown origin

Management of trauma

Trauma is the leading cause of death in the younger age group Deaths occurring after trauma can be prevented by immediate treatment in some cases

Trauma or injury is characterized by a sudden alteration or physiological imbalance resulting from an acute exposure to physical, chemical, thermal or mechanical energy that exceeds the tolerance level of the body

In all trauma cases transfer of energy occurs to body tissues resulting in tissue damage.

The Golden Hour: The critical first period following injury in which lifesaving measures must be undertaken and definitive management should be started to ensure best chance of survival and to reduce morbidity.

The components of major trauma management are

- Reception of the victim
- Primary survey
- Resuscitation phase
- Secondary survey
- Definitive treatment phase
- Follow up and rehabilitation

Reception and documentation

- Pre hospital care of the injured - Retrieval resuscitation and transfer of the victim to the hospital is an important factor in the outcome of treatment
- Follow major Trauma Guidelines in order or simultaneously depending on the nature and urgency of the situation
- Assessment, investigation and management must proceed simultaneously in major trauma cases
- Record the detailed events of the accident including from where, by whom and how the victim was brought to the hospital It is important in medico-legal cases and identifying unconscious patient

Primary Survey:

Is a rapid assessment of vital functions to identify life threatening conditions and allow their immediate correction (ABCDE)

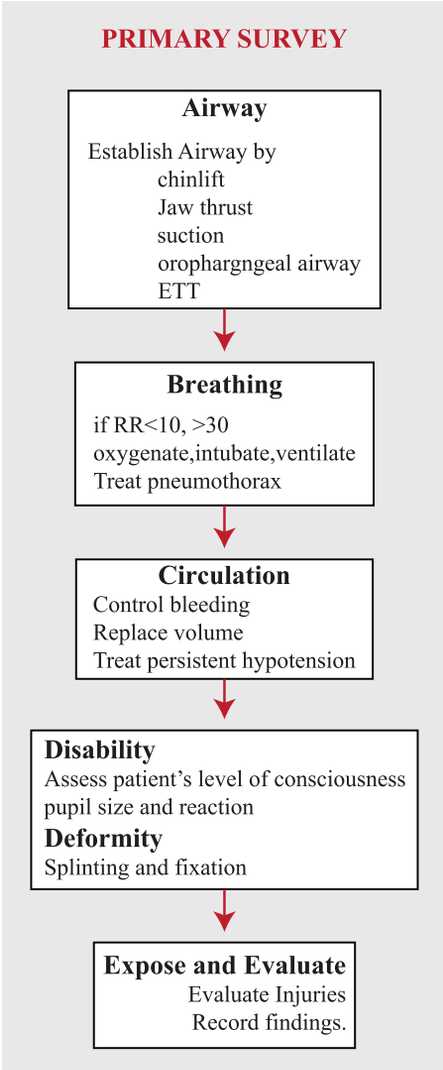
Airway and cervical spine control

Breathing and Ventilation

Circulation with control of bleeding

Disability assessment and Deformity

Exposure and Examination



Airway

- Maintanence of airway is the first and the most important factor in the management of the trauma patient
- Clear airway, mouth, nose and throat of blood, vomitus and secretions by suction
- Maintain airway by chin lift or jaw thrust, suction, oropharyngeal airway or orotracheal intubation
- Immobilize cervical spine in neutral position by semi rigid collars
- Intubate (ETT) whenever required and ventilate

Intubation procedure

- Oxygenate with 100% oxygen through mask or airway
- Cricoid pressure and ET tube held by the assistant (size of the tube is roughly the size of the little finger)
- Lift Tongue with the laryngoscope
- Visualize the vocal cord
- Introduce the E T Tube into the trachea
- Confirm tube position by auscultation
- Inflate cuff to prevent aspiration.

Breathing and Ventilation

- Oxygenate, intubate, ventilate, If Respiratory Rate is <10, >30
- Do not allow the effects of head injury, shock or tissue damage to be compounded by hypoxemia
- Administer high flow oxygen up to 10 l/min by face mask or assisted ventilation
- Treat tension pneumothorax - immedietely. (It is a clinical diagnosis, do not wait for chest X ray), by inserting wide bore canula/ICD
- Drain large hemothorax.
- Occlusive dressing (air tight) and ICD for open pneumothorax

Circulation

- Arrest active bleeding by direct pressure or suturing
- Replace volume with fluids
- Transfuse fully cross matched blood when available
- Reduce of fractures-early
- Surgical hemostasis for uncontrolled bleeding

Causes of persistent hypotension

- Inadequate resuscitation
- Occult bleeding (pelvis,abdomen,chest or femur)

Airway obstruction

- Pneumo and hemothorax
- Pericardial tamponade
- Spinal injury
- Pump failure
- Drugs (beta blockers, nitrates, antihypertensives) and alcohol
- Manage persistent hypotension by adequate resuscitative measures
- Plan urgent surgery when resuscitative measures

fail to stabilize the patient

Disability

- Assess patient's level of consciousness
- Pupil size and reaction to light
- Glasgow coma scale (Eye opening, Motor Response and Verbal response) and APACHE for grading of progression or deterioration of patient's condition
- Hypovolemia may produce severe brain dysfunction, hence should be corrected first.

Exposure and Examination

- Expose the parts
- Evaluate injuries completely
- Record findings

Resuscitative Phase

The initial steps of resuscitation are:

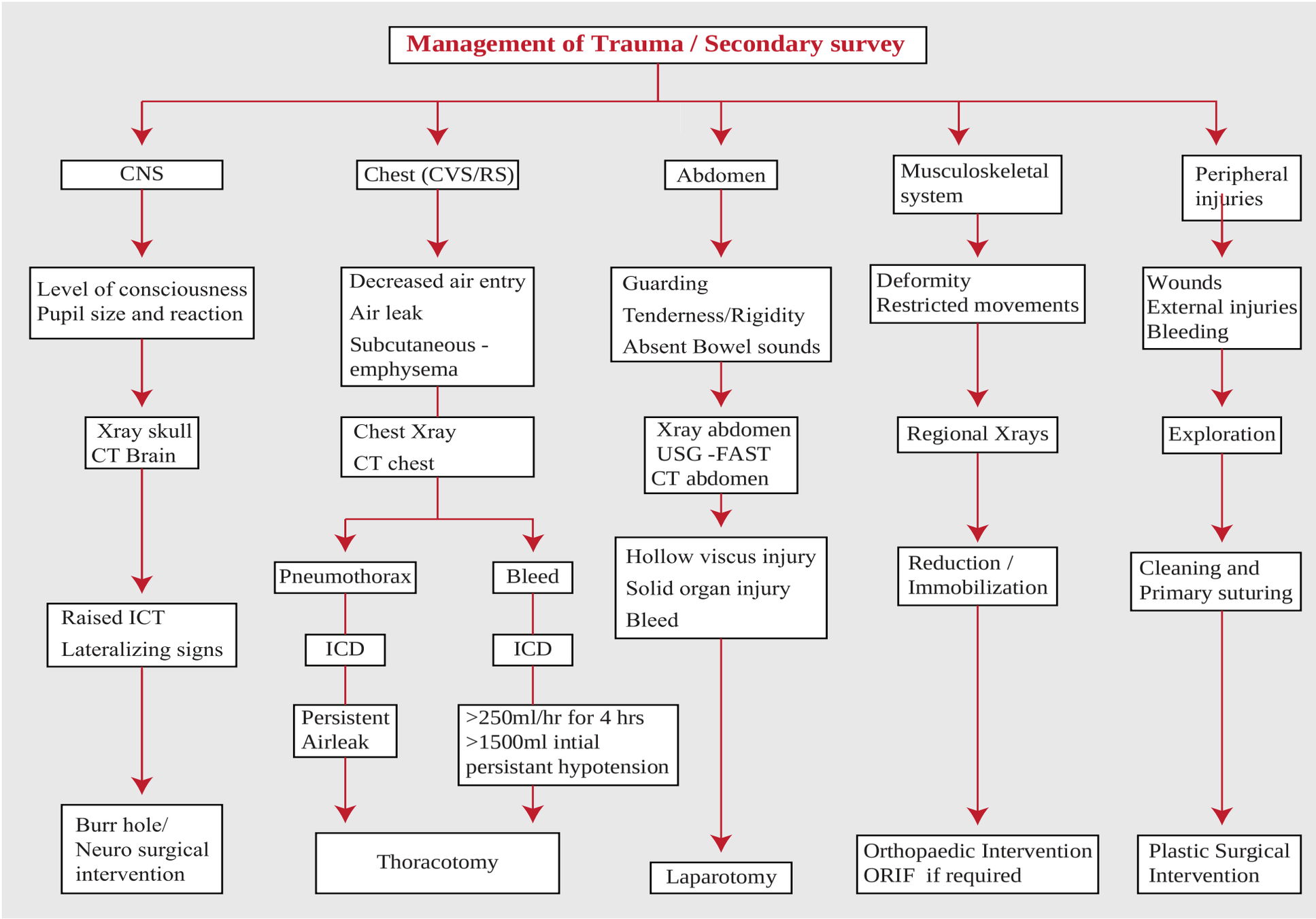
- Establishment and maintenance of secure airway
- Assisted ventilation if necessary with high concentration oxygen
- Arrest of bleeding and rapid restoration of blood volume
- Maintain Breathing and circulation, blood volume and vital parameters
- Record findings at regular intervals before proceeding to the next phase.

Secondary Survey

- Examine in a systematic manner from head to toe to identify all occult injuries, after addressing life threatening emergencies
- Remove all clothing before examination
- Pay special attention to patient's back, axillae and perineum
- Examine oral cavity, nose, ears and scalp
- Digital rectal examination if necessary
- Investigate-essential radiology
  - X-ray chest, abdomen, cervical spine, pelvis, skull and other parts as and when required
  - Sonography (FAST),
  - CT brain for head injury patients.
- Avoid missing injuries and disabilities during secondary survey, since missed injuries are common in trauma patients, eg. Fracture phalanx or toe maybe missed in a case with head injury or multiple fractures.
- Plan for emergency surgery if necessary

Definitive Treatment phase

- Initiate appropriate treatment immediately depending on the nature of the injuries.
- Prioritize injuries - trauma surgeon coordinates and takes decisions
- Undertake simultaneous surgery/repair coordinating with all specialists concerned when multiple organs or systems are affected.
- Tetanus and antibiotic prophylaxis
- Pain relief
- Alleviation of anxiety and fear of the patient and relatives- an integral part of trauma care


Management of Trauma / Secondary survey
CNS
Level of consciousness
Pupil size and reaction
Xray skull
CT Brain
Raised ICT
Lateralizing signs
Burr hole/
Neuro surgical
intervention
Chest (CVS/RS)
Decreased air entry
Air leak
Subcutaneous -
emphysema
Chest Xray
CT chest
Pneumothorax
Bleed
ICD
ICD
Persistent
Airleak
>250ml/hr for 4 hrs
>1500ml intial
persistant hypotension
Thoracotomy
Abdomen
Guarding
Tenderness/Rigidity
Absent Bowel sounds
Xray abdomen
USG -FAST
CT abdomen
Hollow viscus injury
Solid organ injury
Bleed
Laparotomy
Musculoskeletal
system
Deformity
Restricted movements
Regional Xrays
Reduction /
Immobilization
Orthopaedic Intervention
ORIF if required
Peripheral
injuries
Wounds
External injuries
Bleeding
Exploration
Cleaning and
Primary suturing
Plastic Surgical
Intervention

Management of the trauma victim on arrival of the patient at casualty department

- Examine the pulse, BP, level of consciousness, orientation to time, place and person and general condition of the victim.
- Initiate life saving measures first
- Maintain airway, breathing and circulation
- Draw blood for investigations (blood grouping, cross matching, blood sugar etc.)
- Expose and examine wounds thoroughly
- IV access and fluid replacement should be initiated.
- Injection TT and dressing/suturing of obviously bleeding injuries.
- Transfer to emergency room with paramedics
- Record events:
  - * Record history and events simultaneously/ during treatment
  - * Record the pulse BP, Level of consciousness, orientation to time, place and person and general condition of the victim.
  - * Record the Date, Time, and by whom the patient was examined with name in capital letters and designation
  - * Record the date time, place and details of accident, and the person bringing the victim (relative/attendant/police)
  - * Site, size, depth, number and type of wounds should be recorded.
  - * If the victims breath smells of alcohol/under the influence of alcohol, it should be recorded.
  - * If referred from some other hospital/Nursing home/ clinic, the referral note should be attached and the fact should be noted in the accident register.
  - * Sign the Accident Register with the date and name in capital letters.
  - * Record transfer details and sign
  - * Inform RMO/higher authorities about VIPs or mass casualties

During Transfer

- Always accompany the patient during transfer
- Continue fluid resuscitation during transfer
- Hand over the patient to ER/OT staff
- Give details of injuries, treatment and outcome expected and alleviate anxiety of the patient and relatives wherever possible.
- Record the properties of the patient including the jewels worn, clothing and other belongings and hand over to authorities and inform police if no attender is available.

In the Emergency Ward

- Follow the trauma management guidelines
- Primary survey (ABCDE) and resuscitaion
- Maintain airway, breathing and circulation
- Record history with date and time, received time and examination time during resuscitation
- Mode and details of injuries should be noted.
- Life threatening emergencies should be attended first
- Secondary survey for detailed examination and to avoid missing injuries
- Prioritize injuries, coordinate and undertake surgery after resuscitative measures.
- Record clinical condition, treatment instituted, investigation results, expert opinions obtained in chronological order without and delay.
- Avoid overwriting and corrections
- Get consent from relatives, if not available from RMO/other medical officers.
- Undertake surgeries simultaneously in multiple injuries.
- Early surgery and repair of injuries/arrest of bleeding should be done depending on the nature of the injury
- Record the operative findings, procedure done and Post-operative condition of the patient.
- Record clinical condition at reasonable intervals in the Post-operative period
- Follow medico legal requirements and hospital rules at all times
- Inform patient's relatives about the condition of the patient at regular intervals and alleviate their fears.
- Issue the discharge certificate with required details and also issue and certificates required for legal acion

- Always sign the records with date and name in block letters.

Other Services

- Arrange transport for the victim while referring or during discharge (ambulance)
- Issue necessary certificates without delay (AR copy, wound certificate etc.)
- Discuss with the patient's relatives and provide psychological support throughout treatment
- Brain death certification and possible organ donation in brain death cases.
- Complete formalities expedetiously when the patient expires
- Provide psychological support to aggrieved family members
- Arrange post mortem in medicolegal cases
- Send the body to the mortuary without delay
- Explain procedures to be followed
- Handing over bodies to right relatives in case of dispute with help of police and revenue officials
- Help in embalming if transportation to a far off place (embalming is done by anatomy department after Postmortem by forensic medicine department
- Arrange transport (mortuary van)
- Inform social organizations for cremation and last rites if needed by relatives

Requirement for Transporting bodies by air

- Valid certification from authorities - hospital and police
- Embalming of the body and embalming certificate from professor of anatomy
- Needs special air tight coffins specially made for air lifting bodies

Cardio - Thoracic Trauma

Causes

- Blunt injuries
  - Road Traffic Accidents
  - Fall from height
  - Assault
  - Crush injuries
- Penetrating injuries
  - Stab injuries
  - Gunshot wounds
  - Missile injuries
- Chest wall injuries
  - Rib fractures
    - Simple
    - Multiple
    - Flail chest
  - Sternal fracture
  - Shoulder Girdle fracture
  - Thoracic Spine Injury
- Pleuro Pulmonary Injuries
  - Pneumothorax
    - Simple pneumothorax
    - Tension pneumothorax
  - Haemothorax
  - Lung contusion
  - Lung laceration
- Mediastnal Injuries
  - Cardiac Tamponade
  - Aorta, large blood vessel injury
  - Tracheo – bronchial injury

Chest wall injuries

Rib fracture

Symptoms

- Chestpain
- Dyspnea
- Tachycardia
- Cyanosis

Investigations

- Chest X-ray PA view-
- To detect fracture site, number of ribs involved to diagnose associated pneumothorax, hemothorax

Treatment

- Adequate Analgesia
- Chest Physiotherapy
- Intercostal drainage in case of pneumo or hemothorax

Sternal fracture

Symptoms

- Chest pain
- Dyspnea
- Tachycardia
- Hypotension if underlying cardiac injury

Investigations

- Chest X-ray Lateral view
- To detect fracture site, displacement
- CT chest for definitive diagnosis

Treatment

- Adequate Analgesia
- Isolated sternal Fracture: If chest X-ray, ECG are normal and if patient is stable,patient can be discharged.

Shoulder Girdle Injury

Symptoms

- Shoulder pain
- Deformity
- Tachycardia and Hypotension if Underlying subclavian /axillary injury

Investigations

- Chest X-ray PA view
- To detect fracture site, involvement of ribs, diagnose associated pneumothorax.
- Vascular opinion, if distal pulses not felt.

Treatment

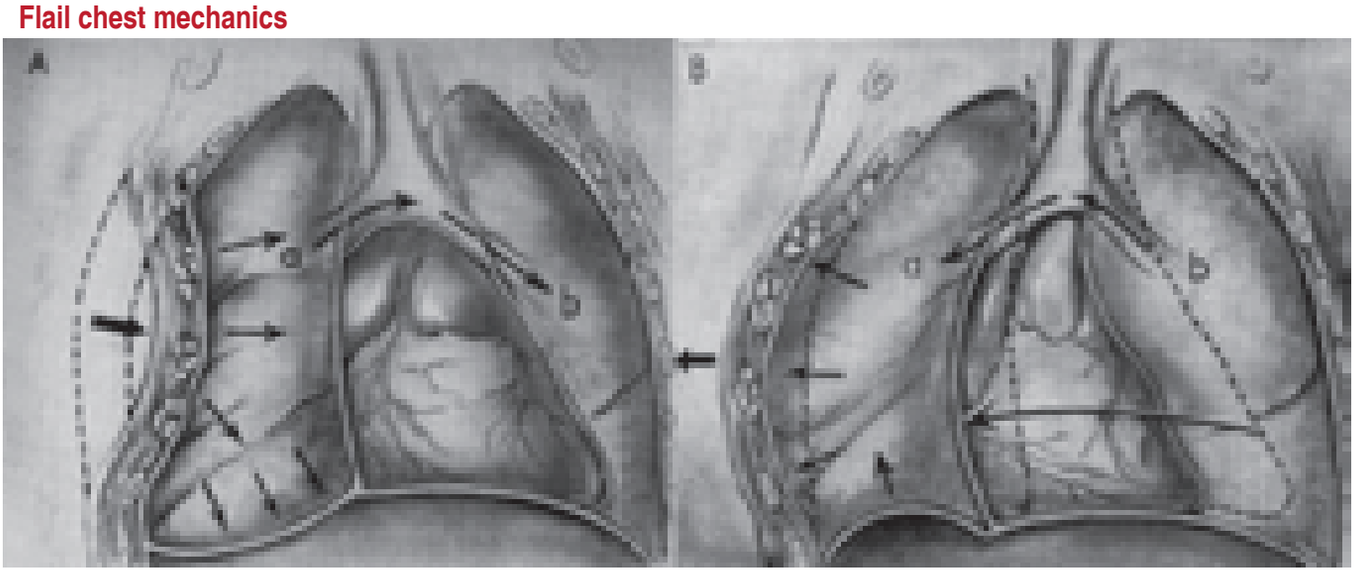
- Refer for ortho consultation after immobilizing and ruling out life threatening associated Injuries

Thoracic Spine Injury

Symptoms

- Back pain
- Weakness of both lower limbs,

Flail chest mechanics



- Neuorgenic shock (don't overload with fluids, give vasopressors), asso.
- Associated rib fractures.

Investigations

- X-ray DL spine AP / lateral.
- To diagnose fractures, dislocation, associated rib fractures.
- MRI spine-to assess extent of spinal cord injury

Treatment

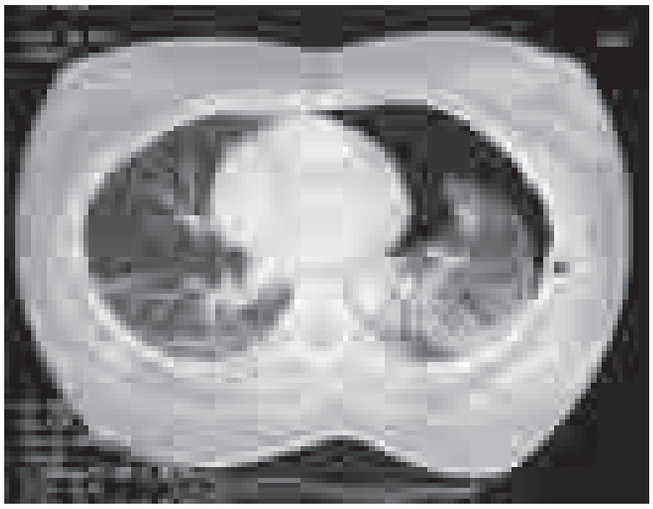
- Rule out cervical spine injury,tension pneumothorax.
- In primary care centre-immobilize spine and referral to tertiary centre for Orthopedic consultation

Pleuro - Pulmonary injuries

Symptoms

- Any of the above injuries I may occur alone or co-exist with other chest and abdominal injuries.
  - Chest pain
  - Dyspnea
  - Tachycardia
  - Cyanosis.
- In Tension Pneumothorax:
  - Hypotension
  - Cyanosis
  - Tracheal hyper resonance.

Pneumothorax - CT



- Persistent hypoxia with low SaO2, low pO2: suspect pulmonary contusion

Pneumothorax

Investigations

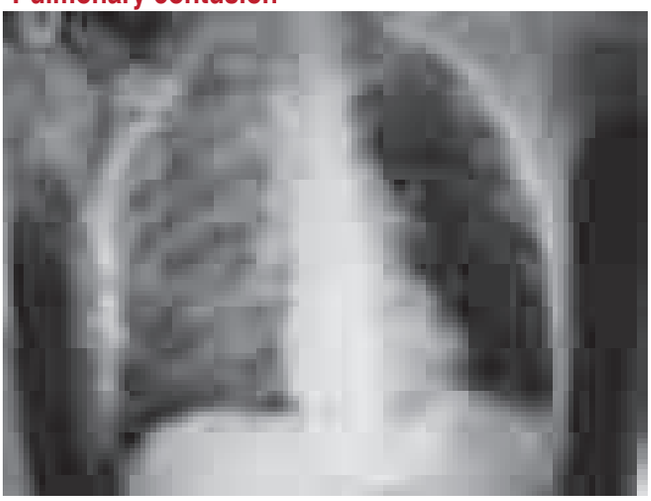
- Chest X-ray PA view
  - To identify collapsed lung borders
  - Hemothorax
  - Tension pneumothorax (look for mediastinal shift)

Treatment

- Urgent Needle decompression in 2nd inter costal space in mid-clavicular line in tension Pneumothorax
- ICD for Pneumo - hemothorax

Spontaneous Pneumothorax types

Pulmonary contusion



Hemothorax

Investigations

- Chest X-ray PA view-Irregular opacity in the pleural cavity if significant collection.
- CT Chest-sensitive for minimal hemothorax.

Treatment

- Intercostal Drainage to drain the blood
- Emergency Thoracotomy
- Initial drain of 1500ml or persistent drain > 200ml / hr.

Pulmonary contusion

Investigations

- Suspect in any chest trauma with persistent hypoxia.
- Chest X-ray PA view - less sensitive, patchy homogenous opacity in any lung zones
- CT Chest - definitive for extent or site of contusion.

Treatment

- Intubation and mechanical ventilation-key to management
- Antibiotics to prevent infection.
- Intercostal drainage, if hemothorax is present.

Mediastinal injuries

Cardiac Tamponade

Symptoms and signs

- Hypotension
- Muffled Heart sounds
- Distended neck veins
- Elevated JVP

Investigations

- Chest X-ray: Widening of cardiac silhouette
- Ultrasound: Blood in pericardial cavity
- ECG: less sensitive, low voltage QRS, ST-T changes.

Treatment

- Fluid resuscitation
- Pericardiocentesis under Ultra sound guidance
- Direct Surgical Decompression: ensures clot removal and complete hemostasis.

Cardiac and Aortic injury

Symptoms

Cardiac contusion can vary from clinically occult and transient cardiac depression to fatal rupture, arrhythmias, LV failure

Mortality 80-90%

Investigations

Diagnosis considered in all patients sustaining decelerating injury or sternal impaction. 2D ECHO is the best method.

Treatment

- Fluid resuscitation
- Pericardiocentesis under Ultrasound guidance.
- Direct Surgical Decompression: ensures clot removal and complete hemostasis.

Tracheo - bronchial injury

Symptoms

- Hemoptysis
- Dyspnea
- Subcutaneous and mediastinal emphysema
- Pneumothorax and air leak.

Investigations

- Chest X-ray: Pneumomediastinum, Pneumothorax, Pleural effusion.
- Bronchoscopy: important in diagnosis and in selective endobronchial intubation.

Treatment

Early surgical correction is needed and immediate referral to a specialist, once vitals are stabilized. It is important to maintain ventilation in the waiting period by using endobronchial tubes or high frequency jet ventilation.

Principles of surgery

"The surgeon should have an eagle's eye, a lion's heart and lady's fingers"

The aim of surgery is to cure patients and provide relief from pain and disabilities.

The major principle is to make surgery safe and beneficial to patients

The safety and success of surgery depend on the following

- Early correct diagnosis
- Thorough preoperative assessment and good preparation
- Smooth conduct of anaesthesia
- Correct surgical technique
- Intensive Post-operative care
- Anticipating complication (s) and early intervention (s)
- Continued follow up and rehabilitation
- Dedicated team

Early correct diagnosis and decision making

- Confirm clinical diagnosis by investigations
- Observe continuously and examine frequently to diagnose and to understand the progression or deterioration of the patient's condition.
- Offer the best option (beneficence), when multiple modalities of treatment are available (surgical and non-surgical).
- Discuss the medical problem with the patient/ relatives/ family physician. Always respect the patient's preference (autonomy) in the treatment
- Weigh the course of the disease and the outcome of surgery, benefits and complications of surgery before planning a procedure
- Prefer minimal access, endo surgery and non-invasive procedures wherever possible for the convenience and rapid recovery of the patient

Preoperative assessment and correction of co-morbidities

- Examine the patient completely to diagnose the problem and assess the physical condition for anaesthetic fitness and surgery.
- Assess the condition of the heart, lung, kidney and CNS functions.
- Treat co-morbid conditions (diabetes, hypertension, cardiac and pulmonary ailments etc) and bring the patient to near normal condition (fluid and electrolyte abnormality, treatment of septicemia etc) before anaesthesia and surgery.

Investigations

- Assess the physical condition by necessary investigations to correct any abnormality
- Record the date of investigations and results in the case sheet.
- Avoid unnecessary and repeated investigations.

Good preoperative preparation

- Prepare the patient physically and mentally for anaesthesia and surgery.
- Plan preoperative strategy, timing of surgery, selection of team and Post-operative requirement of instruments,equipments,ventilators,blood, intensive care, etc.
- Discuss the procedure, the problems expected and the outcome of the procedure before surgery with the anesthetist and the surgical team and be prepared to tackle the unexpected on table
- Discuss frankly about the problem, procedure and explain the possible risks involved in surgery and anaesthesia with the patient/relatives and obtain a detailed informed consent
- Write preoperative instructions clearly in the case sheet with the provisional diagnosis, the surgical procedure planned and sign.

Operation theatre principles

- Maintain theatre discipline strictly
- Practice aseptic techniques always

- Administer premedication preferably in the theatre under the supervision of the Anesthetist.
- Reassure the patient again when he /she is shifted to the operation table.
- Reconfirm the correct patient, the diagnosis and the site of lesion before giving anaesthesia and surgery
- Position the patient on the table properly before washing up.

Correct surgical technique

- Maintain strict asepsis throughout the surgical procedure.
- Plan incision to give adequate exposure.
- Perform the correct procedure required after assessing the condition on the table.
- Handle tissues gently, use diathermy to the minimum.
- Select proper suture materials.
- Close wound in layers, place drains if needed.
- Keep blood loss to the minimum.
- Avoid single unit transfusion, strictly follow blood transfusion norms.

Post-operative care

1. Immediate - In the operation theatre
   * Ensure complete recovery of the patient from anaesthesia.
   * Record the operative procedures in detail and the condition of the patient before leaving the operation theatre
   * Write Post-operative instructions clearly.
   * Send tissues/biopsy material to histo pathological and microbiological examination after proper labelling and writing requisition clearly with proper clinical picture.
2. In the Post-operative ward
   * Monitor closely during the Post-operative period.
   * Maintain fluid, electrolyte balance and glycemic control.
   * Anticipate problems and intervene if required.
   * Care wound and drains properly.
   * Maintain psychological support to the patient by the surgical team, staff nurses, counselors, social workers and the relatives throughout the hospital stay.

Follow-up and rehabilitation

- Follow-up and rehabilitation is essential in case of mutilating surgeries, amputations and in elderly.
- Refer patients to higher centers for possible reconstructive/cosmetic surgery, to enable them to lead an independent and normal life.
- Rehabilitate / provide artificial limb for amputees to make them independent.
- Explain the available welfare measures offered to them by governmental and non-governmental organizations.

Important Note

- Weigh the benefits, complications and natural course of the disease before embarking on surgery.
- Avoid unnecessary surgery.
- Provide emergency care always and institute life-saving measures immediately.
- Refer patients to specialists / higher center for minimal access surgery/non invasive treatment if available / required.
- Ensure safety in emergency surgery and do the minimum necessary procedure.
- Be prepared for the unexpected in surgery all the time.
- Follow medical ethics, moral values and legal formalities strictly.
- Do good and do no harm, always treat patients with tender loving care.

"All surgeons know how to operate, a good surgeon knows when to operate, but a great surgeon knows When not to operate.

Examination of surgical patients

- Elicit complete history before proceeding to examine the surgical patient.
- Examine the patient completely from head to toe.
- Physical examination includes General and Local examination.
- Record findings with date and time in the case sheet and sign.

- A good history may lead to the diagnosis in most of the patients or it may direct the surgeon to the diseased organ or system

General assessment of the surgical patient

Mental status

Record mental status (level of consciousness) in all patients, especially in geriatric, neurological, head injury and in medico-legal patients

Five stages of level of consciousness:

3. Conscious with orientation to time, place and person.
4. Conscious but not oriented
5. Drowsy (semi consciousness).
6. Stupor (unconscious), responds to painful stimuli.
7. Coma (unconscious), no response to painful stimuli.

Build and state of nutrition:

Build and state of nutrition of the patient will give clue to the

Clinical diagnosis

Cachexia in malignancy.

Chronic nature of the disease.

Requirement of preoperative nutritional support (wound healing).

Nutritional status includes hydration.

Facies

By looking at the face the mental state and intelligence, the severity of pain, diseases and effect of treatment can also be gauged. There are some typical facies such as:

'Facies hippocratica' in generalized peritonitis

'Risus sardonicus' in tetanus

'Adenoid facies' in hypertrophied adenoids

Attitude and decubitus

The attitude and position of the patient in bed is informatory.

Example:

The patient with cerebral irritation lies curled up on his side away from light.

Peritonitis patients with pain lie still.

Colicky pain patients become restless and toss on the bed.

Meningitis of the neck will show neck retraction and rigidity.

Fracture neck of femur, the lower limb may be lying in helpless eversion.

Vital Signs

Pulse

Indicates cardiovascular condition and severity of illness

Examine all peripheral pulses for

- Rate – fast,eg: thyrotoxicosis and ap pendicitis slow thready pulse, eg. hypotension and shock
- Rhythm – regular or irregular, in cases of atrial fibrillation
- Condition of arterial wall – thickening (e.g) arteriosclerosis etc.

Respiration

Type:

- Thoracic- in females, massive ascitis, huge abdominal mass etc.
  - * Abdominothoracic – in males

Rate:

Tachypnoea - Fast breathing eg: in fever, shock, hypoxia,

* cerebral disturbances, metabolic acidosis, tetany, hysteria etc.
* Slow and deep respiration is an ominous sign in cerebral compression.
* Dyspnoea (difficulty in breathing) is an ominous sign and has to be attended first before
* attempting to do a complete examination

Temperature:

Normally taken in the mouth, axilla or rectum Rectal or core temperature is usually 1 F more than the peripheral, axillary temperature

Post-operative temperature is an important parameter.

Fever is an early indicator of sepsis

Local examination

"Eyes do not see what the mind does not know"

Inspection

- Make the patient comfortable and examine completely.

- Ensure adequate privacy and expose the parts to be examined.
- Observe the patient in good light, preferably in daylight.
- Examine the female patients in the presence of a female staff nurse.
- Compare with the corresponding normal side wherever possible.
- Postpone detailed examination in acutely and severely ill patients.
- Look for any abnormalities such as presence of swellings, ulcers, sinus, scars, engorged veins, pigmentation etc specially look for pulsations and peristalsis in the abdomen

Palpation

- Confirm the inspection findings
- Feel for the warmth and tenderness
- Palpate gently when the lesion is tender
- Follow detailed and methodical palpation.

Percussion

To find out the presence of gas, fluid in the abdomen (ascitis, effusion, perforated hollow viscus) To differentiate between a solid or hollow viscus (liver, stomach, colon) To access the enlargement of solid organs (upward enlargement of liver)

Auscultation

Auscultate chest and abdomen to make out abnormalities in heart and breath sounds and the presence of peristalsis, murmurs, bruits etc.

Movements

Record range of movement and abnormal movements in orthopaedic cases and in nerve injuries

Measurements

Record measurements of abdominal girth in intestinal obstruction (progression) swellings, ulcers (reduction of size during treatment) injuries in medicolegal cases (length, breadth depth, site, nature) fractures in orthopedic cases (reduction of limb length)

Important Note

Local examination is never complete without the examination of the draining lymph nodes and the examination of the draining area is important when there is lymph node enlargement. eg; oral examination in a case of cervical adenitis.

General examination

In chronic cases, examine the patient as a whole, after completing the local examination. General examination is required mainly for the following purposes:

- Arrive at a complete diagnosis
- Assess the physical condition and co-morbid conditions
- Decide the treatment modality etc.
- Select the type of anaesthesia
- Determine the nature of the operation
- Determine the prognosis

1. Head and neck

   Examine

   * Eyes,
   * Oral cavity,
   * Cranial nerves,
   * Neck nodes,
   * Neck veins,
   * Carotid pulsations,
   * Position of trachea and neck movements.

2. Thorax

   Examine the type of chest,

   * Crowding of ribs,
   * Winging of scapula,
   * Kyphoscoliosis,
   * Chest movements,
   * Intercostal tenderness
   * Presence of any dilated vessels and pulsations,
   * Apex beat,
   * Heart and lungs,
   * Upward enlargement of the liver,
   * Obliteration of liver dullness.

3. Abdomen

   * Examine with patient lying comfortably in the couch fully exposed with hips and legs flexed and abdominal muscles relaxed and patient breathing easily.

* Inspect the type of abdomen: scaphoid-boat shaped (normal) or distended (generalized or localized)
* Abdominal wall – position of the umbilicus, presence of scars, dilated vessels, abdominal reflexes, visible peristalsis or pulsation.
* Hernial orifices, lymph nodes.
* Abdominal examination is incomplete without the examination of scrotum, perineum, inguinal region, back and rectal examination.

4. Examine the back including the spinal column
5. Gynaecological examination should be done in female patients
6. Upper and lower limbs
   * General examination of the arms and hand with particular reference to their vascular supply and nerve supply (power, tone, reflexes and sensations), axillae and lymph nodes, joints, finger nails – clubbing or koilonychia
   * General Examination of legs and feet – with particular reference to the vascular supply and nerve supply (Power, tone, reflexes and sensation), varicose vein, edema, joints.
7. Other examinations:

Examine Sputum, vomit, urine, and stool by naked eye and under microscope, if required.

Physical findings in relation to disease condition and risk

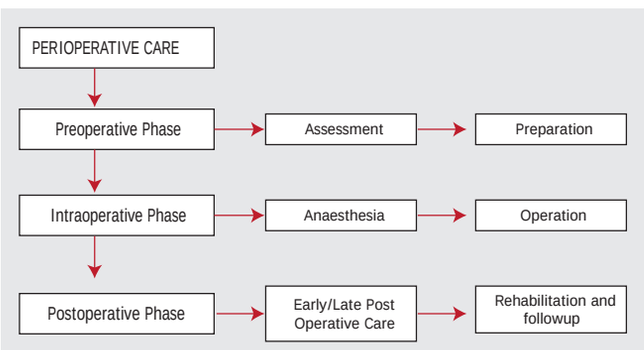
Organ / System	Finding	Disease/Condition	Risk
Eyes	Jaundice	Liver disease	Bleeding, encephalopathy
Skin	Pallor Cyanosis (peripheral and central) Lesions Scars Turgor	Anemia Poor perfusion, Frost bite Dermatosis Prior Surgery Reduced, lost	Heart failure Ischemia Infection adhesion, altered anatomy, dehydration
Nails	Clubbing Cyanosis Koilonychia	Lung disease Lung disease Anemia	Respiratory reserve reduced Heart failure
Heart	Hypertension Hypotension Bradyarrhythmia Tachyarrhythmia Murmur Bruit	Multiple etiologies Multiple etiologies Coronary artery disease CAD, Hyperthyroidism Valvular disease, Atherosclerosis	Heart failure
Lung	Rales Wheeze	CHF, COPD, Pulmonary infection COPD, asthma	Respiratory reserve reduced
Abdomen	Hepatomegaly Mass Lymphadenopathy	Liver disease, cancer, infection	Encephalopathy Delayed healing
Musculo-skeletal	Stiff neck Restriction of movements	Arthritis Arthritis	Immobility Increased falls
Gait	Unsteady waddling Trendelenberg	Alcoholism Bilateral dislocation of hip coxavera muscle dystrophies, polio, perthe's arthritis	Increased fall
Nervous	Sensory impairment Focal deficits	Multiple etiologies CVA, others	Injury Paralysis, convulsion

Important Note

- Undertake life-saving measures first before complete examination in emergency.
- Modify the order of examination in children /elderly / in emergency
- Explain and get patient's permission before physical examination (rectal, vaginal Examination)
- Always examine the female patient in the presence of a staff nurse and ensure privacy
- Never forget to peruse the old records or examine the patient completely including the draining lymph nodes, rectal examination etc

Preoperative assessment and preparation

Perioperative care is a term which encompasses three main components of surgical management, which are interlinked and to be followed step by step meticulously to achieve the best results following surgery.



Preoperative assessment

Preoperative assessment is done to:

- Evaluate the physical status of the patient
- Identify the co-morbid conditions
- Plan treatment modality, assess operability
- Optimise patient's condition preoperatively

Preoperative preparation depends on the following:

* The diagnosis (condition, stage of the diseases)
* Proposed surgical intervention and nature (elective/emergency, major/minor)
* Patient's health, associated risk factors and patient's preferences

Preoperative assessment consists of the following

- Comprehensive history including medication review
- Physical examination
- Nutritional assessment
- Surgical risk assessment and decision making

History and medication review

- Elicit thorough history from the patient in his own words.
- Elicit history from mothers for children and from relatives/caregivers for elders.
- Record presenting complaints and the onset of symptoms and progression of the disease in chronological order.
- Elicit personal history which includes smoking, alcoholism, previous illnesses like recent myocardial infarction, pulmonary tuberculosis, COPD, seizures, jaundice, AIDS, cancer, diabetes, previous surgery and drug allergies.
- Obtain list of medications, over-the-counter drugs, herbal preparations and ingestion of aspirin, NSAID, diuretics, oral hypoglycemic agents, sedatives, etc

Physical Examination

- Assess the physical condition and identify co-morbid conditions.
- Focus on detailed, methodical examination, inspection, palpation, percussion, auscultation, mobility and fixity etc, to arrive at the anatomical diagnosis.
- Proceed to find out the pathological diagnosis such as congenital, inflammatory, neoplastic or degenerative, metabolic or hormonal pathology.
- Continuous/frequent serial observation over a period of time, is sometimes helpful in achieving the diagnosis.
- Examine regional lymph nodes for any enlargement. When lymph nodes are enlarged, draining area should be examined and examination of the lesion is not complete without examination of regional lymph nodes.
- Identify Cardiovascular or respiratory signs such as tachycardia, hypertension, JVP elevation, ar-

rhythmias, murmurs, S3 gallop, edema, cyanosis, rales and rhonchi, bronchospasm and chest deformity

Nutritional Assessment and Preparation :

- Assess the state of nutrition clinically (Body Mass Index) and biochemically.
- Assess fluid status along with the nutritional status.
- Estimate serum albumin level (> 6mg/dl normal).
- Poor nutrition causes poor wound healing (wound dehiscence, infection, development of pressure sores, weakness and loss of function).
- Consider paranteral nutritional supplementation. TPN is useful in gastric outlet obstruction, pancreatitis, alcoholism, malnutrition and Post-operative patients. Parentral nutrition is costly.
- Prefer enteric route as soon as possible to prevent Gut Bacterial Translocation

Cognitive and Functional Assessment

- Record the preoperative level of activity and baseline mental status in the elderly/ neurological patients.
- Mental status allows the physician to recognize delirium.
- To predict potential problems and to plan interventions.
- Poor functional status carries a high surgical risk.

Investigation

- To confirm the diagnosis
- To assess the physical condition of the patient for fitness for anaesthesia and surgery.
- Assess the necessity, benefits, complications and cost of investigation
- Avoid unnecessary and repeated investigations.
- Biochemical -blood urea and sugar; serum creatinine and lipid profile
- Clinical pathological complete urine analysis and blood count
- Radiological -X-ray chest PA view and ultra-sonogram abdomen
- Cardiological -ECG

Special investigations may be carried out depending upon the patients' condition and indications

* For diagnosis: endoscopy, barium meal, enema studies, arteriography, FNAC
* To assess the status of cardiovascular, respiratory, neurological, renal, hepatic and thyroid functions
* For major and high-risk surgery e.g echocardiography, angiography, CT, MRI, ultrasonogram, ABG enzyme analysis etc,.
* Study the investigation reports and correlate with the clinical picture of the patient and write the remarks in the medical record before ordering further investigation or initiating treatment

Diagnosis

Preoperative diagnosis and planning is important for the smooth conduct of surgery.

In emergency, life-saving situations, exploration and restoration of vital functions are more important. eg: head injuries, thoraco abdominal injuries, then peripheral and skeletal injuries

Tissues removed should be sent for histopathological examination properly preserved and labeled for pathological diagnosis.

Surgical risk assessment and decision making

- The benefit of surgery should be weighed against the possible complications and risks.
- Surgical risk assessment also includes the anaesthetic risk.
- Anesthetist should assess the surgical patient preoperatively and his opinion and suggestions should be carried out to bring the patient to near normal levels pre- operatively.

American society of anesthesiologist (ASA) physical status classification has been used successfully to stratify operative risk.

ASA classification

1. Normal healthy patients
2. Patient with mild systemic disease
3. Patient with severe systemic disease that limits activity but it is not incapacitating
4. Patient has incapacitating disease that is a constant threat to life
5. Moribund patient not expected to survive 24

hours with or without an operation

Cardiovascular system

- The capacity to increase the cardiac output in response to intra and Post-operative challenges is a fundamental determinant of survival of the patient.
- Acute fall in ventricular preload leads to hypotension; acute increase leads to ventricular and pulmonary congestion and should be avoided.

High risk patients

- History of ischemic heart disease, congestive cardiac failure, cerebrovascular disease, thromboembolism, and hyperlipidemia
- Preoperative serum creatinine higher than 2 mg /dl

  Preoperative treatment with insulin

Peri-operative strategies

- Complete cardiac work up: ECG, ECHO, stress test.
- Cardiologist opinion and treatment.
- Wait for 4 to 6 weeks after myocardial infarction for elective surgery.
- Stop anti-platelet drugs one week prior to surgery.
- Beta-blocker and aspirin significantly reduces mortality and morbidity in cardiac patient.

Respiratory system

Evaluate the pulmonary functions for thoracic, abdominal and major surgeries.

Investigations

Necessary tests include forced expiratory volume at one second (FEV1) the forced vital capacity and the diffusing capacity of carbon monoxide.

Risk factors for Post-operative pulmonary complications:

- Thoracic, upper abdominal surgery and major surgeries.
- Preoperative history of COPD, purulent productive cough, cigarette smoking.
- Anaesthesia time more than 3 hrs.
- Geriatric, obese and hypoproteinemic patients.

Perioperative strategies

- Stop smoking
- Pre and Post-operative chest physiotherapy
- Vigorous pulmonary toilette and rehabilitation
- Continue bronchodilator therapy
- Use of epidural anaesthesia

Renal system

- Identify cardio vascular, circulatory, haematologic and metabolic derangements secondary to renal dysfunction
- Treat anaemia with erythropoietin.
- Evaluate coagulation - uremia may trigger coagulopathies.
- Preoperative dialysis for chronic end-stage renal disease patients.
- Avoid nephrotoxic agents and maintain adequate intravascular volume to prevent secondary renal insult
- Use narcotics for Post-operative pain control may have a prolonged effect in spite of hepatic clearance in patients with renal impairment.

Hepatobiliary system

Investigation for liver dysfunction

- Serum albumin
- Prothrombin
- Fibrinogen
- CBC
- Serum electrolytes

Malnutrition is common in cirrhotic patients; advice appropriate enteral supplementation.

Endocrine system

- Patients with diabetes mellitus, hyper or hypothyroidism, or adrenal insufficiency have increased physiological stress during anaesthesia and surgery
- Identify complication of diabetes mellitus and maintain glycemic status adequately
- Estimate blood sugar two hours prior to surgery

- Switch over to insulin from oral hypoglycemic drugs for better glycemic control
- Supplment steroid for a presumed abnormal adrenal response to periperative stress in case of steroid use (eg: prednisolone< 5 mg).
- Identify type and degree of endocrine dys function and treat accordingly

Vulnerable groups	Expected problems
• Children • Pregnancy • Immuno compromised • Elderly	• Hypothermia,blood loss and fluid imbalance • Abortion, drugs crossing placental barrier • Florid infection, poor wound healing • Loss of functional reserve and complications

Central nervous system

CNS problesm such as dementia and delirium are associated with poor prognosis special consideration should be given to vulnerable groups

Preoperative check list:

- Consent for surgery

  Record the consent in the casesheet which represents the result of discussion (s) with the patient and family members regarding the risks and benefits of the proposed surgery.

- Get consent preferably written and signed by the patient after making him understand the implications of anaesthetic and surgical risks
- Counseling

  The surgeon should gain the confidence of the patient with his kind approach and frank discussion on the problem, and possible benefits and risks especially in cases involving amputation or possible disability or disfigurement. Preoperative counseling by the doctors, trained staff, social workers and patients who have undergone mutilating surgery such as mastectomy or colostomy, will prevent or reduce depressive effect.

- Prevention of respiratory complications

  Cessation of smoking

  Reducing secretions

  Treating bronchospasm

  Chest physiotherapy

- Aspiration prevention

  Prevention of aspiration is the most important aspect of perioperative care Starving the patient for 6-8 hours prior to surgery Naso gastric aspiration (Ryle's tube) during surgery In emergency, stomach contents to be emptied by aspiration before giving anaesthesia

- Preparation of bowel

  GIT surgery needs complete evacuation and cleansing of alimentary tract. Sterilization of the bowel by oral anti microbial agents should not be done routinely. Routine nasogastric tube aspiration and strong purgatives, enemas are not indicated in elders, since it produces dehydration and exhaustion

Others:

- Blood grouping and Rh typing: Blood grouping and Rh typing should be done; reserve necessary units of blood for possible requirement.
- Sleep: Good sleep should be ensured on the night before surgery (mild sedation)
- Skin preparation: Skin preparation of local area viz., haircut, shaving of local parts should be done, taking care not to injure the skin.
- Bath: Patient should be given a good bath before surgery and draped in operation theatre clothing, cap and overshoes
- Bladder catheterization: Insertion of urinary catheter to prevent Post-operative distension of the bladder and to measure the urine output during surgery are important in major surgeries and especially in elders.
- Pre-medication: Routine pre-medication for anaesthesia is best avoided in the ward and is given in the operation theater under the direct supervision of the anesthetist.

Prophylactic measures:

- Antibiotic propylaxis: Prophylactic antibiotics are essential in elders undergoing valvular surgery with risk of endocarditis, oral, bowel, biliary, pulmonary and urological procedures.
- Immunization: Tetanus immunization by tetanus toxoid injection may be given during the

first consultation to allow time to develop active immunity. It is essential to give inj. Tetanus immunoglobulin 500 units intra muscularly before surgery to achieve passive immunity, especially in trauma cases, road traffic accident injuries and in emergency surgery.

- Thromboembolism: This complication is very common among the elderly especially in caucasians. To prevent thromboembolism, measures such as pneumatic compressive stockings, exercises, early ambulation and anticoagulant therapy are instituted.

Important Note

- Write preoperative orders clearly in the case sheet indicating the diagnosis, the site and side of lesion, blood group, procedure planned and sign.
- Reconfirm the correct patient, diagnosis, and side of the lesion before putting the patient on the operation table and before giving anaesthesia.
- Operation will proceed smoothly if the preparation is good and expected findings are present on the table.

Preoperative assessment and preparation

Preoperative assessment	Preoperative preparation
Complete history chief complaints h/o present illness	Investigation for the diagnosis and co-morbid conditions. assurance, counseling
Past history; Previous medical illness / surgery DM, HT, IHD, laparotomy, hernia)	Treatment of co-morbid conditions. Improve cardiovascular, respiratory functions Control diabetes and hypertension
Medication review non-prescription OTC drugs herbal and native medicines NSAIDs, aspirin, OHA, sedatives H/o drug allergies.	Stop unnecessary OTC drugs, aspirin, and oral hypoglycemic drugs before surgery.
Personal history habits, attitudes, beliefs and life style. H/o smoking and alcoholism.	Stop smoking and alcohol intake. Exercise and chest physiotherapy to improve respiratory reserve.
Family and caregiver history Availability of family support and caregivers, Stress factors	Detailed discussion with patient and their family, (and referring physician) about the diseases, procedures planned and its complications and expected outcome. Ascertain patient's wishes and preference (s). Get advance directives. Informed consent for the procedure.
Physical and general examination Lymphadenopathy, ascities, edema CVS, RS, CNS, abdomen, scrotum and perineum.	Assess general condition, operability, fitness for anaesthesia. Chest physiotherapy and counseling
Cognitive and functional assessment	Baseline assessment of cognitive function and functional level to be recorded
Nutritional assessment	Under nourishment, hypoproteinemia, anaemia, dehydration and electrolytes imbalances to be corrected. Parenteral nutrition in conditions such as gastric outlet obstruction, pancreatitis.
Surgical risk assessment including anaesthetic risk	The benefit of surgery to be weighed against the possible outcome and complications. Preoperative anaesthetic assessment and correction of deficiencies

Anaesthesia

One of the achievements of modern medicine is its ability to keep the patients free from pain through newer and more potent drugs and newer anaesthetic techniques. The patient should not only be free from pain, but also be safe during anaesthesia and surgery. Good anaesthesia should be safe with smooth induction, maintenance and quick reversal without producing any CVS, RS, and CNS complications.

The major risk factors

- Poor general condition and co-morbid condition
- Major / emergency surgery

Choice of anaesthesia depends on

The patient's general condition

- Nature of surgical procedure
- The experience of the anesthetist and familiarity with the procedures

Types of anaesthesia

General anaesthesia

Regional anaesthesia - spinal / epidural / local

Local / regional anaesthesia is given by way of infiltration, field or nerve blocks, and epidural or spinal, (safer option in selected cases.)

Local anaesthesia

Topical and infiltration

- Topical anaesthetic agents are used on the skin, urethra, nasal mucosa and cornea
- Infiltration anaesthesia for very small lesions / biopsies 1% lignocaine into / around the tissues to produce analgesia
- Easily administered
- Starvation not required
- Test dose should be given before infiltration
- Contraindicated in local infection and in coagulation disorders

Regional anaesthesia

Regional anaesthesia is commonly administered in elective surgeries. This involves blockade of major nerve trunks, which innervates the site of surgery.

The two types of regional anaesthesia are spinal and epidural,

In spinal anaesthesia the drug is injected into the subarachnoid space (intrathecal)

In epidural anaesthesia the drug is injected into epidural space.

Advantages

- Advantageous in debilitated patients
- Reduces bleeding, Post-operative respiratory problems and deep vein thrombosis
- Diminishes stress response and CNS complications
- Decreases convalescence time and facilitates early ambulation
- Minimizes requirements of Post-operative analgesia
- Reduces mortality

Disadvantages

- Technically difficult
- Epidural is less reliable
- Supplemental sedation (compensation for inadequate regional anaesthesia) carries risks eg: airway obstruction, pulmonary aspiration and agitation.

Single dose anaesthesia

A single dose epidural or spinal anaesthesia is used to provide short period of very effective analgesia during operation.

Continuous epidural anaesthesia

A sterile epidural catheter is inserted into the epidural space and anaesthetic agent is injected at regular intervals and analgesia provided for many hours or even days. This is particularly valuable in patients with poor respiratory functions in abdominal or thoracic surgeries.

General anaesthesia

GA is faster and reliable

Difficulty in elders- due to cervical spine problem and tracheal narrowing.

Disadvantages

- Difficult intubation
- Myocardial depression

- CNS complications

Causes

Potential causes of intraoperative instability

1. Hypotension,
2. Hypoxia,
3. Hypothermia,
4. Anaphylaxis
5. Malignant hyperthermia

Important Note

Anaphylaxsis

- Causative agents: muscle relaxants, latex, induction agents etomidate, thiopentone, propofol and narcotic agents
- Manifestation: cutaneous eruptions, hypotension, cardiovascular collapse, bronchospasm and death
- Treatment: In suspected cases Inj epinephrine 0. 3 to0.5ml of 1: 1000 sc
- In severe anaphylaxis give epinephrine 0.5 ml IV at 5 -10 minutes interval/ histamine blockade with diphenhydramine and hydrocortisone, fluid boluses, pressors, oro tracheal intubation ,nebulised B2agonists are given. Post-operative monitoring in intensive care

Malignant hyperthermia

Cause: hypermetabolism and muscle injury due to halogenated anaesthetic agent or succinyl choline

Manifestations: Increased sympathetic nervous system activity, muscular rigidity, high fever, hypercar bia, arrhythmia, hypoxaemia and rhabdomyolysis

Treatment: Discontinue the inhalation drug, administer dantrolene sodium 2 - 3 mg/kg IV.

Common causes of failure to breathe after general anaesthesia are:

- Obstruction of the airways, hypoxia or hypercarbia of any cause
- Central sedation from opioid drugs or anaesthetic agents
- Persistent neuromuscular blockade
- Pneumothorax from pleural damage during anaesthesia and surgery
- Circulatory failure leading to respiratory arrest.

Intra operative management

Monitoring of vital parameters

Monitor continuously the vital parameters- pulse, blood pressure, respiratory rate, ECG, oxygen saturation and urine output and immediate intervention done to prevent Post-operative complications.

Three factors such as hypoxia, hypothermia and hypotension are interlinked to each other and produce combined ill effects leading to life threatening complications and hence they need close monitoring. The details are described below

Hypoxia

Monitor continuously by pulse oximetry

Treatment

Administer intraoperative oxygen to prevent hypoxia during surgery. (PaO2 decreases with increasing age due to hypoventilation-pulmonary dysfunction and anaesthetic drugs such as opioids, muscle relaxants and CNS depressants).

Hypothermia

- Measure core temperature by trans-esophageal/ rectal thermometer.
- Prevent heat loss in the OT (body temperature is labile under general anaesthesia, patients are prone to hypothermia during surgery.)
- Cover the patients with sheets, blankets and thermal pads to prevent and treat hypothermia as there is heat loss in the operating room under general anaesthesia.

Hypotension

- Monitor blood pressure continuously
- Infuse fluids appropriately.
- Blood loss, anaesthetic drugs (profound vasodilatation) and muscle relaxant (fluid shifts to extra cellular space) leads to severe hypotension leading to irreversible shock.

Blood transfusions

- Keep blood loss to the minimum
- Avoid unnecessary and single unit transfusions

- Hypotension refractory to volume replacement with fluids require blood transfusion (>2litres of crystalloid)
- Hypoperfusion is an indication for transfusion even if there is no hypotension
- Acute blood loss, rapid loss of more than 20% of blood volume, Hb level of 6gms/dl or 6-9gms /dl with clinical evidence of hypotension and shock require blood
- Problems in blood transfusion includes availability, short shelf life, transmission of diseases and transfusion reactions.

Transfusion norms-

- Check blood grouping and Rh typing preoperatively and record in the case sheet
- Anticipate blood loss prior to surgery, and reserve/order required units of blood.
- Send blood samples immediately on drawing blood properly labeled with all the necessary particulars such as name, age, MRD number (hospital number)and blood group.
- Reconfirm patient's group on receipt of blood sample and donor blood and cross match and confirm compatibility.
- Use the fresh blood for better results (avoid blood stored for more than 21 days).
- Recheck the blood bag received in OT/ward again to reconfirm the correctness of blood group and the correct patient.
- Warm blood to room temperature before transfusion to prevent hypothermia.
- Check and record pulse, blood pressure and clinical condition and time of starting blood with the bag number group etc.
- Observe the patient closely till the transfusion is completed.
- Stop transfusion if the patient complains of
  - Dysponea
  - Hypotension
  - Severe abdominal pain
  - Hemorrhage
  - Hematuria.
- Administer inj calcium gluconate to avoid coagulopathies in multiple transfusion
- Consider blood components (FFP, platelets, fibrinogen concentrate)
- Watch for transfusion reactions (immediate / late).
- Record the clinical condition in the case record after completion.
- Keep the blood samples of donor and the recipient for a minimum of 3 days.

Operation theatre discipline

1. Maintain sterility of operation theatre at all times by regular and effective operation theatre washing, fumigation and autoclaving of instruments so as to avoid infection.
2. Allow minimum persons into the theatre
3. Prevent entry of persons if they have infection
4. Change completely to theatre dress
5. Wear cap and mask and OT shoes properly
6. Be clean – cut nails, cut hair and have bath. Bangles, chains, bindis, rings and flowers should not be worn
7. Do not touch any article with bare hands – use Cheatle's forceps or gloves
8. Avoid unnecessary movements
9. Avoid talking or noise
10. Check the instrument table before surgery and always inform the requirement of instruments to the theatre staff nurse in advance
11. Wash up after positioning the patient and do not leave it to your assistant.
12. Confirm proper positioning of the patient, adequate height of the table and proper air conditioning as well as lighting before surgery
13. Handle diathermy carefully to avoid inadvertent injury to other organs

Important Note

- Assessment and preparation of the patient is essential before anaesthesia and surgery.
- Enquire about allergy to drugs, previous illness, surgery and anaesthesia
- Discuss with the surgeon / patient / patient's relatives regarding the anaesthetic risk and complication and get written consent
- Examine oral cavity for loose tooth and dentures,

tracheal position and spine for mobility, disc space reduction and kyphoscoliosis.

- Ensure gastric emptying before induction of GA to prevent aspiration
- Maintain cardio respiratory, renal function and glycemic control
- Avoid hypoxia, hypotension and hypothermia.
- Reduce exposure to known risk factors for acute organ failure, especially acute renal failure.
- Initiate invasive monitoring if required
- Ensure quick reversal without CNS complication.
- Convert from spinal anaesthesia to general if required
- Continue monitoring and ventilate electively in Post-operative period to optimize recovery

Post-operative care

Numerous physiological and pharmacodynamic changes occur due to surgical trauma, anaesthesia (drugs used for induction, sedation, reversal and muscle relaxants) and in the immediate Post-operative period.

Management of patients after major surgery

- Monitor the cardio-respiratory function in high dependency unit (in high risk patients such as children, elders and following major surgeries) in the intra and Post-operative periods.
- Intervene and adjust therapy every few hours if optimum cardio-respiratory function is to be maintained.
- Expect and treat complications quickly
- Monitor the respiratory functions continuously and ventilate electively if required.

Early Post-operative period

Monitoring of vital parameters is essential

- Blood pressure, temperature, pulse, respiratory rate
- Fluid and electrolytes balance urine output (replace volume with appropriate fluids)
- Maintain glycemic control, liver and renal functions
- Care wound and drains
- Good nutritional intake and bowel movement

Hypoxia

Monitor continuously by pulse oximetry and intervene to prevent hypoxia. Abdominal pain and the effects of opioid drugs given to relieve Post-operative pain can depress respiratory function.

Rx Treatment

Administer oxygen to all elderly patients after surgery to prevent hypoxia. Post-operative hypoxia could persist for several days, especially after abdominal surgery, and also after hip surgery.

Oxygen administration at 2 litres / minute intermittently through twin nasal catheter is an effective way of preventing this hypoxia and is well tolerated by patients.

Hypothermia

There is heat loss in the operating room under general anaesthesia. The young and the older patients are often cold after lengthy procedures and rewarming will take a longer time.

Rx Treatment

It is necessary to cover the patient with warm blankets. In the cold patient, the drugs injected into cold muscles will not be absorbed into the circulation until the muscle warms and the vessels dilate. Hence the first dose of opioid analgesic should always be given intravenously and the intramuscular route reserved for the warm patient.

Hypotension

Anaesthetic drugs and muscle relaxants may lead to profound vasodilatation and lot of fluid is diverted to extra cellular space. The fluid loss is poorly tolerated in older patients and appropriate care and infusion should be started. Hypotension causes poor perfusion thereby leading to hypoxia. Continuous BP monitoring, pulse oximetry and appropriate fluid infusion (25to30ml/kg body weight) are essential.

Rx Treatment

Plasma volume is maintained adequately with appropriate intravenous fluids, blood and its components or plasma expanders. The metabolic response to surgical trauma leads to sodium and fluid retention Monitor to avoid fluid overloading. Fluid overload is hazardous due to poor cardiac function leading to right heart failure in the elderly.

Urine output

- Monitor urine output after major surgery
- Maintain output by ensuring adequate fluid volume initially
- Measure central venous pressure to check the adequacy of fluid replacement
- Restrict the use of indwelling catheters
- Insert bladder catheter aseptically
- Take necessary precautions if kept for prolonged periods

Orientation

Immediately after surgery, many patients are confused (Post-operative cognitive dysfunction). Such confusion lasts longer, if given long-acting sedative and amnesic drugs.

Post-operative analgesia

- Assess pain- ask the patient frequently about their pain level.
- Administer pain medication on a regular basis and whenever necessary.
- Continuous epidural anaesthesia is valuable in patients having abdominal or thoracic surgery with poor respiratory function.
- After minor surgery, oral soluble paracetamol can be given early for relief of less severe pain. Opioid drugs for severe Post-operative pain are best given initially intravenously in incremental doses until the desired effect is achieved. Adequate pain control decreases the Post-operative morbidity and reduces the hospital stay.

Care of drains and wound

Connect drains to proper and sterile containers and measure and record. After major surgery, drains are kept for draining the collected fluid, blood etc., the amount of drainage should be carefully monitored and appropriate fluids / blood replaced. In case of large quantities of drainage in a short time, or severe blood loss, the wound should be re-explored. Wound should be dressed properly and kept clean and dry, change of dressing should be done aseptically if required. Drain sites are to be properly covered with adequate sterile pads.

Late Post-operative period

1. Control of infection
   - Control infection with appropriate antibiotics, if required assess culture and sensitivity of discharge from the wound; or if respiratory or urinary infection is suspected, sputum and urine culture sensitivity.
   - Reduce hospital stay to eliminate the risk of opportunistic infection. Reduce Post-operative respiratory failure in case of upper abdominal surgeries.
2. Deep vein thrombosis (DVT) prophylaxis

   Deep vein thrombosis prophylaxis is advocated for major surgeries and orthopaedic surgery. Infusion of low molecular weight heparin has a vital role in prevention of deep vein thrombosis. Early ambulation reduces deep vein thrombosis.
3. Early mobilization

   Ambulate patient as early as possible to prevent problems like
   - Atelectasis and pneumonia
   - Orthostatic hypotension
   - Decreased cardiac output and stroke volume
   - Urinary retention
   - Negative nitrogen balance
   - Depression and sensory deprivation
   - Decrease tissue sensitivity to insulin
   - Deep vein thrombosis
   - Constipation and fecal impaction
   - Loss of muscle strength
   - Decubitus ulceration.

   Physiotherapy to improve respiratory function and limb exercises to improve muscle strength and movements
4. Prevention of pressure ulcers

   Prevention of pressure ulcers (Decubitus ulcers) is a critical part of Post-operative care
   - Turn the patient frequently in the bed to prevent the pressure ulcers.
   - Use of alpha beds and keeping the back dry as well as early mobilization prevents pressure ulcer.
5. Rehabilitation and follow up
   - Mobilize the patient early to prevent complications and educate patient and family members in the importance of mobilization and

physiotherapy.

* Provide psychological support by trained psychologist, medical social workers and community health workers in cases where no family support is available.
* Encourage the patient to do his /her own day-to-day activities especially in older people to prevent decline in functional status in the Post-operative period.
* Refer elders / patients for proper care and follow up after discharge. (Some may not have proper home or care giver or family members to take care of them; social service organizations and old age care homes may be identified)

Key points

- Monitor Post-operative patients intensively
- Expect complication and intervene early
- Prevent hypotension, hypoxia and infection
- Ambulate early and give physiotherapy

Wound care

"Skin is the best dressing"

Care of wound is the fundamental function of surgical practice from ancient times. Wounds occur in many ways, commonly due to trauma or deliberately made by the surgeons to gain access to deeper structures (incisions). Wound healing is a natural process and all types of wounds heal and the repair process is the same for different kinds of tissues and wounds.

Wounds are of different types and can be classified in many ways

- Acute or chronic, if it persists over 6 weeks
- Open and closed (skin surface not breeched as in blunt injuries)
- Clean or unclean (infected), etc

Traumatic wounds are further classified as:

- Abrasion (tangential force to skin, skin surface is rubbed off)
- Bruise, contusion, and hematoma (blunt injury, closed wound)
- Laceration (shearing force as in falls, road accidents, irregular, ragged edges and injuries with varying depth, damage to deeper structures depending on the force)
- Traction and avulsions. Degloving injuries are caused by shearing forces that separate tissue planes, skin and subcutaneous layers are separated from deep fascia in limbs
- Crush injuries occur due to severe force applied to the tissues in the closed space leading to tissue damage, bleeding exudates and swelling
- Penetrating wounds occur due to sharp objects such as knife, size of the wound may be small but the direction and depth of injury is important, requires thorough and careful exploration.

Chronic wounds

Ulcers

Definition

An ulcer is a discontinuity of an epithelial surface.

Classifications:

- Specific ulcers are defined as ulcers due to specific diseases such as tuberculosis or syphilis.
- Non-specific ulcers are defined as ulcers due to infection of wounds, physical or chemical agents or due to local irritation.
- Malignant ulcers are defined as ulcers due to malignant changes occurring in the layers of the skin e.g epithelioma and rodent ulcer.
- Arterial ulcers occur due to occlusion of blood supply and involve the deeper structures, deep fascia, muscle.
- Venous ulcers due to stasis of blood in the veins involve the skin, subcutaneous and superficial tissues. e.g varicose ulcer

Wound healing

- Inflammatory phase (reactive) limits the damage and further injury to tissues.
- Proliferative phase (repair/regenerative phase) matrix synthesis, neovascularisation and reepithelialisation of the wound.
- Maturation phase (Re-modeling) collagen cross-linking, shrinking and cross contraction of the wound takes place.

Causes of non-healing of ulcers

- Poor blood supply, oedema, chronic smoking
- Malnutrition, diabetes
- Immunosuppressants
- Bacterial contamination
- Non-specific infection turning to specific infection
- Neurological conditions
- Chronic irritation
- Malignant change

Management Guidelines

Acute wound care

- Minimize unnecessary blood loss
- Avoid the formation of a haematoma
- Adequate cleaning, debridement, edema control
- Remove debris and necrotic tissue, irrigate gently with water / normal saline
- Expose viable tissues, excise eschar by sharp mechanical debridement
- Use topical agents, (Chemical debridement) antiseptics (chlorhexidine, povidone iodine, alcohol, hydrogen peroxide, triclosan) antibacterials (silver sulfadiazine, neomycine, polymyxin, bacitracin, mupirocin) for sites, cavities that are difficult to access
- Apply dressings after complete debridement
- Maintain proper wound environment and prevent ischemia
- Keep the bacteria count as low as possible
- Facilitate healing of acute wound, by closure
- Delay closure for several days to allow infection to clear

Post repair wound care

- Apply non adherent dressing for 24 to 48 hours to ensure adequate epithelialization and to prevent contamination of the wound.
- Keep wounds clean and dry.
- Avoid soaking or scrubbing the wound. Gentle blotting of the wound with a towel is better than repetitive wiping of the wound.
- Avoid prophylactic parenteral antibiotics for routine laceration repair.
- Prepare and decontaminate wound to prevent infection rather than using antibiotics.
- Protect scars that are exposed to sunlight with sunscreen for at least 6 to 12 months to minimize subsequent hyperpigmentation.
- Cosmetic effect (good scar) depends on proper wound healing

Chronic wounds management

Identify and treat the predisposing factors

Causes

Diabetes mellitus

Peripheral arterial or venous disease

Severe anaemia

Protein deficiency

Rheumatoid arthritis

Systemic vasculitis

Cushing's syndrome

Systemic steroid therapy.

Treatment

Non-pharmacological

- Encourage daily bath; avoid walking bare foot or using slippers with interdigital bars.
- Reduce standing or excessive walking; wear elastic stockings. Elevation of leg and foot end of the bed while sleeping and leg exercises to activate the calf muscle pump in leg ulcers.

Pharmacological

- Identify the microorganism and treat accordingly.
- Send pus/discharge for culture and sensitivity (microbiology).
- Exhibit appropriate antibiotics. Tubercular ulcer is treated with antitubercular drug for a minimum period of 6 months.

Surgical Treatment

- Clean the wound with normal saline or tap water only (antiseptics delay wound healing).
- Surgical debridement for ulcers associated with necrotic tissue or slough.
- Daily dressing: commonly used dressings are: Occlusive(moisture retentive) dressings Hydrocolloid gel for clean and shallow ulcers Calcium alginate dressing for bleeding wounds

and wounds with a cavity

- Apply split skin graft for early closure of wound. (Chronic ulcers heal by fibrosis and scar formation takes long time to heal)
- Biopsy of the wound edge for pathological diagnosis, in chronic non healing ulcers
- Refer patients with chronic leg ulcer to vascular surgeons or general surgeon with some experience in peripheral vascular problems for surgical treatment

Patient Education

- Teach the care of leg ulcers, wearing socks and proper footwear and compression stocking to prevent ulcers in future
- Massage after bath the healed scar area with an emollient cream such as lanolin or some oil to keep the scar tissue soft and supple to prevent further breakdown.
- Regular use of calf muscle activating
- Chemical agents, enzymes to digest necrotic tissues
- Dressings
- Dressings that absorb moisture such as copolymer starch dressing (alginate)
- Zinc oxide to form a moisture barrier
- Hydro colloid dressing
- Antibiotics after culture and sensitivity, if infection is present
- Vacuum-assisted devices

Surgical management

- Excise dead and devitalized structures
- Cover wound by local/ myocutaneous/ composite/ rotation flaps adequately

General measures for prevention:

- Avoid prolonged lying down in supine position, frequent turning, and use of water or alpha bed
- Educate patients on prevention and care of ulcers

Important Note

- Prevention is the best form of management
- Educate patients / care givers on preventive strategies
- Avoid prolonged lying down in the supine position
- Avoid friction and shearing forces while turning the patient
- Avoid soiling of bed by urine, motion, sweat or discharges
- Turn patient frequently to prevent pressure effects
- Care of the skin, back, perineum and scrotum
- Implement proper bed making
- Use alpha bed/water bed if necessary
- Provide balanced nutrition
- Encourage early ambulation
- Exercises and leg elevation
- Avoid prolonged period of standing or sitting with legs down.
- Good personal hygiene (daily bath) and good nutrition.
- Control of diabetes is necessary.

Pressure ulcers

Definition

A pressure ulcer is defined as a lesion occurring over the bony prominence or on the firm surface, caused by unrelieved pressure, resulting in the damage of the underlying tissue.

Pressure ulcers are the costly complication of immobility and can be easily prevented. Healing of all three phases (extension, transition, and repair) of wound is delayed due to poor nutritional status, presence of neurological conditions and diabetes. In the very old and bedridden patients the problem assumes importance due to the difficulties encountered in the healing and its added complications.

Prevention is the best form of treatment

Management of pressure ulcers

1. Prevention of ulcers
2. Eliminate extrinsic factors
3. Prevention of further deterioration and promotion of healing of existing ulcers
   - Create and maintain proper environment (clean wound, adequate oxygenation)
   - Provide nutritional support
4. Surgical management

* Wound debridement
* Plastic surgical intervention

5. General treatment measure to promote healing
   * Wound cleaning (washing with soap and water, normal saline, avoid irritants)
   * Debridement (removal of dead tissue, eschar excision)
   * Mechanical removal by wet dressing, hydrotherapy

Sutures

Sutures

The purpose of suturing is to hold the tissues united till the natural process of repair and firm union between the cut edges is completed.

Definition

A ligature is a band or tie used around a ductular structure in order to constrict it like tying a blood vessel using catgut or silk.

There are different kinds of suture materials available and the surgeon is expected to select the right kind of suture material for the different types of tissue closure for best results.

An ideal suture or ligature should have the following qualities-

- Adequate tensile strength (tensile strength means minimum amount of tension (by weight) required to disrupt the suture materials. The higher the strength, the better is the material).
- Least tissue reaction
- Non-allergic, non-capillary, non-electrolytic and non-carcinogenic.
- Should produce a secure knot without cutting or fraying.
- Easy to sterilize, cheap, comfortable and handy

Classification of sutures

1. Absorbable suture
   * Natural: Plain and chromic catgut (prepared from sheep's gut) tensile strength depends upon the size of the material, plain catgut lasts upto 7 - 10 days and absorbed by enzymatic proteolysis. Bacterial infection leads to increased proteolysis and rapid absorption of sutures even before the complete union of tissues leading to wound dehiscence.
   * Synthetic: Polyglycolic Acid (Dexon), Polyglactin (Vicryl), Polydiaxanone (PDS) Approximately 60% remains at 2 weeks and 30% remains at 3 weeks. Degradation by hydrolysis is complete by around 60-90 days. Vicryl is an ideal suture material for medium term and widely used for bowel anastomosis.
   * Advantages Great tensile strength and little tissue reactivityEasily handled like silk and knots well
   * Absorbed in 60-90 days
2. Non-Absorbable sutures
   * Natural eg: silk, cotton, linen, metal (stainless steel)
   * Synthetic eg: dacron, nylon, polypropylene, proline

Suture needles

Suture needles are of two types:

Straight and curved needles

- Straight needles are used usually on the surface structures like skin or in bowel anastomosis
- Curved needles are used when suturing in the depth, abdominal and thoracic cavities and in vascular or other anastomosis. Curved needles are usually directed towards the surgeon while suturing.
- Cutting needle - Triangular in cross section. Cutting needles are mainly used to suture skin, aponeurosis, tough deep fascia etc.
- Round needle - Rounded in cross section. Mainly used in bowel anastomosis.
- Atraumatic sutures are special type of suture material in which the needles do not have the eye to thread suture material and the suture is swaged / impregnated into the needle. The diameter of the needle is nearly the same as that of the suture material to prevent/minimize needle puncture leakages. Atraumatic needles are used for vascular anastomosis, bowel anastomosis, subcutaneous tissues etc.

Principles of suturing

- All sutures act as foreign bodies and increase infection
- Use thin atraumatic sutures

- Avoid including large chunks of tissues in sutures or in the ligatures to prevent avascular necrosis and infection
- Suture with right degree of tension
- Prefer monofilament sutures instead of braided silk
- Metallic sutures are permanent and strongest while natural sutures are the weakest
- Synthetic materials have intermediate position and hence ideal choice
- Continuous sutures are as good as interrupted ones; breakage is rare if placed properly.

Good suturing technique:

- Keep the needle tip perpendicular to the skin at about 0.5 cms from the cut edge of the skin and pierce it to the required depth avoiding potential spaces (curved needle directed towards you and straight needle away)
- Turn the needle to the other side of the incision by circular movement of the wrist and the needle tip should come out perpendicular and equidistant from the incision on the other side
- Pull the needle along the curvature of the needle always
- Approximate the skin properly without any overlapping or in folding
- Place the knots on one side of the suture without tension and the thread should be cut less than the inter-knot distance

Skin:

- Usually interrupted silk sutures preferred
- Subcuticular stitch is ideal for skin closure results in thin scar
- Simple or mattress sutures depending on the thickness of the subcutaneous tissue, site and the surgeon's preference
- In malnourished and cancer patients, when delayed healing is expected the sutures are left longer, up to 2 weeks.
- Secondary suturing retained up to 2 weeks.
- If there is swelling, edema, collection or infections remove the particular suture immediately.
- Titanium clips can also be applied for skin closure. painful while removing clips, not very commonly used in general surgery.
- Steri-strips and adhesive glues are also used for better cosmetic results in skin closure.

Face - Monofilament thin non-absorbable simple sutures.

- Remove sutures by 4-5 days
- Heal well due to good blood supply.
- Trunk and Limbs - Preferably monofilament
- Removed around 7 – 10days
- Back- Sutures removed at around 10 days

Aponeurosis and ligments:

Non-absorbable strong monofilament like prolene is used eg: Rectus closure, inguinal hernia repair.

Muscles:

Muscle tissues are sutured by chromic catgut stiches. The first knot snuggly placed without tension and the second knot only tightened to prevent necrosis of the muscle tissue.

Blood vessel:

In blood vessels connective tissue response is limited and hence, prolonged suture support is required

Monofilaments of thin caliber such as 8-0 to 10-0 can be used depending upon the vessel

Intimal suture line must be smooth, to prevent clotting in the suture line

Knots must be secure

Needle must pass from within outwards

Stomach and intestines:

In intestines healing takes place in the serosal layer. Hence serosa to serosa closure is essential to prevent leakage and proper healing. Avoid taking bigger bites of tissues to prevent valvular effect and stenosis. Interrupted sutures prevent the purse-string effect. Usually small intestines require double layer closure, inner chromic catgut or vicryl, and outer layer of silk suture, single layer closure is adequate provided the correct surgical technique is followed.

* Stomach and small bowel - Two layer closure
* Large bowel - Single layer closure

Two layered closure:

- Inner (including all layer of bowel)-Continuous 2/0 or 3/0 vicryl or catgut stiches are locked to get perfect hemostasis
- Outer (seromuscular to ensure serosal opposition) Interrupted 2 0 or 3 0 silk

Genito-urinary tract:

Healing is good and tensile strength recovered within 3 weeks, in genito-urinary tract. Hence, absorbable material (chromic catgut) is good enough for suturing the urethra, bladder and ureter.

Lungs:

Lung tissues are repaired by chromic catgut / vicryl mattress sutures

Wound dehiscence

Definition

Wound dehiscence is defined as the wound giving way after primary closure following surgery. Dehiscence of wound is related to the ratio of collagen synthesis to lysis. More the lysis dehiscence will follow as in infection there is softening of the edge by collagenase activity. Starvation leading to weight loss (<20%) impairs collagen synthesis Steroid given on long term affects wound response to repair

Dehiscence can be prevented by

- Well nourished and oxygenated tissue (early rapid restoration of circulating volume)
- Prevention of collections and infection
- Good surgical technique
- Properly placed incisions
- Gentle handling of tissues
- Precise selection of suture material
- Closure in layers, with drains if required
- Tensionless suture techniques

Treatment

- Re-suturing of the wound edge
- Wash wound thoroughly
- Trim wound edges
- Use proper suture material, (vicryl, silk)
- Suture without tension
- Keep drains, if there is oozing or collection
- Prevent infection- administer antibiotics.
- Promote healing (proteins, vitamins).

Important Note

- Prevention is always better than cure
- Thin serous discharge is the forerunner of wound dehiscence.
- Take all necessary steps to prevent wound gaping at all costs It is alarming and demoralising to see the wound dehiscence in the postop period and has depressing effect on the patient.
- Increases the healing time and cost.

Chapter 2

- Anaphylaxis
- Shock
- Cardio Pulmonary Resuscitation
- BLS
- ACLS
- Burns
- Fluid and Electrolyte Imbalance and Replacement
- Stridor
- Septicaemia
- Coma
- Poisoning
  - Copper Sulphate Poisoning
  - OPC Poisoning
  - Potassium di chromate Poisoning
  - Mozhikizhangu Poisoning
- Emergency Surgical Procedures

Anaphylaxis

It is a generalized hypersensitivity reaction characterized by hypotension, Peripheral circulatory collapse and respiratory difficulty in the from of stridor and dyspnoea. Anaphylaxis can occur due to food, inhaled/ ingested allergens or drugs (Table 2.4). symptoms may occur instantaneously or within a few minutes after an intravenous injection of the offending agent. At times the reaction may develop after 1/2 - 1 hour of the exposure. Anaphylaxis to oral drugs may take 1-2 hours, but in many patients it can be instantaneous.

commonly used agents implicated in anaphylactoid reactions

1. Antibiotics
   * Penicillin and analogues
   * Tetracycline
   * Sulfonamides
   * Streptomycin
2. Local anaesthetics
   * Inj. Lidocaine
3. General anaesthetics & muscle relaxants
   * Inj. Thiopental
   * Inj. Tubocurarine
4. Non-steroidal anti-inflammatory agents
5. Blood products and vaccines
   * Red blood cell, white blood
   * Tetanus cell& platelet transfusions
   * Diphtheria
   * Gamma globulin
   * Snake and spider antivenoms
   * Rabies
6. Diagnostic agents
   * Iodinated radiocontrast agents
7. Venoms
   * Bees, wasps, spiders, jellyfish
8. Hormones
   * Inj. Insulin ป Pituitary extracts
   * Inj. Hydrocortisone ป Vasopressin
9. Extracts of allergens used for desensitization
   * Food
   * Eggs
   * Nuts
   * Milk and milk products
   * shellfish
   * Legumes (peanuts, soyabeans,
   * Kidney beans, chick peas)
   * Citrus fruits
10. Other drugs
    * Protamine
    * ACE inhibitors
    * Parenteral iron
    * Inj. Dextran

Rx Treatment

A Severe anaphylactoid reaction is a life-threatening emergency. Effective treatment depends on prompt diagnosis and rapid supplementation of appropriate therapy.

1. Inj. Adrenaline 1 : 1000, 0.01 ml/kg (maximum 0.3 ml in children and 0.5 ml in aduits) by IM injection. If necessary, dose can be repeated every 15 minutes. If the anaphylaxis is to injection of an allergen extract or to a hymenoptera sting into an extremity, half the dose of adrenaline can be infiltrated locally, subcutaneously after dilution with 2 ml saline. A tourniquet above the site can also slow systemic distribution of allergen. It can be loosened every 3 minutes.
2. For severe anaphylaxis with shock as in all medical emergencies, initial management should be directed at the ABC of resuscitation, namely maintenance of adequate airway - suction, breathing and circulation. if working alone, call for assistance
3. Establish one or preferably two, wide bore intravenous lines. Commence rapid fluid resuscitation with normal saline.
4. If there is severe laryngeal obstruction, bronchospasm, circulatory shock or coma,intubate and commence intermittent positive pressure ventilation.
5. If there has been little or no response to the initial intramuscular dose of adrenaline, administer adrenaline 5 mcg/kg slowly in to the intravenous line. Repeat at 5 minutes intervals depending on response. If the patient remains in shock, start an adrenaline infusion (preferably via a central

venous line), commencing 0.1 mcg/kg/ min in children and 0.2 mcg/kg/min in adults,titration is required to restore blood pressure. Large doses of adrenaline may be needed.

6. If the only manifestation of anaphylaxis is urticaria or angioedema, intial IM dose of adrenaline should be given in addition to H antagonists. If no progression occurs, patient can be kept under observation for at least 12 hours and then discharged.

Additional measures

1. Administer Salbutamol or Terbutaline by aerosol or nebulizer.
2. Inj. Diphenhydramine 1 mg/kg slow intravenously.
3. Inj. Ranitidine 1 mg/kg slow intravenously.
4. Inj. Hydrocortisone 2-6 mg/kg or Dexamethasone 0.1-0.4 mg/kg IV.

Supportive treatment

Observe vital signs frequently and if possible, Monitor electrocardiogram and pulse oximetry.

All patients who have suffered a severe anaphylactoid reaction must be admitted to the hospital. Patients who remain clinically unstable after intial resuscitation should be admitted to an intensive care unit. If nat admitted to hospital and if they respond to the intial treatment, provide infomation to them about possible late reaction.

Patient education

To check and look for the cause (food, drugs etc.) and to avoid it in future.

References

1. General ASpects of Treatment. In: Textbook of Dermatology. Champion RH et al (eds), 6th Edition, Blackwell Science Ltd. London. pp-3289.
2. Urticaria and Angioedema. In: Dermatology in General Medicine. Freedberg IM et al (eds), 5th edition, The McGraw Hill Company Inc., pp-1409.
3. Anaesthesia at the District Hospital. In: World Health Organization in collaboration with the World Federation of Societies of Societies of Societies of Anaesthesiologists, Geneva, 2nd Edition, 2000 World Health Organization.
4. Drugs Used in Anaesthesia. In: WHO Model Prescribing Information, 1989, World Health Organization.
5. Anaesthesia at the District Hospital. In: World Health Organzation in collaboration with the world Federation of Societies of Anesthesiologists, Geneva, 1988, World Heaith Organization.

Shock

Definition

Shock is defined as an acute circulatory failure leading to inadequate tissue perfusion resulting in generalized cellular hypoxia and end organ injury. Shock is classified as

- Hypovolemic shock
- Obstructive shock
- Cardiogenic shock
- Distributive shock

Hypovolemic shock

Reduction in the blood volume

Causes

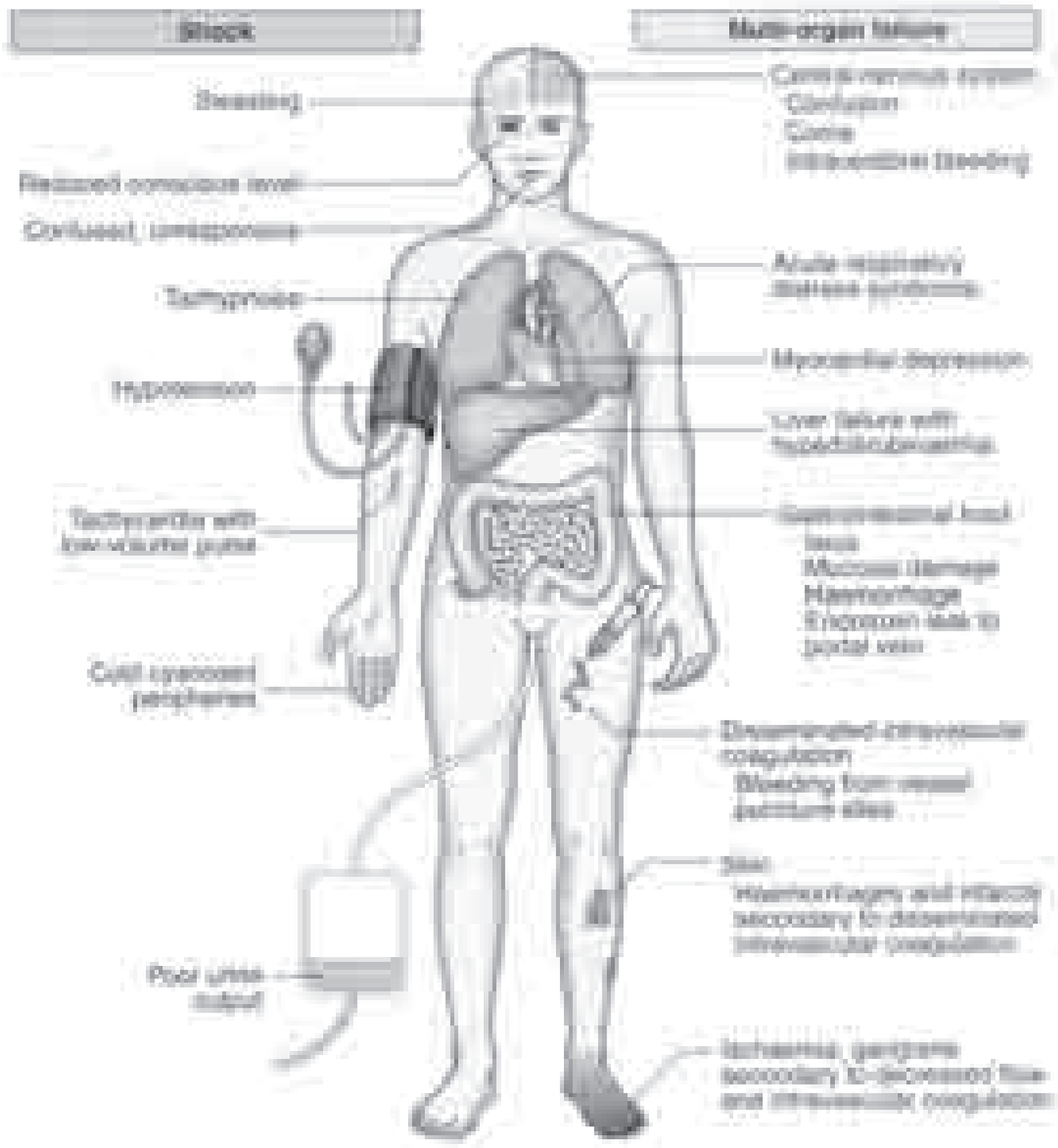
Hypovolemic shock is due to inadequate intake of fluids relative to losses (dehydration) or increased losses due to

- Bleeding
- Diarrhea
- Diuresis
- Excessive sweating or
- Third space loss as in burns, the crush syndrome, perforating gastrointestinal wounds, etc.,

Symptoms

- Breathlessness
- Thirst and dry mouth
- Bluish discoloration / cyanosis
- Cold and pale skin
- Delayed capillary fill (3 seconds or more)
- Depressed mental status or loss of consciousness
- Decreased or no urinary output
- Marked tachycardia >120/min (for children according to age)

Signs of Shock



- Decreased systolic blood pressure (Age appropriate for children)
- Narrowed pulse pressure or immeasurable diastolic pressure
- Weak or absent peripheral pulses/ weak central pulses

Investigations

- CBC- decreases in Hb (blood loss)/ increases in PCV (loss plasma or fluids), Leukocytosis or leukopenia (in sepsis)
- Blood glucose (diabetic ketoacidosis)
- BUN, creatinine
- Electrolyte levels (eg, Na, K, Cl, HCO3)

- Prothrombin time
- ABG
- Urinalysis- myoglobin or hemoglobinuria (in patients with trauma).
- Blood grouping and Rh typing

Rx Treatment

- Admit to hospital
- Supplement with high-flow oxygen
- Support with ventilation if needed
- Start two large bore IV lines
- Resuscitate with fluids
  * One to two litres of isotonic normal saline or ringerlactate for an adult
  * Also blood, albumin, fresh frozen plasma, hexastarch, pentastarch, and dextran 70 are used
  * Fluid requirement has to be calculated properly for children and administered carefully.
- Observe for vital signs
- Continue crystalloid infusion
- Arrange for urgent blood or plasma infusion if little or no improvement is seen with crystalloid infusion; consider early blood transfusion in suspected blood loss as in trauma.
- Raising the hypotensive patient's legs may be useful though, controversial
- Control further loss of blood, plasma or fluid by surgical or pharmacological agents

Fluid therapy for children

- Rapid IV access; if not possible intra osseous access
- Initial fluid bolus for shock
  * Intravenous fluids ringer lactate or normal saline 20ml/kg rapidly in hypovolemic shock over 5-20 minutes. Repeat boluses 2-3 times after reassessment of cardio-pulmonary status at the end of each bolus
- 2 or 3 fluid boluses will be required in hypovolemic or septic shock
- In case of blood loss, attempt replacement with blood after 2 boluses
- Reassess cardio-pulmonary status following each bolus by pulse volume, heart rate, skin perfusion, urine output, sensorium, blood pressure, liver size and lung signs.
- Withhold further boluses if cardiogenic shock occurs (airway instability, pink froth, apnea, development of grunt, retractions, abdominal respirations, fresh rales, gallop rhythm, enlargement of liver).
- Start vasopressor agents (Dopamine or Dobutamine) as intravenous drip

For Pediatric use

- Dopamine
  * Start at 10 mcg/kg/min; titrate dose by increment of 2-3 mcg/kg/min every 15 minutes upto 20 mcg/kg/min
  * Weight X 6 = mg of dopamine to be added to 100 ml of NS; 1ml/hr will deliver 1mcg/kg/mt; Indicated in hypotensive shock only after fluid boluses
- Dobutamine
  * Indicated in cardiogenic shock with normal or increased blood pressure
  * Inotrope of choice
  * dose: 10-20 mcg/kg/min
  * Preparation similar to dopamine.

For adults

- Dopamine
  * 2-4 mcg/kg/min in normal saline as IV drip and adjusted with blood pressure and increase at 5 minutes interval to appropriate hemo- dynamic end point.
- Dobutamine
  * 2-20 mcg/kg/min, as intravenous drip similar to dopamine.
  * Treat the underlying condition
  * Treat the associated metabolic disturbances

Referral

- Hypovolemic shock due to the loss of plasma volume can be managed adequately in the primary health centers and peripheral hospitals.
- Hemorrhagic shock and loss of fluid in the third space may require referral to higher centers.

Obstructive shock

Definition

Obstructive shock is a condition where in there is an obstruction of the blood flow to and from the heart, which may be due to inflow obstruction as in the case of pericardial tamponade, acute pneumothorax or hemothorax and outflow obstruction as in acute pulmonary embolism.

Diagnosis may be made from the history and clinical examination.

Symptoms

- Breathlessness
- Increase in JVP and pulsus paradoxus (pericardial effusion)
- Cyanosis and decrease jugular venous distension (tension pneumothaorax)
- Unilateral swelling (DVT which may lead to pulmonary embolism)

Investigations

- Basic investigations
- X-ray chest
- ECG
- Echocardiogram
- CT scan/perfusion scan

Treatment

- Admit to hospital
- Relieve the obstruction (life saving procedure)
- Proceed for urgent peri-cardiocentesis for cardiac tamponade
- Initiate needle thoracostomy for tension pneumothorax
- Start thrombolysis and proceed for surgical removal if massive pulmonary embolism

Referral

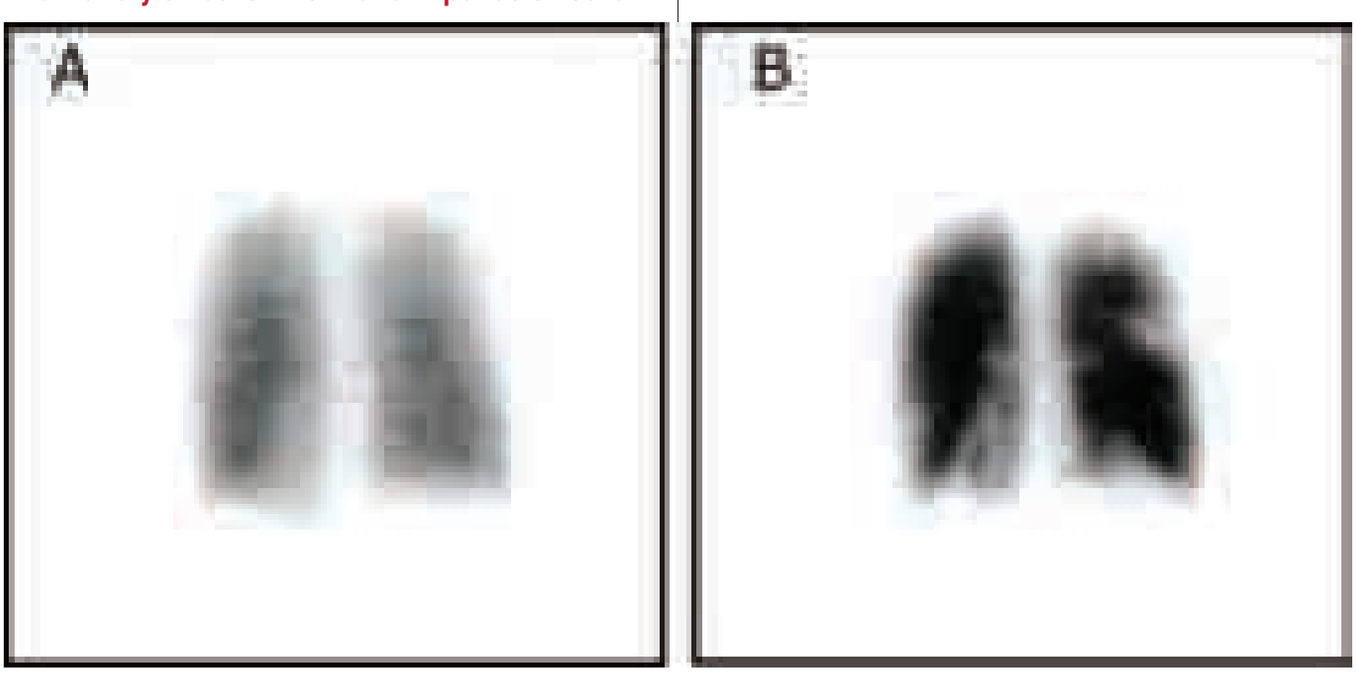
Obstructive shock almost always requires invasive treatment and hence these cases require referral to higher centers after providing supportive and resuscitative measures.

Cardiogenic shock

Cardiogenic shock is due to myocardial dysfunction with decreased cardiac output and evidence of tissue hypoxia in the presence of adequate intravascular volume.

- Hypotension and poor tissue perfusion
- Oliguria
- Clouded sensorium
- Cool, mottled extremities
- Other features of myocardial dysfunction such as
  - * Gallop rhythm

Pulmanory embolism ventilation - perfusion scna



* Pulmonary oedema and hepatomegaly secondary to acute myocardial infarction
* Aortic or mitral regurgitation or ventricular septal rupture (after excluding or correcting factors such as hypovolemia, hypoxia, and acidosis), myocarditis.

Investigations

- Complete blood count
- Blood glucose
- BUN, creatinine
- Electrolyte levels
- ECG
- Chest X-ray
- Urinalysis
- Other special investigations (depending upon the cause)

Treatment

- Admit to hospital
- Provide oxygen by face mask
- Start venous access
- Relieve the pain with strong analgesics (narcotics analgesics)
- Challenge with 250 to 350 ml of fluid if there are no lung crackles (In children smaller volume. i.e-5-10 ml/kg fluid boluses over 20 minutes with careful monitoring)
- Start vasopressor Dopamine drip if SBP remains less than 80 mm/Hg
- Prefer Dobutamine as drip if SBP is more than 80 mm/Hg
- Continue other supportive measures, as required.

Referral

Cardiogenic shock may require invasive treatment depending upon the cause and hence needs referral to higher centers.

Distributive shock

Definition

Distributive shock is a state of low peripheral vascular resistance.

- Conditions that come under this category are
  * Allergic shock / Anaphylactic shock
  * Septic shock and
  * Neurogenic shock
- This condition requires large amount of fluid. End points of fluid resuscitation are the same as for hypovolemic shock at this stage
- If hypotension persists, inotropes and vasopressors may be necessary.

Anaphylactic shock

Anaphylactic shock occurs as a result of injection, ingestion, inhalation, or skin contact of an allergen.

It is an emergency and immediate management saves the lives. If not treated promptly, the outcome is invariably fatal.(for more information, read the chapter on Anaphylactoid and anaphylactic reactions).

Symptoms

- Skin eruptions
- Peri-orbital fullness
- Sweating
- Hypotension
- Breathlessness
- Coughing due to laryngeal edema

Treatment

- Treated with intramuscular epinephrine (0.3-1mg) and volume resuscitation.
- Intravenous corticosteroids, and vasopressors
- Other agents preferred are Inj.chlorpheniramine and H2 receptor blockers

Referral

In most of the occasions referral may not be required if managed properly.

Septic shock

Causes

Septic shock results as a consequence of release of toxin by gram negative bacteria (E.coli,Proteus,Klebseilla) and gram positive cocci and fungi.

Symptoms

- Evidence of volume depletion such as dry mu-

cous membranes, and cool, clammy skin.

- Sometimes patients will be warm along with hypotension.
- Signs and symptoms pertaining to pneumonia, UTI, meningitis. intravenous catheter infection or others.
- Signs of possible infection include
  * Fever
  * localized erythema or tenderness
  * Consolidation on chest examination
  * Abdominal tenderness
  * Meningismus.
- Signs of end-organ hypoperfusion which include
  * Tachypnea
  * Oliguria
  * Cyanosis
  * Mottling of the skin
  * Digital ischemia
  * Abdominal tenderness
  * Altered mental status

Investigations

- Complete blood count (CBC)
- Arterial Blood Gas analysis
- Metabolic parameters
- Microbiological evaluation
- Cultures from relevark sites
- Other investigations as needed

Treatment

- Volume resuscitation with fluids, changes the clinical picture to hyperdynamic shock, with tachycardia, bounding pulse with a widened pulse pressure, a hyperdynamic precordium and warm extremities.
- 500 to 1000 ml fluid over 5 to 10 minutes to increase the mean blood pressure 65 to 75 mm of Hg.
- Take care while administering fluid in children. End points of fluid resuscitation are fall in heart rate, urine output > 0.5 ml / kg/ hour, central venous pressure of 8 – 12 mm Hg, central venous oxygen saturation > 70%, fall in base deficit, and fall in blood lactate concentration
- Norepinephrine as intravenous drip is superior in the treatment of hypotension of septic shock (0.05-5 µgms/kg/min)
- Inj. Hydrocortisone as intravenous (usefulness is controversial)
- Antimicrobial selection may be based on the basis of underlying illness, patients susceptibility and local resistance pattern
- Targetted narrow spectrum treatment is probably justified, if strong clues to the sources of infection exist. Some examples are
  * Pneumonia –second or third generation cephalosporin or macrolide
  * Urinary tract infection – Ampicillin plus Gentamycin
  * Meningitis - third generation Cephalosporin
  * Intra-abdominal.infection - third generation Cephalosporin
  * Primary bacteremia – Piperacillin and Tazobactum?
- Maintain normal blood glucose levels
- Avoid intravenous sodium bicarbonate

Course and outcome

- Early diagnosis and prompt treatment influence the outcome. However, the diagnosis cannot be made in all cases on the basis of initial findings on history taking and physical examination
- With effective antibiotics, volume replacement and vasopressors improvement may appear within 24 to 48 hours
- Prognosis in septic shock is very grave (mortality roughly 50%)
- Common sequence of organ failure is primarily heart and circulation followed by kidneys, respiratory system and brain, tertiary involvement will be liver and hemostatic system.

Referral

Patients not responding need referral to a higher center.

Cardiopulmonary resuscitation (CPR)

CPR consists of a series of maneuvers by which oxygenated blood supply to brain and vital organs is

maintained during cardiopulmonary arrest (CPA) i.e., cessation of respiration and circulation,

In children,CPA is not sudden but end result of long period of hypoxaemia secondary to inadequate ventilation, oxygenation or circulation. Therefore, prompt management of these is essential to prevent CPA, the outcome of which is poor.

Diagnosis of cardiopulmonary arrest

Cardiac arrest

1. Absence of pulse in major arteries (carotid or femoral in older children and femoral or brachial in infants as carotid is difficult to plpate due to short neck).
2. Absence of heart sounds on auscultation.
3. Asystole / ventricular fibrillation on ECG.

Respiratory arrest

Absence of respiration on looking (absent chest movements), listening (absent air flow on bringing ears in front of mouth) and feeling (absent air flow on keeping hands in front of mouth or nose).

Levels of CPR .

There are two levels of CPR.

1. BLS (basic life support) The elements of CPR provided without additional equipment, skill and speed are mose essential.
2. ACLS (Advanced cardiac life support). Use of equipment and drugs for assisting ventilation or circulation.

Basic life support

Call for help. Position the victim supine on firm flat surface with head level with the heart.

1. Airway
   * Clear airway by cleaning blood, secretions, foreign particles(suction if available).
   * Prevent posterior displacement of tongue due to muscle relaxation during CPA , by head tilt and chin or jaw thrust (may use an airway if available). **Head tilt:** Put a hand at forehead and tilt head back to sniffing or neutral position in an infant and little more in older children and adults. (**Caution:** In a patient with suspected cervical spine injury head tilt shoule be avoided) **Chin lift:** Put finger of other hand under bony part **Jaw thrust:** Place 2-3 fingers under each side of lower jam at its angle and lift jaw upward withthe elbow resting on the surface on which victim is lying.
2. Breathing
   * Determine the absence of breathing. Give mouth to mouth / nose/ mask/airway breath(may use bag and mask if available). Inhale and then make a seal around the mouth and nose together in an infant and seal mouth only in older children and adults (nose pinched with the hand used for head tilt) to exhale smoothly. Rate of breaths should be 20/min for infants; 15/min in older child and 10-12/min in adults.

c. Circulation
   * Determine the absence of pulse after 2 breaths (rescue breaths). External cardiac massage if asystole and unresponsive to rescue breaths. In children, perform cardiac massage if HR <60/min with signs of poor perfusion.
   * Rescuer should stand or kneel at the side of the patient so that his hips are on a level with the victim's chest.
   * In a newborn 2 thumbs are positioned side by side on sternum just below the nipple line, with fingers encircling chest and supporting the back and compress sternum by 0.6-1.2cm (120/min).
   * In an infant put index finger at the intersection of intermammary line and sternum. Use 2-3 fingers (index, middle and ring) to compress sternum by 1.5-2.5 cm(100/min) and do not lift the finger when compression is released. Two thumb- encircling hands technique can also be used.
   * In children(1-8 years) use heel of hand on lower half sternum with long axis of heel same as long axis of sternum and compress 2.5-3.5cm (100/min)
   * In adults the heel of one hand is placed on the lower sternum and the other hand placed on top of the first. The elbows should be locked in position with the arms straight and the shoulders over the hands. Sternum should depress by 3.5-5.0 cm and the rate of compression should be 80 to 100/min.(CAUTION: Do not exert pressure on the ribs, costal cartilages

or xipoid)

Combination of ventilation and cardiac massage

If both cardiac and respiratory arrest-Compression: ventilation=1.5: 2 in adults and children>8 years: Children and infants 1-8 years = 5:1; Neonates=3:1.

Advanced Cardiac Life Support (ACLS)

If ACLS facility is available, shift the patient to ACLS as soon as possible. If this is not available then continue cardiac massage till spontaneous HR is more than 60-80/min and continue artificial breathing till adequate respiratory efforts are present (good chest movement, no cynaosis or shock). For ALCS proceed in the folowing order.

1. ECG monitoring (if available)
   * If ventricular fibrillation, defibrillation.
   * In adults.First shock at 200 Joules; if the first is unsuccessful then a second shock at 200-300 Joules. If both fail, additional shocks at 300-360 Joules are given.
   * In children. 2 Joules /kg and can be repeted a few times (if does not revert to normal rhythm). Continue cardiac massage in the mean time.
   * All patients require oxygen(100%) because even with best CPR only a fraction of the cardiac output is provided and alo there are other factors causing ventilation perfusion mismatch.
   * Establish IV line as early as possible to give drugs and fluids and intubation of trachea should be done to continue artificial ventilation.
   * Drugs are used in the following order if indicated.

Inj.Adrenaline

Indication: Asystole, symptomatic bradycardia unresponsive to ventilation, In adults: 1 mg IV every 3-5 minuts. In children: 0.1 ml/kg of 1:10,000 solution (0.01 mg/kg) IV, intra-osseous or 0.1 ml/kg of: 1:1000 solution by endotracheal tube followed by several positive pressure breaths. Can be repeated every 5 min by either route. IV route is preferred and should be used as soon as IV access is achieved (Intracardiac route is not desirable).

Inj.Lignocaine

Indication: Ventricular tachycardia or fibrillation non resposive to recurs after defibrillation.In adults: Initial bolus dose is 1.5 mg/kg. Additional bolus of 0.5-1.5 mg/kg can be given 5-10 miutes during CPR up to a total dose of 3 mg/kg.In children: Inj. 1 mg/kg IV stat followed by infusion at 20-50 mcg/kg/min.

Inj. Amiodarone

Indication: Shock refractory ventricular fibrillation (as an alternative to or after failure of lignocaine).In adults: Initially 300 mg rapid infusion in 20-30 ml saline followed by 150 mg over 10 minutes followed by 1 mg/min for upto 6 h to 0.5 mg/kg/day.

Inj.Atropine

Indication: Vagally mediated bradycardia during intubation, HR<80 or asystole in an infant and symptomatic bradycardia with AV block in any child. Dose and route: 0.02 mg/kg bolus(not <0.1 mg or > 0.5 mg for a child and 1.0 mg for an adult). This dose may be repeated after 5 minutes for a maximum total dose of 1.0 mg for a child and 2.0 mg for an adult.

Inj.Naloxone

Indication: Narcotic overdos or poisoning and newborn resuscitation (if mother has been given morphine or pethidine during labour. Dose and route 0.1 mg/kg IV.

Inj. Sodium bicarbonate (NaHC03)

Not required routinely as it can cause alkalosis later and worsen respiratory acidosis by releasing CO2 in inadequate ventilation. Indication: Hyperkalaemia, significant metabolic acidosis (pH<7.2) or prolonged CPR. In adults and in children: Inj.Sodium bicarbonate 1 mEq/kg stat and 0.5 mEq/kg every 10 minutes in protracted resuscitation.

Inj.Calcium

Indication: Not used routinely now a days unless there is hyperkalemia, hypocalcaemia or calcium channel blocker toxicity. Dose and route: (in children) 0.5 ml/kg of calcium gluconate IV . In adults 10 ml to be given as a slow infusion under ECG monitoring.

Inj.Glucose

Indication - Hypoglycaemia Dose and route:- 0.5 -1 g/kg IV. 5. Try to get ABG, serum electrolytes and blood sugar (dextrose stick/glucometer) - post resuscitation

care.

- Maintain mechnical ventilation for several hours to ensure adequate oxygnation and ventilation.
- Look for and treat seizures.
- Inj.mannitol 0.5-1 g/kg IV if raised intracranial tension.
- Maintain temperature, fluid and electrolyte balance and ABG.
- Treat shock with fluids, dopamine, dobutamine and adrenaline infusion as required.
- Treat the underlying pathology causing CPA.

Monitoring

Pulse should be palpable and chest expansion should be seen during effective CPR, Blood pressure, Sp02, ET CO2 (In intubated patient and if facility available) , ABG should be monitored during and soon after CPR.

Termination of CPR

If asystole persists for >10 minutes after CPR has been performed, ventricular fibrillation eliminated, successful endotracheal intubation accomplished and confirmed, adequate ventilation provided and appropriate medications given.

Patient education

- Explain to parents that many causes of CPA are preventable e.g., injuries (by providing safe environment), poisoning (by keeping drugs out of reach of children), foreign bodies (safe toys and avoid beads, balloons etc., and avoid eatables like peanuts in infants). Young children should be closely supervised.
- Generlal public should be trained in BLS.
- Health care workers should be able to recognize and refer emergencies in time, and also know about BLS.

References

1. Management of Caradic and Respiratory Arrest. In:Davidson's A practice of Anaethesia, 6th Edition, Wylie and Churchill, 1995. pp1409-1425.
2. New Guidelines for Cardiopulmonory Resucitation and Emergency Cardiac Care - Changes in the Management of Cardiac Arrest. JAMA 2001;285(10) 1267.
3. Cardiopulmonory Resuscitation in infants and children. In: principles of Critical Care Ed.Udwadia FE,1995, Oxford University press Delhi.
4. Cardiopulmonory Resuscitation (CPR) In: Harrison's principles of Internal Medicine. Kasper Dt.Braunwald E,Fauci As et al (eds) 16th Edition 2005, MeGraw Hill Company Inc, New York, pp 1608- 1624.

BURNS

Burns are a mojor preventable cause of morbidity and mortality. These can be heat or space heating, moist heat - scalds and fat burns, ionizing radiation, electric burns, friction, chemicals and cold - frost bite.

SALIENT FEATURES

- Burns, pain, anxiety fluid loss and dehydration, local tissue oedema and infection.
- Early complications include shock, toxaemia, sloughing of mucous membranes-gastrointestinal tract and respiratory tract, inhalation injuries, acute renal failure, and haematemessis **(Curling ulcer).**
- Late complications include, protein losing enterpathy, secondary haemorrhage, hypertrophic scar/keloid and contracture.

Rx Treatment

Immediate resuscitation and care

- Clear airway, suspect inhalation injury if history of being trapped in close space, facial burns, stinging of eyebrows/nasal hairs, respiratory distress, hoarseness of voice or stridor, altered consciousness and soot in sputum.
- Check for breathing and circulation and provide support.
- Rule out other associated injuries.
- Insert nasogastric tube in all major burns.

Assess the severity of burns

Assssment includes calculation of surface area of burns: Rule of nine/charts, depth of burns, location of burns, patient;s age and presence of associated injury or disease. **Criteria for admission or transfer to a burns center**

- Burns of more than 20% body surface in an adult.
- Burns of more than 10% body surface area in a child under 10 or adult over 50 years,
- Burns of more than 5% body surface area in an infant.
- Burns of head, face, neck or perineum.
- Respiratory burns or inhalation injury.
- Circumferential burns.

Transfer should be done in a fully equipped ambulance with secured airway and circulatory support.

Fluid and electrolyte imbalance and replacement (In Adults)

Disturbances in fluid and electrolyte balance occur in a wide spectrum of diseases, are not confined to any particular field of medicine and are common following burns, trauma and major surgery.

The conventional and easy method of evaluating disturbances in fluid and electrolyte balane is the frequent measurement of the concentration of serum electrolytes. It is crucial to remember that intracellular and extracellular electrolytes are normally constant, and that major shifts in and out of 'compartments' can occur in disease with minimal changes in serum electrolyte. Compositional changes also involve disturbances in acid-base balance.

Volum changes: volume deficit

1. Obvious causes
   * Vomiting diarrhoeas, intestinal fistulae, nasogastric suction, fluid loss following burns, sequestration of fluid in soft tissue injuries and infections, diuretics, renal disease/adernal insufficiency.
2. Less obvious causes
   * unsuspected inadequate fluid intake, fluid loss through excessive sweating as in high fever, hot humid temperature, haemodialysis, haemofiltration from surgical incisions and in diseases like tetanus.

Management

The first principle is to restore circulating volume through infusion of intravenous fluids. Once this is satisfactorily achieved, distubances in electrolytes and acid-base balance if present need to be rectified. Various fluids used for volume replacement are given below:

Replacement fluids

1. Replacement fluids are used to replace abnormal loss of blood, plasma or other extracellular fluids as first line treatment for hypovolaemia in.
   * Treatment of patients with establised hypovolaemia e.g. haemorrgagic shock.
   * Maintenance of normovolaemia in patients with ongoing fluid lossing e.g. surgical blood loss.
2. Intravenous replacement fluids are the first line of treatment for hypovolaemia. Initial treatment with these fluids may be lifesaving and provide some time to control bleedig and obtain blood for transfusion if it becomes necessary.
3. Crystalloid maintenance fluids, which contain dextrose, are not suitable for use as replacement fluids. Only crystalloid solutions with a somilar concentration of sodium to plasma (normal saline or balanced salt) solutions (Ringer's lactate or Hartmann's solutions) are effective as replacement fluids.These should be available in all hospitals where intravenous replacement fluids are used. Fluids and electrolyte requirement in adults and children are shown in Table 2.3.
4. Crystalloids should be infused in a volume at least three times the volume lost in oeder to correct hypovolaemia.
5. All colloid solution (albumins, dextran, gelatins and hydroxyethyl starch solutions) are replacement fluids. However, they have not been shown to be superior to crystalloids in resuscitation.
6. Colloid solutions should be infused in a volume equal to the blood volume deficit.
7. Plasma should never used as a replacement fluid.
8. Plain water should never be infused intravenously. It will cause haemolysis and will probably be fatal.
9. In addition to the intravenous route, the intraosseous, oral, rectal or subcutaneous routas can be used for the administration of fluids, blood and certain drugs. However, with the exception of intraosseous route, other routes are generally unsuitable in severely hypovolaemic patients.
10. Rectal fluids are administered through a plastic or

rubber enema tube which is inserted into rectum and connected to a bag or bottle of fluid. The fluid rate can be controlled by using a drip giving-set, if necessary. The fluids used need not be sterile. A safe and effective solution for a rectal rehydration is 1 liter of clean drinking water with teaspoon of table salt.

11. Subcutaneous fluids: Occasionally, when other routes of administration of fluids are unavailable a subcutaneous infusion can be used. A cannula or needle is inserted into the subcutaneous tissue (the abdominal wall is a preferred site) and sterile fluids are administered in a conventional manner. Do not give dextrose-containing solutions subcutaneously as they can cause sloughing of tissues.
12. Oral and nasogastric fluids: Oral rehydration can often be used in mildly hypovolaemic patients if the oral route is not contraindicated. Do not use if:
    * The patient is unconscious.
    * The patient has gastrointestinal lesions or reduced gut motility e.g. obstrution.
    * General anaesthesia and surgery is planned imminently.

WHO/UNICEF formula for oral rehydration fluid:

Dissolve in one litre of drinkable water	
Sodium chloride	2.6 g/L
Trisodium citrate / dihydrate	2.9 g/L
Potassium chloride	1.5 g/L
Glucose anhydrous	13.5 g/L
Resulting concentrations	

Na+ 75 mmol/L, C1- 65 mmo1/L, K+ 20 mmo1/L, Glucose anhydrousv 75 mmo1/L, Citrate 10 mmo1/L,

Total osmolarity 245 mmo1/L.

Maintenance fluids

Maintenance fluids are fluids used to replace the normal physiological loss that occurs in a patient through skin, lung, faeces and urine. Since a considerable proportion of the loss is water, maintenance fluids are mainly composed of water in the form of a dextrose solution. Some electrolytes may also be included in these solutions.

All maintenance solutions are crystalloid solutions. Some examples of crystalloids that are suitable that are suitable as maintenance fluids are: 50% dextrose and 4% dextrose in sodium chloride 0.18%

Fluid and electrolyte for adult and children under n0rmol circumstances

Weight	Fluid ml/ kg/24 H	Sodium mmol/ kg/24 H	Potassium mmol/ kg/24 H
Children			
First 10 kg	100 (4*)	3	2
Second 10 kg	50 (2*)	1.5	1
Subsequent kg	20 (1*)	0.75	0.5
Adults			
All weights (kg)	35 (1.5*)	1	0.75

* These figures represent the fluid requirements in m1/kg/hour.

Safety

Before giving any intravenous infusion:

1. Check that the seal of the infusion fluid bottle or bag is not broken.
2. Check the expiry date.
3. Check that the solution is clear and free from visible particles.

Stridor

Causes

Common causes of stridor in children ara (i) congenital laryngomalacia, (ii) croup (acute laryngitis, laryngotracheobronchitis, epiglottitis); In adults, are (i) croup, (ii) allergies and (iii) tumours, Sudden onset of stridor may be caused by aspiration of a foreign boby. Other causes include peritonsillar, retropharyngeal abscesses, angioedema and hypocalcaemic tetany.

Salient features

- Noisy respiration primarily during inspiration produced by turbulent airflow through narrowed air passages.
- Respiratoty distress, restlessness and cyanosis are features of severe airway abstruction.

Treatment

Treatment of common causes of stridor is discussed below in four sections

A. Acute laryngitis/laryngotracheobronchitis

B. Spasmodic croup

C. Epiglotittis

D. Diphtheria

Acute laryngitis, laryngotracheobronchitis (croup)

It is an acute infection of the larynx and is usually caused by viruses. In children, may lead to respiratory obstrution.

Nonpharmacological

- Maintain airway by positioning the patient in lateral position with neck slightly.extended.
- Gentle suction of secretions, if required.
- Oxygen by ventimask/hood at the of 4-6 L/min.
- With the supportive and specific therapy, need for endotracheal intubation/tracheostomy may arise rarely. In case, the patient is detdriorating steadily despite therapy, elective intubation/tracheostomy should be done to prevent respiratory failure.

Pharmacological

Mild cases with minimal stridor (on exertion/crying) do not require any treatment and may need rest only. Moderate (stridor at rest) and severe cases (to be hospitalized immediately) need specific therapy in the form of:

1. Inj. Dexamethasone 0.4 mg/kg IM stat and followed by
2. Inhaled Adrenaline 0.01 - 0.05 mg/kg/dose to be diluted in 3 ml saline every 1-2 hours. A few doses can be administered until side effects viz. tachycardia, tremors etc. apper. or Inhaled Budesonide 500-1000 mcg/dose 12 hourly till response is seen.
3. Intravenous fluids maintenance dose (see respective section on fluids and electrolytes in adults and children).

Spasmodic croup

It occurs most commonly in children 1-3 years of age. It is possibly allergic and recurrent and occurs more often in the evening or night time. It has sudden onset, preceded by mild coryza and hoarseness. Symptoms usually diminish with in few hours.

Taking out the child with spasmodic croup in fresh air may decrease the airway obstructions.

Epiglottitis

It is usually caused by H. influenzae and is a potentially life-threatening condition. Lateral X-ray of soft tissue neck may show swollen epiglottis **(thumb sign)**. It is a medical emergency; airway and specific therapy must be introduced aggressively.

Treatment

1. Nonpharmacological treatment as above in acute laryngitis.
2. Inj. Cefotaxime 100 mg/kg/day divided into 3 does.(or) Inj. Ceftriaxone 100 mg/kg/day (maximum dose 4 g/day) in 2 divided doses. If cephalosporins not available Tab. Chloramphenicol 500 mg 6 hourly. In Children 100 mg/kg/day divided into 6 hourly doses.

Diphtheria

It is usually seen in non-immunized children.

Salient features

- Presents as stridor but laryngeal examination may show a membrane like structure (pseudomembrane), removal of which leads to bleeding.
- Large cervical lymph nodes (Bull neck appearance) and hoarseness.
- Common complications are palatal palsy, III nerve palsy, polyneuritis and myocarditis.

Treatment

1. Supportive treatment as above in acute laryngitis.
2. Immediately refer the child to Infectious Diseases Hospital under supervision and oxygen therapy. In case the child cannot be transferred isolation should be done and following measures should be taken immediately:
3. Inj. Diphtheria antitoxin 20,000 - 40,000 IU, IV or IM for pharyngeal and laryngeal involvements

with disease present for < 48 hours; 40,000 to 60,000 IU for nasopharyngeal infections; 80,000 to 100,000 IU for diffuse involvement that has been present for > 3 days.

4. Inj. Crystalline penicillin 25,000 - 50,000 units/kg/day divided into a doses for 14 days. (or) Inj. Procaine Penicillin 1 Lac - 25,000 - 50,000 units/kg/day in 2 divided doses for 14 days. (or) Syr. Erythomycin 40-50 mg/kg/day divided into 4 doses for 14 days.
5. IV saline infusion over 60 min.

Monitoring and follow up

1. Watch the child for altered sensorium, degree of stridor, pulse rate, respiratory rate and for other features of respiratory distress like intercostal recession etc.
2. Larryseal examination should not be done until facilities for intubation are available because it might lead to sudden respiratory arrest.
3. If child develops stridor at rest, hospitalize immediately.
4. Increasing respiratory distress with alteration of sensorium or cyanosis may be an indication for intubation.

Patient education

- Avoid overuse and misuse of voice.
- If stridor worsens or is noticed at rest, the patient should immediately report to the nearest health facility.
- Children with Diphtheria should be completely immunized after recovery of the current episode and contact to be immunized immediately (See section on immunization).

References

1. Stridor in: Scott Brown's Otolaryngology: 5th Edition, Volume 5, 1987 pp. 420-427.
2. congenital Anomalies of the Larynx. In: Nelson's Textbook of Paediatrics. Behrman RE, Kleigman RM, Jenson HB (eds). 17th Edition, 2003, pp 1409-1410

Septicaemia

Septicaemia is a clinical condition condition associated with invasion of blood stream by microorganisms giving rise to features of **systemic inflammatory response syndrome** (SIRS) i.e. presence of any two of the following: fever/hypothermia, tachypnoea, tachycardia, leucocytosis/ leucopenia. It may be associated with infection at specific sites (e.g. lungs, urinary tract,gastrointestinal tract) or there may be no clear originating focus. Septicaemia occurs more commonly in patients with defective baby defenses. In previously healthy persons, Staphylococcus aureus, Streptococcus pneumoniae and Escherichia coli are the most frequent organisms, while in patients with defective immune systems, Gram-negative bacteria including Pseudomonas aeruginosa may be responsible. Other febrile illnesses due to enteric fever and malaria may be difficult to differentiate from these pathogens clinically. Septicaemia when persists can result in multi-organ dysfunction syndrome requiring immediate to maintain haemostasis.

Salient features

- Sick look, features of SIRS, symptoms of local site of infection of respiratory/gastrointestinal system etc and symptoms due to decreased perfusion of various organs e.g. confusion, disorientation, decreased urinary output,icterus, bleeding diathesis (DIC) and haemodynamic instability like hypotension.

Rx Treatment

Nonpharmacological

1. Care of airway and breathing as given in section on CPR.
2. Removal or drainage of a focal source of infection. Indwelling intravenous catheter, Foley's cather etc should be replaced if considered as a source.
3. General care of skin, orodental hygiene and nutrition supplementation should be taken care of in prolonged severe seosis.

Pharmacological

1. Oxygen therapy - 2-4 liters/min with catheter/mask (to keep SPO2 > 95%).
2. Intravenous fluids - to be guided by haemodynamic status. If in shock, aggressive fluid therapy and mentioned in section on shock to maintain urinary output at more than 1 ml/kg/hour.

3. Antimicrobial agents - Antimicrobial therapy should be initiated as soon as samples for culture are withdrawn from blood and other relevant sistes. Choice of antibiotics depends on suspected organism.

Immunocompetent host

1. Inj. Cefotaxime 150-200 mg/kg/day in 3 divided doses.(or) Inj. Ceftriaxone 100 mg/kg/day (maximum dose 4g/day) in 2 dided doses.
2. Inj. Gentamicin 7.5 mg/kg/day in 2-3 divided doses. (or) Inj. Amikacin 15 mg/kg/day in 2-3 divided doses.
3. Add Penicillin/Vancomycin if Streptococus/Staphylococus organisms are suspected. Inj. Penocillin G aqueous 200,000-300,000 units/kg IV 4 hourly. (or)Inj. Vancomycin 15 mg/kg/day in 2 divided doses

Immunocompromised host

1. Inj. Ceftazidime IV 150 mg/kg/day in 3 divided doses.
2. Inj. Vancomycin 15 mg/kg/day in 2 divided doses.

Follow up and monitoring

- Continuous monitoring of pulse, respiratory rate,blood pressure, capillary filoling time, urinary output and neurological status should be done for early detection of septic shock or multi-organ failure.
- Patient should be referred to tertiary level centre if very sick or shows no signs of improvement after initial therapy.

Patient/parent education

- Immunocompromised patients should be informed about features of early sepsis. Fever in any child with congenital or acquired immunodeficiency stste should be taken very seriously.

References

1. Sepsis and Septic Shock. In: Harrison's Principles of Internal Medicine. Kasper DL, Braunwald E, Fauci AS et al (eds), 16th Edition, 2005, McGraw Hill Company Inc., New York, pp 1606-1612.
2. Sepsis and Shock. I.: Nelson's Textbook of Paediatrics. Behrman RE, Kleigman RM, Jenson HB (eds), 17th edition 2004, WB Saunders, pp 846-850.

COMA

Coma is defined as a prolonged period of uncondciousness and lack of reaction to stimulus. Patients in coma can not be aroused.

Salient features

Following causes affect the functions of reticular - activating system and its connections with cerebrrum.

- Structural damage to brain (haemorrhage, tumors, trauma, localized infections, meningitis, stroke).
- Metabolic disturbances (ischaemia, anoxia, uraemia, diabetes), respiratopry/hepatic/renal failure, dyselectrolytaemia, endocrinopathies, drugs like opiates, barbiturates, benzodiazepines, anti-depressants and cyanide.
- Abnormal elecrical activity - periodic lateralized epileptiform discharge (PLED).

Rx Treatment

Nonpharmacological

- The immediate goal in acute coma is the prevention of further nervous system damage.
- Hypotension, hypoglycaemia, hypercalcemia, hypoxia, hypercapnia and hyperthermia should br corrected rapidly and assiduously.
- An oropharyngeal airway is adequate to keep the pharynx open in drowsy patients who are breathing normally.
- Tracheal intubation is indicated if there is apnoea, upper airway obtruction, hypoventilation or emesis, or if the patient is liable to aspirate because of coma.
- Mechanical ventilation is required if there is hypoventilation or if there is an intracranial mass and a need to induce hypocapnia in order to lawer intracranial pressure.
- Establish intravenous access.

Pharmacological

1. Inj. Glucose (25 or 50%) 50 g IV.
2. Inj. Thiamine 100 mg IV.
3. If opiate overdose is suspected, give Inj. Naloxone 0.8 mg IV. Response is inadequate, double the dose every 15 minutes (for details see section on

opioid intoxication).

4. If benzodiazepine overdose is suspected give Inj. Flumazenil 200 mcg IV slowly. If no response repeat 100-200 mcg after 1 minute. If required give maximum does of 1 mg or give as IV infusion of 100-400 mcg/h if drowsiness recurs.
5. If focal neurological deficit or signs of herniation/decerebration/decortication occurs, CT scan, EEG and neurologic consultation is required.
6. If no clear aetiology and no herniation - CSF examination should be done.
7. If signs of raised intracranial tension (papilloedema, convulsions, decerebrate posture indicating herniation) occurs:
   * Avoid giving free fluid (glucose solution) intravenously.
   * Inj. Frusemide 40 mg IV to maintain adequate urine output of 30-50 ml/h.
   * Inj. Mannitol 1.0 g/kg IV over 10 minutes.
   * Hyperventilate to bring down PCO2 to 25 mmHg.
   * Inj. Dexamethasone 20 mg IV stat and 6 mg 4 hourly.

Children and young adults may have ominous early clinical findings such as abnormal brainstem reflexes and yet recover. Metabolic comas have a far better prognosis than traumatic comas. Glasgow coma scale empirically has predictive value in case of brain trauma (Table 2.5). For anoxic and metabolic coma, clinical signs such as papillary and motor responses after 1 day, 3 days and 1 week have been shown to have predictive value. Absence of cortical waves of the somatosensory evoked potentials has also proved a strong indicator of poor outcome in coma from any cause.

Grading of coma

Glasgow coma scale for head injury (Table 2-5)

Glasgow coma scale

Eye opening (E)	Coma score
Spontaneous	4
To loud voice	3
To pain	2
Nil	1
Best motor response (M)	
Obeys	6
Localizes	5
Withdraws (flexion)	4
Abnormal flexion posturing	3
Extension posturing	2
Nil	1
Verbal response (V)	
Oriented	5
Confused, disoriented	4
Inappropriate words	3
Incomprehensible sounds	2
Nil	1

Note: Coma score=E+M+V. Patients scoring 3 or 4 have an 85% chance of dying or remaining vegetative, while scores above 11 indicative only a 5 to 10% likelihood of death or vegetative state and 85% chance of moderate disability or good recovery. Intermediate scores correlate with proportional chances of recovery.

Reference

1. Coma. In: Harrison's Principles of Internal Medicine. Kasper DL, Braunwald E, Fauci AS et at (eds), 16th Edition 2005, McGraw Hill Company Inc., New York, pp 1624-1631.
2. Head Injury. In: Harrison's Principles of Internal Medicine. Kasper DL, Braunwald E, Fauci AS et al (eds), 16th Edition 2005, McGraw Hill Company Inc., New York, pp 2447-2452.

Poisoning

General considerations

Increasing incidence of poisoning is attributable to rapid development of newer compounds in trade, industry and medicine and easy access to them. A stepwise care approach to a patient of poisoning is helpful in successful management

Stepwise care approach

- Diagnosis - Suspect and identify poison, if possible.
- Treatment includes basic principles, antidotes, symptomatic and supportive.
- Anticipate coomplications, preserve evidence and prevent sequelae as well as recurrence.

Diagnosis

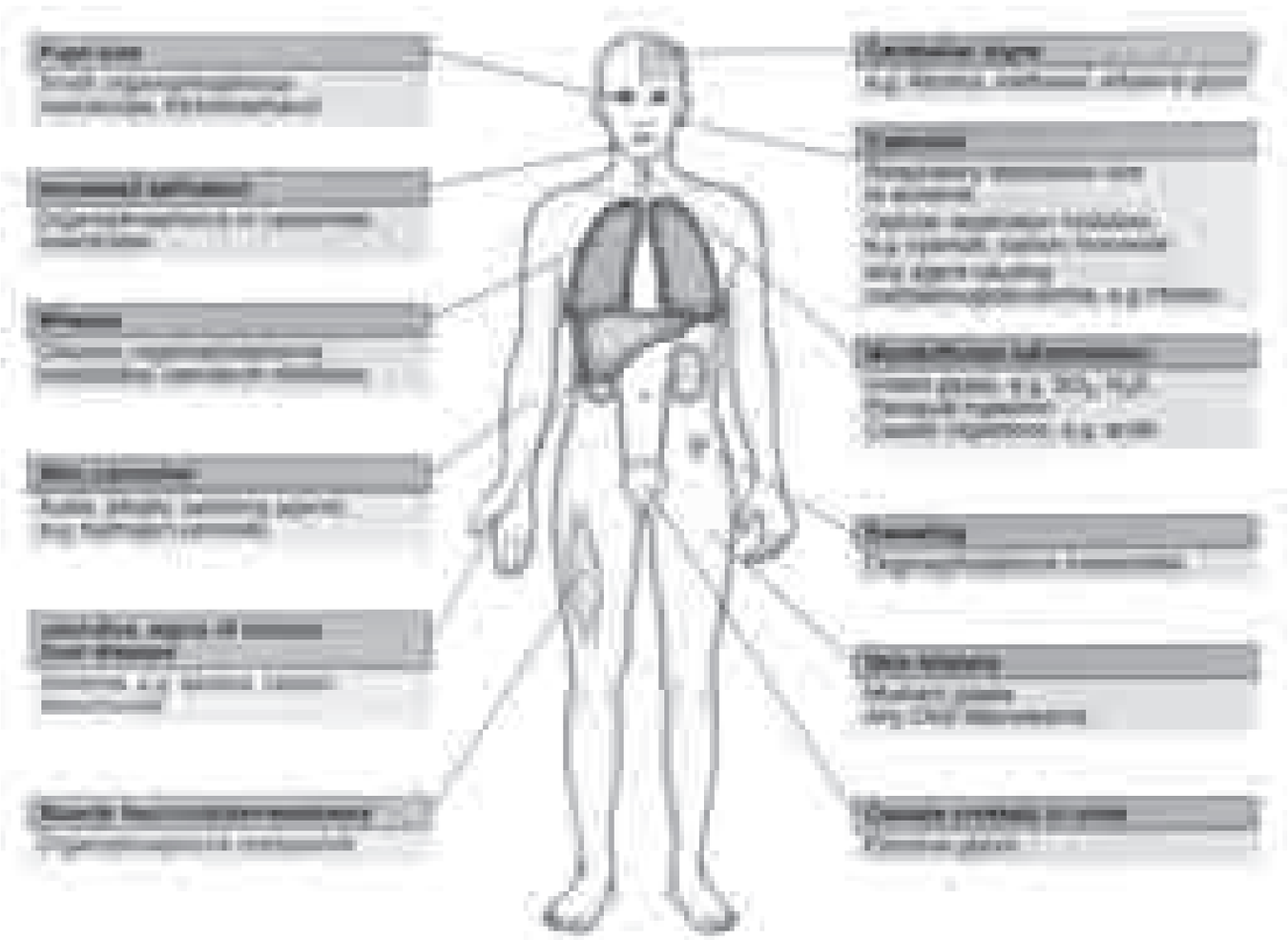
1. Suspicion of poisoning should be aroused by sudden onset of symptoms, uniform and increasing severity of symptoms in a group e.g. food poisoning or industrial poisoning. Unexplained nausea, vomiting, diarrhoea, drowsiness or coma, euphoria, increased psychomotor activity, convulsions, delirium and unusual breath smell are symptoms which in the absence of disease need careful evaluation for suspected poisoning. Signs and symptoms helpful in diagnosis of poisoning are shown
2. Identification of the substance should not take precedence over the first step, since the process is slow and unreliable or lack of proper history might cause confusion. Action of poisons is modified by physical factors like quantity, form, chemical combination, diluion, route of administration and host factors like age, idiosyncrasy, sleep, food and use (abuse) of multiple substances.

Copper sulphate poisoning

Introduction

Copper sulphate is blue in colour and used as an antiseptic, fungicide, herbicide and in crystal growing sets. Copper sulphate is a powerful oxidizing agent and corrosive to mucous membranes. Absorption of copper

Signs and symptomatology of Poisoning



sulphate ranges from 40 to 60% following ingestion.

Copper is transported across the intestinal mucosa which is facilitated by metallothionein. Highest concentration of copper is seen in the liver, brain, heart, kidneys and muscles. Ceruloplasmin renders free copper innocuous with subsequent biliary excretion. Renal clearance is very low.

Symptoms

- Hypersalivation
- Abdominal pain
- Vomiting
- Hematemesis and melaena may be fatal
- Shock due to reduction in the intravascular volume
- Jaundice due to hepatic cell necrosis and haemolysis
- Oliguria, anuria are due to acute tubular injury
- Kidney and liver effects develop 3 to 4 days after ingestion

Investigations

- Urine analysis
- Complete blood count
- Liver function tests
- Blood urea nitrogen (BUN)
- Creatinine
- Blood grouping and Rh typing

Special investigations

- For Hemoglobinuria
- Serum copper and ceruloplasmin
- Methemoglobin
- Creatinine kinase

Treatment

- Admission
- Gastric lavage is absolutely contraindicated
- Egg albumin (5-6 eggs) is used as demulcent (early oral administration has brought down morbidity and mortality).
- H2 receptor antagonist or Proton pump inhibitor as slow IV or orally
- D-Pencillamine 250 mg qid orally
- Inj. Vitamin C 500mgs 8th hourly in intravenous fluid as drip
- Fluid and electrolyte balance
- Supportive management - cardiovascular support
- Monitoring - renal and hepatic function
- Blood transfusion in selected cases

Referral

Referral to higher centre for supportive care and monitoring of cardiovascular, renal, liver and hematological systems.

Prognosis

Prognosis depends upon early diagnosis and appropriate management.

Patient education

- Oral dilution with milk or water (if performed immediately) may be helpful
- Egg albumin (5-6 eggs) is used as demulcent (earlier oral administration reduces complications)

Organophosphorous, Carbamate and Rodenticide Poisoning in Children

Introduction

Poisoning is common among children and takes hundreds of innocent lives every year. Unfortunately, most of these are accidental. Although in India poisoning due to pesticides and rodenticides are less in comparison to kerosene and drug over dosage, the practitioners and pediatricians should be aware of these due to their easy availability and accessibility. There is a need to strengthen the capacity to diagnose and treat such emergencies.

Epidemiology

In the absence of national registers or reliable hospital-based data, one looks forward to tertiary hospital data. Accordingly, organophosphorous (OP) and carbamate poisonings comprise less than one percent of total poisonings. Rodenticide poisoning is far less than that of pesticides.

Causes

- The incidence of pesticide poisoning tends to be

higher among children from lower socio-economic classes of society due to poor storage facilities and parental negligence.

- Inexperience, lack of maturity, illiteracy and inability to assess the risk make them ingest poison accidentally.
- Older children and adolescents may be directly exposed as field workers, while younger children may be brought into treated fields to accompany their parents.
- Work clothes often carry pesticide residues, exposing both workers and family members to harm.
- Stress factors for poisoning are grouped as
  - * Family stress
  - * Death of a parent
  - * Mental illness in a parent
  - * Financial problems
  - * Interpersonal conflicts among parents
  - * Parental alcoholism
  - * Divorce, separation
- Parental stress
  - * Punitive parent
  - * Conflict with parents
- School stress
  - * Poor academic achievement
  - * Examination failure
  - * change of school
  - * Teacher stress

Pathophysiology

- Organophosphorous compounds and carbamates bind to one of the active sites of acetylcholinesterase (AChE)1 and inhibit the functionality of this enzyme by means of steric inhibition
- Carbamylation of esters are more quickly reversible than phosphorylation of the esters
- Phosphorylation of the esters in AchE will undergo “aging” process
- Aging means loss of one alkyl or alkoxy group leading to stable mono alkyl or mono alkoxy phosphoryl AchE.
- The main purpose of AChE is to hydrolyze acetylcholine (ACh) to choline and acetic acid. Therefore, the inhibition of AChE causes an excess of ACh in synapses and neuromuscular junctions, resulting in muscarinic and nicotinic symptoms and signs.
- The pathophysiology of intermediate syndrome is not well defined and various studies are going on.
- In some individuals, neuropathy target esterase (NTE) is targeted to cause Organophosphorous compounds-induced delayed polyneuropathy.

Poisoning dosage

- Children may die of Organophosphorous compounds with very minimal dose of 2mg.(0.1mg/kg)
- Studies showed that young animals were more susceptible than adults of the same species, and that may be applicable to human beings as well.
- The poisoning dose varies from compound to compound. Therefore whatever be the situation, the victims should be observed for at least 48 to 72 hours.

Symptoms

- Children are more vulnerable than adults due to various risk factors such as
  - * Smaller size
  - * Differing metabolism and
  - * Rapidly growing and developing organ systems.
  - * Pediatric patients had predominately CNS depression and severe hypotonia, whereas muscarinic symptoms were infrequent.
- Pesticides can rapidly be absorbed through the skin, lungs, gastrointestinal tract, and mucous membranes
- The rate of absorption depends on the route of administration and the type of organophosphorous or carbamate
- Symptoms usually occur within a few hours after GI ingestion and appear almost immediately after inhalation exposure
- Patients often present with evidence of a cholinergic toxic syndrome, or toxidrome. It is useful to remember the toxidrome in terms of the three clinical effects on nerve endings and they are

* Nicotinic effects at neuromuscular junctions and autonomic ganglia
* CNS effects and
* Muscarinic effects SLUDGE/BBB and DUMBELS

- Nicotinic signs and symptoms include
  * Weakness
  * Fasciculation and paralysis
- CNS effects may lead to
  * Seizures and
  * CNS depression
- Carbamates have less CNS toxicity
- Respiratory failure
  * There seem to be two underlying mechanisms for respiratory failure and they are an early acute mixed central and peripheral respiratory failure, and a late peripheral failure rather than two distinct clinical syndromes.

Potassium dichromate poisoning

Introduction

In the industry chromium salts are used in safety matches, paint pigments (chromium compounds can be red, yellow, orange, and green), leather tanning, chrome plating, and copy machine toner. Also, these are used as corrosion inhibitors, photographic chemicals, and in magnetic tapes. **Salt of hexavalent chromium [Cr (VI)] is toxic.**

Causes

Poisoning happens due to potassium dichromate salts usually as accidental or suicidal, and is rarely occupational or environmental.

- Chromium compounds can be sensitizers as well as irritants
- Cr (VI) is a powerful oxidizing agent
- Exposure can cause irritation and corrosion. Cr (VI) is absorbed from the lungs, gut, and skin. After absorption, Cr (VI) is reduced to Cr (III) which forms protein complexes
- These complexes are taken up by bone marrow, lungs, lymph nodes, spleen, kidney, and liver. 60% of absorbed Cr (VI) is excreted by the kidneys and approximately 10% is eliminated by biliary excretion.

Symptoms

Acute Cr (VI) ingestion	Chronic Exposure
• Abdominal pain, vomiting, thirst and hematemesis, and malena • Features of shock due to reduction in the intravascular volume • Vertigo, convulsions, coma • Effects on kidney and liver develop one to four days after ingestion • Jaundice due to hepatic cell necrosis • Coagulopathy occasionally • Oliguria, anuria due to acute tubular necrosis • Systemic manifestations may develop with dermal contact with Cr (VI) compounds.	• Eczematous dermatitis with edema • Irritation of the conjunctiva and mucous membranes • Nasal ulcers and perforations, keratitis, gingivitis, and periodontitis. • Acute hepatitis with jaundice • Lung cancer is the most serious long-term effect

Investigations

- Urine analysis
- Complete blood count
- Liver function tests
- Blood urea nitrogen (BUN) and creatinine

Special investigation

- Urine chromium levels
- Beta-2 microglobulin

Treatment

- Hospitalization
- Induction of vomiting is contraindicated
- Gastric lavage with magnesium hydroxide or another antacid
- Fluid and electrolyte balance

- Supportive management- cardiovascular support and ventilator support
- Monitoring - renal and hepatic function

Referral

Referral to higher centre for supportive care and monitoring of cardiovascular, renal, liver and hematological systems.

Prognosis

Acute chromium poisonings are often fatal despite treatment and prognosis is variable.

Mozhikizhangu poisoning

Introduction

The scientific name of Mozhikizhangu is **Gloriosa Superba**. It belongs to the family Colchiaceae.

- It is also known as kalappathattai kizhangu and nabhikodi
- Entire plant, especially tubers are extremely poisonous
- Poisoning due to this plant is reported more frequently in certain parts of Tamilnadu
- It is cultivated in South India (e.g. Karur, Mulanur) and then sold in the market for pharmaceutical purposes for the preparation of anti-cancer agents
- It is also grown as an ornamental plant. Toxic properties of the plant are essentially due to active alkaloid colchicine and other derivatives.
- The chemical constituents are absorbed from the gastro intestinal tract and taken up intra-cellularly. 50% is bound to plasma proteins.

Symptoms

- Symptoms appear within 2 to 6 hours after ingestion.
- Numbness and tingling around the mouth.
- Burning and rawness of the throat.
- Nausea, intense vomiting and abdominal pain.
- Bloody diarrhea leading to dehydration.
- Respiratory depression
- Dyspnoea
- Shock
- Hypotension.
- Marked leucopenia, thrombocytopenia, coagulation disorders.
- Oliguria
- Hematuria
- Confusion, seizures, coma and ascending polyneuropathy.
- Alopecia/ dermatitis are late manifestations. (develop about 1 to 2 weeks after poisoning)

Investigations

- Daily full blood counts, coagulation tests
- Serum electrolyte levels and urinalysis
- Liver and renal function tests
- Blood collection for colchicine levels. (Collected and kept in the dark with anticoagulant).

Treatment

- Admission
- Induce vomiting, if patient is conscious and alert
- Carry out stomach wash
- Decontamination of digestive system

Treatment guidelines

- Monitor respiration
- Adequate airway
- Immediate gastric lavage
- Anticipate and treat hypotension with adquate intravenous fluids and vasopressors
- Transfuse compatible blood if required.
- Correct dehydration and electrolyte imbalance
- Provide continuous cardiac monitoring
- Monitor renal function
- Initiate forced alkaline diuresis once shock and dehydration are corrected (anti-colchicines antibodies have been tried).

Emergency surgical procedures

Venous cut down

Anatomical considerations for venous access The primary site for a peripheral venous cutdown is the greater saphenous vein at the ankle, which is located at a point approximately 2 cm anterior and proximal to the middle of the medial malleolus

A secondary site is the antecubital vein , located 2.5

Cm lateral to the medial epicondyle of the humerus at the flexion of the elbow

Saphenous vein cutdown:

- Prepare the skin of the ankle with an antiseptic solution and drape the area
- Infiltrate the skin over the vein with 0.25% lignocaine if the patient is conscious
- A full thickness transverse skin incision is made through the area of anesthesia to a length of 2.5 cm
- By blunt dissection , using a curved forceps , the vein is identified and dissected free of any accompanying structures
- Elevate and dissect the vein for a distance of approximately 2 cm to free it from its bed
- Ligate the distal mobilized vein, leaving a suture in place for traction
- Passatie around the vein, cephalad
- Make a small transverse venotomy and gently dilate the venotomy with the tip of a closed artery forceps
- Introduce a large plastic canula through the venotomy and secure it in place by tying the upper ligature around the vein and the canula
- Use distal suture to further secure the canula and the tubing
- Attach the intravenous tubing to the canula and close the incision with interrupted sutures
- Apply a sterile dressing

Complications of peripheral venous access:

- Perforation of the posterior wall of the vein
- Dissection of the vein
- Hematoma
- Phlebitis
- Cellulitis
- Venous thrombosis
- Arterial transection

Orotracheal intubation Procedure:

- Ensure that adequate ventilation and oxygenation are in progress, and that suction, a range of endotracheal tubes and laryngoscopes are available, together with introducer, boogie and magill forceps
- Connect laryngoscopic blade and handle, check the bulb for brightness
- Have an assistant manually immobilize the head and neck and a second apply cricoid pressure. The person must maintain cricoid pressure irrespective of all other events until directer to discontinue by the intubating doctor
- Use a rapid sequence induction unless patient is unresponsive and flaccid
- Hold the laryngoscope in the left hand
- Insert the laryngoscope into the right side of the patient's mouth displacing the tongue to the left
- Visually examine the epiglottis and the vocal cords
- gently insert the endotracheal tube into the trachea without applying pressure on the teeth or the oral tissues inflate the cuff with enough air to provide adequate seal
- Check the placement by bag- valve - to tube ventilation
- Visually observe the lung expansion with ventilation
- Auscultate the chest and abdomen with a stethoscope to ascertain position
- Release cricoid pressure only when the tube is in satisfactory position and the cuff inflated
- If the endotracheal intubation is not established within 30 seconds, discontinue attempts, ventilate the patient and try again

Complications:

- Esophageal intubation, leading to hypoxia and death
- Right main bronchus intubation, resulting in collapse of the left lung and hypoxia
- Induction of vomiting, aspiration and death
- Dislocation of the mandible
- Inability to intubate
- Airway hemorrhage due to trauma
- Chipping or looseniong of teeth
- Dislocation of cervical spine during hyper extension or hyperflexion

Tracheostomy

Procedure:

- Assemble the necessary equipment(tracheostomty tray, tube, suture- cuffed disposable tracheostomy tubes are now available)
- Place the patient supine with the neck extended
- Surgically prepare and anesthetize the area locally
- Make a transverse skin incision over the lower neck over the trachea - preferably below the 2nd tracheal ring
- Incise the deep cervical fascia, retract the strap muscles laterally
- Expose the trachea beware of the thyroid isthmus, retract it superiorly, ligate any vessels carefully
- Incise the tracheal cartilage after confirming by aspuiration of air, removing a cuff of thr trachea
- Insert a cuffed tracheostomy tube
- Inflate the cuff and ventilate the patient
- Close the incision
- Secure the tube to prevent dislodgement

Complications:

- Asphyxia
- Aspiration
- Cellulitis
- Creation of false passage in the tissues
- Tracheal stenosis
- Hemorrhage or hematoma formation
- Laceration of the esophagus
- Mediastinal emphysema
- Vocal cord paralysis, hoarseness

Intercostal chest drain

Procedure:

- Ensure fluid resuscitation via atleast one large caliber iv line and high flow oxygen by mask
- Assemble the necessary equipment, scrub, don gown and gloves
- Determine the insertion site; usually at the nipple level (5th intercostal space) anterior to tghe midaxillary line on the affected side
- Use a 36fr tube in males, 32fr tube in females
- Prepare and drape the chest at the site of tube insertion
- Locally anesthetize the skin and rib periosteum and intercostal muscles. Use 30- 40ml of 0.5% lignocaine in the adult and infiltrate extensively
- Make a 2 -3 cm transverse incision in the line of the intercostal space at the predetermined site and bluntly dissect through the subcutaneous tissues, just over the upper border of the lower rib
- Puncture the parietal pleura with the tip of the clamp, stretching up the opening and put a gloved finger into the incision to avoid injury to other organs
- Mount the tip of the tube in the jaws of the forceps and advance into the pleural space
- Look for fogging of the tube with expiration and movement of the water column
- Apply an airtight dressing and tape the tube to the chest
- Obtain a chest x-ray
- Obtain arterial blood gases if necessary
- Keep the chest drain bottles emptied regularly

Complications:

- Tube dislodgemnt
- Chest bottle elevation with fluid flowing into the chest cavity
- Damage to intercostal nerve, artery or vein
- Damage to internal mammary artery if puncture is too medial
- Intercostal myalgia
- Introduction of pleural infection
- Laceration or puncture of intra thoracic or intra abdominal organs
- Local cellulitis
- Local hematoma
- Mediastinal emphysema
- Subcutaneous emphysema

Gastrostomy:

- Assemble necessary equipment (laparotomy tray, tube, suture)
- Place the patient in supine position
- Done under GA or regional anaesthesia

Procedure

- Small incision made in the anterior abdominal wall , usually in the midline
- Small incision made in the anterior stomach wall inside a pursestring
- The catheter (malecot/ foley) is introduced and the suture snugged around the catheter
- The anterior wall of the stomach should then be fixed to the anterior abdominal wall to prevent dragging
- Laparotomy wound is closed and the catheter fixed to the skin

Complications:

- Tube dislodgement
- Leak
- Hematoma
- Twist of bowel around any intra peritoneal tube with obstruction

Cardiology

Standard Treatment Guidelines
Tamil Nadu Health Systems Project

Chapter 3

- Rheumatic Fever
- Infective Endocarditis
- Acute Pericarditis
- Cardiomyopathy
- Hypertension
- Angina Pectoris
- Myocardial Infarction
- Congestive Heart Failure
- Pulmonary Embolism
- Arrhythmias

Rheumatic fever

Rheumatic fever is due to an autoimmune reaction to group A streptococci and involves many organs, primarily the heart, joints and the central nervous system.

Causes

Acute Rheumatic fever usually occurs about three to four weeks (range between 1-5 weeks) after an attack of group A streptococcal throat infection. Antigenic "mimicry" between streptococcal M protein and myocardial cells is thought to be the cause. Poor socio-economic conditions predisposes to Rheumatic fever

Important note

Children 5 – 15 years, most commonly affected

Updated Jones Criteria 1992 – AHA

Major
Migratory Polyarthritis
Carditis
Sydenham's chorea
ESR / CRP
Erythema Marginatum
Minor
Fever
Arthralgia
Elevated acute phase reactants
Subcutaneous nodules
Prolonged PR interval
Plus
Supporting evidence of a recent group A streptococcal infection (e.g. positive throat culture or rapid antigen detection test; or elevated or rising streptococcal antibody test). Diagnosis – One major and two minor or two major criteria

Treatment

- Primary Antibiotic treatment
  - * Oral Penicillin V-500 mg bd -10 days (or)
  - * Inj. Benzathine Penicillin G – 1.2 million units in single dose (or)
  - * Erythromycin – 250 mg qid – 10 days – If patient is allergic to Penicillin
- Secondary prophylaxis
  - * Inj. Benzathine Penicillin – 1.2 million units in every 3 weeks (or)
  - * Oral Penicillin V – 250 mg bd – daily (or)
  - * Oral Sulfadiazine
  - * Erythromycin 250 mg twice daily

Duration

- Category of patient
  - * Patient without proven carditis
  - * Patient with carditis (mild mitral regurgitation or healed carditis)
  - * More severe valvular disease
  - * Valvular surgery
- Duration of prophylaxis
  - * For 5 years after last attack or till 18 years of age whichever is longer
  - * For 10 years after last attack or 25 years of age whichever is longer
  - * Lifelong

Arthritis

- Salicylates
  - * Doses upto 2 gram qid in adults and 80-100 mg/kg/day in children till symptoms persist.
- Naproxen
  - * Dose 10-20 mg/kg/day can also be used.

Carditis

Prednisolone 1-2 mg /kg/day can be used for short periods or upto a maximum of 3 weeks

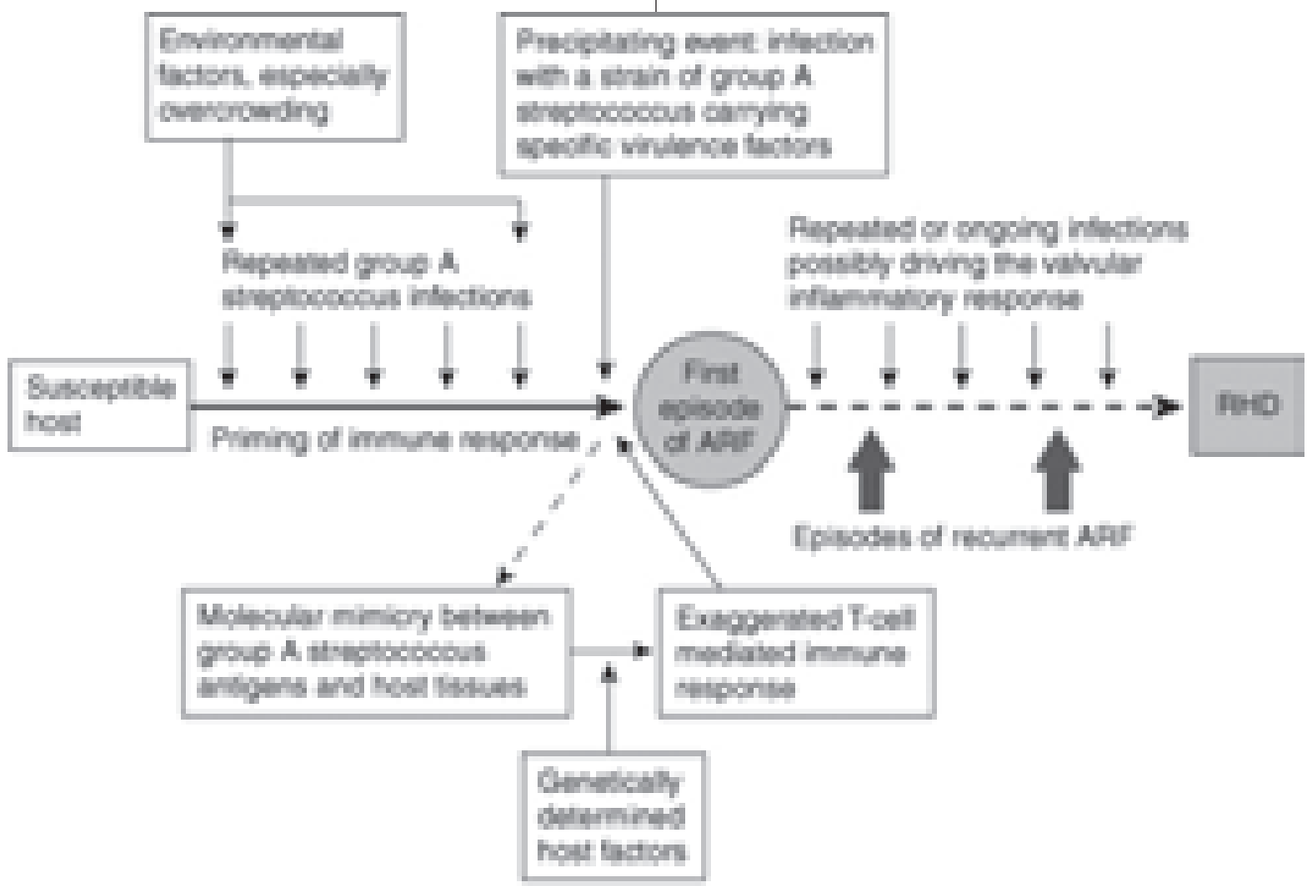
Chorea

Carbamazepine or Sodium valproate are preferred to Haloperidol to reduce the abnormal movements but response may take upto 2 weeks.Treatment should continue upto 1-2 weeks after symptoms subside.

References

- Harrison's principles of internal medicine, 16th edition, pg. 1977 – 1979
- Braunwald's heart disease, 7th edition, Pg. 2093 – 2098

Pathogenesis of Rheumatic fever



Infective endocarditis

Causes

Infective endocarditis is due to the proliferation of micro-organisms in the endocardium.

Salient features

- Acute and subacute endocarditis
- Organisms – Native valve
  - * Viridians streptococci
  - * Staphylococci
  - * HACEK
  - * Enterococci
  - * Streptococcus bovis
- Nosocomial
  - * Staphylococcus aureus
  - * Gram negative organisms
- Prosthetic valve
  - * Staphylococcus aureus
  - * Coagulase negative staphylococci
  - * Facultative gram negative bacilli
  - * Diphtheroids and fungi
- Injection Drug abusers
  - * Right heart – Staphyloccocus aureus
  - * Left heart – Staphyloccocus aureus
  - * Pseudomonas
  - * Candida
  - * Lactobacillus
  - * Corynebacterium
- Polymicrobial endocarditis

Definition

Duke's criteria

2 Major (or) 1 Major and 3 Minor (or) 5 Minor criteria

Major

- Positive Blood culture - Typical organism for infective endocarditis from 2 blood cultures **or** organism consistent with infective endocarditis on 2 cultures, drawn 12 hours apart or all of 3 or 3/4 blood cultures, the first and last drawn 1 hour apart **or** single positive blood culture

for Coxiella burnetti or phase1 IgG antibody for C.burnetti>1:800.

- Evidence of endocardial involvement by ECHO
  - Vegetations
  - Abscess
  - Partial dehiscence of prosthetic valve
- New valvular regurgitation.

Minor

- Predisposing factor : Heart Lesion/drug abuse
- Fever ≥ 38°C
- Haematologic causes
  - Arterial emboli
  - Pulmonary infarct
  - Mycotic aneurysms
  - Intracranial haemorrhage
  - Conjunctival haemorrhage
  - Janeway lesions.
- Immunologic
  - Glomerulonephritis
  - Osler's nodes
  - Roth spots, RF
- Microbiologic evidence
  - Positive blood culture but not meeting major criteria or Serologic evidence of infection

Treatment

- Antibiotics based on Blood culture and sensitivity:
- Streptococci / Enterococci – Penicillin G: 2 – 3 million units I.V. 4th hourly along with Gentamycin 1 mg/kg I.V. 8th hourly for 2 weeks
- Enterococci: 4 to 6 weeks.
- Staphylococci: Oxacillin 2 g i.v. 4th hourly for 4 – 6 weeks
- If Methicillin resistant: Vancomycin 15 mg/kg I.V. 12 hourly for 4 to 6 weeks
- HACEK – Ceftriaxone 2 g/d I.V. for 4 weeks

Prophylaxis

Dental procedures wherein there is manipulation of gingival tissue or periapical regions of the teeth or perforation of the oral mucosa (including surgery on the respiratory tract)

Conditions for which prophylaxis is advised

- Prosthetic heart valves
- Prior endocarditis
- Unrepaired cyanotic congenital heart disease, including palliative shunts or conduits
- Completely repaired congenital heart defects during the 6 months after repair
- Incompletely repaired congenital heart disease with residual defects adjacent to prosthetic material
- Valvulopathy developing after heart transplant

Antibiotic regimens for prophylaxis

- Standard oral regimen
  - Amoxycillin 2 gm 1 hour before procedure
- Inability to take oral medication,
  - Inj. Ampicillin 2gm IV/IM 1 hour before procedure
- Penicillin allergy
  - Clarithromycin or Azithromycin 500 mg 1 hour before procedure
  - Cephalexin 2 gm, 1 hour before procedure
  - Clindamycin 600mg, 1 hour before procedure
- Penicillin allergy, inability to tolerate oral medication
  - Cefazolin or Ceftriaxone 1 gm IV/IM, 30 min before procedure
  - Clindamycin 600 mg IV/IM, 1 hour before procedure

Acute Pericarditis

Definition

Inflammation of Pericardium

Classification

- Acute pericarditis - < 6 weeks
- Subacute pericarditis - 6 weeks to 6 months
- Chronic pericarditis - > 6 months

Causes

Infections

- Viral
  - * Coxsackievirus A and B
  - * Mumps
  - * ECHO virus
  - * Adenovirus etc.,
- Pyogenic
  - * Streptococcus
  - * Pneumococcus
  - * Staphylococcus
  - * Tuberculosis
- Fungal
  - * Histoplasmosis
  - * Coccidiodomycosis
  - * Candida albicans

Non - Infectious:

- * Acute myocardial infarction
- * Post myocardial infarction (Dressler's syndrome)
- * Uremia
- * Neoplasia
- * Myxoedema

Acute Pericarditis

- * Aortic dissection
- * Rheumatic fever
- * Collagen vascular diseases
- * Post radiation
- * Trauma
- * Drug induced
  - » Procainamide
  - » Hydralazine
  - » Phenytoin
  - » Isonicotinic acid hydrazide
  - » Minoxidil

Symptoms

- Chest pain
  - * Retrosternal or left sided relieved by sitting up and leaning forward and intensified by lying supine.
- Pericardial friction rub
  - * High pitched, scratching.
- ECG
  - * Widespread ST–elevation with upward concavity.
- Echocardiography
- Most effective technique available.

Treatment

Usually with antibiotics patients have to be carefully observed as they may develop Pericardial effusion. If cardiac tamponade develops, Peri-cardiocentesis must be carried out at once.

Cardiomyopathy

Definition

The cardiomyopathies are a group of diseases that affect the heart muscle itself and are not the result of hypertension, congenital or acquired valvular, coronary or pericardial abnormalities.

Types

- Dilated cardiomyopathy
- Restrictive cardiomyopathy
- Hypertrophic obstructive cardiomyopathy (HOCM)

Causes

- Idiopathic
- Familial
- Infective – mostly viral, bacterial
- Metabolic causes
- Storage disorders – Glycogen storage disorder.
- Deficiency – Electrolytes, Nutritional
- Connective tissue disorders – SLE, RA
- Neuro muscular – Muscular dystrophy, Myotonic dystrophy
- Toxic
  - * Alcohol
  - * Radiation
  - * Drugs
- Peripartum heart disease

Dilated

- Left and right ventricular enlargement
- Impaired systolic function
- Congestive HF, arrhythmias, emboli.

Restrictive

- Endomyocardial scarring
- Myocardial infiltration resulting in restriction to left and or right ventricular filling.

Hypertrophic

- Disproportionate left ventricular hypertrophy, typically involving septum more than free wall, with or without an intraventricular systolic pressure gradient, usually of a non-dilated left ventricular cavity.

Note

- Sudden cardiac death can occur.
- Double or triple apical impulse

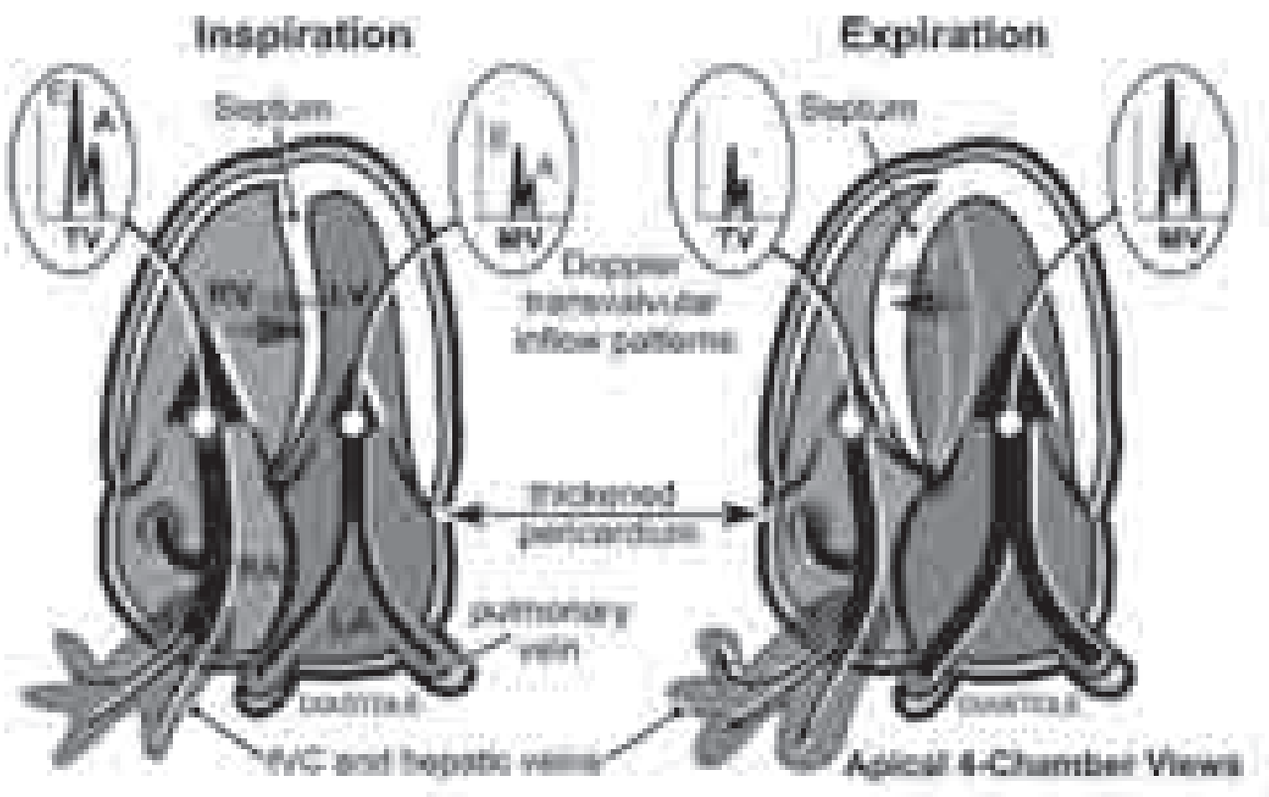
Symptoms

As in Congestive Heart Failure (CHF)

Treatment

- Usual treatment as in CHF
- β-blockers are useful
- Spironolactone can be added

Constrictive Pericarditis



- Chronic anticoagulation can be done

Drugs to be avoided

- Calcium Channel blockers and NSAIDS – in dilated cardiomyopathy
- Digitalis, Nitrates, Vasodilators, β-agonists – (best avoided in HOCM - Hypertrophic obstructive cardiomyopathy)

Hypertension

Definition

- Defined as blood pressure elevation to a level that places the patient at an increased risk for target organ damage
- Blood pressure is considered normal, if blood pressure is less than 120/80 mm Hg.
- High blood pressure may be due to systolic or diastolic or both. Pre-hypertensive 120-139 / 80-89 mm.Hg.
- High Blood Pressure may be of
  - * Stage I : 140-159/90-99mm Hg
  - * Stage II : ≥ 160 / ≥ 100 mm Hg.
- High blood pressure may produce a hypertensive crisis which includes hypertensive emergencies or urgencies.
- If diastolic blood pressure is 120 to 130 mm.Hg, with no target organ damage it is a **hypertensive urgency.**
- If diastolic blood pressure > 130 mmHg and systolic blood pressure >210 mmHg with evidence of target organ damage, it is a **hypertensive emergency** which requires immediate blood pressure reduction..
- Sometimes only systolic blood pressure may be very high (>_140 mm Hg) and this is called isolated systolic hypertension.
- When there is no causative factor it is called **Essential** hypertension. Hypertension may be labile or stable.
- If Hypertension is due to certain systemic diseases it is called **secondary hypertension** which includes renal disease, endocrine disease, vascular disease, etc.,

Symptoms

- Blood pressure elevation is usually discovered in asymptomatic period during routine screening or during certain associated conditions
- Blood pressure recording should be done with a properly calibrated blood pressure apparatus with sitting, supine and standing positions under a non-stressful situation. Minimum two records and both upper limbs or one lower limb record is also necessary.
- Before diagnosis, recording the history is very important which includes family history, drugs,diabetes, duration of hypertension, age at which blood pressure is first detected and other symptoms.
- The physician should carefully look for physical signs such as evidence of target organ damage, carotid bruit, abdominal bruits, pulse deficit, S3 / S4 gallop, cardiac murmurs, JVP, rales, enlarged kidney, peripheral pulses, flushing, markers of hyperlipidemia, obesity, cushingoid features, vessel wall thickening, etc.

Investigations

All hypertensive patients should undergo the following laboratory tests to identify target organ damage or to assess the adverse effect of drug therapy. Tests include

- * Urine analysis
- * Hematocrit
- * Plasma glucose
- * Serum potassium and sodium
- * Blood urea and creatinine
- * X-ray chest
- * Electrocardiography and
- * Lipid profile.

- If secondary hypertension is suspected certain extra investigations are mandatory which include
  - * Serum calcium, phosphorus
  - * TSH
  - * 24 hour urine meta nephrines or VMA, plasma free meta nephrines
  - * USG Abdomen with renal artery Doppler.
- The following differential diagnosis must be ruled out before establishing the diagnosis of hyper-

tension.

* Drug withdrawal,
* Acute stress
* White coat hypertension
* Drug-induced hypertension
  - » Steroid
  - » NSAIDS,
  - » Cocaine
  - » Opioid drugs.

- Sudden withdrawal of anti-hypertensive drugs also will lead to rebound hypertension.
- Any chronic hypertensive patient is examined for clinical evidence of cardiac failure, renal failure, coronary artery disease, cerebro vascular disease.

Rx Treatment

Once blood pressure is confirmed, lifestyle modifications (LSM) need to be outlined to all hypertensive patients whether they need medication or not which includes smoking cessation, stress reduction, judicious use of alcohol, body weight reduction, regular exercise and adequate intake of minerals and vitamins.

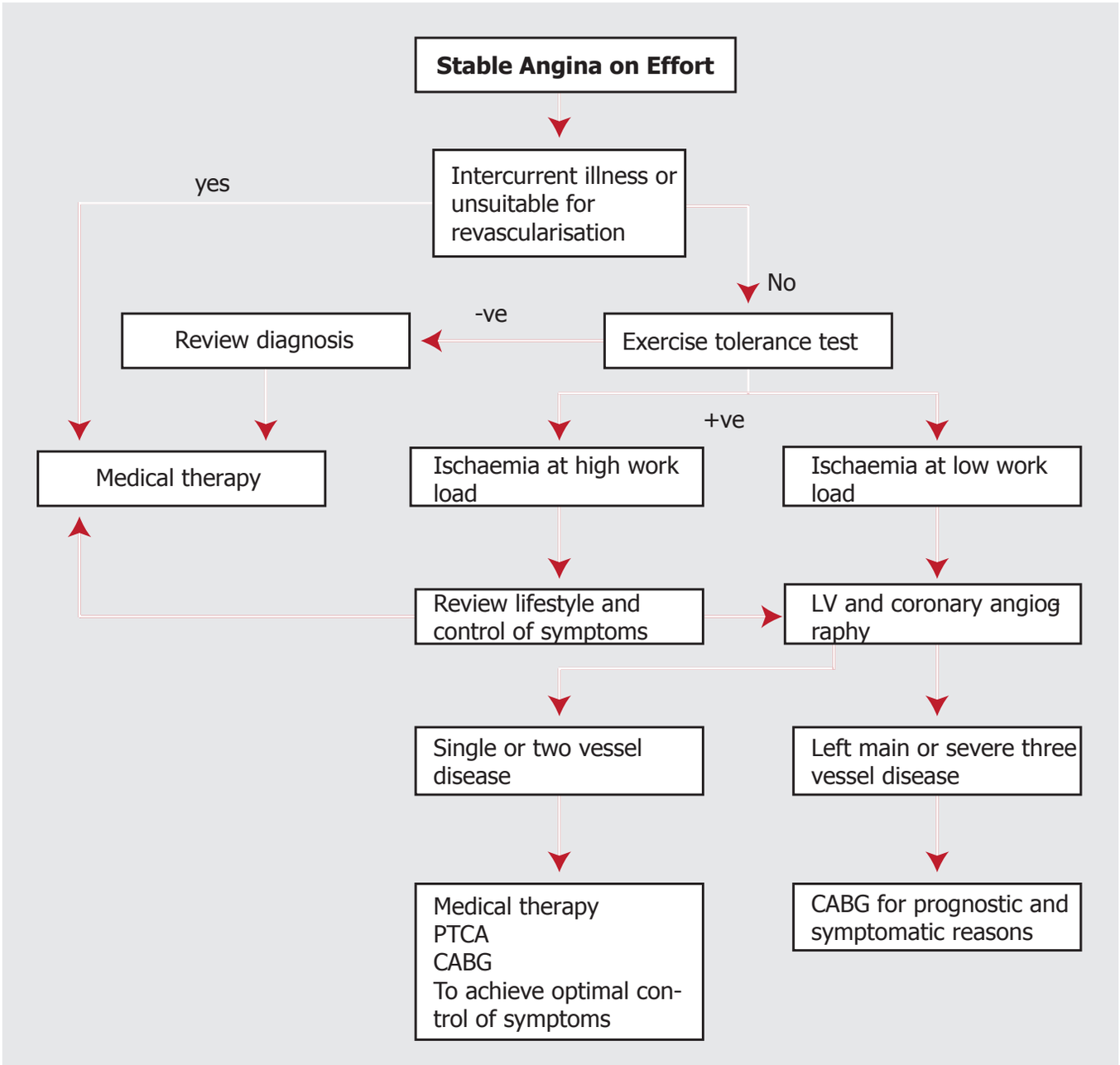
- If cause for hypertension is detected eg., renal, endocrine, drug, etc., it is to be treated appropriately.
- Pre-hypertension behavioral modification follow up
  * Stage I - Thiazide diuretics and calcium channel blockers (or) Beta blockers + ACEI
  * Stage II - Thiazide + ACEI + CCB + beta blockers(Dosages to be adjusted according to blood pressure response)
- Drugs also to be added depending upon coexisting factors such as angina, tachycardia, bronchospasm, renal insufficiency, obesity, LVF, hyper lipidemia, hyper urecemia, diabetes, cost and drug interaction; poor compliance also to be taken into consideration.
- Anti-hypertensive treatment is called for in certain special situations such as hypertensive emergency and urgency
- Intravenous anti-hypertensive drugs such as sodium nitropruside, Nitroglycerine, Labetalol, Esmolol, etc., should be given carefully with correct calculated dose.
- Special care should be given when patient presents with renal failure, stroke, rebound hypertension, pregnancy, aortic dissection, or acute myocardial infarction
- The public health problem of hypertension is enormous and patient education is essential
- Proper treatment is mandatory and will definitely reduce morbidity and mortality.

Angina pectoris

Risk factors for Coronary Artery Disease

- Non-modifiable (fixed)
  * Age
  * Male preponderance
  * Family History
- Modifiable
  * Smoking
  * Hypertension
  * Lipid disorders
  * Diabetes mellitus
  * Haemostatic variables
  * Sedentary life style
  * Stress
  * Obesity / Over weight
  * Dietary deficiencies of antioxidant vitamins and Polyunsaturated fatty acids.

Management of stable angina on effort



Medical therapy may be the only option if there is very poor LV function or diffuse disease
PTCA: Percutaneous Transluminal Coronary Angioplasty
CABG: Coronary Artery Bypass Grafting

Health promotion in Coronary artery disease

- Avoid smoking
- Regular exercise
- Maintain 'ideal' body weight
- Eat a mixed diet rich in fresh fruit and vegetables
- Aim to get no more than 30% of energy intake from fat

Myocardial infarction (MI)

Definition

Myocardial Infarction is due to the formation of occlusive thrombus at the site of rupture of an atheromatous plaque in a coronary artery.

Symptoms

- Chest Pain – Retrosternal / Substernal / Epigastric region
- Prolonged Cardiac Pain – referred to chest, throat, jaws, arms, epigastrium or back associated with sweating, pain on exertion and relieved by rest
- Anxiety, Angor animi (fear of impending death)
- Nausea and vomiting
- Breathlessness
- Collapse / syncope

Physical Signs

- Signs of sympathetic activation, pallor, sweating, tachycardia
- Signs of vagal activation
  - Bradycardia, vomiting
- Signs of impaired myocardial function:
  - Hypotension, Oliguria, cold peripheries
  - Narrow pulse pressure
  - Raised JVP
  - Third heart sound
  - Quiet first heart sound
  - Diffuse apical impulse
  - Lung crepitations
- Signs of tissue damage – Fever
- Signs of complications
  - Ventricular fibrillation
  - Ventricular ectopics
  - Mitral regurgitation
  - Pericarditis
  - Atrial Fibrillation
  - Heart Block.

Treatment

- Bed rest
- Oral Aspirin – 150 – 325 mg
- High flow oxygen
- Intravenous access
- Intravenous Analgesics with opiates
- Intravenous Morphine sulphate 10 mg or Diamorphine 5 mg
- Intravenous Antiemetic
  - Cyclizine 50 mg or Prochlorperazine 12.5 mg
- Consider thrombolysis with intravenous Streptokinase or Alteplase
- Defibrillation therapy if necessary

Streptokinase

- 1.5 million units in 100 ml of saline as an intravenous infusion over 1 hour
- The drug may cause hypotension, which can be managed with fluids and restarting the infusion at a slower rate
- The drug is antigenic.

Alteplase

htpa (human tissue plasminogen activator)

The regimen is given over 90 minutes (bolus dose of 15 mg followed by 0.75 mg/kg of body weight and then 0.5 mg/kg body weight over 60 minutes).

- Consider intravenous β – adrenoreceptor antagonist Inj. Metoprolol 5 mg IV every 2-5 minutes for 3 doses and changed to oral Metoprolol 50 every 6 hours for 48 hours, followed by 100 mg every 12 hours
- Monitor ECG and cardiac enzymes
- Detect and treat complications early

Nitrates

* Sublingual glyceryl trinitrate 300-500 µg
* Intravenous nitroglycerine 0.6 – 1.2 mg/hour
* Isosorbide dinitrate 1 – 2 mg/hour

Anti-coagulants

Subcutaneous Heparin 12,500 units twice daily for 7 days is given in addition to oral aspirin may prevent re - infarction after successful thrombolysis and reduce the risk of thrombo-embolic complications.

Contraindications to thrombolytic therapy

Absolute contraindications to thrombolytic therapy

- Active internal bleeding
- Previous sub-arachnoid or intra-cerebral haem-orrhage
- Non-haemorrhagic stroke within the last 1 year
- Uncontrolled hypertension (systolic >180mmHg,diastolic>110mmHg)

Relative contra-indications to thrombolytic therapy

- Current use of anti-coagulants
- Recent (<2 weeks) invasive surgical procedure
- Recent prolonged (>10 min) cardiopulmonary resuscitation
- Known bleeding diathesis
- Pregnancy
- Hemorrhage prone ophthalmic conditions.
- Active peptic ulcer disease
- Recent history of severe hypertension

Streptokinase should not be used, if given in the preceeding 5 days to 2 years because of the risk of allergic reactions

Congestive Heart Failure (CHF)

Definition

CHF is a clinical syndrome of inadequate cardiac output resulting in fluid retention in the lungs, abdominal organs and peripheral tissue.

Symptoms

- Fatigue, listlessness

CXR-Pulmonary oedema

- Poor effort tolerance
- Decreased urine output
- Breathlessness and pink frothy sputum
- Swelling around ankles

Signs

- Cold peripheries
- Low blood pressure
- Oliguria, Uraemia
- Orthopnoea
- Paroxysmal Nocturnal Dysnoea (PND)
- Bilateral basal inspiratory crepitations
- Raised JVP
- S_3 summation gallop
- Peripheral oedema
- Ascites
- Pleural effusion
- Profuse pink frothy sputum
- Tender hepatomegaly

Factors that may precipitate or aggravate heart failure

- Myocardial ischaemia or infarction
- Intercurrent illness (eg) Infection

- Arrhythmia
- Inappropriate reduction of therapy
- Drugs :– β – adrenoreceptor antagonist, Drugs with fluid retaining property (NSAIDS, Steroids)
- Pulmonary embolism
- Conditions with increased metabolic demand
  - Pregnancy
  - Thyrotoxicosis
  - Anaemia
- Intravenous fluid overload

Compensatory changes in heart failure

- Chamber enlargement
- Myocardial hypertrophy
- Increased heart rate

Rx Treatment

General measures

- Restrict physical activity
- Bed rest in propped up position with a back rest
- Dietary salt (sodium) restriction. (2 – 3 g/day)
- Avoid and discontinue NSAIDS, high dose Beta blockers and calcium channel blockers
- Fluid restriction depending on output and other conditions.

Drug therapy

- Diuretics
- Vasodilators
- ACE inhibitors/Angiotensin II receptor blockers
- Digoxin
- β-adrenoceptor antagonist
- Other drugs – Heparin, Anti coagulants to prevent thromboembolism complicating atrial fibrillation.

Diuretics

- Inj. Frusemide 40 – 80 mg IV stat every 2 to 3 hours with maximum up to 200 mg/day. Maintenance dose is 40 mg IV 12th hourly.
- Tab. Spironolactone 25 – 200 mg / day (or)
- Tab. Chlorthiazide 250 – 500 mg/day (or)
- Tab. Indapamide 2.5 - 5 mg/day (or)
- Tab. Benzthiazide 25 mg + Tab. Triamterene 50 mg/day

Vasodilators

- Venodilators – Organic nitrates
- Iso-sorbide mononitrate

Angiotensin – converting enzyme (ACE) inhibitors

- Tab. Enalapril 2.5 – 20 mg/day (or)
- Tab. Lisinopril 2.5 – 10 mg/day

Angiotensin II receptor antagonist

- Losartan 50 mg once daily

Digoxin

- Is indicated in fast ventricular rate (e.g.) Atrial Fibrillation
- Tab. Digoxin 0.5 mg first day followed by 0.25 mg/day

Heparin

- Inj. Heparin 5000 IU 12 hourly SC if patient is bedridden
- Beta blockers
  - Tab. Carvedilol 3.125 – 25 mg per day in one or two divided doses is useful in all symptomatic or asymptomatic heart failure and in case of reduced ejection fraction (< 40 %.)

Monitoring of CHF patient

- Strict intake – output charting
- Daily weight and abdominal girth measurement
- Serum electrolytes and uric acid
- Watch for any **symptoms of digitalis toxicity**
  - Anorexia
  - Nausea
  - Vomiting
  - Worsening of heart failure.

Pulmonary Embolism

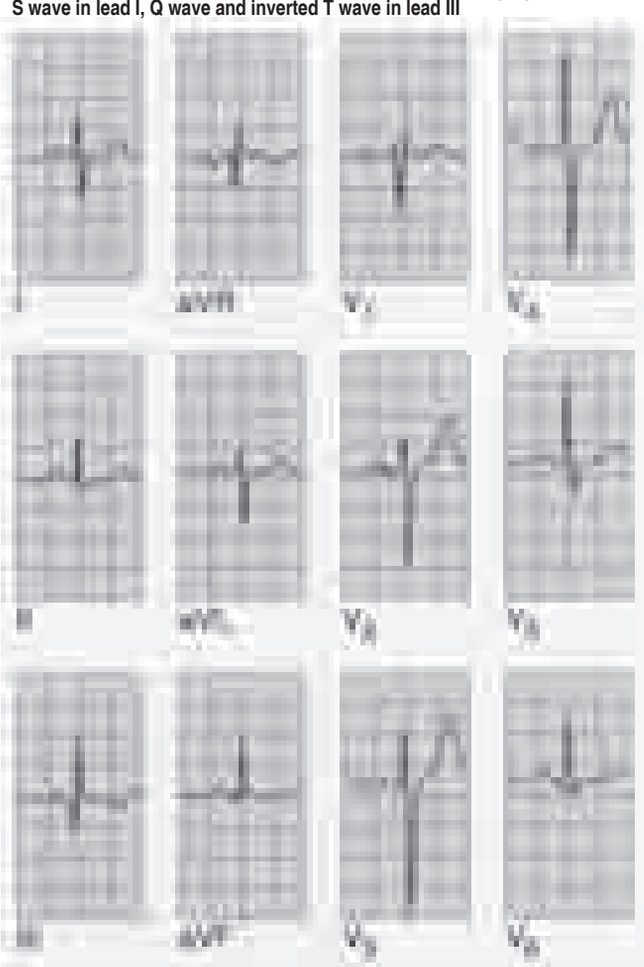
Causes

Risk factors

- Patient factors
  - * Age, obesity, long air travel, immobility (> 4days)
  - * Smoking, Hypertension, Diabetes Mellitus
  - * Pregnancy, Puerperium, Thrombophilia
  - * Anti-phospholipid antibody syndrome
  - * Hypercoagulable states such as
    - » Protein C, Protein S deficiency
    - » Hyper-homocysteinemia etc.
  - * Drugs such as those given for hormone replacement therapy.
- Disease or surgical procedures
  - * Trauma or surgery, especially of pelvis, hip or lower limbs
  - * Cardiac failure

Pulmonary embolism - ECG showing $S_1 Q_3 T_3$ pattern

S wave in lead I, Q wave and inverted T wave in lead III



  - * Malignancy
  - * Nephrotic syndrome
  - * Stroke
  - * Indwelling central venous catheters etc.

Symptoms

- Depends on size of the embolism and associated co-morbidity
- Sudden onset dyspnoea is the most common symptom
- Tachypnoea is the most common sign
- Pleuritic chest pain and haemoptysis are present when infarction has occurred
- Hypotension with acute right ventricular failure in a patient with risk factors of pulmonary embolism

Clinical syndromes of Pulmonary embolism

- * Massive pulmonary embolism
- * Sub-massive pulmonary embolism
- * Small to moderate pulmonary embolism
- * Pulmonary infarction
- * Paradoxical embolism
- * Non-thrombotic embolism
- * Multiple recurrent pulmonary embolism

Features

- * Cardiogenic shock
- * RV hypokinesia, normal blood pressure
- * Normal RV function, normal blood pressure
- * Features of lung consolidation
- * Stroke with pulmonary embolism
- * Commonly air, fat, amniotic fluid, tumour, right sided infective endocarditis
- * Increasing breathlessness over weeks or months

Investigations

- Non-imaging methods
- Imaging methods

Non-imaging methods

[Suspicion of the illness is the most important first step]

Test

- Plasma d-DIMER ELISA
- ABG- Arterial blood gas analysis
- ECG

Comment

- If undetectable, excludes pulmonary embolism
- Type I respiratory failure
- Sinus tachycardia (common)
- RV strain
- Anterior T wave inversion,
- $S_1Q_3T_3$ pattern in ECG

Imaging methods

- Chest X-ray
  - * Can be normal
  - * Cardiomegaly, wedge-shaped opacity, effusion, oligemia, abrupt cut-off of pulmonary artery
- Echocardiography
  - * Detects RV overload and function
- CT Scan (Multi detector – row CT Scan)
  - * Can detect even small pulmonary artery emboli
  - * Can surplant the pulmonary angiography
- Pulmonary Angiogram
  - * Gold standard test,
  - * Done if surgery is planned.
- MR Angiogram
  - * Useful if CT Angiogram is contra indicated
- Venous Ultrasonography
  - * May show thrombosis of deep veins of the lower limb or pelvis
- Ventilation/Perfusion Scanning
  - * Reserved for patients with Renal failure / Contrast allergy

Rx Treatment

Management of Acute pulmonary embolism

- Oxygen therapy / NSAIDs for pain relief
- Avoid Diuretics, vasodilators
- Fluid / Dopamine support if hypotension is present
- Thrombolytic therapy (Streptokinase): In pulmonary embolism with cardiogenic shock
- Anticoagulation
  - * Heparin followed by oral anticoagulation
    - » Life long if there is pro-thrombotic risk
    - » For 3 months if an identifiable reversible risk factor is present
    - » For 6 months in idiopathic pulmonary embolism
    - » Heparin: LMWH is preferred Enoxaparin: 1 mg/kg 12 - hourly Dalteparin : 200 IU/kg sc daily
    - » Warfarin 5 mg (To maintain INR between 2-3)
  - * Inferior Venacava filters: useful in recurrent pulmonary embolism
  - * Surgical embolectomy
  - * Identify the risk factors and treat
- Prophylaxis for Venous thrombo-embolism:
  - * Preventive measures such as early mobilization, intermittent pneumatic compression, graded stock compression.
  - * Anticoagulation prophylaxis (To be combined with intermittent pneumatic compressions and graded stock compressions)

Examples

Anticoagulation prophylaxis	Drugs
Hip/ Knee replacements Hip or Pelvic fracture	Warfarin (INR: 2 - 2.5) For 4 - 6 weeks LMWH (Enoxaparin) 30 mg sc B.D
Gynaec cancer surgery	Warfarin (INR: 2 -2.5) Enoxaparin 40 mg sc OD
Neurosurgery, Ophthalmic surgery or any other surgeries where anti coagulation is contraindicated	Intermittent pneumatic compressions and graded stock compressions
Medical conditions	Compression stockings Heparin 5000 IU q8h Enoxaprin 40 mg sc OD

Cardiac arrhythmias

Causes

- Due to disorders of impulse formation (or)
- Due to disorders of impulse conduction
- Arrhythmia can be:
  - * Brady arrhythmia or Tachy arrhythmia
  - * Paroxysmal or persistent
  - * Hemodynamically stable or unstable

Symptoms

Patients present with palpitations, light headedness, hypotension, dyspnoea, angina, syncope or fatigue. Sudden onset and abrupt termination of palpitations may indicate tachyarrthymia.

History of congenital (Hypertrophic Cardiomayopathy,WPW syndrome, congenital long QT syndrome) or acquired structural heart disease (Ischemic Heart Disease, Cardiomyopathy, valvular or endocrine disease (Thyroid disorder, adrenal disorders) should be obtained.

Investigations

Physical examination includes pulse, Blood pressure, detailed cardio-vascular examination.

Investigations include – 12 lead ECG, continuous ambulatory ECG (Holter) monitoring for 24 – 72 hours event recorder, His Bundle Electrogram, Excercise ECG, EPS (Electro physiology study).

Treatment

Treating the acute attack of arrhythmia

1. Treating the precipitating causes
2. Treating the underlying causes
3. Long term management, if necessary

Cardiac arrhythmias

Brady Arrhythmia

Definition

Any heart rhythm with a rate less than 60 beats/min.

Causes

1. Sick sinus syndrome (caused by SA node dysfunction)

   Presents as

   - * Symptomatic sinus brady cardia
   - * Sino atrial block
   - * Sinus arrest

2. A.V. conduction disorders:
   - * First degree A V Block: Prolongation of PR interval
   - * Second degree A.V.Block

   c. Mobitz Type I Block (Wencke back's phenomenon)
      - » Progressive prolongation of PR interval.
      - » Periodically one P wave is not followed by QRS complex.

   d. Mobitz Type II block
      - » Periodically P wave is not following by QRS complex

5. Third degree A V Block
   - * A V disosiation
   - * Independent atrial and ventricular rhythm

Treatment

In symptomatic acute situation, the heart rate should be increased to alleviate the symptoms by:

- Inj. Atropine 0.5 mg – 2 mg I.V (or)
- Inj. Isoproterenol 1 – 4 micro gram/min I.V.
- On a long-term basis, the appropriate patients have to be provided with a permanent pacemaker.

Tachyarrhythmia

Definition

Any heart rhythm with a rate more than 100 beats/min

Types

They may be: Supraventricular (or) Ventricular

Supraventricular tachycardia

They occur paroxysmally. Onset is sudden with abrupt termination.

Symptoms

- They may be asymptamatic or symptomatic.
- Patients present with palpitation, dizziness, angina.
- Hypotension may be present.
- Polyuria may follow after the acute episode.

Investigations

1. ECG:
   * SVT: Regular narrow QRS Tachycardia. Ventricular rate may be 160 – 200 beats/ min. P wave may be buried in QRS or follow QRS complex.
   * SVT with aberrancy: Regular wide QRS Tachycardia.

Mechanisim:

1. AVNRT (A.V. Nodal Re-entrant tachycardia)
2. O – AVRT (Orthodromic A.V. re-entrant tachycardia using accessory pathway in WPW syndrome)

Treatment

Depends on the haemodynamic status of the patient. If he is haemodynamically stable:

1. Treatment of acute episode includes vagal manoeuvres (Carotid sinus massage, valselva maneuver)
2. If this measure fails, AV Nodal blocking drugs are tried.
   * Inj. Adenosine 6 mg IV bolus followed by 10 ml saline flush.12 mg IV bolus can be repeated if no response after 2 minutes.
   * Contra indications: Sick sinus syndrome or second or third degree A.V. Block
3. Inj. Metoprolol 5 mg IV. Can be repeated after 5 minutes
4. Inj. Verapamil 5 – 10 mg IV bolus over 2 – 3 minutes. Can be repeated after 15 minutes
5. Inj. Diltiazem 0.25 mg/kg IV bolus over 2 minutes. Followed by IV infusion at a rate of 10 mg/ hour.
6. If haemodynamically unstable, prompt synchronized direct current (DC). Cardio version (using 10 to 100 Joules) has to be done.

Maintenance therapy

Once sinus rhythm is established, chronic maintenance therapy includes

1. Oral calcium channel blockers
   * Diltiazem – 30 mg tid (or)
   * Verapamil – 40 mg tid
2. Beta-adrenergic antagonist
   * Metoprolol 25 – 100 mg bid
   * Atenolol 25 – 100 bid
   * Digoxin 0.125 – 0.5 mg od

Curative Therapy

Curative therapy for supraventricular tachycardia involving accessory pathway is radio-frequency ablation.

Ventricular tachycardia

Definition

Runs of three or more consecutive idioventricular beats exceeding a rate of 100 beats/minute.

Types:

* Sustained - If ventricular tachycardia persists for > 30 seconds.
* Non-sustained - 3 beats to 30 seconds.
* Monomorphic - Configuration is uniform
* Polymorphic - Configuration varies from beat to beat

Symptoms

Patients with ventricular tachycardia may present with stable haemodynamic status or features of hypotension and syncope or pulseless state.

Investigations

Echo and electrocardiography

AV Dissociation, capture or fusion beats and an LBBB morphology with right axis deviation.

QRS morphology in precardial leads in VT

	Lead V_1	Lead V_6
If there is LBBB pattern	R > 30 milli seconds	QR or QS
	R to S > 60 milli seconds	
	Notched S wave	
RBBB pattern	Monophasic R	R/S < 1
	QR or RS	

Treatment

1. Patients with non-sustained VT without organic cardiac disease are asymptomatic – need not be treated.
2. Patients with sustained VT in the absence of car-

diac disease are symptomatic and require therapy with Beta blockers, verapamil, Class IA, IC or III or amiodarone.

3. In patients with VT with organic cardiac disease, if there is haemodynamic instability, unsynchronized 200 Joules DC cardioversion will terminate the arrhythmia.
4. If haemodynamically stable, the following drugs can be tried:
   * Inj. Lidocaine – 1 mg/kg IV bolus, 0.5 – 1 mg /Kg boluses may be repeated at 5 min intervals followed by 2 – 4 mg/min maintenance infusion.
   * Inj. Amiodarone may be tried.
   * If these drugs fail, over drive pacing has to be done.
   * To prevent recurrences, sustained VT – ICD implantation is effective.

Prognosis

* Prognosis of ventricular tachycardia depends on the underlying heart disease. Ventricular tachycardia and supraventricular tachycardia with aberrancy are to be differentiated, as the therapy is different in both conditions.

Atrial fibrillation

Common arrhythmia we see in practice

Types:

1. Paroxysmal
2. Persistent
3. Permanent
4. Acute (or) chronic

Causes

Seen in the following conditions:

* Patients with valvular heart disease
* Ischaemic and hypertensive heart disease
* Chronic lung disease, hypoxia, hypercapnia
* ASD
* Thyrotoxicosis
* Lone atrial fibrillation occurring in patients without heart disease
* Acute alcoholic intoxication
* Emotional stress, exercise

Symptoms

- Palpitation, hypotension, angina, syncope or embolic manifestations.
- Pulse : irregularly irregular
- JVP- Loss of 'a' waves.
- Varying intensity of first heart sound.

Investigations

- ECG: No discrete 'p' wave, only 'f' waves are seen
- Varying RR interval
- Rate 350 to 600 beats/minute

Treatment

Acute atrial fibrillation

1. Treating the precipitating factors.
2. If the patient is haemodynamically unstable, electrical cardioversion using 200 Joules is the treatment of choice.
3. If haemodynamically stable, ventricular rate control is adequate by beta blockers or calcium channel blockers.
4. Next step is to convert atrial fibrillation to sinus rhythm. It consists of anti-coagulant prior to cardioversion and following the cardioversion and use of antiarrhythmic drugs to restore sinus rhythm.

Chronic atrial fibrillation:

1. Chronic atrial fibrillation cannot be converted to sinus rhythm or will not remain in the sinus rhythm.
2. In this case, rate control is sufficient by beta blockers, calcium channel blockers or digitalis.
3. To prevent recurrence of atrial fibrillation, Sotalol and Amiodarone and can be used.
4. To prevent the risk of embolism, either Aspirin or Warfarin can be used depending upon the presence of risk factors.

Surgical methods

1. Ablation around pulmonary veins.
2. MAZE procedure, to create zig zag surgical scars in the atria.
3. Catheter ablation of atrioventricular node with pace maker implantation.

Haematology

Standard Treatment Guidelines
Tamil Nadu Health Systems Project

Chapter 4

- Anemia
  - Microcytic Anemia
  - Normocytic Anemia
  - Macrocytic Anemia
- Haemolytic Anemia
- Approach to a bleeding patient
- Platelet Disorders
- Acute ITP
- Coagulation Disorders
- General Considerations

Anaemia-approach to diagnosis

Definition

Anaemia is defined as a reduction in the number of red blood cells, blood hemoglobin content, or hematocrit, below the normal level for the same age and sex.

There are three primary causes of anaemia:

1. Reduced production of red blood cells in the bone marrow
2. Excessive destruction of red blood cells, and
3. Excessive blood loss.

When to suspect anaemia?

When there are symptoms and signs of weakness, fatigue, palpitations, tachycardia, dyspnea, positional dizziness, syncope, increased or new onset angina.

The diagnostic approach consists of

1. History
2. Physical examination and
3. Laboratory evaluation.

History

- Blood loss, intake of Aspirin or other NSAIDs,
- Menstrual history or possible pregnancy in women
- Dietary history, including history of pica
- History of alcohol abuse
- Family history
- History of gastric surgery, distal paresthesias, gait problems -consider B12 deficiency
- History of jaundice, transfusion, new medication, infection
- History of weight loss, fever and chills, cough, dyspnea,
- Other diseases like cancer, HIV, rheumatoid arthritis, thyroid disease and renal disease

Investigations

Initial laboratory tests for anaemia workup

- Complete blood count profile, including red cell morphology, white blood cell differential and reticulocyte count.
- Biochemistry profile-Renal and liver function tests.
- Urine analysis: Hematuria/proteinuria in renal disease, hemoglobinuria in hemolysis.

The Complete blood count and other related tests

Parameters in normal adult

1. Hemoglobin

   Male= 15.5 (+/- 2 mg/dl)

   Female = 13.5 (+/-2)
2. Hematocrit

   Male=46.0 (+/- 6%)

   Female=41.0 (+/- 6%)
3. Red blood cell count

   Male = 4.3 - 5.9 million/uL

   Female = 4.0 - 5.2 million/uL
4. White blood cell count 4500 - 11000 cells/uL
5. Platelet count 1.5 - 4.0 lakh cells/uL
6. Reticulocyte count 0.5 - 1.5 %

Red cell indicies

- Mean corpuscular volume 80 - 90 fl
- Mean corpuscular hemoglobin 27 - 32 picogram
- Mean corpuscular hemoglobin concentration 30 - 36 gm/dl
- Red cell distribution width 11.5 - 14.5

From a pathophysiological perspective, anaemia is either due to

- Decreased production
- Increased loss

Reticulocyte count

The reticulocyte count helps differentiate between these two types

- The reticulocyte must be corrected for the reduction in red cell count to accurately reflect marrow production of erythrocytes and is obtained by the following formula.

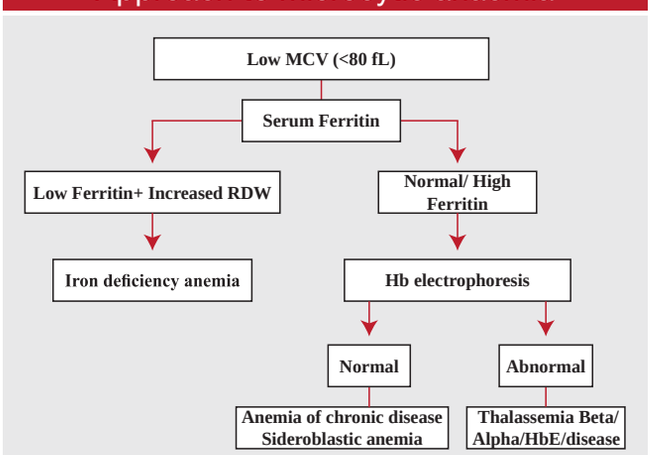
Corrected retic = observed retic x observed hematocrit (%)

- Normal hematocrit: 45

- The normal corrected reticulocyte percentage is 1-2%
- From a practical diagnostic perspective, anaemia is best classified according to Mean corpuscular volume (MCV) as
  - * Microcytic
  - * Macrocytic
  - * Normocytic

Classification of Anaemia based on Red cell size
Microcytic (MCV <80 fL) • Iron deficiency anaemia • Thalassemia • Congenital sideroblastic anaemia • Chronic inflammation • Lead poisoning
Macrocytic (MCV >100 fL) • Vitamin B12 deficiency • Folate deficiency • Liver disease • Hypothyroidism
Normocytic (MCV 80-100 fL) • See table below

Approach to microcytic anaemia



Treatment fo Iron deficiency anaemia

Aim of treatment

1. Correction of anaemia
2. Replenishmint of stores
3. Treatment of the cause of iron deficiency
   - * Transfusion of packed red cells is only indicated if there is significantly symptomatic and severe anaemia
   - * Ferrous sulphate given as non - enteric coated tablets 150 - 200 mg of elemental iron has to given daily in 1 - 3 divided doses. Adverse effects can be reduced by starting with single tablet per day and then increasing the dose slowly. The total duration of therapy is 6 months.
   - * Parenteral iron- Rarely required

Indications

- Malabsorption
- Inflammatory bowel disease

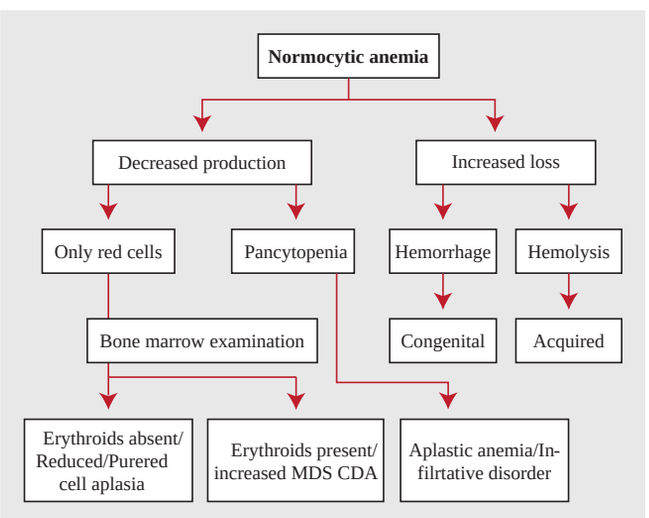
Note:

Iron surcrose can be administered as 200 mg in 100 ml of normal saline as infusion over 20 minutes.

Treatment of megaloblastic anaemia

- 1 mg of intramuscular Cyanocobalamin per day for 3 days, 1 mg weekly for 4 weeks and then 1 mg once in 3 months for 1 year for megaloblastic anaemia
- Megaloblastic anaemia should never be treated with folate alone as neurological disease is precipitated.

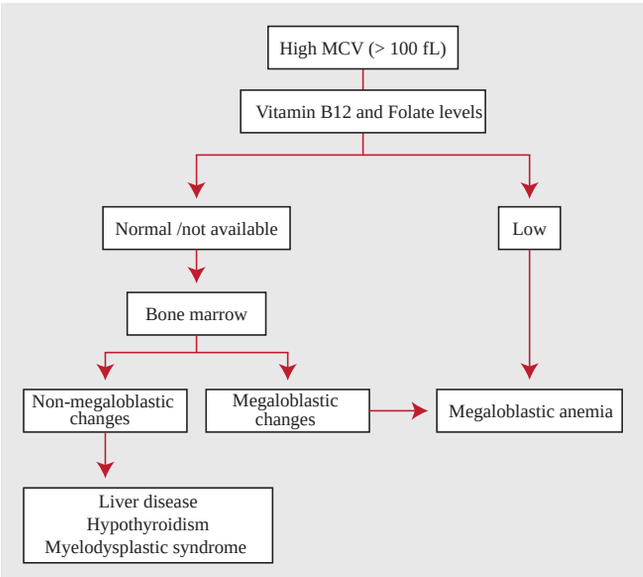
Approach to normocytic anaemia



Microcytic anaemias

An MCV of less than 80 fL with pale, poorly hemoglobinized red cells on peripheral smear characterizes this group of anaemias.

Approach to macrocytic anaemia



Hemolytic anaemia

Anaemia with high reticulocyte count systematic approach

1st step

Look for the evidence of haemolysis (clinical and laboratory)

2nd step

Immune versus Non-immune

3rd step

Peripheral smear examination to find a clue to the diagnosis.

4th step

Ask for confimatory test to establish the diagnosis.

Evidence of hemolysis

Clinical evidence

Chronic long standing from childhood

- Skeletal Abnomalities
  - * Frontal Bossing
  - * Macillary prominence
  - * Harrison's sulcus
  - * Genu valgum
- Jaundice with acholuric urine
- Hepatosplenomegaly
- Chronic leg ulcers

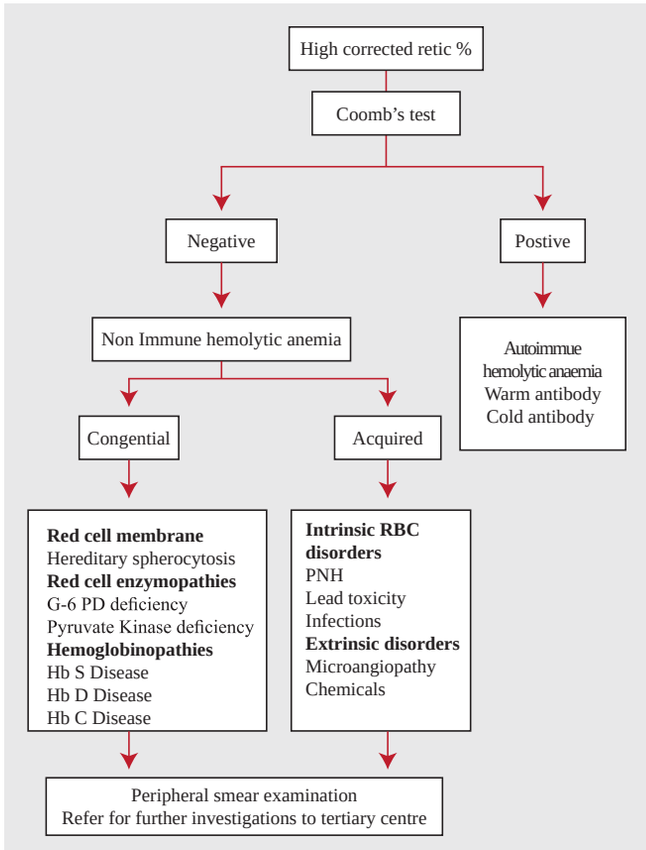
Investigations

Laboratory evidence

Hallmark of hemolysis is presence of an elevated reticulocyte count, with stable or falling hemoglobin.

- Other findings include:
  - * Elevated indirect bilirubin
  - * Elevated serum lactate dehydrogenase (LDH)
  - * Decreased Haptoglobin levels
  - * Hemoglobinemia and hemoglobinuria
  - * Erythroid byperplasia in bone marrow

Approach to hemolysis



Management of hemolytic anaemias

Congential Hemolytic Anaemia

Patients with hemolytic anaemia should not be given oral iron as this may siad to iron overload. Folic acid 205 mg / day should be administered.

Autoimmune Hemolytic Anaemia

Steroids are the mainstayof treatment for patients with autoimmune hemolytic anema. Start pred-nisolone 1 mg / kg / day for- weeks and then taper over- weeks. Steroids should not be continued beyond weeks.

Transfusion is warranted when cardiac or cerebral function is threatened.

If patient is steroid dependent or if there is inadequate response to steroids, Tab Azathioprine to be started at a dose of 1-2mg/kg body weight for 6 months, with monitoring of leukocyte count. Splenectomy to be considered, if not responding to steroids and azathioprine.

Investigations

Recommended laboratory Investigations in the facilities

LEVEL 1:

- Primary health center
  - * Hemoglobin estimation using a spun hematocrit or colorimeter. Preipheral smear examination.

LEVEL II:

- District hospital
  - * Bood cell counter with 3 or 5 part differential and reticulocyte count
  - * Coomb's test.

LEVEL II:

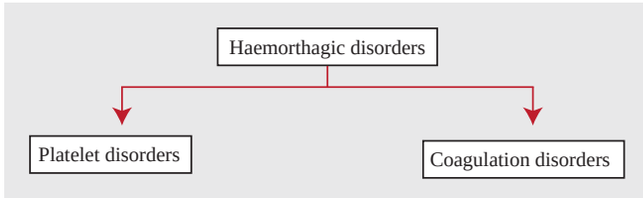
- Tertiary referral centre
  - * Blood cell counter with 5 part differential
  - * Facilities for bone marrow examination including trephice
  - * Serum iron TIBC and ferritin.
- Evaluation of hemolytic anaemia
  - * HPLC/Hb electophoresis
  - * Osmotic fragility
  - * Unstable hemoglobin
  - * Sickle preparation
  - * G6PD estimation
  - * Ham's test and
  - * Sucrose lysis test

Approach to the bleeding patient

Haemostasis may be defined as the process that maintains blood in a fluid state while preventing loss of blood from sites of vascular disruption. A normal haemostatic mechanism maintains a healthy balance between bleeding and clothing.

Primary haemostasis is the function of a platelet plug at sites of injury and occurs within seconds of injury.

Secondary haemostasis describes the coagulation system, resulting in fibrin formation. This is particularly important in bleeding originating from large vessels and in preventing recurrent bleeding hours or days after initial injury.



Acquired disorders are more common than inherited bleeding disorders.

Investigations

The investigation of a suspected bleeding tendency may begin from three differnt perspectives:

Investigating a clinically suspected bleeding tendency

- The investigation begins with history, which may suggest an acquired or congenital disorder of primay or secondary haemostasis
- If the bleeding hostory of family history is significant, appropriate specific testsw and assays should be performed.

Following up an abnormal first-line test

- Platelet Count (Normal range - 1.5 lakhs to 4.5 lakhs)
- Bleeding time (Normal range - 2 to 8 mins)
- Prothrombin time (Normal range - 11 to 16 secs)
- Activated partial thromboplastin time (Normal range - 26 to 40 secs)
- The abnormalities already detected will determine appropriate further investigations.

Investigations of acute hamostatic failure

- This is often required in the context of an acutely ill or postoperative patient
- Investigations are therefore directed toward detecting disseminated intravascular coagulation (DIC) or a previously undetected coagulation defect (congenital or acquired)
  - * The availability of a normal pre-morbid coagulation screen and further questioning to determine a bleeding history is extremely useful in

this situation.

Platelet disorders

Features:

- Purpura
- Bleeding time - prolonged
- Patelet count - normal / abnormal
- PT,APTT - normal
- Bleeding rarely occurs when counts > 50,000 / cu.mm
- < 50,000 / cu.mm - purpura
- < 20,000 / cu.mm - spontaneous bleeding including high risk mucosal bleeds and intracerebral haemorrhage

Inherited qualitative Platelet disorders:

- Bleeding (usually muco - cutaneous)
  - * Purpura
  - * Ecchymosis
  - * Menorrhagia
  - * GI bleeding since childhood
- Family history of bleeding tendencies may be postive.
- Bleding time - prolonged.
- Clot retraction - defective or absent.
- Platelet count and peripheral smear study for platelet aggregates and morphology
- PT, APTT - normal.
- (N.B: APTT could be prolonged in Von eillebrand disease
- Platelet aggregation studies - abnormal in Glanzmann Thrombasthenia and BSS.

Platelet Disorders

S.No:	Quantitative Platelet Disorders (Low platelets/thrombocytopenia)	Qualitative Platelet Disorders (Normal platelet count)
I	• Acute ITP	• Congenital * Glanzmann's-thrombasthenia * Bernard - Soullier syndrome
II	• Drug induced * Cytotoxic Drugs * Cephalosporins * Anti-mlarials * Rifampin	• Acquired * Uraemia * Liver disease * Myelo-proliferative disorders * Cardiopulmonary by pass * Drugs - Aspirin, Clopidogrel etc.
III	• SLE	
IV	• Infections * Malaria * Dengue * HIV - 1 * Infectious Mononucleosis	
V	• DIC	
VI	• Sepsis	
VII	• Thrombotic thrombocytopenic purpura	
VIII	• Hemolytic-uraemic syndrome	
IX	• Ethanol induced	
X	• Hereditary	

Treatment

- Non-transfusional haemostatic drugs and platelet transfusion are the mainstay of therapy in congenital disorders, for major bleeding.
- Aquired disorders require in addition, management of the primary problem.

Thrombocytopenia - non - immune

Points to remember :

1. Platelet transfusion
   * Random platelet transfusion- 1unit/ 10 kg body weight anticipated increase in platelets is 10,000 /cu.mm.
   * One unit of Single Donor Platelet (SDP) transfusion is equivalent to 6 units of Random Platele transfusion. Single Donor Platelets decreases alloimmunization.
2. Porphylactic Platelets :
   * Platelet counts < 10,000 / cu.mm with active bleeding or fever.
   * Platelet count < 50,000 / cu.mm if invasive procedures are contemplated.

Avoid

- Intra muscular injections
- Anti - Platelet drug- NSAIDS.

Acute Idiopathic Thrombocytopenic Purpura

- Bleeding (usually muco-cutaneous)
  * Purpura
  * Ecchymorrhagia
  * Gastro intestinal or urinary bleeding of short duration.
- Presence of underlying disease (eg. SLE) or history of drug intake.

Investigations

- Platelet count-very low
- PT, APTT - normal
- Bone marrow - Normal with adequate megakaryocytes.

Treatment

- Counts > 30,000 / ul
  * Corticosteroids (short course - 2 weeks) if bleeding and steriod responsive. No treatment, if no bleeding.
- Counts < 30,000 / ul
  * Without serious bleed
    » Prednisolone 1 mg / kg / day for 4 weeks and then taper over 2-4 weeks
    » Steroids should not be continued beyond 6-8 weeks.
  * With serious bleeding
    » Prednisolone 1 mg / kg / day plus
    » IV gamma globulin 1 gm / kg / day for 2 days (if possible), or
    » Rh-D immunoglobulin 50 - 75 mg / kg / day given (if possible) IV once in 3-4 weeks to maintain counts around safe level > 50,000 /ul, with appropriate precautions.
- Splenectomy to be considered in adults, if not responding to steroids.
- Other drugs useful in ITP
  * Azathioprine 1-2 mg / kg / day for 6 months
  * Dapsone 2 mg / kg / day for 6 months
- Those unresponsive to above therapies should be referred to a tertiary centre.
- Platelets transfusions are not indicated. However, if there is life threatening bleeding such as intracranial bleeding, then 4-6 platelet units, may be given, if available.

Coagulation disorders

Inherited coagulation factor deficiencies	Acquired coagulation disorder
Factor VIII deficiency -Haemophilia A Factor IX deficiency -Haemophilia B Von-Willebrand disease Rare coagulation disorder	DIC Liver disease Vitamin K deficiency Drugs (heparin, warfarin)

Clinical features

- Servere inherited disorders are associated with bleeding since childhood.
- Bleeding into joints - spontaneous, post traumatic

- Soft tissue including muscle bleeds
- CNS bleed can be life threatening
- DIC is characterised by :
  - * Generalized bleeding associated with features of underlying disease.

Investigations

PT is prolonged in

- DIC
- Liver disease
- Vit K deficiency
- Warfarin therapy

APTT is prolonged in

- DIC
- Liver disease
- Factor VIII deficiency / Factor IX deficiency
- Lupus anticoagulant
- Heparinized patient

Correction Tests Using the PT or APTT

- If there is deficiency of a clothing factor - PT or APTT performed on a 1:1 mixture with normal pooled plasma should give complete correction of prolonged times
- It is then necessary to identify the specific factor(s) that are deficient
- If PT/APTT is prolonged, and normal plasma fails to correct hte prolonged time, an inhibitor should be suspected
- An inhibitor screen and tests for an lupus anticoagulant should be performed.

Specific factory assay

- < 1% : severe deficiency
- 1 % - 5 % : moderate deficiency
- 6% - 30 % : mild deficiency.

Note:

N-Normal

PT- Prothrombin time

APTT-Activated partial thrombo plastin time

TT-Thrombo plastin time

First-line tests used in investigating haemostatic failure

S.No:	PT	APTT	TT	Fibrinogen	Platelet	Condition
1	N	N	N	N	N	Normal haemostasis Disorder of platelet function Factor XIII deficiency Disorder of vascular haemostasis
2	Long	N	N	N	N	Early oral anticoagulation Factor VII deficiency
3	N	Long	N	N	N	Von- Willebrand's disease Circulating anticoagulant, e.g.lupus Factor VIII, IX, XI, XII deficiency
4	Long	Long	N	N	N	Oral anticoagulants Factor V, X, or II deficiency Multiple factor deficiency and liver failure Vitamin K deficiency
5	Long	Long	Long	N or Abnormal	N	Liver disease Fibrinogen deficiency/disorder Hyper-fibrinolysis Heparin (large amount)
6	N	N	N	N	Low	Thrombocytopenia
7	Long	Long	N	N or Abnormal	Low	Liver disease Massive transfusion
8	Long	Long	Long	Low	Low	Acute liver disease Disseminated intravascular coagulation

Treatment

1. Replacement therapy
   * Factor VII / IX concentrates, if available, for patient with haemophilia.
   * Fresh Frozen Plasma (FFP) - 15-20 ml / kg body wt.
   * Cryoprecipitate 1 bag /10 kg body wt. - useful in haemophilia A, VWD (Von-Willebrand Disease), DIC.
   * Correction of anaemia.
2. Treat the cause / precipitating factor, if possible, such as sepsis.
3. Avoid deleterious agents such as anti-platelet drugs
4. Local measures (for haemarthrosis in haemophilia)
   * Pressure bandage
   * Cold compression
   * Immobilisation
   * Physiotherapy after pain subsides
5. Bleeding due to anticoagulants
   * Oral anticoagulants
     » Anti-dote: Inj. Vit K 1 -2 mg, if bleeding with INR>5.0
     » Fresh frozen plasma- 10 - 15 ml/kg if bleeding or for interventions.
     » Withhold drug, till bleeding stops.
   * Heparin
     » Anti-dote: Protamine sulphate 1 mg / 100 unit of heparin given IV
     » Fresh frozen plasma - 10 - 15 ml/kg if bleeding or for interventions.

In bleeding neonates

1. Coagulation factors do not cross from mother to fetus.
2. Physiological deficiencies of coagulation factors can cause prolongation of PT and APTT.
3. Bleeding is more common and severe in pre-term and low birth weight infants.
4. All new borns should receive Vit K 1 mg immediatily after birth.

General considerations

1. Blood sample for coagulation profile should be obtained before transfusion of blood or blood products.
2. If on anticoagulation, the same to be noted while interpreting the results.
3. If only whole blood is available, fresh blood (within 6 hours of bleeding) is preferred to stored blood, after adequate screening.
4. Platelet concentrates should be transfused immediately after procurement from the blood bank.
5. Anti-fibrinolytics such as Trann-exemic acid can be used for mucosal bleeds.
   * For oral bleeding one tablet of Trann-exemic acid (500mg) to be powdered and mixed with 10ml of water to make a paste. This may be kept in the mouth for 10-15 mins and then swallowed
   * If bleeding does not stop by local measures, tablet/capsule can be taken orally
   * Dose - 500 mg three - four times a day for an adult initially and can be increased to 1 gm every 6 hours (Dose in Children : 50-100 mg/kg/day). This may be continued till the bleeding stops
   * For epistaxis, a roller gauze may be soaked in the same paste and the nose packed with it.

Note:

Systemic Trann-exemic acid (oral/intravenous) is contra-indicated in patients with hematuria.

Recommended facilities to be available at various levels of hospitals for evaluation of bleeding disorders:

1. Primary health centre:
   * Blood smear examination for platelets
2. District hospital, in addition to the above
   * Complete blood count.
   * Bleeding time
   * PT and APTT with correction studies with pooled normal plasma
   * Thrombin time
   * Fibrinogen (if possible)
   * Inhibitor screening.
3. Tertiary referral Hospital

* In addition to above
* Factor assays.
* Inhibitor assays
* Von-Willebrand disease work up
* Platelet aggregation studies
* d-Dimer

Respiratory medicine

Standard Treatment Guidelines
Tamil Nadu Health Systems Project

Chapter 5

- Bronchial Asthma
- COPD
- Bronchiectasis
- Pleural Effusion
- Cor Pulmonale
- Tuberculosis
- RNTCP
- Extra Pulmonary TB
- Special Situations
  - TBM
  - Pregnancy
- Vaccination

Bronchial asthma

"Asthma is a chronic inflammatory disorder of the airways in which many cells and cellular elements play a role, in particular, mast cell, eosinophils, T Lymphocytes, Macrophages, neutrophils, and epithelial cells.

In susceptible individuals, this inflammation causes recurrent episodes of wheezing, breathlessness, chest tightness, and coughing, particularly at night or in the early morning

These episodes are usually associated with widespread but variable airflow obstruction that is often reversible either spontaneously or with treatment.The inflammation also causes an associated increase in the existing bronchial hyperresponsiveness to a variety of stimuli".

Factors Precipitating:-

Allergens:-

Inhaled (Dust, Pollen, House dust mite)

Ingested (Fish, Nuts, Strawberries)

Food Additives

Pharmacologic Stimuli:- NSAID's especially aspirin, Beta Blockers

Environmental and Air Pollution:-Cold Air, Tobacco Smoke, Dust, acrid fumes

Occupational Factors:- Grain dust, Wood dust

Infections:- Viral, Bacterial

Exercise

Emotional Stress

Clinical Manifestations:-

Symptoms:

- Episodic breathlessness
- Cough
- Chest Tightness
- Wheezing

Signs:

Tachypnea

Tachycardia

Diaphoresis

Use of accessory muscles

Pulsus paradoxus

Impending respiratory failure

Silent chest

Unable to speak

Respiratory muscle fatigue

Cyanosis

Shock

Investigations

Spirometry:-

FEV1 / FVC normally greater than 0.75 to 0.85%

Reduced FEV1

Reduced FEV1 / FVC ratio indicates airflow limitation

FVC loop shows expiratory flow limitation

FEV1 or FVC improve by at least > 200ml and > 12% of baseline following administration of short

acting bronchodilator indicative of reversibility.

Peak Expiratory Flow:

PEF measurements are made using a peak flow meter and can be an important aid in both diagnosis and monitoring of asthma.

Useful in assessing :

Acute asthma diagnosis

Monitoring of chronic asthma

Diagnosis of Asthma - >15% of diurnal variability

Chest X-Ray:-

To R/O other causes of wheezing

To R/O presence of pneumothorax in severe acute asthma

Signs of hyperinflation:-

Increased lung volume

Hyperlucency

Depressed diaphragm

Increased retrosternal airspace

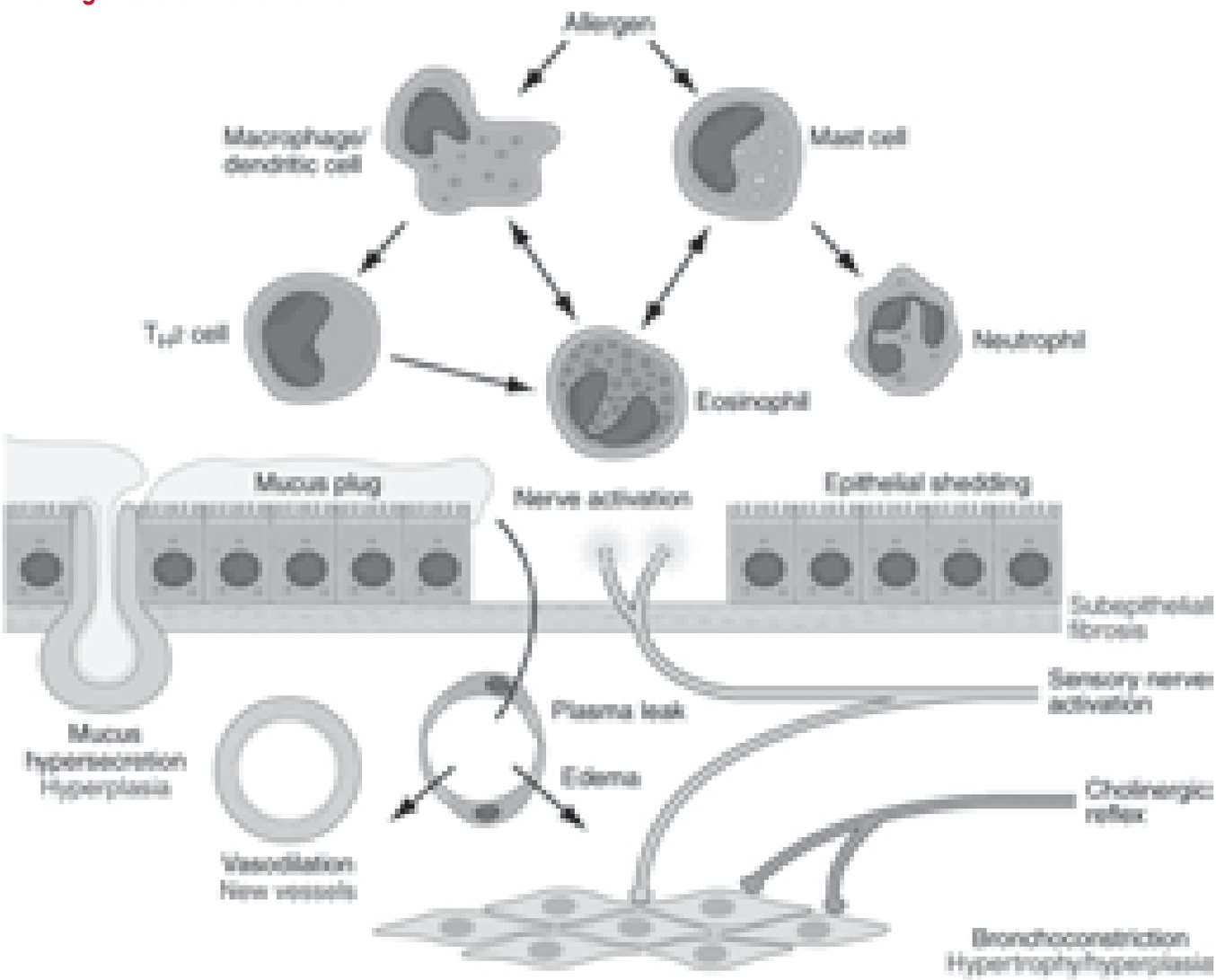
Focal atelectasis due to mucus plugging

Treatment

Pharmacological treatment:-

Beta 2 – agonists- salbutamol, terbutalin, salmeterol, formetrol. Inhalers are preferred over tablets since there are more effective and have less side effects. The short acting beta - 2 agonists should be given only for

Pathogenesis of Bronchial Asthma



'as and when required' basis and not regularly. Beta agonists can be combined with regular use of inhaled corticosteroids.

Inhaled corticosteroids - budesonide, beclomethasone, fluticasone, and should be used in all asthmatics except those with mild intermittent asthma.

Nedocromil and cromolyn sodium – With the advent of more effective inhaled corticosteroids the role of these agents have come down.

Leukotriene receptor agonists - Montelukast, Zafirlukast, Zileuton

Theophyllines – Can be used if asthmatic is still symptomatic when after giving inhaled corticosteroids and Long active beta agonists. Should not be used as the sole drug for treating asthma.

Oral corticosteroids – Prednisolone – should be given only during exacerbation. Oxygen

Non Pharmacological Treatment:-

Environmental control for reduce exposure to allergens

Immunotherapy in selected cases.

Education

Criteria for immediate referral to hospital

- Any life threatening features:
- Silent chest
- Cyanosis
- Feeble respiratory effort
- Bradycardia
- Hypotension
- Exhaustion
- Confusion
- Coma

Any features of severe attack that persist after initial treatment cannot complete sentence in one breath Respiratory rate > 25/min Pulse rate > 110/min

Complications:-

Respiratory

1. Mediastinal and Subcutaneous emphysema
2. Pneumothorax
3. Lobar collapse

Cardiac

1. Cardiac arrest
2. Myocardial infarct
3. Dysrrhythmia

Bio-Chemical

1. Hypokalemia
2. Syndrome of Inappropriate ADH Secretion

Prevention of asthma

Interventions are

1. Avoidance of house dust mite (HDM) exposure.
2. Avoidance of certain food like cow's milk, nuts, eggs and fish
3. Combination of the two.

Chronic Obstructive Pulmonary Disease

Introduction :

COPD is characterized by airflow limitation that is not fully reversible. The airflow limitation is usually both progressive and associated with an abnormal response of the lungs to noxious particles or gases.

- COPD includes :
  - * Chronic bronchitis
  - * Emphysema

Chronic Bronchitis is defined as presence of cough and sputum production for at least 3 months in each of 2 Consecutive years

Emphysema is defined as abnormal and permanent enlargement of airspaces distal to terminal bronchioles with destruction of walls and without fibrosis of airspaces

Etiology :

- Tobacco smoke (active and passive smoking). Recent evidence shows that upto 40% of smokers develop COPD.
- Smoke from biomass fuel. (firewood petrol diesel etc)
- Occupational chemicals (vapors and fumes)

Symptoms

- Cough
- Sputum production
- Dyspnoea

Signs :

- Sitting and bending forward with hands on knees.
- Pursed lip breathing.
- Cyanosis (Ominous sign)
- Distance between sternal and thyroid notch.
  - * Normal – 4 finger breath COPD - less than 3 finger
- Forced expiratory time
  - * Normal 4 secs - COPD - 6 secs and above
- Barrel chest
- Hyper resonant chest
- Diminished breath sound and bilateral wheeze

Diagnosis

Spirometry showing obstruction (Fev1/Fvc <70% even after bronchodilator confirms the diagnosis of COPD.

Treatment

Non pharmacological

- Rehablitation
  - * Exercise
  - * Nutrition
  - * Education
- Oxygen therapy
- Avoid smoking
- Pharmacological
- Bronchodilators inhaled bronchodilators are preferred to oral formulations in view of better efficacy and lesser side effects. Inhaled bronchodilators include short acting beta agonists (salbutamol, terbutaline), long active beta agonists (salmeterol, formoterol), short acting anticholinergics (ipratropium), long acting anticholinergic (tiotropium).

- Glucocorticoids - Inhaled corticosteroids should be given in severe COPD or in those with repeated exacerbation. systemic corticosteroids should be given only in patients with acute exacerbation of COPD.
- Antibiotics only when there is evidence of infection in the form of purulent sputum or fever.
- Surgical Bullectomy Lung transplantation.

Complication:

- Infection
- Cor pulmonale - Peripheral edema, pulmonary hypertension
- Respiratory failure
- Polycythemia. PCV more than 55%

Referral and admission:

- Increased symptoms despite treatment
- Cyanosis and peripheral edema
- Peak expiratory flow less than 100 liters / min
- SaO2 less than 90%
- Pneumonia

References:

www.goldcopd.org

Bronchiectasis

Introduction

Bronchiectasis is defined as permanent irreversible dilatation and distortion of medium sized bronchi.

Etiology

Congenital, Acquired

Acquired

Bronchial obstruction: Foreign body, tumor, bronchial stenosis / stricture extrinsic lymph node compression

Infection

Post measles and whooping cough

Bacterial infection

Mycobacterium tuberculosis and non tuberculous mycobacteria

Fungus (aspergillus species) Allergic broncho pulmonary aspergillosis (ABPA)

Acquired immunodeficiency syndrome

Cystic fibrosis

Primary ciliary dyskinesia (Kartagener's syndrome)

Pulmonary sequestration

Alpha 1 antitrypsin deficiency

Young syndrome (idiopathic obstructive azoo spermia with chronic sinopulmonary infection)

Hypogammaglobulinemia (IgA/IgG deficiency)

Symptoms

Cough with copious sputum mucoid to mucopurulent may be blood stained foul smelling with postural variation. Dyspnoea and wheezing Fever during exacerbations

Signs

Clubbing, halitosis and sinus tenderness

Persistent coarse leathery crackles may alter in character with coughing

Investigations

Sputum examination

Macroscopically large quantity of three layered sputum

Microscopic examination – Gram's stain, Ziel Neelson test for AFB, pyogenic culture and sensitivity, fungal culture and cytology for malignant cells.

Hemogram

Leukocytosis in bacterial infections

Lymphocytosis in chronic infections

Eosinophilia in ABPA

Lymphopenia in AIDS

Chest X-ray PA view

Diagnosis:

HRCT scan is the recommended investigation to confirm the diagnosis of bronchiectasis.

Treatment

Non – pharmacological

1. Chest physiotherapy - coughing, huffing, postural drainage – By this procedure sputum / pus in the

non dependant areas of lung is brought into the main stem bronchi or trachea by the aid of gravity. Then the sputum is cleared by coughing.

2. Physical exercise to be encouraged to aids mucous clearance

Pharmacological

1. Antibiotics. to Amoxicillin, Ampicillin, Cephalaxin for the bacterial infection . Ciprofloxacin or Gentamicin for pseudomonal infections

   Bronchodilators. Salbutamol, theophylline when wheeze is present. Inhaled drugs are preferred over oral.

2. Expectorants and mucolytics. N-acetyl cysteine, Ambroxol, Bromhexine, steam inhalation to thin out the secretions which can be later cleared by chest physiotherapy.
3. Supplemental oxygen.
4. Vaccination Pneumococcal and annual influenza vaccinations in high risk cases may reduce the incidence of frequent exacerbations and hospitalizations.

Surgical

Surgery is only appropriate when bronchiectasis is localized. Emergency surgical resection may be necessary for life – threatening hemoptysis but embolisation of appropriate bronchial artery is usually attempted first.

Natural Course

Bronchiectasis may progress slowly over many years and quality of life is usually impaired.

Complications

1. Acute exacerbations,
2. Haemoptysis
3. Lung abscess
4. Empyema / Pyothorax
5. Metastatic abscess – like cerebral abscess
6. Respiratory failure and cor pulmonale
7. Amyloidosis

Prognosis

Nowadays the natural history of bronchiectasis has changed and the prognosis is much improved However, pseudomonal infection is a bad prognostic sign.

Patient education

Patient is advised to perform postural drainage at least once daily and to increase the frequency to twice or thrice if they suffer an exacerbation.

Prevention

Vaccination against childhood infections prevents post infection bronchiectasis.

Admission

- Acute exacerbation,
- Hemoptysis

Referral to higher centre

- Presence of persistent haemoptysis
- Disease uncontrolled with medical management
- Unilateral and localized disease with repeated infection.

Pulmonary Function Tests

- Obstructive pattern

Pneumonia

Introduction :-

A syndrome of infection that is usually bacterial with symptoms and signs of consolidation of parts of the lung parenchyma .

Etiology : -

Pneumonia is not a single disease It can have over 30 different causes There are five main causes of pneumonia .

- Bacteria
- Viruses
- Mycoplasmas
- Other infectious agents, such as Fungi- including pneumocystis
- Various chemicals

High risk factors with specific pathogens :- Penicillin-resistant and drug-resistant pneumococci

- Age 65 years
- Lactam therapy within the past 3 months

- Alcoholism
- Immuno-suppressive illness (including therapy with corticosteroids)
- Multiple Medical co-morbidities
- Exposure to a child in a day care center

Pseudomonas

- Structural lung disease (bronchiectasis)
- Corticosteroid therapy (10 mg of prednisone per day)
- Broad-spectrum antibiotic therapy for 7 days in the past month
- Malnutrition

Enteric gram-negatives :-

- Residence in nursing home
- Recent antibiotic therapy

Symptoms and signs

- Cough, often producing mucus (sputum) from the lungs
- Mucus may be rusty or green or tinged with blood.
- Fever, which may be less common in older adults Shaking , "teeth-chattering" chills (one time only or many times).
- Fast, often shallow, breathing and the feeling of being short of breath.
- Fast heartuberculosiseat chest wall pain that is often made worse by coughing or breathing.
- Feeling very tired (fatigue) or feeling very weak (malaise).
- Nausea and vomiting
- Diarrhoea.

Classification

Condition	Commonly Encountered Pathogens
Exposure to animals and birds	Histoplasma capsulatum Chlamydia psittaci, Cryptococcus neoformans, Francisella tularensis Coxiella burnetii (Q fever) Legionella species
Travel and voyage	Coccidioidomycosis Pneumonia, Legionella species Burkholderia pseudomallei, avian influenza, SARS Bacillus anthracis (anthrax), Yersinia pestis (plague)
Infective and structural disease of lung	P. aeruginosa, S. aureus Anaerobes, S. pneumoniae, H. influenzae, Drug-resistant pneumococci, MRSA, fungal pneumonia. M. tuberculosis, Atypical mycobacteria
HIV infection (early)	S. pneumoniae, H. influenzae, M. tuberculosis
HIV infection (late)	Cryptococcus, Histoplasma, Aspergillus, Atypical mycobacteria (especially Mycobacterium kansasii)
COPD / Smoker	S. pneumoniae,Hemophilus influenzae Moraxella catarrhalis, Legionella
Nursing home / Residency	H. influenzae, Staphylococcus aureus, Anaerobes, Chlamydia pneumoniae, Tuberculosis

Investigations

- Complete blood count
- Pretreatment blood culture
- Pretreatment sputum culture
- Rapid PCR(Chlamydia and Legionella)
- Cold agglutinin for Mycoplasma
- Chest X-ray
- CT chest

- Bronchoscopy

Treatment

Non:- Pharmacological

- Oxygen Therapy
- Nutrition
- Physiotherapy
- IV Fluids

Pharmacological :-

- Analgesics
- Empirical Antibiotics
- Appropriate antibiotics after culture results

Natural Course

- With treatment , most types of bacterial pneumonia can be cleared within two to four weeks .
- Viral pneumonia may last longer .
- Mycoplasma pneumonia may take four to six weeks to resolve .
- Pneumonia progresses to bacteremia in about 20% and mortialtiy is increased in such patients.

Complications

- Pleural effusion
- Shock and DIC
- Bacteremia
- Lung abscess, pneumothorax ,empyema
- Pericardits , endocarditis , myocarditis
- Respiratory failure
- Meningo - encephalitis , Guillain - Barre Syndrome (GBS)

Prognosis

Criteria for clinical stability :-

- Temperature < 37.8 C
- HR< 100 beats/ min
- RR< 24 breaths/ min
- Systolic BP > 90 mm hg
- Arterial O2 saturation > 90% (or) PO2 < 60 mm Hg on room air
- Ability to maintain oral intake
- Normal mental status

Prevention

- Hand – washing (hospital setting)
- Identification of sources of infection
- Vaccination
- Antibiotic prophylaxis

Referral And Admission

- Patients who fail to respond to amoxicillin or a macrolide are frequently older have coexisting lung disease such as chronic bronchitis and emphysema .
- Those who show inadequate response to oral antibiotics .
- Respiratory rate > 30 breaths/ min
- Multilobar infiltrates
- Confusion/ disorientation
- Uremia (BUN level, >20mg/dl)
- Leukopenia (WBC count, 14000 cells/mm3)
- Thrombocytopenia (platelet count, 1100,000 cell/mm3)
- Hypotension requiring aggressive fluid resuscitation
- Patients who need invasive mechanical ventilation
- Septic shock with the need for vasopressors.

References

www.webmd.com/a-to-guides/pneumonia-symptoms American academy of family physiclans February 1, 2006 vol, 73 No. 3 Thorax, December 1, 2001: 56 (90004) : !

Pleural effusion

Symptoms

- Breathlessness
- Cough
- Fever
- Pleuritic chest pain

Signs:

Decreased breath sounds, dullness on percus-

sion, decreased vocal resonance

Types:

- Transudate
  - * Congestive cardiac failure
  - * Cirrhosis of liver
  - * Nephritic syndrome
  - * Myxoedema
  - * Peritoneal dialysis
- Exudates
  - * Infectious diseases-Tuberculosis, other bacterial, viral, fungal, parasitic
  - * Neoplasm – metastatic disease, mesothelioma, pulmonary embolism
  - * Collagen vascular disease-Rheumatoid Arthritis(RA), Systemic Lupus Erythematosus (SLE)
  - * Gastrointestinal disease- Oesophageal perforation, Pancreatic disease
  - * Uraemia
  - * Chylothorax, Hemothorax
  - * Drug induced- nitrofurantoin, dantrolene, amiodarone

Diagnostic aspects

Chest radiography: lateral decubitus, PA view, lateral view can be taken

- Blunting of costophrenic angle
- Ellis S shaped in larger effusions

Ultrasound chest

To detect

- Small amounts of fluids
- Loculation
- Septation

Treatment

Thoracocentesis

- One space below the maximum dullness in the posterior axillary line just above the rib, should be done.
- If patient develops cough or chest pain, stop the procedure.
- Complications: Pneumothorax (do check x-ray of the procedure)

Management of most common causes

- Tuberculosis
  - * Straw coloured effusion
  - * Cell count - lymphocyte predominance
  - * Pleural fluid protein > 3g/dl
  - * Category III ATT
  - * Category I ATT if presence of co morbid illness / bilateral effusion or Chest X-ray infiltration , Paratracheal nodes
- Para pneumonic effusion
  - * Chest X-ray consolidation features along with effusion
  - * Cell count predominantly neutrophilic
  - * Appropriate antibiotics.
  - * Thoracocentesis to ensure that empyema has not developed
- Malignant effusion
  - * Cytology positive
  - * Refer to higher centre
  - * Treatment of underlying cause in heart failure /nephrotic syndrome / liver cirrhosis

Cor Pulmonale

Definition

Hypertrophy of the Right Ventricle resulting from diseases affecting the function and / or structure of the lung.

Etiology

Parenchymal and Interstitial Diseases	
Chronic Sequelae of Tuberculosis	Cough, expectoration fever,loss of appetite, loss of weight night sweats, hemoptysis.
Cystic Fibrosis	Neonate or child – Failure to thrive Meconium ileus, Rectal prolapse Cough,Recurrent.chest infection. GIT Disturbances, Sinusitis,Diabetes Mellitus,Male – Infertile Female-Subfertile
Pulmonary Embolism	Acute or Chronic,Circulatory Collapse, Dyspnoea, Hemoptysis ,Pleuritic pain, Tachypnoea ,Dyspnoea RR > 20
Pneumonectomy	From History
Pneumoconiosis	Occupational history, coal mining foundary work, welding, fireworks,armament and painting. Clinical Features, Cough, Sputum, Dyspnoea on exertion.
Airway Diseases	
Asthma	Wheeze, Nocturnal cough, Episodic dyspnoea,Chest tightness Triggering factors.
COPD	Dyspnoea, reduced exercise tolerance, productive cough, Wheeze.
Bronchiectasis	Recurrent childhood infection, Viral exanthema Cough, Productive sputum.
Vascular Disease	
Pulmonary Embolism	(Acute Cor Pulmonale)
Primary Pulmonary Hypertension	Exertional dyspnoea, Chest Pain Syncope, Palpitation Edema.
Structural	
Chest Wall Abnormalities	Kyphoscoliosis Neuromuscular disease

Symptoms

- Weight gain
- Right Upper Quadrant discomfort
- Nocturia
- Peripheral edema
- Easy Fatiguability

Signs

- Tachypnoea
- Accessory muscles of respiration acting
- Cyanosis
- Neck vein distention
- Ascites
- Pedal edema
- Liver – enlarged tender
- CVS- Parasternal systolic lift
- Thud felt over Pulmonary area
- S2 accentuated

Investigations

Chest X-ray

Enlarged Pulmonary artery and underlying lung disease

ECG

'P' Pulmonale, R axis deviation RV Hypertrophy

ECHO

To rule out Left Ventricular failure from CAD / Aortic / Mitral Valvular Disease, Congenital Heart Disease.

RBC

Polycythemia

ABG

PO2 < 90%

Treatment

- Non – Pharmacological
- Salt reduced diet
- Diaphragmatic breathing
- Breathing exercises
- Long term O2
- Avoid smoking /Viral Infections
- Prompt treatment of acute respiratory infections.

Pharmacological

- Beta 2 – agonists
- Corticosteroids
- Diuretics
- Digoxin
- Oxygen therapy

Surgical

- Phlebotomy

Patient Education

- Avoid smoking
- Avoid exposure to cold and allergens
- Prompt treatment of acure respiratory infections.

Referral

- RR > 30
- ABG PO2 < 60
- Hypoxemia –Persistent and Worsening
- Cardio – respiratory failure.
- Pneumothorax.

Complications

- Respiratory failure
- Circulatory failure
- Congestive hepatomegaly.
- Neurohypoxia

Prognosis

- Poor.

Tuberculosis and RNTCP

(Revised National Tuberculosis Control Program)

Tuberculosis is caused by Mycobacterium tuberculosis, which is primarily a pulmonary disease but also affects other systems of the body.

In India, there are 14million tuberculosis cases out of which, 3.5million are sputum positive and about 1million sputum positive cases are added each year. About 1000 people per day and one every minute, die of Tuberculosis in our country.

Transmission of disease:

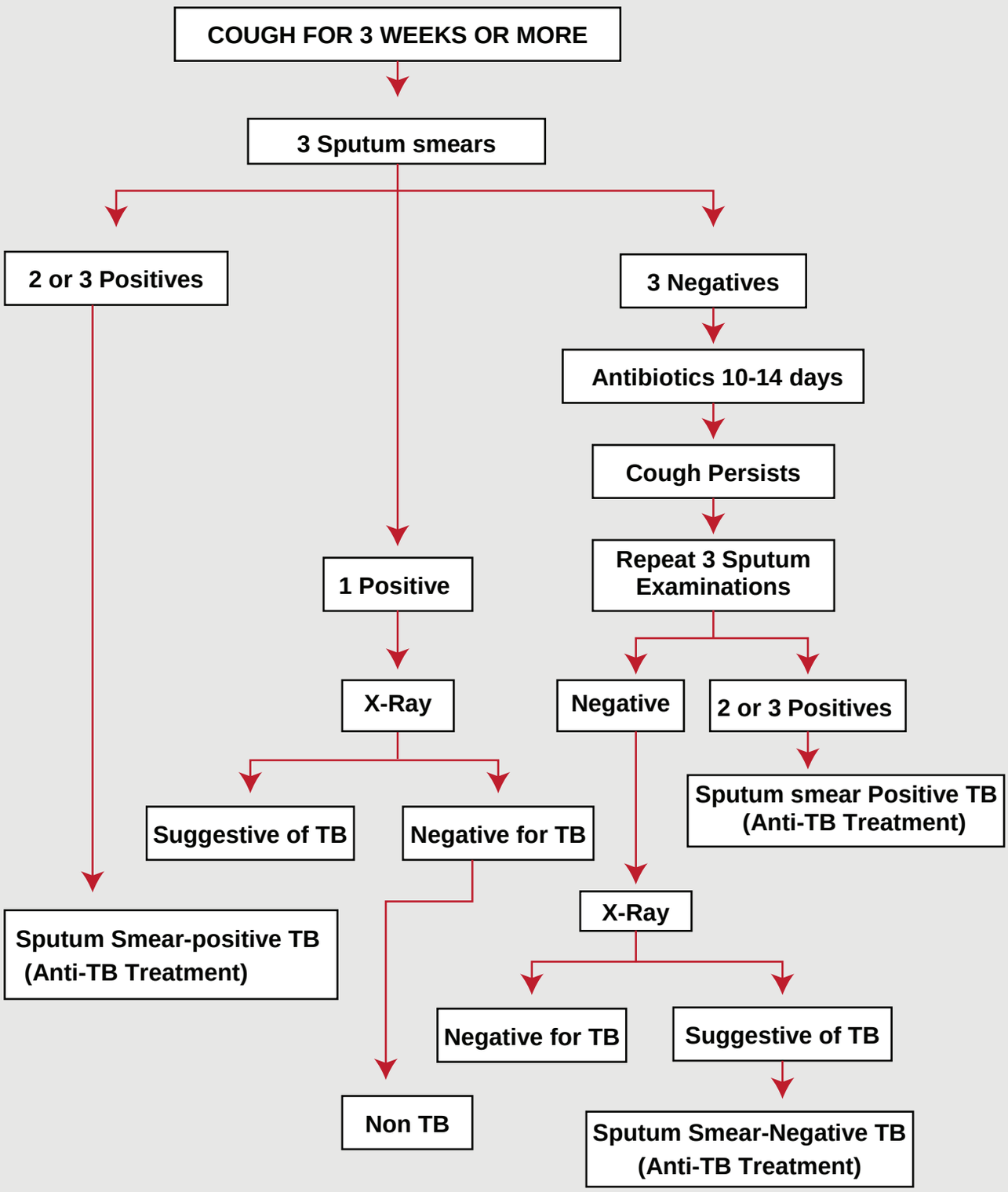
When a sputum positive pulmonary tuberculosis patient coughs, he spreads Tuberculosis bacilli into air, in the form of tiny droplets of 1-5µm.If these droplets are happened to be inhaled by a healthy person, he may be infected with tuberculosis.

Effective treatment of smear positive tuberculosis patient can help to control the spread of disease. Hence, it is the top priority under RNTCP. (Revirsed National Tuberculosis Control Programme)

Goal of RNTCP

To cure atleast 85% of all newly detected sputum positive cases.

Diagnostic algorithm for Pulmonary tuberculosis



Note:

**Antibiotics used in the treatment should not have Anti Tuberculosis activity (e.g. co-trimoxazole. Avoid floroquinolones, rifampicin and streptomycin.)

RNTCP-DOTS:

Directly observed treatment, short course is the way Tuberculosis cases are treated under RNTCP. It is ensured that patients take medicines regularly.

Categories and treatment:

- Category of treatment
- Type of patient
- Regimen
- Category I
- New sputum smear-positive

  Seriously ill new sputum smear-negative

  Seriously ill new extra-pulmonary
- Intensive phase - 2H3R3Z3E3
- Continuation Phase - 4H3R3
- Category II
- Sputum smear-positive Relapse
- Sputum smear-positive Failure
- Sputum smear-positive Treatment After default
- Others
- Intensive phase - 2H3R3Z3E3S3 + 1H3R3Z3E3
- Continuation phase - 5H3R3E3

Category III

- New Sputum smear-negative, not seriously ill
- New Extra-pulmonary, not seriously ill
- Intensive phase - 2H3R3Z3
- Continuation phase - 4H3R3

Note:

The number before the letters refers to no. of months of treatment. The subscript after the letters refers to no. of doses per week.

Patients in categories I, II who have a positive sputum smear at the end of intensive phase should receive an additional month of intensive phase treatment.

IP – Intensive Phase, CP – Continuation Phase. All drugs are given under supervision during IP, thrice weekly on alternate days. During CP, the first dose of each week is given under supervision.

Dosage Strengths:

Drug

Dose(thrice a week)

Isoniazid 600 mg H

Rifampicin 450 mg(600 mg if wt >60 kg) R

Pyrazinamide 1500 mg Z

Ethambutol 1200 mg E

Streptomycin 0.75 g IM(0.5 g if age>50 years)

S

Note:

Smear-negative pulmonary Tuberculosis

Seriously ill:

- Miliary Tuberculosis
- Extensive parenchymal infiltration
- Co-infection with HIV.
- Pulmonary Disease with cavitations
- All forms of Paediatric sputum smear-negative pulmonary Tuberculosis except primary complex.

Extra - Pulmonary Tuberculosis

Tuberculosis can affect any part of body including pleura, lymph node, bone and joints,genito-urinary tract,gastro-intestinal tract and central nervous system.

Diagnosis of extra pulmonary tuberculosis may require specialized tests such as FNAC, biopsy, radiological investigations or strong clinical evidence.

Diagnosis

Any person with cough with expectoration for 3 or more weeks should be investigated for tuberculosis. Sputum microscopy of at least 3 samples is the gold standard investigation to confirm pulmonary tuberculosis. It also indicates the degree of infectivity and helps to monitor response to treatment.

Radiological investigation is unreliable because other pulmonary disease may often resemble tuberculosis and the activity of the disease cannot

be confirmed.

Extra pulmonary Tuberculosis (EPTuberculosis)

Seriously ill EPTuberculosis

- Meningitis
- Pericarditis
- Peritonitis
- Bilateral or Extensive pleural effusion
- Spinal Tuberculosis with neurological involvement.
- Intestinal, Genito-Urinary
- Co-infection with HIV
- All forms of paediatric EPTuberculosis other than lymph node and unilateral pleural effusion

Not Severely EPTuberculosis

- Lymph node
- Unilateral pleural effusion
- Peripheral Joints.
- Hospitalisation:

Extremely ill EPTuberculosis,

- With significant hemoptysis,
- Pneumothorax,
- Large pleural effusion
- Dyspnoea
- Hospitalisation and intensive care.

Special Situations

Tuberculosis Meningitis:

- Patients should be referred to the hospital and treated under category I treatment, with continuation phase lasting 6-7 months.
- Steroids should be given initially to reduce meningeal inflammation and tapered over a period of 6-8 weeks.

During pregnancy:

- All anti tuberculosis drugs used in RNTCP except streptomycin are safe during pregnancy.
- Breast feeding should be continued regardless of mother's Tuberculosis infective status.

Child contacts - < 6 years of age with sputum smear positive case:

- If the child has symptoms of tuberculosis and if it is confirmed by the treating physician – a full course of ATT (CAT III) should be given.
- If the child does not have symptoms:
  - * Tuberculin test: Not available – chemotherapy for 6 months Isoniazid 5 mg/kg.
  - * Tuberculin test: Available – child should be given INH chemotherapy for 3 months and Tuberculin test should be done, then treat as per the notes given below.

Note:

- If induration to tuberculin test <6mm stop preventive chemotherapy and vaccinate with B.C.G (if not vaccinated previously)
- If induration is >6mm, continue INH preventive chemotherapy for another 3 months.

Vaccination:

BCG vaccination does not protect an individual from developing adult type pulmonary tuberculosis. But, several studies indicate that BCG prevents serious forms of Tuberculosis in children.

Gastroenterology

Standard Treatment Guidelines
Tamil Nadu Health Systems Project

Chapter 6

- Aphthous Ulcers
- Acute Esophageal Candidiasis
- GERD
- Peptic Ulcer Diseases
- Vomiting
- Constipation
- IBS
- AGE
- Chronic Diarrhoae
- Ulcerative Colitis
- Amoebic Liver Abscess
- Pyogenic Liver Abscess
- Acute Pancreatitis
- Chronic Pancreatitis
- GI Bleed

Aphthous ulcers

Symptoms

Aphthous ulcers in the oral cavity cause significant pain during chewing food.

Causes

Minor aphthae are recurrent, painful typically single or multiple (1-15 mm) shallow ulcers surrounded by erythematous mucosa occurring anywhere in the oral mucosa. These occur cyclically and heal without a scar in a week's time. Larger and deeper ulcers known as major aphthae, heal with a scar. Those that are vesicular are called herpetiform aphthae and are more numerous. Fever, adenopathy, gastrointestinal symptoms are typically absent.

Important note

Ulcers occurring repeatedly at the same site or slow healing ulcers with systemic symptoms e.g. uveitis, arthritis, fever, adenopathy are worrisome. Malignancy should be excluded.

Treatment

Medical therapy

Antibiotics

1. Topical and systemic antibiotic treatment:

   Tetracycline 250-mg antibiotic capsule dissolved in 180 mL water and used as a "swish and swallow" or "swish and spit" treatment four times per day for several days in adult patients.

   or

   Tetracycline suspension, 250 mg per 5 ml, used in a similar fashion, with 5 ml swished four times per day. (Avoid in children and in pregnancy)

2. Pro-biotics:

   The powder form can be used two or three times a day, placed within the oral cavity and swallowed with sips of water

3. Anti-inflammatory agents

   Pellets of Hydrocortisone 5 mg can be kept on the ulcer base and sucked every 4 hours for 3 to 5 days. Most successful when ulcers are located in the sulci where pellet can be left to dissolve.

Triamcinolone 0.1 percent

- Applied to ulcers two to four times a day. The paste can be applied until the ulcer heals.

  Betamethasone sodium phosphate (Betnesol mouthwash)

- One 0.5 mg tablet dissolved in 5 to 10 ml of water used as a mouthwash qid during ulcer attack. Must be held in mouth for a minimum of 3 minutes for maximum effectiveness; spit out after use. Can be used 6 times a day under strict supervision.

Dexamethasone elixir:

- 0.5 mg per 5 ml as a rinse and expectorated: recommended for extensive oral ulceration
- Warning: secondary fungal infection likely.
- Tab. Prednisolone 0.5 mg/kg/day for 3 to 5 days.

Immune modulators

- Hydrocortisone pellets 5 mg can be kept on the ulcer base and sucked every 4 hours for 3 to 5 days

  or

  Prednisolone 0.5 mg/kg/day for 3 to 5 days.

- Levamisole
  - Dose: 50 mg twice a day for three consecutive days for 4 weeks; no medication for next two weeks
  - Followed by Levamisole 150 mg tablet: half a tablet twice a day for three consecutive days for two weeks.

HIV positive patients

Thalidomide: 200 mg once to twice daily for three to eight weeks (contra-indicated in non-HIV ulcers: significant side effects and terato-genicity)

Do's and Don't's

- Good oral hygiene: Repeated mouth wash with plain water/saline gargle especially after eating
- Ensure toothbrush has aligned bristles
- Avoid chewing betel leaf and other condiments
- Avoid excessive carbonated drinks and spicy or sharp/crispy foods
- Take plenty of green leafy vegetables. Leaves of 'Manathakali" when chewed for 10 minutes two or three times a day, results in spontaneous healing of ulcers
- Using a straw can alleviate pain
- Seek opinion of a dentist, if ulcer fails to heal.

Acute Oesophageal candidiasis

Causes

Commonly occurs as opportunistic infection in individuals with uncontrolled Diabetes mellitus or immuno-compromised conditions (AIDS, malignancy, chronic steroid therapy, cytotoxic drugs). Usually caused by Candida albicans.

Symptoms

Discrete or confluent curdy white adherent plaques on the oropharyngeal mucosa.

- Oral lesions are usually painless but oesophageal lesions produce painful dysphagia

Antifungal therapy for Esophageal Candidiasis			
Antifungal Agent	Form	Strength	Use
Topical			
Nystatin	Suspension	100,000 U	5 ml, swish and swallow q.i.d.
Clotrimazole	Oral lozenge	10 mg	Dissolve 1 lozenge 5 times/day
Amphotericin B	Suspension	1 mg/ml	1 ml, swish and swallow q.i.d.
Amphotericin B	Lozenge	100 mg	q.i.d.
Amphotericin B	Tablet	10 mg	q.i.d.
Systemic			
Ketoconazole	Tablet	200 mg	1-2 tablets / day b.i.d.
Fluconazole	Tablet	100 mg	1 tablet / day
Fluconazole	Solution	10 mg/ml	10 ml /day
Itraconazole	Capsule	100 mg	200 mg /day
Itraconazole	Solution	10 mg/ml	10-20 ml / day b.i.d.

Investigations

Diagnosis is confirmed by demonstration of pseudohyphae on wet smears or culture.

Treatment

Suspension Nystatin local application in mouth and 100, 000 units orally 4 hourly for 5 to 7 days

or

Tab Fluconazole 100 mg /day for 10 to 14 days

Dyspepsia

It is a non-specific group of symptoms related to the upper gastrointestinal tract. It is also referred to as 'non-ulcer dyspepsia' / functional dyspepsia/ GERD

Symptoms

Common symptoms include

- Upper abdominal symptoms simulating an ulcer disease, or heart burn with or without regurgitation simulating gastro- oesophageal reflux disease, heaviness, post-prandial fullness or early satiety (dysmotility type). Symptoms of 'gas' in abdomen is not uncommon.
- Beware of red flag signs or 'alarm' symptoms such as anorexia, weight loss, anaemia, dysphagia, mass abdomen
- Age > 35 years (south Indian), not responding to empirical anti ulcer, anti -GERD treatment

Investigations

The above symptoms suggest an organic disease and are excluded by history and upper endoscopy.

Treatment

Medical therapy

Start empirical therapy for patients below 35 years and in absence of 'alarm symptoms'. UGI scopy is indicated in non-responders.

- In patients with ulcer-like dyspepsia, cap. Omeprazole 20 mg once a day 45 minutes before breakfast for 4 to 6 weeks

  o r

  Ranitidine 150 mg twice a day 45 min. before breakfast and dinner for 4 to 6 weeks Antacids 2 to 3 teaspoon or 2 tabs (chewable) when symptomatic despite above medication
- For those with dysmotility symptoms
  - * Domperidone 10 mg three times a day 30 min. Before breakfast, lunch and dinner (options: other prokinetics: mosapride, itopride, levosulpride).
  - * Avoid metoclopramide due to extrapyramidal side effects
- For reflux type dyspepsia
  - * Cap Omeprazole 20 mg 45 min. Before breakfast 4 to 6 weeks
  - * If regurgitation is predominant one can add

Domperidone 10 mg half an hour before breakfast and dinner (or any other prokinetic)

Duration:

Short courses of therapy (4 to 6 weeks) of the drug may be repeated or long-term treatment may be continued for up to a year. Intermittent therapy or biweekly PPI is also recommended in those requiring long-term treatment.

Anti-H pylori treatment is recommended for

- Those on long term NSAIDs
- Those with Duodenal / gastric ulcers (complicated e.g. bleed).

Recommended treatment for H. Pylori (For one week)

Combination of

Omeprazole 20 twice a day

+

Amoxicillin 500 mg thrice a day

+

Metronidazole 400 mg thrice day

Followed by

Omeprazole 20 mg once a day for three weeks

It is desirable that the anti -H pylori regimen is taken for at least 5 days

Do's and Don't's

- Avoid excess tea, coffee, fried food items.
- Abstain from alcohol and smoking.
- Avoid unnecessary NSAIDs; prefer Paracetamol especially those with ulcer like symptoms or those with documented duodenal/ gastric ulcer.
- Follow meals at regular intervals: 4 th hourly (including snacks).
- Daily exercise to maintain optimum weight.

Gastro-Esophageal Reflux Disorder

Causes

It is a common disorder caused by retrograde flow of gastric contents through an incompetent gastro-oesophageal junction.

Symptoms

Clinically patient presents with

- Retro-sternal pain, heartburn and regurgitation mostly after a meal
- Rare presentations: chronic cough, laryngitis, recurrent pulmonary infections, bronchospasm, otitis media etc

Investigations

Diagnosis is confirmed by endoscopy which based on severity of disease may show mild (minimal or no mucosal change) to severe changes (linear ulcers with or without oesophageal stricture). Endoscopy is not recommended as a routine for screening for GERD since there is a very poor correlation between clinical presentation and endoscopy findings. pH study is the gold standard.

Treatment

If GERD is occasional i.e. once a week or twice a week:

For immediate relief:

- Liquid antacid with or without alginate 10 to 15 ml or
- 2 to 3 tablets chewed: 4 to 6 times a day ½ to 1 hr after a meal if symptoms persist.
- There is no role for long-term maintenance therapy. Ranitidine or Omeprazole will not relieve the symptoms instantaneously.

For Mild GERD

1. Cap Omeprazole 20mg OD/ BD
2. (or Pantoprazole 40 mg OD,
3. Rabeprazole 20 mg OD,
4. Esomeprazole 40 mg OD, or
5. Lanzoprazole 30 mg OD) 45 minutes before a meal for 4 to 6 weeks.
6. If individual is on multi- drug therapy, Pantoprazole or Rabeprazole is recommended.
7. Add pro-kinetic: Domperidone 10 mg three times a day 30 minutes before a meal for 2 weeks. if regurgitation is significant.
8. Long-term therapy with Omeprazole is not associated with major side effects. Drugs are often an alternative to surgery.

For moderate to severe GERD

- Cap Omeprazole 20 mg or any of the available PPIs twice a day 45 minutes before a meal for 4

to 6 weeks i.e. “double dosing”

Follow-up

Maintenance dose

Recommended in few cases using half the recommended dose (Omeprazole 10 mg once a day half - hour before break fast).

For Severe GERD

Long term maintenance therapy / double dose with PPI is recommended. Patient may eventually require surgery.

Surgery

This is indicated in individuals who are young, responding to Proton pump inhibitors (PPI) and are likely to require long term maintenance. It is relatively contraindicated in non-responders to PPI

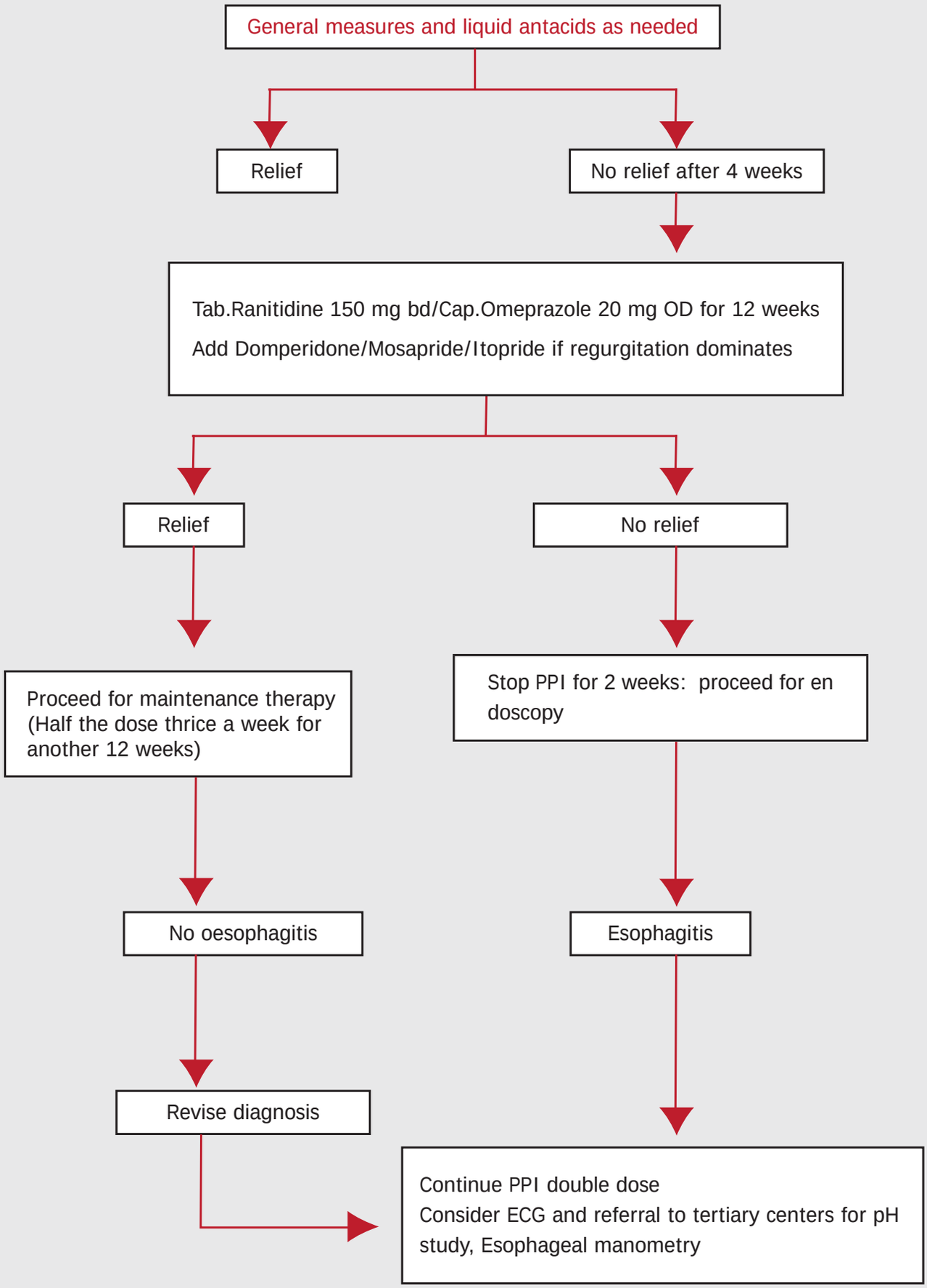
Important note

Do’s and Don’ts

Life style modification

- Reduce fat intake
- Weight reduction
- Stop smoking
- Small regular meals
- Allow 3-hours between last meal and retiring at night
- Avoid tight corsets during sleep
- Avoid alcohol/hot drinks before bed/excessive tea/coffee/garlic pearls
- Combine with a PPI when on treatment for asthma, depression.
- Plenty of water is recommended while swallowing doxycycline, tetracycline, NSAIDs etc.
- Raise head end of bed by 10 cm using a block/brick if nocturnal symptoms are present. Do not raise the head using pillows

Approach to drug therapy in GERD



Peptic ulcer disease

Definition

There is ulceration of the gastric or duodenal mucosa due to acid and pepsin.

Symptoms

- Patient presents with sharp gnawing epigastric pain, worsened (gastric ulcer) or relieved by intake of food (in duodenal ulcer).
- Nocturnal pain commonly awakens the patient at midnight. Patient occasionally gets up in the morning with pain. Typically there are relapses and remissions.
- Complications due to an ulcer disease include an upper GI bleed, perforation and gastric outlet obstruction.
- Helicobacter pylori is responsible for most of the duodenal and gastric ulcers.
- Endoscopy is confirmatory. Empirical treatment is recommended in those with no "alarm symptoms."
- Helicobacter pylori checking is not done routinely.
- Rapid urease test, histology of antral- mucosa is adequate.

Treatment

Medical treatment

Anti H.pylori treatment is recommended for patients on long term NSAIDs, bleeding pepic ulcer

Preferred one-week triple therapy (Table), followed by PPI for 3 weeks.

H.pylori treatment

Drug	Dose (mg)	Frequency	Duration
PPI Clarithromycin Metronidazole	* 500 400	BD BD BD	One week
PPI Amoxicillin Metronidazole	* 500 400	BD TDS TDS	One week
PPI Amoxicillin Clarithromycin	* 500 500	BD BD BD	One week

* Choice of PPI: Pantoprazole 40 mg/Rabeprazole 20 mg/Lansoprazole: 30 mg

Non-Helicobacter pylori

Symptomatic therapy

1. Any PPI for 4 to 6 weeks, 45 minutes before breakfast.
2. H2-Receptor antagonists
   * Ranitidine (150 mg bd) / Famotidine (40 mg od) equally efficacious but takes longer time for symptom relief.
3. Maintenance dose with PPI for patients on long term NSAIDs, IHD patirents

Do's and Don'ts

Stop smoking

Curtail alcohol intake

Avoid NSAIDs, prefer Paracetamol

Avoid foods which aggravate symptoms; no role for bland diet or excess milk

Meals at regular intervals

Vomiting

Definition

Vomiting is forceful expulsion of the gastric contents due to contraction of abdominal musculature and simultaneous relaxation of gastric fundus and lower oesophageal sphincter.

Nausea is an unpleasant feeling, that one is likely to vomit.

Regurgitation is effortless vomiting without forcible contraction of abdominal wall muscles.

Causes

1. Central (due to stimulation of vomiting centre) neurological diseases, raised intracranial pressure
2. Vestibular system disorders
3. Drugs and toxins
4. Toxic and metabolic disorders such as ketoacidosis,
5. Systemic infections
6. Radiation exposure
7. Pregnancy and
8. Psychogenic vomiting.
9. Peripheral
10. Obstructive diseases of the GIT

11. Acute gastritis, gastroenteritis
12. Severe UGI bleed etc.
13. Excessive vomiting can result in electrolyte imbalance.
14. Chronic recurrent vomiting can result in malnutrition.
15. Repeated nausea and retching may result in UGI bleed often mild, consequent to a mucosal tear in oesophageal-cardia junction referred to as a Mallory-Weiss tear.

Investigations

- Evaluation should exclude CNS causes and an Upper GI endoscopy to rule out pathology in the upper GI tract. Screening up to III part of duodenum is possible.
- Barium meal is recommended only when Upper GI endoscopy is normal.
- Psychogenic vomiting is diagnosed by exclusion of organic causes only.

Treatment

Hospitalize the patient to give intravenous fluids if dehydrated. Start oral fluids as soon as the patient can tolerate. Appropriate analgesics if patient has severe pain.

Acute vomiting

Rule out gastric outlet obstruction then

Inj Metoclopramide 10 mg I/M, repeat after 6 hours if needed

or

Tab. Mozapride 5 mg three times a day

or

Tab. Domperidone 10 mg three times a day

or

Tab Metoclopramide 10 mg three times a day

or

Injection Prochlorperazine 5 mg IM. Repeated after 4-6 hours if needed

or

Ondansetron 8 mg stat dose (oral/parenteral) and repeated 8-hrly if required

In pregnancy avoid all drugs, if possible

Promethazine 25 mg oral/injection safe in the first trimester

For motion sickness: Tab Cyclizine 50 mg 3 times daily.

Prevention

- Avoid stale food, cut vegetables/fruits kept in open, drink potable water only.
- Avoid NSAIDS, especially if ulcer symptoms are present.
- Prevent dehydration: Encourage patients to take sips of liquids at short intervals to prevent dehydration.
- Endoscopy is necessary, if symptom persists.
- Prevent motion sickness by avoiding heavy meal before travel.

Constipation

Causes

Commonest cause of constipation is habitual. Important factors include insufficient dietary fibre, physical inactivity, suppression of defecatory urges occurring at inconvenient moments, prolonged stress etc.

Secondary causes include neurological, hormonal, colonic, malignancy, depression. These causes should be considered if there is recent onset of constipation or severe symptoms.

Symptoms

Clinically an individual is considered to be chronically constipated if there is decrease in frequency and liquidity of stool compared to the normal pattern in a particular individual or straining at defecation> 25% of times, passing lumpy/hard stools and sensation of incomplete evacuation.

Investigations

A rectal examination with a short length colonoscopy is a must for all patients with recent onset of constipation irrespective of bleeding per rectum.

When acute, the constipation may be a part of a serious illness such as acute bowel obstruction. These patients present with abdominal pain, vomiting and distension and non-passage of flatus are the typical presentation. These patients should be referred immediately to a higher center after rectal examination, passage of rectal tube (for passage of flatus) and a plain X-ray abdomen.

Treatment

Medical treatment may be given 2 to 4 times a week. Some patients may require these for several weeks to months

Lactulose solution 15 to 20 ml at night

or

Susp. Magnesium sulphate 15 to 20 ml at night

or

Bulk forming agents such as 'isaphgul husk' or 'psyllium seeds' or

Isotonic polyethylene glycol (PEG electrolyte) solution 125 – 250 ml

Do's and Don'ts

Advise patients to take plenty of fluids, high fiber diet – green leafy vegetables, fruits, avoid caffeinated drinks.

Regular walk and exercise ½ to 1 hr daily, abdominal exercise.

To use Indian closet as far as possible (this will straighten the anorectal angle).

Avoiding suppression of urge to defecate, making a regular habit).

Avoid purgative frequently to treat constipation, as it may be habit forming.

Suppository or simple enema is preferred in IHD.

Irritable Bowel Syndrome (IBS)

Definition

A constellation of gastrointestinal symptoms associated with lower bowel symptoms that occur in absence of an organic disease.

Symptoms

Clinically the diagnosis is made when continuous or recurrent symptoms of abdominal pain associated with any of the three features viz. Relief by defecation and /or onset with change in stool frequency or consistency for at least 3 months.

Supportive symptoms of IBS include passage of mucous, abnormal stool passage (straining, urgency, feeling of incomplete evacuation) and feeling of abdominal fullness.

Exclude IBS if individual has alarm symptoms such as fever, weight loss, bleeding per rectum or anaemia.

Treatment

If pain is predominant

Tab. Mebevaerine Hcl 270 mg three times a day given for long term

or

Tab Drotaverine 40 to 80 mg 3 times a day

or

Tab Propantheline Hcl 15 mg 3 times a day

In those with depressive symptoms

1. Tab Amitryptaline 10 mg HS for 4 to 6 weeks
2. In those with diarrhoeal symptoms
3. Tab Loperamide 2 to 4 mg daily for several days/ weeks depending on the clinical response
4. Any IBS patient with change in presentation e.g. change in bowel habit requires re-evaluation.

Do's and Dont's

- Diet should contain high fibre and supplemented with bulk forming agents such as isaphghul husk
- Avoid caffeine and alcohol
- Avoid milk and other dietary constituents, which worsens the symptoms
- Psychotherapy may be helpful in select cases

Acute Gastro-enteritis(AGE)

Symptoms

It is a self-limiting illness characterized by diarrhoea, abdominal cramps, nausea and vomiting, usually caused by viruses or bacteria (E coli, V cholerae, Staph aureus, Bacillus cereus etc). Most of these cause non-invasive or toxic diarrhoea.

Less commonly patient present with diarrhoea with passage of mucous and / or blood in stools. This may be associated with significant symptoms such as fever, malaise etc. These patients are more likely to have invasive diarrhoea caused by bacteria (E coli, Shigella, salmonella, Campylobacter) or parasite amoeba.

Treatment

In acute gastro-enteritis the aim is to correct dehydration and electrolyte imbalance. There is no need to investigate for the etiology immediately. Further investigations are necessary if there is bloody diarrhoea, clinical evidence of toxicity or prolonged diarrhoea.

Medical treatment

1. Indicated only in very ill patients with systemic symptoms associated with bloody diarrhoea, traveller's diarrhoea or in cholera infection. Tab ciprofloxacin 500 mg two times a day for 3 to 5 days
2. In amoebic dysentery Tab Metronidazole 400 mg three times a day for 5 to 7 days or Tab Tinidazole 600 mg twice a day for 3 to 5 days
3. In acute Giardial infection

   Tab Tinidazole 1000 mg single dose or Tab Metronidazole 400 mg three times a day for 3 days Hospitalization is needed when there are clinical signs of dehydration especially in young children or in the elderly, suspected cholera, immunosuppressed patients and those with severe systemic symptoms

Do's and Dont's

In absence of vomiting patient should be asked to take sips of fluid

Fluids used at home can be juices, soups and ORS

Milk and related products are avoided for at least 2 weeks, because of secondary lactase deficiency.

Chronic diarrhoea

Definition

A patient is considered to have chronic diarrhoea if diarrhoea persists for more than a month.

Causes

- Common causes of chronic diarrhoea are parasitosis, sprue and tuberculosis.
- A person is considered to have mal-absorption if the stools are bulky, greasy, frothy, foul smelling stools associated with lot of flatulence indicating mal-absorption.
- A large bowel diarrhoea is typically small volume, loose, watery stools mixed with mucus and/or blood.

Investigations

- After a preliminary blood sugar, thyroid function tests and stool fat globules, patient should be referred to a tertiary centre for further evaluation.
- Barium meal may be of some help as also an ultrasound of the abdomen. A duodenal biopsy from the third part of duodenum may be informative.

Treatment

Medical treatment

There is a role for long-term Tetracycline 250 mg three times a day for 6 months with Tab Folic Acid 5 mg a day, when bacterial overgrowth is suspected.

Anti-cholinergics or nonspecific anti-diarrheal agents are not recommended in the absence of a proper diagnosis.

Other recommended agents

1. Tab Norfloxacin 400 mg BD or Tab Ciprofloxacin 500 mg BD or Cap Doxycycline 100 mg OD or If the above mentioned drugs are contraindicated, Tab Cotrimoxazole 960 mg BD.
2. Tab. Folic acid 5 mg BD
3. Pro-biotics two to three caps three times a day for 2 weeks

The above treatment is given for 3-6 months duration depending upon patient's response. Other minor nutrient supplements are given if there is evidence of specific deficiency.

Ulcerative colitis(UC)

Symptoms

During the first attack the patient often presents with bloody diarrhoea, with systemic symptoms of low to moderate fever, backache, arthralgia.

The first attack is a close mimicker of acute infective diarrhoea. A stool examination followed by sigmoidoscopy is mandatory, especially if the bloody diarrhoea persists for more than a month. It is important to exclude amoebic infection prior to institution of steroids.

Rectum is uniformly involved in these patients. Frequency of stool can provide information on severity of disease: mild (2-4 stool/day), moderate (4-6 stools/day) or severe (> 6 stools/day).

During remission, patient may be asymptomatic or may have extra-intestinal symptoms.

Treatment

These patients require a referral to a tertiary unit. Aim is induction of remission in acute stage and then maintenance of remission.

Medical therapy

Therapeutic decisions depend on disease activity and extent. Patients with severe disease require hospital admission, whereas those with mild/moderate disease can generally be managed as outpatients.

Disease extent can broadly be divided into distal and more extensive disease.

Distal disease (proctitis/procto-sigmoiditis):

Topical management is appropriate.

Extensive disease:

Oral or parenteral therapy is the mainstay of treatment.

Choice of drugs:

Mesalamine preparations and Steroid preparations

Treatment of active left sided, or extensive UC:

- Mesalazine 2–4 g daily or Balsalazide 6.75 g daily are effective first line therapy for mild to moderately active disease.
- Sulpha-salazine has a higher incidence of side effects compared with newer 5-ASA drugs. Selected patients, such as those with a reactive arthropathy, may benefit.
- Prednisolone 40 mg daily is appropriate for patients in whom a prompt response is required, or those with mild to moderately active disease, in whom Mesalazine in appropriate dose has been unsuccessful.
- Prednisolone should be reduced gradually according to severity and patient response, generally over 8 weeks. More rapid reduction is associated with early relapse.
- Long-term treatment with steroids is undesirable. Patients with chronic active steroid dependent disease should be treated with azathioprine 1.5–2.5 mg/kg/day
- Topical agents (either steroids or Mesalazine) may be added to the above agents. Although they are unlikely to be effective alone, they may benefit some patients with troublesome rectal symptoms.
- Severe UC: close monitoring at a tertiary centre.

Maintenance of remission

Lifelong maintenance therapy is generally recommended for all patients, especially those with left sided or extensive disease, and those with distal disease who relapse more than once a year.

Discontinuation of medication may be reasonable for those with distal disease who have been in remission for 2 years and are averse to such medication. However, there is some evidence that maintenance therapy reduces the risk of colo-rectal cancer.

For the maintenance of remission in UC:

- Oral Mesalazine 1–2 g daily or Balsalazide 2.5 g daily should be considered as first line therapy.
- Sulpha-salazine 2–4 g daily has a higher incidence of side effects compared with newer 5-ASA drugs.
- Selected patients, such as those with a reactive arthropathy, may benefit.
- Topical Mesalazine 1 g daily may be used in patients with distal disease with/without oral Mesalazine, but patients are less likely to be compliant.
- Most patients require lifelong therapy, although some patients with very infrequent relapses (especially if with limited extent of disease) may remain in remission without maintenance therapy.
- The advantages and disadvantages of continued treatment with amino-salicylates are best discussed with the patient, especially if a patient has been in remission for a substantial length of time (>2 years).
- Steroids are ineffective at maintaining remission.
- Azathioprine 1.5–2.5 mg/kg/day or mercaptopurine 0.75–1.5 mg/kg/day are effective at maintaining remission in UC. However, in view of toxicity they should be reserved for patients who frequently relapse despite adequate doses of amino-salicylates, or are intolerant of 5-ASA therapy. It is common practice to continue amino-salicylates with azathioprine, but limited evidence that this is necessary.
- Patients with gastrointestinal intolerance of azathioprine may be cautiously tried on Mercaptopurine before being considered for other therapy or surgery.

Important Note

Regular surveillance is necessary for UC lasting

for more than 10 years.

Explain to the patient the chronic nature of the disease and continuation of maintenance treatment for life with regular follow-up. Risk of colonic cancer after 10 years of disease onset must be explained

Do's and Don't's

Milk is preferably avoided during the acute phase of illness.

Amoebic liver abscess

Causes

Liver abscess is the commonest extra-intestinal form of amoebiasis, caused by E. histolytica. The disease usually affects young males, particularly chronic alcoholics, in endemic areas.

Causes

- Clinical manifestations are high grade fever, RUQ pain, which may be dull aching or pleuritic in nature.
- Symptoms are often chronic with intermittent fever and constitutional symptoms.
- Jaundice is uncommon. Liver abscess is less common in the elderly.

Complications

Include rupture of abscess into pleural, pericardial or rarely peritoneal cavity.

Investigations

- The diagnosis is made by an elevated total count,ESR and serum alkaline phosphatase,
- One or more hypo-echoic lesions in liver on ultrasonography.
- Stool is postivie for E histolytica in 30%.
- Antibodies to E. histolytica is high.
- Examination of pus for the parasite is usually negative.

Treatment

1. Tab. Metronidazole 800 mg three times orally (or IV, if necessary) daily for 5-10 days

   or

   Tab. Tinidazole 600 mg 2 times a day for 7-10 days
2. If the patient is very toxic, Inj. Metronidazole 500 mg given 8 th hourly until patient improves. Switch over to oral therapy whenever possible. Followed by Diloxanide furoate (luminal agent for cysts) 500 mg three times a day for 10 days.
3. Chloroquine

   Chloroquine 600 mg orally daily 2 days, followed by 300 mg daily for 2 weeks; dose is calculated as chloroquine base. Drug is active against E. histolytica trophozoites

Indications for drainage of an abscess:

1. If pyogenic abscess cannot be excluded
2. No improvement with medical therapy in 72 hours
3. Impending rupture of abscess (severe pain, pleuritic pain, hiccups)- one very close to the surface of the liver
4. Large left lobe abscess, to prevent rupture in to the pericardium

Follow-up:

1. Monitor the patient for resolution of symptoms with medical treatment and aspirate if any indication.
2. Abscess cavity may persist for several weeks even after cure of infection. Frequent US scan is unnecessary unless patient develops fever etc. Scan may be repeated after 4-6 weeks, after the patient becomes asymptomatic.
3. Tab. Di-loxanide furoate 500 mg 3 times a day for 1 week may be given after a course of Metronidazole

Do's and Don'ts

1. Avoid taking alcohol, specifically if on treatment with Metronidazole
2. Avoid contaminated food and drinking water. Vegetables should be cooked or washed well.
3. Use boiled water (kills the cyst) or bottled water from a known source.
4. Maintain good hygiene during food intake to prevent enteric infections

References

1. Sharma MP, Ahuja V. Management of amebic and pyogenic liver abscess. Indian J Gastroenterol 2001;20:33-6
2. Amoebiasis and infection with free living amoeba. In: Harrison's Principles of Internal Medicine, Kasper DL, Braunwald E, Fauci AS et al (eds), 16th edition, 2005, McGraw Hill Company Inc., New York, pp 1214-18
3. Seeto RK: Amebic liver abscess: epidemiology, clinical features, and outcome. West J Med 1999, 170:104-109. Indications foe aspiration of amoebic liver abscess
4. Hughes MA: Amebic liver abscess. Infect Dis Clin North Am 2000, 14:565-582.

Pyogenic liver abscess

Liver abscess constitutes about 48% of all visceral abscesses.

Causes

Pyogenic liver abscess is usually caused by spread of infection from peritoneum, abdominal viscera such as appendicitis/ diverticulitis/portal pyemia or disease of biliary tract. It is most commonly caused by coliform organisms.

Symptoms

Fever, abdominal pain, toxaemia, features of associated problems such as appendicular pain/ mass etc. Mostly abscesses are small and multiple.

Investigations

Diagnostic investigations include total counts, US scan of the abdomen, blood culture, pus culture. CECT and MRI is seldom indicated.

Treatment

1. Drainage- percutaneous catheter or open surgical- remains the mainstay of treatment for large abscess
2. Patient should be kept nil by mouth and given IV fluids if toxic and sick.

Recommended antibiotics

Metronidazole plus ampicillin and gentamicin, ciprofloxacin, or a third-generation cephalosporin (eg, ceftriaxone or cefotaxime).

Standard dosage

- Ceftriaxone 2 g intravenously every 24 hours, or cefotaxime 2 g intravenously every 8 hours.
- Initial empirical treatment should include broad-spectrum antibiotics.
- Various combinations recommended are:

  Metronidazole : 500 mg I/V three times daily

  Ampicillin : 2 g I/V 6 hourly

  Gentamicin : 2 mg/kg load, then adjust for renal function

  Ciprofloxacin : 400 mg I/V 12 hourly for 10 days

  or

  Inj. Ceftriaxone 1-2 g IV every 24 hours times a day

  or

  Inj. Cefotaxmine 2 g 8-hrly for 10 days Combination of Amoxycillin +Ciprofloxacin +Metronidazole is also a recommended schedule

  In the elderly or those with renal impairment: a Penicillin (such as amoxicillin) plus an injectable Cephalosporin (such as cefotaxime or cefuroxime) plus metronidazole is recommended

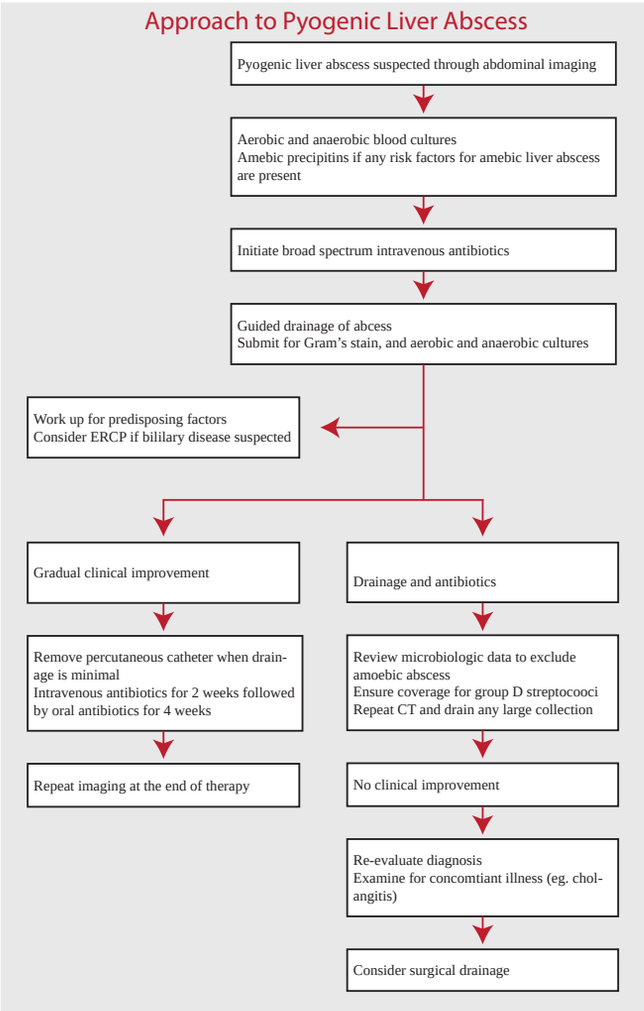
  In penicillin –allergic patients: ciprofloxacin plus clindamycin
- Once the sensitivity is known, antibiotic therapy is amended accordingly. Duration of therapy is usually from 2–4 weeks or longer depending on number of abscesses and the clinical response.

Follow-up:

1. Monitor for clinical improvement and modify the therapy based on culture sensitivity report
2. Abscess should always be drained.
3. Surgery considered if no improvement with medical treatment and percutaneous drainage in 4-7 days.

Important Note

1. Avoid taking alcohol, specifically if on treatment with Metronidazole.
2. Maintain good hygiene regarding food intake to prevent enteric infections.



Reference

- Sharma MP, Ahuja V. Management of amebic and pyogenic liver abscess. Indian J Gastroenterol 2001;20:33-6
- Intra-abdominal Infections and Abscesses. In: Harrison's Principles of Internal Medicine, Kasper DL, Braunwald E, Fauci AS et al (eds), 16th edition, 2005, McGraw Hill Company Inc., New York, pp 749-54

Acute Pancreatitis

Causes

Acute inflammation of the pancreas, usually caused by alcohol or gall stones getting impacted at the ampulla. Rare causes include

* Trauma
* Infections such as mumps, ascariasis and
* Drugs such as diuretics and azathioprine.

Symptoms

- Acute upper abdominal pain, radiating pain to the back, crouched posture.
- In severe cases, there is paralytic ileus, vomiting abdominal distention, jaundice and fever.

Investigations

1. Diagnosis is confirmed by an elevated serum amylase to more than 3 times the normal. One needs to exclude other causes of acute abdomen like gut perforation or infarction. A repeat assay is done after 48 hours.
2. Serum lipase elevation is more specific for pancreatitis. USG or CT scan further helps to confirm the diagnosis

Complications

* Necrosis
* Haemorrhage
* Pseudo-cyst
* Abscess
* Pleural effusion and
* Other end organ failure.

Treatment

- If the disease is mild and there are no signs of hypovolemia: Start intravenous fluids
  * Infusion Ringer lactate followed by 4 to 6 pints of I/V normal saline, maintaining a good urine output
  * Inj KCl 60-80 mmol (20 mmol added to 50 ml of IV fluid)
- Patient is kept nil oral until bowel sounds appear or patient continues to vomit.
- Oral feeds are initiated at the earliest, when the general condition is stable i.e. within 72 hours
- The diet should be low in fat and protein.
- Analgesics are recommended for pain relief
  * Inj Diclofenac Sodium 75 mg IM 2-3 times a day;
  * If pain is not relieved, Inj Tramadol 50 mg I/M, repeated hourly if needed
- Antibiotics are not to be given as a routine. There is no role for octreotide/somatostatin.
- If gallstones with stones in common bile duct are detected on ultrasonogram, emergency ERCP is warranted to disimpact the distal stone.

- Interval cholecystectomy is recommended after resolution of acute pancreatitis.
- In case there is deterioration in general condition or patient has evidence of severe necrotizing pancreatitisof the patient referral to a tertiary centre is recommended.

Markers of severe Pancreatitis include

1. WBC count > 15 000 cells/cu mm
2. Blood urea > 16 mmol/l
3. Calcium < 2.0 mmol/l
4. Albumin < 3.2 g/dl
5. Glucose > 10 mmol/l
6. pO2 < 8 pKa (60 mm Hg)
7. AST>200 iu/l
8. LDH > 600 iu/l
9. C-reactive protein >150 mg/l

Management of acute pancreatitis

All patients with (amylase > 4-fold elevated)

Analgesia (Pethidine, not morphine)

Nil by mouth

Plain abdominal and erect chest x-ray (exclude perforation)

Urinary catheter

Nasogastric tube if vomiting

Assess severity by objective criteria

Mild	Severe
Less than 3/9 markers of severity	More than 3/9 markers of severity
Admit	Transfer to higher center
I/V crystalloids (often sufficient)	Peripheral venous line
Restart diet after 48 to 72 hours if improving	Urgent ultrasound to exclude gallstones
	No gall stones
	Remove catheter at 24 hours
	Consider other causes of acute pancreatitis

Reference

1. Approach to the patient with Pancreatic disease. In: Harrison's Principles of Internal Medicine, Kasper DL, Braunwald E, Fauci AS et al (eds), 16th edition, 2005, McGraw Hill Company Inc., New York, pp 1891-1906
2. Acute and Chronic Pancreatitis. In: Harrison's Principles of Internal Medicine, Kasper DL, Braunwald E, Fauci AS et al (eds), 16th edition, 2005, McGraw Hill Company Inc., New York, pp 1895-1906

Chronic Pancreatitis

Causes

Usually caused by chronic alcohol consumption or due to tropical calcific pancreatitis.

Symptoms

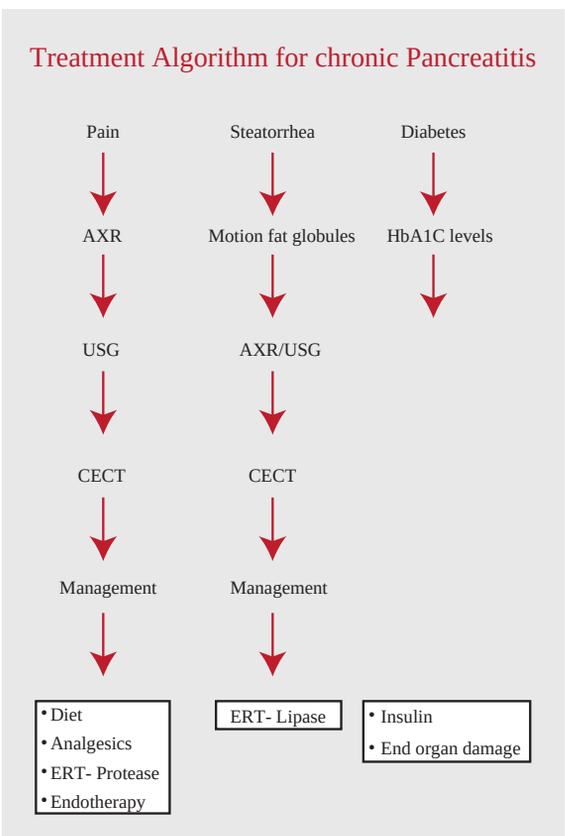
Typically patients present with pancreatic type of pain, steatorrhoea and Diabetes mellitus. Diabetes is often non-ketotic, though blood sugar levels are grossly elevated.

Investigations

The diagnosis is confirmed by plain x-ray abdomen (for calcification in bed of pancreas),stool examination for fat globules and a CECT.

Treatment

Medical treatment



Diet

Daily diet of 2000-3000 calories, consisting of 1.5-2 g/kg of protein, 5-6 g/kg of carbohydrates, and 20-25% of total calories consumed as fat (about 50-75 g) per day.

Oral supplementation of fat soluble vitamins (A, D, E, and K) and vitamin B-12 is recommended.

Small frequent, low fat meals

Medium chain triglycerides

Coconut oil based

Micronutrient supplementation

Diabetic diet

Drugs

- Pancreatic enzyme replacement therapy
- High proteases containing preparation
- Adequate dose to be taken along with a meal
- For non-enteric coated preparation combine with pre-meal H2RA/PPI therapy
- Treatment continued until 8 weeks
- If no response: refer to higher centre for endo-therapy
- Others
  - * Analgesics: Tramadol
  - * For Steatorrhoea:
  - * Medium chain triglycerides
  - * Coconut oil based
  - * Enzyme preparation 25,000 units to 30,000 units of lipase
  - * With every main meal
  - * 10,000 units with every fatty snack
  - * Antioxidants

Enzyme Preparations...local brand

Brand	Form	Lipase	Prot ease	Amylase
Panlipase	Micropearls 2mm	10000	37500	33200
Creon	Minimicro spheres	10000	37500	33200
Digemax	EC tab	20000	1500	15000
Festal	Ec tab	8000	18000	18000
Pankreoflat	Ec tab	6500		
Enzar Forte	Ec tab	4000	15000	15000

Reference

Acute and Chronic Pancreatitis. In: Harrison's Principles of Internal Medicine, Kasper DL, Braunwald E, Fauci AS et al (eds), 16th edition, 2005, McGraw Hill Company Inc.,

New York, pp 1895-1906

Gastrointestinal (GI) bleeding

Definition

Gastrointestinal (GI) bleed may present as a frank or an occult bleeding.

Upper GI bleed is defined as bleeding from any site from pharynx to duodeno-jejunal (DJ) flexure and usually presents as haematemesis or melaena (atleast 50 mL of blood in stool).

A malenic stool is black, tarry, sticky and foul smelling. It is necessary to exclude intake of iron, charcoal containing tablets or bismuth salts.

Lower GI bleed is defined as bleeding occurring beyond the ligament of Trietz is referred to as a lower GI bleed.

Symptoms

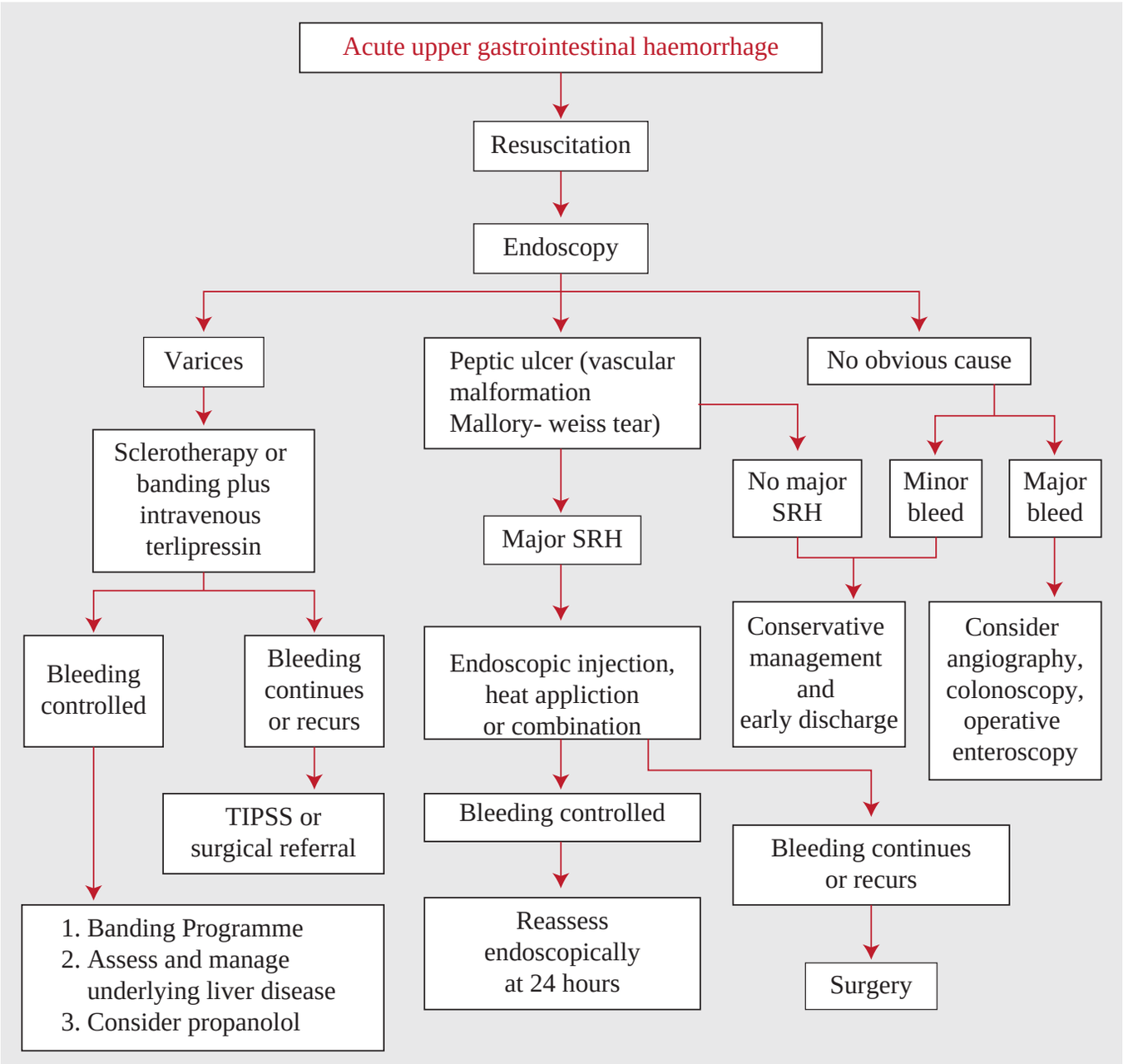
Any individual presenting with haematemesis or melena

- Assess degree of bleed
  - * A patient walking into the casualty with no history of giddiness is likely to have 500 mL of blood loss
  - * Those with giddiness, postural hypotension are likely to have had approximately 1000 to 1500 mL of blood loss
  - * Those coming in a state of shock are likely to have lost more than 1500 mL of blood.

Treatment

Resuscitation is the key to management of any GI bleed especially if moderate to massive. An UGI scopy will not be helpful to deal with an active massive bleed. First assess the clinical status of the individual:

- Assess pulse, blood pressure, postural change in blood pressure
- Pass a ryles tube to exclude ongoing bleed



Investigations

- Collect blood samples for Hb, TC, platelets, coagulation profile, renal and liver function tests, blood grouping and cross matching; Repeat Hb after 6 hrs
- Start normal saline. I/V Ringer lactate, and arrange for blood transfusion if postural hypotension is present.
- Replace blood as soon as possible if moderate or severe bleed or active bleed.
- Intravenous Pantoprazole 40 mg as a stat dose. (If there is a history of NSAIDs or patient is a known case of peptic ulcer disease, immediately give)

Important Note

Do not attempt to give a stomach wash. this can displace a blood clot overlying an ulcer.

- In case the patient is a known case of chronic liver disease with portal hypertension, Inj Octreotide 50 mcg IV immediately followed by 25 mcg/hr infusion. or Somatostatin infusion: 250 micro gm stat followed by hrly dose injection for atleast 5 days Insertion of Sengstaken Blakemore tube can be life saving. Patient can be transferred to a higher centre with the tube in situ.

Reference:

Gastrointestinal bleeding. In: Harrison's Principles of

Internal Medicine, Kasper DL, Braunwald E, Fauci AS et al (eds), 16th edition, 2005, McGraw Hill Company Inc., New York, pp 235-8

Neurology

Standard Treatment Guidelines
Tamil Nadu Health Systems Project

Chapter 7

- Headache
- Dementia
- Epilepsy
- Facial Nerve
- Parkinson's Disease
- GBS
- Stroke
  - Hemiplegia
  - Paraplegia
  - Quadriplegia
- Acute Bacterial Meningitis
- TBM
- Herpes Simplex Encephalitis
- Japanese Encephalitis
- Neurocysticercosis
- Vertigo
  - Peripheral Vestibulopathy
  - Otitic Herpes Zoster
  - Meniere's disease
  - Vestibular Neuronitis

Head ache

Introduction

Many causes of headache have been described in medical literature over the years. For practical clinical purposes, however, all headaches can be classified as one of the primary headache syndromes or as a headache that is caused by or secondary to an underlying disease process or condition. Because primary headaches are the most common, this discussion focuses on the diagnosis and management of those syndromes.

Definition

The primary headache syndromes are migraine, tension-type, and cluster. Migraine and cluster headaches are episodic and recurring conditions.

- Tension-type headache is usually episodic but may be chronic, occurring daily or almost daily for more than 15 days a month.
- None of these headaches are associated with demonstrable organic disease or structural neurologic abnormality.
- Laboratory and imaging test results are generally normal; however, if an abnormality is found, it most likely is not the cause of the headache. Similarly, physical and neurologic examination results also are usually normal, but abnormalities found are not related to the headache.
- During the headache, however, cluster and migraine patients may have some abnormal clinical findings, and many patients with tension-type headache will have demonstrable tightness in the cervical muscles, with limitation of neck motion and/or scalp tenderness.
- Secondary headaches are usually of recent onset and associated with abnormalities found on clinical examination. Laboratory testing and/or imaging studies will confirm the diagnosis.
- Recognizing headaches related to a condition or disease is critical not only because treatment of the underlying problem will usually eliminate the headache but also because the condition causing the headache may be life-threatening.

Prevalence

- Primary headaches account for more than 90% of all headache complaints and, of these, episodic tension-type headache is the most common. Almost everyone has had an occasional headache of this type. Although chronic tension-type headache occurs in only slightly more than 2% of the population, it accounts for a large number of visits to the physician's office and missed work days.
- Several epidemiologic studies conducted in various areas of the world indicate that the prevalence of migraine headache ranges from 12% to 18% of the population. Migraine is three times more common in females. The prevalence of cluster headache is not known. This uncommon condition probably affects less than 0.5% of the population but is underdiagnosed and is often believed to be a sinus problem.
- Cluster headache affects men eight to ten times more frequently than women. Because the three primary headache syndromes tend to begin in persons younger than 50 years, anyone older than 50 with a recent onset of headaches should have a thorough examination and testing to look for an underlying cause.

Symptoms

Migraine

- Most migraine patients do not have an aura; migraine with aura occurs in only 15% to 20% of sufferers.
  - The aura is a well defined visual or neurological deficit lasting less than 1 hour and is followed by the headache within an hour.
  - Most auras are visual, with photopsia (flashing lights) being most common.
  - The aura is initially small, then enlarges or moves across the visual field.
  - A typical migraine aura may occur without a headache. This phenomenon tends to begin later in life.
  - Occasionally, a neurologic aura will occur, with a tingling or weakness that slowly spreads up or down an extremity.
- Many patients with migraine will have prodromal symptoms for many hours or even a day or so before the onset of an attack. These prodromal symptoms are generally changes in mood or personality. Fatigue also is common, and occasionally hyperactivity occurs.
- The migraine attack lasts 6 to 72 hours. This pounding, throbbing pain of moderate to severe

intensity is generally unilateral, but some patients will experience bilateral pain. Pain caused by migraine worsens with physical activity.

- Photophobia and phonophobia are very common, with sensitivity to odors being a little less common. Migraine is a sick headache.
- Nausea occurs in most patients, and vomiting is very common.
- Dehydration may occur, which will increase the pain and disability of the condition. Migraineurs' want to be quiet, inactive, and in a darkened area during the attack.
- Approximately 60% of women experience their worst migraine attacks in conjunction with their menstrual period.

Tension-type headache

- Tension-type headache is characterized by generalized pressure or a sensation of tightness in the head.
  - The discomfort level is usually mild to moderate and does not worsen with activity.
  - Although nausea and photophobia or phonophobia may occur, they generally are not prominent features.
  - Tension-type headache can be episodic (less than 15 days a month) or chronic (more than 15 days a month).
  - Poor posture is often evident, which may play a role in causing tension-type headache.

Cluster headache

- Cluster headache causes intense pain that is generally steady and boring behind one eye.
  - The pain may spread to the temple, face, and even back into the upper neck.
  - It is so intense that most sufferers will pace the floor or do vigorous exercises during the attack.
  - The attacks are short (usually less than 3 hours in duration) and often last only 30 to 45 minutes.
  - They occur from one to several times a day for a period of several weeks or months, then remit, leaving the patient pain free for several months or years, only to recur.
  - During a cluster headache cycle, the attacks of pain often occur at the same time each day, most often waking the patient in the early morning hours.
  - Eighty percent of cluster sufferers experience unilateral tearing, with conjunctival injection and ipsilateral nasal congestion.
  - About 20% of cluster patients have a partial Horner's syndrome with ptosis and meiosis of the affected eye. These symptoms clear as the attack leaves.
  - Alcohol will bring on an attack within a few minutes in a patient.

Investigations

- History
- Clinical examination
- Laboratory Studies to exclude metabolic or endocrine factors
- A high erythrocyte sedimentation rate in a patient older than 50 years with new headache onset suggests temporal arteritis. This diagnosis must be confirmed by temporal artery biopsy.
- CT Scans can be performed to exclude intracranial causes of headache or to rule out lesions that cause neurologic or visual abnormalities associated with headache. Computerized tomography scanning of the paranasal sinuses is useful in evaluating the role of acute or chronic sinus infection in the etiology of the headache.
- Magnetic resonance imaging is currently the best scan for viewing the posterior fossa and occipital areas of the brain.

Treatment

Migraine Management

- Some migraine headaches can be relieved with
  - The use of cold packs
  - Pressure on the temple, and sleep
  - However, most require preventive or abortive medication.
- Educating the migraine patient to recognize and avoid headache triggers helps to reduce the frequency of attacks.
- Common migraine triggers include
  - Weather changes

* The estrogen cycle
* Bright lights
* Strong odours
* Stress
* Food and food additives, and the skipping of meals

- Migraineurs do better and have fewer headaches by following regular eating and sleeping patterns.

Migraine Preventive Medication

- Drugs used for migraine prophylaxis
- Beta-blockers
- Calcium channel blockers
- Nonsteroidal anti-inflammatory drugs (NSAIDs)
- Anti-depressants
- Anti-epileptic drugs

Others

Large doses of riboflavin or magnesium oxide and the serotonin agonist Methysergide

Migraine abortive medication

- All migraine patients suffering an acute attack need to take an abortive drug, whether or not they are taking a prophylactic agent.
- For mild attacks, over-the-counter analgesics (especially those containing caffeine) may be useful.
- Effective agents available by prescription include a
  * Combination of isometheptene mucate
  * Dichloralphenazone
  * Acetaminophen
  * Ergotamine tartrate combined with caffeine
  * Dihydroergotamine mesylate and the triptans.
- Large doses of rapid-acting NSAIDs, such as meclofenamate, ibuprofen, or naproxen sodium, can also prove helpful in mild attacks.
- Ergotamine tartrate combined with caffine (oral, suppository) is quite effective but often causes nausea. Dihydroergotamine (DHE 45) is a very effective abortive agent when used parenterally. It is also available as a nasal spray.
- Intravenous DHE, Prochlorperazine, Divalproex sodium, magnesium and Ketorolac can be very effective in stopping a migraine attack

Tension-type headache management

- The occasional tension-type headache can be alleviated by a hot shower, massage, sleep and through patient recognition and avoidance of stress factors.
- Episodic tension-type headache is usually well treated with analgesics such as aspirin, acetaminophen, and NSAIDS or combinations of these agents with caffeine or sedatives.
- Some patients, particularly those with tension-type headache caused by stress, may benefit from relaxation techniques or biofeedback training. Physical therapy may decrease chronic neck pain caused by increased cervical muscle spasm or postural abnormalities.
- The most effective group of drugs for treatment of chronic tension-type headaches is the tricyclic antidepressants. Amitriptyline HCl, doxepin HCl and nortriptyline HCl are the most commonly used.
- Muscle relaxants such as cyclobenzaprine HCl, orphenadrine citrate, and baclofen may be helpful at times, particularly if increased muscle spasm is present.
- In recent clinical trials, the central-acting muscle relaxant tizanidine HCl was found to be effective in treating chronic headaches, either tension-type or coexisting migraine and tension-type.

Cluster headache treatment

- Because the onset of cluster headache attacks is rapid and may occur several times a day, the best approach to treatment is with daily preventive drugs.
- Effective prophylactic medications include verapamil HCl, prednisone, lithium carbonate, methysergide, and the antiepileptic drugs divalproex and topiramate. High doses of verapamil (480 mg to 720 mg / day) may be necessary.
- Prednisone and methysergide work quickly and often will be used with verapamil or lithium at onset, for a quick response and then tapered while verapamil or lithium is continued. Prednisone is usually prescribed at 60 mg / day initially and then tapered over 2 to 3 weeks.
- The use of 100% oxygen by mask at a flow rate of 8 to 10 L/min for up to 10 minutes will abort

an acute cluster headache in 50% to 60% of patients.

- Ergotamine tartrate, DHE and any of the triptans usually are very effective, but are inappropriate in patients who suffer several attacks a day.
- Fortunately, cluster headache patients do not appear to develop rebound headaches from frequent use of ergotamine tartrate or the triptans, as do migraine sufferers.

Dementia

Dementia denotes a deterioration of intellectual or mental ability with little or no disturbance of consciousness and is usually associated with behavioural and personality changes.

It is generally considered as a disease of the elderly, (senile dementia > 65 year age) though cases occur in the slightly younger age group (pre-senile dementia age < 65 years).

Definition

Dementia is defined as a decline in memory and atleast one other cognitive function such as aphasia, apraxia, agnosia or decline in an executive function such as planning, organizing, sequencing or abstraction.

- This decline impairs social or occupational functioning in comparison to previous functioning.
- The deficit should not be attributed to delirium or psychiatric illness.

[According to the Diagnostic and statistical manual of mental disorders–IV (DSM–IV) criteria]

Incidence

- 1.03% of the whole population
- > 4% of persons more than 65 years, 20% in persons > 85 years.
- > 50 – 60% of patients with dementia are due to Alzheimer's disease.

Causes

Degenerative

- Alzheimer's disease
- Lewy body disease
- Parkinson's disease
- Fronto-temporal dementia
- Progressive supra-nuclear palsy
- Multisystem atrophy
- Huntington's disease

Vascular

- Multiple infarcts
- Binswanger's disease
- Normal pressure hydrocephalus (NPH)
- Subarachnoid hemorrhage
- Vasculitis

MRI of Hydrocephalus due to Aqueduct stenosis

Dilated Lateral Ventricles

Sagittal image

- Single infarcts at strategic locations

Infections

- Fungal meningitis
- AIDS dementia
- Syphilis
- Cruetzfeldt – Jacob disease

Toxic or metabolic

- Vitamin B_{12} deficiency
- Thyroid deficiency
- End organ damage (Liver, Kidney etc.)
- Alcohol Abuse

Traumatic

- Subdural hematoma
- Anoxic brain injury
- Closed head injury

Tumor

- Glioblastoma
- Lymphoma
- Metastatic tumour

Others:

- Symptomatic hydrocephalus

Note

Each of the diseases in degenerative dementias has specific criterias to diagnose.Memory problems are common among the elderly but do not always herald the onset of dementia. Isolated memory impairment (or) mild cognitive impairment (MCI) progresses to dementia at a rate of about 10% per year. But people with MCI have normal activities of daily living and normal general cognitive functioning.

Symptoms

History

It is essential to take history not only from the patient but also from an independent informant. Enquiries about the memory and the difficulties, the patient has in carrying out his Activities of Daily Living (ADL) at home and work place are made. Activities related to shopping, hobbies, handling money, food preparation, dressing etc are enquired about.

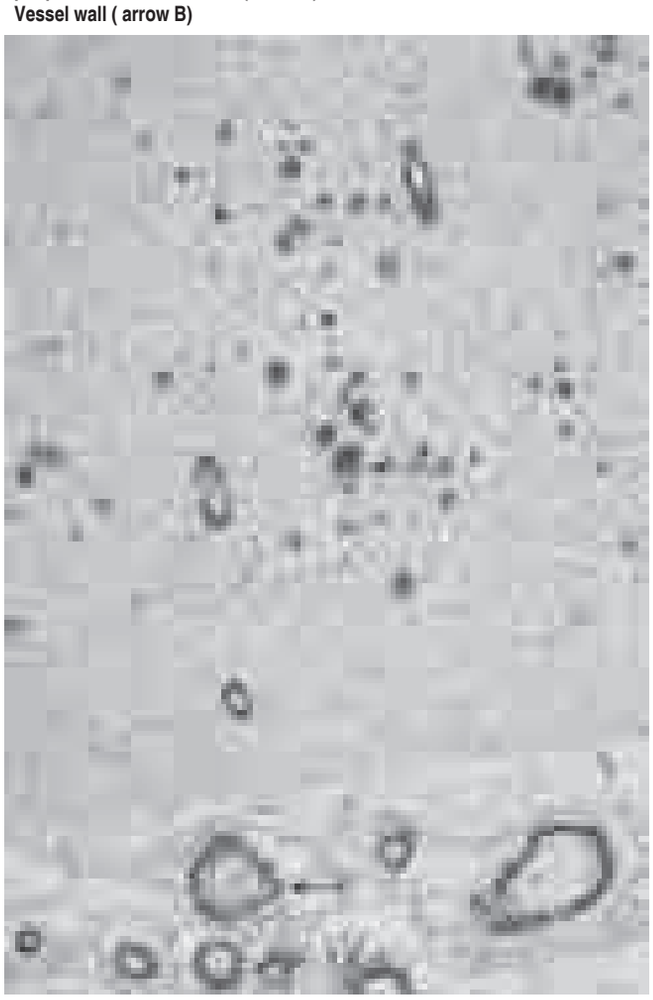
Etiology directed questions such as risk factors for vascular disease, toxin exposure, head injury, medication, systemic illness, and alcohol exposure are asked.

Examination

- Because dementia must be promptly distinguished from delirium, attention is assessed first. If the patient is overtly distractible or otherwise unable to maintain attention, the diagnosis is likely to be delirium, although dementia impairs attention especially in the later stages.
- The ability to register information is evaluated by presenting the names of three objects to patients, who are asked to repeat the names immediately. If patients cannot do so, the problem is usually attention not memory.
- If patients can register information, short term memory is tested after 5 minutes. Asking the patient to name objects with categories (e.g.

Alzheimer's Disease

(section of Neo cortex shows amyloid deposits in plaques in brain substance (arrow A) and in blood Vessel wall (arrow B)



animals, articles of clothing, furniture) is another useful test.

- Functional activities questionnaire is used to evaluate whether cognitive impairment affects a patient's ability to perform instrumental and other complex activities of daily living.

Several versions of bedside mental status testing are available. Folsteins Mini-Mental status examination is the most widely used.

Mini - Mental status examination	
5 For	Orientation to time (year, season, month, date and day)
5 For	Orientation to place (state, country, town, hospital and floor)
5 For	Attention (either serial subtraction in 7s with one point for each of the first five subtractions)
3 For	Registration of three items (e.g. Tree, Ball, Gold)
3 For	Recall of three items after 5 minutes
2 For	Naming a pencil and a watch
1 For	Repeating "no its, and or buts"
3 For	Following a three – staged command
1 For	Following a printed command "close your eyes"
1 For	Writing a sentence
1 For	Copying a diagram of two intersecting pentagons

The MMSE consists of 30 points, the low – normal cutoff is estimated to be 19 for uneducated people, 23 for elementary graduates, 27 for high school graduates, 29 for college graduates. Age factor also should be considered.

A full neurologic examination has to be done with special attention to visual fields, paresis, ataxia, sensory disturbance, extrapyramidal disturbance etc.,

Investigations

Recommended for all patients:

Complete blood cell count, chemistry panel such as sugar, urea, creatinine, electrolytes, liver function tests, thyroid function tests, chest x-ray, vitamin B_{12} level, syphilis serological testing, computed tomography or magnetic resonance imaging and neuropsychological evaluation.

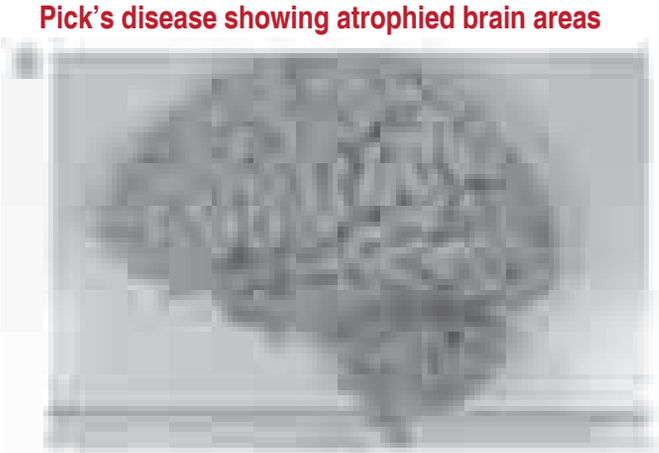
Recommended for selected patients:

Lumbar puncture, Electroencephalography, heavy metal screening, HIV test, SPECT, drug screening, etc.,

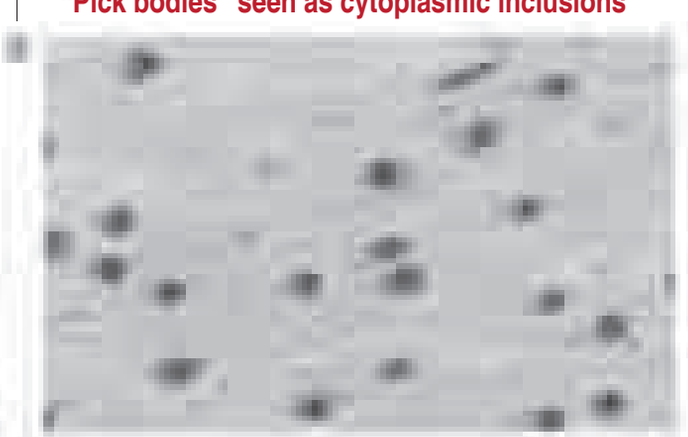
Treatment

- The primary importance of the physician is to identify any treatable causes (reversible dementia) for the symptom. For example vitamin supplements for the B_{12} deficiency states, abstinence of alcohol and vitamin supplements for alcohol-induced dementia, treatment of depression, surgical treatment for NPH, tumours, ART for AIDS dementia and treatment of infections such as syphilis. After about 6-12 weeks of treatment, reevaluation has to be done and improvement should be documented.
- Once it is established that the patient has any untreatable dementing brain disease and the diagnosis is sufficiently certain, a responsible member of the family should be informed of the medical facts and prognosis and assisted in the initiation of social and supportive services.
- The value of newer, centrally acting cholinergic agents and glutamate antagonists in the treatment of Alzheimer disease is clear, but mod-

Pick's disease showing atrophied brain areas



"Pick bodies" seen as cytoplasmic inclusions



est and should be weighed against the need, side effects and economical back ground. The cholinesterase inhibitors Donepezil, Rivastigmine and Galantamine are somewhat effective in delaying the progression of AD and dementia with lewy bodies and also in other forms. Memantine, an NMDA antagonist, may help delay progression of moderate to severe dementia and can be used with other drugs.

- Treatment of dementia does not stop with pharmacotherapy alone. Environment measures such as not exposing the patient to newer places, keeping dangerous things such as guns, sickles, power tools and sharp objects away from reach, closing the wells and tanks, preventing him from driving etc has to be adopted. Patients are to be encouraged to have regular meals, exercise and clean habits.
- Care giver stress is common. Health care professionals must provide support for family members in medical and non-medical issues including financial planning.
- As dementia worsens, highly aggressive interventions and hospital care provide less benefit and may not be worth their costs, discomforts and risks. Palliative care should be routinely offered. Decision about artificial feeding and treatment of acute disorders are best discussed before such a situation occurs and then discussed again when the situation arises. In severe dementia, maximising comfort may be more appropriate than attempts to prolong life.

References

- Principles of Neurology - Adams and Victors
- Neurology in Clinical Practice - Bradley
- Neurological Differential Diagnosis - John Patten

Epilepsy

ICD Code number: G 40.9

Definition

Seizure

Defined as uncontrolled electrical activity in the brain, which may produce a physical convulsion, minor physical signs, thought disturbances or a combination of symptoms.

It manifests as sudden and transitory abnormal phenomenon which can be in the form of altered consciousness, motor, sensory, autonomic and psychic.

Epilepsy

It is a disease characterized by the tendency to develop recurrent seizures due to abnormal / refractory underlying state

Epidemiology

- Around 50 million people in the world have epilepsy.
- It is the commonest and most serious neurological condition.
- Annual incidence is around 50 – 70 per 100,000 population.
- It is more in the developing countries due to primitive obstetric services, CNS infections and head trauma.
- Incidence varies according to age. It is more in the early childhood and older age above 65 yrs.
- Point prevalence is 1 % of the general population.

Clinical aspects

Classification of Seizures

[ILAE: International league against epilepsy]

- Partial Seizure:
  - Simple Partial (with no LOC)
  - Complex Partial (with impaired consciousness)
  - Partial leading to Secondary generalization.
- Generalized seizure:
  - Absence – Typical, Atypical seizures
  - Tonic seizures
  - Clonic seizures
  - Tonic – Clonic seizures
  - Myoclonic seizures
  - Atonic seizures
  - Unclassified seizures

Symptoms

Simple partial seizure

Depend on the site of origin of electrical discharges.

Consciousness is normal.

Motor → Tonic, clonic movements of opposite arm, leg and face.

Sensory → Paresthesia, olfactory, visual and auditory hallucinations.

Autonomic → Sweating and fear.

Psychic → Euphoria, panic, de javu, jamais vu.

Complex Partial Seizure

Also called as temporal lobe epilepsy.

It starts with aura or warning followed by vacant stare, unresponsiveness, "tonic and clonic" jerks followed by automatism in the form of repetitive semi-purposive behavior such as facial grimacing, gesturing, lip smacking, chewing, snapping the fingers, repetitive words, walking, running and undressing. It lasts for 3 –5 minutes with the post - ictal state of few minutes to hours.

Generalized seizure

It is due to widespread involvement of bilateral cortical regions.

Tonic–Clonic seizure (GTCS)

Patient develops an ictal cry followed by tonic posturing of all four limbs and then clonic jerks of both upper and lower limbs, upward gaze, tongue bite and urinary incontinence followed by postictal confusion.

Absence Seizure

Sudden onset of vacant stare, unawareness, eye blinking and lip smacking lasting for 5 – 10 seconds occurring in clusters.

Myoclonic Seizure

Sudden, brief muscle contraction occurs singly or in groups.

Atonic Seizure (Drop Attacks)

Sudden loss of muscle tone leading to fall and injuries to face.

Febrile Seizure

- It occurs in the age group between 6 month to 6 years.
- Sudden cessation of activity with uprolling of eyes, partial or generalized tonic clonic seizures (GTCS).
- It develops on the first day of fever with no evidence of CNS infection.
- Clinically confirmed by Cerebrospinal fluid analysis when required

Differential Diagnosis

- Syncope
- Migraine
- Transient ischemic attack
- Transient global amnesia
- Psychogenic Seizure (Pseudo-seizure)
- Non-Epileptic attack disorder.

Investigations

Diagnosis remains essentially clinical. But few investigations may be useful.

Electro encephalography (EEG)

It is helpful in the classification of seizure and epilepsy, to diagnose non-convulsive status epilepticus and pseudo seizures. Video EEG is ideal as it links EEG activity and clinical manifestation.

CT Brain

Useful for identifying calcification and tumors.

MRI Brain

- The investigation of choice in epilepsy and is superior to CT
- It is very helpful to diagnose
  - * Malformations of cortical development
  - * Hippocampal sclerosis
  - * Arterio-venous malformations
  - * Low grade glioma

Others

- Complete hemogram
- Renal Function Tests
- Sereum electrolytes, are needed routinely to rule out metabolic causes
- Cerebrospinal fluid analysis may be occasionally done if CNS infection is suspected

Treatment

Medical, Surgical.

Medical

* Anti epileptic drugs (AED) play an important role in the management of epilepsy.

When to start Anti epileptic drugs?

- After second GTCS or after first partial seizure.
- After first GTCS when associated with increased chance of recurrence like presence of CNS lesion, birth trauma, abnormal EEG and positive family history.
-

Choice of AED:	First Line AED	Second Line AED
Partial Seizure	CBZ, PHT, SVP, PHB	LTG, TPM.
Absence Seizure	SVP, LTG	ESM, CZP
Myoclonic Seizure	SVP	CZP, TPM, Zonisamide, Valproate, Lamotrigine
GTCS	SVP, CBZ, PHT, PHB	LTG, TPM.
Atonic Seizure	SVP	LTG.
Unclassifiable	SVP	LTG
Febrile Seizure – Intermittent Clobazam or diazepam.		
CBZ, Phenytoin are contraindicated in Absence, Myoclonic and Atonic Seizure.		
[CBZ- Carbamazepine, SVP-Sodium Valproate, PHT-Phenytoin, PHB-Phenobarbitone, LTG-Lamotrigine, TPM-Topiramate, CZP-Clonazepam, ESM-Ethosuximide]		

Anti-epileptic drugs

Dosage Schedule:		Starting dose	Maintenance dose	Frequency
Sodium Valproate	Children:	10 mg/kg/day	20 –40 mg/kg/day	bd –tds
	Adults:	400 mg/day	400 – 3000 mg/day	bd –tds
Carbamazapine	Children:	5 mg/kg/day	10 – 25 mg/kg/day	bd –tds.
	Adults:	200 mg/day	400 – 2000 mg/day	bd –tds
Phenytoin	Children:	5 mg/kg/day	5 – 8 mg/kg/day	od –bd.
	Adults:	200 mg/day	200 – 700 mg/day	od –bd
Phenobarbitone	Children:	5 mg/kg/day	5 – 8 mg/kg/day	od –bd
	Adults:	60 mg/day	60 –240 mg/day	od –bd
Clonazepam	Children:	0.025 mg/kg/day	0.025 – 0.1mg/kg	bd –tds
	Adults:	1 mg/day	2 –8 mg/day	od –bd
Clobazam	Children:	0.25 mg/kg/day	0.5 – 1.0 mg/kg/d	od - bd
	Adults:	10 mg/day	10 -40 mg/day	od –bd
Lamotrigine	Children:	0.5 mg/kg/day	2 –8 mg/kg/d	od - bd.
	Adults:	12.5 - 25 mg/day	100 -800 mg/day	od –bd
Topiramate	Children:	0.5 –1 mg/kg/day	5 – 9 mg/kg/d	bd.
	Adults:	25 – 50 mg/day	100 -500 mg/day	bd

Which Anti epileptic drug?

Principles of AED Therapy:

- It depends on the type of seizure, age of the patient and comorbid illness.
- Monotherapy is always ideal. Polypharmacy is needed in occasional cases
- Start at a low dose, slowly titrate over few weeks to establish effective and tolerable regimen

Strategies for treatment of newly diagnosed epilepsy

Newly diagnosed epilepsy
↓
First AED ⇒ Seizure free (47 %)
↓
Second AED ⇒ Seizure free (13 %)
↓ 40 %
Refractory
↓
Add on AED or Surgery.

Duration of AED

- It should be given for at least a 2 –3 years seizure free period, then slowly tapered and stopped over the next 6 months.
- Monitor for drug side effects and drug-withdrawl seizure.

Surgery

Indications:

- Refractory Epilepsy
- Polypharmacy and intolerable side effects

Types:

- Anterior temporal lobectomy
- Amygdalatomy
- Lesionectomy
- Rarely Corpus callosotomy and
- Sub-pial multiple resection

Prognosis

Majority of the epileptic patients have a good prognosis. 60 – 70 % become seizure free with AED. Around 30 – 40 % continues to have seizure with varying frequency and severity. After drug withdrawal, 10 % can develop a relapse.

Indicators of poor prognosis

- Symptomatic seizure
- High frequency of seizures
- Family history of epilepsy

Mortality

It is 2 –3 times above that of the general population. SUDEP (Sudden Unexpected Death in Epilepsy) is responsible for 17 % of total deaths.

When to admit?

For evaluation of first seizure, recurrent seizures and drug modification.

When to refer?

Persons with Refractory epilepsy on polypharmacy and with intolerable side effects.

References

- Williams H. Trescher and Ronald P. Lesser, 2004, The Epilepsies, Bradley's Neurology in Clinical Practice, 4th edition, pp 1953 – 1990.
- Martin J Brodie, Steven C, Patrick Kwan, 2005, Fast facts in Epilepsy, 3rd edition.
- Allan H.Ropper, Robert H.Brown, Epilepsies and other seizure disorders, Adam's Principles of Neurology, 8th edition, pp 273 – 299.

Facial Nerve Paralysis

ICD Code number: G 51.0

Definition

Facial nerve paralysis can be caused by lower motor neuron level or upper motor neuron level lesions.

Epidemiology

It is the most common disease of the facial nerve. Incidence rate is around 23 / 100,000 annually. It affects males and females equally occurs in all age groups. It is more common in diabetic and hypertensive patients.

Causes

Lower motor neuron palsy

- Bell's palsy - Idiopathic
- Viral infections

* HSV - Herpes simplex virus
* HZV - Herpes zoster virus
* HIV
* Infectious mononucleosis
* Polio

- Bacterial
  * Tuberculosis of mastoid bone or middle ear
  * Lyme disease
  * Syphilis
- Trauma like temporal bone fracture
- Tumors of parotid gland

Upper motor neuron palsy

- Stroke
- Demyelination
- Brain tumors

Features of lower motor neuron (LMN) palsy:

(On the same side of lesion)

- It is characterized by acute onset of illness.
- It reaches maximum paralysis in 48 hours in 50 %, in almost all by 5 days.
- It is usually preceded by pain behind the ear by one or two days.
- Complete paralysis of all muscles required for facial expression.
- Deviation of angle of mouth to opposite side, absence of wrinkling of forehead.
- Effacement of creases and folds, widening of palpebral fissure.
- Inability to close the eye with intact Bell's phenomenon.
- Saliva dribbles from the corner of the mouth.
- Lacrimation and salivation may be affected in some cases.

Features of upper motor neuron (UMN) palsy

(On the opposite side of lesion)

- Upper part of face is spared
- Dissociation of emotional and voluntary face movements
- Preservation of facial reflexes
- Taste and lacrimation is normal

Investigations

It remains essentially clinical. But few investigations may be useful when UMN lesion is suspected

MRI Brain with Gadolinium contrast

CT scan / MRI Brain would reveal the cause of the upper motor neuron palsy

Treatment

- Prednisolone:
  * 1 –2 mg/kg/day (60 – 80 mg/day) for 10 –14 days followed by slow tapering over next 2 – 4 weeks is useful.
  * It is highly effective if started within 3 days of onset of illness.
- Protection of eyes during sleep using eye pads
- Splinting of the lower face to prevent the drooping
- Massaging of weak muscles.
- Facial retraining (mime therapy) with biofeedback.
- Transcutaneous nerve stimulation

Prognosis

80 % of the patients recover completely in one or two months. Incomplete weakness at the onset of illness is good prognostic sign. Taste recovery and partial motor recovery at the end of first week indicates good recovery.

Indicators of poor prognosis

- Complete facial palsy
- No recovery in three months
- Age over 60 years
- Severe pain
- Nerve conduction studies
  * Lack of excitability of facial nerve after 10 days and amplitude difference between the abnormal and normal more than 50 %
- EMG showing denervation after 10 days

Complications

- Facial synkinesis
- Crocodile tears

- Hemi-facial spasm

When to admit?

Can be managed as outpatient most of the time.

When to refer?

No recovery within 3 weeks, suspicion of secondary causes.

References

- Patrick J, Sweeney and Maurice R. Hanson, 2004, Cranial Neuropathies, Bradley's Neurology in Clinical Practice, 4th edition, pp 1953 – 1990.
- Allan H.Ropper, Robert H.Brown, Facial nerve palsies, Adam's Principles of Neurology, 8th edition, pp 1181-1183.

Parkinson's disease

Introduction

By the year 2040, neuro-degenerative diseases are expected to become the second most common cause of death in the elderly. One of the most common neuro-degenerative disorders is Parkinson's disease (PD).

Definition

Parkinson's disease was described by James Parkinson in 1817 as a clinical syndrome presenting with bradykinesia, tremor, and slow, shuffling gait with postural instability. Rigidity was described later, but is included as a key clinical feature in the current diagnosis of Parkinson's disease.

Parkinsonian disease is characterized by a tetrad known as TRAP:

- Resting **T**remor
- Cogwheel **R**igidity
- Bradykinesia / **A**kinesia
- **P**ostural reflexes

Epidemiology

As a rule Parkinson's disease (PD) begins between the ages of 40 and 70, with peak age at onset occurring in the sixth decade. Onset at younger than age 20 is known as juvenile Parkinsonism, which has a different pattern of nigral degeneration and is often hereditary (Parkin gene) or caused by Huntington's or Wilson's disease. Parkinson's disease is more common in men, with a male-to-female ratio of 3:2.

Causes

The exact cause of parkinson's disease remains unclear. A combination of factors is probably responsible for the development of parkinson's disease. Various theories include

- Accelerated aging
  - Decline in pigmented neurons in substantia nigra
- Oxidative stress
  - Combined effects of multiple factors culminating in damage from free radicals.
- Genetic susceptibility
  - Increased incidence of a family history of Parkinson's disease is observed in affected individuals (16% vs. 4% of controls)
- Environmental toxins
  - Cyanide
  - Manganese
  - Carbon disulfide
  - Pesticides
  - Well water
  - Methanol and
  - Organic solvents
- Medications
  - Metoclopramide
  - Domperidone
  - Reserpine containing antihypertensive and
  - Neuro-leptics

Symptoms

- Insidious onset, slowly progressive unilateral rest tremors at a frequency of 4-6 Hz in the hands is the mode of onset of PD in more than 85% patients.
- Slowness of motor activities, masked facies, decreased eye blinking, cogwheel rigidity, stooped posture and decreased arm swing while walking are present in the early stages.
- Early symptoms may be weakness and fatigue, nonspecific pains and discomfort. Soft speech, micro-graphia, shuffling gait and postural instability and falls can also occur. These are the motor manifestations.

- Non-motor manifestations such as insomnia, sleep disturbances (nightmares, restless leg syndrome, periodic leg movements, excessive daytime sleepiness), autonomic disturbances such as orthostatic hypotension, constipation, urinary urgency and frequency, excessive sweating and seborrhea are common.
- Neuropsychiatric manifestations can be mood changes, depression, cognitive and behavioral disturbances.

Differential diagnosis:

- Wilson's disease: If the onset is less than 40 years (even 60 years) serum ceruloplasmin, urine copper levels should be done
- Normal pressure hydrocephalus: differentiated by doing a CT scan - brain.
- Drug-induced Parkinsonism: can be identified by detailed history.
- Vascular Parkinsonism: predominantly in lower limbs with history of stroke and CT scan of brain showing infarcts in basal ganglia.

Investigations

Usually the investigations such as CT brain, MRI brain, blood investigations for Wilsons are done to exclude other causes. PD is usually a clinical diagnosis not warranting sophisticated investigation.

Natural history

The disease progresses inspite of treatment and within 10 years the response to treatment will be reduced leading to dykinesias of various types and the patient will become bedridden within 15 years.

Treatment

Ref Table 1 and Flowchart 1

Medical treatment of Parkinson's disease				
Drug	Type	Dose	Common Side effects	Special feature
Selegiline	Mono amino oxidase inhibitor	5mg-15mg	Insomnia, hypotension	Initiating therapy
Tri-hexyphenidyl	Anti-choloinergic	2mg tds	Dry mouth, confusion	Younger patients with tremors
Amantadine	NMDA antagonist	100mg-300mg	Hallucination	Effective for tremors
Levodopa/ Carbidopa (4:1)		10/100mg-25/200mg 4-6 times a day	Hypotension, Hallucination, confusion, dyskinesias after 5 years of disease	Mainstay of treatment
Pergolide	DOPA agonist	0.05mg-0.5mg tds	Hypotension, Hallucination, confusion, pedal oedema	
Bromocriptine	DOPA agonist	2.5mg tds-20 mg tds	Hypotension, Hallucination, confusion, pedal oedema	
Ropinrole	D2/D3 agonist	0.25mg tds -1-3mg/ tds	Somnolence	High dose(>16mg) for monotherapy, avoid driving vehicles
Pramipexole	D2/D3 agonist	0.125mgtds-0.5-0.75mgtds	Somnolence	High dose(>3mg) for monotherapy, avoid driving vehicles
Entacapone and Tolcapone	COMT inhibitors	100mg 100-200mg	Hepatotoxicity, diarrhoea	To be given with Levodopa/ carbidopa

Conclusion

Diagnosis is easy as Parkinson's disease, is usually diagnosed with clinical examination. Secondary causes and other differential diagnosis should be ruled out by investigations. Treatment usually doesn't alter the course of the illness and has only symptomatic value.

References

- Jancovic J, The extra pyramidal disorders, Cecil Text book of Medicine 20th Edition Philadelphia, Pa: WB Saunders Co 1996:2042-2046
- Harrison's Internal Medicine -16th Edition
- Adam's Principles of Neurology-8th Edition.
- Manual of Neurologic Therapeutics-7th Edition, Lippincot Williams.

Guillain-Barre syndrome (GBS)

ICD Code number: G61.0

Definition

- Guillain - Barre syndrome(GBS) is a heterogenous grouping of immune mediated processes generally characterized by motor, sensory and autoimmune dysfunction.
- In the classic form, GBS is an acute inflammatory demyelinating polyneuropathy characterized by
  - * Progressive symmetric ascending muscle weakness

* Flaccid paralysis, and
* Hyporeflexia with or without sensory or autonomic symptoms

- In severe cases muscle weakness can lead to respiratory failure and death.

General Considerations

In 1859, Landry described a neuropathy characterized by ascending paralysis. Subsequently, Guillain-Barre and Strohl noted the areflexia and albumino-cytological dissociation in the cerebrospinalfluid(Cerebrospinal fluid) associated with this neuropathy.

Other names for this condition are:

* Acute postinfective polyradiculoneuropathy,
* Acute infectious polyneuritis
* Landry-Guillain- Barre- Strohl syndrome
* Postinfectious polyneuritis

Epidemiological aspects

- GBS is the most common cause of acute generalized weakness with an annual incidence ranging from 1-4/lakh population.
- Male to female ratio is 1.5:1.
- A Swedish epidemiological study indicated incidence of GBS is lower during pregnancy and increases in the months immediately following delivery.
- GBS occurs at all ages, but a bimodal distribution with peaks in young adulthood (15-35y) and in elderly persons(50-75y) appears to exist
- Epidemiological studies from Japan show an antecedent Campylobacter jejuni (C.jejuni) in a greater percentage of GBS patients.

Causes

Etiology

* A mild respiratory infection or gastrointestinal infection precedes the neuropathic symptoms by 1-3 weeks.
* Campylobacter jejuni is the most frequent identifiable antecedent infection. Other antecedent illness include viral exanthems and viral infections(Cytomegalovirus, Epstein-Barr virus, HIV), bacterial infections (Mycoplasma pneumoniae, Borrelia burgdorferri) and lymphomas.
* Certain vaccines known to cause GBS are outmoded antirabies vaccine, A1 or New Jersy(Swine) influenza vaccine, Menactra Meningococcal conjugate vaccine
* Anecdotal association include
  » SLE
  » Sarcoidosis
  » Surgery
  » Renal transplantation
  » Thrombolytic use and
  » Snake bites.

Pathogenesis

* GBS is believed to be an autoimmune response, both humoral and cell mediated, to a recent infection. Its relation to antecedent infections and identification of various antiganglioside antibodies suggests that “molecular mimicry” may serve as a possible mechanism for the disease.
* The antibodies formed against the ganglioside like epitopes in the lipopolysacharide layer of some infectious agents cross-react with ganglioside surface molecules of peripheral nerves.
* Symptoms generally coincide pathologically with various patterns of lymphocytic infiltration and macrophage mediated demyelination, depending on the subtypes.
* Recovery is typically associated with remyelination. In axonal form of GBS, myelin sparing axonal damage in the form of direct cellular immune attack on the axon itself is seen.

Major Pathological Subtypes	
AIDP	* Acute Inflammatory Demyelinating Polyneuropathy.
AMAN	* Acute Motor Axonal Neuropathy.
AMSAN	* Acute Motor Sensory Axonal Neuropathy.
MFS	* Miller Fisher Syndrome * Acute Panautonomic Neuropathy.
BBE	* Bickerstaff’s Brain-stem Encephalitis

Acute inflammatory demyelinating polyneuropathy

- Most common. Nearly 40% of patients are seropositive for Campylobacter jejuni
- Lymphocytic infiltration and macrophage mediated demyelination of peripheral nerves are present. Symptoms generally resolve with remyelination.

AMAN

It is a purely motor subtype, more prevalent amongst paediatric age groups. Nearly 70-75% of patients are seropositive for Campylobacter. AMAN is generally characterized by rapidly progressive weakness, ensuing respiratory paralysis, more protracted course and delayed recovery.

AMSAN

Acute severe illness similar to AMAN but also affects sensory nerves and roots. Patients are adults with both motor and sensory dysfunction, marked muscle wasting and poor recovery.

MFS

It is a rare variant that presents with classic triad of ataxia, areflexia and ophthalmoplegia. Patients may also have ptosis, facial palsy, mild limb weakness and bulbar palsy. Anti GQ1b antibodies, reduced or absent sensory nerve action potentials (SNAP) and absent tibial H reflex are characteristic of MFS. Recovery occurs generally within 1-3 months.

BBE

Bickerstaff's Brain-stem Encephalitis

- A Variant of GBS
- Acute onset of ophthalmoplegia, ataxia, disturbance of consciousness, hyperreflexia or Babinski's sign.
- Course of the disease is monophasic or remitting to relapsing
- Prognosis is good
- MRI is diagnostic

Acute Pan-autonomic Neuropathy

Rarest of all variants. Cardiovascular involvement is common. Dysrhythmias are a significant source of mortality in this form of disease. Recovery is gradual and often incomplete.

Other variants of GBS

Regional

Cervico-brachio pharyngeal, often with ptosis, oculopharyngeal weakness, bilateral facial and abducens weakness with distal paraesthesias, Ophthalmoplegia with GQ1b antibodies and predominant paraparesis.

Functional

Generalised ataxia without dysarthria or nystagmus, pure sensory, pure motor, Pan-dysautonomia and axonal.

Symptoms

- In GBS, the maximum deficit develops over days(maximum 28 days), followed by a plateau phase and a gradual recovery.
- Patients may initially present with paraesthesias, sensory symptoms and weakness. The fairly symmetrical weakness of the lower limbs ascends proximally over hours to several days to involve arm, facial, oropharyngeal muscles and in severe cases the respiratory muscles. Progression ends by 1-4 weeks.
- Autonomic symptoms such as palpitation, postural giddiness, facial flushing, venous pooling, urinary retention and constipation can occur.

Signs

- Acute flaccid quadriparesis, hypo or areflexia, impaired propioception and pain, cranial nerve palsies in the form of ophalmoplegia, pupillary abnormalities, bi-facial, palatal and pharyngeal weakness, and dysarthria.
- In severe cases respiratory muscle paralysis occurs which is characterized by poor respiratory effort, reduced vital capacity (<10ml/kg) and reduced single breath count < 20.
- Papilloedema, paralytic ileus and urinary retention can occur.

Diagnostic Criteria

Features required for diagnosis

- Progressive weakness of both legs and arms.
- Areflexia.

Clinical features supportive of diagnosis:

- Progression over days to 4 weeks.
- Relative symmetry of signs.
- Mild sensory symptoms or signs.
- Cranial nerve involvement (bifacial palcies).
- Recovery beginning 2 weeks after progression.
- Autonomic dysfunction.
- Absence of fever at onset.

Investigations

- Elevated Cerebrospinal fluid protein with<10 cells/microlitre.
- Electro-diagnostic features of nerve conduction slowing or block.

Note: Features supporting an axonal process are seen in AMAN and AMSAN.

Investigations

Cerebrospinal fluid examination and serial electrophysiological studies are critical for confirming the diagnosis of GBS.

- Elevated or rising protein levels on serial lumbar puncture and 10 or fewer mononuclear cells/mm^3 strongly support diagnosis.
- Most common electrophysiological abnormalities include prolonged distal motor and F wave latencies, absent or impersistent F waves, conduction block, reduction in distal CMAP amplitudes, with or without temporal dispersion and slowing of motor conduction velocities.
- In cases of axonal degeneration, reduced CMAP and SNAP amplitudes are found.
- MRI- Lumbosacral MRI may demonstrate Gadolinium enhancement of lumbar roots.
- Serum- Elevated serum antibodies to Mycoplasma, C.jejuni and cytomegalovirus can pinpoint the preceding infections.
- Antibodies to GM1 ganglioside are seen in AMAN.
- Antibodies to GQ1b are seen in MFS and ophthalmoplegia.
- Antibodies to GD1a and GT1b are diagnostic of pharyngo brachial cervical variant.
- Biochemistry
  - * Abnormal LFT in 1/3rd of patients.
  - * Hyponatremia in ventilated GBS patients.

Differential diagnosis

- Acute neuropathies
  - * Porphyrias
  - * Diphtheria
  - * Critical illness neuropathy
  - * Toxins-Arsenic, lead, organophosphorus compounds, thallium, neurotoxic fish and shell fish (ciguatoxin, tetradotoxin, saxitoxin), tick paralysis, vasculitis and lymes disease.
- Neuromuscular junction disorders
  - * Botulism
  - * Myasthenia gravis
- Myopathies
  - * Hypokalemia
  - * Hypophosphatemia
  - * Polymyositis
  - * Rhabdo-myolysis
- CNS disorders
  - * Rabies
  - * Poliomyelitis
  - * Transverse myelitis
  - * Basilar artery thrombosis.

Treatment

Emergency department care

- Monitor airway, breathing, circulation. IV fluids, oxygen and assisted ventilation form the initial management.
- Clinical indications for the need of intubation include hypoxia, rapidly declining respiratory function, poor or weak cough reflex and suspected aspiration.
- Typically intubation is indicated whenever the FVC is less than 15 ml/kg (usually a single breath count of 20 is equivalent to VC of 1.5 l), and maximum inspiratory pressure is <30 cm of water.
- Intravenous infusion and use of vasopressor agents in patients with hypotension due to dysautonomia.
- Correction of hyponatremia and use of heparin to prevent pulmonary embolism.
- Plasma exchange (PE) and intravenous immu-

noglobulin (Iv Ig). Both therapies have been shown to shorten recovery time by as much as 50%.

Plasma exchange

- Dosage
  - 200 to 250 ml/kg of plasma in 4-6 treatments on alternate days.
  - The replacement fluid is saline combined with 5% albumin.

Indication

If the patient is unable to walk unaided, if he shows significant reduction of vital capacity and signs of oropharyngeal weakness.

Predictors of responsiveness

Younger patients and patients with preserved CMAPs prior to institution of treatment respond better.

Advantage

In patients who are treated within 2 weeks of onset, there is approximate halving in period of hospitalization and in time required for independent ambulation.

Complications

- Need large bore venous access(subclavian or internal jugular catheterization) which may cause pneumothorax, infection and haemorrhage.
- During and after the procedure, hypotension, hypo-prothrombinemia with bleeding, and cardiac arrhythmias can occur.

Intravenous immunoglobulin

- Dosage
  - 0.4g/kg/day for 5 consecutive days.
- Complications
  - Renal failure
  - Proteinuria
  - Aseptic meningitis
  - Anaphylaxis can occur in patients with congenital IgA deficiency.

Note

- Performing plasma exchange after intravenous immunoglobulin does not make sense and hence follow exchanges after intravenous immunoglobulin.

Corticosteroids

- Are ineffective as monotherapy.
- Two randomised controlled trials, one with conventional dose prednisolone and another with high dose Methylprednisolone have failed to demonstrate any beneficial effect.

Prognosis

- Approximately 3-5% of patients do not survive the illness even in best equipped hospitals.
- In early stages death is due to cardiac arrest related to dysautonomia, ARDS, pneumo or haemothorax.
- Later in the illness, pulmonary embolism and respiratory failure are the main causes of death.
- Majority of patients recover completely but in 10% residual disability is pronounced.

Follow Up

- Average period of mechanical ventilation has been 22 days and period of hospitalization is 50 days in patients with respiratory failure.
- Speed of recovery varies and the pace is steady. It often occurs within few weeks to months.
- In patients with axonal degeneration, regeneration may require 6-18 months or even longer.

When to admit?

Since patient condition may deteriorate unpredictably and rapidly in first few days of illness, virtually all but the mildest cases should be admitted to the hospital for observation of autonomic, motor and respiratory functions.

When to refer?

Patients with significant respiratory distress and cardiovascular autonomic instability need ICU care by skilled personnel and hence they should be referred to higher institutions with facilities for mechanical ventilation and ICU care.

References

- Allan H. Ropper, Robert H. Brown, Diseases of the

peripheral nerves, Adams and victor's PRINCIPLES OF NEUROLOGY, 8th edition, pp1117-1126.

- E.Peter Bosch and Benn E. Smith, Disorders of Peripheral Nerves, Walter G. Bradley's NEUROLOGY IN CLINICAL PRACTICE, 4th edition, pp2336-2345.
- Article from E medicine(www.emedicine.com.) on GBS from internet.

Stroke

Epidemiology

Cerebro vascular disease is a major public health problem. The crude prevalence rate varied from 45-843 per 100,000. There are large human and monetary costs involved in caring for patients with stroke. At least one-third of patients survive with significant disability.

Symptoms

A systematic approach is required to evaluate a patient with a cerebrovascular problem. Stroke usually is sudden in onset with rapid progression to maximal deficit. **Transient ischemic attack** is defined as a temporary episode of focal ischemic neurological dysfunction that resolves completely within 24 hours. This has to be differentiated from other stroke mimics such as migraine and seizures. Strokes can be classified as ischemic and haemmorrhagic stroke.

Causes

- Major causes of cerebral ischemia are cardiac disease, large vessel disease, small vessel disease, and hematological disease
- Ischemic stroke can be classified as thrombotic, embolic and lacunar infarctions.
- Infarctions can be arterial or venous
- **Five common locations of haemorrhage are**
  - Epidural and subdural (secondary to trauma),
  - Sub-arachnoid haemorrhage (AVM or aneurysmal rupture)
  - Intra-cerebral and intra-ventricular hemorrhage (secondary to hyper tension, AVM, aneurysm)

Symptoms

History

One should differentiate stroke / TIA from potential mimics

- Symptoms may include motor weakness, sensory disturbances, cranial nerve disturbances, cognitive abnormalities, seizures, unsteadiness, headache, etc depending on site of ischemia.
- History of heart disease, atherosclerotic risk factors such as hypertension, diabetes, hyper-lipidemia, smoking, sedentary lifestyle, family history of atherosclerotic disease should be asked for.
- History of pain in neck, face, head; pain might suggest arterial dissection.
- History of fever, chills, cardiac symptoms, drug abuse might indicate infective endocarditis.

Examination

- Blood pressure, cardiac rhythm, carotid pulses, bruits, heart sounds, murmurs are recorded.
- Neurological examination allows classification into one of the stroke syndromes.

Major stroke syndromes include

- Middle cerebral artery syndrome with contra lateral gaze paresis, hemi paresis, hemi sensory loss, contra lateral visual field loss and aphasia with left sided neglect with right-sided lesions
- MCA branch syndromes
  - Non-fluent aphasia if involving anterior division and fluent aphasia if involving posterior division of left side.
- Anterior cerebral artery syndrome with predominantly leg weakness and sensory deficits contra laterally and sparing of vision.
- Posterior cerebral artery syndrome with contra lateral homonymous hemianopia with associated memory loss.
- Lacunar syndromes suggesting occlusion of small vessels resulting in pure motor, sensory, isolated dysarthria syndromes
- Stroke mimics
  - Acute Migraine attacks
  - Seizures
  - Hypertensive encephalopathy
  - Metabolic causes
  - Syncope

Management of Stroke

Investigation

- Recommended tests in acute stroke are
  - * Serum glucose
  - * Blood count
  - * Serum electrolytes
  - * Renal function tests
  - * Coagulation studies and
  - * ECG

Neuro imaging

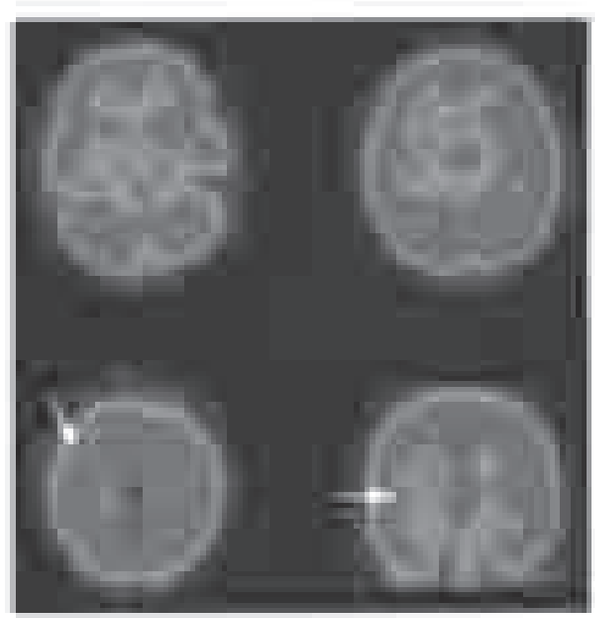
- Emergency non-enhanced CT helps to detect haemorrhage which appears hyper dense
  - * The ability of CT to reveal an ischemic lesion depends on the size, location of lesion, time after onset of symptoms
  - * CT may be normal in small infarctions in posterior circulation, infarcts < 5mm and in acute infarctions
  - * Ischemic stroke is seen as an area of hypodensity that appears 12 to 48 hours after stroke.
- Multimodal MR imaging is
  - * Useful in detecting acute stroke, posterior circulation stroke and lacunar strokes
  - * It is more sensitive to ischemic brain damage than is the CT scan
  - * While the latter reveals hemorrhage immediately after it occurs, softened tissue cannot be seen until several days have elapsed
  - * On the other hand, MRI reveals ischemic damage within a few hours, in both white and gray matter, and diffusion-weighted MRI techniques do so even earlier (with in minutes).
- By MR angiography
  - * One can see all the major cervical and intracranial arteries and may detect the irregular lumen or occlusion of atherosclerosis and even embolic occlusions in more distal vessels
  - * This method has to a large extent has replaced conventional angiography, which is reserved for cases in which the diagnosis is in doubt (e.g., suspected angiitis) or when surgical intervention or long-term anticoagulant therapy is contemplated.

Rx Treatment

Acute therapy of Ischemic stroke

- Time is prime factor and awareness should be created for early medical attention to these patients.
- In general blood pressure should not be lowered unless:
  - * Blood pressure lowering is necessary to fulfill criteria for safe thrombolysis.
  - * Acute myocardial infarction being associated.
  - * Hypertensive crisis with end-organ involvement.
  - * The consensus guidelines from American

CT scan - MCA infarct



SPECT showing Caudate infarct

CT scan - Lacunar infarction

CT scan - Peripherial infarction from a occlusion of MCA

Stroke Association suggest that therapy be withheld unless Diastolic Blood pressure >120 or systolic Blood pressure >220.

- Supplemental oxygen when SpO_2 less than 95%.
- Hyperthermia and hyperglycemia to be corrected.
- When patients present within 3 hours, IV thrombolysis with rtPA improves stroke outcomes.
- Acute use of lowdose Aspirin improves stroke outcome.
- Anticoagulants are useful in cardioembolic stroke, cortical vein thrombosis and arterial dissections.
- Anti-oedema measures are used when signs or symptoms of cerebral edema is present. Mannitol, furosemide, hyperventilation, Head elevation is useful in management of cerebral edema.
- If acute stroke presents within 3-6 hours intra-arterial thrombolysis improves outcome and so patient can be referred to specialty centers for the same

Paraplegia and Quadriplegia

Definition

Paraplegia means weakness of both lower limbs and quadriplegia is weakness of all four limbs.

Causes

Paraplegia or quadriplegia may be caused by lesions of cerebrum, spinal cord, motor roots, peripheral nerves, myoneural junction, or muscles. The causes include:

- Trauma
  - Usually acute onset (seconds or minutes)
  - Cord transection –may be complete or incomplete
  - Disc heriniation/extrusion
  - Shock cord syndrome or spinal concussion, may present with complete cord syndrome, but largely reversible.
  - may be associated with vertebral fracture
- Acute Transverse myelitis
- Anterior spinal artery infarction – atherosclerosis, embolism, hypotension, angiography, vasculitis
- Spinal cord hemorrhage – intramedullary, subarachnoid, subdural or epidural
  - Haematomyelia is usually due to trauma.
  - Other etiologies include

    - » Arteriovenous malformation
    - » Venous infarction
    - » Blood dyscrasias
- Acute immune disorders – includes
    - * GBS
    - * Myasthenia gravis
    - * ADEM
    - * Para-neoplastic myelopathy, and
    - * Acute polymyositis
- Infectious disorders such as acute epidural abcess.
- Hereditary metabolic disorders
    - * Hypokalemic and hyperkalemic periodic paralysis
    - * Acute intermittent porphyria.
- Toxins/miscellaneous
    - * Tick paralysis
    - * Aminoglycosides
    - * Organophosphates
- Cerebral causes of paraplegia such as
    - * Unpaired anterior cerebral artery thrombosis,
    - * Superior sagittal sinus thrombosis
    - * Frontal lobe tumours
- Hysterical paraparesis.

Recurrent paraplegia

e.g. Multiple sclerosis, Vascular malformations of cord.

Chronic para or quadriparesis

- Compressive myelopathy
    - * Disc herniation
    - * Chronic degenerative arthritis and spinal canal stenosis
    - * Tumour
    - * Abscess
- Inflammatory or demyelinating lesions
    - * Multiple sclerosis
    - * CIDP
    - * Para-neoplastic myelopathy
    - * Spinal arachnoiditis
    - * Neuro-sarcoidosis
- Infections
    - * HIV vacuolar myelopathy
    - * Lyme disease
    - * Tuberculosis
- Metabolic or Toxic
    - * Vit B12 deficiency causing subacute combined degeneration
    - * Post radiation therapy myelopathy
    - * Lathyrism
- Hereditary and congenital conditions
    - * Cerebral palsy
    - * Hereditary spastic paraplegia
    - * Arnold-Chiarri malformation with or without syringomyelia
- Central cause of paraplegia such as
    - * Para-sagittal meningioma
    - * Chronic hydrocephalus.

Spinal cord syndromes

Brown-sequard syndrome (cord hemisection)

Anterior cord syndrome

Central cord syndrome

Posterior cord syndrome

Conus medullaris syndrome

Cauda equina syndrome

Spinal`cord lesion has the following triad of clinical features:

- Sensory level (the hallmark of spinal cord disease)
- Spastic weakness
- Bladder and bowel dysfunction

Symptoms

The uppermost level of spinal cord lesion can be localized by **'segmental signs'** like a band of hyperalgesia at the upper end of the sensory level, fasciculation or atrophy of muscles of one or more segments and diminished or absent DTR. They indicate a spinal cord lesion particularly when accompanied by long tract signs.

The state of **'spinal shock'** with flaccid areflexic limbs, resulting from severe and acute transverse

lesions, may last for days or rarely weeks and should not be mistaken for a LMN lesion like polyneuropathy.

Slow spinal cord compression affects the pyramidal tract first, the posterior column next and the spinothalamic tract last.

In occlusion of the anterior spinal artery, the infarction is associated with spinothalamic tract and pyramidal tract involvement with sparing of posterior column.

Major concerns regarding mortality/morbidity due to spinal cord lesions depend on localization.

- Respiratory depression may occur with cranio-cervical and cervical cord lesions.
- Autonomic instability may occur with cord lesions at the mid thoracic level or above.
- Urinary retention.

When the onset of paraplegia is acute, vascular, traumatic, inflammatory and demyelinating causes should be thought of. It represents a **'Neurological emergency'**. Non-spinal causes should also be included in the differential diagnosis.

Spinal epidural abscess is often missed and misdiagnosed, sometimes with disastrous results . Low grade fever and low back pain followed by radicular pain, presenting with rapidly progressive paraplegia or quadriplegia , is characteristic.

Investigations

- X-rays
  - * spine-AP and lateral views
  - * Chest X-ray-PA
- TLC, DLC, ESR, Hb%
- CPK
- ECG
- Cerebrospinal fluid Analysis
- Rheumatology work up
- CT Scan-may not be very informative.
- MRI spine with contrast focusing on the clinical level of lesion.
- MRI of brain is ordered if hemispheric or cortical signs like seizures and confusion are present, in the absence of sensory level on trunk.
- Nerve conduction studies
  - * EMG
  - * RNS. if lower motor neurone involvement is suspected. (RNS system is a deep brain stimulation treatment)

Treatment

- Many disorders of the spinal cord are treated if recognized early as otherwise the results are devastating.
- If a spinal cord disease is associated with absence of demonstrable compression by imaging techniques, it is designated as non-compressive myelopathy.
- In case of compressive lesions of the spinal cord, cauda equina or nerve roots, surgical therapy is usually required.
- Replacement of potassium for hypokalemic periodic paralysis; Inj. Neostigmine for myasthenia gravis; ventilatory support for GBS;
- Hysterical paralysis associated with retained DTR, lack of atrophy and give way weakness should be differentiated from organic lesions causing paraplegia.
- In all cases of spinal injury, associated head injury and other injuries such as abdominal injury should be excluded and movement (especially flexion) of the cervical spine should be avoided. The patient should be placed on a firm, flat surface.
- Once the degree of injury to spine and cord has been assessed, high dose Methyl prednisolone injection is given – bolus of 30mg/kg followed by 5.4 mg/kg/ hour, for 23 hours, usually beginning within 8 hours of injury. Though acute decompressive surgery is done for haematomas and other sources of compression amenable to surgery, most surgeons do not favour surgery with complete spinal cord lesions.
- The spinal epidural abcess, if not surgically treated by laminectomy and drainage at the earliest possible time, before the onset of paralysis, the spinal cord lesion which is party due to ischemia becomes more or less irreversible. Broad spectrum antibiotics in large doses should be given initially.
- In tuberculosis, often there is epidural compression from caseous granulation tissue getting extruded from an infected vertebra and it requires ATT.
- Post infective and post vaccinal acute transverse myelitis is best treated with high dose corticos-

teroids, plasma exchange, or IV immunoglobulin.

- Bladder care and avoidance of pressure sores are important.
- Physiotherapy, assisted devices, orthotics and wheel chairs may all be beneficial in improving the functional abilities of patients with paraplegia.

References :

1. Neurological differential diagnosis – A prioritized approach – Roongroj Bhidayasiri.
2. Principles of neurology – Adams and victor.

Acute Bacterial Meningitis

Most common organisms responsible for bacterial meningitis are

- Haemophilus influenzae
- Neisseria meningitidis and
- Streptococcus pneumoniae, which account for about 75 percent of sporadic cases.
- Infection with L. monocytogenes is now the fourth most common type of nontraumatic or nonsurgical bacterial meningitis in adults.
- The following are less frequent causes
  - Staph. aureus and group A and group D streptococci, usually in association with brain abscess, epidural abscess, head trauma, neurosurgical procedures, or cranial thrombophlebitis
  - E. coli and group B streptococci in newborns; and the other Enterobacteriaceae such as Klebsiella, Proteus, and Pseudomonas, which are usually a consequence of lumbar puncture, spinal anesthesia, or shunting procedures to relieve hydrocephalus
  - Rarer meningeal pathogens include Salmonella, Shigella, Clostridium, Neisseria gonorrhoeae, and Acinetobacter calcoaceticus, which may be difficult to distinguish from Haemophilus and Neisseria.

Epidemiology

Pneumococcal, influenzal (H. influenzae), and meningococcal forms of meningitis have a worldwide distribution, occurring mainly during the fall, winter, and spring and predominating in males. Each has a relatively constant yearly incidence, although epidemics of meningococcal meningitis seem to occur roughly in 10-year cycles. Meningococcal meningitis occurs most often in children and adolescents but is also encountered throughout much of adult life, with a sharp decline in incidence after the age of 50. Pneumococcal meningitis predominates in the very young and in older adults.

Causes

- Meningococcal Meningitis occurs as a cyclical outbreak (New Delhi and Northern parts of India) or sporadically.
- Remember environmental causes for meningitis and look for tickborne, zoonosis or ornithosis; ask for exotic travel or travel to endemic areas; exposure to airborne organism; and elicit behaviour for contact with carriers.
- Whenever cases of meningitis occur, search for infection in the adjacent areas.
- Bacterial meningitis among migrant population and travellers are not uncommon.

Clinical aspects

Symptoms

- Meningitis is a medical emergency
- One should not expect all clinical symptoms and signs in every case of meningitis, as these are influenced by the age, biological status, previous exposure and immune response of the individual on one side, and the nature, number and virulence of the organism on the other side.
- The classical clinical features and laboratory findings may not be demonstrable in many cases now, as the cases are diagnosed on the basis of probability and treatment is started very early before adequate time is given for immunological response.
- If immuno-compromised individuals develop meningitis, they may not show classical findings of meningitis.
- Individuals with deficiency in complement components appear to be susceptible to meningococcal infection.
- Patients with acute bacterial meningitis seek medical attention usually within one to two days after the onset of symptoms.
- Aseptic meningitis has a benign course and is self limited. It is mostly caused by

* Viruses: Entero-viruses
* Infectious mononucleosis
* Leptospirosis
* Disseminated Lyme's disease
* Secondary syphilis, etc.,

- Watch for other system involvement during the course of the meningitis or while presentation in every case of meningitis.
- **In every suspected tuberculosis meningitis, look for BCG scar,** ask for previous history of pulmonary tuberculosis or elicit details on treatment.
- Always look for neurological and non-neurological complications, as these cannot be predicted in a given case.
- Death in meningitis is mostly related to raised Intra cranial pressure (ICP).
- Lumbar Puncture is essential when there are indications.
- The frequency of Cerebrospinal fluid examination depends on the clinical outcome but a repeated examination should be done in 24 to 48 hours, if there has not been satisfactory improvement or if the causative micro organism is more resistant.
- Routine "End of treatment" Cerebrospinal fluid examination is unnecessary.
- Meningitis patients may develop convulsions at any point of time. Hence, **medical personal while transferring a case to higher centre must take care to manage convulsions while on transfer.**
- Meningitis patients who have respiratory complications, multi-organ failure or involvement and deteriorating neurological signs require intensive care management, if available.
- In view of the immediate and late complications due to meningitis, care should be taken to treat the patients without any delay.

Investigations

- Cerebrospinal fluid (Cerebrospinal fluid) should be sent immediately to the laboratory for cytological, bio-chemical, immunological and microbiological analyzes.
- Normal Cerebrospinal fluid in a suspected case does not rule out meningitis.
- Antibiotics given 4 hours before obtaining Cerebrospinal fluid probably do not affect culture results.
- In bacterial meningitis, positive rate for gram staining vary from 60 to 90%, and culture in about 90%. Hence efforts must be made for bacteriological studies, if suspected.
- Cerebrospinal fluid sugar may be normal in Listeria mono-cytogenes infection.
- Cerebrospinal fluid sugar will be around 60% of the blood sugar of an individual normally.
- Cerebrospinal fluid protein may be elevated normally in diabetic patients
- In uncontrolled diabetics with central nervous system infection, the Cerebrospinal fluid sugar is less desirable
- Along with Cerebrospinal fluid glucose, estimate simultaneous blood glucose level.
- Depending upon the clinical scenario, proceed with multiple investigations to identify the cause for meningitis.
- Encourage blood culture in suspected cases of meningitis.
- Meningitis due to opportunistic pathogens and other newer organisms are attracting clinicians, micro biologists and community medicine specialists. Also, they need special media for isolation and identification.
- Various Salmonella serotypes can contribute to epidemic meningitis.
- As meningitis due to more than one organism was reported in one and the same individual rarely, one should not ignore the isolates as contaminants.

Treatment

- Meningitis patients require empirical antimicrobial therapy in high doses and it should be started immediately and be adjusted after the microbiological results arrive.
- Steroids are administered amidst controversies in view of the potential benefits and for short duration.
- Type of anti-microbial required, dose and duration are decided by invading organism.
- In Cryptococcal meningitis Cerebrospinal fluid is

often at high pressure and management may require regular removal of 10 to 15 ml of Cerebrospinal fluid to prevent features of raised ICP.

Prevention

- Vaccination against meningitis is available for few infections only.
- Observe for infection and assess carrier status among other members living or in contact with the case of Meningococcal meningitis.
- **Family members have to be given chemoprophylaxis, if the case turned out to be meningococcal infection.**
- Healthcare workers may be a carrier for the occurrence of meningococcal Meningitis.

Important note

- Risks involved and anticipated complications have to be explained to the care givers of the patients, as case fatality cannot be predicted.
- Timely administration of medications, vigilant observation for complications, good nursing care and adequate supportive measures enhance recovery with minimal complications.
- **Meningitis due to notifiable agents has to be reported to the public health authority immediately.**
- Include microbiologists and public health personnel in the team when you are confronted with meningitis.
- In every case of meningitis look for a cause and a source.
- Ocular fundus examination should be done before lumbar puncture and should be entered in the medical records.
- Performance of CT scan brain before lumbar puncture is controversial but is not generally required if meningitis is considered in non-elderly or immuno-competent individuals.
- Every case of meningitis invariably has predisposing / precipitating factors.
- Be empathetic with patients and care givers but never give assurance.
- Lumbar puncture has to be carried out after obtaining a written informed consent.
- Suspected meningitis case has to be referred to a higher centre after assessment and providing supportive care along with a detailed written report.
- As the implications of meningitis on the individual are many- family, social, professional, national and legal, the documentation of cases should be clear, specific and well written.

Research aspects

Meningitis is a fascinating area for research from the point of view of epidemiology, clinical, therapeutic, preventive, social, and economical aspects.

Tuberculous Meningitis

Tuberculosis presenting as meningitis is the most common form of CNS tuberculosis and constitutes a medical emergency, as a diagnostic and therapeutic delay may have very serious consequences.

Nearly 50% of patients with advanced CNS disease fail to survive and there is a high incidence of serious neurological complications in those who survive.

Causes

Most cases of Tuberculous meningitis are in young children, but primary infection can be acquired at a later age and, in recent years, a large proportion of patients with this condition have been adults. The disease is uncommon, but severe, in pregnant women. There has been an increase in the incidence of tuberculosis related to the epidemic of HIV infection. HIV-infected patients with tuberculosis are at increased risk of meningeal involvement.

The disease commences with the rupture of a meningeal or subcortical lesion with liberation of tubercle bacilli into the Cerebrospinal fluid and the development of many tubercles on the meninges.

The ensuing meningeal inflammation, particularly at the base of the brain, leads to the secretion of thick exudates which may lead to strangulation of the cranial nerves, especially the optic and auditory nerves at the base of the brain and to raised intracranial pressure due to obstruction to the flow of Cerebrospinal fluid.

Raised intracranial pressure is a major complication of tuberculous meningitis and some degree of hydrocephalus occurs in late stages.

Symptoms

Clinically, cases are classified into three stages:

Stage 1

- The patient is fully conscious and rationale with non-specific symptoms such as general malaise, low-grade fever apathy, irritability, personality changes, depression and intermittent headache but with no focal signs and little or no evidence of meningitis
- Symptoms may be limited or even absent in immuno suppressed patients, including those who are HIV-positive.

Stage 2

- The patient is mentally confused and/or has focal neurological signs such as cranial nerve palsies.
- Other symptoms include more severe and persistent headache and vomiting and some degree of photophobia.

Stage 3

- The patient is deeply stuporous or comatosed and or has complete hemiplegia, paraplegia or quadriplegia.

Investigations

- Examination of Cerebrospinal fluid is essential although this should be performed carefully after ruling out raised intracranial pressure
  * This may reveal
  * Lymphocytes
  * A raised protein level and
  * Decreased glucose level, but these parameters may he normal
  * In some cases there is a high polymorph count, which suggests tuberculous meningitis of rapid onset or a non-acid-fast bacterial infection.
- A chest X-ray may be helpful as pulmonary tuberculous lesions are evident in about half the patients
- The diagnosis is confirmed by the microscopic detection of acid-fast bacilli in centrifuged samples of Cerebrospinal fluid, although a very thorough search only detects such bacilli in 10-30% of case. Where a Cerebrospinal fluid 'c1ot' of fibrin is present, acid-fast bacilli may be seen in it
- Culture of Cerebrospinal fluid is far too slow, even with the use of automated systems but, where facilities are available, PCR and related nucleic acid amplification techniques may be useful
- **If in any doubt as to the diagnosis, antituberculosis therapy should be commenced immediately.**
- CT and MRI, where available, are of value in the investigation of tuberculous meningitis as they detect
  * Cryptic lesions
  * Raised intracranial pressure
  * Hydrocephalus and
  * Cerebral infarctions
- Clinically, the differential diagnosis should include
  * Various subacute or chronic meningitis, including partially treated bacterial meningitis, fungal meningitis para-meningeal infections,
  * Neoplastic and granulomatous infiltrations of the meninges (for example carcinomas, leukaemias. lymphomas, sarcoidosis), and
  * Cerebral tumours

Treatment

- The untreated mortality of Tuberculous meningitis is close to 100 percent
- Full treatment must be started when the diagnosis is suspected on clinical grounds, immediately after adequate samples have been taken for microscopy, culture, and immunodiagnosis.
- 'Trial' of chemotherapy is justified when there is clinical suspicion of Tuberculous meningitis, particularly when diagnostic facilities are limited
- There is conflicting evidence regarding the length of treatment, and treatment for 12 months is probably a conservative estimate of the time required for bacterial cure
- The combination of Isoniazid and Rifampicin for 12 months, with Pyrazinamide and Streptomycin during the first 2 months with initial adjuvant steroids, is an effective regimen that has been widely used
- Steroid should be used with IV Dexamethasone for 1st 7days followed by prednisolone 1mg/kg for 1 month and then to be tapered over 3-4 weeks. Duration of steroid therapy may need to be extended depending on clinical picture.

Complications of Tuberculous Meningitis

The complications of Tuberculous meningitis are common, some of which are often serious enough to cause severe morbidity and death in spite of active treatment with anti-tuberculosis drugs

Nursing care is very important during the acute illness, when there are the usual problems presented by unconscious patients, and during the prolonged phase of convalescence and rehabilitation

Anticonvulsants are often needed, especially in children.

Prognosis

- The prognosis is worst and the risk of sequelae highest in those admitted in coma with signs of brain-stem damage
- It is severe in the very young and very old, pregnant women, and those with malnutrition or other diseases
- The outcome of Tuberculous meningitis in HIV-infected patients is similar to that in patients without HIV infection. There are permanent sequelae in 10 to 30 percent of survivors
- Intellectual impairment is especially common in infants and young children

Long-term sequelae

- As many as 60 percent of patients who have seizures during the illness will suffer recurrences.
- Upto 25 percent of survivors will have cranial nerve deficits including blindness, deafness, and squints
- Some 10 to 25 per cent of survivors have some residual weakness after hemiparesis or paraparesis
- About 10 per cent of patients develop Cerebrospinal fluid spinal block at some stage of the illness, but this will recover completely in at least half of them.

Important note

A high index of suspicion which helps in early treatment of Tuberculous meningitis is the cornerstone in preventing the morbidity

BCG Vaccination at birth reduces the risk of infection and is recommended for all infants born in our community.

References

- Oxford Text book of Medicine
- Mansons Tropical diseases
- Bradley.S Text book of Neurology
- Park Text book of Preventive medicine

Herpes Simplex Encephalitis

Epidemiology

- It is the commonest and gravest form of acute encephalitis, accounts for 10% of all encephalitis.
- In untreated cases mortality is 70%
- In treated cases mortality is 20%
- Morbidity in treated and untreated cases is 70%. Survivors may show permanent neurological sequelae.

Causes

Caused by Herpesviruses (HSV–I in 90% of cases). Also causes herpitic lesion of oral mucosa. Type 2 virus causes acute generalised encephalitis in neonates and genital herpitic infection in the mother. Both type I and II cause myelitis. Rarely localized adult type of encephalitis is caused by type 2 virus and diffuse neonatal encephalitis by type I.

Mode of transmission

- Transmitted by respiratory or salivary secretion. Upto 33% of HSV-I encephalitis occurs with primary infection.
- Virus spreads from the olfactory fibres in the nose to orbitofrontal cortex and temporal lobes.
- Mainly due to reactivation and centripetal spread of virus latent in the trigeminal ganglia from a prior infection.

Symptoms

Fever, headache are consistent features.

- Onset may be abrupt with focal or generalized seizures, confusion, stupor and coma (or) protracted with olfactory or gustatory hallucinations, anosmia, temporal seizures, personality change, bizarre or psychotic behavior or delirium, aphasia and hemiparesis.
- Status epilepticus is rare.
- Memory disturbances is evident later in the con-

valescent stage, as the patients goes from stupor to coma.

- Swelling and herniation of one or both temporal lobes through the tentorium may occur leading to deep coma and respiratory arrest during the first few days of illness.

Differential diagnosis

Herpes Simplex Encephalitis must be differentiated from:

- Acute haemorrhagic leucoencephalitis
- Subdural empyema
- Cerebral abscess
- Cerebral venous thrombosis
- Septic emboli from bacterial endocarditis and
- Mitochondrial encephalopathy

Investigations

Cerebrospinal fluid analysis

- Cerebrospinal fluid is under pressure
- Lymphocytic pleocytosis in the range of 10 – 1000 WBC/µl.
- Neutrophils in early stages
- RBC and xanthocromia are found
- Protein content is raised
- Glucose is usually normal
- **HSV DNA** can be detected in Cerebrospinal fluid by PCR technique. Has sensitivity of > 95% and specificity approaching 100% for the diagnosis of HSV. False negative test can occur in the first 48 hours of febrile illness.
- **Fluorescent antibody study** and viral culture of cerebral tissue obtained by brain biopsy in a few atypical cases. Rising titres of neutralizing antibodies can be demonstrated but is not diagnostic.

CT and MRI

- MRI is more sensitive and is preferred. 40% of patients with HSE and normal CT will show demonstrable abnormalities in areas of high signal intensity on T2 weighted images in fronto temporal regions.

EEG

- This is abnormal in the early course of the disease. Shows diffuse slowing, focal abnormalities in temporal regions or Periodic Lateralizing Epileptiform Discharges **(PLEDS).**

Treatment

- Acyclovir is administered intravenously in the dose of 10mg/kg every 8hours in adults and 20mg/kg every 8 hours in neonates and children.
- Treatment is continued for 14 days.
- Side effects with therapy are local irritation of veins and transient impairment of renal function
- Treatment with acyclovir reduces mortality from 70% of 20%.
- Associated brain edema is treated
- Seizures are treated with anticonvulsants
- Foscarnet is an alternate therapy for Acyclovir resistant strains or for those allergic to acyclovir.

Prognosis

- Both mortality and morbidity is governed by the patient's age and state of consciousness at the time of institution of acyclovir therapy. If the patient is unconscious the outcome is poor.
- If treatment is began with in 4 days of illness in a conscious patient, the survival rate is > 90%.

Sequelae

- Includes
  - Korasakoff's amnesia
  - Global dementia
  - Seizures and
  - Aphasia

Important Note

Primary prevention

The large reservoir of persons with asymptomatic HSV–I and HSV–II infection indicates that the sum of efforts to control HSV disease through suppressive antiviral chemotherapy and educational programme will be limited. Control of HSV infection is attained by vaccination. Several vaccines are under investigation.

Japanese Encephalitis

Japanese encephalitis (JE) is caused by Flavivirus, an Arbovirus (arthropod-borne virus). They are transmitted by culex mosquitoes.

Epidemiological aspects

- Japanese encephalitis is numerically one of the most important causes of viral encephalitis worldwide, with an estimated 50,000 cases and 15,000 deaths annually. It is a zoonotic disease, i.e. infecting mainly animals and incidentally man.
- Majority of cases (about 85%) occur among children less than 15 years of age and 10% of cases occur among those over 60 years of age.
- Incidence in India: JE was first recognized in India in 1955 in Tamilnadu. Half of the population in South India has neutralizing antibodies to the virus. JE virus infects several extrahuman hosts such as animals and birds.
- Available evidence indicates that the basic cycles of transmission are:

  Pig → Mosquito → Pig

  The Ardeid bird → Mosquito → Ardeid bird

Causes

- The disease is transmitted to man by the bite of infected mosquitoes. Man is an incidental dead-end host. Man to man transmission has not been recorded so far.
- Infected pigs do not manifest any symptoms of illness and are thus considered as 'amplifiers' of the virus and infect the mosquitoes.
- Culicine mosquitoes act as vectors in the transmission of the viruses. Culex tritaeniorhynchus is the most important vector in South India.
- All those bitten by infected mosquitoes do not manifest the disease. The ratio of overt infection to inapparent infection varies from 1:300 to 1:1000. Thus cases of encephalitis represent only the tip of the iceberg compared to the large number of inapparent infections.

Virology

In common with all flaviviruses, Japanese encephalitis virus has a small (50 nm) lipoprotein envelope surrounding a nucleocapsid comprising of core protein and 11 kb single stranded RNA (3800 kD). At least five genotypes of Japanese encephalitis virus occur in Asia, which relate roughly to the geographical area of isolation.

Symptoms

Incubation Period

- 5 to 15 days. Course of the disease in man may be divided into three stages:
  - **Prodromal stage**
    - » onset is acute with fever, headache and malaise
    - » Duration is 1 to 6 days.
  - **Acute encephalitic stage**
    - » Fever >38°C
    - » Nuchal rigidity
    - » Focal CNS signs
    - » Convulsions and
    - » Altered sensorium progressing to coma.
  - **Late stage and sequalae.**
  - Patients with Japanese encephalitis typically present after a few days of non-specific febrile illness, which may include coryza, diarrhoea, and rigors. This is followed by headache, vomiting, and a reduced level of consciousness, often heralded by a convulsion.
  - In some patients, particularly older children and adults, abnormal behaviour may be the only presenting feature, resulting in an initial diagnosis of mental illness.
  - A proportion of patients make a rapid spontaneous recovery (so called **abortive encephalitis**). Others may present with aseptic meningitis and have no encephalopathic features.
  - Convulsions occur often in Japanese encephalitis, and have been reported in upto 85% of children and 10% of adults. Generalized tonic-clonic seizures occur more often than focal motor seizures. Multiple or prolonged seizures and status epilepticus are associated with a poor outcome.
  - The classical description of Japanese encephalitis includes
    - » A dull flat mask-like facies with wide unblinking eyes

» Tremor

» Generalised hypertonia, and cogwheel rigidity

» These features were reported in 20% to 40% of Indian children

» Ophisthotonus and rigidity spasms, particularly on stimulation, occur in about 15% of patients and are associated with a poor prognosis.

* Changes of respiratory pattern, flexor and extensor posturing, and abnormalities of the pupillary and occulocephalic reflexes are poor prognostic signs and may reflect encephalitis in the brain stem

* However in some patients a clear rostrocaudal progression of brainstem signs, an association with high Cerebrospinal fluid opening pressures, and a reversal of signs on aggressive management of raised intracranial pressure suggests transtentorial herniation.

* Recently a subgroup of patients infected with Japanese encephalitis virus who presented with a poliomyelitis-like acute flaccid paralysis has been identified.

Prognosis

- Case fatality rate varies between 20 to 40 percent. Around half of the survivors have severe neurological sequelae. About 30% of survivors have frank motor deficits. These include a mixture of upper and lower motor neuron weakness, and cerebellar and extrapyramidal signs.
- Fixed flexion deformities of the arms, and hyperextension of the legs with "equine feet" are common. Twenty percent of patients have severe cognitive and language impairment (most with motor impairment also), and 20% have further convulsions. A higher rate of sequelae is reported for children than adults.

Differential Diagnosis

The differential diagnosis of Japanese encephalitis is broad and includes other viral encephalitides (arboviruses, herpes viruses, enteroviruses, and postinfectious and postvaccination encephalomyelitis), other CNS infections (bacterial and fungal meningitis, tuberculosis, cerebral malaria, leptospirosis, tetanus, abscesses), other infectious diseases with CNS manifestations (typhoid encephalopathy, febrile convulsions), and non-infectious diseases (tumours, cerebrovascular accidents, Reye's syndrome, toxic and alcoholic encephalopathies, and epilepsy).

A peripheral neutrophil leukocytosis is seen in most patients, and hyponatraemia may occur as a consequence of inappropriate antidiuretic hormone secretion (SIADH).

Cerebrospinal fluid Analysis

The Cerebrospinal fluid opening pressure is increased in about 50% of patients. High pressures (>250 mm) are associated with a poor outcome. Typically there is a moderate Cerebrospinal fluid pleocytosis of 10-100 cells/ mm^3, with predominant lymphocytes, mildly increased protein (50-200 mg %), and a normal glucose ratio.

Imaging

- In about 50% of patients CT shows bilateral non-enhancing low density areas in one or more of the thalamus, basal ganglia, midbrain, pons, and medulla.
- MRI is more sensitive, typically demonstrating more extensive lesions (typically high signal intensity on T2 weighted images) of the thalamus, cerebral hemispheres, and cerebellum.
- Imaging studies may be useful in distinguishing Japanese encephalitis from herpes simplex encephalitis, where the changes are characteristically frontotemporal.

EEG

- Various electro-encephalographic abnormalities have been reported in Japanese encephalitis including theta and delta coma, burst suppression, epileptiform activity, and occasionally alpha coma.
- Diffuse slowing may be useful in distinguishing Japanese encephalitis from herpes simplex virus, in which changes are characteristically frontotemporal.

Rx Treatment

- Treatment for Japanese encephalitis is supportive, and involves controlling convulsions and raised intracranial pressure when they occur.
- Careful nursing care and physiotherapy are needed to reduce the risk of pressure sores, malnutrition, and contractures.

- Aspiration pneumonia is a common occurrence in patients with a reduced gag reflex
- There is currently no specific treatment for Japanese encephalitis.

Prevention and Control

- Broadly speaking, measures to control Japanese encephalitis include those which interfere with the enzootic cycle of the virus, and those which prevent disease in humans.
- Measures to control breeding of Culex mosquitoes, such as the application of larvicides to rice fields, and insecticide spraying have proved ineffectual.
- Avoiding culex bites by minimising outdoor exposure at dusk and dawn, wearing clothing that leaves a minimum of exposed skin, using insect repellents containing at least 30% DEET (N,N-diethyl-3 methlybenzamide) and sleeping under bed nets are some solutions.

Vaccination

- Vaccination of population at risk is recommended.
- A killed mouse brain vaccine is available.
- For primary immunization, 2 doses of 1ml each (0.5ml for children less than 3 years) should be given subcutaneously at an interval of 7-14 days.
- A booster is given before 1 year for full protection.
- Revaccination is given after 3 years.
- The vaccine is best used in the inter-epidemic period.

Neuro-Cysticercosis

ICD Code :B69.0 G99.8

Definition

Neuro-cysticercosis is a parasitic infection of the central nervous system. Larval forms of the pork tapeworm Taenia solium causes this disease.

Epidemiology

Neuro-cysticercosis is highly endemic in Latin America, Mexico, Eastern Europe, Asia, Africa, and Spain.

Race

Neuro-cysticercosis tends to be diagnosed more frequently in Hispanics.

Sex

No sexual predilection exists.

Age

Reports of cysticercosis are unlikely in children less than 2 years of age. The disease is recognized in children older than 7 years.

Etiology

- The pork tapeworm T. solium can cause two distinct forms of infection in human: adult tapeworms in the intestine or larval forms in the tissues (cysticercosis).
- Humans are the only definitive hosts for T. solium; pigs are the usual intermediate hosts, although other animals may harbor the larval forms.
- By ingesting undercooked pork containing cysticerci, humans acquire infections that lead to intestinal tapeworms.
- Infections that cause human cysticercosis follow the ingestion of T. solium eggs, usually from close contact with a tapeworm carrier.
- Autoinfection may occur if an individual with an egg - producing tapeworm ingests eggs derived from his or her own feces.

Clinical manifestations

- In cysticercosis, the clinical manifestations are variable. Cysticerci can be found anywhere in the body but are most commonly detected in the brain, cerebrospinal fluid (Cerebrospinal fluid), skeletal muscle, subcutaneous tissue, or eye.
- The clinical presentaion of cysticercosis depends on the number and location of cysticerci as well as the extent of associated inflammatory responses or scarring.
- Neurologic manifestations are the most common. Seizures are associated wtih inflammation surronding cysticerci and accompanying inflammation or by Cerebrospinal fluid outflow obstruction from arachnoiditis.
- Signs of increased intracranial pressure, ataxia, or confusion, are often evident. patients with hydrocephalus may develop papilloedema or dis-

play altered mental status.

- When cysticerci develop at the base of the brain or in the subarachnoid space, they may cause chronic meningitis or arachnoiditis, communicating hydrocephalus, or stroke.

Diagnostic criteria for human cysticercosis

Absolute criteria

- Demonstraion of cysticerci by histologic or microscopic examination of biopsy material
- Visualization of the parasite in the eye by funduscopy
- Neuro-radiologic demonstration of cystic lesions containinig a characteristic scolex

Major criteria

- Neuro-radiologic lesions suggestive of neurocysticercosis
- Demonstration of antibodies to cysticerci in serum by enzyme-linked immuno-electrotransfer blot
- Resolution of intracranial cystic lesions spontaneously or after therapy with Albendazole or Praziquantel alone.

Minor criteria

- Lesions compatible with neuro-cysticercosis detected by neuro-imaging studies
- Clinical manifestations suggestive of neurocysticercosis
- Demonstration of antibodies to cysticerci or cycticercal antigen in cerebrospinal fluid by ELISA
- Evidence of Cysticercosis outside the central nervous system (e.g., cigar-shaped soft tissue calcifications)

Epidemiologic criteria

- Residence in a cysticercosis-endemic area
- Frequent travel to a cysticercosis-endemic area
- Household contact with an individual infected with Taenia solium

Diagnosis

Diagnosis is confirmed by either one absolute criterion or a combination to two major criteria, one minor criterion, and one epidemiologic criterion.

Differential diagnosis

- Amoebic Meningoencephalitis
- Cytomegalovirus Infection
- Lyme Disease
- Schistosomiasis
- Toxocariasis
- Toxoplasmosis
- Trichinosis
- Tuberculosis
- Sarcoidosis
- Hydatid disease
- Tuberous sclerosis
- Von Hippel-Lindau disease
- Astrocytoma, Craniopharyngioma, and Medulloblastoma may be suspected

Investigations

Radiological Examination

* Computed Tomography (CT) Scan shows the cyst and granuloma stages of neurocysticercosis.
* Lesions may be located in the cortex or at the gray-white junction and present as punctate hyperdense lesions with ring enhancement.
* CT scanning also detects edema around the cyst, associated with the death of the organism.
* Magnetic resonance imaging (MRI) is the best imaging test overall for the diagnosis. Use of contrast shows larval death, visible as enhancement of the cyst wall, which indicates that the cyst has changed into a granuloma. In addition, MRI shows vasogenic edema around the cyst.
* Soft tissue radiography can be performed to look for extraneural cysts.

Electro-encephalography (EEG)

* May be essential in children with intractable seizures. Periodic lateralized epileptiform discharges (PLEDs) may be present.

Laboratory Tests

* Stool Examination for ova and parasites. Obtain 3 consecutive daily stool specimens. The presence of ova may be the sole diagnostic confirmation in children.

Cerebrospinal fluid Examination

* Cerebrospinal fluid shows lymphocytic pleo-

cytosis (occasionally eosinophilic), decreased glucose and increased protein.

Serology

* Enzyme-linked immunotransfer blot (EITB) assay of the patient's serum for cysticercal antibodies can confirm the diagnosis. Enzyme-linked immunosorbent assay (ELISA) can be used on serum and Cerebrospinal fluid.

Brain biopsy

* Brain biopsy can be performed in cases where the diagnosis remains questionable and the lesion has not resolved.

Rx Treatment

- Consult a neurologist for management of seizures, increased intracranial pressure, and any other neurologic sequelae of this disease.
- Consult an infectious disease specialist for help with a questionable diagnosis, eradication of the organism, and public health issues
- Consult an ophthalmologist to examine the child for any signs of sub-retinal cysts.

Anti-convulsants to stop seizures

Carbamazepine

- Adult dose
  * Initial dose: 200 mg PO bid; increase dose at weekly intervals (by 200 mg/day) until optimal response is achieved
  * Usual dose: 800-1200 mg/day PO divided tds/qid.
- Pediatric dose
  * 10-20 mg/kg/d PO divided bid/tid
  * Increase weekly to achieve optimal clinical response, administered tds/qid.

Note: Unsafe in pregnancy

Adverse drug reactions

Adverse drug reactions include dizziness, drowsiness, and abdominal pain; rarely, rash, agranulocytosis, aplastic anemia, and Stevens-Johnson reaction.

Phenytoin

- Adult dose: 300 mg PO
- Pediatric dose: 4-8 mg/kg/day PO divided bid/tid (or) bd/tds
- Contraindications
  * Arrhythmia.
- Unsafe in pregnancy
- Adverse reactions
  * Gingival hyperplasia, nausea, and vomiting; rarely, Stevens-Johnson syndrome, nystagmus, slurred speech, and ataxia.

Anti-helminthics

Treatment with anti-helminthic drugs can result in complete resolution or significant regression in 80-90% of patients.

- A single dose of Praziquantel (5-10 mg/kg) can be administered to individuals found to have T solium tapeworms in their stool.
- Albendazole - Adult dose 400 mg PO bid for 14 days with meals. (Drug of choice) Pediatric dose - 15 mg/kg/d PO divided bid for 14 days with meals.

Important

To avoid inflammatory response in the CNS, the patient must be started on anticonvulsants and high-dose glucocorticoids.

Adverse effects: Headache, nausea, dizziness, vomiting, alopecia, rash, fever, and pancytopenia.

Surgical Care

Reserve neurosurgical intervention for cases of cysts that have failed to resolve with anti-helminthic treatment and are causing severe neurologic sequelae.

Surgery may be indicated for cases of intraventricular cysts that are resistant to medical management. Shunting may be indicated for hydrocephalus.

Diet

Avoid reinfection. No other specific diet is necessary.

Patient education

- Educate patients and their families regarding prevention
- Emphasize improvement in sanitation, separation of pigs from humans, and food preparation hygiene in endemic areas.

When to admit and refer

Arrange transfer of the patient to higher centres if

neurologic or neurosurgical care is necessary. Inpatient care is necessary for the following clinical states:

- Children who need anti-helminthic therapy for active or multiple cysts require to be hospitalized for the first 72 hours of therapy due to the inherent risk of anaphylactic reaction
- Signs of increased intracranial pressure or apparent need for corticosteroid treatment
- Intractable seizures
- Hydrocephalus, requiring a shunt procedure.

Follow - up

Patients who have single non-viable lesions do not require anti-helminthic treatment and can be managed safely on an outpatient basis.

Arrange neurologic follow-up care to manage seizures and any sequelae Perform a follow-up MRI in 3-6 months or sooner if symptoms worsen or recur.

If a child who was admitted for anti-helminthic treatment is doing well after 72 hours and follow-up care is assured, the child can be discharged to finish therapy at home.

Complications

Hydrocephalus, intractable seizure disorder, hemiparesis, motor and speech delay, blindness have been reported.

Prognosis

In cases with single lesions, prognosis is excellent. In those with multiple lesions, especially extra-parenchymal, prognosis can be poor. Treatment with anti-helminthics results in complete resolution or significant regression in 80-90% of patients. Most children with calcified single lesions do not require anti-helminthic treatment. Seizures control is effective in most children with Carbamazepine and they can be weaned from their anti-convulsants within 1-2 years. The majority of children remain free of seizures.

Bibliography

Rajshekhar V, Joshi DD, Doanh NQ, van De N, Xiaonong Z. Taenia solium taeniosis/cysticercosis in Asia: Epidemiology, impact and issues. Acta Trop. 2003;87:53–60.

Del Brutto OH, Rajshekhar V, White AC, et al. Proposed diagnostic criteria for neurocysticercosis. Neurology. 2001;57:177–183.

Vertigo

Definition

Vertigo is defined as the 'hallucination' of movement, either of self (subjective) or the environment (objective). Usually the patient uses various terms (eg.) bouncing, oscillating, staggering, swimming, twisting rolling, spinning, rocking, light-headedness, imbalance, floating, fainting, falling

For the most part they are benign but always there is the possibility that they signal the presence of an important neurological disorder.

General Considerations

Spatial orientation is largely automatic but complex. Continued sensory monitoring assesses the position of the body in space, in relation to the surrounding environment. The 5 sensory modalities constantly sample position and motion: vision, vestibular sensation, proprioception, touch and pressure, and hearing.

Normally the brain integrates the input from each of these sensory modalities giving a comprehensive image of position and motion in space. This process enables us to maintain balance, move about, and interact with other objects

When the orienting image is unreliable, we become uncertain of position and the result is a sensation of spinning or vertigo.

Clinical Aspects

When a patient presents with this type of dizziness, the clinician must next determine whether the symptom is central (brain) or peripheral (inner ear, 8th cranial nerve) in origin.

Causes

Peripheral causes of vertigo

- Peripheral vestibulopathy
  - Includes labyrinthitis
  - Vestibular neuronitis, and
  - Acute and recurrent peripheral vestibulopathy
- Benign positional vertigo
  - Includes benign positional nystagmus
  - Benign paroxysmal vertigo
- Post-traumatic vertigo
- Vestibulotoxic, drug-induced vertigo
- Meniere's disease

- Other focal peripheral diseases
  - Includes local bacterial infection
  - Degeneration of hair cells
  - Genetic anomalies of labyrinth
  - Cupulolithiasis, tumor of eighth nerve
  - Otosclerosis
  - Fistula of labyrinth, and
  - Rarely focal ischemia

Central neurological causes of vertigo

- Brainstem ischemia and infarction
- Demyelinating disease:
  - Multiple sclerosis
  - Post infectious demyelination
  - Remote effect of carcinoma
- Cerebello-pontine angle tumor
  - Acoustic neuroma
  - Meningioma
  - Cholesteatoma
  - Metastatic tumors
- Cranial neuropathy
- Focal involvement of eighth nerve or in association with systemic disorders
- Intrinsic brainstem lesions (tumor, arteriovenous malformation)
- Other posterior fossa lesions (primarily other intrinsic or extra – axial masses of the posterior fossa, such as hematoma, metastatic tumor, and cerebellar infarction)
- Seizure disorders (rare)
- Heridofamilial disorders such as spinocerebellar degeneration

Systemic causes of vertigo

- Drugs
  - Anticonvulsants
  - Hypnotics
  - Antihypertensive
  - Alcohol
  - Analgesics
  - Tranquilizers
- Hypotension, pre-syncope (including primary cardiac causes and postural hypotension from a wide variety of causes)
- Infectious diseases
  - Syphilis
  - Viral and other bacterial meningitis
  - Systemic infection
- Endocrine diseases
  - Diabetes mellitus
  - Hypothyroidism
- Vasculitis
  - Collagen vascular disease
  - Giant cell arteritis, and
  - Drug–induced vasculitis
- Other systemic conditions
  - Hematological disorders
    - Polycythemia, and
    - Dysproteinemia
  - Sarcoidosis
  - Granulomatous diseases and
  - Systemic toxins

Characteristics of peripheral versus central positional vertigo		
Symptom or sign	Peripheral	Central
Latency (time to onset of Vertigo or nystagmus)	0-40 sec (mean 7.8*)	No latency; begins Immediately
Duration	<1 min	Symptoms may persist (signs and symptoms of single Episode)
Fatiguability (habituation)	Yes	No
(Lessening signs and symptoms with repetition of provocative manoeuvre)		
Nystagmus direction	Direction: fixed torsional, Up, upper pole of eyes toward ground	Direction changing, variable
Intensity of signs and symptoms	Severe vertigo, marked nystagmus, nausea	Usually mild vertigo, less intense nystagmus, rare nausea
Reproducibility	Inconsistent	More consistent

Peripheral Vestibulopathy

Definition

Peripheral vestibulopathy has been described as vestibular neuronitis, labyrithitis, or viral neuro-labyrinthitis.

Vestibular neuronitis, strictly speaking, is characterized by single or recurrent sudden episodes of true vertigo lasting from hours to days and is often associated initially with vomiting.

When the condition is associated with hearing loss, the entire labyrinth is assumed to be involved, and the term labyrinthitis is used.

Symptoms

In the acute phase, most patients present with sudden severe vertigo, nausea, and vomiting without any hearing disturbance or facial weakness. The acute symptoms usually resolve in a few days to a week but may recur in weeks or months.

Causes

- Epidemic and seasonal outbreaks of acute vertigo have suggested an infectious origin caused by viral disease, but this remains largely unproved.
- Viral labyrinthitis can also be part of a systemic viral infection, such as mumps, measles, infectious mononucleosis, or upper respiratory tract viral infections
- Isolated viral infections of the labyrinth are also believed to cause the sudden onset of hearing loss, vertigo, or both, in children and adults.

Otitic Herpes Zoster

Definition

Otitic herpes zoster is an infection characterized by pain in the ear, followed in 1-10 days by a vesicular eruption in the external ear, when the seventh and eighth nerves are affected, there is a combination of facial weakness, hearing loss, and vertigo known as the **Ramsay-Hunt syndrome.** A dysesthetic area of skin may precede, by many days, the appearance of the skin eruption.

Beningn Paroxysmal Positional Vertigo

Definition

Benign paroxysmal positional (or "positioning") vertigo (Blood pressurePV) is a symptom complex suggesting benign peripheral (end-organ) disease. It is a major cause of vertigo.

Symptoms

Historical factors that should lead to the consideration of Blood pressurePV include the following:

- Symptoms associated with certain head positions,
- Episodic rotational vertigo of brief duration,
- Antecedent episode of severe rotary vertigo with or without nausea and vomiting, associated with an upper respiratory tract infection that suggests prior viral neuro-labyrinthitis
- History of head trauma before attacks of vertigo,
- Most severe symptomatology early in the day, with lessening symptoms as the day progress
- Relative absence of spontaneous symptoms without head movement or position change.

The signs and symptoms of benign positional vertigo are transient and rarely last longer than 40 seconds. They usually occur when a certain position is assumed, such as lying down or turning in bed. Depending on whether the symptom (vertigo) or sign (nystagmus) is being emphasized, this condition can be called benign paroxysmal positional nystagmus or Blood pressurePV.

Investigations

Physical examination findings include

- Vertical rotary benign positional paroxysmal nystagmus produced by provocative maneuvers.
- Latency to onset of symptoms once precipitation head position is achieved,
- Short-duration nystagmus (3-30 seconds), and
- Adaptation of nystagmus and symptoms (i.e., disappearance with repeated maneuvers). The findings of the typical nystagmus on assumption of certain head positions are considered the most important physical finding in making the diagnosis of Blood pressurePV.
- In benign paroxysmal positional vertigo, the nystagmus fast phase is horizontal-rotary directed toward the lower ear. The nystagmus fast phase is upward toward the forehead when gaze is directed to the upper ear
- With the eyes in the central orbital position, the nystagmus fast phase is vertical upward and rotary toward the lower ear.

Meniere's Disease

Definition

Meniere's disease is characterized by attacks of severe vertigo and vomiting, tinnitus, fluctuating hearing loss, ill-described aural sensations of fullness and pressure and spontaneous recovery in hours to days.

This is followed by severe vertigo which reaches peak intensity within minutes and slowly subsides over hours, with a persistent sense of dis-equilibration for days after an acute episode.

Consciousness is not lost in such episodes, although of the accompanying vertigo, nausea.

Causes

- The most consistent pathological finding in Meniere's disease is an **increase in the volume of the endolymphatic fluid** and distension of the canals, hence the term endolymphatic hydrops.
- Although some specific causes, such as bacterial, viral, and syphilitic infections, may lead to the same pathological changes and symptoms, most cases are idiopathic

Symptoms

According to the guidelines from AAO-HNS Committee of hearing and equilibrium the major symptoms are described as follows:

- Vertigo
  - * Recurrent, well-defined episodes of spinning or rotation
  - * Duration ranging from 20 min to 24 h
  - * Nystagmus associated with attacks
  - * Nausea and vomiting during vertigo spells are common
  - * No neurologic symptoms with vertigo
- Deafness
  - * Hearing deficits fluctuate

    * Sensorineural hearing loss
    * Hearing loss progressive, usually unilateral
- Tinnitus
    * Variable, often low pitched and louder during attacks
    * Usually unilateral on the affected side
    * Subjective

Vestibular Neuronitis

Definition

This term is based on their clinical findings which suggest that the syndrome is caused by an isolated lesion of the vestibular nerve and its central connections. Vestibular neuronitis is diagnosed using three clinical diagnostic criteria:

- Vertigo: usually sudden onset
- An absence of cochlear symptoms or signs (deafness and tinnitus)
- An absence of associated neurological symptoms and signs.

Vertigo in migraine

Vertigo has been found to occur significantly more frequently in patients with migraine than in controls and a high prevalence of migraine has been found in vertigo sufferers.

Basilar migraine

* Basilar migraine consists of aura and headache. The aura should include at least two of the following: vertigo, tinnitus, decreased hearing, ataxia, dysarthria, double vision, hemi field visual symptoms (both eyes and both fields), bilateral paraesthesia, bilateral hemiparesis, decreased level of consciousness.

Definite migrainous vertigo

* Episodic vestibular symptoms of at least moderate severity
* At least two of the following migrainous symptoms during at least two vertiginous attacks:
    » migrainous headache
    » photophobia,
    » phonophobia
    » visual or other aura.
- Attacks of migraine (outside episodes of vertigo) according to HIS migrainous
- Some central and/or peripheral vestibular abnormalities may be found in vertigo-free periods.
- Other causes ruled out by history, physical examination and other appropriate investigations.

Probable migrainous vertigo

Definition

- Episodic vestibular symptoms of at least moderate severity (rotational vertigo, other illusory self or object motion, positional vertigo, head motion intolerance)
- At least one of the following, in relation to at least one vertiginous attack
    * Migrainous headache
    * Photophobia
    * Phonophobia
    * Migraine-specific triggers e.g. specific foods, sleep irregularities, hormone changes
- Response to migraine prophylactic drugs
- Migraine (outside vertiginous attacks) according to the criteria of the IHS
- Some central and/or peripheral vestibular abnormalities may be found in vertigo-free periods
- Other causes ruled out by history, physical examination and other appropriate investigations

Benign recurrent vertigo

Definition

- Episodic vertigo, occasionally with tinnitus but without hearing loss
- May be accompanied by nausea/vomiting and ataxia
- Nystagmus may be observed during the episode
- Duration: minutes to hours, usually less than one hour, or hours to days
- Episodes of migrainous headaches outside vertiginous episodes, and/or positive family history of migraine
- Normal audiometric findings, or no asymmetry if there is an incidental hearing loss

- Differential diagnosis: vestibular hydrops, other episodic vestibular disorders, epilepsy

Vertigo in Cerebrovascular disease

- Several mechanisms can cause vertigo and dizziness in persons with cerebrovascular disease.
- Vertigo can occur with cerebrovascular disease that involves the vertebrobasilar circulation, which supplies the labyrinth, the lateral pontomedullary region that contains the vestibular nuclei, and the cerebellum.
- In vertebrobasilar artery (VBA) insufficiency, vertigo is sudden in onset, lasts only minutes, is associated with nausea and vomiting, and is usually accompanied by a range of neurologic deficits (eg, extremity weakness, numbness, in coordination, and dysarthria, diplopia, field defects, tinnitus, hearing loss, loss of consciousness, drop attacks).
- Isolated vertigo without additional symptoms can be the presenting manifestation of vertebrobasilar ischemia.

Symptoms

- Symptoms and signs of posterior fossa cerebrovascular disease include vertigo, tinnitus, and ataxia. Symptoms may be transient, permanent, recurrent, or isolated.
- Stroke syndromes that involve the posterior circulation vary depending on the involved territory. Wallenbergh syndrome (ie, lateral medullary syndrome) appears with vertigo, nausea, vomiting, imbalance, ipsilateral facial numbness and weakness, diplopia, dysphagia, and dysphonia.
- Infarction of the dorsolateral ponto-medullary region results in labyrinthine injury (ie, severe vertigo, nausea, vomiting, hearing loss) in addition to the signs and symptoms of Wallenbergh syndrome.
- Cerebellar infarction also occurs with severe vertigo, nausea, vomiting, and ataxia. When cerebellar infarction occurs without any other associated neurologic or audio logic symptoms, the presentation may be attributed to viral neuronitis (VN). Such cases of pseudo-VN usually occur after infarction of the nodulus and uvula, territory supplied by the medial branch of the posterior inferior cerebellar artery (mPICA). Cerebellar signs that affect the extremities can be minimal or absent in mPICA infarction of the vestibulocerebellum. Basilar artery syndrome results from infarction of the pons.
- Symptoms include
  * Vertigo
  * Hearing loss
  * Ataxia
  * Ophthalmoplegia
  * Blindness and regional sensory losses
  * Cerebellopontine angle (CPA) tumors typically result in disequilibrium or unsteadiness rather than a sensation of vertigo. However, sudden change in tumor size with hemorrhage or disruption of regional blood flow to the labyrinth may precipitate vertigo
  * Tumors in the Cerebellopontine angle are most likely to be vestibular schwannomas

Investigations

All the patients are to be subjected to a thorough history, clinical examination and various bed side tests such as

- The Head thrust test
- Dynamic visual acuity
- Head shaking test
- Dix Hall pick's test
- Fukuda's test and Calorie test
- CT and MRI brain
- X-ray neck
- Blood sugar
- Lipid profile
- Carotid vertebral doppler.

The Head Thrust test

- Method
  * The patients's head is held firmly on each side and the patient is asked to fixate on any one point – say the examiner's nose. The head is moved slowly from side to side to see if the eyes are remaining fixed on one point (the examiner's nose).
  * Once the examiner is satisfied that the patient is following the instructions, the head is

rapidly brought back into the midline. If the vestibulo-ocular reflex is normal, the eyes do not move at all.

* If there is vestibular imbalance, the vestibulo ocular reflex on the affected side is hypoactive and the eyes move with the head. When the head is brought back to the midline, there is a re-fixation saccade.
* The examiner carefully looks for this re-fixation movement of the eyes. This saccadic re-fixation only occurs after rotation of the head to the affected side and persists for a very long time.
* The Head Thrust test is very reliable for unilateral vestibular hypo function.

Dynamic visual acuity

- Method
  * Ask the patient to read the smallest line possible on a Snellen's eye chart with best corrected vision. Repeat the visual acuity (VA) while passively shaking the patient's head at 2Hz. Record the number of lines "lost" during the head shakes.
  * If the VOR is normal, the eyes remain fixed on the target line and the visual acuity does not change. If the VOR is hypoactive, the eyes move with the head and are no longer fixed on the target line, resulting in visual degradation.
  * Loss of three or more lines from static VA indicates vestibular dysfunction and is a good test for vestibular hypofunction due to ototoxicity or age.

The head shaking test

The patients head is pitched down 30' and oscillated at 2 Hz for 20 seconds. An abnormal test is elicitation of jerk nystagmus. Post head shake nystagmus is considered pathologic of vestibular imbalance. In most cases a peripheral vertigo is identified with the fast phase beating towards the unaffected (stronger) ear.

The Dick-Hallpike test

Is a manoeuvre which is specifically positive if a patient has benign paroxysmal positional vertigo (Blood pressurePV). It is intended to stimulate the vertical semi circular canals.

The anterior and contralateral posterior semi circular canals are approximately parallel in a plane orientated 45' from the sagittal plane. With the head turned 45' to one side, moving patient from sitting to supine results in rotation in the plane of the canal pair, ie. in the horizontal plane.

In the normal patient, nystagmus occurs during the manoeuvre but not after it. In Blood pressurePV, a change of position causes movement of the endolymph eliciting vertigo. The nystagmus starts at a latency of 4-10 seconds and lasts upto 30 seconds.

Involvement of the posterior semicircular canals results in up-beating nystagmus with a rotary component, the upper pole of the eye beating towards the lower ear. Blood pressurePV results from debris moving freely in the vertical semi circular canals, usually the posterior.

Fukuda test (Stepping test of Underberger)

Ask the patient to march in place with eyes closed and arms outstretched- normally less than 15 degrees or so of rotation is displayed Asymmetry of labyrinthine function is manifested as excessive rotation away from the diseased side.

Calorie test

- The patient's head is ideally tilted forward 30 degrees from the horizontal, each auditory canal is irrigated for 30 seconds, first with water at 30° C, and then at 44° C with a pause of at least 5 minutes between each irrigation.
- In normal persons cold water induces a slight tonic deviation of the eyes to the side being irrigated, followed after a latent period of 20 seconds by nystagmus to the opposite side (direction of fast phase). Warm water induces nystagmus to the irrigated side.

Nystagmus

- The presence of spontaneous or induced nystagmus is of crucial importance in making a diagnosis of peripheral or central causes of imbalance.
- Spontaneous nystagmus of a peripheral origin is usually due to lesions either of the labyrinth or eighth cranial nerve.
- The characteristics of nystagmus of a peripheral origin are as follows
  * Mixed–horizontal plus rotational or tortional.
  * Presence of fixation suppression – if there is

a gaze evoked nystamus while the patient is staring at a blank wall, asking the patient to fixate on your finger, suppresses the nystagmus i.e. the nystagmus is so-to-say "fatiguable".

* The nystagmus is intense, i.e. it intensifies (increases in amplitude) in the direction of the fast phase,
* It is direction fixed i.e. it does not change direction with gaze. Usually in an irritative lesion, it is in the direction of the affected ear and beats towards the unaffected ear if the lesion is destructive.

- Spontaneous nystagmus of central origin is usually due to lesions of the brainstem, cerebellum or rarely certain areas of the cerebrum.
- The characteristics of nystagmus of central origin are quite the opposite of peripheral nystagmus.

Thus the characteristics of central nystagmus are:

- Pure – purely horizontal, vertical or torsional
- Absence of fixation-suppression – it is "non-fatiguable" when one fixates over the examiner's finger
- Less intense – does not intensify in amplitude in the direction of fast gaze
- Direction changing with gaze - left beating nystagmus with left gaze

Nystagmus is dampened by convergence, therefore, do not hold the finger very close to the eyes – hold it at least 14 inches away. Primary position nystagmus is suppressed by fixation. The manoeuvres to suppress fixation are to ask the patient to stare at a totally blank wall, the hand held ophthalmoscope method.

Treatment

Meniere's Disease

- During an acute attack of Meniere's disease, rest in bed is the most effective treatment, since the patient can usually find a position in which vertigo is minimal.
- The anti-histaminic agents
  * Cyclizine
  * Meclizine or transdermal scopolamine are useful in the more protracted cases.
  * Promethazine is an effective vestibular suppressant, as is tri-methobenzamide given in 200-mg suppositories, which also suppresses nausea and vomiting.
- For many years a low-salt diet in combination with ammonium chloride and diuretics have been used in the treatment of Meniere's disease, but the value of this regimen has never been established.
- The same is true for dehydrating agents such as oral glycerol and the more recently popular calcium channel blockers.
- Mild sedative drugs may help the anxious patient between attacks.

Vestibular Neuronitis

During the acute stage, antihistamine drugs, Phenergan, Clonazepam and Scopolamine may be helpful in reducing the symptoms. Vestibular exercises are recommended.

Medical therapy for vertigo

Antihistamines

- Meclizine 25-50mg 3 times / day
- Cyclizine 50mg 2 times / day
- Dimenhydrinate 50mg 2 times / day
- Promethazine 25-50mg / day

Anti-cholinergics

- Scopolamine tablets 0.45-0.50 mg 2 times/day
- Scopolamine Transdermal patch 1/day for 3 days

Sympathomimetics

- Ephedrine 25 mg/day

Anti-emetics

- Tri-methobenzamide 250mg 2 times / day PO/200-mg suppository
- Promethazine 25-50mg / day
- Prochlorperazine 5-10mg 3 times / day PO/25-mg suppository

Tranquilizers

- Diazepam 5-10mg 3 times / day
- Oxazepam 10-60mg / day

- Haloperidol 0.5-5mg 2 times / day

Surgical Treatment

Surgical treatment of chronic peripheral vestibular dysfunction is primarily destructive. In patients with severe Meniere's disease for whom medical therapy has been ineffective and who have severe recurrent disabling attacks, a labyrinthectomy may be performed.

Unfortunately, Meniere's disease may become bilateral, eventually resulting in the need for labyrinthectomy or vestibular nerve section on the contralateral side.

A medical labyrinthectomy may be performed by the use of Aminoglycoside drugs, which are particularly destructive to the peripheral vestibular hair cells.

Surgical or medical labyrinthectomy usually is a last resort for patients who have clearly defined, severe attacks of peripheral vestibulopathy, presumably from Meniere's disease.

References

- Allan H. Ropper, Robert H. Brown, Deafness, Dizziness and Diseases of equilibrium, Adams and Victor's Principles of neurology, 8th edition, pp1117-1126.
- Todd Troost, Lisa.C Arguello, Neurootology, Walter G. Bradley's Neurology in clinical practice, 4th edition, pp746-748.
- Article from E medicine (www.emedicine.com.) on Vertigo from internet.

Nephrology

Standard Treatment Guidelines
Tamil Nadu Health Systems Project

Chapter 8

- Acute Nephritic Syndrome
- Chronic Kidney Disease
- Nephrotic Syndrome
- Acute Renal Failure
- Hyponatremia
- Hypernatremia
- Hypokalemia
- Hyperkalemia

Acute Nephritic syndrome

Definition

Acute post-infectious glomerulonephritis following streptococcal pharyngitis or skin infection is a prototypical lesion of Nephritic syndrome.

Clinical features

Acute Nephritic syndrome is characterized by the following clinical features

- Hematuria
- Proteinuria
- Hypertension
- Oliguria
- Edema
- Decreased GFR
- RBC casts or dysmorphic RBCs in urine.

Causes

It is a spectrum of diseases with various etiologies with a common site of injury the glomerulus. The common causes are:

Post-infectious diseases:
Post-streptococcal glomerulonephritis
Non-streptococcal glomerulonephritis
Infective endocarditis
Pneumcoccal infections
Shunt nephritis
Meningococcemia
Multisystem diseases
SLE
Vasculitis
Henoch-schonlein purpura
Goodpasteur's syndrome
Primary Glomerular diseases
IgA Nephropathy
Membranoproliferative glomerulonephritis

Symptoms

- Puffiness of face
- Pitting bilateral pedal edema
- Cola coloured urine
- Dyspnoea, if pulmonary edema occurs
- Hypertension
- Hypertensive encephalopathy
  - * Seizures,
  - * Altered sensorium

There is usually a latent period of 10 to 14 days for occurrence of symptoms following a respiratory tract infection. This latent period is about 3 weeks following a skin infection.

Investigations

- Urine microscopic examination - dysmorphic RBC's or RBC casts
- Blood urea and serum creatinine levels
- Complement levels: Transient decline in C 3 levels which become normal in 8 weeks
- Investigations for confirming streptococcal infection are:
  - * Tests for serological confirmation of post streptococcal glomerulonephritis: ASO titre, Anti DNA ase - B
  - * Positive throat culture for beta hemolytic streptococci

Treatment

- Bed rest
- Fluid restriction according to volume status
- Salt restriction
- Diuretics for control of volume overload status: loop diuretics are preferred. Intravenous route if pulmonary edema is present.
- Control of hypertension with antihypertensive drugs
- Management of renal failure - may rarely require dialytic support
- Renal biopsy is indicated, if features are not suggestive of post streptococcal glomerulonephritis.

Prognosis:

Epidemic forms of disease in children have a uniformly favorable short and long term prognosis. Sporadic cases, especially in adults may have more serious long term consequences. Following resolution of symptoms, microscopic hematuria can persist for as long as a year.

Chronic kidney disease

Epidemiology

Chronic kidney disease (CKD) is a major public health problem with rising incidence and prevalence. The disease may progress to end-stage renal disease, which is associated with significant morbidity and mortality. The incidence of CKD in the general population is estimated to be 100 to 250 per million people in India.

CKD is a disease which in most instances is silent until late stages. Therefore it is important to identify those at risk and screen them and to initiate appropriate measures to stop the progression of disease.

Causes

- Diabetes Mellitus
- Hypertension
- Glomerular diseases
- Congenital and hereditary diseases like Polycystic Kidney disease and Vescico ureteric reflux.

Investigations

Screening for CKD

Identify people at high risk

- Obesity
- Smokers
- Diabetes
- Hypertensives
- Family history of renal disease

How to screen at risk individuals?

- Measure blood pressure
- Urine for proteinuria, hematuria and other sediments
- Estimate Serum creatinine. Screen them at least yearly.

Diagnosis

Either one of the following establishes the diagnosis:

- Kidney damage for more than 3 months as defined structural or functional abnormalities of the kidney with or without decreased GFR
- Decrease in GFR to < 60ml/min for more than 3 months.

GFR is calculated by the formula:GFR = (140-age) x (Wt in kg) x (0.85 if female) / (72 x Cr in mg/dl)

Staging

Chronic Kidney disease has been divided into five stages.

Stage	Description	GFR (ml/1.73m2/min)
Stage I	Kidney damage with normal or ↑ GFR	> 90
Stage II	Kidney damage with mild ↓ GFR	60 - 89
Stage III	Moderate ↓ in GFR	30 - 59
Stage IV	Severe ↓ in GFR	15 - 29
Stage V	End stage renal disease	< 15

- GFR (Glomerular filtration rate)

Treatment

Step 1:
Look for clues with respect to etiology

- History of hypertension or diabetes mellitus.
- Symptoms related to lower urinary tract, recent infections
- Features of connective tissue disease
- Past medical records suggestive of proteinuria
- Pregnancy related problems
- Past renal evaluation
- Family history of hereditary kidney disease like ADPKD/ Alports syndrome.

Step 2:
Reversible factors in CKD

- Obstructive nephropathy
- Urinary tract infections
- Extra renal infections
- Nephrotoxic drugs
- Hypovolemia
- Congestive cardiac failure
- Pericardial tamponade
- Hypokalemia / hyperuricemia

Step 3:
Modifiable Risk Factors

- Control of blood pressure :
  - In patients with Proteinuria > 1 g / day, BP target is < 125 /75 mm Hg
  - If proteinuria < 1 g / day, BP target is < 135/ 85 mm Hg
- Drugs used to control Hypertension :
  - ACE inhibitors
  - Diuretics
  - Calcium Channel blockers
- Glycemic control : Target HbA1C levels of < 6.5
- Control of Proteinuria : Done by using ACE inhibitors
- Control of hyperlipidemia
  - Total cholesterol < 200 mg/dl , LDL Cholesterol <100 mg /dl is the target
  - Use of HMG CoA reductase inhibitors - statins
- Obesity: Aim for ideal body mass index of < 25
- Cessation of smoking

STEP 4:
Prevent and treat complications

- Malnutrition:
  - Dietary protein intake of 0.6 g / kg body weight / day
  - Dietary calorie intake of 30 - 35 kcals / kg / day
- Cardiovascular disease
  - It is the commonest cause of mortality in CKD patients.
  - Risk for IHD increases by 30% for every increase in serum creatinine by 0.23 mg/dl.
  - At least yearly screening for cardiovascular disease should be done.
- Anaemia:
  - Target hemoglobin of 11 g / dl is desirable in CKD patients. They need iron and erythropoietin injections
- Renal osteodystrophy:
  - Target calcium - 8.8 to 9.7 mg / dl
  - Phosphorous - 3.5 to 5.5 mg /dl
  - Calcium - Phosphorous product < 55 mg^2 / dl
- This is achieved by:
  - Use of calcium or non-calcium containing phosphate binders
  - Phosphorus restriction-avoidance of dairy products, meat , colas.
  - Use of calcitriol

Step 5 :
Prepare for Renal replacement therapy

- Early referral to Nephrologist
- Vaccination:
  - Hepatitis B vaccine 40 micro grams 3 doses - 0, 1 , and 2nd month followed by booster dose at 6 th month .
- Early creation of arterio-venous fistula when the GFR is < 25 ml/min or Serum Creatinine > 4.0 meq/l

Nephrotic syndrome

Definition

- Proteniuria more than 3.5 gm/day in adults
- Proteinuria more than 40mg/m^2/hr in children
- Associated features:
  - Hypoalbuminemia
  - Hyperlipidemia
  - Edema

Causes

Diseases confined to kidney:

- Minimal change disease
- Membranous nephropathy
- Focal segmental glomerulosclerosis
- Membranoproliferative GN
- Mesangioproliferative GN

Systemic diseases:

- Diabetes mellitus
- SLE
- Amyloidosis
- Henoch-schonlein purpura
- Preeclampsia

Hereditary diseases

- Congenital nephrotic syndrome
- Alports syndrome

Complications of Nephrotic syndrome

- Increased predisposition to infections
- Thrombotic tendencies
- Hyperlipidemia
- Hypocalcemia
- Iron resistant hypochromic anemia

Investigations

- Urine analysis: Dipstick test for proteinuria,
- Urine microscopic examination for deposits
- 24 hrs urinary protein: more than 3.5 gm/day establishes the diagnosis
- Renal function tests: Blood urea, serum creatinine – Usually normal in isolated nephrotic syndrome
- USG abdomen

Guidelines for referral

Refer to a tertiary care center if there is a indication for renal biopsy

Indications for Renal Biopsy in Nephrotic Syndrome
• Age > 10 yrs
• Micro - hematuria in urine analysis
• No response to steroid therapy in adequate doses for a period of 4 weeks
• Associated renal failure
• Low C 3 levels
• ANA positive
• Evidence of systemic disease

Treatment

- Steriods:
  - * In children less than 10 yrs, without hematuria, without renal failure, start empirical steroids viz.Prednisolone 2 mg/kg/day for 4 weeks, taper over next 4 weeks
- HMG CoA reductase inhibitors for hyperlipidemia
- Diuretic therapy:
  - * High dose intravenous loop diuretics
- Diet:
  - * Protein 0.8 g / kg /day + 1 g protein per gm lost in urine,35 kcals / kg / day carbohydrates
- ACE inhibitors to reduce proteinuria
- Treatment of infections.
  - * The commonest cause of death in nephrotic syndrome is infection.
  - * Hence treat infection early and aggressively.
  - * Refer for severe uncontrollable infections.

Acute Renal Failure (ARF)

Definition

Acute renal failure is defined as a rapid decline in renal function, reflected by GFR over hours to days as evidenced by raising creatinine values

- ARF can be classified into oliguric vs nonoliguric. (Urine output < 500 ml/day is oliguria)
- ARF complicates upto 5% of hospital admissions and 30% of ICU admissions

Classification:

1. **Pre-renal** - 70%
   - * Hypovolemia - diarrhoea , persistant vomiting, burns, haemorrhage.
   - * Low cardiac output- CCF
   - * Systemic Vasodilatation – Sepsis, Hepato-renal syndrome
   - * Drugs - NSAID's
   - * Management of Pre-renal Failure :
     - » Volume replacement - 0.9 % saline , ringer lactate
2. **Renal** - 25%
   - Can occur due to disease process in glomeruli / tubules / interstitium / vasculature.
     - * **Acute tubular necrosis** can occur due to
     - * Ischemic kidney injury - can occur with any cause of pre-renal failure if prolonged
     - * Nephrotoxic drugs (commonly used)
       - » NSAID 's
       - » Aminoglycosides
       - » Cis-platin
       - » Amphotericin-B
       - » Acyclovir
3. **Post-renal** 5 %
   - * Prostatic hyperplasia with obstruction
   - * Renal stone disease
   - * Carcinoma of cervix

* Malignancies of urinary tract

Evaluation of Renal failure:

- Exclude Pre-renal and postrenal.
- A ultrasound-KUB rapidly rules out the easily correctable post renal causes

Pre-renal

- Usually occurs with volume depletion and by definition renal parenchyma is not damaged
- Urine is characterized by low volume, low sodium (< 20 mmol/l) and high osmolality.
- Treatment should be directed towards the underlying cause.

Renal

Assess for signs and symptoms of uraemia

Symptoms

- Anorexia
- Fatigue
- Mental status changes
- Nausea / Vomiting
- Pruritis
- Shortness of breath

Signs:

- Asterixis
- Myoclonus
- Pericardial rub
- Pedal edema
- Pulmonary rales
- Raised JVP
- Seizures.

Look for clues for etiology of renal failure

CLUES	ETIOLOGY
Sinusitis,Hemoptysis	Pulmonary renal syndromes, Vasculitis
Diarrhoea,Vomiting, Hypotension	Pre-renal hypovolemia
Back Pain	Multiple Myeloma
Trauma,Prolonged immobility	Rhabdomyolysis
Skin rash	Vasculitis,AIN
Liver disease	Hepatorenal syndrome
Prostate symptoms	BPH
Constituonal symptoms	Vasculitis, Malignancy, Infection
Recent surgery	Ischemia, Atheroembolism, Contrast.
Medication	ACEI's, ARB's, NSAID's, Antibiotics
Flank Pain	Obstruction, Pyelonephritis

Blood Investigations

- Urea
- Creatinine
- Serum electrolytes
- Serum calcium
- Serum PO4
- Serum albumin

Urine Analysis

- Urinary pattern
- Pre-renal: small amount, bland few hyaline casts.
- Post-renal: few hyaline casts.
- ATN: Proteinuria usually small amounts,WBC's, epithelial cells, muddy brown appearance.
- Calculate **FeNa (fractional excretion of sodium)**
- Urine Na x Serum Creatinine / Serum Na x Urine Creatinine x100

  <1 – Pre-renal; > 2 – Renal

Indications For Dialysis :

- Hyperkalemia
- Metabolic Acidosis
- Volume Overload

- Encephalopathy
- Pericarditis

Lab tests consistent with ARF (50% increase in creatinine above baseline or 50% decrease in baseline GFR

PRE-RENAL
Features: BUN to Cr > 20:1 FeNa <1 Urine Sp Gr > 1020 Hyaline casts No e/o obstruction No e/o intrarenal cause Treatment : Hydrate Eliminate toxins Treat underlying cause
RENAL
Features: BUN to Cr > 10:1 to 20:1 FeNa >2 Urine Sp.Gr 1.010 to 1.020 Tubular or granular casts in urine **Treatment**: Eliminate toxins Nephrology consultation
POST-RENAL
Features USG : Hydronephrosis Serum and urine test similar to renal cause **Treatment:** CT, KUB Relieve obstruction Urology Consultation

Hyponatremia

Serum sodium reflects the ratio of sodium to that of water.

Hyponatremia is defined as Serum sodium < 135 meq/l

The approach to a patient with hyponatremia:

- First step is to rule out pseudohyponatremia which can occur in conditions like hypertriglyceridemia, para-proteinemia.
- Lab methods involving direct potentiometry does not have this problem.
- Once pseudohyponatremia is ruled out, assess volume status and follow the algorithm.

Treatment of Hyponatremia

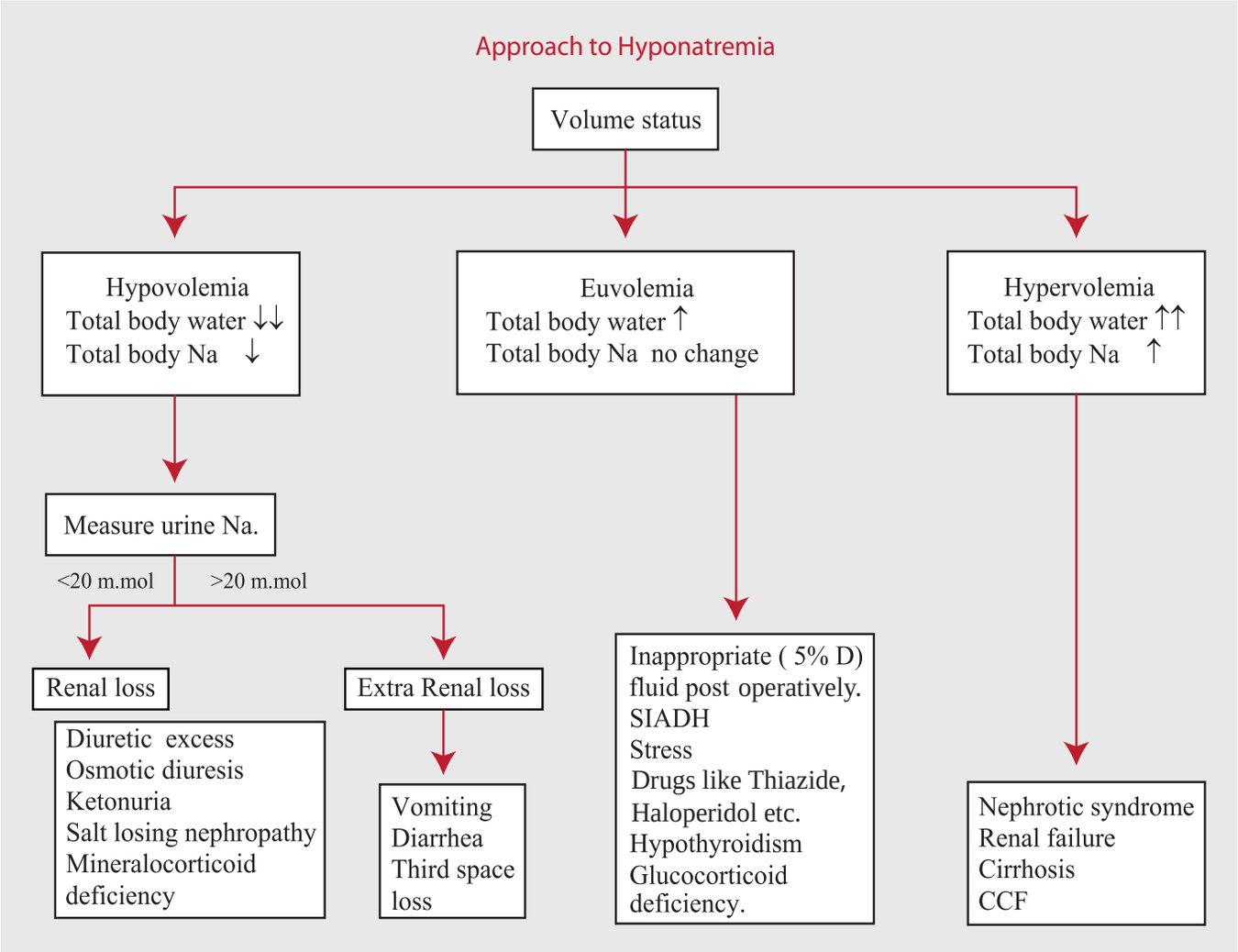
Symptomatology and duration of hyponatremia determine the treatment approach.

- Lethargy
- Headache
- Nausea
- Ataxia
- Psychosis
- Seizures and coma can be manifestations of hyponatremia

Note:

Neurological damage (osmotic demyelination) can occur if

- Acute symptomatic hyponatremia is left untreated
- Chronic symptomatic hyponatremia is corrected too rapidly



Hypernatremia

It is defined as Serum Na > 145 m.mol/l

Thirst and urinary concentrating mechanisms are the important defenses against hypernatremia. Hence it is a rare finding in a conscious patient.

Algorithm to hypernatremia therapy:

Step 1:

* Correct ECF volume status
* Isotonic saline if hypovolemic
* Diuretics if hypervolemic

Step 2:

Calculate free water deficit:

Total body water x [Na/140 - 1] or

0.6 x body weight (kg) x [Na/140 - 1].

Acute hypernatremia can be rapidly corrected. If the hypernatremia is chronic, correct one half using ½ NS (contains 500 ml free water per liter) in first 24 hours.

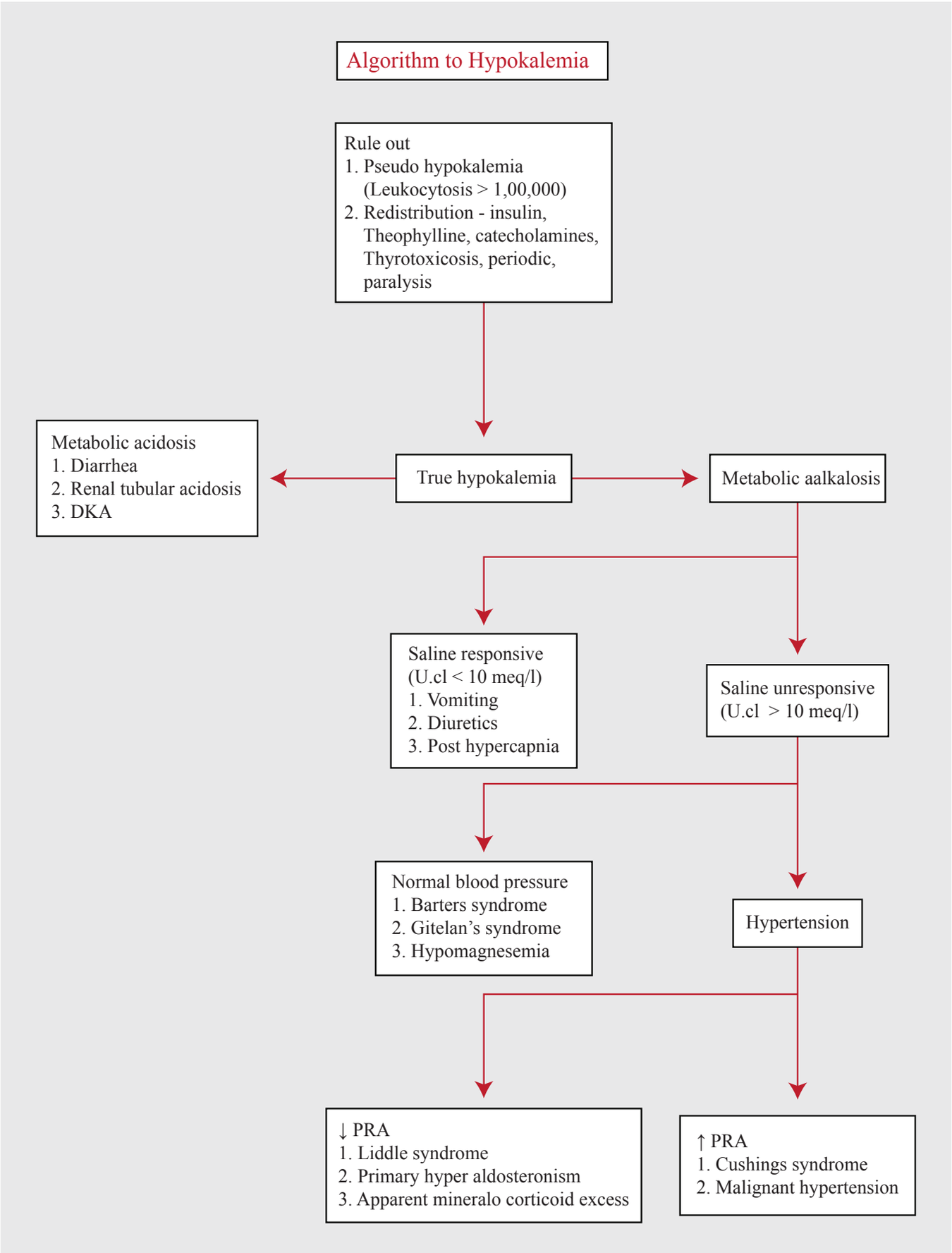
Further correction is done in subsequent 24 hours.

Hypokalemia

- Defined as serum potassium < 3.5 meq/l.
- It can present with muscular symptoms like flaccid paralysis, cardiovascular problems like ventricular arrhythmias and renal involvement like polyuria.

Emergency Trea tment

- Urgent treatment is required in rare situations like periodic paralysis, ventricular ectopics or in the setting of myocardial infarction.
- Parenteral therapy is risky, requires central line and ECG monitoring.
- It is not indicated if the potassium is >3.0. Oral potassium chloride 15 ml tds is usually sufficient if there are no cardiovascular or neurological emergencies.

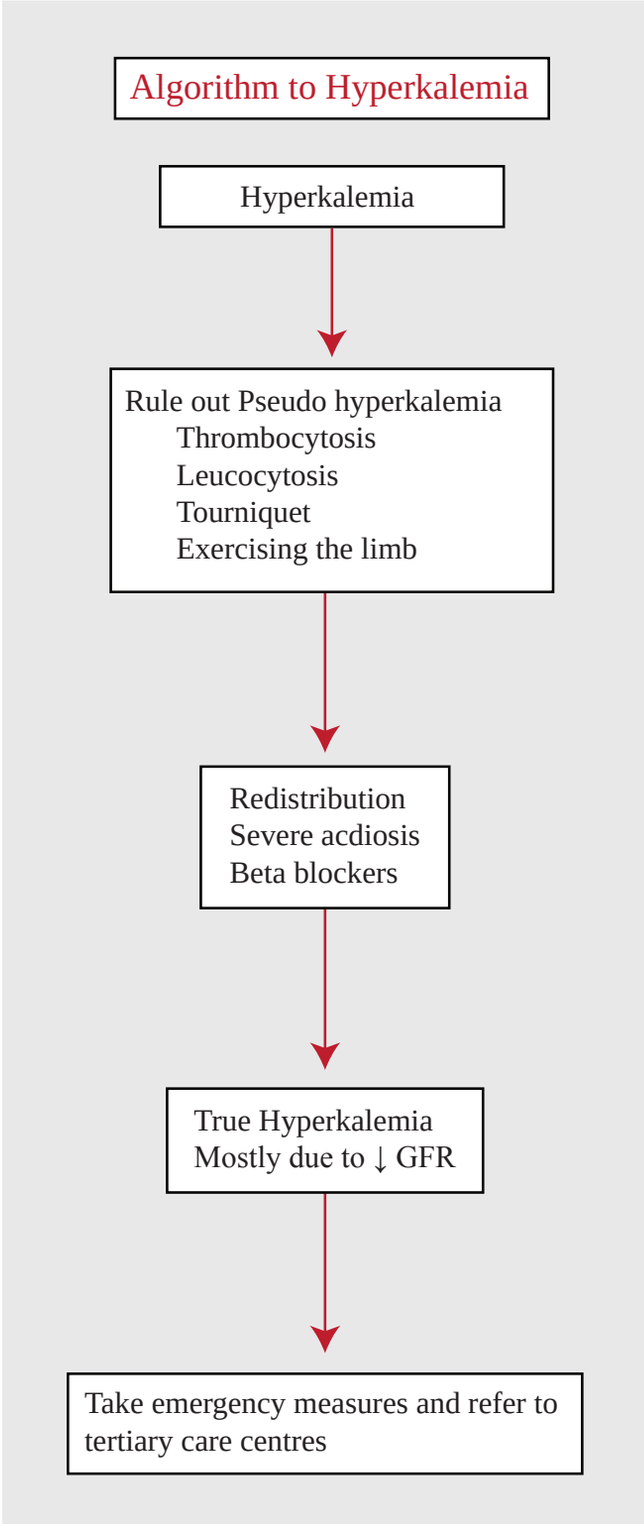

Algorithm to Hypokalemia
Rule out
1. Pseudo hypokalemia
(Leukocytosis > 1,00,000)
2. Redistribution - insulin,
Theophylline, catecholamines,
Thyrotoxicosis, periodic,
paralysis
Metabolic acidosis
1. Diarrhea
2. Renal tubular acidosis
3. DKA
True hypokalemia
Metabolic aalkalosis
Saline responsive
(U.cl < 10 meq/l)
1. Vomiting
2. Diuretics
3. Post hypercapnia
Saline unresponsive
(U.cl > 10 meq/l)
Normal blood pressure
1. Barters syndrome
2. Gitelan's syndrome
3. Hypomagnesemia
Hypertension
↓ PRA
1. Liddle syndrome
2. Primary hyper aldosteronism
3. Apparent mineralo corticoid excess
↑ PRA
1. Cushings syndrome
2. Malignant hypertension

Hyperkalemia

- Hyperkalemia is defined by serum potassium > 5.5meq/l.
- It can be fatal by its effects on conduction system.
- As serum potassium increases ECG becomes progressively abnormal viz.
  - * Tenting of T waves
  - * Prolonged PR and QRS interval
  - * Flattened P waves, sine wave and asystole.
- Muscle paralysis can also be a feature of hyperkalemia.

Emergency treatment of hyperkalemia

- Administer inj.calcium gluconate 10 ml of 10% solution over 10 min. effect lasts for 30-60 mins. Repeat hourly
- Infuse 25% dextrose 100 ml + 10 units of regular insulin. The effect lasts for 4- 6 hours
- Salbutamol nebulisation 10 mg (2- 8 times the normal dose).
- **Note:** All three steps are to be combined. All these can buy time before patient reaches the tertiary care center for dialysis.



Endocrinology

Standard Treatment Guidelines
Tamil Nadu Health Systems Project

Chapter 9

1. Diabetes Mellitus
   * Introduction
   * Classification
   * Approach to a newly diagnosed Diabetic
   * Treatment
     » Oral Hypoglycaemic Agents (OHA)
     » Insulin Therapy
     » Medical Nutrition therapy (MNT)
2. Gestational Diabetes mellitus
3. Complications of DM
   * Hypoglycemia
   * DKA
   * Diabetes and infections
   * HHNKC
4. Microvascular Complications
   * Diabetic Foot
   * Diabetic Retinopathy
   * Diabetic Neuropathy
   * Diabetic Nephropathy
5. Macrovascular Complications
   * DM and CAD
6. Prognosis of Diabetes mellitus
7. Hypothyroidism
8. Hyperthyroidism
9. Hypocalcemia
10. Hypercalcemia

Diabetes Mellitus

Introduction

Diabetes is world-wide in distribution and the incidence of both type 1 and type 2 diabetes is rising. This global pandemic principally involves type 2 diabetes and is associated with several contributory factors including increased lon-gevity,Obesity,unsatisfactory diet,sedentary lifestyle, and increasing urbanisation.

Moreover type 2 diabetes is also commencing at an earlier age in many populations, and in some ethnic groups,such as Hispanic and Afro-Americans ,is now being observed in children and adolescents.India ,a fast developing nation is invariably set to become the diabetic capital of the World .

Definition

Diabetes Mellitus is a pan metabolic disorder characterised by chronic hyperglycemia with disturbances of carbohydrate, fat and protein metabolism due to defects in insulin secretion or insulin action or both.

Symptoms

- Polyuria -frequent passage of urine with increased nocturnal frequency.
- Polydypsia - increased thirst
- Polyphagia - abnormal excessive appetite
- Weight loss

Associated symptoms and signs :

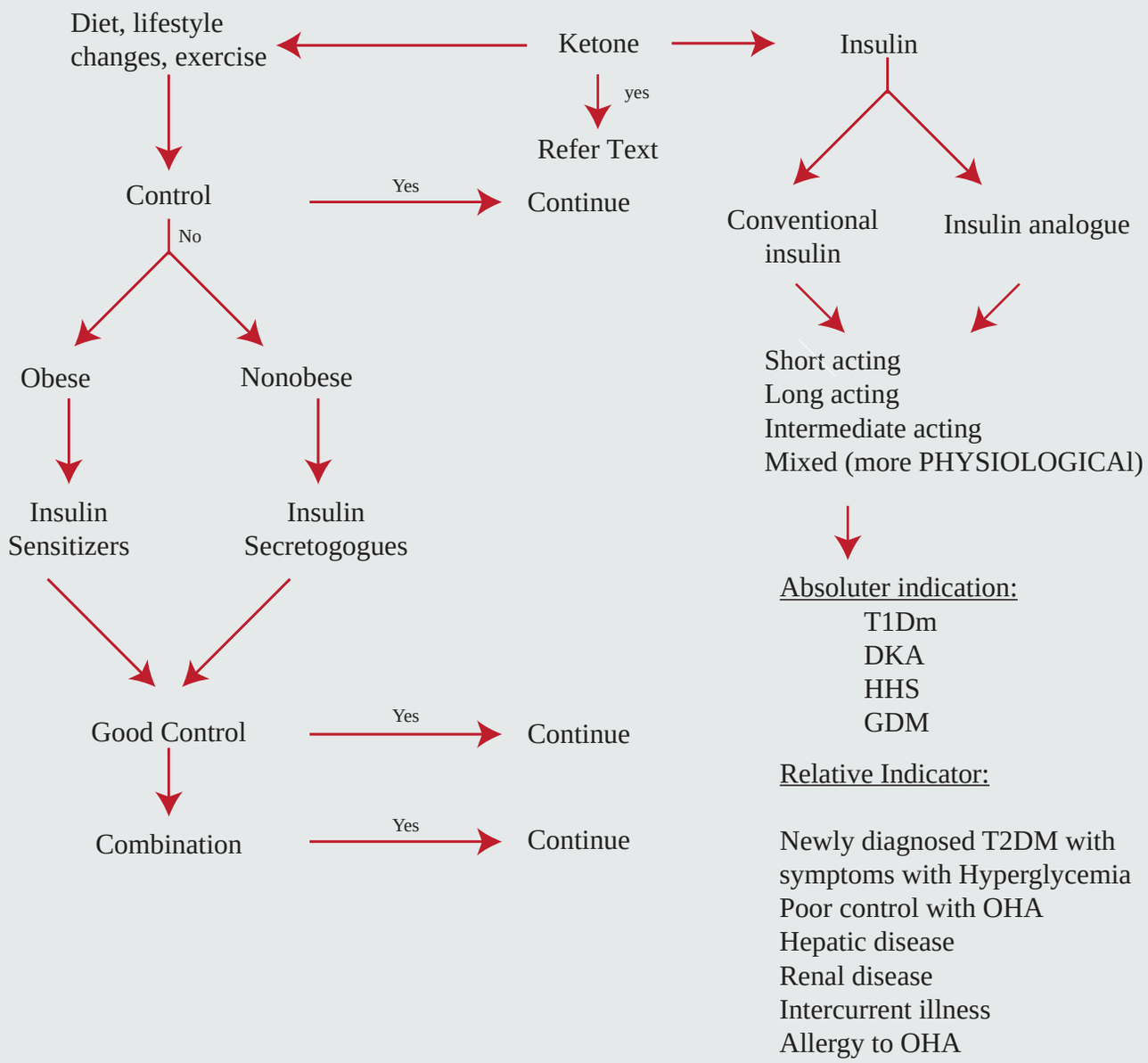
- Giddiness or Dizziness
- Pruritis vulvae or Vaginal candidiasis
- Delayed wound healing
- Burning sensation of feet
- Extreme fatiguability
- Can present with acute or chronic complications of Diabetes mellitus

Classification of Diabetes mellitus:

1. Type 1 Diabetes mellitus – B cell destruction; absolute insulin deficiency
2. Type 2 Diabetes mellitus – Insulin Resistance / Relative Insulin deficiency
3. Other Specific Types:
   * Genetic defects of B cell function – Maturity Onset Diabetes in Young (MODY)
   * Genetic defects of insulin action.
   * Diseases of exocrine pancreas –
     » Trauma
     » Pancreatitis
     » Pancreatectomy
     » Cystic fibrosis
     » Fibrocalculous Pancreatopathy
     » Haemochromotosis.
   * Endocrinopathies.
   * Drug induced or chemical induced –
   * Pentamidine,
   * Nicotinic acid,
   * Glucocorticoids,
   * Diazoxide,
   * Thiazide diuretics.
   * Infections etc.,
4. Gestational Diabetes mellitus (GDM)

Onset / Recognition of glucose intolerance in pregnancy

APPROACH TO A NEWLY DIAGNOSED DIABETIC



Investigations

Diagnosis of Diabetes mellitus: (at least one)

- Symptoms of Diabetes mellitus and Random Blood Sugar > 200 mg% (mg / dl)
- Fasting blood sugar > 126 mg % on more than one occasion
- 2 hours Plasma glucose > 200 mg % during oral glucose tolerance test with glucose – 75g Glucose (or) 1.75gm / kg glucose in children.

Diagnostic values	
Diabetes Mellitus	Fasting blood sugar > 126 mg % Post-prandial blood sugar >200mg%
Impaired Fasting Glucose	Fasting blood sugar> 100 – 125 mg %
Impaired Glucose Tolerance	Post-prandial blood sugar> 140 – 200 mg%

Treatment

Type 1 Diabetes mellitus:

- Strict meal plan
  - * Carbohydrate: 50 - 60%
  - * Protein: 10 – 20%
  - * Fat: 30% (If patient is dyslipidemic, fat should be 15%)
  - * Caloric intake: 30Kcal / kg
- Physical exercise
- Only insulin

Type 2 Diabetes mellitus:

- Strict meal plan
- Physical exercise
- Oral hypoglycemic agents
- Insulin

Oral hypoglycemic agents

Classification:

1. Sulphonyl ureas
2. B-iguanides
3. Sulphonylureas
   - » Glibenclamide
   - » Glipizide
4. Biguanides
   - * Metformin

DRUG	DOSE mg / day	t 1/2	SIDE EFFECTS
Insulin secretogogue (sulfonylureas)			
Glibenclamide	1.25-20	10 hours	Hypoglycemia, weight gain.
Glipizide	2.5-25	2-4 hours	
Glimepride	1-8	9 hours	
Insulin sensitizer (Biguanides)			
Metformin	500-2000mg	1.5 - 4.9 hours	Nausea, Vomiting, GI disturbances.
Newer Drugs (Thiazolidinediones)			
Pioglitazone	15-45mg	16-24 hours	Anaemia, weight gain, fluid retention, elevated liver function tests.

Contraindications for sulphonyl urea therapy

1. Insulin dependent diabetes mellitus (IDDM)
2. Pregnancy
3. Patients with severe infections
4. Allergic reactions
5. Significant liver and kidney disease
6. Patients undergoing surgery

Bi-guanides

Mechanism of action of Bi-guanides

- * Increases insulin sensitivity
- * Increases peripheral glucose uptake
- * Decreases hepatic gluconeogenesis
- * Inhibition of glucose absorption in intestines

Contra-indications to Bi-guanide therapy:

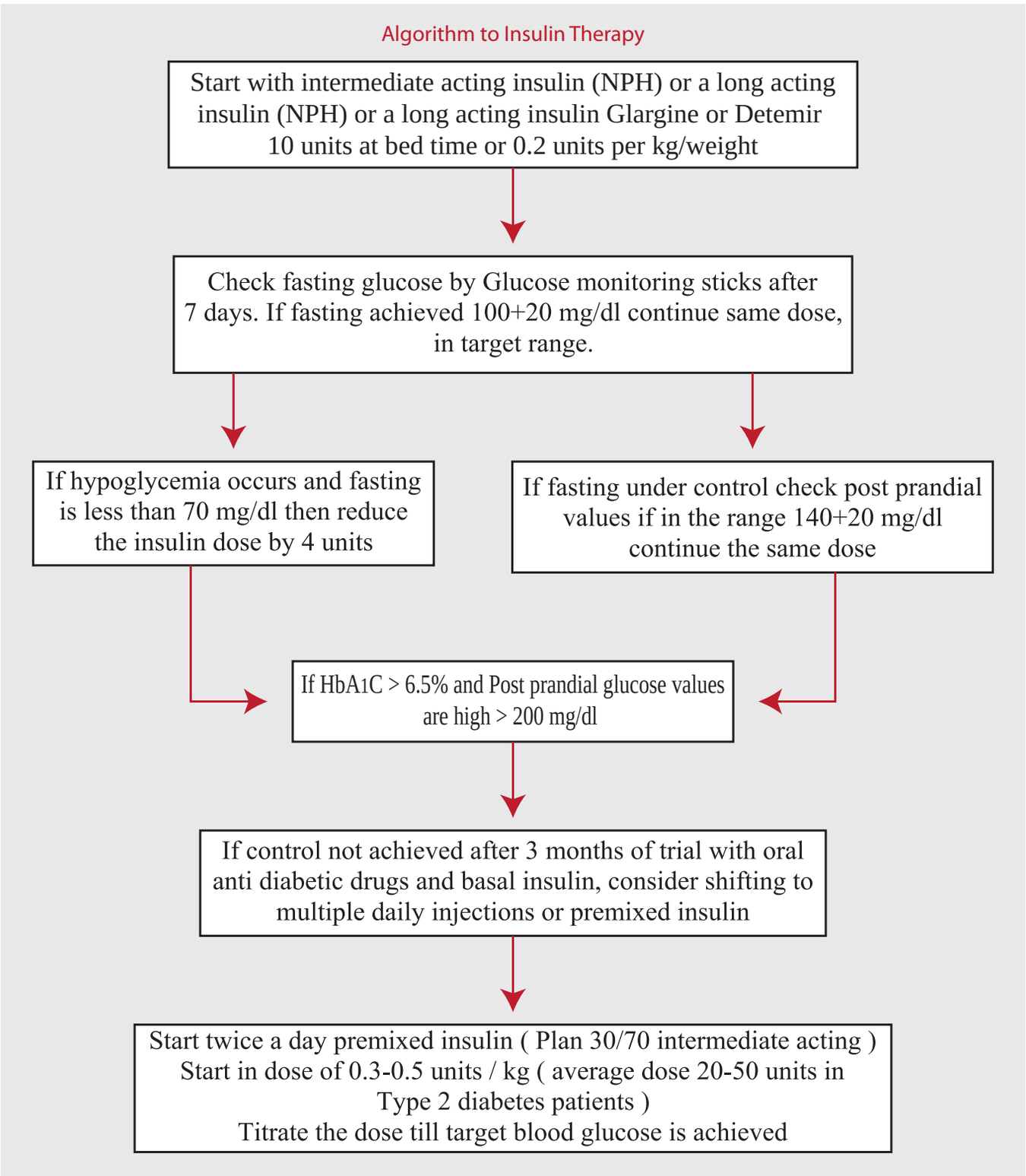
1. Renal failure when creatine clearance < 40 ml / min
2. Arteriography or intravenous urography – as intravenous iodinated products may precipitate lactic acidosis on patients with bi-guanides
3. Advanced liver cell failure

4. Alcoholism
5. Cardiac diseases
6. Diabetes with significant acute and late complications
7. Pregnancy
8. Old age > 70 years

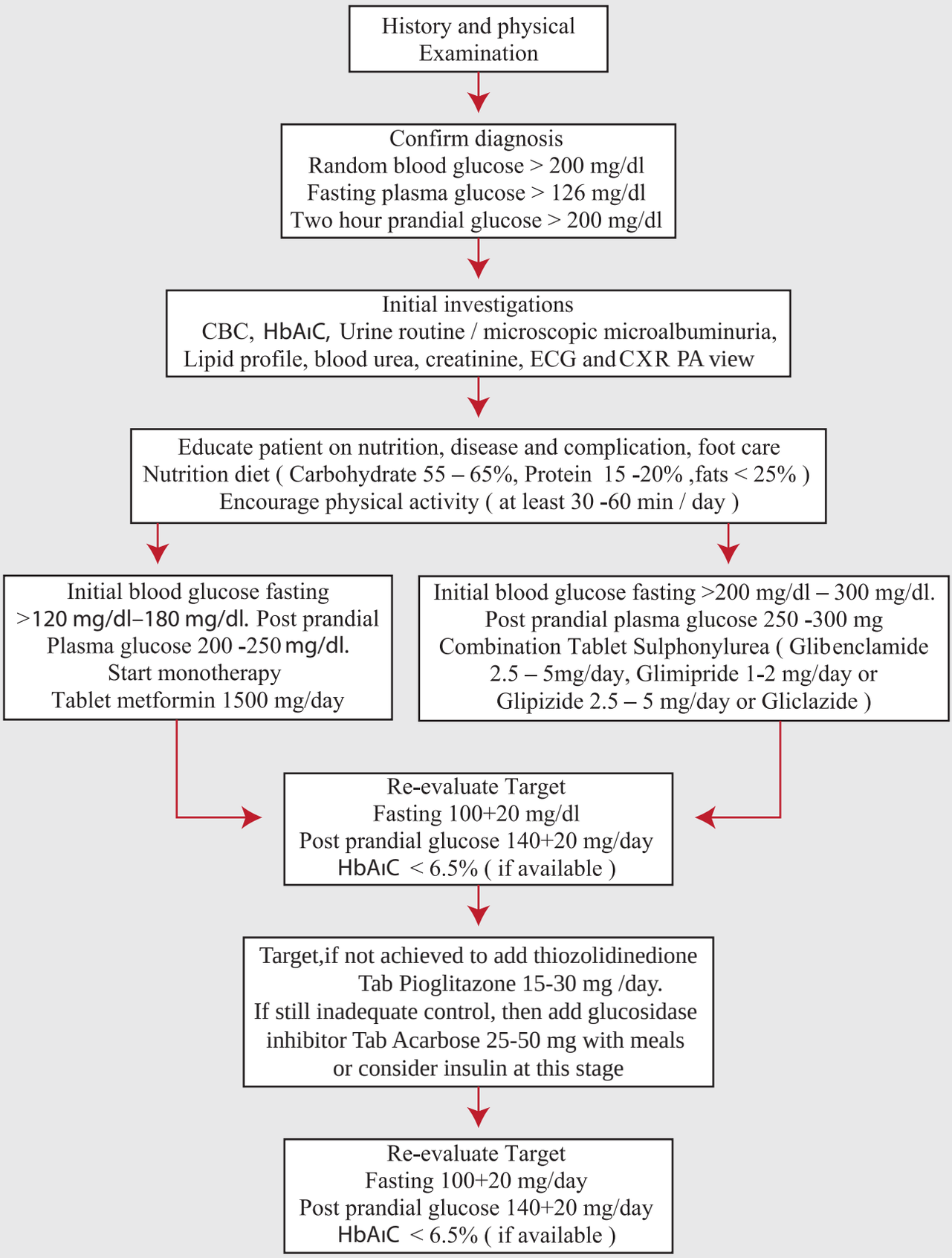
Consider insulin as initial therapy in patients with:

toxicity to islet cells, improve insulin secretion and possibly make oral hypoglycemic agents more effective.

- Lean patients or those with severe weight loss.
- Underlying renal or hepatic disease.
- Hospitalized or acutely ill patients.
- If response to oral hypoglycemics is not adequate. Consider insulin as initial therapy.



Algorithm for treatment of patients of Type 2 diabetes



Medical Nutrition Therapy (MNT)

A non-pharmacological mode of management of diabetes. Medical Nutrition therapy is individualized and should be a tailor made regimen. It is used as a compliment for an oral glucose lowering agent / insulin therapy.

Energy Recommendations:

- This depends on body weight and physical activity.
  - 20 kcal/kg Ideal body weight – Sedentary worker
  - 30 kcal/kg Ideal body weight – Moderate worker
  - 40 kcal/kg Ideal body weight – For heavy worker

In obese people

- Reduce 500 kcal from the calculated energy requirement

For under weight

- Add 500 kcal to the calculated energy requirement
  - Of the total kcal, 45-65 % kcal from carbohydrate and 10-25 % kcal from proteins

Fat recommendation :

- 500ml / month of a blend of oils / individual
- Gingelly oil and any refined vegetable oil could be used (or)
- Rice brand oil and any refined vegetable oil

Fibre recommendation

- 14gm/1000kcal provided.

Fluid recommendation:

- 8 to 10 glasses / day of water except in LVF, CKD, Cirrhosis etc.

MNT in Gestational Diabetes Mellitus:

- 30 kcal / kg Instant Body weight in first trimester
- 30 kcal / kg Instant Body weight + 300 kcal/day in IInd and IIIrd trimester
- Protein - 1gm/kg Instant Body weight + 10gm daily throughout pregnancy
- Avoid hypocaloric diets in obese GDM
- Provide compulsory bed time and evening snack to avoid accelerated starvation and nocturnal hypoglycemia

Gestational Diabetes mellitus:

- Strict meal plan
- Physical exercise
- Insulin

Other specific types

- Strict meal plan
- Physical exercise
- Insulin with or without Oral hypoglycemic agents

Gestational Diabetes mellitus

Definition

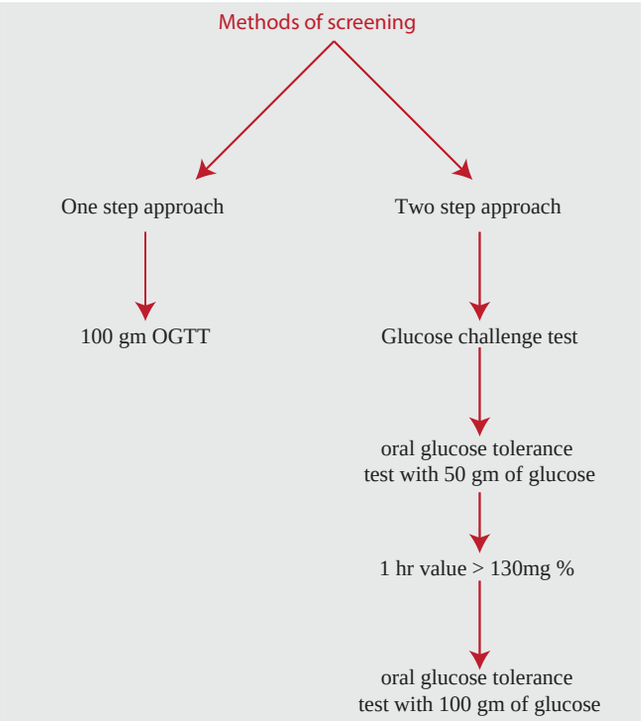
Gestational diabetes mellitus is defined as carbohydrate intolerance of variable severity with onset or first recognition during the present pregnancy.

Risk for gestational diabetes mellitus:

- Age more than 25 years
- Family history of diabetes mellitus
- History of unexplained fetal loss
- History of baby being large for gestational age
- History of congenitally malformed infant
- Maternal obesity
- History of Polycystic ovarian disease
- Polyhydramnios
- Pre-eclampsia
- Unexplained intrauterine death

Methods of screening;

- Spot test: Fasting <90 mg% [normal 2 hr postprandial <120 mg% random <105 mg% values] ADA recommends



Diagnostic Criteria:

Carpentar Coustan (with 100 gm)

F	-	95 mg %
1 hr	-	180 mg %
2 hr	-	155 mg %
3 hr	-	140 mg %

WHO criteria (with 75 gm of glucose)

F	-	95 mg %
1 hr	-	180 mg %
2 hr	-	155 mg %

If any two values equals or crosses normal value, it is termed as **Gestational Diabetes mellitus.**

Important Note

OGTT value should never be treated.

Treatment

1. Medical Nutrition Therapy [refer MNT]
2. Insulin is essential if MNT fails to achieve euglycemia

Target Glycemic Level

Fasting glusose	–	90 mg %
2 hr postprandial	–	120 mg %
Mean glucose	–	105 mg %

Monitoring Glycemic Control

- Blood glucose fasting and postprandial every three days till glycemia is achieved; then every fortnightly, throughout first and second trimester.
- Every week in third trimester.
- Glycemic profile monitoring once in 1st and 2nd trimester and then every month in last trimester.

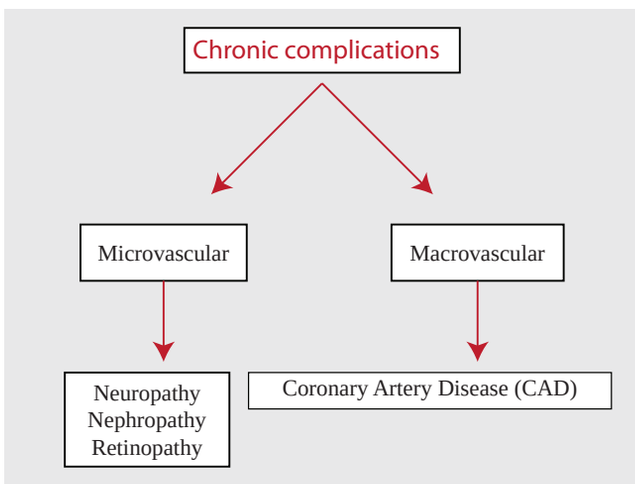
Complications of Diabetes mellitus

Acute complications:

- Hypoglycemia
- Diabetic Keto Acidosis (DKA)
- Diabetes and infections
- Hyperosmolar Hyperglycemic State (HHKNC)

Chronic complications:

Follow the flow chart below



Hypoglycemia

Definition

Hypoglycemia is a clinical emergency occurring in diabetes characterized by either autonomic or neuroglycopenic symptoms (or) biochemically random blood sugar < 70mg, due to antidiabetic agent, food and activity mismatch.

Symptoms

Autonomic symptoms
• Tremor
• Palpitation
• Sweating
• Sensation of hunger
Neuroglycopenic symptoms
• Head ache
• Fatigue
• Irritability
• Disturbed vision
• Mental confusion
• Personality changes
• Convulsion,night mares
• Coma
Hypoglycemic symptoms in children
• Restlessness, rolling (or) frequent falling from the bed
• Sudden changes in behavior
• Irritability
• Crying.

Treatment

- Draw blood sample immediately
- Dextrose supplementation
  - * Conscious**:** Oral Glucose, Sugar, Fruit Juice
  - * Unconscious**:** 50% Dextrose 100ml IV Stat. Followed by 10% Dextrose then by 5% DNS Maintenance (or) Inj. Glucagon 1mg im if not accessible to intravenous route
- Patient still remains unconscious
  - * To rule out cerebral edema
  - * If present IV mannitol + Inj. Dexamethasone 8mg IV
- Stop the antidiabetic agents for 3 days in Type 2 Diabetic mellitus patients and recheck blood sugars. In Type 1 Diabetic mellitus patients recheck blood sugars after 6 hrs and adjust insulin dose accordingly.
- Identify the cause of hypoglycemia
- If recurrent hypoglycemia, rule out
  - * Renal function disorder
  - * Liver function disorder
- Repeat blood sugar value after hypoglycemia correction and monitor blood sugars

Referral:

If the patient remains unconscious even after dextrose administration refer the patient immediately to higher centre for further evaluation.

Important Note

Patient Education:

- Educate patient and his family members about low blood sugars and symptoms
- Never miss a meal after insulin / Oral hypoglycemic agents
- Be cautious of unaccustomed physical activity
- To carry diabetic identity card
- Always carry simple sugar (biscuits and toffee) to avoid low sugars.

Diabetic Ketoacidosis (DKA)

Symptoms

Suspect DKA in a diabetic patient when he has

- Altered sensorium
- Unexplained abdominal pain
- Nausea
- Vomiting
- Breathlessness
- Increased respiratory rate (Kussmaul's breathing)
- Signs of dehydration.
- Thirst
- Polyuria

Investigations

- Blood glucose [usually >250 mg %]
- Blood Urea [may or may not be ↑]
- Serum Creatinine [may or may not be ↑]
- Serum electrocyte [Na ↑ or ↓, K ↑ or ↓]
- Serum Bicarbonate < 10 mmol / l
- Urine sugar [positive]
- Urine Acetone [positive]
- Chest X-ray

- ECG
- Ultrasonogram abdomen / KUB

Treatment

Rehydration with normal saline	
Hours	Volume
1st half hour – 1 hour	1 L
2nd hr	1 L
3rd hr	500 ml-1L
4th hr	500 ml-1L
5th hr	500 ml-1L
Total 1st - 5th hr	3.5-5 L
6th – 12th hr	250-500 ml/hr
NS for first 4 hrs Consider half NS thereafter	

May need to adjust type and rate of fluid administration in the elderly and in patients with cardiac and renal compromise

Insulin infusion

Start regular insulin infusion at 5 units/hr and titrate infusion depending on blood sugars.

Electrolyte imbalance

- If potassium > 5.5, and if patient is anuric – no potassium infusion
- Avoid rapid correction of
  - Dehydration
  - Hyperglycemia and
  - Electrolyte imbalance.

Referral:

- Refer the patient to a higher centre if:
  - Patient is comatose
  - Hypotension requiring ionotropic support
  - Anuric
  - Elevated renal parameters
  - Evidence of septicemia

Diabetes and Infections

Both diabetes and its complications predispose to infections.

Causes

Contributing factor	Mechanism	Effect
Hyperglycemia	Alters leucocyte functions Impairs phagocytosis Defective leukocyte adherence	Predisposes to infection
Neuropathy	Foot trauma	Foot infection
Autonomic-neuropathy	Bladder dysfunction Reduced sweating	UTI Infections Dry skin

Infection and Organisms

Hyperglycemia ketosis syndrome

Secondary infections in a diabetic can aggravate hyperglycemia and leading to ketosis analysis.

Hyperglycemic hyperosmolar non-ketotic coma (HHNKC)

Definition

It is an acute metabolic complication in middle aged and elderly diabetics with high morbidity and mortality.

Causes

Precipated by

- Infection
- Trauma
- Burns
- Infarction
- Hyper-alimentation
- Drugs like
  - Thiazide
  - Cimetidine
  - Phenytoin and
  - Parentral diuretics

Symptoms and signs

- Polyuria
- Polydipsia
- Severe hyperglycemia (Blood sugar > 600 mg%)
- Profound dehydration
- Elevated osmolality
- Hemianopia, muscle fasciculation, seizures

- Altered sensorium, coma

Treatment

1. Fluid replacement: ½ normal saline at the rate of 2 litre in 1st 2 hours and 1 litre in another 2 hours
2. Low dose insulin.
3. Correction of electrolytes and hyperosmolality
4. Low-dose heparin to prevent vascular thrombosis intravascular coagulation.

Distinguishing features between DKA and HHNKC

Criteria	DKA	HHNKC
Age	Younger	Older
Respiration	Hyperventilation, deep	Normal, shallow
Dehydration	Around 10 %	Around 25 %
Consciousness	Diminished	Comatose
Temperature	Normal or low	May be raised
Blood glucose	>300 mg/dl	> 600 mg/dl
Blood urea	42 – 70 mg/dl	60 – 180 mg/dl
Sodium	125 – 140 mmol/l	130 – 155 mmol/l
Pottassium	3 – 6.5 mmol/l	3 – 5 mmol/l
Bicarbonate	< 14 mmol/l	16 – 30 mmol/l
Ketones	+ + To + + +	0 to +

Infection	Organisms	Treatment
Skin and soft tissue infection Carbuncle Necrotising cellulitis	Staphlococcus Streptococcus Gram negative clostridium	Appropriate antibiotics
Eye Stye Blepharits ,dacrocystitis	Streptococcus	Appropriate antibiotics
Dental sepsis Gingivitis Pyorrhea	Anaerobes	Appropriate antibiotics
Ear Malignant Otitis externa	Pseudomonas	Ceftriaxone, Ticarcilin
Nose Mucor mycosis	Rhinocera Fungus	Prognosis bad
Lung Pulmonary TB, Bacterial pneumonia	Mycobacterium	Anti-tuberculosis treatment
Gall Bladder Emphysematous cholecystitis	Clostritidia	Appropriate antibiotics
UTI Asymptomatic bacteriuria Emphysematous pyelonephritis Emphysematous cystitis Balanoposthitis Vulvo vaginitis	E Coli Candida	Appropriate antibiotics Topical Clotrimazole and oral anti fungals if necessary

Micro- vascular complications

Diabetic foot

Causes

Mostly **polymicrobial** infection

Bacteroids are the commonest group

Neuropathy and ischemia lead to DFS

Investigations

1. Blood routine and urine routine
2. Urine culture sensitivity
3. Blood culture sensitivity

Treatment

1. Impatient care
2. Insulin is the treatment of choice except for mild infections
3. Mild infections can be managed with OGLA
4. Always suspect Tuberculosis since Tuberculosis and diabetes are concomitant infections

Diabetic retinopathy

1. Definition and Classification
2. Screening
3. General recommendations
4. Treatment

Definition

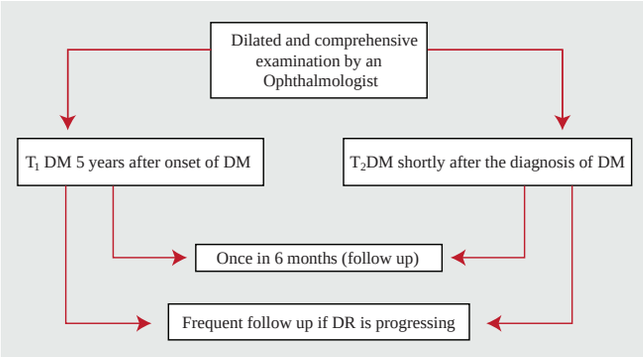
Diabetic retinopathy is a micro vascular complication of diabetes affecting the vascularity of the eye. (Retina)

Classification of Diabetic retinopathy (DR)

Grade	Symptoms
1. Non Proliferative diabetic retinopathy (NPDR)	
a. None	Normal retina
b. Early NPDR	Micro Aneurysms (MA) only
c. Mild NPDR	MA (+) Retinal haemorrhages and / or Hard exudates (HE) (Extra macular i.e > 1 DD from fovea)
d. Moderate NPDR	Mild NPDR (+) Haemorrhages and or Cotton wool spots (>5) and / or
e. Severe NPDR ('4/2/1' rule)	Venous beading / looping (or) IRMA* in one field Moderate NPDR (+) MA / Hages in 4 (or) more fields (OR)
f. Very severe NPDR	Venous beading / looping in 2 (or) more fields (OR) IRMA * in one quadrant
2. Proliferative Diabetic retinopathy (PDR)	Any two (or) more of the 'severe' category
* PDR without high risk changes (HRC)	NVE** or NVD*** <1/2 DD
* PDR with HRC	NVE or NVD> ½ DD Pre-retinal and / or vitreous haemorrhages

*IRMA – Intra Retinal Micro Vascular Abnormalities
**NVE – New Vessel Elsewhere ***NVD- New Vessel Disc.

Screening



- DR is a highly specific vascular complication of both T1 DM and T2 DM patients
- The prevalence of retinopathy is strongly related to the duration of diabetes.
- DR is the most frequent cause of blindness in adults.

DR and Pregnancy

- Eye examination: Once in every trimester, 6 wks. after delivery, upto one year
- Patients should be counseled on the risk of development and / or progression of DR.

General Recommendations

Glycemic control:

- Optimal glycemic control
  - * Reduced risk and progression of DR.
- Reduction of HbA1c by 1%
  - * Reduces the relative risk of development of micro vascular complications by 37%
- Optimal Blood Pressure Control:
  - * Reduced risk and progression of DR.
- Diabetic Nephropathy is associated with DR.

Treatment

- Laser Therapy
  - * Can reduce the risk of vision loss in patients with high risk changes.
- Prompt referral of patients. with any level of macular oedma, severe NPDR or any PDR
- Indications for laser therapy
  - * Clinically Significant Macular Oedma (CSME)
  - * Mild (or) Moderate NPDR
  - * Severe NPDR / PDR with out HRC
  - * PDR with HRC
  - * NVD (or) NVE with Pre retinal haemorrhage and / or Vitreous haemorrhage .

Diabetic nephropathy

Definition

Diabetic Nephropathy is defined as progressive increase in urine albumin, accompanied by rising blood pressure and declining GFR resulting in end stage renal disease associated with retinopathy and increased cardio vascular risk.

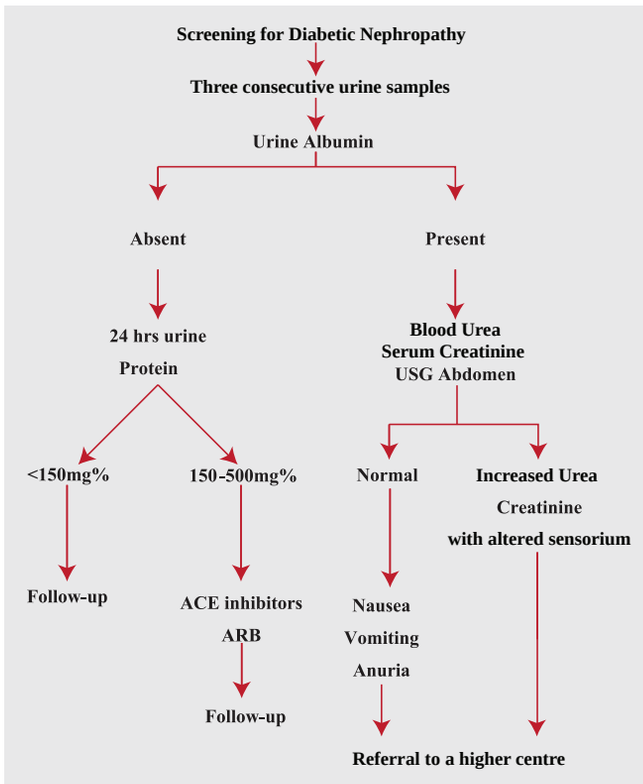
Symptoms

- Puffiness of face
- Swelling of legs
- Abdominal distention
- Reduced urine output.

Signs :

- Bilateral. pedal edema
- Renal angle tenderness
- Abdominal bruit

Investigations



Diabetic Neuropathy

Introduction:

Diabetic Neuropathy is the most common complication of Diabetes mellitus.

- It is a micro-vascular complication
  - * Endo-neural hypoxia due to involvement of micro vascular disease and vasa nervorum involvement.
  - * Hyperglycemia plays a dominant role in the pathogenesis of diabetic Neuropathy.

Polyneuropathy	Mononeuropathy	Autonomic neuropathy
• Sensory • Proximal • Truncal • Motor	• Isolated • Cranial • Truncal • Multiple	• Parasympathetic • Sympathetic

Symptoms

1. Burning, shooting, stabbing pain of both feet
2. "Pins and needles" sensation
3. Absent sensation to several modalities
4. Numbness of both feet
5. Depressed reflexes
6. Muscle paralysis

Clinical Features

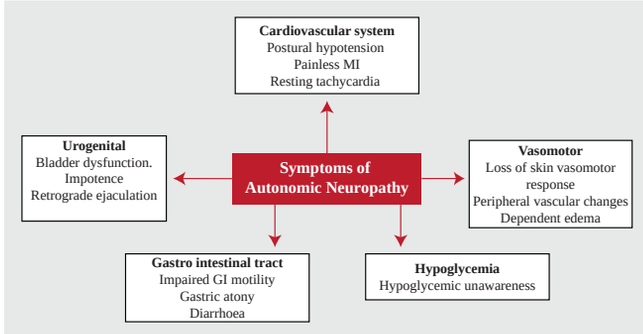
Types:

- Large fibre type
- Small fibre type

Large fibre type
• Unsteady gait • Absent reflexes • Reduced vibration / position sense • Mimics posterior column lesion
Small fibre type
• Pain predominates • Variable reflexes • Variable position / vibration sense

Symptoms

Diabetic Autonomic Neuropathy (AN)



Treatment

1. Strict glycemic control
2. Most of the following drugs offer only symptomatic relief but don't essentially reverse the disease process
   - * Tricyclic antidepressants
   - * Anticonvulsants
   - * Anti-oxidants
   - * Topical Capsaicin
   - * Aldose reductase inhibitors
   - * Methylcobalamine
   - * Pre-gabalin

Referral

Patients with severe sensory motor neuropathy with autonomic neuropathy need referral to a higher centre for further evaluation.

Macrovascular complications in Diabetes mellitus

Diabetes mellitus and heart:

Introduction :

Heart is involved in many ways in diabetic patients and they are

1. Coronary artery disease (CAD)
2. Cardiomyopathy
3. Cardiac autonomic neuropathy
4. Heart failure

Coronary Artery Disease (CAD)

It is a major macrovascular complication seen in diabetics. There is a two to four fold increased risk of CAD in diabetic patients.

Symptoms

- They can present with
  - * Acute coronary syndromes
  - * Classical angina
  - * Silent ischaemia.
- Silent ischaemia and infarction are very common in diabetics due to cardiovascular autonomic neuropathy.
- Atypical symptoms such as
  - * Confusion
  - * Dyspnoea
  - * Restlessness
  - * Unexplained fatigue
  - * Sweating should alert the clinician to rule out silent infarction.
- Diabetes is a risk factor for cardiogenic shock and acute left ventricular failure in the setting of ACS.

Investigations

ECG, ECHO, TMT and advanced investigation if required.

Treatment

- Admission may be required based on the clinical status and ECG changes of the patient.
- It is safe to switch over to Insulin therapy
- Drugs
  - * Aspirin
  - * Cardio Selective beta blockers
  - * ACE- Inhibitors
  - * Statins.

Referral:

Patient may be stabilised and referred to a higher centre for speciality care.

Prognosis

It depends on periodical monitoring and regular follow up:

- Type I Diabetes mellitus:
  - * With good glycemic control screen for complications 5 yrs after diagnosis
- Type 2 Diabetes mellitus:
  - * Screen for complications at the time of detection and yearly afterward.
- Gestational Diabetes mellitus:
  - * Good prognosis for both mother and child if mother achieves and maintains good glycemic control
- Specific types
  - * Depends on etiological factor

Note:

- All patients should be given chronic follow up card/book
- Patient who need to be started on insulin therapy should be taught for insulin injection technique

Hypothyroidism

Causes

Hypothyroidism may be primary; common causes of which are autoimmune, iatrogenic due to I^{131}, antithyroid or lithium treatment and thyroidectomy, or secondary to pituitary or hypothalamic disease.

Symptoms

- Coarse dry skin
- Hoarseness of voice
- Facial puffiness, weight gain
- Cardiac enlargement and/or pericardial effusion,
- Goiter with or without prolonged relaxation phase of deep tendon reflexes.
- **Myxedema coma** is a rare complication of severe hypothyroidism with hypothermia, hypoventilation, hyponatremia, hypoxia, hypercapnia and hypotension.

Investigations

- Diagnosis is confirmed by
- Low serum free T_3 and T_4
- serum TSH raised in thyroid types and low in supra-thyroid types
- Thyro-peroxidase (TPO)
  - * Thyro-peroxidase antibodies are seen in 90-95 % of patients presenting with autoimmune hypothyroidism

Treatment

- Pharmacological
  * Tab. L-Thyroxine 50 – 100mcg/day
  * Dose to be adjusted based on TSH levels
  * Goal is normal TSH (lower half of reference range
  * Measure TSH levels after about 2 months of instituting therapy
  * Adjust by 12.5 or 25 mcg increments if TSH is high; decrement of the same if TSH is suppressed.
  * When full replacement is achieved then follow up measurement at annual intervals and later by a 2-3 year interval
  * Ensure ongoing compliance.

Special treatment considerations

- A hypothyroid woman should be euthyroid prior to conception and during early pregnancy (effect on foetal neuronal development)
- Elderly require less Thyroxine (less by upto 20%) especially those with coronary artery disease, starting dose 12.5mcg/day with similar increments every 2- 3 months until TSH level is normalized.
- In hypothyroidism due to low TSH (supra-thyroid cause is suspected) detailed investigations are required and patient should be referred to a tertiary care level
- Asses the response clinically and by serum TSH (serum T_3 in suprathyroid type) at 8 weekly intervals
- Once euthyroid state is restored, follow-up at 6-12 monthly intervals.

Treatment of Myxedema coma

- Warm blankets, mechanical ventilation for respiratory failure.
- Correction of metabolic disturbances and treat precipitating factors.
- Drugs
  * L-Thyroxine 500 mcg IV bolus, then 50-100mcg IV daily
  * If intravenous preparation not available, the same dose is administered through Ryle's tube.
  * Once acute phase is over, maintain L-Thyroxine as above.
  * Inj.Hydrocortisone 100 mg IV stat, 25-50 mg 8 hourly.
- **Caution**: Avoid sedatives

Patient Education

- L-Thyroxine should be taken as a single daily dose, ideally on awakening, at least 30 minites before breakfast.
- Fibre and bran products (e.g., Isapghola husk) may impair absorption, as also cholestyramine, cholestipol, iron sulphate, sucralfate, aluminium hydroxide
- Metabolism of L-Thyroxine is increased by Phenytoin, Rifampicin, and carbamazepine.
- **Explain to the patient that the treatment is life long.** Do not modify dose or stop treatment without consultation.
- Over treatment may lead to decreased bone mineral density and adverse cardiac complications.

References:

1. An update on management of hypothyroidism and Hyperthyroidism. Arch Intern Medicine 2000; 160: 1067 – 1071
2. Disorders of Thyroid Gland. . In: Harrison's principles of medicine.
   Kasper DL, Braunwald E, Fauci AS et al (eds), 16th edition, 2005, McGraw-Hill Company Inc., New York, pp 2104-2127.

Hyperthyroidism

Causes

Classically occurs in Grave's disease, which is characterized by diffuse goiter, Opthalmopathy and Dermopathy in varying combinations. Other important causes are Toxic Multinodular Goiter (TMN) and toxic adenomas.

Symptoms

- Sweating
- Tremors
- Wide pulse pressure
- Sinus tachycardia and atrial arrhythmias

- Worsening of angina or cardiac failure may predominate in older patients
- Graves' disease
  * Goiter
  * Opthalmopathy and
  * Dermopathy

Investigations

Diagnosis is confirmed by low to undetectable serum TSH and increased Serum free (FT_3) and free (FT_4)

Treatment

Pharmacological

- Adjunctive treatment
  * For adrenergic symptoms such as sweating, tremor and tachycardia. Tab. Propranalol 40 – 120 mg a day.
- Tab. Propylthiouracil 100–150 mg every 6–8 hours or Tab. Carbimazole 10 – 20 mg every 8 – 12 hours;
- After euthyroid state is achieved in 6 – 8 weeks once daily dose possible.
- Review with serum TSH and FT_3 after 3 – 4 weeks treatment has been initiated
- Once controlled reduce to the smallest effective dose or continue initial dose combined with L-Thyroxine
- Drugs are given for an average of 2 years.

Definitive treatment is surgery / ablation of thyroid tissue

- Subtotal thyroidectomy in younger patients (<30 years) in whom antithyroid therapy has been unsuccessful and in very large goiters.
- Radioactive iodine (I^{131}):
- Method of choice in
  * Elderly
  * Younger patients who have completed family with recurrent thyrotoxicosis following surgery or when surgery is refused or contraindicated.

Caution

Radioactive iodine should never be given in pregnancy. In woman of childbearing age if radioactive iodine is planned, a pregnancy test should always be carried out.

Pregnancy

- In pregnant woman surgery should not be performed in 1st or 3rd trimesters
- Antithyroid drugs are less risky but may induce hypothyroidism in the foetus and should be used in the smallest necessary dose to keep serum TSH and FT_4 in normal range.
- Propylthiourcil is preferred – usual maintenance is 200 mg/day. If > 300 mg/day required during 1st trimester, Subtotal thyroidectomy is indicated in 2nd trimester
- Propranalol should be avoided as it can cause foetal growth retardation and neonatal respiratory depression.

Ophthalmopathy

Refer to ophthalmologist.

Initiate therapy in mild cases with elevation of head at night, diuretics to decrease edema, use of tinted sunglasses and 1 % methyl cellulose eye drops to prevent drying and refer patients with severe and progressive exophthalmos to an ophthalmologist.

Toxic multinodular goiter

- Radioactive iodine is the treatment of choice.
- Large doses are usually required
- Treatment with antithyroid drugs given till patient is euthyroid.
- Propranalol may be useful before and after radioactive iodine administration.

Thyrotoxic crisis or thyroid storm

- Refer to a tertiary care centre.
- Life threatening hyperthyroidism with fever, vomiting, diarrhoea, jaundice, delirium and coma;
- Usually precipitated by acute illness such as stroke, infection, diabetic ketoacidosis, trauma, patients undergoing surgery or radioactive iodine treatment in a poorly prepared patient:

Treatment

- Tab. Propylthiourcil 600 mg loading dose, then 200 – 300 mg every 6 hours orally or through Ryle's tube.

  O r

  Tab. Carbimazole 15 – 25 mg 6 hourly.
- 1 hour after the 1st dose of antithy-

roid drug, saturated solution of Potassium iodide (SSKI) 5 drops every 5 hours. O r Lugol's iodine 10 drops 3 times a day. O r Sodium iodide 1 g IV slowly.

- Tab. Propranalol 40 – 60 mg 4 hourly or 0.5 – 2 mg IV every 4 hours.
- Inj. Dexamethasone 2 mg IV 6 hourly
- Continue iodides and dexamethasone until normal metabolic stage is achieved and give supportive treatment such as cooling, antipyretics, antibiotics for infection, intravenous fluids, etc.
- Once euthyroid status is achieved, manage as already outlined.

Patient education

- If fever or sore throat develops on antithyroid drugs complete blood count should be done; discontinue if polymorphs count is <1500/mm^3
- If allergic rash or drug hypersensitivity develops, give antihistamines and preferably change to another drug
- **If agranulocytosis, hepatitis, drug fever, arthralgias develop, preferably stop antithyroid treatment.**
- Iodide - useful in impending Thyrotoxic crisis and patients with severe cardiac disease; must be used only after following antithyroid drugs.

References

- An update on management of hypothyroidism and Hyperthyroidism. Arch Intern Medicine 2000; 160: 1067 – 1071
- Disorders of Thyroid Gland. . In: Harrison's principles of medicine. Kasper DL, Braunwald E, Fauci AS et al (eds), 16th edition, 2005, McGraw-Hill Company Inc., New York, pp 2104-2127.

Hypocalcemia

Causes

Hypocalcemia may be caused by hypoparathyroidism, pseudo hypoparathyroidism, vitamin D deficiency states, chronic renal failure, mala-bsorption syndrome and hypomagnesaemia.

Symptoms

- Circumoral parasthesias
- Muscle cramps
- Confusion
- Tetany
- Convulsion
- Positive Chovstek's and Trousseau's sign

Investigations

- ECG may reveal prolongation of QT interval.
- Total serum calcium <8.5 mg/dl
- In hypoalbuminemia, add 1 mg/dl of calcium to the estimated level for every 1 g fall of albumin below 4 g/dl (corrected serum calcium).

Treatment

Severe symptomatic hypocalcemia

- 20 ml of 10 % calcium gluconate solution over 10 – 15 mins followed by 60 – 80 ml of 10 % solution in 1 L of 5 % distilled water (0.5 – 0.25 mg/kg/hour elemental calcium)
- **Caution:** should not be mixed with bicarbonate solution as it may result in precipitation of calcium carbonate
- If associated with hypomagnesaemia
  - Inj. Magnesium sulphate 1 – 2 g IV day 1 followed by oral Magnesium oxide 600 – 1200 mg 3 times a day to replenish stores.

Asymptomatic Hypocalcemia/ maintenance treatment

- Treat the underlying cause if possible. Usually long-term treatment is required in conditions such as hypoparathyroidism, pseudohypoparathyroidism and chronic vitamin D deficiency states.

Treatment

- Tab. calcium carbonate (40 % elemental calcium by weight) 1 –2 g elemental calcium orally 3 times a day initially and subsequently maintenance dose of 0.4 – 1.5 g 3 times a day
- In chronic renal failure calcium alone gives inadequate results. However, correct concomitant hyper-phosphatemia before instituting following therapy: Vitamin D 5000 IU/day for 1 – 2 weeks, then weekly or bimonthly

- Calcitriol 1,25 (OH)2D3 0.25 mcg orally daily – more expensive but less toxic than vitamin D for hyperphosphatemia
- Advise low phosphate (low cereal) diet and phosphate binding agents e.g., aluminium hydroxide.

Patient education

- Side effects of oral calcium carbonate are dyspepsia and constipation.
- Absorption requires gastric acid and is impaired in achlorhydria or when acid suppression therapy is given.
- Serum calcium should be monitored frequently (daily in severe hypocalcemia, weekly with moderate hypocalcemia for first month) and maintained at 8.0 – 8.6 mg/dl, and PTH and 24 hour urinary calcium within 2 – 4 weeks of starting treatment.
- Once Serum calcium and urinary calcium is normal and PTH falls, maintenance treatment as described with reassessment at 3 monthly intervals.

References

- Diseases of Parathyroid gland and other hyper and hypo-calcemic disorders. In: Harrison's principles of medicine Kasper DL, Braunwald E, Fauci AS et al (eds), 16th edition, 2005, McGraw-Hill Company Inc., New York, pp 2249-2268.
- Calcium In: The Washington Manual of Medical Therapeutics, Ahya SN, Flood K. Paranjothi S eds., 30th edition, 2001. Philadelphia: Lippincott, Williams and Wilkins, pp-60-66

Hypercalcaemia

Causes

The common causes are hyperparathyroidism and malignancy; others include vitamin D action, high bone turnover or renal failure.

Symptoms

- Fatigue, depression, confusion, anorexia, nausea, vomiting, constipation, Polyuria,

Investigations

- short QT interval on ECG and occasionally cardiac arrhythmias. Generally symptoms appear when serum Ca >11.5-12.0 mg/dl; severe hypercalcemia >15 – 18 mg/dl can result in death.

Treatment

Treatment varies with severity; mild Hyper-calcemia can be treated with rehydration only, while severe Hyper-calcemia is treated as a medical emergency.

A. Mild

3. Rehydration – isotonic saline 2 – 4 L/day increases calcium excretion
4. After correcting dehydration administer Inj. Frusemide 20 – 40 m g 2 times a day.
5. Monitor electrolytes especially potassium and magnesium and replace accordingly.

B. Moderate to severe

1. Aggressive preceding approach - isotonic saline 6L Plus Inj. Frusemide upto 100 mg every 1 to 2 hours.
2. Inj. Pamidronate 30 – 90mg IV infusion in 0.9 % saline over 4 - 24 hours; response lasts for weeks.

   Or

   Tab. Aldronate Sodium 10 mg orally in the morning with full glass of water at least 30 mins before any food or drink; remain in upright position for at least 30 mins.

   Or

   Tab. Zolendronate 1 – 4 mg IV in few mins.
3. Inj. Calcitonin 2 – 8 U/kg IV or SC or IM every 6 – 12 ; rapid but mild action.
4. Tab. Prednisolone 40 – 100 mg/day in 4 divided doses may be useful in osteolytic malignancies, vitamin D intoxication and sarcoidosis.
5. Tab. Phosphorous (sodium and potassium phosphate) 1 – 1.5 g/day in 4 divided doses for several days when hypo-phosphatemia is present.
6. Peritoneal dialysis with calcium free dialysate is useful especially in cases complicated by renal failure.

Definitive treatment, wherever possible, is parathyroidectomy in hyperparathyroidism.

Patient education

- In patients with mild asymptomatic hyper-calcemia due to hyperparathyroidism, advise to keep active, avoid immobilization, drink adequate fluids and avoid thiazide diuretics, large doses of vitamin D or A and calcium supplements and cal-

cium containing antacids.

- Check serum calcium and albumin twice a year, renal function and urine calcium once a year and bone density of distal radius once every 2 years.

References:

- Diseases of Parathyroid gland and other Hyper and hypo-calcemic disorders. In: Harrison's principles of medicine Kasper DL, Braun wald E, Fauci AS et al (eds), 16th edition, 2005, Mc Graw-Hill Company Inc., New York, pp 2249-2268.
- Calcium In: The Washington Manual of Medical Therapeutics, Ahya SN, Flood K. Paranjothi S Eds. 30th edition, 2001. Philadelphia: Lippin-cott, Williams and Wilkins, pp-60-62

Rheumatology

Standard Treatment Guidelines
Tamil Nadu Health Systems Project

Chapter 10

- Clinical Approach to Arthritis
- Rheumatoid Arthritis
- Spondyloarthropathies
- Systemic Lupus Erythematosis
- Vasculitis
- Regional Syndromes
- Juvenile Idiopathic Arthritis

Clinical approach to arthritis

Introduction

Arthritis is defined as inflammation of joint or a group of conditions characterized by the presence of some or all of the cardinal signs of inflammation (pain, tenderness or swelling), systemic disturbances (such as prolonged morning stiffness or fever) and laboratory evidence of inflammation (raised ESR, elevated CRP or other acute phase reactants).

Stratification of Patients:

The patients with arthritis can be stratified in to certain broad groups by

- Duration of disease
  - * Acute or chronic arthritis, the dividing line between the two being duration of six weeks
- Age of disease onset
  - * Adult or juvenile arthritis, the latter having disease onset at age less than 16 years
- Number of joints involved
  - * Monoarthritis or oligoarthiritis (single joint)
  - * Pauciarthiritis (two to four joints)
  - * Polyarthritis (five or more joints)
- Presence of inflammation
  - * Inflammatory or non-inflammatory arthritis
- Presence of bilateral involvement
  - * If polyarthritis, symmetrical or asymmetrical

Chronic arthritis

Distinguishing features between various types of arthritis

The conditions that look like arthritis may actually be a manifestation of soft tissue rheumatism. So, it should be first ruled out before proceeding further.

Arthritis and soft tissue rheumatism (STR)

Characteristics	Arthritis	Soft tissue rheumatism
Pain	Deep, diffuse and circumferential	Superficial and sharply localized
Tenderness	Circumferential around the joint	Localized over the affected structure
Pain on active range of motion	Yes	Yes
Pain on passive range of motion	Yes	No
Clinical synovitis or effusion	Yes	No
Crepitus. instability, deformity	Often Yes	No

Although most of the arthritic conditions are inflammatory, there can be exceptions. For example, a hemophilic joint and a Charcot joint in diabetic patients are non-inflammatory in nature requiring altogether a different line of treatment. Osteoarthritis can be both non-inflammatory and inflammatory.

Inflammatory and non- inflammatory arthritis

Feature	Inflammatory arthritis	Non-inflammatory arthritis
Constitutional features (such as fever, weakness, fatigue)	Yes (in certain conditions)	No
Prolonged early morning stiffness (>1hour)	Yes	No
Spontaneous disease flares	Yes	No
Improvement of joint symptoms on joint usage	Yes	No
Elevated ESR, CRP	Yes	No

Mono-articular and polyarticular arthritis

Even if the patient complains of pain in just one joint, it is mandatory to examine all the joints to ensure that other joints are not involved before proceeding further as mono-articular arthritis. The most critical diagnosis to consider in patients with mono-articular symptoms is septic arthritis because bacterial infection can destroy the joint cartilage within a few days.

Causes for Mono-arthritis

Acute	Subacute or chronic
Septic arthritis	Tuberculosis
Gout (uric acid crystal deposition)	Osteoarthritis
Pseudo gout(calcium pyrophosphate crystal deposition)	Rheumatoid arthritis(commonly polyarticular)
Haemarthrosis	Juvenile idiopathic arthritis
Reactive arthritis	Malignancy
Ischemic necrosis	Trauma

Symmetrical and asymmetrical Polyarthritis

If it is a polyarticular presentation, it is most useful to distinguish between symmetrical and asymmetrical joint involvement. This makes a tremendous difference in differential diagnosis. For example, should it be asymmetrical joint involvement, chances are that this would be a case of one of the seronegative spondylo-arthropathies. On the other hand, polyarticular symmetrical presentation would involve differential diagnosis amongst a large number of collagen vascular disorders including rheumatoid arthritis.

Causes for symmetrical and asymmetrical polyarthritis

Causes for Polyarthritis	
Symmetrical	Asymmetrical
• Rheumatoid arthritis	• Ankylosing spondylitis(especially juvenile)
• Systemic lupus erythematosus • Scleroderma • Myositis	• Reactive arthritis
• Polyarticular gout	• Psoriatic arthritis
• Polyarticular septic arthritis (rarely)	• Enteropathic arthritis
• Infective-endocarditis • Endocrine disorders • Viral arthritis • AIDS	• Undifferentiated spondyloarthropathies

Conclusion

A proper history taking and a detailed clinical examination are the keys to successful diagnosis. A simple approach to the arthritic patient as discussed would help the primary care physician to at least make a tentative clinical diagnosis. The management of individual conditions include appropriate laboratory tests and treatment with both non-pharmacological measures like physiotherapy and drugs like NSAIDS and disease modifying agents. The details of the treatment are described in the subsequent chapters, on Rheumatoid arthritis (RA)

Rheumatoid Arthritis (RA)

Definition

Rheumatoid arthritis is a chronic systemic autoimmune inflammatory disease with long term disability, acute intermittent morbidities and sometimes with premature mortalities

Diagnosis

Revised ACR (1988) classification criteria		
Criteria	Characteristics	Duration
1	• Morning stiffness for more than 1 hour	Must be present for at least six weeks
2	• Arthritis of three or more joint areas	
3	• Arthritis of hand joints	
4	• Symmetric arthritis	
5	• Rheumatoid nodules	
6	• Serum Rheumatoid factor	
7	• Radiographic changes	

" Patient is said to have Rheumatoid arthritis if he or she has four out of seven criteria"

Complications

Infections	Entrapment neuropathies
Vasculitis	Atlanto-axial joint subluxation
Deformities	Interstitial lung disease
Tendon and ligament damage	Amyloidosis
Osteoporosis	Drug toxicity

Investigations

Hematological	Bio-chemical	Immunological
Hemoglobin	LFT	Rheumatoid Factor(RF)
TLC, DLC, Platelet count	RFT	Anti-cyclic citrullinated peptide (anti-ccp)
ESR	Lipid profile	C-reactive protein (CRP)

Rheumatoid factor (RF)

- Positivity indicates more severe and active disease.
- Early bony erosions are more common
- Positive patients may require aggressive treatment with combination of disease modifying anti rheumatic drugs. (DMARDS)
- Absent RF, doesn't exclude the diagnosis of Rheumatoid Arthritis
- Investigations are done to assess the disease activity and in follow up to detect early complications as well as to monitor the drug side effects.
- Radiological investigations like plain x-rays, ultrasound and MRI are useful for diagnosis, to assess severity and to detect erosions.
- Cardiac and ophthalmic workup at base line and during follow up.

Treatment

Conservative

Non-Pharmacological	Pharmacological
Health education	NSAIDS
Physiotherapy	DMARDS
Occupational therapy	Immunosuppressants
Supportive devices like splints	Corticosteroids
Rehabilitation	Biological agents

Operative

Open Surgery	Closed Procedures
Release of contractures	Arthroscopy joint Lavage
Repair of tendon rupture	Chemical synovectomy
Joint replacement	Radio-synovectomy

Analgesic and anti inflammatory drugs

- Physician must evaluate the patients individually
  - * Diclofenac sodium (25-50mg bid-tid)
  - * Ibuprofen (300-800mg bid-qid)
  - * Indomethacin (25mg tid-qid)

Risk factors for increased toxicity

- Advanced age

Causes for Polyarthritis

- Acid peptic ulcer disease
- Pregnancy
- Cardiac failure
- Asthma
- Nasal polyposis

DMARDS:
Disease modifying anti-rheumatic drugs therapy

- Early initiation, combination of drugs and regular monitoring of side effects are essential. The names of the DMARDS are furnished below
  - * Methotrexate
  - * Hydroxychloroquine
  - * Sulphasalazine
  - * Leflunomide
  - * Gold salt
  - * D.Penicillamine
  - * Azathioprine
  - * Biologicals

Referral:

Severe cases of rheumatoid arthritis with extraarticular manifestations should be referred to a higher institute for further work up.

Prognosis:

It is highly variable.

Patient education

Regular physiotherapy and drugs intake are essential to preserve the joints.

Spondylo-Arthropathies

Introduction

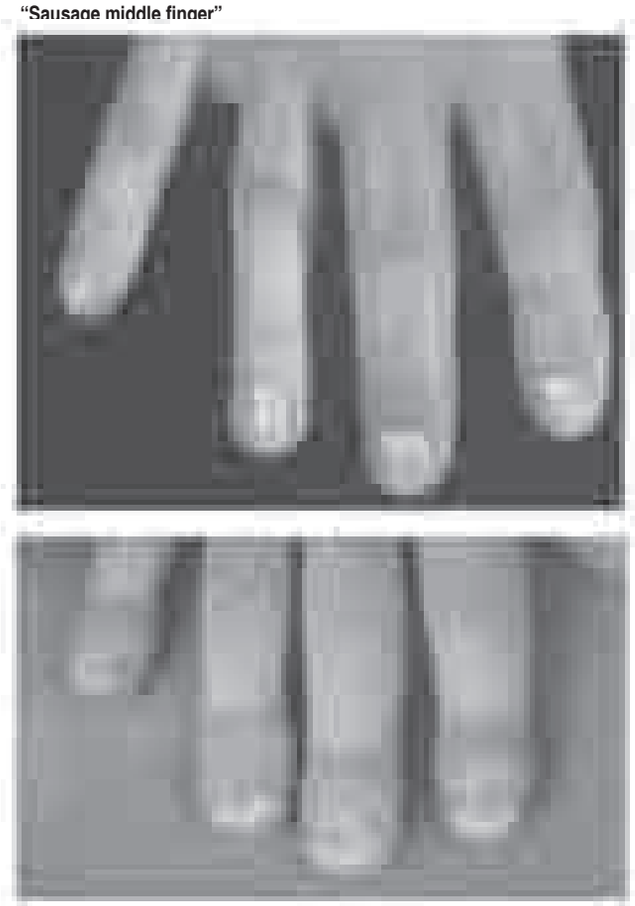
Spondylo-arthropathies (SpAs) constitute a cluster of interrelated and overlapping chronic inflammatory rheumatic diseases that include Ankylosing spondylitis (the proto type of SpAs) reactive arthritis (including Reiter's syndrome), psoriatic arthritis, enteropathic arthritis and pauci-articular late onset arthritis, a form of juvenile idiopathic arthritis. These diseases are not associated with rheumatoid factor

Characteristics of the SpAs

- Absence of the rheumatoid factor and rheumatoid nodules
- Peripheral arthritis
- Spinal inflammation: Inflammatory back pain and sacroiliitis with or without spondylitis
- Peripheral enthesitis
- Familial aggregation
- Association with HLAB27

Psoariatic arthritis

"Sausage middle finger"



Distal inter Phalangeal joint involvement with nail dystrophy

Ankylosing Spondylitis (AS)

Introduction

AS is an HLAB27 associated chronic inflammatory disease of unknown etiology. It affects mainly the sacroiliac joints and the axial skeleton, but peripheral joint involvement may also be an important feature. The disease can be accompanied by extra skeletal manifestations such as

- Acute anterior uveitis
- Aortic incompetence
- Cardiac conduction defects
- Fibrosis of the upper lobes of the lungs
- Neurologic involvement and renal (secondary) amyloidosis

AS is "primary" or "idiopathic" if no associated disorder is present, and "secondary" if the disease is associated with psoriasis or chronic inflammatory bowel disease.

Modified New york 1984 Criteria for Ankylosing spondylitis

- Low back pain of at least 3 months duration improved with exercise and not relieved by rest.
- Limitation of lumbar spine in sagittal or frontal planes.
- Chest expansion decreased relative to normal values for age and sex.
- Bilateral sacroiliitis grade 2 to 4.
- Unilateral sacroiliitis grade 3 or 4.

Definite Ankylosing spondylitis is of

* Unilateral grade 3 to 4
* Bilateral grade 2 to 4
* Sacroilitis and any clinical criterion,

Clinical manifestitions

Articular

Low back pain and stiffness

The pain is felt deep in the gluteal region, is dull in character, is difficult to localize and is insidious in onset. The buttock pain may typically alternate from side to side. The lower lumbar area becomes stiff and painful. Both the stiffness and pain tend to be eased by a hot shower, an exercise program or physical activity.

Chest pain

The pain accentuated by coughing or sneezing which sometimes is characterized as "pleuritic". The pain may be associated with tenderness over sternocostal or costo-sternal junctions. Reduction of chest expansion is often detectable at an early stage of Ankylosing spondylitis.

Tenderness

Tender sites are the costo-sternal junctions, spinous processes, iliac crests, greater trochanters, ischial tuberosities, tibial tubercles and heels (Achilles tendinitis or plantar fasciitis). These sites are called entheses.

Joints

The girdle or "root" joints (hips and shoulders) are the most frequently involved extra axial joints in Ankylosing spondylitis.

Extra-articular

General symptoms

- Fatigue
- Loss of weight
- Low grade fever
- Occur frequently

Eye disease

- Acute anterior uveitis.

Cardiovascular disease

- Ascending aortitis
- Aortic valve incompetence
- Conduction abnormalities
- Cardiomegaly and
- Pericarditis

Pulmonary disease

- Slowly progressive fibrosis of the upper lobes of the lungs is a rare and last manifestation.

Neurologic involvement

- Can be caused by fracture, instability, compression or inflammation
  - Atlanto-axial joint subluxation
  - Atlanto-occipital subluxation
  - Upward subluxation of the axis may occur.
  - Compressive myelopathy
  - Cauda equina syndrome may also occur.

Renal involvement

- IgA nephropathy
- Microscopic hematuria
- Proteinuria may occur
- Amyloidosis is a rare complication

Osteoporosis

- Osteopenia is seen even in early stages of Ankylosing spondylitis.

Laboratory tests

- An elevated ESR or CRP, a mild normochromic anemia, elevations of serum alkaline phosphatase and serum IgA are seen.
- X-Rays show changes in sacroiliac, disco-vertebral, apophyseal, costovertebral and costo-transverse joints.
- CT is usually considered superior to MRI.

Management:

Physiotherapy

- Hydrotherapy and exercises are useful.

Medication

- NSAIDs
  - Indomethacin
  - COX-2 inhibitors like etoricoxib

Second line drugs

- Cortico- steroids are effective for local intra articular treatment
- Sulphasalazine is effective in reducing synovitis in patients with peripheral poly articular involvement but has no results on axial involvement.

Surgery

- Total hip replacement can be done for involvement of the hip joint.

Referral

- All suspected cases should be referred to higher institutions for further management.

Natural course and prognosis

- It is highly variable and characterized by spontaneous remissions and exacerbations.

Patient education

- Smoking should be avoided.
- Regular physiotherapy
- Follow-up is essential for prevention of joint ankylosis

Systemic Lupus Erythematosus

Introduction

It is a common autoimmune multisystem inflammatory disease with diverse clinical and laboratory manifestations associated with an unpredictable course and prognosis. Difficulty in diagnosis, laboratory assessment and treatment are peculiar features of this often devastating illness.

Epidemiology

It is more in females of reproductive age group, but no age is exempt.

In India, the female:male ratio is 11:1.

Types Of Lupus

- Discoid lupus erythematosus
- Subacute cutaneous lupus erythematosus
- Drug induced lupus

Lupus in special situations

In order to exercise special care for lupus in special situations, the details of the same are provided.

Lupus and pregnancy

Patients should be allowed to become pregnant only when the disease is in remission for 6 months both clinically and laboratory wise. Close monitoring is mandatory.

Neonatal Lupus

Occurs in newborns of mothers having anti Ro (SSA) and anti La (SSB) antibodies with the mothers being symptomatic or asymptomatic. New born intensive care is essential.

Symptoms and Signs

- Unexplained fever
- Arthritis
- Muscle pain
- Rashes over the face
- Lymphadenopathy
- Oral ulcers
- Extreme fatigue
- Chest pain on deep breathing-pleurisy
- Unusual loss of hair
- Raynaud's phenomenon
- Photosensitivity
- Eyelid and leg oedema
- Chest pain
- Anaemia
- Hypertension
- Headache
- Dizziness
- Confusion
- Depression
- Behavioural changes
- Seizues
- Stroke etc.
- At different times
  - Renal involvement is usually asymptomatic except for oedema, hypertension and identification of urinary abnormalities.
- Co-morbid states
  - Accelerated atherosclerosis
  - Pro-thrombotic states
  - Osteoporosis
  - Infection

Criteria for diagnosis

American College of Rheumatology (ACR) - in 1999 stipulated that at least 4 of the following 11 conditions must be present, serially or simultaneously.

Acr Criteria For Diagnosis Of Sle	
1.	Malar rash
2.	Discoid rash
3.	Oral ulcers
4.	Photosensitivity
5.	Arthritis (non-erosive, involving at least two peripheral joints and accompanied by tenderness, swelling, or effusion)
6.	Pleuritis or pericarditis
7.	Neurologic disorders (seizures or psychosis)
8.	Hematologic disorder (hemolytic anemia, leucopenia, lymphopenia, or thrombocytopenia)
9.	Renal disorders (persistent proteinuria or cellular casts)
10.	Anti-nuclear antibodies (ANA)
11.	Immunologic disorders (abnormal levels of antibody to native DNA, antibodies to the smith (SM) nuclear antigen, or antiphospholipid antibodies)

Investigations

"No single test can establish but several laboratory tests help in the diagnosis".

Differential diagnosis:

- Includes other systemic connective tissue disorders like
  - Rheumatoid arthritis
  - Dermatomyositis
  - Polymyositis
  - Vasculitis
  - Sjogren's syndrome
  - Primary anti-phopholipid antibody syndrome
  - Fibro-myalgia
  - Drug reactions
  - HIV
  - Hematological disorders

Treatment

- Requires a collaborative approach comprising various specialists
- Patient and family education is of vital importance - about remissions, exacerbations, marriage, pregnancy etc.
- Treatment is individualized and depends on whether it is organ or life threatening or not – mild, moderate, severe or with miscellaneous features.
- Treatment plan has to be charted according to the patient's age, sex, symptoms, flare and life style. Patients should be explained about flares and overall treatment.

Drugs:

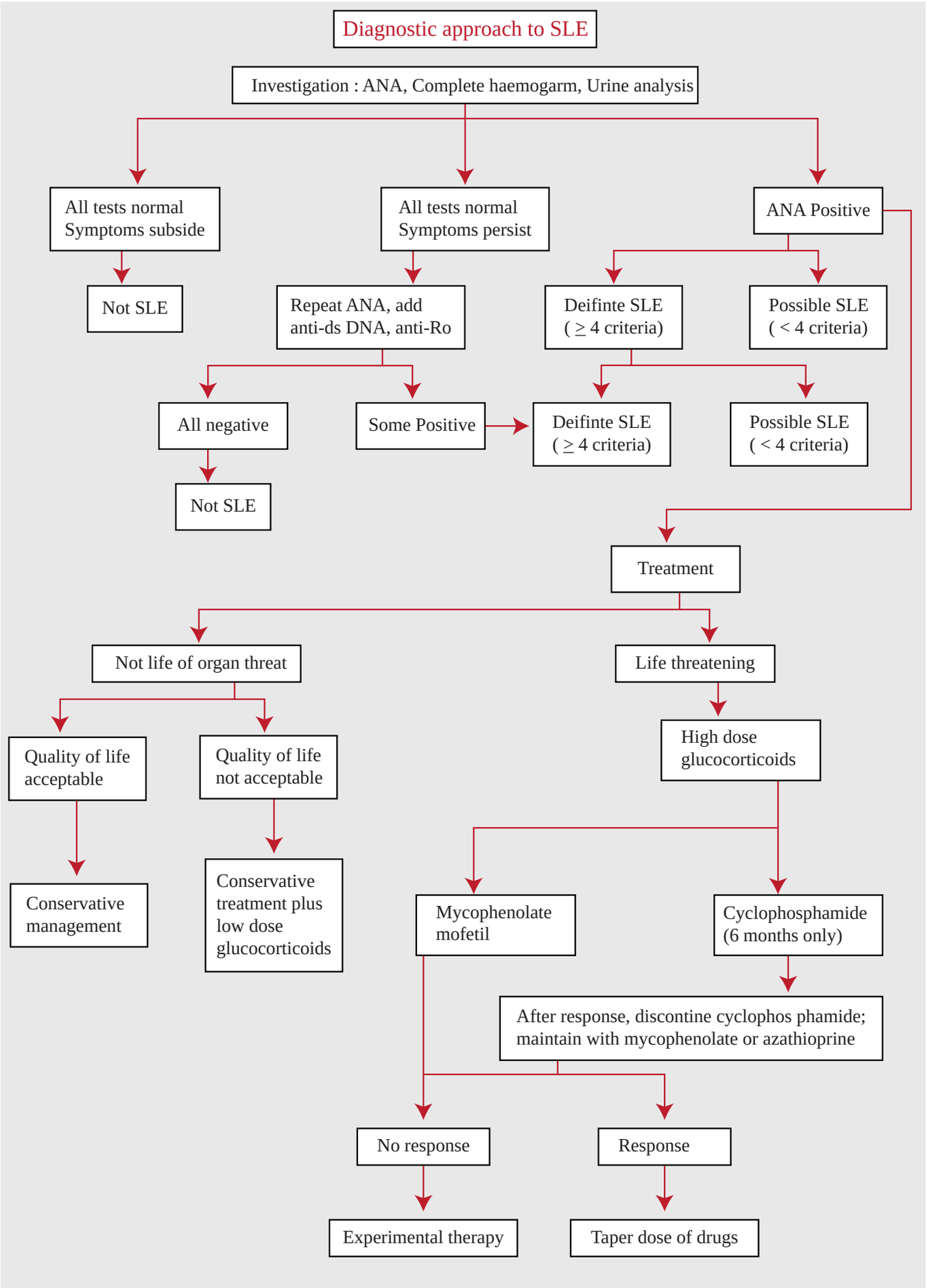
- Ibuprofen should not be given to lupus patients
- Exposure to sun light should be avoided.
- Apart from treatment of the disease (e.g. steroids - upto cyclophosphamide and then biologics etc.) as the situation demands, the following should also be treated.
- Comorbid states: Aspirin for atherosclerosis prophylaxis is desirable. Osteoporosis should be treated.
- Immuno-prophylaxis
  - Vaccinations permitted include
    - Hemophilus
    - Influenza (HIB)
    - Pneumococcal vaccine yearly

Key Message

- Diagnosis can be frustrating and primary care physicians may be the first to suspect and have to manage in conjunction with a Rheumatologist or Physician.
- Natural course is unpredictable with remissions and exacerbations.
- Patient and family members or care providers must be informed or educated about the natural course of the disease, complications, co-morbid status, infection, special situations and long term management.
- Periodic review by a specialist is helpful.
- Referral to a higher centre is desirable for confirming the diagnosis, to assess disease activity or severity, for establishing or amending treatment plans and to improve the outcome.

Reference

- Mary Desmond pinkowish: American College of Rheumatology Ad Hoc committee on Systemic Lupus Erythematosus Guidelines.


Diagnostic approach to SLE
Investigation : ANA, Complete haemogarm, Urine analysis
All tests normal Symptoms subside
All tests normal Symptoms persist
ANA Positive
Not SLE
Repeat ANA, add anti-ds DNA, anti-Ro
Deifinte SLE (≥ 4 criteria)
Possible SLE (< 4 criteria)
All negative
Some Positive
Deifinte SLE (≥ 4 criteria)
Possible SLE (< 4 criteria)
Not SLE
Treatment
Not life of organ threat
Life threatening
Quality of life acceptable
Quality of life not acceptable
High dose glucocorticoids
Conservative management
Conservative treatment plus low dose glucocorticoids
Mycophenolate mofetil
Cyclophosphamide (6 months only)
After response, discontine cyclophos phamide; maintain with mycophenolate or azathioprine
No response
Response
Experimental therapy
Taper dose of drugs

- Guidelines for referral and management of Systemic lupus erythematosus in adults. Arthritis Rheumd. 1999; 42: 1785-1796.
- A Kumar. A Indian Guidelines on the management of SLE. J Indian Rheumatol Assn 2002: 10: 80-96.

Vasculitis

Definition

Vasculitis is a clinical condition where inflammation of the blood vessels occurs resulting in either necrosis of the vessel wall, aneurysmal dilatation, narrowing of the lumen or occlusion of the lumen due to secondary effects of inflammation - thrombus, the final result of which will be infarction of the tissues if there are no adequate collaterals. It should be differentiated from Vasculopathies which are non- inflammatory occlusive conditions.

Epidemiology

- It is observed in all ages from childhood to adulthood. Takayasu's arteritis has a highest incidence in Asia and India.

Etiology

- The cause may be unknown but is usually due to the result of combination of genetics, environmental exposure or infection.

When the cause is not identified they are called Primary vasculitis, and classified on the basis of blood vessel involvement

Primary Vasculitis		
Large vessel	Medium vessel	Immune complex mediated
Takayasu's arteritis	Polyarteritis Nodosa	Goodpasture's disease
Giant cell arteritis	Cutaneous polyarteritis nodosa	Cutaneous leucocytoclastic vasculitis
Cognan's syndrome	Buerger's disease	Henoch- schonlein Purpura
Behcet's disease	Kawasaki's disease	Hypo-complementenemic urticarial vasculitis
	Primary angitis of the central nervous system	Essential cryoglobulinemia Erythema elevatum diutinum

Clinical manifestations are varied and those common to vasculitis of all vessels include

- Fever
- Weight loss
- Malaise
- Arthritis
- Arthralgias
- Seizures
- Nerve palsies
- Myopathies
- Hypertension
- Renal failure
- Wheezing
- Gangrene
- Rashes
- Cerebrovascular accidents and
- Myocardial infarction

Investigations

Depends on the type of vasculitis. Many tests are helpful in the diagnosis or in evaluation of the activity of the disease and in ruling out other mimics of vasculitis. Cases may be referred to higher centre for appropriate evaluation.

A complete blood count, inflammatory markers and individual organ assessment are essential. Special immunological investigations as well as those that rule

out other disorders simulating vasculitis are required at higher centres.

Treatment

Generally accepted protocols for the different type of vasculitis consist of the following. However, the treatment and drugs schedules are individualized.

- Large vessel vasculitis:
  - * Steroids
  - * Methotrexate
  - * Azathioprine
- Medium vessel vasculitis:
  - * Steroids + Cyclophosphamide for induction
  - * Azathioprine or Methotrexate for remission
- Small vessel vasculitis:
  - * Treatment when required with initial steroid therapy and if refractory and absolutely necessary, cytotoxics
- Kawasaki disease in the acute stages requires intravenous immunoglobulin and resistant systemic vasculitis require newer therapies which include Biologicals.

Emergencies in vasculitis requiring specialists care include

- Impending visual loss
- Impending or established gangrene.
- Uncontrolled hypertension.
- Renal emergencies
- Respiratory distress
- Neurological e.g., Seizures, Stroke, Mono-neuritis multiplex, Headache
- Acute abdomen e.g., mesenteric vasculitis and ruptured aneurysm
- Myocardial infarction
- CCF
- Kawasaki disease
- Takayasu's arteritis
- Bleeding from any origin

Key Message:

- Vasculitis is an enigmatic disease with remissions and exacerbations.
- Admission: Institutional management is advisable in emergencies.
- Referral:
  - * Early referral to the higher centre and regular follow up are mandatory.
- Outcome:
  - * Prognosis is variable with unpredictable complications.

Regional Syndromes

Regional syndromes are characterized by pain that affects a single musculoskeletal area (eg: shoulder, hand or knee). This could be of four main origins:

- Peri-articular pain
- Joint pain
- Neurogenic pain
- Referred pain

Osteo - Arthritis HIP

1. Joint space narrowing
2. Sub - chondral sclerosis
3. Marginal osteophyte and cysts

Distinctive features of regional syndromes

	Pariarticular pain	Articular pain	Neurogenic pain	Referred pain
Enquiry	Selective painful movements	All joint movements are painful	Disaestesic. Aggravated by compression of nerve or mobilization of the spine	Unrelated to movement “visceral” timing
Pain on motion	Active> passive selected motions	Active ~ passive several directions	Normal. If root pain, pain on motion of the affected spine segment	Normal
Range of motion	Active motion can be limited by pain. passive motion: full	Can be limited in active and passive motion	Normal	Normal
Resisted mobilization	Pain on specific manoeuvres	No effect	No effect	No effect
Local palpation	Pain upon affected structure	Possible crepitus, swelling, effusion, heat, pain along joint margin	Normal	Normal
Neurological examination	Normal	Normal	May be abnormal	Normal

Diseases affecting the joints

History and examination taken to decide between degenerative and inflammatory joint pain.

Degenerative joint disease

Osteoarthritis is typically associated with paint that

- Increases with repeated use of the joint and
- Is worst at the end of day
- Pain intensity decreases during rest
- Is rarely present at night and
- The patient can usually find a pain - free position

Patients can describe that pain increases again after resting and this may be accompanied by stiffness that subsides in 2-3 minutes. Early morning stiffness associated with degenerative arthritis ceases in a few minutes (<10).

In contrast to active inflammatory disease, pain is worst in the morning and is relieved by continued use of the joint. The patient may have pain during the night, not related to movement in bed and cannot identify pain - rest stiffness may persist for more than 5 minutes.

Associated extra - articular manifestations may accompanay a variety of inflammatory arthritis but they are absent in degenerative joint disease. Then determine

- Which joints are affected and their pattern of distributin
- How the condition began and how it developed over time
- The presence or absence of inflammatory low back pain
- Accompanyng extra - articular manifestations.

Mono-arthritis

It is useful to separate acute (onset over hours to days) from chronic mono-arthrits.

Acute mono-arthrits

The common causes are

- Trauma and Gout, but this condition should be considered infectious (septic arthritis) untill proven otherwise.
- Pseudo - gout (calcium pyrophosphate dihydrate disease)
- Post - traumatic synovitis
- Palindromic rheumatism

- Reactive arthritis
- Psoriatic arthritis and
- Bacterial endocarditis are the other causes

Chronic mono-arthritis

Mono-arthritis can have an indolent course, lasting from weeks to months. The main possible causes include

- Infection (Brucella, Mycobacterium,)
- Crystal induced arthritis and
- Mono-articular presentation of oligo or polyarthritis
- Juvenile idiopathic arthritis
- Reactive arthritis
- Seronegative spondylo-arthropathy

Differential diagnosis must include causes such as osteoarthritis and neuropathic (Charcot's)joints. The aetiological diagnosis of chronic mono-arthritis usually requires synovial biopsy with pathological and bacteriological examination.

Chronic symmetrical additive peripheral polyarthritis

This pattern describes joint inflammation involving, simultaneously, five or more joints (polyarthritis), for more than six weeks (chronic). small joints of the hands and feet are predominantly affected, with or without the wrist and ankle (peripheral), and approximately the same joints are involved on each side of the body (symmetrical). There should be no inflammatory low back pain.

Causes

- Rheumatoid arthritis, and this is the most common pattern
- Other connective tissue diseases such as
  - Systemic lupus
  - Primary Sjogren's syndrome
  - Polymyostitis
  - Mixed connective tissue disease
- Psoriatic arthritis can also present with this pattern

Chronic, asymmetrical oligo/polyarthritis

This describes an asymmetrical arthritis, affecting proximal or distal joints. Dactylitis or involvement of distal interphalangeal joints is common.

- Psoriatic arthritis is the most common cause of this pattern.
- Ankylosing spondylitis (inflammatory back pain is expected in this condition but may no be obvious).
- Reactive arthritis
- Arthritis associated with inflammatory bowel disease.

Proximal oligoarthritis

Patients may present with arthritis involving predominantly proximal joints. The most common causes are the

- Seronegative spondylarthropathies
- Less common causes are
  - Behcet's disease
  - Juvenile idiopathic arthritis and
  - Incipient rheumatoid arthritis

Inflammatory low back pain

This refers to low back pain that persists or predominates at night, does not relieve with rest but rather with movement and is associated with prolonged morining stiffness. This is always a significant clue that deserves clarification.

This is a typical mainifestation of

- Seronegative spondylo-arthropathies
  - Ankylosing spondylitis
  - Psoriatic arthritis
  - Reactive arthritis
  - Spondylitis
  - Inflammatory bowel disease
- It is also a feature of Bechcet's disease and infectious or aseptic discitis

Generalized pain

In this syndorme, pain affects different parts of the body diffusely and imprecisely, with little or no focus on joints.

Vitamin D deficienc;y and Fibro-myalgia accounts for the majority of the cases of generalized pain syndrome.

Juvenile Idiopathic Arthritis (JIA)

JIA includes all forms of arthritis that begins before the age of 16 years, persists for more than 6 weeks and is of unknown cause.

Classification of JIA (International League Association For Rheumatology)

Category	Definition	Exclusions
Systemic onset JIA	Arthritis in> 1 joint with, or preceded by, fever of at least two weeks that is documented to be daily (“quotidian”) for at least 3 days and accompanied by >1 of the following	Psoriasis or a history of psoriasis in the patient or a first - degree realtive
	Evanescent (non-fixed)erythematous rash	Arthritis in an HLA-B27+ male beginning after the 6th birthday
	Generalized lymph node enlargement	AS, Enthesistis - related arthritis, sacroilietis with inflammatory bowel disease, Reactive arthritis, or acute anterior uveitis, or h/o of one of these disorders in a first - degree relative
	Hepatomegaly or splenomegaly or both	Presence of IgMRF on at least 2 occasions at least 3 months apart
	Serositis	
Oligoarticular JIA	Arthritis affecting 1-4 joints during the first 6 months of disease. Two subcategories are recognized:	A,B,C,D above, plus
	Persistent oligo-arthiritis affecting < 4 joints throughout the disease course	Presence of systemic JIA in the patient
	Extended oligo-arthritis affecting > 4 joints after the first 6 months of disease	
Polyarthritis (RF negative)	Arthritis affecting>5 joints during the first 6 months of disease; a test for RF is negative	A, B, C, D, E
Polyarthritis (RF Positive)	Arthritis affecting >5 joints during the first 6 months of disease; >2 tests for RF at least 3 months of disease are positive	A, B, C, E
Psoriatic arthritis	Arthritis and psoriasis, or arthritis and at least 2 of the following:	B, C, D, E
	Dactylitis*	
	Nail pitting and onycholoysis	
	Psoriasis in a first- degree relative	
Enthesitis - related arthritis	Arthritis and enthesitis, or arthritis or enthesitis with at least 2 of the following	A, D, E
	Presence of HLA -B27 antigen	
	Onset of arthritis in a male>6 yr old	
	Acute (Symptomatic) anterior uveitis	
	History of ankylosing spondylitis, enthesitis - related arthritis, sacroilitis with inflammatory bowel disease, Reiter’s syndrome, or acute anterior uveitis in a first - degree relative	
Undifferentiated arthritis	Arthritis that fulfills criteria in no category or in >2 of the above categories	

Table acronyms

RF: rheumatoid factor

Quotidian fever is defined as a fever that rises to 39oc once a day returns to 37oc between fever peaks.

Serosistis refers to pericarditis, pleuritis, or peritonitis, or some combination of the three.

Dactylitis is swelling of >1 digits, usually in an aysmmetric distribution, which extends beyond the joint margin.

A minimum of 2 pits on any or more nails at any time.

Enthesitis is defined as tenderness at the insertion of a tendon, ligament, joint capsule, or fascial to bone.

Inflammaroy lumbosacral pain refers to lumbosacral pain at rest with morning stiffness that improves on movement.

Management of Juvenile idiopathic arthritis

Investigations

There is no specific test for disgnosis of JIA

- Complete blood count
- Anaemia, raised white cell and platelet count consistent, with inflammation.
- ESR and CRP may be elevated
- Rheumatoid factor (RF) and antinuclear antigen (ANA)
- Autoantibodies indicate poor prognosis in JIA.
- Synovial fluid counts, glucose and culture should be performed if septic arthritis is suspected.
- Plain X-rays have a role in excluding trauma, malignancy or infection, but changes caused by Juvenile idiopathic arthritis often occur late in the disease course.
- Ultrasound screening of joints may be used to detect effusions, especially where clinical assessment is difficult (such as hips), and also to guide intra-articular treatment.

Poor progonstic indicators in Juvenile idiopathic arthritis (JLA)

- Active systemic disease at 6 months in Sysemic Onset JIA (fever, need for cortisocsteroid, and thrombocytosis)
- Polyarticular onset and polyarticular disease course
- Extended oligo JIA
- Female
- Rheumatoid factor postive
- ANA positive
- Persistent early morning stiffness
- Tenosysnovitis
- Subcutaneous nodules
- Rapid involvement of the small joints of the hands and feet with erosions
- Hip involvement
- Higher mean ESR
- Generalized lymphadenopathy.

Principles of therapy

The mangement of JIA benefits from input from a large multidisciplinary team including physiotherapists, occupational therapists, podiatrists or orthotists, specialist nurses, community nursing teams, psychologists, social workers, school liaison workers, family support groups, general practitioners, opthalmologists, dentistis or orthodontists, orthopaedic surgeons, and pain management teams.

Recent changes in the medical management of JIA have included increased use of intrarticular and judicious use of oral and/ or intravenous corticosteroids, establishment of Methrotrexate as the first line disease modifying anti - rheumatic drug (DMARD), and the discovery and use of biologic agents or newer treatments of resistant disease. Early, aggressive suppression of inflammation is the principle underlying treatment.

Non - steroidal anti-inflammatory drugs (NSAIDS)

NSAIDS are used for the initial treatment of pain, inflammation and stiffness. side effects are well recoginised and include gastrointestinal disturbance, rash, mood changes, and sleep disturbance.

Doses used in JIA are higher than in other indications. Thers is no evidence to support the use of topical NSAIDS in JIA. There is little evidence base for differences between NSAIDS and choice is usually made according to preferred dosing schedules, availability of liquid preparations, and patient preference.

Doses of common non - steroidal anti-inflammatory drugs (NSAIDS) used for JIA

- Ibuprofen: 10mg/kg three or four times daily, can be given up to 60mg/kg/day in systemic onset JIA in 6 divided doses
- Naproxen: 5-10mg/kg twice daily (suspension 125mg/5ml)
- Diclofenac: 1mg/kg twice or three times daily
- Indomethacin: 0.5 -1mg/kg three times daily

Corticosteroids

Intra-articular corticosteroids if used under proper aseptic condition, are well established for the treatment of oligoarticualar JIA and as an adjunct in the treatment of polyarticular JIA.

The preparations used are

- Traimcinolone hexacetonide
- Triamcinolone acetonide
- Methylprednisolone acetate

  Lignocaine Combinitaion for intra articular injection to be avoided as it can cause anaphylaxis

Intermittent pulsed Methylprednisolone

(30mg/kg/day with a maximum daily dose of lg, given on three consecutive days, repeated one week later) has been shown to provide good short term benefit in JIA, achieving rapid control of disease and allowing for a lower cumulative dialy steroid dosage than conventional oral glucocorticoid treatment.

If long-term oral steroids are required, they should be used at the minimum does possible to control disease. Long acting steroids like betamethasone, dexamehasone are not to be used as they cause prolonged adrenal suppression. Steroid dose is to be given in single dose in the morning.

Doses more than 1mg/kg day of prednisolone are not necessary, in fact can causes more side effects.

Methotrexate has become the second-line agent of choice for persistent, active arthritis because of its effectivenvess and acceptable toxic effects. Improvement in patients is usually seen after 6-12 weeks of continuous and regular therapy.

Dose 10mg/m2 per week.. maximum therapeutic effect with parenteral administration of 15 mg/m2 per week along with supplementation of folic acid 5mg twice weekly to prevent the occurrence of liver enzyme abnormalities.

The side-effect profile

Methotrexate is well recognized and drug interations are rarely significant at doses used. Adverse effects occur in up to 23% of children most commonly nausea, vomiting, mouth ulcers, loss of appetite, alopecia, transient rise in liver enzymes, or leucopenia.

Monitoring Methotrexate toxicity

Complete blood counts, LFT and creatinine initially every month for 3 month then once in every 3 month.

Other disease modifying anti-rheumatic drugs

- Sulfasalazine can be beneficial for late-onset oligo JIA, enthesitis-related or inflammatory bowel realated arthritis.
- It is to be avoided in Systemic onset JIA.
- Dose:
  - Initially 30mg/kg/day in divided doses, then increase by 5mg/kg every week to maximum dose of 50mg/kg/day in dividied doses.
- Monitoring:
  - Total blood Counts monthly for 3 months then every 3rd monthly therafter

Anti-TNF treatment

Antitumour necrosis foctor (TNF) therapy is required for many children who have failed to respond adequately to Methotrexate or have been unable to tolerate Methotrexate because of adverse effects.

Other medical considerations in JIA

- Treatment of growth failure
- Treatment of uveitis - ophtalmology referral
- Dental referral
- Vaccinations
- Treatment of varicella contact if immunosuppressed
- Prevention of osteoporosis
- Orthopaedic referral for joint replacement
- Psychology and counselling
- Pain management
- Letters for school, career advice
- Adolescent care and sexual health.

Infections

Standard Treatment Guidelines
Tamil Nadu Health Systems Project

Chapter 11

- HIV and AIDS
- Opportunistic Infections
- Pneumocystitis Carinii (Jerovici)
- Oesophageal Candidiasis
- Cryptococcosis
- Toxoplasmosis
- Cryptosporidiosis
- Typhoid
- Leptospirosis
- Parasitic Infections
- Amoebiasis
- Giardiasis
- Malaria
- Worm Infestations
- Ascariasis
- Filariasis
- Dengue fever
- Chikungunya
- H1N1 Swine Flu

Infections – Bacterial, viral and opportunistic

HIV and AIDS

HIV infection leads to progressive immunodeficiency and increased susceptibility to infections, including TB. The rate of disease progression is highly variable between individuals, ranging from 6 months to more than 20 yrs

The median time to develop AIDS after transmission is 10 yrs in the absence of anti retroviral therapy (ART).

Symptoms

- The case definition of AIDS is fulfilled if at least 2 major signs and at least 1 minor sign are present where HIV testing facilities are not available. In children if 2 major and 2 minor signs are present (if no other causes for immunosuppression)
- **Major signs and symptoms:**
  - * Weight loss (>10 Kg or > 20 % of original weight)
  - * Diarrhoea (>1 month)
  - * Fever for more than 1 month
- **Minor signs:**
  - * Herpes zoster (shingles)
  - * Pruritic popular rash
  - * Kaposi's sarcoma
  - * Persistent generalized lymphadenopathy
  - * Oral candidiasis
  - * Oral hairy leukoplakia
  - * Persistent painful genital ulceration.
  - * The persistence of either Kaposi's sarcoma or cryptococcal meningitis is sufficient for the case definition of AIDS
- HIV disease is characterized by three phases
  - * Acute primary illness
  - * Asymptomatic chronic illness
  - * Symptomatic chronic illness.
- Essential laboratory investigations:
  - * HIV serology
  - * CD4+T lymphocytes counts (if available)
  - * Complete blood count and chemistry profile
  - * Pregnancy test.
- Supplementary tests indicated by history and physical examination:
  - * Chest X-ray
  - * Urine for routine and microscopic examination
  - * Hepatitis C virus (HCV) and Hepatitis B virus (HBV) serology (depending on test availability and resources)

Important note

It is most important to confirm the diagnosis of HIV infection by tests performed by a trained technician, preferably in a diagnostic laboratory. The test results should include the type of test performed to establish the diagnosis based on WHO guidelines. In case there is any doubt, the test should be repeated in a standard/referral laboratory.

Treatment

Initiation of ART in HIV – infected adults and adolescents

HIV disease staging is important for initiating antiretroviral (ARV) therapy. WHO recommends that in resource-limited settings, in case of HIV infected adults and adolescents, start ARV when they have:

- WHO stage IV of HIV disease **(clinical AIDS)**, regardless of CD4 count
- WHO stages I, II, III of HIV disease, with a CD4 count below 200/mm

If CD4 testing is available

- WHO stage IV disease
- WHO stage I, II or III with CD4 count < 200/mm3

If CD4 testing is unavailable

- WHO stage IV disease irrespective of CD4 count
- WHO stage I, II or III with count < 1200/mm3. Though the total lymphocyte count correlates relatively poorly with CD4 count, in combination with clinical staging it is a useful marker of prognosis and survival

WHO Clinical staging of HIV Disease for adults

Primary HIV Infection

- Asymptomatic
- Acute retroviral syndrome

Clinical stage 1

- Asymptomatic
- Persistent generalized lymphadenopathy (PGL)

Clinical stage 2

- Moderate unexplained weight loss (<10% of presumed or measured body weight)
- Recurrent respiratory tract infections
  - Sinusitis
  - Bronchitis
  - Otitis media
  - Pharyngitis
- Herpes zoster
- Angular cheilitis
- Recurrent oral ulcerations
- Papular pruritic eruptions
- Seborrhoeic dermatitis
- Fungal nail infections of fingers

Clinical stage 3

Conditions where a presumptive diagnosis can be made on the basis of clinical signs or simple investigations

- Severe weight loss (>10% of presumed or measured body weight)
- Unexplained chronic diarrhoea for longer than one month
- Unexplained persistent fever (intermittent or constant for longer than one month)
- Oral candidiasis
- Oral hairy leukoplakia
- Current Pulmonary tuberculosis (TB)
- Severe presumed bacterial infections (e.g. pneumonia, empyema, pyomyositis, bone or
- Joint infection, meningitis, bacteraemia)
- Acute necrotizing ulcerative stomatitis, gingivitis or periodontitis

Conditions where confirmatory diagnostic testing is necessary

- Unexplained anaemia (< 8 g/dl), and or neutropenia (<500/mm3) and or
- Thrombocytopenia (<50 000/ mm3) for more than one month

Clinical stage 4

Conditions where a presumptive diagnosis can be made on the basis of clinical signs or simple investigations

- HIV wasting syndrome
- Pneumocystis pneumonia
- Recurrent severe or radiological bacterial pneumonia
- Chronic herpes simplex infection (orolabial, genital or anorectal of more than one month's duration)
- Oesophageal candidiasis
- Extra-pulmonary Tuberculosis including Tuberculous lymphadenopathy
- Kaposi's sarcoma
- Central nervous system (CNS) toxoplasmosis
- HIV encephalopathy

Conditions where confirmatory diagnostic testing is necessary:

- Extra-pulmonary cryptococcosis including meningitis
- Disseminated non-tuberculous mycobacterial infection
- Progressive multi-focal leukoencephalopathy (PML)
- Candida of trachea, bronchi or lungs
- Cryptosporidiosis
- Isosporiasis
- Visceral herpes simplex infection
- Cytomegalovirus (CMV) infection (retinitis or of an organ other than liver, spleen or lymph nodes)
- Any disseminated mycosis (e.g. histoplasmosis, coccidiomycosis, penicilliosis)
- Recurrent non-typhoidal salmonella septicaemia
- Cerebral or B-cell Non-Hodgkin lymphoma
- Invasive cervical carcinoma
- Visceral leishmaniasis

WHO clinical staging of HIV for children

Clinical Stage 1

- Asymptomatic
- Persistent Generalised lymphadenopathy (PGL)

Clinical Stage 2

- Hepatosplenomegaly
- Papular pruritic eruptions
- Seborrhoeic dermatitis
- Extensive human papilloma virus infection
- Extensive molluscum contagiosum
- Fungal nail infections
- Recurrent oral ulcerations
- Lineal gingival erythema (LGE)
- Angular cheilitis
- Parotid enlargement
- Herpes zoster
- Recurrent or chronic recurrent respiratory tract infections
  - Otitis media
  - Otorrhoea
  - Sinusitis

Clinical Stage 3

Conditions where a presumptive diagnosis can be made on the basis of clinical signs or simple investigations

- Moderate unexplained malnutrition not adequately responding to standard therapy
- Unexplained persistent diarrhoea (14 days or more)
- Unexplained persistent fever (intermittent or constant, for longer than one month)
- Oral candidiasis (outside neonatal period)
- Oral hairy leukoplakia
- Acute necrotizing ulcerative gingivitis /periodontitis
- Pulmonary tuberculosis including tuberculous lymphadenopathy
- Severe recurrent presumed bacterial pneumonia

Conditions where confirmatory diagnostic testing is necessary

- Chronic HIV-associated lung disease including brochiectasis
- Lymphoid interstitial pneumonitis (LIP)
- Unexplained anaemia (< 8g/dl)
- Neutropenia (< 1000/mm3) and or
- Thrombocytopenia (< 50 000/ mm3) for more than one month

Clinical stage 4

Conditions where a presumptive diagnosis can be made on the basis of clinical signs or simple investigations

- Unexplained severe wasting or severe malnutrition not adequately responding to standard therapy
- Pneumocystis pneumonia
- Recurrent severe presumed bacterial infections (e.g. empyema, pyomyositis, bone or joint infection, meningitis, but excluding pneumonia)
- Chronic herpes simplex infection; (orolabial or cutaneous of more than one month's duration)
- Extra-pulmonary TB
- Kaposi's sarcoma
- Oesophageal candidiasis
- CNS toxoplasmosis (outside the neonatal period)
- HIV encephalopathy

Conditions where confirmatory diagnostic testing is necessary

- CMV infection (CMV retinitis or infection of organs other than liver, spleen or lymphnodes onset at age one month or more)
- Extra-pulmonary cryptococcosis including meningitis
- Any disseminated endemic mycosis (e.g. extrapulmonary histoplasmosis, coccidiomycosis, penicilliosis)
- Cryptosporidiosis
- Isosporiasis
- Disseminated non-tuberculous mycobacteria infection
- Candida of oesophagus, bronchi or lungs
- Visceral herpes simplex infection

- Acquired HIV associated rectal fistula
- Cerebral or B-cell Non-Hodgkin lymphoma
- Progressive multifocal leuko - encephalopathy (PML)
- HIV-associated cardiomyopathy or
- HIV-associated nephropathy

Principles and goals of therapy of HIV infection

- Ongoing HIV infection leads to immune system damage and progression to AIDS; goal is restoration and/or preservation of immunological function
- Plasma HIV RNA levels indicates the magnitude of HIV replication and the rate of CD4+ T cell destruction.
- **CD4+ T cell counts indicate the current level of competence of the immune system.**
- Rates of disease progression differ among individuals and treatment decisions should be individualized based upon the plasma HIV RNA levels and CD4+ T cell counts
- Maximal suppression of viral replication is the goal of therapy; then greater the suppression the less likely the appearance of drug-resistant quasi-species
- The goal of anti-retroviral therapy is reduction of HIV-related morbidity and mortality and improvement in quality of life.
- The most effective therapeutic strategies involve the simultaneous initiation of combinations of effective anti-HIV drugs with which the patient has not been previously treated and those that are cross-resistant with anti-retroviral agents that the patient has already received.
- The anti-retroviral drugs used in combination regimens should be used according to optimum schedules and dosages.
- The number of available drugs is limited. Any decisions on anti-retroviral therapy have a long term impact on future options for the patient
- Women should receive optimum anti-retroviral therapy regardless of the pregnancy status.
- The same principles apply to children and adults. The treatment of HIV infected children involves unique pharmacologic, virologic, and immunologic considerations.

Recommended regimens for treatment of AIDS in adults and adolescents

First line regimens	Dose
Zidovudine (ZDV) Lamivudine (3TC) plus Efavirenz (EFZ) or Nevirapine (NVP)	ZDV 300 mg twice daily 3TC 150 mg twice daily EFZ 600 mg once daily NVP 200 mg once daily for 14 days, then 200 mg twice daily
ZDV/3TC/Abacavir (ABC)	ABC300 mg twice daily
ZDV/3TC/RTV-enhanced	
PI[a] or Nelfinavir (NFV)	NFV 1250 mg twice daily
Indinavir (IDV/r)	IDV/r 800 mg/100 mg twice daily used Commonly; 800 mg/200mg to 400mg/100 mg
Lopinavir (LPV/r)	LPV/r 400 mg/100 mg twice daily
Saquinavir (SQV/r)	SQV/r 1000 mg/ 100 mg twice daily

RTV - enhanced PI = Indinavir (IDV) / Ritonavir (r), Lopinavir (LPV), / Ritonavir, Saquinavir (SQV) /Ritonavir. An RTV - enhanced PI regimen is preferred given the potency of these regimens. NFV can be considered as an alternative for the PI component of the second - line therapy if RTV - enhanced PI is not available or if there is a clinical contraindication to its use.

Important Note

Compliance plays an important part in ensuring maximal effect from a given regimen. The simpler the regimen, the easier it is for the patient to follow. It must be explained to the patient that ART requires high levels of adherence for long-term efficacy, most likely will need to be taken life long, and is associated with a range of short term and long term toxicities that can occasionally be life-threatening.

- HIV- DNA PCR or HIV- RNA PCR or immune complex dissociated p24 antigen assays, or HIV culture
- Initiation of ARV can also be considered for children who have advanced WHO pediatric stage II disease including such as severe recurrent or persistent candidiasis outside neonatal period, weight loss, fevers, recurrent severe bacterial infections, irrespective of CD4 count.

WHO recommendations for initiating Anti retroviral therapy in infants and children

AGE	CD4 count	HIV Diagnostic testing	Treatment recommendation
< 18 months	Available	Positive HIV virologic test HIV virologic testing notavailable but infant HIV seropositive or born to known HIV-infected mother. (Note: HIV Ab test must be repeated at age of 18 months)	WHO Paediatric stage IIIb (Irrespective of CD4 cell percentage WHO Paediatric stage I/II disease with CD4 percentage < 20%
> 18 months	Available	HIV antibody seropositive	WHO Paediatric stage IIIb disease WHO Paediatric stage I/II disease with CD4 percentage < 20%
< 18 months	Not available	Positive HIV virologic test HIV virologic testing not available but infant HIV Seropositive or Born to known HIV-infected mother.	WHO Paediatric stage III Treatment not recommended
> 18 months	Not available	HIV antibody seropositive	WHO Paediatric stage III

- The rate of decline of CD 4 percentage (if measurement available) should be factored into decision-making.
- Many of the clinical symptoms in the WHO Paediatric stage II and III disease classification are not specific for HIV infection in resource-limiting setting; thus, in the absence of virologic testing and CD4 cell assay availability, HIV-exposed infants < 18 months of age should generally not be considered for ART regardless of symptoms.

Recommend first-line antiretroviral regimens for children

Zidovudine (ZDV)

- ZDC/3TC is the first choice for children as it has the highest amount of clinical experience
- Other dual NRTI components can be substituted for children, including ZDV/ddl, d4T/ddl, and ddl/3TC. ZDV/d4t should never be used together due to proven antagonism.

Reasons for changing ART

- The reasons for changing ART regimen include **adverse drug effects**, **inconvenient regimens** such as dosing z/number of pills that may compromise adherence, **treatment failure**, **occurrence of active tuberculosis and pregnancy**.
- Treatment failure can occur due to a number of reasons. These include
  - * Unsatisfactory patient adherence
  - * Viral resistance to one or more drugs
  - * Impaired drug absorption, and
  - * Altered drug pharmacokinetics.
- The entire regimen should be changed from a first to a second line combination regimen in case of treatment failure. **A single drug should not be added or changed to a failing regimen**.
- The new second line regimen will need to use drugs which retain activity against the patient's virus strain and ideally include at least three new drugs, in order to increase the likelihood of treatment success.

Prevention of Parent to child transmission (PPTCT)

- Primary prevention of HIV infection among women of child bearing age
  - * By giving information, education and counselling on HIV prevention and care to the general population and couples
  - * Better STI management
  - * Reduction of unsafe transfusions
  - * Condom promotion: safe sex practice, and encouraging partner's involvement in safe sex

discussions.

- Prevention of unintended pregnancy in HIV infected women- information, education and counselling on HIV prevention and care including approach to MTCT prevention
  * Increasing the number of woman who know their HIV sero-status
  * Counseiling of woman and their partners to enable informed choice with regard to potential future pregnancy
  * Condom promotion as a valuable family planning tool.
- Women who test HIV-positive in early pregnancy can make the decision either to continue with the pregnancy or to elect for termination where this is legal and safe.

Occupational HIV exposure and HIV post-exposure prophylaxis (PEP)

PEP involves the management of health care workers who are possibly exposed to HIV. The most important management strategy is prevention of exposure. Nature of exposure and PEP recommendation are given below:

Nature of exposure and PEP recommendations

PEP recommended	PEP recommended	PEP not warranted
High-risk exposure	Intermediate-risk exposure	Low-risk exposure
Large bore hollow needle deep puncture	Less severe percutaneous exposure (e.g., solid needle)	Short exposure (< 1 minute)
Device in patient artery or vein	Mucous membrane exposure or non intact skin: volumes (e.g., a major blood splash) prolonged exposure (>1 minute)	Small volume (e.g. blood visible on device superficial scratch)

Rx Treatment

Management of PEP

- Treatment of exposure site with intermediate first aid
  * Wash skin with soap and water
  * Rinse eyes with eye wash fluid
  * Oral exposure- spit out immediately and rinse mouth several times.
- Prompt assessment of risk and HIV status of exposure source.
- Counselling of health care worker
  * Pretest counselling for hepatitis B and HIV; consider for HBV immunoglobulin and HBV vaccination if not HBV immune; not to give blood until outcome is known; review and post exposure counselling.
- Giving ART if indicated and counsel about adherence and follow up
- If intermediate risk or high risk- commence as soon as possible (preferably within 72 hours)
  * Three drug regimen AZT/3TC/Indinavir 3 times or
  * Nelfinavir or
  * Third drug can be an NNRTI (EFV is preferred over NVP)
- Documentation of the incident
  * Date and time of exposure
  * Details of the event
  * Exposure source
  * Details of PEP given
  * Follow up and outcome.

Tuberculosis and HIV infection

- Tuberculosis and HIV are closely interlinked.
- Tuberculosis is a leading cause of HIV related morbidity and mortality
- HIV is the most important factor fuelling the Tuberculosis epidemic in populations with a high HIV prevalence
- Tuberculosis can occur at any point in the course of progression of HIV infection
- High levels of MDR-TB in some areas threaten tuberculosis control efforts.
- In patients with HIV-related tuberculosis, the priority is to treat tuberculosis, especially smear-positive Pulmonary tuberculosis. However, patients with HIV-related tuberculosis can have ART and anti-Tuberculosis treatment at the same time, if managed carefully

- Careful evaluation is necessary in judging when to start ART. In case, e.g., disseminated tuberculosis and /or CD4 count < 200/mm^3, it may be necessary to start ARV concomitantly with tuberculosis treatment
- On the other hand, for a patient with smear positive pulmonary tuberculosis as the first manifestation of HIV infection, who does not appear to be at high risk of dying, it may be safe to defer ART until the initial phase of tuberculosis treatment has been completed and then use Ethambutol and isoniazid in the continuation phase
- This decreases the risk of immune reconstitution syndrome (temporary exacerbation of symptoms, signs or radiological manifestation of tuberculosis) and avoids the risk of drug interaction between rifampicin and protease inhibitors (PI).

Important Note

Anti-Tuberculosis drug treatment is the same for HIV positive and HIV negative tuberculosis patients, with one exception: **do not give thiacetazone to HIV positive tuberculosis patients** (increased risk of severe and sometimes fatal skin reactions).

severe and sometimes fatal skin reactions).

Opportunistic infections

Causes

- Tuberculosis
- Pneumocystis carinii pneumonia
- Esophageal candidiasis
- Cryptococcosis
- Toxoplasmosis
- Cryptosporidiosis
- Cytomegalovirus (CMV)
- Mycobacterium Avium Complex (MAC) disease

As the compromised immune system makes the individual vulnerable to a variety of illnesses, HIV infection may manifest itself in many different forms. Diagnosis and management of common opportunistic infections is given below.

Pneumocystis carinii pneumonia (PCP)

Pneumocystis carinii is a fungus that infects the lungs

Symptoms

- Sub-acute onset of symptoms over a period of weeks
- Typically fever, dry cough and progressive difficulty in breathing, also weight loss, night sweats and fatigue.

Investigations

- Diagnos is by
  - * X-ray
  - * Induced sputum examination or bronchoscopy
  - * CD4 count < 200
  - *

Treatment

Management of PCP depends on the degree and severity of disease:

- Severe disease—hospitalization, Intravenous TMP / SMX (15 mg/kg/day of TMP for 21 days), supplemental oxygen.
- Patients with severe hypoxemia (PaO2 <70-mmHg breathing room air at rest).,
  - * Should be given cortico-steroids Prednisolone 40 mg twice daily for 5 days followed by 40 mg once daily for 5 days, followed by 20 mg once daily for 11days.
- Moderate disease – an oral agent may be used and management can proceed on outpatient basis,although hospitalization should be considered.
- Recommended oral regimen: TMP/SMX (15 mg/kg/day of TMP) in divided doses for 21 days.
- Mild disease—Oral TMP/SMX as above.

Preventive therapy is indicated when CD4+ cell counts equal or are below (prophylaxis) 200 and / or symptomatic HIV

Alternative therapies

- Dapsone 100 mg once a day for 21 days—preferred second-line option.
- Clindamycin 450 mg 4 times a day + primaquine 15 mg once daily for 21 days.
- Atovaquone 750 mg once a day for 21 days
- Pentamidine (intravenous) 3-4 mg/kg/day for 21 days

Toxicities of treatment

- TMP/SMX hypersensitivity (typically fever and maculopapular rash)
- Nausea and vomiting
- Bone marrow toxicity
- Hepatitis

Stopping maintenance

- There is some evidence that it may be possible to stop maintenance therapy if CD4 counts stay above 200 on anti retro therapy. However there is insufficient data to make current recommendations.

Oesophageal candidiasis

Candidiasis is a fungal infection that frequently occurs in the mouth and vagina. It is considered to be an opportunistic infection when it occurs in the Oesophagus.

Symptoms

- Difficulty in swallowing or retrosternal discomfort
- Weight loss is common
- Presentation - sub acute over weeks
- CD4 count < 100
- Usually made clinically in presence of oral candidasis and dysphagia.
- Endoscopy is indicated only in those who fail to respond to a clinical trial of appropriate treatment.
- The diagnosis of esophageal candidiasis should be reconsidered if oral candidiasis is not present.
- Associated fever and oral ulceration are not common.

Treatment

- Preventive therapy:Not recommended because current drugs effectively treat (prophylaxis) disease.
- Antifungal resistance may develop and drug-drug interactions may occur
- Drugs
  - Flucanazole 100 – 400 mg once a day for 2 weeks is the treatment of choice
- Alternative treatment
  - Amphotericin-B 0.3 – 0.5 mg/kg/day.
- Maintenance Therapy
  - Fluconazole (50 – 100 mg once a day)
- Stopping maintenance therapy
  - There is evidence that patient who achieves CD4 counts > 100 on ART may cease treatment

Cryptococcosis

Cryptococcus is a fungus that is inhaled and that has a predilection for meninges (brain linings)

Symptoms

- Meningitis
  - Headache
  - Nausea
  - Fever
  - Malaise
  - Altered mental status
  - Irritability and
  - Seizures
- Lung involvement may co-exist
  - Cough
  - Chest pain
  - Breathlessness
- Presentation
  - Sub - acute with progressive symptoms over weeks to months or acute with symptoms over days
- CD4 count: <100

Investigations

- Usually by lumbar puncture to test for the presence of cryptococcus or cryptococcal antigen in cerebral spinal fluid. ICP is often raised.
- CSF protein and glucose are generally normal and there may be few white blood cells.

Treatment

- Preferred
  - Amphotericin B (0.5 – 0.8 mg/kg/day) + Flucytosine (100 mg/day) 4 times a day) for 2 weeks then 8 to 10 weeks.

- Alternative Treatment
  * Liposomal Amphotericin
- Maintenance therapy
  * Flucanazole 200 mg once a day
  * Pregnant woman should avoid azole drugs
- Stopping Maintenance
  * Not recommended currently because of few available studies
  * Maintenance Therapy
    » Cohort studies suggest that maintenance therapy can be ceased in patients with sustained CD4 response to ART (CD4>200) for >3 months.

Toxoplasmosis

Toxoplasmosis is a parasite that has a predilection for the brain.

Symptoms

- Altered mental state (confusion, unusual behavior),
- Headaches
- Fever
- Seizures
- Paralysis
- Coma
- Presentation : acute to sub acute over days to weeks
- CD4 count < 100

Investigations

- Typical appearance on CT or MRI scan.Diagnosis is usually presumptive on the basis of appearance on scan. **May show ring shaped contrast enhancing lesions.**
- If no response to empirical anti-toxoplasmosis therapy after 2 weeks then consider brain biopsy, to rule out CNS lymphoma.
- Preventive therapy is indicated when CD4+ cell count is below 200 (for primary PCP prophylaxis)
- Preferred
  * TMP SMX (1 double - strength every 12 hours three times a week: or
  * A single strength or 1 double - strength tablet once a day.
- Stopping preventive therapy
  * CD4+ cell count above 200 for over 3-6 months.

Treatment

- Pyrimethamine 100 – 200 mg loading dose and then 50 – 75 mg once a day given in combination with sulphadiazine 4 – 6 g/day, 4 times a day or Clindamycin 2 – 4 g/day, 4 times a day for 6 – 8 weeks duration depending upon response
- If Sulphadiazine is used then Folinic acid25 mg once a day should be given to prevent haematological toxicity.
- Corticosteroids may be used in the presence of cerebral edema

Alternative treatment

- Pyrimethamine in combination with one of the following:
  * Azithromycin 1 – 1.5 mg/day Atovaquone 3 g/day
  * Dapsone 100 mg/day
  * Clarithromycin 2 g/day

Maintenance therapy:

- Pyrimethamine (25 – 75 mg once a day) + Sulphadiazine (500 – 100 mg 4 times a day for several days with Leucoverin)

Stopping maintenance therapy:

  * Not recommended currently

Cryptosporidiosis

Cryptosporidiosis is a parasite that infects gastro intestinal tract

Symptoms

- Chronic diarrhoea with frequent watery stools
- Abdominal cramps
- Nausea and Vomiting
- Fatigue
- Weight loss
- Loss of appetite
- Fever
- Dehydration

- Electrolyte imbalance (especially sodium and potassium)
- Presentation time frame: acute to chronic presentation over days to weeks or months in some cases
- CD4 count<100

Investigations

Stool examination for detection of oocysts in the stool, on biopsy of small intestine. A specific request for examination for cryptosporidiosis is required (specific lab techniques are needed)

- Preventive therapy: There are no proven effective therapies. (Prophylaxis)
- There is no good evidence that boiling water or the use of water filters prevents the disease.

Treatment

- There are no proven effective therapies
- Symptomatic treatment includes Loperamide, codeine, and Somatostatin analogues.
- Immune recovery induced by ART alone results in excellent clinical responses.
- Maintenance Therapy: There are no proven therapies that prevent Cryptosporidiosis

Important Note

Drug interactions between ARV drugs and drugs used to prevent or treat opportunistic infections:

- Trimethoprim-sulphamethoxazole can give additive hematological toxicity when given together with Zidovudine.
- Antifungal drugs such as Ketaconazole and Flucanazole may inhibit the metabolism of Protease inhibitors. This may result in increased serum levels of Protease inhibitors and increased risk of toxicity.

References

- Infection in the immunocompromised host. In: Oxford Textbook of Medicine, Warrell DA. Cox TM, Firth JD, Benz EJ Jr. (eds), 4th edition, 2003, oxford university press,pp 1.864-1.870
- Diagnosis and treatment of fungal infections. In: Harrison's principles of medicine. Kasper DL, Braunwald E, Fauci AS et al (eds), 16th edition, McGraw-Hill Company Inc., Newyork, pp 1176 – 1179
- Pneumocystis carinii infections. . In: Harrison's principles of medicine. Kasper DL, Braunwald E, Fauci AS et al (eds), 16th edition, McGraw-Hill Company Inc., New York, pp 1194 – 1196
- Programme implementation Guidelines for a phased scale up of access to Antiretroviral Therapy (ART) for people living with HIV/AIDS. National AIDS Control Organization. Ministry of Health & Family Welfare, 2004.
- WHO SEARO Training modules on HIV/AIDS care and Antiretroviral treatment, 2003.
- TB/HIV: A clinical Manual WHO Second Edition, 2004.

Typhoid or enteric fever

Causes

It is caused by Salmonella typhi and Salmonella paratyphi. Salmonella typhi causes a variety of illnesses including

- Asymptomatic carrier state
- Gastro-enteritis
- Enteric fever

Symptoms

- The onset of fever is typically gradual, continuous (temperature upto 40°C) with constitutional symptoms such as
  - Malaise
  - Anorexia
  - Lethargy
  - Headache
  - Constipation or diarrhoea (pea-soup stools) which may be associated with
    - Abdominal pain and tenderness
  - Hepatomegaly
  - Splenomegaly and
  - Change in mentation
- Usually the patient is sick and has a toxic appearance with a coated tongue and a soft splenomegaly.
- Examination may reveal toxic signs with relative bradycardia and mild soft splenomegaly

Investigations

- Diagnosis is suggested by
  - Rising titres of O antibodies (widal test) and
  - Confirmed by isolation of organism in blood, bone marrow, urine or stool.
- Complications such as
  - Hepatitis
  - Peritonitis
  - Meningitis
  - Pneumonitis and
  - Myocarditis can occur usually after first week.

Treatment

Non-pharmacological

- Adequate nutrition and hydration should be maintained ensuring adequate intake either orally or with intravenous fluids (in severely ill)
- Inpatient treatment is recommended if patient is very sick, not accepting orally with inadequate urine output, patient has altered sensorium/ drowsiness or is having very high pyrexia particularly in the second week of illness when the risk of complications increases or if the complications have already ensued.

Pharmacological

- Management of fever
- Antipyretics can cause precipitous fall in temperature and even shock in enteric fever. They should be used judiciously. Therefore hydro-therapy is preferred for fever.

Specific therapy

Multi-drug resistance is prevalent. Antibiotics are recommended on the basis of available culture and sensitivity pattern or epidemiological data.

- Tab. Ciprofloxacin 10 mg/kg in 2 divided doses upto a maximum of 750 mg twice daily for 10-14 days (for 1 week after the fever subsides).
- Tab. Ofloxacin 200-400 mg daily for 5-7 days.
- **If no response after 5 days**
  - Inj. Ceftriaxone 50-60 mg/kg/day IV or IM in 2 divided doses or as a single dose for 7-10 days (preferred in pregnant women patients, children or patients resistant to quinolones) or Tab. Cefixime 200-400 mg daily as a single dose or 2 divided doses for 14 days.
- **If there is no response after 5 days, alternative diagnosis should be considered**
  - Report to physician if abdominal symptoms worsens or occurrence of bleeding per rectum or alteration in sensorium and shock (severe typhoid with high risk of fatality)
- Severe typhoid with shock or patients with enteric encephalopathy should be hospitalized and treated as above plus Inj. Dexamethasone 3 mg/ kg IV first dose followed by 1 mg/kg IV every 6 hourly for 8 doses.
- **Chronic carrier state (**patients who continue to excrete bacilli in stool for more than 1 year)
  - Tab. Ciprofloxacin 750 mg twice a day for 28 days.

Assessment of response to therapy

- The toxic look of the patient decreases and appetite starts returning in 72-96 hours of treatment and gradually fever subsides, touches the baseline for increasing duration. The fever may take as long as 7 days to respond.
- Sometimes the patient may apparently appear to have responded whereas patient may be developing impending shock due to complications. So a careful clinical examination has to be done if there is a precipitous fall in temperature.

Modification or step up therapy if required

- The patient should be monitored for complications and usual indications for inpatient treatment are
  - Myocarditis
    - Fall in perfusion and blood pressure
    - Arrhythmias
  - Altered sensorium
  - Shock
    - Tachycardia
    - Cold clammy skin
    - Diaphoresis
    - Hypotension
  - Perforation peritonitis
    - Acute pain in abdomen

» Guarding, rigidity

» Hypotension

» Bilious vomiting

- In case the patient worsens in 4-7 days, as discussed above, a change in antibiotics is suggested, preferably on the basis of culture and sensitivity report, where available.

Patient/Parent education

- Small frequent feeds should continue. Give plenty of oral fluids and compensate for increased fluid loss from the body due to high-grade fever.
- The treatment should be given till the patient has an afebrile period of 7 days as incomplete treatment increases the risk of relapse and emergence of resistance.
- The caregivers must be informed about the complications.
- Ciprofloxacin and Ofloxacin are very bitter and cause severe nausea and gastritis. Patients should be asked to report any missed dose due to vomiting.
- Three types of vaccines are available for this disease. Readers are requested to refer standard textbooks.

References

1. Salmonellosis: Harrison's principles of Internal Medicine, Kasper DL et al, 16th edition, 2005, McGraw Hill company, New York, pp 897-902
2. Textbook of Paediatric infectious diseases. WB Saunders Co Philadelphia
3. American academy of Paediatric in: Report of the committee on infectious diseases, 25th edition, 2000, Elk Grove Village, Illinois, USA.
4. Salmonella in: Nelson's textbook of Paediatric. Behrman RE, Kleigman, 17th edition, 2004, pp 912-919.

Leptospirosis

Causes

Leptospirosis is an infectious disease caused by the spirochaete Leptospira interrogans.

Symptoms

- Rodents are the most important reservoirs.
- Leptospires may enter the host through abrasions in the skin or through intact mucosa, especially the conjunctiva and the lining of oro- and nasopharynx when they come in contact with water contaminated with leptospira.
- 90% of symptomatic persons have the relatively mild and the usual anicteric form of Leptospirosis, with or with out meningitis.
- Severe Leptospirosis with profound jaundice – **Weil's syndrome** –occurs in 5 to 10% of patients.
- The most common finding in anicteric Leptospirosis is
  - * Fever with conjunctival suffusion
  - * Less common findings include
  - * Muscle tenderness
  - * Lymphadenopathy
  - * Pharyngeal injection
  - * Rash
  - * Hepatomegaly and
  - * Splenomegaly
- Mild jaundice may be present
- Rarely meningitis can occur
- Weil's syndrome, the most severe form of Leptospirosis is characterized by jaundice, renal dysfunction, hemorrhagic diathesis, and a mortality rate ranging from 5 to 15 %.

Investigations

Laboratory support is needed:

- To confirm the diagnosis
- For epidemiological and public health reasons to determine which serovariant caused the infection, the likely source of infection and potential reservoir and its location. The tests used depends on the phase of the infection. During leptospiremic phase (<10days) leptospires inoculate in the blood and can be isolated by blood culture and PCR while in the immune phase, rising antibodies can be detected by serological tests.

Culture

The isolation of leptospirosis by culture of blood, CSF and urine is the most definite way of confirming the diagnosis of leptospirosis. Unfortunately, culture of

blood does not contribute to an early diagnosis as results come late, weeks or even months after inoculation of culture medium.

PCR

PCR is promising on both sensitivity and specificity but is complicated and expensive. Its value for rapid diagnosis is not seen and evaluated and not widely used.

Serology:

The serological tests for diagnosis of leptospirosis have been classified as serovar specific tests and genus specific tests.

Serovar specific tests

1. Microscopic agglutination test (MAT)
2. Genus specific tests

Microscopic agglutination test (MAT)

MAT is the gold standard test for diagnosis of leptospirosis because of its unsurpassed diagnostic specificity. The main advantage is that serovar can be identified, which is of epidemiological importance.The difficulties in utilizing MAT are due to the following factors.

- The antibody titres rise and peak only in second or third week, making it a less sensitive test.
- The high titres of past infection persist for a long time (1-5years) and therefore interfere with the diagnosis of current leptospirosis. Positive titres may represent a rising titre of current infection or declining titre of past infection.
- The cut off titre for diagnosis of current infection depends on whether the area is endemic or non-endemic, for example the cut off titre varies from 1/80 to1/400.Therefore a second sample is usually required. (To demonstrate 4 fold rising titre.) To diagnose current infection, seroepidemiological studies are required for determining the cutoff value
- The test is complicated requiring dark field microscopy and cultures of various live serovars. This may not be available in small laboratories.

Genus specific tests

- The two common tests are the ELISA and macroscopic slide agglutination tests (MSAT). The other tests are latex agglutination test, complement fixation test and haemagglutination tests
- The genus specific tests are the test of choice for the diagnosis of current infection. These tests are simple and more sensitive and become positive earlier than MAT, only as single antigen is used, the genus specific antigen
- These tests detect genus specific antibodies, which is shared by pathogenic and saprophytic leptospira
- These test become positive early in the disease (5-6th day). **Detection of specific IgM antibodies helps in rapid diagnosis of current infection.**

ELISA: These are popular tests and can be performed with commercial kits or with antigen preparation "in house".

MSAT: The slide agglutination test is a simple macroscopic test in which a drop of the dense suspension of leptospira is mixed with drop of serum on a slide is examined by the naked eye for agglutination.

Labaratory Criteria For Diagnosis Of Current Leptospirosis

- Culture
  - * Positive
- MAT
  - * Seroconversion / Four fold rise in the titre
  - * High titre.
- ELISA / MSAT
  - * Positive.

Rx Treatment

Chemotherapy:

The aims of chemotherapy are to eradicate leptospirosis and to prevent complications. Leptospirosis is sensitive to most antibiotics.

- Penicillin is the most effective antibiotic when given early. In severe illness large doses (6—8million units per day) of Benzyl penicillin may be given in divided doses, preferably by IV route, for 5-7days. Fever subsides in 24-36 hours.
- Ampicillin 1g IV qid in severe illness or 500-700mg qid in mild illness.
- Doxycycline 200mg/day, Amoxycillin 500mg qid and Erythromycin 250mg qid are effective.
- Quinolones and Cefotaxime are also effective

against leptospira. Recently there is evidence to suggest that antibiotics are useful even in the late stages of illness.

Symptomatic and supportive treatment:

- The primary importance is the meticulous attention to fluid and electrolyte balance. Hypovolemia and hypotension need prompt and specific treatment with intravenous fluids
- In patients with oliguria, if pre-renal azotemia is suspected, prompt diuresis should be attempted with fluid therapy
- Patients who have no response to therapy should be managed as established renal failure
- Headache and myalgia are treated with analgesics. Fever with anti pyretic
- Restlessness and anxiety with sedatives and anaemia with blood transfusion.

Peritoneal dialysis has been found to be a safe, simple and effective procedure for management of leptospira renal failure due to leptospirosis

If there is contraindication to peritoneal dialysis, hemo-dialysis can be done.

Reference

- Harrison's Text Book of Medicine, 16th edition, Kasper et.al., Mc Graw Hill company inc. in:pp-988-990.
- Oxford Text Book of Medicine .pp: 478-479.
- Manson Bahr Text Book of Tropical Medicine. Pp: 822-826

Parasitic infections
Intestinal protozoal infections

Amoebiasis and Giardiasis are the commonest intestinal protozoal infections. Patients of Amoebiasis and Giardiasis present as asymptomatic carriers.

Amoebiasis (Intestinal)

Causes

Infection is caused by intestinal protozoa- Entamoeba histolytica. Infection usually spreads by infective cysts in stool, which contaminate food and drinking water.

Symptoms

- Lower abdominal pain
- Mild diarrhoea develops gradually and may lead to full blown dysentery. 0-12 stools per day with blood and mucous and little faecal matter.
- Caecal involvement may mimic acute appendicitis.
- Chronic form i.e., amoebic colitis, can be confused with inflammatory bowel disease. Other form of **chronicity may present as amoeboma**
- Untreated or incompletely treated intestinal infection may result in amoebic liver abscess and involvement of other extra intestinal site.

Investigations

- Diagnosis made by demonstration of cysts and/or trophozoites of Entamoeba histolytica in the stool.

Treatment

Asymptomatic cyst passers

Tab. Diloxanide furoate 500 mg 8 hourly for 10 days.

Acute amoebic dysentery and chronic infections

1. Tab. Metronidazole 400-800 mg 8 hourly PO for 10 days.In children 15 mg/kg divided in three doses for 7 days. (o r) Tab. Tinidazole (300 mg, 500 mg and 1 g) 2 g orally as single dose.In children 50 mg/kg as a single dose.
2. Tab. Diloxanide furoate 500 mg 8 hourly for 10 days.In children 20 mg/kg/day in three divided doses for 10 days.

For treatment of amoebic liver abscess see chapter on gastrointestinal diseases

Giardiasis

Causes

Intestinal disease caused by protozoal parasite – Giardia lamblia. The disease spreads by direct faeco-oral transmission.

Symptoms

- Acute Giardiasis – although diarrhoea is common, upper intestinal manifestations such as abdominal pain, bloating, belching, flatus, nausea and vomiting may predominate.
- Chronic Giardiasis – history of one or more epi-

sodes of acute diarrhoea, increased flatus, loose stools, abdominal distension, borborygmi, eructation of foul tasting gas and passage of foul smelling flatus, and weight loss

- Symptoms could be intermittent, recurring and gradually debilitating; severe disease may result in malabsorption, weight loss, growth retardation and dehydration.

Investigations

- Diagnosis is made by the demonstration of cysts and or trophozoites of G.lamblia in the stools.

Treatment

- Tab. Tinidazole 2 g as a single dose in adults.
- In children 50 mg/kg as a single dose.

(or)

- Tab. Metronidazole 400 mg every 8 hours for 7 days in adults.In children 15 mg/kg divided in three doses for 7 days.

Patient education

- These infections spread by ingestion of food or water contaminated with cysts.
- Properly cooked food, use of clean drinking water, proper sanitation and good personal hygiene-hand washing with soap after defecation and before meals may prevent infection.
- Avoiding unpeeled fruits and vegetables may minimize infection.
- Side effects are usually mild and transient and include nausea, vomiting, abdominal discomfort, metallic taste and a disulfiram like reaction, therefore, avoid use of alcohol during treatment.

References

- Infectious Diseases: Protozoal and Helminthic. In: Current Medical Diagnosis and Treatment. Lawrence M Turner Jr, Stephen J McPhee, Maxine A Papadakis, 38th edition, Prentice Hall International Inc. USA, pp 1353-1417
- Drugs used in the chemotherapy of Helminthics. In: The Pharmacological basis of therapeutics. Martin J Wonsiewich and Peter MC Curdy, 9th edition, 1996, Mc Graw Hill Company Inc., USA, pp 1009-1026
- Drugs used in the chemotherapy of Protozoal infections Trypanosomiasis, Leishmaniasis, Basis of therapeutics. Martin J Wonsiewich and Peter MC Curdy, 9th edition, 1996, Mc Graw Hill Company Inc., USA, pp 987-1008.
- Parasitic causes of acute diarrhoea. In: Davidson's principles and practice of medicine. Haslett C Chilvers et al, 19th Edition, 2002, Churchill Livingstone, pp 46

Malaria and National anti-malaria programme (NAMP)

Causes

- Parasitic infection due to protozoa of genus plasmodium transmitted by the female anopheles mosquito. There are four plasmodia species:
  - Plasmodium falciparum
  - Plasmodium vivax
  - Plasmodium malariae
  - Plasmodium ovale

Symptoms

- Malaria is an acute and chronic protozoan illness characterized by paroxysms of fever, chills, sweats, fatigue, anaemia and spleenomegaly.
- Falciparum malaria (severe and complicated malaria) is associated in varying degrees with the following clinical signs
  - Cerebral malaria
    - Mental clouding
    - Coma
    - Convulsions
    - Delirium, and occasionally localizing signs.
    - Hyper pyrexia (>40.5c)
    - Haemolysis
    - Oliguria
    - Anuria
    - Pulmonary edema
    - Macroscopic haemoglobinuria.

Investigations

Diagnosis is made by presence of protozoa in the blood in thick and thin smear slides. Thick smear for easy detection of parasite and thin smear for identification of species.

Note:

That blood films may be negative even in a severe attack because of parasites in the deep capillaries.

Rx Treatment

- All fever cases without any other obvious causes should be presumed as malaria cases and antimalarial drug be given preferably after taking blood smear.
- Chloroquine is the first line antimalarial in the treatment of uncomplicated malaria.
- In high-risk areas **presumptive treatment** 25 mg/kg of chloroquine base is to be given over 3 consecutive days (10 mg/kg on day 1 and 2, followed by 5 mg/kg on day 3) with a single dose of primaquine 0.75 mg/kg on the first day. High risk area is defined as follows:
  - Recorded deaths due to malaria (on clinical diagnosis or microscopic confirmation) with P.falciparum infection during the transmission period in an endemic area during any of the last 3 years.
  - Doubling of slide positivity rate (SPR) during the last 3 years provided the SPR in second or third year reaches 4% or more, or the average SPR of the last year is 5 % or more.
  - P.falciparum is 30% or more provided SPR is 3% or more during any of the last 3 years.
  - An area having a focus of chloroquine resistant. P.falciparum.
- In the low risk areas, presumptive treatment with chloroquine 10 mg/kg single dose.
- Resistance should be suspected if in spite of full treatment and no history of vomiting and diarrhoea, patient does not respond within 72 hours parasitologically. Such patients should be given alternative drug i.e., sulfa-pyrimethamine combination (S-P) and reported to concerned district/ state malaria officer for monitoring of drug sensitivity status.
- Sulfadoxime pyrimethamine is the second line of antimalarial in P.falciparum resistant to chloroquine. The dose is 25 mg/kg of sulfadoxime + 1.25 mg/kg of Pyrimethamine which is 3 tablets for adult (single dose)
- The dose of primaquine for P.vivax cases is 0.25 mg/kg daily for 5 days to prevent relapse and for P.falciparum 0.75 mg/kg single dose for gametocytocidal action. A 14 days primaquine treatment in P.vivax is NOT recommended in the programme.
- Mefloquine can be given to chloroquine or other antimalarial resistant uncomplicated P.falciparum cases only.
- Resistance to chloroquine
  - There must be evidence of falciparum positive blood slide on the first and third days of treatment.

WHO classification of resistance to Chloroquine

- RI: Total disappearance followed by reappearance of the parasite.
- RII: Noticeable fall without disappearance of the parasite.
- RIII: Parasite level almost unchanged, indeed increased

Important Note

Before labeling resistance verify that

- Treatment has in fact been taken.
- Correct dose for weight has been prescribed.
- Patient has not vomited within 30 minutes of taking medication.
- There has not been under dosage due to confusion in the expression of dosage as a chloroquine base and as a chloroquine salt. Equivalence between salt and base: 130 mg sulphate = 150 mg phosphate or diphosphate = 100 mg base.

- In pregnant woman and infants, primaquine is contraindicated. As no data is available to suggest the safety of Artemisinin derivatives in this group, the same cannot be recommended.
- In severe and complicated P.falciparum malaria cases Quinine iv/ Artemisinin derivatives (for adults and non pregnant women only) is to be given irrespective of chloroquine resistance status. In case of non-availability of the above drugs, chloroquine 10 mg/kg in isotonic saline should be infused over 8 hours followed by 15 mg/kg in the next 24 hours. The treatment is to be continued till such time Quinine /Artemisinin derivatives become available.

- The use of Artemisinin derivatives is to be restricted and injections may be used for the treatment of severe and complicated P.falciparum malaria in adults and non-pregnant women only. However oral forms of these derivatives may be used for falciparum malaria resistant to both chloroquine and sulfa-pyrimethamine combination.

Rx Treatment

Patients with uncomplicated malaria can be managed at a primary level but patients with complicated malaria should be admitted and managed in a hospital where facilities for detailed investigation and blood transfusion exist.

- Presumptive treatment in uncomplicated malaria
  - **Low risk area:** with chloroquine 10 mg/kg single dose (maximum dose is 600 mg) to all suspected cases.

High-risk area

Day 1	Chloroquine (600 mg adult)	10 mg/kg
	Primaquine (45 mg adult)	0.75 mg/kg
Day 2	Chloroquine (600 mg adult)	10 mg/kg
Day 3	Chloroquine (300 mg adult)	5 mg/kg

- **Confirmed cases of Malaria**
  - Tab. Chloroquine as in presumptive treatment in "high risk area"
  - In P.vivax Tab. Primaquine 0.25 mg/kg/day for 5 days.
  - In P. falciparum Tab. Primaquine 0.75 mg/kg as a stat dose
  - In high risk areas where presumptive treatment with 500 mg Chloroquine and 45 mg Primaquine has been given chloroquine need not be administered again but primaquine must be given for 5 days.
- Chloroquine resistant P. falciparum cases In Chloroquine resistant P. falciparum cases, second line treatment must be given with 25 mg/kg of sulfadoxine + 1.25 mg/kg of pyrimethamine which is 3 tablets for adult (single dose) followed by primaquine (45 mg)
- In severe and complicated Malaria cases
  - In severe and complicated Malaria cases, irrespective of chloroquine resistance status of the area: Inj. Quinine salt 10 mg/kg 8 hourly IV in 5 % dextrose saline is preferred. Patients should be switched over to oral quinine as early as possible with oral dose 10 mg/kg 8 hourly not exceeding 2 g in a day in any case.
  - Minimum total duration for quinine therapy should be for 7 days including both parentral and oral doses. Or Injectable form of Artemisinin derivatives may be used in non-pregnant adults and G-6-PD deficiency.
  - Any of the following Artemisinin derivatives, dosages as follows:
    - Inj. Artemisinin: 10 mg/kg at 0 and 4 hours followed by 7 mg/kg at 24, 36, 48, and 60 hours.
    - Inj. Artesunate: 2.4 mg/kg IM/IV followed by 1.2 mg/kg after 12 hours then 1.2 mg/kg once daily for total duration of 5 days.
    - Inj. Artemether: 3.2 mg/kg stat followed by 1.6 mg/kg daily for 3-5 days.
    - Inj. Artether: 150 mg daily IM for 3 days in adults only.

Supportive treatment

Treatment of fever, hypoglycemia, electrolyte imbalance, hypotension, renal failure, anaemia, and convulsions appropriately.

Chemoprophylaxis in selective cases

- Chemoprophylaxis is recommended for
  - Pregnant women in high-risk areas and
  - Travellers including service personnel who temporarily go on duty to highly malarious areas.
- Chemoprophylaxis is to be started a week before arriving to malarious area for visitors and for pregnant women prophylaxis should be initiated from second trimester.

Chloroquine sensitive area

- Start with a loading dose of Tab. Chloroquine 10 mg/kg followed by a weekly dose of 5 mg/kg. This is to continue till 1 month after delivery in case of pregnancy and travellers till one month after return from endemic area
- The terminating dose should be 10 mg/kg along

with 0.25 mg/kg of primaquine for 5 days.

- **Caution:** In pregnancy, primaquine should not be given
- Chemoprophylaxis with chloroquine is not recommended beyond 3 years because of its cumulative toxicity.
- In chloroquine resistant areas chloroquine 5 mg/kg weekly and **proguanil** 100 mg daily.

Patient education

- To take measures to stop mosquito breeding and protection from mosquitoes.e.g., mosquito nets, repellents, long sleeves, long trousers.
- Fever without any other signs and symptoms should be reported to nearest health facility.
- Chloroquine should be given with plenty of water after food and not on an empty stomach. If chloroquine syrup is not available for children, the tablet form should be crushed and given with honey and thick syrup.
- Watch for side effects of drugs prescribed. Chloroquine may cause nausea, vomiting, diarrhoea, mild headache and skin allergy/rash.
- If vomiting occurs within 30 minutes of chloroquine intake repeat the dose of chloroquine.
- **Chloroquine, Primaquine and Sulpha-pyremethamine should not be given in patients with G-6-PD deficiency.**
- Patients should be educated about cerebral malaria, and should seek medical help immediately on occurrence of these symptoms.

Reference

- National Antimalaria Programme (NAMP) drug policy, Government of India, DGHS, Directorate of National Antimalaria Programme. 22-Shamnath Marg, New Delhi.
- Malaria (Plasmodium) in: Nelson's text book of Paediatrics, Behrman RE, Kleigman RM, HB (eds), 17th edition, 2004, Harcourt Publishers International Company, pp 1139-1143.

Worm infestation

Hookworm Infestation

The majority of worm infestations are asymptomatic.

Causes

Infection is caused by A.duodenale and N.americanus. The infective larvae penetrate through skin usually the foot and travel through subcutaneous tissue to the intestines. The adult forms live in the jejunum and feed on blood, thus leading to chronic blood loss and anaemia.

Symptoms

- Most of the affected individuals may be asymptomatic
- Patients usually present with symptoms of anaemia (hypochromic microcytic)
- Pruritic maculopapular dermatitis (ground itch) at site of skin penetration by infective larvae.
- Serpigenous tracts of subcutaneous migration in previously sensitized host.
- Mild transient pneumonitis because of larvae migration through lungs.
- Intestinal manifestations – epigastric pain often with post-prandial accentuation, inflammatory diarrhoea.
- Major consequences – progressive iron deficiency anaemia and hypoprotenemia.

Investigations

- Diagnosed by demonstration of ova of A.duodenale and or N.americanus in the stool and occult blood.

Treatment

- Tab. Mebendazole 100 mg 12 hourly for 3 days in children above 2 years of age. (Caution: contraindicated in children less than 2 years) or
- Tab. Pyrantel palmoate (250 mg); syr. (250 mg/5 ml) 10 mg/kg body weight once daily for 3 days. (Caution: not recommended in children below 1 year of age)
- In children more than 1 year Susp. Pyrantel palmoate 10 mg/kg as a single dose. Or Tab. Albendazole 400 mg to be chewed as a single dose.
- In children between 1-2 years of age syr. Albendazole 200 mg as a single dose. In children more than 2 years syr. Albendazole 400 mg as a single dose.

Patient education

- Hookworm infestation occurs through skin penetration by the infective larvae.
- The disease can be prevented by use of boots and gloves while working in the fields.
- The deworming agents should not be used in pregnancy, lactation and along with alcohol.
- Side effects of these drugs are generally mild which may include nausea, abdominal pain, headache, dizziness, malaise and skin rash.

Ascariasis (Roundworm infestation)

Causes

Ascariasis is caused by Ascaris lumbricoides, the largest nematode parasite of humans reaching upto 40 cm in length. The worm is located in the large intestine.

Symptoms

- Most infected individuals have low worm burden and are asymptomatic.
- Features of pulmonary involvement because of larval migration include irritating non-productive cough, bronchospasm or pneumonitis and burning substernal discomfort aggravated by coughing or deep inspiration, dyspnoea, fever, eosinophilic pneumonitis.
- Heavy intestinal infection- pain in abdomen, small bowel obstruction that may get complicated by perforation, intussusception or volvulus
- Aberrant migration of a large worm may cause
  - * Biliary colic
  - * Cholangitis
  - * Cholecystitis
  - * Pancreatic and
  - * Oral expulsion of the worm.

Treatment

- Tab. Mebendazole 100 mg 12 hourly for 3 days in children above 2 years of age. (**Caution:** contraindicated in children less than 2 years) or
- Tab. Pyrantel pamoate 11 mg/kg as a single dose. or
- Tab. Albendazole 400 mg as a single dose.
- In heavy infestation, however, a 2-3 day course is indicated. (**caution:** contraindicated in pregnancy)
- In children between 1-2 years of age syr. Albendazole 200 mg as a single dose. In children more than 2 years, syrup. Albendazole 400 mg as a single dose.

Note

Nasogastric suction, IV fluids, and instillation of piperazine through nasogastric tube may manage partial intestinal obstruction. Complete obstruction and other surgical complications require surgical referral for intervention.

Patient education

- Infection occurs by faecal-oral route.
- Proper sanitation and good personal hygiene-hand washing with soap after defecation and before meals may prevent infection.
- Infection can be minimized by avoiding unpeeled fruits and vegetables and use of clean drinking water.

References

- Infectious Diseases: Protozoal and Helminthic. in Current Medical Diagnosis and Treatment. Lawrence M Turner Jr, Stephen J McPhee, Maxine A Papadakis, 38th edition, Prentice Hall International Inc. USA, pp 1353-1417
- Drugs used in the chemotherapy of Helminthics. in the Pharmacological basis of therapeutics. Martin J Wonsiewich and Peter MC Curdy, 9th edition, 1996, Mc Graw Hill Company Inc., USA, pp 1009-1026
- Drugs used in the chemotherapy of Protozoal infections Trypanosomiasis, Leishmaniasis, Basis of therapeutics. Martin J Wonsiewich and Peter MC Curdy, 9th edition, 1996, Mc Graw Hill Company Inc., USA, pp 987-1008.
- Helminthes not associated with eosinophilia. In: Davidson's principles and practice of medicine. Haslett C Chilvers et al, 19th Edition, 2002, Churchill Livingstone, pp 72-73.

Filariasis

Causes

- Lymphatic filariasis results from infection of

* Wucheriria bankcroftii
* Brugia malayi
* Brugia timori

Symptoms and signs

- Manifests in both acute and chronic form with or without fever.
- Adeno-lymphangitis
  * Hydrocele
  * Elephantiasis
  * Chyluria
  * Involvement of spermatic cord-Funiculitis, Lymphocoele, Epi-didymoorchitis
  * Tropical Pulmonary Eosinophilia (TPE)
  * Abscesses
  * Mono-articular arthritis
- Suspected when a patient presents with a
  * Sudden onset of fever
  * Acute groin pain with swollen tender lymph gland and
  * Oedematous swelling of the legs.

Investigations

- Blood examination
- Thick film: Blood is taken at the time when peak concentration of microfilaria is expected i.e., **between 8:30 PM and 12 mid night**
- Membrane filter concentration (MFC) methods
  * Most sensitive method for low-density microfilaremia.
- DEC provocation test
  * Microfilaria can be induced to enter the blood stream during daytime. Di-Ethyl- Carbazine (DEC) 100 mg orally is given and blood is drawn after 15-20 minutes.

Treatment

Medical treatment

- Diethylcarbazine (DEC) – 6 mg/kg/day – in divided doses for 12 days
- Treatment is repeated about every 6 months for as long as the person remains microfilaremic or has symptoms.
- For Tropical Pulmonary Eosinophilia: 3 week course of DEC is given.
- Symptomatic treatment: for treating acute attacks of filarial fever and lymphadenitis
  * Analgesics
  * Antipyretics and
  * Bed rest
- Management of lymph-edematous limbs:
  * Elevation of affected limb.
  * Elastic bandage, stocking, massage.
  * Prevention of superficial bacterial/fungal infections.

Surgical treatment

Prior to any surgical procedure, a course of DEC is recommended.

Chronic hydrocoele – Excision and eversion of sac.

Scrotal elephantiasis – Surgical removal of grossly elephantoid skin and scrotal tissues with preservation of penis and testicles.

Limb elephantiasis – Lympho-venous procedures followed by removal of excess of subcutaneous and fatty tissue from affected extremities and adequate postural drainage and physiotherapy.

Filarial control in community

1. Mass therapy: abandoned now.
2. Selective therapy
   * Given only to those who are microfilaria positive
   * Given for both filarial cases and human carriers
   * 6 mg/kg/day – 12 doses to be completed in 2 weeks
   * Endemic areas – dose is repeated every 2 years.
3. DEC medicated salt
   * 1 to 4 gram of DEC/kg of salt for at least 6-9 months.

Vector control:

Larva control: Organophosphorus larvicides –

* Temephos

* Fen-thion
* Removal of pistia plant.

Adult mosquito control: Space spray with pyrethrum.

Reference

- Oxford Text Book of Medicine. Pp: 787-792.
- Manson Bahr Text Book of Tropical Medicine. Pp: 1321 to 1368.
- Park's Text Book of Preventive and Social Medicine. Pp: 213-216.

Dengue

Causes

- Dengue is the most important emerging tropical viral disease of human beings in the world today. All four dengue virus (Dengue 1, 2, 3 and 4) infections may be asymptomatic or may lead to
  * Undifferentiated fever, dengue fever (DF)
  * Dengue hemorrhagic fever (DHF)
    » With plasma leakage that may lead to hypovolemic shock
  * Dengue shock syndrome (DSS).

Symptoms

- Dengue fever is an acute febrile illness of 2-7 days with two or more of the following manifestations
  * Headache
  * Retro-orbital pain
  * Myalgia
  * Arthralgia
  * Rash
  * Hemorrhagic manifestation (petechiae and positive tourniquet test) and
  * Leucopenia
- Dengue hemorrhagic fever (DHF) if one or more of the following are present
  * Positive tourniquet test
  * Petechiae, purpura or ecchymosis
  * Bleeding from mucosa
  * Haematemesis, malaena
  * Thrombocytopenia (platelets 100,000/mm^3 or less) and evidence of plasma leakage.
- Dengue shock syndrome (DSS)
  * All the above criteria of DHF plus signs of circulatory failure.

Note:

The tourniquet test is performed by inflating a blood pressure cuff to a mid-way between the systolic and diastolic pressure

Dengue fever and DHF during febrile phase

Non-pharmacological

- Rest

Pharmacological

- Tab. Paractemol 500 mg 6 hourly (not more than 4 times in 24 hours)
- ORS in patients with dehydration
- **Caution:** No role of antibiotics, steroids; do not use aspirin or ibuprofen

Important Note

To report immediately if patient develops any of the following danger signals: severe abdominal pain, passage of black stools, bleeding into the skin or from the nose or gums, sweating and cold skin.

Dengue hemorrhagic fever: grade I (positive tourniquet test) and grade II (spontaneous bleeding) and thrombocytopenia < 100,000, Hct rise > 20%.

- Immediate hospitalization.
- As above in dengue fever.
- Send sample for blood grouping and cross matching.
- IV fluids if patient has persistent vomiting or Hct rise >20 %, continue IV fluids for 12-24 hours. (**Caution:** IV fluid therapy before leakage is not recommended) Monitor vitals and urine output on an hourly basis. Based on periodic haematocrit, platelet counts and vital signs review and revise treatment.

DHF grade III (with circulatory failure) and grade IV (profound shock with undetectable

blood pressure and pulse)

Immediately admit the patient in a hospital where trained personnel can manage shock and where blood transfusion facilities are available.

- If a patient had already 1000 ml of IV fluids and still vitals are not stable, the haematocrit should be repeated and (a) if the haematocrit is increasing, IV fluids should be changed to colloidal solution preferably dextran or (b) if the haematocrit is decreasing, fresh whole blood transfusion 10 ml/kg/dose should be given.
- In case of continued or profound shock give colloidal fluid following the initial fluid bolus (Do not give IV fluids indiscriminately leading to either fluid overload or under transfusion in shock patients).
- In case of persistent shock when, after initial fluid replacement and resuscitation with plasma or plasma expanders, the haematocrit continues to decline, internal bleeding should be suspected. It may be difficult to recognize and estimate the degree of internal blood loss in the presence of haemoconcentration
- Give fresh whole blood in small volumes of 120 ml/kg at one time
- Give platelet rich plasma transfusion in exceptional cases when platelet counts are below 5000-10000/mm^3
- Monitor pulse, BP, and temperature every 15-30 minutes
- After blood transfusion, continue fluid therapy at 10 ml/kg/h and reduce stepwise to bring it down to 3 ml/kg/h and maintain for 24-48 hours.
- During convalescent phase (2-3 days) after recovery from the state of shock, advise rest, normal diet

Signs of recovery

* Stable pulse, respiration and blood pressure
* Normal temperature
* No evidence of bleeding
* Return of appetite
* No vomiting
* Good urinary output
* Stable haematocrit
* Convalescent confluent petechial rash.

Criteria for discharging patients:

- Absence of fever for at least 24 hours without the use of anti pyretic agents.
- Return of appetite.
- Visible clinical improvement.
- Good urine output.
- Minimum of three days after recovery from shock.
- No respiratory distress from pleural effusion and no ascites.
- Platelet count of more than 50,000/mm^3.

Patient education

- Since this disease can rapidly become very serious and lead to a fatality, carefully watch for danger signs and immediately report to a doctor. Do not wait.
- **The complications usually appear between the third and fifth day of illness.**
- Give large amounts of fluid along with normal diet.
- All control efforts should be directed against mosquitoes. Efforts should be intensified before transmission season and during rainy season.

Reference

- Guidelines for treatment of Dengue fever/DHF in small hospitals, 1999, WHO Regional office for South-East Asia, New Delhi.

Chikungunya

Causes

Chikungunya is caused by an alpha virus closely related to O' nyong-nyong virus.

Main vector-Aedes aegypti.

Symptoms

- Acute self-limiting illness.
- Incubation period – 2 to 4 days. Bi-phasic disease.
- Abrupt onset presenting as fever with severe joint pain.
- After 1 – 4 days, fever subsides; there will be a

afebrile period of 3 days, fever returns with an itching maculopapular rash on trunk and extensor surfaces of limbs.

- After another 3 – 6 days, fever subsides and there is complete recovery.
- Crippling arthropathy can occur intermittently for upto 4 months, in some cases even upto 5 years.

Investigations

- Leucopenia.
- Ig M antibody detectable by ELISA – first detected by 48 hours and can be detectable upto 6 months.

Treatment

- Symptomatic treatment with anti-inflammatory drugs to relieve arthralgia.
- Chloroquine phosphate can be used in refractory arthralgia.
- No licensed vaccines or no specific treatment available at present.
- Measures to reduce Aedes aegypti population and avoiding mosquito bites.

Reference:

- Oxford Text Book of Medicine .pp: 378-379.
- Manson Bahr Text Book of Tropical Medicine. Pp: 624-626.

SWINE FLU
H1N1 VIRUS INFECTION
(Syn: Swine influenza / S-OIV infection / H1N1 - A virus infection)

Definition

It is respiratory disease of pigs affecting human beings, caused by type A Influenza viruses with regular outbreaks in pigs. Source of the virus in swine are avian, human and swine. All three viruses re-assort and form a new virus which is a mixture of all three. At present there are four types H1N1, H1N2, H3N2 and H3N1. The present pandemic is by Influenza A H1N1 type.

Characteristics of the Influenza viruses

Influenza viruses belong to the orthomyxoviridae family, with three separate genera A, B, and C based on antigenicity of the nucleoprotein (NP) and matrix (M) protein.

Influenza A has subtypes based on surface antigens of Hemagglutinin (H) 16 distinct subtypes (H1 to H16) and Neuraminidase (N) antigens 9 distinct (N1 to N9) subtypes. Only H1, H2, H3, N1, and N2 have been associated with epidemics.

Epidemiology

Epidemics of H1N1 - A begin abruptly, peak over a 2 to 3 week period and generally last for 2–3 months. There is an increase in the number of children with febrile respiratory illnesses followed by increase in rates of influenza-like illnesses among adults. Eventually there is a spurt in hospital admissions for pneumonia, exacerbations of chronic pulmonary disease and worsening of pre-existing congestive heart failure.

Transmission of H1N1 virus

The infectious period is one day before and until 7 days after the case's onset of illness. Data available indicate that this virus is transmitted in ways similar to other influenza viruses. When an infected person coughs or sneezes large-particle respiratory droplets are released. These droplets being large, generally travel only a short distance (<1 meter).

Droplets cannot remain suspended in the air for long and hence settle on the surfaces. Because of the above, spread is either by a direct contact between source and recipient persons or by respiratory-droplet contaminated surfaces. All respiratory secretions and bodily fluids (diarrheal stool) of (H1N1) patients should be considered potentially infectious.

High risk population for H1N1 virus infection

Populations at high risk are children less than5 years old and persons aged above 50 years. Pregnant women, adults and children with chronic organ dysfunction (pulmonary, cardiovascular, hepatic, hematological, neurologic, neuromuscular or metabolic disorders) or immuno suppression (caused by medications or by HIV) and residents of nursing homes, other chronic-care facilities are also more vulnerable for H1N1 infection.

Case Definitions:

A confirmed case of H1N1 infection is defined as a person with an acute febrile respiratory illness with laboratory confirmed H1N1 infection at CDC by one or more of the following tests: Real-time RT-PCR, viral culture or four fold increase in H1N1 virus specific neutralizing antibodies.

A probable case of H1N1 is a defined as a person with acute respiratory illness who is positive for influenza A but negative to H1 and H3 by RT-PCR

A suspected case of S-OIVA (H1N1) is defined a person with an acute febrile respiratory illness who has had close contact with a person who is a swine-origin influenza confirmed case or travelled to a community in the United States or internationally where there are one or more confirmed swine-origin influenza cases or resides in a community where there are one or more confirmed swine-origin influenza A (H1N1) cases.

Acute respiratory illness

Defined as recent onset of at least two of the following: rhinorrhea or nasal congestion, sore throat, cough (with or without fever or feverishness).

Clinical Features of (H1N1) virus infection

Symptoms include fever, headache, cough, sore throat, rhinorrhea, myalgia, fatigue, vomiting, or diarrhea. Patients appear flushed and the skin is hot and dry. Pharynx is normal despite a severe sore throat. There may be mild cervical lymphadenopathy. Illness generally resolves over 2–5 days and recovery occurs in 1 week. Cough may persist 1–2 weeks longer and post-influenzal asthenia may persist for several weeks. Frank dyspnea, hyperpnoea, cyanosis, diffuse rales and signs of consolidation are indicative of pulmonary complications.

Little is currently known about this infection in children since it is difficult to distinguish from illnesses caused by other respiratory pathogens by symptoms. Symptoms of severe disease may include apnea, tachypnoea, dyspnoea, cyanosis, dehydration, altered mental status and extreme irritability.

Complications of H1N1 infections

The complications are dominantly respiratory and include secondary bacterial pneumonia with or without sepsis, bronchiolitis, status asthmaticus, sinusitis, otitis media and croup. Exacerbations of underlying chronic medical conditions are frequent. Other rare complications are viral pneumonia, myocarditis, pericarditis, myositis, rhabdomyolysis, acute and post-infectious encephalopathy, encephalitis, febrile seizures and status epilepticus.

H1N1 related Pneumonias

Usually bacterial pneumonia is common. It is characterized by reappearance of fever, cough, production of purulent sputum with physical and x-ray signs of consolidation. Common Organisms are Str.pneumoniae, Staph. aureus, and H. influenzae. The pneumonia responds to antibiotic therapy when instituted promptly.

Primary Viral pneumonia is a least common but most severe complication that has a predilection for individuals with cardiac disease particularly those with mitral stenosis. Manifestations include persistent fever, dyspnoea, and eventual cyanosis. Sputum though scanty can contain blood. Diffuse rales may be noted in advanced cases with X-ray showing diffuse interstitial infiltrates and/or ARDS.

Testing for swine-origin influenza A (H1N1) virus

Upper respiratory specimens, nasopharyngeal swab or wash, nasal aspirate or tracheal aspirate should be tested by the state public health laboratory. Real time RT-PCR, viral cultures are to be performed to confirm the H1N1 infection.

Supportive measures for H1N1-A virus infection

- Bed rest
- Hydration with oral or parenteral fluids and nutritional support
- Cough suppressants generally are not indicated but codeine-containing compounds may be employed if the cough is troublesome
- Management of the metabolic consequences of dehydration and increased metabolic demand.
- Fever management with antipyretics like acetaminophen 4th hourly. ('aspirin' is contraindicated in children and teenagers).
- Cold sponges, ice bags, ice baths, ice water enemas and sprinkling a patient with water is the optimal approach for hyperpyrexia.

Antiviral drugs for S-OIV (H1N1)

H1N1 - A virus is susceptible to neuraminidase inhibitor antiviral medications

Zanamivir and Oseltamivir are the two drugs widely used in the present epidemic of (H1N1) virus infection. It is resistant to the adamantane antivirals amantadine and rimantadine. For oseltamivir-resistant human A (H1N1) viruses, either zanamivir or combinations of oseltamivir and rimantadine or amantadine provide adequate empiric treatment. Oseltamivir (Tamiflu) is administered orally while Zanamivir by inhalation.

Antiviral treatment guidelines

Treatment is considered for confirmed, probable or suspected cases of S-OIA infection. RT-PCR or viral

culture testing must be done to confirm S-OIA (H1N1) infection. Priority is given for hospitalized patients and patients at higher risk of complications. Antivirals are to be started within 48 hours of onset of illness. Reduction in mortality or duration of hospitalization is noted even if antiviral therapy is started after 48 hours. Duration of treatment is five days.

Antiviral Chemoprophylaxis for (H1N1) virus infection

Chemoprophylaxis is recommended for high risk individuals with household contacts of confirmed case and health care workers or public health workers who were not using personal protective equipment (PPE). It may be considered in high risk individuals with household contacts and HCW of suspected case of H1N1 and travelers to the countries where the epidemic is going on.

Post exposure chemoprophylaxis considered for contact during the infectious period. The duration of chemoprophylaxis post-exposure is 10 days after the last known exposure to a confirmed case. Pre-exposure protection is given during the potential exposure period and is continued for 10 days after the last known exposure to a confirmed case.

Adverse effects

Adverse effects of Oselatamivir include nausea and vomiting which might be less severe if it is taken with food. Rarely anaphylaxis, Toxic Epidermolysis Necrosis (TEN), Erythema Multiforme (EMF), Steven Johnson's syndrome, transient neuropsychiatric events (self-injury or delirium) have been reported. Persons receiving oseltamivir should be monitored closely for abnormal behaviour.

Zanamivir is not recommended for treatment for patients with underlying airway disease since they can produce cough and symptoms of bronchitis. Allergic reactions, including oropharyngeal or facial edema, nausea, diarrhoea, headache, dizziness may occur. Occasionally they may develop seizures, confusion or abnormal behaviour.

Antivirals for Pregnant women with H1N1 infection

No clinical studies have been conducted to assess the safety of these antivirals in pregnancy but no adverse effects have been reported among women or infants born. Although pregnancy is not a contraindication to Oseltamivir or Zanamivir they are used during pregnancy, only if the potential benefit justifies the potential risk to the fetus.

Antiviral drugs and dosages Recommended for H1N1 influenza infection

OSELTAMIVIR (TAMIFLU)			
Age and groups	Weight	Treatment	Chemopro-phylaxis
Adults		75 mg caps. twice daily	75 mg caps. Once daily
Children	40 Kg and above	75 mg caps. twice daily	75 mg caps. Once daily
	25 Kg to 40 Kg	60mg caps. twice daily	60 mg caps. Once daily
	15 Kg to24 kg	45 mg caps. twice daily	45 mg caps. Once daily
	Less than 15 Kg	30 mg caps. twice daily	30 mg caps. Once daily
ZANAMIVIR			
Adults and children		Two 5 mg (total 10 mg) inhalation	One 5 mg inhalation once daily

General Precautions for health care personnel

- Avoid close contact with the sick person
- Keep at least 6 feet distance from the patient
- Hand hygiene
- Use face protection (medical or surgical mask / eye-visor or goggles / face shield)
- Use a gown and clean gloves

Personal Protective Equipments (PPE) while treating suspected case

- Fit tested disposable N95 respirator
- Eye protection (goggles or eye shield)
- Disposable non sterile gloves and gown while going closer to the patients

Precautions while treating non suspected case

- Standard surgical mask for the patients
- Respiratory hygiene
- Using non-sterile glove for contact with the patient, patient secretions or surfaces that may

have been contaminated

- Hand washing or cleansing with alcohol based disinfectant after contact

Respiratory protection for health care personnel

- Use face mask or an N95 respirator that fits snugly on your face
- Wear an N95 respirator if you help a sick person with respiratory treatments using a nebulizer or inhaler
- Avoid re-using disposable facemasks and N95 respirators
- After you take off a facemask or N95 respirator, clean your hands with soap and water or an alcohol-based hand sanitizer
- Hand hygiene

Surveillance of healthcare personnel

- If symptoms of infection develop, health worker is instructed not to report for work
- If at work already- should cease patient care activities
- Asymptomatics who have had unprotected exposure should have chemoprophylaxis

Guidelines for community with confirmed cases of H1N1

- If anyone develops ILI (influenza like illness) they must self isolate themselves
- Period of isolation
  - 7 days after the symptoms started or atleast 24 hours after the symptoms resolved
- If the person should go into the community
  - Should wear a mask
  - Handkerchief or tissues may be used for covering if mask is not available
- Household members
  - Should be given infection control instruction
  - Should do meticulous hand washing
  - Should remain home at the earliest sign of infection

Personal protection where there is risk of infection

- Covering the mouth and nose with a tissue while sneezing or coughing
- Throwing the tissue in the trash after use
- Washing the hand with soap and water especially after sneezing and coughing
- Alcohol based hand cleaners are useful
- Touching of eyes, nose and mouth to be avoided
- Avoiding close contact with infected persons

Precautions for staff providing care to patients

- Use a medical or surgical mask
- Emphasize hand hygiene and provide hand hygiene facilities and supplies.
- Use face protection (medical or surgical mask and eye-visor or goggles, face shield) and use a gown and clean gloves.
- Do not forget hand hygiene after PPE removal.

References

www.cdc.gov/h1n1flu/guidance/

www.who.int/csr/disease/swineflu/guidance/health_professionals/en/index.html

Harrison's principles of internal medicine CMDT 2009.

Geriatrics

Chapter 12

- Falls
  - Introduction
  - Assessment
  - Evaluation
  - Treatment
- COMA
  - Definition
  - Investigations
  - Treatment
- Geriatric Surgery
  - Introduction
  - Geriatric Case Principals
  - Treatment
  - Special Considerations
  - Keypoint

FALLS

Introduction

Falls are one of the major problems faced by the elderly and it is considered as a "Geriatric Giant". Recurrent falls is an important cause of morbidity and mortality in the elderly and a marker of poor physical and cognitive status.

The incidence increases as age advances, and is a leading cause of death due to its complications. The morbidity due to falls are fractures, soft tissue injury, restricted mobility and psychological trauma. More than 80% of hip fractures in the elderly are due to falls. These lead to increased health care utilization. The psychological fear may lead to "Post Fall Syndrome" a state in which an elderly person restricts his mobility or becomes dependent on others to move, in spite of normal neurological status.

Definition

Fall is best defined as a sudden unintentional change in position, causing a subject to land on the ground or on lower level not as a result of major intrinsic or extrinsic hazards. A recurrent fall is defined as two or more fall events occurring within a period of six months.

About 1/3 of the community living elderly fall in a year. Some studies indicate that 25-50% of community–dwelling elderly fall at least once annually and half of them have multiple falling episodes.1 The incidence of falls is higher in the institutionalized elderly, owing to their poor health and higher reporting rates.

Causes

Basically, the upright human body is unstable with a very small base of support relative to its height. During physical activities the balance is maintained by complex neuromuscular feedback mechanism. The central structures (cortex, basal ganglia, brain stem and cerebellum) coordinate the sensory inputs from visual, vestibular and proprioceptive organs and control the effector organs i.e. the musculoskeletal action of lower limbs, neck and trunk. Aging changes include impairment of vision and hearing, reduction of proprioceptive and vibratory sensation, increased sway, altered gait and slower righting reflexes. These physiological changes alone do not cause a fall but increase the liability to fall. Most often the cause of a fall is multifactorial. It is due to multiple underlying problems such as physical illness, cognitive decline, medications and environmental hazards. The risk factors for falls in elderly 1-5 are listed below

Assessment of falls:

Screening:

Falls and their sequalae are potentially preventable and hence annual screening for falls is recommended. For individuals who have experienced one or more falls, a further evaluation is recommended.

Assessment includes a complete detailed history regarding the fall, individual's fear of falling, the presence of acute or chronic illness and medications.

The pneumonic "SPLATT" is useful for recalling the fall circumstances.

S	Symptoms that occurred immediately prior to fall or with the fall episode (light headedness, dizziness, vertigo, palpitations, chest pain, dyspnoea, sudden focal neurological deficit, aura, syncope, or urinary / faecal incontinence.
P	Previous history of falls
L	Location of fall
A	Activity at the time of fall
T	Timing of fall and length of time on ground
T	Trauma or injury as a result of fall.

- Assessment of individual's fear of falling is done by self efficacy tests.
- Depression is assessed with the Geriatric Depression Scale (GDS).
- Medication history is elicited in detail with specific attention given to newly started drugs or for which the dose has been increased recently.
- Functional assessment with ADL score is done.

RISK FACTORS FOR FALLS

Intrinsic Factors
1. Old Age / Female sex / Low body mass index
2. Neurological
* Cognitive impairment
* Postural instability
* Parkinsonism
* CVA / Gait disorders
* Peripheral neuropathy
* Sleep disturbance
3. Visual impairment
4. Musculoskeletal
* Foot disorders
* Muscle weakness of lower limbs
5. Cardiovascular
* Arrhythmias
* Postural hypotension
* Cardiac failure
6. Medication
* Polypharmacy
* Sedatives / hypnotics
* Diuretics /Antidepressants
7. Acute Illness
* Acute pneumonitis / Myocudial infection.
8. Metabolic disturbances
9. Behavioral
* Alcohol intoxication
10. Psychological
* Depression

Extrinsic Factors
1. Ground surface
* Uneven surface
* Slippery Floors
* Steps
2. Lighting
* Poor lighting
* Glare from lamps
3. Furniture
* Low lying furniture
* Chairs without arms
4. Improper walking aids and footwear

Symptoms

This includes assessment of vision, gait and balance and lower extremity joint function. Detailed neurological examination including cognitive screening using Mini Mental State Examination (MMSE) is done. Cardiovascular assessment and evaluation of various intrinsic and extrinsic factors are done.

Investigations

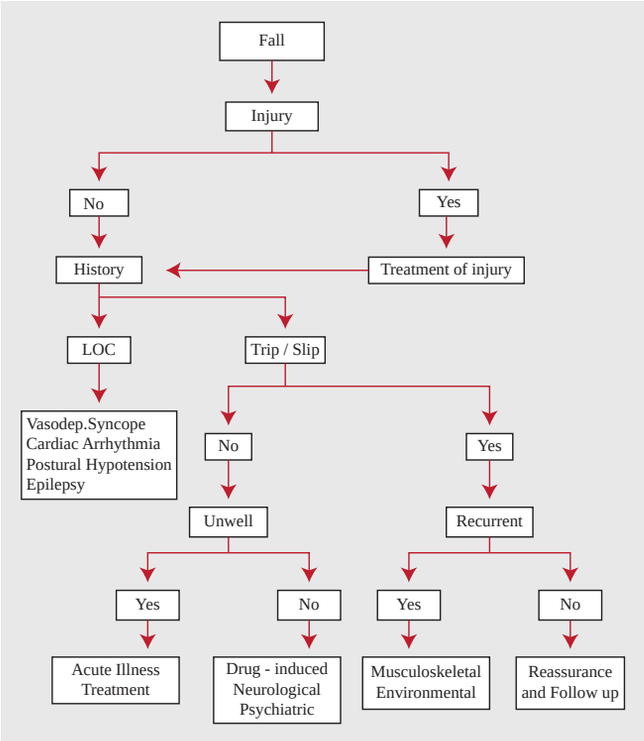
Injurious falls require X-Ray or other imaging studies depending on the site of injury and condition of the patient.

Specialized Assessment

This includes assessment of gait and balance by various tests such as the

* Functional Reach (FR) which measures dynamic balance
* Berg balance scale- a measure of functional activity
* Timed get up and go test and
* Performance oriented mobility assessment which is a measure of balance and gait.

Algorithm for Evaluation of Falls:



Treatment

- Home safety evaluation is done for falls that occur at home, with regard to extrinsic factors.
- Treatment of the injury and the associated illness. After the patient is stabilized the treatment of the primary cause of fall should be implemented. These include physiotherapy (balance training and muscle strengthening exercise) use of elastic stockings for orthostatic hypotension and specific drug therapy, dose adjustment and review of medications.

Fall prevention

1. Fall-related education
2. Environmental assessment and modification
3. Modification of medication regimen
4. Exercise program to improve strength, balance and endurance.
   * Muscle strengthening / resistance exercise
   * Balance training
   * Aerobic / endurance training
   * Flexibility exercises and
   * Tai-chi- an ancient martial art form of China. The slow and direct rhythmicity of movements contribute to development of movement strategy to prevent falls in actual life.

Important Note

Falls occur frequently in elderly with increased morbidity. A number of intrinsic and extrinsic risk factors are associated with a fall. Falls are often multifactorial. Hence a detailed evaluation is mandatory, particularly in those with recurrent falls. Falls may be prevented by targeted, multifaceted interventions.

References :

1. Tinetti ME, speechley M, Ginter SF : Risk factors for falls among elderly persons living in the community. N Engl J Med 1988; 319 : 1701-7.
2. Nevitt MC, Cummings SR, Kidd s et al: Risk factors for recurrent non syncopal falls. JAMA 1989; 261:2663-68.
3. O' Loughin JL, Robitaile Y, Boivin JF et al: Incidence of and risk factors for falls and injurious falls among the community dwelling elderly. Am J Epidemiol 1993; 137: 342-354.
4. Gragmans WC, Ooms ME, Hofstee HM et al: Falls in the elderly: a prospective study of risk factors and risk profiles. Am J Epidemiol 1996; 143:1129-1136.
5. Leipzig RM, Cumming RG, Tinetti ME: Drugs and falls in older people : a systematic review and meta-analysis : I. Psychotropic drugs. J Am Geriatr soc 1999; 47:30-39.
6. Tideiksaar R : Preventing falls : how to identify risk factors, reduce complications, Geriatrics 1996; 51(2) : 43-46, 49-53.
7. Tinetti ME, Richman D, Powell L : Falls efficacy as a measure of fear of falling. J Gerontol A 1990; 45:M239-M243.
8. Powell LE, Myers AM : The activities specific balance confidence scale : J Gerontol A 1995; 50 : M28 – M34.
9. Anonymous : Guideline for the prevention of falls in older persons. J. Am Geriatr Soc 2001; 49:664 – 672.
10. Close J, Ellis M, Hooper R et al: Prevention of falls in the elderly trial (PROFET): a randomized controlled trial. Lancet 1999; 353:93-97.

COMA

Coma is defined as a prolonged period of uncondciousness and lack of reaction to stimulus. Patients in coma can not be aroused.

Salient features

Following causes affect the functions of reticular - activating system and its connections with cerebrrum.

- Structural damage to brain (haemorrhage, tumors, trauma, localized infections, meningitis, stroke).
- Metabolic disturbances (ischaemia, anoxia, uraemia, diabetes), respiratopry/hepatic/renal failure, dyselectrolytaemia, endocrinopathies, drugs like opiates, barbiturates, benzodiazepines, anti-depressants and cyanide.
- Abnormal elecrical activity - periodic lateralized epileptiform discharge (PLED).

Rx Treatment

Nonpharmacological

- The immediate goal in acute coma is the prevention of further nervous system damage.
- Hypotension, hypoglycaemia, hypercalcmia, hypoxia, hypercapnia and hyperthermia should br corrected rapidly and assiduously.
- An orophatyngeal airway is adequate to keep the pharynx open in drowsy patients who are breathing normally.
- Tracheal intubation is indicated if there is apnoea, upper airway obtruction, hypoventilation or emesis, or if the patient is liable to aspirate because of coma.
- Mechanical ventilation is required if there is hypoventilation or if there is an intracranial mass and a need to induce hypocapnia in order to lawer intracranial pressure.
- Establish intravenous access.

Pharmacological

1. Inj. Glucose (25 or 50%) 50 g IV.
2. Inj. Thiamine 100 mg IV.
3. If opiate overdose is suspected, give Inj. Naloxone 0.8 mg IV. response is inadequate, double the dose every 15 minutes (for details see section on opioid intoxication).
4. If benzodiazepine overdose is suspected give Inj. Flumazenil 200 mcg IV slowly. If no response repeat 100-200 mcg after 1 minute. If required give maximum does of 1 mg or give as IV infusion of 100-400 mcg/h if drowsiness recurs.
5. If focal neurological deficit or signs of herniation/decerebration/decortication occurs, CT scan, EEG and neurologic consultation is required.
6. If no clear aetiology and no herniation - CSF exemination should be done.
7. If signs of raised intracranial tension (papilloedema, convulsions, decerebrate posture indicating herniation) occurs:
   * Avoid giving free fluid (glucose solution) intravenously.
   * Inj. Frusemide 40 mg IV to maintain adequate urine output of 30-50 ml/h.
   * Inj. Mannitol 1.0 g/kg IV over 10 minutes.
   * Hyperventilate to bring down PCO2 to 25 mmHg.
   * Inj. Dexamethasone 20 mg IV stat and 6 mg 4 hourly.

Children and young adults may have ominous early clinical findings such as abnormal brainstem reflexes and yet recover. Metabolic comas have a far better prognosis than traumatic comas. Glasgow coma scale empirically has predictive value in case of brain trauma (Table 2.5). For anoxic and metabolic coma, clinical signs such as papillary and motor responses after 1 day, 3 days and 1 week have been shown to have predictive value. Absence of cortical waves of the somatosensory evoked potentials has also proved a strong indicator of poor outcome in coma from any cause.

Greding of coma

Glasgow coma scale for head injury (Table 2-5)

Glasgow coma scale

Eye opening (E)	Coma score
Spontaneous	4
To loud voice	3
To pain	2
Nil	1
Best motor response (M)	
Obeys	6
Localizes	5
Withdraws (flexion)	4
Abnormal flexion posturing	3
Extension posturing	2
Nil	1
Verbal response (v)	
Oriented	5
Confused, disoriented	4
Inappropriate words	3
Incomprehensible sounds	2
Nil	1

Note: Coma score=E+M+V. Patients scoring 3 or 4 have an 85% chance of dying or remaining vegetative, while scores above 11 indicative only a 5 to 10% likelihood of death or vegetative state and 85% chance of moderate disability or good recovery. Intermediate scores correlate with proportional chances of recovery.

Reference

1. Coma. In: Harrison's Principles of Internal Medicine. Kasper DL, Braunwald E, Fauci AS et at (eds), 16th Edition 2005, McGraw Hill Company Inc., New York, pp 1624-1631.
2. Head Injury. In: Harrison's Principles of Internal Medicine. Kasper DL, Braunwald E, Fauci AS et al (eds), 16th Edition 2005, McGraw Hill Company Inc., New York, pp 2447-2452.

Geriatric surgery

Longevity has increased all over the globe due to advances in science and health care delivery systems, resulting in an increased elderly population. There is not only a large number of elderly population in (absolute term) but they also live longer than ever before to contract many diseases and problems

As age advances there is progressive and generalized deterioration of organ function with loss of reserve. The rule of thirds states that 1/3 of decline in function is due to disease; another 1/3 due to disuse and the rest due to ageing. Ageing changes can be minimized and postponed by balanced diet, exercise, and avoiding smoking and alcohol

Health status of the elderly is generally poor in addition to their physical, mental, social and financial problems. They have multiple medical disorders like diabetes, hypertension, ischemic heart disease, neurological problems and as age advances they may develop cancers of various organs.

Principles of geriatric surgery:

- Maximize independent function
- Reduce disabilities, suffering and pain
- Enhance quality and dignity of life

Best forms of health care in the elderly:

- Respect elderly patients at all times by listening to their statements
- Execute health promotional activities- good nutrition, exercise, social activities
- Implement preventive measures like stopping smoking, chewing tobacco and drinking alcohol (it is never too late to stop smoking and drinking) and to switch over to a healthy life style
- Screening of common diseases, early detection and intervention

Geriatric care principles:

- Improve quality of life, it is more important than prolonging life
- Honor the patient's wishes while investigating and treating
- Improve the general condition and nutritional status
- Identify co - morbid condition and correct them before surgery
- Explain the procedure, possible risks and complications of surgery
- Get detailed informed consent in writing for all procedures
- Initiate treatment early
- Consider alternative modalities of treatment instead of high risk surgery
- Modify the treatment regimen considering the ageing physiology
- Take up proactive measures so as to prevent ia-

trogenic complications

- Assess the capabilities of the patient and the family or caregivers as it is essential to make a good and safe managemnet plan
- Provide continued, comprehensive interdisciplinary team care

History taking in elders:

- Elicit history , including medication review
- Past history, treatment taken, polypharmacy
- Get all old records wherever possible
- Rely on history given by the relative/ care taker or by the referring physician if no history can be elicited from the patient
- Completely rely on physical findings, investigations if no history is possible

Physical examination:

- Complete general and physical examination
- Examine in comfortable surroundings
- It may be necessary to postpone the examination as per the patient's wishes
- Examine the patient in multiple sittings if required

Investigations

- Special tests to be carried out if any abnormality is suspected
- Routine and repeated investigations should be avoided
- Costly and invasive investigations for diagnosis need not be done in case of very old and very ill patients, if no treatment is contem plated

Diagnosis:

- Multiple pathological problems with multiple symptoms are common in the and no single diagnosis is common for all symptoms
- General measures are to be taken to keep the geriatric patient comfortable and free from pain when the diagnosis is delayed or not possible

Treatment

- Always aim at complete cure
- Consider the general condition and co morbid conditions
- Always weigh the benefit of surgery against the possible risks and complications
- Consider alternatives to high risk surgery and non operative managemant
- Always provide general supportive measrues and tender loving care when no specific treatment of the problem is contemplated

Special considerations:

- Preoperative:
- Preop assessment and preparation
- Detect and treat comorbid conditions- Diabetes, IHD, Hypertension
- Improve respiratory function, preop physiotherapy
- Correct anemia, hypoprotenemia, treat infective foci
- Preoperative counselling

There is generalised deterioration of organ function and loss of reserve, hence minor degrees of fluctuation in blood volume and BP can tilt the balance in the elderly

Anaesthesia:

- Good anaesthetic techniques likie smooth induction, maintanence and quick reversal without any cerebral complications
- During anaesthesia, adequate cerebvral and coronary blood flow, maintanence of fluid and electrolyte balance , urinary output and glycemic control are of paramount importance in the elderly

Surgery:

- Do the minimum necessary procedure
- Avoid blood loss/ replace immedietely
- Avoid hypotension, hypoxia and hypothermia
- Neat and fast surgery to keep anaesthesia time to the minimum

Post-operative care

Immediate:

- Maintain vital signs and blood pressure
- Adequate oxygenation and fluids to avoid hypotension, hypoxia and hypothermia
- Prevent cerebral anoxia and avoid delirium

- Adequate analgesia, early mobilization and physiotherapy to prevent pulmoray complications

Early postoperative care:

Avoid prolonged parenteral nutrition, use gut early Prevent respiratory and wound infections Avoid prolonged bed rest , ambulate early

In elderly immobility my lead to:

- Loss of muscle mass
- Decreased tissue sensitivity to insulin
- Orthostatic hypotension, tachycardia, decreased cardiac output, and decreased stroke volume
- Urinary retention, urinary tract infection
- Negative nitrogen balance
- Depression, sensory deprivation
- Deep vein thrombosis
- Constipation, fecal impaction
- Decubitus ulcers

Key points:

- Always take the patient's / relatives signature in writing when deciding on hospitalization, ordering costly and troublesome investigations , initiating intensive and invasive monitoring and treatment
- Manage elders judiciously without pushing them to financial difficulties
- Good communication with the patients, family members, caregivers and the family physicians will help to get the expressed preferences
- Medical ethics, moral values and legal formalities should be followed strictly while taking management decisions
- Reassurance and tender loving care should be given life long.

Paediatrics

Standard Treatment Guidelines
Tamil Nadu Health Systems Project

Chapter 13

Neonatology

- Guidelines for management of normal New born
- High risk new born
- Neonatal resuscitation
- Management of LBW babies
- Neo natal seizures
- Jaundice in the new born
- Respiratory distress in new born
- Bleeding in neonates
- Hypothermia in new born
- Neo natal transport
- Surgical problems in neo nates

Paediatrics

- Altered level of consciousness
- Febrile seizures
- Acute respiratory infection
- Pneumonia
- Bronchiolitis
- Empyema
- Approach to fever in children
- Malaria
- Dengue
- Typhoid
- UTI
- Leptospirosis
- Viral hepatitis
- Tuberculosis
- Rheumatic fever in children
- Nephrotic syndrome
- Acute nephritic syndrome
- Protein energy malnutrition
- Anaemia
- Management of scorpion sting
- Management of snake bite
- Management of Poisoning in children

Guidelines for Management of Normal Newborn

Prior to delivery

Review maternal history to anticipate problems in neonate.

Check list for New born kit

1. Mucus catheter (Lee's) / Bulb sucker
2. Warm towels
3. Warmer
4. Equipment for resuscitation
5. Test tube for cord blood
6. Disposable syringes and needles
7. Emergency drugs including Inj.vitamin K 25% dextrose, 10%calcium gluconate, adrevaline, naloxone.

At delivery

1. Keep the baby on level with mother. Do not milk the cord. Clamp 5 cm away from the baby, cut it and tie with double ligatures. Leave the cord uncovered. Collect cord blood for grouping, Coomb's test, VDRL, screening tests if necessary.
2. Resuscitate, if required. Assess Apgar.
3. Thermo regulation. Dry the baby well with a pre-warmed towel soon after birth. Wrap in dry, warm sheet / blanket and place with mother. No bath need be given. Head should be dried thoroughly.
4. Clean eyes from medial to lateral with separate moist sterile swabs if necessary.
5. Weigh the baby, assess gestation.
6. For identification, tie wrist tag.
7. Record time and date of birth.
8. Record passage of meconium, urine.
9. Carry out a preliminary examination. Rule out life-threatening congenital anomalies.
10. Administer Vitamin K 1 mg intramuscularly
11. Put the baby to the breast soon after birth, preferably in the first half hour.
12. Normal newborns must be kept with mother.

Care of the normal newborn

1. After delivery the baby is placed next to the mother and leaves the labour room with the mother. Baby should be kept with the mother in the postnatal ward.
2. Feeding
   * Exclusive breast milk
3. No pre-lacteal feeds including water, glucose water or honey.
4. Clothing
   * Soft, white, clean clothes.
5. No pins, hooks or buttons, only ties.
6. Easy to put on at neck and arms.
7. Napkins: cotton napkins may be used instead of disposable napkins.
8. Care of cord
   * Left open to dry.
9. Baby bath
   * No full water bath should be given immediately after delivery to ensure maintenance of temperature
   * Baby can be cleaned with oil / warm water to remove excess of vernix and meconium
   * In a busy, crowded ward it is wiser to avoid baby baths to prevent cross infection
   * At home, baby is safely bathed on extended legs in the Indian way. Oil and powder are not necessary. A mild soap should be used.

Cleaning

Baby should be cleaned everyday and each time when urine or stools are passed. Bottom should be dipped in warm water and perineum cleaned from above and below.

Vitamins

Normal, full-term babies do not require vitamin supplements.

Immunization

Baby should be given BCG, hepatitis B (wherever given) and the first dose of oral polio before discharge.

Stools and urine

The first stools of the baby are dark green / black, sticky and is called meconium. It is passed in the first

24 hours after birth. After initiation of feeds the colour slowly changes to green, yellow (transitional) and finally yellow. The normal baby who is breastfed can pass upto 7 - 8 bright yellow, pasty stools or one stool per day (pasty) or once in two to three days.

A baby must pass urine in the first 48 hours of life and should pass urine at least 5 - 6 times a day. The frequency can vary upto 15 - 20 times a day.

- Feeding schedule will depend on the gestational age and clinical status of newborn (Given in the chapter on low birth weight)
- Treatment will depend on the individual condition

High risk newborn

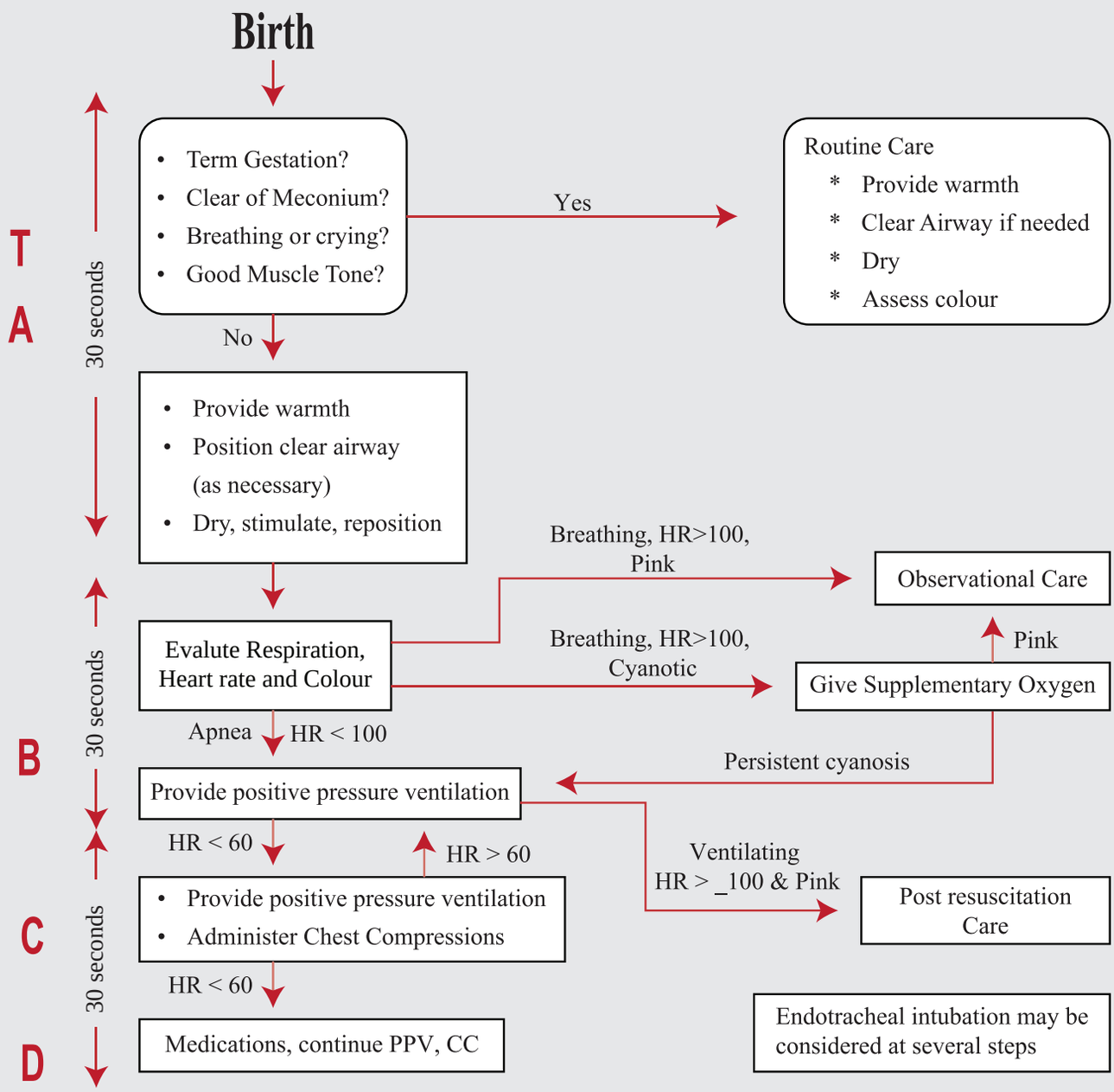
High risk newborns are required to be kept in observation areas according to the specific indications as follows

Treatment areas for high risk newborns

With mother under observation	Observation area (nursery)	Premature unit	NICU
1. Infants of elderly / very young primigravida 2. Rh - ve mother 3. Maternal medication 4. Tubal ligation done in mother 5. Pre-eclampsia 6. PROM 7. LSCS, vacuum, forceps delivery 8. Meconium stained liquor, mild asphyxia (asymptomatic infant) 9. Infant weighing 2-2.5 kg 10. Previous death of a sibling 11. Birth trauma	1. Infants of mother with toxaemia of pregnancy 2. Infected mother 3. Mother with post-partum psychosis 4. IDM 5. Polycythemia 6. Birth trauma 7. Hyper-bilirubinemia	8. Pre-term less than 37 weeks of gestation. 1. Babies weighing less than 2 kg.	1. Severe asphyxia 2. Symptomatic meconium aspiration 3. Severe respiratory distress syndrome 4. Life threatening congenital anomalies. 5. Congenital infections 6. Septicaemia 7. Seizures 8. Bleeding neonate 9. Congestive cardiac failure 10. Necrotising-enterocolitis (NEC) 11. Severe anaemia 12. Post-operative surgical neonate

Neonatal resuscitation

T - Temperature
A - Airway
B - Breathing
C - Circulation
D - Drugs



Management of Low Birth Weight (LBW) babies

Infants weighing less than 2.5 kg.

These babies can be preterm or small for gestational age. (SGA)

Aetiology

- Maternal
  - * Infections
  - * Chronic diseases
  - * Malnutrition
  - * Trauma
  - * Drugs
- Obstetric factors
  - * Toxaemia
  - * Antepartum haermorrhage
  - * Premature rupture of membranes
- Uterine factors
  - * Structural abnormality - bicornuate or septate uterus
  - * Premature rupture of membranes
- Fetal factors
  - * Multiple pregnancies
  - * Congenital anomalies
  - * Chromosomal anomalies
  - * Intrauterine infections

Management of term LBW and Pre-term babies

LBW (Pre-term) : Problems

- Birth asphyxia
- Hypothermia
- Feeding difficulties
- Infections
- Hyper-bilirubinemia
- Respiratory distress syndrome (RDS)
- Retinopathy of prematurity (ROP)
- Apnoeic spells
- Intraventricular hemorrhage (IVH)
- Hypoglycemia
- Metabolic acidosis

LBW (Small for gestational age) : Problems

- Birth asphyxia
- Meconium aspiration syndrome
- Hypothermia
- Hypoglycemia
- Infections
- Polycythemia

LBW - Issues in delivery

- Transfer mother to a well - equipped centre before delivery.
- Skilled person needed for effective resuscitation.
- Prevention of hypothermia - topmost priority.

LBW - Indications for hospitalization provided below.

Decision to choose the referral hospital may be decided by the treating doctor.

- Birth weight < 1800 g
- Gestation < 34 weeks
- Unable to feed
- Sick neonate irrespective of birth weight and gestation

Treatment guidelines for LBW babies

- Prevention of Hypothermia
  - * Keeping warm at home by skin to skin contact - Kangaroo Mother Care (KMC).
  - * Keeping the baby warm in hospital - skin to skin contact; warm room and bed; warmly wrapped; radiant warmer; incubator
- Feeding
  - * Weight < 1200 g; Gestation < 30 wks
    - » Start initial intravenous fluids (refer fluid therapy guidelines)
    - » Introduce lavage feeds once stable.
    - » Shift to paladai / spoon feeds over next few days. Later on breastfeeds.
  - * Weight 1200 - 1800 g; Gestation 30-34 wks
    - » Start initial gavage feeds.
    - » Paladai / spoon feeding after 1 - 3 days. Shift to breast feeds as soon as baby is able to suck.

    - » May need intravenous fluids, if sick.
  - * Weight > 1800 g; Gestation > 34 wks
    - » Breast feeding
    - » Paladai / spoon feeding, if sucking not satisfactory on breast.
    - » Shift to breast feeds as soon as possible.
- LBW - Feeding schedule
  - * Begin at 60 to 80 ml/kg/day; Increase by 15 ml / kg/day; maximum of 180 - 200 ml/kg/day. First feed at 2 hours of age, then every 2 hourly.
- LBW - Weight pattern
  - * Pre-terms lose 2 to 3% weight every day for 1st week with a cumulative loss of 15-20%. Weight gain commences by 10-15 days
  - * Excessive weight loss or inadequate weight gain consider: Cold stress, anaemia, poor intake, sepsis etc,
- LBW
  - * Supplements
    - » Vitamin : Vit. K 1.0 mg IM at birth
    - » Vit A 1000 IU / day and
    - » Vit D 400 I.U / day from 2 weeks of age.
  - * Iron oral 2 mg/kg/day from 8 weeks of age
  - * Calcium and phosphorus: 100 mg/kg/day
- Danger signals (early detection and referral)
  - * Lethargy, refusal of feeds.
  - * Hypothermia
  - * Tachypnea, grunt, gasping, apnea
  - * Seizures, vacant stare
  - * Abdominal distension
  - * Bleeding
  - * Icterus over palms / soles
  - * Sudden pallor
- LBW babies require dose monitoring and follow up of
  - * Growth monitoring - Head circumference and weight
  - * Developmental assessment and early stimulation.
  - * Intraventricular hemorrhage screening by ultrasound cranium on day 1, 3, 7 and at 4-6 wks.
  - * Screening tests for hearing - at discharge
  - * Retinopathy of prematurity screening at one month of age.
  - * Screening for osteopenia of prematurity

Neonatal Seizures

Seizures are abnormal involuntary movements affecting part or whole of the body, occurring due to an excessive synchronous electrical discharge of neurons within the CNS.

Points to remember

- Neonatal seizures are not easy to recognize because of varying clinical patterns.
- Seizures often result from underlying CNS dysfunction and they cause CNS damage.
- Intravenous administration of anticonvulsants in correct doses is essential as absorption by other routes is erratic.

Aetiology

- Asphyxia
- Intracranial haemorrhage.
- Metabolic
  - * Hypoglycemia
  - * Hypocalcemia
  - * Hypomagnesemia
  - * Hyponatremia
  - * Hypernatremia
- Meningitis, encephalitis
- Polycythemia
- Pyridoxine deficiency
- Inborn errors of metabolism
- Congenital malformations of CNS
- Narcotic drug withdrawal.

Clinical presentation

Classical tonic clonic convulsions of older children are not seen in neonates. Seizures present as:

- Subtle convulsions
  - * Abnormal eye movements - Blinking, fluttering of eyelids, horizontal deviation of eye balls with nystagmus.

    * Oro-buccal - lingual movements - Sucking, chewing, drooling, lip smacking, etc.,
    * Abnormal limb movements - Swimming, rowing, cycling
    * Apnoea.
- Focal clonic : Common in multi-focal disorders, focal lesions.
- Multi-focal clonic.
- Generalized tonic spasms : Common in preterm babies.
- Myoclonic jerks.

Suggested investigations

- Blood sugar, Dextrostix
- Serum calcium, magnesium.
- Serum electrolytes.
- Haematocrit.
- Lumbar puncture.
- Ultrasonography of cranium
- CT Scan
- Urine and Plasma screening for inborn errors of metabolism.
- EEG
- Sepsis screening

Note

Diazepam is not safe in neonates as it interferes with vital function, its sedative effect exceeds 24 hours and sodium benzoate, the preservative used increases the risk of bilirubin encephalopathy.

Others

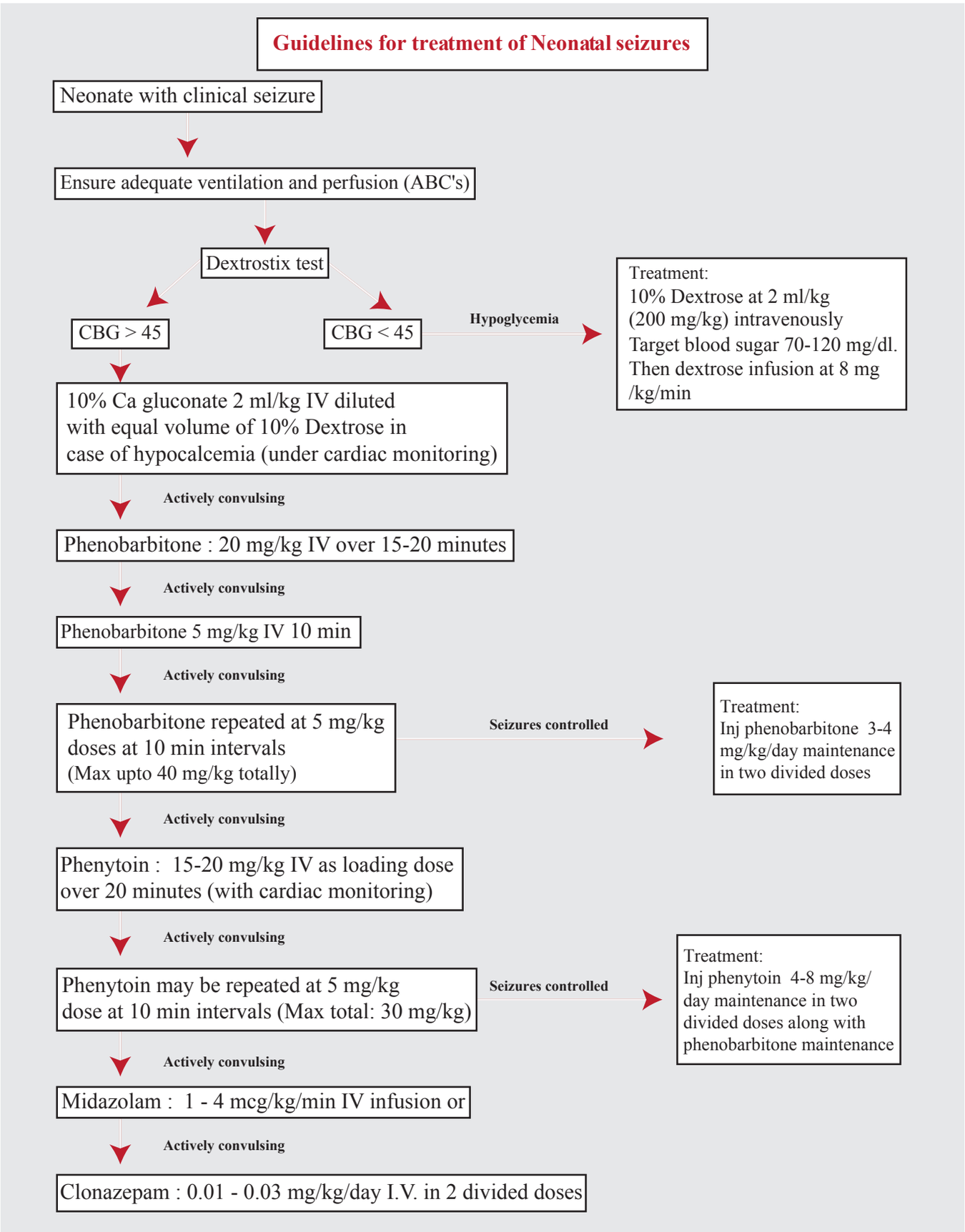
- Hypomagnesemia
    * Magnesium sulfate, 50% solution : 0.2 ml/kg, IM;
- Refractory seizures
    * Pyridoxine : 50-100 mg/kg IV (with EEG monitoring)

Duration of anticonvulsant therapy - guidelines in Neonatal period

- Neurological examination normal
    * Discontinue therapy
- Persistently abnormal
    * Consider etiology
    * Obtain EEG
    * Continue phenobarbitone
    * Discontinue phenytoin
    * Re-evaluate in a month

One month after discharge

- Neurological examination normal
    * Discontinue phenobarbitone after 2 weeks
- Persistently abnormal
    * Obtain EEG
    * No seizure activity
    * Discontinue phenobarbitone over 2 weeks
- Seizure activity persists
    * Continue phenobarbitone until 3 months of age and reassess in the same manner.


Guidelines for treatment of Neonatal seizures
Neonate with clinical seizure
Ensure adequate ventilation and perfusion (ABC's)
Dextrostix test
CBG > 45
CBG < 45
Hypoglycemia
Treatment:
10% Dextrose at 2 ml/kg
(200 mg/kg) intravenously
Target blood sugar 70-120 mg/dl.
Then dextrose infusion at 8 mg
/kg/min
10% Ca gluconate 2 ml/kg IV diluted
with equal volume of 10% Dextrose in
case of hypocalcemia (under cardiac monitoring)
Actively convulsing
Phenobarbitone : 20 mg/kg IV over 15-20 minutes
Actively convulsing
Phenobarbitone 5 mg/kg IV 10 min
Actively convulsing
Phenobarbitone repeated at 5 mg/kg
doses at 10 min intervals
(Max upto 40 mg/kg totally)
Seizures controlled
Treatment:
Inj phenobarbitone 3-4
mg/kg/day maintenance
in two divided doses
Actively convulsing
Phenytoin : 15-20 mg/kg IV as loading dose
over 20 minutes (with cardiac monitoring)
Actively convulsing
Phenytoin may be repeated at 5 mg/kg
dose at 10 min intervals (Max total: 30 mg/kg)
Seizures controlled
Treatment:
Inj phenytoin 4-8 mg/kg/
day maintenance in two
divided doses along with
phenobarbitone maintenance
Actively convulsing
Midazolam : 1 - 4 mcg/kg/min IV infusion or
Actively convulsing
Clonazepam : 0.01 - 0.03 mg/kg/day I.V. in 2 divided doses

Jaundice In The Newborn

Points to remember

- Jaundice is common in neonates and is predominantly of the indirect type.
- It is important to distinguish between physiological and pathological jaundice and establish an aetiology for the latter.
- Jaundice is clinically appreciable in newborns with serum bilirubin values of 7 mg% or more as compared to 2 mg% in adult and is best appreciated not in the sclera but by blanching the body skin in good day-light.
- All newborns should be screened at least twice a day for jaundice.
- The most accurate method to assess and monitor jaundice is by estimating bilirubin in serum, especially in dark babies and babies under phototherapy, where clinical judgement is not to be relied upon.
- Babies with asphyxia, acidosis, hypoglycaemia (sick neonate) and pre-terms run a higher risk of bilirubin encephalopathy (kernicterus) at lower levels of bilirubin.

Criteria For Pathological Jaundice

- Clinical jaundice in first 24 hours of life.
- Total serum bilirubin increasing by more that 5 mg%/day.
- Total serum bilirubin more than 12.9 mg% in full-term.
- Direct serum bilirubin more than 2 mg%.
- Clinical jaundice persisting for more than one week in full-term and for more than 2 weeks in pre-term infants.

  [Look for various causes related to time of appearance of jaundice after birth and associated clinical findings]

Evaluation

- Clinical determination of jaundice by Kramer's criteria.
- Jaundice in the newborn presents a cephalocaudal pattern of appearance.

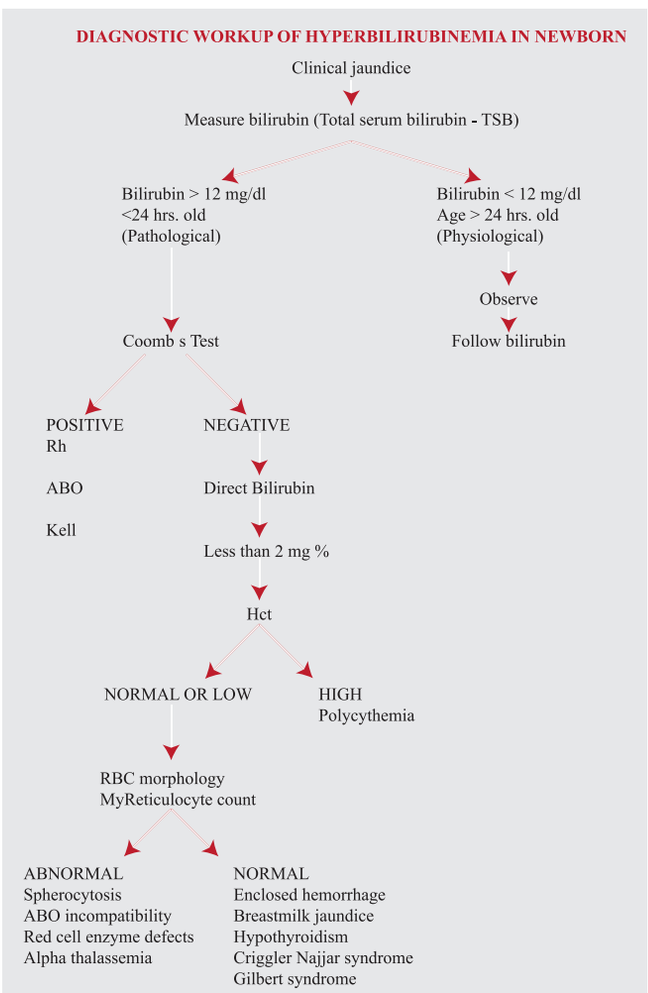
Clinical Determination Of Jaundice By Kramer's Criteria.

Area of body	Range of serum bilirubin (mg%))
Head and neck	4 – 8
Upper trunk	5- 12
Lower trunk and thigh	8 – 16
Arms and lower limbs	11 – 18
Palms and soles	more than 15 mg

Investigations

Following investigations must be done in each case of neonatal jaundice.

- Serum bilirubin direct, indirect.
- Blood grouping of mother and child ABO and Rh.
- Direct Coomb's test in infant
- Hematocrit and peripheral smear for RBC morphology and reticulocyte count.
- Indirect Coomb's test in mother if she is Rh negative



Treatment modalities of Hyperbilirubinemia

- Hydration
- Phototherapy
- Exchange transfusion
- Drugs to increase conjugation

Hydration

Continued and frequent breast feeding - 8 - 10 times / day

Phototherapy (Table I and II)

- Special blue lights to be used
- 45 cm distance between baby and phototherapy unit
- Eyes and genitalia should be covered
- Double surface phototherapy is preferred
- Watch for side effects (diarrhoea, skin rash, hyper / hypothermia)

TABLE 1 - Guidelines for phototherapy according to AAP

Healthy, term newborn (>37 weeks)

Age (hours)	Consider Phototherapy TSB (mg/dl)	Phototherapy TSB (mg/dl)
< 24		
25 – 48	>12	> 15
49 – 72	>15	>18
>72	>17	>20

Note: TSB-Total serum bilirubin

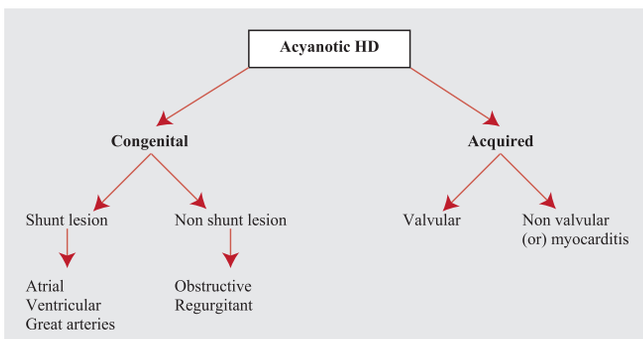
TABLE II – Phototherapy indications

Based on birth weight and health of the newborn

Birth weight (gms)	Healthy; TSB (mg/dl)	Sick; TSB (mg/dl)
< 1000	5 - 7	4 - 6
1001 – 1500	7-10	6-8
1501 – 2000	10 - 12	8 - 10
2001 – 2500	12 - 15	10 - 12
	TERM	
>2500	15-18	12-15

Points to remember

- Try to establish diagnosis before instituting phototherapy by carrying out necessary investigations.
- Check blue lights functioning; life of these lights is 1500 - 2000 h. (approx. 3 months). Keep lights at a distance of 18" from the baby.
- When blue tube lights are not available, four pairs of white tube lights may be used instead.
- Change the position of the baby after every 2 h.
- Babies can be taken out of phototherapy for breast feeding
- Monitor baby's temperature 2 hourly
- Monitor fluid balance-daily weight and urine output. Increase fluids as necessary
- Shield the eyes in both sexes to prevent retinal damage and genitals in males to prevent mutation defects in adulthood
- Monitor rise or fall of bilirubin every 12 hourly.
- Do not give phototherapy for direct hyperbilirubinemia.
- Exchange transfusion



Choice of Blood

- If baby or mother is Rh - ve use only Rh -ve Blood
- Always cross-match donor's blood with both mother's and baby's blood.

* Cord bilirubin more than or equal to 4.5 mg% and Hb less than 11 g%.
* Rate of rise of bilirubin > 1 mg/dl despite phototherapy
* In LBW babies, indirect bilirubin > (weight in g)/100.
* Exchange earlier at levels of 2 mg% less for following criteria:
  » Sepsis
  » RDS
  » Asphyxia
  » Acidosis
  » Hypoglycemia

Respiratory Distress In Newborn

Presence of two or more of the following indicates respiratory distress.

- Respiratory rate more than 60 / min.
- Cyanosis.
- Use of accessory muscles of respiration, nasal flaring, intercostal, subcostal retraction.
- Expiratory grunt.

Points to remember

1. Commonest problem in neonates with a wide differential diagnosis.
2. Caused by respiratory and non-respiratory conditions.
3. History and clinical examination are useful, but X-ray chest is a must.
4. Surgical problems should be suspected and ruled out.
5. Respiratory parameters, i.e., clinical and arterial blood gases (if possible) to be monitored regularly.

Aetiology

Respiratory

- Development anomalies: Upper airway obstruction, tracheo - oesophageal fistula, diaphragmatic hernia, etc.,
- Parenchymal disease: Hyaline membrane disease, pneumonia, meconium aspiration, etc
- Transient Tachypnea of Newborn (TTN)

Cardiac

Metabolic

- Such as metabolic acidosis

Neurologic

- Such as intraventricular hemorrhage

Others

- Polycythemia
- Sepsis
- Hypothermia

Diagnostic approach

Time of onset of respiratory distress

At birth	Later onset
• Congenital airway obstruction. • Developmental anomalies of respiratory track • HMD, Meconium aspiration. • Transient tachypnoea of new born	• Pneumonia • Air-leak syndromes. • Bronchopulmonary dysplasia.

Progress

- Early onset, progressive increase
  * HMD
  * Meconium aspiration
  * Diaphragmatic hernia
  * Lobar emphysema
- Early onset, later improvement
  * Transient tachypnoea of new born

Treatment Guidelines of Respiratory Distress In New Born

Ask

1. Time of onset
2. Age
3. EDD
4. Birth weight
5. Feeding problems

Assess

1. Temperature
2. Downe score
3. Capillary Refill Time (CRT)
4. SaO2
5. Gestational age
6. Present weight
7. Blood pressure
8. Heart rate

Downe Scoring

Sign	0	1	2
Respiratory rate	<60	60 to 80	>80 or apnoea
Cyanosis	None	In room air	In 40% oxygen
Retractions	None	Mild	Mod and severe
Grunt	None	Audible with stethoscope	Audible without steth
Air entry	Good	Delayed or decreased	Barely audible

Look for

- Patency (nasal and oesophageal)
- Chest asymmetry
- Apical impulse
- Scaphoid abdomen
- Hepatomegaly

Investigations

- Chest X-Ray (with NGT in situ)
- Septic screening (Total and differential count, Band – neutrophil ratio, CRP, micro ESR)
- Blood culture
- ABG if feasible
- Serum electrolytes
- Renal Function Tests (RFT), Blood glucose

Management of Respiratory distress

- Maintain temperature 36.5o C - 37.5o C
- Positioning (Supine position with head and neck in neutral position)
- Oxygen-according to Downe's Score
  - 0 to 3 Oxygen by hood (51 / min)
  - 4 to 7 CPAP with Oxygen
  - Above 7 Intermittent Positive Pressure Ventilation (IPPV) / Synchronized Intermittent Mandatory Ventilation (SIMV)
- If glucose is low - Bolus 10% dextrose 2 ml. / kg. (2 boluses)
- Maintenance fluid If CRT is more than 3 sec.: 10 ml/kg of normal saline, monitor liver size If still no improvement in CRT, commence inotropes
  - Dopamine 5 to 10 mcg / kg / min or Dobutamine 10 to 20 mcg / kg / min
  - If bradycardia inspite of all the above
    - Adrenaline infusion 0.1 mcg / kg. / min.
- Antibiotics
  - Ampicillin at 50 mg. / kg /dose BD if < 7 days and 50 mg/kg/dose TID if > 7 days +
  - Gentamycin 5 mg/kg in 2 divided doses
  - Cefotaxime at 50 mg/kg/dose BD if < 7 days and 50 mg/kg/dose TDS if > 7 days (in place of ampicillin in a very sick neonate)
- Documented metabolic acidosis : Correct the acidosis, treat the underlying cause.

Specific Management: Depending on individual condition

Bleeding In Neonates

Points to remember

1. Coagulation factors do not cross from mother to fetus.
2. Physiological deficiency of clotting factors such as II, VII, IX, X results in prolongation of Prothrombin Time (PT) and Partial Thromboplastin Time (PTT).
3. Bleeding is more common and severe in preterm and LBW infants due to accentuation of this deficiency.
4. All newborns should receive Vitamin K immediately after birth.

Common causes

1. Hemorrhagic disease of newborn (HDN) due to Vitamin K deficiency.
2. Disseminated intravascular coagulation (DIC)
3. Thrombocytopenia of any cause.
4. Inherited deficiency of clotting factors.
5. Increased capillary fragility causing bleeding into skin. e.g. breech delivery, traumatic delivery.

Approach

Maternal History

Condition	Increased risk due to
Antenatal infections TORCH	Thrombocytopenia
Drugs given to mother, i.e. phenytoin, phenobarbitone, aspirin, anticoagulants	Early onset HDN
H/o bleeding in mother, e.g. ITP, SLE in mother	Thrombocytopenia
Details of labour: hypoxia, trauma, etc.	DIC

Family history

H/o Bleeding in previous sibling, or other members of family.

Present history

- Day on which bleeding observed.
- Day 1 -Early onset HDN.
  - * Afibrinogenemia
  - * Hypofibrinogenemia
  - * Factor XIII deficiency
  - * DIC.
- Day 2
  - * Classical HDN
- H/o administration of Vit. K at birth

Physical examination

- First asses the baby, i.e. sick versus well baby from it's appearance, cry and activity.

Site of bleeding	Likely cause
• GIT	• Swallowed maternal blood, HDN
• Mucosa, skin, subcutaneous tissue	• Thrombocytopenia, Trauma, DIC
• Umbilicus, circumcision site	• Factor XIII def., DIC, coagulation factor def

1. Look for signs of sepsis, jaundice, hepato splenomegaly.
2. Cephalhematoma.
3. Hemangioma.

Investigations

Basic screening tests such as 'Platelet count, PT, PTT should be done before giving Vit K or blood transfusion.

Tests such as bleeding time and clotting time are not sensitive and do not help to reach diagnosis.

Treatment

- Hemorrhagic disease of newborn
  - * Vit K 0.5-1mg IV. if bleeding continues
  - * Fresh frozen plasma (FFP) 10-15 ml/kg IV if active bleeding, can be repeated 12 hourly if needed
  - * If Hb is less than 10 g then fresh blood transfusion 10-15 ml/kg
  - * Prevention: 1 mg Vit K IM to every newborn at birth is the preventive measure for this condition.
- DIC
  - * Treat the underlying cause, i.e. appropriate antibiotics in sepsis, correction of acidosis, hypoxia
  - * Replacement therapy with FFP 10-15 ml/kg every 12 hourly
  - * Exchange transfusion using fresh blood less than 12 hours old every 12 hourly, till condition stabilizes.

Hypothermia In Newborn

BABIES AT RISK FOR HYPOTHERMIA

- All newborns in first 12hrs of life
- All preterm and low birth weight babies
- Asphyxiated babies

- Associated sepsis and meningitis
- Intraventricular hemorrhage
- Maternal sedatives

Signs and symptoms of hypothermia	
Acrocyanosis	Tachypnea
Cool extremities	Respiratory Distress
Decreased peripheral perfusion	Bradycardia
Lethargy	Apnea
Poor feeding	Abdominal distension
Shock	Increased gastric residuals
Weight loss	Pulmonary hemorrhage
Poor weight gain	

Definition
Normal axillary temperature
Cold Stress
Moderate Hypothermia
Severe Hypothermia
Temperature
36.5°c – 37.5°c
36°c – 36.4°c
32°c – 35.9°c
<32°c
Implication
Concern
Danger
Grave
Very grave
Action
Warm the baby
Urgent skilled care
Referral
Referral

Treatment Of Hypothermia

Hypothermic newborns should be re-warmed quickly. The room temperature should be at least 25°C (77°F). Cold clothes should be removed and replaced with pre-warmed clothes and a cap. It is very important to continue feeding the baby. If the infant is too weak to breast-feed, breast milk can be given by nasogastric tube, spoon or cup.

At the hospital

The methods to use include:

- Skin to skin contact
- Warm room or bed
- 200 watts bulb
- Radiant warmer
- Air heated incubator

Radiant warmer (open care system)

Radiant warmers provide an intense source of radiation energy. Suggested abdominal skin temperature settings for infants nursed under radiant warmer or servo mode incubator.

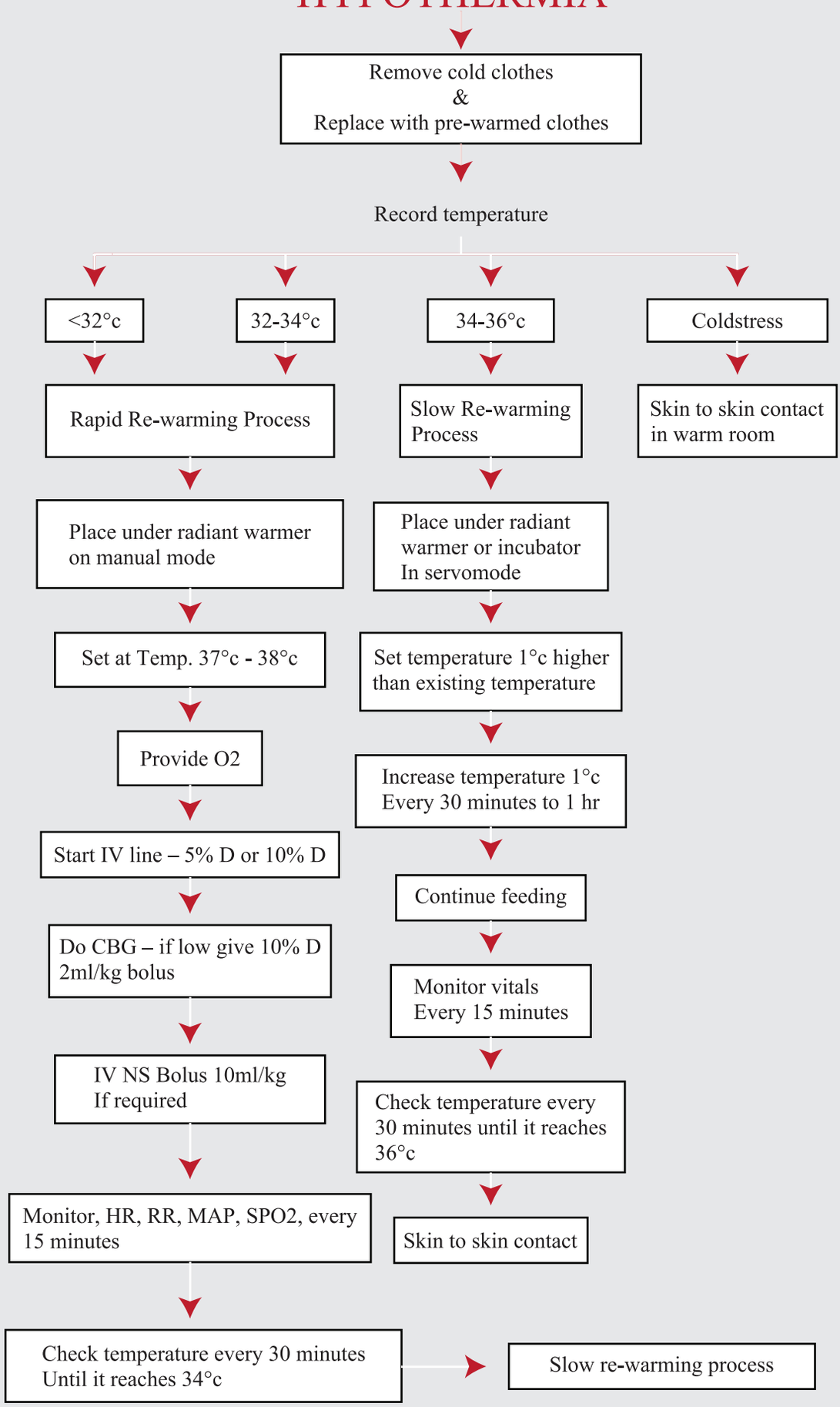
<1kg	36.9°C
1-1.5kg	36.7°C
1.5-2 kg	36.5°C
2.0-2.5kg	36.3°C
>2.5kg	36°C

Care of skin probes

Apply the probe over anterior abdominal wall and frequently inspect the probe for detachment from skin surface to prevent over heating of the baby.

"It is important to be aware that hypothermia can be a sign of infection. every hypothermic newborn should therefore be assessed for infection"

HYPOTHERMIA



Prevention of Hypothermia

Concept of "warm chain"

Baby must be kept warm at the place of birth (home or hospital) and during transportation for special care either from home to hospital or within the hospital.

The "Warm Chain" is a set of ten interlinked procedures carried out at birth and later, which will minimize the likelihood of hypothermia in all newborns.

1. Warm delivery room (>25°C)
2. Warm resuscitation
3. Immediate drying
4. Skin to skin contact between baby and the mother
5. Breast feeding
6. Bathing and weighing postponed
7. Appropriate clothing and bedding
8. Mother and baby together
9. Warm transportation
10. Training/awareness of healthcare providers

The kangaroo mother care (KMC)

Kangaroo Mother Care (KMC) is a special way of caring for low birth weight babies. It

- Assists in maintaining the temperature of infant
- Facilitates breast-feeding
- Helps to increase the duration of breast-feeding
- Improves mother infant bonding

Eligibility Criteria

All stable LBW babies are eligible for KMC. However, sick babies needing special care should be cared under radiant warmer initially, KMC should be started after the baby is hemo-dynamically stable.

Guidelines for practicing KMC include:

1. Birth weight >1800 g; These babies are generally stable at birth. Therefore, in most of them KMC can be initiated soon after birth.
2. Birth weight 1200-1799 g; Many babies of this group have significant problems in neonatal period. It might take a few days before KMC can be initiated. If such a baby is born in a place where neonatal care services are inadequate, he should be transferred to a proper facility immediately after birth, along with the mother / family member. He should be transferred to a referral hospital after initial stabilization and appropriate management, One of the best ways of transporting small babies is by keeping them in continuous skin to skin contact with the mother / family member during transport.
3. Birth weight <1200g; Frequently, these babies develop serious prematurity-related morbidity often starting soon after birth. They benefit the most from in-utero transfer to the institutions with neonatal intensive care facilities. It may take days to weeks before baby's condition allows initiation of KMC.

Kangaroo positioning

- The baby should be placed between the mother's breast in an upright position.
- The head should be turned to one side and in a slightly extended position. This slightly extended head position keeps the airway open and allows eye to eye contact between the mother and the baby.
- The hips should be flexed and abducted in a "frog" position; the arms should also be flexed.
- Baby's abdomen should be at the level of the mother's epigastrium. Mother' s breathing stimulates the baby, thus reducing the occurrence of apnea. Support the baby's bottom with a sling / binder.

Kangaroo mother care

Monitoring

Make sure that baby's neck position is neither too flexed nor too extended, airway is clear, breathing is regular, color is pink and baby is maintaining temperature. Mother should be involved in observing the baby during KMC, so that she herself can continue monitoring at home.

When should KMC be discontinued?

When the mother and baby are comfortable, KMC is continued for as long as possible, at the institution and then at home. Often this is desirable until the baby's gestation reaches term or the weight is around 2500g. She starts wriggling to show that she is uncomfortable, pulls her limbs out, cries and fusses every time the mother tries to put her back skin to skin. This is the time to wean the baby from KMC.

Bathing the baby

Bathing should be avoided immediately after birth. Preferably give bath to normal baby on second day in summer. In winter bathing may be avoided for several days. In small and /or LBW baby postpone bath till cord falls or preferably till weight is 2.5kg.

Neonatal Transport

Temperature maintenance during transport (Weakest link in warm chain)

- Ideally transport of a newborn should be in an orderly manner i.e. a neonate who is found to be sick by a health worker at home visit should be referred to a PHC
- If the facilities or expertise at the PHC not adequate enough to manage this sick neonate, he should be referred to the FRU and thereafter to the Medical College. Sickest of the neonates require referral to an apex institution or a tertiary care centre.
- Prepare well before transport: It is of utmost importance that a neonate is stabilized before the transport is begun, as an unstable neonate is going to deteriorate on the way and may reach the referral facility in a moribund state defeating the very purpose.
- The neonate should be assessed for temperature maintenance, airway patency, breathing efforts, state of circulation, fluid and hydration status, medications to be administered and feeding that is to be provided.
- If, on assessment any of the above parameters is found to be compromised, remedial action should be immediately taken.
    - Communicate and write a note
    - Assess and stabilize
    - Correct hypothermia
    - Encourage mother to accompany
    - Arrange a provider to accompany
- Ensure warm transport
    - Skin to skin care (Kangaroo Mother care)
    - Cover the baby fully with clothes (or cotton) including the head and the limbs. Avoid undressing the infants for cleaning, weighing or examinations. Carry the baby close to chest of the mother Improvised containers: Thermocol box with pre-warmed lines or plastic bubble sheet or silver swaddler may be used during transport.
    - Transport incubator

Provide other care during the transport

- Ensure warm feet
- Ensure an open airway
- Check breathing
- Provide feeds
- Record the vitals including the temperature before, during and after the transport.
- Take the baby to the nearest referral facility, by the shortest route, and by a safe mode.
- During Procedures
    - Perform the procedure under a functioning radiant warmer
    - Monitor the temperature frequently during the procedure

Fluid And Electrolyte Management In Newborn

Treatment guidelines for fluid and electrolytes

Day 1: > 1500 g

Term babies > 1500 g 60 ml/kg is given. To meet the glucose requirements of 4-6 mg/kg/min, this is given as 10% Dextrose.

Day 1: < 1500 g

- Because of the higher insensible water loss, ba-

bies with birth weight 1000 – 1500 g require 80 ml/kg of 10%D.

- Extremely premature babies < 1000 g require 100 ml/kg and this is given as 5%D to maintain glucose homeostasis.

After Day 1:

Sodium and potassium should be added only after 48 hrs of age each in a dose of 2 – 3 mEq/kg/day. Glucose infusion ideally should be maintained at 4 – 6 mg/kg/min in a volume independent manner if possible.

In preterms, sodium is supplemented at 3 – 5 mEq/kg/day till 32 – 34 wks post-menstrual age. Calcium may be used in a dose of 4 – 6 ml/kg/day of calcium gluconate for the first 3 days in certain high risk situations such as prematurity, infant of diabetic mothers, birth asphyxia etc.,

Treatment guidelines for fluid and electrolytes			
Weight	>1500 g	1000 – 1500 g	<1000 g
D1	60	80	100
D2	75	95	120
D3	90	110	130
D4	105	120	140
D5	120	130	150
D6	135	140	160
D7 and >	150	150	170
Increments	15-20	10-15	10-20
Max	150	150-170	160-180

Monitoring of fluid and electrolyte status

Body weight

- At least once a day
- Term neonates lose 1 – 2% of their body weight daily in the 1st week with a cumulative loss of 5 – 10%
- Pre-terms lose 2 – 3% daily in 1st week with a cumulative loss of 15 – 20%
- Weight gain should have commenced by 7 – 10 days in a term neonate and by 10 – 15 days in pre-terms

I/O charting

Input output charting has to be done meticulously including boluses, NG aspirates, CSF by lumbar puncture, diarrheal loss, fluids used for injection and flushing. These will be of importance especially in VLBW neonates.

Clinical assessment

The usual physical signs of dehydration are unreliable in neonates.

- A 10% dehydration (100 ml/kg) presents as
- Sunken eyes
- Depressed fontanelle
- Cold and clammy skin
- Poor skin turgor and
- Oliguria
- With 15% dehydration (150 ml/kg
- Shock ensues. As the normal range of blood pressure is wide, and blood pressure correlates poorly with volume status, they are not to be relied upon for assessing hypo-volemia
- Rather, importance is given to capillary refill time and core – peripheral temperature
- For hyper-volemia, look for weight gain, oedema, features of CCF and pulmonary oedema.

Principles of Deficit correction

- Water deficit
  - * Half of it is replaced over the first 8 hours and the other half over the next 16 hours.
- Sodium deficit
  - * Replaced over 24 hours
- Potassium deficit
  - * If large, replaced over 48 – 72 hours
- Maintain urine output 1-3 ml/kg/hour

Surgical Problems In Neonates

Tracheo-esophageal fistula (TEF)

Points to remember

1. TEF should be suspected in a neonate who soon after birth presents with excess salivation, respiratory distress, and in whom there is difficulty in passing a naso-gastric tube.
2. In suspected cases of TEF, even if the naso-gastric tube passes smoothly, it may be due to the fact that the tube gets coiled in the upper blind pouch. Therefore always confirm that the lower end of the tube is in the stomach by x-ray in suspected cases.
3. Prognosis depends on early diagnosis and prevention of aspiration of gastric contents, or milk into lungs.

4. Look for associated cardiac, renal, vertebral, lower GI-anomalies prior to surgical correction.

Clinical features

1. Antenatal : H/o polyhydramnios
2. Babies are commonly pre-term or small for gestational age (SGA).
3. Excessive oral and pharyngeal secretions with frothing at mouth.
4. Choking, coughing and cyanosis on feeding.
5. Failure to pass naso-gastric tube, resistance is encountered 8 - 10 cm from the upper gum line.
6. Symptoms and signs of pneumonia and sepsis due to aspiration of gastric contents with super-added infection may be present in cases that are diagnosed late.

Investigations

- Plain X-ray of neck and chest with naso-gastric tube in position
  - * Upper blind eso-phageal pouch is dilated with air and coiled up tube is seen.

Treatment

1. Nurse the baby in an upright position of 45o and carry out frequent suction to prevent aspiration.
2. Nil by mouth. start IV fluids.
3. Antibiotics if pneumonitis or sepsis.
4. Supportive treatment
5. Referral to higher centre

Congenital Diaphragmatic Hernia

Abnormal presence of abdominal viscera in the thoracic cavity above the diaphragm.

Signs and symptoms

1. Moderate to severe respiratory distress with or without cyanosis may be present right from birth or later
2. Scaphoid abdomen
3. Heart sounds better heard on side opposite to the hernia
4. Tympanic note on percussion of chest, and bowel sounds heard in chest

Investigations

1. Antenatal diagnosis by ultrasound.
2. Post natal diagnosis by X-ray.
   - * Mediastinal shift.
   - * No diaphragm visualized
   - * Bowel loops in chest.

Treatment

1. Anticipate the problem if diagnosed ante-natally. Do not ventilate with bag and mask. Use endotracheal tube for ventilation.
2. Head high position.
3. Pass naso-gastric tube and continuous aspiration.
4. Correction of fluid and electrolyte imbalance and metabolic acidosis.
5. Referral to higher centre.

Intestinal Obstruction

1. Intestinal obstruction should be suspected if there is maternal H/o polyhydramnios.
2. Abdominal distension and failure to pass meconium are considered cardinal signs of this condition.

Investigations

- Abdomen X-ray erect
  - * Level of obstruction may be determined by configuration of fluid levels on X-ray.

Clinical features

- Vomiting: Non-bilious or bilious
- Abdominal distension
- Upper abdominal
  - * Duodenal atresia
  - * Annular pancreas
- Generalized distension
  - * Obstruction at the level of jejunum and lower ileum.
- Failure to pass meconium
  - * Imperforate anus

Treatment

1. Nil orally

2. Deflation and aspiration of stomach with naso-gastric tube.
3. Fluid and electrolyte correction and monitoring.
4. Referral to higher centre.
5. Thermoregulation is extremely important during transport and in the operation theatre.

Recognition of a critically ill child

Appearence of the child

AVPU scale	
• Alert	
• Voice:	Responsive to voice
• Painful:	Responsive to painful stimuli
• Unresponsive	

Other features in appearance (Refer to algorithm)

Airway

Asses airway as clear, maintainable by positioning, or not maintainable if it needs advanced intervention

Breathing

Look for breathing movements; categorize as normal, increased or decreased or absent.

Assessment of breathing		
Respiratory rate: Rapid respiratory rate (tachypnea) by age		
• Age	<2 months	60/mt or more
• 2 months	<12 months	50/mt or more
• 12 months	< 5 years	40/mt or more

- Work of breathing
  - * Look for nasal flaring, grunting
  - * Intercostal, subcostal and suprasternal retractions
  - * Presence of these indicates increased work of breathing
  - * Increased work of breathing (IWB), head bobbing and see saw respirations are late signs.
- Air entry and breath sounds
  - * Look for unequal air entry, stridor, wheeze and silent chest
  - * Based on the above, classify the child as normal, in respiratory distress or respiratory failure
- Check oxygen saturation if available.
- Tidal volume
  - * Clinically assessed by chest expansion and auscultation for distal air movements simultaneously

Circulation

Heart rate in children	
* Newborn	80 - 200 / mt
* 1-8yrs	80 - 180 / mt
* >8 yrs	60 - 160 / mt

- Pulses
  - * Comparison of central and peripheral pulses to be done
  - * Both should be of equal volume
  - * In early shock peripheral pulse volume is decreased.
- Skin Perfusion
  - * Skin Temperature
  - * Color and Capillary refill time (CRT) are altered in shock.
- Blood pressure
  - * Use the following systolic blood pressure as cut off for defining hypotension

Blood pressure (Hypo-tension)	
• New born	<60 mm Hg
• Up to 1 year	<70 mm Hg
• 2 years and above	<70 + (2 x age in years) mm Hg
• After 10 years	<90 mm Hg

  - * Based on above circulatory assessment classify as normal, compensated shock or hypotensive shock.
- Based on the appearance, breathing and circulatory status, physiologic status of a critically ill child is characterized as:
  1. Stable
  2. Respiratory distress characterized by IWB and increased respiratory rate
  3. Respiratory failure characterized by cyanosis, altered sensorium, poor muscle tone and poor respiratory efforts.
  4. Compensated shock –Shock with normal BP

5. Hypotensive shock
6. Cardiorespiratory failure – Shock + Respiratory failure

Note: (3 - 6 need urgent referral after stabilizing.)

Initial resuscitation and stabilization

When a child is assessed to be critically ill, initial management comprises of taking care of airway, breathing and circulation.

a. Airway: is kept patent – if necessary by positioning, suctioning and or intubation.
b. Breathing: In respiratory distress only supplementation with O2 in a non-threatening manner is sufficient in respiratory failure; ventilatory support is often needed
c. Circulation: Vascular access and volume expansion with isotonic fluids such as RL or NS.(20ml / kg) If unable to get a venous access or in hypotensive shock immediate intra-osseous access should be obtained
d. If child presents with seizures, take care of airway and breathing and control seizures with intravenous Diazepam or Lorazepam. If no intravenous access, intramuscular Midazolam 0.15 mg/ kg is relatively safe, effective and fast.

Treatment Of Acute Respiratory Distress And Respiratory Failure Beyond Newborn Period	
Diagnosis	**Treatment**
Respiratory distress 1. Tachypnea 2. Increased work of breathing. 3. Presence of stridor, wheeze, grunt, retractions 4. Colour	• Keep the child in position of comfort (infant in mother's lap) • Oxygen in a non-threatening manner (by simple mask 5 l/min or by hood 10-12 l/min or non-rebreathing mask.10-12 l/min • Monitor Spo2 • Cardiopulmonary assesment • Anticipate worsening and be prepared for ventilatory support • Specific management for underlying illness
Respiratory failure (RF) In addition to features of respiratory distress child will have • Restlessness, anxiety • Altered level of consciousness: drowsiness, lethargy, unresponsiveness • Signs of exhaustion, sweating, head bobbing • Loss of tone and posture • Cyanosis is a late finding • Bradypnea, shallow breathing • Heart rate and perfusion will be abnormal • SpO2 < 92% (pulse oximetry) (RF can occur without respiratory distress in CNS depression, muscular weakness and very ill children) Diagnosis of RF and decision to intubate and ventilate should be made on clinical signs	• Airway positioning, suctioning • Bag – mask ventilation with 100% O2 using a reservoir • Prepare to intubate and ventilate • If there is no improvement or deterioration occurs after intubation and ventilation consider 'DOPE' * Displaced tube * Obstructed tube * Pneumothorax * Equipment failure • Assess circulation – RL / NS boluses if shock present • Ongoing Cardiopulmonary assesment and monitoring is important • Never leave the child unattended during transport, till the child recovers.

This is only the general guidelines for respiratory distress and respiratory failure. Specific management of illnesses is described separately under specific diseases.

Treatment Of Shock In Children

Shock can be

1. Hypovolemic shock
2. Septic shock
3. Cardiogenic shock

Features of shock: Tachypnea, tachycardia, normal or low BP, weak or absent peripheral pulses/central pulses, delayed capillary fill, cool peripheries, oliguria, change in mental status.

General principles

1. Resuscitation
2. Treat the underlying condition
3. Treat the associated metabolic disturbances
4. Supportive therapy to prevent or treat multi-organ dysfunction syndrome

Resuscitation: Maintain airway and breathing. Administer oxygen by a non-rebreathing mask 10-15 L/min in spontaneously breathing children.

Vascular access

- Large bore IV catheter
- Intra-osseous (IO): When IV access not obtained or child is in hypotensive shock, immediate IO access; proximal tibia preferred site in infants and small children

Fluid therapy

- Initial fluid bolus for shock
  - * RL or NS 20ml/kg rapidly in hypo-volemic shock. Repeat boluses 2-3 times after reassessment of cardiopulmonary status at the end of each bolus
- Cardiogenic shock due to sepsis
  - * 100- 240ml / kg in aliqouts of 20 ml /kg at 20 minutes per hour may be needed in the initial 6 hours of rescuscitation
- 2 or 3 fluid boluses will be required in hypovolemic or septic shock
- In case of blood loss, attempt replacement with blood after 2 boluses of fluids
- Reassess cardio-pulmonary status following each bolus: by pulse volume, heart rate, skin perfusion, urine output, sensorium, blood pressure, liver size and lung signs.
- Withhold further boluses if cardiogenic shock occurs (airway instability, pink froth, apnea, development of grunt, retractions, abdominal respirations, fresh rales, gallop rhythm, enlargement of liver).
- Child with shock refractory to fluid boluses needs to be shifted to a tertiary care facility urgently as the delayed referral will increase the mortality in shock.

Inotropes may be needed in cardiogenic and septic shock

Dopamine

- Start at 10 mcg/kg/min; titrate dose by increment of 2-3 mcg/kg/min every 15 minutes upto 20 mcg/kg/min
- Weight × 6 = mg of dopamine to be added to 100 ml of NS; 1ml/hr will deliver 1mcg/kg/mt; Indicated in hypotensive shock after fluid boluses.

Dobutamine

- Indicated in cardiogenic shock with normal or increased blood pressure
- Inotrope of choice, dose: 10-20 mcg/kg/min preparation similar to dopamine.

Epinephrine

- In case of low blood pressure

Treat underlying condition and correct any metabolic imbalance, documented hypo-glycemia, hypo-kalemia and metabolic acidosis.

Caution in replacing Sodium bicarbonate and potassium replacement in hypoxia on shocked child

Anaphylactic shock

- Epinephrine 0.1 ml/kg 1:10000 deep IM, 0.1ml/kg 1:1000 IV,
- Oxygen. Early intubation to secure airway, rapid volume expansion with NS or RL ; repeated boluses to restore perfusion.
- Steroids, antihistamines

Airway: To be maintained; Oropharyngeal suction; Nasogastric decompression

Breathing: Spontaneous breathing: oxygen through NRM 10-15 litres/min

Apneic: BMV with 100% O2 10 litres/min

If not improved, consider intubation

Circulation: IV access and correct shock; 25% Dextrose 2ml/kg if hypoglycemic

Treatment of status epilepticus

STATUS - EPILEPTICUS
Seizures > 5 min or sudden unconsciousness
Not regained consciousness in between two episodes

↓

0 Min: Inj. Lorazepam 0.05-0.1 mg/kg; IV; max.4mg/dose: at 2mg/min
(or)
Inj.Diazepam 0.2mg/kg; IV; max.10mg/dose
(or)
Inj. Midazolam 0.2mg/kg; IM (If IV access immediately not available)

↓

10 Min: Inj. Lorazepam/ Diazepam - Same dosage (Second dose)

↓

20 Min: Inj.Phenytoin 20mg/kg IV Loading dose; max.50mg/min at 1mg/kg/min
Mix with NS as phenytoin is incompatible with glucose containing solutions
Monitor HR, perfusion and blood pressure.
If fit continues additional 5mg/kg up to a max of 30mg/kg

↓

60 Min: Plan intubation with Midazolam 0.2 mg/kg bolus
IV Phenobarbitone 20mg/kg at 2mg/kg/min

↓

Plan Shift to ICU; Start Midazolam infusion at 1µg /kg/min
Increase every 15 min upto 20µg/kg/min

↓

IV Sodium valproate 15 –20mg/kg loading dose over 1-5 min
If seizures stop at any stage, continue to evaluate the cause for seizures and complications.

When to refer:

- Seizures refractory after phenobarbitone
- Persistent altered level of consciousness

Treatment Of A Child With Altered Level Of Consciousness (ALOC)

How to recognize ALOC?

An older child not responding to simple commands and simple questions appropriately. In an infant > 8 weeks, absence of eye contact and unresponsiveness to mother's call. Irritablity, restlessness, refusal of feeds, excessive sleepiness, inconsolable cry, failing to cry even with painful stimuli such as injections are indicative of ALOC. Rapid assessment can be done by AVPU scale

History

Try to find out if the problem is acute, chronic or acute on chronic illness. Acute symptoms: Fever, headache, vomiting, ALOC, drug ingestion, injury, predisposing illness. Underlying Chronic illness such as CHD, CRF, seizure disorder, developmental delay, bleeding disorder. History should point towards the gross categories as follows:-

- Infection, Raised ICP, trauma, bleeds, systemic illness.
- History not correlating to the presentation/ vacuum in the history should alert about poisoning, animal bites and metabolic encephalopathies.

Clinical features

Tachypnea, shock Hypertension, oedema Mastoid swelling, ear discharge. Pallor, jaundice, bleed Bite marks, fang-marks, cellulitis	Arrythmia, murmur Hematoma of scalp Neurocutaneous markers Bulging AF Hepatosplenomegaly

- Raised intra-cranial pressure is suspected with neurogenic hyperventilation, neurogenic stridor, unequal pupils, dilated non-reactive pupil, abnormal posture, presence of Cushing's triad with bradycardia, hypertension and abnormal breathing pattern may be seen in advanced cases.
- Fundus examination and Glasgow Coma Scale (GCS)

Investigations

- Complete blood count
- Smear for MP
- Blood glucose for hypoglycemia and hyperglycemia,
- Serum electrolytes for hyponatremia, hypernatremia and other imbalances,
- Blood Urea and serum creatinine, calcium
- SGOT, SGPT, CSF analysis if no contraindications.

Other investigations as necessary

PT, PTT, X-ray bones, Cultures, MSAT, Widal, Viral studies, CT scan, EEG, Gastric aspirate analysis in poisoning.

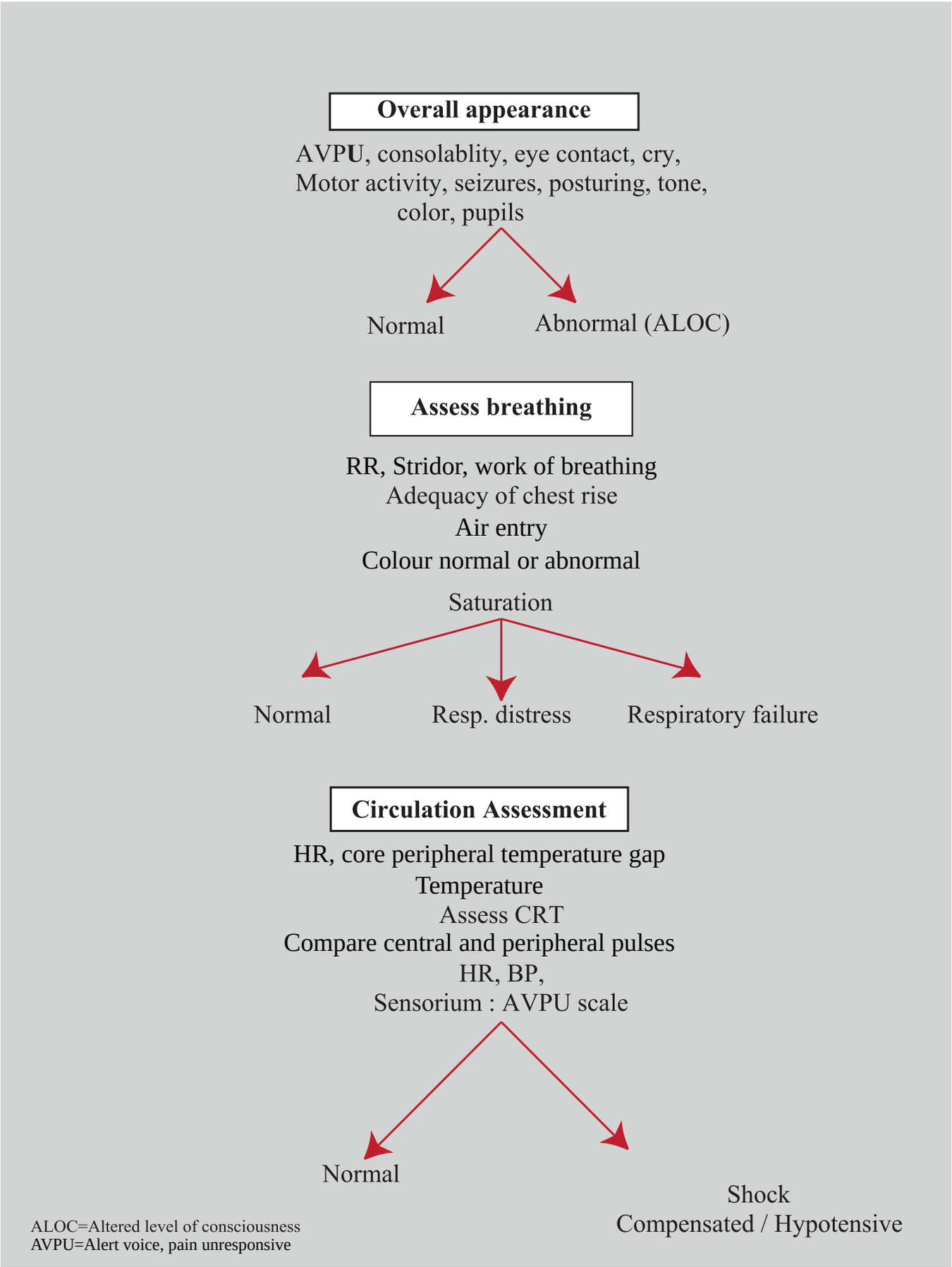
Treatment

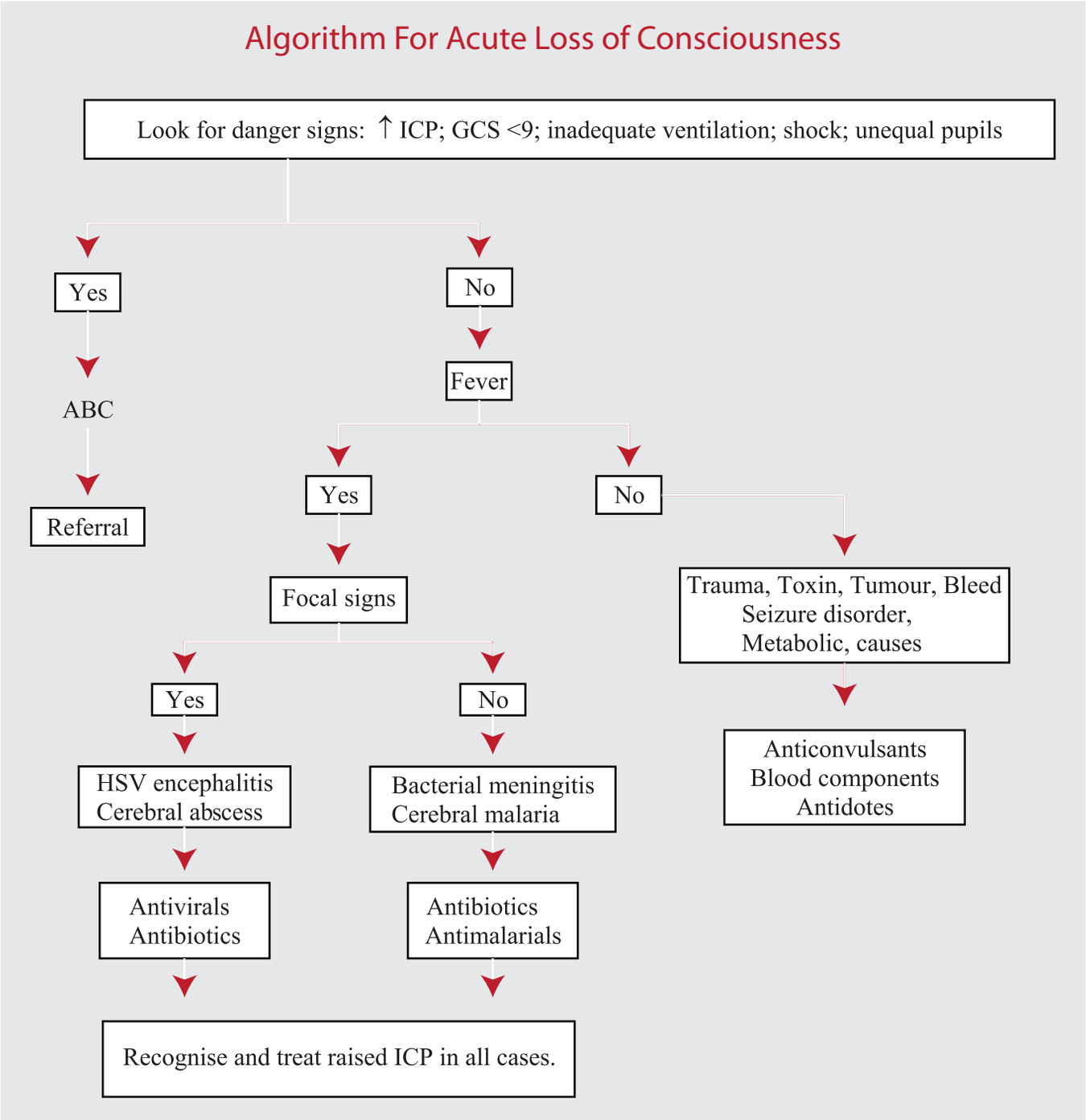
- ABC as mentioned earlier
- Position the child in semi-recumbent posture
- Specific management depending on underlying condition
- Other supportive measures
  - * Anti-convulsants as per management of Status epilepticus
  - * Anti-edema measures in cases with raised ICP Keep the head elevated 15- 30 degrees; Control fever and seizures; 20% mannitol 1.5 ml/kg eight hourly if not in shock
  - * Ventilatory support
  - * Blood components therapy if indicated
  - * Electrolyte/Glucose correction
  - * Neurosurgical intervention.
- Supportive care
  - * Eye care, skin care, bladder care.
- Monitor urine output, electrolytes, renal parameters, blood glucose.
- Nutritional support
  - * IVF as Dextrose with ½ NS at the following rate
  - * Up to 10 Kg 4 ml/kg/hour
  - * 10 –20 Kg 40 ml + 2ml/kg.hour for every kg above 10
  - * >20 Kg 60ml +1ml/kg/hour for every kg above 20
- Start oral feeds as early as possible.

Indications for referral

1. Worsening sensorium

2. Suspected increased intra-cranial pressure
3. Suspected intracranial focal pathology





Note

ALOC: Acute loss of consciousness

ICP: Intracranial pressure

HSV: Herpes simplex virus

Febrile Seizures

- Common between 6 months and 5 years of age
- Family history of febrile convulsion may be present
- Occurs frequently when the core temperature is more than 39oC (102oF)
- Convulsions typically of generalized tonic, clonic type
- Duration of seizures from few seconds to 10 minutes
- Causes of febrile seizures are commonly
  - Respiratory infection
  - Ear infection
  - Acute dysentery
- Nearly 50% of children have recurrent febrile seizures
- No evidence of underlying meningeal disease
- Febrile seizures occurring less than one year of age for the first time have more recurrences.

Rx Treatment

Prevention of further febrile seizures is mainly by early and effective treatment of fever. Only few children require long-term anti-convulsants to prevent recurrence.

- Tepid sponging
- Reassurance to the parents
- Antipyretics – Paracetamol 10-15 mg/kg/dose
- Symptomatic treatment
- Anticonvulsant not required routinely
- Intermittent diazepam 0.3 mg/kg/dose twice daily starting on the day of fever for 2 to 3 days (low dose clobazam is the other option).

Indications for anticonvulsant prophylaxis

- Atypical febrile seizures
  - Age below 6 months or above 5 years
  - Focal seizures and
  - Prolonged seizures – more than 15 minutes
- Post-ictal palsy
- Inter-ictal EEG abnormality
- Family history of febrile / afebrile seizures
- Recurrent febrile seizures
  - Long-term Sodium valproate or phenobarbitone may be used
  - Intermittent diazepam or Clobazam at the onset of fever is the other option

Treatment of Acute Respiratory Infection

Acute pharyngotonsillitis

- Fever, sore throat, foul breath, difficulty in swallowing, voice change, referred otalgia and cough are the clinical features
- Nasal symptoms may be seen (nasopharyngitis)
- Aetiology can be bacterial
  - Group A β-hemolytic streptococcus – GABHS or
  - Viral: Rhinovirus
- Clinical differentiation between bacterial (streptococcal) and viral aetiology is not foolproof, but the following features help

Nasal discharge, conjunctival involvement or cough besides throat involvement are more common with viral infection; severe pharyngeal congestion with yellow, thick exudates, tender anterior cervical lymphadenopathy, toxicity and absent coryza or cough point towards streptococcal tonsillitis.

Rx Treatment

- Supportive care: Increased fluid intake, paracetamol
- Antibiotics in suspected streptococcal tonsillitis to reduce morbidity and prevent long-term complications such as rheumatic fever:
  - Oral Penicilin V
    - 250 mg bid X 10 days for children < 30 kg
    - 500 mg bid X 10 days for children > 30 kg
  - Oral Amoxycillin
    - 40 mg/kg/day in 3 doses X 10 days for children < 30 kg
    - 250 mg tid X 10 days for children > 30 kg
  - Inj. Benzathine Penicillin 12 lac units deep IM once (6 lac units for children < 30 kg)
- If allergic to Penicillin
  - Oral erythromycin 40 mg/kg/day in 3 doses for 10 days

Pneumonia

- ARI (Pneumonia) is responsible for 20% of Under 5 mortality.
- Aetiology varies with age
  - * Upto 3 months of age
    - » Gram negative bacteria and Group B streptococcus
  - * 3 months – 5 years
    - » Streptococcus pneumoniae
    - » H.influenzae, viruses
    - » Staph. aureus
  - * > 5 years of age
    - » S pneumoniae
    - » Mycoplasma pneumoniae
    - » viruses

Clinical features

- Fever
- Fast breathing (RR: Up to 2 months of age: 60/ minutes or more; 2 months - up to 12 months: 50 or more; 12 months – up to 5 years: 40 or more) count RR for full 1 minute when child is not crying
- Increased work of breathing – IWB (Nasal flaring, chest indrawing and grunt)
- Crackles/ bronchial breathing on auscultation is heared if pneumonia is confined to a lobe

Rx Treatment

- Chest X-ray need not be done routinely if no complication (pleural effusion, non improvement) suspected
- Children up to three months of age and children with severe pneumonia (IWB, not feeding, cyanosis, intermittent apnea, signs of dehydration) are treated as in-patients with parenteral antibiotics.
- They may also require close monitoring (clinical and pulse oximetry), IV fluids, oxygen and sometimes ventilatory support
- All pneumonias are treated as 'bacterial' with antibiotics

For non-severe pneumonia (domiciliary oral treatment and follow up)

- 3 months – 5 years
  - * Amoxycillin 40 mg/kg/day in 3 doses X 7-10 days or
  - * Chloramphenicol 50-100 mg/kg/day in 4 doses X 7-10 days
- 5 years plus
  - * Amoxycillin (First line)
  - * Erythomycin or chloramphenicol or coamoxi – clav (second line)

For severe pneumonia and children upto 3 months of age (Hospitalization and IV antibiotics)

- Upto 3 months (for 7 - 10 days)
  - * Inj cefotaxime 100 mg/kg/day in 3 or 4 divided doses

    +
  - * Inj Garamycin 5-7.5 mg/kg/day 2 divided doses or
  - * Inj Amikacin 15 mg/kg/day 2 divided doses
- 3 months – 5 years:
- First line for 7 to 10 days
  - * Inj Ampicillin IV 100 mg/kg/day in 4 divided doses or
  - * Inj Chloramphenicol 50-100 mg/kg in 4 divided doses
- Second line
  - * Inj Cefotaxime 150-200 mg/kg/day

In all age groups

- If staph. pneumonia is suspected treat for 14 days:
  - * Inj cefotaxime / ceftriaxone

    +
  - * Inj cloxacillin 100-200 mg/kg/day in 4 divded-doses (with supportive measures, IV fluids and oxygen administration)

When to refer

- Respiratory distress not improving
- Respiratory failure as indicated by increasing fatigue, cyanosis or altered sensorium

Bronchiolitis

- Viral in aetiology (Respiratory syncitial virus)
- Most frequently in children < 12 months of age
- Initial URI symptoms followed by
  - * Increasing cough
  - * Respiratory distress
  - * Wheeze and
  - * Feeding difficulty

Investigations

- CXR: Hyperinflated lungs with patchy infiltrates

Treatment

- Supportive measures such as oxygen by hood (10 litres / mt) or by mask (5 lits / mt); IV fluids if child is not able to feed orally.
- Monitoring (RR, Respiratory distress, pulse oximetry)
- A trial dose of Nebulised salbutamol / epinephrine if wheezing is marked
- As child improves wean off oxygen and increase oral feeds.
- If child develops severe respiratory distress, increasing hypo-xemia, cyanosis or fatigue – ventilatory support may be required.

Guidelines For Diagnosis And Treatment Of Empyema

- Characterised by presence of pus or microorganisms in the pleural fluid
- Occurs as a complication of pneumonia
- Staph aureus, Streptococcus pneumoniae, Hemophilus influenzae and Streptococcus pyogenes are the common organisms
- Common symptoms are
  - * Fever
  - * Chills
  - * Toxemia
  - * Respiratory distress
  - * Grunt and
  - * Chest pain (pleuritic pain)

On examination

- Decreased chest expansion
- Diminished breath sounds and
- Dullness on percussion on affected side and
- Mediastinal shift to opposite side

Investigations

- CXR: obliteration of costophrenic angle; diffuse homogenous opacity
- USG chest: size, site of effusion, adhesions or loculations can be made out
- Diagnostic thoracocentesis usually in fifth intercostal space over mid-axillary line using a large bore needle
- Pleural fluid for Gram stain, culture and sensitivity
- Pleural fluid pH and sugar are reduced and protein is elevated.

Treatment

- Treatment comprises of chest drain and antibiotics

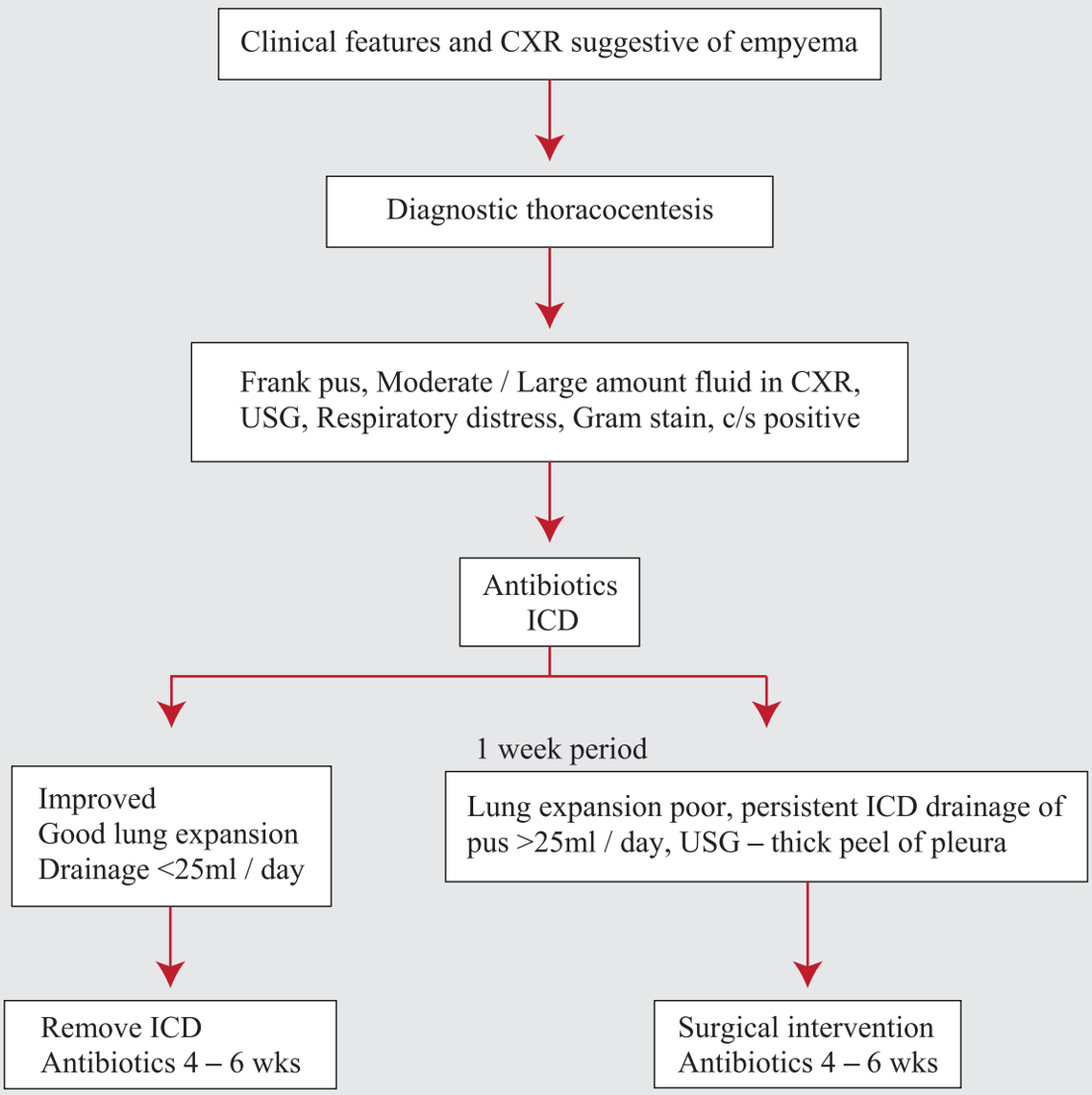
Chest drainage

- Using an intercostal drainage tube inserted in the region of maximal dullness (usually V or VI intercostal space in axillary region) and connecting to a sterile under water drainage bottle
- Chest drainage is kept till the drainage decreases to < 25 ml/day and there is good lung expansion.
- If there is no chest expansion by clinical or radiological methods, surgical opinion is sought

Antibiotics

- Cloxacillin with cefotaxime or ceftrioxone is the first line antibiotic; switch over to oral antibiotics after child becomes afebrile and chest tube is removed. Total duration of 4-6 weeks of antibiotic therapy.
- Cloxacillin: 100-200 mg/kg/day in 4 div. doses
- Cefotaxime: 150-200 mg/kg/day in 3 or 4 div. doses
- Supportive care: oxygen, good nutrition

ALGORITHM: TREATMENT OF EMPYEMA



Approach to Fever

History

- Type of fever
- Associated symptoms – chills / rigor, cough, sore throat, ear pain, urinary symptoms, bleeds etc
- Previous illness and treatment if any
- Feeding difficulty, respiratory distress

Clinical examination
Check Temperature. Blood pressure, Pulse, Perfusion
Skin: Rashes, Bleed, Cyanosis
Eyes: Pallor, Icterus
Mouth: Ulcer, Thrush
Ear: Discharge, Redness, Tenderness
Throat: Congestion, Tonsillitis
CNS: Meningeal irritation, Altered sensorium
Abd: Hepatomegaly, Splenomegaly
RS: Tachypnea, Retraction, Creps, Wheeze

Basic investigations in high risk group and fever beyond 5 days in low risk:

- Total count, differential count, peripheral smear,
- Platelet count
- Urine analysis, urine c/s
- Blood culture and sensitivity
- Chest x-ray
- C-reactive protein
- Mantoux test
- CSF analysis if required

Other investigations

- Liver function test
- Renal function test
- USG abdomen
- Blood for leptospirosis
- Serology for dengue
- Widal
- Bone marrow

Notification

Remember to Inform the health authorities in Notifiable diseases.

Referral

- Fever with unconsciousness
- Fever with shock
- severe respiratory diseases
- Bleeding diathesis
- Refractory seizures

Treatment Of Malaria In Children

Approximately 2.48 million malaria cases are reported annually from South Asia of which 75% cases are contributed by India alone. Plasmodium falciparum resistance to standard antimalarial drugs particularly chloroquine is on the rise.

Clinical features

- Fever: Though the classical intermittent fever is described in Malaria, any type of fever can occur.
- Chills, rigors and sweating are the other characteristic features.
- Pallor and splenomegaly are often seen in Malaria
- High grade fever, jaundice, altered sensorium and shock can occur in falciparum malaria (complicated malaria)
-

Investigations

- Microscopy
  - Light microscopy of well-stained thick and thin films. Blood sample collection for smear should be done before starting antimalarials and smears should be prepared soon after blood collection.
- Rapid diagnostic tests
  - These are immunochromatographic tests to detect plasmodium antigens in blood
- Histidine rich protein (HRP-II) test
  - To detect P.falciparum (both asexual stages and young gametocytes)
- Parasite lactate dehydrogenase (LDH) test
  - To detect falciparum and vivax malaria.

Treatment

- Chloroquine 10 mg base/kg stat followed by 5 mg/kg at 6, 24 and 48 hours.

or

- Chloroquine 10 mg base / kg stat followed by 10 mg / kg at 24 hours and 5 mg / kg at 48 hours (Total dose 25 mg / kg).
- In case of vivax malaria to prevent relapse, Primaquine 0.25 mg / kg / dose for 5 days
  - * Age group 12 months – 5 years
  - * 1 tablet Primaquine tablet containing 2.5 mg base once daily for 5 days
- Primaquine should not be given to children up to 1 year of age and during pregnancy.

Antimalarial therapy of severe and complicated malaria (due to P. falciparum)

IV Quinine or parenteral Artemisinin

Quinine:

- 20 mg salt / kg (loading dose) diluted in 10 ml / kg of isotonic fluid by infusion over 4 hours.
- After 12 hours of starting the loading dose, give maintenance dose of 10 mg / kg over 2 hours.
- This maintenance dose is to be repeated every 8 hours until the patient can swallow, then quinine tablets 10 mg / kg 8 hourly to complete a 7 day course.

or

Artesunate

- 2.4 mg / kg / IV loading dose followed by 1.2 mg / kg at 12 and 24 hours, then 1.2 mg / kg daily for 6 days.
- If patient is able to swallow, then the daily dose can be given orally.

or

Artemether

- Artemether 3.2 mg / kg IM loading dose followed by 1.6 mg / kg daily for 6 days.
- If the patient is able to swallow, then the daily dose can be given orally.
- At the end of therapy a single dose of Sulfadoxime – Pyrimethamine (calculated as 25 mg / kg of sulfadoxime) or Mefloquine 25 mg / kg (in 2 doses of 15 mg / kg and 10 mg / kg 4-6 hours apart) is to be given.
- A single dose of Primaquine (0.75 mg / kg) is to be given for gametocytocidal action.

Supportive management in complicated malaria

- Continuous clinical monitoring
- Care of Airway, Breathing and Circulation (A B C)
- Blood Sugar values.
- Promt treatment of seizures

Dengue Infection

Dengue infection is caused by one of the four serotypes of Dengue virus (Dengue 1 to Dengue 4) transmitted by the mosquito vector, Aedes aegypti. WHO estimates that 20 million dengue cases occur every year worldwide of which, about 5 lakh cases are those of Dengue Hemorrhagic fever with case fatality rate of 1-5%. Dengue infection causes two important clinical syndromes:

- Dengue fever
- Dengue hemorrhagic fever (DHF) including Dengue Shock Syndrome (DSS).

Dengue fever

It is a benign, non-fatal febrile illness indistinguishable from other common febrile illnesses.

- Acute fever – biphasic (initial fever of 1-7 days, then afebrile for 1-2 days followed by fever again).
- Headache, myalgia, arthralgia
- Rash (Macular, generalized rash seen in the first 48 hours of fever)
- Lymphadenopathy
- Leukopenia
- A second rash may appear within 1-2 days of defervescence.

DHF / DSS

- Fever of 2-7 days duration.
- This is followed by rapid deterioration in the form of restlessness, irritability, abdominal pain, vomiting, liver enlargement.

WHO criteria for DHF

- Fever of 2-7 days
- Thrombocytopenia (platelet count of <1,00,000/cmm)
- Bleeding manifestation (skin and mucosal bleeding)
- Evidence of plasma leak (Greater than 20% rise in average hematocrit level for age and sex, greater than 20% drop in hematocrit level following volume replacement or pleural effusion, ascites or hypoproteinemia)

DSS

Above criteria plus circulatory failure characterized by:

- Rapid, weak pulse
- Narrow pulse pressure (<20 mm/kg)
- Hypotension
- Cold, clammy skin

The critical period of fluid leak and shock is the peri-defervescence period, i.e. about 1-2 days before and 1 – 2 days after the fever subsides. Hence close watch is required from 2nd day of fever for early signs of shock.

If this phase is not recognized and treated promptly, fatal shock and hemorrhage can occur rapidly. Plasma leakage, sometimes sufficient to cause shock, is the most important complication of dengue infection in children

Investigations

- In dengue fever non-specific features such as leucopenia and mild thrombocytopenia may occur.
- In DHF, increase in haematocrit of more than 20% of base line value and thrombocytopenia (<1,00,000/cmm).
- Moderate elevation of SGPT and hypoalbuminemia
- X-ray: Pleural effusion, usually right sided.
- USG abdomen: Ascites
- Dengue serology: It has some pitfalls and limitations. In early dengue infection (first 5 days of fever) serology may be negative. A single sample may be sent for IgG and IgM dengue antibodies after 5 days of illness
- The ELISA tests (IgM capture ELISA and IgG capture ELISA) and rapid immunochromatographic card tests are most commonly used.

In primary dengue infection IgM is positive (80% by day 5, 99% by day 10-20). In secondary dengue infection there is a brisk and rapid IgG response with a slower and lower IgM response.

Treatment

- In uncomplicated dengue fever, rest, oral paracetamol and oral fluids will be sufficient. Avoid NSAIDs
- All dengue patients must be observed for complications for at least 2 days after recovery from fever.
- Parents must be informed of the following danger signs which require hospitalization:-
  - * Abdominal pain
  - * Any bleeding
  - * Irritability, drowsiness
  - * Altered sensorium
  - * Poor feeding
  - * Sweating and cold skin

Danger signs are features of shock (rapid, weak pulse, narrowing of pulse pressure, hypotension), hematocrit of >40 or rising hematocrit, platelet count of <1,00,000/cmm or evidence of plasma leakage.

Gradation of severity of Dengue Hemorrhagic fever

Grade	Symptoms	Lab findings
DHF I	Dengue fever features plus positive tourniquet test	Platelets < 1,00,000/ cmm Hct rise ≥ 20%
DHF II	Above signs plus spontaneous bleeding	Platelets < 1,00,000/ cmm Hct rise ≥ 20%
DHF III	Above signs + circulatory failure (weak pulse, hypotension,restlessness)	Platelets < 1,00,000/ cmm Hct rise > 20%

DHF IV	Profound shock with undetectable BP and pulse	Platelets < 1,00,000/ cmm Hct rise > 20%
DHF III and IV= Dengue shock syndrome (DSS)		

DHF grade I and II

- DHF I can be managed with oral rehydration therapy and close monitoring. If oral fluids are not tolerated and for DHF II, IV fluids are required
- Early volume replacement of lost plasma with isotonic crystalloid solution can reduce the severity of disease and prevent shock
- Usually IV fluid therapy for 12-24 hours with frequent clinical and lab (Hct) monitoring is required. IV fluid therapy is not recommended in dengue infection when plasma leak is not suspected.z

DHF grades III and IV

- All children with DSS should be managed in the hospital, where facilities for frequent clinical and htaematocrit monitoring are available.
- Guidelines for volume replacement in DHF III & IV (DSS) are depicted in fig. 2.
- To ensure adequate volume replacement and to prevent excessive fluid administration the rate of fluid administration should be carefully adjusted during the 24-48 hour period of plasma leakage.
- Fluid overload may occur due to
  - Excess and /or too rapid IV fluids
  - Inappropriate use of hypotonic rather than isotonic crystalloid solution
  - Continuation of IV fluids for too long even after the plasma leak has resolved
  - Unnecessary use of large volume of IV fluid in children with severe leak.
- Fluid overload may cause respiratory distress from massive pleural effusion, ascites and pulmonary oedema. This can be dangerous.

Clinical Features of recovery

- Child becomes more alert,active
- Wants feed
- Urine output improves
- Clinical signs of shock disappear

Typhoid Fever

- Caused by Salmonella typhi
- Also by Salmonella paratyphi

Clinical pattern:

- Ranges from mild gastroenteritis to severe bacterial sepsis
- Fever, coated tongue
- Vomiting, abdominal distension, loose stools
- Altered sensorium can be present in severe cases
- Hepatomegaly and soft splenomegaly may be present

Complications:

- Intestinal perforation
- Hemorrhage
- Toxic encephalopathy

Diagnosis:

- Blood culture – 1st week
- Widal – 2nd week
  - Significant if 'O'titer >1:160

Differential Diagnosis

- Septicemia, malaria, leptospirosis, UTI

Rx Treatment

- Uncomplicated typhoid: Oral cefixime 15-20mg/kg/day (I Pine for 14 days or Oral chloramphenicol 50-75mg/kg/day or TMP-SMX 40mg/kg/day of SMX for 14 days
- IV fluids if child doesn't take oral feeds
- Injection ceftriaxone 75-100 mg/kg in two divided doses – 14 days
- Oral cefixime can be used for follow up thereby after improvement to complete 14 days course.
- Injection Ciprofloxocin 10-15 mg/kg in two divided doses – 2 weeks in life-threatening infection resistant to other drugs
- Antipyretics
- Tepid sponging

Prevention

- Typhoid vaccine (0.5 ml) available in single dose or multi dose vial

- Take care of hygiene, sanitation, cooked food
- Eat well cooked food including boiled milk

Notification:

- Notify authorities

Urinary Tract Infection (UTI)

- Approximately 8% of girls and 1-2% of boys are likely to get an UTI during childhood.
- A significant proportion of children less than 2 years developing UTI have underlying urinary tract anomalies, most often vesico ureteric reflux (VUR). UTI in a setting of VUR may lead to renal scarring, an important cause of chronic renal disease. Early recognition and treatment of UTI and urinary anomalies is essential to prevent such complications.

Clinical features of UTI

- Neonates
  - * Sepsis like features with fever or hypothermia, lethargy, poor feeding, Poor weight gain, jaundice and shock; urinary symptoms may be absent.
- Infants and children below 2 years
  - * Unexplained fever; urinary symptoms minimal or absent.
- Adolescents
  - * Mostly related to lower urinary tract such as dysuria, frequency, urgency and suprapubic pain. Renal parenchymal involvement is indicated by high fever, chills, rigors and flank pain.

Investigations

- Urine analysis
  - * May suggest UTI in the form of increased leukocytes in urine. Gram stain of centrifuged urine specimen may show bacteria.
- Dipstick for nitrite reduction and leukocyte esterase may help in rapid diagnosis.
- Urine culture
  - * This is the only confirmatory test for UTI.

Every effort must be made to properly collect and send a urine sample before antibiotic is started. In infants and young children UTI should be suspected if there is unexplained fever.

- A midstream clean catch specimen is ideal. Soap or antiseptic solution should not be used before collection. In infants urine can be obtained by suprapubic aspiration.
- Common organisms responsible for UTI are
  - * E. coli
  - * Occasionally Klebsiella
  - * Staph epidermidis or Strep fecalis may be responsible.
- A colony count of > 105 colony forming units (CFU) / ml of single species in a clean catch specimen indicates significant bacteriuria. Presence of any bacteriuria in suprapubic specimen is significant.

Treatment

For the purpose of management UTI is divided into complicated and uncomplicated UTI.

Complicated UTI

- Temperature >390C, persistent vomiting, renal angle tenderness and systemic toxicity are features of complicated UTI
- Infants below 3 months of age and those with complicated UTI should receive parenteral antibiotics initially.

Options

1. Cefotaxime 100-150 mg/kg/day in 3 div doses
2. Ceftriaxone 75 mg/kg/day in 1-2 doses
3. Gentamycin 5-7.5 mg/kg/day single dose
4. Amikacin 15-20 mg/kg/day single dose

- In young infants (< 3 months) entire treatment is parenteral
- For older children, after first 2-3 days, oral antibiotics may be started based on antimicrobial sensitivity
- Total duration of treatment is 10-14 days.

Oral antibiotics

Amoxicillin	20 - 40 mg/kg/day in 2-3 doses
Cefadroxil	30 mg/kg/day in 2 doses
Cephalexin	50 mg/kg/day in 3 doses
Cefixime	8 mg/kg/day in 2 doses
Ciprofloxacin	10 - 20 mg/kg/day in 2 doses

Uncomplicated UTI

Children >3 months of age and those who do not have features of complicated UTI can be treated with oral amoxicillin or cefadroxil for 7 to 10 days (based on sensitivity). Though fluoroquinolones are effective and safe for UTI, they are not the first-line antibiotics

Other investigations

To identify urologic abnormalities that predispose to renal scarring the following studies are recommended.

1. Ultrasonography
2. Voiding cytourethrography: For diagnosing VUR and defining bladder and urethral anatomy
3. Radionuclide scintigraphy: For identifying pyelonephritis and renal scarring.
4. Special investigations to rule out predisposing causes

Antibiotic prophylaxis

Recommended in

- Infants with UTI pending evaluation
- Children with VUR
- Recurrent febrile UTI

Cotrimoxazole (1-2 mg/kg/day of Trimethoprim), Cephalexin (10 mg/kg/day) or low dose Cefixime are commonly used in prophylaxis.

Leptospirosis

- Leptospirosis is a zoonotic disease which has recently emerged as a major public health issue. Rats and dogs are the major reservoirs in urban areas.
- Human infection occurs through contact with urine of an infected animal or the organisms entering through abrasions in skin or through conjunctiva; contamination of water supplies also can result in outbreaks.

Clinical features

Biphasic clinical presentation; variable, non-specific clinical features at different phases

I phase (Acute or septicemic phase)

- Usually lasts for a week
- Fever, headache, photophobia, myalgia, abdominal pain, conjunctival suffusion and skin rash.
- Differential diagnosis in this phase includes viral fever, dengue, typhoid fever, malaria, viral hepatitis and pneumonia.

II phase (Immune phase)

- This follows a brief febrile period of variable duration after the I phase of illness, may last for 4-30 days.
- Liver and kidneys are the most commonly involved organs
- Some develop jaundice (Weil's disease) followed by renal failure
- Many patients do not have jaundice. They may develop fever, rash and headache.
- Pulmonary manifestations such as cough, dyspnoea and hemoptysis may occur in a few.
- Unusually CNS manifestations (encephalitis, aseptic meningitis), CVS features (arrhythmias) or hepatobiliary manifestations (cholecystitis, pancreatitis) may occur.

Investigations

- Urinalysis: proteinuria, pyuria, microscopic hematuria
- Leukocytosis with thrombocytopenia in severe forms
- Features of hepatic and / or renal involvement
- Organisms (Leptospires) can be visualized in blood or urine by dark-field microscopy or immunofluroresence (High degree of false positivity)
- Cultures from blood, CSF or urine though are confirmatory, are tedious to carryout.
- Antibody detection (serology): commonly used tests in spite of certain limitations
    - e. Microscopic agglutination test (MAT) is the standard serologic test 1 : 100 / MSAT - 2+
    - f. Enzyme Linked Immunosorbent Assay (ELISA) for detection of Ig M antibodies increasingly used in view of its simplicity.

Treatment

1. Supportive care including adequate hydration.
2. In case of renal failure, peritoneal / hemodialysis.
3. Antibiotics:

* Effective antimicrobials should be instituted early.
* Parenteral penicillin G 2-3 lacs / kg / day in 4 to 6 divided doses for 7 days is the treatment of choice.
* Doxy / Tetracyclines in children more than eight years of age
* Oral amoxicillin, erythromycin and 3rd generation cephalosporins (Cefotaxime / Ceftriaxone) are also effective.

Refer early those children with

- Altered level of consciousness
- Renal / hepatic failure or multi-organ dysfunction
- Respiratory distress, shock

Viral Hepatitis

Aetiology

- Water born viruses: Hepatitis A and E
- Spread by blood and body fluids
  * Hepatitis B and C
- Hepatitis A virus (HAV) accounts for 20-25% of clinical hepatitis
- HAV and HEV transmission occurs primarily by faeco-oral route through contaminated water.
- HAV transmission can also occur person-to-person and also through food handlers.

Hepatitis A Infection

Clinical features

Hepatitis in children due to HAV may be anicteric or icteric and these children may have mild symptoms to severe and prolonged forms.

Prodromal phase (few days to 1 week): Anorexia, fatigue, abdominal pain nausea, vomiting, fever.

Icteric phase: Jaundice appears. High-coloured urine, pale stools; Itching may occur; fever, nausea and vomiting subside.

Convalescent phase: All symptoms gradually subside. Normalcy returns after variable period.

In children all these phases usually have a short duration.

Investigations

1. Increase in liver enzymes (SGPT, SGOT).
2. Increase in serum bilirubin; usually does not exceed 10mg/dl
3. Detection of IgM anti HAV indicates recent infection

Fulminant Hepatitis A

Very rarely hepatitis A may lead on to fulminant hepatitis characterized by deepening jaundice, GI bleeding, ascites and altered sensorium. Underlying chronic liver disease is a risk factor.

Treatment

- Only supportive treatment
- Dietary restriction does not have any role
- Potentially hepatotoxic drugs are to be avoided
- Antiviral agents do not have any role

Hepatitis E Virus (Hev) Infection

- HEV infection generally occurs in outbreaks, primarily after first decade of life.

Clinical features

- Mild gastroenteritis followed by jaundice, anorexia, abdominal pain, nausea, vomiting, fever and pruritus.
- Self-limited course; Can be severe in pregnant women
- Serological diagnosis: IgG, IgM and IgA antibodies to HEV antigens by enzyme immunoassay.

Hepatitis B Infection

Spread occurs through contact with infected blood and body fluids. Vertical transmission (from mother to newborn) and later horizontal transmission through body fluids can occur.

Clinical features

- If infection is acquired in perinatal period 90% cases will develop chronic hepatitis; if it is acquired 1-5 years of age, then 20-50% risk of developing chronic hepatitis; older children and adults have <5% chance of progressing to chronic hepatitis.
- Children may be asymptomatic or may present with acute viral hepatitis.
- Extra- hepatic features such as arthralgia and skin manifestations (urticaria, maculopopular lesions) may occur.

- Other immune mediated features such as polyarteritis, glomerulonephritis and papular acrodermatitis may occur.
- The course is insidious and lasts longer than HAV infection
- Chronic HBV infection includes chronic hepatitis, cirrhosis and hepatocellular carcinoma.

Investigations

Serology

- Following acute infection, HBs Ag (Hepatitis B surface antigen) appears first. It may eventually disappear after clinical recovery.
- HBe Ag is normally present during the acute phase and is indicative of a highly infective state.
- HBV DNA is also detectable in this phase.

Treatment

Antiviral drugs (Interferon and lamivudine) are used in the treatment of chronic HBV infection with the aim of suppressing HBV replication and ultimately elimination and also to decrease necro inflammation in liver and prevent hepatic decompensation. However they are not used routinely.

Prevention of Hepatitis B infection

Immunization with 3 doses of hepatitis B vaccine is effective (either starting at birth or later at 6 weeks along with DPT) (Ref. Chapter. Immunization).

If mother is HBsAg positive, newborn baby is given hepatitis B immunoglobulin in addition.

Screening blood for HBsAg before transfusion and safe injection practices also will prevent hepatitis B transmission.

Hepatitis C Infection

Hepatitis C virus (HCV) is fast emerging as an important cause of chronic hepatitis and primary hepatocellular carcinoma. Blood transfusion is the principal mode of infection followed by vertical transmission.

Clinical features

- Acute hepatitis can occur in a small minority of patients.
- Chronic infection is characterized by chronic hepatitis, cirrhosis and hepatocellular carcinoma slowly developing over decades.

Investigations

- The following lab tests are useful:-
  - * Anti HCV antibodies
  - * HCV RNA levels
  - * Liver biopsy to stage the necro inflammatory changes.

Treatment

- To get a sustained virological response by clearing of HCV RNA.
- Interferon α and Ribavirin are used under specific indications.

Pyogenic Meningitis

- Causative organisms depend on the age group
- In the neonatal period and upto 2 months the common organisms are
  - * Gram negative bacilli
  - * Group B streptococci
  - * Listeria monocytogens etc
- In the age group more than 2 months the common organisms are
  - * Hemophilus influenzae
  - * Streptococcus pneumoniae
  - * Neisseria meningitidis etc

Symptoms and signs

Upto 2 months of age

- Lethargy unconsciousness
- Not taking feeds
- Bulging anterior fontanalle
- Movements less than normal
- Hyperthermia or hypothermia
- Feeble cry
- Irritability or incessant cry
- Seizures

>2 months

- Fever
- Headache
- Vomiting
- Irritability

- Bulging anterior fontanalle (Upto 18 months)
- Altered level of consciousness
- Neck stiffness
- Seizures
- Petechiae / rashes
- Hypotension

Investigations

Diagnosis of Pyogenic Meningitis

1. Early diagnosis depends on high index of suspicion
2. Definite confirmation is by CSF analysis only. There are very few contra indications for lumbar puncture such as
   * Signs of raised intracranial tension
   * Bleeding diathesis
   * Infection at the site of Lumbar Puncture (LP)
3. CSF has to be sent for:
   * Cell count
   * Smear for Gram stain
   * Biochemistry (CSF protein raised; sugar decreased <40% of simultaneous blood sugar)
   * Culture and sensitivity
   * P. C. R. tests / Latex agglutination tests may help in rapid diagnosis and also in partially treated cases of pyogenic meningitis
4. Indications for neuro–imaging
   * When the diagnosis is doubtful
   * Signs of increased intracranial tension
   * Focal neurological signs
   * When the clinical response is not satisfactory

Treatment

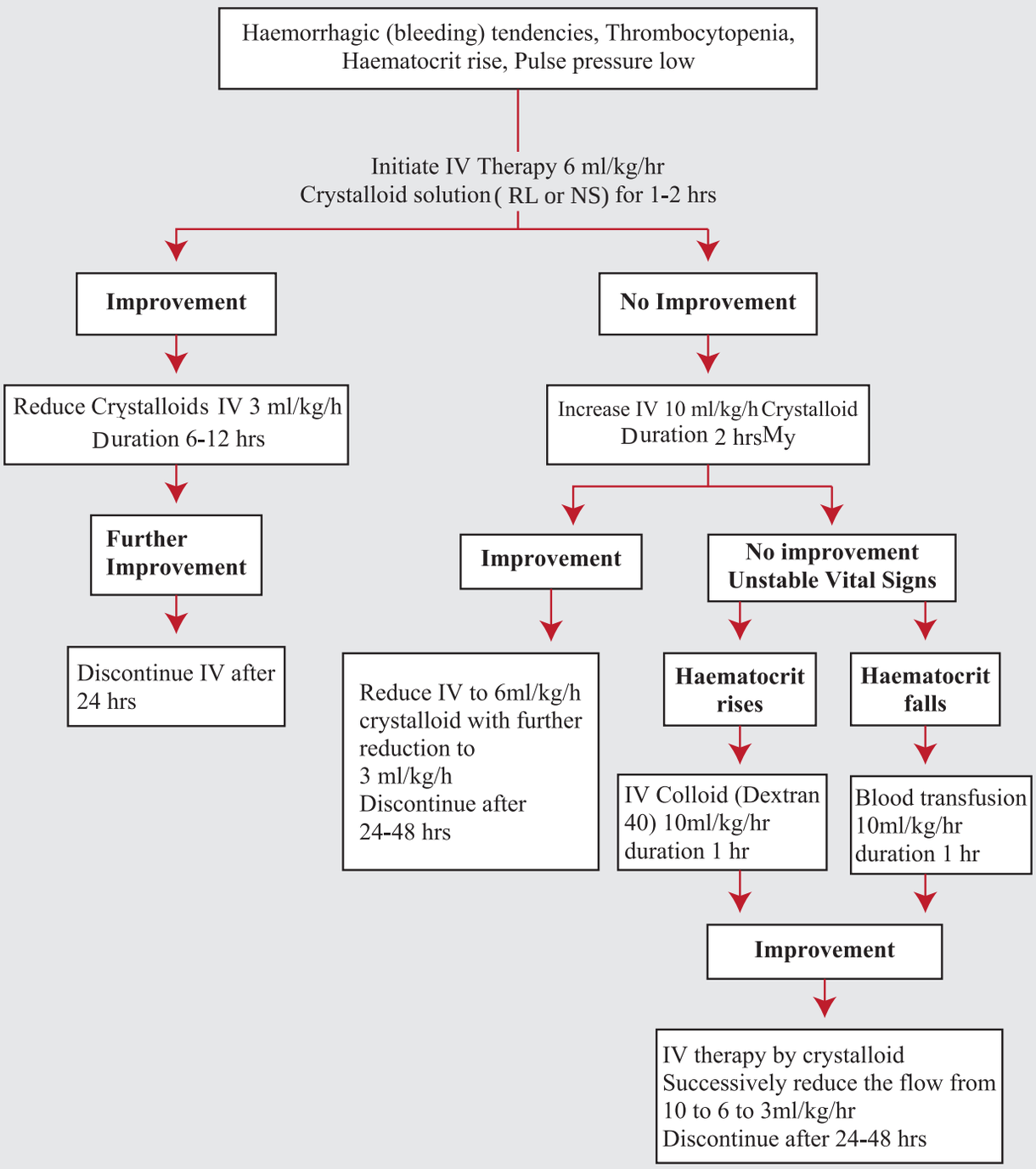
Selection of antibiotics as per age, etiology and immune state.

1. For immuno competent children Inj. Ceftriaxone 100 mg/kg/day IV in 2 div. doses for 10 – 14 days Or Inj. Cefotaxime 200mg/kg/day IV in 3 or 4 div. doses for 10 – 14 days
   * Modify antibiotic based on clinical improvement and C/S report if necessary
   * Duration of antibiotic for Gram negative meningitis (Usually in newborn): 14-21 days
2. Steroids: Inj. Dexamethasone - 0.1 to 0.2 mg/kg/dose, once in 6 – 8 hours
   * Not indicated in newborn
   * To be given 1 – 2 hours before first dose of antibiotics.
3. When to repeat C. S. F. analysis?
   * Young children
   * No satisfactory clinical response
   * Gram negative bacterial meningitis
4. Other supportive measures:
   * Stabilization of airway, breathing and circulation
   * Fluid balance: No need to restrict intake. Maintain normal circulating volume
   * Anti-oedema measures:
     » Mannitol (20%) - 1 – 2 ml/kg/dose, once in 8 hours
     » Inj. Dexamethasone – 0.25 – 0.5 mg/kg/dose, may be repeated once in 6 hours
     » Frusemide 1 mg/kg per dose
   * Control of fever
   * Anticonvulsants - Phenobarbitone or Phenytoin
5. During follow-up look for
   * Head circumference Hearing
   * Neuro cognitive defects
   * Epilepsy
   * Cranial nerve palsies

Referral

- Complicated or difficult cases
- When therapeutic response is not satisfactory
- Modify if meningococcal meningitis is suspected

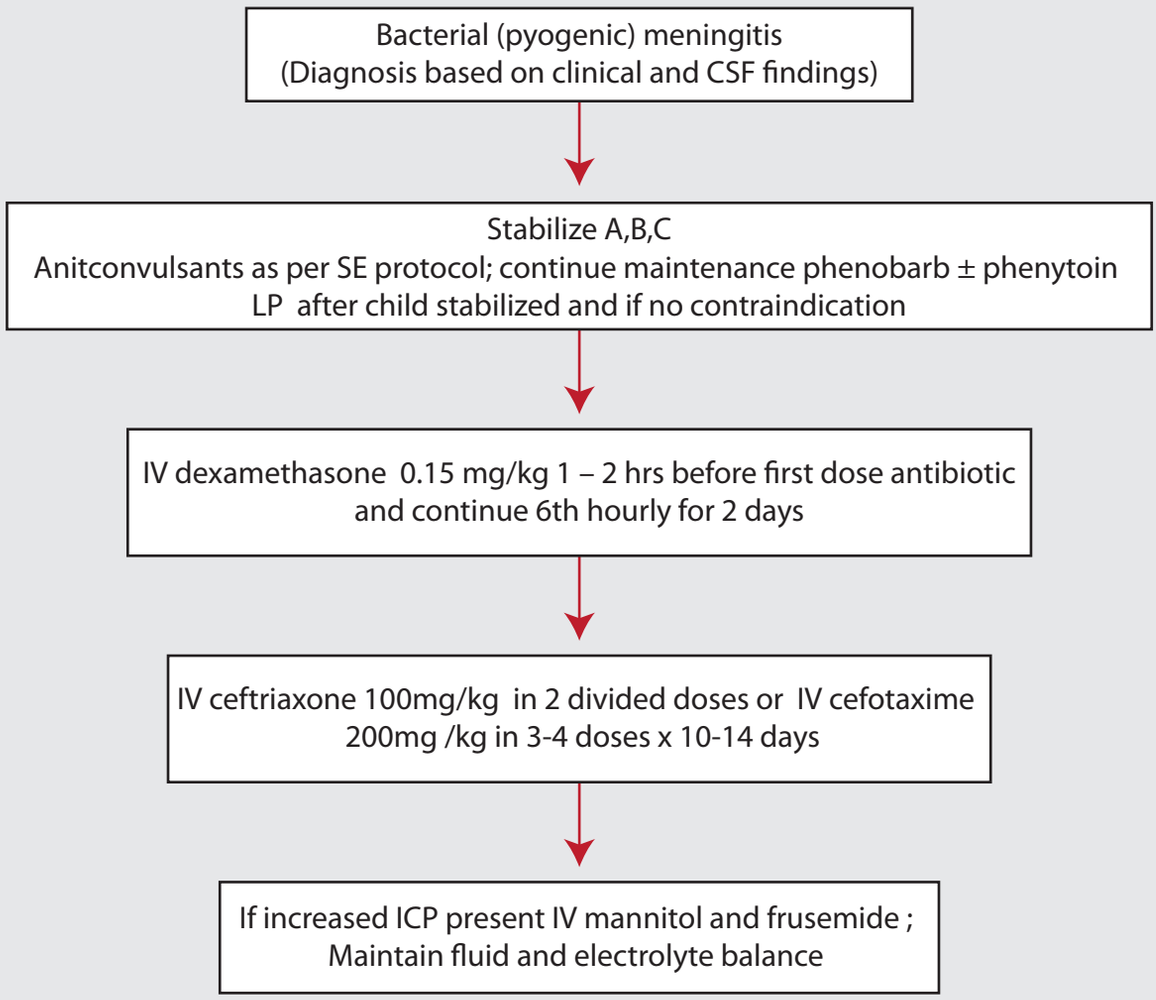
Volume replacement flow chart for patients with DHF grades I and II
Guidelines for Treatment of Dengue haemorrhagic fever in small hospitals



Improvement: Haematocrit falls, pulse rate and blood pressure stable, urine output rises
No improvement: Haematocrit or pulse rate rises, pulse pressure falls below 20mmHg, urine output falls
Unstable vital signs: Urine output falls, signs of shock

***Source:** *IAP specialty series on Pediatric Infectious Diseases, 1st Edition, Mumbai, 2006; pp779-281*

ALGORITHM: MANAGEMENT OF BACTERIAL MENINGITIS



Neuro-imaging if diagnosis doubtful or focal pathology or no improvement or complications such as hydrocephalus

Management Of Pediatric Tuberculosis Under RNTCP

- Diagnosis

  Suspect when:

  * Fever and / or cough for more than 3 weeks
  * Weight loss ± / no weight gain
- Based on:
  * Clinical presentation
  * Sputum examination wherever possible
  * Chest X-ray
  * Mantoux test
  * Contact history
- Tuberculous meningitis suspected in presence of:
  * Neurological symptoms such as irritability, refusal to feed, headache, altered sensorium
  * Vomiting

Treatment of Pediatric Tuberculosis

- DOTS is the recommended strategy for treatment in adults as well as in children
- All paediatric TB patients should be registered under RNTCP
- Category of treatment

- Category I: This category is generally prescribed to new sputum smear-positive patients. They have a high bacillary population with higher chances of having naturally occurring drug-resistant mutants. Therefore, four drugs are prescribed during the Intensive Phase (IP). Category II: These are cases that have had previous anti-tuberculosis treatment. Therefore, the chances of harboring resistant bacilli are higher. Hence, a 5 drug regimen is prescribed in the intensive phase, and the total duration of treatment is 8 months. Category III: These are sputum smear-negative cases with a low bacillary population. There is a lower chance for drug-resistant mutants. Therefore, a 3 drug regimen is prescribed.

Principles in treatment

Patients are classified into three categories for the purpose of treatment. The number of drugs and the duration of treatment are different in the three treatment categories of RNTCP (Table 2).

In patients with TBM on category I treatment, the four drugs used during the intensive phase should be HRZS(instead of HRZE).

- Continuation phase of treatment in TBM and spinal TB with neurological complications should be given for 6-7 months, thus extending the total duration of treatment to 8-9 months.
- Steroids should be used initially in hospitalized cases of TBM and TB pericarditis and reduced gradually over 6-8 weeks.
- In all instances, before starting a child on category II treatment, s/he should be examined by a pediatrician or TB expert.
- Any patient, pulmonary or extra-pulmonary, who is known to be HIV positive based on voluntary sharing of results and / or history of anti-retroviral therapy, is considered seriously ill For the purpose of categorization, HIV testing should not be done. Also, HIV status should not be revealed / recorded in any RNTCP document.
- All efforts should be made to ensure that every dose of medicine in the intensive phase and at least the first dose every week in the continuation phase are directly observed.
- Treatment observation should be done by someone who is accessible and acceptable to the patient and accountable to the health system. One of the most important responsibilities is to ensure that during the intensive phase of treatment (which is 2 to 4 months of directly observed administration of drugs) patients are swallowing every dose of their drugs under the direct observation of a DOT provider.
- To ensure proper drug administration, observe the DOT provider administering drugs to the patients and speak directly to patients to determine whether they have been receiving the correct number and type of drugs.
- After the patients swallow their drugs in the presence of a DOT Provider, those receiving streptomycin should be given the injections with sterile syringes and needles.

Chemoprophylaxis

- Recent infection with tubercle bacilli is one of the risk factors for disease development. The younger the child, the higher is the risk of breakdown of infection into disease. Therefore, child contacts of smear – positive tuberculosis cases, especially those below 6 years of age, must be screened for symptoms of tuberculosis.
- In cases of symptoms being present, the diagnostic algorithm for pediatric tuberculosis should be followed and the child should be given a full course of anti-tuberculosis treatment if she/he is diagnosed as a tuberculosis case.
- For asymptomatic children and those who are not found to be suffering from tuberculosis , chemoprophylaxis with isoniazid (5mg per kg body wt) should be administered daily for a period of six months. This is regardless of the BCG vaccination status.

Monitoring

- Child is reviewed at the end of 2 months of treatment. Satisfactory response is identified by improvement in symptoms and weight gain.
- Clinical follow-up and treatment is continued. On review, unsatisfactory response is assessed by non-adherence to treatment, weight loss and worsening symptoms. This child is referred to pediatrician or a tuberculosis specialist to decide whether he has to receive Category II treatment (Non-responder).

RNTCP Treatment Categories And Regimens For Children

Treatment category	Type of patients	Treatment Regimen***	
		IP	CP
Category I	New sputum smear – positive PTB, New sputum smear – negative PTB seriously ill * New extra – PTB, seriously ill*	$2H_3R_3Z_3E_3$***	$4H_3R_3$
Category II	Sputum smear – positive relapse, sputum smear – positive treatment failure Sputum smear – positive treatment after default	$2H_3R_3Z_3E_3S_3$ + 1 $H_3R_3Z_3E_3$	$5H_3R_3E_3$
Category III	New sputum smear – negative, not seriously ill ** New extra – PTB, not seriously ill **	$2H_3R_3Z_3$	$4H_3R_3$

* In children that are seriously ill, sputum smear negative pulmonary tuberculosis (PTB) includes all forms of sputum smear negative PTB other than the primary complex including military TB, cavitary disease. Seriously ill extra pulmonary TB (EPTB) includes TB meningitis (TBM), disseminated TB, TB pericarditis, TB peritonitis and intestinal TB, bilateral extensive pleurisy, spinal TB with or without neurological complications, genito–urinary TB, and bone and joint TB.

Note:

** Not seriously ill sputum smear-negative PTB includes primary complex. Not seriously ill EPTB includes lymph node TB and unilateral pleural effusion

*** Prefix indicates month and subscript indicates thrice weekly

IP = Intensive Phase CP = Continuation Phase

Suggested Pediatric Dosages For Intermittent Therapy

Drugs	Dosage (Thrice a week)
Isoniazid	10- 15 mg/kg
Rifampicin	10 mg /kg
Pyrazinamide	30-35 mg/kg
Ethambutol	30 mg /kg
Streptomycin	15 mg / kg

Immunization

National Immunization Schedule

Age	Vaccines
Birth	BCG, OPV-O
6 weeks	DPT 1, OPV 1
10 weeks	DPT 2, OPV 2
14 weeks	DPT 3, OPV 3
9 months	Measles
16-18 month	DPT booster, OPV 4
5 years	DT
10 years	TT
16 years	TT
Pregnant women	TT (2 doses at 4 weeks interval)

BCG Vaccine

Supplied as a lyophilized (freeze-dried powder) preparation; reconstituted with the recommended diluent supplied. Once reconstituted, the vaccine should be used within 4-6 hours.

Dose: 0.1ml

Route: Intradermal over deltoid insertion site of left arm

Following the vaccination there is no immediate reaction. A papule develops in 2-4 weeks, ulcerates and heals slowly over 6-12 weeks leaving a small scar.

Oral polio vaccine (OPV)

- Routine immunization with 6 doses
- National schedule 5 doses

- In Tamil Nadu, one more dose given with measles vaccine at 9 months.
- To achieve polio eradication and to interrupt circulation of wild polio virus in community annual polio immunization in the form of pulse polio (2 doses in Tamil Nadu) is given to all children upto 5 years irrespective of their prior immunization status.
- Polio vaccine is temperature sensitive, hence proper maintenance of cold chain till the vaccine is delivered to the child is extremely important. Dose: 2 drops orally

DPT Vaccine

It is a combination of diphtheria toxoid, whole-cell killed pertussis and tetanus toxoid. Dose: 0.5ml. Route: IM – over anterolateral aspect of thigh (Avoid injection over gluteal region). The protection following 3 doses is more than 95% for diphtheria and tetanus and 70-80% for pertussis. Following an injection, fever, local pain and induration may last for 1-2 days. Paracetamol may be given for these symptoms

Tetanus toxoid (TT)

Tetanus toxoid is given at 10 years and 16 years. Following this, booster doses are required only after every 10 years. There is no need to give Inj TT for every wound if the child has received the immunizations at the recommended age.

Measles vaccine

Measles vaccine is supplied in freeze – dried state (lyophilized powder) with the required diluent. The vaccine does not contain any antibacterial preservative. Hence strict aseptic technique should be used during reconstitution and administration. Reconstituted vaccine should be used within 4 hours.

Dose: 0.5ml

Route: Subcutaneously over arm or anterolateral thigh. Fever, coryza and mild measles like illness may occur in up to 10% of children between 5-10 days after immunization.

Other vaccines under consideration for inclusion in National Immunization schedule

Hepatitis B vaccine

It is a recombinant DNA vaccine. Immunogenicity is more than 95% with various schedules (Birth, 6 and 14 weeks; 6, 10 and 14 weeks at the same time of DPT vaccine but at a different site; Birth, 1 month and 6 months).

Dose: 0.5ml (10 microgram in children)

Route: IM over anterolateral thigh or deltoid region. Local pain and erythema may occur following the vaccination.

Important points to remember in immunization

1. Proper handling of vaccines and maintenance of cold chain is important
2. Minor illnesses such as cold, cough or mild diarrhea are not contraindications for immunizations.
3. If due to some reason a vaccine is delayed, the schedule need not be repeated; it should only be completed.
4. If any adverse reaction follows immunization, report to health authorities immediately.

Acute Flaccid Paralysis (Afp) Surveillance

Definition of AFP

- Sudden onset of weakness and floppiness in any part of the body in a child less than 15 years of age or paralysis in a person of any age in whom polio is suspected.
- AFP surveillance helps to detect reliably areas where polio virus transmission is occurring. To ensure that no case of polio is missed, all cases of AFP within 6 months of onset are reported and investigated with collection of two stool specimens within 14 days of onset.
- The non-polio causes of AFP include
  - Guillain – Barre syndrome (GBS)
  - Transverse myelitis and
  - Traumatic neuritis.

Stool sample collection

- 2 stool samples from the child at a minimum interval of 24 hours.
- Attempt to collect the stool samples within 14 days of onset of paralysis to maximize the probability of positive result. If not possible, the specimens should still be collected up to 60 days of paralysis onset.
- Each stool sample should be 8 grams (approximately the size of adult thumb).
- It should be transported to regional laboratory

under proper cold chain (20 – 80C) conditions so that it arrives at the lab in a good condition (no desiccation, no leak, with adequate documentation and evidence that the cold chain was maintained).

- Document the proper address of child so that follow up examination for residual paralysis can be carried out between 60-90 days following paralysis onset.

Outbreak response immunization (ORI)

- After AFP investigation and stool collection, ORI is organized in the community to administer one dose of OPV to approximately 500 children below 5 years of age form that locality / village.

Virological classification of AFP cases

- An AFP case is 'confirmed' as polio only by isolation of polio virus from any stool specimen.
- An AFP case is classified as "non-polio AFP" if wild polio virus is not isolated from adequate stool specimens.
- If stool specimens are inadequate, then the classification will depend on 60 days follow up examination. If the 60 days follow up examination shows no residual weakness, the case is classified as "non-polio AFP".

Reporting

- All cases of AFP should be reported to the notified surveillance medical officer immediately for further evaluation and action.

Treatment

- Supportive measures
- Avoid IM injection
- Ventilatory support may be required in progressive paralysis

Rheumatic Fever In Children

- Caused by Group A β hemolytic streptococcus
- Children from 5 to 15 years are the most susceptible.
- Increased number of cases occur in socially and economically disadvantaged groups

Diagnosis Of Rheumatic Fever - Modified Jones Criteria

- Major
  - Carditis
  - Migratory polyarthritis,
  - Chorea
  - Subcutaneous nodules
  - Erythema marginatum
- Minor
  - Fever
  - Arthralgia
  - Previous rheumatic fever
  - Increase in ESR, CRP
  - Prolonged P-R interval in ECG

For diagnosis of Rheumatic fever 2 major or 1 major and 2 minor criteria Plus Evidence of previous Group A streptococcus infection.

Differential Diagnosis

- Juvenile rheumatoid arthritis
- Infective endocarditis
- Septic arthritis
- Serum sickness

Investigations

- Throat culture for Group A streptococcus
- ASO for recent streptococcal infection
- Chest X-ray
- ECHO

Management

- Treat streptococcal infection:
- Inj. Benzathine Penicillin G 12 lakhs units single dose deep IM (6 lacs units for < 27 kg)

or

- Oral penicillin V 250 mg bid (500 mg bid for >30 kg) for 10 days

or

- Oral erythromycin 40 mg/kg/day in 3 divided doses for 10 days.
- Aspirin 90-120 mg/kg/day for 4 weeks

In carditis

- Prednisolone 2 mg/kg/day – 2 to 3 weeks and taper over another 2-3 weeks
- To treat cardiac failure as applicable

Treatment of rheumatic chorea

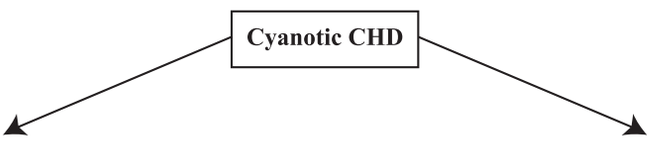
- Haloperidol 0.5 mg/day in 2 divided doses

Prevention

- Primary (Refer treatment of streptococcal infection given above)
- Secondary
  - * Inj. Benzathine Penicillin 12 lakhs units (6 lakhs <27 kg) once in every 3-4 weeks
    or
  - * Oral Penicillin V 250 mg BID daily

Congenital Heart Diseases (CHD)

- Incidence 58 per 1000 live births
- Commonest CHD – VSD, ASD, PDA, TOF



↑**PBF** (Pulmonary Blood Flow)

↓**PBF**

DORV (Double outlet right ventricle)

TOF

TGA with VSD

VSD with PS

Truncus arteriosus

Common atrium

Symptoms

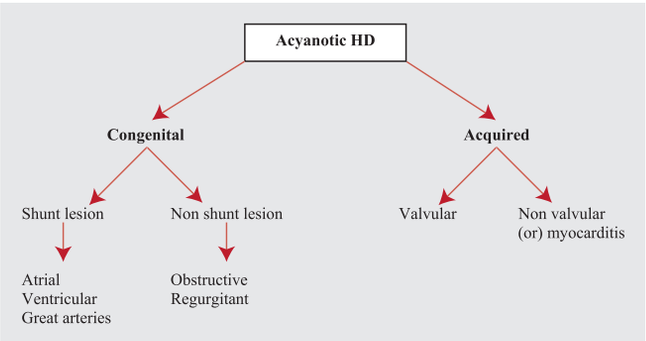
- Recurrent respiratory tract infections
- Failure to thrive
- Diagnosed usually in first 3 months of life
- Feeding problem
- Respiratory distress
- Presence of CCF

Severe cyanosis at birth

- Cyanotic spells
- Squatting episodes
- Ejection systolic murmur

X-ray

- Cardiomegaly
  Small heart
- Pulmonary plethora
  Pulmonary oligemia
- ECHO diagnostic
- Treat CCF as applicable (Ref. Chapter on CCF)
- Surgery is the definitive treatment



Approach –Acyanotic heart disease

- To look for shock or arrhythmia
- Look for facial dysmorphism
- Look for congenital malformation
- Growth parameter

Clinical features

Pulse

- Absence of arterial pulse in lower limb / radio femoral delay – coarctation of aorta
- Low volume pulse – aortic stenosis
- Collapsing pulse - aortic regurgitation, PDA
- Pulsus paradoxus – pericardial effusion
- Pulsus alternans – LVF

Blood Pressure

- Wide pulse pressure – aortic regurgitation, AV fistula
- Narrow pulse pressure – valvular aortic stenosis

Parasternal heave

- Present in right ventricular hypertrophy

Thrills

- Aortic area – aortic stenosis
- Pulmonary area – pulmonary stenosis
- Left sternal lower border – VSD

Various features of acyanotic CHD:

- VSD – asymptomatic or symptomatic
  - * some times presents with CCF
  - * Presence of pansystolic murmur in the left lower sternal border
- PDA – continuous murmur in the left first intercostal space
- ASD – mostly asymptomatic
  - * Wide split of second heart sound
- Coarctation of aorta – signs of acute circulatory shock in first 6 weeks of life
  - * Weak lower limb pulses

Investigations

Echocardiogram is diagnostic for acyanotic CHD

To treat CCF appropriately (Refer chapter 34)

Appropriate surgical management for each disease

Congestive Heart Failure (CHF)

Principles

- Control of excessive salt and water retention with diuretics
- Improve cardiac contractility with digoxin
- Prevent and reverse neurohormonal changes that lead to progressive worsening of cardiac status with beta blockers, ACE inhibitors

Digoxin

- Drug of choice in chronic CHF with atrial fibrillation
- In CHF with sinus rhythm it gives symptomatic benefit

Dose

- Total digitalizing dose
  - * In children 30-40 mcg/kg ½ the total dose stat: ¼ after 8 hrs; ¼ after 16 hrs
- Daily maintenance dose
  - * ¼ of total digitalizing dose. Once daily or 2 divided doses

Note:

Hypokalemia may aggravate digitalis toxicity especially with concomitant diuretic administration. Use oral Kcl supplement or use potassium sparing diuretic such as spironolactone.

Oral Diuretics

- Furosemide: 1-2 mg/kg/day
- Hydrochlorothiazide: 1-1.5 mg/kg/dose every 12-24 hours
- Spironolactone: 1-2 mg/kg/day

ACE inhibitors

- Indicated for all patients with congestive heart failure.
- Captopril: 0.1 to 0.5 mg/kg/dose oral every 8 to 12 hourly up to 4 mg/kg/day
- Enalapril: 0.1 mg/kg/dose oral every 12-24 hourly up to 0.5 mg/kg/day

β-blockers

- Integral part of congestive heart failure therapy nowadays.
- Metaprolol or carvedilol is used

Diet

- Calories - Recommended daily dietary allowance plus 20-30% in shunt lesions
- Avoid salty foods and additional salt in cooking
- Iron supplementation

When to refer

- Severe respiratory distress
- Acute pulmonary oedema
- Refractory CCF
- Cardiogenic shock

Nephrotic syndrome in children

Nephrotic syndrome is a clinical entity characterized by

- Massive proteinuria (>50mg/kg/day)
- Hypoalbuminemia (<2.5gm/dl)
- Edema
- Hyperlipidemia.

Definitions

- Frequent relapses
  - * Either 2 or more relapses within 6 months of

initial response or 3 or more relapses within a 12 month period.

- Steroid dependent
  * 2 or more consecutive relapses while steroids are still being tapered or within 4 weeks of stopping steroids.
- Steroid resistant
  * Failure to respond to oral Prednisolone of 2mg/kg/day for 4 weeks or to three doses of pulse Methyl prednisolone

Classification

- Congenital or Primary (idiopathic)
- Secondary. In children majority of NS are primary or idiopathic; 90% of the idiopathic NS are minimal change NS.

Clinical examination

- Periorbital puffiness/gain in weight, generalized edema with pleural effusion, ascites, diminished urine output.

Investigations

Urine Investigations

Proteinuria

- Preferable early morning specimen
- 24 hour urine protein
- The ratio of spot urine protein to creatinine (PCR) more than 3 is diagnostic of NS.
- Urine microscopy: broad waxy casts, hyaline and granular casts.
- Urine culture: Rule out infection before institution of steroid therapy.

Blood Chemistry

- Blood urea, serum creatinine
- Serum total protein will be reduced
- Serum albumin level is less than 2.5gms/dl.
- Serum cholesterol and triglycerides are raised in a nephrotic child
- Serum electrophoretic pattern will reveal a decrease in albumin level and a raised alpha 2 and beta globulins.
- Mantoux and chest X-ray should be done to rule out tuberculous infection.
- Ultrasound of the kidneys is routinely done to confirm the number, site and size of the kidneys.

Problems in nephrotic syndrome

Infection, acute renal failure, increased thrombo embolic manifestations, hypocalcemia

Management

- Diet
  * A balanced diet adequate in both calories and proteins (1-2gms/kg) is recommended.
  * Salt is restricted to 1-2gms/day if edema is present.
  * Fluid restriction is advised only if gross edema is present.
- Diuretics
  * Fluid is restricted to insensible losses in massive edema. Fluid restriction is not necessary in moderate edema Diuretics such as frusemide (1-2mg/kg) / spironolactone (2mg/kg) are used. In refractory cases 20% salt poor albumin 2ml/kg or 20ml/kg plasma followed by intravenous frusemide is advised.
- Infections: Associated infections such as UTI, skin infection, ARI, tuberculosis or peritonitis must be controlled, prior to steroid therapy.

Specific

Oral prednisolone 60mg/m2/ day daily for 6weeks

If urine is protein free it is followed by 40mg/m2/ alternate days for next 6 weeks and stopped. (APN regimen). More than 50% of children who respond within two months do so within first two weeks and 95% within first month.

Relapses: There is no single clinical or histological test to predict frequent relapses but occurrence of first relapse soon after initial attack predicts further relapses. Regular follow up in OPD to detect relapses at early stages before edema develops is sufficient to start therapy.

Frequent Relapses (>2 relapses in 6 months): The standard prednisolone schedule is started and tapered to a low dosage of 0.5mg/kg/48hrs in order to minimize side effects. Most children can tolerate a dosage of 0.5mg/kg/48 hrs and treatment should be given for a period of 12-18 months.

Infrequent Relapse (Less than two relapses in 6 months): Standard steroid therapy is started and if urine is protein free it is continued for 2 weeks and switched over to alternate days for four weeks and stopped.

Steroid Resistant: About 10-15% of the patients do not show remission of proteinuria and this is termed as a steroid-resistant nephrotic syndrome. For these patients alternate therapy can be considered.

Alternate Treatment

Alternate drugs should be considered in the following circumstances:

- Relapse on Prednisolone dosage of more than 0.5mg/kg/48hrs, steroid toxicity, pubertal age and steroid resistant NS.

Immunosuppressives

- Levamisole
- Cyclosporin A
- Tacrolimus
- Angiotensin converting enzyme inhibitors(ACE) and
- Angiotensin receptor blockers
- Pulse methyl prednisolone
- Lipid lowering agents.

Parental Counselling

The parents should be educated about the course and outcome of the illness.

Referral

Resistant nephrotic syndrome, frequent relapses, and complications need referral.

(missing algorithm for management of nephrotic syndrome book pg 70)

Acute Nephritic Syndrome

Etiology

Post-infective glomerulonephritis

Commonest cause is streptococcal infections of the throat and skin.

Clinical features

Abrupt onset of hematuria, edema, proteinuria, hypertension, oliguria, azotemia

Investigations

- Colour of urine (cola coloured)
- Urine deposits / RBC cast/proteinuria
- Urine proteinuria
- 24 hours urine protein
- Spot protein creatinine ratio (0.3 –3)
- Urea/creatinine/electrolytes
- Chest skiagram
- USG abdomen
- ASO/CRP
- Serum C3 levels if indicated

Monitoring

Blood pressure

Daily weight

Urea, creatinine

Intake/output

Complications

Pulmonary edema, hyertensive encephalopathy , hyperkalemia, uremic encephalopathy

Treatment

Control of edema

- Salt free diet / Inj Frusemide 1- 2 mg/kg/dose/
- Fluid restriction in severe edema-restrict to insensible water loss(400ml/m2 /day)

Control of hypertension

- T. Alpha methyl dopa 20- 40 mg/kg/day
- T.Enalapril 0.3 – 0.5 mg/day
- Sublingual Nifedepine 0.25 – 0.5 mg/kg/dose 6 hourly

Infection control

Appropriate antibiotics to control infection

Hyperkalemia

- Sodabicarbonate 1 ml/kg.dose intravenously very slowly
- 10% calcium gluconate 1 ml/ kg/dose IV slowly
- Inj Frusemide 1- 2 mg/kg/dose

- Salbutamol nebulisation
- Sodim polysterone sulfonate 1 gm/ kg/dose every 6 hours
- Peritoneal dialysis

Pulmonary edema

Supportive oxygen therapy, ventilation, Inj. Frusemide 5 mg/kg/IV

Hypertensive encephalopathy

- Sublingual Nifedepine 0.5 mg/kg/dose
- Seizure control with Diazepam, Phenytoin

Indications for referral

Pulmonary edema, persistent anuria, uremic encephalopathy and rapid deterioration of renal function

Protein Energy Malnutrition (PEM)

Definition of PEM

WHO (1973) has defined PEM as a range of pathological conditions arising from coincident lack, in varying proportions of protein and calories, occurring most frequently in infants and young children and commonly associated with infections.

Welcome classification of PEM

Weight for age	Oedema	Type
Less than 60%	+	Marasmic kwashiorkor
Less than 60%	Nil	Marasmus
60 – 80%	+	Kwashiorkor
60 – 80%	Nil	Under nutrition

Weight for age

- 1-7 years (age x 2) + 8 = Expected Weight
- >7 years (age x 7) - 5 ÷ by 2 = Expected weight

Look for the following

- Visible severe wasting
- Edema both feet
- Very low weight for age
- Look for associated infection
- Look for underlying vitamin/ trace element deficiencies.

Investigations

- To determine cause and degree of malnutrition
- To identify infections, infestations, vitamin and mineral deficiency
  - * Examination of stools
  - * Urine routine, culture and sentivity
  - * Hemoglobin, smear study
  - * Blood sugar, electrolytes
  - * Mantoux, X-ray chest
  - * Blood grouping
  - * Serum protein, albumin, globulin
  - * HIV status

Treatment

Phase I (24 – 48 hours - Resuscitative phase): Children presenting with life-threatening complications:

1. Stabilize airway, breathing and circulation
2. Look for life-threatening complications:
   - * Hypoglycemia
     - » Treat with 10% Glucose IV 2ml/kg followed by infusion with dextrose containing IVF
   - * Hypothermia
     - » Maintain warmth
   - * Hypokalemia
     - » Provide adequate potassium in the fluids at 40 meq/litre (2ml KCL /100ml of maintenance fluid)
   - * Shock
     - » Treat with normal saline boluses
   - * Dehydration
     - » Correction as per guidelines
3. Treatment of associated problems:
   - * Anemia
     - » Treat with oral iron after smears, stool study; 6 mg/kg elemental iron/day
   - * CCF
   - * Severe Vit. A deficiency

* Sepsis management with parenteral antibiotics

Phase II (Restorative phase)

Identify and treat underlying cause and continue to treat already identified complications. Oral feeds can be gradually started.

Phase III (Rehabilitative phase)

Dietary Treatment

1. Principle
   * Diet according to Recommended Dietary Allowance (RDA) and calculated for ideal weight for age
2. In Practice
   * Correct dietary imbalances
   * Increase whatever the child routinely takes at home
   * Add sugar and oil (within RDA) to make up calories (upto 30% of required calories can be in the form of sugar and oil)
   * Low cost: cereal - pulse preparation (NIN mix) as a nutritional supplementation.
3. Vit A supplementation (as in table)
4. Nutritional monitoring and follow up
5. Deworming with Albendazole 400mg as a single dose in children 2 years and above.

Cereal pulse multi-mix preparation NIN mix

Ragi	-	40 gm
Wheat	-	35 gm
Roasted Bengal Gram	-	15 gm
Ground Nuts	-	10 gm

This provides 13 gm of protein and 360 calories per 100 gm.

Treatment of vitamin A deficiency

Oral Oil miscible Vitamin A	< 1 year	> 1 year
Immediately	1 lakh IU	2 lakhs IU
Next day	1 lakh IU	2 lakhs IU
After 2 – 4 weeks	1 lakh IU	2 lakhs IU

Injection Vitamin A (water soluble)

- Oral vitamin A for infant < 6 months is 50,000 units/dose
  * In the presence of diarrhea and vomiting, Keratomalacia
    » Below 1year - 50,000 units IM/dose
    » Above 1 year - 1 lakh units IM/dose
- Given on day 1 and day 2 and day 14.
- Encourage green leafy vegetables, yellow vegetables, milk fortified with vitamin A.
- Prophylactic vitamin A single dose of vitamin 100,000 units at 9 months followed by 200,000 units at 18, 24, 30, 36 months.

When to refer in PEM

- Children with fulminant sepsis needing parentral nutrition
- Children suspected to have any life-threatening complication
- Children with no weight gain despite adequate therapy.

Management of Anemia

The most common form of anemia is iron-deficiency anemia caused by reduced dietary intake. It is easily treatable with supplemental iron and early intervention may prevent later loss of cognitive function. Following is the range of Hemoglobin (Hb) values in well-nourished children

Hemoglobin (Hb) values in well-nourished children

2 week	13	-	20g/dl
3 months	9.5	-	14.5g/dl
6m-6 yrs	10.5	-	14g/dl
7 – 12yrs	11	-	16g/dl

Simple recognition is by looking at the palmar pallor in comparison to examiner's palm.

Clinical features

Irritability, pallor (usually not seen until hemoglobin levels are less than 7 g per dl),pica, glossitis, systolic murmur, growth delay, nail bed changes, tachypnea, tachycardia, splenomegaly, hematuria, congestive heart failure, lethargy, poor scholastic performance.

Investigations

- Complete blood count showing reduced Hb, HCT, MCH, MCV, MCHC

- Smear: hypochromic microcytic RBCs for iron deficiency and malarial parasite.
- Stool examination for ova and cyst (Hookworm ova)
- Special investigations as required in selected cases

Note:

An increase in hemoglobin levels of greater than 1.0 g per dL (10.0 g per L) by four weeks after iron therapy is diagnostic of iron-deficiency anemia and warrants continuation of therapy for two to three additional months to properly replenish iron stores. During this time, further dietary intervention and patient education can be provided. Further diagnostic work up will be guided by the type of anemia as suggested by the CBC/ smear study.

Treatment

Oral iron therapy at the dose of 6 mg/kg of elemental iron /day for 3 months. Blood transfusion is needed if Hb% is < 4 g/dl or if the Hb% is 4 – 6 g/dl and the child has respiratory distress

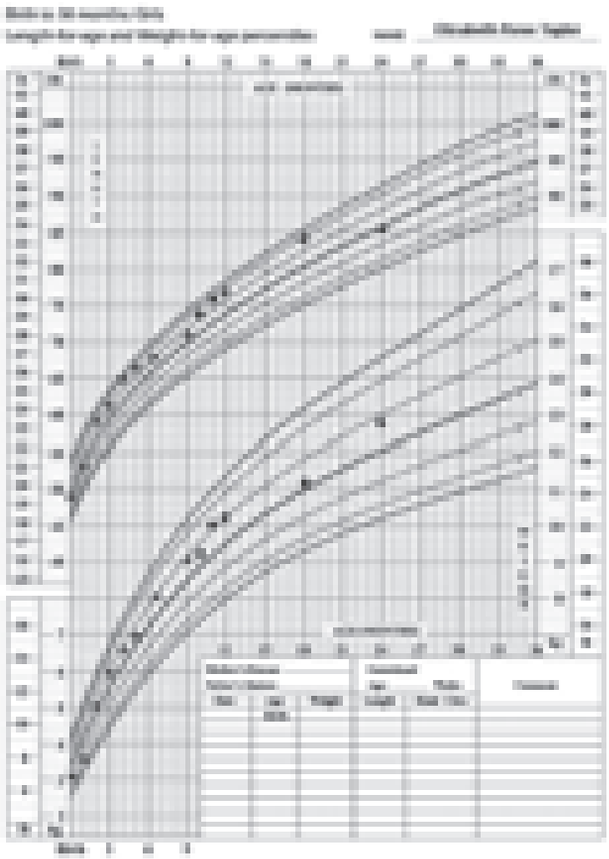
- Slow/ small volume transfusion in malnourished children
- Whole blood 10 ml/ kg slowly over 3 hours with
- Frusemide 1 mg/ kg IV at the start of the transfusion
- If the child has signs of heart failure give 1 ml/ kg of packed red blood cells
- Monitor the pulse and breathing every 15 minutes during transfusion.
- If breathing increases by 5 breaths / minute or Pulse by 25 beats/minute transfuse slowly.
- After transfusion if the Hb% is still low do not repeat the transfusion within 4 days.

IMNCI

Integrated management of neonatal and childhood illness programme by WHO and Government of India Guidelines for oral iron therapy:

Give Iron and Folic Acid (IFA) therapy

Growth chart



Age/wt	IFA tab	IFA syp	IFA drops
2 – 4 months (4-6kg)	Nil	1ml	0.5 –1ml
4-12months (6-10kg)	1 tab	1.25ml	1-1.5ml
12m to 3 years (10-14kg)	11/2 tab	2ml	1.5-2ml
3-5 years (14-19Kg)	2 tabs	2.5ml	2-3ml

- "IFA tab" ferrous sulphate 100 mg and folic acid 100mcg/tab(20 mg elemental iron/tab)
- "IFA syp" ferrous fumarate 100 smg and folic acid 50mcg/5ml(20 mg elemental iron/5ml)
- IFA drops ferrous ammonium citrate 20 mg elemental iron and folic acid 20mcg/1ml
- Deworm the child and / or treat if co-existing malaria exists.
- Reassess after 4 weeks.
- If the child's status has not improved after 4 weeks of therapy rule out causes other than iron deficiency of anemia or look for occult bleed.

Prophylaxis

- All children more than 6 months of age need one pediatric IFA or 5 ml syp or 1ml drops for 100 days.
- All adolescent girls need at least 100 tabs of IFA.
- Dietary counselling

Indications for referral

- Severe pallor
- Presence of heart failure

Management of scorpion sting

All children with a scorpion sting need to be admitted and observed for 24 hours.

Clinical features

1. Only local effect: pain, paresthesia at site of sting
2. Features of "Autonomic storm"

   Vomiting, hypersalivation, profuse sweating, brady / tachycardia, restlessness, cold peripheries, hypertension, priapism
3. Features of cardiac involvement
   - * CCF (Cardiomegaly, hepatomegaly, tachycardia, S3 gallop)
   - * Cardiogenic shock (Above features + shock+ hypotension)
   - * Pulmonary oedema (Respiratory distress, wheeze, rales, pink frothy sputum)
   - * ECG changes (Arrhythmias, ST-T changes, myocardial infarction pattern)

Rx Treatment

Depending on type of presentation:

1. Only local effect
   - Oral paracetamol 15 mg / kg + Inj Xylocaine 1% infiltration around sting site for older children, admit and
   - Observe for systemic features for 24 hours.
2. Autonomic storm
   - * Maintain hydration
   - * If shock occurs: oxygen, IV boluses of RL or NS 20 ml/kg . (Profuse vomiting and sweating can cause hypovolemic shock)
   - * Tab Prazosin
     - » Dose: 30mcg /kg per dose (1mg tablet 1/4 for every 10kg)
     - » To prevent first dose hypotension, keep child supine for 1 hour after prazosin
     - » Adequate hydration, repeat prazosin after 3-4 hours if clinical features persist, Continue prazosin for 2 more doses 6 hourly after clinical recovery.

Features of recovery

Warm peripheries, child quiet, sleeping comfortably, normal heart rate.

- Cardiac involvement
- Needs close monitoring and management

When manifesting initially or later with cardiogenic shock / pulmonary oedema

- * Oxygen by NRM 10L/min
- * Ventilatory support by early intubation and ventilation in pulmonary edema
- * Dobutamine infusion – start at 10 mcg/kg/min; increase dose by 2mcg/kg/min upto 20 mcg/kg/min till shock improves.
- * Tab Prazosin through NGT
- * Avoid digoxin, diuretics, morphine, lytic cocktail and steroids.

MANAGEMENT OF SCORPION STING

Scorpion sting

↓

Scorpion sting with persistent tachycardia, tachypnea, ALOC and shock needs PICU care.

Management of snake bite

Not all snake bites cause envenomation. It is important to remember that bite marks are of no use in determining whether the patient was bitten by a venomous or non-venomous species. Those who do not show manifestation should be kept for 24 hours under observation, given a tetanus toxoid injection if indicated and discharged.

Confirm snake bite

- Snake seen / killed /fang marks

Clinical Features

Local (cobra and viper)

- Pain
- Swelling
- Ecchymosis
- Bleeding from bite site
- Progressive oedema

Hemorrhagic (usually viper)

- Prolonged clotting time (CT), prothrombin time (PT), hematuria, DIC
- 20 minute whole blood clotting test (20 WBCT) is the standard test for coagulopathy
  - * A few ml of fresh venous blood is left undisturbed in a clean, dry, test tube for 20 minutes and then gently tilted
  - * If the blood is still liquid this is evidence of coagulopathy and confirms that the bite species is viperine.

Neurotoxic(Cobra and krait):

- * Paresthesia over tongue and mouth, ptosis, ophthalmoplegia, difficulty in speaking and swallowing, respiratory paralysis, shock

Nephrotoxic (Russell's viper, hump-nosed viper)

- * Renal failure is a frequent complication. Hence renal parameters need to be monitored.

Rx Treatment

1. Supportive measures
   - * ABC
   - * Ventilatory support for respiratory failure
   - * IV access/correct shock, if present
   - * Broad spectrum antibiotics/tetanus prophylaxis
2. Neurological involvement (ptosis, respiratory failure)
   - * IV Atropine 0.02mg/kg followed by IV Neostigmine 0.05mg/kg
   - * Repeat after 1-2 hours later if neurological features persist
3. Anti-snake venom(ASV)

Indications for ASV

- * Systemic envenomation as evident by 20 minutes WBCT
- * Spontaneous bleeding
- * Neurological impairment
- * Severe local swelling

Severe local symptoms are defined by swelling rapidly crossing a joint or involving half of the bitten limb, in the absence of tourniquet. Increased swelling one hour following removal of tourniquet is an evidence of venom generation. Purely local swelling is not adequate ground for ASV administration.

1. ASV should be administered over one hour. There is no benefit in administering each dose over a long period. ASV test doses are no more recommended. They have no predictive value in anaphylactoid or late serum reactions and may presensitize the patient to the protein. Eight to ten vials is the initial starting dose for both adults and children.
2. Adverse reactions to ASV

- At the first sign of any of the following:
  - * Urticaria, itching, fever, shaking chills, nausea, vomiting, diarrhea, abdominal cramps, tachycardia, hypotension, bronchospasm, angioedema.

Rx Treatment

- * ASV should be discontinued
- * 0.01mg/kg of 1:1000 Adrenalin should be given IM.
- * 2 mg/kg of Hydrocortisone IV
- * 0.2 mg/kg of Chlorpheniramine maleate IV
- * If no improvement in 10- 15 minutes 2nd dose of Adrenaline. Repeat till a maximum of 3 doses totally.
- * If patient has recovered, ASV is restarted slowly over 10-15 minutes
- * Observation.

1. In anti – hemostatic bites after the initial dose no further doses will be required for 6 hours. 20WBCT is done every 6 hours, and this will determine the need of additional ASV doses
   - * In neurotoxic bites after the first dose ASV and neostigmine is given, if no improvement in 1-2 hours, 2nd and final dose of ASV is given.

* A second dose of 10 vials may be needed in some cases
* Carefully assess for neck muscle weakness/ respiratory failure
* Further ASV will achieve nothing in this setting. Patient will either recover or require mechanical ventilation.

2. Monitoring
   * Clinical (Pulse, respiration, BP, urine output, hematuria, neurological status) Clotting time 6 hourly till stabilized and daily for 2 or 3 days more
   * Monitor the local effects and also the tender lymphadenitis proximally in the bitten limb.
3. Tourniquets/ Pressure immobilization methods are not recommended in snake bites based on current literature. Incision, suction, electric shock, cryotherapy and washing the wound are contraindicated.

Confirm : Snake seen or killed / Fang marks. Reassure the patient that only 50% of bites by venomous species envenomate the patient. Immobilise the limb (like a splint)

↓

Look for; local swelling, bleeding, progressive oedema, blisters, bulbar palsy, respiratory paralysis, abnormal coagulation, hematuria

↓

Stabilise ABC's: Ventilatory support needed in neurotoxic bites
To decide on ASV as follows

No local/ systemic effects /mild swelling only	Spontaneous bleed/ ↑20WBCT	Neurotoxic signs	Severe local reactions
↓	↓	↓	↓
Observe for 24 hours	8- 10 vials of ASV over one hour	Maintain ABC Monitor ECG, SPO_2 8 - 10 vials of ASV over one hour	Swelling rapidly crossing joint, swelling > than half of the bitten limb
↓	Monitor:20WBCT 6 hours later	↓	↓
Monitor – vitals, CT, hematuria	↓	Atropine 0.02mg/kg IV followed by Inj. Neostigmine 0.05mg/kg IV	8 - 10 vials of ASV
	If coagulation is abnormal	↓	↓
	↓	If improvement is seen continue Neostigmine .If no improvement in 1-2 hours 2nd and final dose of ASV	Monitor: vitals, CT, hematuria, neurologic effects
	Further dose of ASV	↓	↓
		Recovery or require mechanical ventilation. No more ASV	Broad spectrum antibiotics Surgical intervention if necessary.

Management of Poisoning

Principles

1. Rapid assessment, stabilization and maintenance of ABC
2. Focused history and physical examination
3. Supportive measures such as control of seizures and oxygen administration
4. GIT decontamination (reduction of absorption)
5. Antidotes
6. Frequent monitoring
7. Unknown substance ingestion and when in doubt admit and observe

Resuscitation and stabilization for children presenting with

Altered level of consciousness, seizures, shock, brady /tachy cardia, inadequate respiratory effort (bradypnea), respiratory distress and cyanosis.

GIT decontamination

1. Gastric lavage is indicated in all types of poisons except for a few
   * Maximum benefit if done within 1 hour
   * Warm saline 50-100 ml aliquots
   * Patient in left lateral position
   * Use largest NG tube/ orogastric tube which can be comfortably inserted
   * Protect airway

Contra indications

1. Corrosives, kerosene poisoning keep the first sample of return lavage for toxicology
2. Activated charcoal

   Can be used if available

   Effective for all poisons except iron, lithium, organophosphates

   Dose 1gm/kg mixed with water or fruit juice and given orally or through NG tube

Contra indications

- Corrosives, kerosene, GI bleed, ileus

3. Whole bowel irrigation

   PEGLEG

   Through NG tube

   15ml/kg/hr till rectal effluent is clear

   (Maximum of 500 ml/hr in preschool children and 1000 ml/hr in older children)

   Monitor electrolyte imbalance and hydration

Remember

- Early intubation and ventilation for brief periods useful in some poisonings
- Cardiac rhythm disturbances to be looked for
- Monitoring of vitals most important
- Majority of poisoning cases recover with supportive care
- Specific antidotes available only for a few poisons

Kerosene poisoning

1. Admit the child even if asymptomatic for 24 hours
2. No gastric lavage
3. Chest X-ray if persistent cough, lung signs or respiratory distress present
4. Oral feeds if no respiratory distress
5. Respiratory distress:
   * Oxygen by NRM 10l/min or by simple mask 5l/min
   * Monitoring of vitals, SaO2 (pulse oximetry)
   * IV fluids- maintenance
   * Manage as for “respiratory distress protocol”

Insecticide (Organophosphate) poisoning

1. Admit in intensive care unit
2. Gastric lavage
3. Stabilise ABC
4. Look for respiratory failure early ventillatory support
5. Control seizures if present
6. IV atropine 0.05mg/kg/dose every 5min till secretions dry up.
7. Then continue atropine at less frequent intervals for 24 hours before stopping
8. IV pralidoxime (P2AM) 25-50mg/kg in NS over 10 min in case of respiratory muscle paralysis: repeat after 1hour if necessary
9. Continuous monitoring

Neem oil poisoning

Presentation like status epilepticus with encephalopathy

Treatment

* Stabilize and maintain ABC
* Management of seizures as per “status epilepticus protocol”

Algorithm for Management of Poisoning in Children

Only history of ingestion No symptoms

Stable vitals, normal sensorium

↓

No contra indication for gastric lavage

↓

Gastric lavage with 50-100 ml NS till returns clear: Take care to prevent aspiration

↓

Send first return sample for toxicology

↓

Admit, monitor vitals for 24 hrs

↓

Supportive/ specific therapy

Kerosene ingestion

No respiratory distress / no lung signs
Admit & observe for 24 hrs

↓

Persistent cough, lung signs, respirtory. distress: Check with chest X-ray

↓

Supportive measures:
O_2
IV fluids
Manage respiratory distress / respiratory failure
No gastric lavage

Suspected poisoning in a sick child (Seizures, altered sensorium, shock, inadequate respiration)

Stabilize ABC: Take blood for investigations.

↓

25% dextrose 2 ml/kg
For seizures: Anti convulsants as for status epilepticus protocol

↓

Maintain adequate ventilation
Continue to treat shock

↓

Gastric lavage with air way protection

↓

IMCU admission: Monitoring

↓

Specific antidotes if needed:

a. IV Atropine + Pralidoxime for organo phosphate poisoning
b. IV Desferoxamine for iron
c. IV Methylene blue for methemoglobinemia (Dapsone)
d. IV Pyridoxine for INH

In emergency situations contact the poison control center at Chennai for guidance.
Telephone number: 044 2530 5959

Abbreviations used

ABC	-	Airway, Breathing, Circulation
NICU	-	Neonatal intensive care unit

PROM	-	Prolonged rupture of membranes
IDM	-	Infant of diabetes mother
RDS	-	Respiratory distress syndrome
NEC	-	Neonatal enterocolitis
HR	-	Heart rate
PPV	-	Positive pressure ventilation
CC	-	Chest compression
LBW	-	Low birth weight
SGA	-	Small for gestational age
KMC	-	Kangaroo mother care
IVH	-	Intra ventricular hemorrhage
ROP	-	Retinopathy of prematurity
CNS	-	Central nervous system
EEG	-	Electro encephalogram
TSB	-	Total serum bilirubin
AAP	-	American Academy of Pediatrics
EDD	-	Expected date of delivery
Sa O_2	-	Oxygen saturation
NGT	-	Naso-gastric tube
CRT	-	Capillary refill time
DIC	-	Disseminated Intra-vascular coagulation
TORCH	-	Toxoplasmosis, other, rubella, cytomegalovirus, herpes
ITP	-	Immune thrombocytopenic purpura
SLE	-	Systemic lupus erythematosus
FFP	-	Fresh frozen plasma
Hb	-	Hemoglobin
PHC	-	Primary health centre
FRU	-	First referral unit
VLBW	-	Very low birth weight
RR	-	Respiratory rate
NRM	-	Non re-breathing mask
BMV	-	Bag mask ventilation
ALOC	-	Altered level of consciousness
PT	-	Prothrombin time
PTT	-	Partial thromboplastin time
CXR	-	Chest X-ray
ICD	-	Inter costal drainage
SE	-	Status epilepticus
ICP	-	Intracranial pressure
VSD	-	Ventricular septal defect
ASD	-	Artial septal defect
PDA	-	Patent ductus arteriosus
TOF	-	Tetrology of Fallot
TGA	-	Transposition of great arteries
PS	-	Pulmonary stenosis

CONGENITAL DIAPHRAGMATIC HERNIA

Scaphoid abdomen.

Heart sounds better heard on side opposite to the hernia.

Tympanic note on percussion of chest.

Bowel sounds heard in chest

↓

X- Ray shows

Mediastinal shift.

NO diaphragm visualized

Bowel loops in chest.

↓

CONFIRMS CDH

↓

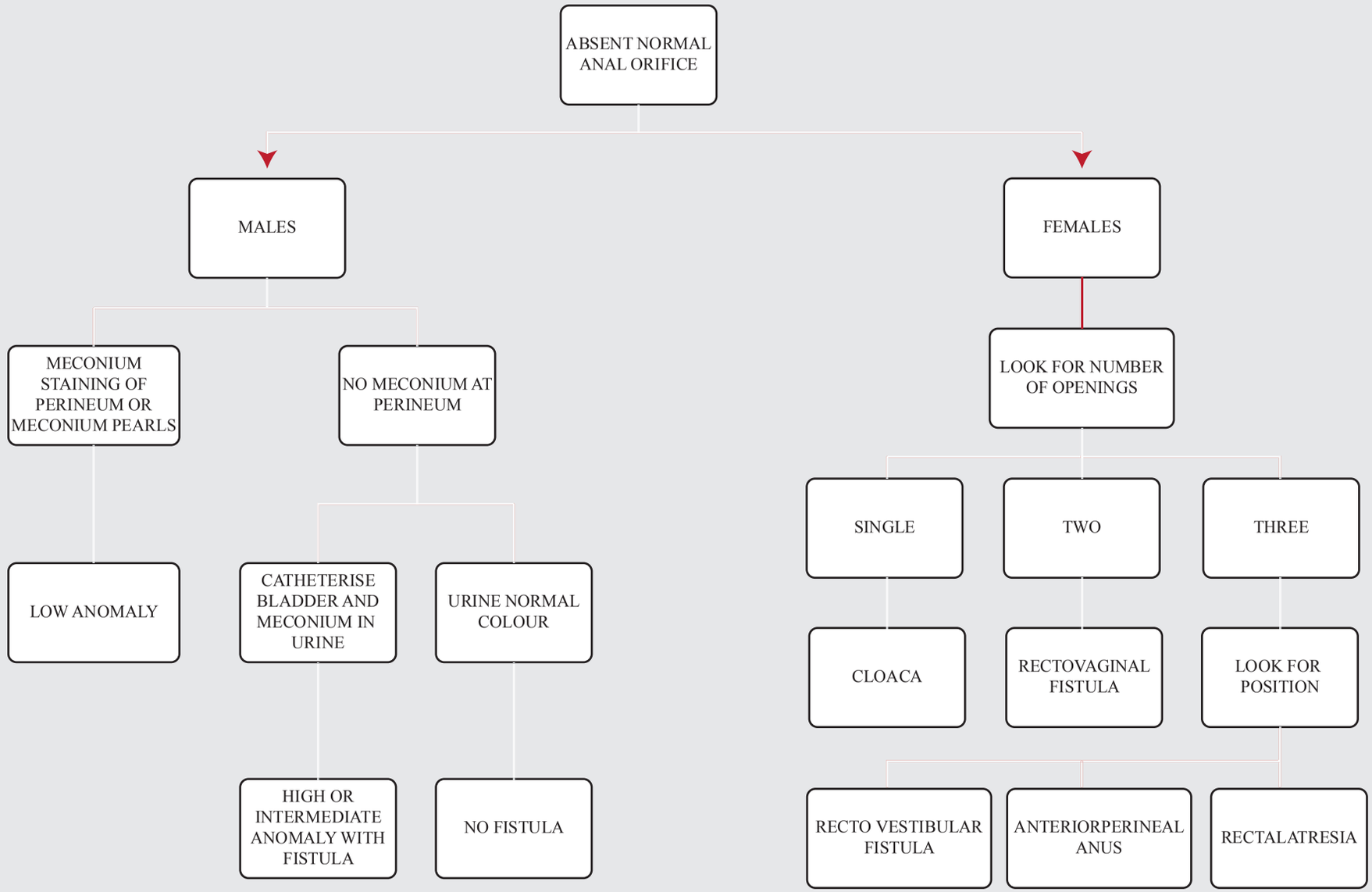
RESSUSCITATE

NO BAG AND MASK VENTILATION

↓

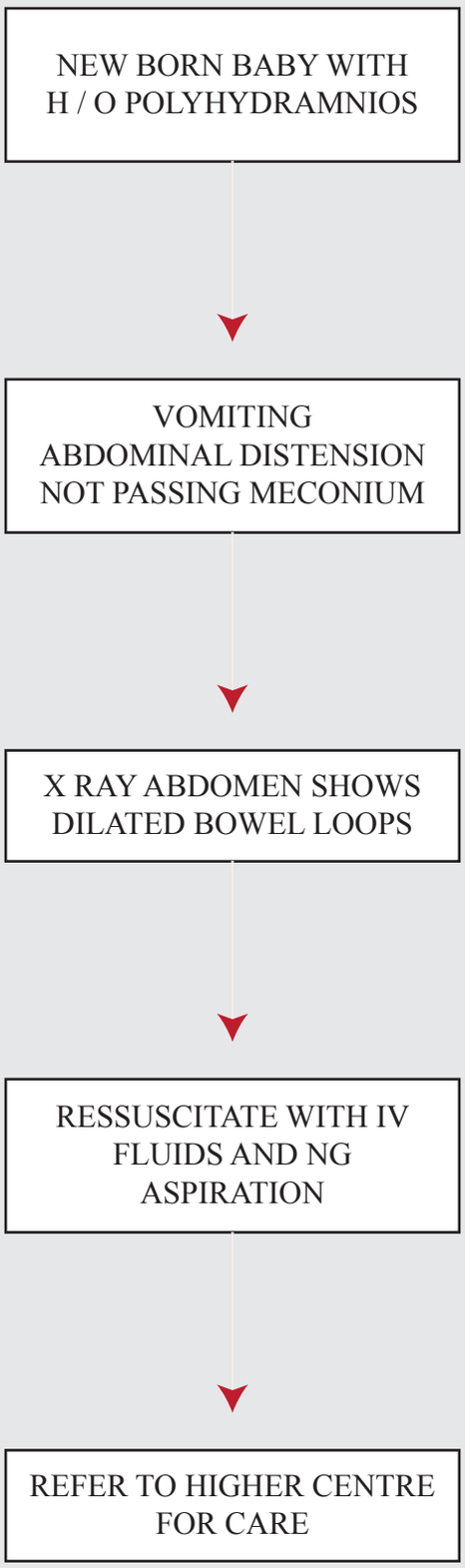
REFER TO HIGHER CENTRE

ANORECTAL MALFORMATION OR IMPERFORATE ANUS

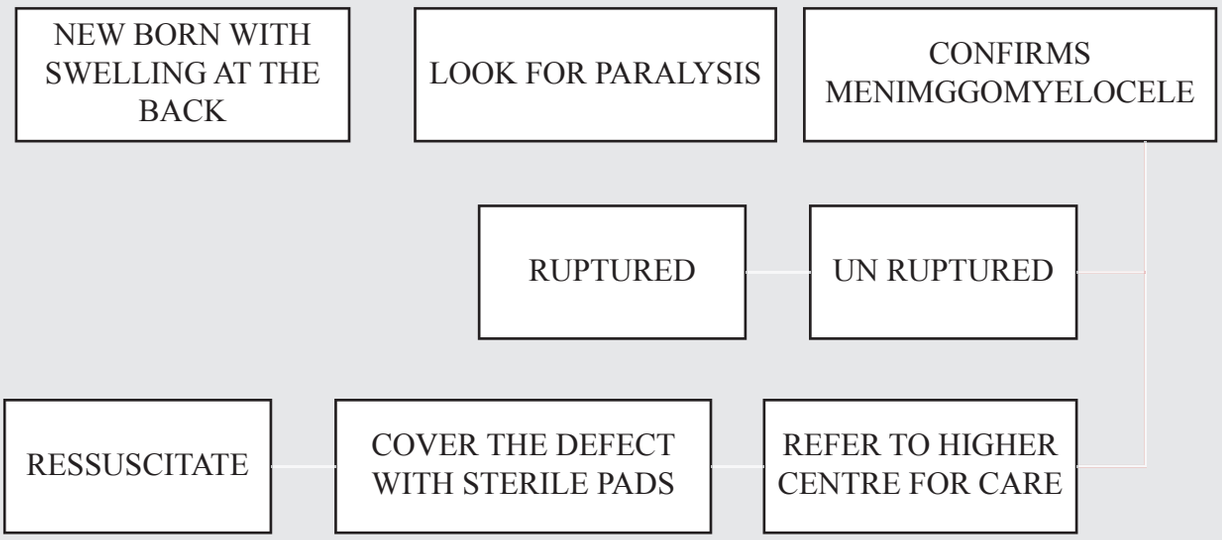


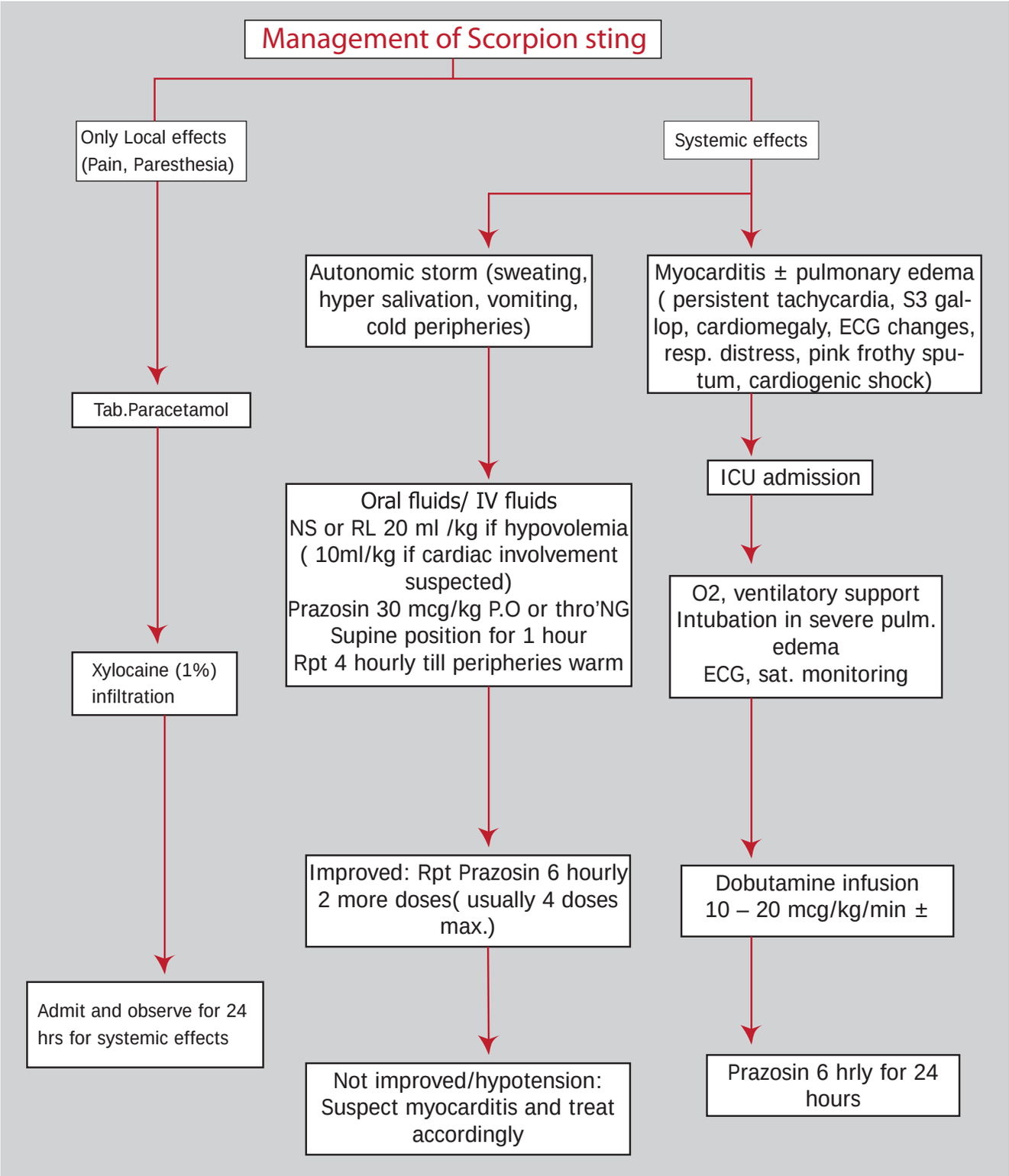
all cases need confirmation with xrays and refer after ressuscitation.

INTESTINAL OBSTRUCTION



SPINA BIFIDA




Management of Scorpion sting
Only Local effects (Pain, Paresthesia)
Systemic effects
Autonomic storm (sweating, hyper salivation, vomiting, cold peripheries)
Myocarditis ± pulmonary edema (persistent tachycardia, S3 gallop, cardiomegaly, ECG changes, resp. distress, pink frothy sputum, cardiogenic shock)
Tab.Paracetamol
ICU admission
Oral fluids/ IV fluids
NS or RL 20 ml /kg if hypovolemia
(10ml/kg if cardiac involvement suspected)
Prazosin 30 mcg/kg P.O or thro'NG
Supine position for 1 hour
Rpt 4 hourly till peripheries warm
O2, ventilatory support
Intubation in severe pulm. edema
ECG, sat. monitoring
Xylocaine (1%) infiltration
Improved: Rpt Prazosin 6 hourly 2 more doses(usually 4 doses max.)
Dobutamine infusion
10 – 20 mcg/kg/min ±
Admit and observe for 24 hrs for systemic effects
Not improved/hypotension: Suspect myocarditis and treat accordingly
Prazosin 6 hrly for 24 hours

Obstetrics and Gynaecoloy

Standard Treatment Guidelines
Tamil Nadu Health Systems Project

Chapter 14

1. Normal Pregnancy
2. Normal Labour
3. Obstructed Labour
4. Contraception
5. Bleeding in early pregnancy
6. Ectopic pregnanacy
7. MTP
8. Anaemia in pregnancy
9. Hypertensive disorders of pregnancy
10. Heart disease complicating pregnancy
11. Gestational Diabetes mellitus
12. Post dated pregnancy
13. Post Caesarean pregnancy
14. HIV and pregnancy
15. Chromosomal disorders
16. Ante partum haemorrhage
17. Post partum haemorrhage
18. Post meno -pausal bleeding
19. DUB
20. Screening guidelines for cancer

Normal Pregnancy

Antenatal Care

Effective antenatal care (ANC) can improve the health of the mother and give her a chance to deliver a healthy baby. Regular monitoring during pregnancy can help detect complications at an early stage before they become life-threatening emergencies. However, one must realize that even with the most effective screening tools currently available, one cannot predict which woman will develop pregnancy-related complications. Hence, every pregnant woman needs special care. As the Medical officer (MO) in charge, you must remember the following:

- Recognize that "Every Pregnancy is at risk"
- Ensure that ANC is used as an opportunity to detect and treat existing problems.
- Make sure that services are available to manage obstetric emergencies.
- Prepare pregnant women and their families for the eventuality of an emergency.

  The important components of ANC are discussed below.

Early registration

Timing of the first visit/registration

The first visit or registration of a pregnant women for ANC should take place as soon as the pregnancy is suspected. Every married women in the reproductive age group should be encouraged to visit her health provider or inform you if she believes herself to be pregnant.

Ideally, the first visit should take place in the first trimester, before or at the 12th week of pregnancy. However, even if the woman comes late in her pregnancy for registration, she should be registered, and care given to her according to the gestational age.

During regular monthly meeting, emphasize to the auxiliary nurse-midwives (ANMs) and other health workers to inform the pregnant woman and her family member about the timings of the ANC clinic organized at the primary health centres (PHCs) /community health centres (CHCs) and government hospitals. Explain the importance of seeking timely ANC, during their outreach visits

Importance of early registration

Early registration is required to:

- Assess the health status of the mother and obtain baseline information on blood pressure, weight haemoglobin, etc.
- Screen for complications early and manage them appropriately by referral as and when required
- Help the woman recall the date of her last menstrual period (LMP)
- Give the woman the first dose of tetanus toxoid injection (Inj.TT) well in time (after 12 weeks of pregnancy)
- Help the woman access facilities for an early and safe abortion if she does not want to continue with the pregnancy. Be alert to the possibility that the abortion might be an attempt at female foeticide.(Refer to the Government of India (GoI) Guidelines for the Medical termination of pregnancy (MTP))
- Build a good rapport with the pregnant woman. Allow plenty of the to counsel the woman and her family.
- Start the woman on a regular does of iron folic acid during the first trimester

Record – keeping

Complete the antenatal card for every woman registered/examined by you . Hand over the card to the woman. Instruct her to bring the card with her for all subsequent check-ups/visits , and carry it along with her at the time of delivery.

Record this information in the PHC/CHC/government hospitals antenatal register

Antenatal check-up

Numbering and timing of visits

Ensure that every pregnant woman makes at least 4 visits for ANC including the first visit/registration and any home visits by the ANC/lady health visitor (LHV). These are sufficient and, for pregnancies without complications, studies have shown that additional visits do not improve the maternal or perinatal outcome.

The first visit is recommended as soon as the pregnancy is suspected. This is meant for registration of the pregnancy and the first antenatal check-up. The second visit should be scheduled between the 4th and 6th month (around 26 weeks). The third one should be planned in the 8th month (around 32 weeks), and the fourth in the 9th month (36-40 weeks).

Preparing for the ANC clinic

Before beginning each ANC clinic, ensure that all the required instruments/equipment ,e.g stethoscope, foetoscope, gloves, sphygmo-manometer, weighing scale, inch tape, are available and in working condition.

Greet every pregnant woman in a friendly manner at each visit.

Listen to the woman's problems and concerns and offer advice or refer to a CHC or a first referral unit (FRU), GH as appropriate. Remember, every woman needs social support during pregnancy.

Confirm that pregnancy is wanted. If not, and the woman wishes to go in for an abortion, offer her manual vacuum aspiration (MVA)/(MTP services if they are available at your PHC/CHC, or refer her to the nearest CHC/FRU/GH where safe abortion services are available services are available. (You are advised to follow the GoI guidelines for the Medical Termination of Pregnancy.) This is important, especially during the first visit when MTP is still feasible.

Conduct the antenatal examination in a room/ enclosure that allows privacy for conducting an abdominal palpation.

Record all finding on the antenatal card, and in the antenatal register.

History – taking

During the antenatal visits, take a detailed history of the woman (i) to diagnose the pregnancy (first visit only, if required); (ii) to identify any complications during previous pregnancies which may have a bearing on the present one: and (iii) to identify any medical or obstetric condition(s) that may complicate the present pregnancy (first and subsequent visits)

While taking the history, ask the following questions:

Date of the last menstrual period

Remember that the LMP refer to the **first** day of the woman's last menstrual. Ensure that the woman, which telling you her, LMP is NOT referring to the date of the first **missed period**. This mistake will lead to a miscalculation of the gestational age and expected date of delivery (EDD) by4 weeks.

If the woman is unable to remember the exact date, encourage her to remember some major event/festival, etc. Which she might link with her LMP. A calendar with the Indian system of months, dates and local festivals might come in handy. If the exact date of the LMP is not known, and it is still early pregnancy, the gestational age can be calculated from the size of the uterus on vaginal examination done during the first Trimester. However , if the woman comes to you late in the pregnancy,assess the fundal height to estimate the gestational age. You may also ask for the date when the foetal movement were first felt. This id known as **"quickening"** and is felt at around 20 weeks of gestation.

If the woman has undergone a test to confirm the pregnancy, ask her the approximate date when it was done, and also after how many days of amenorrhoea. This will assist you in estimating her LMP.

The LMP is used to calculate the gestational age at the time of check-up and the EDD. In cases with uncertain LMP calculate the EDD based on the above estimates. Make a special note on the records of these cases that the LMP, and therefore the EDD, are approximated.

The following formula for calculation of the EDD is based on the assumption that the menstrual cycle of the woman was regular before conception and it was a 28-30 days' cycle. If the period of the menstrual cycle is more than 30 days, add the additional number of the cycle (beyond 28 days) to the EDD calculated below.

EDD=LMP+9 months+7 days (+ additional days, if any)

Age of the woman

This is required as woman below 16 years of age or above 40 years have greater chances of having pregnancy related complications.

Order of the pregnancy

Primigravidas and those who have had 4 or more pregnancies are at a higher risk of developing complications during pregnancy and labour.

Birth interval,

Research shows that woman who have spaced their children less than 36 months a part have greater chances of delivering a premature and low birth-weight (LBW)baby, with consequently increased risk of infant mortality.

An interval of less than 2 years from the previous pregnancy or 3 months from the previous abortion increases the chances of the mother developing anaemia.

Symptoms during the present pregnancy

You must ask for symptoms that might be causing the woman some discomfort, and also for symptoms which indicate that a complication may arise. Ask the woman for the following symptoms in the present pregnancy.

Symptoms that indicate discomfort

- Nausea and vomiting
- Heartburn
- Constipation
- Increased frequency of micturition

Symptoms which indicate that a complication may arise

- Vaginal discharge
- Palpitations easy fatiguability and breathlessness at rest
- Generalized swelling of the body: puffiness of the face
- Vaginal bleeding
- Decreased or absent foetal movements
- Leaking of watery fluid per vaginum (P/V)
- Decreased urinary output

  (see also Table ,latter in this chapter for the presumptive diagnosis and management of these symptoms and signs)

Previous pregnancies

It is essential to ask a woman about her previous obstetric history, especially if she had suffered from any complications. This is important as some complications may recur during the present pregnancy.

Ask the woman about:

- The total number of previous pregnancies (including the present one, "gravida")and deliveries ("parity")
- Abortion(s)
- Premature birth(s)
- Stillbirth(s) or neonatal loss
- Hypertensive disorders of pregnancy (if not known,ask for a history of convulsions in previous pregnancies)
- Obstructed labour
- Malpresentation, such as breech delivery
- Antepartum haemorrhage(APH)
- Postpartum haemorrhage(PPH)
- Assisted delivery (forceps or vacuum extraction)
- Delivery by caesarean section
- Birth weight of the previous baby
- Any surgery on the reproductive tract (e.g. myomectomy, removal of the septum, cone biopsy, cervical cerclage, uterine perforation following an MTP, etc.)
- Isommunization (Rh –ve) in the previous pregnancy (history of any costly injection given within 72 hours of the previous delivery)

Ask especially for notes on the previous pregnancy, if available.

- Previous stillbirth or neonatal loss
- History of three or more consecutive spontaneous abortions
- Birth weight of the previous baby >4500 g
- Hospital admission for hypertension or pre-eclampsia in the previous pregnancy
- H/o surgery on the reproductive tract
- Isoimmunization (Rh-ve) in the previous pregnancy

History of any systemic illness

- Rule out any personal history of systemic illnesses such as
  - Hypertension
  - Diabetes
  - Heart disease
  - Tuberculosis
  - Renal disease
  - Epilepsy
  - Asthma
  - Skin rashes
  - Jaundice

Family history of systemic illness

If the woman does not have any of the above-mentioned systemic illnesses, ask for a family history of hypertension, diabetes and tuberculosis. If present, such a history predisposes the woman to developing the same herself during pregnancy (e.g hypertensive disorders of pregnancy, gestational diabetes, etc). As pregnancy is a physiologically stressful period in a woman's life, it can unmask the underlying tendency to develop these disorders.

Also ask for a family history of thalassaemia, or whether anybody in her family has received blood

transfusions. You must also ask for a family history of delivery of twins and/or the delivery of an infant with congenital malformation, as the presence of such a history in the increases the chances of the woman giving birth to a child with the same defect

History of drug intake or allergies

It is important to find out whether the woman is allergic to any drug, or if she is taking any drug that might be harmful to the foetus. Find out whether she had undergone any treatment or taken drugs for infertility. If yes, then the woman has a higher chance of twins and other multiple pregnancies.

History of intake of habit- forming or harmful substances

Ask the woman if she takes tobacco (chewing or smoking) and /or alcohol. If yes, she needs to be counselled to discontinue them during pregnancy, as they harm the developing foetus. Even after the delivery, the woman should be advised to continue to abstain from taking alcohol and tobacco as their use may lead to other complications such as addiction and/ or cancer.

Physical examination

This activity will be nearly the same during all the visits. Initial readings may be taken as a baseline and compared with the later readings.

General examination

Weight : A pregnant woman's weight should be taken at each visit. The weight taken during the first visit/registrations should be treated as the baseline weight.

Normally, a woman should gain 9-11 kg during her pregnancy. After the first trimester, a pregnant woman gains around 2 kg every month or 0.5 kg per week. To calculate the expected weight gain since her previous visit, multiply the number of weeks elapsed since the previous visit by 0.5 kg. This should be compared with the actual weight gained.

If the diet is inadequate, with less than the required amount of calories, the woman might gain only 5-6 kg during her pregnancy. Suspect an inadequate dietary intake if the woman gains less than 2 kg per month. Put her on food supplementation. Take the help of the ANM or refer the woman to the anganwadi worker (AWW) of her village for food supplementations, especially for those categories of woman who needs it the most [see later in this chapter under Counselling for "Diet and rest "]A low weight gain usually points towards intra-uterine growth retardation (IUGR) and results in an LBW baby.

Excessive weight gain (more than 3 kg in a month) should arouse the suspicion of pre-eclampia/twins (multiple pregnancy). Check the woman's blood pressure, and test her urine to check if she has proteinuria

- Keep the following points in mind while taking the weight:
  - * The weighing machine should be checked for "zero error" before taking the weight
  - * The woman should be wearing light clothing
  - * She should stand erect on the weighing machine, in such a way that her weight is evenly distributed on the platform.
  - * The weight must be measured to the nearest 100 g

Blood pressure: Measure the blood pressure of pregnant woman **at every visit** to rule out hypertensive disordered in pregnancy.

If the blood pressure is high (more than 140/90 mmHg: or diastolic more than 90mmHg), check the blood pressure again after 1 hour. If it is still high, test the woman's urine for the presence of albumin, as the combination of a high blood pressure and proteinuria is sufficient to categorize the woman as having pre-eclampsia. These woman needs to be managed as per the guidelines.

If the diastolic blood pressure of the woman is above 110 mmHg, it is a danger sign pointing towards severe pre-eclampsia. Refer the woman to the CHC/FRU/GH **immediately** after giving her a dose of Nifedipine.

A woman with pregnancy-induced hypertension (PIH)/pre-eclampsia require hospitalization for daily/ alternate day monitoring of blood pressure, the level of protein in the urine and foetal condition.

Pallor: Pull down the lower eyelid and look at the lower palpebral conjunctiva, and also the nails, palms, tongue and oral mucosa of the woman for the presence of pallor. If is an indication that the woman is anaemic, investigate her hemoglobin (Hb) level [see later in this chapter under investigation for "Haemoglobin estimation"]

Respiratory rate (RR): It is important to check the RR especially if the woman complains of breathlessness. If the RR is more than 30 breaths/minute and pallor is present [see above in this chapter under examination for "pallor"], it indicates that she has severe anaemia.

If the RR of the woman is >30 breaths/minute , and

she has other associated medical problems, refer her to the specialist at the FRU/GH for further investigation and management of any systemic illness, if present.

Generalized oedema: The presence of generalized oedema or puffiness of the face should arouse the suspicion of pre-eclampsia.

Breast examination

Observe the size and shape of the nipples for the presence of inverted or flat nipples. Try and pull out the nipples to see if they are protractile (i.e. can be pulled out easily). Flat nipples that are protractile do not interfere with breast feeding.

If the nipples are inverted, advise the woman to roll her nipples between the thumb and the index finger, pulling at the nipples simultaneously.

Another technique for correcting inverted nipples includes the use of a 10 cc or 20 disposable plastic syringe. Cut the barrel of the syringe from the end where the needs is attached. Take out the plunger and put it in from the opposite end, which is the cut end of the syringe. Push the piston forward fully , and place the open end of the barrel so that it encircles the nipple and areola. Ask the woman to pull back the plunger and create negative pressure. The nipple will be sucked into the barrel and thus be pulled out in the process.

- Crusting and soreness of the nipples must be looked for. If present, the woman must be advised regarding breast hygiene. If the nipples do not heal, look for infection or any other cause and treat accordingly.
- The breasts must be palpated for any lumps or tenderness. If present refer the woman to the surgical specialist at the FRU/GH.

Vaginal examination

- Vaginal examination is required, especially during the first visit, to confirm the pregnancy.
- This is also used to measure the gestational age, by estimating the size of the uterus during the first trimester of pregnancy, before the uterus becomes an abdominal organ.
- A per speculum (P/S) examination may be done during the first and last antenatal visit, especially if the woman complains of discharge P/V. This would help in defining whether it is a vaginal or a cervical discharge, and the type of discharge, thus assisting you in marking a diagnosis of reproductive tract infection (RTI), and managing the case accordingly.

Abdominal examination

Examine the abdomen to monitor the progress of the pregnancy and foetal growth, and to check the foetal lie and presentation.

Fundal height: The fundal height indicates the progress of the pregnancy and foetal growth. The uterus becomes an abdominal organ after 12 weeks of gestation. The gestation age (in weeks) can be estimated from the fundal height (in cm)after 12 weeks of gestation.

If the height of the uterus is more than that indicated by the period of amenorrhoea, the possible reasons could be:

- wrong date of LMP
- Full bladder
- Multiple pregnancy
- Polyhydramnios
- Hydatidiform mole
- Pregnancy with a pelvic tumour

If the height of the uterus is less than that indicated by the period of amenorrhoea, the possible reasons could be:

- Wrong date of LMP
- Intrauterine growth retardation (IUGR)
- Missed abortion
- Intrauterine death (IUD)
- Transverse lie

Foetal lie

Palpate for the foetal lie and assess whether it is longitudinal, transverse or oblique. Remember , if a malpresentation is diagnosed before 36 weeks, no active management or intervention is recommended at that point of time.

You must be able to recognize lie. Missing it can be disastrous because there is no mechanism by which a woman with a transverse lie can deliver vaginally. This woman would need a caesarean section, and hence should be referred to an FRU/GH where emergency obstetric care services are available, including the facility for a caesarean section. Failure to do a timely caesarean section in such a woman can lead to obstructed labour, rupture of the uterus, and death of the woman

Foetal presentation

Check the foetal presentation, especially in the case of a longitudinal lie, to see whether the presenting part is the vertex (normal), or any other part the cephalic end (face, brow),or the breech [see annexure 2b: :"Determining the foetal lie and presentation"

Foetal heart rate (FHR)

If the FHR is between 120 and 160 beats per minute, it is normal. Both foetal bradycardia (FHR<120 beats/ minute) and foetal tachycardia (FHR more than 160 beats per minute) indicate foetal distress.

Remember that the FHR is not heard before the 24th week of pregnancy ;hence the FHS needs to be checked from the second visit only.

- Multiple pregnancy :
  - * This must be suspected if the following are present on examination:
  - * An unexpectedly large uterus for the estimated gestational age
  - * Feeling multiple foetal parts on abdominal palpation
  - * Polyhydramnios, as it is often associated with multiple pregnancy.

If a multiple pregnancy is suspected, refer the woman to an GH/FRU's Obstetrics and Gynaecology specialist for confirmation and, if confirmed, arrange for delivery in the GH/FRU.

Assessment of the pelvis

Examination of the pelvis is required to assess if it is adequate for delivering the baby vaginally. This should be done during the last ANC visit (at about 16 weeks of gestation) to rule out any cephalo-pelvic disproportion (CPD).

Remember, it is not advisable to give a pregnant woman any medication during the first three months of

Common symptoms and signs that may be encountered in a pregnant woman
Probable diagnosis and action required to be taken at the PHC level

Symptoms	Signs/investigation	Most probable diagnosis	Action(s) to be taken
Excessive vomiting especially after the first trimester, inability to retain anything taken orally	The woman may be dehydrated	Hyperemesis	Admit her for a few days at the PHC and manage as Hyperemesis gravidorum
Palpitations, easy fatiguability, breathlessness at rest	Conjunctival and/or palmar pallor present Hb level <7 g/dl	Severe anaemia	Start the woman on a double dose of IFA tablets. Give her Albendazole (second trimester onwards only). Monitor the Hb level after one month. Advise her for delivery at the FRU.
Puffiness of the face, generalized body oedema	Blood pressure >140/90 mmHg Proteinuria absent Blood pressure >140/90 mmHg Proteinuria present	Hypertensive disorder of pregnancy Pre-eclampsia	If the blood pressure is <160/110 mmHg, advise home management with rest and regular follow up. If the blood pressure is >160/110 mmHg, start on Nifedipine. Start the woman on antihypertensive medication. Refer to an FRU for further management. Advise her on the danger signs of imminent eclampsia and eclampsia and refer to an FRU.
Heartburn and nausea	Reflux	Hypertensive disorder of pregnancy	Advise the woman to avoid spicy and rich foods. Ask her to take cold milk during attacks. If severe, antacids may be prescribed.
Increased frequency of urination up to 10-12 weeks of pregnancy Increased frequency of urination after 12 weeks, or persistent on urination	Tenderness may be present at the sides of abdomen and back. The body temperature may be raised	May be physiological due to pressure of the urinary bladder Urinary tract infection	Reassure her that if will be relieved on its own Management of Urinary tract infection.

Constipation		Physiological	Advise the woman to take more fluids, leafy vegetable and a fibre-rich diet. If not relieved, prescribe Isabgol, 2 tablespoonfuls to be taken at bedtime, with water or with milk Do not prescribe strong laxatives as they may initiate uterine contractions.
Bleeding P/V, before 20 weeks of gestation. Bleeding P/V, after 20 weeks of gestation	Check the pulse and blood pressure to assess for shock Ask for history of violence Check the pulse and blood pressure to assess for shock	Threatened abortion/spontaneous abortion/hydatidiform mole/ectopic pregnancy Spontaneous abortion due to violence Antepartum haemorrhage	Carry out an MVA to evacuate the retained products of conception Ask the ANM to put the woman in touch with local support groups. Do not carry out a vaginal examination Refer to an FRU/GH.
Fever	The body temperature is raised Blood peripheral smear is positive for malarial parasite	Site of infection somewhere, including possible sepsis Malaria	Try to ascertain the cause of fever. Start the woman on antibiotics Manage according to the NAMP guidelines for malaria in pregnancy. Treat the malarial fever.
Decreased or absent foetal movements (Note: Foetal movements are felt only after about 4 months of gestation)	FHS heard, and within the normal range of 120-160 beats/minute FHS heard, but the rate is <120 beats/minute, or >160 beats/minute FHS not heard	Baby is normal Foetal distress Intrauterine foetal death	Reassure the woman Re-check the FHS after 15 minutes. If the FHS is still out of the normal range, manage as given under the management of “foetal distress” Inform the woman and her family that the baby might not be well If labour pains are present, conduct the delivery in the usual manner If there ate no labour pains, refer to an FRU for induction of labour to terminate the pregnancy.
Vaginal discharge, with or without abdominal pain		RTI/STI	Start treatment as per the Guidelines for RTI/STI
Leaking of watery fluids P/V	Wet pads/cloths	Premature or prelabour rupture of membranes	Manage as given under the management of “PROM”

Testing the urine for the presence of sugar

This test is required to rule out the presence of gestational diabetes. Though not a sensitive method, it is specific and useful as a cheap screening test.

Testing the urine for bacteriuria

This is important, especially in women complaining of burning micturition. You are advised to carry our this test if the facilities for the same exist in your PHC/GH laboratory.

Interventions

Iron – folic acid supplementation

Stress the need for increased requirements of iron during pregnancy and the dangers of anaemic to every pregnant woman.

All pregnant women need to be given one tablet of IFA (100 mg elemental iron and 0.5 mg folic acid) every day for at least 100 days. This is the prophylactic of IFA.

Start IFA at the prophylactic dose as early as possible, preferably soon as the pregnancy is registered. However, ensure that the woman is able to tolerate the intake of IFA, as iron has a tendency of aggravating the nausea and vomiting, which are part of morning sickness during the first trimester.

If a woman is anaemic (Hb < 11 g/dl or she has pallor), give her two tablets of IFA per day for three months. This means a woman with anaemia in pregnancy needs to take at least 200 tablets of IFA. This is the therapeutic dose of IFA.

A woman with sever anaemia (Hb < 7 g/dl, or those who have breathlessness and tachycardia due to anaemia) should be started on the therapeutic dose of IFA and also be investigated to detect the cause of anaemic. She may require injectable iron preparations, if iron deficiency is found to the mother. Tell her:

- Though the tablets should be taken preferably early in the morning on an empty stomach, she may take the tablets with meals or at night. This will help avoid nausea.
- She should not worry about black stools. This is normal while taking IFA tablets.
- If she has constipation, ask her to drink more water.
- These side-effects are not serious.
- She should avoid taking the tablets with tea, coffee or milk as they reduce the absorption of iron.
- Tablets containing calcium should not be taken at the same time than before, advise her not to stop the tablets despite feeling better.
- Although IFA tablets may take a woman feel less tired than before, advise her not to sop the tablets despite feeling better.
- She should return to you if she has problems in taking IFA tablets.

Injection tetanus toxoid (Inj.TT) administration

Administration of two doses of Inj. TT to a pregnant woman is an important step in the prevention of neonatal tetanus. The first date of TT should be give just after the trimester, or as soon as the woman registers for ANC, whichever is later. The second dose is to be given one month after the first dose, but preferably at least one month before the EDD because, if the gap between the second dose of TT and the EDD is less than 4 weeks, the efficacy of the vaccine is reduced.

Inj. TT is to be given as 0.5 ml per dose, deep intramuscular (IM) in the upper arm,

If the woman has received Inj. TT during a previous pregnancy, a single dose of injection if sufficient. However, in case of doubt, give two injections.

Inform the woman that there may be slight swelling, pain and/or redness at the injection for a day or two.

Malaria prophylaxis

You are advised to follow the guidelines of the National Anti-Malaria Programme (NAMP) for malaria prophylaxis. At the time of printing of this document, the NAMP recommends that in Malaria-endemic areas of India, pregnant women should be given intermittent malaria prophylaxis.

If a pregnant woman is diagnosed with Malaria, start the treatment for Malaria (in accordance with the NAMP guidelines), as malarial fever can cause more harm to the health of the mother and die baby than the drugs used for its treatment.

Counselling

Birth preparedness and complication readiness

It is estimated that 4 out of 10 pregnant or postpartum women will experience some complication related to their pregnancy, and in about 15% of them, the complication will be potentially life-threatning and will require emergency obstetric care. Since most of these complications cannot be predicted, every pregnancy necessitates preparation for a possible emergency.

Birth preparedness

Identification of a skilled provider for birth

All pregnant women should be helper to reach a decision regarding the health provider they want for conducting their delivery: A skilled birth attendant (SBA) should be preferred over a unskilled one. (Note that traditional birth attendants (TBAs), trained or untrained , do not fall into the category of "skilled birth attendants"). Other factors such as the condition of the pregnancy (Complicated or uncomplicated), the distance to the health facility, transport facilities situation etc. all need to be kept in mind finally reaching a decision about the choice of birth attendant.

All pregnant women must be encouraged to opt for an institutional delivery.

Explain to her why delivery at a health facility is recommended. Tell her that

- Any complication can develop during delivery, complications are not always predictable: they can cost the life of the mother and/or the baby.
- A health facility has staff, equipment, supplied and drugs available to provide the best care, if needed . It even has a referral system should the need arise to refer to a higher facility.

Identify support people: People are needed to help the woman care for her children and/or household, arrange for transportation, and/or accompany the woman to the health facility in an emergency. Advise the woman and her family to identify such people and to seek help fro either the close relatives of the woman or community-based health functionaries such as the ANM, AWW, accredited social health activist (ASHA) and the TEA. Ask the ANM of the area to assist the woman for this purpose.

Finances: The woman and her family should be given an estimate of the expected expenses for the delivery and related aspects (such as transport etc.). They should be advised to keep some emergency fund, or have a source for emergency funding, should a complication arise and more money is required the initially anticipated. You should also be aware of the existing schemes that provide funds for maternal health, and any other schemes that may be launched From time to time (for example, refer to the provisions made for woman living below the poverty line under the Janini Suraksha Yojana launched in 2005). Help the women and their families access these schemes and receive the allocated funds to pay for their delivery.

Signs of labour: Advise the woman to come to the PHE- or contact the SBA if she has any one of the following sings which indicate labour:

- A bloody, sticky discharge P/V
- Painful abdominal contractions every 20 minutes or less
- The bag of waters has broken, and she has clear fluid coming out P/V ("leaking").

Complication readiness

Danger signs The woman and her family/caretakers should be informed about potential danger signs during pregnancy, delivery and the postpartum period. She must be told that if she has of the following pregnancy, delivery or the postpartum/post – abortion period, she should immediately visit an FRU or the PHC, WITHOUT WAITING, be it day or night.

Danger signs: visit an FRU

- Any bleeding P/V during pregnancy, and heavy (>500 ml) vaginal bleeding during and following delivery .
- Severe headache with blurred vision
- Convulsions ore loss of consciousness
- Labour lasting for more than 12 hours.
- Failure labour of the placenta within 30 minutes of delivery
- Prerm labour (onset of labour before 34 weeks of gestation)
- Cases with leaking P/V(PROM)
- Continous severe abdominal pain
- All cases of medical illness associated with pregnancy, such as diabetes mellitus, heart disease, asthma, etc. at the onset of labour pains.

Danger signs: Visit a 24 hour PHC

Advice the woman to visit a FRU is she has any of the following conditions:

- High fever with or without abdominal pain, and the woman is too week to get out of bed (indicating infection/sepsis)
- Fast or difficult breathing (dyspnoca) Decreased or absent foctal movements
- Excessive vomiting wherein the woman is unable to take anything orally, leading to a decreased urinary output.

Location of the nearest health centre/FRU: the woman and her family members should be aware of

the nearest health facility, both the PHC where 24-hour functioning emergency obstetric care services are available and the FRU, where facilities for a blood transfusion and surgery are available.

Identification of transportation facilities: Delay in reaching a health care facility is one of the major "delays" responsible for maternal morality. If the woman has decided to deliver at a health facility, a vehicle/EMRI at 108 should be identified which should be available whenever the woman needs it, to take her to that health facility.

Even if the woman decides to deliver at home, a vehicle should be identified and ideally be kept ready to transport her to the nearest health facility or referral centre in case she develops some complications needing immediate referral and care.

The various schemes available for assisting the woman with transportation facilities should be Kept in mind. Also keep yourself informed regarding any new schemes diet may be launched from time to time.

Preparedness for blood donation: Haemorhage, both anterpartum and postpartum, is an important cause of maternal mortality: Blood transfusion cane be life-saving in such cases. As blood cannot be "bought" one needs voluntary donors to replace the blood before it is issued for transfusion,. Such donors (2-3 in number) must be ready, should the need arise.

Diet and rest

The woman should be advised to eat more than her normal diet throughout her pregnancy. Remember, a pregnant woman needs about 300 extra kcal per day compared to her usual diet. She should be told that she needs these extra calories for:

- Maintenance of her health as a mother
- The needs of the growing foetus
- Successful lactation

Diet chart during pregnancy and lactation

Food Item (in grams)	Adult woman		
	Moderate work	Additional in pregnancy	Additional in lactation
Cereals	440	35	60
Pulses	45	15	30
Leafy Vegetables	100	-	-
Other Vegetables	40	-	-
Roots and tubers	50	-	-
Milk	150	100	100
Oil and Fat	25	-	10
Sugar or Jaggery	20	10	10

- Special categories of women have been identified who should be given priority for additional nutrition during pregnancy. They include the following:
  - Women with a reduction in the dietary intake below habitual levels during pregnancy.
  - Women who have an increased level of physical activity above the usual levels during pregnancy.
  - Women with a combination of both the above-mentioned factors.
  - Adolescent girls who become pregnant
  - Women who become pregnant during lactation
  - Women who become pregnant within two years of the previous delivery.

The woman's food intake should be especially rich in proteins, iron, vitamin A and other essential micronutrients.

- The other members of the family, especially those who take decisions regarding the type of food brought home and /or given to the pregnant woman, such as her husband and mother-in- law, should also be taken into confidence and counselled regarding the recommended diet for the pregnant woman. Ask for their assistance to help ensure that the woman eats enough and avoids hard physical work.
- Some of the recommended dietary items are ce-

reals, milk and milk products such as curd, green leafy vegetables and other vegetables, pulses, eggs and meat, including fish and poultry (if the woman is a non-vegetarian) nuts (especially groundnuts), jaggery, fruits, etc. Give examples of the types of food, suggested preparations, if possible, and how much to eat.

- Tell her about the locally available foods rich in iron such as groundnuts and jaggery. Tell the woman to avoid taking tobacco, tea, coffee or milk, especially within one hour of a meal, as they have been shown to interfere with the absorption of iron. Also advise her to take foods rich in proteins and vitamin C (e.g. lemon, amla, guava, oranges, etc.) as both help in the absorption of iron.
- The diet should be rich in fibre so that she does not have constipation
- The diet should be advised keeping in mind the socio-economic conditions, food habits and taste of the individual
- Food taboos must be looked into while counselling the woman regarding her dietary intake. If there are taboos about nutritionally foods, the woman should be advised against these taboos. In certain communities, food taboos exist for sex selection of the foetus. These, especially omission of certain foodstuffs from the diet, should be strongly discouraged.
- If a woman has PIH, she should be encouraged to eat a normal diet with no restrictions on fluid, calorie and/or salt intake: such restrictions do not prevent PIH from converting into pre-eclampsia, and may be harmful to the foetus.
- The woman should be advised to refrain from taking alcohol or smoking during pregnancy.
- The woman should be advised not to take any medication unless prescribed by a qualified health practitioner.
- The woman should be advised to sleep for 8 hours at night and rest for another 2 hours during the day. She should be advised to refrain from doing heavy work, such as construction work and full-time farm labour work, as it can adversely affect the birth weight of the baby. The other members of the household should be taken into confidence and advised to help the woman in carrying out her routine household chores.
- All pregnant women should be told avoid the supine position, especially in late pregnancy, as it affects both the maternal and the foetal physiology. During pregnancy, the pressure exerted by the uterus on the main pelvic veins results in a reduced quantity of circulating blood reaching the right side of the heart. This causes reduced oxygenation to the brain and can therefore lead to a fainting attack, a condition referred to as the **supine hypotension syndrome** it can also result in abnormal FHR patters, and may also cause a reduction in the placental blood flow. If the supine position is necessary, a small pillow under the lower back at the level of the pelvis is recommended.

Sex during pregnancy

- It is safe to have sex throughout the pregnancy, as long as the pregnancy is “normal”.
- Sex should be avoided during pregnancy if there is a risk of abortion(h/o previous recurrent spontaneous abortions), or a risk of a preterm delivery (h/o previous preterm labour).
- Some women experience a decreased desire for sex during pregnancy. The husband should be informed that this is normal and the woman’s consent should be sought before engaging in sex. This is extremely important as forced and unsafe sex can have adverse consequences on the health of the mother and die foetus, resulting in an abortion or preterm labour.

Infant and young child feeding

Pregnancy is the ideal time to counsel the mother regarding the benefits of breast feeding her baby. Though breast feeding is almost universal in India, a few points need to be emphasized to the would-be-mother.

Initiation of breast feeding

Counsel the mother that breast feeding should ideally be initiated within half an hour of a normal delivery (or within two hour of a caesarean section, or as soon as the mother regains consciousness, incase she undergoes a caesarean section).

It is common practice in India to delay initiation colostrums (the first milk) is thrown away, And pre-lacteal feeds are given instead. This has obvious disadvantages. One, the pre-lacteal feed may not be

hygienic and can cause an intestinal infection in the baby. Second, the baby is deprived of colostrums, which is very rich in protective antibodies.

Most importantly, the sucking and rooting reflexes in the child, which re essential for the baby to successfully start breast feeding, are the strongest immediately after delivery, making the process of initiation much easier for the mother and the baby. These reflexes gradually become weaker over the span of a few hours, thus making breast feeding difficult later on.

Exclusive breast feeding for 6 months: Emphasize to the mother that only breast milk and nothing but breast milk should be given to the baby for the first 6 months, not even water. Assure the mother that breast milk contains enough water to quench the baby's thirst even in the peak of summer.

Demand feeding: this refers to the practice of breast feeding the child whenever he/she "demands" it, as can be made out by the child crying. The practice of feeding the child by the clock should be actively discouraged. After a few days of birth, most children will develop their own "hunger cycle" and will feed every 2-4 hours. Remember that each child is different as far as the feeding requirements and timings are concerned.

The practice of giving night feeds should be actively encouraged. Often, there is a misconception that breast feeding the baby at night disturbs the mother's sleep, thus denying her of adequate rest. Inform the woman and her husband that this is not so. Night feeds help the baby to sleep more soundly.

Rooming in: This refers to the practice of keeping the mother and baby in the same room and preferably on the same bed. This is usually practiced in India. This practice should be encouraged as it has certain advantages.

- Makes demand feeding easier to practice, as the mother can hear the child cry.
- Keeps the baby warm, thus preventing hypothermia in the newborn.
- Helps build a bond between the mother and the baby.

Complementary feeding at 6 months: The mother should be told that after 6 months of age, breast milk alone does not meet the nutritional requirements of the baby. The baby needs supplementary food, **in addition to breast milk**. Advise the mother to begin with semi-solid soft food devoid of spices, supplemented with a small amount of ghee/butter/oil. The frequency of feeds and the quantity of each feed I should be increased gradually. Over a period of time, the bay may be given solid foods. A one year –old child should start eating from the family pot, and should have an intake that is half of the adult diet. Feeding bottles should be strictly discouraged.

Contraception

The woman should be advised regarding birth spacing (or limiting, as the case may be). Explain to the woman and her partner that after birth, if she has sex and is not exclusively breast feeding, she can become 5 pregnant as early as six weeks after delivery. Therefore, it is important to start thinking early about what family planning method they will use.

Ask about the couple's plans for having more children. If they desire more, advise them that a gap of 3-5 years between pregnancies is healthier for both the mother and the child.

Compulsory institutional deliveries

Every pregnant woman should be advised and encouraged to go in for an institutional delivery. However, about 60% of the deliveries in India still occur at home. There are medical/obstetric conditions during a pregnancy when the chances of a complication occurring are increased , and a home delivery may be risky and potentially life-threatening. Under such conditions, explain to the woman why the delivery needs to be at a facility level Only and strongly advise her to deliver only in a institutional setting. Such conditions/ complications are

- Mild pre-eclampsia
- PPH in the previous pregnancy
- More than 5 previous births
- Previous assisted delivery
- Maternal age less than 16 years
- H/o third-degree tear in the previous pregnancy

FRU

- Severe anaemia
- Severe pre-eclampsia /eclampsia
- APH
- Transverse foetal lie or any other malpresentation
- Caesarian section in the previous pregnancy
- Multiple pregnancies
- Premature rupture of membranes (PROM)
- Medical illness such as diabetes mellitus, heart disease, asthma, etc. during pregnancy.

Diagnosis of labour

Intermittent contractions after 22 weeks of gestation, contractions associated with blood-stained discharger or watery vaginal discharge should raise a suspicion of the onset of labour. Normal vaginal discharge should not be confused with slow. The onset of labour can be confirmed by the following;

- Cervical effacement-progressive shortening and thinning of the cervix during labour
- Cervical dilatation {see Annexure 8:"Assessing cervical dilatation:}

Stages of labour

- The First stage of labour starts with the onset of labour pains to full dilatation of the cervix. This stage takes about 12 hours in primigravidas and half that time for subsequent deliveries.
- The second stage starts from full dilatation of the cervix to the delivery of the baby. This stage takes about 2 hours for primigravidas and only about half an hour for subsequent deliveries.
- The third stage starts after the delivery of the baby and ends with the delivery of the placenta. This stage takes about 15 minutes to half an hour, irrespective of whether the woman is a primigravida or multi-gravida.
- Frequent monitoring for one hour immediately after delivery is critical to detect PPH. This period is sometimes referred to as the fourth stage of labour.

Assessment of the progress of labour

The progress of labour is assessed by:

- Assessing the changes in cervical effacement and dilatation (by conducting a P/V examination)
- Assessing the progress in foetal descent (by conducting a abdominal and/or a P/V examination)

Abdominal examination to access the descent of the presenting part

Abdominal palpation should be conducted to assess the descent of the presenting part. If the head is above the symphysis pubis it is fully palpable and mobile. If the head is entirely below the symphysis pubis it is not palpable abdominally.

Vaginal examination to assess the stage and progress of labour

- Always examine the abdomen before examining the vagina.
- **Do not** shave the perineal area.
- Prepare clean gloves swabs and pads.
- Wash your hands with soap and water before and after each examination. Carry out the vaginal examination under strict aseptic conditions.
- Always inform the woman and take her verbal consent before carrying out a vaginal examination.
- Perform a vaginal examination gently. Do not start a vaginal examination during a contraction.
- **Remember,** do not carry out a vaginal examination if the woman is bleeding at the time of labour or at any time after 5 months (20 weeks) of pregnancy. Manage this as a case of "Vaginal bleeding in late pregnancy".
- Clean the vulva and perineal area with a mild antiseptic solution. Use a cotton swab soaked in antiseptic solution to clean the vulva. Wipe the vulva in the anterior to posterior direction. Use a swab only once.
- Place the woman in the supine position with her legs flexed and apart.
- Separate the labia with the thumb and forefinger of the left hand and clean the area once again. Use two fingers of the right hand (index and middle fingers) and insert them gently into the vaginal orifice without hurting the woman.
- During a vaginal examination, determine the following:
  - * Cervical effacement
  - * Cervical dilatation in cm
  - * The presenting part. Try and judge if it is hard, round and smooth (the head), If not try and identify the presenting part.
  - * Incase the vertex is not the presenting part, manage the case as a malpresentation. Such cases need to be referred to a higher health facility.
  - * The position or the station of the presenting part.
  - * Feel for the membranes. Are they intact?
  - * If the membranes have ruptured, check

whether the colour of the amniotic fluid is clear or meconium stained.

* Feel for the umbilical cord. If it is felt, it is a case of prolapsed cord. If the cord pulsations are felt, refer the woman to an FRU/GH immediately. Explain to the woman and her family that a caesarean section may be required. Manage the woman as given under the management of "prolapsed cord".

The stage of labour can be decided as follows

- If the cervix is dilated 1-3cm, and the contractions are weak than 2 in 10 minutes this is the first stage of labour: but the woman is not in active labour yet.
- If the cervix is dilated> 3 cm, but not fully, the woman is still in the first of labour. However, she is now in active labour.
- Full cervical dilatation (10 cm; the cervix is no longer felt on vaginal examination) a bulging thin perineum, a gaping vagina and anus, and the head visible through the introitus, even in between contractions indicate the second stage of labour, and that delivery is imminent.
- Remember, vaginal examinations are rarely required more frequently than once every 4 hours.
- Oxytocic drugs such as Inj. Oxytocic IM, should not be given before delivery of the baby. The use of oxytocic drugs is associated with an increased incidence of rupture of the uterus and consequent severe APH.

Supportive care to the woman during labour

- Explain all the procedure, seek permission for examination and carrying out the procedures, an discuss the finding with the woman.
- Keep the woman informed about the progress of labour.
- Praise the woman, encourage her and reassure her that things are going well.
- Ensure and respect the privacy of the woman during examinations and discussions.
- Always wash your hands with soap and water before examining the woman.
- Ensure cleanliness of the birthing area.
- Enema should **not** be routinely given during labour. Enema should be given only when needed. E.g., when the woman complains of constipation on admission or at the onset of labour, or if the woman wishes to have an enema.
- Encourage the woman to empty her bladder frequently. Remind her every 2 hours or so.
- The presence of a second person or a birth companion of the woman's choice in additional to a SBA is beneficial. Birth companions provide comfort, emotional support, reassurance, encouragement and praise. On a practical level too, the presence of a second person is valuable, in that if at any point during the labour additional assistance is required, or in a emergency this second person can be useful, even if it is only to seek help. But one must ensure cleanliness ad concentrate on preventing infection.
- Women should be allowed to remain mobile during labour, especially the first stage, as this helps in having a shorter and less painful labour.
- The woman should be free to choose any position she desires and feels comfortable in during labour and delivery. She may choose from the left lateral, squatting, kneeling, or even standing (supported by the birth companion) positions. Remember, given a choice, the woman will often change positions as no position is comfortable for very long.
- To relieve the woman of pain and discomfort, a change in position and mobility is helpful. Encourage the birth companion to massage the woman's back if she finds this helpful, to hold the woman's hand and sponge the woman's face between contractions.
- Other non-pharmacological methods of relieving pain during labour include:
- Calm and gentle voice of the birth attendant
  * Offering the woman encouragement, reassurance and praise
  * Relaxation techniques performed by the woman such as deep breathing exercises and massage.
  * Placing a cool cloth on the woman's forehead
  * Assisting the woman in voiding urine and in changing her position.
- Women who are not at risk of requiring general anaesthesia can have light, easily digested, low-fat food during labour, if they wish. The advan-

tage of having food far outweigh any risks related to a full stomach and the use of general anaesthesia. This is because labour requires large amounts of energy.

- In women who have not eaten for some time, or who are undernourished, the effects of labour can quickly lead to physiological exhaustion, dehydration and ketosis (maternal acidosis), which can lead to foetal distress. Therefore, encourage the woman to eat and drink as she wishes throughout labour.

Normal delivery

Management of the first stage of labour

Not in active labour

The cervix is dilated 0-3 cm and contractions are weak, less than 2 in 10 minutes.

- Monitor the following every hour.
  - * Frequency (once in how many minutes), intensity (how strong), and duration (for how many seconds does it last) of contractions.
  - * FHR. The normal FHR is between 120 and 160 beats/minute.
  - * The presence of any sign that denotes an emergency (such as difficulty in breathing, shock, vaginal bleeding convulsions or unconsciousness)
- Monitor the following every 4 hours:
  - * Cervical dilatation (in cm)
  - * Temperature
  - * Pulse
  - * Blood pressure
- Record the time of rupture of the membranes and the colour of die amniotic fluid.
- Never leave the woman alone
- If after 8 hours, the contractions are stronger and more frequent, but there is no progress in cervical dilatation with or without rupture of die membranes, this is a case of non-progress of labour. Refer the woman immediately to an FRU/GH.
- On the other hand, if after 8 hours, there is no increase in the intensity/frequency/ duration of contraction and the membranes have not ruptured and there is no progress in cervical dilatation, ask the woman to relax. Advise her to send for you again when the pain/discomfort increase, and/or there is vaginal bleeding, and/or the membranes rupture.
- If the membranes were already ruptured on admission, but even after 8 hours there is no increase in die frequency/intensity of contractions, refer die woman to an FRU/GH (prolonged latent phase) for induction of labour.

In active labour

The cervix is dilated 3 cm or more.

- Monitor the following every 30 minutes
  - * Frequency, intensity and duration of the contractions
  - * FHR
  - * Presence of any emergency sign.
- Monitor the following every 4 hours:
  - * Cervical dilatation (in cm)
  - * Temperature
  - * Pulse
  - * Blood pressure
- Again , do not leave the woman alone.
- Start maintaining a Partograph once the woman is in active labour.

Simplified Partograph

The Partogaph is a graphic recording of the progress of labour and salient features of the **mother and foetus**. It is a tool to assess the progress of labour and recognize the need for action and referral at the appropriate time. The instructions for filling the Partograph are given below.

Foetal condition

- The FHR should be counted and recorded every half-an hour. Count the FHR for one full minute. The rate should preferably be counted immediately following a uterine contraction. An FHR of >160 beats/minute or < 120 beats/minute indicates foetal distress. Each of the small boxes in the vertical column of a Partograph represents half-hour intervals.
- Simultaneously record the condition of the membranes and colour of the amniotic fluid as visible at the vulva every 30 minutes as:
  - * Intact membranes (mark T)
  - * Clear liquor (mark ‘C’)

* Meconium stained (mark 'M')
* no liquor (mark 'A')

Labour

- Start plotting on the labour graph only after the woman is in active labour. The woman is said to be in active labour when the cervical dilatation is more than 3 cm and least 2 goof contractions. (i.e. each lasting for more than 20 seconds) occur in 10 minutes.
- Start recording the cervical dilatation (in cm) when the woman reports in labour and then every four hours.
- The initial recording is placed to the left of the alert line (cervical dilatation must be 3 cm and above, i.e., the woman must be in active labour before you start plotting the graph). Normally the line should continue to remain to the left of the alert line. Write the time accordingly in the row for time.
- If the alert line is crossed (the graph moves to the right of the alert line) indicates prolonged labour, and you should be alert that labour is not progressing as it should. Note the time when the alert line is crossed. Start preparing for referral to an FRU/GH.
- Crossing of the action line (the graph moves to the right of the action line) indicates the need for intervention and referral. There is a difference of 4 hours between the alert and the action line. By the time the action line is crossed, the woman should ideally have reached the FRU/GH for receiving appropriate and timely intervention.
- Record the number of good contractions (lasting more than 20 seconds) in 10 minutes every half –an-hour and accordingly, blacken the boxes on the Partograph.

Maternal condition

Record the maternal pulse and blood pressure every half-an-hour and plot them on the graph. Record both the systolic and the diastolic blood pressure using a vertical arrow, with the upper end of the arrow representing the systolic blood pressure and the lower end indicating the diastolic blood pressure. Use crosses to mark to pulse.

Intervention

Mention here any drug that you have administered during labour, including the dose and route of administration, and when. Also include the food items and liquids consumed by the woman during that period.

Management of the second stage of labour

- If the cervix is fully dilated or the perineum is thin and bulging with the anus gaping and the head of the baby visible at the vaginal introitus, it is the second stage of labour.
- Monitor the following every 5 minutes.
  * Frequency, duration and intensity of contractions FHR
  * Perineal thinning and bulging
  * Visible descent of the foetal head during contractions
  * Presence of any signs indicating an emergency.
- The upright positions such as standing, sitting, squatting and being on all fours makes pushing easier. Therefore, if the woman finds it difficult to push, or there is slow descent of the presenting part, you should change the position of the woman.
- The woman should be allowed to push down when she has contractions if she has the urge to do so during the second stage of labour.
- Bearing down efforts are required after the cervix is fully dilated, and even more so when the head is distending the perineum. Occasionally , the woman feels the urge to push before the cervix is fully dilated. This should be discouraged as it can result in oedema of the cervix which may delay the progress of labour.
- To prevent pushing at the end of the first stage of labour (before the cervix is fully dilated), teach the woman to pant, i.e. to breathe with an open mouth, take in two short breaths followed by a long breath out.
- Asking the woman to hold her breath and asking her to bear down in the second stage of labour should **not** be done. Holding the breath is potentially harmful. It may reduce the quantity of blood reaching the uterus and placenta. It may also reduce the supply of oxygen to the foetus.
- Giving the woman oxytocics to shorten second

stage of labour is **not** advisable.

- Avoid ironing the perineum (or using the "Sweep and stretch" technique) to hasten delivery.

Episiotomy

- There is no evidence that routine episiotomy decreases perineal damage, future vaginal prolapse or urinary incontinence.
- Remember, whenever an episiotomy is required, a **right paramedian episiotomy** is preferred.
- Indications for conducting an episiotomy
  - * Complicated vaginal delivery (refer to a higher health facility in case of a malpresentation)
  - * H/o third-or fourth-degree perineal tears
  - * Foetal distress
  - * Instrument /assisted delivery
- Ensure a controlled delivery of the head by taking the following precautions
  - * Encourage the woman to push only during pains (a contraction)
  - * Keep one hand gently on the head as it advances with the contractions.
  - * Support the perineum with the other hand during delivery and cover the anus with a pad held in position by the side of the hand.
  - * Leave the perineum visible (between the thumb and the index finger).
  - * Ask the mother to breathe steadily and to not push during delivery of the head.
  - * Encourage rapid breathing with the mouth open.
  - * Do NOT apply fundal pressure to hasten delivery of the head.
- Feel gently around the baby's neck for the presence of the umbilical cord around the neck. If the cord is present around the neck.
  - * And if it is loose, deliver the baby through the loop of the cord, or slip the cord over the baby's head.
  - * If the cord is tight, clamp it and cut the cord, and then unwind it from around the neck.
  - * Delivery of the shoulders and the rest of the baby.
  - * Wait for spontaneous delivery of the shoulders. This usually occurs within 1-2 minutes.
  - * Perineal tears can be prevented by delivering one shoulder at a time. If there is difficulty in delivering the shoulder, suspect shoulder,dystocia. Ask the woman to take a position with extreme flexion at the knees and hips with the knees wide apart. The shoulder may be released from behind the symphysis pubis and may deliver. If not, then refer the woman immediately to an FRU/GH. Fortunately, shoulder dystocia is rare in India.
  - * Apply gentle pressure downwards to deliver the anterior shoulder.
  - * Then lift the baby up, towards the mother's abdomen, to deliver the lower (posterior) shoulder.
  - * The rest of the baby's body smoothly follows out.
  - * Place the baby on the mother's abdomen or in the baby tray.
- Note the time of delivery

Cutting the cord

- Tie and cut the cord after 2-3 minutes of delivery, during which time the cord will normally stop pulsating. This will result in an increased amount of blood transfused into the foetal circulation, and thus help avoiding neonatal anaemia.
- Put ties tightly around the cord at 2 cm and 5 cm from the baby's abdomen.
- Cut between the ties with a sterile blade.
- Look for oozing of blood from the stump. If these is oozing , place a second tie between the baby's skin and the first tie.

Give immediate newborn care

- If the baby does not cry in 30 seconds, take steps to resuscitate the baby.
- Ensure warmth to the baby to prevent hypothermia.
- Rule out the presence of another baby by palpating the abdomen and trying to feel for foetal parts.
- It is recommended that the umbilical cord stump by left dry, and only routine daily care be given with clean safe water. Do not apply any substance

to the stump.

- Note the Apgar score of the baby at 1 minute and at 5 minutes after delivery.
- Care of the newborn: The newborn needs to be taken care of. The elements of essential newborn care are given in box

Elements of essential newborn care

* Maintain the body temperature and prevent hypothermia
* Maintain the airway and breathing
* Breast feed the newborn
* Take care of the cord
* Take care of the eyes

- Leave the baby on the mother's chest for skin-to-skin contact.
- Cover the baby to prevent lose of body heat. If the room is cool, use additional blankets to cover the mother and the baby.
- Encourage the mother to initiate breast feeding.

Active management of the third stage of labour

The active management of the third stage of labour consists of the following three activities.

Uterotonic drug

Giving a uterotonic drug (one that enhances the contraction of the uterine muscles) has been shown to be effective in preventing PPH.

Although Inj. Oxytocin (in a dose of 10 units intramuscular) is the drug of choice for preventing PPH, due to administrative difficulties, Misoprostol can now be used for the same purpose. Three tablets of 200 mcg each of Misoprostol (a total dose of 600 mcg) should be given immediately after delivery of the baby. It should be given either sub-lingually or orally.

Before giving Misoprostol, ensure that there is no additional baby(ies). This can be done by palpating the abdomen and ruling out the presence of foetal parts.

Administration of Misprostol has a few common side-effects. These are not dangerous, and therefore should not cause worry. However, you must inform the mother and her companion(s), if any about these to ease any apprehensions, should these side-effects occur. Shivering and gastro-intestional disturbance are common side-effects.

Controlled cord traction(CCT)

This is a technique to assist the expulsion of the placenta and helps to reduce the chances of a retained placenta and subsequent PPH.

Do **not** exert excessive traction on the cord while performing controlled cord traction. Never squeeze or push the uterus to deliver the placenta.

Examine the placenta carefully to ensure that none of the pieces are missing. Retained placental fragments or pieces of membrane will cause PPH. This can be suspected if a portion of the maternal surface of the placenta is missing or there are torn membranes with vessels. Ensure that the placenta is delivered completely with all the membranes.

Uterine massage

- This technique helps in contraction of the uterus and thus prevents PPH.
- Immediately after delivery of the baby, massage the uterus by placing your hand on the woman's abdomen until it is well contracted.
- Repeat the massage every 15 minutes for the first 2 hours. Ensure that the uterus does not become relaxed (soft) after the massage is stopped.
- If the placenta is not delivered within 30 minutes of giving Misoprostol , and the woman is not bleeding, try and remove the placenta again by controlled cord traction.
- Empty the bladder, and encourage the woman to breast feed.
- If the placenta cannot be delivered after another 20 minutes, and the woman is not bleeding, empty the bladder, initiate breast feeding and repeat controlled cord traction.
- The placenta may separate. If it does not separate and the woman is still not bleeding, refer her to a FRU/GH.

Immediate postpartum care

- The first one hour after delivery of the placenta is sometimes referred to as the fourth of labour.
- After delivery of the placenta, check that the uterus is well contracted, i.e. it is hard and round, and there is no heavy bleeding. Repeat the checking every 5 minutes. If the uterus is not well contracted, massage the Uterus and expel the clots. If bleeding continues even after 10 minutes, manage as given under "postpartum

hemorrhage".

- Examine the perineum, lower vagina and vulva for tears.
- Estimate and record the amount of blood lost throughout the third stage and immediately afterwards. If the loss is around 250 ml, but the bleeding has stopped, observe the woman for the next 24 hours.
- Monitor the following every 10 minutes for the first 30 minutes, then every 15 minutes for the next 30 minutes, and then every 30 minutes for the next three hours.
  * Blood pressure, pulse, temperature
  * Vaginal bleeding
  * Uterus, to make sure that it is well contracted
- Look for vaginal and/or perineal tears
- Clean the woman and the area beneath her. Put a sanitary pad or a folded cloth under her buttocks blood. This will also help in estimating the amount of blood, lost, by counting the number of pads/cloths soaked. Help her change her cloths, if necessary.
- Ensure that the mother has enough sanitary napkins or clean cloths to collect the vaginal blood.
- Dispose of the placenta in the correct, safe and culturally appropriate manner. Use gloves while handling the placenta. Put the placenta into a leak-proof bag. Incinerate the placenta or bury it at least 10 m away from a water source, in a 2 m deep pit refer to the guidelines laid down by the state or your institution for the same.
- Keep the mother and the baby together, do not separate them. Encourage early breast feeding.
- Encourage the woman to eat and drink, and rest.
- Encourage the woman to pass urine. If the woman has difficulty in passing urine, or the bladder is full (as evidences by a swelling over the lower abdomen) and she is uncomfortable, help her pass urine by gently pouring water over her vulva.
- Weigh the baby.
- Ask the birth companion to stay with the mother. Do not leave the mother and the newborn alone. Ask the companion to watch the woman and call for help if any of the following occurs.
  * The bleeding increases
  * The woman feels dizzy.
  * The woman has severe headache.
  * The woman has visual disturbance.
  * The woman has epigastric distress.
  * The woman complaints of breathlessness.
  * The woman complains of increased abdominal or perineal pain.
- Enter the following information in the labour register:
  * Name of die the woman
  * Age of the woman
  * Parity
  * ANC received (or not) : mention the number of ANC visits received
  * Mode of delivery (normal or assisted)
  * Birth weight of the baby
  * Apgar score of the baby at 1 minute and 5 minutes after delivery
- Do not discharge the woman before 24 hours after delivery. This is a crucial period for the occurrence and management of PPH. The woman must be kept under observation during this time.

Counselling

Counsel the woman regarding the aspects discussed below.

Postpartum care and hygiene

Advise and explain to the woman

- To always have someone near her for the first 24 hours after delivery to respond to any change in her condition.
- Not to insert anything into the vagina
- To wash the perineum daily and after passing stools. Wash in an anteroposterior direction from the vulva to the anus.
- To change the perineal pads every 4-6 hours, or more frequently, if there is heavy lochia.
- To wash cloth pads, if used, with plenty of soap and water and dry them in die sum.
- To bathe daily.
- To have enough rest and sleep.
- To avoid sexual intercourse for the first six weeks or until the perineal wound heals, whichever is later.

- To wash her hands before handling the baby.

Nutrition

- Advise the woman to eat a greater amount and variety of healthy foods. Give her examples of the types of food and how much to eat.
- Reassure the mother that she can eat normal food: These will not harm the breast feed baby.
- Spend more time on nutrition counselling with very thin woman and adolescents.
- Determine if there are important food taboos, especially against foods that are nutritionally healthy. Advise the woman against these taboos.
- Talk to family members such as her husband and mother-in-law, to encourage them to help ensure that the woman eats enough and avoids heavy physical work.
- Drink plenty of water

Contraception

Advise the woman regarding birth spacing or limiting as the case may be.

Registration of birth

Emphasize to the woman that she must get the birth of the baby registered with the local Panchayat, or any other appropriate registering authority. This is a legal requirement. Also the birth certificate issued is an important document stating the date of the child, and is required for many purposes, e.g. to gain admission to a school.

Postpartum visit

- Inform the woman about the next routine postpartum visit.
- As the woman is kept under observation for the first 24 hours after delivery, the first postpartum visit is taken care of during her stay at the PHC/GH health facility.
- The second postpartum is it should be planed within 7-10 days after delivery. Either ask the ANM of that area to pay a visit to the woman and her baby, or ask the woman to return to the PHC/GH for a postpartum check-up.
- If the woman misses her postpartum visits, inform her regarding the danger signs (see below) and when to return.

Danger signs

- For the following symptoms and signed in the mother, advise the woman and her family to go to an FRU immediately, day or night, **without waiting.**
  - Excessive vaginal bleeding, i.e soaking more than 2 or 3 pads in 20-30 minutes after delivery, OR bleeding increase rather than decreases after the delivery.
  - Convulsions
  - Fast or difficult breathing
  - Fever and weakness: inability to get out of bed
  - Severe abdominal pain
- Advise the woman that she should visit you at the PHC as soon as possible, in case she suffers from any of the following symptoms.
  - Fever
  - Abdominal pain
  - The woman feels ill
  - Swollen, red or tender breasts, or some nipples
  - Dribbling of urine or painful micturition
  - Pain in the perineum, or pus draining from the perineal area
  - Foul-smelling lochia.
  - Breast engorgement, crack nipple.

Obstructed Labour

Obstructed labour can occur due to

* CPD (Cephalo-pelvic disproportion)
* Hydrocephalus
* Brow presentatio
* Shoulder presentation and
* In tumour complicating pregnancy

Diagnosis

History of

- Prolonged labour
- Prolonged rupture of membranes
- Delivery attempted elsewhere
- Slow progress in labour, no progress in second stage
- High head, in spite of full cervical dilatation
- Patient is anxious, in agony, exhausted and the tongue is dry
- The pulse is rapid

Abdominal examination

* The uterus is hard and tender
* The uterus is contracted on the baby making palpation difficult
* Presence of "retraction ring of Bandl" (demarcation between the upper and lower segment will be clearly visible)
  - » A ridge that may form around the uterus at the junction of the upper and lower uterine segments during the prolonged 2nd stage of an obstructed labour
  - » The lower segment is abnormally distended and thin and the upper segment is abnormally thick.The ring which may be felt and seen abnormally is a warning of impending uterine rupture.

- The fetal heart is absent or irregular

Vaginal examination

* Vagina is hot and dry
* Large caput on the presenting part

Diagnosis of Rupture uterus

- Patient shows signs of shock
- Uterine contractions cease
- Fetal parts are easily palpable
- Vaginal bleeding may be present
- Fetal heart is absent
- Presenting part recedes from pelvis

Management Of Threatend rupture

- Resuscitation
- Arrange blood
- Correct dehydration and hypovolaemia
- Antibiotics
- Take immediate steps to empty the uterus.
- If facilities are not available, shift the patient to the nearest higher centre.
- Before shifting – give sedation
  * Terbutaline inhalation 2 puffs to relax the uterus

Female contraception

Definition

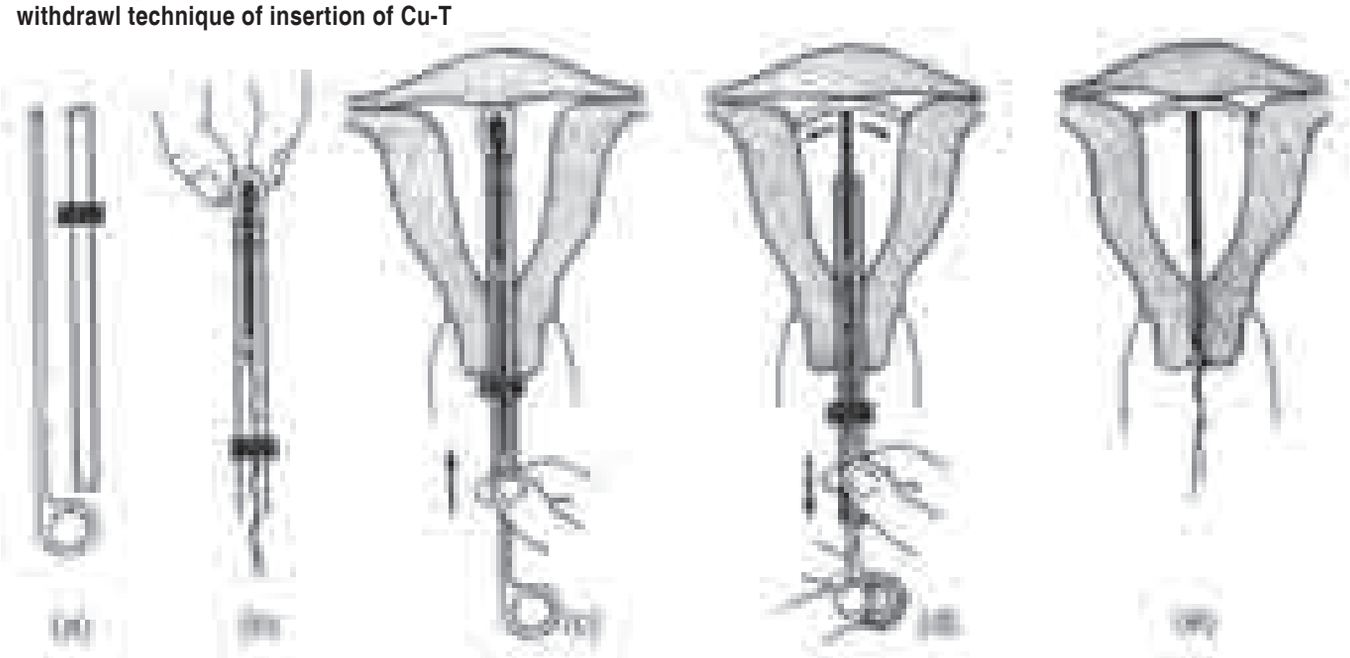
A method or system which allows intercourse and yet prevents conception is called a contraceptive method.

Temporary Methods of Female Contraception:

* Natural methods
* Barrier contraception
* Intra Uterine Device (IUD)
* Suppression of ovulation

1. Natural Methods:
   * Abstinence is determined by subtracting 18 days from shortest cycle and 10 days from longest cycle.
   * Temperature method
   * Withdrawal method
2. Barrier Contraception:
   * Diaphragm
   * Fem shield
   * Today sponge

withdrawl technique of insertion of Cu-T



Diaphragm	Fem shield	Today Sponge
Dome shaped thin rubber with rubber covered metal rim.	Newly developed loose fitting sheath made OD polyurethane ring at the close end, coital independent, does not slip off easily, protects against STD	Mushroom shaped polyurethane sponge containing nonoxynol-9, should be placed higher in vagina, remains effective for 24 hrs, used only once

3. Intrauterine Device:
   * Plate of polyethylene with cu-wire wrapped ideal for spacing
   * Effective reversible and long term method
   * Inserted by withdrawal technique
   * Inserted soon after menstruation, after abortion and delivery

Acts by

* Foreign body reaction
* Prevents implantation by enzymatic and metabolic changes
* Progesterone-alteration of cervical mucus
* Copper is spermicidal

Time of insertion:

After the cessation of menstruation (within 10 days of LMP) This is used as an emergency contraception within 5 days of unprotected intercourse to prevent pregnancy

It should not be used in nulliparous women

It should not be inserted in PID, tumours of the genital tract and during amenorrhoea

Complications

Immediate	Early	Late
Difficulty in insertion	Perforation	PID
Vasovagal attack	Spotting	Ectopic pregnancy
	Infection	Menorrhagia
	Expulsion	

Advantages

- One time insertion gives protection for 3 years
- No systemic side effects
- No adverse effect on lactation

4. Hormonal Contraception
   * Combined pill
   * Triphasic pill
   * Mini pill

- Acts by inhibiting ovulation
- Atrophic changes in endometrium
- Alters cervical mucus

- Pregnancy rate 0.1/100 women
- Pill should be taken everyday regularly
- If missed for one day,next day 2pills should be taken, if missed for 2days some other method should be used along with this

Combined Pill

Benefits	Contraindications
Dysmenorrhoea	Obesity, History fo thrombosis, smoker for more than 35 years
Pre-menstrual tension	Heart disease, Hypertension, Diabetes
Prevents anaemia	Chronic Liver disease, Breast cancer, thyroid disease
Lowers the incidence of benign breast disease, functional ovarian cyst, ovarian and uterine malignancy, PID	Patients on rifampicin / sodium valproate
Rheumatoid Arthritis	4-6 weeks prior to planned surgery
	Lactating women
	People who are having tumours of the genital tract

Triphasic pill

No adverse effect on carbohydrate and lipid metabolism

Minipill

- Lactating mothers
- Women more than 35 years
- Can be used in patients who have contraindications for estrogen

Injectable Contraceptives

DMPA (Depot Medroxy Progesterone Acetate) as monthly, once in 3 monthly or 6 monthly injection

- Advantages
  - No worry of missing pill
  - Good compliance
- Disadvantages
  - Break through bleeding
  - Delay in return of fertility

Implants

Norplant 1and2

- Pregnancy rate 0.2 to 1.3/100 women years
- Advantages
  - Used in lactating women , over 40 years
- Disadvantages
  - Break through bleeding
  - Irregular cycles

Other preparations

1. Sialistic vaginal rings: kept in vagina for 3 weeks, pregnancy rate 3 to 5/100
2. Skin patches: 3 for 3 weeks followed by 1 week patch free interval pregnancy rate 1 to 2.8/100 women years
3. Centchroman 30mg on 1st day of menses then twice weekly for 12 weeks, weekly there after Pregnancy rate 1.83/100 women years

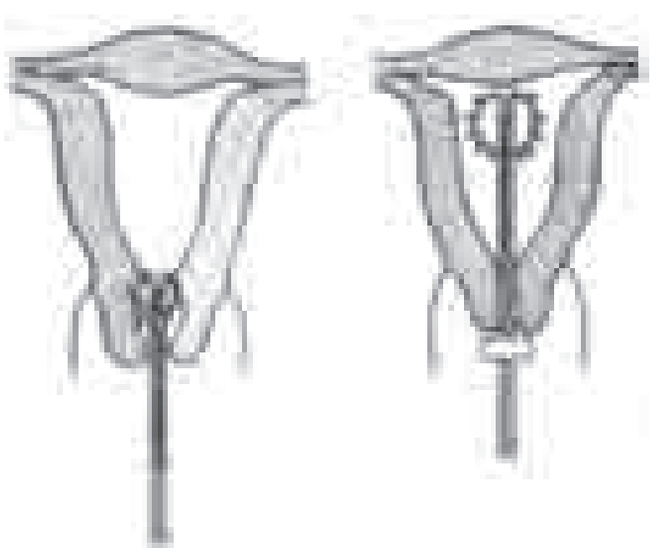
- Permanent Methods in Female
  - Tubal ligation
  - Mini Laparotomy
  - Laparoscopic-sialistic ring/diathermic cauterisation

Male contraception

Temporary

1. Gossypol: 10 to20mg/day for 3 months then 20mg twice weekly Other drugs:
   - Testosterone enanthate

Technique of Insertion of Multiload IUD's



* GnRH
* MDPA/desogesteron

All cause suppression of spermatogenesis

Permanent Methods

- Vasectomy

Bleeding in early pregnancy

Miscarriage (abortion) is defined as the expulsion of the products of conception (either spontaneously or assisted) before the period of viability. According to WHO, the expelled fetus should weigh 500 gms or less.

Types of Miscarriage

- Spontaneous
- Induced

Spontaneous miscarriage

- Incidence
  * 15% - 20% of all pregnancies result in spontaneous miscarriage
  * First trimester miscarriage is more common than the second trimester miscarriage

Aetiology

- Idiopathic
- Genetic–chromosomal abnormality
- Blighted ovum
- Multiple pregnancy
- Uterine anomalies
- Endocrinological causes
  * Hypothyroidism
  * Uncontrolled diabetes
  * PCOD – due to luteal phase defect.
- Infections
  * TORCH complex
  * Hyperpyrexia due to any infection

Types of Spontaneous miscarriage

- Threatened miscarriage
  * H/o Period of amenorrhoea
  * Bleeding per -vaginum (slight or more)
  * Pain abdomen
  * Expulsion of fleshy mass
- Per- vaginal examination
  * Uterus corresponds to the period of pregnancy
  * Os closed
- USG
  * Viable intra-utierine pregnancy with detectable fetal cardiac activity
  * If fetal heart activity is detected, there is a 97% chance of the pregnancy continuing

Rx Treatment

- Rest
- Progesterone or HCG supplementation are not justified.
- Rh-Anti D injection to be given if the patient is Rh -ve

Haemorrhage or bleeding in early pregnancy
Cervical Abortion

Inevitable miscarriage features

- History of period of amenorrhoea (usually up to 14 weeks)
- Bleeding PV usually heavy
- Pain abdomen due to expulsion

On examination

- Uterus smaller for the period of amenorrhea
- Os open with products partially protruding through the os
- If left alone complete expulsion takes place in 80% of cases in 3 days
- Can be confirmed by USG which shows a normal endometrial echo.
- If bleeding is profuse there are 2 options
  - * Medical
    - » If pregnancy is < 9 weeks, by giving 400µg of Misoprostol PV 2 doses, 4-6 hours apart
    - » If more than 12-14 weeks - Syntocinon drip.
  - * Surgical
    - » Suction or digital evacuation followed by Inj.Methergine.
  - * Conservative management is possible if the gestation sac is <50 mm
  - * If surgical procedure is done – antibiotics to be given
  - * Inj.Rh-Anti D to be given if Rh –ve mother

Complete abortion

- Usually follows inevitable abortion
- Bleeding stops
- Per vaginal examination
  - * Uterus almost normal size
  - * Os closed
  - * No bleeding
- USG
  - * Uterus is normal
  - * No gestational sac
  - * Normal uterine cavity echo is seen
  - * Os closed
- If slight bleeding is present, Inj. Methergine can be given
- Rh-Anti-D to be given for a Rh-negative mother.

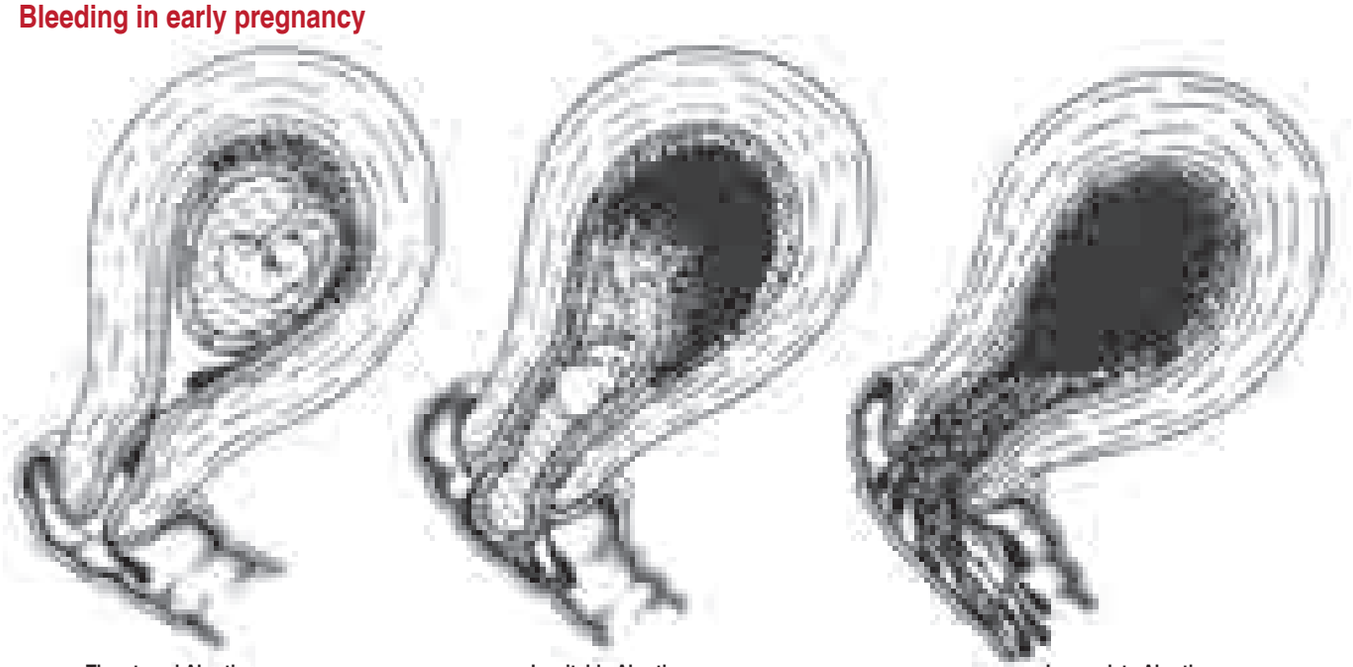
Blighted ovum

- Embryo has stopped developing
- Gestational sac still remains
- Empty sac > 20 mm diameter on USG
- To be managed as missed miscarriage.

Missed miscarriage

Retention of the products of conception after the

Bleeding in early pregnancy



Threatened Abortion | Inevitable Abortion | Incomplete Abortion

death of the fetus or embryo after an initial attempt to expel.

Clinical findings

- Initial spotting or slight bleeding
- Regression of early signs and symptoms of early pregnancy, i.e., relief from vomiting.
- On examination, uterus is small for the period of gestation.
- Os closed
- No bleeding or brownish discharge.
- USG
  - * Embryo of 6 weeks size with no demonstrable fetal cardiac activity.
  - * Same features when the scan is repeated after 1 week.
  - * Irregular gestational sac.

Treatment

- Check clotting time and bleeding time
- Medical
  - * If less than 9 weeks, Mifepristone followed by Vaginal Misoprostol 400µg 6th hrly x 3 doses
  - * Check by USG for completion after 1 week.
- Surgical
  - * Dilatation and surgical evaluation

Septic abortion

- May be due to criminal interference
- Incomplete/missed abortion with infection

Clinical findings

- History of interference
- Fever with chills
- Foul smelling discharge
- Tenderness on examination

Note:

Refer the patient to higher centre after an initial dose of intravenous antibiotic and antipyretic

Recurrent miscarriage

- Incidence < 1%
- 3 or more consecutive pregnancy wastages
- May be first or second trimester
- Refer to higher centres

Ectopic Pregnancy

Definition

Any pregnancy occurring outside the uterine cavity is termed as ectopic pregnancy

Incidence

Common in women in the age group of 25-34 years.

Sites

- Tubes–commonest site
- Ovaries
- Cervical
- Abdominal

Tubal Pregnancy (95%)

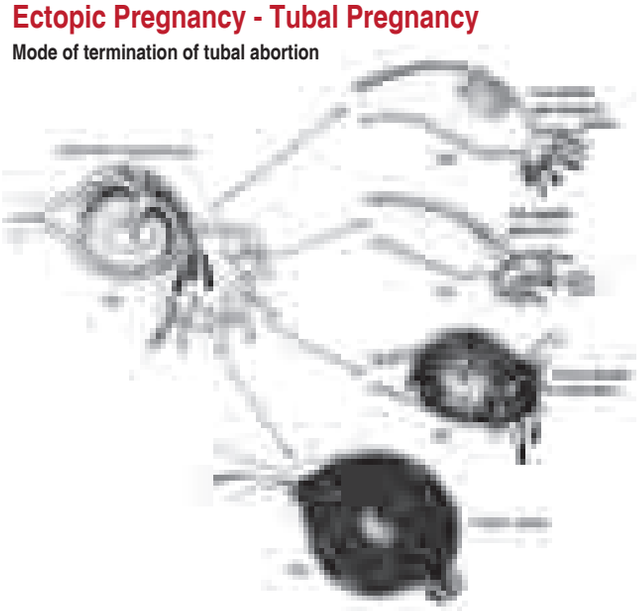
Risk factors include:

- A history of infertility
- Pelvic inflammatory disease
- Pelvic operations (particularly tubal surgery)
- Previous tubal pregnancy
- Assisted conception (particularly IVF if tubes are patent and damaged)
- Presence of an intrauterine device.

Chlamydial and gonococcal tubal infections causes

Ectopic Pregnancy - Tubal Pregnancy

Mode of termination of tubal abortion



tubal block and may give rise to ectopic pregnancy.

Symptoms and signs

- Amenorrhoea of about 6 to 8 weeks except in cornual and abdominal pregnancy
- Unilateral lower abdominal pain followed by spotting or vaginal bleeding
- Shoulder tip pain
- The uterus may be slightly enlarged with cervical excitation and tenderness
- Tender mass may be palpable to one side of the uterus.
- Un-ruptured pulsatile mass if felt per vaginally

Diagnosis

- Ectopic should be ruled out in all suspected cases
- The diagnosis is missed in nearly 25% of the cases on clinical grounds.
- Even when it is made, the presentation-to-treatment interval is over 48 hours in 40-50%, and over 1 week in 20-25%
- When a woman of reproductive age presents with unexplained abdominal pain with or without vaginal bleeding, ectopic should be strongly suspected and immediately refer to a higher centre

Differential diagnosis

- Threatened or incomplete miscarriage
- Rupture or torsion of ovarian cyst
- Pelvic inflammation – bilateral involvement, USG can differentiate each condition.
- Bleeding corpus luteum – laparoscopy / laparotomy

Investigations

- For women in clinically stable conditions
- If a urine test is positive and an ectopic pregnancy is suspected, carry out a serum assay.
  - When the β-HCG value is < 1500 IU/L and if there is an empty uterine cavity, ectopic pregnancy is more likely.
  - Failure to double the value of β-HCG by 48 hours along with an empty uterus is very much suggestive.
- Laparoscopy
  - The gold standard for diagnosis if index of suspicion is high. Very early tubal pregnancies (3-4% of total) can still be missed at laparoscopy.

Treatment guidelines

All suspected cases of ruptured ectopic should be admitted.

- If the patient is shocked, resuscitate
- Arrange for blood transfusion, if required.
- The priorities are to stop haemorrhage and prevent further bleeding
- Conservative surgery is less likely to be possible under such circumstances
- If facilities are not available, arrange for immediate transfer to higher centre.
- Start two intravenous lines. Haemaccel infusion can be started.

Conservative management

- Not possible in primary health centre
- Refer to higher centre
- Admission
- Monitor vitals
- Evaluate by USG
- Collect blood for β-HCG
- Keep emergency surgical facilities ready

Medical management

Methotrexate as intramuscular injection with suitable precautions.

Surgical treatment

- Laparotomy
  - Indicated in emergencies where people are not trained in laparoscopy
  - Salphingectomy in ruptured ectopic
  - Salphingostomy in un-ruptured ectopic
- Laparoscopy
  - Salphingectomy
  - Salphingostomy
  - Inj. Methotrexate into the sac

Prognosis

Explain that recurrence of tubal pregnancy is 12%. As the maternal mortality is very high in improperly managed ectopic pregnancy, i a high degree of suspicion, early diagnosis and referral is mandatory.

Medical termination of pregnancy

Medical Termination of Pregnancy (MTP act) was passed by the parliament in 1971, and came into force on 1st April 1972. Worldwide, approximately 40 million legal abortions, and about 10 to 22 million illegal abortions take place every year. In India alone 10-12 million abortions take place annually, resulting in 15-20 thousand maternal deaths, mainly due to illegal abortion.

The MTP Act

Grounds for Performing MTP

- Medical Grounds
- Eugenic Grounds
- Humanitarian Grounds
- Social Grounds
- Failure of contraceptive method

The Persons who can perform the MTP

A registered medical practitioner with post graduate training or qualification in OandG, or trained at a specialized and recognized training centre for MTP.

* First Trimester MTP–Single registered medical practitioner
* Second Trimester MTP – Two registered medical practitioners

The Place for Performing MTP

1. A hospital established and maintained by the government.
2. A place recognized and approved by the government, under this act.

Counselling

Both supportive and contraceptive counseling should be given.

Consent

The written and informed consent of the patient on a specially prescribed form should be obtained. The consent of the legal guardian must be obtained if the women are under the age of 18 years or when lunatic, even if she is older than 18 years.

Clinical Assessment

One must obtain a thorough history and perform complete general, systemic and gynecologic examination for :

- Confirmation of pregnancy
- Assessment of exact period of gestation.
- Associated gynecological disorders and inflation
- Associated medical diseases.

Investigation

- Hemoglobin Level
- Urine for Albumin and Sugar

Others are

- Blood grouping and Rh typing
- Blood sugar, urea and creatinine.
- X-ray chest
- ECG on all leads
- HIV and HbsAg screening

Specific Investigation

USG: to rule out extra-uterine pregnancy/ uterine and or fetal malformations.

Methods of MTP

Trimester	Medical	Surgical
Ist Trimester	The protocol is as follows Day 1: 600 micro grams (3 tablets) of misoprostol given as a single dose – the woman is observed for half an hour and then allowed home. Day 3: 400 micro grams of oral misoprostol (prostaglandin) is administered. Day 14: Follow up to confirm abortion has occurred, if not surgical MTP is done.	• Menstrual Regulation (upto 42 days of LMP) • Suction Evacuation • Manual Vacuum Aspiration (MVA) • Dilatation and Evacuation
2nd Trimester	• Extra ovular instillation of drugs • Intracervical or Extraovular instillation of Prostaglandin gel (PGE2) • Mifepristone and Misoprostol • Prostaglandin F2 alpha (250 mg) can be given IM every 3 hours for a maximum of 10 doses.	Dilatation and evacuation in cases of incomplete expulsion

Post-Operative Care

Instructions on discharge

1. Normal recovery and resumption of activity. (normal menstrual period could be expected within 4 to 6 weeks)
2. Contraceptive counseling
3. Prescribe Medication (Iron and Folic acid tablets for one month)

Follow Up

- The women is instructed to report immediately to the centre if she experiences any of the following
- Bleeding more than normal menstruation
- Severe abdominal pain, distension and vomiting
- Fever, chill or malaise
- Foul smelling discharge
- Routine visit at the centre after 2 weeks, during which an internal examination is performed to rule out continuation of pregnancy, incomplete abortion and pelvic infection

Complications

Symptoms

Immediate	Delayed and Remote
I Complications of local anesthesia • Systemic toxic reaction • Hypersensitivity and allergic reaction **II Complications during MTP Procedure** • Excessive bleeding • Perforation • Shock	**Delayed complications** • Incomplete evacuation • Continuation of pregnancy • Infection **Remote Complications** • PID • Chronic pelvic pain • Ashermann's syndrome • Adherent placenta in future pregnancies • Psychosomatic symptoms • Prolonged Pregnancy

Management of bleeding

- Check curettage
- Intravenous Ergometrine 0.25 – 0.5 mg
- Oxytocin infusion
- Bimanual massage of uterus for 2 minutes

Refer to higher centre if bleeding persist

Management of perforation

- Stop the procedure as soon as perforation is suspected
- Start intravenous infusion
- Shift the patient to higher centre. Prolonged Pregnancy
- Do not attempt to replace the omentum or intestinal loop, if it has herniated through the perforation.

Anaemia In Pregnancy

In India the prevalence of anaemia in pregnancy is 80% and that of severe anaemia is 10-15%

Causes

Causes of anaemia in pregnancy are the same as those encountered in the non-pregnant state. However, iron deficiency anaemia is the commonest type of anaemia in pregnancy. In about 40-50% of cases, there is an associated folic acid deficiency.

Definition

- Anaemia in pregnancy is defined as hemoglobin concentration of less than 11 g/dl and haematocrit of less than 33
- It is further classified depending on hemoglobin levels as
  - Mild 10-11 g%
  - Moderate7-10 g% and
  - Severe < 7 g%

Symptoms

- In mild to moderate cases
  - Weakness
  - Exhaustion
  - Lassitude
  - Anorexia
  - Glossitis
  - Stomatitis
- In severe cases
  - Palpitations
  - Dyspnoea
  - Oedema
  - Cardiac failure

Investigations

- Haemoglobin
- Haematocrit
- Total RBC count
- Peripheral smear (to identify the type of anemia)
- Haematological indices
- Plasma proteins
- Stools for ova and cyst

Treatment

Iron deficiency anaemia

All cases of severe anaemia need to be admitted especially those with features of anoxia or cardiac failure.

Non - pharmacological

- Diet rich in iron-jaggery, green leafy vegetable, sprouted pulses, meat, cooking food in iron utensils.
- Diet rich in protein-pulses, lentils, milk and milk products, nuts.

Pharmacological

- Oral iron therapy
  - Ferrous -sulfate and Ferrous fumarate
  - Recommended dose is 200 mg elemental iron daily in divided doses
  - Not to be taken with meals, milk, coffee or tea.
  - Continue therapy till blood picture returns to normal and then continue with 100 mg elemental iron for 3 months to build up the stores
  - Government of India recommends minimum of 100 mg of elemental iron and 5 mg folic acid for 100 days starting at 20 weeks (common side effects are epigastric pain, nausea, vomiting, constipation, and diarrhoea)

- De-worming to be done after first trimester, if necessary
  - * Tab. Mebendazole 100 mg 2 times a day for 3 days. (or) Tab. Albendazole 400 mg as single dose.

Monitoring of response to therapy

Subjective improvement of feeling better, weight gain and improved appetite after 1-2 weeks. reticulocyte response observed in 5-10 days (increases to 5-6%) and rise in Hb/ hemato-crit in 2-3 weeks

If no improvement in 3 weeks, re-evaluate for incorrect diagnosis, non-compliance,defective absorption, continuing loss, associated deficiencies.

Role of parenteral therapy is limited as rate of rise of haemoglobin with parenteral iron, is similar to oral iron preparation.

Specific indications

- Severe intolerance to oral iron
- Mal-absorption
- Non-compliance and
- Moderate to severe anaemia in advanced pregnancy.

Total dose of iron to be given is calculated using the following formulae

% deficiency of Hb x weight in 1bs x 0.03+300mg.

(or)

- Simple method
  - * 250mg of elemental iron needed for each gram of Hb deficit.

- Caution
  - * Emergency drugs to be kept Ready for resuscitation in case of anaphylactic reaction
- Intramuscular
  - * 100 microgram in alternate buttocks daily

Indications for blood transfusion:

Severe blood loss, severe anemia beyond 36 weeks of pregnancy or anaemia refractory to oral and parentral therapy or anaemic patient with anoxia or cardiac failure.

- Management of anaemic patients during labour
  - * Propped up position, oxygen therapy
  - * Sedation and pain relief
  - * Digitalization may be required in cardiac failure due to severe anaemia
  - * Cut short 2nd stage using forceps
  - * Active management of 3rd stage of labour with Inj. Methylergometrine maleate 0.2mg IV at the delivery of anterior shoulder. Inj. Methylergometrine to be avoided in patients of anaemia with cardiac failure.
  - * Packed cell transfusion if necessary and if Hb %<5g after giving diuretics.

Megaloblastic anaemia in pregnancy

Folic acid and Vitamin B12 deficiency

Symptoms

Patients may be asymptomatic or may have vomiting, diarrhoea, pallor, yellow colour of the skin, hepatosplenomegaly, and polyneuropathy.

Investigations

Diagnosis is by MCV>96fl, MCH > 33pg and MCHC normal. peripheral smear macrocytic anaemia with hyper segmentation of neutrophils, “Howell -jolly” bodies, neutropenia and thrombocytopenia

Treatment

- Tab. Folic acid 5mg daily to be continued for atleast 4 weeks in puerperium.
- Inj. Cyanocobalamin 250mcg IM every month.

Dimorphic Anaemia

Treatment

Both iron and folic acid in therapeutic doses.

Patient eductaion

- Dietary advice-milk, green leafy vegetables, egg and fruits.
- Common side effects of therapy should be explained to the patient
- Explain to the patient that stools turn black after oral iron therapy, so no need for concern.
- Iron supplementaion should continue for at least 3 months in postpartum period.

- Adequate spacing of at least 3 years between two pregnancies.

References

- Hematological disorders. In: William's Obstetrics, Eds Cunningham HG, Gant NF, Loveno KJ at 21st Edition, 2001, Mcgraw Hill Company Inc., PP1307-1323.
- Hematogical Problems during pregnancy. In: practical Guide to High Risk Pregnancy and Delivery, Fernando Arias (ed),2nd Edition, 1192, Harcourt Asia Pvt Ltd., PP245-642.
- Anaemia and Pulmonary Tuberculosis. In: Practical Obstetric Problems. Ian Donald (ed)5th Edition,1998, B1 publications, pp 198-226.
- Medical and Surgical illness Complicating Pregnancy. In: Textbook f Obstetrics, DC Dutta (ed), 4th Edition, 1998, New Centra Book Agency (P) Ltd., PP 277-292.
- Haematinics and Erythropoietin. In: Essential of Medical Pharmacology, KD Tripathi (ed), 4th Edition, 1999, Jaypee Brothers Medical Publisher Pvt Ltd., PP 580-595.

Hypertensive disorders of Pregnancy

Hypertensive disorders in pregnancy include

- Pregnancy induced hypertension (hypertension with no proteinuria)
- Pre-eclampsia (hypertension with proteinuria)
- Eclampsia (pre-eclampsia with superadded convulsions)
- Chronic hypertension (hypertension antedates pregnancy and persisting post-partum)
- Chronic hypertension with superadded pre-eclampsia or eclampsia.

  Pre-eclampsia arises after the 20th week of gestation, characterized by hypertension and proteinuria. Oedema may also be present.

PIH is defined as

- A Blood pressure of 140/90 mm of Hg or more
- An increase in the systolic pressure by 30 mm of Hg or more
- An increase in the diastolic pressure of 15 mm of Hg or more

Proteinuria

Proteinuria is defined as a protein concentration of 0.3 g /L or more in at least two random urine samples collected at 6 or more hours apart.

Oedema

An excessive weight gain of 1 kg or more in a week (or 3 kg in a month) is indicative of pre-eclampsia (the normal weight gain is about 0.5 kg per week, or 2 kg in a month). Oedema in a case of pre-eclampsia seen in

- The front of the legs or dorsum of the foot and over the ankles
- Hands / fingers
- Face, eyelids
- Abdominal wall
- Sacral areas and vulva

Classification of Pre-eclampsia		
Findings	Mild Pre-eclampsia	Severe Pre-eclampsia
Blood Pressure	The diastolic pressure rises 15-20 mm of Hg above the usual level or the absolute level of Blood pressure is >140/90 mm of Hg but <160/110 mm Hg	The diastolic pressure rises >20 mm of Hg above the "usual level or the absolute level of Blood pressure is >160/110 mm of Hg.
Proteinuria	Present, but 2+ or less	3+ or persistently greater
Generalized oedema (including the face and hands)	May or may not be present	Present
Imminent signs Headache	Absent	Present
Visual disturbances	Absent	Present
Upper abdominal pain	Absent	Present
Oliguria	Absent	Present
Diminished foetal movement	Absent	Present

Eclampsia

It is characterized by convulsions/fits followed by more or less prolonged coma. The woman usually has hypertension and proteinuria. The convulsions may occur in the antepartum, intrapartum or the postpartum period.

Imminent eclampsia

Imminent eclampsia means that an eclamptic fit is likely to occur very soon.

Symptoms of imminent eclampsia	Signs of Imminent eclampsia
• Severe headache • Drowsiness • Mental confusion • Visual disturbances • Epigastric pain • Nausea, vomiting • Decreased urinary output	• A sharp rise in the blood pressure • Increased proteinuria • Exaggerated knee jerk

The most common causes of maternal death in eclampsia are aspiration of vomitus, kidney failure, intracerebral haemorrhage, and multi-organ failure (e.g. heart + liver + kidneys).

Pre-disposing condition

- Primigravidas (especially young teenagers and women over the age of 35 years)
- Obese women
- Women with essential hypertension
- Women with multiple pregnancy
- Women with diabetes, hydatidiform mole, polyhydramnios
- Women with a h/o pre-eclampsia or eclampsia in a previous pregnancy
- Women with a family history of eclampsia.

Treatment guidelines at PHC level

- Monitor the blood pressure and urine for proteins during prenatal care
- Counsel the women and her family about the dangerous symptoms of pre-eclampsia and the importance of prenatal care.
- Early referral of a woman with eclampsia
- Knowledge of management strategy
- Proper equipment and drugs made available
- Carry out timely management of complications arising due to eclampsia.

Eclampsia	
Effects on the mother	Effects on the foetus
• Respiratory * Asphyxia, aspiration of vomitus, pulmonary oedema, bronchopneumonia) • Cardiac * Heart failure • Renal * Acute renal failure • Hepatic * Liver necrosis • HELLP syndrome * Haemolysis * Elevated liver enzymes * Low platelet count) • Haemorrhage * Due to coagulation defect, i.e. disseminated intravascular coagulopathy (DIC), which is often associated with eclampsia. • Visual problems * Temporary blindness due to oedema of the retina • Injuries * Fractures * Tongue bite	• Hypoxia * This may lead to permanent brain damage, which may result inS • Physical handicap • Cerebral palsy • Mental retardation • IUGR (Intra- Uterine Growth Retardation) • Still birth

Diagnosis of hypertension during pregnancy

The hypertensive disorders of pregnancy include chronic hypertension (elevation of the Blood pressure before 20 weeks of gestation) and PIH.

Differential diagnosis of Hypertensive dSisorders of Pregnancy	
Symptoms and signs	Probable diagnosis
Blood pressure 140/90 mm of Hg or more before the first 20 weeks of gestation	Chronic hypertension
Blood pressure 140/90 mm of Hg or more before 20 weeks of gestation + Proteinuria	Chronic hypertension with superimposed pre-eclampsia.
Two readings of Blood pressure 140/90 mm of Hg or more taken at least 4 hours apart, after 20 weeks of gestation + Proteinuria	Pregnancy-induced hypertension (PIH)

Pre Eclampsia and Eclampsia

Pregnancy Induced Hypertension

Woman with PIH disorder may progress from mild disease to a more serious condition.

Definition

It is defined when systolic blood pressure is >140 mm of Hg or diastolic blood pressure is >90 mm of Hg recorded on two occasions 4 hours apart or a single recording of diastolic blood pressure >110 mm Hg.

Pre eclampsia

If the above is seen with proteinuria, it is called Pre eclampsia. Peripheral edema not included in this.

Classification

- Hypertension without proteinuria or edema.
- Mild / Severe / Eclampsia
- Chronic hypertension with super imposed mild pre-eclampsia
- Chronic hypertension

Remember

- Mild pre-eclampsia often has no symptoms
- Increasing proteinuria is a sign of worsening pre-eclampsia.

- A small proportion of woman with eclampsia have normal blood pressure
- Mild pre-eclampsia may progress rapidly to severe pre-eclampsia. The risk of complication including eclampsia increases greatly in severe pre-eclampsia.

Investigations

- Urine - Albumin, sugar
- Urine – Culture and sensitivity
- Urine – 24 hr Urinary protein
- HG %,PCV%
- Platelets, BT, CT, Serum fibrinogen
- Look for any coagulopathy.
- 75 gms GTT
- Blood Urea, Sr.Creatinine, Sr.Uric acid
- Sr.Bilirubin, Sr.Proteins, SGOT,SGPT,SAP
- Ophthalmologist opinion, regarding status of fundus

Management of Pre-eclampsia

- Monitor blood pressure,Urine albuminuria and fetal condition weekly
- If there are signs of severe fetal growth restriction or fetal compromise, admit the woman for assessment and possible expedient delivery.
- Counsel the woman and her family about danger signal indications of pre-eclampsia or eclampsia.
- If the observation remains stable allow to proceed with normal labour and child birth.

Mild Pre-eclampsia

Gestational age <37 Weeks

- If pre-eclampsia -signs remain unchanged follow up twice a week as an outpatient.
  - Monitor Blood pressure
  - Urine proteinuria
  - Reflexes
  - Fetal condition
  - Give adequate rest
  - Encourage normal diet
  - Start anti-hypertensives if diastolic blood pressure goes >100 mm Hg
- If blood pressure or Albuminuria raises, admit the patient
  - Give normal diet
  - Monitor blood pressure/urine proteinuria,
  - Give anti-hypertensives for blood pressure,
  - Monitor fetal well being by USG for serial growth,liquor status, Doppler study of umbilical artery
- When diastolic blood pressure decreases to normal and biophysical profile is normal, send the patient home with the advice of
  - Normal diet,plenty of water
  - Adequate rest
  - Continue anti-hypertensives
  - To check blood pressure, urine albumin twice weekly
  - To report back if blood pressure or urine albumin raises or imminent symptoms reveals
- When diastolic blood pressure remains stable
  - Keep the woman in hospital
  - Monitor fetal growth symphysis-fundal height
  - If growth restriction,deliver her
  - If not continue until term
- When blood pressure or urine albumin continues to raise, manage as severe Pre-eclampsia

Gestational age >37 Weeks

- If there are sign of fetal compromise, assess the cervix and expedite delivery.
- If the cervix is favourable, (soft,thin,partly dilated),rupture the membrane and induce by Oxytocin.
- If the cervix is unfavourable ,ripen the cervix with Cervi-prime or deliver by Caesarean section.

Severe Pre-eclampsia and Eclampsia

- Both are managed with the exception that delivery must occur within 12 hOUrs of onset of convulsions in eclampsia.
- All cases of severe pre-eclampsia should be managed actively.
- Symptoms and signs of “impending eclampsia” (blurred vision, hyperreflexia) are unreliable and expectant management is not recommended.

Management during a convulsion

- Give anticonvulsive drugs
- Gather equipment (airway, suction, bag and mask,oxygen) and give oxygen at 4-6 l/minute
- Protect the woman from injury but do not actively restrain her.
- Place the woman on her left side to reduce risk of aspiration of secretions,vomit and blood.
- After the convulsion aspirate the mouth and throat as necessary.

General management

- If diastolic blood pressure remains above 110 mm Hg,give antihypertensive drugs.
- Reduce the diastolic blood pressure to less than 100 mm Hg but not below 90 mm Hg.
  - * Start an IV line and infuse fluids.
  - * Maintain strict fluid balance chart.
  - * Monitor the amount of fluids administered and urine output to ensure that there is no fluid overload.
  - * Catheterize the bladder to monitor urine output and proteinuria.
- If urine output is less than 30 ml per hour:
  - * Withhold MgSo4 and infuse IV fluids(NS/RL) at 1 L in 8 hours.
  - * Monitor for the development of pulmonary edema.
- Never leave the woman alone. A convulsion followed by aspiration of vomit may cause death of the woman and fetus.
- Observe vital signs,reflexes and fetal heart rate hourly.
- Auscultate the lung bases hourly for rales indicating pulmonary edema.
  - * If rales are heard withhold fluids and give frusemide 40 mg IV once.
- Assess clotting status with a bedside clotting test.
  - * Failure of a clot to form after 7 minuted or a soft clot that breakdown easily suggests coagulopathy.

Anticonvulsive drugs

- A key factor in anti-convulsive therapy is adequate administration of Anti-convulsive drugs (ACD)
- Convulsions in hospitalized women are most frequently caused by “under-treatment”
- Magnesium sulphate (MgSo4) is the drug of choice for preventing and treating convulsions in severe pre-eclampsia and eclampsia.
- If MgSo4 is not available, Diazepam may be used, although there is a greater risk for neonatal respiratory depression because, Diazepam passes the placenta freely.
- A single dose of diazepam to abort a convulsion seldom causes neonatal respiratory depression.
- Long-term continuous intravenous administration, increases the risk of respiratory depression in babies, who may already be suffering from the effects of utero-placental ischaemia and preterm birth.
- The effect may last several days.

Schedules for severe Pre-eclampsia and Eclampsia

Loading Dose

- MgSo4 20% solution, 4 gm IV over 5 minutes.
- Follow promptly with 8 g of 50% MgSo4 solution, 4 g in each buttock as deep intramuscular injection with 1 ml of 2% Lignocaine in the same syringe.
- Ensure that aseptic technique is practiced when giving MgSo4 deep IM inj.
- Warn the woman that a feeling of warmth will be felt when MgSo4 is given.
- If convulsions recur after 15 minutes , give 2 g MgSo4 50% solution intravenous over 5 minutes

Maintenance Dose

- 4 g MgSo4 50% soln + 1 ml Lignocaine 2% IM every 4 hours into alternate buttocks.
- Continue treatment with MgSo4 for 24 hours after delivery or the last convulsion,whichever occurs last.

Before repeat administration, ensure that

- Respiratory rate is atleast 16 per minute.
- Patellar reflexes are present.

- Urinary output is atleast 30 ml per hour over 4 hours.

Withhold or delay drug if

- Respiratory rate falls below 16 per minute.
- Patellar reflexes are absent.
- Urinary output falls below 30 ml per hour over proceeding 4 hours.

Keep Antidote ready

- In case of respiratory arrest
  - * Assist ventilation (mask and bag,anaesthesia apparatus,intubation).
  - * Give injection Calcium gluconate 1 g (10 ml of 10% soln)IV slowly until respiration begins to antagonize the effects of MgSo4.

Antihypertensive Drugs

If the diastolic pressure is 110 mm Hg or more,give anti-hypertensives. The goal is to keep the diastolic pressure between 90 mm Hg and 100 mm Hg to prevent cerebral haemorrhage.

Hydralazine is the drug of choice.

- Give Hydralazine 5 mg slow intra-venously every 5 minutes until blood pressure is lowered. Repeat hourly as needed or give Hydralazine 12.5 mg IM every 2 hours as needed.
- If Hydralazine is not available,give Labetolol 10 mg IV:
  - * If response is inadequate(diastolic Blood pressure remains above 110 mm Hg)after 10 min , give Labetolol 20 mg IV
  - * Increase the dose to 40 mg and then 80 mg if satisfactory response is not obtained after 10 min of each dose. or Nifedipine 5 mg under the tongue.
  - * If response is inadequate (diastolic blood pressure remains above 110 mm Hg) after 10 min ,give an additional 5 mg under the tongue.

Note:

There is a concern regarding a possibility for an interaction with MgSo4 that can lead to hypotension.

Delivery

- Delivery should take place as soon as the woman's condition has stabilized.
- Delaying delivery to increase fetal maturity will risk the lives of both the woman and the fetus.
- Delivery should occur regardless of the gestational age.
- In severe pre-eclampsia, delivery should occur within 24 hours of the onset of symptoms.
- In eclampsia, delivery should occur within 12 hours of the onset of convulsions.
  - * Assess the cervix.
  - * If the cervix is favourable(soft,thin,partly dilated), rupture the membranes with an amniotic hook or a Kocher's clamp and induce labour using oxytocin or prostaglandins.
  - * If vaginal delivery is not anticipated within 12 hours (for eclampsia) or 24 hours (for severe pre-eclampsia), deliver by caesarean section.
  - * If there are fetal heart rate abnormalities (less than 100 or more than 180 beats per minute), deliver by caesarean section.
  - * If the cervix is unfavourable(firm,thick,closed) and the fetus is alive , deliver by caesarean section.
  - * If safe anaesthesia is not available for caesarean section or if the fetus is dead or too premature for survival
    - » Aim for vaginal delivery.
    - » If the cervix is unfavourable(firm,thick,closed), ripen the cervix using Misoprostol, Prostaglandins or a Foley catheter.

Note:

- If caesarean section is performed,ensure that:
  - * Coagulopathy has been ruled out.
  - * Safe General anaesthesia (GA) is available
  - * Spinal anaesthesia is associated with the risk of hypotension.This risk can be reduced if adequate IV fluids (500-1000 ml)are infused prior to administration of anaesthetic.
  - * Do not use local anaesthesia or ketamine in women with pre-eclampsia or eclampsia

Postpartum care

- Anti-convulsive therapy should be maintained for 24 hours after delivery or the last convulsion,whichever occurs last.

- Continue anti-HT as long as the diastolic pressure is 110 mm Hg or more.
- Continue to monitor urine output.

Referral for tertiary level care

Consider referral of women who have

- Oliguria that persists for 48 hours after delivery.
- Coagulation failure (e.g.Coagulopathy or haemolysis,elevated liver enzymes and low platelets (HELLP Syndrome).
- Persistelt coma lasting more than 24 hours after convulsion.

Complications of Pregnancy induced hypertension

- Complications may cause adverse perinatal and maternal outcomes.
- Prevent them by early diagnosis and proper management.
- Manage complications as follows
  - * If fetal growth restriction is severe, expedite delivery.
  - * If there is increasing drowsiness or coma, suspect Cerebral haemorrhage.
    - » Reduce blood pressure slowly to reduce the risk of cerebral haemorrhage.
    - » Provide supportive therapy.
  - * If heart,kidney or liver failure is suspected, provide supportive therapy and observe.
  - * If a clotting test shows failure of a clot to form after 7 minutes or a soft clot that breaks down easily, suspect coagulopathy.
  - * If the woman has IV lines and catheters,she is prone to infection. Use proper infection prevention techniques and closely monitor for signs of infn.
  - * If the woman is receiving IV fluids,she is at risk of circulatory overload. Maintain a strict fluid balance chart and monitor the amt of fluids administered and urine output.

Chronic hypertension

- Encourage additional periods of rest.
- High levels of blood pressure maintain renal and placental perfusion in chronic hypertension.Reducing blood pressure will result in diminished perfusion. Blood pressure should not be lowered below its pre-pregnancy level. There is no evidence that aggressive treatment to lower the blood pressure to normal levels improves either fetal or maternal outcome:
  - * If the woman was on anti-hypertensive medication before pregnancy and the disease is well-controlled,continue the same medication if acceptable in pregnancy.
  - * If diastolic blood pressure is 110 mm Hg or more, or systolic blood pressure is 160 mm Hg or more,treat with anti-hypertensive drugs.
  - * If proteinuria or other signs and symptoms are present,consider superimposed pre-eclampsia and manage as mild pre-eclampsia.
- Monitor fetal growth and condition.
- If there are no complications,deliver at term.
- If pre-eclampsia develops ,manage as mild pre-eclampsia or severe pre-eclampsia.
- If there are fetal heart rate abnormalities(less than 100 or more than 180 beats per minute),suspect fetal distress.
- If fetal growth restriction is severe and expected date of pregnancy is accurate, assess the cervix and consider delivery.

Note

Assessment of Gestation by ultrasound in late pregnancy is not accurate.

- If the cervix is favourable (soft,thin, partly dilated),rupture the membranes with an amniotic hook or a Kocher's clamp and induce labour using oxytocin or prostaglandins.

4. If the cervix is unfavourable(firm ,thick, closed), ripen the cervix using prostaglandins or a Foley's catheter.
   - * Observe for complications including abruptio- placentae and superimposed pre-eclampsia.

Heart disease complicating Pregnancy

Incidence: 1% among pregnant women Types Acquired

- Rheumatic heart disease
  - * Most common type of heart disease in India contributing to 90-95% cases. Commonest le-

sion – 'Mitral Stenosis

* Mitral regurgitation
  » Aortic Stenosis and
  » Aortic regurgitation

- Pre-disposing factors: Low socio-economic Status, over crowding.
- Congenital – ASD, VSD, PDA, PS, 5% cyanotic lesions – Fallots tetrology, Eisen-mengers syndrome.
- Other Cardiac lesions: Mitral valve prolapse, peripartum cardiomyopathy, arrhythmias, myocardial infarction.

Cardiovascular systems changes during pregnancy

- During pregnancy, labour, delivery and puerperium heart rate increases in response to physical stress.
- Rule of five
  * At 5 weeks
  * At 5 months
  * At 5 weeks before delivery
  * At 5 hours after the onset of labour
  * At 5 minutes after delivery

Cardiovascular systems changes during pregnancy

Parameter	Modification	Magnitude	Peak
Cardiac output	Increasing	30-50%	28-32 weeks
Heart rate	Increasing	10-15 beats	Between 14-20 weeks maintained till term
Plasma volume	Increasing	40-45%	32 weeks
RBC volume	Increasing	15-20%	30-32 weeks
Resistance changes systemic circulation	Decreasing	20%	16-24 weeks
Pulmonary circulation	Decreasing	34%	34 weeks
Systolic blood pressure	Decreasing	9%	
Diastolic blood pressure	Decreasing	Slightly more on diastolic	28 weeks
oxygen consumption	Increasing	+20%	Term

Clinical indicators of Heart disease during pregnancy

- Symptoms:
  * Progressive dyspnoea
  * Orthopnoea
  * Nocturnal cough
  * Hemoptysis
  * Syncope
  * Chest pain
- Signs
  * Tachycardia
  * Tachypnoea
  * Cyanosis
  * Clubbing
  * Persistent neck vein distension

* Systolic murmur of grade 3/6 or greater
* Diastolic thrill and murmur
* Rales, Wheeze
* Persistent arrhythmia
* Persistent split second sound

- ECG
- Echo
  * Diagnostic for valvular lesion

New york heart association (NYHA) classification of Heart disease.

- Class I: Uncompromised – No limitation of physical activities.
- Class II: Slight limitation of physical activity.
- Class III: Marked limitation of physical activity
- Class IV: Severely compromised.

Risk factors that precipitate heart failure

- Infection – Urinary tract, dental, and respiratory tract
- Anemia
- Obesity
- Hypertension
- Arrhythmias
- Hyperthyroidism
- Drugs – Beta blockers
- Excess intake of caffeine, alcohol, calorie diet
- Physical activity, emotion, Anxiety

Time of admission/Time for refferal from PHC and secondary care hospital to tertiary centres

- Grade I: At 34 weeks
- Grade II: At 28th week specially in case of unfavourable social surrounding
- Grade III and IV: As soon as pregnancy is diagnosed patient should be kept in hospital throughout pregnancy.

Indications for pregnancy termination

- Primary Pulmonary Hypertension
- Eisen-menger's Syndrome
- Dilated cardiomyopathy
- Marfan syndrome with cardiovascular involvement
- Pulmonary AV fistulae
- Any uncorrectable cardiac lesion in functional class III or IV refractory to medical treatment. Done in first trimester. Earlier the best

Anticoagulant treatment

- Indications
  * Artificial (Mechanical) valve replacement
  * Atrial fibrillation
  * Congenital heart disease with pulmonary hypertension.
- Anticoagulant of choice
  * As soon as pregnancy is diagnosed – Stop warfarin
  * Administer Heparin – 5000 U SC BD till completion of 12 weeks
  * From 13th week – Warfarin 3 mg once daily at the same time each day.
  * Discontinue at 36 weeks and administer Heparin – till 7 days postpartum, then continue Warfarin.

Rheumatic fever prophylaxis

- Inj: Benzathine penicillin 12 Lakh units IM every 21 days

Infective endocarditis prophylaxis

- Given during I stage of labour

Incase of PROM and before caesarean section. (Half an hour before).

- Inj.Ampicillin 1 gm IV Inj. Gentamicin 1.5 mg/kg IV repeated after 6 hour. Two additional doses repeated every 8 th hourly

General principles for management of Cardiac patients in labour

I Stage

- Bed rest in semi recumbent position.
- Avoid IV fluid overload[Rate of infusion 50 ml/hr]
- Adequate Analgesia
  * Morphine 15mg
  * IM Pethidine 100mg

* Epidural analgesia

- O2 should be avaliable
- Monitor – Pulse, Blood pressure,RR,Temperature,O2 saturation – 15 minutes.
- Urine output – hourly
- IE prophylaxis.
- Monitor fetal heart rate.
- Keep anti-failure measures ready

II Stage

- Avoid straining, lithotomy position
- Second stage curtailed by outlet forceps or ventouse

III Stage

- No place for prophylactic Methergine.
- If excess bleeding pervaginum 10 units IM syntocinon given

Management of Puerperium

- Hourly pulse, blood pressure, respiratony rate, input output chart to be maintained
- Antibiotic prophylaxis
- Anti-failure drugs and Anticoagulants
- Breast feeding is not contraindicated unless there is failure of Gr III and IV.

Temporary Contraception

- Barrier – safe, effective and prevents STD.
- Oral steroidal contraception – contra indicated as it may precipitate thrombo-embolic phenomenon
- IUCD – contra indicated for fear of infection.

Permanent sterilisation

- Vasectomy

  After 6 weeks of delivery

  At a tertiary care centre.

Gestational Diabetes mellitus (GDM)

Definition

- It is a glucose intolerance of any severity occuring or detected for the first time during pregnancy irrespective of whether insulin is required for treatment or not and whether it reverts back to normal or not after delivery.

Effects Of GDM

- Mother
  * Incidence Of Infections Like Urinary tract infection, Monilial vulvo Vaginitis
  * Skin infections, pruritus
  * Weight gain
  * Incidence Of hydramnios 50%
  * PIH 30%
  * Operative delivery, difficult labour, maternal injuries subsequent to macrosomia.
  * Puerperal Sepsis
- Fetus
  * Sudden IUD
  * Incidence of malformation
  * Fetal wastage
  * Prematurity
  * Macrosomia
  * Incidence of RDS
  * Hypoglycaemia
  * Perinatal morbidity

Investigations

Screening and diagnosis

- Various screening test are available
- Casual blood glucose – cut off level 100 mg%
- Fasting less than 90 mg%
- 2 hours Post prandial less than 120 mg%

One step screening and diagnostic test (GTT) Glucose tolerance test

- It is very convenient for the pregnant women
- The load on the laboratory is reduced
- Allows screening and diagnosis for all antenatal

patients and

- Should be done during 1st visit
- If negative to be repeated at 24-28 weeks and 32-34 weeks.
- As per WHO criteria antenatal patients are requested to come irrespective of meal status and 75 grm glucose is given and blood sugar estimated after 2 hours.
- A value of 140 and above is considered as GDM

Treatment

- Exercise
- Medical Nutrition Therapy (MNT)
- Drugs

Exercise

- No weight reduction during pregnancy
- Only ergometric exercise (flexion and extension of arms and legs)
- Walking

Medical Nutrition Therapy

- Calorie requirement according to bmi and occupation
- 30Kcal/kg body weight
- Carbohydrate: 50-60%.
- Increase the fibre diet.
- Protein : 1-1.5 gm /kg body weight
- The remaining should be fat
- The meals should be split – 3 major meals and 3 – 4 snacks.
- Especially morning breakfast should be spilt into two.

Aim of the treatment

- Blood sugar < 90 mg%
- Post-prandial <120 mg%
- Mean blood glucose 100 mg%

Drugs

- Usually premixed insulin are used during pregnancy
- Short and intermediate 50 – 50 – as there is post prandial hyper-pglycaemia.
- Controlling post-prandial hyperglycaemia is important to control perinatal mortality.

Monitoring the control

- In pregnancy the insulin requirements increases as gestation advances
- So frequent monitoring and adjusting the dose is important to maintain euglycaemia.
  - Once in a month upto 28 weeks
  - Once in 15 days from 28 – 36 weeks
  - Once in a week from 36 week onwards.
- Provided every time euglycaemia is achieved otherwise frequent monitor is required.

Obstetric management in antenatal period

- Regular antenatal period check up
- Dating Scan – as early as 8 weeks
- Anomalies scan – Targeted scan at 20 weeks
- Assessment of fetal well being by modified biophysical profile by 32 weeks onwards.

Management during Labor

- Glycaemic control should be continued during labor since sudden IUD can occur due to lactic acidosis and electrolyte imbalance.
- So hourly blood glucose monitoring and insulin infusion as and when required is essential
- Anticipate shoulder dystocia

Timing of delivery

- Well controlled mother with GDM with good obstetric history can be allowed for spontaneous onset of labor.
- Induction of labour at 38 weeks.
- GDM is not an indication for LSCS.
- The thumb rule is GDM should not be allowed post term

Post-partum

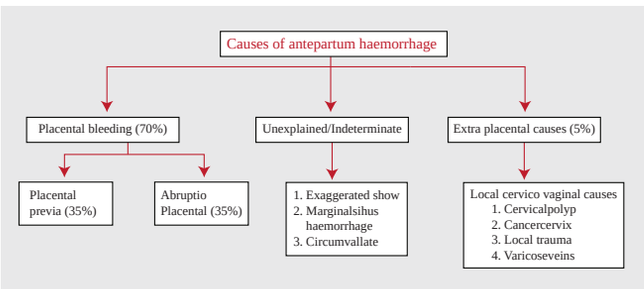
- The insulin requirement drastically falls down after delivery and usually there is no need for insulin postpartum.
- Prophylactic antibiotic

Contraception

- Permanent contraception if family is completed.
- Temporary contraception like barrier method or IUCD can be used.

Follow-up

- Fasting and post prandial blood sugar estimation should be done 15 days after delivery.
- Fasting <100 mg%
- Post-prandial < 120 mg%
- GTT after 6 weeks with 75 grms glucose load should be done as a method of long term follow-up.



Timing of referral

- At 38 weeks
- Suspected macrosomia
- Hydramnios
- Poor control

Post dated pregnancy

Definition

Any pregnancy that crosses 42 weeks or 294 days of gestation is considered as post-term pregnancy and any pregnancy that crosses the expected date of delivery (EDD) is considered as post dated or prolonged pregnancy.

Statistics

The incidence –4 to14 %; approximately 11 %

Causes

- Wrong dates (most common)
- Heredity
- Maternal factors
  - * Primiparity
  - * Elderly Multiparae
  - * Previous post-term pregnancy and
  - * Sedentary life style
- Anencephaly
- Placental sulphatase deficiency

Symptoms

- Diminution of liquor
- Liquor becomes cloudy

Post maturity syndrome

Grade – I – Skin wrinkled and pealing but not meconium stained.

Grade – II – As in stage 1 with meconium staining of fetal skin membranes and card.

Grade–III – Infant and Placenta stained yellow because meconium has been passed for several days.

Investigations

- Menstrual History can be relied upon if
  - * Patient was not on oral contraceptive pills
  - * Patient has had 3 regular cycles before the last one in normal duration and amount of flow
- Ultra Sono Gram
  - * One USG in first trimester (7-11 weeks)
  - * Two USGs in the second trimester (12-28 wks) 3 to 4 weeks apart
- Serum or Urine HCG Test Positive plus 36 weeks
- Fetal Heart sound
  - * With Fetoscope at 20 weeks
  - * With Doppler at 10 weeks
- Uterine size on palpation in the first trimester
- Date of quickening
  - * Primipara at 28 weeks
  - * Multipara at 26 weeks

Diagnosis

Symptoms

- Diminished fetal movement.
- Decline in amniotic fluid volume.
- CTG late deceleration of FHR.
- Fall in serum oestriol.
- Cord and the umbilical vessels are normal – Dop-

pler umbilical will be normal hence not reliable.

Bishop's score				
Parameters	Score			
	0	1	2	3
Cervical dilation in cms	Closed	1-2	3-4	5+
Cervical effacement in %	0-30	40-50	60-70	80+
Fetal Station	-3	-2	0, +1	+2, +3
Cervical consistency	Firm	Medium	Soft	
Cervical position	Posterior	Middle	Anterior	

A Bishop's score < 6 is suggestive of poor induceability.

Investigations

Ultrasound

- Amniotic fluid index (AFI),
- Fetal size
- Placental maturity
- Amniotic fluid volume is important because cord compression due to oligohydramnios is the most common cause of intrapartum fetal distress

AFI	<5 -	Oligohydramnios
	5 -	Decreased liquor
	0 -	Normal liquor
	*	Increased liquor
	>25 -	Polyhydramnios

- Fetal size estimation is important because macrosomia which is common in post-dated pregnancy can commonly lead to shoulder dystocia or other complications during labour.
- Regarding placental maturity, grade III placenta or calcified placenta are usually associated with post dated pregnancy.

Treatment

Complications

Oligohydramnios
Placental Insufficiency
Meconium Aspiration syndrome
Fetal hypoxia and acidosis
Shoulder dystocia
Birth trauma

Prevention

To prevent the complications of prolonged pregnancy all antenatal mothers should attend regular antenatal check up using her records to help confirm her gestational age. And once pregnancy has crossed term,proper fetal surveillance should be done.

Follow up

All post-dated mothers should be clearly advised to come for fetal surveillance tests at 41 weeks and twice between 41 and 42 weeks

Referral

In peripheral centres after making sure that her EDD is correct patients beyond term should be referred to higher centres for fetal surveillance

Important Note

Assessment is best done at 41 weeks with reactive NST (FHR >15 beats lasting for 15 seconds)

Positive CST is an indication for LSCS

If cervix is favorable induction can be done, if unfavorable no .18 Foley's catheter with 30ml balloon kept in the cervix for 12-24 hrs to improve the bishop score by 3 points- 92%success

CTG monitoring is ideal

Foetal distress is diagnosed by – steadily rising baseline FHR, repeated bradycardia and late deceleration

Foetal distress can lead to hypoxic ischemic encephalopathy

Post cesarean Pregnancy

Definition of Caesarean Section

Removal of child through an incision in the abdominal wall of an intact uterus. The word 'caesarean' is derived from the Latin word "caedere" means to cut.

Reasons for increasing incidence in caesarean rate

- Increase in repeat section
- Continuous fetal monitoring is a demand
- Rising induction rates and sequelae
- Improvement in neonatal set up for low birth weight, very low birth weight and IUGR babies.
- Fear of litigation for cerebral palsy
- Professional mothers

Recommendations of ACOG for selection of candidates for VBAC (VBAC- vaginal birth after caesarean section)

- Not more than one prior low transverse caesarean section delivery
- Clinically adequate pelvis
- No other uterine scars or previous rupture
- Surgeon immediately available throughout active labour and performing emergency caesarean section
- Availability of anaesthesia and personnel for emergency caesarean section

The success rate depends on

- Clinically adequate pelvis.
- The indication of previous caesarean section
  - The success rate may be high as 90% if previous caesarean section was done for non-recurring indication and 60% when done for potentially recurring indication like delivery dystocia.
- Previous vaginal delivery
  - Vaginal delivery before or after caesarean improves prognosis

Management of labour following caesarean section

- Refer post caesarean delivery to higher centres
- Informed consent for trial of scar
- Keep operation theatre ready for delivery
- Keep compatible blood available.
- All post caesarean deliveries should only be managed in equipped hospitals with personnel readily available if emergency caesarean section is needed

First stage of Labour

- Provide sedatives to decrease anxiety (inj. Pethidine/inj. Fortwin)
- Monitor fetal heart and maternal pulse rate.
- Administer Oxytocin carefully
- Plot parto-graph
- Constant attention

Warning signs of uterine rupture

- Irregularities of fetal heart rate
- Pain and tenderness in the lower abdomen
- Blood stained urine
- Maternal tachycardia
- Vaginal bleeding
- In cases, where fetal presenting part has entered the pelvis, not able to detect station by per vaginal examination

Terminate the trial of labour for emergency caesarean section

If progress is unsatisfactory with PROM and unengaged head, it is safe to do caesarean section.

Second stage of labour

- If there is good progress
- Monitor as above
- Use outlet forceps

Third stage of labour

- Active management
  - If no excess bleeding
  - No need for uterine exploration
- Deliver placenta by active management of third stage of labour
  - If there is delay in placental separation
  - Excess bleeding
  - Blood stained urine

Post-natally

- Patient to be monitored in labour room for four to six hours after delivery.
- A study of risks and complications between trial of labour and planned elective cesarean section showed uterine rupture risk and perinatal mortality to be higher in trial of scar
- No difference was found in maternal mortality.
- To conclude Vaginal birth after caesarean section (VBAC) can be safely practiced and incidence of caesarean section rates can be dramatically reduced, provided patient selection for VBAC is done properly in a well equipped hospital.

Post operative care:
Principles of initial care

- Place the woman in the recovery position.
- Position the woman on her side with her head

slightly extended to ensure clear air way.

- Place the upper arm in front of the body for easy access to check blood pressure
- Assess woman's condition immediately after the procedure.
- Check vitals pulse rate, blood pressure, temperature, respiratory, every 15 minutes in first hour, then every 30 minutes for the next hour
- Assess the level of consciousness every 15 minutes until the woman is alert.
- Ensure clear air way and adequate ventilation
- Look for vaginal bleeding
- Check the intravenous lines.
- Check the Foley's catheter position, look for the colour and quantity of urine in uro-bag and make a note of it.
- Maintain vitals chart and I/O chart

Post- operative fluid infusion

To maintain normo-volemia in woman with surgical fluid losses.

- Intravenous fluids should be normally given at 100 ml/hour for first 24 hours provided there is no associated medical risk restricting fluid infusion
- In case, the woman presents with heart disease / severe anaemia, fluid to be transfused very carefully to avoid the risk of pulmonary embolism and Congestive cardiac failure (CCF)
- In patients with existing renal disease, fluid infusion to be calculated as urine output in the previous hour + 30 ml for the next hour.
- Crystalloid replacement fluids, Normal saline/ Dextrose normal saline and balanced electrolyte solution Ringer lactate to be infused, initially.
- Dextrose solutions are poor replacement fluids. They are not preferred to replace fluid loss.
- Colloids have no super added advantage over crystalloids for resuscitation.
- Check electrolytes if woman is receiving intravenous fluids for more than 48 hrs.

Analgesia

- Adequate post operative pain control is important.
- Good post operative pain control regimens include
  - Non-narcotic mild analgesics
  - Inj. Tramadol intramuscular whenever needed.

Bladder care for 8 hours

If the urine is clear, no need to keep the catheter in situ except in conditions like

- Uterine rupture
- Prolonged/Obstructed labour
- Massive perineal oedema
- Periperal sepsis and risks

For 7 days

In bladder injury due to uterine rupture, caesarean section and laporotomy.

Assessment of gastro-intestinal function

- If surgical procedure is uncomplicated,start liquids 12 hour after surgery.
- If there are signs of infection or if caesarean section is done for rupture uterus, Obstructed labour, wait till bowel sounds return.
- When woman passes flatus ,start soft solids.
- Continue intravenous fluids till woman takes oral fluids well.
- Ensure woman is having regular diet prior to discharge.

Antibiotics

- Pre-operative antibiotics to be given
- Continue for 5 days
- If there is fever or signs of infection, investigate the patient and treat with appropriate antibiotics

Dressing and wound care

- If the blood or fluid is leaking through initial dressing,reinforce.
- If bleeding increases or covers half the dressing, replace with another.
- If dressing is loose, reinforce.
- Replace dressing on the 4th post operative day.
- Daily wound care to be given

Suture removal

- In case of SPT (Supra- pubic transverse) scar, remove sutures on 7th post- operative day.
- In case of RPM (Right paramedian) scar,remove on 8th post- operative day.

Ambulation

- Encourage foot and leg exercises and mobilize the patient as early as possible within 24 hrs.

Specific case management:

Heart Disease

- Propped up position, start nasal oxygen.
- Check vitals every 15 minutes.
- Restrict intravenous fluids.
- I/O chart.
- Adequate analgesia
- Antibiotics. (Infective endocarditis prophylaxis is must.)
- Intravenous fluid infusion to be done carefully to avoid the risk of fluid overload
- To avoid thrombo-embolic complication, continue anti-coagulants (Heparin 5000 units s/c, to be started after 12 hours)

Pre-eclampsia

- Check blood pressure and vitals every hour.
- Intravenous fluid replacement to be done carefully to avoid fluid overload.
- Continue anti-hypertensives according to blood pressure.
- Input output chart
- If patient had been given Magnesium Sulphate for imminent symptoms,continue Magnesium Sulphate post operatively for 24 hours
- While on Magnesium Sulphate regimen check
  - Respiratory rate
  - Urine output
  - Deep tendon reflexes
  - Response to pupils equality reacting to light
- Look for coagulation profile BT/CT/PT.
- Repeat the investigations on first day post op.

Eclampsia

- Ensure clear air way.
- Ensure adequate ventilation, if needed connect the patient to mechanical ventilator.
- Continue anti-convulsants.
- Check vitals every half an hour.
- Anti-hypertensives according to blood pressure
- Check the level of consciousness frequently

Diabetes mellitus complicated Pregnancy:

- Check vitals.
- Fluid replacement with crystalloids.
- Check blood sugar immediate post op and every 4th hourly
- Establish another intravenous line with dextrose 5% with insulin, infuse according to glycemic status of patient.

HIV In Pregnancy

The incidence of HIV in India is increasing rapidly with over 5 million infected people in 2004. The commonest source of infection is sexual (85%), other sources are IV drug abuse (6.7%), Blood products (2.7%) and perinatal (2.5%).

Following viral entry, there is a window period of 6-12 weeks after which patient becomes HIV positive. Thereafter there is a latency of 2-10 years before full blown AIDS is manifested.

HIV infection is diagnosed by detection of antibodies, that is ELISA (Positive 12 weeks after infection) and Western Blot tests (Positive after 72 hours) or by antigen screening. (PCR and P24 antigen – positive after 2 weeks).

90% of Pediatric HIV infection is due to vertical transmission (mother to child transmission).

In India, the incidence of HIV in asymptomatic antenatal population is 0.5 – 0.8%. With no interference, the risk of transmission of HIV to the baby is 30%. With interference, this risk can be brought down to less than 1%.

Prevention of Mother to child transmission components (MTCT)

1. Pre-Test counselling – Mandatory for all pregnant patients
2. Testing for HIV – by ELISA
3. Seropostive – confirm by 2 ELISA tests or western blot test
4. Screen for other infections STD, HEP-B, tuberculosis, CMV
5. Post test counselling
6. Evaluation of HIV status – CD4 count, Viral loads culture.
7. Anti-retrovial inverventions after 34 weeks

Anti-Retrovial inverventions	
Regimen - I	Zidovudine 300 mg bd + 300 mg every 3 hours in labor (Decreased transmission by 51%)
Regimen – II	Single dose Nevirapine 200mg at onset of labour + Nevirapine syrup 2 mg/kg within 72 hours of birth for the baby (Decreased transmission by 37%)

Intrapartum management

- Elective caesarean decreases Mother to child transmission (MTCT)
- Universal work precaution for health care personnel
- Vaginal disinfection
- Early cord clamping
- Thorough baby bath
- Proper disposal of infected material in hypochlorite solution.

Post Partum

- Breast feeding – Increases risk of transmission by 20%
- If breast-feeding cannot be avoided
  - * Advice exclusive breast feeding for 3 months
  - * Abrupt weaning because formula feeds along with breast milk increases GI inflammation and increases transmission risk.
- Advice condom usage

Follow up of baby

- Less than 18 months – PCR Testing
- After 18 months – ELISA TESTING

Clinical markers of chromosomal and Genetic disorders

The following clinical markers may help the clinician to identify common genetic disorders

Head and Face

Shape of Head

Microcephaly	–	All mental retardation
Brachycephaly	–	Down's syndrome
Trigonocephaly	–	Craniostenosis Crousons disease Apert syndrome
Hydrocephalus	–	Neural tube defects

Hair

- Soft silky hair - Down syndrome
- White forelock - Waardenburgh syndrome

- String like hair - Kinky's minky hair syndrome.

Face

Dysmorphic features typical of chromosomal disorder

- Short or broad forehead
- Hyper-trichosis of face and forehead
- Hyper-teliorism
- Epicanthic fold – mongoloid slant – Down syndrome
- Anti-mongoloid slant – Noonan/Turner Syndrome
- Depressed nasal bridge
- Long philtrum
- Open mouth with protruded tongue
- Low set ears – other ear anomalies.
- Large ears – in male - Fragile x syndrome
- Pre-auricular skin tags
  - * Golden Harr Syndrome
  - * Treacher collins Syndrome
- Absent ears (auricles)
  - * Golden Harr Syndrome
  - * Treacher collins Syndrome

Eyes

- Congenital cataract

Brush field spots in iris	-	Down Syndrome
Hypo-pigmented iris	-	Ocular Albinism
Lens dislocation	-	Marfans Syndrome
Coloboma of Eyes	-	Golden Haar Syndrome
Ptosis – Myasthenia Gravis		

Cleft lip and Palate

- Present in may disorders of chromosomes and also as an independent entity.

Pitting of lips

- Ellis van creveld syndrome.

Teeth

- Peg like – hypoplastic – ectodermal dysplasia
- Late development of teeth – Down's syndrome

Neck

Webbing	–	Turner/Noonan syndrome
Short Neck	-	Klippel feil sequence
Torticollis	-	Congenital with many anomalies.

Chest

Broad and Shield like	–	Turner/syndrome
Pectus Excavatum	–	Noonavan syndrome
Accessory Nipples	–	In many disorders
Absent Pectoralis Major	–	Poland syndrome

Arms

- Cubitus Valgus (wide carrying angle) – Turner syndrome
- Dwarfism with shortness of upper arm / fore arm or hands and feet – Achondroplasia
- Forearm anomalies – Radial ray defect in Fanconis anaemia

Fingures and toes :

Brachy-dactlyly	–	Down's syndrome.
Cleino-dactyly	–	Down's syndrome
Campto-dactyly	–	Bony Disorders
Ectro-dactyly	–	In some chromosomal disorders (Absent digits)
Syndactyly	–	In many disorders
Poly-dactyly	–	In many disorders
Sandal cleft feet	–	Down's syndrome

Crease anomalies

Single Palmar crease – Down's syndrome

Hypoplastic Nails

- Ectodermal dysplasia
- Ellis Van creveld syndrome

Shoulder

Sprengel's shoulder	–	Scapular defect

Abdomen

Umbilical hernia	–	Cretinism / MPS
Hepatosplenomegaly	–	Storage disorder
Other Masses	–	Tumors
Inguinal Hernias	–	Female to rule out testicular feminization sndrome

External Genitalia

Males

Microphallus /	–	5α reductose deficiency
Hypospadiasis	–	Praeder willi syndrome LMB syndrome
Undescended testes	–	In many disorders
Large genitals inboys with MR	–	Fragile x syndrome precocious puberty (Male Congenital adrenal hyperplasia)

Females

Clitoromegaly-Congenital adrenal hyperplasia

Lower limb

Genu Valgum	–	Rickets
Congenital talipes equino varus	–	Part of a syndrome or independent entity

Feet

Pedal oedema at birth	–	Turner syndrome, Milroy's disease
Large big Toe	–	Rubinstein Taybi syndrome
Sandal Cleft	–	Down's syndrome (Trisomy 21)
Short 4th digit	–	Turner syndrome
Rocker bottom left (Trisomy 18)	–	Edward syndrome
Flat Feet	–	In many disorders
Polydactyly	–	Laurence-Moon-Biedl-Syndrome

Spine and Cranium

- Occipital encephalocoele
- Anencephaly — Neural tube defects
- Meningo-myecoele
- Spinama Bifida
- Gibbus – Mucopolysaccharidosis – Hunters syndrome
- Scoliosis
- Kyphosis — In skeletal dysplasia
- Lordosis

Skin : Neurocutaneous markers

- Haemangiomas
- Neurofibromas
- Tuberous sclerosis
- Ash-leaf macules
- Hypo-pigmented patches
- Mongolian Patches
- Congenital Icthyosis
- Port-wine stain – on face – Sturge weber syndrome

Overall appearance

- Dwarfism — Indicates Hormonal disorders.
- Hirsutism

Antepartum Haemorrhage (APH)

Definition

Antepartum hemorrhage is defined as the bleeding from genital tract after 28th week of pregnancy and before the birth of the baby.

Causes of APH

Placenta Previa

Normally, the placenta is located in the upper part of the body of the uterus encroaching to the fundus adjacent to the anterior or posterior wall with equal frequency.When the placenta is implanted partially or completely over the lower segment, it is called Placenta previa.

Risk factors

- Advancing maternal age
- Multiparity
- Multiple gestation
- Previous caesarean section or any other scar in the uterus.
- Smoking
- Placental abnormality (succenturiate lobes)

Degrees of Placenta previa

1. First degree/type I - Low lying- placenta is dipping into the lower uterine segment such that the placental edge does not reach the internal os.
2. Second degree/type II - Anterior and posterior - marginal- edge of placenta is at the margin of the internal os.
3. Third degree/type III - Internal os is partially covered by the placenta (placenta covers the internal os when closed but does not entirely do so when fully dilated)
4. Fourth degree/type IV - Total – internal os is completely covered by the placenta.

Clinical features Symptoms and signs

Recurrent causeless and painless bleeding after 28 weeks

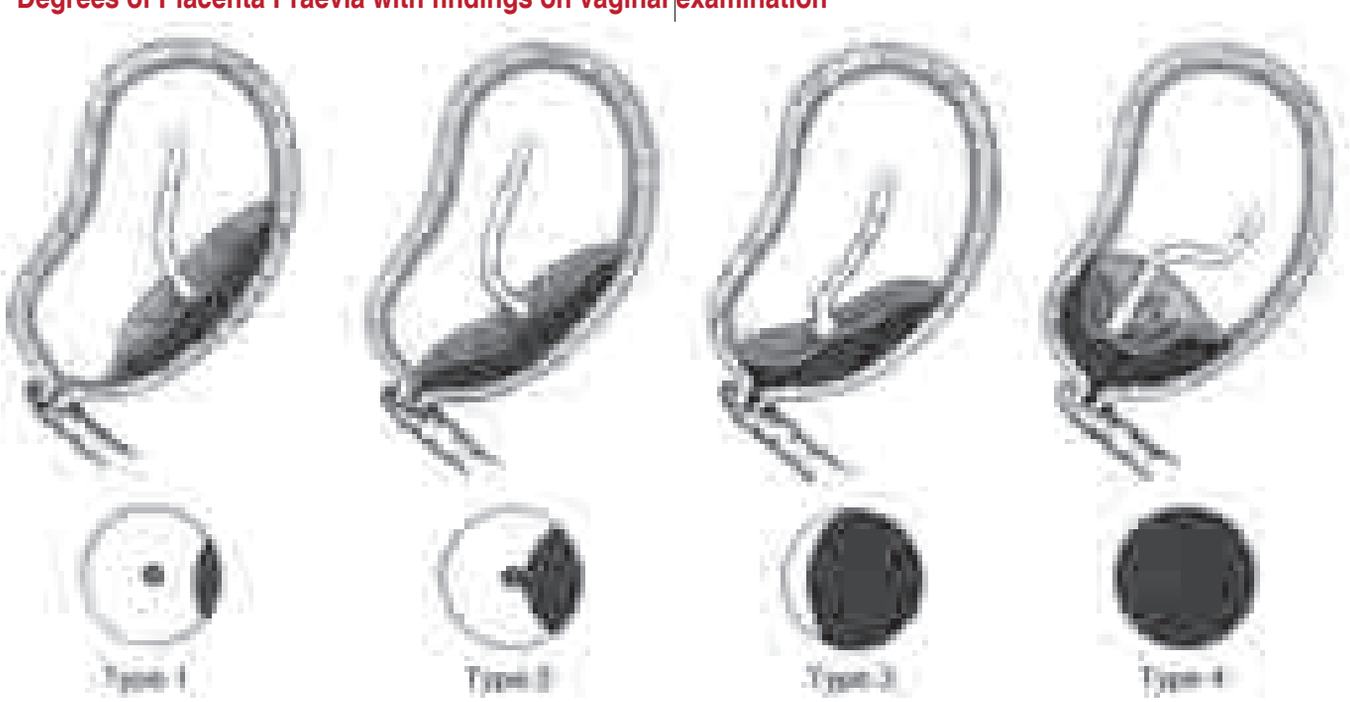
Examination

General:

- Anaemia
- Tachycardia
- Hypotension proportionate to the visible blood loss.

Per abdomen

Degrees of Placenta Praevia with findings on vaginal examination



- Size of the uterus proportionate to the period of gestation.
- Uterus feels soft, non tender
- Persistence of mal-presentation
- High up presenting part.
- Soft boggy feel over the lower uterine segment.

Per vaginal

Double setup vaginal examination to be done in the theatre, only after keeping everything ready for caesarian section

Diagnosis

Ultrasound is the gold standard for diagnosis

Differental diagnosis

- Abruptio placenta
- Vasa previa
- Circumvallate placenta
- Other local cervical lesions.

Complications of Placenta previa

- Maternal
  - * Hypo-volemic shock
  - * Premature Labour
  - * Complications due to malpresentation
    - » Early rupture of membranes
    - » Cord prolapse
    - » Increased operative interferences
  - * Post-partum Haemorrhage
  - * Sepsis
- Fetal
  - * Low birth weight – mostly due to prematurity.
  - * Asphyxia
  - * IUD – Intrauterine death of the fetus.
  - * Congenital malformations

Indications of caesarian section in placenta previa

- Type III and IV and type II posterior
- Mal-presentations and other complications
- Profuse bleeding per vaginum which is not controlled by amniotomy in type I, II (anterior)

At PHC Level

Initial resuscitation has to be done as soon as the patient comes to the medical attention.

- Bed rest
- IV lifeline
- Blood for grouping, typing and cross matching
- IV fluids – according to the amount of blood loss.

Referral

All cases of placenta previa irrespective of gestational age should be transferred to a referral hospital where conditions for blood transfusion, caesarian section and neonatal resuscitation are available.

Abruptio Placenta

Definition

Abruptio placenta/Accidental haemorrhage is one form of APH where hemorrhage occurs as a result of premature separation of normally situated placenta.

Risk factors

- Maternal hypertension and PIH
- Multiparity
- Advancing age of the mother
- Poor socioeconomic status
- Folic acid deficiency
- Smoking
- Sudden uterine decompression – as occurs when the membranes rupture in a case of hydramnios
- Multiple pregnancies.
- Uterine fibroids complicating pregnancy.
- Cocaine abuse
- Pre-term rupture of membranes
- Trauma

- External Cephalic Version

Types of Abruption

- Concealed type
  - No external bleeding
  - Blood collects behind the separated placenta , behind membranes and behind the presenting part
- Revealed type
  - Blood collected due to placental separation escapes by dissecting under the membranes and seen externally.
- Mixed type
  - This is the commonest type where the blood is partially concealed and partially revealed.

Clinical features

Symptoms

Abdominal pain and discomfort followed by vaginal bleeding

- Vaginal bleeding
- Uterine tenderness / abdominal pain
- Fetal distress and fetal death
- Pre-term labour
- High frequency contractions
- Dead fetus

General examination

- Tachycardia
- Hypotension
- Pallor

Abdominal examination

- Uterine height disproportionately enlarged to the period of gestation
- Uterus feels tense and tender
- Fetal parts difficult to make out.
- Fetal heart sounds usually absent (except in mild/ very early cases)

Differential diagnosis

- Placenta previa
- Acute hydramnios
- Rupture uterus
- Other acute abdominal conditions like torsion of a ovarian cyst
- Tonic uterine contractions.

Distinguishing features of placenta previa and abruptio placentae

Clinical Features	Placenta Previa	Abruptio Placenta
Nature of bleeding	Painless, apparently causeless, recurrent. Bleeding is always revealed.	Painful often attributed to PIH or trauma, continuous bleeding. Revealed / concealed / Mixed.
Character of blood	Bright red	Dark coloured
General condition of the patient	Proportionate to visible blood loss	Worser than the proportion of visible blood loss (in concealed/ Mixed types)
Features of Pre-eclampsia	Not relevant	Present in 1/3rd of cases

Abdomination Examination

Height of uterus	Proportionate	May by dispropor tionately enlarged in concealed types.
Feel of uterus	Soft and relaxed	May be tense, tender and rigid
Malpresentation	Common, Head is high, floating	Unrelated. Head may be engaged
Fetal heart sounds	Usually present	Usually absent

Clinical classification (Page classification)

- Grade 0
  - * Clinical features may be absent. The diagnosis is made after inspection of placenta following delivery
- Grade I
  - * External bleeding (+) Uterus – irritable, tenderness may or may not be present
  - * Shock is absent
  - * FH is good
- Grade II
  - * Uterine tetany and uterine tenderness always present.
  - * Shock is absent
  - * Fetal distress / death.
- Grade III
  - * Marked uterine tenderness
  - * Shock is present
  - * Fetal death
  - * Associated coagulation defects/oliguria may complicate

Complications of Abruptio Placentae

- Hypovolemic shock
- Renal failure
- Coagulation failure
- Post partum hemorrhage

Management at PHC level:-

- Initial resuscitation should be done
- IV lifeline and fluids
- Blood for Hb, coagulation profile (platelets, fibrinogen Prothrombin time, activated partial thromboplastin time)
- Continuous bladder drainage – To monitor urinary output hourly and to detect haematuria.

Mild cases

In mild cases, when the patient is in labour , ARM ± oxytocin infusion with careful monitoring of urine output, coagulation profile, general condition of the patient.

Severe cases

All cases of severe abruption should be transferred to be referral centre where facilities for blood and fresh frozen plasma transfusions, caesarean section/neonatal resuscitation are available.

Post Partum Haemorrhage

Definition

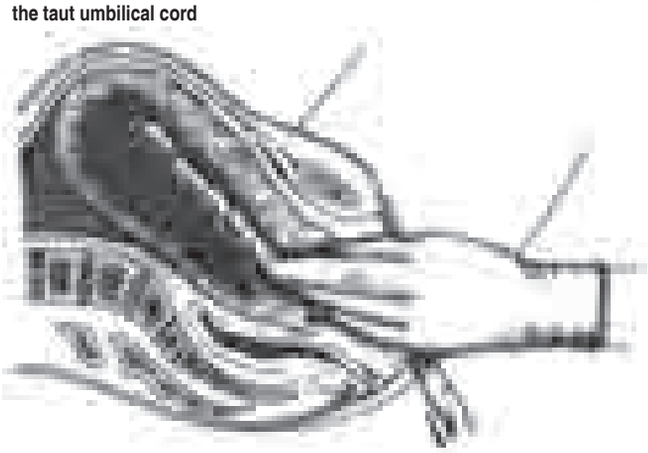
PPH is defined as the loss of 500 ML of blood or more from the genital tract during the 1st 24 hours of delivery.

Predisposing Factors :

1. Uterine atony
   - * Over distension of the uterus (Twins and Hydramnios)
   - * Prolonged labour
   - * Grand multi
   - * APH
   - * Uterine fibroids
   - * General Anesthesia (Halothane)
   - * Oxytocin augmentation of labour
2. Retained Placenta
   - * Placenta Accreta
   - * Accessory lobe of placenta
   - * Retained cotyledons
3. Lacerations of the genital tract
   - * Cervical tear
   - * High vaginal tear
   - * Mid vaginal tear

Manual removal of Placenta

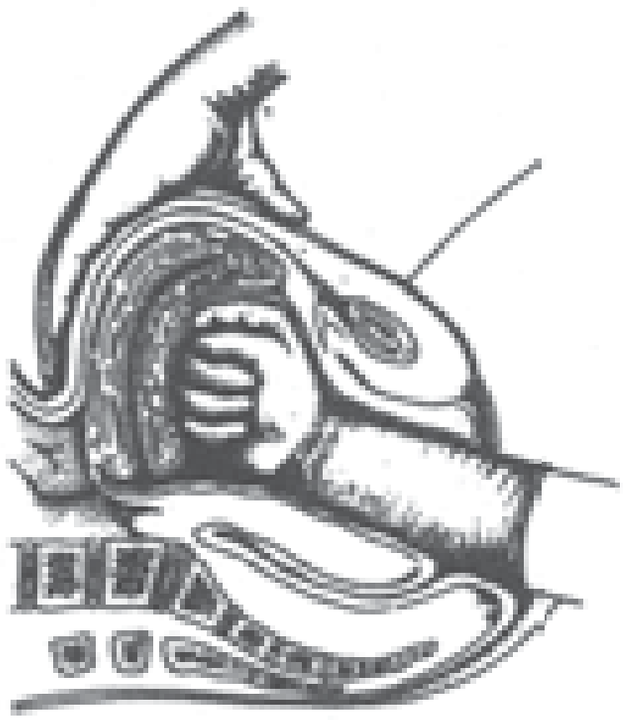
Introduction of hand into the Uterus in a cone shaped manner following the taut umbilical cord



Method of Placental separation by

a. Keeping the back of the hand in contact with the Uterine wall
b. With slicing movements of the hand

Bimanual compression of the uterus



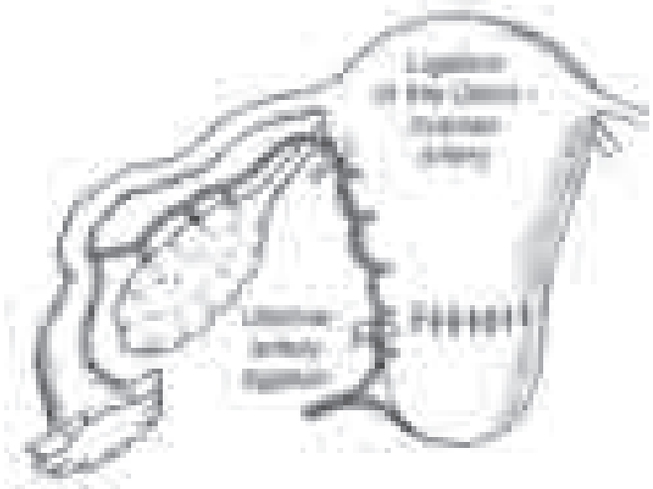
* Low vaginal tear
* Vulval Hematoma
* Rupture of the Uterus

4. Coagulation Disorder
   * Abruptio Placenta
   * Blood Transfusion
   * Severe Preeclampsia
   * ITP
5. Uterine inversion

Key Points

- In a shocked patient due to rupture of the uterus or inversion corrective measures and resuscitation should be done simultaneously .
- In correcting inversion the part that prolapsed last should be repositioned first.

Ligation of the utero - ovarian artery



B - Lynch brace suture for control of Atonic PPH

Post menopausal bleeding

Definition

Post menopausal bleeding is the vaginal bleeding occurring after 1 year of amenorrhea.

Incidence

Occurs in 5%.

Malignancy of genital tract accounts for 30 – 50% of PMB

Risk Factors

- Obesity
- Diabetes
- Hypertension

- Estrogen replacement therapy
- Tamoxifen

Causes

Genital:

Endometrial causes	Cervix
• DUB(metropathia haemorrhagica) • Senile endometritis • Endometrial polyp • Tubercular endometritis • Endometrial hyperplasia • Endometrial carcinoma • Sarcoma **Ovary** • Benign tumors such as • Brenner tumor, hormone sec tumor such as • Granulosa cell tumor • Theca cell tumor	• Erosion, polyp, cervicitis, carcinoma cervix, decubitus ulcer in prolapse **Vagina** • Senile vaginitis, vaginal tumors, foreign body like ring pessary **Vulva** • Vulvitis, benign and malignant lesions

Non-Genital

(a) HT, bleeding diathesis

(b) Urinary tract – urethral caruncle, papilloma, carcinoma bladder

(c) Bowel– hemorrhoids, anal fissures, rectal cancer.

Symptoms

- Irregular bleeding
- Spotting
- Brownish discharge
- Post-coital bleeding

Dysfunctional uterine bleeding

Definition

Bleeding from uterus in the absence of organic disease of the genital tract.

Classification:

Presentation can be classified into 2 groups based on

* Age Group
* Bleeding Pattern

Adolescent	Reproductive	Perimenopausal
Perimenarchel	20 - 40 years Ovulatory R/o complications of pregnancy	40 years Anovulatory R/O benign and malignant diseas

Intermenstrual Bleeding

R/o Polyp

Submucosal Fibroid

Carcinoma Cervix

Average Blood loss during menses 30-40ml ,> 80ml is Menorrhagia

Investigations

1. CBC
2. PT, aPTT
3. Thyroid Function Test
4. Pregnancy Test
5. Pap Smear
6. Culture for Gonorrhea with Chlamydia
7. Gonadotrophin Levels
8. USG
9. Curettage
10. Diagnostic Hysteroscopy

Treatment

Exclusion of organic disease of genital tract

Pubertal Age Group

- Reassurance
- Menstrual Calendar
- Iron and Vitamin Supplementation
- Hormones
- NSAIDS
- Antifibrinolytics
- Blood Transfusion
- Re evaluation

- D and C – Normal Haemostasis fails

C.L-corpus luteum O.C.P-oral contraceptive pill

DUB in Perimenopausal Age Group

Investigations to rule out benign and malignant disease of the genital tract and manage accordingly.

Screening guidelines for early detection of Cancer

Introduction

The goal of cancer screening is a very practical one - to detect cancer at an early stage when it is treatable and curable .Screening is defined as early detection of asymptomatic or un-recognized disease by the application of acceptable,inexpensive tests or examination in a large number of persons.

In India cancer of the breast has emerged as the top ranking cancer in 2002 followed by uterine cervix, oral cavity, stomach and ovary in females.

The annual CIR [Crude Incidence Rate] and ASR [Age Specific Rate] for breast cancer in 2002 is 26.8 and 29.3/100,000 and for cancer cervix is 21.3 and 24.2/ 100,000 population [MMTR report 2005]

Selected health care organizations has given guidelines for screening general population.The American Cancer Soceity [ACS] guidelines is given below along with the ICMR recommendations for screening of cervical cancer and breast cancer in India.

Screening for Breast cancer

ACS guidelines for the early detection of breast cancer in average risk women emphasize a process that begins when a woman is 20 years of age and consist of a combination of clinical breast examination (CBE), counselling to raise awareness of breast symptoms and regular mammography beginning at age 40.

Between the ages 20 and 39 years, women undergo clinical breast examination every 3 years. Although A C S no longer recommends that all women conduct regular breast self examination BSE, women should be informed about potential benefits, limitations associated with breast self exam.

Instructions on the technique of BSE can also be given. ACS recommends that average risk women should begin annual mammography at the age of 40 years. Benefits include a reduction in the risk of dying from breast cancer, less aggressive therapy and a greater range of treatment options.

There is no set age at which mammography should be discontinued. As long as a woman is in good health and would be candidate for breast cancer treatment, she should continue to be screened with mammography There is benefit in choosing digital mammography over screen – film mammography especially in younger women with dense breasts.

In India, since the breast cancer is on the rise, awareness about the importance of recognizing symptoms for breast cancer is essential. Breast – self examination should be advised to every woman beginning at the age 20 years. Presently mammography is used for diagnostic purposes not for screening because it is not available in most places.

Screening for cervical cancer

ACS recommends that cervical cancer screening should be approximately 3 years after the onset of vaginal intercourse, but not later than age of 21 years.

Annual screening with conventional cytology smears, or biennial screening using liquid based cytology, is recommended until the age of 30 years. At or after 30 years, a woman who has had 3 consecutive, technically satisfying normal/negative cytology results may undergo screening every 2 – 3 years using conventional or liquid based cytology.

Alternatively, after 30 years of age, women who have the same history of normal cytology results may undergo HPV DNA testing with conventional or liquid based cytology every 3 years.

Average risk for women aged 70 years and older with an intact cervix may choose to cease cervical cancer screening if they have no abnormal /positive cytology tests within the 10 year period before the age of 70 years.

Women with a history of cervical cancer should follow the same guidelines as average risk women. Women who are immunocompromised by organ transplantation, chemotherapy or corticosteroid treatment or who are HIV+ should be tested twice during the first year after diagnosis and annually thereafter.

There is no specific age to stop screening for women with h/o cervical cancer and who are immuno-compromised including HIV+ve patients.

Women with a h/o CIN 2/3 or women for whom it is not possible to document the absence of CIN 2/3 before as the indication for the hysterectomy, should continue until there is a 10 year history of no abnormal positive cytology tests.

Average risk of women who have had sub total

hysterectomy, should be screened like average risk women who have not undergone hysterectomy.

In India, ICMR guidelines state that every women at age of 30 years should undergo PAP smear at least once as it helps in diagnosis of early cervical cancer.

Screening for Colo-rectal cancer for men and women, aged > 50 years have the following options as given by the ACS

1. Annual Fecal Occult blood test (FOBT)
2. Flexible sigmoidoscopy every 5 years
3. Annual FOBT and flexible sigmoidoscopy ever 5 years
4. Double contrast barium enema [DCBE] or
5. Colposcopy every 10 years

Screening for Endometrial cancer

- The ACS states that there is insufficient evidence to recommend screening at average risk or somewhat increased risk, for endometrial cancer due to h/o unopposed Estrogen therapy, Tamoxifen therapy, late menopause, null-iparity, infertility, diabetes, obesity, or hypertension
- Women at high risk due to known hereditary nonpolyposis colon cancer (HNPCC) associated genetic mutation carrier status should consider beginning annual testing for early endometrial cancer detection at an age of 35. The endometrial biopsy is still the most common technique used to obtain endometrial tissue.

Screening guidelines

Screening for Cervical cancer

- Age group
  - 3 years after sexual activity, not later than 21 years
  - In countries with limited resources all women around the age of 30 years should have one Pap test.
- Method
  - Pap Smear (or)
  - VIA (visual inspection after application of 5% acetic acid) in countries with limited resources.
- Pre-cautions
  - Prior to a pap smear

Advice

- Not to douche for 48 hours
- Not to use vaginal cream for 1 week
- To abstain from coitus for 24 hours
- Smear to be done on 7th day after menstrual period

Technique

- PAP TEST includes samples from ecto-cervix and endo-cervix
- Use bivalved speculum
- Place the endocervical brush or cotton tipped swab and roll it firmly against the canal
- Remove the endocervical brush or cotton tipped swab and place samples on the glass slide
- Place the spatula against the cervix with the longer protrusion in the cervical canal
- Rotate the spatula 360° firmly against the cervix , scrape the entire transformation zone
- Place the sample immediately from the spatula onto the glass slide and fix it (spray fixative or placing it in a bottle of fixative – 95% ethanol.

Follow Up

Abnormal smears should be referred for colposcopy and biopsy

Screening For Breast Cancer

- Self breast examination from the age of 20 years
- Clinical examination once in 3 years by a physician
- Baseline mammogram from age of 40 years and once in 2 years afterwards
- Annual mammography from the age of 50 years
- In countries with limited resources, mammogram is available for diagnostic purposes not for screening.

Screening for Endometrial cancer

- Not recommended for general population or women with average risk or somewhat high risk for endometrial cancer

* Women at high risk (due to hereditary non-polyposis colon cancer carrier status) should consider annual testing from the age of 35.
* Endometrial biopsy is the most common technique used to obtain endometrial tissue.

Screening for Colorectal cancer

* Men and women aged > 50 years
* Annual Fecal occult blood test (FOBT)
* Flexible sigmoidoscopy every 5 years

Chapter 15

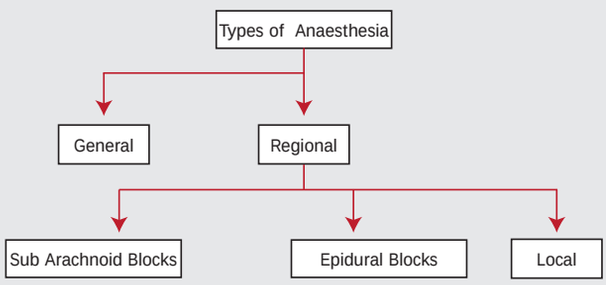
Principle and Practice of Anaesthesia
Components of Anaesthesia
Requirements of Anaesthesia
Complications of Anaesthesia

Principles and Practice of Anaesthesia

Components of Anaesthesia

The four basic components of Anaesthesia are :

- Amnesia
- Analgesia
- Muscle relaxation
- Abolition of reflexes with maintenance of physiologic homeostasis



General Anaesthesia

Advantages	Limitations
• Better muscle relaxation • Better analgesia and amnesia • Effective oxygenation and ventilation	• Polypharmacy • Delayed recovery • Less cost effective

Regional Anaesthesia

Advantages	Limitations
Sub Arachnoid Blocks • Technically easier • Cheaper • Cost effective Epidural Blocks • Used for providing longer duration of surgeries and post operative analgesia • analgesia	Sub Arachnoid Blocks • Cannot be performed in patients with bleeding diathesis, ↑ ICT • Cannot used for surgeries on the chest,neck and face Epidural Blocks • Patchy block • Accidental dural puncture

Local Anaesthesia

Advantages	Limitations
• Better patient acceptance • Avoidance of poly pharmacy • Early discharge of patients	• Can be used only for superficial surgeries.

Requirements for anaesthesia

In Upgraded PHC's

Surgeries usually performed are

- Simple, superficial surgeries like lipoma excision,
- Incision and drainage, puerperal sterilization, conduct of normal delivery,
- Simple suturing
- ENT Procedures like foreign body removal from nose, ears, etc.

These can be done under local anaesthesia (or) intravenous general anaesthesia. The following are required.

1. Instruments
   - * Boyle's apparatus with Oxygen Source
   - * Simple face mask – Size 3,4
   - * AMBU Bag
   - * Endotracheal tubes
   - * Laryngoscope
   - * Venflons
   - * Airways
   - * Urinary Catheters to assess urinary output
   - * Foot suction and electrical suction
   - * New born resnscitation kit
2. Monitors
   - * Stethoscope
   - * BP apparatus
   - * Pulse oximeter
   - * ECG Monitor
   - * Defibrillator
3. Drugs
   - * Induction Agents
   - * Inj Thiopentone sodium

* Inj Propofol
* Inj Ketamine
* Benzodiazepines
* Inj Diazepam
* Inj Midazolam

4. Muscle Relaxants
   * Inj Suxamethonium
   * Inj.Vecuronium
5. Opiods
   * Inj Fentanyl, Inj Pentazocine (Fortwin)
6. Local Anesthetics
   * Inj Lignocaine 2%
   * Inj Bupivacaine 0.5%
7. Emergency Drugs
   * Inj Adrenaline
   * Inj Atropine
   * Inj Furosemide
   * Inj Dopamine
   * Inj Dexamethasone
   * Inj Sodium Bicarbonate
   * Inj Deriphylline
   * 25% Dextrose, Inj Hydrocortisone,
   * Inj Neostigmine.
8. Intravenous Fluids
   * Ringer Lactate
   * 0.9% Normal Saline
   * 5 % Dextrose in 0.9% Normal Saline
   * Colloids.
9. Obstetric Drugs
   * Inj.Oxytocin
   * Inj Methyl ergometrine
   * Inj Prostaglandin F2 alpha
   * Inj Ranitidine
   * Inj Metoclopramide
   * Inj Magnesium sulphate prophylaxis

District headquarters hospitals

In District head quarters hospitals, surgeries that require general anaesthesia, regional anaesthesia and local anaesthesia can be done. The requirements include.

1. Instruments
   In addition to the instruments required in PHCS, the following are needed :
   * Pediatric masks and circuits.
   * Laryngeal Mask Airways, Stylet, Bougie.
   * Magill's forceps.
   * Spinal needles.
   * Inter costal Drainage set.
   * Vaporizers (Halothane, Isoflurane) in Boyle's Machine
   * Defibrillators.
   * New born resuscitation kit.
2. Drugs
   In addition to the drugs required in PHCS, the following are needed
   * Muscle Relaxants
     » Non – Depolarizing relaxants like vecuronium, Pancuronium
   * Antihypertensive Drugs
     » Inj Nitroglycerine, Beta blockers
   * Reversal Agents
     » Inj Neostigmine
   * Anticholinergics
     » Inj Glycopyrrolate, Inj Atropine
3. Monitors

Capnography - To monitor End Tidal Carbondioxide (ETCO2) where laparoscopic surgeries are done.

Conduct of Anaesthesia

Anaesthesia can be conducted taking 5 P's into consideration. They are:-

- Pre-operative assessment of Patients.
- Patient preparation.
- Preparation of Operation theatre.
- Premedication.
- Plan of anaesthesia.

1. Preoperative Assessment
   * Elicit proper history.
   * Enquire about associated comorbid conditions like Hypertension / Diabetes / Ischemic heart

disease/ Epilepsy / Allergy to Drugs/ Asthma.

* Enquire about past h/o surgery.
* Evaluate the vital parameters like heart rate, blood pressure, respiratory rate.
* Do proper systemic examination of cardio-vascular system, respiratory system.
* Check the blood investigations like blood sugar,serum creatinine, urine albumin and sugar, ECG, X-Ray Chest.
* Evaluate the patient's airway if difficulty in intubation is anticipated.

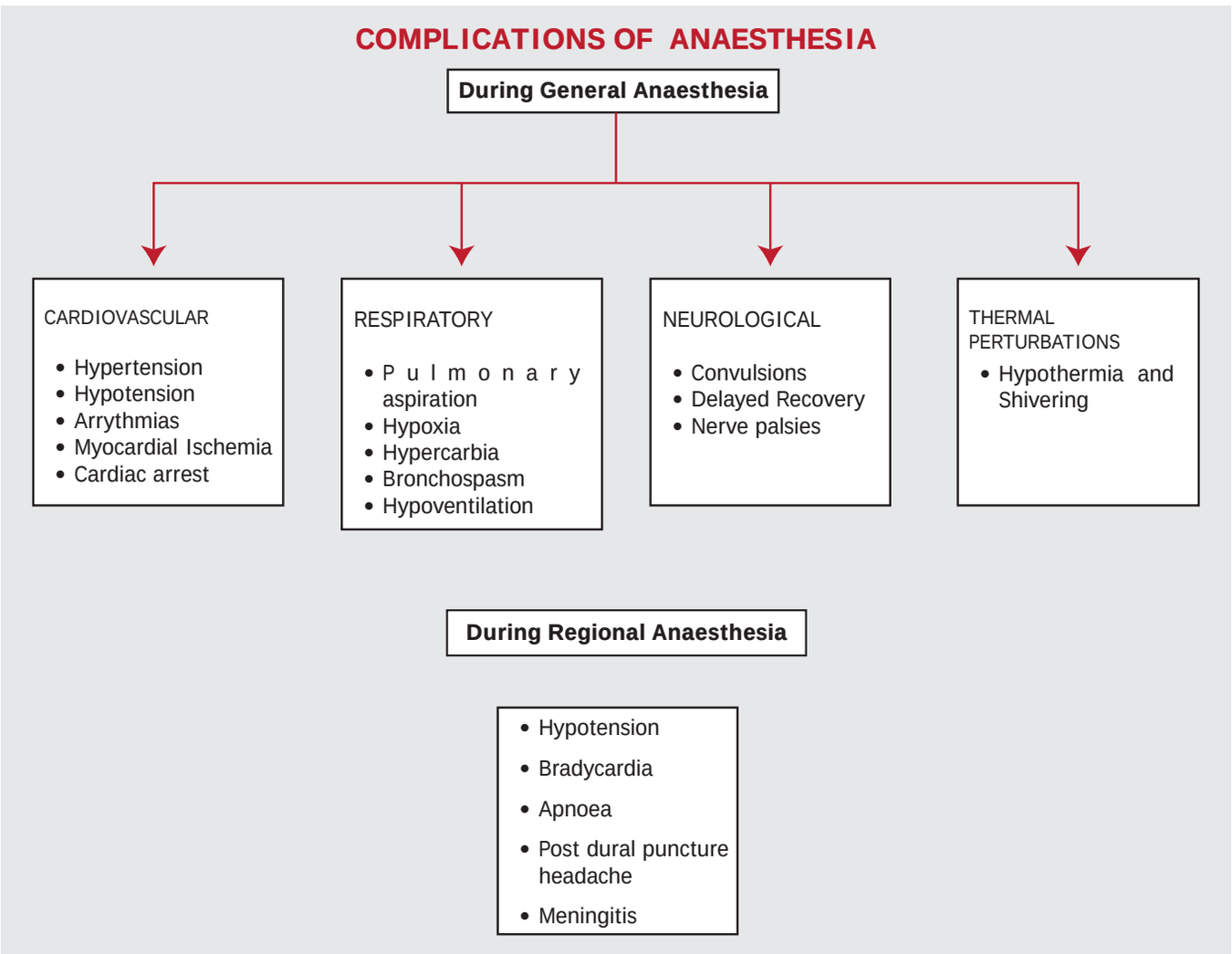
2. Patient Preparation
   * Explain the risks and possible effects of an-aesthesia to patients.
   * Obtain consent for surgery from patient and relatives.
   * Obtain high risk informed consent for life threatening surgeries.
   * Advise appropriate fasting guidelines.
   * Advise to take appropriate medications.
   * Administer lignocaine test dose to the pa-tients and monitor.
   * Administer tetanus toxoid injection to pa-tients.
3. Preparation of Theatre
   * Sterilization of theatre.
   * Perform Boyle's Machine check .
   * Arrange Monitors.
   * Keep intravenous fluids and drugs ready.
   * Keep the Instruments ready.
   * Take necessary steps for disposal of biomedi-cal waste.
4. Pre-medication
   * Pre-medication is the administration of drugs preoperatively to the patients to relieve anxiety ,to induce sedation and to provide analgesia and amnesia.
   * Verify patient and patient documents before administering the drugs.
   * Benzodiazepines and opiods are the com-monly used premedicaments.
   * Administer the drugs Intramuscularly 1 hour before surgery.
5. Plan of anaesthesia
   * Anaesthetic plan is based on the condition of patient, site of surgery, choice of the surgeon and anesthetist.
   * Positioning of patient is important in any anaesthetic plan to avoid pressure on the nerves and vessels.
   * Plan the anaesthetic management that is best suited for the individual patients.

Anaesthesia to different group of patients

1. Hypertensive Patients
   * Advise to take all antihypertensive drugs till the morning of surgery.
   * To avoid ace intibitor on the day of surgery to avoid intrap fall in BP
   * Keep antihypertensive drugs like Nitroglycer-ine, Nifedipine and Metoprolol ready.
   * Keep volume expanders like colloids ready.
2. Diabetic Patients
   * Advise to skip the morning dose of insulin and oral hypoglycemics.
   * Check fasting blood sugar, urine ketones and serum electrolytes on the morning of sur-gery.
   * Place the diabetic patients first in the list of surgery.
   * Specific care of the patients with autonomic disturbance .
   * Keep short acting regular insulin and glucom-eter ready for intraoperative use.
   * Assess target organ function and monitor accordingly.
3. Obstetric Patients
   * Prepare the operation theatre ready for emergency LSCS.
   * Follow anti-eclamptic regimens for patients with pregnancy induced hypertension .
   * Verify the availability of blood.
   * Keep the difficult intubation kit ready.
   * Take care of prophylactic measures to treat aspiration (Mendelson Syndrome).
   * Keep the neonatal care equipment ready (Infant resuscitation bag, smaller endotra-

cheal tubes, laryngoscopy, suction apparatus, infant radiant warmer, oxygen hoods and emergency drugs for resuscitation.

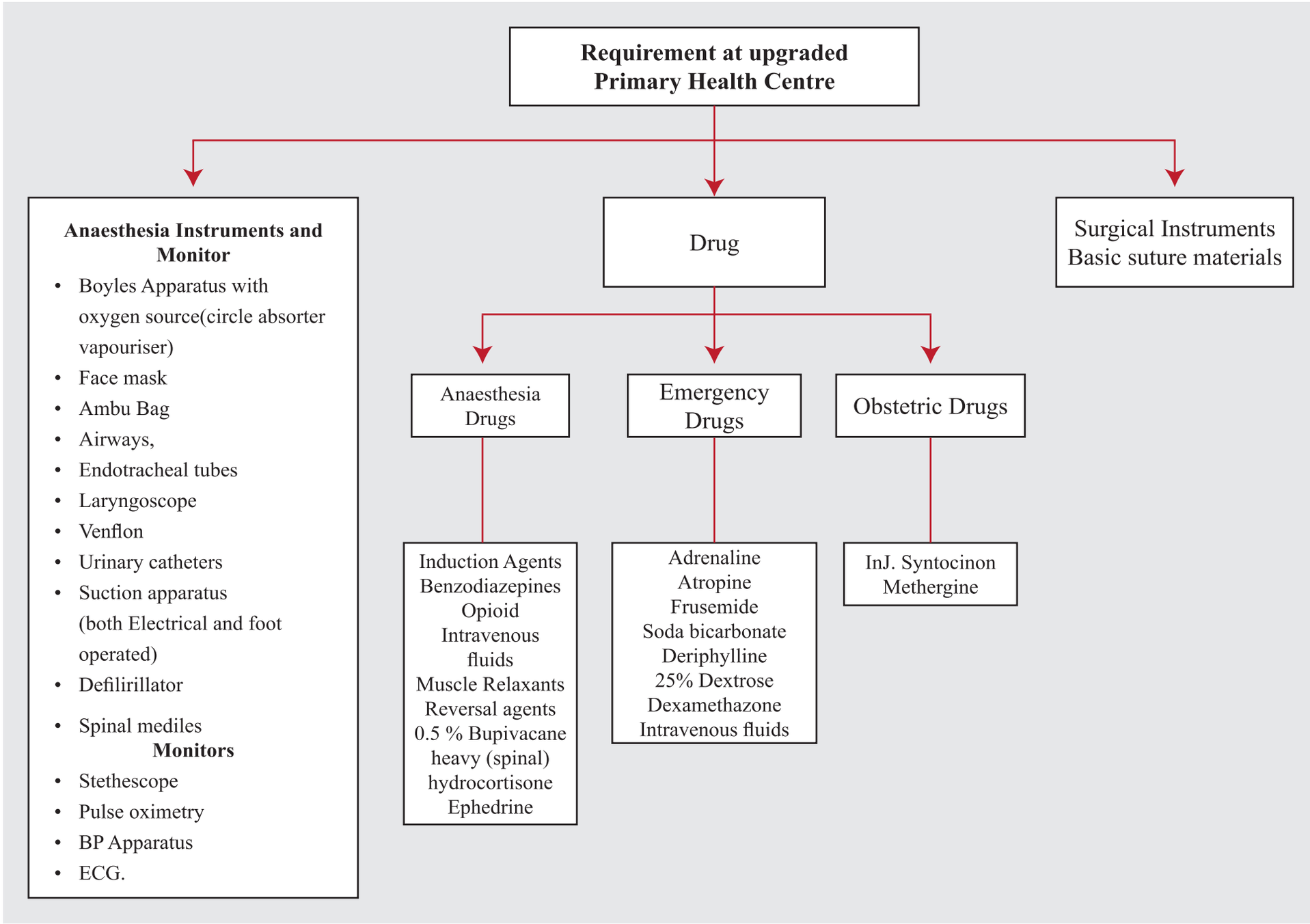
4. Trauma Patients
   * Stabilize the patient taking into consideration ABC (Airway, Breathing and Circulation) of resuscitation.
   * Stabilize the cervical spine (with collar for suspected C-spine injuries).
   * Place the inter costal drainage for chest injuries.
   * Splint the fractures.
   * Assess the GCS (Glasgow coma score). If < 8, intubate for airway protection.
   * Transport the patient to referral centre with portable ventilator, oxygen cylinders and Bain circuit on stand by.
   * Inform the patient details to referral centres prior to or during transport.



Complications Of Anaesthesia During General Anaesthesia

Management of common complications:

1. Aspiration:
   * Follow appropriate fasting guidelines.
   * Administer anti-aspiration prophylaxis like H2 blockers, proton pump inhibitors, metoclopramide.
   * Intubate by Rapid sequence technique in full stomach patients.
2. Hypercarbia
   * Ventilate appropriately as hypoventilation is the commonest cause.
3. Hypertension
   * Maintain good plane of anaesthesia .
   * Use opiods for adequate pain relief.
4. Hypotension
   * Manage blood loss appropriately.
   * Preload the patient adequately before spinal anaesthesia.
5. Post Operative Apnoea :
   * Commonly due to over dosage or sensitivity of barbiturates, opiods and inhalational agents and inadequate reversal .
   * Titrate the doses of opiods. Use appropriate dose to avoid respiratory depression.Use Morphine in a dose of 0.1 mg/kg.
   * Keep Flumazenil (antidote for benzodiazepine) and Naloxone (antidote for opiods) ready, to manage complications of overdosage.
6. Nerve Palsies
   * Position the patient appropriately to prevent pressure on the nerves.
   * In prone position-avoid pressure on eye ball and avoid overabduction of arms to prevent Brachial plexus injury.
   * In lateral decubitus position,keep axillary roll beneath axilla to prevent Brachial plexus injury.
   * In lithotomy position,care must be taken to avoid injury to the lateral popliteal nerve.
7. Hypothermia and Shivering
   * Increase theatre temperature (21°C for adults and 28°C for children).
   * Use warm intravenous fluids.
   * Use warm blankets and forced air warming to combat hypothermia.
   * Use I.V. Pethidine and I.V.Pentazocine to treat shivering.


Requirement at upgraded Primary Health Centre
Anaesthesia Instruments and Monitor
• Boyles Apparatus with oxygen source(circle absorter vapouriser)
• Face mask
• Ambu Bag
• Airways,
• Endotracheal tubes
• Laryngoscope
• Venflon
• Urinary catheters
• Suction apparatus (both Electrical and foot operated)
• Defilirillator
• Spinal mediles
Monitors
• Stethescope
• Pulse oximetry
• BP Apparatus
• ECG.
Drug
Anaesthesia Drugs
Induction Agents
Benzodiazepines
Opioid
Intravenous fluids
Muscle Relaxants
Reversal agents
0.5 % Bupivacane heavy (spinal)
hydrocortisone
Ephedrine
Emergency Drugs
Adrenaline
Atropine
Frusemide
Soda bicarbonate
Deriphylline
25% Dextrose
Dexamethazone
Intravenous fluids
Obstetric Drugs
InJ. Syntocinon
Methergine
Surgical Instruments
Basic suture materials

Requirement in Headquarters Hospital
(In addition to the requirement of PHC)

Anaesthesia Instruments

- Pediatric Mask and circuits
- LMA, Stylet,
- Bougie
- Magills forceps
- Spinal needles
- Intercostal drainages set
- Vaporizers
  - Halothane
  - Isoflurane
- Defibrillator
- Epidural Catheter needle

Monitors

- ECG monitor
- Pulseoximeter
- Capnography monitor if laparoscopic surgeries are conducted.

Anaesthesia Drugs

- Thiopentone
- Propofol
- Ketamine
- Suxamethonium
- Pancuronium,
- Vecuronium
- Neostigmine
- Fentanyl
- Morphine
- Benzodiazepine Antidotes
  - Flumazeanil
  - Naloxone
- Nitroglycerine
- Betablockers

Obstetric requirements

- Magnesium Sulphate
- Prostaglandins
- Ranitidine
- Metoclopramide
- Other anti hypertensives

CONDUCT OF ANAESTHESIA (5 Ps)

Preoperative Assessment of patients

- History
- Blood group
- Co-morbid conditions
- Previous History of allergy
- Examination of respiratory system
- Blood investigation
- Airway evaluation
- History of epilepsy
- Details of drug regimen of the patient

Patient Preparation

- Explanation of procedure and risks.
- Consent for surgery
- Fasting guide lines
- Tetanus prophylaxis
- Lignocaine test dose
- Advise regarding any alterations in drug administration before surgery in patients with co-morbid conditions.

Preparation of theater

- Sterilisation of the operation theatre
- Machine check
- ensure working condition of monitors.
- IV fluids and drug availability
- Bio-medical waste disposal arrangements.

Premedication

Benzodiazepine or opioid and anti - sialogogue one hour before surgery

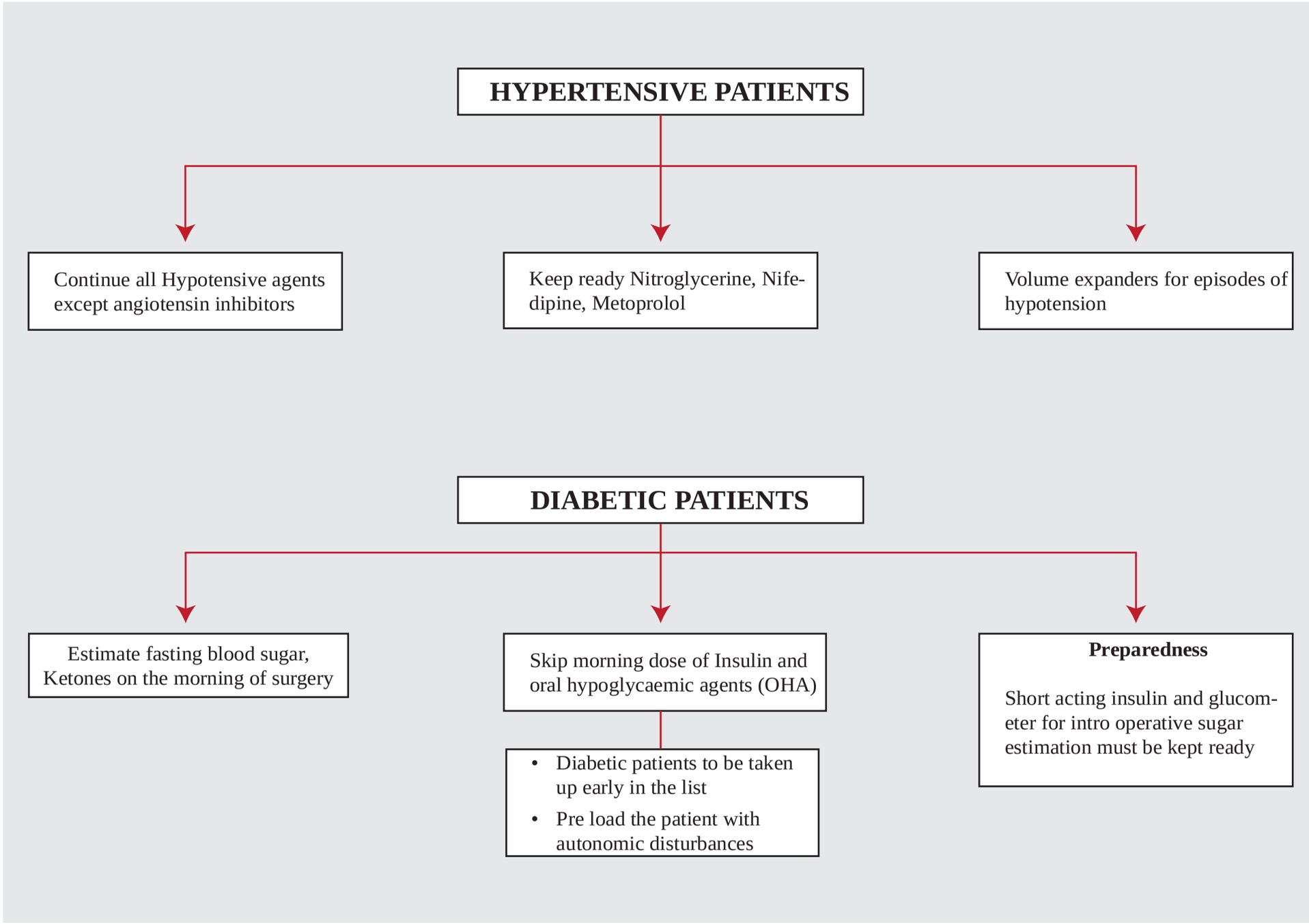
Plan of anesthesia

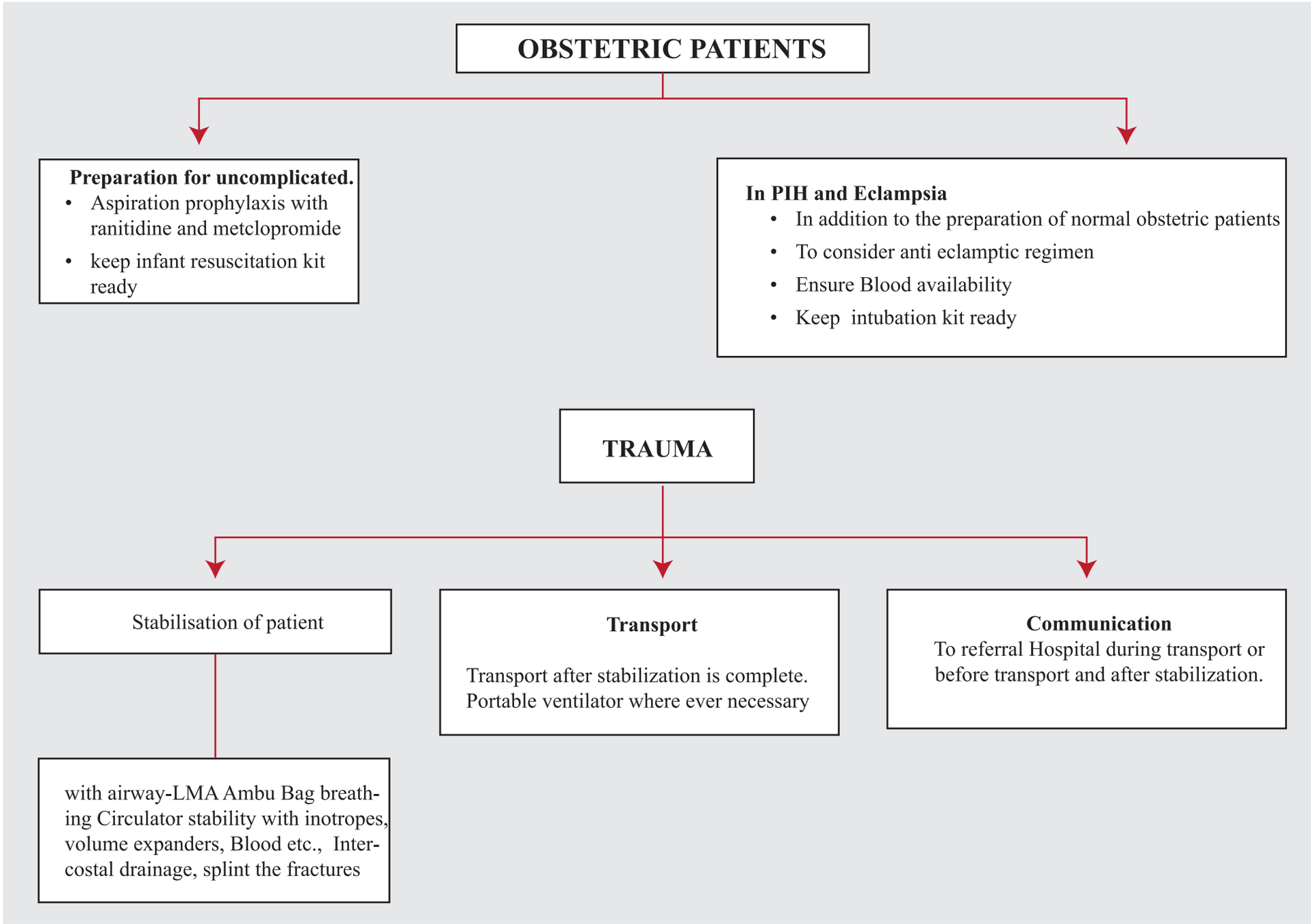
Plan depend on patients' condition

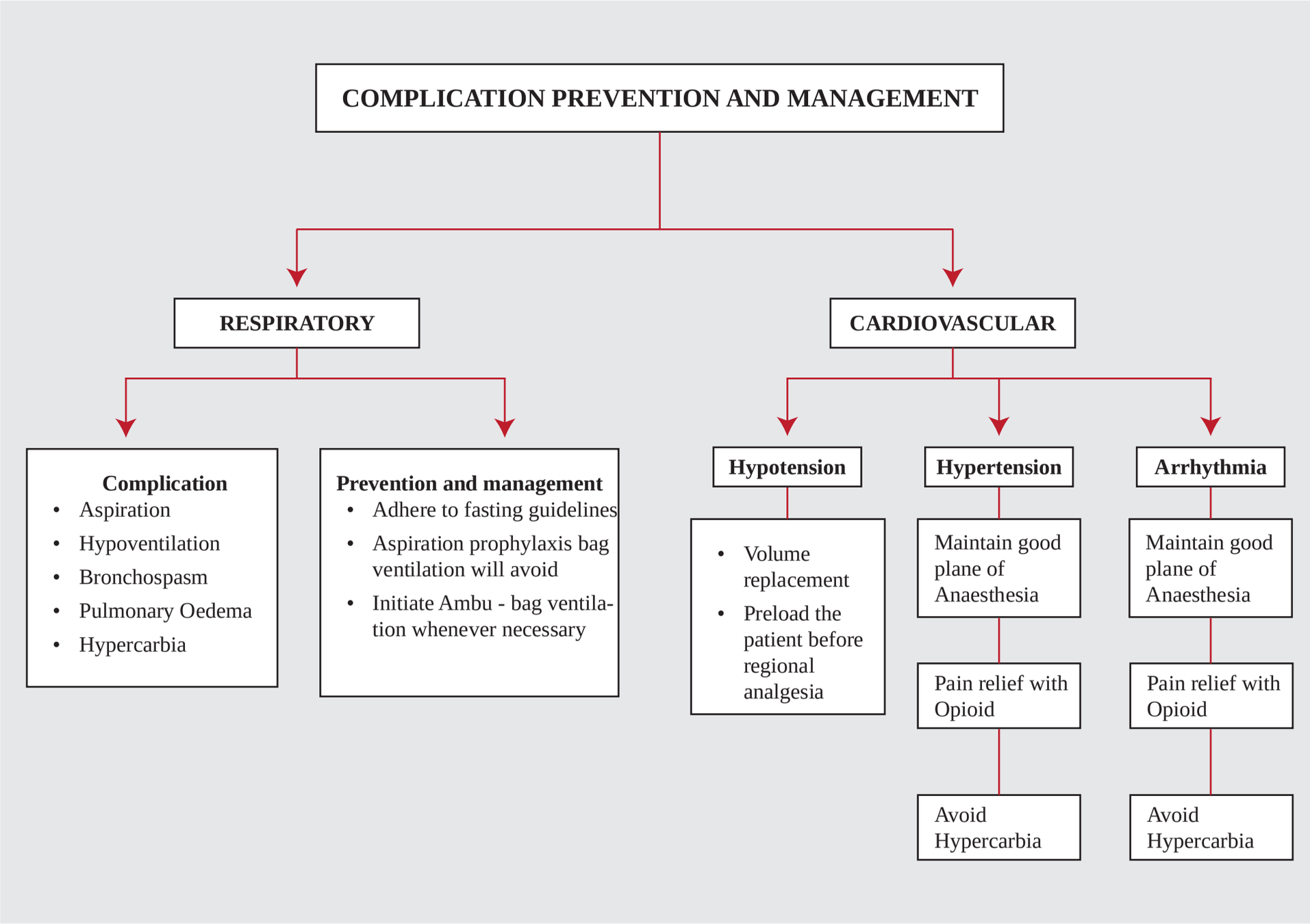
- Hypertensives
- Diabetes mellitus
- Obstetric patients
- Trauma.

Site of surgery

Careful positioning of the patient. Avoid pressure on nerves and vessels.


HYPERTENSIVE PATIENTS
Continue all Hypotensive agents except angiotensin inhibitors
Keep ready Nitroglycerine, Nifedipine, Metoprolol
Volume expanders for episodes of hypotension
DIABETIC PATIENTS
Estimate fasting blood sugar, Ketones on the morning of surgery
Skip morning dose of Insulin and oral hypoglycaemic agents (OHA)
• Diabetic patients to be taken up early in the list
• Pre load the patient with autonomic disturbances
Preparedness
Short acting insulin and glucometer for intro operative sugar estimation must be kept ready


OBSTETRIC PATIENTS
Preparation for uncomplicated.
• Aspiration prophylaxis with ranitidine and metclopromide
• keep infant resuscitation kit ready
In PIH and Eclampsia
• In addition to the preparation of normal obstetric patients
• To consider anti eclamptic regimen
• Ensure Blood availability
• Keep intubation kit ready
TRAUMA
Stabilisation of patient
with airway-LMA Ambu Bag breathing Circulator stability with inotropes, volume expanders, Blood etc., Intercostal drainage, splint the fractures
Transport
Transport after stabilization is complete. Portable ventilator where ever necessary
Communication
To referral Hospital during transport or before transport and after stabilization.


COMPLICATION PREVENTION AND MANAGEMENT
RESPIRATORY
CARDIOVASCULAR
Complication
• Aspiration
• Hypoventilation
• Bronchospasm
• Pulmonary Oedema
• Hypercarbia
Prevention and management
• Adhere to fasting guidelines
• Aspiration prophylaxis bag ventilation will avoid
• Initiate Ambu - bag ventilation whenever necessary
Hypotension
• Volume replacement
• Preload the patient before regional analgesia
Hypertension
Maintain good plane of Anaesthesia
Pain relief with Opioid
Avoid Hypercarbia
Arrhythmia
Maintain good plane of Anaesthesia
Pain relief with Opioid
Avoid Hypercarbia

COMPLICATION PREVENTION AND MANAGEMENT (contd...)

NEUROLOGICAL

Post operative Apnoea

- Common cause
  - * Over dose of narcotics
  - * Manage with antidotes
- Apnoea in spinal anaesthesia
  - * complication of total spinal
  - * manage aggressively with establishing ventilation

Convulsion

- Manage with anticonvulsants and look for the cause and act accordingly

Nerve palsy

- Can be avoided with proper positioning
- In prone position-avoid pressure on eyeball, over abduction of arms
- Axillary roll to avoid brachial plexus injury
- In lithotomy position, avoid injury to lateral popliteal nerve.

THERMAL PERTURBATIONS

Hypothermia and Shivering

- Improve theatre temperature
- Use warm blankets
- Forced air warming
- Intravenous fluids
- I.V Pethidine or I.V Pentazocine

Malignant Hyperthermia

Refer text

Surgical Topics

Chapter 16

1. Varicose veins.
2. Deep vein thrombosis.
3. Lymphadenopathy and lymphoma.
4. Breast disease.
5. Hernia.
6. Hydrocele.
7. Anal fissure.
8. Haemorrhoids.
9. Fistula in ano.
10. Blunt injury abdomen.
11. Peptic ulcer disease.
12. Carcinoma stomach.
13. Appendicitis.
14. Intestinal obstruction.
15. Jaundice.
16. Cholelithiasis.
17. Emergency surgical procedures.

Varicose veins

Varicose veins are dilated, tortuous veins caused by dilation of normal veins due to increased venous pressure. They are visible surface manifestations of an underlying syndrome of venous insufficiency.

They occur in varying severity and in different clinical presentations. 2% of these patients present with skin changes and morbid chronic venous ulcers.

Causes

- Hereditary, history of phlebitis
- Female, pregnancy, pelvic tumours
- Prolonged standing
- Varicose veins may also occur secondary to DVT and arteriovenous malformations.

Varicose veins may involve:

- Greater saphenous vein, (GSV)
- Lesser saphenous vein (LSV) with their tributaries (or)
- Perforator veins, which connect them to the deep venous system of the leg
- Combination of the above veins

Clinical presentation depends upon the severity of the disease:

Symptoms

- Aching pain in the leg
- Tiredness
- Discomfort
- Ankle edema
- Eczema
- Hyperpigmentation
- Lipodermatosclerosis
- Venous ulceration

Signs

Clinical examination is aimed at assessing the presence and anatomical distribution of venous reflux and the patency of deep veins. The clinically relevant tests performed at the bedside are:

Trendelenburg test – To assess saphenofemoral or Saphenopopliteal valve reflux

Three bandage test – To assess site of perforator reflux

Perthes test – To assess patency of deep venous system

Examination of the pulse in both lower limbs is mandatory

Patients are categorized according to the clinical, etiological, anatomical and pathological (CEAP) status in order to serve as a guide to therapy, and for documentation or follow up purposes.

CEAP Classifications	
C - CLINICAL 0-no visible signs of venous disease Telangiectasia/reticular veins Varicose Veins Edema Skin changes including pigmentation, Venous eczema, lipodermatosclerosis Healed ulceration Active ulcer A- Asymptomatic S- Symptomatic	A - Anatomical Distribution Superficial veins Deep veins Perforator veins Alone or in combination
E - Etiological Primary Secondary Congenital	P - Pathophysiological dysfunction Obstruction Reflux Combination of both

Investigations

- Basic investigations
- USG abdomen
- Doppler study
- Duplex scan (mandatory)
- Venogram (in selected patients)

Clinical approach to varicose veins

Treatment

Compression therapy:

Graduated compression stockings are used The commonly used stocking is class ii with pressure of 24 to 32 mm hg (they aid in fluid resorption, resolution of edema, thereby increasing nutrients to skin and subcutaneous tissues, skin capillary density and cutaneous oxygenation.)

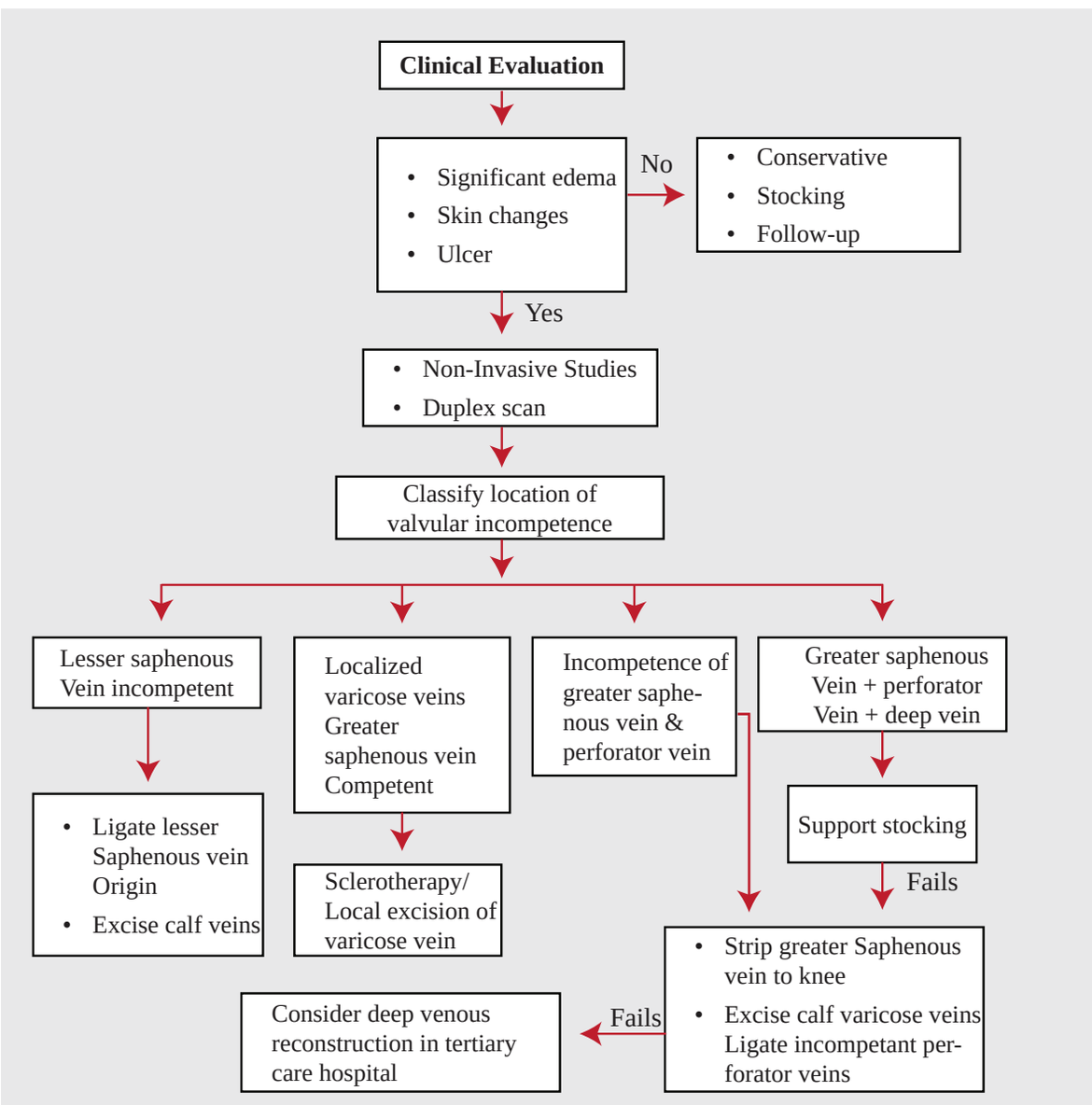
Pharmacological therapy

- Topical zinc cream application
- Venotonic drugs like flavonoids
- Hemorheological agents have adjuvant role

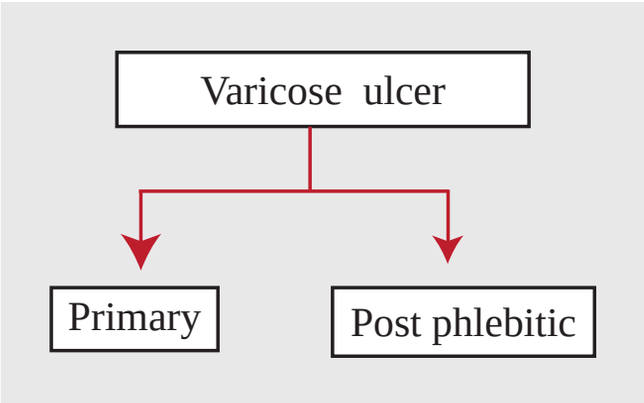
Trendelenberg procedure:

- Flush ligation of the saphenofemoral junction with stripping of the GSV upto below knee.
- Saphenopopliteal reflux is treated by flush ligation of the saphenopopliteal junction
- In tertiary care hospitals, newer modalities for treatment include
  - * Laser/ radiofrequency ablation of the GSV.
  - * Stab avulsion phlebectomy, or
  - * SEPS through subfascial endoscopic perforator surgery

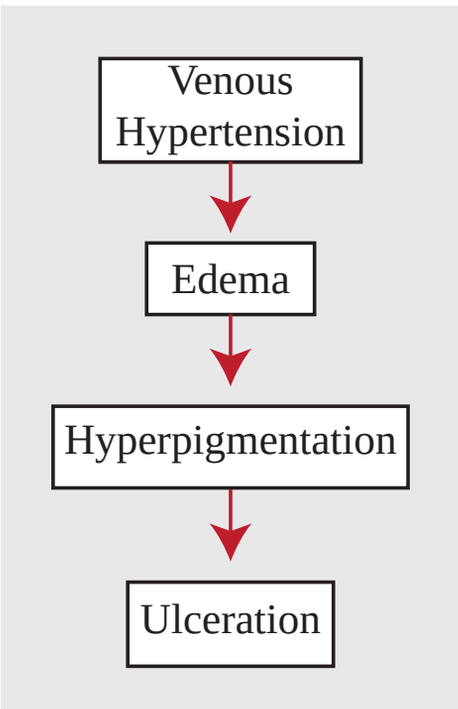
Sclerotherapy is used as a treatment option for reticular veins, telangiectasia, recurrent, residual and isolated varicose veins. Commonly used agents are sodium tetradecyl sulfate, polydocanol and ethanol and they are injected carefully to avoid extravasation and skin necrosis. Contraindication to sclerotherapy is allergy to the sclerosing agents.



Varicose ulcer Classification



PATHOGENESIS



Treatment

- Reduce venous hypertension by elevation and compression
- Small ulcer - treat infection and allow primary healing
- Large ulcer - treat the curable cause; split skin graft if necessary
- Post phlebitic, evaluate further, conservative management.

Conservative management

- Compression therapy
- Graded compression stockings
- Medical care / supportive, aim wound healing, promotes granulation
- Control infection with appropriate antibiotic
- Reduce the edema by elevation
- Blood transfusion if necessary
- Treatment of primary cause

Venous haemorrhage:

- Due to varicose veins and venous ulcer
- Bleeds profusely on minor trauma

Treatment

- Compression,
- Elevation,
- Blood transfusion if necessary,
- Treatment of primary cause,
- Regular dry dressing of the wound.

Deep vein thrombosis

Definition

Deep vein thrombosis is the formation of a clot in one of the deep veins of the body commonly in the deep veins of the lower limb.

The blood clot can partially or completely block the flow of blood. DVT may arise spontaneously or after injury or immobility to the limb. The complications of DVT such as pulmonary embolism and post-thrombotic syndrome are the most common cause of hospital death and source of substantial long-term morbidity.

- Calf vein DVT is the commonest
- Ileofemoral DVT is the most symptomatic
- Inferior vena cava thrombosis is the most lethal

Causes Three 'I's

- Individual related
  - Smoking
  - Hormone replacement therapy
  - Obesity
  - Long distance air /car travel
- Illness / injury related
  - Previous DVT
  - Hypercoagulable states
  - Myocardial infarction
  - Trauma
  - Advanced cancer

- Intervention related
  - * Recent surgery
  - * Immobilization/paralysis
  - * Central venous catheterizations
  - * (Three D's – Diameter, Drugs, Duration)

Symptoms	Assessment
Asymptomatic or silent Symptomatic • Pain • Limb swelling • Pyrexia • Dilated superficial veins • Tenderness along the deep veins • Pulmonary embolism	• Moses'sign – pain on pressing the calf muscle is the most reliable • Homans'sign-pain in the calf on dorsiflexion of the foot * not reliable * may promote embolisation and hence should not be done

Progression of the disease

- DVT may lead to limb threatening complications
- Phlegmasia alba dolens (painful white leg)
- Thrombosis involving both superficial and deep veins, phlegmasia cerulea dolens (blue leg).
- They may also primarily present with symptoms of pulmonary embolism.

Important Note

Any unilateral limb swelling must be considered to be DVT unless proved otherwise.

Differential Diagnosis

- Cellulitis
- Arthritis
- Muscle spasm
- Muscle haematoma
- Ruptured Bakers cyst
- Kidney/heart/liver failure
- Lymph edema

Investigations

- Complete blood count
- Clotting profile
- D-dimer estimation
- Hypercoagulation screening
- Doppler ultrasound
- Duplex scanning (gold standard)
- Venography
- Magnetic resonance venogram

Treatment of Deep vein thrombosis

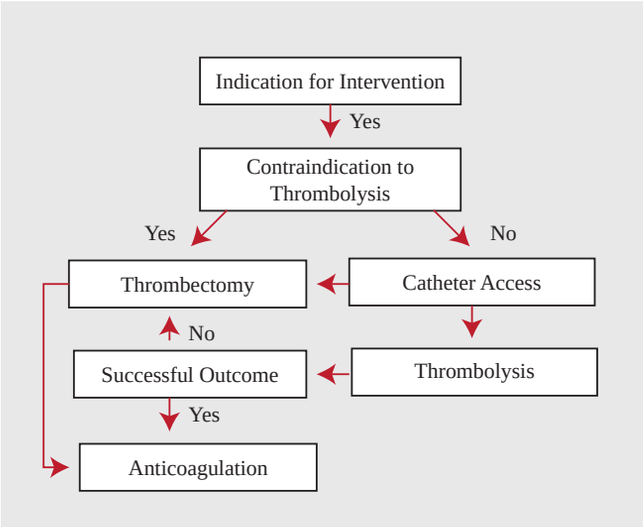
- Absolute bed rest
- Limb elevation
- Early mobilization
- Compression stocking
- Unfractionated heparin
- Low molecular weight heparin

In tertiary care hospitals

- Catheter directed thrombolysis
- Venous thrombectomy
- IVC filters limited indication in patients with ileofemoral thrombosis in whom heparin therapy is contraindicated and patients with pulmonary embolism with poor cardiorespiratory reserve who will not tolerate another bout of embolus.

All patients with DVT have to be given oral anticoagulants for a minimum period of six months: coumarin derivative (warfarin).

Ilio-Femoral Thrombosis



DVT prophylaxis

DVT prophylaxis is mandatory in patients undergoing surgery. According to the level of risk, patients are categorized into low, medium, high and highest risks. Early diagnosis and prompt treatment with prophylaxis whenever indicated can reduce the mortality (pulmonary embolism) and long-term morbidity (chronic venous insufficiency) remarkably.

DVT risk level and prophylaxis:

Lymphadenopathy and lymphoma

Lymphedema

Lymphatics drain the lymph from the tissue spaces into the veins. Hence they are vulnerable to various infections and enlargement of lymph node occurs.

Definition

Lymphedema is due to accumulation of lymph in the subcutaneous extra cellular extra vascular compartment resulting in enlargement of body parts.

Common sites:

Lower limbs, upper limbs, scrotum, penis, and breast

Lymphedema of leg: Types:

- At birth- lymphedema congenita
- At puberty – lymphedema precox
- At later life –lymphedema tarda

Primary and Secondary

Primary - Lymphatic aplasia, Lymphatic hypoplasia, Milroy's disease – familial

Secondary (Acquired)

- Filariasis – common, associated with fever, chills, rigor, red streaks and painful nodes
- After repeated infection
- Malignant infiltration and obstruction of lymph nodes
- After block dissection of inguinal or axillary nodes
- Following radiotherapy to lymphnodes

Risk level	Description	Prophylaxis
Low	Minor surgery, no risk	No specific measures, early mobilization
Medium	Minor surgery with risks	Low density unfractioned heparin (LDUH) Low molecular weight heparin (LMWH) Elastic stockings (ES) Intermittent pneumatic compression (IPC)
High	Major surgery with risks	Low density unfractioned heparin(LDUH) Low molecular weight heparin(LMWH) Intermittent - pneumatic compression (IPC)
Highest	Major surgery Prior VTE Molecular hypercoagulable state, (cancer, hip or knee surgery, major trauma, spinal cord injury)	LMWH IPC/ES + LDUH/ LMWH Oral anti-coagulants

Treatment

Conservative

- Elastic compression bandage
- Elevation
- Exercise and massaging
- Avoid skin injuries
- Antibiotics, diuretics, Diethylcarmapazine, warfarin used to treat medically.

Surgical

- Reduction procedures
  - * Subcutaneous excision and grafting
  - * Excision and primary skin suturing
- Lymphatic bypass procedures
  - * Nodo-venous shunt
  - * Omental transposition
- Excision with bypass
  - * Swiss roll operation- dermal flap buried deeply

Tuberculous lymphadenopathy

Tuberculous lymph adenits is the most common lymph node enlargement in the younger age group. The incidence is reducing due to improved nutritional status and improved health care facilities.

Characteristic features:

Usually cervical lymph nodes are affected. 80% are from the tonsillar crypt involving the jugulo-digastric nodes in the anterior triangle. The posterior triangle nodes commonly involved from adenoids. Rarely supraclavicular nodes are involved secondary to lung involvement or the ileo cecal region.

Symptoms

- Neck node enlargement is the usual presentation
- Matting of nodes in tuberculosis is due to periadenitis.
- Nodularity is due to matted lymph nodes
- Collar stud abscess and sinus are seen in advanced stages.
- Constitutional symptoms such as evening rise of temperature, loss of appetite and weight may be present.

Stages of TB lymphadenitis

1. Stage of lymphadenitis
2. Stage of periadenitis
3. Stage of cold abscess
4. Stage of collar-stud abscess
5. Stage of sinus

Investigations

TC, DC, Hb% ESR

X-Ray chest

Biopsy of the lymph node

Treatment

- As per Revised National Tuberculosis Control Programe

Effect of treatment

- Improvement in general health
- Regression of the lymph node enlargement,
- Return of appetite and weight gain
- Esr will come down

Role of surgery:

For diagnostic purpose – FNAC, Biopsy

For therapeutic purpose – Non-dependent drainage of collar stud abscess

Excision of fibrotic nodes (residual) after ATT completion

Excision of sinus and scars

Important Note

- Suspect TB in chronic lymphadenitis in the younger age group
- Confirm by biopsy of the node.
- Ensure complete course of the treatment to prevent drug resistance.
- Screen family members, prevent contact with open cases (sputum positive)
- Follow-up long term
- Suspect concomitant HIV infection

Lymphoma

Lymph nodes may be enlarged due to neoplastic changes Suspect lymphoma if the nodes are:

- Painless
- Non-tender
- Soft
- Progressive
- Non-resolving with antibiotic or ATT

Hodgkins lymphoma

It is a lympho-reticular malignancy involving lymph nodes, spleen, liver etc.

Characterized by painless nodal enlargement, not matted and involvement of midline (axial) systems.

Usually idiopathic, Epstein Barr viral infection is also postulated as an etiological agent.

Classification

REAL classification (Revised European American Lymphoma)

1. Lymphocyte predominant nodular
2. Classic Hodgkin's lymphoma
3. Nodular sclerosis
4. Lymphocyte rich
5. Lymphocyte depletion
6. Mixed cellularity

Symptoms

- Bimodal involvement- in children and middle age (30-50 yrs)
- Males are mostly affected
- More than one group of nodes are involved
- Nodes are hard and more than 2cms
- Mostly starts in the left posterior triangle of neck as bunch of grapes
- Contiguous and centripetal spread involving the neck, axilla, mediastinum, para aortic and inguinal region
- Smooth, firm, round bordered painful hepatosplenomegaly
- Ascites
- Intermittent irregular fever
- Osteoblastic painful secondaries, mainly lumbar vertebrae
- Superior venacaval obstruction in case of mediastinal node involvement

Clinical Staging

Stage I

* Involvement of one anatomical region or
* Single extra lymphatic organ or Single extra lymphatic site

Stage II

* Involvement of two or more groups confined to same side of diaphragm

Stage III

* Involvement of nodes on either side of diaphragm with or without spleen

Stage IV

* Involvement of one or more extra lymphatic organ with or without

Lymphnode.

H – Liver

P – Pleura

M- Marrow

O- Bones

A – Asymptomatic
B – Symptomatic

- Fever above 38o C or night sweats
- Pruritis
- More than 10% weight loss in 6 months
- Bone pain
- Anaemia

Investigations

- Complete blood count
- Hemogram - anaemia in advanced stage
- Peripheral smear – to rule out leukemia
- Blood urea and creatinine – in renal involvement or in extraneous uretral obstruction
- Chest X ray – mediastinal nodes and pleural involvement
- Ultrasonogram abdomen – para-aortic nodes, liver and spleen involvement
- C.T scan abdomen - minimal nodal involvement
- Intravenous pyleogram – renal excretory function

- Excision biopsy of the node – to confirm the pathological diagnosis
- Mediastinoscopy – in isolated mediastinal node involvement
- Bipedal lymphangiography – rarely done

Treatment

Stage I and II - Radiotherapy

Depending on the region involved

- Local regional – one group involvement
- Mantle field – bilateral neck
- Extended mantle field – neck and mediastinum
- Inverted Y field – para aortic and bilateral inguinal nodes

Stage III and IV - Chemotherapy

MOPP regime

M – mechlorethamine 6mg/m2 Day 1 and 8

O – Oncovin (vincristine) 1.4mg/m2 Day 1 and 8

P – Procarbazine 100 mg oral Day 1 to 10

P – Prednisolone 15 mg 8th hourly Day 1 to 10

* Minimum of 6 cycles or 2 cycles after complete remission
* 10 year survival rate is 80%

Complications:

Infertility, acute myeloid leukemia, bone marrow suppression etc.

Other regime:

ABVD: (Adriamycin, Bleomycin,Vinblastine, Decarbazine)

Staging Laparotomy:

In stage I and early stage II cases:

(Especially supra diaphragmatic) It includes

- Splenectomy with its hilar nodes for biopsy
- Wedge and needle biopsy of both the lobes of liver
- Para-aortic lymph node biopsy
- Iliac and celiac node biopsy
- Iliac crest bone biopsy
- Oophoropexy in young females

Non-Hodgkins lymphoma

Causes

Physical

- Radiation

Chemical

- Benzene
- Pesticides
- Petroleum products

Biological

Viral eg : EBV, HSV 8

Bacterial eg: H.pylori

Genetic eg: Ataxia telangiectasia

Autoimmune disorders - Sjogren's syndrome.

Classification

Working formulation of NHL

* Low grade
* Intermediate grade
* High grade

Pathological

- B - cell NHL
  * Small Lymphocytic lymphoma
  * Follicular lymphoma
  * Burkitt's lymphoma
- T - cell NHL
  * Cutaneous T-cell lymphoma
  * Mycosis fungoides
  * Sezary syndrome
  * Lymphoblastic lymphoma

Extra-lymphatic involvement:

- Seen only in non-Hodgkin's lymphoma
- GI tract is the most common site

Investigations

- Peripheral blood smear must be done to rule out

leukemia

- Lymph node biopsy for confirmation of lymphoma, excise the largest and significant node completely

Treatment

Radiotherapy

Stage I and II (Low grade)

Chemotherapy

Stage I and II (intermediate and high grade)
Stage III and IV

Role of Surgery in Lymphoma:

- Lymphnode / extralymphatic site biopsy for diagnosis
- MALT lymphoma, early stages, post-RT residual disease (eg- total parotidectomy, total thyroidectomy, gastrectomy)
- MALT lymphoma, early stages, primary surgical treatment (eg – lung and intestine)
- Primary testicular lymphoma - orchidectomy

Important Note

- Any swelling in the neck is lymph node enlargement unless proved otherwise
- Excision biopsy is preferred to incision biopsy
- Examination is not complete without looking for the draining areas of the nodes
- In younger age group rule out infective causes and primary malignancy
- Rule out secondary nodal deposits in the elderly

Benign Breast Disease

Breast disease is associated with psychological, emotional and social problems. About 30% of women suffer from benign breast disease in their life time. Commonly present as breast lump or pain.

The aim of treatment is to cure the condition, to treat symptomatically and to give psychological relief to the patients.

Benign lesions of the breast:

Fibrocystic Disease - ANDI (aberration of normal development and involution) Fibroadenomatosis, cyclical nodularity, cysts and fibroadenoma occur as a result of aberration of normal development and involution, due to disturbance in the normal cyclical changes occurring in the breast during the menstrual cycles. Changes occurring in the breast are cyst formation, fibrosis, hyperplasia of duct acinar epithelium and papillomatosis.

Symptoms

Lump seldom discrete, common in upper outer quadrant, usually bilateral with cyclical mastalgia.

Treatment

- Reassurance
- Periodical checking (self-examination of breast)
- Drugs – Evening primrose oil, danazol, tamoxifen, analgesics.

Benign neoplasm

- Epithelial
  - * Duct Papilloma
  - * Pure adenoma
- Connective tissue
  - * Neurofibroma
  - * Lipomas
- Mixed Fibroadenoma

Duct Papilloma

Single

From large lactiferous duct

Bloody discharge

Age 35-50

Premalignant

Treatment

Microdochectomy

Fibro adenoma (breast mouse)

- Common in18-28 years
- Single lobule hyperplasia
- Freely mobile mass
- Well encapsulated lesion
- Giant fibroadenoma (size>5cms)
- Treatment: Enucleation, Incision - circumareolar, avoid radial incision and in exposed parts of chest.

Phyllodes Tumour (Serocystic disease of Brodie cystosarcoma phyllodes)

- Commonly seen above 40 years of age
- Large bosselated, mobile, rarely cystic lesion
- Spreads via blood stream
- Wide local excision
- Mastectomy (subcutaneous with nipple and areola preserved) in recurrent tumour.

Breast Abscess

Causes

- Abscess occurs in breast feeding women.
- Caused by highly virulent strains of penicillin resistant staphylococcus aureus and anaerobic streptococci.
- Abscess usually large and fluctuation may not be present.
- Antibiotics when given for long periods results in antibioma.
- Breast abscesses can be classified into mastitis neonatorum, lactating epidemic or sporadic mastitis, and non-lactating breast abscesses.

Treatment

Non-pharmacological:

Rest and support to the breast and to continue breast feeding from both the breasts, however, in case of larger abscess shift to bottle feeding. Milk should be evacuated by mechanical means, breast pump, to prevent painful engorgement of breast.

Pharmacological:

In early stage (induration only):

- Antibiotics and anti-inflammatory drugs
- In case of no improvement of large abscess:
- Antibiotics as above.
- Incision and drainage of pus through thinned skin over the abscess (Large abscesses require operation under intercostal block or general anaesthesia)
- Daily dressing.
- In some cases suppress lactation with hormones if the mother finds breast-feeding too painful.

Patient education

- To maintain good hygiene and to continue breast feeding from both the sides unless it is a large abscess and very painful
- Advise on timely weaning of the infant.

Important Note

- Avoid unnecessary antibiotics to prevent antibioma
- Do not wait for fluctuation in breast abscess
- Aspirate with wide bore nedle to confirm diagnosis
- Always drain dependently
- Avoid incision in the upper inner quadrant

Early Breast Cancer

Definition

Early breast cancer is defined as the condition in which the tumor is confined to the breast Size >2 cm with no obvious metastasis.

TNM classification is internationally accepted and widely used to describe the extent of the tumour; presence of regional lymph nodes and metastasis- early breast cancer is up to the T2N1M0

T.N.M. Classification

T – Primary tumour

T0- No demonstrable tumour

T1S- Carcinoma in situ, Non-infiltrating intraductal carcinoma, Paget's disease of nipple without tumour

T1- Tumour diameter 2 cm or less without fixation

T2- 2 cm or 5 cm without fixation

T3- Tumour greater than 5cm

T4 -Direct extension of a tumour of any size to skin or chest wall (including peau d' orange)

Skin tethering and nipple retraction does not affect T classification

N – Regional lymph nodes

N0 -No palpable homolateral axillary nodes

N1- Mobile homolateral axillary nodes

N2- Fixed homolateral axillary nodes

N3- Homolateral supraclavicular, infraclavicular or

oedema of arm

M – Distant metastases

M0 - No distant metastasis M1- Distant metastasis

Screening for Breast Cancer

- Beneficial in women over 50 years of age.
- Screening detects breast cancer early (primary tumour is smaller and lower incidence of lymph node metastasis.
- Early detection of breast cancer by a year (clinical examination and mammography) lowers death rate in screened patients

High Risk factors

- Strong family history of breast or endometrial cancer
- Carcinoma of the contralateral breast
- A history of intraductal papilloma or intraductal carcinoma
- Childless women and those conceiving after 30 years of age
- Breasts with dense parenchyma with prominent duct patterns (Fatty breasts with minimal connective tissue elements are at least risk)

Diagnosis of Breast Cancer

Triple assessment includes:

1. Physical examination of the breast (self examination of breast fortnightly)
2. Aspiration cytology
   Cells of epithelial origin can be examined within minutes of sampling. False-positive and false-negative reporting is rare.
3. Imaging methods/ Mammography
   * Mammography is useful to detect non-palpable breast cancer and for screening
   * Routine mammographic screening should not be performed under 50 years of age (risk of radiation carcinogenesis due to cumulative irradiation following repeated mammography.
   * The incidence of false-positive and false-negative reporting is high (10-20 per cent on average)

Biopsy

- Trucut biopsy of the lesion for histopathological
- ER, PR status
- Her2-neu status

Rx Treatment

Aim

- To achieve a cure
- To conserve the breast if possible
- To achieve loco regional control of disease

Treatment options

- Surgical management
- Radiotherapy
- Chemotherapy
- Hormonal therapy

Surgical management:

Surgical management includes

- Modified radical mastectomy (Patey's) (Pectoralis major muscle and nerve to lattismus dorsi are retained)
- Radical mastectomy (Halsted) (Both pectoralis major and minor along with the nerve is removed with loss of anterior axillary fold)
- Breast conservative procedures (quandrantectomy lumpectomy and wide local excision)
- Simple mastectomy for ulcerated, foul smelling lesions of the breast (toilet mastectomy- nipple areolar complex with the involved skin and breast tissues are removed)
- Breast reconstruction and cosmetic surgeries after complete treatment

Radiotherapy:

Post-operative radiotherapy consists of 5000 to 6000 cGy units totally with 200 cGy per day, 5 days per week for 5 to 6 weeks.

Indications for post-operative radiotherapy

- Tumour margin positive
- High-grade tumour
- Axillary clearance not fully possible
- Tumour size more than 5 cms

Hormonal therapy

Depending upon the estrogen receptor status, hormonal therapy is given.

Tamoxifen is the drug of choice in case of estrogen positive patients. Dose - 20mg/day for 5 years.

In older persons >70 years aromatase inhibitors (anastazole , letrazole)

Chemotherapy

Chemotherapy consists of primarily a 3-drug regime

CMF regime - cyclophosphamide, methotrexate and 5-fluorouracil for 6 cycles

Alternate regime includes FAC regime (5-fluorouracil, adriamycin and cyclophosphamide)

Do Blood count Hb TC, DC before every cycle of chemotherapy

Avoid if the WBC count is less than 2000/cmm/l and resuscitate

Advanced Breast Cancer

Advanced breast cancer includes

- Breast cancer staged beyond T2 N1 at presentation
- Recurrent local disease
- Metastatic disease

Rx Treatment

- Simpler hormonal manipulation and major endocrine ablation
- Chemotherapy
- Radiotherapy
- Surgery for the relief of specific complications
- Treatment of hypercalcaemia

Patients likely to respond to hormone manipulation

- Long recurrence-free interval
- Good response to previous hormone therapy
- 5 years post-menopausal women, at presentation with breast cancer
- Advanced local disease without metastases
- Primarily bone metastases rather than soft tissue metastases
- Oestrogen receptor-positive tumours

Hormonal manipulation

- Tamoxifen (Tamoxifen is a synthetic antioestrogen, which inhibits the binding of oestradiol to receptor. High correlation between response and positive oestrogen receptor assay Response rate approaches 50 %)
- Aminoglutethimide (Inhibits adrenal steroid hormonal synthesis, produces a medical adrenalectomy and must be accompanied by adequate glucocorticoid and mineralocorticoid replacement)
- Corticosteroid therapy (Effect is independent of age and menopausal status. Benefits pain from bone metastases and decreases oedema around cerebral and pulmonary metastases. Important in the treatment of hypercalcaemia)
- Oophorectomy (pre-menopausal and early post-menopausal patients, tumour remission rate 30 %)
- Androgen therapy (secondary hormonal therapy, failed remission or reactivation following oophorectomy or tamoxifen)
- Oestrogen therapy (secondary hormonal therapy. Useful in postmenopausal women without evidence of oestrogen secretion. Tumour remission rate increases with increasing years past the menopause, further remission on stopping oestrogen in 30 per cent of responders)

Major endocrine ablation

Bilateral adrenalectomy

- Major surgical procedure
- Fluid and electrolyte management problems with a permanently deficient response to 'stress'

Hypophysectomy

- Contraindicated in the absence of objective response to simpler hormonal manipulation.

Chemotherapy

- Cyclical combination chemotherapy with cyclophosphamide, adriamycin and 5-fluorouracil
- Intra-cavitary alkylating agents by thoracentesis and instillation of nitrogen mustard or thio-TEPA provides good palliation of malignant pleural effusion

Radiotherapy and surgery for the relief of

specific complications Radiotherapy

- To control locally advanced breast cancer without metastases, localized skin and node metastases
- Pain of localized bone metastases, when signs of mediastinal and spinal cord compression and following internal fixation of pathological fracture

Surgery

- Laminectomy for spinal cord compression
- Treatment of pathological fracture by internal fixation
- Prophylactic internal fixation of bone metastases

Treatment of hypercalaemia

- I.V. saline corrects dehydration and provides calcium diuresis
- Corticosteroids
- Discontinue any recently commenced hormonal therapy
- Oral or I.V. inorganic phosphate therapy

Important Note

- Confirm diagnosis in suspected breast lump by FNAC/ biopsy
- Use multi modality treatment
- Give emotional support
- Advice reconstructive surgery
- Follow up long term

Hernia

Definition

Hernia is the protrusion of viscus or a part of viscus through the wall that contains it. The commonest variety of hernia is protrusion of viscus through the abdominal wall eg. inguinal, femoral, umbilical, and incisional.

The hernia is the commonest condition seen in surgical practice.

Symptoms

The presence of inguinal hernia gives rise to swelling in the groin, discomfort and problems in ambulation. The complications of hernia such as obstruction and strangulation pose grave risk to the patients. The emergency surgical correction of strangulated hernia with bowel resection and anastomosis carries high mortality.

Inguinal Hernia

Types of inguinal hernia

Based on clinical presentation

* Complete / incomplete
* Reducible / irreducible / obstructed / strangulated

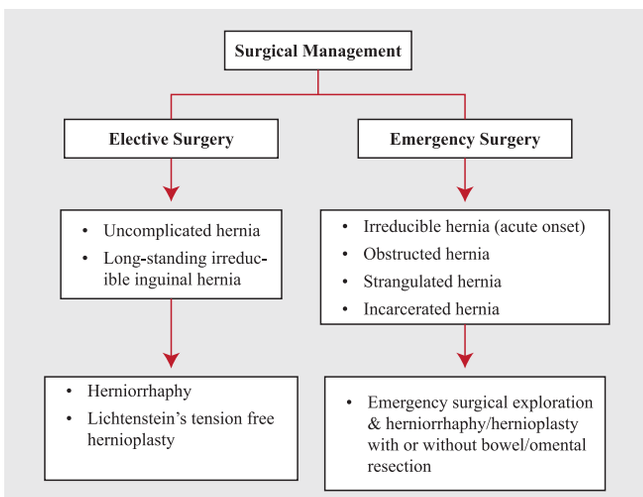
Based on contents

* Omentocele / Enterocele / Cystocele

Special types

* Littre's hernia (when appendix is a content)
* Maydl's hernia (when two loops of intestine gets obstructed at the neck)
* Richter's hernia (when a part of the circumference of bowel gets obstructed)
* Sliding hernia (when a part of the sac is formed by sigmoid colon on the left side and caecum on the right)

Inguinal Hernia



Symptoms	Signs
• Swelling in the groin region following may be present. • Difficulty in passing stools • Difficulty in voiding urine • Chronic cough • Pain (when tendency to herniate, or complicated)	• Swelling in the groin in standing or lying posture. • Cough impulse present. • Reducibility (fully, partly) • Irreducibility and tenderness present if complicated.

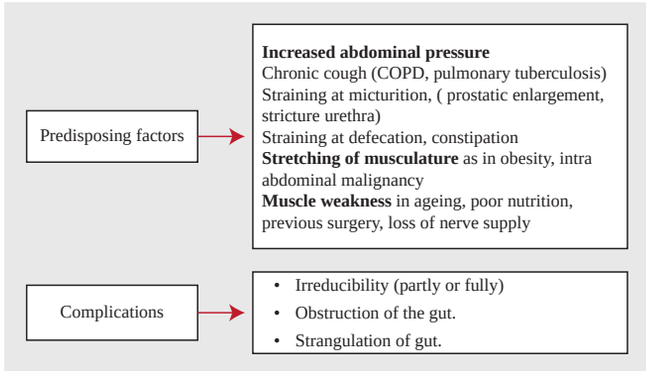
Treatment

Preoperative assessment

- Complete history including medication list
- General examination to identify possible cause of hernia
- Comorbid conditions
- Abdominal examination including digital rectal examination
- Pulse, BP, cardiac workup, ECG, ECHO
- Chest workup. Chest X-ray, pulmonary function test
- Blood sugar estimation to rule out diabetes
- Renal workup, abdomen X-ray, ultrasonogram
- Investigations to rule out intra-abdominal malignancy

Preoperative preparation

- Pre-operative treatment of comorbid conditions (anemia, diabetes, hypertension, IHD)
- Treatment of the cause of hernia (pulmonary tuberculosis, constipation, transurethral resection for benign hypertrophy of prostate)



Herniorrhaphy

- Open the inguinal canal through the inguinal incision.
- Lateralise the ilioinguinal nerve.
- Define inguinal ligament and conjoint tendon.
- Open cremastric fascia and internal spermatic fascia.
- Separate the cord structures from sac.
- Dissect upto the neck of the sac (extraperitoneal fat is seen) and ligate at a high level.
- Perform herniotomy (in case of indirect hernia, inspect and reduce only viable contents)
- Strengthen the posterior wall of the inguinal canal by suturing the inguinal ligament with conjoint tendon using non-absorbable sutures (if the muscle strength is adequate)
- Close wound in layers.

Liechtenstein's Tension-Free Hernioplasty

- After opening the inguinal canal, identify and open sac, return hernial contents to abdominal cavity.
- Transfix/suture the neck of the sac.
- Strengthen posterior wall by placing prolene mesh, fix the mesh at pubic tubercle medially and inguinal ligament below and conjoint tendon above by prolene stitches.
- Place cord structures back into the canal and close wound in layers.

In Emergency Exploration

- Through inguino-scrotal incision open the canal and deliver sac with its content.
- Open the sac, inspect the contents and let out the collected fluid if any.

- Assess viability of bowel/omentum and then plan accordingly.
- Reduce the contents if viable then excise the sac and perform herniorraphy.
- Resect and anastomose the bowel / excise omentum if not viable followed by herniorraphy.

Assessment of bowel viability in case of strangulated hernia

- Colour
- Texture
- Return of peristalsis
- When in doubt, use oxygenation and hot compresses

Bowel resection and anastomosis

- Isolate the non-viable loop by placing sterile packs around
- Use occlusion clamps at the viable ends
- Apply crushing clamps to the non-viable parts of bowel
- Excise the non-viable part of the bowel between the clamps
- Remove more on the anti-mesentric border of the bowel to prevent avascular necrosis
- Avoid spillage of the contents during resection
- Excise mesentry close to the bowel
- Check for viability of the cut edge of the bowel
- Prefer two-layered closure with inner all layer continuous absorbable sutures and outer seromuscular interrupted non-absorbable sutures.
- Prevent valvular effect and maintain the bowel patency to the maximum
- Secure perfect hemostasis
- Close mesenteric rent to prevent internal herniation

Postoperative management

- Complete bed rest
- ½ hourly PR, 2 hourly BP
- Intake / Output chart
- IV fluids. (4 to 6 hours)
- Antibiotics (preferably oral cap amox)

Postoperative complication

- Pain
- Urinary retention
- Infection
- Post herniorrhaphy hydrocele
- Recurrence

Important Note

- Treat the predisposing factors of hernia such as Pulmonary tuberculosis, constipation, Beningn prostatic hypertrophy etc
- Look for direct and indirect sac and ensure complete repair
- Dissect and ligate the sac at the high level
- Repair from the most medial aspect near pubic tubercle (medial recurrence is most common)
- Hernioplasty if there is muscle weakness
- Prevent infection in the post operative period
- Avoid heavy manual work for three months to prevent recurrence

Refer: Table - 1 page 34

Hydrocele

Definition

Hydrocele is an abnormal collection of serous fluid in the tunica vaginalis covering the testicles or within some part of processus vaginalis.

Hydrocele is the commonest cause of swelling in the scrotum. Simple surgical correction such as eversion or excision of sac can cure the problem. It is important to examine the patients completely including the hernial orifices, back and the perineal region.

- Primary hydrocele cause not known (common)
- Secondary hydrocele secondary to a disease in testis or epididymis

Types of hydrocele:

- Congenital hydrocele (sac communicates with the peritoneal cavity)
- Infantile hydrocele (sac is closed at the deep inguinal ring)
- Funicular hydrocele (sac closed at the upper end of the testis)

Vaginal hydrocele (sac completely encircles the testis)

- Encysted hydrocele of the cord (loculated cyst present anywhere between the deep ring to the testis)
- Hydrocele en bisac (huge sac with cross fluctuation)

Causes

- Defective absorption of fluid by the tunica vaginalis
- Excessive production of fluid within the sac
- Lymphatic obstruction
- Communication with peritoneal cavity

Symptoms

In the majority the swelling of the scrotum may be the only complaint.

Occasionally patient may complain of pain (due to heaviness or complications)

Examination

- Swelling confined to scrotum (can get above the swelling, cf. Inguinal hernia)
- Fluctuation test positive (cystic swelling)
- Transillumination test positive (except in thickened, fibrosed and calcified sac)
- Irreducible
- Testis not felt separately (not in secondary hydrocele)

Complications

- Infection
- Rupture
- Haematocele
- Hernia of the hydrocele sac
- In long standing hydrocele (calcification of the sac, atrophy of the testis may be present)
- Skin excoriation due to in-drawing of the penis and dribbling of urine.
- Decubitus ulcer in the scrotum.

Treatment

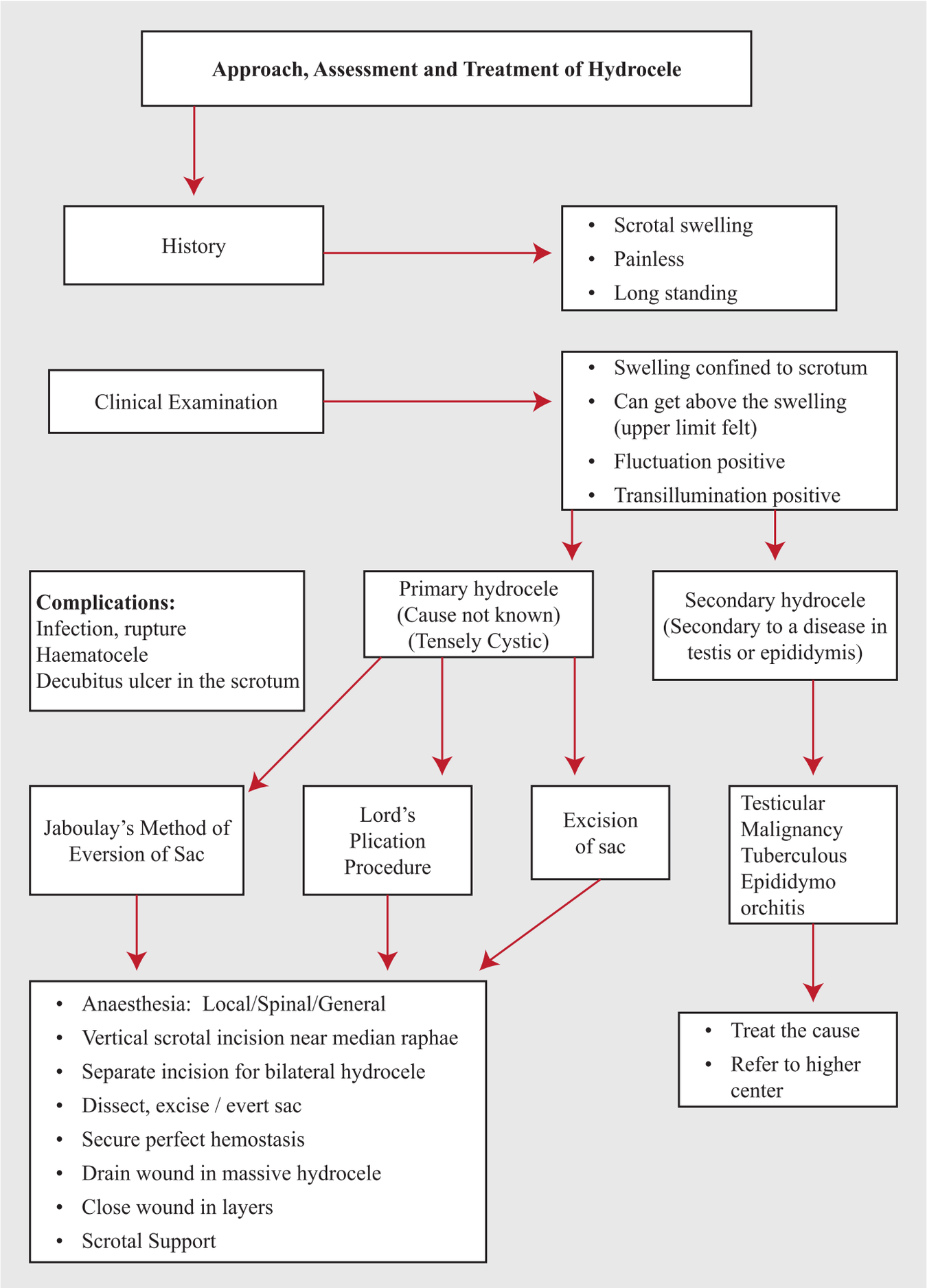
- Jaboulay's method of eversion of sac In this procedure the hydrocele fluid is let out after opening the sac through scrotal incision and then eversion of sac is done so that the secreting surface of the tunica vaginalis is turned outside
- Lord's procedure: In case of small hydrocele and thin sac, plication of the sac is done
- Excision of the sac done in case of massive hydrocele with thickened sac
- In cases of secondary hydrocele due to testicular malignancy, the management protocol for malignancy should be followed in a tertiary care hospital

Postoperative complications

- Hematoma – drain aseptically
- Infection -treat conservatively /antibiotics after culture and sensitivity if required

Important Note:

- If the hydrocele is associated with absent testicular sensation, rule out malignancy.
- When it is inflamed and painful, rule out filariasis, epididymo-orchitis
- When the hydrocele is heavy, rule out hematocele
- When there is florid fungal infection of scrotal skin, treat the infection first.
- When the hydrocele is small and the patient is unfit for anesthesia, defer surgery
- When in doubt (malignancy, associated hernia), reconfirm the diagnosis (ultrasonogram)


Approach, Assessment and Treatment of Hydrocele
History
Scrotal swelling
Painless
Long standing
Clinical Examination
Swelling confined to scrotum
Can get above the swelling (upper limit felt)
Fluctuation positive
Transillumination positive
Complications:
Infection, rupture
Haematocele
Decubitus ulcer in the scrotum
Primary hydrocele (Cause not known) (Tensely Cystic)
Secondary hydrocele (Secondary to a disease in testis or epididymis)
Jaboulay's Method of Eversion of Sac
Lord's Plication Procedure
Excision of sac
Testicular Malignancy Tuberculous Epididymo orchitis
Anaesthesia: Local/Spinal/General
Vertical scrotal incision near median raphae
Separate incision for bilateral hydrocele
Dissect, excise / evert sac
Secure perfect hemostasis
Drain wound in massive hydrocele
Close wound in layers
Scrotal Support
Treat the cause
Refer to higher center

Anal Fissure

Definition

Anal fissure is an ulcer or a crack in the anal canal or anal verge that may extend from mucocutaneous junction to dentate line. Anal fissure is of particular concern if acute because the degree of patient discomfort, pain and disability are extremely high.

The types of fissure are acute and chronic:

Acute anal fissure is a deep tear through the skin of the anal margin extending into the anal canal. There is little inflammatory induration or edema of its edges. There is accompanying spasm of the anal sphincter muscle.

Chronic anal fissure is characterized by inflamed indurated margins, and a base consisting of either scar tissue or the lower border of the internal sphincter muscle. Chronic anal fissure does not heal with conservative measures.

Causes

- Trauma to anal canal due to passage of hard stools, scarring, stricture, stenosis or previous surgery
- Sexually transmitted diseases:- Syphilis, Herpes, Cytomegalo viral infection, Chancre
- Inflammatory bowel diseases:- Crohn's disease, Ulcerative colitis
- Tuberculosis
- Immunodeficiency state

Symptoms

- Pain during defecation and/or after defecation
- Slight bleeding / streaking

Examination

- Acute fissure
  - * Digital rectal examination not possible because of anal spasm.
- Chronic fissure
  - * Skin tag
  - * Thickened underlying muscles
  - * Floor may be fibrosed
  - * Fissure as such

Investigations

Basic investigations

Colonoscopy

Barium enema

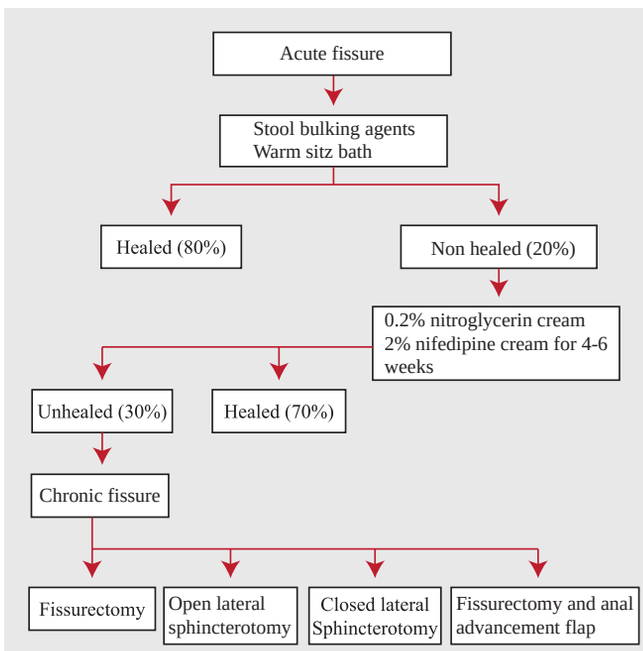
Treatment

Conservative management Surgery

- Laxatives/stool bulking agents.
- Ointment to relax anal sphincter and improve blood flow like Diltiazem / Nifedipine cream / Glyceryl trinitrate ointment
- Botulinum toxin
- Sitz bath

Surgery

- Lateral internal sphincterotomy:-
  - * The internal sphincter is divided laterally, away from the fissure itself.
  - * The procedure is done by open or closed method.
  - * Healing is usually complete by 3 weeks
- Anal advancement flap



Haemorrhoids

Definition

Hemorrhoids are dilated veins occurring in relation to the anus. (commonly known as piles).

Hemorrhoids develop due to

- Loss of anchoring connective tissue in the anal cushions
- Downward displacement or prolapse of the anal cushions
- Abnormal dilatation of veins in the internal hemorrhoidal venous plexus
- Carcinoma of rectum – compression / thrombosis of superficial rectal veins
- Benign prostatic hyperplasia – chronic straining
- Chronic constipation – straining at stools

Classification

1. By descent
   * Grade 1 bleeding
   * Grade 2 protrusions with spontaneous reduction
   * Grade 3 protrusions regressing with manual reduction
   * Grade 4 irreducible protrusions
2. By location
   * External – arise from inferior haemorrhoidal plexus and are covered by modified squamous epithelium, occur below pectinate line
   * Internal – arise from superior haemorrhoidal plexus, arise above pectinate line
   * Interno-external – when both are present

Haemorrhoids – Clinical Aspects

Symptoms

- Bleeding, bright red and painless, frequent bleeding may lead to anemia
- Prolapse
- Pain on prolapse
- Mucus discharge
- Pruritus/ itching

Investigations

- Direct Visualisation
- Digital Rectal Examination (DRE)
- Proctoscopy
- Basic investigation
- Blood grouping and Rh typing
- USG Abdomen
- Colonoscopy

Complications

- Profuse hemorrhage
- Fibrosis
- Thrombosis
- Infection
- Pyelophlebitis
- Ulceration
- Strangulation
- Gangrene
- Suppuration
- Portal pyemia

Active treatment

- Sclerotherapy - 2 to 5ml of 5% phenol in almond oil injected around the pedicle aseptically
- Banding - by modified Barron's band applicator if available
- Photo coagulation
- Hemorrhoidectomy

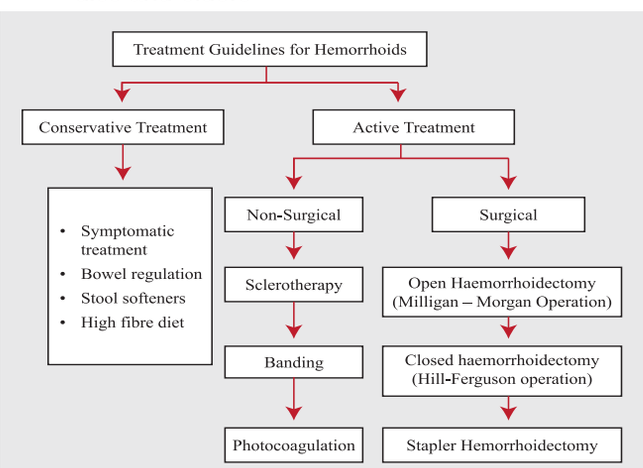
3. Open (Milligan – Morgan operation)
   * Patient is put in lithotomy/left lateral position
   * Pile mass is removed with a cuff of skin in the anal verge.
   * After removal of all the three primary hemorrhoids the appearance of anus should look like a clover.
4. Closed (Hill-Ferguson operation)
   * Patient is placed in prone jack knife position
   * Hemorrhoids are excised.
   * Mucosal defect is primarily closed with catgut sutures or daxon.
5. Stapler

* In recent times pile masses are removed with specially designed stapler whereby the pile mass is excised with the stapler and the mucosal continuity is restored.

Post Operative Care	Post Operative Complication
• Watch for bleeding • Sitz bath twice daily in the postoperative period • Analgesics • Stool softners and laxatives • Avoid digital rectal examination in the early postoperative period	Early • Pain • Acute retention of urine • Reactionary Hemorrhage(<24 HRS) • Constipation Late • Secondary hemorrhage(>24 hrs) • Anal stricture • Anal fissure • Fecal incontinence

Important note

- Surgery in failure of conservative management or in severe cases
- Colonic carcinoma to be ruled out (colonoscopy)
- Anaemia to be corrected preoperatively (blood transfusion if required)
- Muco-cutaneous bridges should be preserved during surgery to prevent anal stenosis
- Conservative treatment preferred in pregnant ladies and elders



Fistula In Ano

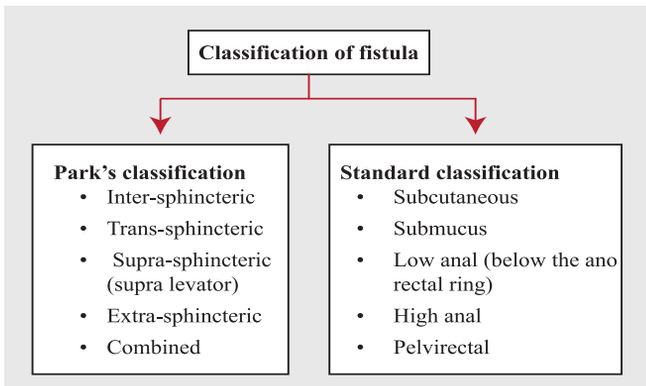
Definition

A fistula in ano is a tract lined by granulation tissue, which connects the anal canal mucosa with the skin around the anus. The fistula continues to discharge and the constant reinfection from the anal canal prevents healing of the fistula. Surgical treatment alone offers permanent cure.

Symptoms

The most frequent presenting complaints of patients with anal fistula are:

- Swelling
- Pain
- Discharge



Check for causes	Identification of fistulous tract	Investigation (When abdominal pathology is suspected)
Perianal abscess Injury in anal canal Tuberculosis HIV Crohn's disease Colloid carcinoma	Physical examination Digital rectal examination. Proctoscopy Probe test Injection technique dye injection (methylene blue or indigo carmine) or hydrogen peroxide Fistulography	Colonoscopy Sigmoidoscopy Barium enema Anorectal manometry

Surgical procedure

Principles of operative treatment

- Identify the tract
- Incise / excise the tract
- Send for biopsy

Fistulotomy

If the fistulous tract is superficial and both internal and external opening are made out, then opening of the tract with a probe in the tract is ideal. The wound then heals by secondary intention.

Fistulectomy

This procedure involves identification of fistulous tract and removing the entire tract up to the internal opening. Wound healing will be much faster when compared to the fistulotomy.

Supra-Sphincteric Fistula

Fistulotomy can be accomplished distal to the internal opening by dividing the lower portion of the internal and external sphincters. The cephalad component including the internal opening is treated by means of Seton division.

Seton

Seton means, "bristle" a seton (non-absorbable suture material) is introduced through the fistulous tract and the two ends brought through the internal and external opening and tied together. Controlled tightening of the seton will result in division of the fistulous tract over days to weeks. This is used mainly in extra-sphincteric fistula where incontinence is expected if fistulotomy is done.

Other procedures

- Endorectal advancement flap
- Transposition of fistulous tract
- Use of fibrin glue
- Diversion colostomy

Abdominal Injury

The abdomen has only muscle layers protecting the internal organs anteriorly and laterally and hence injuries to internal structures are common.

Mechanism of Injury

Two types of injury occur depending upon the nature of the offending agent.

Blunt trauma

Occurs when the offending agent is blunt and wide area of contact occurs at the time of impact. Severity of the injury depends on the force of the offending agent.

Penetrating trauma

Occurs when injured by sharp instruments and the depth and direction of the wound is more important than the size of the wound.

Causes

- Accidents (traffic, industrial and disasters)
- Falls
- Assaults
- In children (child abuse, bicycling, swimming, etc)

Investigations

The history of the traumatic event is important in determining the nature and severity of the intra-abdominal organ injury.

Physical Examination

- Life saving measures such as ABCDE
- Airway patency
- Bleeding control
- Circulation maintenance
- Deformity (cervical stablisation and fracture immobilisation) should be done first before transport to the hospital.
- Examine head to toe thoroughiy on arrival of the trauma patient to identify all the injuries Categorise patient immediately into hemodynamically stable or unstable.
- Hemodynamically unstable patient will have the following features:
  - * Cold clammy extremities
  - * Restlessness
  - * Breathlessness
  - * Sweating
  - * Altered level of consiousness
  - * External injuries
- Stabilize the hemodynamically unstable patient first, investigate later
- Cardiothoracic, head and spinal injuries get prior-

ity over abdominal and other skeletal and surface injuries or they should be tackled at the same time.

- Do emergency laparotomy based on clinical examination instead of wasting precious time (golden hour) in investigations, in hemodynamically unstable patient

Proceed in the following order for hemodynamically stable patient:

- Complete clinical examination
- Plain X-ray abdomen – Erect or left lateral position
- Diagnostic peritoneal lavage
- Ultra sound abdomen (FAST)
- Abdominal CT (if available)
- Rarely, diagnostic laparoscopy

Blunt injury abdomen can be broadly divided into

- Hollow viscous injury
- Solid organ injury
- Vascular and other injuries

Depending upon the location, blunt injury can be classified into

- Parietal injury, intra peritoneal injury, retroperitoneal injury

Parietal injuries

- The layers of the abdominal wall alone is injured
- Peritoneum is not breeched
- Require simple suturing in layers after through cleaning

Hollow Viscus Injury

- Hollow viscus injury includes injury to stomach, duodenum, small intestine and large intestine etc.,
- Bleeding, shock, bowel viability and peritoneal contamination with its attendant complications pose grave threat to life
- Management of blunt injury abdomen depends upon the severity of injury.

Rx Treatment

General principles in the management of blunt injury abdomen

- Resuscitate and institute life saving measures first.
- Do not delay an emergency operation
- Have sufficient quantities of blood, proper team and supportive systems.
- Tackle other injuries such as fixation of fractures at the same time if required.
- Explore the wound if the peritoneum or pleura is breached
- Observe continuously for 48 hours, or till the patient's general condition improves or passes flatus and urine.
- Intervene immediately if required
- Follow up continuously (clinical and radiological) and record the findings

General principles in the management of hollow viscus perforation

- Manage small breach in the serosa without damage to the mucosa with interrupted chromic catgut / vicryl sutures.
- Repair with primary suturing (two layered suturing), small rent involving the mucosa of the intestines if it involves less than half the circumference of the lumen (to prevent luminal stenosis- close the wound transversely).
- Limited resection and anastomosis for injuries involving major area with more than half the circumference of the lumen and vascular compromise.
- Peritoneal toileting with diversion colostomy and closure of abdomen with drain followed by definite repair at a later stage.
- Defer primary resection and anastomosis

Major large bowel laceration and with peritoneal contamination:

- Partial gasterectomy and hemicolectomy depending upon the site and severity of injury.
- Special care needed for duodenal injuries (retroperitonial duodenal injuries may be associated with injury to pancreas and biliary system. Iso-

lated duodenum injury is rare)

- Resect the non viable bowel before closure. Ensure the viability of the bowel in mesenteric tear with vascular compromise, and in solid organ injury

Solid organ injury includes injury to spleen, liver, pancreas and kidney. Spleen is the most common organ to be injured in case of blunt injury abdomen.

General principles in the management of solid organ injury

- Minor solid organ laceration in which the patient is hemodynamically stable and there is no further blood loss intraperitoneally or retroperitoneally-patient can be managed well by conservative measures like
  - * Adequate fluid resuscitation
  - * Oxygenation
  - * Maintaining adequate urine output
  - * Blood transfusion if needed
  - * Adequate bed rest.
- Splenic injury, splenectomy for major hilar injuries and stellate tears of spleen
- Splenorraphy or conservative management for minor spleenic lacerations
- Liver injury, suturing using blunt needles (chromic /vicryl) passed through gelfoam or pledgets to control bleeding.
- Excise and suture liver edge lacerations
- Hepatectomies can be done for major injuries taking necessary precautions.
- Pancreatic injuries are very rare due to the anatomical location and occur along with splenic injury or duodenal injury.
- Renal injuries can vary from injury involving only the parenchyma or involving the collecting system, which has to be dealt with accordingly. Tears in the ureter can be sutured to prevent leak. When there is urine leak abdomen should be drained via catheter placed in the cavity.

Vascular injury

- Vascular injuries are usually associated with other injuries and may be fatal if not attended to immediately.
- Retroperitoneal hematomas are the commonest and are usually managed conservatively. If there is a huge collection and liquifaction it should be drained. Adequate blood volume should be replaced to keep the general condition stable.

Important Note

Always admit the patient and investigate and observe for 24-48 hours

Conservative management is possible but strict and close monitoring is essential.

Be prepared to open the abdomen if necessary

Rising pulse rate, air hunger, dyspnoea, increasing pain, tenderness and abdominal girth have poor prognosis

Stable general condition, soft abdomen, return of bowel sounds, absence of pain, passing clear urine and flatus have favourable prognosis

Peptic Ulcer Disease

Peptic ulcer disease

Peptic ulcers (discontinuity in gastric mucosa) occurs commonly in the younger age group and 10% of peptic ulcers are gastric ulcers. If untreated, they develop alarming complications such as perforation and hemetemesis, increasing the morbidity and mortality.

Symptoms

- Pain (periodic)
- Vomiting (due to pyloric obstruction)
- Bleeding (hemetemesis and malena-due to erosion of underlying vessels)
- Weight loss (due to prolonged vomiting)

Clinical Examination

Epigastric tenderness

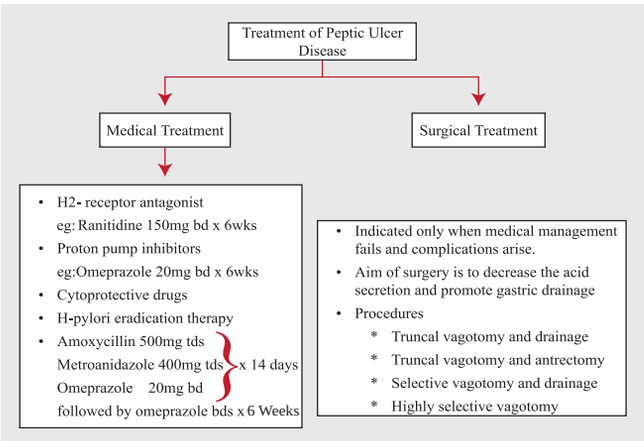
- Anaemia (due to chronic blood loss)
- Signs of complications such as
  - * Mass
  - * Visible gastric peristalsis (Due to outlet obstruction)

Investigations

- Basic investigations
- Gastroduodenoscopy
- Ultrasound abdomen

Treatment

Aims to relieve pain, promote ulcer healing, prevent ion of complication



General measures Lifestyle modification:

- Avoid smoking, alcohol, betel nut chewing
- Timely intake of food, avoiding oily and fried foods
- Supervised consumption or avoidance of NSAIDs

Complications of peptic ulcer:

- Perforation
- Haematemesis
- Gastric outlet obstruction.

Duodenal Perforation

Duodenal perforation is one of the common complications of acid peptic disease. NSAIDS appear to be more responsible for most of these perforations.

Symptoms

- Abdominal pain
- Distention
- Rigidity (may not be pronounced)
- Shock (may be present)

Investigations

- Basic investigations
- Plain x-ray chest and abdomen erect or left lateral decubitus position in ill patients (to identify the air under the diaphragm)
- Ultrasonogram abdomen. (To identify the free fluid abdomen)
- Diagnostic peritoneal lavage if necessary

Treatment

- Assess initially and resuscitate – simultaneously
- Conservative management
- Nasogastric aspiration
- Abdominal girth measurement
- Intravenous antibiotics
- Correct fluid and electrolyte imbalance
- Monitor urine output, pulse rate and temperature.

Surgical treatment

- Failure of conservative management
- Laparotomy followed by closure of perforation with omental patch
- Closed flank drainage in patients who are unfit for anaesthesia
- Laparoscopic perforation closure with flank drain

Haemetemesis

Causes

usually occurs due to

* Bleeding peptic ulcer
* Ingestion of nsaids or
* Erosion of blood vessels

Management

- Resuscitation and initial assessment - simultaneously
- Airway protection
- Adequate oxygenation
- Hemodynamic stabilization
- Maintenance of vital functions
- Followed by endoscopy / definite procedures (Injection therapy)
- Surgery it endoscopy fails.
- Observation for re-bleeding Patients require intensive observation for 72 hours in the ward and should be observed for any sign of rebleed.

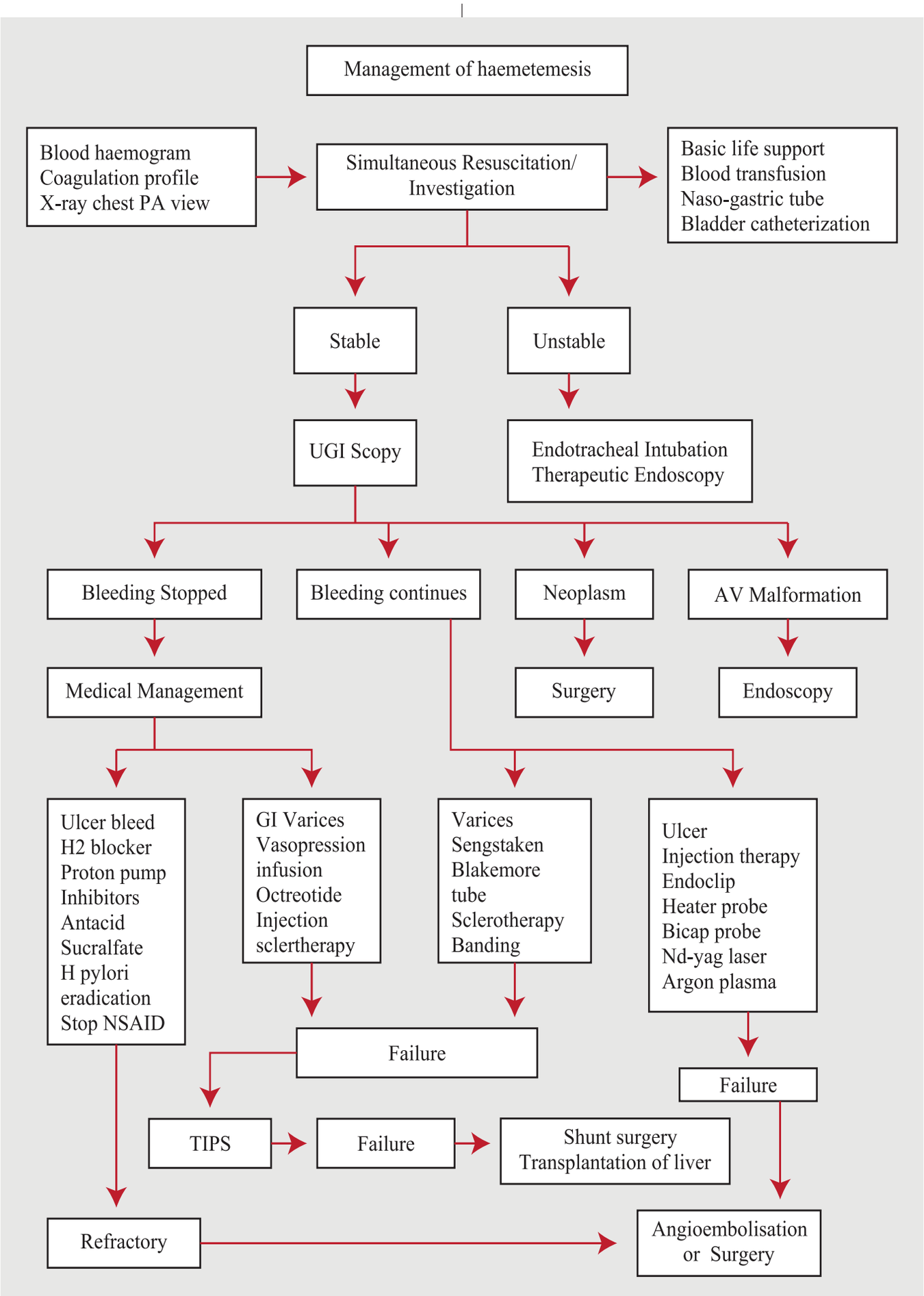
Symptoms of rebleeding

- Rise in pulse rate
- Fall in blood pressure

- Haemetemeisis or malena
- Pallor
- Decrease in urine output
- Postural hypotension
- Fall in hemoglobin or hematocrit value

Treatment

- Resuscitative measures
- Definitive procedure should be undertaken after thorough investigations
- Poor prognosis in the elderly


Management of haemetemesis
Blood haemogram
Coagulation profile
X-ray chest PA view
Simultaneous Resuscitation/ Investigation
Basic life support
Blood transfusion
Naso-gastric tube
Bladder catheterization
Stable
Unstable
UGI Scopy
Endotracheal Intubation
Therapeutic Endoscopy
Bleeding Stopped
Bleeding continues
Neoplasm
AV Malformation
Medical Management
Surgery
Endoscopy
Ulcer bleed
H2 blocker
Proton pump
Inhibitors
Antacid
Sucralfate
H pylori
eradication
Stop NSAID
GI Varices
Vasopression
infusion
Octreotide
Injection
sclertherapy
Varices
Sengstaken
Blakemore
tube
Sclerotherapy
Banding
Ulcer
Injection therapy
Endoclip
Heater probe
Bicap probe
Nd-yag laser
Argon plasma
Failure
Failure
TIPS
Failure
Shunt surgery
Transplantation of liver
Refractory
Angioembolisation
or Surgery

Gastric Outlet Obstruction (GOO)

GOO is the sequelae of healing and scarring of peptic ulcer.

Symptoms

Patients may present with vomiting of stale food and ball rolling movement, chronic cases may present with dehydration and weight loss.

Investigations

- Basic investigation
- Barium meal series
- Upper gastrointestinal endoscopy and biopsy if indicated

Treatment

- Surgery vagotomy with drainage procedure (posterior, vertical, short loop, iso peristaltic, retrocolic gastrojejunostomy)

Refer : Table 2 page 35

Carcinoma stomach

Gastric cancer is a common malignant disorder causing death, especially in men. Fortunately there is decline in incidence due to better preservation of food, awareness, screening programmes and decreasing incidence of Helicobacter Pylori infection. The peak incidence occurs around 60 years, for both sexes. Common in the lower socioeconomic groups, it is rare under 30 years of age. Antral and prepyloric tumors are the most common followed by those in the body and fundus.

Risk factors Environmental and dietetic factors

- Polycyclic hydrocarbons - especially generated by high temperature pyrolysis of animal fat and aromatic aminoacids in grilled barbecued meats.
- Smoking
- Highly-spiced, salted or pickled foods, high consumption of animal fat, excess alcohol intake and dietary nitrates
- Protein malnutrition, viral infection, bile reflex into stomach may lead to – mucosal damage and achlorhydria leading to cancer of stomach.

Others

- Atrophic gastritis, pernicious anemia, adenomatous and regenerative gastric polyps familiar polyposis, blood group A
- Previous partial gastrectomy,
- Helicobacter Pylori infection

Classification (Lauren)

- Intestinal type (older age group)Localized expanding or ulcerative lesions remains locally and spreads through nodes (Less tendency to disseminate)
- Diffuse type (younger age group)Highly invasive, invade the stomach wall without any arrangement into tubular or glandular structures (Linitis plastica) spread through submucosal and subserosal lymphatic plexus, penetrates the gastric wall.

Symptoms

- Dyspepsia, indigestion, malaise, postprandial fullness, loss of appetite, weight loss, dysphagia, haematamesis, malena, vomiting and pain.
- Anemia, weight loss, hypoalbuminemic, enlarged left supraclavicular node, palpable mass in the epigastrium, jaundice, hepatomegaly and ascitis.

Staging TNM – Four grades of tumor penetration

T1 – Confined to mucosal and submucosal lesions

T2 – Involves as far as subserosa

T3 – Serosa

T4 – Spread to contiguous structures

NO – No metastasis to nodes

N1 – Perigastric Lymphnodes within 3 cms

N2 – Regional Lymphnodes more than 3 cms

MO – No distant metastasis

M1 – Evidence of distant metastasis

Involvement of lymph nodes beyond level N2 is regarded as distant metastasis. The N3 groups of nodes are involved around the aorta, retropancreatic, hepatoduodenal and mesenteric regions.

Investigations

- Endoscopy with multiple biopsy, brush cytology
- Air contrast barium meal

- Ultrasonogram
- CT scan abdomen
- Laproscopy.

Treatment

- Adequate surgical resection cures or increases long-term survival.
- Palliative resection is effective in relieving the symptoms
- By-pass (AGJ) or intubation with expandable metalic stent (Palliation to relieve obstruction)

Principles in curative surgery

- Appropriate resection with adequate tumor free margin.
- Regional lymph node clearance corresponding to the location of the tumor in the stomach.
- Physiological, safe and well functioning reconstruction.

Indications for total gastrectomy:

- When the proximal distance from the cardia is less than the required length to achieve a safe tumor free margin.
- When the growth involves two or all the three sectors (cardia, body and pylorus)
- Diffuse carcinoma irrespective of size.

Curative resection

- There is no peritoneal or hepatic disease
- The serosa is not involved with the tumour
- The resection margins are free of tumour by histological examination

Operative procedure

- Open abdomen through upper midline incision.
- Assess operability (ascites, peritoneal, mesenteric, liver metastasis, posterior fixity).
- Proceed with palliative procedures if metastasis is present and proceed with curative resection if absent.
- Free Greater omentum near colonic attachment.
- Ligate right gastro epiploic vessel, divide the lesser omentum.
- Ligate right gastric artery dissect around the first part of duodenum.
- Transect duodenum between occlusion clamps
- Close supple duodenal stump with single layer closure (Vicryl). Reinforce with purse-string sutures to invert the duodenal stump if necessary; select jejunum two feet from the GJ and anastamose with the proximal stomach.
- Resect with clear margins of one inch all around the tumour.
- Anastomose with Iso peristaltic loop of jejunum
- Take care to prevent purse string effect or occlusion of the afferent or efferent loop.
- Clear lymphnodes one level above the involved nodes
- Perform jejunostomy to prevent duodenal blow out and bile reflux

Chemotherapy

Indicated in residual tumours, down grading of tumours preoperatively

- Regimen 1: EAP (Etoposide, adriamycin and cisplatin)
- Regimen 2: 5 FU, doxirubicin or epirubicin

Radiotherapy

Adjuvant radiotherapy indicated in selected cases.

Palliative Procedures

- Palliative gastrectomy
- Anticolic gastro jejunostomy
- Intubation with self expandable metalic stents
- Laser resection
- Feeding jejunostomy

Early gastric cancer

Definition

A well-differentiated adenocarcinoma with size less than 2 cms

Treatment

- Laser therapy
- Endoscopic mucosal resection

- Combined endoscopic and laparoscopic resection of the tumour.

Important Note

- Suspect malignancy in cases of recent onset of anaemia, dyspepsia in the old age
- Endoscopy is indicated in patients not responding to H2 receptors blocker
- Laparoscopic assessment is the best way to determine operability

Appendicitis

Definition

Appendicitis is the condition in which inflammation of the vestigial organ, the appendix, occurs. There are various forms of appendicitis depending upon the clinical presentation

- Acute
- Sub-acute
- Chronic

Causes

- Faecolith obstruction
- Infection

Symptoms and signs

- Usually occurs in the 2nd and 3rd decade. More common in females
- Pain around umbilicus initially and later shifting to right iliac fossa (McBurney's point)
- Fever
- Increasing pulse rate
- Nausea, dyspepsia
- Tenderness in the McBurney's point, rebound tenderness in right iliac fossa.
- Mass in late presentation

Investigations

- Blood investigations (leucocytosis, raised ESR)
- Ultrasound abdomen
  - * Probe tenderness in early stages
  - * Presence of faecolith
  - * Appendicular wall and mesoappendix thickening
  - * Non-peristalsis segment of small bowel in RIF
  - * Periappendicular fluid collection
  - * Mass in RIF in late stage
- X-ray abdomen-erect (to rule out calculus disaease)

Treatment

- Conservative treatment (Oschener-Sherren regimen)
  - * Young females
  - * Very early stage of presentation
  - * Pulse rate is below 100
  - * Appendicular mass
- Conservative treatment should be monitored continuously and it is abandoned and surgery planned
- When the pulse rate rises continuously
- Signs of perforation and peritonitis

Surgical treatment

- Appendicectomy (Emergency / Elective) Open or Laproscopic(gold standard)
- Interval appendecectomy Usually done 4 weeks after an acute attack or after complete regression of mass
- Drainage procedures to drain the purulent discharge in case of appendicular perforation where appendicectomy could not be done.

Appendicectomy procedure

- Anesthesia - spinal or general
- Open abdomen by a curved lanz or gridion incision centering lateral to the Mcburney's point.
- Identify the base of the appendix, Caecum is the lateral most structure and the tenia coli convergence is the indicator.
- Divide mesoappendix between ligatures.
- Ligate the base of the appendix with catgut and excise it with 11-blade knife without soiling the peritoneum.
- Bury the base by purse-string or Z stitch after chemical cautering (betadine) the exposed mucosa.
- Achieve hemostasis and close wound in layers.

In case of difficulty in retrieving the appendix then the incision should be modified to Rutherford Morrison (muscle cutting) incision. Right paramedian incision in case of doubtful diagnosis and in females.

If the appendix is found to be macroscopically normal then other intra abdominal pathology has to be ruled out (Meckel's diverticulum, acid peptic disease, tubo-ovarian mass and other gynaecological problems etc)

Complications of appendicectomy

- Wound infection
- Caecal perforation and faecal fistula
- Inguinal hernia rarely (if ilioinguinal nerve is damaged accidentally)

Intestinal obstruction

Intestinal obstruction is a common problem seen in the elderly. It presents as an acute condition requiring surgical correction in the emergency ward. Often the classical acute symptoms and signs are masked. The atypical presentation of intestinal obstruction causes unnecessary delay, increasing the morbidity and mortality. Intestinal obstruction may be classified into two types:

Dynamic obstruction:

Where peristalsis is occurring against a mechanical obstruction.

Adynamic obstruction:

This is of two forms:

- Peristalsis may be absent (eg. Paralytic ileus)
- Peristalsis may be present in a non-propulsive form (eg. Pseudo-obstruction, mesenteric vascular occlusion)

By anatomical consideration, intestinal obstruction is classified as:

- Small bowel obstruction
  - High
  - Low
- Large bowel obstruction

By Presentation

- Acute
- Chronic
- Acute on chronic
- Sub-acute

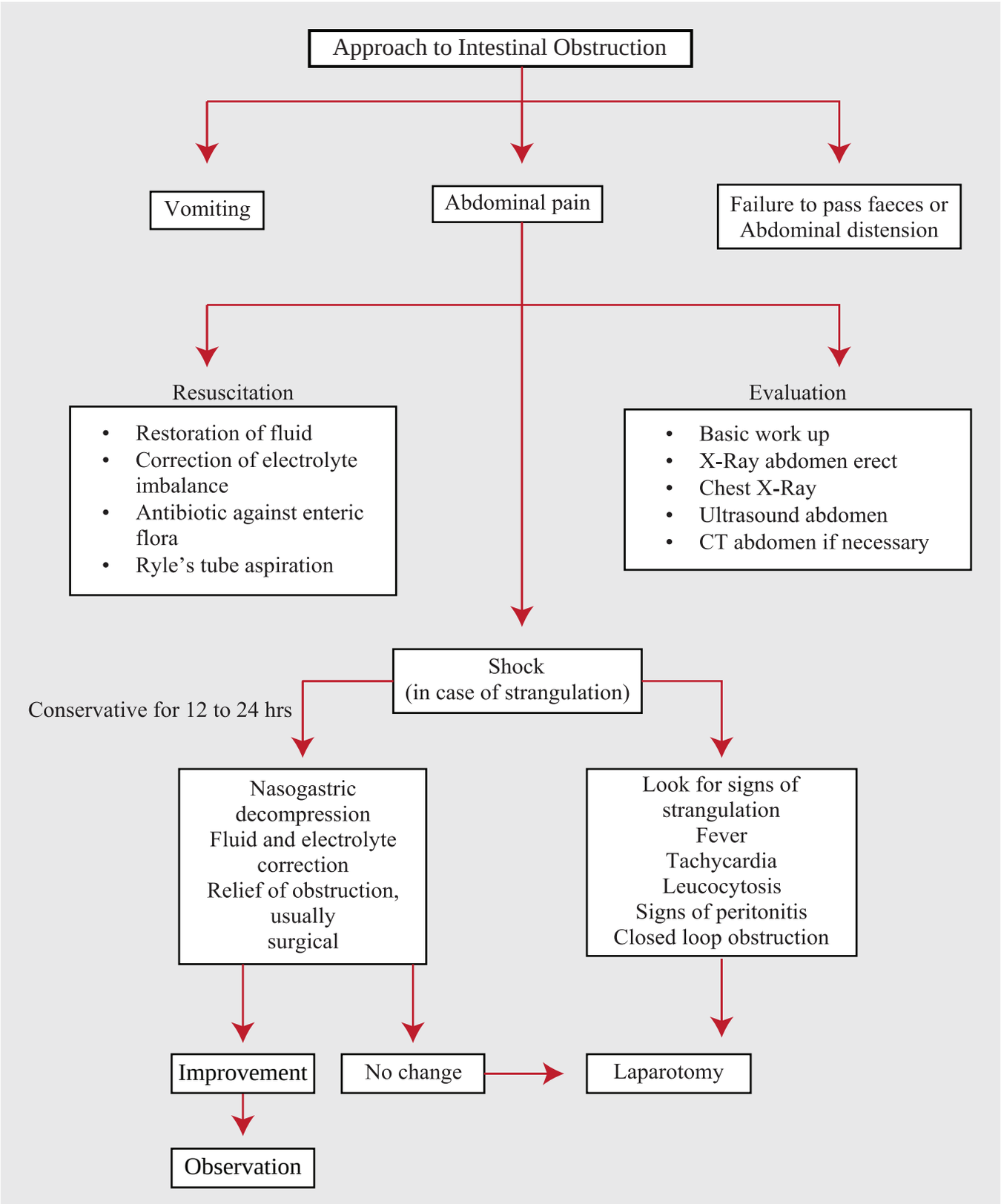
Causes

- Adhesions
- Carcinoma
- Fecal impaction
- Inflammatory
- Pseudo-obstruction
- Obstructed hernia

Symptoms

The type of surgical procedure required will depend upon the nature of the cause:

- Division of adhesion
- Excision
- Bypass
- Proximal decompression


Approach to Intestinal Obstruction
Vomiting
Abdominal pain
Failure to pass faeces or Abdominal distension
Resuscitation
• Restoration of fluid
• Correction of electrolyte imbalance
• Antibiotic against enteric flora
• Ryle's tube aspiration
Evaluation
• Basic work up
• X-Ray abdomen erect
• Chest X-Ray
• Ultrasound abdomen
• CT abdomen if necessary
Shock (in case of strangulation)
Conservative for 12 to 24 hrs
Nasogastric decompression
Fluid and electrolyte correction
Relief of obstruction, usually surgical
Look for signs of strangulation
Fever
Tachycardia
Leucocytosis
Signs of peritonitis
Closed loop obstruction
Improvement
No change
Laparotomy
Observation

Jaundice

Definition

Jaundice is a clinical state characterized by yellowish discolouration of the skin, sclera, mucus membrane and serum. Clinically manifested when the bilirubin is more than 3 mg %. the three types of jaundice are Pre Hepatic , Hepatic, Post Hepatic

Symptoms

- Dark coloured urine
- Pale coloured stools
- Generalized pruritis – especially in the palms and soles of the feet.
- Charcot's triad - fever with chills, abdominal pain, dark urine
- Raynaud's pentad - the above with hypotension and mental obtundation
- History, examination and screening investigations in a jaundiced patient is important to avoid unnecessary surgery in cirrhotics or in hepatitis since these are associated with morbidity and mortality.

Investigations

Grey Scale USG (the following findings may be seen)

- Dilated biliary radicals
- Biliary stones
- Abnormal pancreas or abnormal lymph gland node
- Changes in liver texture
- Dilated portal vein
- Subphrenic abscess or liver abscess

If biliary tree is not dilated then proceed with liver biopsy If the biliary tree is dilated the nature and extent should be defined by ERCP and / or percutaneous trans hepatic cholangiography

Endoscopy

- Upper GI endoscopy
- ERCP

ERCP

- Performed using side viewing duodenoscopy. Duodenal loop should be examined for distortion, mucosal ulceration, infiltrate or diverticula.
- It is contraindicated in recent acute pancreatitis or in presence of pancreatic pseudocyst.
- Can be performed via afferent loop of Polya gastrectomy
- Pervious sphincterotomy or sphincteroplasty may facilitate cannulation ot papilla
- Success rate varies

	HEPATIC	OBSTRUCTIVE
History	anorexia, malaise and cigarette aversion Usually painless (acute hepatitis painful) Alcohol or hepatotoxic drugs	Few systemic symptoms are usually painful. Pruritus dark urine and pale stools symptoms of cholangitis previous biliary surgery
Examination	Hepatomegaly + Signs of liver failure and portal hypertension Venepunture marks for drug abuse	Hepatomegaly + Palpable gall bladder (Courvoisier's sign) evidence of advanced malignant disease scratch marks.
Screening investigation	Transaminases >1000 units Albumin < 3gm/dl Uro bilinogen A/G reversal suggests cirrhosis	Alkaline phosphatase > 13kA or 250 IU Urinary bilinogen
Barium meal or endoscopy	Varices +	No Varices Duodenal infiltration or deformity

Findings in ERCP

- A filling defect in the biliary tree
- A dilated CBD
- The site and extent of stricture of CBD
- Particularly valuable in post cholecystectomy syndromes
- Carcinoma pancreas obstructing the main pancreatic duct
- Enable cytology and study of pure pancreatic

juice

- Endoscopic sphincterotomy and extraction of retained stones in biliary tree (Basket or Balloon extraction)

Complications:

- Cholangitis
- Septicaemia
- Acute pancreatitis

Percutaneous transhepatic cholangiography (PTC)

Direct puncture of biliary radical and injection of dye under radiographic control is done. Outline biliary tract.

Contra indication

- Ascitis
- Bleeding tendency

Complication

- Peritonitis

MRCP

Non-invasive investigation to outline the entire biliary tree and to locate the site of lesion.

Preoperative management

- Correction of clotting dysfunction
- Adequate hydration
- Mannitol to prevent renal failure
- Prophylactic antibiotics
- Nutritional support
- Drainage

Surgical management:

Choledocholithiasis Removal of calculi from the biliary tree at laparotomy should be accomplished by drainage procedure if there are multiple stones or biliary sludge.

* Sphincteroplasty
* Choledocho – duodenostomy in poor-risk older patients with dilated duct

Carcinoma of Common Hepatic Duct

A slow growing scirrhous tumour presents with jaundice and pruritus. May be a small palpable nodule at the hilum of the liver and distal biliary tree and Gallbladder are collapsed.

- Minority of patient's tumour is resectable and continuity is restored by hepatico jejunostomy using Roux – en-y
- Many focal infiltrate into the liver or distant metastasis palliation can be achieved by dilatation of the stricture or Choledochotomy and insertion of trans hepatic stents liver transplantation can be planned in patients with out spread.\

Traumatic biliary strictures

- Presents as intermittent cholangitis
- Previous cholecystectomy
- Biliary stasis results in stones and sludge in the intrahepatic ducts
- Stricture is usually at the level of Common hepatic duct; may be high as a terminal portion of hepatic duct

Surgical Treatment:

Identification of patent ducts above the stricture

- Removal of stone and sludge
- Hepatico jejenostomy Roux – en-y or hepatodochojejunostomy Roux – en –y
- Transhepatic tube used to facilitate and splint the anastomosis
- If ducts are too small access to the hepatic ducts and confluence can be achieved by hepatotomy and jejunal anastomosis.

Cholelithiasis

Gallstones are stones present in the gall bladder and in the biliary tree. Gallstones may be silent or give rise to problems if it obstructs the flow of bile or is associated with co-morbid conditions such as diabetes. Gallstone may be single (solitary) or multiple. Asymptomatic solitary stone found in the incidental USG does not require treatment. Multiple, small, migrating and obstructing stones present more problems than a single stone.

Types of stones

- Cholesterol, black pigment and brown pigment stones

Causes

Causative factor – Metabolic, infective, stasis of bile

Associated disorder – Diverticulosis of colon, Hiatus hernia (saint's triad)

Complications

- Acute cholecystitis
- Chronic cholecystitis
- Mucocele
- Carcinoma
- In the bile duct - Obstructive jaundice CholangitisAcute pancreatitis
- In the intestine - Acute intestinal obstruction, Gall stone ileus

Calculous cholecystitis

Symptoms

Symptoms are either due to obstruction of the bladder neck by a stone or inflammation of the gall bladder.

Right hypochondrial pain - Colicky in nature

Radiating to shoulder

Pain during deep inspiration (Murphy's sign)

Nausea, vomiting.

Flatulent dyspepsia - Fullness after food

Belching

Heartburn

Investigations

- USG – whole abdomen
- LFT
- HIDA scan

Complications

Infection - Causative organisms are E-coli,Klebsiella, Salmonella

Mucocele

Pyocele

Perforation

Local Abscess

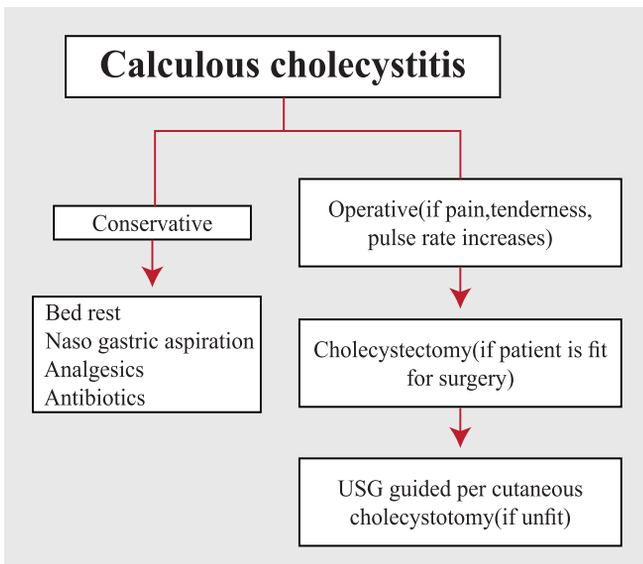
Peritonitis

Treatment

Medical

- Analgesics
- Antispasmodics
- Anti-emetics
- Low fat diet
- Dissolution therapy by bile acids (Ursodeoxycholic acid, Chenodeoxycholic acid)

Surgical



Open cholecystectomy

- Kocher's sub costal incision
- Retract liver stomach and colon
- Expose Calot's triangle
- Define biliary anatomy
- Ligate cystic duct and artery
- Dissect GB from liver bed
- Close with drain

Intra operative golden rule

- Identification of Calot's triangle.
- If cystic duct is too long, too big- beware.

Indications for choledochotomy

Indications for choledochotomy at cholecystectomy, if facilities of ERCP/ peroperative cholangiography / MRCP is not available.

- Stones in the ducts
- H/Ojaundice, rigor, fever, pruritis – Charcot's tri-

ad.

- Dilated CBD > 1cm
- Abnormal liver function test with raised alkaline phosphatase.

Acalculous cholecystitis

Causes

- Cholesterosis
- Polyposis
- Adenomyomatosis
- Cholecystitis glandularis proliferens.
- Typhoid gallbladder

Treatment

Cholecystectomy - open/ laparoscopic

cholecystostomy

Emergency surgical procedures

Venous cut down

Anatomical considerations for venous access The primary site for a peripheral venous cutdown is the greater saphenous vein at the ankle, which is located at a point approximately 2 cm anterior and proximal to the middle of the medial malleolus

A secondary site is the antecubital vein , located 2.5 Cm lateral to the medial epicondyle of the humerus at the flexion of the elbow

Saphenous vein cutdown:

- Prepare the skin of the ankle with an antiseptic solution and drape the area
- Infiltrate the skin over the vein with 0.25% lignocaine if the patient is conscious
- A full thickness transverse skin incision is made through the area of anesthesia to a length of 2.5 cm
- By blunt dissection , using a curved forceps , the vein is identified and dissected free of any accompanying structures
- Elevate and dissect the vein for a distance of approximately 2 cm to free it from its bed
- Ligate the distal mobilized vein, leaving a suture in place for traction
- Passatie around the vein, cephalad
- Make a small transverse venotomy and gently dilate the venotomy with the tip of a closed artery forceps
- Introduce a large plastic canula through the venotomy and secure it in place by tying the upper ligature around the vein and the canula
- Use distal suture to further secure the canula and the tubing
- Attach the intravenous tubing to the canula and close the incision with interrupted sutures
- Apply a sterile dressing

Complications of peripheral venous access:

- Perforation of the posterir wall of the vein
- Dissection of the vein
- Hematoma
- Phlebitis
- Cellulitis
- Venous thrombosis
- Arterial transection

Orotracheal intubation Procedure:

- Ensure that adequate ventilation and oxygenation are in progress, and that suction, a range of endotracheal tubes and laryngoscopes are available, together with introducer, boogie and magill forceps
- Connect laryngoscopic blade and handle, check the bulb for brightness
- Have an assistant manually immobilize the head and neck and a second apply cricoid pressure. The person must maintain cricoid pressure irrespective of all other events until directer to discontinue by the intubating doctor
- Use a rapid sequence induction unless patient is unresponsive and flaccid
- Hold the laryngoscope in the left hand
- Insert the laryngoscope into the right side of the patient's mouth displacing the tongue to the left
- Visually examine the epiglottis and the vocal cords
- gently insert the endotracheal tube into the trachea without applying pressure on the teeth or the oral tissues inflate the cuff with enough air to

provide adequate seal

- Check the placement by bag- valve - to tube ventilation
- Visually observe the lung expansion with ventilation
- Auscultate the chest and abdomen with a stethoscope to ascertain position
- Release cricoid pressure only when the tube is in satisfactory position and the cuff inflated
- If the endotracheal intubation is not established within 30 seconds, discontinue attempts, ventilate the patient and try again

Complications:

- Esophageal intubation, leading to hypoxia and death
- Right main bronchus intubation, resulting in collapse of the left lung and hypoxia
- Induction of vomiting, aspiration and death
- Dislocation of the mandible
- Inability to intubate
- Airway hemorrhage due to trauma
- Chipping or looseniong of teeth
- Dislocation of cervical spine during hyper extension or hyperflexion

Tracheostomy

Procedure:

- Assemble the necessary equipment(tracheostomty tray, tube, suture- cuffed disposable tracheostomy tubes are now available)
- Place the patient supine with the neck extended
- Surgically prepare and anesthetize the area locally
- Make a transverse skin incision over the lower neck over the trachea - preferably below the 2nd tracheal ring
- Incise the deep cervical fascia, retract the strap muscles laterally
- Expose the trachea beware of the thyroid isthmus, retract it superiorly, ligate any vessels carefully
- Incise the tracheal cartilage after confirming by aspuiration of air, removing a cuff of thr trachea
- Insert a cuffed tracheostomy tube
- Inflate the cuff and ventilate the patient
- Close the incision
- Secure the tube to prevent dislodgement

Complications:

- Asphyxia
- Aspiration
- Cellulitis
- Creation of false passage in the tissues
- Tracheal stenosis
- Hemorrhage or hematoma formation
- Laceration of the esophagus
- Mediastinal emphysema
- Vocal cord paralysis, hoarseness

Intercostal chest drain

Procedure:

- Ensure fluid resuscitation via atleast one large caliber iv line and high flow oxygen by mask
- Assemble the necessary equipment, scrub, don gown and gloves
- Determine the insertion site; usually at the nipple level (5th intercostal space) anterior to tghe midaxillary line on the affected side
- Use a 36fr tube in males, 32fr tube in females
- Prepare and drape the chest at the site of tube insertion
- Locally anesthetize the skin and rib periosteum and intercostal muscles. Use 30- 40ml of 0.5% lignocaine in the adult and infiltrate extensively
- Make a 2 -3 cm transverse incision in the line of the intercostal space at the predetermined site and bluntly dissect through the subcutaneous tissues, just over the upper border of the lower rib
- Puncture the parietal pleura with the tip of the clamp, stretching up the opening and put a gloved finger into the incision to avoid injury to other organs
- Mount the tip of the tube in the jaws of the forceps and advance into the pleural space
- Look for fogging of the tube with expiration and movement of the water column
- Apply an airtight dressing and tape the tube to

the chest

- Obtain a chest x-ray
- Obtain arterial blood gases if necessary
- Keep the chest drain bottles emptied regularly

Complications:

- Tube dislodgemnt
- Chest bottle elevation with fluid flowing into the chest cavity
- Damage to intercostal nerve, artery or vein
- Damage to internal mammary artery if puncture is too medial
- Intercostal myalgia
- Introduction of pleural infection
- Laceration or puncture of intra thoracic or intra abdominal organs
- Local cellulitis
- Local hematoma
- Mediastinal emphysema
- Subcutaneous emphysema

Gastrostomy:

- Assemble necessary equipment (laparotomy tray, tube, suture)
- Place the patient in supine position
- Done under GA or regional a naesthesia
- Surgically prep
- Small incision made in the anterior abdominal wall , usually in the midline
- Small incision made in the anterior stomach wall inside a pursestring
- The catheter (malecot/ foley) is introduced and the suture snugged around the catheter
- The anterior wall of the stomach should then be fixed to the anterior abdominal wall to prevent dragging
- Laparotomy wound is closed and the catheter fixed to the skin

Complications:

- Tube dislodgement
- Leak
- Hematoma
- Twist of bowel around any intra peritoneal tube with obstruction

Jejunostomy

Procedure:

- Assemble necessary equipment (laparotomy tray, tube, suture)
- Place the patient in supine position
- Done under GA or regia onal a naesthesia
- Surgically prepare
- Small incision made in the anterior abdominal wall , usually in the midline
- Small incision made in the jejunal wall inside a pursestring
- The catheter (feeding tube/ foley) is introduced and the suture snugged around the catheter
- The bowel wall should then be fixed to the anterior abdominal wall to prevent dragging
- Laparotomy wound is closed and the catheter fixed to the skin

Complications:

- Tube dislodgement
- Leak
- Hematoma
- Twist of bowel around any intraperitoneal tube with obstruction
- Intususception

Colostomy Procedure:

- Assemble necessary equipment (laparotomy tray, tube, suture)
- Place the patient in supine position
- Done under GA or regiaonalanaesthesia
- Surgically prepare
- Small incision made in the anterior abdominal wall , usually in the midline
- Smal loop of colon- transverse/ sigmoid based on indication is picked up
- A disk of skin and fat removed at a corresponding area of the abdominal wall avoiding folds, bony prominences and away from any incisions or scars

- An opening made in the rectus sheath and the muscle retracted
- The bowel wall is fixed to the skin and the rectus sheath and then opened along the tinea.
- The bowel wall should then be fixed to the anterior abdominal wall to prevent dragging
- Laparotomy wound is closed
- Colostomy bag is fixed

Complications:

- Parastomal hernia
- Loop kinking and obstruction
- Leak
- Hematoma
- Twist of bowel around any intraperitoneal tube with obstruction Intu sureeytor

Urethral Catheterization

Required in many patients in the perioperative and ICU wards

Procedure:

- Prepare the external genitalia
- Anesthetize the urethra with liberal amount of-lignocaine jelly
- A small foley type catheter is satisfactory (usually 12- 14 fr, fr refers to the circumference in mm)
- Insert the catheter in the external meatus
- Once urine flow is confirmed, the bulb is inserted
- Avoid excessive pressure and follow the natural curves of the urethra
- Abandon in case of bleeding or suspected false passage and seek expert help

Complications:

- False passage
- Tube block
- Retained tube
- Stricture

Percutaneous Suprapubic Cystostomy

This is an alternative to urethral catheterization , especially where urethral catheterization has failed and is relatively simple when there is an enlarged palpable bladder

Procedure:

- Local anesthetic is injected into the midline skin and abdominal wall 5 cm above the pubis
- Confirmation that the swelling is indeed the bladder can be obtained by a spiration
- A 1-2 cm incision is made in the skin and linea alba through which a trocar and canula is introduced into the bladder the trocar is withdrawn and a self retaining catheter is inserted down the canula into the bladder
- The propreitory suprapubic catheterization sets have a canula which splits into 2 portions.

Complications:

- Hematoma and hemorrhage
- False passage
- Damage to intra abdominal organs
- Tube dislodgement
- Leak
- Urinary extravasation and ascites

Chapter 17

Section 1 – General

1. Infections In Orthopaedics
2. Arthritis
3. Congenital Disorders
4. Metabolic Disorders Of Bone
5. Immunological Joint Diseases
6. Bone Tumours
7. Regional Conditions
8. Common Spinal Disorders
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Section 2 –Traumatology

1. General fracture management
2. Pathological fractures
3. Paediatric fractures
4. Polytrauma
5. Fractures of upper limb
6. Fractures of pelvis and acetabulam
7. Fractures of lower limb
8. Named fractures
9. Dislocations
10. Ligamentous injuries
11. Spine trauma
12. Amputation
13. Abbrervations
14. Referrences

Infections in orthopaedics

Acute Bacterial Septic Arthritis

Causes

Usually due to hematogenous spread.

Source

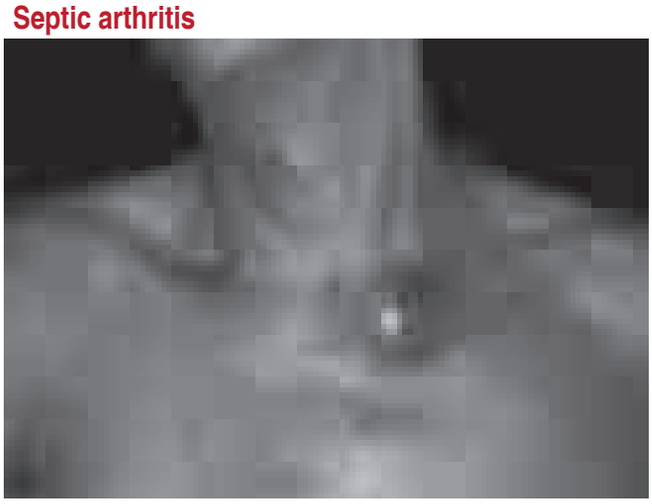
- Trauma
- Surgical procedures,
- Immunosuppressed status.

Most common organism	Staphylococcus aureus
Type	• Acute • Sub acute
Site:	• Knee most commonly involved
Clinical Features	• Pain and swelling • Constitutional symptoms • Restricted movement.

Investigations

- TLC, DLC ESR
- Aspiration of fluid - > 50000 wbc / cu.mm
- Gram stain sensitivity(30%)
- Culture sensitivity (90%)
- Blood culture
- X-ray
- MRI and Bone scan will show increased uptake

Septic arthritis



Treatment

- Immobilise the joint with splint for pain relief
- Medical therapy
- Antibiotics
- If organism unknown- treat it with broad spectrum antibiotics
- Cloxacillin or cefazolin if Staph. Aureus suspected.
- Ceftriaxone or Cefotaxime if gram negative or gonococcus is suspected
- Treat with intravenous antibiotics for 3 - 6 weeks.

Surgical Therapy.

Once diagnosed, immediate arthrotomy should be done. Arthroscopic debridement may be done at specialized centres.

Hematogenous osteomyelitis

Etiology	Children Usually caused by • Staph aureus • Strep pyogenes or Hemophillus influenzae (if < 5 yrs) Adults • Usully Staph aureus.
Clinical features	• Pain • Swelling • Constitutional symptoms • Restricted movement of joints and discharge.
Types	Acute • Illness less than 10 days • No previous episode Sub acute • No systemic symptoms • Radiological change and illness more than 10 days Chronic • Previous episode • Radiological change and illness more than 1 month

Investigations

Get atleast 2 blood cultures.

Oral therapy of Osteomyelitis

- Antibiotic concentration in bone are only 6-14% for most antibiotics, so IV therapy is usually needed to achieve, high serum levels.
- Best data for Ciprofloxacin – Rifampin combination.
- Oral Clindamycin also useful as 98% of drug penetrates into bone.
- Always make sure that the organism is sensitive.
- Treat for atleast 2 weeks with IV antibiotics initially.

Surgical Therapy

- Debridement
- Sequestrectomy
- Saucerisation
- Sinus tract excision
- Bone graft with or without myocutaneous flap.
- External fixator for stability may be done at tertiary centre

Skeletal Tuberculosis

Source

- Always secondary.

Site

- Most common site is spine
- Followed by hip and knee.
- Commonly occurs in first three decades of life.

Agent

- M. tuberculosis.

Clinical Features

- Mono articular
- H/o night cries
- Decreased joint movement
- Wasting
- Cold abscess

Investigations

- TLC, DLC, ESR
- X-ray
  - * Early: De-calcification
  - * Late: Joint destruction.
- Biopsy of regional lymph nodes.
- Exploratory arthrotomy – send tissue for culture.
- Confirm diagnosis by biopsy or culture and start treatment

Principles Of Treatment

General support – Protein rich diet.

Chemotherapy – Anti-tuberculous treat ment as per RNTCP.

Local treatment

Traction in acute and early stages

Operative

- Capsulotomy
- Synovectomy
- Curettage.

Abscess

Conservative if tense, then aspiration or incision and drainage.

Prognosis

Uncomplicated heal by conservative management alone.

Recovery after chemotherapy takes about 3 months.

Tuberculous Osteomyelitis

Tuberculous foci within the bone.

clinical Feature

Pain and tenderness

Abscess.

Radiology

Irregular cavity with sclerosis.

Treatment

As per RNTCP

Tuberculosis Of Spine

Most common region in spine- lower dorsal spine.

Most common site in vertebrae – Para discal / intervertebral disc space.

Clinical Features

- Back stiffness, pain, constituitional symptoms, paraplegia
- Physical findings
  - * Muscle spasm
  - * Wasting of back muscles
  - * Paravertebral abscess
  - * Sinuses
  - * Spastic or flaccid paralysis.
- Spinal deformity
  - * Kyphosis
  - * Scoliosis
  - * Lordosis
  - * Paravertebral thickening.

Investigations

Dealt in introduction.

Xray findings

Average number of vertebrae involved -3.

Earliest – disc space narrowing.

Late – compression / collapse.

Paravertebral shadow- cold abscess.

CT scan

Fine calcification in paravertebral soft tissue shadow.

MRI

Cord compression / Gallium scan- disseminated TB.

Biopsy and USG to assess size of cold abscess.

Management

ATT as per RNTLCP, rest , brace.

Indications For Surgery

- Neurological symptoms
- Kyphosis severe or progressing
- Resistance to chemotherapy
- Cord compression and spinal instability.

Procedure includes

- Aspiration for cold abscess
- Minimal debridement for costotransversectomy
- Radical debridement- before abscess or neuro-logical complications develops

Complications

- Paraplegia
- Cold abscess
- Sinuses
- Secondary infections
- Amyloidosis
- Fatality

Pott's Paraplegia

Most commonly associated with TB dorsal spine

Causes

Inflammatory or mechanical

Early onset paraplegia

within 2 yrs of onset of disease or with active disease

Late onset

After 2 yrs of onset of disease or with healed disease.

Motor functions first affected, clonus – most prominent early sign of pott's spine

Treatment

- Surgical indication for failed conservative, rapid onset paraplegia, recurrence, fusion for stability.
- Procedure includes
  - * Costotransversectomy for tense abscess
  - * Anterolateral decompression
  - * Bone grafting.

Tb hip

Synovial membrane most commonly affected. Most common early symptom- painful limp.

Investigations

Stage -1	Synovitis
Stage -2	Early arthritis
Stage -3	Advanced arthritis
Stage -4	Advanced arthritis with sub-luxation /dislocation

Treatment

Synovitis, early arthritis – chemotherapy with traction, if not successful synovectomy, arthrotomy.

Late arthritis

If ankylosis is sound then immobilise in plaster of paris. If not acceptable corrective osteotomy or arthrodesis in proper position. If patient does not want stiff hip then excision arthroplasty may be done.

TB Knee

Clinical Features and management

- Flexion, posterior subluxation, lateral subluxation, lateral rotation, abduction of tibia.
- Synovitis: chemotherapy+traction+joint aspiration.
- Early arthritis: chemo +synovectomy +joint debridement.
- Advanced arthritis: arthrodesis.

Osteoarthritis

Synonym: Degenerative joint disease

Definition

Chronic joint disorder in which there is progressive softening and disintegration of articular cartilage

Clinical Features

- Pain
- Stiffness after rest
- Difficulty in climbing stairs
- Difficulty in getting up from squatting position
- Deformity- varus/valgus
- Fixed flexion contracture

Investigations

- X-RAY:
  - * Joint space narrowing
  - * Subchondral sclerosis
  - * Peripheral osteophytes
  - * Cysts close to the articular surface

Treatment

- Supportive
  - * weight reduction
  - * Isometric exercise knee; Quadriceps strengthing exercise
- Drug therapy includes analgesics
  - * Calcium, antacids / H2 blockers / proton pump inhibitors

Intermediate treatment

- Intra - articular steroids
- Realignment osteotomy- done in mobile and stable joint

Late treatment

- Total joint replacement.

Congenital Disorders

Congenital Torticollis (Wryneck)

- Usually due to contracted sternocleidomastoid muscle.
- Head tilted towards and chin rotated away from affected side.
- A firm swelling at junction of mid and distal 3rd of the muscle may be felt.
- Associated with congenital anomalies in cervical spine.
- Treatment includes stretching exercises, if deformity persists for 2 yrs surgical correction-lengthening of sternomastoid muscle.
- Early identification and immediate implementation of treatment gives good results. Earlier the treatment. Better are the results

Congenital Dislocation Of Hip (CDH)

If displacement of femoral head from its normal position within the acetabulum is found at birth, it is regarded as CDH.

Subluxation:

Incomplete dislocation

Dislocation :

Femoral head completely out of acetabulum

Clinical Features

- More common in females
- Asymmetric groin folds
- Shortening of limb
- Ortoloni test
- Barlow and Telescopy test Positive
- X-ray shows broken shentons line
- Head lying outside the acetabulum
- Dysplastic acetabulum/femur.

Treatment

- Early identification and immediate implementation of treatment gives good results

Various splints include

- Von rosen splint
- Pavlik harness
- Triple diapers
- Craig splint

Treatment

Age 1-3 Years

Initially closed reduction is tried. If it doesn't give the needed result then surgical correction opted for. Surgery is planned after a period of traction. If the child is more than 18 months only surgical procedures can help.

The methods are

- Removal of the interposed soft tissue from the joint
- Innominate osteotomy
- Femoral osteotomy

More than 3 Years

Preliminary traction followed by open reduction

The methods are

- Removal of limbus
- Salter innominate osteotomy
- Pemberton pericapsular osteotomy of ilium
- Rotation osteotomy of femur

Complications

- Avascular necrosis of femoral head
- Neglected cases develop painful instability of hip and late OA, hip
- Persistent dislocations presenting in adulthood with OA can be treated with Total hip replacement arthroplasty.

Congenital Clubfoot

Congenital Talipes Equino Varus (CTEV)

Etiology

Unknown but mostly attributed to mechanical causes

- Germ plasm defect and primary soft tissue abnormalities
- Features are usually obvious at birth.
- Foot is twisted and turned inwards so that it faces posteromedially
- Foot is plantarflexed, inverted and adducted at forefoot.The leg muscles are smaller and heel cord is tight and associated internal tibial torsion may be present.

X-ray

- Talus plantarflexed and neck elongated
- Talo-navicular dislocation
- Calcaneum inverted
- Medial curving of metatarsals, in late presenting cases.

Treatment

Conservative

Aim of treatment is to produce and maintain a

plantigrade foot. Preferably should be started within a day or two after birth. Manipulative correction and maintenance in cast

Methods

- Kite
- Ponseti
- French
- Ponseti produces best results.

Surgical correction

Should be opted for in cases that have failed conservative line of management

Soft tissue procedure

- Closed Tendo Achilles Tenotomy
- Lengthening of medial and posterior structures.
- Talonavicular reduction and maintenance.

Methods

- Turco
- Carroll
- External fixator correction
  - * JESS: Less than 7 years
  - * Ilizarov: More than 7 years upto 12 years

Bone procedures

Done in case of deformity that has persisted for more than 3 yrs

- In addition to posteromedial release lateral column shortening and / or medial column lengthening procedures can be done.
- Late / relapsed cases

Options

- Soft tissue and bone procedures with tendon transfers, Gradual correction using Ilizarov method.
- Neglected cases can be treated by Triple arthrodesis.

Metabolic Disorders Of Bone

Osteoporosis

Definition

Osteoporosis is defined as abnormally porous bone and its strength is less than normal for a person of that age and sex.

Investigations

- X-rays
- Special investigations(if needed) includes
- Dual energy x-ray absorptiometry
- Single energy absorptiometry
- Quantitative computed tomography

Diagnosis

MD > 2.5 SD below the average for premenopausal women for that population group.

x-ray features:

- loss of trabecular definitions
- thinning of cortices

Primary osteoporosis

Physiological bone depletion that normally accompanies ageing and loss of gonadal activity.

Post menopausal osteoporosis

- Complaints of back pain
- Increased thoracic kyphosis

Treatment

- Physiotherapy
- Bisphosphonate
- Vitamin D
- Calcium-both diet and drug
- HRT in selected cases.

Calcium	Best taken with food
Vitamin D	< 50 yrs 200 IU/ day 50 to 70 yrs 400 IU / day > 70 yrs 600 IU / day.
Estrogen	0.3 mg / day in selected cases if there is no contraindication Selective estrogen receptor modulator (SERM) Raloxifene (60 mg / day) Tamoxifene Bisphosphonate-Risedronate -steroid induced osteoporosis

Alendronate	(70 mg once weekly given with full glass of water before breakfast) in severe cases.
Calcitonin	Nasal spray (200 IU / day), Injectable calcitonin, limited use.

Secondary osteoporosis

Causes

- Nutritional
- Endocrine
- Drug induced
- Malignant disease

Treatment

- Identify the cause and treat it accordingly
- Correct the nutritional deficiency
- Correction of hormonal imbalance

Surgery

- Injection of polymethylmethacrylate into the vertebral body
- Vertebroplasty
- Kyphoplasty

Rickets

Definition

Metabolic disease of childhood in which the osteoid, the organic matrix of bone fails to mineralise, due to interference with calcium metabolism.

Causes

- Vitamin D deficiency
- Malabsorption
- Renal disease
- Celiac disease
- Hepatic osteodystrophy
- Anti - epileptic drugs

Clinical features includes

- Bone pain during rest and excessive perspiration
- Generalised weakness
- Broadened forehead
- Craniotabes
- Pigeon chest
- Rachitic rosary
- Harrison's sulcus
- Enlargement of metaphyseal segments
- Coxa vara,knock knee
- Bow legs
- Delayed dentition

Investigations

Serum calcium	-	normal or decreased
Serum phosphorus	-	decreased
Alkaline phosphotase	-	increased
Urinary calcium	-	decreased

Treatment

- Single oral dose of 6 lakh IU of vitamin D
- 2nd dose after 3 to 4 weeks(if no sclerotic change is seen in x-ray)
- If the child responds to above treatment maintanence dose of 400 IU of vitamin D is given

Prevention of deformity

- Ricket splints

Osteomalacia

Definition

A condition marked by softening of the bones (due to impaired mineralisation, tense accumulation of osteoid) with pain, tenderness, muscle weakness, anorexia and loss of weight, resulting from deficiency of vitamin D and calcium.

Causes

- Decreased vitamin D absorption from intestines, derangement of vitamin D and phosphorus metabolism

Clinical features

- Generalised weakness/muscle pain
- Symptoms related to dietary

- Renal and GIT symptoms may be seen
- Deformities encountered are kyphosis, coxa vara, protrusio acetabuli.

X- rays

- Generalised demineralisation
- Loss of transverse trabeculae
- No sub-periosteal resorption of bone
- Presence of looser's zone

Investigations

Serum calcium: Normal or decreased

Serum phosphorus: Normal or decreased

Serum alkaline phosphatase: Increased

Treatment

- Calcium - 0.5 to 3 gm/day
- Vitamin D - 10,000 IU/day
- High protein diet

Renal Osteodystrophy

Definition

Bone is diseased due to glomerular failure and renal tubular disease.

Causes

- Congenital polycystic kidney
- Congenital hydronephrosis
- Chronic glomerulonephritis
- Chronic interstitial nephritis
- Chronic pyelonephritis

Clinical features

- Thirst
- Polydypsia
- Polyuria
- Stunted growth
- Genu valgum
- Dwarfism

Treatment

- Treat the underlying renal disease
- Organic acid with sodium citrate helps absorbtion of calcium from intestines
- Vitamin D

Fluorosis

Definition

It is a metabolic disorder leading to osteoporosis.It is a public health problem in India. Fluorosis results when fluoride content of drinking water exceeds 1 PPM

Clinical features

- Mottling of enamel of upper incisors
- Posterior longitudinal ligament of spine is thickened and may compress the cord.

Investigations

- Reveal high fluoride level

Treatment

- Encouraged to drink defluorinated water

Prevention

- By drinking defluorinated water

Immunological Joint Diseases

Rheumatoid arthritis

Definition

Most common inflammatory disease of joints characterised by proliferative and destructive changes in synovial membrane , periarticular structure , skeletal musles and perineural sheaths due to wide spread vasculitis of small arterioles.

Causes

- Exact cause not known
- Malfunction of cellular and humoral immune system may be the probable cause

Diagnostic Criteria

Four out of seven criteria should be fulfilled to make a diagnosis of rheumatoid arthritis (American college of Rheumatology)

ACR Diagnostic criteria for RA
• Early morning stiffness for minimum of 1 hour everyday, atleast for 6 weeks
• Arthritis or swelling of 3 or more joints for more than 6 weeks
• Arthritis or swelling of hand joints-wrist , meta-carpal) for more than 6 weeks
• Serum rheumatoid factor present
• Radiographic features of rheumatoid arthritis
• Rheumatoid nodules
• Symmetrical arthritis

Clinical features

- Pain
- Swelling
- Stiffness of small joints
- History of remissions and exacerbations of symptoms with seasonal variations

Extra articular features :

- Subcutaneous nodules
- Wide spread vasculitis
- Osteoporosis
- Eye changes like Episcleritis, Secondary glaucoma, Scleromalacia perforans
- Lung affections like Pleurisy, Pleural effusion, Caplan's syndrome
- Cardiac involvement includes Pericardial effusion, Arrythmias and Heart block

Orthopaedic deformities

- Symmetrical peripheral joint swelling
- Ulnar deviation of the hand
- Boutonniere's deformity
- Swan-neck deformity
- Trigger fingers and trigger thumb
- Z–deformity of the thumb

Treatment

Improving general condition of the patient Splints to reduce pain deformity

Drug therapy

- NSAIDS,DMARD
- Gold,penicillamine
- Sulphasalazin
- Antimalarial drugs
- Dapsone
- Levamisole
- Corticosteroids
- Anti – cytokine agent : Monoclonal anti-body to TNF (Infliximab), Etanercept

Immunosupressive therapy

- Azathioprine
- Leflunomide
- Cyclosporine
- Cyclophosphamide
- Treatment to be coordinated with Rheumatologist

Surgical procedures

- Synovectomy
- Osteotomy
- Arthrodesis
- Arthroplasty

Ankylosing Spondylitis

Definition

Chronic progressive inflammatory disease of sacroiliac joints and the axial skeleton, insidious onset,age less than 40 years, morning stiffness,improvement with exercise,persistence for more than 3 months

Extra articular manifestations

- Acute iritis
- Pericarditis
- Aortic incompetance
- Subluxation of atlanto-axial joints

Treatment

- Conservative treatment –rest
- NSAIDS
- Physiotherapy / back exercise

- Occupational therapy
- General preventive measures like genetic counselling

Surgical treatment

- Spinal osteotomy
- Total hip replacement,
- Total knee replacement

  Recent trends: Tumor necrosis factor antagonist (Etanercept) is being used.

Bone Tumours

Primary neoplasms of the skeleton are rare, amounting to only 0.2% of the overall human tumour burden. However,children are frequently affected and the etiology is largely unknown. Significant progress has been made in the histological and genetic typing of bone tumours. Furthermore, advances in combined surgical and chemotherapy have lead to a significant increase in survival rates even for highly malignant neoplasms, including Osteosarcoma and Ewing sarcoma.

Primary bone tumours

Benign Tumors

- Osteoid Osteoma
- Commonest true benign tumor of bone

  Age group: 5-25yrs
- Commonest site: Diaphysis of long bone. eg tibia.
- Clinical features– Night pain, relieved by salicylates.
- Pathology- Consists of a nidus surrounded by dense sclerotic bone
- X-ray - zone of sclerosis surrounding a nidus
- Treatment- complete excision of nidus with sclerotic bone.
- Prognosis - good

WHO CLASSIFICATION OF BONE TUMORS	
Cartilage Tumours	Osteochondroma , Chondroma , Enchondroma , Periosteal chondroma , Multiple chondromatosis , Chondroblastoma , Chondromyxoid fibroma Chondrosarcoma
Osteogenic Tumours	Osteoid osteoma, Osteoblastoma, Osteosarcoma, Conventional chondroblastic, Fibroblastic, Osteoblastic, Telangiectatic, Small cell, Low grade central, Secondary Parosteal, Periosteal high grade surface tumours
Fibrogenic Tumours	Desmoplastic fibroma, Fibrosarcoma
Fibrohistiocytic Tumour	Benign fibrous histiocytoma, Malignant fibrous histiocytoma
Neuroectodermal Tumour	Ewing sarcoma
Haematopoietic Tumours	Plasma cell myeloma, Malignant lymphoma,
Giant Cell Tumour	Giant cell tumour, Malignancy in giant cell tumour
Notochordal Tumours	Chordoma
Vascular Tumours	Haemangioma , Angiosarcoma
Smooth Muscle Tumours	Leiomyma, Leiomyosarcoma
Lipogenic Tumours	Lipoma,Liposarcoma
Neural Tumours	Neurilemmoma
Miscellaneous Tumours	Adamantinoma, Metastatic malignancy
Miscellaneous Lesions	Aneurysmal bone cyst, Simple cyst, Fibrous dysplasia, Osteofibrous dysplasia, Langerhans cell histiocytosis, Erdheim-Chester disease, Chest wall hamartoma
Joint Lesions	Synovial chondromatosis

Osteochondroma

- Commonest benign tumor of bone, arises from epiphysis extending to metaphysis
- Age group - adolescents
- Clinical features- painless swelling around a joint, Sessile or pedunculated
- Multiple site involvement is called Diaphyseal achalasis
- Complications- bursitis, neuropathy, limitations of movement., malignant transformation (chondrosarcoma) occur rarely
- Xray-bony growth made up of mature cortical bone and marrow. Cartilaginous gap not visible .
- Treatment- excision including periosteum.

Fibrous Dysplasia

- Normal bone is replaced by fibrous tissue.Erodes the cortices of bone from within.
- Thin layer of sub - periosteal bone forms around the mass, so bone appears expanded. Site- upper end of femur, tibia, ribs.
- Mono-ostotic one bone is affected. Poly-ostotic many bones are affected.
- Clinical features- pain, deformity, pathologic fractures
- Xray- ground glass appearance
- Treatment- curettage and bone grafting

Osteoclastoma (Giant Cell Tumor)

- Common bone tumor with variable growth potential
- Age group-20- 40yrs
- Site - starts in epiphysis and extends into metaphysis
- Commonly lower end of femur, upper end of tibia
- Pathology
  - Cell of origin is uncertain
  - Tumor consists of undifferentiated spindle cells with multinucleated giant cells
- Clinical feature-swelling, vague pain
- X-ray
  - Lytic expansile lesion
  - Eccentric location
  - “Soap bubble appearance” - pathognomonic

Treatment

- Excision with reconstruction,
- Curettage with or without supplementary procedures like chemical ablation, cryotherapy, PMMA implantation.
- Amputation: aggressive tumors, recurrence
- Radiation: tumor involving vertebrae.

Primary malignant bone tumors

- Osteogenic Sarcoma
- Usually in males aged 10 to 30.
- Occurs most often in femur, but also in tibia and humerus;
- Occasionally, in fibula, ileum, vertebra, or mandible.
- Tumor arises from bone-forming osteoblast and bone-digesting osteoclast

Treatment

- Surgery
  - Tumor resection
  - High thigh amputation
  - Hemi-pelvectomy
  - Inter-scapulothoracic surgery
- Radiation and or chemotherapy or combination of all

Parosteal Osteogenic Sarcoma

- Usually in females ages 30 to 40
- Occurs most often in distal femur, may also be in the humerus, tibia, and ulna.
- Develops on surface of bone and progresses slowly.

Treatment

- May be a combination of Surgery
  - Tumor resection, possible amputation
  - Inter-scapulothoracic surgery
  - Hemi-pelvectomy
- Chemotherapy

Chondrosarcoma

- Usually in males ages 30 to 50
- Occurs most often in pelvis, proximal femur, ribs, and shoulder girdle.
- Develops from cartilage, and grows slowly.
- Usually painless, locally recurrent and invasive

Treatment

- Hemipelvectomy
- Surgical resection (ribs)
- Radiation, and or Chemotherapy

Multiple Myeloma

Malignant neoplasm derived from plasma cells.Most commonly affects pelvis, vertebrae, skull, ribs.

- Pathology
  - * Bone is simply replaced by tumor tissue, no reactive new bone formation.
  - * Tumor cells have eccentric nucleus with clumped chromatin.

Investigations

- Urine - Bence Jones Protein present in 30% of cases
- Blood- very high ESR, A/G ratio reversal
- Serum electrophoresis- abnormal spike in gamma globulin region in 90% cases.

Treatment

- Chemotherapy: Melphalan is the drug of choice. Given in combination with Vincristine, Prednisolone and some times Cyclophosphamide. Cycles are repeated 3-4weeks for 6-12 cycles.
- Splintage of diseased part.
- Radiotherapy- useful in case of neurological compression.
- Surgical intervention in advanced cases.

Metastatic bone tumors

- The most common bony malignancies are metastatic carcinomas.
- Metastatic lesions represent the most common cause of pathology fractures due to a neoplasm.
- Usually are multiple, but can be solitary.
- The most common primaries are breast, prostate, lung,kidney and thyroid, in that order.
- Renal metastasis are quite vascular and have cold bone scans.

Clinical features

- Patients with known primary malignancy presents with symptoms suggestive of secondaries in bone.
- Bony pain, commonest site is spine.
- Pathological fractures most common in spine.

Investigations

- Xray- majority are osteolytic, few are osteoblastic. eg., male- prostatic secondaries, female-breast secondaries
- Blood- high ESR, elevated serum calcium, elevated serum acid phosphatase in prostatic secondaries.

Treatment

- Symptomatic relief of pain, and prevention of pathologic fractures
- Chemotherapy
- Radiotherapy

Regional Conditions

Peri arthritis shoulder

Significant restriction of both active and passive shoulder motion

Clinical Features

Night pain, inability to reach overhead and reach away from the body

Investigations

- X-ray
- Blood-rule out metabolic causes
- Ultrasound(Dynamic ultra sound)
- MRI (Thickness of the capsule and synovium >4mm) – To rule out other pathology

Treatment

- Supportive-Diathermy, Ultrasound, TENS,
- Medications-oral,topical,
- Intra-articular steroids.

- Streching Exercises
- Manipulation under general anaesthesia(symtomatic > 6months)

Contraindications

- Severe osteopenia
- Rotator cuff tear
- Long term Insulin dependant Diabetes mellitus (>20 years)
- No improvement with previous procedure

Surgical Release

Open or Arthroscopic release at specialized centres.

Suraspinatus Tendinitis

clinical features

- Age 20-40yrs
- Shoulder Pain
- Inability to abduct shoulder.

On examination,

Tender spot below acromion,

Abduction in range of 60-120o is painful-diagnostic.

X-ray: usually normal

Treatment

- Acute stage – rest shoulder in cuff and collar
- Anti inflammatory drugs
- USG massage
- Chronic - local infiltration of hydrocortisone and lignocaine.
- After pain subsides active exercises

Rupture of Rotator Cuff

- Due to degeneration of Supraspinatus tendon in old age. It is impinged against Acromion leading to attrition of tendon. Can later rupture.
- Clinically - pain, inability to initiate abduction, arm drop sign.
- Xray- normal
- Arthrography –shows communication between joint and subacromian bursa.

Treatment

- Rest
- Analgesics
- Short wave diathermy
- Exercise
- Total rupture needs surgical repair

Tennis Elbow

Inflammation of common extensor origin.

Clinical feature

- Pain lateral side of elbow
- Sense of weakness in lifting objects

Investigations

- Cozens test
- Thomsens test
- Chair test are positive
- X-ray: normal

Treatment

- Rest
- Anti-inflammatory drugs
- Exercises
- Ultrasonic therapy
- Local Inj. of Hydrocortisone

Cubitus Valgus

- Outward deviation of forearm at elbow.
- Normal carrying angle is 10o in male and 15o in females.
- Causes
  - * Non union of fracture lateral condyle of humerus
  - * Destruction of lateral condyle due to sepsis.
- Tardy-ulnar nerve palsy may be present - treated with anterior transposition of ulnar nerve.
- Gross deformity – supracondylar osteotomy.

Cubitus Varus

- Inward deviation of forearm at elbow.
- Most common cause is malunion of supracondylar fracture.
- Full Range of movement
- Severe deformity needs –supracondylar osteotomy.

De Quervian's Disease

Chronic constrictive tenosynovitis of abductor pollicis longus and extensor pollicis brevis.

Clinical feature

Diffuse pain at distal end of radius laterally,tenderness and painful movements of thumb

Treatment

- Early- splint wrist and thumb in full extension
- Anti inflammatory drugs
- Local administration of Inj. hydrocortisone
- Later soft - tissue release can be done

Hip

Slipped Capital Femoral Epiphysis

Boys between 10-15 yrs are affected.Associated with obesity, hormonal imbalance, impaired sexual development.

Clinical features

- Pain, Limp
- Leg Extenally Rotated, adducted,
- Limitation of abduction and internal rotation,
- Leg is shorter

Investigations

- Xray –slipping of epiphysis
- Trethowan's sign

Treatment

- Mild- surgical fixation with multiple pins.
- Moderate- internel fixation after reducing the slip.
- Severe- cervical or inter trochanteric osteotomy.
- Complications-chondrolysis, AVN, secondary osteoarthritis.

Avascular Necrosis Of Femoral Head

Death of bone due to gradual vascular impairment or sudden infarction. Clinically there is pain in groin, radiates to thigh and knee, local tenderness, limitation of movements., loss of abduction and rotation, preserved flexion.

Radiological features

Stage 1	* Pre radiologic stage – no radiologic findings
Stage 2	* Osteoporosis, sclerotic cystic areas
Stage 3	* Partial collapse * Flattening of femur head * Increased density and deformity of head
Stage 4	* Secondary deterioration of the articular cartilage * Narrowed joint space * Osteoarthritic changes

Other investigations

- Radio isotope scanning
- MRI – most sensitive to detect ischemic necrosis
- Bonemarrow pressure measurements
- Intramedullary venography
- Core biopsy of femur head

Treatment

- Early- bedrest,traction, weight reduction, withdrawal of steroid and alcohol.
- Stage 2- core decompression of head, muscle pedicle bone grafting.
- Stages 3, 4-osteotomy and replacement arthroplasty

Knee

Genu Valgum

- Outward deviation of leg at knee, acquired, unilateral or bilateral.
- Cause-Injury or septic destruction of lateral half of lower femoral epiphysis.
- Bilateral- bone softening as in rickets, osteomalacia,rheumatoid arthritis

Treatment

- Mild
  - * Boots with Robert jones heel.
- Moderate
  - * Boots with long outside bar upto the level of greater trochanter and knee straps.
- Severe
  - * Macewen's osteotomy.

Genu Varum

- Lateral bowing of legs at knee. Usually bilateral.
- Seen in rickets, pagets, osteoarthrosis of knee.

Treatment

- Mild
  - * Boots with long inner rod extending to the groin and leatherstraps across tibia and knee.
- Severe
  - * Osteotomy

Plantar Fasciitis

Aseptic inflammation of plantar fascia

Clinical features

- Pain both heels
- Unable to bear weight
- Tenderness over medial tuberosity of calcaneum

Investigations

Xray- lateral view of calcaneum may show spur

Treatment

- Soft sole pad in the heel
- Ultrasound Therapy
- Analgesics
- Local Hydrocortisone

Common Spinal Disorders

Spondylolisthesis

Spondylolisthesis is defined as anterior/posterior slipping or displacement of one vertebra over another. It may be developmental/acquired .Stable/unstable. Trapezoid shaped vertebral body and dome shaped sacrum are indicative of instability and significant dysplasia.

Classified as

- Dysplastic
- Isthmic
- Degenerative
- Traumatic
- Pathological

Clinical Features

Children

- * Asymptomatic; but seek medical attention only for postural deformity or gait abnormality.

Adults

- * Complain of back ache, leg pain, clinical step off at lumbo sacral junction, hamstring tightness on straight leg raising, lordosis, spastic gait.

Investigations

- X-rays –antero posterior,oblique and standing-lateral
- Bone scan- shows increased uptake in pars plana.
- CT SCAN- may show acute stress fracture
- MRI - shows extent of disc injury at the level of listhesis

Treatment

Non operative:

For mild slips- restriction of activities, spinal brace, abdominal and trunk exercises, patient monitored at periodic intervals.

Operative:

Persistent symptoms, inspite 9 months to 1 year of treatment; tight hamstrings; abnormal gait; pelvic trunk deformity, developmental neurological deficits.

Various modalities like Postero lateral fusion/anterior interbody fusion/posterior interbody fusion with instrumentation.

Lumbar disc disease:

Low back ache is a very common problem faced in day to day life. Not all of them , are caused by disc pathology. Clinical features suggestive of disc pathology

include backache associated with para spinal spasm, streTLCh pain and leg pain.

Investigations

- X-rays lumbosacral spine AP and lateral views.
- CT scan, MRI scan to show bony architecture and cord pathology

Treatment

- Rest
- Analgesics
- Education about proper back posture
- Physiotherapy interferential therapy, traction
- Epidural steroid injections eTLC

Operative

- Cauda equina syndrome especially with bowel and bladder involvement
- Patients with persistent pain not relieved by conservative measures for 6-8 weeks
- Unilateral leg pain extending below knee that has lasted atleast 6 weeks
- Discectomy is performed usually by posterior approach and pateints will have good post operative pain relief

Spinal Deformities

Kyphosis

Is a posterior curvature of the spine.

Types

- Smooth rounded kyphosis
  - * Scheuermann's disease
- Angular kyphosis
  - * Tuberculosis spine
  - * Traumatic fusion

Scoliosis

Definition:

Scoliosis is defined as lateral curvature of the spine. Scoliosis is named according to the level and side to which the main convexity of the curve is directed.

- The common patterns are
  - * Thoracic scoliosis,
  - * Thoracolumbar,
  - * Cervicothoracic scoliosis depending upon the site of primary curve.

Classification

- Postural
- Structural
- Idiopathic
  - * Idiopathic is the most common form
- Sub-types:
  - * Infantile
  - * Juvenile and adolescent.
- Neuropathic
- Myopathic
- Osteopathic

Clinical Features

Rib hump, prominence of posterior chest wall. Test done to decide whether deformity is mobile or rigid Make the patient lie in the lateral position on the concave side. Curvature is diminished in mobile cases.

Investigations

- Radiography of the whole spine.
- Degree of the curve is measured by the Cobb's angle.
- Skeletal maturity by the fusion of the iliac apophysis(Risser sign) is an indicator of end of growth of spine.

Treatment

- Mild cases-conservative
- Moderate cases- spinal and breathing exercise with correction by Milwaukee Brace.
- Severe cases- Cobb's angle > 40 deg. Surgical correction with instrumentation like Harrington Rod. Fusion carried out with cancellous bone graft.

Cervical Spondylosis

It is a degenerative condition of the cervical spine over 50 yrs of age. Involves the intervertebral discs, posterior intervertebral joints. Commonest at C5-C6.

Clinical Features:

- Pain and stiffness

- Radiating pain to shoulder or downwards on the outer aspect of the forearm and hand.
- Giddiness on and off
- On examination there is loss of normal cervical lordosis and limitation in neck movements.
- Tenderness over the lower cervical spine or in the muscles of the paravertebral region.

Investigations

X ray cervical spine AP and Lateral :

- There is narrowing of the intervertebral disc spaces,
- Osteophytes at the vertebral margins,
- Narrowing of the intervertebral foramen presenting with radicular symptoms best observed with oblique view and MRI neck.

MRI - cervical cord compression (arrow)

Treatment

- Symptoms of cervical spondylosis undergo spontaneous remissions and exacerbations.
- Aim of the treatment is to assist the natural resolution of the temporarily inflamed soft-tissue.
- Advise proper neck posture, neck muscle exercises.
- Generally the treatment consists of analgesics, hot fomentation, traction- if stiffness present, cervical collar.
- Rarely, when spinal cord is compressed by osteophytes, surgical decompression can be done.

Lumbar spondylosis

It is a degenerative disorder of the lumbar spine , degeneration begins in the intervertebral joint, then involves posterior facet joints.

Clinical Features

- Low backache,intially worse during activity, but later present almost all time.
- The pain may radiate down the limb upto the calf.
- Spinal movements are limited terminally,
- Straight leg raising test may be positive if the nerve root compression is present

Investigations

X-ray-lumbosacral spine- AP and Lateral view. See for reduction of the disc space, osetophyte formation, narrowing of joint space of the facet joints and subluxation of one vertebra over another.

Treatment

- In acute stage
  - * Bed rest
  - * Hot fermentation and analgesics are advised.
- As symptoms subside, then spinal exercise, lumbar corset.
- Rarely spinal fusion may be necessary.

Neuromuscular Disorders

Cerebral palsy (Little's disease)

Definition

It is a non-progressive neurological disorder of children affecting the locomotor system.

Etiology

- Prenatal causes
  - Genetic factors
  - Prematurity
  - Maternal infection
  - Kernicterus.
- Natal causes
  - Cerebral anoxia
  - Birth injury.
- Postnatal causes
  - Infection- encephalitis
  - Meningitis
  - Head injury.

Types:

Spastic type- most common, due to damage to the cerebral cortex. Forms 70% of cases. Grouped according to the limb involved as monoplegia, paraplegia, hemiplegia and quadriplegia.

Clinical features:

- Generally the child is brought in the age between 2 to 4 yrs with delayed milestones.
- On examination, patient presents with adductor spasm at the hips, equines spasm at feet, exaggerated knee and ankle reflexes- patellar and ankle clonus.
- In the upper limb, shoulder in abduction and internal rotation, wrist is flexed and the thumb is drawn into the palm, and fingers are flexed, at the metacarpophalangeal joints.

Treatment:

- Aim :

  To maintain and develop whatever physical and mental capabilities the child has.
- The main stay of the treatment is Physiotherapy with rehabilitation such as occupational therapy and speech therapy.
- Methods of controlling the spasticity are drugs like
  - Diazepam
  - Baclofen
  - Botulinium toxin injection
  - Phenol nerve block etc.,
- Orthopedic treatment consists of the prevention and correction of deformities.
- Surgical treatment at specilised centres includes:
  - Tendon lengthening
  - Tenotomy of contracted tendon
  - Tendon transfer operation
  - Neurectomy
  - Stabilization of joints- Triple arthrodesis.

Traumatology

General fracture management

Definition

Disruptions of bone tissue are called fracture, visible disruption of articular cartilage is also called fracture. (Rockwood and green).

Types of fractures

- Closed fracture and open fracture
- Incomplete fracture or greenstick fracture and complete fracture
- Linear Fractures- transverse, Oblique
- Spiral, Comminuted (more than two fragments),
- Segmental Fracture, Fracture with bone loss and Impacted Fractune.
- Stress fracture
- Pathological fracture.

Gustilo Anderson classifition of open fracture	
Type I	Open fracture with wound < 1cm Bone is not exposed.
Type II	Wound > 1cm without extensive soft tissue damage / skin flaps avulsion.

Type III A	Open fracture with extensive soft tissue damage, but with adequate soft tissue coverage of bone. It also includes segmental fracture, comminuted fracture with laceration < 1cm.
Type III B	Extensive soft tissue loss with periosteal stripping with bony exposure.
Type III C	Open fracture with an arterial injury that require immediate repair regardless of size of the wound.

Clinical features

- Pain
- Swelling
- Inability to use the affected limb
- Deformity
- Crepitus
- Abnormal mobility
- Loss of transmitted movement.

Investigations

- Routine X-ray AP
- Lateral view
- Special views if necessary

Management includes

- Conservative management for closed fracture and Type –I open fracture for which closed manual reduction tried and patient put on appropriate plaster of paris splint for 3-4 weeks.
- Surgical management
  Open reduction with internal fixation with appropriate implant Mangled extremity severity score 7-16 Amputation, 3-6 --- Limb salvage in Gr-II, III Fractures
- Indications are
  * TypeII, IIIA,B,C Open fractures
  * Displaced intraarticular fracture
  * Unstable pelvic fractures
  * Unstable spinal injuries
  * Unstable long bone fracture
  * Unstable fracture in which appropriate nonoperative management has failed
  * Displaced Epiphyseal physeal finjuries with propensity for growth arrest
  * TypeIII, IV. Fracture with compartment syndrome
  * Impending or displaced pathological fracture.
  * Major avulsion fracture with musculotendinous unit disruption
  * Nonunion, malunion with or without infection in which previous non operative or surgical treatments have failed.

Timing of surgery

Emergency	• Emergency for Open fractures, Irreducible Dislocation of major joints • Fractures with lacerations or deep excoriations in the operative field,
24-72 hrs.	• Spinal injuries with deteriorating neurological deficit / fractures, • Dislocations impairing neurovascular element, or overlying soft tissues • Fractures with compartment syndromes • For Re-debridement of severe open fractures, • Long bone stabilization in polytrauma, • Hip fractures and Unstable fracture-dislocation.
Elective	• Elective operation can be delayed upto 3-4 weeks
Note:	• POP splint, skin or skeletal traction, appropriate splint can be used in patients prior to emergency / elective surgery to maintain reduction and to relieve pain.

Initial management of open injuries:

- Record vitals, start lifeline, IV fluids, IV antibiotics, analgesics
- Immediadely debride the the wound of contaminated wound, devitalized tissue look for 4 C's,

consistency, colour, contractility, circulation.

- Irrigate copiously with saline – Grade I – 3 L saline, Grade II – 6 L saline ,Grade III – 9 L saline.
- Enlarge the wound if necessary for adequate debridement.
- Remove contamination in the medullary canal.
- If wound can be closed, suture the surgically created wound, put loose stitch for other wounds over a drain if necessary. If closure is not possible, leave the wound open.

Complications

- Acute
  - Shock
  - ARDS
  - Thromboembolism
  - Neurovascular injuries
  - Crush syndrome
  - DVT.
- Chronic
  - Delayed union, Non-union, Malunion
  - Growth disturbances
  - Joint stiffness
  - Volkmann's ischemic contracture
  - Myositis ossificans
  - Post-traumatic arthritis
  - Avascular necrosis
- Peculiar to open fractures are
  - Infection
  - Tetanus
  - Gas gangrene
  - Osteomyelitis
  - Hypovolemic shock

Adult Respiratory Distress Syndrome (ARDS)

Common in long bone or multiple injuries, pelvic fractures

Clinical features

- Tachycardia greater than 140/min
- Tachypnoea
- Pyrexia
- Cyanosis
- CNS depression
- Petechial rashes

Investigations

- X-ray chest shows – "snow storm" appearance
- Retinal petechiae if PaO2 < 60 mmHg
- Decrease in platelet count
- ECG shows prominent S- wave

Management

- Artificial ventilation with ventilatory support, PEEP- 10-15 mmHg, Tidal volume <6ml/kg,
- Low molecular weight heparin
- Intravenous steroids
- Antibiotics.

Thrombo-Embolism

- Thrombo-Embolism occurs in fractures of femur tibia, pelvis, spine
- Virchows triad:
  - Venous stasis
  - Vascular damage
  - Hypercoagulability
- Deep vein thrombosis (DVT)

Clinical features include

- Calf pain
- Swelling
- Tenderness
- Increased temperature
- Pitting oedema
- Erythema
- Enlarged superficial veins

signs

- Homan's sign +ve.
- Moses's sign +ve

Treatment

- Early ambulation

- Stocking application
- Intermittent compression
- Drugs
  - * Aspirin 600 mg/day
  - * Heparin
  - * Low dose warfarin 2.5 – 16mg/day

Neuro - Vascular Injuries

Common Vessels injury in upper limb trauma

- Subclavian vessel
  - * Fracture clavicle
- Axillary vessel
  - * Proximal humerus fracture
- Brachial vessels
  - * Supracondylar fracture of humerus, posterior dislocation of elbow.
- Anterior interosseous artery
  - * Fracture both bones forearm

Common vessels injury in lower limb trauma

- Femoral vessels
  - * Posterior dislocation of hip, fracture femur
- Popliteal artery and its branches
  - * Supracondylar fracture of femur, posterior dislocation of knee , tibial fractures, ankle injuries.

Causes of injury

- Reflex vasospasm,
- Compression by fracture fragtments or haematoma,
- Incomplete or complete tear,
- Tight bandages.

Clinical features

5 P's

- * Pain
- * Pulselessness
- * Pallor
- * Paraesthesia
- * Paralysis and cold extremities

Investigations

- Routine X-rays, Doppler angiogram.

Treatment

- Prompt Open reduction itself relieves the vascular compromise
- Address the cause like repair of artery tear.
- Amputation in irreversible loss of blood supply.

Common Nerves Injured

Upper limb

- Brachial plexus
  - * Clavicle fracture
- Axillary nerve
  - * Proximal humerus fracture
- Radial nerve
  - * Shaft of humerus fracture, Supracondylar fracture of humerus
- Median nerve
  - * Posterior dislocation of elbow, wrist injury
- Posterior interosseous nerve
  - * Monteggia fracture
- Ulnar nerve
  - * Hook of hamate fracture.

Lower limb

- Sciatic nerve
  - * Posterior dislocation of hip
- Femoral nerve
  - * Anterior dislocation of hip, shaft of femur fracture.
- Common peroneal nerve
  - * Dislocation of knee
- Posterior tibial nerve
  - * Proximal tibial fracture,ankle injury
- Lateral popliteal nerve
  - * Fracture neck of fibula.

Crush syndrome

Severe crush injury of limbs and muscles results

in release of myoglobins leading to renal failure.

Treatment is managing acute renal failure

Compartment syndrome

- It is due to ischaemic necrosis of structures of anterior compartment of forearm / legs.
- It is defined by the following signs (6P's)
  - * Pain
  - * Pallor
  - * Paraesthesia
  - * Paralysis
  - * Pulselessness
  - * Positive Passive stretch.

Claw hand

- Compartment pressure > 30 mmHg of diastolic pressure.
- Treatment
  - * Emergency surgical decompression by, fasciotomy.

Delayed union

Union is considered delayed, when healing has not advanced at the average rate for the location and the type of fracture, usually 3-6 months.

Treatment

Conservative management with functional cast for additional 4-12 weeks or ORIF with appropriate implant with or without bone grafting.

Non-union

A diagnosis of Non-union is justified either by clinical or roentgenographic, shows healing has ceased, minimum of 9 months elapsed since injury and the fracture shows no visible progressive signs of union for 3 months. Time period – long bone 6 - 9 months. Fracture neck of femur – 3 months.

Treatment

- ORIF with appropriate implant with bone graft, or bone graft substitute, or bone morphogenic protein, or only bone graft.
- Ilizarov external fixator application.
- Low intensity ultrasound.
- Eletrical and electromagnetic stimulation.

Malunion

A malunited fracture is one that has healed with the fragments in a non - anatomical position.

Treatment

Corrective osteotomy with internal or external fixation with or without bone grafting.

Joint stiffness

Due to inappropriate fracture immobilization, intraarticular fractures, periarticular adhesion soft tissues, capsules and muscle contractures.

Treatment

- Physiotherapy,
- Exercises,
- Manipulation under anaesthesia,
- Surgical excision and lengthening of contractures.

Myositis Ossificans

- It is a reactive lesion (ossification) occurring in soft tissues and in stripped periosteum followed by trauma and ill advised massage.
- Clinical features
  - * Pain
  - * Swelling
  - * Loss of movements
  - * Tenderness
  - * Late stage bony hard swelling

Investigations

- X-ray
  - * Little evidence in early stage
  - * Definite in late stages.

Treatment

- Conservative treatment with splint for immobilization.
- Drugs:
  - * Low dose Indomethacin
  - * Calcitonin

* Later active physiotherapy and passive mobilization is avoided.

Avascular Necrosis

- It occurs when blood supply to a segment of bone is affected

Causes

- Extensive stripping of soft tissue and periosteum
- Loss of unique and unidirectional blood supply to bone like talus, scaphoid, neck of femur
- Steroid therapy
- Caissons disease
- Radiation therapy

Clinical features

- Pain
- Limp
- Loss of movements
- Rest pain

Investigations

- MRI
- Bone scan
- Radiographs

Treatment

- Early stages- protective brace.
- Decompression with bone graft.
- Later stage replacement surgery.

Pathological Fractures

Definition

A Pathological fracture is defined as a fracture that occurs through abnormal bone.

- Fracture that occur in bones that lacks normal bio-mechanical and viscoelastic properties.
- Due to intrinsic process- osteopenia,replacement of bone with tumours.
- Extrinsic processes- weakness caused by radiation or hole in bone by biopsy or internal fixation.
- Due to generalized process- Idiopathic Osteopetrosis

* Osteoporosis due to chemotherapy
* Cushing syndrome
* Hyperparathyroidism
* Osteogenesis imperfecta, Rickets, Renal osteodystrophy
* Leukemia
* Neuro - fibromatosis
* Due to localized Process - Unicameral bone cyst
* Aneurysmal bone cyst
* Osteomyelitis
* Fibrous dysplasia
* Langerhan's histiocytosis
* Enchondroma
* Osteochondroma
* Osteosarcoma
* Chondroblastoma
* Peri-prosthetic osteopenia
* Myelo-meningocoele
* Cerebral palsy

Treatment

* Treat the Cause
* Curettage
* Bone grafting
* Open reduction and internal fixation with appropriate implant with bone grafting
* Replacement surgery

Paediatric Fracture

The bones of children are more malleable and hence plastic type of bowing injury is more common. Bones are weaker, periosteum is thicker, so one side of the fracture remains intact. Thick periosteum helps to decrease the displacement, stabilize the reduction. Physis is the weakest point. Fractures adjacent to a joint in plane of motion will remodel. Varus, valgus and rotational malalignment may not get corrected readily.

Fractures of Necessity

For these fracture, surgery is always necessary.

- Lateral humeral condyle fracture
- Femoral neck fracture

- Distal tibial epiphysis fracture

Physeal injuries

Salter and Harris classification of Physeal Injuries	
Type –I	Epiphyseal separation through physis only with or without displacement.
Type –II	Triangular metaphyseal spike attached to separated epiphysis. Thurston Holland sign.
Type –III	Physeal separation with fracture through the epiphysis into the joint. If there is displacement joint incongruity occurs.
Type –IV	Fracture through the metaphysis, physis, epiphysis.
Type –V	Compression fracture of the physis producing growth arrest,diagnosed retrospectively.
Type –VI	Bruise or contusion to the periphery of the physis producing growth disturbances.

Management

Majority of children's fracture are treated conservatively. Few fractures require open reduction and internal fixation or closed pinning.

Open fractures in Children

Frequent and vigorous debridement with irrigation every 36-48 hours adequate stabilization of fracture will reduce the rate of non-union and secondary infection. Rarely after closed reduction hematogenous osteomyelitis occurs at the fracture site.

Polytrauma

Poly trauma is defined as

- Injury severity score (ISS) 16 or above
- Systolic blood pressure below 80 mm Hg
- Glasgow coma score less than 15
- Higher fluid resuscitation requirements
- Chest, head, abdominal organ injuries
- Fractures of more than one long bone

Stage Of Care:

- Acute resuscitation period (1-3hrs)
  - From the first point of contact with medical service to the control of acute life threatening conditions.
  - Rapid systemic assessment to identify life threatening conditions.
  - Then Airway, Breathing, Circulatory (ABC) support should be made.This involves airway control, thoracocentesis, rapid control of external bleedings, vigorous fluid and blood replacement therapy.
  - Then complete diagnostic check up if there is no acute life threatening situation
- Primary stabilization period: (upto 48 hrs)
  - From the control of acute life threatening situation and complete stability of respiratory, haemodynamic and neurologic symptoms.
  - Here acute management of fractures associated with arterial injuries and acute compartment syndrome are managed.
  - Fractures are temporarily stabilized with external fixators and compartments
- Secondary regeneration period (2 - 10days)
  - In this, general condition of the patient is stabilized and monitored.
  - Systemic inflammation and multiple organ dysfunction syndrome are managed
  - Tertiary reconstruction and rehabilitation period (weeks to months) after trauma
  - Necessary surgical procedures.
  - Definitive treatment of complete mid-phase fractures.
  - Specialized procedures to achieve fracture correction or joint reconstruction.

Hypothermia

- Due to intrinsic and androgenic causes.
- Core temperature below 34°C associated with increased mortality. May be due to depletion of high energy phosphates (Adenosine triposphate) in trauma patients
- Hypothermia can induce coagulative states so that the cardiac sequel disordered enzymatic function leads to reduced platelet activity and altered fibrinolysis

Treatment

* Active and passive rewarming techniques - use of warming blankets
* Conductive heat devices
* Preclinical use of fluid warmess and extra corporeal devices.

First examination for rapid assessment

- Respiratory:
  * Checking airway, first priority,
  * If any respiratory problem, must be corrected immediately
- Blood pressure and pulse:
  * Recorded at 15 minutes interval on a special chart - continued later at 30 minutes interval
- State of consciousness, pupil size, pupil reaction are charted
- The skull :
  * Examined for bruising and scalp lacerations, inspected and palpated to exclude depressed fractures .
  * Bleeding around eyes or conjunctival hemorrhage are recorded.
- Neck movements :
  * Examined for restriction and pain to rule out cervical spine injuries.
- The chest:
  * Examined for fractures of rib by gentle compression.
  * Palpation of skin to exclude, surgical emphysema .
  * Movement of chest wall - for any withdrawing to rule out - flial Rib segment (Paradoxical respiration)
- The Abdomen:
  * Palpated for tenderness, guarding of muscle wall.
  * Any brusing/seatbelt marks recorded.
  * Spleen, liver and kidney are carefully examined to rule out rupture .
  * Rigidity, absence of abdominal reflexes and loss of bowel sounds are important signs of Intra abdominal injury. If intra abdominal injury is suspected recording of girth measurement is a must.
- Pelvis :
  * Marks of staining around iliac crest, inwards and outwards - major pelvic disruption : minor injuries detected by local tenderness
  * Penis and perineum examined for bruising
  * Tip of urethra examined to exclude fresh bleeding
- The Spine:
  * Back inspected for bruising and abrasions for detecting the direction of violence.
  * Examine the spinous processes for tenderness or widening of interval between spinous segments for fracture dislocation of Thoraco lumbar region.
  * Alignment of spinous processes - any disturbance of alignment - Rotating deformity
- Limbs:
  * Deformity - Fractures of ankle, leg or thigh .
  * Dislocation of Hip: limb flexed adducted and internally rotated.
- Neurological Examination:
  * Muscle tone, power, sensation, reflexes are noted especially when there is spinal/head injury
- Peripheral pulses :to rule out vascular injuries.
- Wounds : Assessment of extent and depth
- Unconscious patient :
  * Examination of CNS to exclude spinal cord injuries.
  * X-ray cervical spine, skull in case of head injuries.
  * Careful attention of Airways.
  * Urine output by catheterization .
  * Blood should be taken for Grouping and Cross matching .
  * IV fluids-Initially : Isotonic electrolyte solution (saline, Ringer lactate or Hartmans 50% in a bounds of 1-2 litter for an adult and 20 ml/kg for a child.
  * Serial haematocrit monitoring until haemodynamic stability is documented.

Golden Hour

First one hour after injury, with threefold increase in mortality for every 30 minutes

Resuscitation

Follows ABCDE

Airway
Breathing
Circulation
Disability
Exposure

Airway control

- Inspect the Upper airway
- Ensure air patency
- Extend the neck; Pull the jaw forward, press the tongue down.
- Estabilish Nasal/Endotracheal/Nasotracheal airway as needed.
- Tracheostomy may be necessary

Breathing

- Restoration of ventilation and oxygenation
  - * Tension pneumothorax:
    - » Diagnosed by tracheal deviation, unilateral absent breath sounds, and distended neck veins.
    - » Treated by insertion of large bore needle in to 2nd intercostals space at the midclavicular line then placing chest tube
  - * Open Pneumothorax :
    - » Diagnosed by sucking chest wound .
    - » Treated by Occlusive dressing, not taped on one side to allow air to escape, followed by surgical wound closure, and a chest tube
  - * Flail chest with pulmonary contusion:
    - » Features: Paradoxical movement of chest wall with ventilation (breathing) .
    - » Managed with towel clip – hold the rib and prevent it to retract and refer to higher centre, positive end expiratory pressure may be needed
  - * Hemothorax:
    - » Diagnosed by opacity on x-ray chest, diminished/absent breath sounds. Managed by Chest tube placement
  - * Cardiac Arrest
    - » 1st priority - Ensure adequate pulmonary ventilation (O2, cleaning airway with sucker, tongue drawn forward and neck hyper extended) (mouth to mouth resp, neck closed while using this method / positive pressure, a simple face mask and mouth airway.
    - » If this measures fail, endotracheal intubation - positive pressure ventilation
    - » If this dose not re-establish normal cardiac rhythm, then 2nd External cardiac massage-60 compressions/min over the left side of xiphisternum, 1 breath for every 5 compressions -sternum pushed 4-6cms, compression method - hands interlaced .
    - » To combat the acidosis in cardiac arrest 100ml equivalents of sodium bicorbonate given as IV infusion.
    - » Cardiac stimulants - Adrenaline 4-10ml in 1:10000 dilution, Calcium Chloride 5 to 10ml, 1% 50% or Isoprenaline (0.1 to 0.2mg) may be used.

Circulation

- In trauma, the first shock is hemorrhagic until proven otherwise.
- Minimum 2 large bore - intravenous lines placed in antecubital fossa or groin.
- Atternatively long saphaneus vein cutdown or intraosseous (tibia) infusion for children <6years of age

Initial managenent of patient in shock

- Direct control of obvious bleeding by direct pressure - (preferrable), tourniquet clamping of blood vessels
- Large-bore intravenous access
  - * Ringer lactate infusion
  - * Blood replacement as indicated, by serial haematocrit estimation
- Traction with Thomas Splints or extremity splints to limit hemorrhage from unstable fractures
- Consideration of angiography or immediate op-

erative intervention for hemorrhage control

Haemorrhagic shock

- Diagnosed by hypotension, tachycardia, seen in patients with large open wounds, active bleeding, pelvic and/femoral fractures and abdominal or thoracic trauma.
- In the absence of open haemorrhage bleeding into voluminous space (chest, abdomen, pelvis, thigh) must be ruled out.
- This may require peritoneal lavage, angiography, CT, MRI.
- Managed by aggressive fluid replacement, blood transfusion Angiographic embolisation, operative intervention, fracture stabilisation etc.,

Classification of Haemorrhage

Type	Event	Clinical signs	Management
Class - I	<15% loss of circulating blood volume.	Normal blood pressure Pulse or capillary refill	Replaced by crystalloids
Class - II	15 - 30% loss of circulating blood volume	Tachycardia with Normal blood pressure	Replaced by crystalloid.
Class - III	30 - 40% loss of circulating blood volume.	Tachycardia Tachypnoea Hypotension	Managed by crystalloid and blood replacement
Class - IV	40% loss of circulating blood volume	Marked tachycardia and hypotension	Managed with immediate blood replacement.

Blood Replacement:

- Fully cross matched blood is preferrable,
- In case of life threatening situations O-negative blood can be used.
- Warming of blood prior to administrations prevents hypothermia..
- Monitor coagulation factors, platelets and calcium levels.
- Indications for Immediate Surgery:
  - * Haemorrhages secondary to liver, splenic, renal parenchymal injury - Laporatomy;
  - * Aortic, caval or pulmunary vessel tears - Thoracotomy
  - * Depressed skull fracture or acute intracranial hemorrhage – Craniotomy.
- Disablity (Neurologic Assessment)
  - * Assess level of conciousness by GCS,
  - * Pupillary response sensation
  - * Motor response in all extremities,
  - * Rectal tone and sensation.

The Glascow Coma Scale	
Eye opening	Verbal (Non intubated)
4 – Spontaneous 3 – Verbal stimuli 2 – Painful stimuli 1 – No response	5 – Oriented and talks 4 – Disoriented and talks 3 – Inappropriate words 2 – Incomprehensible sounds 1 – No response
Verbal (Intubated)	Motor Activity
5 – Seems, able to talk 3 – Questionable ability to talk 1 – Generally unresponsive	6 – Verbal command 5 – Localizes to pain 4 – Withdraws to pain 3 – Decorticate 2 – Decerebrate 1 – No response

- GCS = Eye opening score + Verbal (intubated or nonintubated) score + Motor score.
- GCS if < 13, systolic BP < 90, RR< 10 / min or > 30 / min warrants Emergency trauma care.
- (Ref: Harrison's Principles of Int med 17th edition)

Radiographic evalution:

- X rays of cervical spine AP and Lateral
- Lumbar spine AP and Lateral
- Pelvis AP view
- Skull AP and Lateral
- Abdomen erect, AP view
- X-ray of limbs
- Ultra sound abdomen

- CT, MRI

Assess the concomittent injuries such as head injuries, thoracic injuries, genitourinary injuries and refer the patient to higher centers for tertiary care management.

Pelvic Fracture

- It is an emergency in orthopaedics which involves multispeciality intervention.
- Mode of injury:
  - * High energy injuries
  - * Crush injuries
  - * Impact injuries
- Associated with complications and other fractures

Clinical features:

- Pain and tenderness at affected side
- Range of movements painful
- Shock,
- Intra-abdominal / Urethral / Vascular injuries.

Management

- Evaluate ABCDE and stabilise the patient
- Evaluate for other associated injuries head, chest, abdomen and spine.
- If haemodynamically stable, assess radiologically with x-ray pelvis AP view,inlet and outlet view.If there is no or minimal displacement, treat conservatively with strict bed rest and analgesics
- If there is displacement with anterior opening type(unstable #) then assess the displacement
- If it is < 2.5cm – treat conservatively
- If it is >2.5cm – external fixation or open reduction and internal fixation.
- If there is unstable # with vertical displacement then treat with ORIF
- If haemodynamically unstable, then stabilise the pelvis with external fixator at the casualty itself without shifting the patient and follow the above steps.

Associated complications and treatment

- Shock-blood transfusion and fluid replacement ,
- Intervention by Radiologist have a roll in embolisation, of bleeding pelvic vessels.
- Urethral ruputure-diagnosed by blood in urethra, perineal hematoma, distended bladder, managed by Urologist .
- Bladder rupture-extravastation of urine-Urologist and Surgeon.
- Bowel and intra-abdominal injuries are managed by General surgeon
- Thrombosis-DVT prophylaxis, vascular opinion must be obtained
- Post operatative:
  - * Infection rate (0-25%) managed accordingly
  - * Thrombo-embolism,
  - * Pin tract infection,
  - * Death inevitable in certain situations.

Named Fractures

Galeazzi or Piedmont Fracture

- Fracture of the radial diaphysis at the junction of the middle and distal thirds with associated disruption of the distal radio ulnar joint.
- Reverse Galeazzi Fracture – Fracture of the distal ulna with associated disruption of the distal radio-ulnar joint.
- Mode of injury:
  - * Galeazzi fracture- direct trauma to the wrist on the dorsolateral aspect or fall onto an outstretched hand, with forearm in pronation.

Clinical

Patient presents with wrist pain which is aggravated by stretching of the distal radio-ulnar joint .

Investigations

X-ray forearm with elbow and wrist-AP and lateral.

Treatment

- Operative- fracture of necessity- open reduction and internal fixation with plate and screw osteosynthesis. and k wires for joint disruption.

Complication

- Infection

- Neuro-vascular injury
- Radio-ulnar synostosis
- Recurrent dislocation
- Malunion, non-union

Monteggia Fracture

Fracture of the proximal ulna with radial head dislocation.

- Mode of injury
  - * Forced pronation of forearm,
  - * Axial loading,
  - * Forced abduction of the elbow

Clinical

- * Patient presents with elbow swelling deformity,
- * Crepitus and painful range of movements,
- * Associated nerve injuries.

Investigation

- * X-ray elbow and forearm with wrist-AP and lateral view

Treatment

- Conservative:
  - * Closed reduction and casting for pediatric patients
- Operative:
  - * Closed reduction of radial head with open reduction and internal fixation of ulna with plate and screw osteosynthesis.

Complications

- Infection
- Radial head instability
- Nerve injury

Bennett Fracture

- Intra articular fracture through base of first metacarpal in which shaft is displaced laterally by unopposed pull of abductor pollis longus.
- Treatment : closed or open pinning by reducing the fragment by traction and supination. And immobilization with cast for 4 weeks.

Rolando Fracture

- Comminuted fracture of base of thumb metacarpal that does not result in diaphyseal displacement.
- X-ray AP, oblique view is taken.
- Treatment- ORIF with K wires or minifragment T plate fixation or Ex-Fix application.

Dislocations

Shoulder Dislocation

- Most common dislocation

Types

- Anterior
- Posterior
- Inferior (luxatio erecta)

Mechanism

- Most commonly indirect/direct violence
- Posterior dislocation is common in electric shock and convulsions

Clinical Features

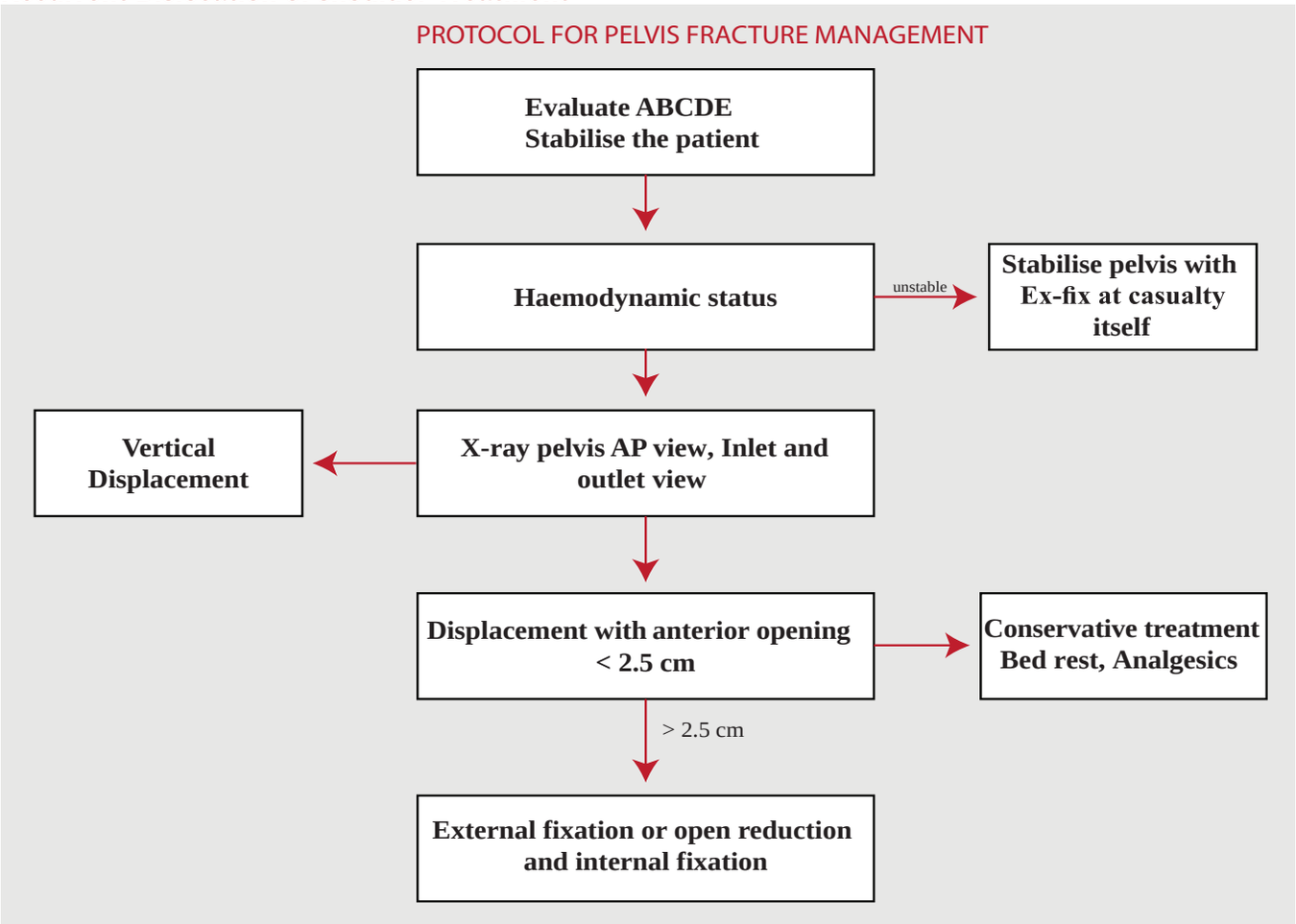
- Anterior
  - * Patient comes with injured shoulder in abduction and external rotation
- Posterior
  - * No striking deformity, shoulder in adduction and internal rotation
- Luxatio erecta
  - * Salute possession abduction forward elevation
- Severe pain, neuro vascular deficit, more with latter two
- Xray- AP, Scapular Y, Axillary view, CT scan

Rx Treatment

- Reduction – Traction – Counter Traction, Abduction, extension, external rotation – reduction
- Stimson – sedation and prone position with 5 lb weight
- Surgery
  - * Irreducible dislocation
  - * Displaced fracture dislocation with ORIF
- Complications
  - * Recurrent dislocation

* Axillary nerve and artery injury
* Brachial plexus injury
* Stiffness

Recurrent Dislocation of shoulder Treatment



Elbow Dislocation

- Most common
  * Posterior Dislocation
- Mechanism:
  * Posterior - elbow hyperextension, valgus stress, arm abduction, forearm supination.
  * Anterior-direct force over posterior forearm with elbow in flexed position.

Clinical features

- Gross instability
  * swelling
  * Three point test is positive
- Associated injuries- radial head and coronoid #

Investigation

- X-ray elbow AP and lateral.

Treatment

- Closed manual reduction under GA, followed with above elbow posterior slab 90 degree flexion.

Operative Indications

- Redislocation
- Non-concetric reduction
- Surgery: Open reduction and repair of soft tissues, hinged external fixation, cross-pinning of the joint.

Complications:

  * Loss of motion
  * Neurologic compromise
  * Vascular injury
  * Compartment syndrome
  * Redislocation
  * Heterotopic bone
  * Myositis ossificans

Hip Dislocation

Types of dislocation

- Posterior
- Anterior
- Central
- Posterior dislocation
  * Is the most common type of dislocation. Occurs mainly due to dash board injury;
- Anterior dislocation
  * Occurs because of blow to the back in squatted position
- Central dislocation
  * Occurs due to direct blow over the trochanter

Clinical Features:

- Posterior
  * Limb in flexion, adduction, internal rotation, limb shortening
  * Sciatic nerve injury
- Anterior
  * Limb in flexion, abduction, external rotation, limb lengthened
  * Injury to femoral nerve.

Investigations

- X-ray
- Hip AP and lateral
- CT, MRI

Treatment

- Resucitate, CMR attempted with in-line traction with patient lying supine, under general anesthesia
- Methods used are the classical Watson-jones, Bigelow and reverse bigelow, Allis, Stimson gravity method.
- Maintain it with skeletal traction.
- If irreducible,non concentric, ipsilateral neck # or acetabular # then open reduction is done

Complications:

- Osteonecrosis
- Posttraumatic osteoarthritis
- Recurrent dislocation
- Neurovascular injury
- Femoral head fractures
- Heterotopic ossification
- Thrombo-emblism.

Knee Dislocation

- Mode of injury: high energy/low energy
  * Hyperextension with or without varus/valgus

Clinical features:

- Gross knee distortion is present,
- A neurovascular examination must be done
- First reduce the dislocation
- Then take x-rays AP and lateral, 45 degree oblique, patellar sunrise views
- MRI
- Arthroscopy
- Assess the ligament injuries

Treatment

- Immediate closed reduction, avoid direct pres-

sure over the popliteal space after reduction, splint at 20 -30 degrees of flexion

- Operative indications:
  * Unreduced, residual soft tissue interposition
  * Open injuries. ORIF with ex- fix.
  * Reconstrucion of ligaments at later setting

Complications:

- Limited range of movements
- Ligamentous laxity and instability
- Vascular compromise
- Nerve traction injury

Ligamentous injuries

Ligamentous injuries around the knee

Mode of injury: indirect, twisting or bending forces on the knee.

Mechanism of injury

- Medial collateral ligament- valgus force
- Lateral collateral ligament – varus force
- Anterior cruciate ligament – hyperextension
- Posterior cruciate ligament-backward force on tibia.
- Menisceal injury - twisting injury with semi-flexed knee

Clinical features

- Patients present with swelling, tenderness over the corresponding anatomical sites with or without haemarthrosis, with knee instability.
- Medial collateral ligament- valgus stress test +ve at 300 knee flexion.
- Lateral collateral ligament- varus stress test +ve at 300 knee flexion
- Anterior cruciate ligament- Anterior Drawer test +ve and Lachmann test +ve
- Posterior cruciate ligament- Posterior Drawer test +ve
- Menisceal injuries
  * Young patient actively engaged in sports,
  * Recurrent episodes of pain and locking of knee
  * History of classical twisting injury to the knee followed by a swelling appearing overnight..
  * McMurray,s and Apley's Grinding test positive

Investigations

X-ray knee AP and Lateral, MRI, Arthroscopy

Treatment

- Conservative: collateral ligament injuries and minimal grade ligamentous injuries are treated conservatively with cylinder cast or a Robert Jones bandage for a period of 3-6 weeks
- Operative:
  * Anterior cruciate ligament- arthroscopic evaluation and reconstruction, open method is also used
  * Menisceal injuries- arthroscopic evaluation and excision, meniscorraphy done.

Ligamentous Injuries Around Ankle Mode of injury

- Twisting of ankle
- Axial loading with torsion
- Commonly lateral-collateral ligament is sprained; that too anterior talo-fibular component is involved

Clinical

- swelling
- Painful eversion and inversion
- Non-ambulatory
- Inversion of plantar-flexed foot painful for anterior talfibular ligament sprain
- Inversion in neutral position for complete lateral collateral ligament- painful
- Eversion in neutral position for medial collateral ligament is painful

Investigations

X rays- ankle AP and Lateral, stress views.

Treatment

- Conservative
  - Rest, ice-packs compression, elevation
  - Short leg cast- 2 to 6 weeks depending upon severity of the ligament injury.

Achilles Tendon Rupture

Mode of injury

Closet injuries

Clinical:

- Sharp pain
- Thompson test +ve
- Needle test +ve

Investigations

- X-ray ankle-lateral view
- MRI

Treatment

- Aim is to restore normal musculotendinous length and tension for normal function of the gastrocnemius-soleus complex.
- Conservative: Initially AK slab – Dorsally for 2 weeks with foot in plantar flexion, then 6 to 8 weeks in neutral position, then physiotherapy is recommended.

Operative:

Various suturing techniques with long cast followed with physiotherapy.

Complication:

- Infection
- Stiffness of ankle

Spine trauma

- The spinal cord injury is a devastating condition of major public health importance
- The higher mortality and morbidity of these injuries produce a burden on the health system of any country
- The cervical region and Thoraco-lumbar junction (T11-L2) are most commonly affected followed by the thoracic and lumbar segments
- In cervical region, the canal is narrowest between C4 and C6. Hence injury is most common at this level
- At the level of C1-C2, 1/3 rd of the canal space is free, which provides a margin of safety
- The canal in the thoracic spine is narrower than the cervical spine leading to significant neurological deficits
- The three column model of Denis is still the best model to assess the stability of injuries.
- Male: Female = 4:1, Mean age 30 years
- Most common – Motor vehicle crash (50%)

Primary mechanisms of spinal cord injury	
Mechanical force	Injuries
• Impact plus persisting compression	• Burst fracture dislocation • Disc rupture
• Impact alone	• Hyper-extension
• Flexion distraction (lap belt) injury	• Bowel rupture • Major vessel injury • Liver, spleen, urologic injury • Burst fracture • Laminar fracture • Fracture–Dislocation
• Laceration • Transection	• Missile injuries

Cervical Spine Injuries

- Cervical spine injuries are the most common site for spinal injuries accounting for about 50-64%. 10% of patients become tetraplegic
- Initial management of the spinal cord injury of patient includes: Check airway, breathing, circulation, patient should be transported after immobilization
- Shifting to be made in long spine boards, Log rolling techniques, scoop stretches, kendrick's extrication device etc.
- Medical treatment of hypotension due to neurological shock, bradycardia, fluid management and lung functions.. Ryles tube to avoid aspiration. Catheterisation of the bladder. Documentation of neurological status
- Steriods: Methyl prednisolone, within 8 hours after initial injury, 30 mg / kg over 15 minutes, followed by 5.4 mg / kg / hr for 24 hrs

Spinal realignment

- Initial spinal realignment can be obtained by skeletal traction using: Crutch field tong/Gardner wells tong/Halo-ring
- Non operative management done in
  - * Stable compression injuries of the body, without neurological deficit
  - * Undisplaced lamina fractures, lateral mass fractures
  - * Spinous process fractures and unifacetal stable fractures after closed reduction

Spine Lesions

C6 -C7 Facet dislocation

Myelogram showing widening of cervical cord due to Astrocytoma

MRI shows Posterior Epidural compression from secondaries to the posterior arch of first thoracic vertebra

- Immobilisation in rigid cervical brace for a period of 8-12 weeks
- Rigid cervical brace may be in form of
  * Halo ring
  * Minerva jacket
  * Somi brace
  * Philadelphia collar or
  * Extended cervical brace
- Unstable cervical spine fractures with or without neurological deficit are generally treated with open reduction
- Operative treatment may involve
  * Open reduction, decompression, stabilization and fusion
  * Approach and stabilization can be done either by anterior, or posterior approach or by both.

Management of Dorsolumbar spinal injuries

- The management starts at the scene of accident even before precise diagnosis is made.
- Associated injuries are also to be looked for and managed accordingly
- 60% of Dorsolumbar fractures occur between T11 and L2 levels, predominantly young males 4:1 mean age of 30 years, after sustaining high velocity injuries.
- X-rays – should be taken, focusing appropriate levels of injury. A good quality AP and lateral views of the D-L spine gives maximum information. The alignment, shape of vertebral-body and the neural arches are assessed.

Unstable spine

- The injury is said to be unstable when there is loss of vertebral body height of greater than 50% and angulation of more than 20%,
- High velocity injuries and those with neurological deficit are considered to be unstable.
- Whenever posterior column is damaged or more than 1 column involvement these are evidences of mechanical instability

Investigations

- C.T. Scan
  * Very useful in assessing the fracture stability and canal compromise. It can also demonstrate occult posterior column injuries
- MRI Scan
  * Very useful in assessing the status of neural elements and ligamentous structures.
- Myelogram
  * Incomplete neurological deficit has better prognosis
- Cauda equina lesion have better prognosis than cord lesions
- Frankel grading is very useful in documenting the functional degree of neurological deficit

Management

(Summary of NASCIS I, II and III protocols - National Acute Spinal Cord Injury Study. Ref: Rockwood Vol.2, 6th edition page-1411)

Steroids

- Methylprednisolone bolus 30 mg / kg, then infusion 5.4 mg / kg / hour, given for 24 hours, if patient presents within 3 hours of injury and
- Infusion for 48 hours if patient presents ,within 3-8 hours after injury

Non-operative Treatment:

- Implicated in simple wedge compression fractures stable burst fractures and chance fractures.
- Bedrest, Pillow reduction followed by mobilization in Hyper extension cast/brace for a period of 6-12 weeks are recommended.
- Exercises to improve spinal extensors and abdominal muscles are mandatory.

Operative Treatment :

- For unstable fractures, burst fractures with canal compromise of more than 50%, incomplete cord lesion and cauda equina lesions.
- Aim of Surgery
  * To restore anatomy supplemented with / without instrumentation.
- Fusion
  * Posterior intertransverse and interbody fusion will suffice in most cases.
- Decompression
  * Through posterolateral (or) anterior approach

* Especially in cases with neurological deficits

- Also cases with complete cord lesion can be treated surgically for pain relief or easy nursing care and better rehabilitation.

Rehabilitation of Post-spinal trauma

- Pressure sores
  * Over Bony prominences like sacrum/trochanters/heels/ischium/knees
  * ASIS (Anterior superior iliac spine). It requires padding/frequent changing of position, use of water beds.
- Bladder Care
  * Conservation with antibiotics and proper care of the bladder and urinary tract with frequent changing of catheter.
- Bowel care
  * Constipation to be avoided, for which laxatives and digital evacuation is necessary for several weeks
- Nutritional
  * Intravenous lipid and protein infusion.
- Muscle spasm
  * Dantrolene: Oral dose 25–100mg qid, reduces spasticity
  * Diazepam–Acts in brain on specific reports enhancing GABA ergic transmission. It reduces tone by supraspinal rather than spinal action.
  * Baclofen–Acts as selective GABA (B) receptor agonist
- Sexual functions
  * Improved by vacuum devices, vaso active agents and sildenafil.
- Pain
  * Reduced by NSAIDS

Conclusion

Management of acute spinal injuries needs a multi disciplinary and holistic approach with a dedicated team and appropriate management techniques with rehabilitation and psychological training of the patients.

Amputation

Indication

- The only absolute indication is the irreversible ischemia in a diseased or traumatised limb
- Other indications include
  * Gangrene due to large vessel obstruction (atherosclerosis, embolus
  * Small vessel involvement in dibetes
  * Buerger disease
  * Raynauds disease
- Moist gangrene
- Spreading cellulitis
- Neoplasm (osteosarcoma)
- Arterio-venous fistula
- Severe trauma
- Severe rest pain
- Severe contracture or paralysis
- Most significant predictor of amputation in Diabetes is peripheral neuropathy – measured by insensitivity to the Semmes-Weinstein 5.07 monofilament
- Langes absolute indication for amputation
  * Type III-C Open tibial fracture with complete disruption of tibial nerve
  * Crush injury with warm ischemia time >6 hours

Mangled Extremity Severity Score

- Helps to assess the need for amputation.
- Score > or = 7 is an indication for amputation.

Types

- End-bearing and cone-bearing.
- Best cosmetic and functional results with cone bearing.
- Cone bearing includes above knee and below knee amputation.
- End-bearing includes Gritti-stokes, through knee, Syme's amputations.

Ideal Stumps

Forearm	–	18 cm from Olecranon
Arm	–	20 cm from Acromion
Thigh	–	12 cm above knee joint line
Leg	–	14 cm from knee joint line

Below Knee (BK) Amputation:

- Two types of skin flaps
  - * Long posterior flap
  - * Skew flap
- The rule is that the total length of flap should be atleast 1.5 times diameter of leg at the level amputation
- In Long posterior flap
  - * Muscles and flap are at the same level or distal, nerves are pulled down gently and transected and cut proximal to bone cut, fibula resected 3cm proximal to the level of amputation
- In Skew flap
  - * Two equal long flaps join anteriorly 2.5 cm from tibial crest, overlying anterior tibial component and posteriorly at exact opposite point on circumference of leg. Ideal level is at the musculotendinous junction of gastrocnemius muscle.
- Lisfranc amputation
  - * At the level of tarsometatarsal joint
- Hopart amputation
  - * At the level of mid-tarsal joint
- Syme's amputation
  - * At the Level of bone section is at the distal tibia and fibula 0.6 cm, proximal to the periphery of ankle joint and passing through dome of ankle centrally.

Referrences

- Campell's operative orthopaedics- 10th edition, (2003)
- Rockwood and Green's fractures in adults -6th edition, (2005)
- Rockwood and Green's fractures in children- 6th edition, (2005)
- Current diagnosis and treatment orthopedics- 4th edition, (2006)
- Hand book of fractures – Kenneth J. Koval, Joseph D. Zuckerman- 3rd edition, (2006)
- Essential orthopaedics – J. Maheshwari, 5th edition.
- Natarajan's text book of orthopaedics and traumatology- 6th edition.
- Pocketbook of orthopaedics and fractures- Ronald McRae-2nd edition, (2006)
- Turek orthopaedics- principles and their application- 6th edition, (2005)
- Apley system of orthopaedic and fractures- 8th edition, (2001)
- Mercer's orthopaedic surgery- 9th edition, (2003)
- Tachdjian paediatric orthopapedics – 3rd edition, (2002)
- Harrisons princiles of internal medicine -17th edition

ENT

Standard Treatment Guidelines
Tamil Nadu Health Systems Project

Chapter 18

Ear

- External Ear Wax
- Furuncle
- Otomycosis
- Malignant Otitis Externa
- ASOM
- CSOM
- Deaf Mutism

Nose

- Acute Sinusitis
- Epistaxis
- Allergic Rhinitis
- Acute Parotitis
- Bacterial Parotitis

Throat

- Acute Tonsillitis
- Chronic Tonsillitis
- Hoarseness of Voice
- Stridor
- Acute Airway Obstruction

External Ear-Wax

Symptoms

Blocked ear

Pain

Tinnitus

Reflex cough on manipulation

Otoscopy

Brownish-black mass in external auditory canal.

Treatment

Soft Wax

* Aural syringing
* Removal with wax hook / scoop

Impacted Wax

* Softening of wax using coconut oil / olive oil / soda bicarbonate / glycerine / liquid paraffin

Treatment

* Syringing / removal using wax hook
* Removal under General anesthesia (GA) for children and un-cooperative patients.

Keratosis obturans

Collection of desquamated epithelium mixed with wax in external auditory canal is considered as keratosis obturans.

Symptoms

Severe pain

Difficulty in hearing

Serosanginuous discharge.

Otoscopy

Cerumen admixed with desquamated debris.

External canal granulations

Bone erosion

Treatment

Removal under GA using microscope.

Foreign bodies

* Animate - Insects
* Inanimate
* Organic - Seeds
* Non-organic - Stones, beads, cotton buds, match sticks etc.

Treatment

Insects

- Immobilised using oil and removed with forceps

Vegetable foreign bodies

- Removal with foreign body hook. (Syringing not advisable).

Non-organic foreign bodies

- Removal with syringing
- Removal with foreign body hook

Impacted foreign bodies

- Removal under GA using the microscope

Furncle

Definition

Furuncle is the infection of the root of a hair follicle usually caused by staphylococcal aureus. Seen in cartilaginous external auditory canal

Symptoms

Severe pain

Blocked ear

Movement of pinna painful

Signs

Tragal tenderness

Post-auricular lymphadenitis

Treatment

- Local
  * Packing ear with 10% icthamolglycerine.
  * Local heat application (fomentation)
- Systemic
  * Antibiotics (anti-staphylococcal) like Ampi-cloxacillin
  * Analgesics and control of diabetes if present

Otomycosis

Otomycosis is the fungal infection of the ear canal. Secondary growth occurs in patients using topical antibiotics for otitis externa or middle ear suppuration.

Symptoms

- Irritation and itching of ear
- Discharge (thick white debris like.)
- If the discharge is
  - * White blotting paper – white candida
  - * Black mass with mycelia – aspergillus niger
  - * Pale blue and green – Aspergillus fumigatus.

Investigations

Direct otoscopic examination

Microscopic examination in 10% KOH

Culture in sabourd's dextrose agar

Treatment

- Removal of fungal debris by ear toileting – syringing, suctioning, mopping.
- Anti-fungal ear drops (clotrimazole)
- Nystatin 1,00,000 units / ml ® against candida.
- 2% salicyclic acid in alcohol or 10% boric acid in alcohol.
- Application of gentian violet, keep ear dry.

Malignant otitis externa

Definition

External otitis in diabetic patients or those using immuno-suppressive drugs due to pseudomonas aeruginosa infection.

Symptoms

Severe pain

Swelling around the ear / facial weakness.

Otoscopy

External canal inflammation

Granulation

Cranial nerve paralysis

Facial palsy is common

Investigations

To exclude diabetes / CT scan temporal bone to rule out osteomyelitis of temporal bone.

Treatment

- Control of diabetes
- Avoid self-instrumentation
- Anti-pseudomonal antibiotics (Ciprofloxacin, carbenicillin, amikacin, tobramycin, ticarcillin, third generation cephalosporins, high dose by parenteral routes).

Acute suppurative otitis media (ASOM)

Common in children; associated with URI and exanthematous fevers.

Symptoms

- Pain
- Fever
- Ear discharge
- Hearing impairment and tinnitus

Symptoms in children:

Fever / vomiting / loose motion / sleeplessness / incessant cry / irritability

Otoscopy

Congested and bulging tympanic membrane

Mucopurulent discharge

Clinical Examination

Signs of upper respiratory tract infection

Treatment

- Treating focal sepsis (acute adeno tonsillitis, rhinosinusitis, URI allergy) and underlying condition
- Treatment of acute suppurative otitis media(ASOM)
- Systemic antibiotics
  - * Amoxicillin/ Ampicillin 20 to 40 mg/kg/day in divided doses x 10 days
  - * If allergic to penicillin - cotrimoxazole, erythromycin, lactamase- producing antibiotics. amoxycillin – Clavulanate, augmentin, cefuro-

xime axetil, cefixime (dose to be adjusted in pediatric cases)

- Decongestants- systemic and local
- Antipyretic, analgesics
- Ear toilet if discharge is present
- Dry local heat
- Surgical Myringotomy

Chronic Suppurative Otitis Media (CSOM)

- CSOM is classified as:
  - * Tubo tympanic disease (safe ear)
  - * Attico-antral disease (unsafe ear)

Tubo tympanic disease

Symptoms

- Ear discharge
- Mucopurulent / intermittent / not foul smelling / profuse / not blood stained
- Associated with Upper repiratory tract infection
- Hearing loss (conductive)
- Tinnitus

Stages

Active

Inactive (no discharge, only hearing loss)

Organisms

Proteus

Pseudomonas

Klebsiella

E. Coli.

Staphylococcus aureus

Bacteroides fragilis

Anaerobic streptococci

Otoscopy

Discharge mucopurulent / mucoid / dry

Perforation – central perforation

Polyp

Conductive deafness (Tuning fork test)

Middle ear mucosa

Normal in quiescent stage

Edematous

Velvety in active stage

Investigations

- Examination under microscope
- Audiogram
- Pus culture and sensitivity
- X-ray mastoids
  - * Both ears sclerotic
  - * May be pneumatised with clouding of air cells to assess co-morbid status

Treatment

1. Aural toileting
2. Antibiotic ear drops (with or without steroids)
   - * Neomycin
   - * Polymyxin
   - * Chloromycetin
   - * Gentamycin
3. Irrigation with 1.5% acetic acid
4. Treatment of primary source (adenotonsillitis and sinusitis, if present)
5. Surgical

- Inactive stage
  - * Pin hole perforation
  - * Chemical cautery with 30% trichloroacetic acid / 10% silver nitrate
- Large perforation
  - * Myringoplasty
- Active stage
  - * Cortical mastoidectomy with tympanoplasty.

Attico antral disease (unsafe ear)

Intracranial complications are common and morbidity is more due to the presence of **cholesteatoma**.

Symptoms

- Ear discharge
  - * Scanty
  - * Foul smelling

    * Blood stained in nature
- Continuous Hearing Loss
    * Conductive / mixed
    * Vertigo (inner ear involvement)
- Symptoms of extra-cranial complications
    * Fever (acute mastoiditis)
    * Vertigo, tinnitus, vomiting
    * Swelling around pinna (subperiosteal abcess)
    * Postaural sinus
    * Facial weakness
- Symptoms of intra-cranial complications
    * Fever
    * Meningitis (high grade)
    * Lateral sinus thrombosis
    * Fever with rigor
    * Headache
        » Meningitis
        » Extradural abscess
    * Brain abscess
        » Vomiting
        » Increased ICT

Clinical examination

Otoscopy

- Foul-smelling blood-stained discharge
- Granulation
- Polyps
- Attic perforation or postero-superior perforation
- Fistula test positive in cases of labyrinthine erosion

Investigations

1. Examination under microscope
2. Pure tone audiogram
3. Pus culture and sensitivity
4. X – ray mastoids (both ear)
5. High resolution CT – temporal bone
6. To assess for co-morbid status

Treatment

1. Treatment is essentially surgical
2. Modified radical mastoidectomy
3. Conservative treatment is tried in cases where surgery cannot be contemplated (Elderly >65 yrs or patients who are unfit for surgery)
4. Modes of conservative management
    * Cholesteatoma if small and easily accessible - suction clearance.
    * Periodic check ups are essential.
5. Polyps can be removed by cup forceps, and granulations cauterized by chemical agents such as silver nitrate, trichloroacetic acid
6. Aural toileting
7. Dry ear precautions.

Deaf Mutism

Definition

Deaf mutism is the inability to acquire speech due to profound or high degree of congenital or early acquired childhood sensory neural deafness in both ears.

Causes

* Prenatal
* Natal or perinatal
* Post natal

Prenatal causes

1. Genetic defects
2. Maternal infection
    * Toxoplasmosis
    * Rubella
    * CMV
    * Herpes type 1 and 2 and
    * Syphilis
3. Drugs during pregnancy
    * Aminoglycosides
    * Quinine
    * Chloroquine.
4. Radiation to mother in first trimester
5. Other factors: nutritional deficiency, diabetes, toxemia and thyroid deficiency

Perinatal causes

* Anoxia
* Prematurity
* Birth injuries, eg. Forceps delivery
* Neonatal jaundice
* Neonatal meningitis
* Ototoxic drugs

Post-natal causes

* Genetic
* Non-genetic

Identification of deafness

- History
- Normal responses of hearing child
  * **Neonatal period**
    » Moro's reflex
    » Auriculo palpebral reflex
  * **Infant**
    » Moro's reflex
    » Auriculo palpebral reflex
    » Oculogyric reflex
    » Clap test
    » At 18 months - 2 words sentence
- Audiometric examination
  * Free field audiometry
  * Visual reinforce audiometry
  * Play audiometry

Investigations

1. Brain Stem Evoked Audiometry (BERA) - is of value to find out the threshold of hearing infants, particularly the high-risk groups and in the diagnosis of retro-cochlear pathology
2. Otoacoustic emissions - Low intensity sounds produced by movements of the outer haircells of the cochlea and are produced either spontaneously or in response to acoustic stimuli
3. Electro-cochleography - It measures the electrical potentials arising in the cochlea and CN VIII in response to auditory stimuli within 1st 5 milliseconds
4. Impedance audiometry
5. High-resolution CT scan

Treatment

- Bilateral hearing aid
- **Cochlear implants.** They are electronic devices that convert sound signals to electrical impulses which then directly stimulate the cochlear nerve
- Auditory training
- Language communication

Referral

- If deaf mutism is suspected, the child should be referred to a higher centre.

Nose

Acute sinusitis

Definition

Acute sinusitis indicates inflammation of mucosa of paranasal sinuses.

1. Multi-sinusitis ís inflammation of more than one of the sinuses
2. Pansinusitis is inflammation involving all the sinuses and can be unilateral or bilateral
3. Acute maxillary sinusitis – is the commonest type of sinusitis
4. Acute ethmoidal sinusitis – is more common in infants and children

Symptoms

1. Pain
   * Maxillary sinusitis –pain over the sinus / upper jaw. May be refered to frontal area , temple, teeth
   * Frontal sinusitis –vacuum headache / office headache - starts early in morning, reaches maximum by midday and gradually subsides in afternoon
   * Ethmoidal sinusitis –mainly over bridge of the nose / at inner canthus/behind the eye
   * Sphenoidal sinusitis –retro orbital /retro auricular / occipital region
2. Purulent nasal discharge
3. Post-nasal drip

4. Disorders of smell –hyposmia / anosmia
5. Periorbital edema
6. Constitutional symptoms – fever , malaise
7. Tenderness
   * Maxillary –pressure over canine fossa
   * Frontal – over floor of the sinus
   * Ethmoid –on either side of bridge of nose
8. Cheek –edematous and reddish in maxillary sinusitis
9. Edema of lid –frontal sinusitis

Investigations

- X ray paranasal sinuses – Water's view - haziness or opacity /fluid level.
- CT-Paranasal sinuses in selected cases and in those cases where surgery is planned.

Epistaxis

Definition

Epistaxis is defined as bleeding through the nose.

Causes

Children – nose picking

Adolescents (especially males)

* Juvenile nasopharyngeal angiofibroma

Adults – idiopathic

Elderly – hypertension /neoplasm

History about bleeding

Quantity of blood loss

Duration

Which side of bleeding

Anterior /posterior

H/o trauma

H/o bleeding tendencies elsewhere

H/o chronic drug intake

H/o chronic liver disease

Family history

Chronic alcohol intake

Investigations

- Hemodynamic status
- Baseline investigatons
  * Complete hemogram
  * Coagulation profile
  * Blood grouping and typing
- X ray paranasal sinuses
- CT paranasal sinuses – if necessary

Treatment

- Resuscitative measures initiated if history and clinical parameters suggestive of hemodynamic-compromise

When to admit?

- Admit, treat, and observe for 24 to 48 hours
- If stable and no active bleeding, consider discharge

Allergic Rhinitis

Allergic rhinitis is an IgE mediated type I hypersensitivity reaction of mucus membrane of nasal cavity sometimes affecting paranasal sinuses due to mucosal continuity.

Symptoms

Seasonal allergic rhinitis	Perinneal allergic rhinitis
Sneezing Watery rhinnorhoea Nasal obstruction Itching of eyes and nose Itching of throat and ears	Viscous purulent rhinitis Loss of smell and taste Symptoms of associated sinusitis Symptoms of Eustachian tube dysfunction Sneezing and itching – less common

Signs

- Crease on tip of nose with crusting –**Darrier's line**
- Periorbital puffiness
- Dark circles around the eye
- Crease in the lower eyelid due to Muller's muscle spasm –**Dennie morgan line**
- Conjuctival congestion

- Epiphora
- Allergic salute

Anterior rhinoscopy

- Pale boggy edematous nasal mucosa
- Mulberry turbinate
- Polyps may be seen
- Posterior rhinoscopy
- Post-nasal drip

Investigations

- Peripheral blood eosinophilia
- Nasal smear – shows eosiniophilia
- X ray nose and paranasal sinuses
- Skin test
  - * Prick test
  - * Scratch test
  - * Intra-dermal test
  - * Radio allegro sorbent test –in vitro for IgE
  - * Paper radio immuno sorbent test

Treatment

1. Avoidance of allergen
2. Medical management
   - * Antihistamines
     - » Chlorpheneramine maleate
     - » Cetrizine
     - » Levocetrizine
   - * Intra-nasal steroids
     - » Budesonide
     - » Fluticasone
     - » Beclomethasone
   - * Sodium chromoglycate
3. Immunotherapy- attempted results variable
4. Surgery
   - * To correct co-existing structural abnormalities ,septal correction, polypectomy, Inferior turbinectomy, submucosal diathermy

Referral

Non-responders as well as individuals with recurrent episodes may be referred to higher centres.

Acute Parotitis

Viral

Mumps – Paramyxovirus

Usually affects < 15 years, seen predominantly in children

Incubation period –2-3 weeks

Symptoms

Bilateral parotid swelling

Pain over gland, ear pain

Low-grade fever

Arthralgia

Malaise

Headache

Investigations

Haemagglutination inhibition test

Complement fixation test

Treatment

Supportive measures- bed rest, adequate hydration, antipyretics

Prevention – MMR vaccine

HIV

Cystic enlargement associated with lymphoproliferative disorders

Gradual painless enlargement of one or more salivary glands

Epithelial-lined simple cysts or HIV-associated lymphoma

Investigations

FNAC , CT, MRI

Benign cyst – conservative management

Lymphoma – surgical management is diagnostic and therapeutic

If HIV is suspected, refer the case to a higher centre

Bacterial Parotitis

Definition

Mechanical blockage of salivary duct or due to stasis of salivary secretions coupled with secondary infection

results in bacterial parotitis. It is prevalent in debilitated patients.

Retrograde migration of intra-oral bacteria contributes to development of acute bacterial parotitis Sialolithiasis is the cause of acute parotitis in non-debilitated patients.

Symptoms

- Pain
- Swelling
- Tenderness

Induration of gland

Erythema of skin and oral mucosa

Fever

Investigations

- Leucocytosis
- Milking of gland for purulent discharge through the duct for culture and sensitivity
- Palpation and X- ray for stone

Organisms

Staphylococcus aureus

Streptococcus species

Haemophilus species

Anaerobic species

(Many produce beta-lactamases)

Treatment

- Appropriate antibiotics –oral or parental
- Adequate hydration
- Sialogogues
- Oral hygiene
- Warm compression
- Bimanual massage to milk purulent discharge from the gland
- Stone in the distal duct –expressed manually or removed after enlarging the duct orifice with a small incision. Failure indicates an abscess, which is confirmed by CT scan or USG
- Surgical drainage –elevation of anteriorly-based flap over superficial parotid fascia, parallel to branches of facial nerve with placement of external drain

Facial Palsy

Facial palsy can present as complete or incomplete palsy.

One third of facial palsy shows recovery within three weeks, and progresses to complete recovery

Medical management starts as soon as patient presents with symptoms

- Monitor progress of palsy using serial EMG and nerve excitability tests repeated every week
- Acoustic reflex should be monitored every week for the first sign of return of nerve function

Treatment

- Refer chapter Neurology-Facial palsy
- Eye care-pad, goggles and artifical tears
- Physiotherapy – active and passive
- Drugs
  - * Acyclovir 200-400 mg 5 times a day
- Vasodialators – xanthine nicotinate
- Ascorbic acid

Mutivitamins- vit B1,B6,B12

If there are no signs of recovery within 7 days then opt for surgical decompression

Surgical approaches:

Indications:

- Complete denervation
- Paralysis more than 4-6 weks
- Incomplete return of function within 60 days
- Recurrent facial palsy
- Nerve excitability tests shows a difference of 3.5 mA between both sides
- Schirmer's test –tear flow decreased more than 50%

Procedures:

- Surgical decompression
- Middle cranial fossa approach
- Bell's palsy shows skip lesions –so complete or total decompression

Indications for surgery

- No improvement after three weeks of medical treatment
- Worsening inspite of medical treatment

- Can be done upto two years after onset of palsy
- After two years consider - plastic surgical repair

Throat

Acute tonsillitis

- 80 –90% viral in origin - adenovirus, Epstein-barr virus are common viruses
- Secondary bacterial infection occurs - due to Group A beta hemolytic streptococcus, haemophilus influenza, staphylococcus

Symptoms

Throat pain

Difficulty in swallowing

Pyrexia

Cervical lympadenopathy

Refered otalgia

Voice change

Trismus –due to irritation of pterygoid muscles

Inflamed tonsils with exudates

Tender jugulo digastric nodes

Treatment

Initially symptomatic treatment with Paracetamol Systemic antibiotics such as:

- Penicillin –first choice
- Amoxycillin
- Erythromycin

(After throat swab culture and sensitivity)

Chronic tonsillitis

Chronic inflammation of tonsil starts as an acute inflammation .Minute abscesses get walled by chronic inflammation.

Types:

1. Chronic parenchymatous /hypertrophic tonsillitis:
   * Tonsils are uniformly enlarged and congested
   * They meet in midline “kissing tonsils”
2. Chronic follicular tonsillitis:
   * Beads of white discharge on surface of tonsil
3. Chronic fibrotic tonsillitis:
   * Tonsils are small and inflamed
   * Occur in adults
   * Anterior pillars are congested

Symptoms

- Repeated attacks of sore throat with little remission between attacks
- Odynophagia
- Fever
- Cough and irritation in throat
- Halitosis –Foul breath
- Thick speech , difficulty in breathing, swallowing and snoring

Signs

- Hypertrophied tonsils
- Congestion of anterior pillar
- Enlarged non-tender jugulodigastric nodes
- Extrusion of purulent discharge on pressure over the tonsil with a tongue depressor – Irwin Moore’s sign

Investigations

- Complete hemogram
- Blood sugar /urea /serum creatinine
- Blood grouping and typing
- Chest X-ray
  * PA view
- X ray skull lateral view
- ECG

Treatment

1. Conservative management
   * Antibiotics
     » Amoxycillin
     » Erythromycin
     » Cephalexin
   * Analgesics – Paracetamol
   * Bed rest
2. Surgery – Tonsillectomy indications:
   * Recurrent episodes of tonsillitis
     » 7 or more episodes in a year
     » 5 or more episodes /year x 2 years
     » 3 or more episodes/year x 3 years

* Peritonsillar abscess
  » Hypertrophied tonsil causing airway obstruction, difficulty in speech and in swallowing
* Systemic diseases
  » Rheumatic heart diease
  » Glomerulonephritis

Hoarseness of voice

Definition

Hoarseness is described as a hard discordant voice, uncharacteristic of one's age, sex and character. The voice may sound weak, scratchy or husky.

Causes

- Puberty
- Excessive voice abuse
- Laryngitis
- Allergy
- Alcohol / tobacco abuse
- Tonsillitis
- Foreign body in the throat
- Vocal cord nodules / polyp / paralysis
- Ingestion of caustic liquid
- GERD
- Cancer larynx

When to consult a doctor

Hoarseness lasting for more than three weeks, throat pain not from a cold or flu, hemoptysis, difficulty in swallowing, lump in neck etc.,

Investigations

- Complete blood count, differential count.
- Throat swab for culture and sensitivity
- X-ray neck / chest
- Laryngeal mirror examination
- Medical gastroenterology and thoracic medicine evaluation
- CT- scan neck / chest

Treatment

1. Laryngitis – avoid smoking / alcohol
2. GERD
   * Acid reducing drugs
   * Prokinetic drugs
   * Avoid spicy meals
3. Vocal nodules / polyp
   * Voice rest
   * Speech therapy
   * Surgery
4. Allergy – inhaled steroids, avoidance of allergens, desensitization
5. Foreign body in throat – removal
6. Cancer of larynx
   * Early stage: Radiotherapy,
   * Advanced stage: Surgery

Prevention:

1. Things to do
   * Voice rest
   * Drink plenty of water
2. Things to avoid:
   * Breathing foul air
   * Using tobacco or marijuana
   * Alcohol
   * Voice abuse
   * Clearing throat continuously
   * Whispering loudly for long time
   * Trying to change one's natural voice

Stridor

Definition

Stridor is noisy, harsh and difficult breathing due to obstruction of the airway It is classified depending on its occurrence with respect to the phase of respiration.

1. Inspiratory
2. Expiratory
3. Biphasic
   * Inspiratory stridor occurs due to an obstruction at or above the level of vocal cords.
   * Expiratory stridor is due to obstruction at bronchiolar level. eg. asthma
   * Biphasic stridor occurs in tracheal and subglottic stenosis.

In children the stridor of laryngo-tracheo-bronchitis has a crowing quality and is called **'Croup'**

Causes

Acute

- Laryngotracheobronchitis (6 months – 2years)
- Aspiration of foreign body
- Bacterial tracheitis (< 3 years)
- Re-tropharyngeal abscess
- Peritonsillar abscess (adolescent and preadolescent)
- Spasmodic croup
- Allergic reaction
- Epiglottitis (2 to 7 years)

Chronic

- Laryngo-malacia
- Subglottic stenosis
- Vocal cord paralysis
- Laryngeal web
- Laryngeal cyst
- Hemangioma / papilloma
- Tracheo-malacia / tracheal stenosis
- Growth larynx

Symptoms

- Restlessness, anxiety and distress, labored breathing
- Noisy breathing, predominantly on inspiration
- Difficulty in swallowing and speaking
- In children : cyanosis, chest retraction, tachypnea, altered level of consciousness, feeding difficulties

Patient may have completely normal vital signs, normal oxygen saturation ; but this should be reassuring. Low oxygen saturation and cyanosis are ominous signs of impending complete upper airway obstruction. When patient progresses to complete airway obstruction, stridor and noisy breathing ceases, and unless intervention takes place the patient dies.

Treatment

- Humidified oxygen
- Steroids-hydrocortisone 100 mg iv
- Antibiotics – Ampicillin / third generation cephalosporins
- Epinephrine – given subcutaneously

Epiglottitis

Causes

- Inflammatoy
- Viral

Investigations

- Throat culture

Treatment

- Antibiotics – Ampicillin

Acute airway obstruction

Adult

Rule out

- Underlying malignancy
- Abscess
- Laryngeal cyst
- Laryngocele

Assess severity

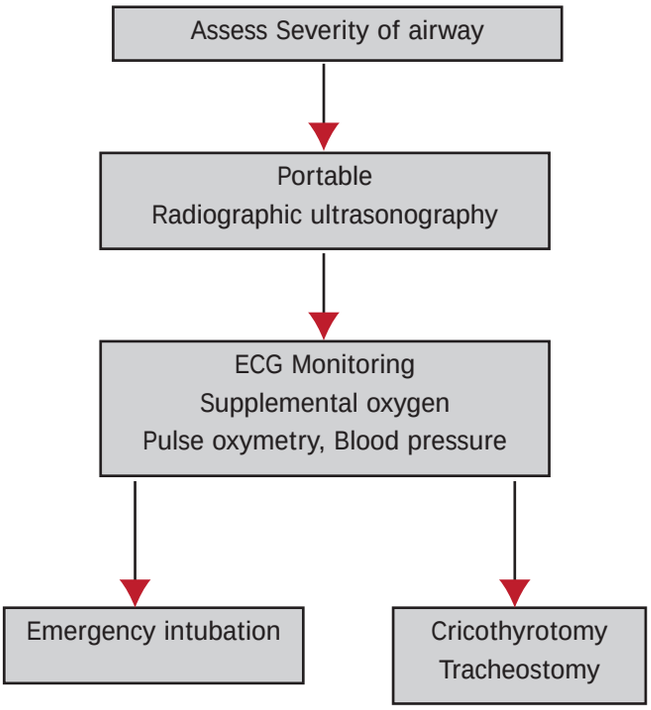
Emergency treatment

- Head extension with lower jaw thrust forward
- Suctioning for clearing secretions
- Ventilatory mask with 100% oxygen
- Endotracheal intubation or tracheostomy

In stable patient

- Plain X-ray neck
- Fibreoptic endoscopy / micro-laryngoscopy / bronchoscopy to find out the cause

Acute airway obstruction-treatment guidelines



Refer the patient to a higher centre as early as possible after initial resuscitative measures.

Ophthalmology

Standard Treatment Guidelines
Tamil Nadu Health Systems Project

Chapter 19

1. Stye
2. Hordeolum Internum
3. Chalazion
4. Refractive Errors
5. Conjuctival Allergic Disorders
6. Primary Open Angle Glaucoma
7. Lens Induced Glaucoma
8. Neovascular Glaucoma
9. Senile Cataract
10. Nuclear Cataract
11. Uveitis
12. Retinal Detachment
13. Diabetic Retinopathy
14. Orbital Cellulitis
15. Trachoma
16. Vitamin A Deficiency
17. Chemical Injuries of the Eye
18. Intraocular Foreign Bodies

Stye (Hordeolum Externum)

Definition

It is acute suppurative inflammation of the Zeis glands

Etiology

Bacterial - Staphylococcus

Symptoms

- Pain
- Lid swelling
- Watering

Sign

A painful swelling at the lid margin

Treatment

- Hot compresses 2-3 times per day
- Antibiotic eye drops like Ciprofloxacin six times per day
- Systemic antibiotics and anti-inflammatory tablets for three or four days
- When pus points out it should be let out by pulling the affected lash – Epilation

Hordeolum Internum

Definition

It is acute suppurative inflammation of the Meibomian gland

Symptoms

Pain, lid swelling and watering

Sign

A painful swelling away from the lid margin

Treatment

- Antibiotic eye drops six times per day
- Systemic antibiotics and anti-inflammatory tablets for three or four days
- The pus should be drained by incising it

Chalazion

Definition

It is a chronic granulomatous inflammation of the Meibomian gland

Etiology

- Uncontrolled diabetes mellitus in adults
- Refractive errors

Symptoms

Swelling of the lid and watering

Sign

Painless nodular swelling away from the lid margin

Course

- Spontaneous resolution
- If secondarily infected, then it is called as internal hordeolum
- It may burst through the conjunctiva
- Very rarely a malignant change may occur (Meibomian carcinoma) especially in old age with a history of recurrence

Treatment

- Incision and curettage under local anaesthesia
- In Marginal chalazion
  - * injection of Depot steroids: 0.1 to 0.2 ml of Triamcinolone 40 mg/ml may be helpful
- Correction of refractive errors if any

Information to the patient

If a chalazion recurs soon after removal, or is rapidly increasing in size, or ulcerates, a biopsy must be done to rule out malignancy.

Refractive Errors

Definition

Refractive error is an error in the focusing of light by the eye and a frequent reason for reduced visual acuity.

Prevalence

- Global prevalence - 800 million to 2 . 3 billion
- Severe refractive error - 5 million
- Uncorrected refractive error. - 153 million

- Of this 153 million, 90% live in developing countries according to

Types

Myopia (short sight)

- Eye cannot see distant objects clearly. Image is focused in front of the retina and happens when the eyeball is elongated.

Hypermetropia (Long sight)

- Eye cannot see closer objects clearly. Image is focused behind the retina.and happens when the eyeball is shorter in length.

Astigmatism

- No single point focus is formed. Refraction is unequal in different meridians.

Presbyopia

- It is a physiological process which affects accomodation, starts around 40 years of age. Here the near point recedes due to loss of elasticity of lens and weakening of ciliary muscle, necessary for accommodation

Clinical Aspects:

- Indistinct distant vision
- Headache and pain in the eyes
- Eye strain.

Diagnostic Aspects:

- Confirmed by eye care professionals during eye examination. In combination with retinoscopy the Doctor instructs the patient to read the eye chart while changing the lenses objectively to estimate the refractive error.
- Cycloplegic agents are used to measure accurately the amount of error , especially in children.
- Automated refractor is an instrument that is sometimes used in place of retinoscopy for objective estimation.

Rx Treatment

- Very mild refractive error - No symptoms; leave it uncorrected.
- Symptomatic:
  - * Corrected by spectacles
  - * Contact lens
  - * Refractive surgery
  - * combination of all.

Spectacles:

• Myopia	-	spherical concave lenses.
• Hypermetropia	-	spherical convex lenses.
• Astigmatism	-	cylindrical or sphero cylindrical lenses.
• Presbyopia	-	spherical lens depending on age and ocupation of the patient.

Bi focal lenses:

Upper segment corresponds to distant vision and lower segment for near.

Multi focal lenses:

- * No separating lines.
- * Near vision power gradually increases. So, it is convenient for all distances.

Contact lenses:

- Normal field of vision, cosmetically better, aberrations associated with spectacles eliminated.
- They do not reduce the degree of myopia.
- Commonly used lenses are soft contact lens, semi soft or gas permeable, soft toric lenses for astigmatism, bi focal contact lenses for refractive errors

Operations:

- LASIK:
  - * Laser Assisted In situ Keratomileusis.
  - * It is an out patient surgical procedure.
  - * Microsurgical instruments and laser are used to reshape the cornea and improve the way the eye focuses the light rays.
  - * Ideal candidate should be 18 years of age, not pregnant or nursing, free of any other eye disease and should not have any change in power in the last few years.

* The refractive error must be within the range of 0.5 D to 12 D of myopia, 0.5 D to 4 D of hypermetropia, or 6 D to 8 D of astigmatism.

- LASEK: Laser Assisted In situ Epi Keratomileusis.
- PRK: Penetrating keratoplasty
- Phakic IOL implantation.
- Refractive lens exchange.
- Conductive keratoplasty for presbyopia using radio-frequency current.
- Follow up-growing children upto 18 to 20 years every year and later every two years.

Complications:

- High myopes
  * Retinal detachment,
  * Open angle glaucoma.
- Hypermetropes
  * Angle closure glaucoma.

Prevention:

To avoid consanguineous marriage to prevent genetic predisposition.

Conjunctival Allergic Disorders

Introduction

The conjunctiva is ten times more sensitive than skin to allergens.Inflammation of conjunctiva due to allergic or hypersensitive reaction to local or systemic allergens, which may be immediate or delayed. Conjunctivitis can occur as an allergic reaction to common allergens like pollens, grass, animal dandruff, house dust. More common in young males between 4-20 years and in summer, it is prevalent in tropics, less in temperate zones and rare in cold climate

Classification

- Simple allergic conjunctivitis
  * Mild,non specific
  * Recurrent episodes occur with renewed contact with the allergen.
- Vernal keratoconjunctivitis (spring catarrh)
  * Recurrent , bilateral ,self limiting ,allergic inflammation of conjunctiva
  * Periodic seasonal incidence in young patients
  * Usually males between 4 to 20 years.
  * Family history of atopy is common.
  * Common in summer and greater prevalence in tropics, less in temperate zones and non - existent in cold climates
- Atopic keratoconjunctivitis
  * Adult equivalent of vernal keratoconjunctivtis associated with atopic dermatitis
- Giant papillary conjunctivits
  * Inflammation of conjunctiva with formation of very large sized papillae. Can occur due to contact lens, protruding suture ends, ocular prosthesis, etc.
- Phlyctenular keratoconjunctivitis
  * Nodular allergic reaction by conjunctival and corneal epithelium to endogenous allergens mainly tuberculosis.
- Contact dermatoconjunctivitis
  * Allergic disorder involving conjunctival skin or lids along with surrounding area of face.
  * Can occur due to chemicals, dyes, cosmetics or topical eye drops.
- Granulomatous conjunctivitis
  * Chronic, specific inflammation of conjunctiva associated with regional lymphadenitis occurring in tuberculosis, sarcoidosis, syphilis, leprosy

Symptoms

- Intense itching, burning, watery discharge, photophobia.
- Contact dermatoconjunctivitis is associated with weeping eczematous reaction of areas in contact with the allergen

Signs:

- Simple allergic conjunctivitis
  * Chemosis, hyperemia of conjunctiva, lid edema
- Vernal keratoconjunctivitis
  * Palpebral form: shows cobble stone or giant papillae on tarsal conjunctiva
  * Bulbar form:

» Dusky red congestion , thick ropy discharge

» Whitish raised dots along the limbus-Trantas spots

- Phlyctenular conjunctivitis
  * Necrotizing conjunctivitis with pustules or miliary form
  * With multiple phlyctens, corneal involvement with fascicular ulcer
- Punctate epithelial keratitis
  * Ulcerative vernal conjunctivitis, pseudogerontoxon or classical
  * Cupid bow outlining the limbal area

Diagnosis

- Viral conjunctivitis
- Bacterial conjunctivitis
- Foreign body in the fornix / conjunctiva

Treatment

- Removal of offending drug/allergens
- Steroid eye drops in acute and severe cases for short duration;
- Subtarsal steroid injection is severe cases of vernal keratoconjunctivitis.(Steroids need to be used cautiously due to chances of steroid induced glaucoma)
- Mast cell stabilizers (sodium cromoglycate, olopatidine) to prevent recurrence
- Cycloplegic eye drops/ointment in case of corneal involvement
- Treatment of primary conditions: Tuberculosis, septic foci, parasitic infestation

Course and Outcome

- Resolves spontaneously once allergen is eliminated, but recurrence is common.
- Vernal keratoconjunctivitis: self limiting course and usually disappears by 10 - 15 years of age.

Prevention

Avoid areas in which the allergen is present (eg. dusty environments). Start using mast-cell stabilizers before contact with the allergen is eliminated (eg. in seasonal allergic conjunctivitis)

Information to patients

- The condition can only be controlled and not cured.
- It is self-limiting in cases that begins in child hood.

Steroid drops should be avoided at all cost to prevent steroid - related sight threatening complication

Primary Open Angle Glaucoma (POAG)

Definition

Primary open angle glaucoma is a bilateral chronic slowly progressive optic nerve head neuropathy with characteristic morphological and functional changes associated with an intraocular pressure higher than the nerve fibres can tolerate, with open angles.

Primary open angle glaucoma is more likely in

- Patients more than 40 years of age
- Race: Blacks
- Positive family history
- Diabetics
- Myopes
- Steroid responders

Clinical Aspects

Symptoms

- Patients are asymptomatic till gross field changes occur.
- Unexplained chronic headache
- Occasional pain or redness
- Frequent change of presbyopic glasses
- Delayed dark adaptation
- Visual field defects

Signs:

- Anterior chamber normal depth
- Pupil reaction-normal till late stages
- Increased intra-ocular pressure
- Variation of IOP > 5mm Hg between two eyes
- Open angles by Gonioscopy
- Increased cup-disc ratio in fundus examination
- Glaucomatous field defects

Diagnosis

- Optic disc changes
- Classical field defects
- Increased IOP
- Gonioscopy

Optic Disc Changes:

- Focal enlargement of inferior temporal quadrant
- Bayonetting double angulation of blood vessels
- Thinning of neuroretinal rim
- Nasal shifting of blood vessels
- Splinter hemorrhages at disc margins
- Baring of circumlinear vessels
- Laminar dot sign

Visual Field Defects:

- Diffuse nerve fibre loss
- Sparing of blind spot
- Paracentral scotomas / Siedel's scotoma
- Bjerrums or arcuate scotoma / ring scotoma
- Roennes nasal step
- Tubular field
- Only paracentral temporal island of vision
- No perception of light

Rx Treatment

Topical:

1. Beta blockers:
   * Timolol,betoxalol,levobunalol-0.25% - 0.5% Twice daily application reduces aqueous secretion by direct action on secretory epithelium of ciliary body
   * Epinephrine and dipivalyl epinephrine decreases aqueous production by vasoconstriction and increases outflow by stimulation of beta receptor in outflow channels.
   * Latanoprost 0.005% (prostaglandin analogue) increases uveoscleral outflow
2. Topical carbonic anhydrase inhibitors:
   * Dorzolamide 2% - decreases aqueous production
   * Cholinergic agents: Pilocarpine increases outflow
3. Alpha antagonists:
   * Prazosin,thymoxamine,dipiprazole
   * Causes miosis through blockage of iris dilator muscle
4. Alpha agonists:
   * Apraclonidine-inhibits aqueous formation
   * Brimonidine-in addition increases uveoscleral outflow.

Systemic Agents

- Carbonic anhydrase inhibitors decrease aqueous production.
  * Acetazolamide 250mg
  * Dichlorphenamide 50mg
  * Methzolamide 50mg
  * Ethoxazolamide 125mg.

Neuro Protective Agents

- It had been suggested that Betaxalol 0.5% and Brimonidine 0.5% drops have a neuroprotective effect.
- Systemic NMDA antagonists, sodium and calcium channel blockers and others are under investigation
- A large randomised clinical trial with Mementine has been concluded: the results are awaited.

Laser

Argon laser trabeculoplasty

- Burns the trabecular meshwork for 360 degrees by shrinkage and fibrosis of collagen
- Opening of inter-trabecular spaces causes increase in aqueous outflow.

Surgical Management

- Partial thickness filtering surgery
- Sub-scleral Trabeculectomy

Course and Outcome

- POAG is a slowly progressive disease leading to optic nerve damage and finally optic atrophy
- A concise explanation about the nature of the disease is given to the patient

* How to instill drops, intervals between medication
* Adverse effects of drops
* Follow up once in 6 months to one year with IOP measurements, fundus, field evaluation and gonioscopy
* Filtering surgery if the patient is not responsive to maximum medical treatment, laser treatment or intolerant to medication.
* Neuro protective agents are given along with all the modes of treatment
* Regular treatment and proper follow up will prevent optic nerve damage and further disease progress

Lens Induced Glaucoma

Lens induced glaucoma is a secondary glaucoma resulting from abnormalities in size, shape, site of the lens or release of lens protein due to hypermaturity of the cataract / trauma and following inflammation.This type of secondary glaucoma may show characteristics of closed angle or open angle. Unless the condition is relieved by the removal of the lens as early as possible, a permanent rise in the intraocular pressure causing damage to the optic nerve.

Clinical types:

- Phacomorphic
- Phacolytic
- Phacoanaphylactic/phacotoxic

Symptoms

- Sudden onset of pain
- Progressive in nature
- Defective vision.

Signs:

- Cornea may be normal or hazy.
- Anterior chamber is shallow in phacomorphic, deep in phacolytic type with pseudohypopyon.
- Pupilary area: Mature, hyper mature or intumescent lens
- Aqueous flare cells and keratic precipitates
- Intra ocular pressure is raised to more than 22 mm/Hg (Schiotz tonometer)

Diagnosis:

- History of recent surgery or trauma in one or other eye will be present in phacoanaphylactic type.
- Examination of the contra lateral eye is essential to rule out primary glaucoma.
- It is made mainly on clinical grounds. Tonometry is done to measure the increased intra ocular pressure.
- Slit-lamp examination is done to rule out associated uveal inflammation.
- Fundoscopy is done to know the extent of the optic nerve damage.

Treatment:

- It is mainly surgical.
- Early cataract extraction is the definitive treatment.
- Medical treatment is indicated to control intra ocular pressure before surgery.
- It is achieved with topical hypotensive drugs and systemic acetazolamide.
- The uveal inflammation is controlled with topical steroids.
- Systemic steroids are needed if the reaction is very severe.

Course and outcome:

Depends upon the early removal of the primary cause. If left untreated, severe lens induced uveitis occurs and the eye goes into a recurrent chronic inflammatory state with loss of function. The prognosis is usually good if treated early under cover of anti inflammatory drugs along with adequate control of intra ocular pressure

Referral

All cases of lens induced glaucoma are advised to get admitted under the supervision of the Ophthalmologist

Prevention:

The condition is preventable. Patients are advised to get operated before the stage of hyper maturity so that the complication of lens induced secondary glaucoma does not arise. This necessitates creation of awareness amongst health care professionals in the primary level of health delivery system.

Neo Vascular Glaucoma

Definition

It is one of the important entities causing great morbidity and visual loss. It is a secondary glaucoma due to the formation of a fibrovascular membrane on the surface of the iris and angle.Initially the fibrovascular membrane covers the trabecular mesh work and the angle is open. With time, this tends to contract to form a zipper like mechanism with peripheral anterior synechiae. This gives rise to secondary angle closure glaucoma.

Causes

- Diabetic retinopathy especially proliferative type with new vessels
- Central retinal vein occlusion
- Central retinal artery occlusion
- Eales disease
- Carotid artery disease
- Anemia
- Leukemia
- Any chronic retinal proliferative retinopathy
- Chronic recurrent iridocyclitis

Symptoms

- Severe pain with redness and watering
- Defective vision / loss of vision
- Increased intra ocular pressure

Signs

- Circumciliary congestion
- Hazy cornea due to edema
- Contracted pupil
- Deep anterior chamber with inflammatory reaction
- Tufts of new vessels near the pupillary margin
- Extension of neo vascular membrane over the ciliary body scleral spur and angle structures
- Extensive peripheral anterior synechiae
- Ectropion uveae
- Bullous keratopathy
- Intractable glaucoma

Investigations

- Clinical features with slit lamp examination
- Fundoscopy to rule out the underlying retinal pathology
- Gonioscopy to find out whether the angle is open or closed
- USG to find out the retinal pathology if the media is hazy
- Ultrasound Bio-Microscopy (UBM) of the anterior segment
- Cardiologist's opinion to rule out carotid artery disease and coronory ischemia

Treatment

Medical management.

- Topical atropine 1% drops
- Topical steroid drops
- Topical hypotensive agents and
- Systemic acetazolomide.

Surgical Management

- Antiglaucoma surgery at the avascular area preferably with pharmaocological modulation.
- Glaucoma valve implant surgery can be tried.

Complications:

- An intractable glaucoma leading to a painful non-functioning blind eye.
- In later stages, in a non-functioning painful eye, cyclo destructive procedures are done

Prevention

Diagnosing the causative factors and treating them properly. At the primary level, awareness amongst the diabetics about the possibility of developing this condition should be created. Screening the diabetics once in a year should be stressed upon..

Senile Cataract

Introduction

Senile cataract is one of the leading causes of preventable blindness of the developing world. With

advancing age, the eye lens undergoes progressive oxidative damage of its membranes and proteins, giving rise to this particular condition. It is common after 50 years of age. In todays modern eyecare, this condition is manageable as a daycare procedure with excellent success rates. Through NPCB, this is done free of cost in all Government hospitals

Causes

- Advancing age
- Congenital (hereditary or intra - partum insult to fetus)
- Trauma
- Complication of an intraocular inflammation or tumour
- Long - term steroid use

Pathology of Senile cataract

- Lamellar separation:
  - * Characteristic demarcation of cortical fibres due to separation by fluid. Can be seen only with a slit lamp
- Incipient stage:
  - * Wedge shaped spokes of opacities with clear areas between them in the periphery. The bases of wedge shaped opacities are present in the peripheral area.
  - * Most common in the lower nasal quadrant. Under oblique illumination they appear grey. Under the ophthalmoscope, retinoscope and slit lamp they appear black against red glow.
- Intumescent stage:
  - * There is progressive hydration of the cortical layers causing swelling of the lens, making the anterior chamber shallow.
- Immature stage:
  - * There is progressive opacification of the cortex. Iris shadow can been seen because some clear lens substance is present.
- Mature stage:
  - * The entire cortex becomes opaque. The swelling subsides
  - * Iris shadow is absent.
- Hypermature stage:
  - * The lens becomes more and more shrunken and the cortex disintegrates. Owing to shrinkage the lens and iris become tremulous and the AC becomes deep. Finally degeneration of the suspensory ligament occurs and this may lead to luxation of the lens.
  - * At the stage of maturity the cortex may become fluid and the nucleus sinks down. The liquefied cortex is milky and the nucleus appears as a brown mass which alters with the position of the head-called as Morgagnian cataract.

Nuclear Cataract

Early onset, may occur soon after 40 years of age.There is increased tendency of nucleus to go for sclerosis.

Grades of nuclear cataract

- Greyish
- Yellowish
- Amber
- Brown/Black

Symptoms/ Signs

- Dimunition of vision/decreased visual acuity
- Blurring of vision, glare.
- Coloured haloes- in the intumescent stage
- Impairment of vision
- Progressive myopia- due to increased refractive index of the nucleus.So, a previously presbyopic patient maybe able to read without the aid of spectacles, referred to as ‘‘second sight’’

Treatment

No medical treatment is possible.

Surgical

- Extra capsular cataract extraction with IOL implantation
- Small incision cataract surgery with IOL implantation
- Phaco-emulsification with IOL implantation

Referral

- NPCB through its DBCS in all districts is working towards reduction of prevalence. Please refer to

nearest operating centre.

- Surgery with IOL implantation is done free of cost in all government hospitals

Uveitis

Definition

Inflammation of the uveal tract is termed uveitis.

Classification

Based on the anatomic location of the inflammation, uveitis may be classified as

- Anterior
- Intermediate
- Posterior
- Panuveitis
- Recurrent/chronic anterior uveitis

Causes

- Auto-immune
- Infectious

Acute anterior uveitis

Symptoms

- Pain
- Redness
- Lacrimation
- Photophobia
- Decrease in vision

Signs

- Ciliary/diffuse congestion
- Keratic precipitates
- Cells and flare in anterior chamber
- Minimal (spill over) cells in anterior vitreous

Chronic anterior uveitis may often be aysmptomatic and may present with a white eye and complications like cataract and secondary glaucoma.

Intermediate uveitis

Symptoms

- Decrease in vision
- Floaters

Signs

- Minimal (spill over) cells in anterior chamber
- Vitreous haze and cells
- Cystoid macular edema (CME)
- Snow banking

Posterior uveitis

Symptoms

- Decrease in Vision
- Floaters

Signs

- Minimal (spill over) cells in anterior chamber
- Vitreous haze and cells
- CME
- Focal inflammatory lesions in the fundus (choroiditis/retinitis/retinal vasculitis/neuroretinitis)

Panuveitis:

Symptoms and signs of both anterior and posterior uveitis occurs in this condition

Symptoms

- Pain
- Redness
- Lacrimation
- Photophobia
- Decrease in vision

Signs

- Ciliary/diffuse congestion
- Keratic precipitates
- Cells and flare in anterior chamber
- Vitreous haze and cells
- Focal inflammatory lesions in the fundus (choroidits/retinitis/retinal vasculitis)

Complication

- Complicated cataract
- Secondary glaucoma
- CME
- Cyclitic membrane

- Retinal detachment
- Phthisis bulbi

Investigations

There is no such thing as a panel of investigations appropriated to uveitis. A careful history is more likely to yield results than a panel of poorly chosen and expensive tests.

Things to be done in all patients with Uveitis

Detailed history

- Previous episodes, treatment taken and response to treatment
- Duration of present episode, treatment taken and response to periocular steroid
- Any associated systemic conditions specifically Tuberculosis, Syphilis, HIV infection, Rheumatic problems, Malignancies, Diabetes, Hypertension

Ocular examination

- Visual acuity
- Document RAPD
- Accurate recording of slit lamp findings - especially, characterization of keratic precipitates, grading of cells and flare
- IOP recording and Gonioscopy
- Dilated fundus examination with scleral indentation. If view to the fundus is too hazy, B scan should be done.

General examination Naming and meshing

After completion of examination, 'naming and meshing' by a uvea specialist should be done to arrive at a short list of most likely diffrential diagnoses.

Anterior Uveitis

- First episode of non-granulomatous anterior uveitis without any positive systemic symptoms/ signs No investigations

First episode of anterior uveitis with positive symptoms/signs of systemic disease

Investigations

- Investigations pertaining to the particular symptom/sign (eg. low back pain - do X ray SI joints (bilateral oblique view) and Xray LS spine AP/ Lateral views

Recurrent/chronic anterior uveitis

Investigations

- Based on the short list of most likely differntial diagnoses arrived at by naming and meshing
- Baseline investigations (useful also for Pre-steroid work up in patients who require systemic steroid therapy)
  - Chest Xray- to rule out Tuberculosis, Sarcoidosis
  - Mantoux test
  - TPHA-to rule out syphilis
  - ELISA for HIV
  - Blood sugar evaluation / Glycosylated Hb
  - Hb,TLC,DLC,ESR
  - Urine microscopy
  - Screening tests to rule out any active systemic infections

Intermediate/posterior/panuveitis

Investigations

- Same as for recurrent anterior uveitis
- FFA and OCT - particularly in case of CME and retinal vasculitis

Treatment

- Topical steroids - Prednisolone acetate drops (frequency to be decided based on the degree of inflammation; if moderate to severe, given hourly; if mild, less frequently)
- Cycloplegics
  - Atropine drops tid if severe uveitis
  - Cyclopentolate drops bd if mild to moderate
- Antibiotic drops are not necessary
- Systemic steroid therapy is generally not required
- Refer for further evaluation and management (Periocular or systemic steroids/ immunosuppressants/specific therapy)
- Urgency of referral should be based on the sever-

ity of disease.

Retinal Detachment (RD)

It is one of the major ocular disease conditions causing visual loss.

Causes

- High myopes
- Trauma
- Family history
- Cataract surgeries are major risk factors.

Definition

It is a separation of neurosensory retina from retinal pigment epithelium due to accumulation of fluid in the sub-retinal space through a retinal break, hole or a tear

Symptoms

- Occurrence of flashes
- Floaters in the vision
- Loss of vision

Types

- Rheumatogenous RD
- Tractional RD – Diabetic retinopathy
- Exudative RD
  - Choroidal tumors
  - Inflammatory diseases
- Combined RD

Investigations

- History
- Vision testing - will be decreased
- IOP - will usually be low
- Mild Uveitis
- Detailed dilated fundus examination by indirect ophthalmoscopy will yield details regarding the extent and nature of RD; determine if macula is involved or not.
- Localize the break /hole/ tear by fundus evaluation
- Examine for any associated Choroidal detachment, posterior vitreous detachment
- Look for shifting fluid in exudative RD
- B-scan to confirm if fundus view is hazy
- Evaluate the fellow eye in detail - treat if any lattice, retinal break with laser or cryotherapy

Treatment

RD surgery can be done only by tertiary eye care centres with infrastructure and trained personnel.

- External procedure: Scleral buckling surgery with cryopexy with or without sub retinal fluid drainage.
- Internal procedure:
  - Pars plana vitrectomy with endo laser
  - Post-operative vision is better if the macula was not detached pre-operatively and the RD is treated within 48 hours

Chances of recurrence of the RD as well as the possible need for multiple surgeries should be explained to the patient.

Diabetic Retinopathy

India has become the diabetic capital of the world. There is a high incidence of Diabetic Retinopathy, among patients who are suffering from chronic diabetes mellitus of more than 15-20 years duration. Early diagnosis and intervention is very crucial in dealing with this malady

Classification

NPDR(non proliferative)	PDR(proliferative)
Mild Moderate Severe	High risk Advanced Diabetic Eye disease Vitreous Hemorrhage Traction RD Neovascular Glaucoma
Maculopathy CSME (clinically significant)	

Fundus

- Veins dilated and tortuous
- Dot and Blot hemorrhages

- Hard exudates
- Cottonwool spots
- Micro-aneurysms
- NPDR stage
  * New vessels and traction bands seen
- PDR stage
  * Vitreous hemorrhage and traction RD complicate the picture
- Maculopathy runs as a distinct disease entity with maximum visual defect

Diagnosis

- Symptom analysis: failing vision,floaters and diabetic status
- Dilated fundus examination
- Fundus flouroscein examination (FFE)
  * Iodine based dye injected into ante-cubital vein and fundus pictures taken and digitally analysed.
  * Leaks and ischemic areas can be picked up
- Optical coherence Tomography:
  * Non-invasive retinal tissue pictures

Treatment

- NPDR:
  * Periodic observation
  * Antioxidants
  * Control of Diabetes.
- CSME:
  * Focal/Grid photocoagulation
- PDR:
  * Pan retinal photocoagulation
  * Intravitreal injection of VEGF inhibitors
    » Bevazizumab
    » Ranibizumab)
  * Vitrectomy may be needed for Vitreous hemorrhage,tractional RD

Prevention

Today Telemedicine has taken sophisticated technology to the rural masses Yet the cornerstone is increasing awareness to public and stress the need for advocacy

Orbital Cellulitis

Definition

Orbital cellulitis is a condition in which the active infection of the orbital soft tissue is present posterior to the orbital septum. The onset of orbital cellulitis is an emergent situation requiring definitive therapy. This condition needs to be urgently treated, since purulent optic neuritis can cause permanent loss of vision.

Causes

Bacterial infections can occur from 3 primary sources:

- Direct spread from sinuses (>90%) viz. frontal, ethmoidal and maxillary sinuses
  * Dacryocystitis
  * Dacryoadenitis
  * Dental infections.
- Direct inoculation
  * Following trauma or skin infection of face and eyelids.
- Bacteremic spread from a distant source
  * Otitis media
  * Pneumonia.

Symptoms

- Recent onset of lid edema
- Proptosis and chemosis
- Blurring of vision
- Reduction in ocular motility
- Pain on movement of the globe

Differential Diagnosis:

- Facial cellulitis
- Blunt orbital trauma
- Dacryocystitis
- Dacryo-adenitis
- Other orbital space occupying lesions

Investigations

- X-ray Para nasal sinuses

- Haemogram
- Culture and sensitivity of
  - * Fluid obtained by incision and drainage from the infected site
  - * Periorbital abscess
  - * Material obtained from middle meatus
- CT scan

Treatment

Includes immediate hospitalization, parenteral broad spectrum antibiotics(change the antibiotics once culture and sensitivity reports are available). Frequent assessment (6 to 12 hours) of visual acuity, visual field, ocular motility, degree of proptosis, fundus examination(optic disc and retinal vessels), temperature and blood count.

- * Nasal decongestants to facilitate sinus drainage
- * Incision and drainage of pointing abscess

Surgical options

- Definitive surgery is indicated if the patient fails to respond to I.V. antibiotics after 24 to 48 hrs or if there is reduction in visual acuity/ocular motility
- Surgical interventions in the form of
  - * External ethmoidectomy
  - * Incision and drainage of abscess cavity
  - * Trephination of frontal sinuses
  - * Antral lavage of maxillary sinus

Complications:

- Orbital apex syndrome
- Septic optic neuritis
- Cavernous sinus thrombosis,
- Meningitis
- Brain abscess..

Trachoma

Definition

Trachoma is defined as a specific communicable keratoconjunctivitis usually of chronic nature caused by Chlamydia trochomatis, primarily affecting the superficial epithelium , characterized by the formation of follicles and papillary hyperplasia of palpebral conjunctiva and pannus, the natural resolution of which is by cicatrisation involving potentially considerable visual disability.

Trachoma is considered one of the leading cause of preventable blindness world wide .It is also known as Egyptian Ophthalmia.The serotypes A ,B, Ba and C of Chlamydia trochomatis are the causative agents

Trachoma is seen in dry, dusty environment where poverty and poor hygiene predominates. Trachoma is highly contagious and spreads by direct contact. Approximately 360 million people in the world are suffering from trachoma of which 6-9 million are blind.

Clinical Aspects

Symptoms

- Foreign body sensation or grittiness
- Itching
- Watering, photophobia, redness
- Discharge is usually scanty, but maybe more due to secondary infections

Signs:

- Bulbar congestion
- Velvety papillary hypertrophy
- Follicles - Seen in the upper tarsal conjunctiva or limbus (Herbert's pits - pathognomonic) or bulbar conjunctiva.
- Pannus - Fine subepithelial neovascularisation arranged vertically with round cell infiltration seen in the upper limbus

Investigations

- Giemsa staining
- Cell cultures
- Direct fluorescent antibody test (DFA)- fluorescent labeled monoclonal antibodies to group specific or species specific antigen.
- ELISA or Enzyme Labelled ImmunoAssay (EIA)- Identification of Chlamydial antigen
- PCR
- Ligase Chain Reaction (LCR)

Treatment

The WHO has advocated the "SAFE" strategy for treatment of trachoma.		
S	-	Surgical treatment for trichiasis
A	-	Antibiotics
F	-	Facial cleanliness
E	-	Environmental improvement.

Medical Management

Topical

- 1% Tetracycline eye ointment or
- 1% Erythromycin eye ointment 4 times daily for 6 weeks, 20% or 40 % sulphacetamide
- 40% Sulphacetamide eye drops 3 times daily with 1% Tetracycline eye ointment HS for 6 weeks

Systemic

- Tetracycline or erythromycin 250 mg 4 times daily for 3-4 weeks
- Doxycycline 100 mg OD for 3-4 weeks
- Azithromycin 20mg/kg single dose
- Sulphonamides 3 weeks

Surgical Management

- Tarsectomy
- Excision of the fornix
- Surgery of trichiasis and entropion
- Pannus treated by cryoapplication / peritomy
- Concretions removed with hypodermic needle

Complications

Eyelids

- Trichiasis
- Madarosis
- Brow ptosis
- Eyelid retraction
- Secondary blepharospasm
- Cicatricial entropion
- Chalazion

Conjunctiva

- Loss of fornices
- Parenchymatous xerosis
- Symblepharon
- Pigmentation
- Pseudopterygium

Cornea

- Herbert's pits
- Healed pannus leading to hazy cornea
- Corneal opacities
- Trachomatous nodular keratopathy
- Descemetocele, Corneal perforation and Phthisis bulbi

Prevention

- Mass treatment / Blanket treatment: Given in places where there are > 5% prevalence of severe and moderate trachoma in children of >10 years.

Treatment

- 1% Tetracycline eye ointment BD for 5 consecutive days every month for 6 months.

Selective treatment

Given in areas with low to moderate prevalence of trachoma. Treatment given by case finding.

Information to patients:

- Importance of good enviromental sanitation
- Importance of ocular hygiene
- Seek treatment early to prevent complications, which may lead to blindness
- Spread of the infection is by contact and flies

Vitamin A deficiency

The major cause of Nutritional Blindness is vitamin A deficiency.

Symptoms

The characteristic ocular manifestations of Vitamin A deficiency range from night blindness to Xerophthalmia (corneal melting). Xerophthalmia is frequent between six months to three years of age.

WHO classification of Vitamin A deficiency

X 1 A	Conjunctival xerosis (dryness of the conjunctiva)
X 1 B	Bitot spots
X 2	Corneal xerosis (drying of cornea)
X 3 A	Corneal ulceration (<1/3 of corneal surface)
X 3 B	Corneal ulceration (>1/3 of corneal surface)
X N	Night Blindness
X S	Corneal scars
X F	Xerophthalmic fundus.

Daily requirements

- School children, adolescents, adults 2250 IU (750 micro grams)
- Children (0-4 yrs)- 1000 to 2000 IU (300 to 400 micro grams)
- Pregnancy and lactation 3000 to 3500 IU (750 micro grams + 300 micro grams)

Treatment schedule for Xerophthalmia (WHO recommendation)

- Immediately upon diagnosis 200000 IU Vitamin A orally
- Next day 200000 IU Vitamin A orally
- Within 1-4 weeks 200000 IU Vitamin A orally
- Children of six to eleven months old or less than 8 kg, half the above dose
- Children less than six months old 1/4th of the above dose

Vitamin A Prophylaxis

- Increasing the dietary intake of foods rich in Vitamin A like dark,green,leafy vegetables , spinach , carrot , etc.,
- Periodic administration of large doses of Vitamin A in the high risk group like children with severe PEM(Protein Energy Malnutrition) , Children with measles , diarrhea or other infections like malaria, chicken pox
- Fortification of food items with Vitamin A like bread, butter, milk, etc.,

Chemical Injuries Of The Eye

Introduction

Chemical injury to the eye is by no means an uncommon occurrence and constitutes a markedly heterogenous collection of accidents varying in severity from the complete and sudden loss of an eye to a trivial irritation. Most common agents causing chemical injury are ammonia, dye (alkali) and sulphuric acid (acids)

Clinical Features:

Symptoms

- Distressing pain
- Lacrimation
- Spasm of lids
- Loss of vision (variable)
- Absence of pain (if corneal nerves are damaged

Signs:

- Conjunctival Injection
- Corneal epitheliopathy
- Corneal edema
- Perilimbal ischaemia
- Anterior chamber activity
- Raised IOP
- Rarely necrotic retinopathy

Roper hall (ballen) staging of alkali burns:

GRADE - I	GRADE - II	GRADE - III	GRADE IV
Excellent	Good	Guarded	Poor
Corneal epithelial damage	Cornea hazy but iris details seen	Total loss of corneal epithelium stromal haze	Cornea opaque; obscuring view of iris or pupil
No ischaemia	Ischaemia <1/3rd of limbus	Ischaemia 1/3rd to 1/2 of limbus	Ischaemia >1/2 of limbus

Diagnosis:

Diagnosis of chemical injury is by typical history, clinical presentation and slit lamp findings.

Treatment: Immediate:

- Continuous copious irrigation with water or ionic solutions to bring the pH to near neutral.
- Removal of particulate matter
- Debridement of devitalized tissue

Medical Management:

- Admit in case of severe injury
- Topical steroids for first 7 days to relieve inflammation; if used for more than 10 days, it may cause ulceration and perforation.
- Topical antibiotics (0.5% chloramphenicol qid)
- Topical cycloplegics (1% cyclopentolate bd or tid)
- Topical lubricants (every four to six hourly)
- Systemic NSAIDs for pain relief
- Topical or oral carbonic anhydrase inhibitors +/- beta blockers for raised Intraocular presssure

Surgical Management:

- Tenonplasty
- Amniotic membrane grafting
- Corneal epithelial stem cell transplant [autologous (other eye) or allograft (live donors)
- Keratoplasty: complete penetrating keratoplasty including stem cells or lamellar keratoplasty

Complications

- Dry eyes
- Corneal ulcer
- Lid scarring
- Symblepheron
- Ankyloblepharon
- Phthisis bulbi

Referral

All cases of severe injury with significant limbal ischaemia (1/3rd), raised IOP, with central corneal haze obscuring iris details.

Information to Patients:

- Hazards of chemicals in day to day use
- Importance of using protective gear at work place
- First aid and importance of copious irrigation
- Seek immediate medical help
- Regular follow-up as advised by the ophthalmologist

Intraocular Foreign Body

Intraocular foreign bodies should always be treated as a surgical emergency. Such injuries should never be considered trivial. Foreign bodies are of 2 types:

- Inert: Gold, silver, platinum, glass, plastic, porcelain.
- Reactive: Iron, copper, wood, organic matter

Type of injury		
While hammering	-	iron
Car accident	-	glass
Blast injury	-	multiple intraocular foreign bodies
Gardening	-	organic matter
Sites of lodgement		
Anterior chamber	-	15%
Lens	-	8%
Posterior segment	-	70%
Orbit	-	7%

Clinical aspects:

The patient will come with a history of injury or something fell into the eye.

Symptoms

Pain

Redness

Photophobia

Watering

Discomfort.

Signs:

- Conjunctiva may show lodgment of iron, paddy, husk, insects etc.
- Common location is middle of subtarsal sulcus in the upper lid which may abrade the cornea.
- Also seen in cornea and bulbar conjunctiva.
- Blepharospasm(painful spasm of eyelids), discomfort, watering when the foreign body is in the cornea.
- Wound of entry may be present over the cornea, iris, lens.

Diagnosis:

5. X ray orbit
   * PA view and lateral view - To see radio opaque foreign body like iron.
   * Sweet technique: Limbal rings used to locate the foreign body.
6. Ultrasonogram - Both radio-opaque and radiolucent foreign bodies can be localized.
7. CT scan - Metallic and non-metallic foreign bodies can be precisely localized
8. MRI scan - Contraindicated for ferromagnetic objects

Rx Treatment

- Extraocular foreign bodies like those in conjunctiva can be removed with the help of a cotton swab under topical anaesthesia.
- Corneal foreign bodies can be removed with a cotton swab or 26 G needle under topical anaesthesia.
- Foreign bodies over the angle can be removed through limbus or through the wound of entry.
- Foreign bodies over the lens, the entire lens along with the foreign body is removed.
- Foreign body in the posterior segment small free floating foreign bodies can be removed with intraocular magnet endoscopically.
- Non-metallic foreign bodies are removed with intraocular forceps endoscopically.
- Regular follow up is a must to rule out infections..
- Foreign bodies should be removed in the acute phase within 24 hours.
- Iron and copper can cause severe reaction, if left alone.
- Associated injuries like traumatic cataract and retinal tear with retinal detachment will affect the final outcome.

Prevention:

- Protective goggles should be worn at the workplace.
- Wearing seat belts, helmets while traveling.
- As the visual prognosis is relatively poor, prevention is always better than cure.

Psychiatry

Standard Treatment Guidelines
Tamil Nadu Health Systems Project

Chapter 20

1. Basic Communication Skills
2. Management of Confused Patient – Delirium
3. Dementia
4. Management of Psychosis
5. Alcohol Dependence
6. Antipsychotic Medications
7. Temper Tantrums
8. Nocturnal Enuresis
9. Management of Suicidal Ideations and Attempted Suicide
10. Management of Unexplained Medical Symptoms

Basic communication skills

A good Doctor-patient relationship is the cornerstone of good medical practice. Good communication skills contribute to clinical competence, improves diagnostic ability, enhances patient compliance, increases patient satisfaction and reduces costs. While the lack of time is often mentioned as a reason for poor communication it is often due to poor training. This module briefly mentions these issues.

Common errors which inhibit communication

Doctors with poor communication skills employ "distancing tactics" that inhibit communication. These include:

- Dismissing patient's worries; immediate and automatic response to queries rather than finding nature and magnitude of out patient's concerns
- Providing false, inappropriate or premature reassurance without a patient hearing.
- Offering generalizations as soon as patient mention's his/her fears without hearing all the issues.
- Passing the buck and leaving the patient stranded.
- Selectively addressing the patient's physical cues and complaints while neglecting emotional signals and concerns.

Basic requirements for good communication

The basic requirements which facilitate good communication are listed below:

- Approach
  - The ability to establish a good relationship with patients is important for good doctors.
  - Communication techniques do not work unless the doctor is convinced of its efficacy.
  - Empathy, warmth and respect for and interest in the patient's welfare are the core of interpersonal skill.
  - The diverse skills to handle the different kinds of patients and the many situations require a professional approach to mastering these issues.
- Environment
  - Ensuring privacy encourages disclosure. Using curtains to create an illusion of privacy is useful. Providing comfortable seating, with the patient and the doctor at the same level, aids communication.
- Verbal techniques
  - The use of a brief and culturally appropriate greeting at the beginning of the interview is useful.
  - A personal query adds warmth. Asking open ended questions (e.g. How are you feeling?) allows the patient to present their problems and issues instead of closed questions (e.g. Is the pain better today?) which tend to bias the reply towards favorable responses.
  - Allowing time for the patient to answer and providing space often helps elicit patient's problems.
  - Picking up verbal cues from the patient and repeating the patient's "last three words" encourage the patient to elaborate his/her thoughts. The use of simple language and avoiding jargon is mandatory.
- Non-verbal issues
  - Leaning forward, maintaining eye contact, nodding appropriately and listening actively are powerful signals of interest which are a potent factor in improving the doctor-patient relationship.

Factors essential to success of counselling

- Factors common to most good counsellors include establishing a warm, confiding relationship with the patient.
- The provision of psychological support to handle the illness and its consequences is necessary. The arousal of hope and the expectation that the distress will reduce are useful.
- Good counsellors need to also re-organize the patient's perspectives about their illness providing them with information, clarifying their doubts and removing misconceptions about their condition. Providing an orderly account of the issues is mandatory.
- Many patients with stress-related problems often shop for miracles from doctors. The responsibility for improvement, when the distress is stress related, should be gently yet firmly transferred

from the doctor to the patient.

- The patient should be encouraged to use a variety of stress reduction strategies including exercise, leisure activities, yoga, meditation and religion which should be incorporated into their daily routine.
- Patients and relatives should be asked to consider stress in their homes and at work which can contribute to the symptoms and to distress. They should be advised to discuss the different solutions possible.
- Doctors should act only as facilitators and should allow patients to come up with their own solutions for stress.

Conclusion

Good communication and counselling skills make a good physician.

The Management of The Confused Patient Delirium

A common psychiatric presentation among hospitalized patients in a general medical and surgical setting is the patient with delirium.

Delirium or an acute confusional state is a transient global disorder of cognition. It is more common among the hospitalized, the frail and elderly, postoperative patients, those admitted to intensive care units and those with compromised mental state (e.g. dementia). The condition is a medical emergency associated with increased morbidity and mortality rates. The steps include:

- Establish the clinical presentation of delirium.
- Monitor vital signs and provide supportive management.
- Identify the cause of delirium and treat it
- Consider the differential diagnoses
- Provide medication for control of symptoms
- Educate and support the family
- Review the patient regularly

Step I: Establish the clinical presentation of delirium

- Delirium manifests clinically with a wide range of neuropsychiatric abnormalities. The clinical hallmarks are
  - * Decreased attention span and a waxing and waning type of confusion.
- The major symptoms include
  - * Clouding and fluctuation of consciousness,
  - * Difficulties in attention and disorientation to time, place and person
  - * Visual and auditory hallucinations and paranoid delusions may also be present
  - * The patient may have associated neurological symptoms like dysarthria, dysphasia, tremor, asterixis etc
- The disturbance is characterized by an acute onset of symptoms, over hours or days and may fluctuate in severity from hour to hour with worsening particularly at night. A detailed history and clinical examination is necessary to establish the clinical presentation.

Step II: Monitor vital signs and provide supportive management

- Vital functions (e.g. heart rate, blood pressure, respiration, temperature, intake and output, etc) should be regularly monitored.
- Fluid and nutrition should be managed.
- The patient's medications should be carefully reviewed; nonessential medications should be discontinued, and doses of needed medications should be kept as low as possible.
- Severely delirious patients who are agitated and wandering, benefit from constant observation and reassurance which may help avoid the use of physical restraints
- Reorientation techniques such as the use of calendars, clocks and family photos may be helpful
- The environment should be stable, quiet and well lit
- Support from a familiar nurse and family should be encouraged
- Sensory deficits should be corrected with eyeglasses and hearing aids.

Step III: Identify and treat the cause of delirium.

- The common laboratory tests which may need to be done to establish causation include

* Complete blood cell counts
* Electrolytes
* Blood glucose
* Renal and liver function tests and
* Urine analysis
* Neuroimaging
* EEG
* ECG and pulse oxymetry are also useful

- Common reversible causes of delirium include: hypoxia, hypoglycemia, hyperthermia, anticholinergic medication, alcohol or sedative withdrawal.
- Others include: infections, metabolic abnormalities, structural lesions of the brain, postoperative states, sensory or sleep deprivation, fecal impaction, urinary retention, and change of environment
- Often, particularly in the critically ill and in elderly hospitalized patients, delirium may have multiple etiologies
- Occasionally, no clear etiology is immediately apparent.

Step IV: Consider other differential diagnoses

- Delirium must be distinguished from a psychotic disorder, which is not generally associated with confusion or a change in the level of consciousness.
- The acuteness of onset, fluctuating severity and disturbance of consciousness of delirium is helpful in distinguishing it from dementia where the patient is generally alert, the onset is usually gradual and does not fluctuate.

Step V: Provide medication for control of symptoms .

- Antipsychotics are the medication of choice in delirium. Older neuroleptics such as Haloperidol are useful but have many adverse neurological effects.
- Newer neuroleptics such as risperidone, olanzapine, and quetiapine relieve symptoms while minimizing adverse effects
- Smaller dose than used in functional psychosis are required Examples:
  * 0.25-1 mg/day of haloperidol
  * 0.5-1 mg/day of resperidone
  * 2.5-5 mg/day of olanzapine
  * 25-100 mg/day of quetiapine.
  * Benzodiazepines are reserved for delirium resulting from seizures (e.g. 1-3 mg lorazepam/day), withdrawal from alcohol /sedative hypnotics (e.g. 2-8 mg lorazepam/day), or when unknown substances may have been ingested.
- They must be used cautiously as they can cause respiratory depression, especially in patients who are elderly or debilitated patients. Attempt to taper the medication once symptoms are in control.

Step VI: Educate and support the family

- Educate the family regarding the etiology and course of the problem
- Providing reassurance that delirium often is temporary and is the result of a medical condition may be beneficial to both patients and their families
- Suggest that family members or friends visit the patient, usually one at a time, and provide a calm and structured environment.

Step VII: Regular review

Regular and daily review of the clinical status is required. Psychotropic medication can be gradually tapered after the mental status improves.

Referral

Many patients with delirium settle with the treatment

Medication for use in psychosis and their side-effects, indications, dosage in primary care and duration of treatment

Medication	Indications	Starting dose/ day	Adult dose/ day	Common side-effects	Duration of treat-ment
Non-sedative oral medication					
Haloperidol	Psychosis without marked agitation	1.5- 3 mg	5-10 mg	Tremor, rigidity, bradykinesia, dystonia, akathisia, tardive dyskinesia	At least 2 years after a first episode of psychosis. Needs to be continued for many years if there is a recurrence or a relapse of symptoms on withdrawal of drug.
Risperidone		1-2 mg	4-6 mg		
Sedative oral medication					
Chlorpromazine	Psychosis with marked agitation and sleep disturbance	100-200 mg	600 mg	Tremor, rigidity, bradykinesia, dystonia, akathisia, tardive dyskinesia	
Olanzapine		5 mg	10-15 mg	Sedation, weight gain, elevation of blood sugars and lipids, akathisia	
Intramuscular parentral medication					
Haloperidol	Psychosis with marked agitation	5 mg	10 mg repeated every hour till patient settles (maximum 30 mg /day)	Sedation, tremor, rigidity, bradykinesia, dystonia, akathisia	
Olanzapine		10 mg	10 mg repeated every hour till patient settles (maximum 20 mg /day)	Sedation, akathisia	
Depot intramuscular parentral medication					
Fluphenazine decanoate	Psychosis with poor medication compliance	25 mg every month	50 mg every month	Tremor, rigidity, bradykinesia, dystonia, akathisia, tardive dyskinesia	
Other medication					
Lorazepam (oral)	Extreme agitation, restlessness	1-2 mg	4-6 mg	Sedation, falls in elderly	Gradually taper within 1-2 weeks
Trihexyphenidyl	Tremor, rigidity, bradykinesia, dystonia, akathisia	2 mg	4-6 mg in divided doses	Dry mouth, constipation, urinary retention	Attempt withdrawal after 3-4 months without symptoms
Promethazine (injection)	For acute dystonia; or along with Haloperidol for extreme disturbance	25 mg	25-50 mg	Sedation	Single dose, intramuscular

Common antidepressant medication, their side-effects, indications, dosage in primary care and duration of treatment

Medication	Indications	Starting dose/ day	Adult dose/ day	Common side-effects	Duration of treatment
Non-sedative medication					
Fluoxetine	Unexplained somatic symptoms, depression, panic, phobia, obsessive compulsive disorder without sleep disturbance	20 mg morning	20 mg for depression and persistent pain; 60 mg for obsessive compulsive disorder	Nausea, vomiting, diarrhoea, restlessness	3- 6 months followed by a tapering schedule. Can be continued for many years if relapse of symptoms on withdrawal
Sertraline		25 mg morning	50-100 mg		
Sedative medication					
Imipramine, Amitriptyline, Dothiapin	Unexplained somatic symptoms, depression, anxiety, panic, phobia associated with disturbed sleep	25 mg bedtime	50-100 mg	Sedation, dry mouth, constipation, giddiness	
Mirtazapine		7.5 mg bedtime	15 mg	Sedation, giddiness	

Common sedatives for short term use

Medication	Indications	Starting dose/ day	Adult dose/ day	Common side-effects	Duration of treatment
Diazepam	Sleep disturbance, anxiety, agitation	2.5 mg	5-10 mg for sleep; 5-20 mg in divided doses for agitation	Sedation, falls in elderly, **potential for addiction**	Less than one month
Lorazepam		0.5 mg	1-2 mg for sleep; 4-6 mg in divided doses for agitation		

of the underlying cause, antipsychotic medication and supportive measures. However, psychiatric consultation may be indicated for management of behavioral problems such as severe agitation or aggressive behavior.

Diagnosing And Managing Dementia

A significant proportion of the elderly population, especially those attending hospital facilities, report subjective memory loss. The condition is a cause of severe disability and burden.

The steps include:

1. Recognize the symptoms of dementia
2. Perform a physical examination and laboratory investigations to exclude treatable causes
3. Manage risk factors
4. Educate the patient and family
5. Suggest life style modifications
6. Discuss psychosocial interventions
7. Prescribe medication for dementia
8. Manage complications
9. Support care givers
10. Review patient regularly

Step I: Recognizing the symptoms of dementia

Diagnosing dementia involves the elicitation of possible dysfunction in multiple cognitive domains including problems with memory, aphasia, agnosia, apraxia and the loss of executive function. A simple categorization of such complaints includes:

- Age related cognitive decline
  - * Impairment in memory without social and occupational impairment
- Mild cognitive impairment
  - * Impairment of memory with consequent problems in social and occupational functioning
- Dementia
  - * Progressive loss of memory; associated with social and occupational impairment; and one of the following dysfunctions:
    - » Aphasia- difficulty in understanding speech, inability to express ideas
    - » Apraxia- difficulty in dressing despite intact motor function
    - » Agnosia- difficulty in recognizing common objects despite intact sensory functions
    - » Loss of executive function- inability to plan and execute activities

Dementia should be differentiated from normal (age related cognitive decline) and the mild cognitive impairment by the presence of impairments in multiple domains of cognitive functions.

Step II: Physical examination and laboratory investigations to exclude treatable causes

Dementia is a syndrome caused by diverse diseases of the brain including: cerebrovascular disease, tumors, chronic infections (HIV, syphilis), degenerative diseases (Alzheimer's, Parkinson's, Huntington's), subdural haematoma, normal pressure hydrocephalus. The risk for dementia increases with the following systemic diseases: hypertension, diabetes, hypothyroidism, vitamin deficiencies (B12, folic acid and niacin) hypercholestrolemia.

A physical examination and appropriate laboratory tests (e.g. complete blood counts, creatinine, sugars, lipids, TSH, CT scan) will be necessary to exclude treatable causes of dementia.

Step III: Managing risk factors

High blood pressure, uncontrolled diabetes mellitus, and hyperlipidemia need to be managed with appropriate medication. Possible nutritional deficiencies will need vitamin supplementation.

Step IV: Educating the patient and family

The disability and burden of dementia is huge. Educating the patient and especially the family and caregivers is mandatory. Discussing the nature of the condition, the need to manage risk factors, the role of medication and psychosocial treatments is necessary. The fact that life style modification and psychosocial interventions is the mainstay of treatment needs to be emphasized.

Step V: Life style modifications

- A simple and regular daily routine is essential. The fact that changes in the environment and in the daily routine increase the confusion and the consequent difficulty needs to be highlighted.
- Regular exercise (walking) is helpful.
- Simple recreation within the daily schedule is helpful.

- Activities which are unfamiliar (meeting new people, new situations) should be minimized.
- The house may need to be modified to help elderly people cope with failing memory.

Step VI: Psychosocial interventions

- A simple and structured daily routine for meals, bath, exercise and bedtime needs to be in place.
- Labelling of switches and containers is useful
- The use of memory aids like calendars, clocks, dairies for appointments, lists for reminders help.
- Simple instructions before activities, introduction of new and unfamiliar people and situations is mandatory
- Simplifying clothes and accessories, using Velcro and elastic, goes a long way in keeping people with failing abilities, independent
- Daily and regular attempts to orient people with dementia are necessary.

Steps VII: Medication for dementia

- The currently available medication helps delay the dementing process. Acteyl choline esterase inhibitors are indicated.
- Donepezil-starting dose 5 mg at bedtime; increase to 10 mg after one month and
- Rivastigmine-starting dose 1.5 mg twice a day; gradually increased to 6 mg per day.

Step VIII: Managing complications

- Delirium (acute episodes of disorientation and confusion) may occur.
- Medical causes like urinary tract infections, pneumonia need to be diagnosed and managed.
- Depression (crying episodes, pervasive sadness) may require antidepressant medication.
- Hearing voices, persecutory beliefs, suspicious ideas, agitation, aggression and wandering need to be managed with small doses of antipsychotic medication (e.g. Haloperidol 0.25 tds).

Step IX: Supporting care givers

The burden on caregivers and on family members is considerable and can result in depression. Psychological support, counselling and occasionally antidepressant medication may be required.

Step X: Regular review

A regular review of the patient's condition is mandatory for fine tuning medication and for providing support and advice to relatives.

Referral

Patients who are difficult to manage and those with treatable conditions not responding to therapy require specialist attention.

The Management Of Psychosis

Psychotic syndromes are seen infrequently in general practice settings. However, general practitioners play an important role in the detection of early symptoms, providing care in a low-stigma environment of general medical settings and in supporting relatives of patients with psychotic illness. The steps in management include:

1. Acknowledge the distress
2. Identify the syndrome of psychosis
3. Perform a focused physical examination and laboratory investigations
4. Elicit the patient's beliefs about problems
5. Elicit the relative's perspective on the patient's symptoms.
6. Provide essential information about the illness to patient and relatives
7. Prescribe medication
8. Discuss the psychosocial aspects of the patient's care
9. Provide support to the caregivers
10. Give a specific appointment for review

Step I: Acknowledge distress

- Patients may be distressed because of delusions (false beliefs which are not shared by patient's culture) and hallucinations (hearing non existent voices or seeing visions) which may be frightening
- Relatives are often scared and puzzled by their relative's odd or grossly abnormal behaviour
- Acknowledging their distress reflects an empathetic attitude and contributes to establishing an effective rapport and a therapeutic alliance which are essential in facilitating improvement.

Step II: Identify the psychotic syndrome

- The symptoms of psychosis include
  - Delusions
  - Hallucinations and
  - Grossly abnormal behaviour
- Psychosis can be diagnosed if any one or more of these symptoms are present
- Based on the duration of symptoms it can be classified as
  - Acute: Less than 1-2 months or
  - Chronic: If the duration is longer.

Step III: Focused history, physical examination and laboratory investigations

- A focused history, physical examination (especially neurological) and investigations are cardinal to identify psychotic syndromes that are secondary to physical disease or substance abuse.
- A clinical presentation of disorientation, poor attention, impaired concentration or long-term impairment of memory would suggest medical causes.
- A history of common physical disorders that can manifest with psychotic symptoms such as head injury, seizures, infections, space-occupying lesions of the central nervous system and thyroid disorders need to be elicited.
- Recommended investigations include a full blood examination, erythrocyte sedimentation rate, serum creatinine and blood sugars.
- Additional tests that may need to be done include electro-encephalogram (EEG) and cranial computed tomography (CT).

Step IV: Elicit patient's beliefs on current problems

Asking patients what they think or fear is necessary- to make a diagnosis, as well as in addressing issues of safety. (E.g. `My wife is plotting to kill me; I heard a message over the radio. I'll either kill her or myself before that').

Step V: Elicit relative's perspective on symptoms

It is important to understand what the patient's family think is wrong with the patient and why this has happened to them. Their beliefs need to be discussed and understood in their cultural context prior to presenting biomedical explanations and providing reassurance.

Step VI: Provide essential information to the patient and the family

- Inform relatives that agitation and strange behaviour are symptoms of a mental disorder
- Symptoms may come and go over time; medication can help by reducing current difficulties as well as in preventing relapse
- Providing information about a multifactorial etiology (brain pathology, genetic and environmental factors) often helps to reduce distress, guilt and blame in the relatives
- Reassure the relatives that recovery often takes place in small steps

Step VII: Prescribe medication

- Antipsychotic medication will reduce psychotic symptoms
  - Drugs such as Chlorpromazine (100- 600mg/ day) and Olanzapine (5 - 15 mg/day) are preferable for individuals who are agitated or have problems sleeping
  - Haloperidol (5-15 mg/ day) and Risperidone (2-6 mg/day) are less sedative agents
  - Common side effects of antipsychotics include extra-pyramidal symptoms (tremor, rigidity), dystonia (abnormal posture of neck, tongue), sexual dysfunction, weight gain and elevation of blood sugars and lipids.
- For extremely disturbed patients intramuscular haloperidol (5 or 10 mg IM) may be given; this can be combined with injection promethazine (25 to 50 mg IM) for greater sedation
- De-pot antipsychotics (e.g. fluphenazine decanoate 25 mg IM once in 2-4 weeks) can be given to patients who are poorly compliant with oral medication.
- General principles of drug treatment include use of the lowest possible dosage for the relief of symptoms, gradual increase in doses in the elderly and close monitoring for side effects
- Smaller doses are generally required for psychosis that is secondary to medical conditions. The dosage schedule and possible side-effects must be discussed with the patient and family.
- In general, after a single episode of psychosis,

medication must be continued for at least 18 to 24 months

- In case of a relapse or in chronic psychosis, the medication must be continued for longer periods. In addition to antipsychotic medication, short-term benzodiazepines (lorazepam 2-4 mg /day) may be required to reduce agitation or assist with sleep.
- Anticholinergic drugs (trihexyphenidyl 2-6 mg/ day) may be required to prevent/ reduce antipsychotic-induced extra pyramidal side effects
- Parentral Promethazine (25-50mg IM stat) should be given to relieve acute dystonia.

Step VIII: Discuss psychosocial management

- The family should supervise the patient and should avoid confrontation unless it is necessary to prevent harmful or disruptive behaviour.
- The patient should be encouraged to maintain his or her daily routine of activities and re-enter work when possible.
- Offering rewards and praise, even if tasks are not perfectly done will help to maintain and increase such behaviour.
- Offer patients health promotion and prevention measures such as smoking cessation, weight control, screening for diabetes and sexual health.

Step IX: Provide support to the care-givers

Engage the caregivers collaboratively in the treatment process. Family members need to be supported as they often have to cope unaided with the unpredictable and socially awkward behaviour or persisting under-activity of their loved ones as well as concerns about the fate and long term care of adult children. In addition to providing support themselves, general practitioners may encourage families to benefit from the services provided by available support organisations.

Step X: Give a specific appointment for review

- Regular review of progress is necessary for all patients, with scheduled appointments every few days for acute psychosis and two to four weeks for follow-up
- Monthly or less frequent reviews for people with psychosis who are stabilized will be needed
- Periodic check-ups also help to monitor compliance with medication, observe for and manage side effects and support and guide relatives.

Referral

Referral to specialists should be considered as an emergency when the patient is at risk of harm to himself or others due to suicide, violence or neglect. It may also be required if there is a relapse, refusal to take medication, lack of response to prescribed medication, problematic side-effects or concerns about co-morbid drug or alcohol use.

The Management Of Alcohol Dependence

Primary care settings offer an important opportunity to identify and treat people with problems related to alcohol. Brief interventions and counselling delivered by general practitioners can be successful for many patients. The steps in management include:

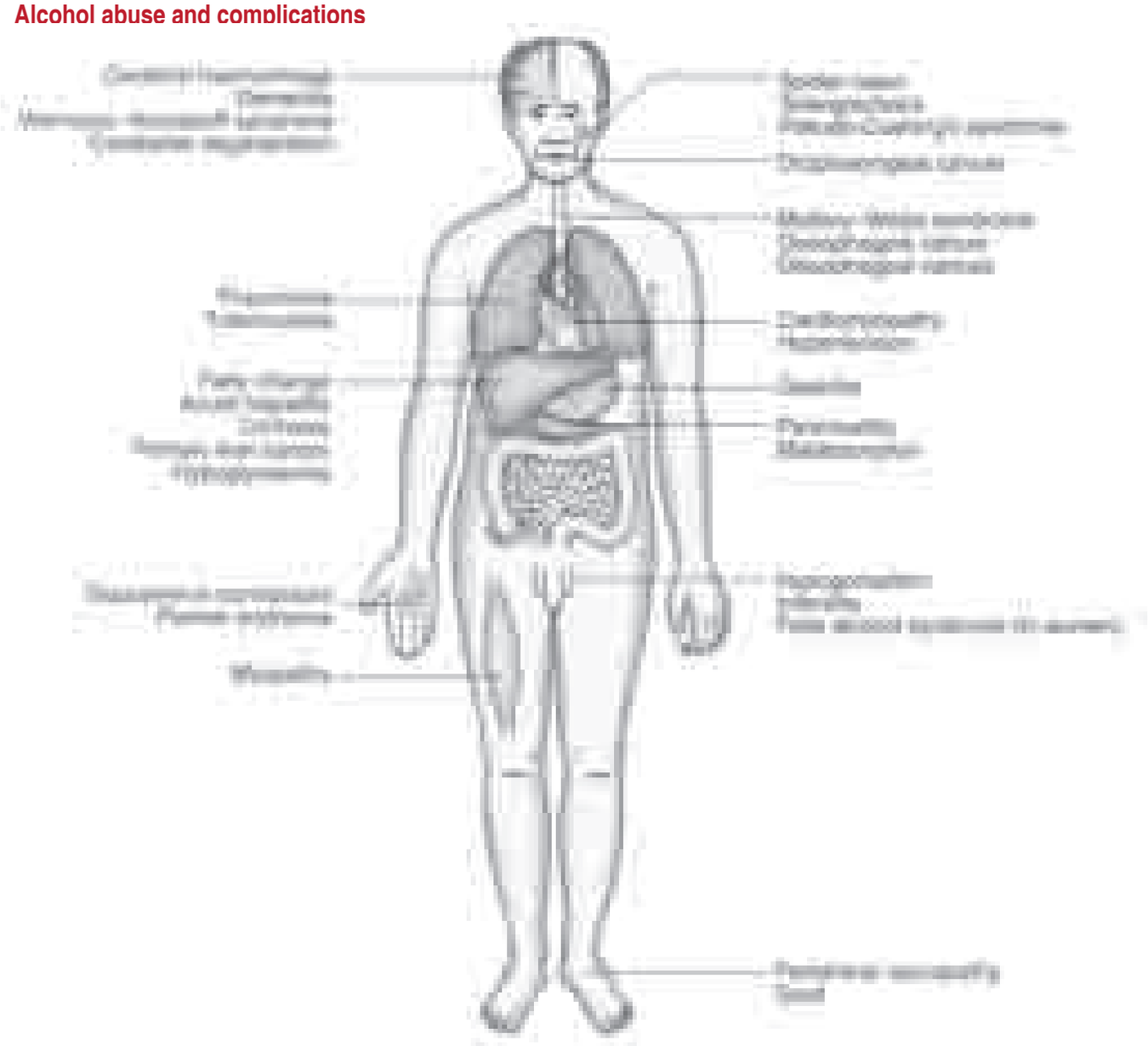
1. Assess physical and mental state
2. Perform a focused physical examination and laboratory investigations
3. Consider general medical management
4. Detoxify
5. Educate patient and relatives
6. Identify cues
7. Provide counselling
8. Support abstinence
9. Transfer responsibility
10. Appointment for review

Step I: Assessment

The duration, amount and pattern of drinking needs to be examined. Harmful use is defined as drinking that result in physical or mental damage.

- Dependence states are characterized by a
  - Strong desire to consume alcohol
  - Difficulty in controlling it's use
  - Withdrawal features such as insomnia, anxiety, tremors and sweating when alcohol use is ceased
  - Tolerance, or drinking large amounts of alcohol without appearing intoxicated
  - Continued use of alcohol despite harmful consequences and neglect of other activities due to alcohol

Alcohol abuse and complications



* Social and occupational impairment.

A simple instrument used to assess alcohol dependence (problem drinking indicated by 2 or more affirmatives) is known by the mnemonic CAGE.

CAGE Questionnaire
• Have you ever thought of **Cutting down** on your drinking?
• Have you ever been **Annoyed** by people telling you to cut down on your drinking?
• Have you ever felt **Guilty** about your drinking?
• Do you need a drink in the morning to get you going (an **Eye-opener**)?

Step II: Focused history, physical and mental state examination and laboratory investigations

- A focused history and physical examination is necessary to identify alcohol related problems, including liver disease, vitamin deficiencies, injuries and peripheral neuropathies.
- A mental state assessment of the level of consciousness, orientation to time, place and person, attention, concentration and the presence or absence of psychotic, anxiety or depressive symptoms is mandatory.
- Medical investigations should be ordered including: electrolytes, full blood examination, liver function tests, serum creatinine and blood sugars.

Step III: General medical management

- Patients need to be assessed and treated for vitamin deficiencies, dehydration, injuries and infections.
- Vitamin supplements for those whose dietary intake of vitamins has been poor is mandatory
- Parentral Thiamine should be given if low reserves are suspected.
- Tetanus toxoid should be given to those with injuries.

Step IV: De-toxification

- Patients with a withdrawal syndrome may require benzodiazepines such as chlordiazepoxide or lorazepam.
- People who drink only in the evenings can receive small bedtime doses of 10-30 mg of Chlordiazepoxide (5-15 mg of Diazepam or 1-3 mg of Lorazepam).
- Higher and divided doses (of 40-80 mg per day of Chlordiazepoxide or 4-8 mg of Lorazepam) will be necessary for those who have been drinking in the morning or in those with withdrawal symptoms which occur in the daytime.
- The dose can be gradually tapered and stopped over 7-15 days after withdrawal symptoms have subsided.

Step V: Education and feedback

- Educate the patient about the various harmful effects of alcohol including the physical, psychological and social issues.
- Provide feedback by linking the alcohol use to existing medical problems, (such as hypertension or gastritis), abnormal blood tests (LFT) or psychosocial problems (marital, legal or work related).
- Discuss the biochemical basis of developing tolerance and withdrawal symptoms and discuss habit formation over time.

Step VI: Identification of cues

- Helping the patient to identify high-risk situations or cues in which drinking is likely to occur, such as family celebrations, spending time in the company of friends who consume alcohol or stressful situations at work, is necessary.
- Discuss with the patient techniques and healthy alternatives for managing such high-risk situations.
- Regular and frequent meals, the control of money, staying away from bars and liquor shops and developing healthy leisure activities are useful techniques.

Step VII: Counseling for specific problems

- Patients with specific problems such as marital discord, work related issues or financial problems would need help with problem solving.
- Asking patients to look for specific solutions to their difficulties and giving them time to examine the issues is obligatory.

Step VIII: Supporting abstinence

- Anxiety and depression often co-occur with alcohol misuse.
- The patient may have been using alcohol to self-medicate in order to reduce these symptoms. If these symptoms such as sleep disturbance and depression persist for more than two weeks following detoxification, antidepressants and counselling should be considered.

Step IX: Transfer responsibility

- Emphasizing that change in the habit is the patient's responsibility and that only he himself can make a decision to change and make life better is mandatory.
- Since alcohol use is often associated with a certain lifestyles, giving up the habit requires a change in philosophy, attitude and that particular way of life.

Step X: Follow-up and reinforcement.

- To ensure the long-term effectiveness of the brief intervention described above, ensure regular follow-up visits with the patient and re-inforce the abstinent behavior.
- Daily supervision and dispensing of drugs is essential in the first few days
- Frequent follow-ups are advisable thereafter to adjust dose of medication, assess whether the patient has returned to drinking
- Check for serious withdrawal symptoms and maintain support.

Referral

- Hospital de-toxification is recommended in patients at risk of a complicated withdrawal syndrome (with a history of seizures or delirium tremens), multiple drug use or dependence and those with severe co-morbid medical or psychiatric disorder.

A note on the management of Delirium tremens

- Delirium following the withdrawal of alcohol is called Delirium tremens. This is a Medical emergency which usually occurs within the first 48-72 hours after alcohol withdrawal. Symptoms include
  - Clouding of consciousness and confusion
  - Vivid hallucinations and illusions
  - Tremors
  - Insomnia
  - Agitation
  - Fearfulness and
  - suspiciousnes
- Hospitalization is recommended with frequent monitoring of the pulse rate, blood pressure, respiration and level of consciousness.
- Treat infections and ensure adequate hydration and nutrition.
- Intramuscular Thiamine 100mg must be administered once a day for a few days
- Oral supplements of Vitamin B complex and Thiamine 100 mg daily must also be given.
- Keep the patient in an evenly lit, quiet room, where dark shadows, bright lights, loud noises and other excessive stimuli are avoided.
- Patients with a history of withdrawal seizures and Delirium tremens require doses upto 100-120 mg equivalent of Chlordiazepoxide (i.e. 50-60 mg of Diazepam or 10-12 mg of Lorazepam).
- Closely supervise the medication and adjust it based on the severity of the patient's withdrawal symptoms and response.
- For patients in withdrawal delirium, antipsychotic drugs in low doses can be used along with the benzo-diazepines to reduce agitation.

A note on managing other substance abuse and dependence

People who abuse other substances and are dependent on them can also be managed with this protocol.

Managing temper tantrums in children

Temper tantrums describe a range of behaviours in children which are difficult to manage. They can involve shouting, screaming, hitting, kicking, rolling on the floor and even breath holding. Parents can seek help from physicians as children are difficult to manage during these episodes. Temper tantrums are equally common in boys and girls and usually occur between the ages of 1 and 5 years. While occasional tantrums are normal part of childhood, regular tantrums suggest that the child is frustrated by his inability to get his own way, have his or her desire fulfilled or are unable to communicate effectively.

- Assess the tantrum
- Exclude mental retardation, autism and hyperactivity
- Maintain record of baseline tantrum
- Suggest preventive measures
- Discuss positive reinforcement
- Provide advice on managing tantrums
- Give appointment for review

Step 1: Elicit details

- The nature and frequency of the tantrum has to be elicited from parents.
- The triggers which start the process, the factors which terminate it and the consequences of such behavior need to be identified.
- Identify common situations, patterns of triggers and consequences of such behavior.

Step II. Exclude mental retardation and autism

- Developmental delays in children suggest mental retardation and disorders like autism
- Problems in vision and hearing and chronic physical illness can also contribute to tantrums
- Inability to concentrate on tasks and marked restlessness may be suggestive of hyperactivity.
- While these conditions can also be treated with this protocol, they may also require specialist help to manage underlying conditions.

Step III. Maintain record of baseline tantrum

- The maintenance of a detailed record of baseline temper tantrums is helpful in assessing progress.
- The parents or relatives should be encouraged to maintain a dairy of events and their relationship to the tantrums.

Step IV. Simple common sense measures for prevention

- The following strategies usually help avoid tantrums
  - * Make sure that the child is getting enough attention to prevent the child seeking attention through the tantrum
  - * Give the child choice over little decisions so that he/she feels a sense of control (e.g. Do you want apple juice or mango juice? Rather than no choice at all.)
  - * Keep objects which the child should not play with, out of sight
  - * Distract your child when you see that child is going to start a tantrum
  - * Provide age appropriate toys and games so that the child achieves a sense of mastery
  - * Know when your child is tired, hungry, sleepy and take appropriate action
  - * Parents should choose your battles carefully and be consistent with discipline

Step V. Positive reinforcement

- The parents should be encouraged to maintain a star chart or calendar.
- Days without tantrums should be marked with red stars while those with tantrums without. Lavish praise for good behavior and providing stars is mandatory.
- A material reward (e.g. chocolates, ice creams, books, etc) for a good behavior of 3-5 days will go a long way in reinforcing such behavior. Punishment and shaming usually make the problems worse.

Step VI. Advice on managing tantrums

- Parents and relatives should be reassured that temper tantrums are common in children and can be managed with simple measures. The following are useful tips
  - * Keep calm. Getting upset, frustrated or using physical punishment only complicates matters
  - * Try to understand the situation, the cause of the tantrum
  - * Calm the child and help him/her face the disappointment, provide comfort
  - * Ignoring some tantrums helps but always be around so that the child is safe
  - * Gentle reasoning, giving viable options may also help
  - * Take children to a calm place to cool down
  - * After a tantrum offer help to calm down, offer praise for calming down
  - * Don't reward tantrums.

Step VII. Appointment for review

The appointment for review is to monitor progress. The child should be praised for even small progress and should be encouraged to take control of emotions. The measures to prevent tantrums and the positive reinforcement strategies should be reviewed.

Referral

Children, who are in danger of hurting themselves, are destructive and in whom the above strategies do not work may require specialist help.

The diagnosis and management of Nocturnal enuresis

Nocturnal enuresis is defined as the involuntary passage of urine during sleep in the absence of any identified physical abnormality in children above 5 years of age. It is commoner among boys and its prevalence reduces exponentially with age. Children with such problems present to general physicians and can be managed in primary care. The steps in management include:

- Elicit clinical details.
- Perform physical examination and relevant laboratory investigations
- Maintain record of baseline bedwetting
- Suggest simple common sense measures
- Recommend positive reinforcement
- Discuss reduction of stress and resolution of conflicts
- Consider placebos
- Prescribe tricyclic antidepressants
- Discuss responsibility with child and with parents
- Give an appointment for review

Step 1. Elicit clinical details

- The nature and frequency of the enuresis in relation to age has to be elicited from parents.
- Specifically whether there were periods when partial or complete bladder control was achieved and the circumstances of the loss of control.
- Details of sleep arrangements and parental attitudes to the enuresis should be elicited
- Stress, including marital discord, single parent situations, academic stress, issues related to inconsistent and very strict discipline, physical and sexual abuse and sibling rivalry needs to be explored
- Primary enuresis (with complete absence of bladder control without any significant period) should be differentiated from secondary enuresis (where control was achieved and the child dry for at least 6 months
- Similarly, enuresis during sleep should be distinguished from incontinence during periods when the child is awake.

Step II. Physical examination and laboratory investigations

- Urological conditions
- Examples: urological abnormalities and obstruction like
  - * Urethral valves
  - * Meatal stenosis
  - * Bladder neck obstruction and
  - * Recurrent urinary infections
- Neurological problems
- Examples:
  - * Spina bifida
  - * Spinal tumors
  - * Epilepsy and
  - * Mental retardation
- These are common causes for the inability to achieve any bladder control in childhood have to be excluded.
- The possibility of a urinary tract infection can be ruled out with a simple test for urine microscopy.
- The identification of specific causes for the lack of bladder control mandates specific treatment for the identified cause.
- Primary and daytime incontinence after the age of 5 years suggests the possibility of a physical disease while nocturnal and secondary enuresis suggests psychological causation implying environmental stress.

Step III. Maintain record of baseline bed wetting

The maintenance of a detailed record of baseline nocturnal enuresis is helpful in assessing progress. The parents or relatives should be encouraged to maintain a dairy of events and their relationship to nocturnal enuresis.

Step IV. Simple common sense measures

- The parents should be advised to restrict fluid intake by the child in the evening and before bedtime
- Insisting on voiding before sleep is mandatory. Similarly, sleep interruption and voiding is helpful.
- The child should be completely awake before he passes urine so that he is aware of the process.

Step V. Positive reinforcement

- The parents should be encouraged to maintain a star chart or calendar
- Dry nights should be marked with red stars while those with the incontinence without. Lavish praise for dry nights and obtaining stars is mandatory.
- A material reward (e.g. chocolates, ice creams, books, etc) for dry periods of 3-5 days will go a

long way in reinforcing such behavior

- Punishment and shaming usually make the problems worse.

Step VI. Reduction of stress and resolution of conflicts

- Psychological stress is commonly associated with nocturnal enuresis
- Exploring common stress at home and at school is a vital part of management
- The identification of stress, including marital discord, single parent situations, academic stress, issues related to inconsistent and very strict discipline, physical and sexual abuse and sibling rivalry, etc, and their resolution is mandatory.
- Parents should be advised on problem solving and the need for reduction of conflicts at home and at school in order for improvement in the child's nocturnal enuresis
- Often enuresis is the child's attempt at resolving stress which he/ she cannot handle
- Discussing the connection between nocturnal enuresis and stress and encouraging the parents to address these issues is cardinal to success.

Step VII. Using placebos

Using placebos (e.g. Vitamin tablets and syrups) along with the common sense measures, the positive reinforcement and the reduction of stress and the resolution of conflict goes a long way in reducing the problem. However, placebos are short term solutions and the resolution of conflicts and stress is required for long term success.

Step VIII. Tricyclic antidepressants

- Tricyclic antidepressants (e.g. Imipramine, Amitriptyline) are useful for children with long standing nocturnal enuresis
- Small doses of 12.5 -25 mg at bedtime for a period of 3 to 6 months help many children
- This should be used in combination with other measures.

Step IX. Discussing responsibility

Encouraging the child to take responsibility for his / her bladder control is necessary. Discussing the responsibility of parents and guardians in exploring, identifying and reducing stress and resolving conflicts through healthy strategies is mandatory for success. Reiterating the connection between nocturnal enuresis and stress is necessary. Parents should be encouraged to solve these problems and resolve conflicts.

Step X. Appointment for review

The appointment for review is to monitor progress. The child should be praised for even small progress and should be encouraged to take control of his/her bladder. The measures to prevent enuresis and the positive reinforcement strategies should be reviewed. The reduction of stress and the resolution of conflicts should be discussed with parents. Medication can be prescribed if necessary.

Referral

Primary enuresis may require specialist assessment and intervention. Secondary nocturnal enuresis not responding to treatment mentioned should be referred to a specialist.

The Management Of Suicidal Ideation And Attempted Suicide

Physicians are often called upon to assess people who have attempted suicide and those who admit to such ideation. The assessment is a complex task as suicide is a result of interaction between personality, mental illness, current mood, stress, social support, religious views and responsibility. However, this simple checklist will help in assessment and management. Suicidal attempts are more a cry for help to cope with distress rather than a definite wish to die.

The steps:

1. Ask questions to elicit suicidal thoughts and plans.
2. Identify the risk and protective factors related to suicide.
3. Listen to the patient.
4. Enlist patient co-operation.
5. Prescribe medication.
6. Suggest general coping techniques
7. Discuss specific stress reduction strategies
8. Convey a sense of hope.
9. Enlist family support.
10. Regularly review the suicide risk

Step I: Ask questions to elicit suicidal thoughts and plans

- Direct but tactful enquiry regarding having experienced thoughts of suicide, plans made for self-harm and details of past attempts are mandatory.
- Most patients with suicidal ideation will admit it on enquiry
- Ask questions such as: "How often do you think about suicide?", "Do you feel as if you're a burden or that life isn't worth living?", "Do you have a plan to end your life?", "How much control of your suicidal ideas do you have?", "Can you suppress them or call someone for help?", "What stops you from killing yourself e.g., family, religious beliefs, etc.?", "Do you have access to means of killing yourself?"

Step II: Identify risk and protective factors related to suicide

Risk and protective factors for suicide are many and can occur in varying combinations making prediction difficult. The following factors imply increased risk:

- Factors associated with completed suicides
  - Male gender
  - Older age
  - Being widowed or divorced
  - Living alone
  - Alcohol or substance use
  - Having a chronic medical illness and
  - Family history of suicide are associated with high risk
- Details of previous attempt
  - Serious intent to die, use of lethal method, meticulous planning and the presence of suicidal note are associated with high risk.
- Evaluation of current status
  - Severe depression or psychosis, acknowledgement of detailed plans for future attempts, presence of hopelessness, low frustration tolerance and poor social supports are associated with high risk.

Step III: Spend time listening to the patient

Management of suicidal risk starts with listening to the patient's point of view without providing premature reassurance. Time should be spent in understanding the patient and his or her stress, coping, personality, supports and context.

Step IV: Enlist patient co-operation

- In patients who are not intoxicated or psychotic it is sometimes possible to enter into a "no-harm contract". This is an agreement between the patient and the physician where the patient agrees not to harm him/herself for a specific time (e.g., 24 to 48 hours).
- This is followed up by frequent visits to the doctor or contact by telephone. Such a contract, which is renewed once the specific time period ends, is not legally binding but may help in tiding over the acute period of crisis.

Step V: Prescribe medication

- Antidepressant medication is useful in the treatment of depression and to reduce anxiety
- Selective serotonin reuptake inhibitors such as Fluoxetine or Sertraline are preferred to tricyclic antidepressants which can be lethal in the event of an overdose
- Antipsychotics will help to reduce psychotic symptoms and people with substance use disorders need to undergo detoxification and de-addiction.

Step VI: Suggest general techniques

Measures like yoga or meditation, regular physical exercise, involvement in religious activities, hobbies and leisure help to improve a person's coping and are useful for those under stress.

Step VII: Discuss specific coping strategies

- Patients with specific problems resulting in distress and suicidal ideation will need help with problem solving
- A detailed discussion on the immediate steps for stress reduction may be necessary
- The patient and family should be asked to think of long term and permanent solutions. Some problems which may not be solvable will require acceptance and a change in attitude to assist in coping.

Step VIII: Convey a sense of hope

Sources of strength, self-esteem and deterrents to self-harm such as children in the home, family

relationships, religion, work and positive support systems need to be identified and focused upon to convey a sense of hope and purpose. This also helps the patient focus on the positive aspects of his/her life rather than solely on the difficulties.

Step IX: Enlist family support

Enlisting the family's support is essential. They will need to play a crucial role in the provision of support, keeping him/her supervised at all times and removing access to lethal means. Issues within the family may be the cause of conflict; in this case, involving the family may be necessary for problem solving.

Step X: Regularly review the suicidal risk

- Regular review of progress is necessary for such patients with scheduled appointments, more frequently (e.g. within 48 hours) during the period of acute crisis
- Suicide risk and safety issues will need to be reassessed at various points throughout treatment, as suicidal risk can change with time.
- Some patients may in fact be at increased risk for suicide as the patient regains his or her energy to carry out their plans while feelings of hopelessness and depressed mood persist
- The provision for emergency contact if the situation worsens is also mandatory.

Referral

Patients with moderate risk should be referred to psychiatrists while those with high risk will also require hospitalization and close supervision to prevent suicide.

The Management Of Unexplained Medical Symptoms

Symptoms

Patients with common psychiatric and psychosocial problems often present to primary care. These disorders include anxiety, depression, phobia, panic, obsessive compulsive disorder and problems secondary to acute and chronic stress. While very few patients report with psychological distress as a presenting complaint, the majority usually report distressing physical symptoms for which known medical causes are not found.

The steps:

1. Acknowledge distress caused by the physical symptoms.
2. Elicit the patient's perspective on her/his symptoms.
3. Do a focused physical examination and laboratory investigations to rule out medical problems.
4. Provide specific reassurance for the symptoms.
5. Discuss alternative explanations for the patient's symptoms.
6. Prescribe medication.
7. Suggest general coping techniques.
8. Discuss specific stress reduction strategies.
9. Transfer responsibility for improvement to the patient.
10. Provide an appointment for review.

Step I: Acknowledge distress

- Acknowledging the distress caused by the physical symptoms reassures the patient that their symptoms have been carefully considered rather than dismissed
- It reflects an empathetic attitude, contributes to establishing an effective rapport and the formation of a therapeutic alliance which are essential in facilitating improvement.

Step II: Elicit patient's perspective on symptoms

- Asking patients what they think or fear is wrong with them is useful in addressing specific concerns (e.g. `It could be cancer')
- These beliefs need to be discussed prior to presenting alternative biomedical explanations
- Eliciting such explanations will also allow for focused examination, investigations and specific reassurance.

Step III: Focused history, physical examination and laboratory investigations

- Physical disease, substance dependence and psychosis have to be excluded with a focused history, physical examination and laboratory investigation.
- This will go a long way in reassuring the patient that medical causes have not been overlooked.

Step IV: Reassure patients about symptoms

- Being told that there is no serious medical problem underlying their symptoms is effective in reducing the health concern for many patients.
- The emphasis should be on providing reassurance while acknowledging the reality and distress of the symptoms.

Step V: Providing an alternate explanation

- Alternative explanations for the patient's symptoms (e.g. the individual's tendency to interpret innocuous bodily sensations as bodily dysfunctions or symptoms related to stress) are useful
- Simple explanations of possible links between anxiety and stress causing physical symptoms or how depression lowers the pain threshold are useful.

Step VI: Prescribe medication

- Most patients expect medication
- Antidepressant medication is useful and can be prescribed if depression, anxiety, panic, phobia or obsessive compulsive symptoms are present
- They can also be prescribed in conditions where pain is incapacitating (e.g. headache, irritable bowel syndrome, atypical chest pain).
- Tricyclic antidepressants such as dothiepin, imipramine and amitriptyline (25-150 mg/day) are especially useful in patients with insomnia
- Common side effects include sedation, giddiness, dry mouth and constipation.
- Serotonin selective reuptake inhibitors (Fluoxetine 20mg/day and Sertraline 25-100 mg/day) are non-sedative and have fewer side effects than the tricyclic agents, though they can produce nausea and restlessness.
- Mirtazapine (7.5-15 mg/day) is a sedative antidepressant that does not have specific cardiac side effects.
- In general, antidepressants are prescribed for 3-6 months followed by a tapering schedule. They can be continued for many years if a relapse of symptoms occurs on withdrawal.
- Patients without symptoms described above can be given vitamins and the placebo response often helps them cope with their circumstances.
- Benzodiazepines are best avoided as they produce dependence if prescribed for more than one month.

Step VII: Suggest general stress reduction strategies

- Recommending general psychological measures like
  - Yoga
  - Meditation
  - Regular physical exercise
  - Involvement in religious activities
  - Hobbies and leisure improve coping and are useful for those under stress

Step VIII: Explore possible specific measures to reduce stress

- Patients with specific problems will need help with problem solving
- Specific education and treatment is helpful for sexual misconceptions, sexual dysfunction and the lack of contraception
- Often life situations may be difficult to resolve and may require acceptance and change in coping strategies.

Step IX: Transfer responsibility for improvement

- Most patients expect cures from Doctors and come back with the same or new complaints for the physician to resolve
- The responsibility for improvement should be gently but firmly transferred to the patient
- The offer of psychological support will help patients cope with stress.

Step X: Give a specific appointment for review

- Regular review of progress is necessary for most patients with scheduled, brief appointments every two to four weeks, avoiding "as-needed" appointments
- A brief, focused physical examination must be performed at each visit that helps rule out any new or worrisome condition
- The physician should gradually shift the focus away from the patient's physical symptoms to the psychosocial context that may be most affecting

them

- Restoration of function must be the treatment goal rather than complete symptom elimination.

Referral

Psychiatric evaluation and specialist counseling may be necessary for incapacitating mental disorders, intractable interpersonal difficulties and for persistent sexual dysfunction and substance dependence.

Appendix

- Anxiety
  - The apprehensive anticipation of future danger or misfortune accompanied by feelings of dysphoria or somatic symptoms of tension.
- Depression
  - The persistent sadness, low mood and loss of interest or pleasure in nearly all activities, often associated with sleep disturbance, loss of appetite and weight, decreased libido and suicidal ideation.
- Obsessive compulsive disorder
  - The presence of recurrent obsessions (persistent ideas, thoughts, impulses, or images which are experienced as intrusive and inappropriate and cause marked anxiety or distress), or compulsions (repetitive behaviors or mental acts who aim is to reduce anxiety) which are often time consuming, distressing and cause impairment in functioning.
- Panic
  - The sudden onset of intense apprehension, fearfulness and terror often associated with a feeling of impending doom which last for short duration (less than 30 minutes) and may be associated with anticipatory anxiety and avoidance.
- Phobia
  - A persistent, irrational fear of a specific object or situation that results in a compelling desire to avoid it or to enduring it with dread.

Dermatology

Standard Treatment Guidelines
Tamil Nadu Health Systems Project

Chapter 21

Pyoderma

Bacterial infection of skin

Impetigo

* Starts as an erythematous macule.
* Develops into a vesicle with erythematous halo.
* Vesicle breaks down with oozing.
* Dries to form golden yellow crust.
* Spreads by auto inoculation.

Folliculitis

* Follicular oriented pustules without peri-follicular oedema.

Furuncle

* Erythematous, painful, tender.
* Form discrete follicular nodules with perifollicular oedema.

Carbuncle

* Infection of multiple adjoining hair follicles.
* Presents with painful, tender plaque which ulcerate.

Ecthyma

* Deeper infection.
* An ulcer covered with adherent crust.
* Heals with scarring.
* Rule out Diabetes mellitus in case of recurrent furunculosis and carbuncle.

For recurrent pyodermas

* Topical application of Neosporin or Fusidic acid to carrier sites especially anterior nares and natal clefts for 2 weeks.

Treatment guidelines:

* Good personal hygiene.
* Wash with soap and water.
* Topical antibacterial cream. Mupirocin / Fusidic acid
* Systemic antibiotics
  » Azithromycin 250 mg bd for 7 days
  » Ampicillin 250 mg qid for 7 days

Erysipelas

- Erysipelas is a superficial form of bacterial cellulitis.
- Usually seen over the face or leg.
- H/o minor injury may be present.
- Presents as sharply defined erythematous tender swelling.

Rx Treatment

- Systemic antibiotics. Roxithr mycin 150 mg bd for 10 days. Ampicillin + Cloxacillin 500 mg bd for 7 days.
- Non steroidal anti-inflammatory drugs.
- Serratiopeptidase.

Cellulitis

- Cellulitis is a deep subcutaneous infection with lymphangitis and adenopathy.
- Borders of the lesion are poorly demarcated.
- Vesicles and bulla may be seen over the surface in both conditions.

Rx Treatment

- Systemic antibiotics. Roxithr mycin 150 mg bd for 10 days. Ampicillin + Cloxacillin 500 mg bd for 7 days.
- Non steroidal anti-inflammatory drugs.
- Serratiopeptidase.

Cutaneous Tuberculosis

Clinical types

- Lupus vulgaris
- Tuberculosis verrucosa cutis
- Scrofuloderma

Lupus vulgaris

* Reddish brown plaque which spreads with foci of scarring.
* Has an active advancing edge and areas of scarring.

Tuberculosis verrucosa cutis

* Indurated warty papule or nodule or a plaque. with surrounding erythema.
* There may be fissuring and discharge from the surface.
* Irregular extension leads to serpigenous margin.

Scrofuloderma

* It occurs over the underlying focus of tuberculosis like caseating lymph gland, bone, joint etc.
* Presents as painless bluish red swelling which breaks down to form sinus or undermined ulcer.

Treatment

- Antituberculous treatment – 6 months
  * 4 drugs – 2 months
  * 2 drugs – 4 months
  * Refer RNTCP guidelines

Leprosy

Non lepromatous

* Hypopigmented anaesthetic patches.
* Peripheral nerve thickening.
* Anaesthesia along the distribution of the involved nerve.

Lepromatous

* Multiple vague ill defined hypopigmented macules.
* Ear lobe infiltration.
* Madarosis.
* Glove and stocking type of anesthesia.

Treatment

Paucibacillary

* Single nerve involvement
* < 5 patches
* Negative acid fast bacilli.

Tuberculoid Leprosy

single lesion with a well defined active edge and anaesthesia within the lesion

Lepromatous Leprosy

Nodules over face with loss of eyebrows and early saddle nose deformity

* Rifamipicin 600 mg once monthly for 6 months
* Dapsone 100mg daily for 6 months

Multibacillary

* More than one nerve involvement
* >5 patches
* Positive acid fast bacilli.
* Rifampicin 600 mg once a month
* Dapsone 100mg daily
* Clofazimine 300 mg once a month or 50 mg daily
* Total duration of treatment is for 3 years

Scabies

- Multiple itchy, papules and vesicles.
- Occurs in web spaces, hands, wrist, elbow, axilla, abdomen and glans penis. Characteristic lesions are burrows.
- Common on children and those living closely

Complication

Secondary infection

Differential diagnosis

Insect bite allergy – lesions over exposed areas, web spaces are free.

Treatment

1 % gamma benzene hexachloride

- Lotion applied over the whole body below the neck repeated after 8 days
  * Contact period 12-24hrs
  * Administer cautiously in pregnancy and infants
  * For children dilute with equal parts of water
- Systemic antibiotics for secondary infection.
- 5% Permethrin cream can also be applied as above.
- All family members to be treated.
- T. cetrizine 1 HS for 10 days for itching
- Crusted scabies, scabies in infants – refer to higher centre.

Chicken pox

Prodromal symptoms

- Fever
- Body pain
- Starts as multiple erythematous macule, then progresses to vesicles and crusting stages
- Centripetal distribution
- Polymorphism in each affected site

Complication

* Secondary infection
* Pnuemonitis
* Encephalitis
* Thrombocytopenia

- One attack confers long lasting immunity

Treatment

- In healthy children
  * Symptomatic treatment with rest, analgesics and topical calamine lotion.
- Antiviral treatment for varicella in adults
  * Severe varicella and in immunocompromised individuals preferably started within 1 to 2 days
  * Oral acyclovir - children 20mg / kg qid
  * Adults 800 mg 5 times daily for 10 days

- Severe varicella or systemic complication refer to higher centre or infectious disease hospital

Herpes zoster

Prodromal symptoms

- Pain at the site.
- Closely grouped reddish papules vesicles and pustules in continuous or interrupted band in one or adjacent dermatomes.
- Usually unilateral and does not cross the midline.
- Thoracic segments most commonly involved.

Herpes - Oral

Herpes - zoster

Herpes - eye

- Disseminated herpes zoster occurs in immuno-compromised individuals.

Differential diagnosis

Herpes simplex

Complication

- Secondary infection.
- Ophthalmic involvement in herpes zoster ophthalmicus.
- Post-herpetic neuralgia.

Rx Treatment

- Topical
  - 1 % silver sulphadiazine
  - Acyclovir cream
- Systemic
  - Adults -- T. Acyclovir 800 mg 5 times daily for 10 days or
  - T. famcyclovir 500 mg tid – 7 days

Molluscum Contagiosum

- Discrete skin coloured waxy hemispherical firm papules. with central umbilication.
- Seen singly or in clusters.
- Common in children.
- Extensive and giant Molluscum contagiosum seen in immuno-compromised and in HIV states.

Treatment

- Cryotherapy with liquid nitrogen.
- Electrocautery.
- Topical 10 % trichloroacetic acid.
- Spontaneous resolution possible.

Warts

Benign proliferative lesions of cutaneous epithelium. Presents as different clinical types.

Clinical types

- Common warts
- Plane warts
- Filiform or digitate warts
- Plantar warts

Common warts

* Firm keratotic papules with rough horny surface.

Plane warts

* Multiple smooth, flat or slightly elevated round or polygonal papules (1 to 5 mm).

Filiform or digitate warts

* Composed of one or more finger like projections 2 to 10 mm in length.

Plantar warts

* Sharply defined rough keratotic lesion.
* Surrounded by a smooth collar of thickened horn.

Differential diagnosis

Callosity

Treatment

- Spontaneous resolution possible.
- Topical keratolytics.
  * 16.7 % salicylic acid and 16.7 % lactic acid in collodion base.
- Trichloroacetic acid applied weekly.
- Topical retinoic acid.
- Topical 5 – flurouracil ointment.
- Electrocautery - liquid nitrogen therapy.
- Podophyllin 25 % for genital wart.

Tinea capitis

Tinea capitis presents with different clinical types and is more common in children

Grey patch

* Well defined scaly circular patches.
* With partial alopecia showing broken off hair.

Black dot

* Broken off hair near the surface give appearance of black dot.
* Diffuse and poorly circumscribed lesions with low grade folliculitis.

Kerion

* Boggy purulent inflammatory nodules and plaques with sinus formation and pus discharge which leads to thick crusting and matting of adjacent hairs.
* Lymphadenopathy present
* Heals with scarring alopecia

Favus

* Perifollicular erythema and matting of hair with fetid odour.

- Heals with scarring alopecia.

Treatment

Topical cream

* Clotrimazole
* Miconozole
* Terbinafine

Systemic anti-biotics

* Griseofulvin 250 to 500 mg bd for 2 weeks; preperably 4 weeks
* Systemic antibiotics in case of secondary infection
  » Tab Fluconazole 150mg one/wk x 4weeks

Tinea corporis

- Well defined scaly annular patches.
- With or without vesicles pustules at margin.
- Single or multiple scattered lesions.

- Itching +

Treatment

Topical cream

* Whitfield ointment
* Miconozole
* Terbinafine

Systemic

* Griseofulvin 250 to 500 mg bd for 10 days
* Tab. Fluconazole 150 mg one/ week x 4-6 weeks

Intertrigo

- Caused by candida infection.
- Sharply demarcated.
- Polycyclic erythematous eroded patches with satellite pustules.
- Seen over intertrigenous areas, groin, inbetween toes, below the neck in children.
- Rule out underlying Diabetes mellitus.

Treatment

- Keep intertrigenous areas dry.
- Topical miconozole / terbinafine cream.
- Systemic ketoconozole 200 mg / d for 7 days.
- Treat underlying Diabetes mellitus.

Urticaria

- Elevated erythematous itchy swellings
- Transient in nature
- Worsened by scratching
- Acute: < 6 weeks
- Chronic: > 6 weeks
  * Angio-oedema involves the deeper structures.
- Provoking factors
  * Drugs
  * Food
  * Infection
  * Stress
  * Systemic diseases

Treatment

- Eradication of the etiological factor
- In acute urticaria with laryngopharyngeal edema 0.5 to 1 ml of subcutaneous epinephrine.
- Antihistamine
  * Loratidine 10 mg / day
  * Cetrizine 10 mg / day
- Severe extensive cases
  * Prednisolone 0.5 to 1 mg / kg per day
- Recurrent and severe cases refer to higher centres

Psoriasis

- Erythematous well defined scaly plaques
- Predominantly extensor aspect of the body
- Scales are silvery and loosely attached to the lesion
- **Koebner phenomeno**n is positive – new lesion occurs at the site of trauma
- Scalp
  * Scaling that extends beyond the hair line
- Nails
  * Collection of keratotic material under the nail
  * Lifting the distal part of the nail from the nail bed and
  * Nail pitting
- Joints may be involved

Treatment

- Topical keratolytics – salicylic acid
- Liquid paraffin for external use.
- Topical coal tar, anthralin
- Topical tazoretine cream
- Potent topical steroids for localized lesion
- Systemic
  * Methotrexate
  * Retinoids
  * Patients with extensive involvement, arthropathy, pustular psoriasis, erythro dermic psoriasis -- Refer to higher centre.

Note:

Systemic Steroids should be avoided

Lichen Planus

- Well defined, violaceous, polygonal flat topped papules
- Itching +
- Common sites: legs, forearm
- Koebner phenomenon +ve
- Involvement of hair leads to alopecia
- Oral mucosa: lacy network of whitish patches

Treatment

- Topical steroids
- T. Cetrizine 10 mg hs for continuous itching
- T.Dapsone 100mg daily
- Generalized forms ---- systemic steroids
- Refer to higher centre for extensive lesions

Pityriasis Rosea:

- Initial lesion 2 to 6 cm circular asymptomatic erythematous scaly plaque – **Herald patch**
- Within days crops of papules and oval patches with wrinkling surface and a border of fine scales
- Trunk and proximal extremities are commonly involved.
- Lesions heal with hyper or hypo pigmentation.

Differential diagnosis:

- Psoriasis
- Secondary syphilis
- Tinea corporis

Treatment

- Mostly do not require treatment
- Symptomatic treatment
- Topical moisturizers
- T.cetrizine, if itching is present

Acne

- Common in adolescents.
- Present as comedones, papules , pustules,nodules abscess, cysts, scars
- Sites - Face, chest, back, shoulder.
- Sequelae: hyper pigmentation and scarring

Differential Diagnosis:

- Acne rosacea.
- Drug induced acneform eruption (monomorphic)

Treatment

Grade I

* Comedones / occasional papules:
* Removal of comedones
* Topical retinoic acid / benzoyl peroxide

Grade II

* Papules, comedones, pustules
* Topical antibiotics
  - » Clindamycin
  - » Erythromycin 2 to 4 %
* Benzoyl peroxide
* Retinoic acid

Grade III

* Predominant pustules, nodules, abscess
* Topical benzoyl peroxide
* Retinoic acid
* Systemic
  - » Tetracycline
  - » Erythromycin
  - » Doxycycline
* Isotretinoin 1 mg / kg 16 to 24 weeks

Grade IV

- Mainly cysts, abscess, widespread scarring
- Systemic antibiotics as in grade iii
- Aspiration of cysts
- Intra-lesional steroids for cysts
- T. Dapsone

Pityriasis Alba

- Asymptomatic round or oval hypo-pigmanted scaly patches usually on the face.
- Single or multiple.
- Spontaneous resolution but may recur.
- Age: 3 years to puberty.

Differential diagnosis

- Early vitiligo.
- Pityriasis versicolor.
- Leprosy.
- Post inflammatory hypo pigmentation.

Rx Treatment

- Reassurance
- Bland emollients for external application
- Multivitamins
- Treatment of worm infestation if any

Miliaria

- Small discrete itchy non-follicular erythematous confluent macules or papules forming sheets
- Pricking sensation +.
- Occurs more in summer and heat condition.

Rx Treatment

- Avoidance of excessive exposure to heat
- Calamine lotion
- Antihistamines
- Vitamin-C
- Loose cotton dresses
- Plenty of oral fluids etc.,

Sexually Transmitted Infections (STI)

Early syphilis

Primary syphilis

- Causative agent - Treponema pallidum
- Incubation period - 9 to 90 days
- Genital ulcer (primary chancre) - classical lesion
- Regional lymphadenopathy in 50% of cases
- Primary chancre resolves in 3 to 6 weeks.
- After 3 weeks of appearance of chancre, VDRL becomes reactive
- Treponema pallidum can be demonstrated from the chancre
- In the case of untreated primary syphilis, patient may develop secondary syphilitic lesions in 3-6 months interval.

Secondary syphilis

- Skin rash
  - Macular
  - Papular
  - Papulosquamous
  - Psoriatiform
  - Annular
  - Pustular
  - Follicular
- Rash is generalized, bilaterally symmetrical, non-itchy
- Mucosal lesions
  - Snail track ulcers
  - Condylomata lata
- Painless discrete, rubbery lymph nodes, systemic manifestations.
- Highly reactive VDRL
- Treponema pallidum can be demonstrated from the skin lesions.

Early latent syphilis

- No signs and symptoms
- 2-year period is demarcation between early and late latent syphilis.

Rx Treatment

For all the above 3 conditions,

Injection: Benzathine Penicillin 2.4 mega units deep IM after test dose (or)

Injection: Procaine penicillin 1.2 mega units deep IM daily for 10 days

In Penicillin allergic patients

- Cap.Doxycycline 100 mg orally b.d x 2 weeks (or)
- Cap.Tetracycline 500 mg orally qid x 2 weeks (or)
- Tab.Erythromycin 500 mg orally qid x 2 weeks.

Follow-up

After treatment the patient should be examined clinically and serologically once a month for first 3 months and quarterly till the end of second year.

Late syphilis

In untreated early syphilitic patients will have the late syphilitic manifestations after 5-15 years from the

onset of infection.

- Late latent syphilis
  - After 2 years from the onset of infection.
  - No signs and symptoms
  - Non-infectious
- Late benign syphilis (gumma)
  - After 5-7 years from the onset of infection.
  - Cutaneous gumma
  - Visceral gumma
- Cardiovascular syphilis
  - After 10-15 years from the onset of infection.
  - Uncomplicated aortitis
  - Aortic regurgitation
  - Coronary ostial stenosis
  - Aortic aneurysm
- Neuro-syphilis
  - After 10-15 years from the onset of infection
  - Meningitis, meningo-vascular
  - General Paresis of Insane (GPI)
  - Tabes dorsalis
  - CNS gumma

Rx Treatment

Late Latent Syphilis and Late Benign Syphilis (Gumma)

- Injection
  - Benzathine Penicillin 2.4 mega units IM weekly for 4 consecutive weeks (or)
  - Procaine Pencillin 1.2 mega units IM daily for 21 days.

Cardiovascular syphilis and neuro syphilis

- Injection
  - Procaine Pencillin 1.2 mega units IM/day for 21 days under the cover of steroids.

In Penicillin allergic patients,

- Cap.Doxycycline 100 mg orally b.d x 4 weeks (or)
- Cap.Tetracycline 500 mg orally qid x 4 weeks (or)
- Tab.Erythromycin 500 mg orally qid x 4 weeks.

Follow-up:

The patient should be monitored at 6 months interval clinically and serologically along with repeat cerebro spinal examination.

Congenital syphilis

Untreated early syphilitic mother may deliver a congenital syphilitic child.

Early

- Skin lesions - Rash, Bullae, Condylomata lata
- Lymphadenitis, Hepatosplenomegaly, Osteochondritis
- CNS, Kidneys, Lungs, Testes involvement

Late

- Interstitial keratitis
- Neurosyphilis
- Bone and joint involvement
- Sensori-neural deafness
- Gummatous lesions
- Cardiovascular syphilis involvement

Stigmata

- Bulldog facies
- Hutchinson's teeth
- Rhagades
- Salt and pepper Fundus
- Gummatous scars

Rx Treatment

Early

- Inj. Procaine penicillin 50,000 units / kg /day IM for 10 days (or)
- Inj. Bezathine Penicillin 50,000 units / kg /day IM single dose

Late

- Inj. Procaine Pencillin 50,000 units / kg /day IM for 20 days

Patient education

- To seek early and appropriate treatment.
- To avoid sexual contact until the treatment is completed.
- Insisting epidemiological treatment of sex part-

ners, with regular follow-up.

Prevention

- Sexual abstinence.
- Avoid high risk behaviour.
- Safer sex practices.
- Consistent and correct usage of condoms.

Chancroid

- Causative agent - Haemophilus ducreyi
- Incubation period - 1 to 14 days
- Multiple, painful, non indurated ulcers with ragged and undermined edges. Floor is covered by a yellowish grey necrotic exudate overlying granulation tissue that bleeds on manipulation.
- In 50% of the patients, a tender inguinal adenitis (usually unilateral) occurs (Inflammatory Bubo).
- "School of fish appearance" in smears

Rx Treatment

- Erythromycin 500mg orally qid for 7 days (or)
- Azithromycin 1g oral stat (or)
- Ciprofloxacin 500mg oral bd for 3 days (or)
- Inj. Ceftrioxone 250mg single dose

Lymphogranuloma Venereum (LGV)

- Caused by Chlamydia trachomatis serovars L1, L2, L3
- Incubation period - 3 to 12 days
- Primary – Transient genital ulcer
- Secondary – Inflammed and enlarged inguinal lymph nodes (bubo)
- Tertiary complications include-
  - elephantiasis of genitalia
  - Fistulae
  - Strictures
  - Carcinoma

Rx Treatment

  - Doxycycline 100mg oral bd for 3 weeks (or)
  - Erythromycin 500mg oral qid for 3 weeks

Venereal granuloma

- Causative agent – Calymmatobacterium granulomatis
- Incubation period – 1 to 3 months
- Granulomatous ulcer
- Calymmatobacterium granulomatis (Donovan bodies) seen inside large mononuclear cells in tissue smears

Rx Treatment

- Doxycycline 100mg oral bd for 14 days (or)
- Tetracycline (or) Erythromycin 500mg oral qid for 14 days

Gonorrhoea

- Causative agent - Neisseria gonorrhoeae
- Incubation period – 1 to 14 days
- Presents as urethritis in male and cervicitis in female
- Complications:
  - Male
    - Posterior urethritis
    - Infection of cowper's and tyson's gland
    - Epididymitis
    - Prostatitis
    - Seminal vesiculitis
    - Peri-urethral abscesses
  - Female
    - Salpingitis
    - Peritonitis
    - PID
    - Bartholinitis
    - Proctitis
    - Chronic urethritis

Other forms

- Disseminated gonococcal infection
- Gonococcal arthritis
- Meningitis
- Anorectal and
- Pharyngeal gonorrhoea

Treatment

- Azithromycin 2g oral stat (or)
- Inj. Ceftriaxone 250mg IM stat (or)
- Cefixime 400mg oral stat.

Non-gonococcal urethritis

- Causative agent
  - Chlamydia trachomatis
  - Ureaplasma urealyticum
  - Mycoplasma genitalium
- Urethritis
- Increased polymorphonuclear leucocytes in urethral smear or sediment of first voided urine

Treatment

- Doxycycline 100mg oral bd for 7 days (or)
- Tetracycline / Erythromycin 500mg oral qid for 7 days

Chlamydial Infections

- Agent – Chlamydia trachomatis
- Incubation period – 1 to 3 weeks.
- Males
  - Urethritis
  - Littritis
  - Epididymitis
  - Prostatitis
  - Proctitis
  - Reiter's syndrome
- Female
  - Cervicitis
  - Urethritis
  - Bartholinitis
  - Endometritis
  - Salpingitis
  - Fitz Hughcurtis syndrome

Treatment

- Azithromycin 1gm oral stat (or)
- Doxycycline 100mg oral bd for 7 days (or)
- Erythromycin 500mg qid for 7 days

Bacterial Vaginosis

- Polymicrobial syndrome
  - Gardnella vaginalis
  - Mobilincus
  - Mycoplasma hominis
  - Bacteroids
- Excessive, homogenous, uniformly adherent vaginal discharge
- Elevated vaginal pH > 4.5
- Positive Amine test (Whiff test)
- Presence of "Clue-cells" (Epithelial cells studded with organisms)

Treatment

- Tab. Metronidazole 400mg BD for 7 days (or)
- Tab. Metronidzole 2g stat dose.

Trichomoniasis

- Caused by Trichomonas vaginalis
- Incubation period – 4 to 28 days
- Copious, homogenous, malodorous, yellowish green vaginal discharge
- Punctate hemorrhages over cervix - Strawberry cervix
- Demonstration of motile trichomonads in wet film smears under direct microscopy.
- Culture – gold standard for diagnosis

Treatment

- Tab. Metronidazole 400mg bd for 7 days (or)
- Tab. Metronidzole 2g stat dose

Candidiasis

- Predominantly caused by Candida albicans
- Predisposed by
  - Pregnancy
  - Diabetes
  - Immunosuppression
  - HIV
- Causes Balanoposthitis in males
  - Clinically erythema and swelling with a macular/ papular rash over glans penis and foreskin

and superficial ulceration can be seen

* A white subpreputial discharge, increased skin markings, fissuring of the glans and foreskin occasionally regional lymphadenopathy may be present.

- Causes vulvo vaginal candidiasis in females - oedema, fissures, erosions, curdy white discharge.
- Demonstration of candida by 10% KOH – yeast cells, mycelia seen

Treatment

- Topical ointments
- Tab. Fluconazole 150mg stat (or)
- Clotrimazole vaginal pessaries

Wart (Condylomata Acuminata)

- Caused by Human Papilloma Virus
- Incubation period – 1 to 8 months
- Skin coloured, multiple verrucous lesions

Treatment

- Topical podophyllin
- Cryotherapy
- Cautery
- Surgery
- Imiquimod etc.

Herpes Genitalis

- Predominantly by HSV-2. also by HSV-1
- Incubation period – 5 to 14 days
- Ballooning degeneration and giant cells seen in HPE
- Primary – severe, associated with systemic symptoms. starts as grouped vesicles - ulcerate - polycyclic margins
- Recurrence - less severe
- Complications
  * CNS involvement, dissemination
  * Secondary infection
  * Recurrence
- Transmissible during pregnancy and delivery

Treatment

- For Primary
  * Tab. Acyclovir 200mg 5 times a day for 7 days.
- For Recurrence
  * Tab. Acyclovir 400mg 3 times a day for 5 days.

Syndromic Managemen Of Std

The common STD syndromes

- Genital ulcer diseases
- Urethral discharge
- Vaginal discharge
- Bubo (inguinal swellings)
- Lower abdominal pain in female
- Scrotal swelling
- Ophthalmia neonatorum

Syndromic approach for STIs is a useful and practical strategy for offering, high quality, effective and acceptable care for prevention and treatment of sexually transmitted infections (Guidelines from National AIDS Control Organization (NACO) has been used).

Genital Ulcer

Herpes

Acyclovir – 200 mg x 5 times x 7 days

Syphilis

Inj.Benzathine Penicillin 2.4 M.U

and

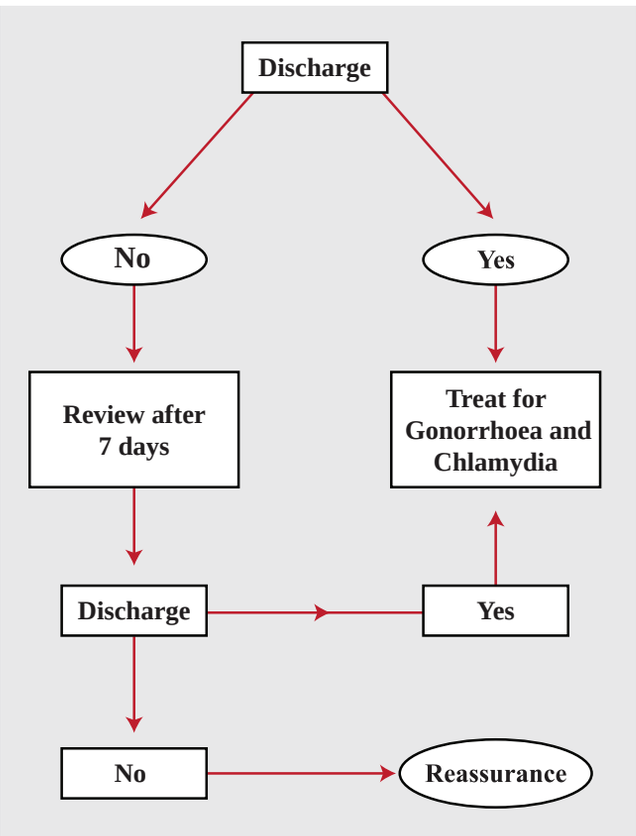
Chancroid - Azithomycin - 1 gm

Inj. Ceftriaxone 250 gm

Azithromycin – 1 gm

(or)

Inj.Ceftriaxone 250 mg



Urethral discharge (male)

For Gonococcal and Chlamydial infection

1. Azithromycin – 2 gm OD

   (or)

2. Cefixime – 400 mg PO – single

   +

   Doxy – 100 mg bd x 7 days

   (or)

3. Inj.Ceftrioxone – 250 mg single

   +

   Doxy – 100 mg bd x 7 days

Vaginal discharge

For gonococcal and chlamydial infection

- Azithromycin – 2 gm single dose

  (or)

- Cefixime - 400 mg single dose

  +

- Doxycycline - 100 mg bd x 7days

For trichomoniasis and bacterial vaginosis

Inguinal bubo

If bubo with ulcer is present follow Genital ulcer treatment.

Only Bubo – Doxycycline 100 mg bd for 21 days.
For Trichomoniasis and Bacterial vaginosis

- Metronidazole – 2 gm stat

  (or)

  Metronidazole – 400 mg bd x 7 days

- For candidiasis

  Fluconazole - 150 mg

  (or)

  Clotrimazole pessary – 100 mg intravaginal x 6 days

Lower abdominal pain (female)

For gonococcal, chlamydial, trichomoniasis, bacterial vaginosis

- Azithromycin - 2 gm single dose

  +

  Metronidazole – 400 mg bd x 14 days

  (or)

- Inj.Ceftriaxone 250 mg single dose

  +

  Doxy 100 mg bd x 14 days

  +

  Metronidazole – 400 mg bd x 14 days

  (or)

- Tab. Cefixime 400 mg

  +

  Doxy 100 mg bd x 14 days

  +

Metronidazole – 400 mg bd x 14 days

Scrotal swelling

For gonococcal and chlamydial infection

- Azithromycin 2 gm od

  (or)

  Inj.Ceftriaxone 250 mg single dose

  +

  Doxy 100 mg bd x 14 days

  (or)

- Cefixime 400 mg orally – single dose

  +

  Doxy 100 mg bd x 14 days

Ophthalmia neonatorum

For gonococcal and chlamydial infection

- Inj.Ceftriaxone 50 mg / kg single dose

  +

  Erythromycin 50 mg / kg / day / 4 divided dose x 14 days

  or

- Kanamycin 25 mg / kg single dose

  +

  Erythromycin 50 mg / kg / 4 divided dose x 14 days

Organ transplant Guidelines

Standard Treatment Guidelines
Tamil Nadu Health Systems Project

Chapter 23

1. Organ Transplant Act
2. Organ Transplant Rules
3. Organ Transplant GO 75
4. Organ Transplant GO 287

Ministry of Law, Justice and Company Affairs
(Legislative Department)
New Delhi, the 11th July, 1994

The following Act of Parliament received the assent of the President on the 8th July, 1994 and is hereby published for general information:-

THE TRANSPLANTATION OF HUMAN ORGANS ACT, 1994

No.42 OF 1994
[8th July, 1994]

An Act to provide for the regulation of removal, storage and transplantation of human organs for therapeutic purposes and for the prevention of commercial dealings in human organs and for matters connected therewith or incidental thereto.

Whereas it is expedient to provide for the regulation of removal, storage and transplantation of human organs for therapeutic purposes and for the prevention of commercial dealings in human organs;

And whereas Parliament has no power to make laws for the States with respect to any of the matters aforesaid except as provided in articles 249 and 250 of the Constitution;

And whereas in pursuance of clause (1) of article 252 of the Constitution, resolutions have been passed by all the Houses of the Legislatures of the States of Goa, Himachal Pradesh and Maharashtra to the effect that the matters aforesaid should be regulated in those States by Parliament by law;

Be it enacted by Parliament in the Forty-fifth Year of the Republic of India as follows:

Chapter I
Preliminary

Short title, application and commencement

1. (1). This Act may be called the Transplantation of Human Organs Act, 1994.
(2). It applies, in the first instance, to the whole of the States of Goa, Himachal Pradesh and Maharasthra and to all the Union territories and it shall also apply to such other State which adopts this Act by resolution passed in that behalf under clause (1) of article 252 of the Constitution.
(3). It shall come into force in the States of Goa, Himachal Pradesh and Maharashtra and in all the Union territories on such date as the Central Government may, by notification, appoint and in any other State which adopts this Act under clause (1) of article 252 of the Constitution, on the date of such adoption; and any reference in this Act to the commencement of this Act shall, in relation to any State or Union Territory, means the date on which this Act comes into force in such State or Union Territory.

Definitions

2. In this Act, unless the context otherwise requires:
(a) "advertisement" includes any form of advertising whether to the public generally or to any section of the public or individually to selected persons;
(b) "Appropriate Authority" means the Appropriate Authority

appointed under section 13;

(c) "Authorisation Committee" means the committee constituted under clause (a) or clause (b) of sub-section (4) of section 9;

(d) "brain-stem death" means the stage at which all functions of the brain stem have permanently and irreversibly ceased and is so certified under sub-section (6) of section 3;

(e) "deceased person" means a person in whom permanent disappearance of all evidence of life occurs, by reason of brain-stem death or in a cardio-pulmonary sense, at any time after live birth has taken place;

(f) "donor" means any person, not less than eighteen years of age, who voluntarily authorizes the removal of any of his human organs for therapeutic purposes under sub-section (1) or sub-section (2) of section 3;

(g) "hospital" includes a nursing home, clinic, medical centre, medical or teaching institution for therapeutic purposes and other like institution;

(h) "human organ" means any part of a human body consisting of a structured arrangement of tissues which, if wholly removed, cannot be replicated by the body;

(i) "near-relative" means spouse, son, daughter, father, mother, brother or sister;

(j) "notification" means a notification published in the Official Gazette;

(k) "payment" means payment in money or money's worth but does not include any payment for defraying or reimbursing –

(i) the cost of removing, transporting or preserving the human organ to be supplied; or

(ii) any expenses or loss of earnings incurred by a person so far as reasonably and directly attributable to his supplying any human organ from his body;

(l) "prescribed" means prescribed by rules made under this Act;

(m) "recipient" means a person into whom any human organ is, or is proposed to be, transplanted;

102 of 1956

(n) "registered medical practitioner" means a medical practitioner who possesses any recognized medical qualification as defined in clause (h) of section-2 of the Indian Medical Council Act, 1956, and who is enrolled on a State Medical Register as defined in clause (k) of that section;

(o) "therapeutic purposes" means systematic treatment of any disease or the measures to improve health according to any particular method or modality; and

(p) "transplantation" means the grafting of any human organ from any living person or deceased person to some other living person for therapeutic purposes.

Chapter II

Authority for the removal of human organs

Authority for removal of human organs

3. (1). Any donor may, in such manner and subject to such conditions as may be prescribed, authorise the removal, before his death, of any human organ of his body for therapeutic purposes.

(2). If any donor had, in writing and in the presence of two or more witnesses (at least one of whom is a near relative of such person), unequivocally authorized at any time before his death, the removal of any human organ of his body, after his death, for therapeutic purposes, the person lawfully in possession of the dead body of the donor shall, unless he has any reason to believe that the donor had subsequently revoked the authority aforesaid, grant to a registered medical practitioner all reasonable facilities for the removal, for therapeutic purposes, of that human organ from the dead body of the donor.

(3). Where no such authority as is referred to in sub-section (2), was made by any person before his death but no objection was also expressed by such person to any of his human organs being used after his death for therapeutic purposes, the person lawfully in possession of the dead body of such person may, unless he has reason to believe that any near relative of the deceased person has objection to any of the decease person's human organs being used for therapeutic purposes, authorize the removal of any human organ of the deceased person for its use for therapeutic purposes.

(4). The authority given under sub-section (1) or sub-section (2) or, as the case may be, sub-section (3) shall be sufficient warrant for the removal, for therapeutic purposes, of the human organ; but no such removal shall be made by any person other than the registered medical practitioner.

(5). Where any human organ is to be removed from the body of a deceased person, the registered medical practitioner shall satisfy himself, before such removal, by a personal examination of the body from which any human organ is to be removed, that life is extinct in such body or, where it appears to be a case of brain-stem death, that such death has been certified under sub-section (6).

(6). Where any human organ is to be removed from the body of a person in the event of his brain-stem death, no such removal shall be undertaken unless such death is certified, in such form and in such manner and on satisfaction of such conditions and requirements as may be prescribed, by a Board of medical experts consisting of the following namely:

(i) the registered medical practitioner in charge of the hospital in which brain-stem death has occurred;

(ii) an independent registered medical practitioner, being a specialist, to be nominated by the

registered medical practi tioner specified in cause (i), from the panel of names approved by the Appropriate authority;

(iii) a neurolo gist or a neurosurgeon to be nominated by the registered medical practitioner specified in claus e (i), from the panel of names appr oved by the Appropriate Authority; and

(iv) the registered medical practitioner treating the person whose brain-stem death has occurred.

(7). Notwithstanding anything c ontained in sub-section (3), where brain-stem death of any person, less than eighteen years of age, occurs and is certified under sub-section (6), any of the par ents of the deceased person may give a uthority, in such form and in suc h manner as may be pr escribed, for the removal of any human or gan from the body of the deceased person.

Removal of human or gans not to be authorised in certain cases.

4. (1). No facilities shall be granted under sub-section (2) of section 3 and no authority shall be given under sub-section (3) of that section for the removal of any human organ from the body of a dec eased person, if the perso n required to grant suc h facili ties, or empowered to giv e such authority, has reason to believe that an inquest may be required to be held in r elation to such body in pursuance of the provisions of any law for the time being in force.

(2). No authorit y for the remo val of any human organ from the body of a deceased person shall be given by a person to whom such body has been entrusted solely for the purpose of interment, cremation or other disposal.

Authority for removal of human or gans in case of unclaimed bodies in hospital or prison.

5. (1). In the case of a dead body lying in a hospital or prison and not claimed by any of the near relatives of the deceased person within forty-eight hours from the time of the death of the c oncerned pers on, the aut hority for the removal of any huma n organ from the dead body which so remains unclaimed may be given, in the prescribed form, by the person in charge, for the time being, of the management or control of the hos pital or prison, or by an employee of such hospital or prison authorised in this behalf by t he person in char ge of the management or control thereof.

(2). No authorit y shall be giv en under sub-section (1) if the person empowered to give such authority has reason to believe that any near relative of the deceas ed person is likely to claim the dead body even through such near relative has not come forward to claim the body of the deceased person wit hin the time specified in such s ub-section (1).

Authority for removal of human or gans from bodies sent for post-

6. Where the body of a person has been sent for post-mortem examination-

(a) for medico-legal purposes by reason of the death of such pers on hav ing been caused by ac cident or any other unnatural cause;

mortem examination for medico-legal or pathological purposes.

OR

(b) for pathological purposes,

the person competent under this Act to give authority for the removal of any human organ from such dead body may, if he has reason to believe that such human organ will not be required for the purpose for which such body has been sent for post-mortem examination, authorize the removal, for therapeutic purposes, of that human organ of the deceased person provided that he is satisfied that the deceased person had not expressed, before his death, any objection to any of his human organs being used, for therapeutic purposes after his death or, where he had granted an authority for the use of any of his human organs for therapeutic purposes after his death, such authority had not been revoked by him before his death.

Preservation of human organs.

7. After the removal of any human organ from the body of any person, the registered medical practitioner shall take such steps for the preservation of the human organ so removed as may be prescribed.

Savings

8. (1). Nothing in the foregoing provisions of this Act shall be construed as rendering unlawful any dealing with the body or with any part of the body of a deceased person if such dealing would have been lawful if this Act had not been passed.

45 of 1860

(2). Neither the grant of any facility or authority for the removal of any human organ from the body of a deceased person in accordance with the provisions of this Act nor the removal of any human organ from the body of a deceased person in pursuance of such authority shall be deemed to be an offence punishable under section 297 of the Indian Penal Code.

Restrictions on removal and transplantation of human organs.

9 (1). Save as otherwise provided in sub-section (3), no human organ removed from the body of a donor before his death shall be transplanted into a recipient unless the donor is a near relative of the recipient.

(2). Where any donor authorizes the removal of any of his human organs after his death under sub-section (2) of section 3 of any person competent or empowered to give authority for the removal of any human organ from the body of any deceased person authroises such removal, the human organ may be removed and transplanted into the body of any recipient who may be in need of such human organ.

(3). If any donor authorizes the removal of any of his human organs before his death under sub-section (1) of section 3 for transplantation into the body of such recipient, not being a near relative, as is specified by the donor by reason of affection or attachment towards the recipient or for any other special reasons, such human organ shall not be removed and transplanted without the prior approval of the Authorisation Committee.

(4). (a) The Central Government shall constitute, by notification, one or more Authorisation Committees

consisting of such members as may be nominated by the Central Government on such terms and conditions as may be specified in the notification for each of the Union Territories for the purposes of this section.

(b) The State Government shall constitute, by notification, one or more Authorisation Committees consisting of such members as may be nominated by the State Government on such terms and conditions as may be specified in the notification for the purposes of this section.

(5). On an application jointly made, in such form and in such manner as may be prescribed, by the donor and the recipient, the Authorisation Committee shall, after holding an inquiry and after satisfying itself that the applicants have complied with all the requirements of this Act and the rules made thereunder, grant to the applicants approval for the removal and transplantation of the human organs.

(6). If, after the inquiry and after giving an opportunity to the applicants of being heard, the Authorisation Committee is satisfied that the applicants have not complied with the requirements of this Act and the rules made thereunder, it shall, for reasons to be recorded in writing, reject the application for approval.

Chapter III
Regulation of hospitals

Regulation of hospitals conducting the removal, storage or transplantation of human organs

10. (1). On and from the commencement of this Act:

(a) no hospital, unless registered under this Act, shall conduct, or associate with, or help in, the removal, storage or transplantation of any human organ;

(b) no medical practitioner or any other person shall conduct, or cause to be conducted, or aid in conducting by himself or through any other person, any activity relating to the removal, storage or transplantation of any human organ at a place other than an place registered under this Act; and

(c) no place including a hospital registered under sub-section (1) of section 15 shall be used or cause to be used by any person for the removal, storage or transplantation of any human organ except for therapeutic purposes.

(2). Notwithstanding anything contained in sub-section (1), the eyes or the ears may be removed at any place from the dead body of any donor, for therapeutic purposes, by a registered medical practitioner.

Explanation: For the purposes of this sub-section, “ears” includes ear drums and ear bones.

Prohibition of removal or transplantation of human

11. No donor and no person empowered to give authority for the removal of any human organ shall authorise the removal of any human organ for any purpose other than therapeutic purposes.

organs for any purpose other than therapeutic purposes.

Explaining effects, etc., to donor and recipient.

12. No registered medical practitioner shall undertake the removal or transplantation of any human organ unless he has explained, in such manner as may be prescribed, all possible effects, complications and hazards connected with the removal and transplantation to the donor and the recipient respectively.

Chapter IV
Appropriate Authority

Appropriate Authority

13. (1). The Central Government shall appoint, by notification, one or more officers as Appropriate Authorities for each of the Union territories for the purposes of this Act.

(2). The State Government shall appoint, by notification, one or more officers as Appropriate Authorities for the purposes of this Act.

(3). The Appropriate Authority shall perform the following functions, namely:

(i) to grant registration under sub-section (1) of section 15 or renew registration under sub-section (3) of that section;

(ii) to suspend or cancel registration under sub-section (2) of section 16;

(iii) to enforce such standards as may be prescribed, for hospitals engaged in the removal, storage or transplantation of any human organ;

(iv) to investigate any complaint of breach of any of the provisions of this Act or any of the rules made thereunder and take appropriate action;

(v) to inspect hospitals periodically for examination of the quality of transplantation and the follow-up medical care to persons who have undergone transplantation and persons from whom organs are removed; and

(vi) to undertake such other measures as may be prescribed.

Chapter V
Registration of Hospitals

Registration of hospitals engaged in removal, storage or transplantation of human organs.	14.	(1). No hospital shall commence any activity relating to the removal, storage or transplantation of any human organ for therapeutic purposes after the commencement of this act unless such hospital is duly registered under this Act. Provided that every hospital engaged, either partly or exclusively in any activity relating to the removal, storage or transplantation of any human organ for therapeutic purposes immediately before the commencement of this Act, shall apply for registration within sixty days from the date of such commencement: Provided further that every hospital engaged in any activity relating to the removal, storage or transplantation of any human organ shall cease to engage in any such activity on the expiry of three months from the date of commencement of this Act unless such hospital has applied for registration and is so registered or till such application is disposed of, whichever is earlier. (2). Every application for registration under sub-section (1) shall be made to the Appropriate Authority in such form and in such manner and shall be accompanied by such fees as may be prescribed. (3). No hospital shall be registered under this Act unless the Appropriate authority is satisfied that such hospital is in a position to provide such specialised services and facilities, possess such skilled manpower and equipments and maintain such standards as may be prescribed.
Certificate of registration	15.	(1). The Appropriate Authority shall, after holding an inquiry and after satisfying itself that the applicant has complied with all the requirements of this Act and the rules made thereunder, grant to the hospital a certificate of registration in such form, for such period and subject to such conditions as may be prescribed. (2). If, after the inquiry and after giving an opportunity to the applicant of being heard, the Appropriate Authority is satisfied that the applicant has not complied with the requirements of this Act and the rules made thereunder, it shall, for reasons to be recorded in writing, reject the application for registration. (3). Every certificate of registration shall be renewed in such manner and on payment of such fees as may be prescribed.
Suspension or cancellation of registration	16.	(1). The Appropriate Authority may, suo moto or on complaint, issue a notice to any hospital to show cause why its registration under this Act should not be suspended or cancelled for the reasons mentioned in the notice. (2). If, after giving a reasonable opportunity of being heard to the hospital, the Appropriate Authority is satisfied that there has been a breach of any of the provisions of this Act or the rules made thereunder, it may, without prejudice to any criminal action that it may take against such hospital, suspend its registration for such period as

it may think fit or cancel its registration:
Provided that where the Appropriate authority is of the opinion that it is necessary or expedient so to do in the public interest, it may, for reasons to be recorded in writing, suspend the registration of any hospital without issuing any notice.

Appeals

17. (1). Any person aggrieved by an order of the Authorisation Committee rejecting an application for approval under sub-section (6) of section 9, or any hospital aggrieved by an order of the Appropriate Authority rejecting an application for registration under sub-section (2) of section 15 or an order of suspension or cancellation of registration under sub-section (2) of section 16, may, within thirty days from the date of the receipt of the order, prefer an appeal, in such manner as may be prescribed, against such order to:

(i) the Central Government where the appeal is against the order of the Authorisation Committee constituted under clause (a) of sub-section (4) of section 9 or against the order of the Appropriate Authority appointed under sub-section (1) of section 13; or

(ii) the State Government, where the appeal is against the order of the Authorisation Committee constituted under clause (b) of sub-section (4) of section 9 or against the order of the Appropriate Authority appointed under sub-section (2) of section 13.

Chapter VI
Offences and Penalties

Punishment for removal of human organ without authority.

18. (1). Any person who renders his services to or at any hospital and who, for purposes of transplantation, conducts associates with, or helps in any manner in, the removal of any human organ without authority, shall be punishable with imprisonment for a term which may extend to five years and with fine which may extend to ten thousand rupees.

(2). Where any person convicted under sub-section (1) is a registered medical practitioner, his name shall be reported by the Appropriate Authority to the respective State Medical Council for taking necessary action including the removal of his name from the register of the Council for a period of two years for the first offence and permanently for the subsequent offence.

Punishment for commercial dealings in human organs

19. Whoever –

(a) makes or received any payment for the supply of, or for an offer to supply, any human organ;

(b) seeks to find person willing to supply for payment any human organ;

(c) offers to supply any human organ for payment;

(d) initiates or negotiates any arrangement involving the making of any payment for the supply of, or for an offer to supply, any human organ;

(e) takes part in the management or control of a body of persons, whether a society, firm or company, whose activities consist of or include the initiation or negotiation of any arrangement referred to in clause (d); or

(f) publishes or distributes or causes to be published or distributed any advertisement-

(a) inviting persons to supply for payment of any human organ;

(b) offering to supply any human organ for payment; or

(c) indicating that the advertiser is willing to initiate or negotiate any arrangement referred to in clause (d),

shall be punishable with imprisonment for a term which shall not be less than two years but which may extend to seven years and shall be liable to fine which shall not be less than ten thousand rupees but may extend to twenty thousand rupees:

Provided that the court may, for any adequate and special reason to be mentioned in the judgement, impose a sentence of imprisonment for a term of less than two years and a fine less than ten thousand rupees.

Punishment for contravention of any other provision of this Act.

20. Whoever contravenes any provision of this Act or any rule made, or any condition of the registration granted, thereunder for which no punishment is separately provided in this Act, shall be punishable with imprisonment for a term which may extend to three years or with fine which may extend to five thousand rupees.

Offences by companies.

21. (1). Where any offence, punishable under this Act, has been committed by a company, every person who, at the time the offence was committed was in charge of, and was responsible to the company for the conduct of the business of the company, as well as the company, shall be deemed to be guilty of the offence and shall be liable to be proceeded against and punished accordingly:

Provided that nothing contained in this sub-section shall render any such person liable to any punishment, if he proves that the offence was committed without his knowledge or that he had exercised all due diligence to prevent the commission of such offence.

(2). Notwithstanding anything contained in sub-section (1), where any offence punishable under this Act has been committed by a company and it is proved that the offence has been committed with the consent or connivance of, or is attributable to any neglect on the part of, any director, manager, secretary or other officer of the company, such director, manager, secretary or other officer shall also be deemed to be guilty of that offence and shall be liable to be proceeded against and punished accordingly.

Explanation: For the purposes of this section:

(a) "company" means any body corporate and includes a firm or other association of individuals; and
(b) "director", in relation to a firm, means a partner in the firm.

Cognizance of offence

22. (1). No court shall take cognizance of an offence under this Act except on a complaint made by:
(a) the Appropriate Authority concerned, or any officer authorised in this behalf by the Central Government or the State Government or, as the case may be, the Appropriate Authority; or
(b) a person who has given notice of not less than sixty days, in such manner as may be prescribed, to the Appropriate Authority concerned, of the alleged offence and of his intention to make a complaint to the court.

(2). No court other than that of a Metropolitan Magistrate or a Judicial Magistrate of the first class shall try any offence punishable under this Act.

(3). Where a complaint has been made under clause (b) of sub-section (1), the court may, on demand by such person, direct the Appropriate Authority to make available copies of the relevant records in its possession to such person.

Chapter VII
Miscellaneous

Protection of action taken in good faith.

23. (1). No suit, prosecution or other legal proceeding shall lie against any person for anything which is in good faith done or intended to be done in pursuance of the provisions of this Act.

(2). No suit or other legal proceeding shall lie against the Central Government or the State Government for any damage caused or likely to be caused for anything which is in good faith done or intended to be done in pursuance of the provisions of this Act.

Power to make rules.

24. (1). The Central Government may, by notification, make rules for carrying out the purposes of this Act.

(2). In particular, and without prejudice to the generality of the foregoing power, such rules may provide for all or any of the following matters, namely:
(a) the manner in which and the conditions subject to which any donor may authorise removal, before his death, of any human organ of his body under sub-section (1) of section 3;
(b) the form and the manner in which a brain-stem death is to be certified and the conditions and requirements which are to be satisfied for that purpose under sub-section (6) of section 3;
(c) the form and the manner in which any of the parents may give authority, in the case of brain-stem death of a minor, for the removal of any

human organ under sub-section (7) of section 3;

(d) the form in which authority for the removal of any human organ from an unclaimed dead body may be given by the person incharge of the management or control of the hospital or prison, under sub-section (1) of section 5;

(e) the steps to be taken for the preservation of the human organ removed from the body of any person, under section 7;

(f) the form and the manner in which an application may be jointly made by the donor and the recipient under sub-section (5) of section 9;

(g) the manner in which all possible effects, complications and hazards connected with the removal and transplantation is to be explained by the registered medical practitioner to the donor and the recipient under section 12;

(h) the standards as are to be enforced by the Appropriate authority for hospitals engaged in the removal, storage or transplantation of any human organ under clause (iii) of sub-section (3) of section 13;

(i) the other measures as the Appropriate Authority shall undertake in performing its functions under clause (vi) of sub-section (3) of section 13;

(j) the form and the manner in which an application for registration shall be made and the fee which shall be accompanied, under sub-section (2) of section 14;

(k) the specialised services and the facilities to be provided, skilled manpower and the equipments to be possessed and the standards to be maintained by a hospital for registration, under sub-section (3) of section 14;

(l) the form in which, the period for which and the conditions subject to which certificate of registration is to be granted to a hospital, under sub-section (1) of section 15;

(m) the manner in which and the fee on payment of which certificate of registration is to be renewed under sub-section (3) of section 15;

(n) the manner in which an appeal may be preferred under section 17;

(o) the manner in which a person is required to give notice to the Appropriate Authority of the alleged offence and of his intention to make a complaint to the court, under clause (b) of sub-section (1) of section 22; and

(p) any other matter which is required to be, or may be prescribed.

(3). Every rule made under this Act shall be laid, as soon as may be after it is made, before each House of Parliament, while it is in session, for a total period of

thirty days which may be comprised in one session or in two or more successive se ssions, and if, before the expiry of the session immediately following the session or the successive s essions af oresaid, both Houses agree in making any modificat ion in the rule or bot h Houses agree that the rule should not be made, the rule shall thereafter have effect only in such modified form or be of no effect, as the case may be; so, however that any such modification or annulment shall be without prejudice to the validity of anything previously done under that rule.

Repeal and saving
28 of 1982
29 of 1982

25. (1). The Ear Drums and Ear Bones (Authority for Use for Therapeutic Purpos es) Act, 1989 and the Ey es (Authority for Use for Therapeutic Purposes) Act, 1982 are hereby repealed.

(2). The repeal shall, howeve r, not affect the previous operation of the Acts s o repealed or anything duly done or suffered thereunder.

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Part II—Section 2

(Supplement)

NOTIFICATIONS BY GOVERNMENT

HEALTH AND FAMILY WELFARE DEPARTMENT

Amendment to the Transplantation of Human Organs Rules, 1995

[G.O. Ms. No. 179, Health and Family Welfare (Z-1), 18th June 2009, Aani 4, Thiruvalluvar Aandu-2040.]

No. II(2)/HF/358/2009.—The following Amendment Notification of Government of India, Ministry of Health and Family Welfare, dated 4th August 2008 in republished:—

G.S.R. 51 (E).—In exercise of the powers conferred by sub-section (1) of Section 24 of the Transplantation of Human Organs Act, 1994 (42 of 1994), the Central Government hereby makes the following rules, namely:—

1. Short title and Commencement:

(1) These rules may be called the Transplantation of Human Organs Rules, 1995.

(2) They shall come into force on the date of their publication in the Official Gazette.

2. Definitions:

(a) "Act" means the Transplantation of Human Organs Act, 1994 (42 of 1994);

(b) "Form" means a form annexed to these Rules;

(c) "Section" means a section of the Act;

[1](d) "National Accreditation Board for Laboratories" (NABL) means a Board set up by the Quality Council of India (set up by the Government of India) for undertaking assessment and accreditation of testing and calibration of laboratories in accordance with the international standard ISO/IEC 17025 and ISO 15189;

[1]Inserted vide Gazette notification dated 04-08-2008

[2](e) the Registered Medical Practitioner, as defined in clause (n) of section 2 of Transplantation of Human Organs Act, 1994 includes an allopathic doctor with MBBS or equivalent degree under the Medical Council of India Act.

[2]Inserted vide Gazette notification dated 04-08-2008

[3](f) words and expression used and not defined in these Rules, but defined in the Act, shall have the same meanings respectively assigned to them in the Act.

[3]Re-numbered as clause (f) vide Gazette notification dated 4th August 2008. of the earlier clause (d)

3. Authority for Removal of Human Organ:

Any donor may authorize the removal, before his death, of any human organ of his body for therapeutic purposes in the manner and on such conditions as specified in [4]Form 1(A), 1(8) and 1(C).

[4]Amended vide Gazette notification dated 04th August 2008.

4. Duties of the Medical Practitioner:

[5](1) A registered medical practitioner shall, before removing a human organ from the body of a donor before his death, satisfy himself-

Substituted vide Gazette notification dated 04th August 2008.

(a) that the donor has given his authorization in appropriate Form 1 (A) or 1(8) or 1(C).

(b) that the donor is in proper state of health and is fit to donate the organ, and the registered medical practitioner shall sign a certificate as specified in Form 2.

(c) that the donor is a near relative of the recipient, as certified in Form 3, who has signed Form 1 (A) or 1 (B) as applicable to the donor and that the donor has submitted an application in Form 10 jointly' with the recipient and that the proposed donation has been approved by the concerned competent authority and that the necessary documents as prescribed and medical tests, if required, to determine the factum of near relationship, have been examined to the satisfaction of the Registered Medical Practitioner l.e, Incharge of transplant centre.

(d) that in case the recipient is spouse of the donor, the donor has given a statement to the effect that they are so related by signing a certificate in Form 1 (8) and has submitted an .J application in Form 10 jointly with the recipient and that the proposed donation has been ' approved by the concerned competent authority under provisions of sub-rule(2) of rule 4A.

(e) In case of a donor who is other than a near relative and has signed Form 1(C) and submitted an application in Form 10 jointly with the recipient, the permission from the Authorisation Committee for the said donation has been obtained.

(2) A registered medical practitioner shall before removing a human organ from the. body of a person after his death satisfy himself:-

(a) that the donor had, in the presence of two or more witness (at least one of whom is a near relative of such persons) unequivocally authorised as specified in Form 5 before his death, the removal of the human organ of his body, after his death, for therapeutic purposes and there is no reason to believe that the donor had subsequently revoked the authority aforesaid;

[6](b) that then person lawfully in possession of the dead body has signed a certificate as specified in Form 6 [7]"

[6]Substituted vide Gazette notification dated 04th August 2008.

[7]The need for Certificate in form 7 deleted by Gazette Notification, dated 04th August 2008.

(3) A registered medical practitioner shall, before removing a human organ from the body of a person in the event of his brainstem death, satisfy himself:-

(a) that a certificate as specified in Form 8 has been signed by all the members of the Board of medical experts referred to in sub-section (6) of Section 3 of the Act;

(b) that is the case of brain-stem death of a person of less than eighteen years of age, a certificate specified in-:Form 8 has been signed by all the members of the Board of medical experts referred to in sub-section (6) of Section 3 of the Act and an authority as specified in Form 9 has been signed by either of the parents of such person.

[8]4-A Authorisation Committee:

(1) The medical practitioner who will be pali of the organ transplantation team for carrying out transplantation operation shall not be a member of the Authorisation Committee constituted under the provisions of clauses (a) and (b) of sub-section(4) of section 9 of the Act.

(2) Where the proposed transplantation is between a married couple, the Registered Medical Practitioner i.e. Incharge of transplnnt centre must evaluate the factum and duration of marriage and ensure that documents such as marriage certificate, marriage photograph etc. are kept for records along with the information on the number and age of children and family photograph depicting the entire immediate family, birth certificate of children containing particulars of parents.

(3) When the proposed donor or recipient or both are not Indian Nationals/citizens whether 'near relatives' or otherwise, Authorisation Committees shall consider all such requests.

(4) When the proposed donor and the recipient are not 'near relatives', as defined under clause(i) of section 2 of the Act, the Authorisation Committee shall evaluate that,-'

(i) there is no commercial transaction between the recipient and the donor and that no payment or money or moneys worth as referred to the Act, has been made to the donor or promised to be made to the donor or any other person;

(ii) the followilig shall specifically be assessed by the Authorisation Committee:-

(a) an explanation of the link between them and the circumstances which led to the offer being made;

(b) reasons why the donor wishes 'to 'donate;

(c) documentary evidence of the link, e.g. proof that they have lived together, etc.;

(d) old photographs showing the donor and the recipient together;

(iii) that there is no middleman or tout involved;

(iv) that financial status of the donor and the recipient is probed by asking them to give appropriate evidence of their vocation and income for the previous three financial years. Any gross disparity between the status of the two must be evaluated in the "backdrop of the objective of preventing commercial dealing;

(v) that the donor is not a drug addict or known person with criminal record;

(vi) that the next of the kin of the proposed unrelated donor is interviewed regarding awareness about his or her intention to donate an organ, the authenticity

of the link between the donor and the recipient and the reasons for donation. Any strong views or disagreement or objection of such kin shall also be recorded and taken note of.'

[8]Inserted vide Gazette notification dated 04th August 2008.

5. Preservation of Organs

The organ removed shall be preserved according to current and accepted scientific methods in order to ensure viability for the purpose of transplantation.

[9]6. The donor and the recipient shall make jointly an application to grant approval for removal and transplantation of a human organ, to the concerned competent authority or Authorisation Committee as specified in Form 10. The Authorisation Committee shall take a decision on such application in accordance with the guidelines in rule 6-A."

[9]Substituted vide Gazette notification dated 04th August 2008.

[10]6(A) Composotion of Authorisation Committees.

1. There shall be one State level Authorisation Committee.

2. Additional authorisation committees may be set up at various levels as per norms given below, namely:—

(i) no member from transplant team of the institution should be a member of the respective Authorisation Committee. All Foreign Nationals (related and unrelated) should go to [6]Authorisation Committee' as abundant precaution needs to be taken in such cases;

(ii) Authorisation Committee should, be Hospital based in Metro and big Cities if the number of transplants exceed 25 in a year at the respective transplantation centres. In smaller towns, there are State or District level Committees if transplants are less than 25 in a year in the respective districts.

(A) Composition of Hospital Based Authorisation Committees: (To be constituted by the State Government and in case of Union territory by the Central Government).

(a) the senior most person officiating as Medical Director or Medical Superintendent of the Hospital;

(b) two senior medical practitioners from the same hospital who are not part of the transplant team;

(c) two members being persons of high integrity, social standing and credibility, who have served in high ranking Government positions, such as in higher judiciary, senior cadre of police service or who have served as a reader or professor in University Grants Commission approved University or are self-employed professionals of repute such as lawyers, chartered accountants and doctors (of Indian Medical Association) etc.; and.

(d) Secretary (Health) or nominee and Director Health Services or nominee.

(B) Composition of State or District Level Authorisation Committees: (To be constituted by the State Government and in case of Union, territory by the Central Government).

(a) a Medical Practitioner officiating as Chief Medical Officer or any other equivalent post in the main/major Government Hospital of the District.

(b) two senior medical practitioners to be chosen from the pool of such medical practitioners who are residing in the concerned District and who are not part of any transplant team.

(c) two senior citizens, non-medical background (one lady) of high reputation and integrity to be chosen from the pool of such citizens residing in the same district, who have served in high ranking Government positions, such as in higher judiciary. senior cadre of police service or who have served as a reader or professor in University Grants Commission approved University or are self-employed professionals of repute such as lawyers, chartered accountants and doctors (of Indian Medical Association) etc., and

(d) Secretary (Health) or nominee and Director Health Services or nominee.

(NOTE: Effort should be made to have most of the members' ex-officio so that the need to change the composition of committee is less frequent.)

[10]Inserted vide Gazette notification dated 04th August 2008.

[11]6B. The State level committees shall be formed for the purpose of providing approval or no objection certificate to the respective donor and recipient to establish the legal and residential status as a domicile state. It is mandatory that if donor, recipient and place of transplantation are from different states, then the approval or 'no objection certificate' from the respective domicile State Government should be necessary. The institution where the transplant is to be undertaken in such case the approval of Authorisation Committee is mandatory.

[11]Inserted vide Gazette notification dated 04th August 2008.

[12]6C. The quorum of the Authorisation Committee should be minimum four. However, quorum ought not to be considered as complete without the participation of the Chairman. The presence of Secretary (Health) or nominee and Director of Health Services or nominee is mandatory.

[12]Inserted vide Gazette notification dated 04th August 2008.

[13]6D. The format of the Authorisation Committee approval should be uniform in all the institutions in a State. The format may be notified by respective State Government.

[13]Inserted vide Gazette notification dated 04th August 2008.

[14]6E. Secretariat of the Committee shall circulate copies of all applications received from the proposed donors to all members of the Committee. Such applications should be circulated along with all annexure, which may have been filed along with the applications. it the title of the meating, the Authorisation Committee should take note of all relevant

contents and documents in the course of its decision making process and in the event any document or information is found to be inadequate or doubtful, explanation should be sought from the applicant and if it is considered necessary that any fact or information requires to be verified in order to confirm its veracity or correctness, the same be ascertained through the concerned officer(s) of the State! Union territory Government.

[14]Inserted vide Gazette notification dated 04th August 2008.

[15]6F. The Authorisation Committee shall focus its attention on the following, namely:—

[15]Inserted vide Gazette notification dated 04th August 2008.

(a) Where the proposed transplant is between persons related genetically, Mother, Father, Brother, Sister, Son or Daughter (above the age of 18 years).

the concerned competent authority shall evaluate:—

(i) results of tissue typing and other basic tests;

(ii) documentary evidence of relationship e.g. relevant birth certificates and marriage certificate, certificate from Sub-divisional magistrate/ Metropolitan Magistrate/or Sarpanch of the Panchayat;

(iii) documentary evidence of identity and residence of the proposed donor e.g. Ration Card or Voters identity Card or Passport or Driving License or PAN Card or Bank Account and family photograph depicting the proposed donor and the proposed recipient along with another near relative;

(iv) if in its opinion, the relationship is not conclusively established after evaluating the above evidence, it may in its discretion direct further medical tests as prescribed as below:

(a) the tests for Human Leukocyte Antigen (HLA), Human Leukocyte Antigen- B alleles to be performed by the serological and/or Polymerase chain reaction (PCR) based Deoxyribonucleic acid (DNA) methods.

(b) test for Human Leukocyte Antigen-DR beta genes to be performed using the Polymerase chain reaction (PCR) based Deoxyribonucleic acid (DNA) methods. .

(c) the tests referred to in sub-rules (i) and (ii) shall be got done from a laboratory accredited with National Accreditation Board for Laboratories (NABL).”

(d) where the tests referred to in (i) to (iii) above do not establish a genetic relationship between the donor and the recipient, the same tests to be performed on both or at least one parent, preferably both parents. If parents are not available. same tests to be performed on such relatives of donor and recipient as are available and are willing to be tested failing which, genetic relationship between the donor and the recipient will be deemed to have not been established.

(b) The papers for approval of transplantation would be processed by the registered medical practitioner and administrative division of the Institution for transplantation, while the approval will be granted by the Authorisation Committee.

(c) Where the proposed transplant is between a married couple (except foreigners, whose cases should be dealt by Authorisation Committee):

The concerned competent authority or authorisation committee as the case may be must evaluate all available evidence to establish the factum and duration of marriage and ensure that documents such as marriage certificate, marriage photograph is placed before the committee along with the information on the number and age of children and a family photograph depicting the entire immediate family, birth certificate of children containing the particulars of parents.

(d) Where the proposed transplant is between individuals who are not “near relatives”, The authorization committee shall evaluate:—

(i) that there is no commercial transaction between the recipient and the donor. That no payment of money or moneys worth as referred to in the sections of the Act, has been made to the donor or promised to be made to the donor or any other person. In this connection the Authorisation Committee shall take into consideration:—

(a) an explanation of the link between them and the circumstances which led to the offer being made;

(b) documentary evidence of the link e.g. proof that they have lived together etc.;

(c) reasons why the donor wishes to donate; and

(d) old photographs showing the donor and the recipient together.

(ii) that there is no middleman/tout involved;

(iii) that financial status of the donor and the recipient is probed by asking them to give appropriate evidence of their vocation and income for the previous three financial years. Any gross disparity between the status of the two, must be evaluated in the backdrop of the objective of preventing commercial dealing;

(iv) that the donor is not a drug addict or a known person with criminal record;

(v) that the next of kin of the proposed unrelated donor is interviewed regarding awareness about his/her intention to donate an organ, the authenticity of the link between the donor and the recipient and the reasons for donation. Any strong views or disagreement or objection of such kin may also be recorded and taken note of;

and

(e) When the proposed donor or the recipient or both are foreigners:—

(i) a senior Embassy official of the country of origin has to certify the relationship between the donor and the recipient.

(ii) Authorisation Committee shall examine the cases of Indian donors consenting to donate organs to a foreign national (who is a near relative), including a foreign national of Indian origin, with greater caution. Such cases should be considered rarely on case to case basis.

(f) In the course, of determining eligibility of the applicant to donate, the applicant should be personally interviewed by the Authorisation Committee and minutes of the interview should be recorded. Such interviews with the donors should be videographed.

(g) In case where the donor is a woman greater precautions ought to be taken. Her identity and independent consent should be confirmed by a person other than the recipient. Any document with regard to the proof of residence or domicile and particulars of parentage should be relatable to the photo identity of the applicant in order to ensure that the documents pertain to the same person, who is the proposed donor and in the event of any inadequate or doubtful information to this effect, the Authorisation Committee may in its discretion seek such other information or evidence as may be expedient and desirable in the peculiar facts of the case.

(h) The Authorisation Committee should state in writing its reason for rejecting approving the application of the proposed donor and all approvals should be subject to the following conditions:—

(i) that the approved proposed donor would be subjected to all such medical tests as required at the relevant stages to determine his biological capacity and compatibility to donate the organ in question.

(ii) further that the psychiatrist clearance would also be mandatory to certify his mental condition, awareness, absence of any overt or latent psychiatric disease and ability to give free consent.

(iii) all prescribed forms have been and would be filled up by all relevant persons involved in the process of transplantation.

(iv) all interviews to be video recorded.

(i) The authorisation committee shall expedite its decision making process and use its discretion judiciously and pragmatically in all such cases where the patient requires immediate transplantation.

(j) Every authorised transplantation centre must have its own website. The Authorisation Committee is required to take final decision within 24 hours of holding the meeting for grant of permission or rejection for transplant. The decision of the Authorisatlon Committee should be displayed on the notice board of the hospital or Institution immediately and should reflect on the website of the hospital or Institution within 24 hours of taking the decision. Apart from this, the website of the hospital or institution must update its website regularly in respect of the total number of the transplantations done in that hospital or institution along with the details of each transplantation. The same data should be accessible for compilation, analysis and further use by respective State Governments and Central Government.

[15]Inserted vide Gazette notification dated 04th August 2008.

7. Registration of Hospital

(1) An application for registration shall be made to the Appropriate Authority as specified in Form 11. The application shall be accompanied by a fee of rupees one thousand payable to the Appropriate Authority by means of a bank draft or postal order.

(2) The Appropriate Authority shall, after holding an inquiry and after satisfying itself that the applicant has complied with all the requirements, grant a certificate of registration as specified in Form 12 and shall be valid for a period of five years from the date of its issue and shall be renewable.

[16](3) before a hospital is registered under the provisions of this rule, it shall be mandatory for the hospital to nominate a transplant coordinator.

[16] Inserted vide Gazette notification dated 4th August 2008.

8. Renewal of Registration

(1) An application for the renewal of a certificate of registration shall be made to the Appropriate Authority within a period of three months prior to the date of expiry of the original certificate of registration and shall be accompanied by a fee of rupees five hundred payable to the Appropriate Authority by means of a bank draft or postal order.

(2) A renewal certificate of registration shall be as specified in Form 13 and shall be valid for a period of five years.

(3) If, after an inquiry including inspection of the hospital and scrutiny of its past performance and after giving an opportunity to the applicant, the Appropriate Authority is satisfied that the applicant, Since grant of certificate of registration under sub-rule (2) of Rule 7 has not complied with the requirements of this Act and Rules made there under and conditions subject to which the certificate of registration has been granted, shall, for reasons to be recorded in writing, refuse to grant renewal of the certificate of registration.

[17]9 Conditions for Grant of Certificate of Registration

No hospital shall be granted a certificate of registration under this Act unless it fulfils the following requirement of manpower, equipment, specialized services and facilities as laid down below:—

A General Manpower Requirement Specialised Services and Facilities:

(1) 24 hours availability of medical and surgical, (senior and junior) staff.

(2) 24 hours availability of nursing staff, (general and speciality trained).

(3) 24 hours availability of Intensive Care Units with adequate equipments, staff and support system, including specialists in anaesthesiology, intensive care.

(4) 24 hours availability of laboratory with multiple'discipline testing facilities including but not limited to Microbiology, Bio-Chemistry, Pathology and Hematology and Radiology departments with trained staff.

(5) 24 hours availability of Operation Theater facilities (OT facilities) for planned and emergency procedures with adequate staff, support system and equipments.

(6) 24 hours availability of communication system, with power backup, including but not limited to multiple line telephones, public telephone systems, fax, computers and paper photo-imaging machine.

(7) Experts (Other than the experts required for the relevant transplantation) of relevant and associated specialties including but not limited to and depending upon the requirements, the experts in internal medicine, diabetology, gastroenterology, nephrology, neurology, paediatrics, gynaecology immunology and cardiology etc. should be available to the transplantation centre.

B Equipments:

Equipments as per current and expected scientific requirements specific to organ or organs being transplanted. The transplant centre should ensure the availability of the accessories, spare-parts and back-up/maintenance/service support system in relation to all relevant equipments.

C Experts and their qualifications:

(A) Kidney Transplantation:

M.S. (Gen.) Surgery or equivalent qualification with three years post M.S. training in a recognised center in India or abroad and having attended to adequate number of renal transplantation as an active member of team.

(B) Transplantation of liver and other abdominal organs

M.S. (Gen.) Surgery or equivalent qualification with adequate post M.S. training in an established center with a reasonable experience of performing liver transplantation as an active member of team.

(C) Cardiac, Pulmonary, Cardio-Pulmonary Transplantation:

M.Ch. Cardio-thoracic and vascular surgery or equivalent qualification in India or, abroad with at least 3 years experience as an active member of the team performing an adequate number of open heart operations per year and well-versed with Coronary by-pass surgery and Heart-valve surgery.

(D) Cornea Transplantation:

M.D./M.S. Ophthalmology or equivalant qualification with one year post M.D./M.S. training in a recognised hospital carrying out Corneal transplant operations.

[17] Substituted vide Gazette notification dated 4th August 2008.

10. Appeal

(1) Any person aggrieved by an order of the Authorisation Committee under sub- section (6) of Section 9 or by an order of the Appropriate Authority under sub- section (2) of Section 15 and Section 16 of the Act, may, within thirty days from the date of receipt of the order, prefer an appeal to the Central Government.

(2) Every appeal shall be in writing and shall be accompanied by a copy of the order appealed against.

[F.No. S. 12011/12/2007-MS]

VINEET CHAWDHRY,

Joint Secretary to the Govt. of India.

1. Principal rules were published in the Gazette of India notification No: S-12011/2/1994-MS dated the 4th February, 1995 Extraordinary, under G.S.R.No, 51 (E).

2. Amendment to the rules were published in the Gazette of India notification, No: S.1201/12/2007-MS dated 31-7-2008 Extraordinary, under G.S.R. 571 (E) dated 4th August, 2008.

[18]FORM 1(A)

(Page 1 of 2)

(To be completed by the prospective related donor)

(See Rule 3)

My full name is..... .....
and this is my photograph

Photograph of the Donor
(Attested by Notary Public)

To be affixed and attested by Notary Public after it is affixed.

My permanent home address is

.....

.....Tel: ....

My present home address is

.....

.....Tel: ....

Date of birth ....(day/month/year)

* Ration/Consumer Card number and Date of issue & place..... ....
(Photocopy attached)

and/or

* Voter's I-Card number, date of issue, Assembly constituency..... .....
(Photocopy attached)

and/or

* Passport number and country of issue ....
(Photocopy attached)

and/or

* Driving Licence number, Date of issue, licensing authority..... .....
(Photocopy attached)

and/or

* PAN.....

and/or

* Other proof of identity and address..... .....

I hereby authorize removal for therapeutic purposes/consent to donate my.....(state w hich organ) to my relative .....(specify son/daughter/father/mother/brother/s ister), whose name is .....and who was born on.....(da y/month/year) and whose particulars are as follows:

[18]Form 1A inserted vide Gazette notification dated 4th August 2008

Photograph of the Recipient
(Attested by Notary Public)

To be affixed and attested by Notary Public after it is affixed.

* Ration/Consumer Card number and Date of issue & place: ……
(Photocopy attached)

and/or

* Voter's I-Card number, date of issue, Assembly constituency ……
(Photocopy attached)

and/or

* Passport number and country of issue .....
(Photocopy attached)

and/or

* Driving Licence number Date of issue, licensing authority .....
(Photocopy attached)

and/or

* PAN.....

and/or

* Other proof of identity and address .....

I solemnly affirm and declare that:

Sections 2,9 and 19 of The Transplantation of Human Organs Act 1994 have been explained to me and I confirm that:

1. I understand the nature of criminal offences referred to in the sections.
2. No payment of money or money's worth as referred to in the sections of the Act has been made to me or will be made to me or any other person.
3. I am giving the consent and authorisation to remove my..... (organ) of my own free will without any undue pressure, inducement, influence or allurement.
4. I have been given a full explanation of the nature of the medical procedure involved and the risks involved for me in the removal of my……(organ). That explanation was given by .....(name of registered medical practitioner).
5. I under the nature of that medical procedure and of the risks to me as explained by that practitioner.
6. I understand that I may withdraw my consent to the removal of that organ at any time before the operation takes place.
7. I state that particulars filled by me in the form are true and correct to my knowledge and nothing material has been concealed by me.

....
Signature of the prospective donor Date

NOTE:—To be sworn before Notary public, who while attesting shall ensure that the person/persons swearing the affidavit(s) Signs(s) on the Notary Register, as well.

* √ wherever applicable.

[19]FORM 1(B)

(To be completed by the prospective spousal donor)

(See Rule 3)

My full name is..... .....
and this is my photograph

Photograph of the Donor
(Attested by Notary Public)

To be affixed and attested by Notary Public after it is affixed.

My permanent home address is

.....

.....Tel: .....

My present home address is .....

.....

.....Tel: .....

Date of birth ....(day/month/year)

I authorize to remove for therapeutic purposes/consent to donate my.....(state which organ) to my husband/wife.....whose full name is..... .....and who was born on.....(day/month/year) and whose particulars a re as follows:

Photograph of the Recipient
(Attested by Notary Public)

To be affixed and attested by Notary Public after it is affixed.

* Ration/Consumer Card number and Date of issue & place..... ...
(Photocopy attached)

and/or

* Voter's I-Card number, date of issue, Assembly Constituency..... .....
(Photocopy attached)

and/or

* Passport number and country of issue ....
(Photocopy attached)

and/or

* Driving Licence number, Date of issue, licensing authority..... .....
(Photocopy attached)

and/or

* PAN.....

and/or

* Other proof of identity and address..... .....

[19]Form 1B inserted vide Gazette notification, dated 4th August 2008

I submit the following as evidence of being married to the recipient:-

(a) A certified copy of a marriage certificate

OR

(b) An affidavit of a 'near relative' confirming the status of marriage to be sworn before Class-I Magistrate/Notary Public.

(c) Family photographs

(d) Letter from member of Gram Panchayat/Tehsildar/Block Development Officer/MLA/MP certifying factum and status of marriage.

OR

(e) Other credible evidence

I solemnly affirm and declare that:

Sections 2,9 and 19 of The Transplantation of Human Organs Act, 1994 have been explained to me and I confirm that:

1. I understand the nature of criminal offences referred to in the sections.
2. No payment of money or money's worth as referred to in the Sections of the Act has been made to me or will be made to me or any other person.
3. I am giving the consent and authorisation to remove my..... (organ) of my own free will without any undue pressure, inducement, influence or allurement.
4. I have been given a full explanation of the nature of the medical procedure involved and the risks involved for me in the removal of my.....(organ). That explanation was given by ....(name of registered medical practitioner).
5. I under the nature of that medical procedure and of the risks to me as explained by that practitioner.
6. I understand that I may withdraw my consent to the removal of that organ at any time before the operation takes place.
7. I state that particulars filled by me in the form are true and correct to my knowledge and nothing material has been concealed by me.

....

Signature of the prospective donor Date

Note:—To be sworn before Notary Public, who while attesting shall ensure that the person/persons swearing the affidavit(s) signs(s) on the Notary Register, as well.

* √ wherever applicable.

[20]FORM 1(C)

(To be completed by the prospective un-related donor)

(See Rule 3)

My full name is..... .....
and this is my photograph

Photograph of the Donor
(Attested by Notary Public)

To be affixed and attested by Notary Public after it is affixed.

My permanent home address is..... .....

.....Tel: ……

My present home address is

.....

.....Tel: ....

Date of birth ....(day/month/year)

* Ration/Consumer Card number and Date of issue & place:..... ....
(Photocopy attached)

and/or

* Voter's I-Card number, date of issue, Assembly Constituency..... .....
(Photocopy attached)

and/or

* Passport number and country of issue .....
(Photocopy attached)

and/or

* Driving Licence number, Date of issue, licensing authority..... .....
(Photocopy attached)

and/or

* PAN.....

and/or

* Other proof of identity and address..... .....

Details of last three years income and vocation of donor..... .....

.....

.....

[20] Form 1C inserted vide Gazette notification, dated 4th August 2008

I hereby authorize to remove for therapeutic purposes/consent to donate my.....(state which organ) to a person whose fully name is ..... and who was born on

.....(day/month/year) and whose particulars are as follows:

Photograph of the Recipient
(Attested by Notary Public)

To be affixed and attested by Notary Public after it is affixed.

* Ration/Consumer Card number and Date of issue & place:..... .....
(Photocopy attached)

and/or

* Voter's I-Card number, date of issue, Assembly Constituency..... .....
(Photocopy attached)

and/or

* Passport number and country of issue..... .....
(Photocopy attached)

and/or

* Driving Licence number Date of issue, licensing authority..... .....
(Photocopy attached)

and/or

* PAN.....

and/or

* Other proof of identity and address..... .....

I solemnly affirm and declare that:

Sections 2, 9 and 19 of The Transplantation of Human Organs Act, 1994 have been explained to me and I confirm that:

1. I understand the nature of criminal offences referred to in the Sections.
2. No payment of money or money's worth as referred to in the Sections of the Act has been made to me or will be made to me or any other person.
3. I am giving the consent and authorisation to remove my..... (organ) of my own free will without any undue pressure, inducement, influence or allurement.
4. I have been given a full explanation of the nature of the medical procedure involved and the risks involved for me in the removal of my.....(organ). That explanation was given by ....(name of registered medical practitioner).
5. I under the nature of that medical procedure and of the risks to me as explained by that practitioner.
6. I understand that I may withdraw my consent to the removal of that organ at any time before the operation takes place.
7. I state that particulars filled by me in the form are true and correct to my knowledge and nothing material has been concealed by me.

....
Signature of the prospective donor Date

Note:—To be sworn before Notary Public, who while attesting shall ensure that the person/persons swearing the affidavit(s) signs(s) on the Notary Register, as well.

* √ wherever applicable.

[21]FORM 2

[*See* rule 4(1) (b)]

(To be completed by the concerned Medical Practitioner)

I, Dr..... possessing qualification of..... .....registered as medical practitioner at serial no .....by the..... .....Medical Council, certify that I have examined Shri / Smt./Km. .....S/o, D/o., W/o Shri..... ..... aged .....who has given informed consent about donation of the organ, namely (name of the organ).....to Shri/Smt./Km.. ....who is a 'near relative', of the donor/other than near relative of the donor; who had been approved by the Authorisation Committee/Registered Medical Practitioner i.e. Incharge of transplant centre (as the case may be) and that the said donor is in proper state of health and is medically fit to be subjected to the procedure of organ removal.

Place: .....

.....
Signature of Doctor

Date: .....

Seal

To be affixed (pasted) and attested by the doctor concerned.

The signatures and seal should partially appear on photograph and document without disfiguring the face in photograph.

Photograph of the Donor
(Attested by doctor)

To be affixed (pasted) and attested by the doctor concerned.

The signatures and seal should partially appear on photograph and document without disfiguring the face in photograph.

Photograph of the recipient
(Attested by the doctor)

[21] Form 2 substituted vide Gazette notification dated 4th August 2008

[22] FORM 3

[See Rule 4(1) (C)]

I, Dr./Mr./Mrs..... working as..... ..... at .....and possessing qualification of..... .....certify that Shri/ Smt./ Km.....S/o, D/o, W/o Shri/Smt. .... aged.....the donor and Shri/Smt..... ..... S/o, D/ o, W/o. Shri/Smt.....aged.....the proposed rec ipient of the o rgan t o b e d onated by the said donor are related to each other as brother/sister/mother/father/son/daughter as per their statement and the fact of this relationship has been established not established by the results of the tests for Antigenic Products of the Human Major Histocompatibility Complex. The results of the tests are attached.

Place .....

Signature

Date .....

(To be signed by the Head of the Laboratory)

Seal

---

[22] Form 3 substituted vide Gazette notification dated 4th August 2008

FORM 4

[See Rule 4(1) (d)]

I, Dr./Mr./Mrs..... .....possessing qualification of .....registered as medical practitioner at Serial No.. .....by the.....Medical Council, c ertify that:—

(i) Mr.....S/o..... ..... aged.....resident of..... and Mrs ..... D/o, W/o ..... aged ..... res ident of ..... are related to each other as spouse according to the statement given by them and their statement has been confirmed by means of following evidence before effecting the organ removal from the body of the said Shri/Smt/ Km.....(Applicable only in the cases where considered necessary).

OR

(ii) The Clinical condition of Shri/Smt ..... mentioned above is such that recording of his /her statement is not practicable.

Plalce.....

Signature of Regd. Medical Practitioner

Date.....

FORM 5

[See Rule 4(2)(a)]

I.....S/o,D/o,W/o.....
.....aged.....resident of..... .....in the present of persons mentioned below hereby unequivocally authorise the removal of my organ/organs, namely,.....

Signature of Donor

(Signature) Dated:

1.Shri/Smt./Km.....S/o,D/o,W/o.....
..... aged.....resident of.....

(Signature)

2.Shri/Smt./Km.....S/o,D/o,W/o..... aged.....
resident of.....is a near relative to the donor as..... .....

Dated.....

FORM 6

[See Rule 4(2)(b)]

I.....S/o,D/o,W/o.....
aged.....resident of.....having lawful posses sion of the dead body of Shri/ Smt./Km.....S/o.D/o.W/o.....aged ..... resident of.....
having known that the deceased has not expressed any objection to relative of the said deceased person has objection to any of his/her organs being used for therapeutic purposes authorise removal of his/her body organs, namely.....

Dated .....
Place .....

Signature

Person in lawful possession of the dead body.
Address.....
.....

[23]FORM 7

FORM 8

[See Rules 4(3)(a) and (b)]

We, the following members of the Board of medical experts after careful personal examination hereby certify that Shri/Smt./Km.....Aged about ..... son of/wife of/daughter of..... Res ident of..... is dead on account of permanent and irreversible cessation of all functions of the brain-stem. The tests carried out by us and the findings therein are recorded in the bmin-stem death Certificate annexed hereto.

Dated..... Signature.....

1. R.M.P.—Incharge of the Hospital In which brain-stem death has occurred. Appropriate
2. R.M.P. nominated from the panel of Names approved by the Authority.
3. Neurologist/Neuro-Surgeon nominated deceased from the panel of names approved by the Appropriate Authority.
4. R.M.P. treating the aforesaid person.

BRAIN-STEM DEATH CERTIFICATE.

(A) PATIENT DETAS.....

1. Name of the patient: Mr./Ms.....
   S.O./D.O./W.O. Mr./Ms.....
   Sex.....Age.....
2. Home Address: .....
   .....
   .....
3. Hospital Number: .....
4. Name and Address of next of kin or person responsible for the patient (if none exists, this must be specified) .....
   .....
   .....
5. Has the patient or next of kin agreed To any transplant? .....
   .....
6. Is this a Police Case? Yes.....No.....

(B) PRE-CONDITIONS:

1. Diagnosis: Did the patient suffer from any illness or accident that led to irreversible brain damage? Specify details.....
.....
.....

Date and time of accident/onset of illness..... .....

Date and onset of non-responsible coma..... .....

2. Findings of Board of Medical Experts:

(1) The following reversible causes of coma have been excluded: Intoxication (Alcohol)

[23]Form 7 deleted vide Gazette notification dated 04.08.2008

Depressant Drugs

Relaxants (Neuromuscular blocking agents)

Examination	*First Medical Examination*		*Second Medical Examination*	
	1st	*2nd*	*1st*	*2nd*
Primary Hypothermia				
Hypovolaemic shock				
Metabolic or endocrine disorders				
Tests for absence of brain-stem functions				

(2) Coma

(3) Cessation of spontaneous breathing

(4) Pupillary size

(5) Pupillary light reflexes

(6) Doll's head eye movements

(7) Corneal reflexes (Both sizes)

(8) Motor response in any cranial nerve distribution, any responses to stimulation of face, limb or trunk.

(9) Gag reflex

(10) Cough (Tracheal)

(11) Eye movements on coloric testing bilaterally.

(12) Apnoea tests as specified.

(13) Were any respiratory movements seen?

Date and time of first testing: ....

Date and time of second testing: ....

This is to certify that the patient has been carefully examined twice after an interval of about six hours and on the basis of findings recorded above, Mr./Ms.....is declared brain-stem dead.

1. Medical Administrator Incharge of the hospital
2. Authorised specialist.
3. Neurologis/Neuro-Surgeon
4. Medical Officer treating the Patient.

NB. (I) The minimum time interval between the first testing will be six hours.

(II) No. 2 and No. 3 will be co-opted by the Administrator Incharge of the hospital from the Panel of experts approved by the appropriate authority.

FORM 9

[See Rule 4 (3) (b)]

I, Mr./Mrs.....son of/wife of..... .....Resident of ..... hereby authorise removal of the organ/organs, namely,..... .....for therapeutic purpose from the dead body of my son/daughter Mr/Mrs..... ..... aged.....whose brain-stem death has been duly certified in accordance wit h the law.

Signature .....

Name .....

Place .....

Date .....

[24]FORM 10

(*Page 1 of 2*)

APPLICATION FOR APPROVAL FOR TRANSPLANTATION (LIVE DONOR)

(To be completed by the proposed recipient and the proposed donor)

[See Rule 4 (1) (c)(d)(e)].

To be self attested across the affixed photograph.	To be self attested across the affixed photograph.
Photograph of the Donor (Self-attested)	Photograph of the recipient (Self-attested)

Whereas I ..... S/o, D/o, W/o Shri/Smt..... .....aged ..... residing at..... .....have been advised by my doctor..... .....that I am suffering from.....and may be benefited by transplantation of .....into my body.

And whereas I ..... S/o, D/o, W/o. Shri/Smt..... .....aged ..... residing at..... .....by the following reason(s):—

(a) by virtue of being a near relative i.e.....

[24] Form 10 substituted vide Gazette notification dated 04-08-2008

(b) by reason of affection/lattachment/other special reason as explained below :—

.....

.....

.....

I would therefore like to donate my (name of the organ)..... .....to Shri/Smt.....

We.....and.....
(Donor) (Recipient)

hereby apply to Authorization Committee for permission for such transplantation to be carried out.

We solemnly affirm that the above decision has been taken without any undue pressure, inducement, influence or allurement and that all possible consequences and options of organ transplantation have been explained to us.

Instructions for the applicants:—

1. Form 10 must be submitted along with the completed Form 1 (A), or Form 1 (B) or Form 1 (C) as may be applicable.
2. The applicable Form i.e., Form 1 (A) or Form 1 (B) or Form 1 (C) as the case may be, should be accompanied with all documents mentioned in the applicable form and all relevant queries set out in the applicable form must be adequately answered.
3. Completed Form 3 to be submitted along with the laboratory report.
4. The doctor's advice recommending transplantation must be enclosed with the application.
5. In addition to above, in case the proposed transplant is between unrelated persons, appropriate evidence of vocation and income of the donor as well as the recipient for the last three years must be enclo sed with this application. It is clarified that the evidence of income does not necessarily mean the proof of income tax returns, keeping in view that the applicant(s) in a given case may not be filing income tax returns.
6. The application shall be accepted for consideration by the Authorisation Committee only if it is complete in all respects and any omission of the documents or the information required in the forms mentioned above, shall render the application incomplete.
7. As per the Supreme Court's judgement dated 31-03-2005, the approval/No Objection Certificate from the concerned State/Union Territory Government or Authorisation Committees is mandatory from the domicile State/Union Territory of donor as well as recipient. It is understood that final approval for transplantation should be granted by the Authorisation Committee/Registered Medical Practitioner i.e., Incharge of transplant centre (as the case may be) where transplantation should be done.

We have read and understood the above instructions.

Signature of the Prospective Donor	Signature of Prospective Recipient
Date:	Date:
Place:	Place:

FORM 11

APPLICATION FOR REGISTRATION OF HOSPITAL TO CARRY OUT ORGAN TRANSPLANTATION

To

The Appropriate Authority for organ transplantation
(State of Union Territory)

We hereby apply to be recognised as an institution to carry out organ transplantation. The required date about the facilities available in the hospital are as follows:

(A) Hospital:

1. Name:
2. Location:
3. Government/Private:
4. Teaching/Non-teaching:
5. Approached by:

Road:	Yes	No
Rail:	Yes	No
Air:	Yes	No

6. Total bed strength:
7. Name of the disciplines in the hospital:
8. Annual budget:
9. Patient turn-over/year:

(B) Surgical Team:

1. No. of beds:
2. No. of permanent staff members with their designation:
3. No. of temporary staff with their designation:
4. No. of operations done per year:
5. Trained persons available for transplantation (Please specify Organ for transplantation):

(C) Medical Team:

1. No. of beds:
2. No. of permanent staff members with their designation:
3. No. of temporary staff members with their designation:
4. Patient turnover per year:
5. No. of potential transplant candidates admitted per year:

(D) Anaesthesiology:

1. No. of permanent staff members with their designations:
2. No. of temporary staff members with their designations:
3. Name and No. of operations performed:
4. Name and No. of equipments available:
5. Total No. of operation theatres in the hospital:
6. No. of emergency operation-theatres:
7. No. of separate transplant operation theatre:

(E) I.C.U./H.D.U. Facilities:

1. I.C.U./H.D.U. facilities: Present.....Not present..... .....
2. No. of I.C.U. beds:
3. Trained:—

   Nurses:

   Technicians:
4. Name and member of equipments in I.C.U.

(F) Other Supportive Facilities :

Data about facilities available in the hospital:

(G) Laboratory Facilities :

1. No. of permanent staff with their designations:
2. No. of temporary staff with their designations:
3. Names of the investigations carried out in the Dept.:
4. Name and number of equipments available:

(H) Imaging Services:

1. No. of permanent staff with their designations:
2. No. of temporary staff with their designations:
3. Names of the investigations carried out in the Dept.:
4. Name and number of equipments available: .

(I) Haematology Services :

1. No. of permanent staff with their designations:
2. No. of temporary staff with their designations:
3. Names of the investigations carried out in the Dept.
4. Name and number of equipments aviailable:

(J) Blood Bank Facilities : Yes ..... No .....

(K) Dialysis Facilities : Yes ..... No .....

(L) Other Personnel :

1. Nephrologist	Yes/No
2. Neurologist	Yes/No
3. Neuro-Surgeon	Yes/No
4. Urologist	Yes/No
5. G.I. Surgeon	Yes/No
6. Paediatrician	Yes/No
7. Physiotherapist	Yes/No
8. Social Worker	Yes/No
9. Immunologists	Yes/No
10. Cardiologist	Yes/No

The above said information is true to the best of my knowledge and I have no objection to any scrutiny of our facility by authorised personnel. A Bank Daft/cheque of Rs. 1,000/- is being enclosed.

Sd/-

HEAD OF THE INSTITUTION

FORM 12

CERTIFICATE OF REGISTRATION

This is to certify that ..... Hospital located at ..... has been inspected by the Appropriate Authority and certificate of registration is granted for performing the organ transplantation of the following organs:—

1. .....

2. .....

3. .....

4. .....

4. This certificate of registration is valid for a period of five years from the date of issue.

Signature ..... Signature.....

FORM-13

[*See* sub-rule 8 (2)]

OFFICE OF THE APPROPRIATE AUTHORITY

This is with reference to the application dated .....From..... (Name of the hospital) for renewal of certificate of registration for performing organ transplantation, under the Act.

After having considered the facilities and standards of the above-said hospital, the Appropriate Authority hereby renews the certificate of registration of the said hospital for the purpose of performing organ transplantation for a period of five years.

Appropriate Authority .....

Place .....

Date .....

V.K. SUBBURAJ,
Principal Secretary to Government.

PRINTED AND PUBLISHED BY THE DIRECTOR OF STATIONERY AND PRINTING, CHENNAI
ON BEHALF OF THE GOVERNMENT OF TAMIL NADU

Abstract

Brain death – Declaration of brain death made mandatory in Government Medical College Hospitals in Chennai – Procedure for declaration of brain death – orders issued.

Health and Family Welfare (Z1) Department

(Thiruvallur Aandu - 2039
Sarvajith, Masi Matham -20)

G.O. (Ms) No. 75 Dated : 03.03.2008
Read :

G.O. (Ms) No. 6, Health and Family Welfare Department Dated 8.1.2008

Order:-

In the Government Order read above, declaration of brain death has been made mandatory in Government Medical College Hospitals in Chennai. In para 5 of the above order, it was decided that the procedures to be followed for declaring brain death and the personnel authorised to certify the same would be issued separately.

2.Whereas the Transplantation of Human Organs Act of 1994 (THO Act) and the Transplantation of Human Organs Rules, 1995 (THO Rules) are the only pieces of legislation available wherein brain death certification procedures have been elaborately laid down, it is hereby decided that the procedures outlined therein will also be adopted as brain death certification procedure in Tamilnadu. This order will also elaborate on the above format to ensure its applicability to the state.

3. Form 8 of the THO Act and Rules as found in the Annexure-I to this order are prescribed as the brain death certification format to be utilised for any given situation requiring certification that a person is dead on account of permanent and irreversible cessation of all functions of the brain stem. The tests prescribed therein and the findings required shall remain the same.

4. According to Form 8 of the said Act and Rules, when such certification is required, there shall be two medical examinations conducted by a team of doctors after a minimum interval of six hours and the findings made based on the tests prescribed therein.

5. One aspect of the above form requires further clarification and this is provided in Annexure-II of this order.

6. According to Form 8 of the above Act and Rules, four Doctors are authorised to certify Brain death and this provision is clarified further.

(A) Doctor No.1 is the 'R.M.P. In charge of the hospital in which brain-stem death has occurred'. Accordingly, the Registered Medical Practitioner in charge of the hospital in which brain-stem death has occurred shall refer to either the Head of the Institution, RMO,

ARMO, Duty RMO or the RMP in charge of the Hospital. (No clearances are required from the Appropriate Authority in this category).

(B) Doctor No. 2 is the R.M.P. (Physicians, Surgeons or Intensivists) nominated from the panel of names approved by the Appropriate authority'. Accordingly, a panel of names shall be sent by the Dean/Medical Superintendent/Medical Director to the Approprate Authority namely the Director of Medical and Rural Health Services and on approval shall then be utilised as the panel from which a R.M.P. shall be nominated for each brain death certification. Each hospital may determine its own procedure for this duty.

(C) Doctor No.3 is 'Neurologist/Neuro-Surgeon nominated from the panel of names approved by the Appropriate Authority'. Again, a panel of names shall be sent by the Dean/Medical Superintendent/Medical Director to the Appropriate Authority namely the Director of Medical and Rural Health Services and on approval shall then be utilised as the panel from which one specialist as in the category therein shall be nominated for each brain death certification. Each hospital may determine its own procedure for this duty.

(D) Doctor No.4 is the R.M.P. treating the aforesaid person. This does not require any clarification and shall be the R.M.P./ Doctor on duty treating the patient. (No clearances are required from the Appropriate Authority in this category).

Note: i) In the event of lack of authorised personnel in Category 3 above in the hospital concerned, a request may be made to any other member of the panel from another hospital.

ii) The 1st and 2nd Medical examination as defined in Form-8 of the THO rules shall be conducted by category 2 and 3 Doctors from the panel approved by the Appropriate Authority.

7. Although it has been made mandatory for the three Medical College hospitals in Chennai to follow this procedure, the same procedure shall be applicable to all hospitals inclusive of private hospitals which wish to certify Brain Death as and when required. Accordingly, the categories that require for the panel to be approved shall be done so on submission to the Appropriate Authority (Director of Medical and Rural Health Services).

8. The Director of Medical Education and the Director of Medical and Rural Health Services are directed to periodically organise awareness workshops on the provisions of the above order.

(By order of the Governor)

V.K.Subburaj,
Secretary to Government

To
The Director of Medical Education, Chennai 600 010.
The Director of Medical & Rural Health Services, Chennai 600 006.
The Dean, Government General Hospital, Chennai 600 003.
The Dean, Government Stanley Hospital, Chenna-600 001.
The Dean, Government Kilpauk Medical College Hospital, Chennai – 600 010.

/forwarded by order/

Section Officer

Annexure - I
FORM 8

[See rule 4(3)(a) and (b) of the THO Rules 1995]

We, the following members of the Board of medical experts after careful personal examination, hereby certify that Shri/Smt./Km. …… aged about …… s/o, d/o, w/o, Shri. ……... resident of ……. is dead on account of permanent and irreversible cessation of all functions of the brain-stem. The tests carried out by us and the findings therein are recorded in the brain-stem death Certificate annexed hereto.

Dated ……. Signature ……

1. R.M.P., Incharge of the Hospital in which brain-stem death has occurred.
2. R.M.P., nominated from the panel of names approved by the Appropriate Authority.
3. Neurologist/Neuro-Surgeon nominated from the panel of names approved by the Appropriate Authority.
4. R.M.P., treating the aforesaid deceased person.

BRAIN-STEM DEATH CERTIFICATE

Patient Details:

1. Name of the patient — Shri/Smt./Km. ……
   ……
   S.O./D.O/W.O. — Shri. ……
   ……
   Sex …… Age ……
2. Home address — ……
   ……
   ……
3. Hospital Number — ……
4. Name and Address of next of kin or person responsible for the patient (if none exists, this must be specified) — ……
   ……
   ……

.....

.....

5. Has the patient or next of kin agreed to any transplant? .....

.....

.....

6. Is this a Police Case? Yes ..... No .....

Pre-conditions

1. Diagnosis: Did the patient suffer from any illness or accident that led to irreversible brain damage? Specify details ……
……
…
……
…

Date and time of accident/onset of illness ……

Date and onset of non-responsible coma

……

2. Findings of board of Medical Experts:

(i) The following reversible causes of coma have been excluded: -
Intoxication (Alcohol)
Depressant Drugs
Relaxants (Neuromuscular blocking agents)

	First Medical Examination		Second Medical Examination	
	1st	2nd	1st	2nd
Primary hypothermia				
Hypovolaemic shock				
Metabolic or endocrine disorders				
Tests for absence or brain-stem functions				

(ii) Coma

(iii) Cessation of spontaneous breathing

(iv) Pupillary size

(v) Pupillary light reflexes

(vi) Doll's head eye movements

(vii) Corneal reflexes (Both sizes)

(viii) Motor response in any cranial nerve distribution, any responses to stimulation of face, limb or trunk

(ix) Gag reflex

(x) Cough (Tracheal)

(xi) Eye movements on coloric testing bilaterally

(xii) Apnoea tests as specified

(xiii) Were any respiratory movements seen ?

.....

.

Date and time of first testing : .....

Date and time of second testing : .....

This is to certify that the patient has been carefully examined twice after an interval of about six hours and on the basis of findings recorded above.

Shri./Smt./Km. ..... is declared brain-stem dead.

1. Medical Administrator Incharge of the hospital
2. Authorised Specialist.
3. Neurologist/Neuro-Surgeon
4. Medical Officer treating the patient.

NB. I. The minimum time interval between the first testing and second testing will be six hours.

II. No. 2 and No. 3 will be co-opted by the Administrator In charge of the hospital from the Panel of experts approved by the appropriate authority.

ANNEXURE - II

Guidelines for Apnoea Tests:

Patient should have a temperature of more than 35° centigrade euvolemic and with Systolic pressure =/> 90 mm of Hg.

i. The first Apnoea test should be performed only after 4 hours of Coma associated with absence of brain stem reflexes. In the case of Anoxic brain damage, this period should be extended to 12 hours.
ii. The Physician involved in certifying Brain death shall be present during Ventilator removal to attest the presence of apnoea if found.
iii. Ventilator manipulation is performed to raise the PaCo2=/>40 mmHg.
iv. The patient should be hyper oxygenated with 100% oxygen for 15 minutes, while still on the ventilator, prior to the apnoea test.
v. Either a blood gas or trending of ETCO2 should be used to determine the adequacy of the baseline prior to the test. SPO2 should be monitored during apnoea test.
vi. Place the patient on 100% oxygen through a tracheal catheter with the tip towards the end of the tube with a continuous 6L/min O2 flow.
vii. The patient is taken off the ventilator in the presence of the physician certifying brain death. The patient is kept off the Ventilator for a variable period of time (usually 3 to 8 minutes) to allow the PaCo2 to rise =/>55 mmHg or =/>15 mmHg over baseline. During this time the patient is observed for respiratory movements.
viii. Test interpretations;
    a. Positive test – implying apnea despite adequate stimulation
        i. Patient remains apneic, without respiratory movements
        ii. PaCo2 is =/> 55 mmHg or =/>15 mmHg from baseline
    b. Negative test – Implying apnea is not present
        i. Respiratory efforts noted at any time during the test
    c. Indeterminate test
        i. - PaCo2 <55 mm Hg or there is less than 15 mm Hg increase over baseline.
    d. Indeterminate tests can either be repeated or another confirmatory test utilized.
ix. Apnoea test should be aborted if the patient develops hypotension, or significant cardiac arrhythmias.
x. These norms will vary for patients less than 12 years and patients with major chest trauma.

ABSTRACT

Health & Family Welfare Department – Organ Transplant – Cadaver Organ Transplant Program - Procedure to be adopted for cadaver transplant by the Government and Private Hospitals approved for organ transplant by the Appropriate Authority – orders issued.

HEALTH AND FAMILY WELFARE (Z1) DEPARTMENT

Thiruvalluvar Aandu 2039
Aavani-20

G.O. (Ms) No.287 Dated : 05.09.2008

Read :

1. G.O. (Ms) No. 6, Health and Family Welfare Department Dated 8.1.2008.
2. G.O. (Ms) No. 75 Health and Family Welfare Department Dated 3.3.2008.

ORDER:-

Considering the large number of patients who are suffering on account of serious organ ailments ranging from heart, liver, kidney etc and could otherwise lead healthy lives if they had the opportunity to have transplant surgery and also as there is a need for streamlining the procedures for cadaver transplants in Government and private hospitals, the Government have analysed the suggestions and opinions brought forward by medical professionals related to this field and the combined opinion of the Director of Medical and Rural Health Services and Director of Medical Education and issue the following orders.

2. All hospitals, approved for transplantation of human organs, and who are willing to participate in the arrangements for cadaver organ transplant program as dictated by this Government Order shall indicate their willingness to the convenor, cadaver transplant program, Tamil Nadu. All participating hospitals will upload details of their waiting list of prospective cadaver organ recipients through an online form to a computer database that will be maintained by the Transplant Coordinator of the Government General Hospital,

Chennai. (hereinafter referred to as Convenor, Cadaver Transplant Program, Tamil Nadu) Assistance for the maintenance of the web-based application may be had from NGO's working for the cause of organ donation.

3. The database will maintain prioritization lists for
   (i) each hospital
   (ii) for all Government hospitals combined
   (iii) for all private hospitals combined and
   (iv) for Government plus private hospitals combined, based on preset criteria determined in this order.

4. When the family of the brain dead patient is willing to donate his/her organ(s) to benefit others, the following procedures shall be strictly adhered to:-

(i) The procedures in declaring brain death shall be adhered to as laid out in the Government Order second read above.

(ii) Form 6 as laid out in the Transplantation of Human Organs Rules, 1995 shall duly be signed by the person(s) in possession of the brain dead patient and in the case of children below the age of eighteen years, the appropriate form namely Form 9 of the Transplantation of Human Organs Rules, 1995 requires to be signed by the persons concerned before organ retrieval.

(iii) Organ(s) retrieval shall not be carried out on a brain dead patient merely due to an earlier declaration by the said patient in Form 5 of the Transplantation of Human Organs Rules, 1995. While such a declaration shall presuppose the previous intentions of the brain dead patient to donate his/her organ(s), consent in Form 6 of the Transplantation of Human Organs Rules, 1995, is necessary to continue with the process of organ retrieval.

(iv) Each hospital should have its own waiting list for each organ, which will include the date of registration. The criteria as well as the prioritized waitlist, continually updated, should be made available online to the Convenor, Cadaver Transplant program, Tamil Nadu. Some hospitals may prefer date of registration. Exceptions to the already notified criteria must be substantiated with reasons to the Convenor, Cadaver Transplant program, Tamil Nadu when a request is made otherwise. The decision of the Convenor, Cadaver Transplant Program, Tamil Nadu is final.

(v) Any individual needing organ transplant through cadaver donation can be registered through only one hospital at a given time. She or he is free to change the registration to any other transplant

hospital, but the original date of registration will continue to apply for purposes of prioritization of organ allocation.

5. Following the above criteria for allocation, the organ(s) of the brain dead patient shall be shared in the following order, based on the respective prioritization list.

(i) If there is a patient who is to be a multi organ recipient and a matching organ donor is available, then the multi organ recipient takes precedence over all others on the regular waiting list. The Convenor, Cadaver Transplant program, Tamil Nadu will take the appropriate decision regarding allocation criteria when such a situation arises.

(ii) Considering the peculiar nature of certain liver ailments, a provision is made, which is as follows:
Potential liver recipients in hospitals are to be listed in one of the two categories namely 'urgent' or 'standard'.

(a) URGENT: Those on the urgent list are those who have:

(i) Hepatic Artery Thrombosis following a liver transplant.
(ii) Primary Non function of a graft
(iii) Fulminant hepatic failure.

These conditions do not require a waiting time on the list.

(b) STANDARD: This list refers to all patients who need a liver transplant but do not fulfill criteria for urgent listing. Patients on the standard list have to be registered for more than 24 hours to be listed in this category.
The Liver is to be allotted to participating hospitals in turn.
Note: Patients on the urgent list supersede the standard list and the hospital misses its regular turn on the rota.

(iii) Similarly, potential heart recipients in hospitals are to be listed in one of the two categories namely 'urgent' or 'standard'.

(a) URGENT:

(i) Patients with Left Ventricular Assist Device (LVAD).
(ii) Followed by patients with Intraaortic Balloon Pump (IABP)

(b) STANDARD: Sick, but stable patients waiting at home for a heart transplant.
A Heart is to be allotted to participating hospitals in turn.

(iv) Likewise for lungs, prioritization would be made according to the urgency of transplant and allotted to participating hospitals in turn.

(v) For kidneys no out of turn allocation would be permitted and the sharing criteria in the following para shall be followed.

6. Sharing of Organs for waitlisted recipients, retrieved from cadaver donors in Government Institutions:

(i) First priority to the list of the Government Hospital where the deceased donor is located, for liver, heart and one kidney. The other kidney would be allocated to the general pool in the priority sequence as listed below.

(ii) Second priority to the combined Government Hospitals list

(iii) Third priority to the combined Private Hospitals list

(iv) Fourth priority to Government Hospitals outside the State, (in order to maximize organ utilization)-provided earlier information and such a request has been registered with the Advisory committee / Convenor, Cadaver Transplant Program, Tamil Nadu.

(v) Fifth priority to Private Hospitals outside the State (in order to maximize organ utilization) provided earlier information and such a request has been registered with the Advisory committee/ Convenor, Cadaver Transplant Program, Tamil Nadu.

(vi) Finally, if the organ(s) remains unutilized by the above criteria, it may be offered to a foreign national registered in a Government or Private hospital within and then outside state.
(This is to ensure that there is no wastage of organs donated)

7. Sharing of Organs for waitlisted recipients retrieved from cadaver donors in Private Hospitals, which are transplant centers.

(i) First priority to the list within the Private Hospital where the deceased donor is located, for liver, heart and one kidney. The other kidney would be allocated to the general pool in the priority sequence as listed below.

(ii) Second priority to the combined list of Government and Private Hospitals.

(iii) Third priority to Government / private hospitals outside the state, (in order to maximize organ utilization) provided earlier information and such a request has been registered with the Advisory committee / Convenor, Cadaver Transplant Program, Tamil Nadu.

(iv) Finally, if the organ(s) remains unutilized by the above criteria, it may be offered to a foreign national registered in Government or private hospital within and then outside the state, provided earlier information and such a request has been registered with the Advisory committee /Convenor, Cadaver Transplant Program, Tamil Nadu.

8. Whenever a deceased donor becomes available in a hospital, the concerned hospital shall contact the Transplant Coordinator or a member of his team at the Government General Hospital, Chennai who will then make allocations based on the above. MOHAN Foundation and National Network for Organ Sharing, (NNOS), NGO's promoting organ transplantation may assist the Transplant Coordinator, Government General Hospital, Chennai to facilitate this arrangement and ensure that most number of cadaver organs donated are utilized to benefit organ failure patients.

9. Considering that a number of practical issues are involved such as

(i) establishing formats and procedures for recipient listing, organ allocation and transfer
(ii) coordination between hospitals where donor / recipient are located
(iii) working towards a coordinating body that would be institutionalized and fine-tuning identification criteria to determine the beneficiaries-
(iv) Proposing policy initiatives from time to time.
(v) Need for watching the working of the cadaver organ transplantation program,

the Government have decided to form an Advisory committee that would address the above issues and ensure stability in functioning of the cadaver organ transplant program

10. This Advisory committee shall be headed by the Secretary, Health or his nominee as Chairman and the committee shall consist of:

(i) Secretary, Health or his nominee-Chairman
(ii) Convenor, Cadaver Organ Transplant Program, Tamil Nadu (i.e Transplant Co-ordinator, Government General Hospital, Chennai.)
(iii) Director of Medical Education or preventative
(iv) Director of Medical and Rural Health Services or representative
(v) Transplant team member, Government Stanley Hospital, Chennai
(vi) Transplant team member, Kilpauk Medical College Hospital, Chennai.
(vii) Transplant team member, Government General Hospital. Chennai.
(viii) One senior police officer of DIG rank or above as nominated by the Director General of Police, Chennai.
(ix) Member from MOHAN Foundation, Chennai.
(x) Member from National Network for Organ Sharing, (NNOS) Chennai.
(xi) One transplant team member from three different hospitals that currently have largest cadaver donation experience.

The Advisory committee shall in turn nominate four sub-committees to assist in its functioning for

(i) Liver
(ii) heart
(iii) kidney
(iv) other organs- to determine the severity of illness for listing a patient for transplant.

11. The Advisory committee shall also propose within three to six months to Government the Coordinating body that needs to be formed and institutionalized to periodically give inputs on organ sharing and allocation and to further-streamline the program.

(BY ORDER OF THE GOVERNOR)

V.K.SUBBURAJ,
PRINCIPAL SECRETARY TO GOVERNMENT

To

All Registered Transplant Hospitals,
through the Director of Medical and
Rural Health Services, Chennai - 600 006.
The Director of Medical & Rural Health Services, Chennai 600 006.
The Director of Medical Education, Chennai 600 010.
The Dean, Government General Hospital,Chennai 600 003.
The Dean, Government Stanley Hospital, Chenna-600 001.
The Dean, Government Kilpauk Medical College Hospital,
Chennai – 600 010.
The Transplant Co-ordinator, Government General Hospital,
Chennai - 600 003.
The Director General of Police, Chennai - 600 004.
Copy to:
The Senior P.A. to Minister for Health, Chennai - 600 009.
The P.S. to Secretary to Government,
Health and Family Welfare Department, Chennai - 600 009.

/FORWARDED BY ORDER /

SECTION OFFICER

Dentistry

Standard Treatment Guidelines
Tamil Nadu Health Systems Project

Chapter 22

- Ethics in Dental Practise
- Bleeding Gums
- Periodontal Disease
- Dental Caries
- Dental Trauma
- Co morbid Medial Conditions
- Precancerous Lesions
- Trismus
- Oral Cavity Disease
- Facio Maxillary Conditions
- Dental Impactions
- Dental malocclusion
- Cyst and Tumours
- Drug Interactions

Ethics in Dental practice

1. The Dentist's primary duty of serving the public is discharged by giving the highest type of service of which he is capable and by avoiding any conduct that leads to lowering the esteem of the profession of which he is a member.
2. Selection of patients: in serving the public, a Dentist may exercise reasonable discretion in selecting patients for his practice. However, he may not refuse to accept a patient into his practice or deny orthodontic service to a patient solely because of the patient's race, creed, colour or national origin.
3. The Dentist should inform the patient of the proposed treatment, and any reasonable alternatives, in a manner that allows the patient to become involved in treatment decisions.
4. Confidentiality regarding patient's records: relevant information in the records should be released to another Dental practitioner assumes that the Dentist requesting the information is the patient's present Dentist.
5. If it is necessary for a treating Dentist to consult with another Dentist or physician with respect to the patient, and the circumstances do not permit the patient to remain anonymous, the treating Dentist should seek the permission of the patient prior to the release of data from the patient's records to the consulting practitioner. If the patient refuses, the treating Dentist should then contemplate obtaining legal advice regarding the termination of the Dentist/patient relationship.
6. A Dentist has the obligation of seeking consultation whenever the welfare of the patient will be safeguarded or advanced by having resource to those who have special skills, knowledge and / or experience
7. The use of professional letterheads in connection with a Dentist's efforts to promote a commercial endeavor is undignified and might tend to lower public esteem for the profession.
8. It is unethical for a Dentist to use patients for teaching or research purposes without their fully informed consent or to mislead a patient as to identify the Dentist who performs the service or procedure.
9. Dentists shall be obliged to make reasonable arrangements for the emergency care of their patients of record. Dentists shall be obliged when consulted in an emergency by patients not of record to make reasonable arrangements for emergency care.
10. In a malpractice suit both parties have a right to present expert testimony through witnesses. A Dentist acting as a witness may not be disciplined merely for presenting his professional opinionA Dentist has an obligation not to prescribe, dispense or promote the use of drugs or other agents where complete formulas are not available to the profession. He also has the obligation not to prescribe any therapeutic agent, the value of which is not supported by scientific evidence.
11. It is unethical for a Dentist to allow the use of offers or his person as part a commercial advertisement
12. Dentists shall be obliged to protect the health of their patients by only assigning to qualified auxiliaries those duties which can be legally delegated. Dentists shall be further obliged to prescribe and supervise the patient care provided by all auxiliary personnel working under their direction.
13. It is unethical for a Dentist to practice while abusing controlled substances, alcohol or other chemical agents which impair the ability to practice.
14. A Dentist who contracts any disease or becomes impaired in any way that might endanger patients or Dental staff shall, with consultation and advice from a qualified physician or other authority, limit the activities of practice to those areas that do not endanger patients or Dental staff.
15. All Dentists, regardless of their bloodborne pathogen status, have an ethical obligation to immediately inform any patient who may have been exposed to blood or other potentially infectious material in the Dental office of the need for postexposure evaluation and followup and to immediately refer the patient to a qualified health care practitioner who can provide postexposure services. The Dentist's ethical obligation in the event of an exposure incident extends to providing information concerning the Dentist's own bloodborne pathogen status to the evaluating health care practitioner, if the Dentist is the source individual, and to submitting to testing that will assist in the evaluation of the patient. If a staff member or other third person is the source individual, the Dentist should encourage that person to cooper-

ate as needed for the patient's evaluation.

16. Once a Dentist has undertaken a course of treatment, the Dentist should not discontinue that treatment without giving the patient adequate notice and the opportunity to obtain the services of another Dentist. Care should be taken that the patient's oral health is not jeopardized in the process.
17. A Dentist has the general obligation to provide care to those in need. A decision not to provide treatment to an individual because the individual is infected with HIV hepatitis b virus, hepatitis c virus or another bloodborne pathogen, based solely on that fact, is unethical. Decisions with regard to the type of Dental treatment provided or referrals made or suggested should be made on the same basis as they are made with other patients. As is the case with all patients, the individual Dentist should determine if he or she has the need of another's skills, knowledge, equipment or experience. The Dentist should also determine, after consultation with the patient's physician, if appropriate, if the patient's health status would be significantly compromised by the provision of Dental treatment.
18. Patients should be informed of their present oral health status without dis - paraging comment about prior services.
19. Dentists shall not represent the care being rendered to their patients in a false or misleading manner.
20. A Dentist who recommends and performs unnecessary Dental services or procedures is engaged in unethical conduct
21. Dentists should not induce their patients to purchase products or undergo procedures by misrepresenting the product's value, the necessity of the procedure or the Dentist's professional expertise in recommending the product or procedure.
22. A Dental practice should not seek to attract patients on the basis of partial truths which create a false impression.

Bleeding gums

Definition

Bleeding gums can be a sign that you are at risk for, or already have, gum disease. However, persistent gum bleeding may be due to serious medical conditions such as leukemia and bleeding and platelet disorders.

Causes

Bleeding gums are mainly due to inadequate plaque removal from the teeth at the gum line. This will lead to a condition called ginigivitis or inflamed gums.

If plaque is not removed through regular brushing and Dental appointments, it will harden into what is known as tartar. Ultimately, this will lead to increased bleeding and a more advanced form of gum and jawbone disease known as periodontitis

Other causes of bleeding gums include:

- Any bleeding disorder
- Brushing too hard
- Hormonal changes during pregnancy
- Idiopathic thrombocytopenic purpura
- Ill-fitting dentures
- Improper flossing
- Infection, which can be either tooth or gum-related
- Leukemia
- Scurvy
- Vitamin K deficiency

Diagnosis

Apart from clinical examination of gingiva diagnostic tests that may be performed include:

Blood studies such as a CBC or blood differential

X-rays of the teeth and jawbone

Treatment

In case of bleeding disorders and leukemia, patient to be referred to a Medical consultant.

Vitamin Supplements.

In case of any abscess, depending on the nature, it is drained / antibiotics is prescribed

In the next appointment OHI and scaling, changing the dentures.

Periodontal disease

Definition

An infectious disease resulting in inflammation within the supporting tissues of the teeth, progressive attachment loss, and bone loss.

There are different types, or classes, of periodontitis. The most common class is chronic periodontitis the typical type that most adults older than age 35 have. Periodontitis that begins in childhood or early adulthood is called aggressive periodontitis. Both types may be generalized or localized.

Clinical features

- Swollen gums
- Bright red or purplish gums
- Gums that feel tender when touched
- Bad breath
- Periodontal pocket depth
- Clinical attachment loss(Gingival recession)
- New spaces developing between your teeth
- Pus between your teeth and gums
- Mobile teeth
- A change in the way your teeth fit together when you bite
- Risk factors
- Factors that can increase your risk of periodontitis include:
- Gingivitis
- Local factors like plaque and calculus.
- Poor oral health habits
- Tobacco use
- Diabetes
- Older age
- Heredity
- Decreased immunity, such as that occurring with leukemia or HIV/AIDS
- Poor nutrition
- Hormonal changes, such as those related to pregnancy
- Ill-fitting Dental restorations
- Lower socioeconomic status
- Diagnosis
- Clinical examination
- IOPA Radiograph which shows horizontal / vertical bone loss.
- Treatment
- OHI and scaling and recall.
- Scaling and root planning and recall.
- Flap surgery in case of vertical bone loss.

Dental caries

Dental caries is an infectious microbial disease of the teeth that results in localized dissolution and destruction of the calcified tissues.

- It is a multi-factorial disease, in which there is interplay of factors as the host (teeth, saliva, etc.), microflora, substrate (diet) and time.
- Pit and fissure caries can be prevented by pit and fissure sealant application and smooth surface caries by topical fluoride application.
- Incipient caries appears as white spot lesion. It can be differentiated from developmental hypo-calcifications in that, incipient caries disappears on hydration and reappears on drying, but developmental hypo-calcifications remain unaltered.
- Incipient caries can be remineralized, which is aided by topical application of fluoride or CPP-ACP and do not need restorative treatment.
- For restoration of anterior teeth, aesthetic material like composite is used.
- For posterior restorations, materials like amalgam, hybrid composite are used. When tooth destruction is more, metal inlays and onlays can be used. Ceramic inlays and onlays can be used in areas of aesthetic interest.
- Basically, the patient's caries risk has to be assessed and diet counseling and oral hygiene instructions have to be given accordingly to minimize the risk and incidence of caries.

Dental Trauma

Traumatic injuries to the teeth result in damage to many Dental and periradicular structures. The various effects of trauma to teeth are,

1. Crown fracture
2. Crown root fracture
3. Root fracture
4. Luxation injury
5. Avulsion

Crown fracture:

- If it involves only enamel and dentin (uncomplicated fracture), aesthetic restoration of the tooth is sufficient.
- If there is involvement of pulp (complicated fracture), vital pulp therapy can be done, for which optimal time is first 24 hours.
- Partial pulpotomy has higher success rate (95%) than direct pulp capping (80%).
- In traumatic exposure after 72 hours in immature teeth, full pulpotomy can be considered for apexogenesis. In mature teeth, pulpectomy should be done.
- In non-vital teeth with open apex, apexification is indicated to create an apical stop.

Crown root fracture:

- If the tooth can be maintained from a periodontal point of view, pulp is treated as in a crown fracture.

Root fracture:

- In this coronal segment is reduced quickly and functional splint is placed for 2 to 4 weeks.
- When attended in time, pulp necrosis is remarkably low. It will occur only 25% of the time and in majority of cases, necrosis will occur only in the coronal segment and only that is RC treated.

Luxation injury and Avulsion:

- Lateral luxation, extrusion and avulsion also needs emergency splinting and later RCT, if required.

Immediate vitality testing may be misleading because of the 'stunned pulp' and it has to be repeated at 3 weeks, 3, 6 and 12 months to monitor the status of the pulp.

Management of patients with special needs

As soon as the patient enters the clinic, help the patient to relax. Vital signs have to be recorded. After proper diagnosis and formulation of treatment plan, the patient's caretaker has to be informed about the treatment required and informed consent should be obtained from them for the treatment.

Communication:

Good communication with the patient has to be established by

1. Speaking to the patient in his or her level of understanding (in case of mentally challenged people
2. Smiling often, making gestures, showing pleasant facial expressions, using simple sign language (In case of hearing impaired or non – verbal clients) physical contact and praise

Administration of sedatives:

In case of patients who get agitated even during normal Dental procedures presedation, sedation and manual restraint procedures to undergo Dental procedures can be given

Presentation :

Drugs like anticholinergics and antiemetics can be given.

Sedation :

This is given to reduce hypertonicity, involuntary movements and anxiety. Drugs like diazepam or nitrous oxide can be used

Restraints :

1. Patient's head has to be stabilised during the procedures to avoid sudden movement with the help of an assistant
2. If required soft wrap with nylon fastners (Velcro) can be used across the lap or shoulders to prevent the patient from the chairduring treatment

Note:

The patient's caretaker has to be present during the time of sedation and during the Dental procedures to reassure the patient.

The Dental procedures have to be done only after the patient calms down

The Dentist or the Dental assistant has to remain in the operatory after administration of sedation and

constantly monitor the vital signs.

Management during Dental treatment:

1. Short appointments have to be fixed and only little procedures need to be done at a time.
2. There must be short periods of rest in between procedures
3. Adjustable mouth props can be used to keep the patient's mouth open during the procedures. The mouth prop can be inserted into the patient's mouth a little ahead of time for the patient to get accustomed to it.
4. Proper finger rests and cheek retraction are required all through the procedures
5. Rubber dam has to be used in order to protect the working area from hyperactive tongue movement.
6. Floss should be attached to small objects such as cotton rolls that become dislodged
7. Gauze shields should be used to prevent tooth aspiration during extractions.
8. Throughout the procedure the Dentist must be relax, and follow the tell – show – do technique before each step in the procedure.

Dismissal Of The Patient:

If the patient had been given sedation, then he/she has to be sent to their living place only after they become responsive and their vital signs are stable.

Co-morbid Medical Conditions

Bleeding problems / patients on anticoagulants

Initially a thorugh medical history is to be taken regarding the details of the bleeding problem, its duration of its existence, duration of anticoagulant therapy and name of the specific anticoagulants used.

Diagnostic Tests:

1. Bleeding problems secondary to liver disease:
   * PT - prothrombin time
   * PTT - partial thromboblastin time
   * INR - international normalized ratios
2. Aspirin and other non-steroidal anti-inflammatory agents.
   * Bleeding time.
3. Thrombocytopenia
   * CBC with a differential (which will give platelet count)
   * Bleeding time
4. Anticoagulant warfarin
   * PT
   * INR

Dental treatment should be defferred in case of patients with:

1. Bleeding time greater than 10 minutes
2. Platelet count less than 60,000
3. PTT greater than 45 seconds
4. PT greater than 22 seconds
5. INR greater than 3.5
   * If bleeding parameters are greater than above, medical coordination is required, for example, physician should decrease anticoagulant or provide packed platelets or prescribe supplemental vitamin K until bleeding parameters are brought into line consistent with Dental treatment.
   * If hemophilic, have a specialist nearby and administer proper replacement factors and run necessary tests to ensure patient is within safe parameters.
   * During Dental procedures minimize physical trauma and pack extraction sites that have the potential to bleed with local pressures and other coagulation procedures, i.e. Gelfoam. Obtain primary closure on any surgical sites, if possible.
   * Establish primary closure and/or put pressure on potential/actual bleeding site.

Be alert for:

1. Easy or prolonged bleeding with minimal trauma (i.e. probing, wedge placed between teeth for amalgam matrix)
2. Easy bruising / multiple bruises

Precautions to be taken :

1. Assure that the patient is aware of necessary lab tests and that should be done close to the time of Dental treatment (within a week, or closer if they have had previous problems). Some bleed-

ing parameters can change quickly.

2. Drugs to be avoided:
   * Drugs like Erythromycin and Ketoconazole which inhibit warfarin metabolism.
   * Drugs that can prolong bleeding, such as Aspirin or other non-steroidal anti-inflammatory drugs.
3. Encourage patient to keep you informed of any drug changes and their use of any over-the-counter medications.
4. If patient calls from home following treatment, instruct them to apply pressure with gauze or cloth to bleeding site for 10-30 minutes, continuously.
5. If bleeding persists, have patient come into office immediately or to a Medical Emergency Room.

For patients with cardiac problems

A thorough medical history to find if nature of cardiac problem (functional / organic)

Diagnostic Tests:

Medical consult to identify type of heart murmur and whether prophylactic antibiotics are needed

Management during Dental treatment:

1. If patient needs prophylactic antibiotics, follow American Heart Association guidelines below:

Premedication requirements for patients with valvular heart disease or congenital cardiac defects. If in doubt, have patient consult their physician as to need.

Standard Regime

Amoxicillin 500 mg.

Disp 4 tablets

Take 4 tablets (2.0 g) 1 hour before procedure.

Note:

- Children 50 mg/Kg. Do not exceed adult dose
- No second dose is required for adults or children

Standard regime for patients allergic to amoxicillin/penicillin

Clindamycin 150 mg.

Disp 4 tablets

Take 4 tablets (600 mg) 1 hour before procedure.

(or)

Azithromycin 250 mg

Disp 2 tablets

Take 2 tablets (500 mg) 1 hour before procedure.

(or)

Clarithromycin 250 mg

Disp 2 tablets

Take 2 tablets (500 mg) 1 hour before procedure.

(or)

Cephalexin 500 mg.

Disp 4 tablets

Take 4 tablets (2 g) 1 hour before procedure.

(or)

Cefadroxil 500 mg

Disp 4 tablets

Take 4 tablets (2 g) 1 hour before procedure.

Note:

Cephalosposins should not be used in individuals with immediate type I hypersensitivity reaction (urticaria, angiodema, or anaphylaxis) to penicillins.

Children's dosage. (Do not exceed adult dose)

Clindamycin 20 mg/kg

Cephalexin 50 mg/kg

Cefadroxil 50 mg/kg

Azithromycin 15 mg/kg

Clarithromycin 15 mg/kg

Patients unable to take oral medication

Ampicillin 2 g IV or IM within 30 minutes before procedure.

Children: 50 mg/kg IV or IM within 30 minutes before procedure.

For patients unable to take oral medication and allergic to ampicillin, amoxicillin, penicillin

Clindamycin 600 mg IV within 30 minutes before procedure.

Children: 20 mg/kg IV within 30 minutes before procedure.

Cefazolin 1 g IV or IM within 30 minutes before procedure

Children: 25 mg/kg IV or IM within 30 minutes-before operation.

Note :

Cephalosporins should not be used in individuals with immediate-type I hypersensitivity reaction (uticaria, angiodema, or naphylaxis) to penicillin.

1. If patient states they're unsure whether prophylactic antibiotics are needed and contact with physician. If not possible then treat with standard guidelines if emergency or refer patient for medical consult to establish need or lack of need for antibiotic prophylaxis.
2. Document, in the chart, the time and dosage of antibiotics taken for prophylaxis.

Be alert for:

Flu-like symptoms within two days, most commonly within two weeks, rarely within four weeks following Dental procedures. Such symptoms can be signs of bacterial endocarditis, even if the patient has been properly prophylaxed. If they have such they should see their physician.

Precautions to be taken :

1. Good oral hygiene.
2. Proper teeth cleaning, chlorhexidine rinse prior to extractions to decrease magnitude of possible bacteremias.
3. Gingivitis, and, especially, periodontitis, increases the frequency, intensity, and duration of bacteremias.
4. The patient has to be insisted on taking their prophylactic antibiotic medication within the proper timeframe.

Cardiovascular problems

High blood pressure, arrhythmia, congestive heart failure and Angina pectoris

A thorough history is to be taken regarding the type and duration of cardiac problem, the specific symptoms and the medications taken for the same

Diagnostic tests:

1. High blood pressure:
   * Take blood pressure at the beginning and at the end of appointment.
2. Arrhythmia's
   * Take patient's peripheral (radial, carotid) pulse and feel for arrhythmia
3. Congestive heart disease
   * Stress test by specialist

Management during Dental treatment:

1. High blood pressure.
   * Blood pressure of 140/90 or less, no special precautions needed.
   * 141/91 - 160/110, depending on Dental procedure and patient's level of anxiety, consider some type of sedation or delay elective treatment until blood pressure controlled.
   * Greater than 161/111, no elective treatment until blood pressure under control. Even if Dental emergency treatment is needed, consult with specialist first to control BP. Consider sedation with benzodiazepine (valium) or nitrous oxide.
   * In patients with controlled high blood pressure, using local anesthetic with a vasoconstrictor such as 1:100,000 epinephrine or it's equivalent is appropriate. The ADA suggests a maximum of 40 µg (2 cartridges of 1:100,000 epi) then wait for at least 10 minutes. If no problems arise, additional cartridges can be administered.
   * For patients with blood pressure above 140/90, epinephrine impregnated retraction cord should be avoided.
2. Arrhythmia or congestive heart failure
   * If patient's arrhythmia or congestive heart failure is controlled, no special precautions necessary.
   * If patient has an arrhythmic or congestive heart failure (angina pectoris) episode, Dental treatment should be delayed. If arrhythmia occurs in the midst of treatment and if the treatement must be completed, discontinue until heart rhythm stabilized (may require hospitalization for cardioversion), then complete treatment quickly and calmly.
   * If angina pectoris occurs, stop treatment, administer oxygen, minimize stress and wait until the pain resolves. Continue as needed, if necessary, and patient if feels capable of com-

pleting to a safe stopping point

* Local anesthetic with vasoconstrictor (1:100,000 epinephrine or equivalent) Is appropriate. 1:50,000 concentration of epinephrine or equivalent should be avoided.

Epinephrine impregnated retraction cord should not be used.

Be alert for:

1. High blood pressure.
   * Request patient inform you if they feel as though their blood pressure is Increasing or if they are getting a headache. Some patients feel jittery,others feel as though there is increased pressure behind the eyes.
   * Profuse bleeding, beyond what would be expected.
2. Arrhythmia
   * Patient to inform you if they feel an arrhythmia. Sometimes this manifest as a coughing or catching feeling in the chest. Other times it is a feeling of light headedness.

Precautions To Be Taken :

Be reassuring with the patient. Under no circumstances should you panic as that will only increase the patient's anxiety which will cause the blood pressure to increase or the arrhythmia to intensify or be prolonged.

Central Nervous System

A thorough history is to taken regarding the duration, symptoms and precipitating factors of the central nervous system problems

Diagnostic tests:

1. Stroke
   * If patient is taking anticoagulant, then assess bleeding status (follow bleeding problems management protocol)
2. Seizure
   * If patient is unclear about types of seizure or medications, and seizures are poorly controlled, then Medical consultation for the above information will be needed.

Management during Dental treatment:

1. Stroke
   * No special treatment considerations are necessary except those that the patient notes could be of value (modifying Dental treatment procedures based on the patient's perceived needs has an enormous positive psychological benefit for the patient).
   * Depending on what areas have lost function, especially if the head and neck or oral cavity area are affected, certain types of Dental prostheses may or may not be effective, i.e. removable prostheses may not be effectively retained without adequate muscle tone, so fixed prostheses or implant may be needed.
2. Seizures
   * Schedule patient early morning when they are well rested.
   * Patient should be instructed to take their medication properly for at least the Several days prior to the Dental appointment.
   * Patient should be questioned at Dental appointment whether in fact they have taken the medication correctly.
   * If seizure occurs, it should be allowed to run its course. The primary concern will be protection of the patient so they don't hurt themselves and the protection of the Dentist and staff so the patient doesn't hurt them.
   * Following a seizure, the decision to continue or discontinue treatment is based on the patient's condition (does the patient feel like he/she can complete the procedure) and the treatment needed.

Be alert for:

1. Stroke:
   * Signs of recurrence of stroke, such as slurred speech, confusion, loss of balance and inability to hold saliva in mouth, and transient ischemic attacks (TIA) which may manifest as fainting and dizziness, with spontaneous recovery.
   * Alert patient's guardian to any new stroke signs or symptoms so physician can follow up.
   * If patient is taking anticoagulants, review bleeding problems protocol for additional

alerts.

* If stroke has effected swallowing, use suction frequently.
* If stroke has effected eyelids, protect/cover eyes as needed.

2. Seizures:
   * Be alert to Dental / oral damage secondary to seizure.
   * Be aware of possible gingival hyperplasia secondary to Dilantin sodium.

Precautions to be taken:

Strokes and seizures:

Minimize stress, avoid procedures that may cause spiking of blood pressure, consider pre-procedural anti-anxiety medication such as Valium, if patient is fearful.

Seizures:

Good oral hygiene. The better the oral hygiene, the less likely or less severe gingival hyperplasia secondary to Dilantin sodium.

Diabetes Mellitus

A thorough history is to be obtained on duration of existence of the disorder and the medications taken

Diagnostic tests:

1. Fasting blood sugar (reflects current control, that day). (> 126 mg/dL)
2. Random plasma glucose > 200 mg/dL with symptoms (polyurina, polydipsia, unexplained weight loss)
3. 2 hour plasma glucose > 200 mg/dL following a 75g glucose load
4. Fructosamine test (reflects average control over last 2-3 weeks).
5. Glycated hemoglobin (reflects average control over last 6-8 weeks)(>7% = problem)

Management during Dental treatment:

1. Patient should have taken a balanced meal (includes fat and protein as well as carbohydrates) within the last two hours before coming to the Dental appointment.
2. Patient should have taken their medications (if they take medications).
3. Food (Power Bar or some other balanced nutritional supplement) should be available if appointment lasts longer than two hours.
4. Early morning appointments.

Be alert for:

1. Periodontal problems.
2. Candidiasis / xerostomia.
3. Poor response to treatment, especially periodontal therapy.
4. Poor healing.
5. Slow healing.
6. Any Dental infection should be treated promptly i.e. with antibiotics and appropriate incision and drainage.

Immuno suppression

Diseases:

HIV, leukemia, primary immunosuppressive diseases

Medications:

Cancer chemotherapeutic agents, immunosuppression drugs used in organ transplant patients, corticosteroids to suppress severe auto-immune diseases.

A thorough history regarding durations of existence of the problem and the duration of medication is to be obtained.

Diagnostic tests:

1. CBC with a differential (especially platelet count, if planning surgery).
2. T-suppressor cell count (HIV patients).
3. Viral load (HIV patients).

Management during Dental treatment:

1. Depending on severity of immunosuppression, laboratory tests, primarily CBC with differential, should be done immediately (within 5 days) of major invasive procedure, i.e. extractions, scaling and root planning, periodontal surgery.
2. If white cell count is below 2,000, no elective treatment until white cell count restored.
3. If platelet count is less than 60,000, no elective

treatment should be done.If emergency treatment is needed with the risk of bleeding, then have Physician administer the patient fresh platelet transfusion prior to procedure.

4. If patient is severely immunosuppresed and infection is present, consider prophylactic antibiotics prior to oral surgical or periodontal surgical procedures.
5. Institute aggressive treatment of any Dental infection, including antibiotics, incise and drain, and proceed with any necessary endodontic procedure or extraction.
6. Aggressively control any periodontal disease with proper cleaning and supplemental medication such as clorhexidine rinse.

Be alert for:

1. Periodontal infections
2. Yeast infections
3. Viral infections
4. Periapical problems, impacted teeth, poorly done endodontic procedures,oral ulcerations.

Precautions to be taken:

1. Prior to organ transplant or when patient is most immunocompetant, consider aggressive Dental therapy to remove / resolve any possible Dental problems, i.E. Scale / root plane for periodontal disease, extract impacted teeth, complete any needed or expected endodontic procedures. Consider extracting teeth with compromised endodontic prognosis.
2. Good oral hygiene.
3. Prophylaxis for viral and fungal infections.

Patient is advised to alert Dentist or physician at first sign of any infection.

Infectious Diseases:

A thorough history regarding the type of disease, duration, and type of medication to be taken.

Diagnostic tests:

1. Tuberculosis
   * If tuberculin test is positive, then an x-ray should be done.
   * If x-ray is positive, or if there is obvious active infection, then sputum test for tuberculosis baccilum should be done.
2. Hepatitis
   * Hepatitis antigens and antibodies should be done.
   * If patient has active hepatitis, then liver function should be done or request physician provide information as to liver function and coagulation status.
3. HIV
   * Current laboratory tests including T-cell count, viral load, CBC with a differential to give platelet count and white count should be done (follow protocol for immunosuppression).
4. Herpes / FLU
   * No specific laboratory tests need be done.
   * If patient is interested in which type of herpes they have, type 1 versus type 2, then antibody tests can be done.

Management during Dental treatment:

1. Tuberculosis
   * No elective treatment rendered until physician says patient is not infectious (sputum negative).
   * If emergency treatment is necessary, patient should be treated in a level 3 Infection control facility with hepafilter mask and laminer airflow.
   * In an actively infected patient, the air expelled when coughing is infectious and should be avoided.
2. Hepatitis
   * Since all patients are treated as though they are infectious and universal Precautions are applied, no special precautions are necessary when treating a patient actively infected with the hepatitis virus (If patient is having liver problems secondary to hepatitis, then review liver protocol).
3. HIV
   * If patient is HIV infected but has had no medical problems, then no special precautions are needed.
   * Since all patients are treated as though they are infectious, the usual Universal precautions are adequate for management.

* If patient has signs and symptoms of immunosuppression, follow protocols for patients with immunosuppression.
* Review the patient's medications and any Dental medications that may be used, to insure no drug interaction.

4. Herpes / FLU
   * Since all patients are treated as though they are infectious, the normal Universal precautions apply and patient is safe for treatment.
   * If patient is feeling so poorly that they don't feel strong enough for Dental treatment, they should be re-appointed.
   * If patient having herpes attack, no special precaution is necessary though Patient may want to have herpetic ulcer lubricated or even topical anesthetic applied to minimize discomfort associated with manipulation of oral cavity.

Be alert for:

1. Tuberculosis
   * Oral ulceration or head and neck ulceration, advanced forms of tuberculosis can manifest as what is termed caseating necrosis. Clinically it appears as an ulceration.
   * These ulcers have a high content of tubercular bacilli. Patients with such ulcerations should not receive elective Dental treatment until their T.B. infection is resolved.
2. Hepatitis
   * Be alert for signs of jaundice. Follow the protocol for liver dysfunction.
3. HIV
   * Be alert for oral manifestations of immunosuppression such as oral yeast infections, viral infections and periodontal problems. Follow the protocol for Immunosuppression.
   * Be alert for poor healing response and bone sequestration following extractions.
4. Herpes / FLU
   * With herpes, avoid traumatizing tissue as it may trigger a herpes attack.
   * If patient knows that herpes attack is precipitated by trauma, consider prophylactic antiviral medication.

Precancerous lesions

Carcinoma in situ

Similar age, sex distribution and sites as invasive oral carcinoma. Often adjacent to areas of invasive carcinoma.

Gross: Often like erythroplakia (red-velvety).

Rx Treatment

excision or irradiation.

Leukoplakia

White patch or plaque, 5 mm or more, on oral mucousmembrane, that cannot be removed by scraping, not due to another disease entity such as lichen planus or candidiasis, and not reversed by removal of irritants. Most common location is buccal gingival gutter; lesions in floor of mouth are often dysplastic. Treatment: vitamin A, Bcomplex and estrogens, X-ray therapy, surgical excision.

Erythroplakia (dysplastic leukoplakia)

Red, velvety, eroded area, level or depressed; treatment: Treatment depends on the histologic findings. If the biopsy shows dysplasia or carcinoma, total excision is indicated.

Speckled leukoplakia

Leukoplakia and erythroplakia; often has candida infection also. Overall 4% risk for carcinoma; highest if speckled or warty or occurs in floor of mouth or ventral surface of tongue. Biopsy needed if no response to tobacco or alcohol cessation. Gross: occurs anywhere in oral mucosa; solitary or multiple, variable appearance.

Verrucous hyperplasia

Slow growing, chiefly exophytic, and persistent, often multifocal. May represent precursor lesion to verrucous carcinoma, dysplasia or other carcinomas. Treatment: surgical excision.

White oral lesions

Lichen planus

Oral lichen planus is typically presents as bilateral white lesions (papules, plaques, or reticular areas) in the buccal and lingual mucosa. Lesions may be soft, creamy patches of thrush can be wiped off the mucosa, leaving erythema, and also symptomless.

Management: Topical corticosteroids are useful in controlling symptoms. Avoid smoking, treat predisposing

causes (such as xerostomia), and improve oral hygiene. Chlorhexidine has some anti-candidal activity, and antifungal drugs can be used.

Hairy leucoplakia

This is an asymptomatic white lesion not removable by wiping that is typically seen on the lateral margins of the tongue in immunocompromised patient. These lesions usually have an irregular surface and may have hair-like projections. Treatment is usually not required, but it resolves with acyclovir or anti-retroviral agents, tretinoin (Retin-A) or podophyllin also used. While this condition may resemble thrush, hairy leukoplakia lesions cannot be wiped off, unlike the lesions of thrush.

Actinic Cheilitis

Actinic cheilitis is a common premalignant process involving the lower lip vermilion resulting from long-term exposure to ultraviolet sunlight. The lesion develops slowly and clinical changes include atrophy of the lower lip vermilion border with blurring of the margin between the vermilion zone and the cutaneous portion of the lip. The lesion may eventually exhibit rough, scaly areas that appear leukoplakic. The scales may be peeled off but will recur within a few days. As the lesion progresses ulcerations may develop and this develop suggests the transformation to early squamous cell carcinoma.

Actinic cheilitis is probably irreversible, but patients are encouraged to use sunscreen containing lip balms in order to prevent progression of the lesion. Areas exhibiting induration, ulceration or leukoplakia should be biopsied. Clinically severe cases without invasion may be treated by lip shave (vermilionectomy).

Candidiasis

Grossly appears as superficial curdy, gray-white inflammatory membrane.

Types:

Pseudomembranous (thrush) - removable

Hyperplastic - non-removable

Mucocutaneous candidiasis - non-removable

Endocrine syndrome - non-removable.

Rx Treatment

Antifungal agent to act locally in the mouth. Examples include nystatin (eg Nystan oral suspension), Amphotericin (eg Fungilin lozenges) or Miconazole (eg Daktarin oral gel).

White spongy nevus

The mucosa appears thickened and folded or corrugated with a soft or spongy texture and a peculiar white opalescent hue. Management: No treatment.

Papilloma

Papillomas appear as pedunculated or sessile, white or normal colored cauliflower-like projections that arise from the mucosal surface. Mangement: Conservative surgical excision

Leukoedema

Leukoedema is a common oral mucosal condition of unknown etiology. Clinically the lesion appears gray-white and may be folded resulting in a wrinkled appearance. The lesion does not rub off and is typically bilateral and is most often reported on the buccal mucosa although it can involve the floor of the mouth. The process disappears or is greatly diminished when the buccal mucosa is stretched. The lesion may appear more prominent in smokers. No treatment is required.

Frictional Keratosis

Frictional keratosis is produced by mechanical irritation and is thought to be a normal hyperplastic response similar to a callus on the skin. The lesion appears clinically similar, if not identical to “leukoplakia” and therefore a search for a traumatic etiology is required in order to rule out a premalignant or malignant lesion. Clinically the lesion appears white and folded with a rough surface. The lesion does not rub off. The white lesion is reversible with the removal of the trauma.

Morsicatio buccarum / linguarum

Chronic chewing on either the buccal mucosa or tongue produces a white ragged lesion. The lesion may appear as a linear or diffuse lesion and commonly involves the lateral surface of the tongue. The lesion is usually found in people who are under stress. Lesions are usually bilateral on the buccal mucosa and may also involve the lips. The lesions appear thickened and ragged with intervening areas of erythema, erosion or traumatic ulceration. No treatment is necessary. Acrylic shields can be fabricated to protect the areas from chronic low-grade trauma.

Fordyce Granules

Fordyce granules are sebaceous glands that occur on the buccal mucosa. Their presence is considered a variation of normal. The lesions present as multiple yellow or yellow-white papules on the buccal mucosa and lateral portion of the vermilion of the upper lip. They may also be present in the retromolar area and the anterior tonsillar pillar. They are more common in adults. The lesions may appear rough to the patient but are otherwise asymptomatic. No treatment is required.

Snuff lesion (smokeless tobacco lesion)

The lesion develops on the mucosa where smokeless tobacco is held. The usual appearance is white, wrinkled or corrugated mucosa. Gingival recession is a common manifestation with cervical erosion of teeth a less frequent finding. Symptoms are uncommon. Treatment: Biopsy should be done to rule out dysplasia, otherwise no treatment is necessary.

Trismus

Motor disturbances of the trigeminal nerve,especially spasm of the masticatory muscles,with difficulty in opening the mouth(Lock jaw).

Causes

Clinical Examinations:

Inspection,palpation,Auscultation in and around the temporomandibular joint and muscles of mastication.

Investigations

- OPG
- CT scan
- MRI

Management:

1. Before ustarting treatment reassure the patient
2. Anti inflammatory drugs and analgesics
3. Antibiotics in cause of infection
4. Space infection- incision and drainage
5. Muscle relaxants and mouth gags followed by physiotheraphy
6. Tetanus – IM antitetanus injection followed by Antibiotics
7. Tetany – IV of calcium gluconate 10 mg
8. Splints(soft as well as anterior bite planes)
9. Fractures-Reduction and fixations either closed or open under LA or GA.
10. Coronoidectony –coronoid hyperplasia
11. Surgery

Trismus

Causes

Articular

1. Ankylosis
   * Fibrous
   * Bony
2. Arthritis
   * Stills disease (Children)
   * Rheumatoid (Adults)
3. Pyogenic arthritis
4. Osteoarthritis
5. Psoriatic arthritis
6. Reiter syndrome
7. Gout
8. Harie strumpell disease
9. Congenital syphilis
10. Fracture condyle

Extra Articular

1. Myositis ossificans of the masseter
2. Scarring of the temporalis
3. Fibrosis of pterigomandibular raphae due to cleft palate surgery
4. Submocous fibrosis
5. Masticatory space infection
   * Pericoronits
   * Medial pterigoid muscle infections
   * Submassetric space infection
   * Mumps
   * Parotid infections
6. Malignancies of the oral cavity
   * either due to infiltration or due to pain
7. Coronoid hyperplasia
8. fratures involving coronoid, zygomatic arch and zygomatic complex
9. Osteomyelitis
10. systemic causes
    * Tetanus
    * Tetany
    * Conversion disorder.

Oral Cavity Disease

Recurrent Aphthous Stomatitis (RAS)

Patients with RAS will complain of recurrence of one or more painful oral ulcers at intervals ranging from days to months. RAS usually begins in childhood or adolescence and may decrease in both frequency and severity with age. Shallow ulcers caused by RAS are confined to the soft mucosa of the mouth, or nonkeratinized mucosa that are not immediately adherent to bone

Minor aphthae are less than 1cm in diameter and heal completely in 7 to 10 days. The minor aphthae usually involve a prodromal stage of tingling and burning for 1 to 2 days and usually occur in clusters of up to 5 ulcers. These lesions are shallow and round to oval in shape with a gray to yellow membrane. Minor aphthae are very painful for about 4 days, then heal completely without scarring after several more days. Major aphthae are uncommon and involve irregular deep ulcers of 1 to 3 cm in size. They may have a raised border and can take 4 to 6 weeks to heal. Major aphthae can leave extensive scarring and distortion with healing, and patients are rarely lesion free. The irregular and chronic nature of these lesions often necessitates a biopsy to rule out squamous cell carcinoma

The role of nutrition is controversial. Deficiencies in vitamins, zinc, and iron have been implicated as the occurrence of RAS improved somewhat with replacements. Some patients with gluten-sensitivities may experience outbreaks that resolve with a gluten-free diet, but lesions in the majority of patients do not respond with dietary measures. Sensitivities to foods such as nuts, chocolate, cereals, tomatoes, dairy products and citrus fruits have been implicated also, and the avoidance of such foods may decrease outbreaks. Trauma, as mentioned above, may incite outbreaks.

Preparations of benzocaine, diclonine, or benzydamine can be effective. Also, as described above, mixtures of lidocaine, diphenhydramine, and Kaopectate may provide some relief. Other therapies that have been reported include hydrogen peroxide, phenol, silver nitrate, topical antimicrobials, antivirals, and antiseptic mouthwashes. These treatments are generally not very effective. The mainstay of treatment of RAS is topical steroid application. Triamcinolone 0.1% in a cream, paste or an aqueous base is the most commonly used. If applied in the prodromal stage, outbreaks can be prevented or even aborted. Beclomethasone spray has also been shown to be of benefit in treating RAS. If patients have a large number of lesions or long duration of attacks, a “burst regimen” of systemic steroid treatment may be used in addition to topical therapy. For RAS major that is difficult to control, intralesional triamcinolone injection will often promote ulcer healing. Because oral candidiasis has been reported in patients using steroid sprays and solutions, prophylaxis with antifungal agents should be considered in these patients.

Squamous cell carcinoma

Squamous cell carcinoma (SCC) is the most common malignancy of the oral cavity. These lesions often begin as mixed white or red lesions and occur most often on the tongue, floor of mouth, or soft palate. Later lesions will often involve irregular ulcers with indurated or heaped margins Factors on examination are course enlarged cervical nodes.

Oral Lichen Planus - OLP

Aetiology

The aetiology of OLP is unclear, Usually no aetiological factor is identifiable but a minority of cases are related to Reactions to amalgam or gold, or occasionally to other Dental materials.

Clinical Features

The oral lesions of OLP are usually:

Bilateral; Posterior; In the buccal (cheek) mucosa; Not seen on the palate; Lesions are usually white and may be asymptomatic or may cause soreness, especially if erosive.

Presentations include:

A network of raised white lines or striae (reticular pattern) White papules, White plaques, simulating leukoplakia, erosions are less common; if persistent, irregular, and painful, with a yellowish slough. Occasionally it is red and atrophic. Lichen planus is one of the most common causes of desquamative gingivitis. Symptomatic OLP may respond to topical corticosteroids, and patients should be informed about the condition. Recalcitrant lesions can be managed with intralesional corticosteroids or topical cyclosporin. Systemic immunosuppressive agents, including corticosteroids, azathioprine, cyclosporin or dapsone – or, rarely, vitamin A derivatives such as etretinate – may be required but should be given only by a specialist in view of the serious adverse effects possible.

Behcet's Syndrome

Behçet described a symptom complex consisting of recurrent aphthous ulcers of the mouth, as well as recurrent painful ulcers of the eyes and genitals.The diagnosis of behçet's disease is made on the basis of the clinicopathologic findings, which may be confused clinically with stevens-johnson syndrome and reiter's disease. The oral manifestations of behçet's disease may be treated in the same manner as those not associated with the disease.

Benign Mucus Membrane Pemphigoid (BMMP) and Bullous Pemphigoid:

BMMP occurs in the 40 to 50 year old age group and affects women twice as often as men. Tense oral bullae form and may persist for days before rupturing, forming large erosions that usually scar with healing. Involvement of the conjunctivae is relatively common and may lead to blindness. Bullous pemphigoid affects older patients and cutaneous lesions are more common than mucosal lesions. This condition is often associated with the prescribing of various medications. The lesions are similar but may heal with less scarring. Treatment is often difficult and multiple sites are involved but potent topical steroids or intralesional steroids can be effective. Systemic steroids may be required for severe cases for ocular involvement. Steroids combined with immunosuppressants maybe needed in refractory cases.

Pemphigus Vulgaris:

The lesions almost invariably initially involve the oral mucosa and the bullae are so easily ruptured that painful irregular ulcers are often the presenting lesion. Gentle rubbing of adjacent uninvolved skin may denude the epithelium, producing an ulcer or vesicle (positive Nikolsky's sign). As this causes a lesion, this test is no longer considered appropriates. Treatment with high dose steroids has greatly reduced the mortality and morbidity of pemphigus but significant morbidity from steroids has been reported as doses of 100 mg/day are often required for initial control. The dose can be tapered down to 20 mg/day over three months.

Linear IgA Disease

Linear IgA disease is a subepidermal blistering disorder. It sometimes follows infection and is rarely caused by drug allergy. Vancomycin is the most frequently associated drug, although diclofenac, cotrimoxazole, cyclosporin, lithium, penicillin, phenytoin, and sodium hypochlorite have been implicated in case reports. Drug-induced disease resolves with withdrawal of the offending agent. Most patients with linear IgA disease improve or clear with Dapsone 50 to 100mg daily. Although the condition may eventually be cured, many patients require long-term treatment as a reduction in dose of medication results in further blistering.

Erythema multiforme

Immune-mediated vesiculo-bullous eruption, are seen especially in younger men. It is characterized by serosanguinous exudates on the lips, and often target-like lesions on the skin.

The major form (Stevens-Johnson syndrome) causes widespread lesions affecting mouth, eyes, skin and genitals.

The minor form of erythema multiforme is much more common and affects only one site.

Oral lesions include:

Lips – cracked, bleeding, crusted, swollen.

Ulcers – diffuse and widespread. Oral lesions progress through macules to blisters and ulceration, typically most pronounce in the anterior parts of the mouth. Extensive oral ulceration may be seen. Management

Specialist referral is indicated, particularly in patients with major forms such as Steven-Johnson syndrome, who may need hospital care.Oral hygiene should be improved with 0.2% aqueous chlorhexidine mouthbaths. In addition, major erythema multiforme should be treated with systemic corticosteroids and/ or azathioprine or other immunomodulatory drugs. Levamisole and thalidomide have been used to some effect on occasion. Minor erythema multiforme may respond to symptomatic treatment and topical corticosteroids, but systemic steroids may still be required.

Candidiasis

Candidiasis is the most common type of fungal infection involving the oral mucous membranes. The causative organism, Candida albicans.

Oral candidiasis usually presents with profuse creamy white plaques, which cover any portion of the mouth, rub off easily, leaving a bright red, raw, bleeding surface. In some cases it may present as a brightly erythematous mucosa with only scattered white plaques. Chronic hyperplastic cadidiasis is the form of the disease that presents clinically as a leukoplakic lesion that does not rub off the underlying mucosa. The use of systemic antifungal therapy such as Fluconazole or Itraconazole may be initiated under specialist supervision.

Oral purpura

Blood blisters in the mouth are not uncommon in elderly people.

Clinical features

Blood blisters are seen in the mouth or pharynx, mainly on the soft palate (sometimes termed angina bullosa haemorrhagica) and occasionally on the lateral border of the tongue in elderly people. There is rapid onset, with breakdown in a day or two to a large round ulcer. Topical analgesics may provide symptomatic relief.

Gingival enlargement

Gingival enlargement, the currently accepted terminology for an increase in the size of the gingiva,is a common feature of gingival disease.

Causes

- Gingivitis
- Gingival enlargement may also be associated with theadministrationofthreedifferentclassesofdrugs, all producing a similar response anticonvulsants, such as phenytoin, succinimides and valproic acid calcium channel blockers, such as nifedipine and verapamil.The dihydropyridine derivative isradipidine can replace nifedipine and does not induce gingival overgrowth. Cyclosporin, an immunosuppressant also produces enlaregement of gingiva, tacrolimus is an available alternative which results in much less severe gingival overgrowth than cyclosporin, but is similarly as nephrotoxic
- Vitamin C deficiency
- Pyogenic granuloma
- Poorly fitting dentures
- Pregnancy
- Puberty
- Leukemia
- Sarcoidosis
- Fibroma / papilloma
- Gingival cyst
- Underlying osseous lesions
- During eruption.
- Physical irritation of the gingiva by improper restorative and orthodontic appliances

Clinical features

Inflammatory Pregnancy and puberty	Drug induced	Vit c deficiency	Leukemia
Gingiva is deep red, soft, friable, bleed easily, In pregnancy, sometimes tumor like lesion occurs.	Lesion is mulberry shaped, firm, pale pink, resilient, no tendency to bleed	Gingiva is deep red,soft, and friable, bleed easily and surfacenecrosis with psuedomembrane formation	Bluish red firm, friable and bleed easily

Diagnosis

Complete medical history and clinical examination.

Treatment

In case of drug induced enlargement, drug is changed with alternative drugs after consulting the Medical practitioner.

In Leukemic patient's proper blood investigations including bleeding and clotting time, platelet count should be checked before treatment. Antibiotics may be given systemically the evening before and for 48 hours to reduce the risk of infection

Inflammatory
• OHI and scaling • Scaling and root planning • Gingivectomy
Drug induced
• OHI and scaling • Gingivectomy/ flap operation
Pregnancy
• OHI and scaling • Scaling and root planning • Tumor like enlargement is treated by surgical excison
Puberty
• OHI and scaling • Scaling and root planning • Severe cases, surgical removal.
Leukemia
• OHI and Daily use of chlorhexidene mouth-washes • Scaling and root planning

Intra oral Swelling

The swelling of the intra oral regions may be due to trauma, cysts, tumours, infections, immune reactions, developmental abnormalities. Chronic swelling of intra oral regions may be painless. Intra oral swelling may or may not accompanied by extra oral swelling. The differentiation of intra oral swelling may be achieved on the basis if presence of ulceration and color of the lesion.

Causes of intra oral swelling;

- Pink:
  - * Fibro epithelial polyp
  - * Drug induced hyperplasia
  - * Warts and condyloma
  - * Focal epithelial hyperplasia
  - * Crohns diseases
  - * Orofacial granuloma
  - * Squamous cell carcinoma
  - * Salivary gland tumours
- Red:
  - * Denture induced hyperplasia
  - * Pyogenic granuloma
  - * Giant cell granuloma
  - * Squamous cell carcinoma
  - * Scurvy
- White:
  - * Squamous papillae
  - * Squamous cell carcinoma
- Blue:
  - * Mucocele and ranula
- Normal mucosal color:
  - * Sialolith
  - * Bony exostosis.

Cyst:

The cyst occurring in the oral cavity may be odontogenic cyst or non odotogenic cyst. The most common odontogenic cyst occurring in the oral cavity is Radicular cyst followed by dentigerous cyst and odotogenic keratocyst. All cyst presents clinically with a swelling with or without a sinus. Intra oral cyst may be infected or not. Infected cyst on aspiration may present with pus.Infected cyst may present with pain and tenderness. Cystic contents may be clear fluid, cloudy exudates, pus, straw color fluid. Radiologically most of the cysts presents with a clear well defined radiolucencies with cortical border. The dentigerous cyst may present with a impacted tooth. Radicualr cyst may present with a non vital tooth.

Tumours:

The tumours presenting as intra oral swelling may be odontogenic or non odotogenic variety, may be central or peripheral, epithelial or connective tissue origin. The most common tumour occurring in oral cavity is squamous cell carcinoma. Tobacco, alcohol, smoking are the predisposing factors. The patient may present with a swelling , ulceroproliferative growth with involvement of lymphnodes.Pain may accompany if it involve the adjacent structures or supper added with infections. Radio graphically the tumour may involve bone with erosion or infiltration of adjacent structures. Maxillary carcinoma usually extends into the nasal septum involving maxillary sinus and orbital floor. Metastatic carcinoma of mandible can also occur which results in paresthesia of lips. Diagnosis is confirmed by histopathological examination. The treatment of oral cancer is surgery, radiotherapy or surgery combines with radiotherapy. Among the connective tissue tumours

primary tumour of bone which is more common is osteosarcoma. The prognosis of osteosarcoma is poor. It's a radio resistant tumour. The treatment is surgery may or may not combined with chemotherapy

Trauma:

It's the common cause of intra oral swelling. The trauma to the oral structures may or may not accompanied with fracture of maxillae or mandible. The maxillary fractures are classifies as Lefort I, II, III fractures. The mandibular fractures are classified according to the location as symphysis, para symphysis, body, angle, ramus, condyle or coronoid fractures. The intra oral swelling due to trauma may also due to haematoma without fracture of bone. Truama to the Dental structures may result in avulsion, extrusion, intrusion, or subluxation of tooth. Clinically the patient present with swelling, trismus, deformed occlusion , step deformities and pain.

Infections:

Infections is of Dental origin or soft tissue. Odontogenic infection may be due to pulpitis or periodontal infection. The sequelae of Dental infection are granuloma, cyst, abscess, cellulitis.

Bacterial, viral, or fungal infections may manifest as intra oral swelling. The most important clinical characteristics of infection is it would be accompanied by pain and Inflammation. The treatment should be aimed at removing the cause of infection like extraction of the offending tooth or conservative management together with systemic antibiotics.

Oro facial pain

Taxonomy of IASP defines PAIN is an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage.

Emergency nature of pain

- Creates an emergency for the patient and motivates for seeking treatment
- Emergency nature of pain relates to the significance of patient attaches to it
- Relates to more to the fear that it generates then to actual intensity of discomfort.

Levels of pain processing

- Nociception
- Pain
- Suffering
- Pain behavior

Neural pathways of pain

- Transduction
- Transmission
- Modulation
- Perception

Primary pain carrying fibres

- A delta fibers
- C fibers

Classfication of Orofacial pain

Axis I(Physical conditions)

Somatic pain

- Superficial somatic pain
- Deep somatic pain musculoskeletal pain visceral pain
- Neuropathic pain episodic neuropathic pain continous neuropathic pain

Axis II (Psychologic conditions)

- Mood disorders
- Anxiety disorders
- Somatoform disorders
- Other conditions

Trigeminal neuralgia(Tic Douloureux)

- Clinical features
  - * Age- middle and old age group
  - * Gender- females commonly
  - * Episodes of intense shooting stabbing pain- few seconds and complete disappears
  - * Quality of pain- Electric shock like, unilateral, along the course of the affected nerve
  - * Maxillary branch most commonly affected
  - * Trigger zones: precipitated by light touch areas along the course of the nerve. E.g.; nasolabial fold, corner of lips
- Pain aggravated by
  - * Shaving, showering , eating, speaking ,expo-

sure to wind

- No of attacks- varies from 1- 2 per day to several per minute

Sweet diagnostic criteria

1. The pain is paroxysmal
2. The pain may be provoked by light touch to the face (trigger zones)
3. The pain is confined to the trigeminal distribution
4. The pain is unilateral
5. The clinical sensory examination is normal.

Treatment

- Drug therapy
  - Baclofen
  - Carbamazepine
  - Gabapentin
  - Phenytoin
  - Lamotrigine
  - Pimozide
  - Topiramate
  - Oxcarbazepine
  - Tiagabine
- Surgery(refractory cases)
  - Peripheral surgeries
  - Gasserian ganglion
    - Percutaneous radiofrequency
    - Thermocoagulation
    - Glycerol block
    - Compression of ganglion
    - Microvascular decompression of nerve

Glossopharyngeal neuralgia (ninth cranial nerve neuralgia)

- Characterized by severe episodic paroxysmal pain in the tonsils and ear
- Less intense than trigeminal neuralgia
- Similar clinical features as Trigeminal Neuralgia
- Precipitating factors
  - Yawning , talking
  - Chewing, Swallowing, contact with food
- Trigger zones
  - Pharynx, post tongue, ear, infraauricular retro molar area
- Differential Diagnosis:
  - Geniculate neuralgia
  - Temporo mandibular joint disorders or masticatory disorders.
- May occur with trigeminal neuralgia

Treatment

- similar to Trigeminal neuralgia
- Good response to carbamazepine and Baclofen

Atypical Facial pain/ atypical odontalgia

- Diagnostic features: considered atypical facial pain if
  - Facial pain not fulfilling other criteria
  - Chronic facial pain
  - Thoroughly investigated with no underlying cause
  - Differentiation of nerves
  - Phantom tooth pain
  - Vascular / neuropathic /
  - Associated with psychogenic component
- Clinical features:
  - Dull aching pain with apparent cause
  - Women- 4th to 5th decade
  - No trigger zones

  H/o onset coinciding with Dental procedures

  H/o multiple medications, multiple Dental procedures with temporary relief and recurrence

  Pain may remain in one area or may migrate

  Symptoms may be unilateral , bilateral or cross the midline

  May involve maxillae or mandible.

Treatment

- Least manageable form
- Tri cyclic antidepressants, Monoamine oxidase inhibitors
- Benzodiazepines- clonazepam,Transcuaneous electric nerve stimulation

- Sympathetic nerve blocks
- Psychotherapy and behavioral approaches

Oral Care of the patient receiving Radiation therapy

Acute complications:

1. Taste alteration(hypogeusia)
2. Mucositis
3. Infections

Chronic complications:

1. Trismus
2. Xerostomia
3. Radiation caries
4. Osteoradionecrosis

Oral Cancer Prior to Radiation:

A through oral examination should be performed before radiotherapy including

- Full –mouth radiograph and a panaromic radiograph
- Identification of periodontal diseases
- Bleeding index
- Pocket depth
- Tooth mobility
- Assessment of oral hygiene
- Assessment of the patients motivation to comply with the necessary preventive regimen
- Identification of all restorative needs
- Caries lesions
- Defective restorations
- Fractured tooth
- Identification of peripical infections unerupted teeth , root tips and other pathologic conditions.
- Preprosthetic surgical needs
- Potential source of irritation
- Removal of softliners which become colonized with yeast and can become a source of irritation to friable mucosa.

Oral care during Radiation Therapy:

Patient needs to be monitored weekly.

Palliative treatment of mucositis includes salt baking soda rinses Dilute chlorhexidine rinses 3- 4 times a day Rinses with diphenhydramine, kaolin with pectin and topical anaesthetics can provide topical pain relief Monitored for candidal infection Salivary substitute should be used for lubrication Good oral hygiene and daily fluoride should be reinforced

Oral care after radiation therapy:

- Patient should be observed once or twice in 1st month
- Recall schedule to prevent radiation caries, periodontal diseases and osteoradionecrosis
- Oral hygiene and fluoride protocols needs to be reinforced
- Caries lesion and demineralisation need to be treated immediately
- Placement of fluoride releasing liner or base

Ulcerative lesions of the oral cavity

Trauma

Trauma is the most common cause of ulceration of the oral mucous membranes. Traumatic ulceration may result from physical, chemical or thermal injury to the tissue. Acute traumatic ulceration is characterized by a break in the mucosa with a shallow base and nonraised margins. Depending on the cause, they may be diffuse or localized. These lesions are at least mildly painful. Traumatic injuries to the oral mucosa are treated by removing the responsible irritant, after which healing is usually uneventful. Healing may be expedited by cleansing the tissues with a mild saline rinse with half strength peroxoide.

Chemical and thermal injuries

Chemical and thermal injuries to the oral mucosa are often more painful, requiring analgesics during the healing period. Cancer chemotherapy agents are extremely powerful drugs that have as a side effect the potential for disruption or destruction of the oral tissues. These lesions are treated palliatively with cleansing mouth rinses, topical anesthetics, antimicrobial agents, or with the use of analgesics.

Infection

Infectious causes of chronic oral ulceration are rare. Viral infection will seldom lead to development of a chronic oral ulcer in an immunocompetent patient.

Although secondary, nonspecific bacterial infection of chronic oral ulcer is common. An example of a chronic ulcerative disease caused by specific bacterial infection would include a gumma in tertiary syphilis, a tuberculous ulcer, and actinomycosis. Tuberculous ulcer is shallow and has indurated margins. Deep mycotic infections, such as histoplasmosis or blastomycosis, will typically cause chronic, deep-based ulcers that may be granulomatous and friable in appearance.

Radiation

Reactions that result from radiation therapy for malignancies in the oral cavity and oropharynx may be quite severe. Radiation mucositis is an early and acute reaction usually beginning during the second week of radiation. It usually presents as erythema followed by spotty mucositis. The spotty mucositis will coalesce to form areas of ulceration covered by a yellow-white pseudomembrane with a bright erythematous border. The lips are often involved, with a tenacious pseudomembrane and crusting being noted in the areas of ulceration. Exquisite pain and burning may be present even at rest and are exacerbated by spicy foods. Treatment is both supportive and therapeutic and includes oxidizing mouth rinses described above to help break up the thick, ropelike saliva covering the mucous membranes. By doing so, other supportive or therapeutic medications will have a greater effect. Local anesthetic rinses described above will reduce some of the discomfort and are particularly useful before meals. Topical antibiotic solutions may aid in preventing secondary infection.

Dental pain

Dental pain of pulpal origin can be because of pulpitis, apical periodontitis or apical abscess.Pulpitis may be acute or chronic.

Acute reversible pulpitis:

- Sharp momentary pain on exposure to sweet or cold present.
- It does not occur spontaneously and does not continue after the stimulus is removed.
- On vitality testing, there is early response and the pain will not linger for more than 10 to 15 seconds after cessation of stimuli.
- Treatment – Removal of the etiology and restoration of the tooth. RCT is not required.

Acute irreversible pulpitis:

- Sharp pain usually caused by hot or cold stimuli and can occur spontaneously.
- Lingering pain after removal of the stimulus is present.
- Night pain is a classical sign of irreversible pulpitis.
- On vitality testing, early response with pain lingering for more than 15 seconds is present.
- Treatment – RCT

Chronic pulpitis :

- Dull pain is present and there is delayed response in vitality testing.
- Treatment – RCT.

Apical periodontitis :

- There is pain on mastication for the patient.
- Pain on percussion is present clinically.
- Treatment – RCT.

Alveolar abscess :

- The patient has severe throbbing pain with swelling of the overlying soft tissue.
- Vitality test will be negative.
- Treatment –Drainage established and completion of RCT after subsidence of symptoms is done.

Discoloured teeth

- Tooth discolorations can be classified as either extrinsic or intrinsic.
- Extrinsic discolorations are found on outer surface of the teeth and may be due to stains from tobacco products, foods, beverages, mouth rinses, chromogenic microorganisms, etc. It can be removed by scaling and polishing.
- Intrinsic discolouration is stain within the enamel and dentin caused by the incorporation of substances within these structures. It may be because of pulp necrosis, intrapulpal haemorrhage, calcific metamorphosis, drug (eg. : tetracycline) and diseases (eg.: porphyria, erythroblastosis fetalis)
- Vital and Non-Vital bleaching techniques help in removing the stains in most of the cases

Non-Vital Bleaching

- Walking bleach technique is commonly followed. In this, after isolation of the tooth, GP is removed 2mm apical to the clinical crown.
- Polycarboxylate or GIC is placed over it to create a barrier.
- Sodium perborate mixed with saline is placed in the pulp chamber and IRM is given to seal the access opening.
- The mix should be changed weekly. Usually 1 to 3 sittings are required to achieve optimal tooth lightening.
- After successful bleaching, calcium hydroxide is placed in the pulp chamber for two weeks and then a composite restoration is given.

Vital Bleaching

- Various inoffice and outside office procedures exists (eg. Night guard bleaching).
- Common inoffice technique is using Mckenzie technique. In this 1 part anesthetic ether, 5 parts Hcl and 5 parts 30% Hydrogen peroxide is used.
- The solution is freshly mixed. After proper isolation, the bleaching solution is applied over the teeth. 3 to 5 sittings may be needed.
- There may be transient sensitivity. But there is no deleterious effect on pulp.
- Microabrasion, macroabrasion and veneering are the other treatment options for addressing discolouration not amenable to bleaching.

Facio - Maxillary Conditions

Any injury to either a soft tissue or hard tissue of face caused by an assault , road traffic accidents, sharp instruments , fall ,sports injuries or an urban violence and social trends will cause loss of soft tissue and hard tissue which has to be handled by an oral or Dental surgeon meticulously at the time of casuality.

Soft tissue injuries:

Abrasion:

Loss of epidermis and raw bleeding surface on the skin

Contusion:

Inury to the soft tissue without an external communication with swelling

Ecchymosis:

Extravasation of blood into soft tissue which results in discolouration

Haematoma:

Accumulation of blood into the tissue plains results in odema

Avulsion or Degloving injury:

Soft tissue being pulled away from its attachments leaving an exposed bone surface

Hard tissue injuries:

Fracture:External force beyond the modulus of elasticity of the bone

Types:

Traumatic fractures

Pathological fractures: fractures occurring due to normal physiological force in a diseased bone eg ; fractures occurring in resorbed jaw bones.

Simple fracture:

Closed linear fractures of bone.

Compound fracture:

Fractures which communicate to the exterior through skin or mucous membrane

Greenstick fracture:

It occurs in immature bone where one surface is compressed and opposing surface is streached leads to fracture.

Dentoalveolar fracture:

Fracture of dentate alveolus may occur as a separate clinical entity or in conjucation with other facial bone fractures and usually associated with injury to teeth like fracture,subluxation or avulsion. Fratures of Maxillary tuberosity and antral floor are relatively common complications which occurs during exodontia.

Zygomatico maxillary complex fractures:

As Zygomatic bone is closely associated with frontal,temporal and maxillary bones they are usually involved when injury occurs in the region of zygomatico frontal,zygomatico temporal and zygomatico maxillary sutures. Zygomatico maxillary complex fractures usually involves orbit also but severity of involvement depends on degree and direction of displacement.Types of displacement are

* Minimal or no displacement
* Inward and downward displacement
* Inward and posterior displacement
* Outward displacement
* Communition of the complex as a whole

Zygomatic arch fractures:

Fractures involving the zygomatic arch alone without involvement of the orbit.

Types:

1. Minimal or no displacement
2. V- type fracture in the arch
3. Comminuted fracture

Signs and symptoms of zygomatico maxillary complex fractures:

1. Flattening of cheek
2. swelling
3. Periorbital haematoma
4. Sub conjunctival haemorrhage
5. Ecchymosis and tenderness intra orally over the zygomatic buttress
6. Limitation of occular movement
7. Diplopia
8. Enopthalmus
9. Tenderness in the orbital rim and fronto zygomatic suture

10. Step deformity in the orbital margin
11. Seperation of the frontozygomatic suture
12. Limitation of mandibular movement due to impingement on the coronoid process
13. Anaesthesia of cheek,temple, upper teeth and gingival
14. Possible gagging posterior segment in the injured side

LeFort I fracture (Guerin fracture)

This is a horizontal fracture above the level of nasal floor.The frature line extends backwards from the lateral margin of anterior nasal aperture below zygomatic buttrsss to cross the lower third of the pterigoid laminae.

LeFort I fracture may occur as a single entity or inassociation with LeFort II or III fractures.

Signs and symptoms:

1. Ecchymosis in the buccal segment beneath each zygomatic arch
2. Ecchymosis of the greater palatine foramen area(Guerin sign)
3. Swelling of upper lip
4. variable amount of mobility in the tooth bearing segment of the maxilla
5. Percussion of upper premolar teeth will produce a distinctive “cracked pot” sound

LeFort II fracture (Subzygomatic or pyramidal fracture)

This involves nasal bone,medial wall of orbit,frontal process of maxilla,infra orbital margin,lateral wall of the antrum below the zygomatico maxillary suture and devides the pterigoid plate half way up.

Signs and symptoms:

1. Step deformity in the infra orbital margin
2. mobility in the midface at the nasal bidge and the infra orbital margins
3. paresthesia of cheek
4. Gagging of occlusion and retro positioning of maxilla as a whole
5. Mid line or para medial split in the pyramidal block

LeFort III fracture(supra zygomatic or high tansverse fracture,craniofacial dysjunction)

Starts below the fronto nasal suture involves the ethmiod bone including The cribriform plate and orbit as a whole below the optic foramen separating the frontozygomatic suture and the roof of the pterigoid plate.The entire middle third becomes detached from the cranial base.

Signs and symptoms:

1. Tenderness and deformity of zygomatic arches and separation at the front zygomatic suture
2. Dish face deformity
3. Lengthening of face
4. Profuse cerebrospinal fluid rhinorrhea
5. Depression of ocular levels and “hooding of eyes”
6. Enophtholmos
7. Flattening of malar area with step deformity and tenderness over the infra orbital margin
8. Tilting of occlusal plane and gagging on one side only
9. Mobility of whole of facial skeleton as a single block

Facial deformities

Facial asymmetry can be analyzed in frontal and profile analysis

- Frontal estimation
  - * Rule of fifths- face is divided into central, medial and lateral equal fifths.
    - » Separation of the eyes and the width of the eyes must be equal
    - » Nose and chin should be centered in the chin
    - » The interpupillar line coincides with the width of the mouth.
    - » Nasal length should be 1/3rd of the total facial height
  - * Vertical facial thirds-face divided from hairline to the base of the nose, base of the nose to the bottom of the nose and bottom of the nose to chin –all three should be equal
    - » Lower third of the face is slightly longer
    - » Lower third has a proportion of 1/3rd to 2/3rd.
- Profile analysis-
  - * Facial profile-land marks are soft tissue na-

sion, subnasale and soft tissue position-the first line dropped from soft tissue nasion to subnasale and second line from subnasale to soft tissue pogonion- this helps in analyzing antero-posterior positioning of the jaws.

» Straight profile-the line form a straightline

» Convex profile- class11 skeletal pattern

» Concave profile-class111 skeletal pattern

* Facial divergence-inclination of the face in relation to forehead

» Posterior divergent- seen in class11 cases

» Straight face-class1 cases

» Anterior divergent-class111 cases

* Head type-determined by the maximum skull width and maximum skull length

» Mesocephalic-avearge shaped head

» Brachycephalic-broad and round head

» Dolichocephalic-long and narrow head

* Facial form-estimated by dividing morphological facial height by bizygomatic width

» Mesoprosopic-average facial form

» Euryprosopic-broad and short facial form

» Leptoprosopic-long and narrow facial form

* Treacher Collins syndrome-diagnosis by

» External ear deformed/hypoplastic

» Hypoplasia of malar bones

» Ptosis of eyelid

» Micrognathism

» Investigation- tomographic examination

» Radiographic

* Hemifacial microsomia-diagnosis by

» Macrostomia, symmetric microtia(smallear)

» Hypoplasia of condylar and ramal portions

» Ear abnormality and hearing loss

» Upper eyelid colobomas

» Parotid gland does not develop

» Facial asymmetry increases with age

* Aperts syndrome-hypertelorism

» High arched palate

» Midface deficiency

» Investigation- radiographic assessment

* Crouzons syndrome

» Shows shallow orbits with proptosis

» Midface deficiency

* Fetal alchoholic syndrome

» Short palpebral fissures

» Flat midface

» Short nose and thin upper lip

* Cleft lip and cleft palate-most common

» infant faces problem of regurgitation and-later stages speech deformities

» initial management of feeding plate

* Chin deformity - in this type of face defect / deformity the chin is unusually small (mirognathia) or may be unusually large (macrognathia)

* Upper jaw (maxillary) deformity - one of the most common type of upper jaw (maxillary) deformity is called vertical maxillary excess. In this facial defect, there is excess bone of the upper jaw, the face appears long, the chin is recessed and the nose large in the profile view.

* Lower jaw (mandibular) deformity - there are two most common mandibular deformities; mandibular excess (protrusion) and mandibular deficiency (retrusion).

* Mandibular asymmetry- most commonly due to acceletrated growth of condyle on one side or decreased growth of condyle on other side investigation by clinical examination and postero-anterior cephalometry

Fractures of the Mandible

Fractures of mandible are more common than the fractures of middle third. The commenest of mandibular fractures is the fracture of condyle and angle fracture if one side is involved.followed by para symphysis,subcondylar,body, symphysis and rarely coronoid fracture.

Causes

1. Direct impact
2. Indirect impact
3. Excessive muscular contracture

The direction and type of impact is more important in mandibular fractures than in middle third fractures.

Types:

1. Unilateral fracture
2. Bilateral fracture
3. multiple fracture
4. comminuted fracture

Signs and symptoms:

1. Tenderness ,Swelling and ecchymosis at the sites of injury
2. Anterior Open bite in case of bilateral condylar fracture
3. Deformity seen and crepitus elicited in the fractured site
4. Sub lingual haematoma in case of para symphsis fracture
5. Limitation in opening and closing the mouth properly
6. Bleeding from the external ear without condylar movement suggestive of condylar fracture
7. Mobility in the fractured segments
8. Parade ground fracture: fracture involving bilateral condyle wiyh symphsis

Investigations

1. OPG
2. PNS view
3. IOPA
4. PA view mandible
5. Sub-mentovertex view
6. Lateral oblique view
7. CT scan
8. 3D CT scan

Management:

1. Airway maintenance with cervical spine control
2. Breathing and ventilation
3. Circulation and haemorrhage control
4. Disabilty and neurologic status
5. Exposure(undress) with temperature control

Soft tissue management:

1. Wound debridement with antiseptic solution
2. Look for neurovascular cut injury.if necessary ligate the vessels and reposition the neurovascular bundles
3. Primary closure within six hours of injury
4. Layerwise closure is necessary
5. Antibiotics and analgesics mandatory

Hard tissue management:

Depending upon the type and site of fracture closed or open reduction is done.Tooth in the line of fracture should be removed.

Closed reduction: Maxillo mandibular fixation. Usually 4 to 6 weeks is required depending upon the type of fracture.Condylar fracture requires a period of 3 weeks.

Open reduction: Done under local or general anesthesia

Procedures:

* Miniplate fixation with screws
* Transosseous wiring

Post operative care:

1. Soft diet and liquids
2. Control of infection
3. Oral hygiene maintainence by using mouth rinses
4. Reassurance of the patient

Dental Impaction

Definition

It is a failure of tooth to erupt into the oral cavity beyond the chronological age due to mechanical impingement, this may be a soft or hard tissue obstruction.The most commonly impacted tooth is mandibular third molar followed by upper third molar, upper canine and lower canine.

Aetiology:

1. Genetic or familial
2. Lack of space in the alveolar arch.
3. Discrepancy of the jaw size and the width of the tooth
4. Dense sclerotic bone or scar of the over lying mu-

coperiosteum

5. lack of eruptive force from the developing roots
6. Pericoronitis or Operculitis
7. Dental caries
8. Masticatory space infection

Classification:

Winters classification for Third molars:

It gives the position of the impacted tooth in relation to the long axis of the second molar ,the thickness of soft tissue covering in relation to the external oblique ridge and the depth at which the point of application to be located.

Types:

1. Mesio angular
2. Vertical
3. Horizontal
4. Disto angular
5. Buccoverted
6. linguoverted
7. Inverted

Investigations

1. IOPA
2. Palatal
3. Intermediate
4. Horizontal
5. Vertical
6. Ectopic eruption (Maxillary sinus,Nasal cavity,orbital cavity)

Management:

1. Antibiotics and analgesics
2. Incision and drainage in case of space infection
3. Surgical removal of tooth

Dental Malocclusion

Malocclusion irregularities in tooth position arch relation

1. Proclination of incisors- always rules out underlying cause like abnormal habits.
   * Mild proclined incisors
     » Hawley's appliance
     » If space present distal to canine then hawley's with long labial bow
   * Moderate proclined incisors-
     » Roberts retractor
     » High labial bow with apron spring
   * If severe proclination- refer to an orthodontist
2. Spacing in the dentition-rule out underlying cause by clinical examination
   * If because of single tooth malformation-single tooth can be capped
   * If because of microdontia-refer to orthodontist for fixed appliance
   * If generalized anterior spacing-hawley's appliance with the base plate trimmed on the palatal aspect
   * If median diastema –
     » Upper hawley's appliance with two finger springs to move the centrals mesially
     » Upper base plate with split labial bow
     » If midline diastema due to flaring and proclination of incisors the simple hawley's appliance with short labial bow
3. Crowding in dentition-
   * If mild crowding is present in developing dentition- no treatment needed except for constant observation
   * If moderate crowding present then guidance of eruption of canines and premolars should be given and also can be managed by expansion and observation
   * If severe crowding in developing dentition
     » Expansion
     » Guidance of eruption
     » Serial extraction
     » Extraction and fixed appliance
4. Anterior cross bite
   * Tongue blade
   * Avoid early mandibular displacement and traumatic occlusion
   * Hawley appliance with posterior bite plane and zspring to correct the tooth in cross bite
   * Upper anterior expansion with posterior bite

plane

* Inclined plane if the bite is deep

5. Posterior cross bite
   * If single tooth cross bite then cross elastics can be given
   * Schwarz type lateral expansion with posterior bite plane for occlusal clearance
   * Hawley appliance with posterior bite plane and t springs or z springs to correct the tooth in cross bite
   * Quad helix ,w arch, coffin spring type of appliance for both unilateral and bilateral expansion
6. Canine mal position
   * Buccally placed canine
     » Buccal canine retractor when palatal and distal movement is needed
     » Helical loop retractor when the sulcus depth is shallow
   * Palatally placed canine-
     » Hawley appliance with z spring and posterior bite plane whenonly buccal movement is required
     » Palatal canine retractor with posterior bite plane when bothdistal and buccal movement of canine is required
     » Radiographic examination is essential to rule out impacted and other pathology
7. Buccally placed premolars needs spring for palatal movement
8. Deep over bite-upper hawley's with flat anterior bite plane
9. Anterior open bite
   * Upper hawley's with tongue spike if due to habits
   * Posterior bite plane to intrude molars
   * Dentoalveolar problems oriented due to malocclusion
     » Difficulty in chewing
     » Speech difficulty
     » Difficulty in maintaining oral hygiene
     » Tendency to bite cheek / roof of the mouth
     » Suffers pain in the facial muscles on the jaw
     » Deflection of permanent teeth to erupt in wrong position

Preventive aspects

1. At birth-teeth present (natal teeth)-commonly mandibular anteriors
   * Management-no extraction needed except when interference in breast feeding and extreme mobility (danger of inhalation)
2. Primary dentition— lack of spacing –suggestive of crowding in permanent dentition
3. Transition from primary to mixed dentition(6-12yrs) ugly duckling stage(spacing between incisors and laterals)
   * No treatment needed and not to be considered as mo
   * Asymmetry in the eruption between both sides of an arch with a difference of 6months needs radiographic examination to rule out the cause
4. Premature loss of deciduous tooth – if natural there will not be much of impact
   * If due to extraction,caries or fracture then it may lead to arch form collapse due to drifting of tooth into extracted site resulting in impaction of the permanent successors, asymmetry of arch form
   * Refer to orthodontist for a space maintainer
5. Retained deciduous tooth- if permanent successors present and there is a difference of 6months compared to contra lateral side –retained deciduous can be extracted after confirming the presence of permanent tooth
   * Investigation-radiographic examination
6. Infraoccluded submerged tooth-results in ectopic eruption of molars and canines-these tooth can be extracted if they are
   * Submerged below the gingival level
   * If root formation of permanent successor going to complete
   * Investigation-radiographic.
7. Impacted permanent first molars-results in crowding-overlying deciduous tooth should be extracted and refer to orthodontist .

8. Dilacerations-causes failure of eruption
   * If severe – extraction is preferable
   * If mild – refer to orthodontist for traction to align the tooth
9. Supernumerary tooth-causes failure of eruption displacement, crowding and sometimes no effect
   * Extraction is preferable
10. Permanent dentition (after 12yrs) - malocclusion should be referred to orthodontist

Cyst And Tumors Of Odontogenic And Non Odontogenic Origin

Cyst:

It is a pathological cavity lined by epithelium which may contain liquid,semi solid or air.It can occur in both hard and soft tissues.Soft tissue cysts are surrounded by a definite connective tissue wall or capsule.

Classification:

Congenital cysts:

1. Thyroglossal
2. Branchiogenic
3. Dermoid and epidermoid

Developmental cysts:

Fissural types:

1. Naso alveolar
2. Median
3. Nasopalatine
4. Median palatal
5. Globulomaxillary

Retention types:

1. Mucocele
2. Ranula
3. Plunging ranula

Bone cysts:

1. Solitary bone cysts
2. Aneurysmal bone cyst

Odontogenic epithelial origin:

Keratinizing(kerato cyst):

1. Primordial cyst
2. Extra follicular dentigerous cyst
3. Calcifying odontogenic cyst
4. Gingival cyst of adults

Non keratinizing cysts :

1. Periodontal cyst:
   * Periapical
   * Lateral
   * Residual
2. Dentigerous cyst:
   * Pericoronal
   * Lateral
   * Residual
3. Eruption cysts
4. Glandular odontogenic cysts

Cysts of the maxillary antrum:

1. Benign mucosal cyst
2. Surgical ciliated cyst

Diagnostic features:

1. Expansion of alveolar bones
2. Egg shell crackling on palpation
3. Dull or hallow sound on percussion of the tooth in the cystic area
4. Apical cysts are always associated with non vital tooth
5. An acute infection of cyst will produce neuroparaxia
6. Impacted tooth may be associated with dentigerous cyst

Investigations

1. Aspiration: done under aseptic condition with topical anaesthesia.Cystic fluid is straw coloured and contain cholesterol crystals. Protien content of 4gms/dl suggests odontogenic keratocyst .
2. Radiographic examination:Well circumscribed round or oval areas of radio leucency with a radio opaque margin
3. Biopsy: a small portion of the cystic linig is taken under local anaesthesia for HPE.Proper closure is mandedatory after biopsy for prevention of infection.

Management:

The treatment of cysts hinge on four salient principles

1. Removal of the cyst lining
2. Conservation of erupted healthy tooth in the cystic area
3. Preservation of the adjoining healthy structures
4. Restoration of the affected area to its anatomy

Surgical procedures:

There are two standard procedures irrespective of type of cyst present in oral cavity or elsewhere in the body. They are

1. Marsupialization
2. Enucleation

Marsupialization:

Done under local or general anesthesia. Incision is placed and muccoperiosteal flap is elevated ,adequate window(fenestration) is made on the outer wall of the cyst and the contents are evacuated.the linig is left in situ and its margins are sutured with the adjacent mucosa.

Enucleation:

Enucleation is en masse removal of the cyst log with its lining.The space is closed with mucoperiosteal flap, and is filled by blod clot which eventually forms normal bone.

Methods:

- Enucleation and packing
- Enucleation with primary closure
- Enucleation with primary closure and reconstruction with bone grafts

Tumors:

Neoplasm: A neoplasm is often considered as an independent uncoordinated new growth of tissue which is potentially capable of unlimited proliferation and which will not regress after removal of the stimulus which produced the lesion.

Classification:

Clinical features:

- Benign tumors:
  - * Encapsulated tumor
  - * Base of tumor is of two types Sessile
  - * Pedunculated-attached by a stalk to the surface.
  - * Not involving the adjacent tissue
  - * Painless and slowly growing
- Malignant tumors:
  - * Ulcero proliferative lesion
  - * Invading into adjacent structures
  - * Pain, Parasthesia
  - * Trismus
  - * Bony erosion
  - * Wobbling teeth
  - * Not encapsulated
  - * Palpable Lymph nodes
  - * Pathological fracture of the bone

Examination:

Examination of the ulcer should be done like this

- * Site
- * Size
- * Shape
- * Margin-Indurated , everted.

Lymph node examination is also done.

Investigations

1. Clinical examination
   - * Inspection
   - * Palpation
2. Biopsy
   - * Exisional
   - * Incisional
3. FNAC
4. Conventional radiographs
   - * OPG
   - * PNS
   - * Occlusal
   - * Lateral oblique
5. CT scan

6. MRI
7. Vascular lesion: If the aspirated fluid from the lesion clots in about 5 minutes it confirms a vascular lesion

Odontogenic tumors (Modified WHO classificaion)

Benign

- Odontogenic epithelium without odontogenic ectomesenchyme.
  - Ameloblastoma
  - Squamous odontogenic tumor
  - Pindborg tumor(CEOT)
  - Adenamatoid odontogenic tumor
- Odontogenic epithelium with odontogenic ectomesenchyme with or without hard tissue formation
  - Ameloblastic fibroma
  - Ameloblastic fibro - dentinoma
  - Ameloblastic fibro - odontoma
  - Odonto ameloblastoma
  - Complex odontoma
- Miscellaneous
  - Myxoma

Malignant

- odontogenic carcinoma
  - Malignant ameloblastoma
  - Primary intra osseous carcinoma
  - Clear cell odontogenic carcinoma
  - Ghost cell odontogenic carcinoma
- Odontogenc sarcoma
  - Ameloblastic fibrosarcoma
  - Ameloblastic fibro - dentinosarcoma
  - Ameloblastic fibro - odontosarcoma

Non Odontogenic tumors

Benign

- Papilloma
- Myoblastoma
- Fibroma
- Lipoma
- Myxoma
- Leiomyoma
- Gingival tissue origin
  - Pyognic granuloma
  - Pregnancy tumor
  - Peripheral giant cell tumor
  - Epulis
  - Gingival hyperplasia
- Vascular origin
  - Blue nevus
  - Haemangioma
  - Lymphangioma
  - Haemangioendothelioma
  - Haemangiopericytoma
- Odontogenic tumors of jaws:
  - Torus palatinus
  - Torus mandibularis
  - Multiple exostosis
  - Osteoma
    - Peripheral
    - Endosteal

Malignant

- Carcinoma
  - squamous cell carcinoma
  - Verrucous carcinoma
  - Undifferntiated carcinoma in children
  - Adenocarcinoma
  - Multiple primary carcinoma
  - Malignant melanoma
  - Metastatic carcinoma
  - Transitional cell carcinoma
- Sarcoma
  - Fibrosarcoma
  - Odontogenic fibrosarcoma
  - Neurogenic fibrosarcoma
  - Synovial fibrosarcoma
  - Rhabdomyosarcoma
  - Liemyosarcoma
  - Lymphosarcoma
  - Reticulum cell sarcoma
  - Angiosarcoma
  - Ewings tumor
  - Multiple myeloma
  - Osteogenic sarcoma
  - Chondrosarcoma

Radiographic Findings Of Odontogenic Tumors:

Ameloblastoma- multilocular radioleucency or honeycomb appearance

Pindporg tumor- driven snow appearance

Adenomatoid odontogenic tumor (AOT) - Unilocular radioleucency below lateral incisor and canine in anterior maxillary segment.

Cementoblastoma-IOPA shows relation to the apex of the tooth.

Odontomas- Irregular mass of calcified material surrounded by radioleucent band and tooth like structures seen.

TNM classification

T

Primary tumor

Tx

Primary tumor that cannot be accessed.

T0

No evidence of primary tumor

T1

Tumors greater than 2cms or less in its greatest diameter

T2

Tumors greater than 2 cms and less than or equal to 4 cms in its greatest diameter

T3

Tumors greater than 4 cms in its greatest diameter

T4

(Lip) Tumor invades through cortical bone, infra alveolar nerve, floor of mouth, skin of facei.e., chin and nose.

T4a

(Oral cavity) Tumors invade in to cortical bone, into deep structures (Extrinsic muscles of tongue),Maxillary sinus or skin of the face

T4b

Tumor involving masticatory space, pterigoid plates,skull bone, and/or Internal carotid artery.

N

Regional lymph nodes

Nx

Nodes cannot be assessed.

N1

Ipsilateral lymph node less than or equal to 3 cms.

N2A

Ipsilateral node greater than 3 cms but less than or equal to 6 cms

N2B

Ipsilateral multiple nodes less than or equal to 6 cms

N2C

Bilateral nodes less than or equal to 6 cms

N3

Nodes greater than 6 cms which are fixed

Mo

No metastasis

M1

Distant metastasis

Staging of TNM group:

Stage 0

Tis No Mo

Stage I

T1 No Mo

Stage II

T2 No Mo

Stage III

T3 No Mo

T1 N1 Mo

T2 N1 Mo

T3 N1 Mo

Stage IV A

T4a No Mo

T2 N2 Mo

T4a N2 Mo

Stage IV B

Any T N3 Mo

T4b any N Mo

Stage IV C

Any T Any N M1

Treatment plan:

- Excision of lesions which are less than 1 cm diameter
- If lesion is more than 2 cms excision with wide margin

  For lesions of T3 and T4 excision of muscle , bone and the overlying skin

Drug interactions

Drug interaction

Is a situation in which a substance affects the activity of a drug, i.e. the effects are increased or decreased, or they produce a new effect that neither produces on its own.

Drug interactions may be the result of various processes. These processes may include alterations in the pharmacokinetics of the drug, such as alterations in the Absorption, Distribution, Metabolism, and Excretion (ADME) of a drug. Alternatively, drug interactions may be the result of the pharmacodynamic properties of the drug, e.g. the co-administration of a receptor antagonist and an agonist for the same receptor.

Metabolic drug interactions

- Many drug interactions are due to alterations in drug metabolism
- Enzyme induction- drug A induces the body to produce more of an enzyme which metabolises drug B. This reduces the effective concentration of drug B, which may lead to loss of effectiveness of drug B. Drug A effectiveness is not altered. Eg. Cytochrome p450 oxidases.
- Enzyme inhibition- drug A inhibits the production of the enzyme metabolising drug B, thus an elevation of drug B occurs possibly leading to an overdose.
- Bioavailability - drug A influences the absorption of drug B.

Drug interactions significant in Dentistry:

Amoxycillin:

1. Probenecid retards the excretion of penicillin, so a dose of penicillin persists longer when taken with it, and it allowed patients to take less penicillin over a course of therapy
2. By inhibiting colonic flora it may interfere with deconjugation and nterohepatic cycling of oral contraceptives- failure of oral contraception.
3. Amoxicillin and clavulanic acid have synergisic effects.

Erythromycin:

- Inhibits hepatic oxidation of many drugs- increase plasma levels of theophyline, carbamazepine, valporate, ergotamine, warfarin, terfenadine, astemizole, cisapride.

Ciprofloxacin:

1. Plasma concentration of theophylline, caffeine, and warfarin are increased by ciprofloxacin due to inhibitor of metabolism.
2. NSAIDs may enhance the CNS toxicity of Fluroquinolones. Seizures have been reported.
3. ANT acids, Sucralfate, and iron salts given concurrently reduce absorption of fluoroquinolones.

NSAIDS:Aspirin:

1. Displaces warfarin , sulphonyl ureas , phenytoin and methotrxate from bining sites on plasma proteins. Toxicity may occur
2. Inhibits tubular secretion of uric acid and antagonizes uricosuric action of probencid
3. Blunts diuretic action of furosmide and thiazides and reduces the action of spironolactone
4. Aspirin reduces preotein bound iodine levels by displacement of thyroxine.

Ibuprufen:

1. Should be avoided the use with anticoagulant
2. Decrease diuretic and anti hypertensive action of thiazides, furosemide, and beta- blockers

Chapter 24

Guidelines for Searching Medical Literature Online

Why Not Google, Yahoo, Bing etc

- Because all these search engines do not differentiate between authentic sources (medical journals) and non authentic sites (personal blogs, advertisement sites etc)

WHAT IS PUBMED?

- PubMed is a popular index to medical journal literature
- Can search and retrieve a list of articles on a broad or very narrow topic. Eg:
  - Lung diseases in children
  - Does drug 'A' administered by IV to infants cause growth retardation

PubMed Overview

- NLM has been indexing the biomedical literature since 1879, to help provide health professionals access to information necessary for research, health care, and education.
- What was once a printed index to articles, the **Index Medicus**, became a database now known as MEDLINE. MEDLINE contains journal citations and abstracts for biomedical literature from around the world.
- Since 1996, free access to MEDLINE has been available to the public online via PubMed.
- PubMed is a Web-based retrieval system developed by the National Center for Biotechnology Information (NCBI) at the National Library of Medicine. It is part of NCBI's vast retrieval system, known as Entrez.
- PubMed is a database of bibliographic information drawn primarily from the life sciences literature.
- PubMed contains links to full-text articles at participating publishers' Web sites as well as links to other third party sites such as libraries and sequencing centers.
- PubMed provides access and links to the integrated molecular biology and chemistry databases maintained by NCBI.

What's in PubMed?

- Most PubMed records are MEDLINE citations.
- Other records include those in different stages of processing (including records provided directly from the journal publisher) but destined to be MEDLINE citations.
- A relatively small number of records that are included in PubMed but not selected for MEDLINE.

MEDLINE Citations

- PubMed provides access to MEDLINE®, the National Library of Medicine's premier bibliographic database containing citations and author abstracts from approximately 5,200 biomedical journals published in the United States and in other countries.
- The scope of MEDLINE includes such diverse topics as microbiology, delivery of health care, nutrition, pharmacology and environmental health. The categories covered in MEDLINE include everything from anatomy, organisms, diseases, psychiatry, and psychology to the physical sciences.
- MEDLINE currently contains over 17 million references dating back to 1948.
- New material is added Tuesday through Saturday.
- Coverage is worldwide, but most records (about 90%) are from English-language sources or have English abstracts.
- Approximately 79% of the citations are included with the published abstract.
- The National Library of Medicine leases MEDLINE data to researchers and commercial vendors, but PubMed provides free access to MEDLINE directly from NLM.

MEDLINE® Basic Bibliographic Citation

One MEDLINE citation represents one journal article and is composed of fields that provide specific information (Title, Author, Language, etc.) about the journal article. The following information is generally provided:

- Title of the journal article
- Names of the Authors
- Abstract published with the article
- Controlled Vocabulary search terms (Medical Subject Headings)

- Journal Source Information
- First Author Affiliation
- Language in which the article was published
- Publication Type (description of the type of article, e.g., Review, Letter, etc.)

A sample MEDLINE citation is displayed.

How Citations Get Into PubMed

- Records are either supplied electronically by publishers or created using scanning and Optical Character Recognition (OCR) at NLM.
- Citations are immediately made available via PubMed. All citations go through a quality control process, and citations from MEDLINE journals are indexed.
- All citations display a status tag, which indicates their stage of processing.

The next several screens explain the various status tags.

Publisher Supplied Citations

- Most PubMed citations are transmitted electronically to PubMed directly by publishers. This accelerated method of entering citations means that you have access to published articles very quickly.
- These citations have a PubMed Unique Identifier (PMID) and the status tag **[PubMed - as supplied by publisher]**.

What is needed to search Medical Literature Online

- Basics - for Clinicians
  - Understand PubMed's scope and content.
  - Understand how the MeSH vocabulary is used to describe and retrieve citations.
  - Build a search using MeSH and PubMed search tools (Details, Limits, History, etc.)
- Advanced (for Research)
  - Manage your results using display, sort, the Clipboard, save, print, e-mail and order features and My NCBI filters.
  - Save your search strategies.
  - Link to full-text articles and other resources.
  - Use special queries and other PubMed/NCBI tools.

Navigating PubMed

PubMed's home page displays:

- the page header
- a black bar that provides access to other Entrez databases
- a database selection menu, where you can choose between PubMed and other Entrez databases
- a query box where you enter your search terms
- feature tabs with access to additional tools and features
- a link to the Advanced Search, where you can construct a tailored search and/or apply limits
- a sidebar with links to PubMed's Help, oth-

er PubMed Services and related resources

- the footnote

Building the Search

- Understand how PubMed translates basic searches.
- Use Limits to narrow your search.
- Understand and use Boolean operators.
- Use PubMed search tools and related databases to construct a search.
- Build your own search using search field tags.

Developing a Search Strategy

Before you can search for any information, you should first develop a search strategy.

What is a Search Strategy?

A search strategy is a plan that helps you look for the information you need.

Search Strategy Tips

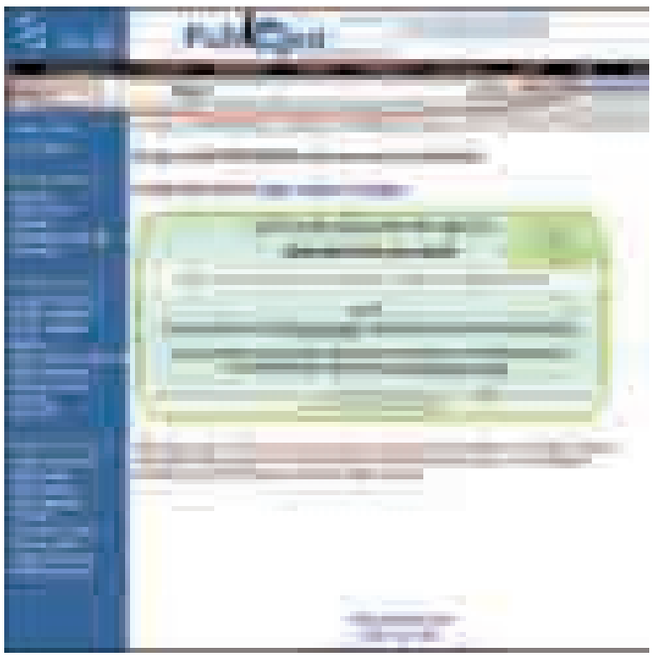
- Identify the key concepts.
- Determine alternative terms for these concepts, if needed.
- Refine your search to dates, study groups, etc., as appropriate.
- Practice helps. Strategies and styles will differ according to personal choice and professional discipline.

How It Works

To search PubMed, you can simply enter search terms in the search box. PubMed contains many records, so be as specific as possible.

Regardless of the page you are on, the Home page, a results screen, the Clipboard, or any of the others -- the search box will always be available to you.

Any combination of search terms can be typed in the search box. Click the go button or Enter key to launch the search.



Automatic Term Mapping

What is searched?

PubMed uses an Automatic Term Mapping feature to search for **unqualified terms**. When you click **Go**, PubMed will look for a match in several lists.

It looks first for a match for your phrase as a

- **Subject** in the
  - **MeSH Translation Table.** If it doesn't find a match, it looks for your phrase as a
- **Journal** in the
  - **Journals Translation Table**, then it searches for
- **Author and Investigator names** in the
  - **Full Author Translation Table,** in the
  - **Author Index,** in the
  - **Full Investigator Translation Table,** and in the
  - **Investigator Index.**

As soon as PubMed finds a match, the mapping stops. That is, if a term matches in the MeSH Translation Table, PubMed does not continue looking in the next table.

If no match is found, PubMed breaks apart the phrase and repeats the process until a match is found.

The phrases and individual terms are also searched in All fields with the exceptions of multi-word Substance Names and MeSH headings that include stand-alone numbers or letters, which are searched in All Fields as phrases only.

Details

Clicking **Details** is useful to see how PubMed translated your search Sometimes you may find that PubMed interpreted your search in a way you had not expected. This may help you understand your retrieval and may also point out a misspelled word.

The Details page shows:

- The search box with the actual strategy and syntax used to run the search. It may include MeSH vocabulary term mappings as well as mappings from the PubMed phrase index. The display will include any error messages, such as stop words truncation warnings, and misspellings.
- The Result number displays the total number of retrieved citations for the current search and can be used to link to those results.
- The Translations area shows how each term was translated.
- The User Query area shows the search as you entered it in the query box.

Automatic Term Mapping (continued)

Again, the first place PubMed looks for a match for your search is in the**MeSH Translation Table.** This table contains:

- MeSH Headings
- **Subheadings**
- **Publication Types**
- Entry Term mappings (also known as synonyms) for MeSH terms
- Mappings derived from theUnified Medical Language System® (UMLS®)
- Supplementary Concepts and synonyms to the Supplementary Concepts

1. If the term **renal transplant** is searched, PubMed will find a match for this in the MeSH Translation table.
2. Renal transplant is a synonym for the MeSH term, Kidney Transplantation.
3. PubMed will search using the MeSH term as well as the term originally entered. In addition, both the entered term and the "mapped to" MeSH term will also be searched in All Fields.

   PubMed automatically searches the MeSH headings as well as the more specific terms underneath that heading (if there are any) in the MeSH hierarchy. This is referred to as exploding the term.

The **Journals Translation Table**contains:

- Full journal titles
- MEDLINE title abbreviations
- **International Standard Serial Numbers (ISSN)**

If you searched for **the journal of cell biology**, for example, PubMed would translate this as **"J Cell Biol"[Journal] OR "the journal of cell biology"[All Fields]**.

What if a journal name is also a MeSH heading -- **Cell**, for example? PubMed will first check in the MeSH Translation Table, find all matches (in this case, the terms **cells** and**cellular phone**) and stop the mapping process. The search will also include [All Fields], therefore it will find the journal name, but the results will include many more citations than from the journal, **Cell**.

Consider using the Search by... feature of Advanced Search to find journal titles that might be confused with subject terms.

Searching for journal titles in the Journals Database will be covered in a later module of this course.

Automatic Term Mapping (continued)

The **Full Author Translation Table** includes:

Full author names for articles published from 2002 forward, and to journals that publish using the full names of authors.

Full author names can be entered in natural or inverted order

julia s wong wong julia s

Omit periods after initials, and put all suffixes (e.g., Jr) at the end. When searching a full name in inverted order, a comma following the last name is generally optional:

herron bruce j herron, bruce j

However, for some names it is necessary to distinguish which name is the last name. [Show Me]

ryan, jamesjames, ryan

Consider using advanced search to find author names.

The Author Index:

If the phrase is not found in the MeSH or Journal Translation Tables and even if it is found in the Full Author Name Translation Table, PubMed checks the

Author Index for a match.

Enter the author's name in the form of last name plus initials.

PubMed will automatically truncate the author's name to account for varying initials.

To turn off automatic of an author's name, surround the name with double quotes and use the **[au]**tag.

If you are searching with last name only, be sure to use the **[au]** tag.

The Full Investigator Translation Table and Investigator Index:

If the phrase is not found in the MeSH or Journal Translation Tables and even if it is found in the Full Author Name Translation Table or Author Index, PubMed checks the Full Investigator name Translation Table, then Investigator Index for a match.

The names in the Full Investigator Translation Table and the Investigator Index are formatted and searchable in the same way as the Full Author Name Table and Author Index (see above). see more information about investigator names.

If no match is found?

There are times when PubMed is unable to match a search term with the MeSH, journals, author or investigator tables or indexes.PubMed will then search the individual words in All Fields. These individual terms will then be combined (ANDed) together.

In the illustration, the search criteriapressure point is translated by PubMed.

Pressure matched in the MeSH Translation Table, but point did not match in any of the tables, and it is, therefore, searched in All Fields.

Search for:	If:	Action:
Head lice shampoo	No match found.	Removes term on the right to rerun Automatic Term Mapping process.
Head lice	Match found in MeSH Translation Table.	**Head lice** will be searched as **("pediculus"[MeSH Terms] OR "pediculus"[All Fields] OR ("head"[All Fields] AND "lice"[All Fields]) OR "head lice"[All Fields])**
Shampoo	No match found.	**Shampoo** will be searched as **shampoo [All Fields]**
How PubMed Searches the query **head lice shampoo**		

Summary

In summary, let's see how PubMed searches for the search query **head lice shampoo**.

PubMed will then combine the results (with AND) to produce a single search strategy:

(("pediculus"[MeSH Terms] OR "pediculus"[All Fields] OR ("head"[All Fields] AND "lice"[All Fields]) OR "head lice"[All Fields]) AND shampoo[All Fields]

Stopwords

PubMed also refers to a list of commonly found words that are referred to as "**Stop words**" Stopwords are words that, if indexed, could potentially return every document in the database if the word was used in a search statement.

Consequently, commonly found words are not indexed and PubMed will ignore them.

Go to PubMed's help to view the list of stopwords.

Spell Check Feature

Spell check suggests alternative spellings for search terms that include misspellings. Results from a search with the closest common word will display, or an option to link to an alternative spelling to provide users with an easy way to retrieve results for untagged words that are misspelled.

Terms entered with a search tag (e.g., [mh]; [majr]; [tw]) will **not**generate alternative spellings.

Note:

- The alternative spellings are not based on a dictionary but rather the frequency with which a term appears in PubMed.
- The spell checking function will not display an alternative spelling for misspellings that have a high frequency of occurrence in PubMed or for terms with numbers or fewer than 5 characters.
- The feature works best for terms with multiple words.

APPENDICES

Appendix A : Normal Values

Before you refer to these, please understand that

Laboratory results ought to be interpreted with caution. The so called normal values differ from individual to individual

Table A 1 : Chemical Constituents of Blood

		Reference Range	
Constituent	**Specimen**	**SI Units**	**Conventional Units**
Acetoacetate	P	<100 μmol/L	<1 mg/dL
Albumin	S	35-55 g/L	3.5-5.5 g/dL
Aldolase		0-100 nkat/L	0-6 U/L
Alpha$_1$ antitrypsin	S	0.8-2.1 g/L	85-213 mg/dL
Alpha fetoprotein (adult)	S	<30 μg/L	<30 ng/mL
Aminotransferases	S		
Aspartate (AST, SGOT)		0-0.58 μkat/L	0-35 U/L
Alanine (ALT, SGPT)		0-0.58 μkat/L	0-35 U/L
Ammonia, as NH_3	P	6-47 μmol/L	10-80 μg/dL
Amylase	S	0.8-3.2 μkat/L	60-180 U/L
Angiotensin-converting enzyme (ACE)		<670 nkat/L	<40 U/L
Anticonvulsant drug levels: see Table 360-8			
Arterial blood gases			
[HCO_3^-]		21-28 mmol/L	21-30 meq/L
P_{CO2}		4.7-5.9 kPa	35-45 mmHg
pH		7.38-7.44	
P_{O2}		11-13 kPa	80-100 mmHg
β-Hydroxybutyrate	P	<300 μmol/L	<3 mg/dL
Bilirubin, total	S (Malloy-Evelyn)	5.1-17 μmol/L	0.3-1.0 mg/dL
Direct	S	1.7-5.1 μmol/L	0.1-0.3 mg/dL
Indirect	S	3.4-12 μmol/L	0.2-0.7 mg/dL
Calcium, ionized		1.1-1.4 mmol/L	4.5-5.6 mg/dL
Calcium	P	2.2-2.6 mmol/L	9-10.5 mg/dL
Carbon dioxide content	P (sea level)	21-30 mmol/L	21-30 meq/L
Carbon dioxide tension (P_{CO2})	Arterial blood (sea level)	4.7-5.9 kPa	35-45 mmHg
Carbon monoxide content	Blood	Symptoms with 20% saturation of hemoglobin	
Chloride	S (as Cl^-)	98-106 mmol/L	98-106 meq/L
Cholesterol: see Table A-9			
Complement	S		

C3		0.55-1.20 g/L	55-120 mg/dL
C4		0.20-0.50 g/L	20-50 mg/dL
Coproporphyrins (types I and III)	U	150-460 μmol/d	100-300 μg/d
Creatine kinase	S (total)		
Females		0.17-1.17 μkat/L	10-70 U/L
Males		0.42-1.50 μkat/L	25-90 U/L
Creatine kinase-MB		0-7 μg/L	
Creatinine	S	<133 μmol/L	<1.5 mg/dL
Erythropoietin	S	5-36 U/L	
Fatty acids, free (nonesterified)	P	180 mg/L	<18 mg/dL
Ferritin	S		
Women		10-200 μg/L	10-200 ng/mL
Men		15-400 μg/L	15-400 ng/mL
Fibrinogen: See "Hematologic Evaluations: Platelets and Coagulation Parameters"			
Fibrinogen split products: See "Hematologic Evaluations: Platelets and Coagulation Parameters"			
Glucose (fasting)	P		
Normal		4.2-6.4 mmol/L	75-115 mg/dL
Diabetes mellitus		>7.8 mmol/L	>140 mg/dL
Glucose, 2 h postprandial	P		
Normal		<7.8 mmol/L	<140 mg/dL
Impaired glucose tolerance		7.8-11.1 mmol/L	140-200 mg/dL
Diabetes mellitus		>11.1 mmol/L	>200 mg/dL
Hemoglobin	B (sea level)		
Male		140-180 g/L	14-18 g/dL
Female		120-160 g/L	12-16 g/dL
Hemoglobin A_{1c} Up to 6% of total hemoglobin			
Iron	S	9-27 μmol/L	50-150 μg/dL
Iron-binding capacity	S	45-66 μmol/L	250-370 μg/dL
Saturation		0.2-0.45	20-45%
Lactate dehydrogenase	S	1.7-3.2 μkat/L	100-190 U/L
Lactate dehydrogenase isoenzymes	S (agarose)		
Fraction 1 (of total)		0.14-0.25	14-26%
Fraction 2		0.29-0.39	29-39%
Fraction 3		0.20-0.25	20-26%
Fraction 4		0.08-0.16	8-16%
Fraction 5		0.06-0.16	6-16%
Lactate	P, venous	0.6-1.7 mmol/L	5-15 mg/dL
Lipase	S	0-2.66 μkat/L	0-160 U/L
Lipids: see Table A-9			
Lipids, triglyceride: S see "Triglycerides"			
Lipoprotein: see Table A-9			
Lipoprotein (a)	S	0-300 mg/L	0-3 mg/dL
Magnesium	S	0.8-1.2 mmol/L	1.8-3 mg/dL
Myoglobin	S		
Male		19-92 μg/L	

Female		12-76 μg/L	
Osmolality	P	285-295 mmol/kg serum water	285-295 mosmol/kg serum water
Oxygen content	B, arterial (sea level) B, venous arm (sea level)		17-21 vol% 10 to 16 vol%
Oxygen percent saturation (sea level)	B, arterial	0.97 mol/mol	97%
	B, venous, arm	0.60-0.85 mol/mol	60-85%
Oxygen tension (P_{O2})	Blood	11-13 kPa	80-100 mmHg
pH	B	7.38-7.44	
Phosphatase, acid	S	0.90 nkat/L	0-5.5 U/L
Phosphatase, alkaline	S	0.5-2.0 nkat/L	30-120 U/L
Phosphorus, inorganic	S	1.0-1.4 mmol/L	3-4.5 mg/dL
Porphobilinogen	U	None	None
Potassium	S	3.5-5.0 mmol/L	3.5-5.0 meq/L
Prostate-specific antigen (PSA)	S		
Female		<0.5 μg/L	<0.5 ng/mL
Male: <40 years		0.0-2.0 μg/L	0.0-2.0 ng/mL
ε40 years		0.0-4.0 μg/L	0.0-4.0 ng/mL
PSA, free, in males 45-75 years, with PSA values between 4 and 20 μg/mL	S	>0.25 associated with benign prostatic hyperplasia	>25% associated with benign prostatic hyperplasia
Protein, total	S	55-80 g/L	5.5-8.0 g/dL
Protein fractions	S		
Albumin		35-55 g/L	3.5-5.5 g/dL (50-60%)
Globulin		20-35 g/L	2.0-3.5 g/dL (40-50%)
$Alpha_1$		2-4 g/L	0.2-0.4 g/dL (4.2-7.2%)
$Alpha_2$		5-9 g/L	0.5-0.9 g/dL (6.8-12%)
Beta		6-11 g/L	0.6-1.1 g/dL (9.3-15%)
Gamma		7-17 g/L	0.7-1.7 g/dL (13-23%)
Pyruvate	P, venous	60-170 μmol/L	0.5-1.5 mg/dL
Sodium	S	136-145 mmol/L	136-145 meq/L
Transferrin	S	2.3-3.9 g/L	230-390 mg/dL
Triglycerides	S	<1.8 mmol/L	<160 mg/dL
Troponin I	S	0-0.4 μg/L	0-0.4 ng/mL
Troponin T	S	0-0.1 μg/L	0-0.1 ng/mL
Urea nitrogen	S	3.6-7.1 mmol/L	10-20 mg/dL
Uric acid:	S		
Men		150-480 μmol/L	2.5-8.0 mg/dL
Women		90-360 μmol/L	1.5-6.0 mg/dL

Urobilinogen	U	1.7-5.9 μmol/d	1-3.5 mg/d
NOTE: B, blood; P, plasma; S, serum; U, urine.			

Table A-2. Metabolic and Endocrine Tests

		Reference Range	
Substance	**Specimen**	**SI Units**	**Conventional Units**
Adrenocorticotropin (ACTH), 8 A.M.	P	1.3-16.7 pmol/L	6.0-76.0 pg/mL
Aldosterone, 8 A.M., (patient supine, 100 mmol/L Na and 60-100 mmol/L K intake)	P	<220 pmol/L	<8 ng/dL
Aldosterone	U	14-53 nmol/d	5-19 μg/d
Androstenedione	P		
Women		3.5-7.0 nmol/L	1-2 ng/mL
Men		3.0-5.0 nmol/L	0.8-1.3 ng/mL
Angiotensin II, 8 A.M.	P	10-30 nmol/L	10-30 pg/mL
Arginine vasopressin (AVP), random fluid intake	P	1.4-5.6 pmol/L	1.5-6.0 ng/L
Calciferols (vitamin D)	P		
1,25-dihydroxyvitamin D [1,25(OH)$_2$D]		40-160 pmol/L	16-65 pg/mL
25-hydroxyvitamin D [25(OH)D]		20-200 nmol/L	8-80 ng/mL
Calcitonin	P		
Women		≤8 ng/L	≤8 pg/mL
Men		≤4 ng/L	≤4 pg/mL
Catecholamines			
Epinephrine	U	<275 nmol/d	<50 μg/d
Free	U	<590 nmol/d	<100 μg/d
Metanephrine	U	<7 μmol/d	<1.3 mg/d
Norepinephrine	U	89-473 nmol/d	15-80 μg/d
Vanillylmandelic acid (VMA)	U	<40 μmol/d	<8 mg/d
Chorionic gonadotropin, β subunit (β-hCG), men and nonpregnant women	P	<3 IU/L	<3 mIU/mL
Cortisol			
Free	U	25-140 nmol/d	10-50 μg/d
8 A.M.	P	140-690 nmol/L	5-25 μg/dL
4 P.M.	P	80-330 nmol/L	3-12 μg/dL
Dehydroepiandrosterone (DHEA)	P	7-31	2-9 ng/dL

		nmol/L	
11-Deoxycortisol (compound S)	P	<30 nmol/L	<1 μg/dL
DHEA sulfate	P	1.3-6.8 μmol/L	500-2500 μg/dL
Estradiol	P		
Women (higher at ovulation)		70-220 pmol/L	20-60 pg/mL
Men		<180 pmol/L	<50 pg/mL
Gastrin	S	40-200 ng/L	40-200 pg/mL
Glucagon	P	50-100 ng/L	50-100 pg/mL
Gonadotropins			
Follicle-stimulating hormone (FSH)	P		
Women			
Mature, premenopausal, except at ovulation		1.4-9.6 IU/L	1.4-9.6 mIU/mL
Ovulatory surge		2.3-21 IU/L	2.3-21 mIU/mL
Postmenopausal		34-96 IU/L	34-96 mIU/mL
Men		0.9-15 IU/L	0.9-15 mIU/mL
Luteinizing hormone (LH)	P		
Children, prepubertal		1.0-5.9 IU/L	1.0-5.9 mIU/mL
Women			
Mature, premenopausal, except at ovulation		0.8-26 IU/L	0.8-26 mIU/mL
Ovulatory surge		25-57 IU/L	25-57 mIU/mL
Postmenopausal		40-104 IU/L	40-104 mIU/mL
Men		1.3-13 IU/L	1.3-13 mIU/mL
Growth hormone, after 100 g oral glucose		<2 μg/L	<2 ng/mL
Hemoglobin A_{1c}	WB	0.038-0.064	3.8-6.4%
17-Hydroxycorticosteroids	U	5.5-28 μmol/d	2-10 mg/d
5-Hydroxyindoleacetic acid (5-HIAA)	U	≤31.4 μmol/d	≤6 mg/d
17-Hydroxyprogesterone	P		
Women			
Follicular phase		0.6-3 nmol/L	0.2-1.0 μg/L
Luteal phase		1.5-10.6 nmol/L	0.5-3.5 μg/L
Men		0.2-9.0 nmol/L	0.06-3.0 μg/L
Insulin, fasting	S, P	43-186 pmol/L	6-26 μU/mL

Insulin-like growth factor (somatomedin C, IGF-1/SM C)	S		
16-24 years		182-780 μg/L	182-780 ng/mL
25-39 years		114-492 μg/L	114-492 ng/mL
40-54 years		90-360 μg/L	90-360 ng/mL
>54 years		71-290 μg/L	71-290 ng/mL
17-Ketosteroids	U		
Women		20-59 μmol/d	6-17 mg/d
Men		20-69 μmol/d	6-20 mg/d
Oxytocin			
Random		1-4 pmol/L	1.25-5 ng/L
Ovulatory peak in women		4-8 pmol/L	5-10 ng/L
Parathyroid hormone	S	10-60 ng/L	10-60 pg/mL
Parathyroid hormone-related protein	P	<1.3 pmol/L	<1.3 pmol/L
Progesterone	P		
Women, luteal, peak		6-60 nmol/L	2-20 ng/mL
Men, prepubertal girls, preovulatory women, postmenopausal women		<6 nmol/L	<2 ng/mL
Prolactin	S	2-15 μg/L	2-15 ng/mL
Radioactive iodine uptake, 24 h (range varies in different areas due to variations in iodine intake)			5-30%
Renin (adult, normal-Na diet)	P		
Supine		0.08-0.83 ng/(L⊇s)	0.3-3.0 ng/(mL/h)
Upright		0.28-2.5 ng/(L⊇s)	1.0-9.0 ng/(mL/h)
Resin triiodothyronine (T_3)		0.25-0.35	25-35%
Reverse T_3 (rT_3)	P	0.15-0.61 nmol/L	10-40 ng/dL
Semen analysis: see Chap. 335			
T_3	P	1.1-2.9 nmol/L	70-190 ng/dL
Testosterone	P		
Women		<3.5 nmol/L	<1 ng/mL
Men		10-35 nmol/L	3-10 ng/mL
Prepubertal boys and girls		0.17-0.7 nmol/L	0.05-0.2 ng/mL
Thyroglobulin	S	0-60 μg/L	0-60 ng/mL
Thyroid stimulating hormone (TSH)		0.4-5.0 mU/L	0.4-5.0 μU/mL
Thyroxine (T_4)	SR	64-154	5-12 μg/dL

		nmol/L	
NOTE: P, plasma; S, serum; SR; serum radioimmunoassay; U, urine; WB, whole blood.			

Table A 3 : Vitamins and Trace Minerals

		Reference Range	
	Specimen	**SI Units**	**Conventional Units**
Carotenoids	S	0.9-5.6 μmol/L	50-300 μg/dL
Ceruloplasmin	S	270-370 mg/L	27-37 ng/dL
Copper	S	11-22 μmol/L	70-140 μg/dL
Folic acid	RC	340-1020 nmol/L cells	150-450 ng/mL cells
Folic acid	S	7-36 nmol/L cells	3-16 ng/mL cells
Lead	S	<1 μmol/L	<20 μg/dL
Vitamin A	S	0.7-3.5 μmol/L	20-100 μg/dL
Vitamin B_1 (thiamine)	S	0-75 nmol/L	0-2 μg/dL
Vitamin B_2 (riboflavin)	S	106-638 nmol/L	4-24 μg/dL
Vitamin B_6	P	20-121 nmol/L	5-30 ng/ml
Vitamin B_{12}	S	148-443 pmol/L	200-600 pg/mL
Vitamin C (ascorbic acid)	S	23-57 μmol/L	0.4-1.0 mg/dL
Vitamin D_3,1,25-dihydroxy	S	60-108 pmol/L	25-45 pg/mL
Vitamin D_3,25-hydroxy	P		
Summer		37.4-200 nmol/L	15-80 ng/mL
Winter		34.9-105 nmol/L	14-42 ng/mL
Vitamin E	S	12-42 μmol/L	5-18 μg/mL
Zinc	S	11.5-18.5 μmol/L	75-120 μg/dL
NOTE: P, plasma; RC, red cells; S, serum.			

Table A 4 : Renal Function Tests

	Reference Range	
	SI Units	**Conventional Units**
Clearances (corrected to 1.72 m^2 body surface area):		
Measures of glomerular filtration rate:		
Inulin clearance (Cl)		
Males (mean ± 1 SD)	2.1 ± 0.4 mL/s	124 ± 25.8 mL/min
Females (mean ± 1 SD)	2.0 ± 0.2 mL/s	119 ± 12.8 mL/min
Endogenous creatinine clearance	1.5-2.2 mL/s	91-130 mL/min
Urea	1.0-1.7 mL/s	60-100 mL/min
Measures of effective renal plasma flow and tubular function:		
p-Aminohippuric acid clearance (Cl_{PAH}):		
Males (mean ± 1 SD)	10.9 ± 2.7 mL/s	654 ± 163 mL/min
Females (mean ± 1 SD)	9.9 ± 1.7 mL/s	594 ± 102 mL/min
Concentration and dilution test:		
Specific gravity of urine:		

After 12-h fluid restriction	ε1.025	ε1.025
After 12-h deliberate water intake	≤1.003	≤1.003
Protein excretion, urine	<0.15 g/d	<150 mg/d
Males	0-0.06 g/d	0-60 mg/d
Females	0-0.09 g/d	0-90 mg/d
Specific gravity, maximal range	1.002-1.028	1.002-1.028
Tubular reabsorption, phosphorus	0.79-0.94 of filtered load	79-94% of filtered load

Table A 5 : Classification of Total Cholesterol, LDL-Cholesterol, and HDL-Cholesterol Values

	Total Plasma Cholesterol			LDL-Cholesterol			HDL-Cholesterol	
	SI, mmol/L	**C, mg/dL**		**SI, mmol/L**	**C, mg/dL**		**SI, mmol/L**	**C, mg/dL**
Desirable	<5.2	<200		<3.36	<130		>1.55	>60
Borderline	5.20-6.18	200-239		3.36-4.11	130-159		0.9-1.55	35-60
Undesirable	ε6.21	ε240		ε4.14	ε160		<0.9	<35

NOTE: LDL, low-density lipoprotein; HDL, high-density lipoprotein; SI, SI units; C, conventional units

SOURCE: Modified from the report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults: Second Report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol (Adult Treatment Panel II). Circulation 89:1329, 1994.

Table A 6 : Drug Levels

	Therapeutic Range		Toxic Level	
Drug	**Conventional Units**	**SI Units**	**Conventional Units**	**SI Units**
Acetaminophen	10-30 μg/mL	66-199 μmol/L	>200 μg/mL	>1324 μmol/L
Amikacin				
Peak	25-35 μg/mL	43-60 μmol/L	>35 μg/mL	>60 μmol/L
Trough	4-8 μg/mL	6.8-13.7 μmol/L	>10 μg/mL	>17 μmol/L
Amitriptyline	120-250 ng/mL	433-903 nmol/L	>500 ng/mL	>1805 nmol/L
Amphetamine	20-30 ng/mL	148-222 nmol/L	>200 ng/mL	>1480 nmol/L
Barbiturates, most short-acting			>20 mg/L	>88 μmol/L
Bromide			>1250 μg/mL	>15.6 mmol/L
Carbamazepine	6-12 μg/mL	26-51 μmol/L	>15 μg/mL	>63 μmol/L
Chlordiazepoxide	700-1000 ng/mL	2.34-3.34 μmol/L	>5000 ng/mL	>16.7 μmol/L
Clonazepam	15-60 ng/mL	48-190	>80 ng/mL	>254

		nmol/L		nmol/L
Clozapine	200-350 ng/mL	0.6-1 μmol/L		
Cocaine	100-500 ng/mL	330-1650 nmol/L	>1000 ng/mL	>3300 nmol/L
Desipramine	75-300 ng/mL	281-1125 nmol/L	>400 ng/mL	>1500 nmol/L
Diazepam	100-1000 ng/mL	0.35-351 μmol/L	>5000 ng/mL	>17.55 μmol/L
Digoxin	0.8-2.0 ng/mL	1.0-2.6 nmol/L	>2.5 ng/mL	>3.2 umol/L
Doxepin	30-150 ng/mL	107-537 nmol/L	>500 ng/mL	>1790 nmol/L
Ethanol			>300 mg/dL	>65 mmol/L
Behavioral changes	>20 mg/dL	>4.3 mmol/L		
Legal intoxication	>80 mg/dL	>17 mmol/L		
Ethosuximide	40-100 μg/mL	283-708 μmol/L	>150 μg/mL	>1062 μmol/L
Flecainide	0.2-1.0 μg/mL	0.5-2.4 μmol/L	>1.0 μg/mL	>2.4 μmol/L
Gentamicin				
Peak	8-10 μg/mL	16.7-20.9 μmol/L	>10 μg/mL	>21μmol/L
Trough	<2-4 μg/mL	<4.2-8.4 μmol/L	>4 μg/mL	>8.4 μmol/L
Imipramine	125-250 ng/mL	446-893 nmol/L	>500 ng/mL	>1784 nmol/L
Lidocaine	1.5-6.0 μg/mL	6.4-26 μmol/L		
CNS or cardiovascular depression			6-8 μg/mL	26-34.2 μmol/L
Seizures, obtundation, decreased cardiac output			>8 μg/mL	>34.2 μmol/L
Lithium	0.6-1.2 meq/L	0.6-1.2 nmol/L	>2 meq/L	>2 mmol/L
Methadone	100-400 ng/mL	0.32-1.29 μmol/L	>2000 ng/mL	>6.46 μmol/L
Methotrexate	Variable	Variable		
Low-dose (1-2 weeks)			>9.1 ng/mL	>20 nmol/L
High-dose (48 h)			>227 ng/mL	>0.5 μmol/L
Morphine	10-80 ng/mL	35-280 μmol/L	>200 ng/mL	>700 nmol/L
Nitroprusside (as thiocyanate)	6-29 μg/mL	103-499 μmol/L		
Nortriptyline	50-170 ng/mL	190-646 nmol/L	>500 ng/mL	>1.9 μmol/L
Phenobarbital	10-40 μg/mL	43-170		

		μmol/L		
Slowness, ataxia, nystagmus			35-80 μg/mL	151-345 μmol/L
Coma with reflexes			65-117 μg/mL	280-504 μmol/L
Coma without reflexes			>100 μg/mL	>430 μmol/L
Phenytoin	10-20 μg/mL	40-79 μmol/L	>20 μg/mL	>79 μmol/L
Procainamide	4-10 μg/mL	17-42 μmol/L	>10-12 μg/mL	>42-51 μmol/L
Quinidine	2-5 μg/mL	6-15 μmol/L	>6 μg/mL	>18 μmol/L
Salicylates	150-300 μg/mL	1086-2172 μmol/L	>300 μg/mL	>2172 μmol/L
Theophylline	8-20 μg/mL	44-111 μmol/L	>20 μg/mL	>110 μmol/L
Thiocyanate				
After nitroprusside infusion	6-29 μg/mL	103-499 μmol/L		
Nonsmoker	1-4 μg/mL	17-69 μmol/L	>120 μg/mL	>2064 μmol/L
Smoker	3-12 μg/mL	52-206 μmol/L		
Tobramycin				
Peak	8-10 μg/mL	17-21 μmol/L	>10 μg/mL	>21 μmol/L
Trough	<4 μg/mL	<9 μmol/L	>4 μg/mL	>9 μmol/L
Valproic acid	50-150 μg/mL	347-1040 μmol/L	>150 μg/mL	>1040 μmol/L
Vancomycin				
Peak	18-26 μg/mL	12-18 μmol/L		
Trough	5-10 μg/mL	3-7 μmol/L	>80-100 μg/mL	>55-69 μmol/L

Table A 7 : Instructions For Collection And Transport Of Specimens For Culture

Type of Culture (Synonyms)	Specimen	Minimum Volume	Container	Other Considerations
BLOOD				
Blood, routine (blood culture for aerobes, anaerobes, and yeasts)	Whole blood	10 mL in each of 2 bottles for adults and children; 5 mL, ifpossible,	See below.[a]	See below.[b]

		in each of 2 bottles for infants; less for neonates		
Blood for fungi/*Mycobacterium* spp.	Whole blood	10 mL in each of 2 bottles, as for routine blood cultures, or in Isolator tube requested from laboratory	Same as for routine blood culture	Specify "hold for extended incubation," since fungal agents may require 4 weeks or more to grow.
Blood, Isolator (lysis centrifugation)	Whole blood	10 mL	Isolator tubes	Use mainly for isolation of fungi, *Mycobacterium,* or other fastidious aerobes and for elimination of antibiotics from cultured blood in which organisms are concentrated by centrifugation.
RESPIRATORY TRACT				
Nose	Swab from nares	1 swab	Sterile culturette or similar transport system containing holding medium	Swabs made of calcium alginate may be used.
Throat	Swab of posterior pharynx, ulcerations, or areas of suspected purulence	1 swab	Sterile culturette or similar swab specimen collection system containing holding	See below.[c]

			medium	
Sputum	Fresh sputum (not saliva)	2 mL	Commercially available sputum collection system or similar sterile container with screw cap	*Cause for rejection:* Care must be taken to ensure that the specimen is sputum and not saliva. Examination of Gram's stain, with number of epithelial cells and PMNs noted, can be an important part of the evaluation process. Induced sputum specimens should not be rejected.
Bronchial aspirates	Transtracheal aspirate, bronchoscopy specimen, or bronchial aspirate	1 mL of aspirate or brush in transport medium	Sterile aspirate or bronchoscopy tube, bronchoscopy brush in a separate sterile container	Special precautions may be required, depending on diagnostic considerations (e.g., *Pneumocystis*).
STOOL				
Stool for routine culture; stool for *Salmonella, Shigella,* and *Campylobacter*	Rectal swab or (preferably) fresh, randomly collected stool	1 g of stool or 2 rectal swabs	Plastic-coated cardboard cup or plastic cup with tight-fitting lid. Other leak-proof containers are also acceptable.	If *Vibrio* spp. are suspected, the laboratory must be notified, and appropriate collection/transport methods should be used.

Stool for *Yersinia, E. coli* O157	Fresh, randomly collected stool	1 g	Plastic-coated cardboard cup or plastic cup with tight-fitting lid	*Limitations:* Procedure requires enrichment techniques.
Stool for *Aeromonas* and *Plesiomonas*	Fresh, randomly collected stool	1 g	Plastic-coated cardboard cup or plastic cup with tight-fitting lid	*Limitations:* Stool should not be cultured for these organisms unless also cultured for other enteric pathogens.
UROGENITAL TRACT				
Urine	Clean-voided urine specimen or urine collected by catheter	0.5 mL	Sterile, leak-proof container with screw cap or special urine transfer tube	See below.[d]
Urogenital secretions	Vaginal or urethral secretions, cervical swabs, uterine fluid, prostatic fluid, etc.	1 swab or 0.5 mL of fluid	Transwab containing Amies transport medium or similar system containing holding medium for *Neisseria gonorrhoeae;* modified Todd-Hewitt broth for group B *Streptococcus* surveillance cultures	Vaginal swab samples for "routine culture" should be discouraged whenever possible unless a particular pathogen is suspected. For detection of multiple organisms (e.g., group B *Streptococcus, Trichomonas, Chlamydia,* or *Candida* spp.), 1 swab per test should be obtained.
BODY FLUIDS, ASPIRATES, AND TISSUES				

Cerebrospinal fluid (lumbar puncture)	Spinal fluid	1 mL for routine cultures; ε5 mL for *Mycobacterium*	Sterile tube with tight-fitting cap	Do not refrigerate; transfer to laboratory as soon as possible.
Body fluids	Aseptically aspirated body fluids	1 mL for routine cultures	Sterile tube with tight-fitting cap. Specimen may be left in syringe used for collection if the syringe is capped before transport.	For some body fluids (e.g., peritoneal lavage samples), increased volumes are helpful for isolation of small numbers of bacteria.
Biopsy and aspirated materials	Tissue removed at surgery, bone, anticoagulated bone marrow, biopsy samples, or other specimens from normally sterile areas	1 mL of fluid or a 1-g piece of tissue	Sterile "culturette"-type swab or similar transport system containing holding medium. Sterile bottle or jar should be used for tissue specimens.	Accurate identification of specimen and source is critical. Enough tissue should be collected for both microbiologic and histopathologic evaluations.
Wounds	Purulent material or abscess contents obtained from wound or abscess without contamination by normal microflora	2 swabs or 0.5 mL of aspirated pus	Culturette swab or similar transport system or sterile tube with tight-fitting screw cap. For simultaneous anaerobic cultures, send	*Collection:* Abscess contents or other fluids should be collected in a syringe (see above) when possible to provide an adequate sample volume and an anaerobic environment.

			specimen in anaerobic transport device or closed syringe.	
SPECIAL RECOMMENDATIONS				
Fungi	Specimen types listed above may be used. When urine or sputum is cultured for fungi, a first morning specimen is usually preferred.	1 mL or as specified above for individual listing of specimens . Large volumes may be useful for urinary fungi.	Sterile, leak-proof container with tight-fitting cap	*Collection:* Specimen should be transported to microbiology laboratory within 1 h of collection. Contamination with normal flora from skin, rectum, vaginal tract, or other body surfaces should be avoided.
Mycobacterium (acid-fast bacilli)	Sputum, tissue, urine, body fluids	10 mL of fluid or small piece of tissue. Swabs should not be used.	Sterile container with tight-fitting cap	Detection of *Mycobacterium* spp. is improved by use of concentration techniques. Smears and cultures of pleural, peritoneal, and pericardial fluids often have low yields. Multiple cultures from the same patient are encouraged. Culturing in liquid media shortens the time to detection.
Legionella	Pleural fluid, lung biopsy, bronchoalveolar	1 mL of fluid; any size tissue	(	(

	lavage fluid, bronchial/transbronchial biopsy. Rapid transport to laboratory is critical.	sample, although a 0.5-g sample should be obtained when possible		
Anaerobic organisms	Aspirated specimens from abscesses or body fluids	1 mL of aspirated fluid or 2 swabs	An appropriate anaerobic transport device is required.[e]	Specimens cultured for obligate anaerobes should be cultured for facultative bacteria as well.
Viruses[f]	Respiratory secretions, wash aspirates from respiratory tract, nasal swabs, blood samples (including buffy coats), vaginal and rectal swabs, swab specimens from suspicious skin lesions, stool samples (in some cases)	1 mL of fluid, 1 swab, or 1 g of stool in each appropriate transport medium	Fluid or stool samples in sterile containers or swab samples in viral culturette devices (kept on ice but not frozen) are generally suitable. Plasma samples and buffy coats in sterile collection tubes should be kept at 4 to 8 C. If specimens are to be shipped or kept for a long time, freezing at –80 C is usually	Most samples for culture are transported in holding medium containing antibiotics to prevent bacterial overgrowth and viral inactivation. Many specimens should be kept cool but not frozen, provided they are transported promptly to the laboratory. Procedures and transport media vary with the agent to be cultured and the duration of transport.

			adequate.	

[a] For samples from adults and children, two bottles (smaller for pediatric samples) should be used: one with dextrose phosphate, tryptic soy, or another appropriate broth and the other with thioglycollate or another broth containing reducing agents appropriate for isolation of obligate anaerobes. For special situations (e.g., suspected fungal infection, culture-negative endocarditis, or mycobacteremia), different blood collection systems may be used (Isolator systems; see table).

[b] *Collection:* An appropriate disinfecting technique should be used on both the bottle septum and the patient. Do not allow air bubbles to get into anaerobic broth bottles. *Special considerations:* There is no more important clinical microbiology test than the detection of blood-borne pathogens. The rapid identification of bacterial and fungal agents is a major determinant of patients' survival. Bacteria may be present in blood either continuously (as in endocarditis, overwhelming sepsis, and the early stages of salmonellosis and brucellosis) or intermittently (as in most other bacterial infections, in which bacteria are shed into the blood on a sporadic basis). Most blood culture systems employ two separate bottles containing broth medium: one that is vented in the laboratory for the growth of facultative and aerobic organisms and a second that is maintained under anaerobic conditions. In cases of suspected continuous bacteremia/fungemia, two or three samples should be drawn before the start of therapy, with additional sets obtained if fastidious organisms are thought to be involved. For intermittent bacteremia, two or three samples should be obtained at least 1 h apart during the first 24 h.

[c] Normal microflora includes alpha-hemolytic streptococci, saprophytic *Neisseria* spp., diphtheroids, and *Staphylococcus* spp. Aerobic culture of the throat ("routine") includes screening for and identification of beta-hemolytic *Streptococcus* spp. and other potentially pathogenic organisms. Although considered components of the normal microflora, organisms such as *Staphylococcus aureus, Haemophilus influenzae,* and *Streptococcus pneumoniae* will be identified by most laboratories, if requested. When *Neisseria gonorrhoeae* or *Corynebacterium diphtheriae* is suspected, a special culture request is recommended.

[d] (1) Clean-voided specimens, midvoid specimens, and Foley or indwelling catheter specimens that

yield ε50,000 organisms/mL and from which no more than three species are isolated should have organisms identified. (2) Straight-catheterized, bladder-tap, and similar urine specimens should undergo a complete workup (identification and susceptibility testing) for all potentially pathogenic organisms, regardless of colony count. (3) Certain clinical problems (e.g., acute dysuria in women) may warrant identification and susceptibility testing of isolates present at concentrations of <50,000 organisms/mL.
e Aspirated specimens in capped syringes or other transport devices designed to limit oxygen exposure are suitable for the cultivation of obligate anaerobes. A variety of commercially available transport devices may be used. Contamination of specimens withnormal microflora from the skin, rectum, vaginal vault, or another body site should be avoided. Collection containers for aerobic culture (such as dry swabs) and inappropriate specimens (such as refrigerated samples; expectorated sputum; stool; gastric aspirates; and vaginal, throat, nose, and rectal swabs) should be rejected as unsuitable.
f Laboratories generally use diverse methods to detect viral agents, and the specific requirements for each specimen should be checked before a sample is sent.

Table A 9 : Urine Analysis

	Reference Range	
	SI Units	**Conventional Units**
Acidity, titratable	20-40 mmol/d	20-40 meq/d
Ammonia	30-50 mmol/d	30-50 meq/d
Amylase		4-400 U/L
Amylase/creatinine clearance ratio [$(Cl_{am}/Cl_{cr}) \infty 100$]	1-5	1-5
Calcium (10 meq/d or 200-mg/d dietary calcium)	<7.5 mmol/d	<300 mg/d
Creatine, as creatinine		
Women	<760 μmol/d	<100 mg/d
Men	<380 μmol/d	<50 mg/d
Creatinine	8.8-14 mmol/d	1.0-1.6 g/d
Glucose, true (oxidase method)	0.3-1.7 mmol/d	50-300 mg/d
5-Hydroxyindoleacetic acid (5-HIAA)	10-47 μmol/d	2-9 mg/d
Protein	<0.15 g/d	<150 mg/d
Potassium (varies with intake)	25-100 mmol/d	25-100 meq/d
Sodium (varies with intake)	100-260	100-260 meq/d

	mmol/d	

Table A 10 : Stool Analysis		
	Reference Range	
	SI Units	**Conventional Units**
Bulk		
Wet weight	<197.5 (115 ± 41)g/d	<197.5 (115 ± 41) g/d
Dry weight	<66.4 (34 ± 15) g/d	<66.4 (34 ± 15) g/d
α_1 Antitrypsin	0.98 (±0.17) mg/g dry weight	0.98 (±0.17) mg/g dry weight
Coproporphyrin	600-1500 nmol/d	400-1000 μg/d
Fat (on diet containing at least 50 g fat), measured on a ε 3-day collection		
Fat		
Percent of dry weight	<0.30	<30.4%
Coefficient of fat absorption	>0.95	>95%
Fatty acid		
Free	0.01-0.10	1-10% of dry matter
Combined as soap	0.005-0.12	0.5-12% of dry matter
Nitrogen	<1.7 (1.4 ± 0.2) g/d	<1.7 (1.4 ± 0.2) g/d
Protein content	Minimal	Minimal
Urobilinogen	68-470 μmol/d	40-280 mg/d
Water	~0.65	~65%

Table A 11 : Other Gastrointestinal Tests		
	Results	
Test	**SI Units**	**Conventional Units**
Absorption tests		
D-Xylose: after overnight fast, 25 g xylose given in oral aqueous solution		
Urine, collected for following 5 h	33-53 mmol (or >20% of ingested dose)	5-8 g (or >20% of ingested dose
Serum, 1 h after dose	1.7-2.7 mmol/L	25-40 mg/dL
Vitamin A: a fasting blood specimen is obtained and 200,000 units of vitamin A in oil is given orally	Serum level should rise to twice fasting level in 3-5 h	Serum level should rise to fasting level in 3-5 h
Bentiromide test (pancreatic function): 500 mg bentiromide (chymex) orally; *p*-aminobenzoic acid (PABA) measured		
Plasma		>3.6 (±1.1) μg/mL at 90 min
Urine	>50% recovered in 6 h	>50% recovered in 6 h

Gastric juice		
Volume		
24 h	2-3 L	2-3 L
Nocturnal	600-700 mL	600-700 mL
Basal, fasting	30-70 mL/h	30-70 mL/h
Reaction		
pH	1.6-1.8	1.6-1.8
Titratable acidity of fasting juice	4-9 μmol/s	15-35 meq/h
Acid output		
Basal		
Females (mean ± 1 SD)	0.6 ± 0.5 μmol/s	2.0 ± 1.8 meq/h
Males (mean ± 1 SD)	0.8 ± 0.6 μmol/s	3.0 ± 2.0 meq/h
Maximal (after SC histamine acid phosphate, 0.004 mg/kg body weight, and preceded by 50 mg promethazine, or after betazole, 1.7 mg/kg body weight, or pentagastrin, 6 μg/kg body weight)		
Females (mean ± 1 SD)	4.4 ± 1.4 μmol/s	16 ± 5 meq/h
Males (mean ± 1 SD)	6.4 ± 1.4 μmol/s	23 ± 5 meq/h
Basal acid output/maximal acid output ratio	≤0.6	≤0.6
Gastrin, serum	40-200 μg/L	40-200 pg/mL
Secretin test (pancreatic exocrine function): 1 unit/kg body weight, IV		
Volume (pancreatic juice) in 80 min	>2.0 mL/kg	>2.0 mL/kg
Bicarbonate concentration	>80 mmol/L	>80 meq/L
Bicarbonate output in 30 min	>10 mmol	>10 meq

Table A 12 : CEREBROSPINAL FLUID[a]

	Reference Range	
Constituent	**SI Units**	**Conventional Units**
Osmolarity	292-297 mmol/kg water	292-297 mOsm/L
Electrolytes		
Sodium	137-145 mmol/L	137-145 meq/L
Potassium	2.7-3.9 mmol/L	2.7-3.9 meq/L
Calcium	1.0-1.5 mmol/L	2.1-3.0 meq/L
Magnesium	1.0-1.2 mmol/L	2.0-2.5 meq/L
Chloride	116-122 mmol/L	116-122 meq/L
CO_2 content	20-24 mmol/L	20-24 meq/L
P_{CO2}	6-7 kPa	45-49 mmHg
pH	7.31-7.34	
Glucose	2.2-3.9 mmol/L	40-70 mg/dL
Lactate	1-2 mmol/L	10-20 mg/dL
Total protein	0.2-0.5 g/L	20-50 mg/dL
Albumin	0.066-0.442 g/L	6.6-44.2 mg/dL
IgG	0.009-0.057 g/L	0.9-5.7 mg/dL
IgG index[b]	0.29-0.59	
Oligoclonal bands (OGB)	<2 bands not present in matched	

	serum sample	
Ammonia	15-47 μmol/L	25-80 μg/dL
Creatinine	44-168 μmol/L	0.5-1.9 mg/dL
Myelin basic protein	<4 μg/L	
CSF pressure		50-180 mmH_2O
CSF volume (adult)	~150 mL	
Leukocytes		
Total	<5 per μL	
Differential:		
Lymphocytes	60-70%	
Monocytes	30-50%	
Neutrophils	None	

[a] Since cerebrospinal fluid concentrations are equilibrium values, measurements of the same parameters in blood plasma obtained at the same time are recommended. However, there is a time lag in attainment of equilibrium, and cerebrospinal levels of plasma constituents that can fluctuate rapidly (such as plasma glucose) may not achieve stable values until after a significant lag phase.

[b] IgG index = $\frac{\text{CSF IgG(mg/dL)} \infty \text{ serum albumin(g/dL)}}{\text{Serum IgG(g/dL)} \infty \text{ CSF albumin(mg/dL)}}$