STANDARD TREATMENT GUIDELINES FOR THE STATE OF MIZORAM
1st EDITION, 2024

DEPARTMENT OF HEALTH AND FAMILY WELFARE GOVERNMENT OF MIZORAM

DISCLAIMER

The following treatment guidelines are provided for informational and educational purposes and are intended to serve as a general reference for healthcare professionals in the state of Mizoram. While every effort has been made to ensure the accuracy and reliability of the information presented herein, these guidelines should not be considered as a substitute for professional medical advice, diagnosis, or treatment. Healthcare providers are encouraged to exercise their clinical judgment and consider individual patient characteristics and preferences when making treatment decisions. The State of Mizoram, its health department, and any affiliated organizations do not assume any liability for the use of these guidelines or any consequences arising from their application.

PREFACE

With great pride and commitment, we introduce the Standard Treatment Guidelines for the State of Mizoram, a comprehensive resource designed to elevate healthcare standards and foster optimal health outcomes across our diverse communities. Crafted through collaboration among healthcare professionals, researchers, and policymakers, these guidelines embody our collective dedication to ensuring equitable access to evidence-based care for every resident of Mizoram.

In a rapidly evolving healthcare landscape, these guidelines serve as a beacon of guidance, offering standardized protocols for the diagnosis, management, and treatment of prevalent health conditions in our region. Grounded in the latest scientific evidence and tailored to the unique needs and challenges of Mizoram, they provide a framework for healthcare providers to deliver quality care while respecting individual patient preferences and local healthcare contexts.

We understand that effective healthcare extends beyond adherence to protocols; it necessitates a holistic understanding of patients' socio-cultural backgrounds and preferences. Thus, while these guidelines offer essential recommendations, we encourage healthcare providers to integrate their clinical expertise, cultural competence, and patient-centered approach into their practice, ensuring that care delivery remains responsive and compassionate.

The development of these guidelines would not have been possible without the invaluable contributions of numerous individuals and organizations committed to advancing healthcare in Mizoram. Their dedication and expertise have enriched this document, making it a vital tool in our collective mission to improve the health and well-being of our communities.

As we embark on this journey towards better healthcare, let us embrace these guidelines as a foundation for continuous learning, adaptation, and innovation. Together, let us strive to uphold the highest standards of care and to ensure that every individual in Mizoram receives the quality healthcare they deserve.

LIST OF CONTRIBUTORS (EXPERT COMMITTEE MEMBERS)

1. Dr. Biakthansangi, Public Health Financing Expert, CEO MSHCS
2. Dr. Robert L Khawlhring, Psychiatry, DHME
3. Dr.Lalduhawmi Pachuau ,Paediatrics ,Kulikawn Sub-District Hospital
4. Dr. Lalnunmawi Sailo, Anesthesiology, Zoram Medical College
5. Dr. Lalromawii, Clinical Pharmacology, Zoram Medical College
6. Dr. Vanlalduhsaki, Community Medicine, Zoram Medical College
7. Dr. Lalsangliana Ralte, Dental Medicine, Zoram Medical College
8. Dr. Laldinthari, Dermatology and STD, Zoram Medical College
9. Dr. Zorinsangi Varte, Medicine, Zoram Medical College
10. Dr. Gracy Laldinmawii, Microbiology, Zoram Medical College
11. Dr. Vanremmawii, Obstetrics and Gynaecology, Zoram Medical College
12. Dr. Zoramthara Zadeng, Ophthalmology, Zoram Medical College
13. Dr. Lalnuntluanga Sailo, Pulmonary Medicine, Zoram Medical College
14. Dr. Laltlanzovi, Pathology, Zoram Medical College
15. Dr. Wendy L Ralte, Radiology, Zoram Medical College
16. Dr. C. Vanlalhlua, General Surgery, Zoram Medical College
17. Dr. R. Lalnuntluanga, ENT, Civil Hospital, Aizawl
18. Dr. Lalrinmawia, Orthopedics, Civil Hospital, Aizawl
19. Dr. Lalthankimi Ralte, Emergency Medicine, Civil Hospital, Aizawl

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INTRODUCTION

The Standard treatment guidelines (STG) are the specific guiding principles with systematically developed statements to assist practitioners and prescribers make decisions about appropriate healthcare for specific circumstances.

Treatment Guidelines are designed to support the decision-making process in patient care. The guidelines are based on a systematic review of evidence-based health care delivery system by the selected experts within the State.

The key features of STG are

1. A few diagnostic criteria are listed for each health problem with clear and concise drug and dosage information.
2. Credibility because guidelines are developed by the most respected clinicians and revisions based on actual experience.
3. Doctors and other health care providers will use the same standard treatment.
4. Drug supply should be standardized i.e., matched to the recommended treatments and drugs on the list of essential drugs.
5. As bacterial resistance patterns change or other factors alter therapeutic preferences, the standards are revised to reflect current recommendations.

The main purpose of STGs is

1. To describe appropriate care based on the best available scientific evidence and broad consensus.
2. To reduce inappropriate variation in practice.
3. To provide a more rational basis for referral.
4. To provide a focus for continuing education.
5. To promote efficient use of resources.
6. To act as a focus for quality control, including audit.

RATIONAL DRUG USE

The Alma-Ata declaration, during the International Conference on Primary Health Care in 1978, reaffirms that health is a fundamental human right and the attainment of the highest possible level of health is the most important worldwide social goal. Medicines are an integral part of any health care system. They not only save lives and promote health, but prevent epidemics and diseases too. Medications are undoubtedly one of the weapons of mankind to fight disease and illness. Accessibility to medication is a fundamental right of every person.

A DRUG is a substance which may have medicinal, intoxicating, performance enhancing or other effects when taken or put into a human body or the body of another animal and is not considered a food or exclusively a food.

A medication or MEDICINE is a drug taken to cure and/or ameliorate any symptoms of an illness or medical condition, or may be used as preventive medicine that has future benefits but does not treat any existing or pre-existing diseases or symptoms.

The selection of essential medicines is only one step towards the improvement of the quality of health care; selection needs to be followed by appropriate use. Unfortunately, because of inappropriate use, the effective medicines of yesterday become ineffective today. Thus, in addition to achieve improve accessibility of essential medicines (availability and affordability), it is equally necessary to use the medicines appropriately, known as using rationally. The appropriate use of medicines selected in the EDL promotes rational use of medicines. Rational use of drugs is an issue of the utmost importance.

Unfortunately, in real practice, prescribing patterns do not always conform to these criteria and can be classified as "INAPPROPRIATE" OR "IRRATIONAL" PRESCRIBING. Irrational Prescribing can be regarded as "pathological" prescribing.

IRRATIONAL DRUG USE is a very serious global public health concern. Rational use could be greatly improved if a fraction of the resources spent on medicines were spent on improving use. Worldwide, more than 50% of all medicines are prescribed, dispensed, or sold inappropriately; while 50% of patients fail to take them correctly. "Arithmetically, that would mean that less than a quarter of medicines prescribed are used appropriately. Moreover, about one- third of the world's population lacks access to essential medicines.

Common patterns of irrational prescribing may be manifested in the following forms:

- The use of drugs, when no drug therapy is indicated.
- Antibiotics for viral URI infections.
- The use of a wrong drug for a specific condition requiring drug therapy.
- Tetracyclines in child hood diarrhea requiring ORS and Zinc.
- The use of drugs with doubtful / unproven efficacy.
- The use of antimotility agents in acute diarrhea.
- The use of drugs of uncertain safety status.
- The use of Baralgan etc.'
- Failure to provide available, safe and effective drugs.

  Failure to vaccinate against measles, tetanus, etc.
- The use of correct drug with incorrect administration, dosage and duration.

  The use of IV metronidazole, when oral or suppository formulations would be appropriate.
- The use of unnecessary expensive drugs
- The use of third generation, broad - spectrum antimicrobial, when a first line, narrow spectrum agent is indicated.
- Indiscriminate use of injections.
- Multi-drug use or polypharmacy: The number of drugs per prescription is often more than needed, with an average of 2.4 up to ten drugs, while generally one or two drugs would have sufficed. Multi-drug use is also common among consumers who purchase their drugs (over the counter drugs).
- Excessive use of antibiotics for treating minor ARI.
- Minerals and tonics for malnutrition.
- Factors underlying the irrational use of drugs
- Patients - Drug misinformation

  Misleading beliefs

  Patient demands / expectations.
- Prescribers - Lack of education and training-

Lack of objective drug information

misleading beliefs about drugs efficiency -With the fear that another physician will prescribe antibiotics and get the credit.

- Work place - heavy patient load.

  Pressure to prescribe.

  Lack of adequate lab capacity Insufficient

  staff to handle heavy load

- Drug supply - unreliable supply system - Shortage of Drugs

  Expired drugs supplied

- Drug Regulation- Non-essential drugs available.

  Non-formal prescribers.

  Lack of regulation enforcement.

- Industry - Promotional activities

  Misleading claims.

Impact of irrational use of drugs

Incorrect use of medicines occurs in all countries, causing harm to people and wasting

resources. Consequences include:

- Reduction in the quality of drug therapy leading to increased morbidity and mortality.

  Antimicrobial resistance.

- Adverse drug reactions and medication errors - Harmful reactions to medicines caused by wrong use, or allergic reactions to medicines can lead to increased illness, suffering and death.
- Wastage of resources leading to reduced availability of other vital drugs and increased cost. Out-of-pocket purchases of medicines can cause severe financial hardship to individuals and their families.
- Eroded patient confidence -Exacerbated by the overuse of limited medicines, drugs maybe often out of stock or at unaffordable prices and as result erode

patient confidence. Poor or negative health outcomes due to inappropriate use of medicines may also reduce confidence.

The inappropriate use of medicines is widespread. It is costly and extremely harmful both to the individual and the population as a whole. Increased incidence of adverse drug events and development of drug resistance is another serious issue.

A classic example is antimicrobial resistance. There is increasing anti-microbial resistance, with resistance of up to 70-90% to original first-line antibiotics for dysentery (shigella), pneumonia (pneumococcal), gonorrhea, and hospital acquired infections

The consequence of irrational drug use- The antimicrobial misuse problem

The antimicrobial resistance is one of the world's most serious public health problems. Many of the microbes that cause infectious diseases no longer respond to common anti-microbial drugs such as antibiotics, antiviral and anti-protozoal drugs.

The discovery of antimicrobials is one of the most important advances in health in human history – decreasing suffering from disease and saving lives. Antimicrobial agents are considered "miracle drugs" that are our leading weapons in the treatment of infectious diseases.

Antimicrobial resistance (AMR) is the ability of microorganisms that cause disease to withstand attack by antimicrobial medicines. From drugs used to treat common bacterial infections, to do complex combinations now fighting HIV infection, resistance is increasingly being detected and is spreading rapidly. In some parts of the world, once powerful medicines against malaria and tuberculosis have now become virtually useless. AMR is rapidly becoming a major public health risk and is threatening to undo decades of advances in our ability to treat diseases. It is challenging our whole understanding of how we control communicable diseases.

Emergence of resistance is a natural phenomenon that follows use of anti-microbials but it is being accelerated by inappropriate anti-microbial use and higher consumption is associated with higher resistance. The extensive misuse of anti-microbial agents leads to bacterial pathogens becoming resistant, thereby rendering treatment ineffective. "The rapid and alarming spread of anti-microbial

resistance around the world has not been matched by a concerted and powerful public health response." Moreover, fewer new antibiotics are being developed to replace those rendered ineffective through resistance.

Bacteria naturally develop resistance due to the selective pressure applied by both responsible and irresponsible use of antibiotics, which effectively limits the efficacy of any one antibiotic over time. This realization coupled with negligible innovation in antibiotics over the last few decades highlights the critical need for responsible use of antibiotics to try & slow down the further development of antimicrobial resistance.

Promoting the rational use of drugs

The concept of rational drug use is age old, as evident by the statement made by the Alexandrian physician, Herophilus, in 300 B.C that "Medicines are nothing in themselves, but are the very hands of god if employed with reason & prudence."

In simplest words rational use means "patient receiving appropriate drug to clinical needs, in adequate dose for the sufficient duration and at the lowest cost possible."

WHO definition of Rational Use of Drugs

"Rational use of drugs requires that patients receive medications appropriate to their clinical needs, in doses that meet their own individual requirements for an adequate period of time, and the lowest cost to them and their community"

The definition implies that rational use of drugs, especially rational prescribing should meet following criteria:

- Appropriate indications

  The decision to prescribe drug(s) is entirely based on medical rationale and that drug therapy is an effective and safe treatment.

- Appropriate Drug

  The selection of drugs is based on efficacy, safety, suitability and cost considerations.

- Appropriate Patient

No contra indications exist and the likelihood of adverse reaction is minimal, and the drug is acceptable to the patient.

- Appropriate Information

  Patients should be provided with relevant, accurate, important and clear information regarding his or her conditions and the medication(s) that are prescribed.

- Appropriate Monitoring

  The anticipated and unexpected effects of medications should be appropriately monitored.

Guidelines for rational use of drugs

- Prescribing a drug only when genuinely indicated.
- Using drugs indicated for specific conditions
- Choosing drugs which are effective.
- Using single-ingredient drugs.
- Choosing drugs which are relatively safe.
- Choosing cheaper alternatives.

Steps required to rationalize the use of drugs in the Market are

- Elimination of new drugs, which are expensive and not necessary because other drugs with proven efficacy already exist in the market.
- Elimination of useless, hazardous and harmful drugs which have irrational combinations.
- Use of essential drugs list.
- Marketing of drugs by their generic names.

Generic vs brand names

Every medicine has a generic name. It is almost always the name of the drug's active compound. Brand names are added by the marketing department of

pharmaceutical companies. To stimulate research and offset the cost of developing new medications, the FDA allows a company that develops a drug to be the only one to sell it for a specified period. When that's over, other companies can sell a medication made with the same active ingredient. These are the generics.

Generic drugs are chemical clones of their brand-name counterparts. A generic drug is identical or equivalent to a brand name drug in dosage form, safety, strength, route of administration, quality, performance characteristics and intended use. In other words, their pharmacological effects are exactly the same as those of their brand-name counterparts. What's different is the look of the drug and the inactive ingredients. These can make a difference in how the drug works for some individuals, but that's uncommon.

Though generic drugs are chemically identical to their branded counterparts, some consumers are reluctant to use generic formulations, thinking they are inferior to "the real thing" because generic drugs are often substantially cheaper than the brand-name versions. Doctors are also a big part of the problem. Up to half of physicians hold negative perceptions about generic drugs. Prescribing a brand- drug when a generic is available "is a huge source of wasteful spending that can be prevented,"

It's hard to resist a patient's request for a brand-name drug. Doctors are often evaluated on how satisfied their patients are—it's easier to say yes than risk a negative evaluation. They tend to have packed schedules, and it takes less time to write the brand-name prescription than it does to explain why the generic will do just fine. Some practitioners are influenced, consciously or not, by their interactions with drug company representatives.

Generic Drugs Are as Good as Brand Names! Health professionals and consumers can be assured that FDA approved generic drugs have met the same rigid standards as the innovator drug.

The essential medicines should be the first choice during medical practice. Finally, health care providers should take care of their clients, the patients, by spending some time with them explaining the appropriate use of prescribed medicines. The patients should be accepted as the partner in drug therapy prescribing.

THE CONCEPT OF ESSENTIAL MEDICINES

According to the WHO: Essential medicines are those that satisfy the priority health care needs of the population. They are selected with due regard to public health relevance, evidence on efficacy and safety, and comparative cost-

effectiveness Essential medicines are intended to be available within the context of functioning health systems at all times in adequate amounts, in the appropriate dosage forms, with assumed qualify and adequate information, and at a price the individual and the community can afford. The implementation of the concept of essential medicines is intended to be flexible and adaptable to many different situations; exactly which medicines are regarded as essential remains a national responsibility." Basically, an essential drugs policy means the availability of minimum number of rational drugs that will satisfy the health care needs of the majority of people.

Advantage of an Essential Drugs List

1. Medical Advantages

- It is medically, therapeutically and scientifically sound, and it ensures rational use of drugs.
- If limits the use of irrational and hazardous drugs and decreases the risks of iatrogenesis (drug and doctor induced disease).
- It improves the possibility of monitoring adverse drug reactions in patients.

2. Economic Advantages

- It is economically beneficial to the nation because it prevents wastage of scarce resources on non-essentials.
- The economies of scale achieved in the larger production of priority drugs brings down their prices.
- It curtails the aggressive marketing of non-essential formulations.
- It is economically beneficial to the patient because it prevents wastage on irrational and non-essentials.

3. Social Advantages

- It responds to the real health needs of people.
- It facilitates the dissemination of correct information about the drugs to health personnel, medical practitioners and consumers in general.
- It makes it imperative to draw up priorities to meet the most urgent needs of the people for essential health care.

4. Administrative Advantages

- It is organizationally sound because it makes qualify control of drugs, because of the limited number of drugs to be monitored. -
- It facilitates the streamlining of production, storage, and distribution of drugs, because of the smaller number of drugs involved.
- It helps in the clear identification of drugs.
- It facilitates the fixing of prices as well as revision/ withdrawal of duties, sales tax etc.

Selection Criteria

Which treatment is recommended and which medicines are selected depend on many factors, such as patterns of prevalent diseases, treatment facilities, the training and experience of available personnel, financial resources, genetic, demographic and environmental factors? The following criteria are used by the WHO Expert Committee on the selection and use of Essential Medicines: -

- Only medicines for which sound and adequate evidence of efficacy and safety is available should be selected.
- Relative cost-effectiveness is a major consideration for choosing medicines within the same therapeutic category.
- In comparisons between medicines, the total cost of the treatment & not only the unit cost of the medicine must be considered and should also be compared with its efficacy.
- In some cases, the choice may also be influenced by other factors such as properties or by local considerations such as the availability of facilities for manufacture or storage.
- Each medicine selected must be available in a form in which adequate quality, including bioavailability, can be ensured; its stability under the anticipated conditions of storage and use must also be determined.
- Most essential medicines should be formulated as single compounds.
- Fixed dose combination products are selected only when the combination has a proven advantage in therapeutic effect, safety, adherence or in decreasing the emergence of drug resistance in malaria, tuberculosis and HIV/AIDS

The process by which medicines are selected is critical. Selection of the medicines should be liked to evidence-based standard clinical guidelines. The essential medicines list needs to be specific addressing the disease burden, keeping in mind the healthcare needs of the majority of the population. The medicines should be available at affordable costs and with assured quality. The medicines used in the various national health programmes, emerging and reemerging infections should be addressed in the list. As the list needs to be developed locally and should be based on evidence not merely on individual's experience, it is necessary first to develop clinical guidelines, called Standard Treatment Guidelines (STG). Then Essential Drug List is compiled based on STG.

The **ESSENTIAL DRUG LIST** for the State of Mizoram has been developed which addresses the prevalent diseases and specific conditions. This will further save the limited resources available under the free drug initiative.

On proper implementation of Essential Medicine Concept, it would be very helpful to reduce morbidity and mortality rates associated with the drug use. It also will improve the allocation of the resources leading for better availability of necessary drugs with proper costs. Overall, patients will be benefited with decreased risk of unwanted affects such as adverse drug reactions and the emergence of drug resistance. Promoting the rational use of medicines would definitely help mankind to fight the disease and illness for a better tomorrow.

ACCIDENT AND EMERGENCY STANDARD TREATMENT GUIDELINES

A. ACUTE GASTRITIS

It may present as retrosternal pain, heart burn and regurgitation, mostly occurring after meals

Diagnosis:

1. History
2. Response to a therapeutic trial with PPI for 1 week
3. In case of alarming symptoms Upper GI Endoscopy

What are the first line drugs for gastritis?

1. Proton pump inhibitors (PPIs) are the first-line therapy to treat gastritis due to H. pylori infection like Pantoprazole 40 mg. Omeprazole 20mg, Rabeprazole 20mg, Esomeprazole 40 mg which can be taken bedtime or in the early morning when stomach is empty for 4 weeks
2. Antacids can be used as a treatment for gastritis like sucralfate or sucralfate combined with oxetacaine etc.
3. Avoid drinking alcohol and eating foods that seem to irritate your stomach which may include those that are spicy, acidic, fried or fatty, weight loss, take small frequent meals, stop smoking
4. Regular homemade meals, avoid staying up late night, stress-free life, drink lots of water, lifestyle changes are cheap and easy to follow remedy

B. ACUTE GASTROENTERITIS

It is a self-limiting illness characterized by diarrhoea, abdominal cramps, nausea and vomiting caused by viruses or bacteria like E Coli, V Cholerae, S Aureus etc. Most of these are non-invasive or toxic diarrhea. Less commonly, patients present mainly with diarrhea with passage of mucous and/or blood in the stools.

Treatment:

1. Oral rehydration therapy is still the key treatment for acute gastroenteritis
2. Homemade food, soup taken in sips is also very useful
3. In ill patients with systematic symptoms with associated bloody diarrhea, traveler's diarrhea Tab Ciprofloxacin 500 mg twice a day for 3-5 days
4. In Amoebic dysentery Tab Metronidazole 800 mg 3 times a day for 7 days
5. In Cholera, Tab Doxycycline 100 mg twice a day for 5 days
6. Patients with clinical signs of dehydration especially young children or elderly may be hospitalized

Interventions to prevent diarrhoea, including safe drinking-water, use of improved sanitation and hand washing with soap, can reduce disease risk.

Severe dehydration constitutes a medical emergency requiring immediate resuscitation with IV fluids. IV access should be obtained and patients should be administered a bolus of 20-30 ml/Kg Lactate Ringer (LR) or normal saline (NS) solution over 60 minutes. RL may independently improve pH in such cases, as lactate gets converted to bicarbonate in vivo.

The WHO recommends treating all episodes of blood in the stools with antibiotics and to use ciprofloxacin as the first-line drug. Alternatives are pivmecillinam, azithromycin, and ceftriaxone.

C. FEVER

The overall mean temperature for healthy adult is 98.4-degree F. Temperature greater than 98.4 is considered to have fever. Fever maybe continuous, intermittent or remittent

Diagnosis:

1. If the history and physical examination suggest that it is likely to be more than a simple URI or viral fever, investigations are indicated
2. Workup will depend upon the extent and pace of illness which includes complete haemogram with ESR, blood for malaria, blood culture, Widal test, Urine analysis including culture
3. If fever persist for more than 2 weeks Chest Xray to be done even in the absence of respiratory symptoms
4. Ultrasonography in some cases of acute fever such as in amoebic liver abscess

Treatment:

1. Hydrotherapy with tepid water, rest and plenty of oral fluids
2. Tab Paracetamol 500-1000 mg 6-8 hourly- if fever does not come down after 2 hours Tab Ibuprofen 400-600mg 8 hourly
3. Antibiotic/antimalarials depending upon the cause suggested by clinical and laboratory evaluation

In most cases of fever, patient may either recover spontaneously or a diagnosis is reached after repeated clinical evaluation and investigations. If no diagnosis is reached in up to 3 weeks, patient is said to be having Pyrexia of unknown origin (PUO) and should be managed accordingly

D. UPPER GASTROINTESTINAL BLEEDING

UGI bleed may present as occult or overt bleed. Upper GI bleed is defined as bleeding from any site from pharynx to duodenojejunal flexure or more specifically up to ligament of Treiz and usually presents as hematemesis or melaena (black, tarry, sticky, foul smelling stools)

Features:

- Occult or overt bleed or fresh blood
- For diagnosis of GI bleed, need clinical history, examination and radiological/ endoscopic examination
- Active bleed is indicated by presence of fresh blood in vomitus, nasogastric tube aspirate, malena, passage of fresh blood in the stool

Treatment:

1. Acute GI bleed is an emergency and needs active management. Blood transfusion if haemoglobin <7g/dl
2. Maintain vital signs (BP, respiration, airways, temperature)
3. Insert a large bore IV cannula and send blood samples for Hb, TLC, platelets, coagulation profile, renal and liver function test, blood grouping and cross matching
4. Start IV fluids like Normal saline/Ringers Lactate/polymer. Replace blood as soon as available, if moderate to severe bleed or active bleed
5. Inj PPI IV 6 hourly for 72 hours
6. If variceal bleed is suspected, Inj Octreotide 50 microgram stat followed by 25 microgram/hour infusion or Inj Terlipressin 1-2 mg IV given 4-6 hourly

E. HYPERTENSION

Usually asymptomatic and discovered on routine measurement of blood pressure. Secondary hypertension presents as a part of a symptom complex as in acromegaly, cushings disease, renovascular or renal parenchymal disease, connective tissue disorders or coarctation of aorta

Features:

- Non-specific symptoms are fatigue, headache, epistaxis
- Uncontrolled hypertension can lead to target organ damage such as coronary artery disease(CAD), LVH, CVA, TIA, retinopathy, PVD, renal disease
- Associated risk factors are age >55years in males and >65 years in females, smoking, diabetes mellitus, hyperlipidemia, family history, obesity, sedentary lifestyle and ethnic group

Precautions to be taken while measuring BP:

The auscultatory method of BP measurement with a properly calibrated and validated instrument should be used. BP should be measured using an appropriate size cuff, bladder length 80% of arm circumference and width 40% of arm length in both upper limbs and atleast one lower limb in both supine and erect posture. Patient should have been resting for atleast 5 minutes and should not have consumed coffee or smoked during the last 30 minutes before measuring the BP. At least 2 measurements should be made. Systolic BP is the point at which the first of 2 or more sounds are heard and diastolic BP is the point before the disappearance of sounds

Management:

1. Reduce dietary sodium intake to no more than 100 mmol per day
2. Lifestyle modification- exercise like brisk walking, swimming or jogging at-least 20-30 minutes in a week
3. Weight control using a combination of dietary and exercise measures to maintain normal body weight
4. Moderation of alcohol consumption
5. Cessation of smoking
6. Control of other risk factors
7. Antihypertensive drug choices:
   a) Diuretics-elderly, obese, heart failure
   b) Beta blockers-young, CAD, vascular headache, AF

c) Calcium channel blockers-old age, CAD, AF, PSVT
d) ACE inhibitors-young, LVF, diabetes
e) Alpha blockers-potassium, diabetes, dyslipidemia

8. Thiazide diuretics should be used with caution in patients with diabetes, gout or history of hyponatremia
9. Beta blockers should be avoided in patients with bronchial asthma, reactive airway diseases or second- or third-degree heart block
10. ACE inhibitors should not be used in patients with history of angioedema
11. Drug combination used for additive effect and minimization of the side effects
12. Hypertension in pregnancy is treated with alpha methyldopa, beta blockers, vasodilators for safety of the foetus

Commonly used antihypertensive drugs are:

1. Tab Hydrochlorothiazide 12.5-25 mg OD or BD
2. Tab Benzthiazide 25mg+Triamterene 50 mg per day
3. Tab Indapamide 2-5 mg daily
4. Tab Atenolol 25-100 mg daily or Metoprolol 25-150 mg BD (Caution-contraindicated in asthma, peripheral artery disease, uncontrolled hypothyroidism, myocardial defects)
5. Tab Amlodipine 2.5-20 mg daily or Cap nifedipine 20-80 mg as sustained release daily (caution- may cause peripheral edema in some individual)
6. Tab Enalapril 2.5 mg and maybe increased to 40 mg daily or Lisinopril 2.5- 20 mg daily (may cause dry cough in some individual)
7. Tab Losartan 25-50 mg OD or BD or Telmisartan 40 mg orally OD
8. Tab Prazosin 1-20 mg/day in divided doses; first dose given at bedtime
9. Tab Terazosin 1-5 mg daily bedtime
10. Tab Clonidine 0.1-0.6 mg BD
11. Tab Methyldopa 25-1000 mg BD
12. Accelerated Hypertension with sudden rise in BP 220/130mmHg or more without papilloedema - Hospitalize

-Inj Enalapril 1.25-5mg IV 6 hourly

Or

Inj Nitroprusside 0.25-1microgram/kg/min iV infusion (dose to be titrated

with BP)

or

Inj Nitroglycerin 5-100 microgram/min infusion

Or

Inj Labetalol 20-80 mg IV every 5-10 min up to a total of 300 mg Follow up should always be adviced to the patient of hypertension

F. URTICARIA

It is a non- specific vascular response to a wide variety of stimuli. Acute urticaria presents with erythematous wheals, which may be associated with swelling of loose connective tissue(angioedema) affecting the lips, scrotum, larynx and trachea

Treatment:

1. Soothing applications like cold water sponging and clearance of airway in case of laryngeal edema
2. Tab Pheniramine maleate 25mg TDS for 1-2 weeks. In children 0.15 mg/dose 3or 4 times a day. The dose should be adjusted according to response and tolerance
3. Tab Hydroxyzine 10-5 mg TDS
4. Tab Cetirizine 10 mg OD
5. In severe cases, antihistaminic can be started IV and once controlled, patient is maintained on oral preparations

G. TRAUMA OF DIFFERENT ORIGIN

Cardiac arrest associated with trauma: Survival rates of 0-3.7% are reported for victims of traumatic cardiac arrest. Consider if there are reversible causes of cardiac arrest and treat which include hypoxia, hypovolemia, diminished cardiac output secondary to pneumothorax or pericardial tamponade and hypothermia

BLS modifications- A jaw thrust should be used instead of a head tilt-chin lift to patient airway. Stop any visible hemorrhage using direct compression and appropriate dressings.

If the patient is completely unresponsive despite rescue breathing, provide standard CPR and defibrillation as indicated

Management: Pre hospital care

1. Airway management
2. Chest trauma- clinically assess for presence of pneumothorax for the purpose of triage or intervention.
3. Hemorrhage - Use simple dressings with direct pressure to control external hemorrhage. In patients with major limb trauma use a torniquet or bandage roll, rubber tubing, crepe bandage, if direct pressure has failed to control life-threatening hemorrhage. Inj. Tranexamic 1g IV over 10 min followed by 1 g IV over 8 hours as soon as possible with major trauma and active or suspected active bleeding
4. Analgesic for pain
5. Spine trauma-Carry out full in-line spinal immobilization by placing two blocks on either side of the head. IV fluid bottles can also be used to prevent movement
6. Fractures- Do not irrigate open fracture of the long bones, hindfoot or midfoot. Use saline soaked dressing covered with an occlusive layer.

HOSPITAL CARE: -

Airway management- Quickly assess by clinical examination of the thorax and respiratory function and observe for following: on inspection, palpation and percussion of the thorax together with pulse oximetry and in ventilated patients. Airway assessment reveals one of three clinical scenarios:

a) Patient is conscious, alert, talking- give high flow oxygen via face mask
b) Patient has a reduced conscious level but airway control and gag reflex present-there is no need for immediate intervention. Endotracheal intubation maybe done later, depending on the clinical condition

c) Patient has a reduced conscious level, gag reflex absent-

d) Secure airway with endotracheal intubation. Patients with multiple injuries must be preoxygenated before anesthesia. If intubation failed for more than 3 attempts, consider alternative methods like emergency cricothyroidoctomy or surgical airway for ventilation

e) For anesthesia Inj. Ketamine 1-2 mg/Kg

f) Monitor and control ventilation by frequent ABG analysis

Assessment of Chest Injuries:

a) Decompress immediately in case of tension pneumothorax with acute severe respiratory distress. Use open thoracostomy instead of needle compression, if experts are available.

b) Observe the patients after chest decompression for signs of recurrence of the tension pneumothorax

c) In case of open pneumothorax, cover it with a simple occlusive dressing and observe for the development of a tension pneumothorax

d) In case of perforating chest injuries, remove embedded foreign bodies during surgery only under controlled conditions after opening up the chest and immediate exploratory thoracotomy

e) Immediate chest Xray as part of the primary survey to assess chest trauma with severe respiratory compromise. CT also advisable

f) Manage hemorrhage and shock

SPINAL TRAUMA

Assessment for spinal injury: -

- On arrival at the hospital, use a prioritizing sequence to assess people with suspected trauma
- History and thorough clinical examination for spinal injury including the functions associated with it must be carried out
- The spine is suspected to be stable, unless any of the following 5 criteria are present: **impaired consciousness, neurologic deficit, spinal pain, intoxication, trauma in the extremities**
- Assume the presence of spinal injury in unconscious patients until evidence to the contrary is found
- Carry out full in-line immobilization if polytrauma with suspected traumatic

brain injury or if this assessment cannot be done

- Procedure for spinal immobilization: Let the patient find a position where they are comfortable with manual in-line spinal immobilization.
- Manually stabilize the head with the spine in-line using the following stepwise approach:
    - Fit an appropriately sized semi-rigid collar unless contraindicated by a compromised airway, known spinal deformities
    - Reassess the airway after applying the collar
    - Place and secure the person on a stretcher

Radiological investigations: -

- Cervical spine Xray if CT is not available
- MRI if after CT there is a neurological abnormality which could be attributable to spinal cord injury
- Perform an x-ray as the first-line investigation for people with suspected regions. Perform CT if the x-ray is abnormal or there are clinical signs and symptoms of a spinal column injury without abnormal neurological signs or symptoms in the thoracic or lumbosacral spinal column injury.
- Do not use the following medications, aimed at providing neuroprotection and prevention of secondary deterioration, in the acute stage after acute traumatic spinal cord injury: methylprednisolone, nimodipine, naloxone. Do not use medications in the acute stage after traumatic spinal cord injury to prevent neuropathic pain from developing in the chronic stage

HEAD INJURY

- The management of head injury patients should be guided by clinical assessments and protocols based on the Glasgow Coma Scale (GCS)
- Symptoms and signs of severe forms may appear immediately as in concussion or contusion or may appear after a few minutes to hours as in acute subdural hematoma.
- Patients with history of unconsciousness at any time since injury, amnesia for the incident or subsequent events, severe and persistent headache, nausea, vomiting, bleeding from nose/ear, seizures or presence of black eye, suspected fracture of skull and hematoma of scalp indicate severe form of head injury and require hospitalization.

Severe Head Injury (GCS <8 or less): - Patients with suspected open or depressed skull fracture, haemotympanum, panda eyes, CSF leakage from ear or nose, battles sign, persistent confusion, behavioral change, post-traumatic seizures, coma, focal neurological signs and features of raised intracranial pressure require immediate attention and should be admitted to the hospital. A CT should be done in all cases and treated as follows:

Check and maintain airway and breathing

- Check and maintain airway and breathing
- Check circulation by pulse, volume, rate and BP
- Establish IV access
- IV fluids according to volume loss: NS/ crystalloids are a fluid of choice
- Check for and stabilize extracranial injuries
- A head injury maybe accompanied by a cervical injury. If spinal injuries are excluded, then transfer the patient in a side position with head down, to a tertiary care centre where neurosurgical interventions are available
- If spinal injury is suspected then transfer the patient on a hard board, place two sand bags on either side of the head
- In case of raised intracranial pressure with signs of transtentorial herniation (pupil widening, decerabrate rigidity, extensor reaction to painful stimuli, progressive clouded consciousness), give IV Mannitol 20% 0.25-1g IV over 30- 60 minutes every 3-4 hourly
- Do not administer glucocorticoids in traumatic brain injury

H. CHRONIC LIVER DISEASE

Chronic liver disease (CLD) is a progressive deterioration of liver functions for more than six months, which includes synthesis of clotting factors, other proteins, detoxification of harmful products of metabolism, and excretion of bile. It refers to chronic conditions that do progressive damage to your liver. Viral infections, toxic poisoning and certain metabolic conditions are among the common causes of chronic liver disease.

CLD progresses in roughly 4 stages:

1. Hepatitis
2. Fibrosis
3. Cirrhosis
4. Liver failure

Symptoms:

- Jaundice- yellowish coloration of skin, eyes, mucus membrane
- Dark-colored urine
- Light colored stool
- Indigestion
- Weight and muscle loss
- Musty-smelling breadth
- Hepatic encephalopathy
- Pruritis
- Nail clubbing
- Ascites with associated pedal edema

Complications:

1. Decompensated cirrhosis with liver failure
2. Portal Hypertension with associated Upper GI bleeding
3. Liver cancer

Management:

1. Antiviral
2. Diuretics
3. Corticosteroids
4. Immunosuppressants

5. Liver transplant

In chronic liver disease, prescribe drugs to a minimum, prefer drugs that do not need hepatic metabolism for elimination, lowest effective dose of a drug, avoiding hepatotoxic drug. Drugs that precipitate hepatic coma are: anxiolytics, hypnotics, frusemide, phenobarbitone, promethazine, opioids. Ask the patient to bring a list of all the prescription and non- prescription medication, including herbs, vitamins and supplements to every physician's appointment. If the patient is taking several medications, make sure the ingredients are not the same. Explore if the patient drinks a significant amount of alcohol daily, avoid or restrict the use of acetaminophen

I. RESPIRATOTY TRACT INFECTION/ PNEUMONIA

Pneumonia is an inflammation in alveolar tissue, most often caused by a microbial agent. Inhalation is the commonest route of infection. The most frequent inhalational pneumonia is the community acquired pneumonia (CAP), which is most commonly caused by Streptococcus pneumoniae(typical). Nosocomial pneumonia is likely to be caused by gram-negative bacilli or Staph aureus. Aspiration pneumonia, is polymicrobial including anaerobes. Age is an important predictor of infecting agent

Features:

- Sudden onset of fever, productive cough, chest pain, shortness of breath and in some cases pleuritic chest pain; systemic symptoms like headache, body ache and delirium are more severe with atypical pneumonia
- New focal signs on physical examination of the chest
- In contrast, the elderly patients differ in their presentation.
- The atypical pneumonia syndrome is characterized by a more gradual onset, a dry cough, SOB and prominence of extrapulmonary symptoms (myalgia, headache, fatigue, sore throat, nausea, vomiting and diarrhoea) and abnormalities on chest Xray despite minimal signs of pulmonary involvement (other than rales)
- The "primary atypical pneumonia" caused by M pneumoniae results in a violent, episodic cough with small mucoid sputum preceded by fever with or without chills and maybe accompanied by profound weakness
- Confirmation of the etiological diagnosis is by blood culture/sputum culture and/or molecular diagnostic methods

Treatment:

CURB65 score for the initial severity assessment and treatment of pneumonia. Antibiotics are the mainstay of treatment. The choice maybe modified based on response and sputum culture

CURB65 severity score (1 point for each feature)

- C=confusion
- U=Urea>7mmol/l
- R=Respirator rate=30/min
- B=BP<90or DBP=60mmHg
- Age=65 years

Plan A: Outpatient treatment-previously healthy without risk factors

Cap Azithromycin 500 mg once daily OR Cap Doxycycline 200 mg OD for 10 days

Plan B: Presence of comorbidities like chronic heart/lung/liver/renal disease/diabetes/alcoholic liver/malignancies/use of immunosuppressant drugs etc.,

1. Inj. Amoxycillin 1G 8 hourly IM/IV OR Cap Amoxycillin+Clavulunate 2G BD OR Inj. Ceftriaxone 2G IV OD OR Tab Cefpodoxime 200 mg BD
2. Cap Azithromycin 500mg OD for 5 days OR Cap Clarithromycin 500 mg BD for 10 days OR Cap Doxycycline 200mg loading dose followed by 100 mg OD for 10 days

Plan C: Inpatient, non-ICU treatment

1. Inj. Cefotaxime 1-2G IV 8-12 hourly OR Inj. Ceftriaxone 2G IV OD OR Inj. Ampicillin 1g IV 8 hourly. Patients allergic to penicillin, Levofloxacin 750mg OD may be given
2. Inj. Azithromycin 500mg IV OD OR Clarithromycin 500 mg IV BD
3. For selected patients, Inj. Ertapenem 1g daily IV plus Inj. Azithromycin 500 mg IV OD or Clarithromycin 500mg BD or Doxycycline 200mg loading dose followed by 100 mg OD for 10 days. Monitor with C- reactive protein concentration in patients with community-acquired pneumonia on admission to hospital and repeat after 48-72 hours, if clinical progress is uncertain

Plan D: Inpatient, ICU treatment

1. A Beta lactam (cefo/ceftria) plus Azithromycin OR Inj. Levofloxacin 750 mg by slow IV infusion over 90 minutes every 24 hours
2. If pseudomonas is suspected:

   Inj. Piperacillin + Tazobactam 4.5g IV/IM 6 hrly or Inj. Cefepime 1-2g IV 8 hrly or Inj. Imipenem 500 mg IV 8 hrly or Meropenem 500 mg-1g IV 8 hourly

Supportive treatment:

1. Concomitant use of bronchodilators (salbutamol, terbutaline) is beneficial for bronchospasm
2. Analgesic for fever and body ache
3. Noninvasive/ invasive ventilator

GENERAL MEDICINE STANDARD TREATMENT GUIDELINES

DIAGNOSIS OF DIABETES MELLITUS

Fasting levels are lower when compared to the corresponding post-prandial levels from the same site.

In the fasting state, venous= capillary values but in the post prandial state, venous is less thancapillary values as the rise in posr prandial insulin pushes glucose into tissues.

Whole blood glucose values are lower than plasma values as red cell glucose concentration is lowerthan plasma.

In the post prandial state, capillary whole blood and venous plasma values are the same For

glucose, 1 mmol/L =18 mg%

CRITERIA FOR TESTING FOR DM

1. Symptomatic patients (Polyuria, polydipsia, polyphagia, unexplained weight loss).
2. All individuals at age 45 or older. If results are normal, they should be repeated atappropriate intervals e.g. 1/year or at 3 year intervals.
3. Test should be done in younger individuals if:
    I. They are obese (BMI >30 for men and >28.6 for women)
    II. First degree relative with DM
    III. Are members of high risk ethnic population
    IV. Are hypertensive (BP > 140/90 mm of Hg)
    V. Have HDL cholesterol of less than/=35mg% and/or triglycerides of more than/=250mg%
    VI. On a previous testing had IGT (Impaired glucose tolerance) or IFG (Impaired fastingglucose)
    VII. Presenting with acute MI/stroke, peripheral neuropathy, proteinuria, peripheralvascular disease.
4. In pregnancy if GDM (Gestational DM) is suspected-

Criteria for diagnosing
Diabetes Mellitus
American Diabetes Association:
Clinical Practice Recommendations 2002

1. Symptoms of diabetes plus casual plasma glucose concentration >200mg/dL (11.1 mmol/l).Casual is defined as any time of the day without regard to time since last meal. The classic symptoms of DM include polyuria, polydipsia and unexplained weight loss.
   OR
2. FPG > 126 mg/dl (7.0 mmol/l). Fasting is defined as no calorie intake for at least 8 hours.

OR

3. 2-h PG >200mg/dl (11.1 mmol/l) during an OGGT #. The test should be performed as described by WHO, using a glucose load containing the equivalent of 75 g anyhydrousglucose dissolved in water.
   In the absence of unequivocal hyperglycemia with acute metabolic decompensation, these criteria should be confirmed by repeat testing on a different day. The third measure (OGGT- oral glucose tolerance) test is not recommended for routine clinical use.

#Values for diagnosing DM and other forms of hyperglycemia based on OGTT (75gm of glucose) (Ref: 1998 WHO consultation group)

CMCH laboratory values correspond to the venous plasma values. Glucose concentrations should NOT be measured on serum unless red cells are immediately removed, otherwise glycolysis will result in an unpredictable underestimation of the true concentrations. For epidemiological purpose of screening for DM, 2-h post glucose load value exceeding 200mg% in venous plasma is sufficient asmentioned above.

OGTT (75 gm of glucose) Ref : 1998 WHO consultation group

Fasting values reflect ENDOGENOUS glucose production Post prandial values reflect EXOGENOUSintake

AIMS OF TREATMENT IN DIABETES MELLITUS

- Save life
- Alleviate symptoms
- Prevent long term complications
- Reduce risk factors- Smoking, HT, Obesity, Hyperlipidemia
- Educate and encourage self management

TARGETS FOR CONTROL
ADA Recommendations 2006

BMI <27
Lipids:
LDL Cholesterol <100 mg% HDL
Cholesterol >45 mg%
Trigycerides<150 mg%

Blood Pressure < 130 /80 mm Hg

Glucose:	Plasma	Whole Blood
	Fasting 90 – 130 mg%	80 – 120 mg%

	Bedtime 110 – 150 mg%	100 – 140 mg%
HbA, C	7%	

Less stringent goals are appropriate for the very old, those with limited life expectancy and those with frequent hypoglycemic attacks. Elevated post challenge (2hour OGTT) has been associated withincrease cardiovascular risk irrespective of the value of fasting plasma glucose. There are pharmacological agents that primarily modify post prandial glucose and monitoring post prandial glucose 2 hours after a meal with an aim to reduce it to < 180mg% lowers HbA,C in those with high HbA,C and normal fasting glucose. However, there are no studies are available to show the efficacy of such a regimen to reduce complications in those with either Type I or Type II Diabetes mellitus.

Specify therapy for Post Prandial Hyperglycemia:

- Metformin
- Repaglinide
- Acarbose
- Insulin lispro

INSULIN THERAPY IN DIABETES LELLITUS

Insulin can be bovince porcine, human. Human insulin is structurally similar to the psysiological insulin secreted by the human pancreas, whereas porcine insulin differs from human by 1 amino-acid and bovine differs from human by three amino-acids.

Human insulins produced by genetic re-engineering techniques are now used universally after reduction in prices. However, monocomponent bovine and porcine insulin are still available for verypoor patients.

Strength: The commonly available strengths are – 40 IU/ml and 100IU/ml. It is important to use the syringes corresponding to the strengths of insulin to avoid hypoglycaemia.

Storing: Can be stored temperatures of 2-8 °C (first shelf of the refrigerator) if needed to store for long periods. It can however be kept at room temperature (Indian conditions), away from sunlightfor about 4-6 weeks with no apparent loss in biological activity.

Purity: Categorised into-

a) Highly purified insulin: with < 10ppm of pro-insulin like substances.
b) Monocomponent insulin: with < 1ppm of pro-insulin like substances.

Newer insulin: these are insulin analogues: Lispro, Glargine

Time duration and effect:

INSULINS BY COMPARATIVE ACTION CURVES				
Insulin type	**Onset**	**Peak (h)**	**Usually effective Duration (h)**	**Usual maximum Duration (h)**
Animal				
Regular	0.5-2.0h	3-4h	4-6	6-8
NPH	4-6h	8-14	16-20	20-24
Lante	4-6h	8-14	16-20	20-24
Human				
Lispro	<15mins	0.5-1.5	2-4	4-6
Regular	0.5-1h	2-3	3-6	6-10
Glargine		Peakless	24	24

1

2 A. Hypoglycemia

Clinical Features:

Early warning: Shaking, trembling, sweating, paresthesis in lips and tongue, hunger, palpitations,headache.

Neuroglycopenia;

MILD: Diplopia, difficulty in concentrating, slurring of speech

MODERATE: Confusion, behaviour change,

SEVERE: Restlessness with sweating, seizures, focal neurological deficits-hemiplegia (specially inelderly patients)

Prevention is most important. All DM patients should always carry sugar/ sweet food substances. Atthe onset of early warning symptoms, they should take 10-20 g of glucose immediately.

Management is by early recognition and bolus intravenous glucose 50% -50ml/25ml. Repeat bolusesmay be needed. If the patient has also been on OHA's, IV infusion of 5% dextrose/ DNS should be considered, till 48h at least as an inpatient till the drug gets washed out of the system, with close monitoring of sugars.

Repeated attacks of hypoglycaemia warrant a look for causes of reduced need for insulin, as well asdose reduction after proper titration.

1. B. DIABETIC KETO – ACIDOSIS

Ketoacidosis results due to lack of insulin. In practice it is usually due to:

a. Stopping Insulin/ reducing the dose- error/ deliberately;
b. Resistance to insulin during infections/ other stresses (acute MI, surgery, etc)
c. Unrecognized onset of IDDM

Clinical assessment:

Recognising DKA:

- Drowsiness
- Dehydration(5L deficit)
- Kussmaul's breathing
- Hypotension

Investigations:

Confirm diagnosis by:

- Hyperglycemia (blood glucose)
- Ketonemia (Urine acetone)
- Acidosis (blood gases)

Treatment:

Principles of treatment:-

1) The plasma glucose level invariably falls more rapidly than plasma ketone level. Insulin administration should not be stopped because glucose concentrations approach normal; Insulin should be infused along with glucose with adequate potassium correction till ketonesare cleared up.
2) The response to treatment is measured more accurately by arterial pH and onion gap rather than plasma ketone bodies which may erroneously give a rising value during the initial phaseof treatment.

Treatment of precipitating factors: Antibiotics for infection (Cefotaxime and Gentamicin) ,management of situations like MI, strokes, etc.

1. C. NON-KETOTIC HYPEROSMOLAR COMA (NKHOC):

A metabolic emergency in which the increased blood glucose causes increase in the osmolality, causing osmotic diuresis and therefore severe dehydration in the absence of ketosis and acidosis.The lack of ketosis is due to the presence of some circulating insulin (suboptimal levels) .

Clinical assessment: Severe dehydration (approximately 10-12L of fluid deficit), obtundation of the sensorium (stupor/coma). Usually elderly; profound dehydration can predispose to hypercoagulablestate causing stroke, AMI, arterial insufficiency in the limbs.
Investigations: Increased plasma osmolality and hyperglycemia in the absence of urinary ketone bodies and a normal pH on ABG. Calculation of osmolality can be done from the values of serum Na,K, Glucose and Urea.

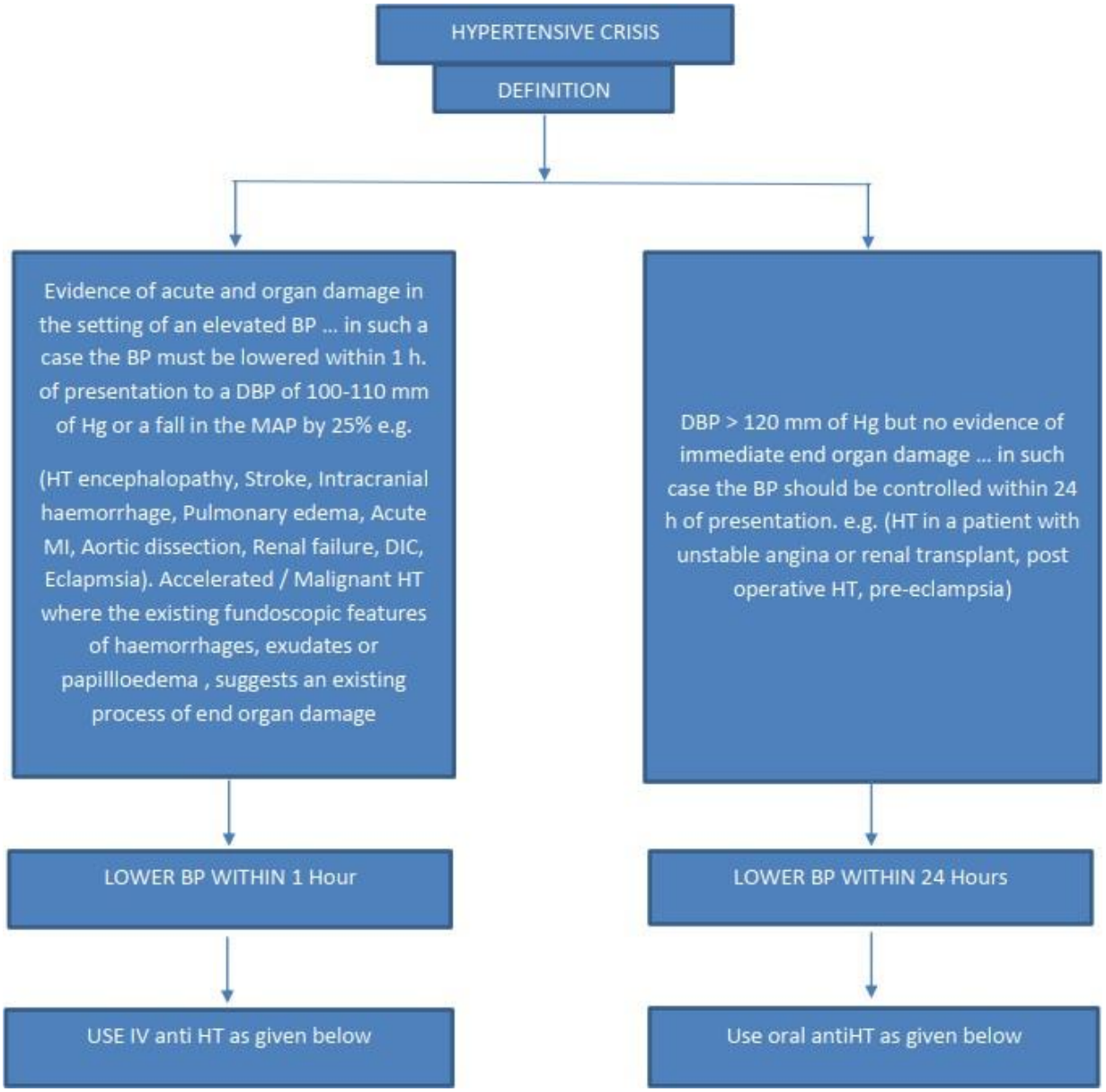
Formula: 2x (Na+K) + Glucose (mmol/L) + Urea (mmol/L)

Or 2x (Na+K)+0.055 x Glucose (mg%) +0.166 x Urea(mg%)

Treatment: Mostly as in DKA. The fluid deficit is more (10-12Ls); therefore to be placed carefully withQ2hly Na and K monitoring. Use 1/ 2 or 1/4 NS as serum sodium is high. Central venous pressure monitoring is absolutely essential because the volume of

correction is large and the rate of replacement should be adequate. DVT prophylaxis can be considered.

HYPERTENSIVE CRISIS



END ORGAN DAMAGE:

- HT encephalopathy: Presence of CNS dysfunction in a setting of SEVERE HT. Headache,depressed consciousness, vomiting, +/- seizures, +/- focal neurological deficits (if thesepersist, consider a cerebrovascular accident.)
- Retinopathy: cotton wool exudates, haemorrhages, papilloedema.Stroke/ Intracranial haemorrhage: focal deficits, raised ICT, vomiting, headache, loss of consciousness , +/- neck stiffness.

Do not treat HT in acute ischemic STROKE unless one of the following co-exists:

1. DBP=>130mm of Hg
2. Evidence of end organ damage
3. HT encephalopathy

- CCF/ unstable angina/MI
- Renal: worsening of proteinuria, hematuria, renal function.
- Hematological : Microangiopathic haemolytic anemia, DIC, Thrombocytopenia.
- HELLP syndrome (severe HT in pregnancy – ecclampsia): Hemolysis, elevated liver enzymes,low platelets.

INVESTIGATIONS:-

As in case of a hypertensive to look for end organ damage.**TARGET ORGAN DAMAGE**……Brain,Heart, Kidney, Vessels.

SCREEN FOR SECONDARY HT……. Electrolytes (K+), 24hr VMA and potassium in urine, renal USGDoppler, tests for Cushings (overnight DST)

Look for associated risk factors-Lipids, Blood glucose. Consider the

following in case of a pregnant lady:-

- Platelets (thrombocytopenia),
- Peripheral blood smear (schistocytes in microangiopathic hemolysis),
- LFT (elevated enzymes)

MANAGEMENT OF HT CRISIS

In the presence of end organ damage, parenteral anti-hypertensives are recommended.

- For patients with acute ischemic coronary symptoms, NITROGLYCERINE is preferref tonitroprusside. (IV infusion)
- In pregnancy, alpha-methyl dopa, nifedipine and labetolol appear safe.
- Captopril is the drug of choice for Scleroderma renal crisis.
- In aortic dissection, combine nitroglycerine/nitroprusside with a betasdrenergic blocker eg.Propranolol to prevent further dissection.

Drugs	Dosages	Onset	Peak	Side-effects
Nitroglycerine	5mcg /min, can go upto 400 mcg/min at increments of 5-10 mcg till desired effect.	1-2 mins	3-5 mins	Headache, vomiting Tachyphylaxis
Sodium nitroprusside	50-100mcg/min, (0.3-10 mcg/kg/min)	Immediate	2-3 min	Vomiting, Thiocyanate toxicity

Do not start these drugs in the casualty without close monitoring before transferring the patient tothe ICU/WARD.

Monitor during patient transport also.

Add on cerebral antioedema measures to bring down the ICT.

Management:- in the absence of acute end organ damage, the anti HT therapy should be throughoral route.

Drugs	Dose	Onset	Peak	Duration	Side effects
Aldomet	250mg, repeat 6-8h	2-4h	4-6h		Headache, Vomiting
Minoxidil	1.25-20 mg PO 12 hourly	1 hour	>1hr	24h	Tachycardia angina

Furosemide (Lasix) can be added on to any of these for enhancement of action. (Dose 40-80 mg PO,repeat 6th hourly).

*CAUTION- Sublingual Nifedipine should not to be used as it can cause a catastrophic fall in bloodpressure.

3 MANAGEMENT OF AN ACUTE ATTACK OF BRONCHIAL ASTHMA

AIMS OF MANAGEMENT

- Prevent death
- Restore clinical condition and lung function to best possible levels as soon as possible.
- Maintain optimal function and prevent early relapse.
- RECOGNIZE SEVERE ASTHMA- DO NOT GO BY AMOUNT OF WHEEZING!!
  PR>110/min, respiratory rate >25/min, Pulsus-paradoxus>10mm of Hg, PEFR<150 or 40% ofexpected/ prior value on the same patient or <1 Sentence / Breath
- VERY SEVERE ASTHMA
  Silent chest with dyspnoea, cyanosis, exhaustion, confusion or unconsciousness Bradycardia
  Features on ARTERIAL BLOOD GAS ANALYSIS
  -Normal/High $PaCO_2$ in a breathless person
  -Hypoxia : PaO_2<60 mm Hg irrespective of treatment with Oxygen
  -Acidosis

MANAGEMENT

- Admit, Bed resr
- Do not sedate
- Humildified Oxygen - use the highest concentration
  - Set a high flow rate
- Inhaled Beta 2 agonists
  Eg. Salbutamol 2.5-5 mg or Terbutaline 5-10mg
  Nebulised with oxygen/ with an air compressor/ by multiple actuations of a metered dose inhaler into a spacer device (2-5 mg i.e..20-50 puffs 5 puffs at a time).

How to administer?

Nebulized Salbutamol (5mg/ml) or Terbutaline (10mg/ml) given as 1 ml in 2-3ml of Normal Saline, toalternate with Ipratropium 0.2-0.5 mg every 4 hourly.

- Subcutaneous 1:1000 Adrenaline 0.3-0.5 ml, rpt 20-30 mins.x3 doses ECG monitoring
- Teerbutaline S.C.0.25-0.5ml (1mg/ml): repeat 30 mins interval x 2-3 doses
- If has been steroid dependent,
  OR
  FOR ALL CAUSES OF SEVERE ASTHMA give:-
  I.V Methylprednisolone 120- 180 mg/Q6 Hrly OR

Hydrocortisone 100 mg- 200 mg, Q6 Hrly
Oral Prednisole 100 mg Q 6 Hrly is as effective as injectable steroid.
Patient is to be discharged on oral steroids.
Do not taper until evidence of improvement noted objectively.
Thereafter a 2 week tapering schedule along with inhaled steroids Slower reduction in patients with history of respiratory failure.

- Aminophylline drip 0.5-0.9 mg/kg/h
- Hydration
- Antibiotics: prulent sputum does not mean infection, if suspected, macrolides/tetracyclinescan be started after a blood culture is taken.

INVESTIGATIONS:-

- Chest Radiograph- look for Pneumothorax, Consolidation and Pulmonary oedema
- ECG – in older patients
- Blood leucocyte counts, renal function tests, electrolytes.
- ABGases to look for onset of respiratory failure.

MAY REQUIRE VENTILATORY SUPPORT IF…….

1) PEFR <33% of predicted value
2) Cyanosis, hypoxia (PaO_2<60 mm of Hg)
3) Bradycardia/hypotension
4) Altered sensorium
5) High/rising Pa CO_2>45 mm of Hg. (Type II respiratory failure)
6) Systemic acidosis

4 CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

ESTABLISH DIAGNOSIS….

Check for sputum AFB before treating chronic cough as COPD! RECOGNIZE THE

EXACEBRATING FACTORS (MNEMONIC- 'DIPLOMAT')

- Drugs….beta blockers
- Infections three principle causative agents…H.Influenzae, S.Pneumoniae, M. Catarrhalis
- Pneumothorax……has to be ruled out –X-ray chest in expiration
- LVF….caused by hypoxia, acidosis, CO_2 retention, polycythemia, Bernheim effect
- Oxygen…hypoxic drive lost.
- Muscle fatigue…….
- Atelectasis……..
- Thromboembolism (pulmonary)……bedridden for prolonged duration.

CHRONIC OBSTRUCTIVE PULMONARY DISEASE

MANAGEMENT OF ACUTE EXACERBATION

General Measures

- ABCs as for any critically ill patient
- Adequate hydration: IV fluids
- Stop or treat the Precipitating factors
- Use Sedation with care.
- Non invasive ventilation is useful for acute exacerbations without respiratory muscle fatiguein conscious patients.
- Mechanical ventilation….indicated when there is respiratory arrest, refractory acidosis/hypoxia, severe respiratory muscle fatigue.

Specific Measures

- BRONCHODILATORS…variable degree of response in patients based on reversibility of thespasm. Nebulized Ipratropium bromide (0.5 mg) is alternated with short acting beta agonists.
- Xanthines….improves diaphragm activity (0.5-0.9mg/kg/hr aminophylline)
- Antibiotics…….amoxycillin for H.Influenza, macrolides for Mycoplasma and S.Pneumoniae.
- Corticosteroids……a subset of COPD patients respond to steroids; tapering over 2 wks.

LONG TERM THERAPY

- STEROID THERAPY: Unpredictable outcome in certain subgroups; record PFT before startingand document any change after 2-3 mo.
  Supplement with calcium and vitamin D.
- INHALERS: Ipratropium, Beta 2 stimulants.
- ORAL XANTHINES: Deriphylline.
- OXYGEN THERAPY: Increases survival; indicated when:-

1. COPD (Pa O_2 55-59 mm of Hg) is complicated with failure of another system e.g. COR-PULMONALE, PULMONARY HT,PSYCHOLOGIC IMPAIRMENT/ HEMATOCRIT >55%
2. Resting room air PaO_2 is <55mm of Hg or saturation <89% on two occasions
3. PaO_2>60 mm of Hg. At rest but falling to <55 mm of Hg. with exercise/sleep.

At least 16 hrs / day of oxygen is recommended. Flow rate of oxygen is to be titrated afteradmitting into the hospital so as to maintain O_2 saturation at 85-90% and yet not removethe hypoxic drive for respiration.

VENESECTION – If PCV > 55%, with CCF/cerebral dysfunction.

5 ANAPHYLAXIS

DIAGNOSIS

- HISTORY .....of skin test , drug ingestion/ injection, vaccination, radiocontrast study.
- CF……hypotension (vascular collapse) , flushing, pruritus, urticarial, angioedema, stridor (laryngeal edema) wheezing (bronchoconstriction), tachycardias, arrhythmias, pink frothysputum (pulmonary edema).
- PRESENTATION .....Severe respiratory distress and/or
  Vascular collapse with/without
  The other clinical features
- **MANAGEMENT……..**
  ADRENALINE....(1st drug)
  Dosage……0.5-1.0ml 1:1000 promptly deep intramuscular route…do not waste time ongetting IV lines!!
  Repeat ……every 15 mins. Until BP stable.
  If IV line available, adrenaline infusion 10mg/mt for 10 mts then 1-4 mg/mt Maintain airway and breathing…… intubate if necessary, oxygen.

ANTIHISTAMINES

Inj. Avil 50-100 mg IV. Or
Inj. Phenergan 25-50 mg IV –or-
InjChlorpheneramine Maleate 10-20 mg slow IV over 1 min. Maintain antihistamine cover for 24-48 hrs.

CORTICOSTEROIDS… Prevents late deterioration in severe cases – Not first line. Inj. Hydrocortisone 200- 500 mg IV or
Inj. Dexamethasone 4-8 mg IV
Continue for 12-24 hrs….. withdraw thereafter If symptoms reappear, add on another dose
INTRAVENOUS VOLUME MAINTENANCE…
Infuse adequate volumes of crystalloids /colloids/ plasma expanders as needed.
MONITOR… for 24 hrs for late phase IgE response OTHER MEASURES…
AMINOPHYLLINE…..if airway obstruction is present OXYGEN……in all patients
VASOPRESSORS……. If BP remains low even after adequate infusion of fluids
Patients who have been on beta blockers may need higher doses of ADRENALINE – or consider usingGLUCAGON.

6 BITES, STINGS

6A SNAKE BITES

FIRST AID:-

1. Focus on getting the victim to definite medical care.
2. Light Tourniquet to occlude lymphatic flow if bite is on an extremely…….pulses must be felt (prevent ischemia). One should be able to insinuate a finger under the tourniquet. Prevent walking if lower limb bitten.
3. Immobilisation of the limb, splinting.
4. No cooling / incision at site bitten.

HOSPITAL MANAGEMENT:-

- Quick history, brief but thorough examination
- Vital signs, cardiac rhythm, oxygen saturation
- IV Access.
- Investigate…. Blood for grouping, cross match, CBC, peripheral blood smears, renal andhepatic functions, CT/ platelet/ PT/PTT (DIC state).
- Urea, creat..potassium (ARF), Urine (microscopy).
- In severe cases…….ABG, ECG, C Xray
- Treat shock…..if volume resuscitation fails to bring up the BP try vasopressors.
- Invasive hemodynamic monitoring (central line)

DRUG THERAPY:-

- Tetanus immunoglobulin 250 U IV
  (avoid IM if clotting abnormality exists)
- Anti snake venom: Be ready for anaphylaxis- Give slowly at first. If anaphylaxis occurs stop & treat promptly- skin tests for sensitivity are not useful as anaphylaxis can occur even after a negative skin test.

HIGH DOSE – Regimes are not followed.

LOW DOSE – 2 Ampoules of ASV diluted in 500 ml of 5% Dextrose over 1 hour, followed by 1 vialover 4 hours until clotting parameters normalized and then 1 vial as an infusion over 24 hours.

Regime followed in medical ICU :

- 2 ampoules ASV (diluted as above) in 1 hour
- 3 ampoules in 500 ml dextrose q 12 hours x 36 hours
- 1 ampoules in 500 ml dextrose X 24 hours

Local injection at the site of snake bite is **not recommended**.

- **MONITORING:-**
  Single breath count
  Chest expansion
  Clotting time, PT, PTT

6B DOG BITES

GENERAL MEASURES

Local wound therapy :- Scrub with soap and flush with water. Chemical cleansing with 1% cetrimide(cetavation) inactivates the rabies virus.

Tetanus toxoid: 1 ml IM stat. (If previously immunized) Tetanus immunoglobulin – TIG – 250 unitsIM for unimmunized.

Antibiotics – Cover gram positive, gram negative and anaerobes.

SPECIFY THERAPY

Not previously vaccinated

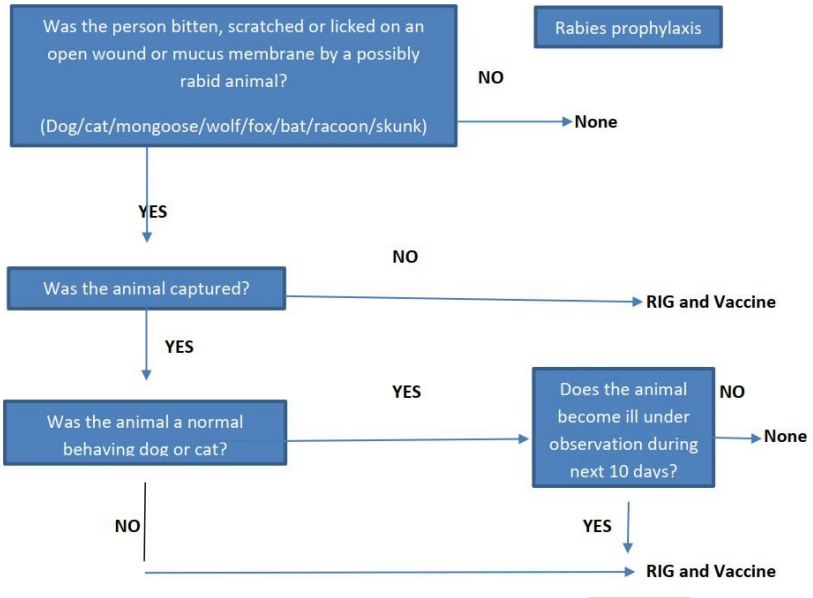
HRIG (Human Rabies Immunoglobulin) 20 IU/kg body weight. If anatomically feasible, half of it should be infiltrated around the wound(s) and the rest should be administered IM in the gluteal area. Never give more than the recommended dose. Do not use in the same syringe/ in the samesite as used for vaccination.

Vaccine- HCDV (Human Diploid Cell Vaccine) or RVA – 1.0 ml IM (deltoid area), one injection ondays 0,3,7,14 and 28.

Previously vaccinated –

HRIG – should not be given.
Vaccine- HDCV or RVA 1.0ml in the deltoid area IM on days 0 and 3. (Vaccine

should never be administered in the gluteal area)



6C SCORPION STING

OUT OF 86 SPECIES, ONLY 2 ARE POISONOUS

SCORPION VENOM is essentially acidic, contains neurotoxin, cardiotoxin, coagulases, proteases,phospholipase A, amylase and serotonin (causes pain).

Action of venom – causes complex effects on sodium channels – opens them – preventing inactivation- spontaneous depolarization- prolonged action potential – excessive firing of neurons.

CLINICAL FEATURES:- The venom simulates sustained release of acetylcholine and cetacholaminesresulting in initial cholinergic and late adrenergic symptoms.

CLINICAL SETTING:- Usually stung at night while sleeping; unknown sting with excruciating localpain; stamped upon accidentally.

IMMEDIATE SYMPTOMS:-

Local effects – intense pain (or itching, paraesthesiae, hyperesthesiae, numbness) is the most common symptom; slight local edema and tender local lymphadenopathy. Frank gangrene cansupervene.

General effects – More in children because the toxin per Kg body weight is more in them. Malaise, restlessness, lacrimation, rhinorrhoea, salivation, perspiration, nausea, vomiting, more so in youngerchildren called 'AUTONOMIC STORM'

Early:- vomiting, profuse sweating, piloerection, alternating tachy and bradycardia, abdominal colic,diarrhoea, loss of sphincter control, priapism.

Late:- severe/ life threatening

- **CVS:-** Cardiogenic Shock
- **Respiratory:-** Acute pulmonary edema
- **CNS:-** Encephalopathy

6D BEE and WASP STINGS GENERAL MEASURES

- Remove embedded stings from the skin with a blade or by brushing not by **forceps** as youmight squeeze in some more of the venom.
- Clean and disinfect the site of sting with soap and water , followed by spirit and then apply icepacks to slow the spread of venom. A tourniquet may slow the spread of venom.
- Tetanus toxoid 1 ampoule IM stat into the deltoids(if immunised) + Tetanus immunoglobulin (if unimmunised)
- Elevation of the affected site.
- Analgesics and oral antihistaminics topical Calamine lotion.
- Large local reactions may need oral therapy with Prednisolone.
- Patients with numerous stings should be monitored for coagulopathy and evidence of renalfailure.

SPECIFIC MEASURES

- Anaphylactic reactions can be managed with subcutaneous 0.3-0.5ml of 1: 1000 dilution of **Adrenaline**, repeated every 20-30 mins if necessary.
- Parenteral antihistaminics, bronchodilators, steroids, oxygen, intubation may be required asin any other form of anaphylaxis.

7 HEAT STROKE

WHEN TO SUSPECT?..... CNS dysfunction in a previously healthy individual with 'fever' and history of exposure to predisposing risk factors.

History:

- Exposure to heat stress, either from increased endogenous production or from environmental sources, is essential for the diagnosis of heat exhaustion or heat stroke.

HEAT EXHAUSTION

- Symptoms are often nonspecific and may be insidious in onset. The symptoms oftenresemble a viral illness.
- Fatigue and weakness
- Nausea and vomiting
- Headache
- Dizziness
- Muscle cramps and myalgia
- Irritability

HEAT STROKE

- This condition may be characterized by all of the symptoms of heat exhaustion.
- The critical feature of heat stroke is central nervous system dysfunction, which has a sudden onset in 80% of cases and may include bizarre behaviour, hallucinations or alterations of mental status .
- Sweating may be present . Anhidrosis, while a classic feature of heat stroke, may be absentover half the time, especially with exertional heat stroke, and is usually a late finding.

Physical examination :

Always ask for a core body temperature to be taken for patients who come to the Emergency Department with CNS Dysfunction and 'Fever'.

MANAGEMENT OF A POISONED PATIENT

GENERAL MEASURES CONSIDER

- Potency
- Quantity
- Duration of exposure
- Presence of other ingredients, i.e. co-ingestants
- Early identification of toxin
- Risk factors
- Exclude hypoglecemia/other metabolic defects
- ECG monitoring

FEATURES CLUSTERS FOR LIKELY POISONS

CLINICAL FEATURES	LIKELY POISONS
Coma, hypertonia, hyperreflexia, extensor plantar, Dry mouth, myoclonus, strabismus, mydriasis, sinus Tachycardia, urinary retention	Tricyclic antidepressants
Coma, hypotonia, hyporeflexia, non-excitable Plantar response,hypotension	Barbiturates, Benzodiazapines And alcohol combinations
Coma, miosis, reduced respiratory rate	Opiod analgesics
Nausea, vomiting, tinnitus, deafness, sweating Hyperventilation, tachycardia	Salicylates
Restlessness, agitation, mydriasis, anxiety tremor Tarchycardia, convulsions	Sympathomimetics
Parasympathetic activity like abdominal cramps,diarrhoea, Salivation, flaccid paralysis of limb, respiratory and ocular Muscles in various degrees, miosis, repiratory failure(typeII) 'kerosene'smell	Carbamates Organophosphates, Pesticides, herbicides

PRIMARY CARE

Like any critically ill patient with some exeptions: 'ABCDEFGH' AIRWAY:

Assess AIRWAY for obstruction; Insert oropharryngeal airway; Nurse in semi-

prone position

BREATHING: Assisted ventilation with oxygen, AVOID HYPOVENTILATION CIRCULATION:

Establish venous access; Avoid HYPOTENSION.

Decontamination: Remove from the site if gaseous poison, wash body surface or remove clothing if Organophosphorus poisoning.

Drugs:

Antidotes:

Multidose activated charcoal: Do not give if bowels sounds are absent/in corrosive poisoning. Not effective in metal poisonings. Multidose regimes 1-2 gm/kg q 4 hourly. Airway should be protected if patients is unconscious. 70% sorbitol as an osmotic cathartic.

Specific : Flumazenil, Atropine Naloxone.

ELECTROLYTES/ECG: Correct dys-electrolytemias; Na, K, Creatinine. ECG monitoring for earlyRecognition and treatment of fatal arrhythmias.

FLUID BALANCE: Catheterize, Intake-output chart, IV fluids, Central venous line if haemodynamicallyunstable. Keep urine output high (>100-150 ml/hour)

GAVAGE: Gastric lavage/stomach wash-

Indications :- any unknown poison ingested; duration of ingestion not known; even when period of ingestion is > 4hrs. still worthwhile in certain poisons where gastric emptying is delayed.

Caution:- in comatose patients with ET tube in place

Contraindication :- corrosives, volatile hydrocarbons should be followed by activated charcoal.

HAEMODIALYSIS : If indicated

Psychiatric assessment and support, once patient is stablilised.

MANAGEMENT OF SPECIFIC POISON

PRINCIPLES OF MANAGEMENT

- Maintain vital signs
- Eliminate remaining poison
- Combat side effects and complications

ORGANOSPHOSPHORUS:

PRIORITIES IN MANAGEMENT

- General Management as of any poisoning.
- If gag reflex present? If present give stomach wash. If absent, intubate and give stomachwash.
- Activated charcoal 50 gms stat and repeat if required.
- Start oxygen 6liters by mask.
- Inj. Atropine 1 mg IV increments, until bronchial and oropharyngeal secretions have ceased:maintainance therapy of continuous infusion of 0.05 mg/kg/hr. or IV 1 mg p r n dosages justto maintain HR above 100/min. Taper over 24-48 hours slowly with subcutaneous 1 mg doses q 6 hourly.
- Inj. Diazepam 10-20 mg IV only if the patient has seizures.
- Pralidoxime use in early cases (controversy exists)

INVESTIGATIONS

ROUTINE BLOOD TESTS (Hb, TC/DC, Na, K, Creat)

PLASMA PSEUDOCHOLINESTERASE <2000 (Normal= 3000-8000 ug/mL) A B Gases (to look for type IIRespiratory failure)

CHEST X-RAY to look for chemical aspiration pneumonitisECG to look for ventricular arrhythmias

COMPLICATIONS TO LOOK FOR AND MANAGE

Aspiration pneumonitis

Ventricular arrhythmias

Intermediate syndrome (develops from day 1-4 after after ingestion-needs ventilator support ifrespiratory failure sets in)

Atropine psychosis (Rx with IM Haloperidol 2.5-5.0 mg; reduce atropine dose)
Convulsions (Rx IV Diazepam)

CARBAMATES

- Like Organo-Phosphorus compounds, similar but short-lived effects due to spontaneous dissociation of carbamate-enzyme complex. There is no intermediate syndrome and CNS isless affected.
- Symptomatic cases require atropine; fairly quick recovery is a rule.

PYRETHROIDS

- Coma, Convulsion, Pulmonary Edema in severe cases.
- Only supportive therapy

AROMATIC AND HALOGENATED HYDROCARBONS (eg ENDOSULPHAN)

- Gastric Lavage (after taking necessary precaution against aspiration and chemical pneumonitis)
- Activated Charcoal
- Signs and symptoms are milder**, (pupillary constriction is however absent)**
- Treatment is mostly supportive, and involves mainly correction of dyselectrolnytemias,treating aspiration pneumonia)
- Treat as OP Poisoininge.gAtropinization, **if pupillary constriction is present** and OP poisoining cannot be ruled out. If certain about the nature of the consumed compound,**Atropine is relatively contraindicated** as it can worsen sympathetic overactivity.
- Seizures to be treated with Diazepam IV.

OLEANDER (WHITE, YELLOW)
(CERBERA THEVATIA)

CF:-Nausea, Vomiting, Abdominal Pain, Diarrhoea O/E **:-**
Hypotension, Bradyarrythmia, Syncope

MANAGEMENT

- ABCDE as for any other poisoining.
- Monitoring serum potassium (Hyperekalemia)
- IV fluids and dopamine for hypotension
- Atropine for bradyarrhythmia
- ECG monitoring
- Cardiac pacing for uncontrolled bradycardia

KEROSENE (PARAFFIN OIL) POISONING

Properties causing toxicity : Low surface tension, Low viscosity.

CF :- Pulmonary Toxicity within 1-8 hours of ingestion – due to the poison aspirated into respiratory tract.

It is possible to have symptoms without radiological features and vice versa. X- Ray abnormalities are maximum at 72 hours.

Management :- Avoid Emesis and Gastric lavage.

In severe cases, ventilation- Oxygen and PEEP can be used.

If amount of consumption is large, Gastric Lavage to be considered, within 1 hour, with cuffed ET- Tube intubation. Corticosteroids/ Antibiotics do not significantly alter the morbidity and mortality.

METHYL-ALCOHOL POISONING

- **CF :-** Coma after a latent period of 8-36h.

If dilated pupils not reacting to light is seen permanent blindness is likely to ensue. Features of hypokinensis, rigidity and extrapyramidal signs develop in cases of putaminal necrosis. Fundoscopyto look for optic neuritis.

- **Management :-**

1. Gastric lavage if presents within 1h, of ingestion.
2. Reversal of metabolic acidosis, Bicarbonate infusion in large doses.
3. Monitor for hypernatremia and fluid overload, both of which can be as a result of acidosis correction.
4. **Inhibition of methanol oxidation :-** Administer 50 gm ethanol, (125 ml of gin/whisky/ vodka)PO as loading dose, followed by 10-12g/h IV infusion, or 12-15g/h IV in known alcoholics or 17-22 g/h IV in patients on haemodialysis. Ethanol can be administered in the dialysate fluid (1-2d/l) if the patient is receiving peritoneal dialysis.
5. **Indications for haemodialysis :-** Consumption of > 30 gm of methanol. Blood methanolconcentration> 500 mg/l

Severe metabolic acidosis, pulmonary edema.Fundoscopic changes of optic neuritis. CNS

dysfunction (putaminal necrosis)

6. **Folinic** acid 30 mg IV q6hrly. (To protect against ocular toxicity)

CORROSIVE POISONING (INGESTION)

ALKALI and ACID POISONING

When suspected, **Diagnostic Endoscopy** (Beyond the first observable Oesophageal/Gastric lesion is mandatory, even if deep penetrating and circumferential burns burns are present) and **NG tube insertion** under direct visualization can be done to prevent rupture in the acute setting and for feeding later by living it in place for prolonged (3-4 weeks) periods. Should be done within 12-24 hours after ingestion of an alkali.

AVOID GASTRIC LAVAGE, NEUTRALIZATION; CAN ASPIRATE AND WORSEN STRICTURES.

- If FIRST DEGREE burns, (edema and erythema)….. can be discharged home if taking orally.
- If SECOND DEGREE burns (erythema, blistering, superficial ulcerations, fibrinous exudate)….. admit, nil, orally, total parenteral nutrition for initial 7 days. Oral fluids on day 7th. Restart solids as tolerated.
- If THIRD DEGREE burns (erythematous, deep ulceration, friability, eschar formation, perforation) should be managed in ICU setting. TPN/feeding jejunostomy required until GI lesions heal.

If evidence of perforation and peritonitis, or gastric necrosis at endoscopy, or persistently alkaline gastric pH , laparotomy and resection of necrosed tissue should be considered.

- Look for features of third degree burns (erythematous, deep ulceration, fibrinous exudate)….. admit, nil orally, total parenteral nutrition for intial 7 days. Oral fluids on day 7th.Restart solids as tolerated.
- If THIRD DEGREE burns (erythematous, deep ulceration, friability, eshar formation, perforation) should be managed in ICU setting. TPN/feeding jejunostomy required until GI lesions heal.

If evidence of perforation and peritonitis, or gastric necrosis at endoscopy, or persistently alkaline gastric pH, laparotomy and resection of necrosed tissue should be considered.

- Look for features of third degree burns on endoscopy, full thickness burns with necrotic slough/charred tissue; or GI perforation/peritonitis at initial presentation – **proceed with laparotomy.**
- Grade I and II a can be managed in ICU setting. TPN/feeding jejunostomy required until GI lesions heal.

If evidence of perforation and peritonitis, or gastric necrosis at endoscopy, or persistently alkaline gastric pH, laparotomy and resection of necrosed tissue should be considered.

- Look for features of third degree burns on endoscopy, full thickness burns with necrotic slough/charred tissue; or GI perforation/peritonitis at initial presentation- **proceed with laparotomy.**
- Grade I and Grade II a can be managed in wards; II and III needs ICU care, parenteral nutrition.
- Steroids are contraindicated because they mask the features of peritonitis.
- Antibiotics only if there is an evidence of infection.
- Correction of metabolic acidosis or alkalosis.

ACETAMINOPHEN POISONING

HISTORY:- of an acute overdose (>/= 140 mg/kg) of grug accidenta;/suicidal

PHARMACOKINETICS OF THE DRUG :- T1/2 life = 2-4 hrs; can be prolonged in cases ofhepatotoxicity. Rapidly absorbed from stomach and small bowel.

CLINICAL FEATURES :-
Early featured :- nonspecific of hepatoxicity; within 2-4 hrs. nausea, vomiting, diaphoresis and pallor.

CNS depression is unusual unless depression is unusual unless depressant drugs have been congested. Hepatotoxicity develops within 24-48 hrs. Renal failure may also supervene.

LOOK FOR FEATURES OF HEPATOTOXICITY :-

- PT is prolonged by twofold.
- S.BILIRUBIN is > 4mg/dL anytime 3rd to 5th after ingestion of drug drug.

IN PATIENTS WHO RECOVER, **LFT RETURNS TO NORMAL WITHIN 1 WEEK;** histological structure returns to normal; within 3 months.

If serum acetaminophen levels are not available, judge the chance of hepatotoxicity based on thefollowing approximation.

Ingested dose	**Hepatotoxicity**
250mg/kg	Probable
>140 mg/kg	Possible
>70 mg/kg	Possible in high risk cases.

High Risk – patients with Alcoholism, Malnutrition with HIV virus, or patients on therapy withBarbiturates, Phenytoin, and Rifampicin.

Antidote therapy :-

- **N-Acetyl cysteine** therapy provides protection against Acetaminophen induced hepatotoxicity because of its anti-oxidant action and the improvement it brings about in the microvascular perfusion of the liver in any kind of fulminant hepatitis, **only if it is initiated within 24 hours of poisoining.**

- There are some evidences showing that within24-36h also it can be helpful, but the consensus is that it is useful only if administered within 24h of overdose.

- **Protective effect** is greatest when the treatment is initiated within 8 hours of poisoning. Thereafter protective effect decreases steadily with time.

- Both Oral and IV therapy are equally effective.

- Preparations :- 10% NAC (100 mg/ml), 20% (200mg/ml).

- ORAL :-

Dilute 10% NAC (1:2) in water/coke/pepsi to make a 5% solution (50 mg/ml). Initial dose 140 mg/kg,maintenance 70mg/kg q4hly for 17 doses. A total of 1330 mg/kg over 72h.

IV :-

- Use 20% NAC, 150 mg/kg in 200ml of 5% Dextrose over 15mins. 50mg/kg in 500 ml of 5% Dextrose over 4h; followed by 100mg/kg in 1000ml of 5% Dextrose over 16h. Total dose : 300mg/kg over 20h.
- Expect and treat dose dependent diarrhoea that occurs 72h after NAC therapy. Self limiting, resolves in 90% of the cases.

TRICYCLIC ANTIDEPRESSANTS (TCAD)

- ABCDEFG as for all poisons

- CARDIAC MONITORING FOR ARRHYTHMIAS – Usually occurs within 6 hrs of overdose. (Do not treat if haemodynamically stable) IV Lignocaine 50-100 mg if ventricular tachyarrhythmia sets in. A **QRS prolongation** of >0.16s correlates best with subsequent seizures and ventricular arrhythmias. **Tachycardia is the most sensitive marker of TCAD overdose.**

- GASTRIC LAVAGE – (>250 mg ingested)AND ACTIVATED CHARCOAL.
- SEIZURE TREATMENT- Usually within 6 hrs. of ingestion.
- ALKALINIZATION OF BLOOD – to maintain arterial pH at 7.45-7.55. Monitor closely for hypokalemia. This reverses the membrane depressant effects of TCA.
- LOOK FOR URINARY RETENTION AND CATHETERIZE – Acute urinary retention can occur as a part of anti-cholinergic effect.
- In case of CO-TOXICITY WITH BARBITURATES……. Caution in using Flumazenil because it unmasks the seizure potential of TCAD.
- T1/2=24-30 hours; therefore monitoring should be done till serum levels fall below toxic levels by approximate calculations. DO NOT MEASURE TCAD LEVELS IN BLOOD because itis rapidly metabolized to active metabolites.

OTHER POISONS : START ABCDEFGH as indicated

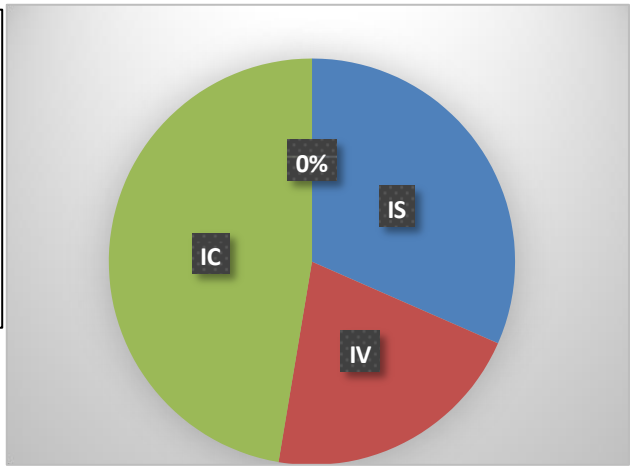
POISON	**SPECIFIC MEASURES**
• Phenothiazine	Benztropine 1-2 mg IV for cute dystonias
• Lithuim Carbonate	Forced diuresis/haemodialysis
Nephrogenic Dl-Indomethacin/thiazide	
• Anti convulsants	Diazepam for convulsions
• Opiates	Naloxone 0.4-2.4 mg IV/IM
• Salicylates	Alkaline diuresis
• Theophylline	Correct hypokalemia,
Charcoal Haemoperfusion	
• Barbiturates	Haemodialysis if sensorium is depressed
• Benzodiazepines	Flumazenil 0.5mg IV repeat doses till 1-3mg

FLUID AND ELECTROLYTE DISTURBANCES

1. BODY FLUIDS

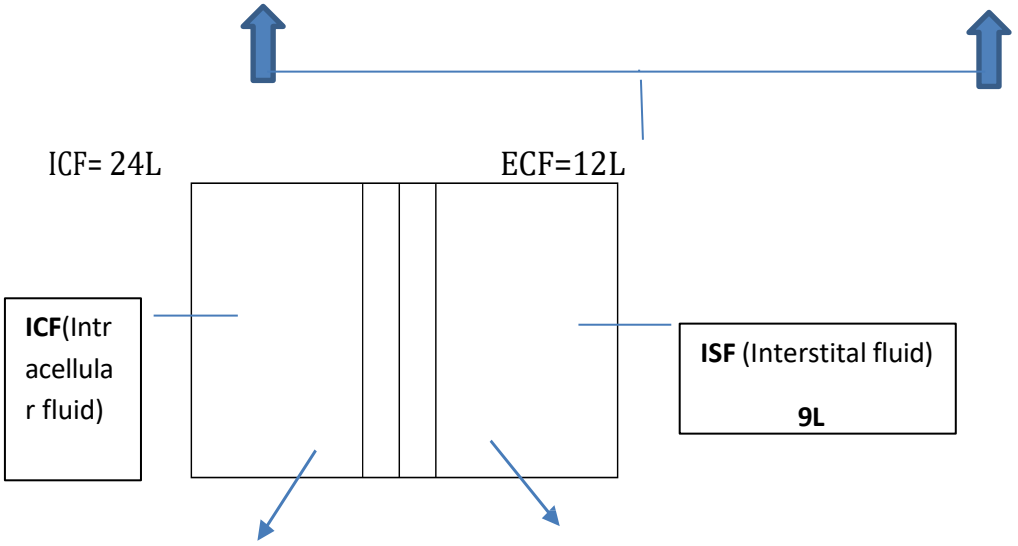
DISTRIBUTION OF BODY FLUIDS

60 % OF WEIGHT IS TOTAL BODY WATER 2/3RD IS INTRACELLUAR (IC)



1/3RD IS EXTRACELLULAR OF WHICH 1/4TH IS INTRAVASCULAR (IV), VASCULAR. 3/4TH IS INTERSITIAL (IS)

THEREFORE, IN A 60 Kg MAN TBW=36L (60X0.6)



RBC PLASMA

(2L) (3L)

RBC + PLASMA = IV (5L)

RBC fluid is part of ICF

- DAILY FLUID ORDERS :-

QUANTITY :Normal individual, NPO, maintenance order. 100 ml/kg body weight x first 10 kgs + 50 ml/kg x next 10 kgs + 20 ml/kg thereafter (More useful for children)
For adults, about 3 litres of IV fluids, equally divided between NS and 5% glucose) Daily requirements of

- Potassium= 60 meq (4.5 KCl)
- Magnesium = 8-20 meq (1-3 of MagSO4)
- Na=100 meq,
- Glucose (To reduce catabolism of proteins by half) = 100-150g (2 litres of 5% Glucose).
- Proteins 40g/day

Type of fluid : Usually a balance of NS & 5% Glucose.
For post op 1st day only dextrose…… 2nd day to 3rd day introduce DNS into fluid order. After 3rd day, usual orders.
Supplement potassium if <3days of NPO.

- **Volume excess…..**Look for increase in weight, pedal edema, ascites, raised JVP, lung basecrepts.
  … ……Causes CCF, nephrotic syndrome, cirrhosis, renal failure.
- **Volume depletion…..**Look for weight loss, excessive thirst, dry mucous membranes,tachycardia, orthostatic hypotension, shock.
  …….. Causes vomiting, diarrhea, diuretics, renal (salt losing) disease, DM, DI, excessiveinsensible loss, inadequate oral intake.
- **Evaluation…..**

Investigations :- Electrolytes, blood urea nitrogen, creatinine, glucose, urine spot sodium,potassium.

INTERPRETATION OF THE FOLLOWING :-

- Serum Osmolality = 2x(Na+ K (meq/L)) + Glucose, 18 + Blood Urea, 6 (MG/DL)

NORMAL = 285-305 mosm/kg

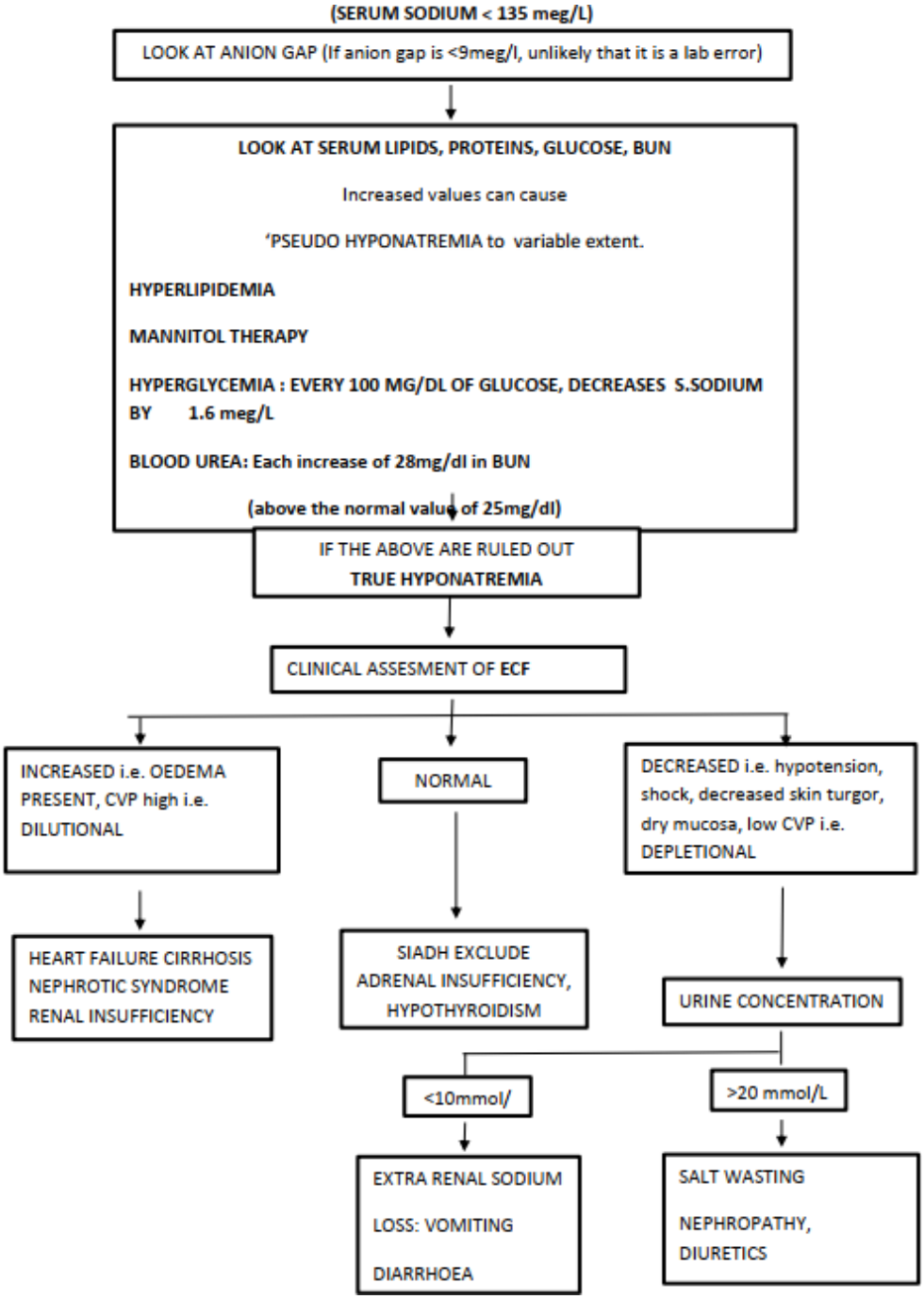
- Urine spot sodium > 20 meq/ l is consistent with renal salt wasting. (Diuretics, ACEI, hypoaldosteronism, salt losing nephropathy.) When less than 10 meq/L it means avidconversation of sodium to comepensate for extra renal losses.
- **Correction…..**
- Goal is to restore normovolemia
- Fluid should be as close in composition as the fluid lost.
- Monitor weight daily, look at cumulative balance, maintain intake-out put charts.
- In shock/or during large volume replacements, **central venous line is mandatory**
- Look for signs of overcorrections (JVP, basal crepts, anasarca)

- Mild volume contractions, replace by mouth.
- More severe requires IV therapy. **Individualize therapy ; most often it isthe 'blanket protocol therapy' that harms the patient.**
- Amount and type of fluid is determined by serum sodium values.
- With haemorrhage, anemia, might need volume replacement with blood or colloids.
- Quantity of fluid depends on
    - Deficit to start on
    - Maintenance needed
    - Ongoing loss

1. ELECTROLYTES

SODIUM

1. HYPONATREMIA
(SERUM SODIUM < 135 meg/L)



CORRECTION:- (17.5meq = 1gm. 1000 ML of NS has 9 gms of sodium 154mmol/lt)

- **REPLACEMENT = BODY WT. X 0.6 X (130- SERUM Na)** (Sodium is replaced for all the compartments, and therefore in the equation 60% of the body weight is considered thoughthe intravascular volume is only 5% i.e. all the compartments are in equilibrium due to osmotic drive and the major ion involved is sodium).
- The plasma sodium concentration should not rise by more than 10-12 mmol/l/24hr or >25mmol/l/first 48 hours.
- When it is a fall known to have occurred within 12-24 hours, it is safe to correct at a fasterrate than if hypernatremia is more chronic.

In the **Depletional** form……. Oral/IV Correction

- If mild, asymptomatic ….. Oral Salt
- If symptomatic / severe …… IV
- Fluid replacements to be corrected over 48-72 hrs.
- Fast sodium correction can cause seizures and permanent neurological abnormalities (**Central pontine myelinosis-** The neuronal response to an extracellular increase in osmolality (and therefore to prevent the outflow of intracellular water) is to break up its
- proteins into 'IDIOOSMOLES' (smaller particles over a period of time) which increase sodiumand fluid at this stage can cause cellular swelling due to influx of water into the cell owing toits higher osmolality – therefore causing cell damage. (a.k.a. osmotic demyelination syndrome ODS)
- Total deficit is calculated by using the above formula
- Half of this is given in 1st 8-12 hrs, followed by the rest in over next 12-24 hrs.

In the **dilutional** form restrict fluids, cautious use of diuretics

- In emergency (like seizures), use 3% saline for correction (same formula as above)
- Correct over a duration of 48-72 hrs.
- Change in serum sodium = (Na I – Na S) (Total Body Water + 1) NaI =

Sodium Conc. In Infusate

Na S is Serum Sodium Conc.

Na concentrations in NS = 154 meg/L Na conc. in RL = 130meg/L
Na conc. in 3% saline = 513meg/L

In the **SIADH** type….

1. Dilutional hyponatremia i.e. plasma hypo-osmolality proportional to the hyponatremia
2. Urine osmolality greater than plasma osmolality (usually)
3. Persistent high urine sodium (>50 mml/l)
4. Absence of hypotension, hypovolemia and edema forming states
5. Normal thyroid, renal and adrenal functions

- In emergency, use hypertonic saline as above
- Restrict fluids to 500-800 ml/day, increased salt intake
- Demeclocycline 600-1200 mg/day
- Lithium
-

Note : Hiccups is a common manifestation of hyponatremia. CF-

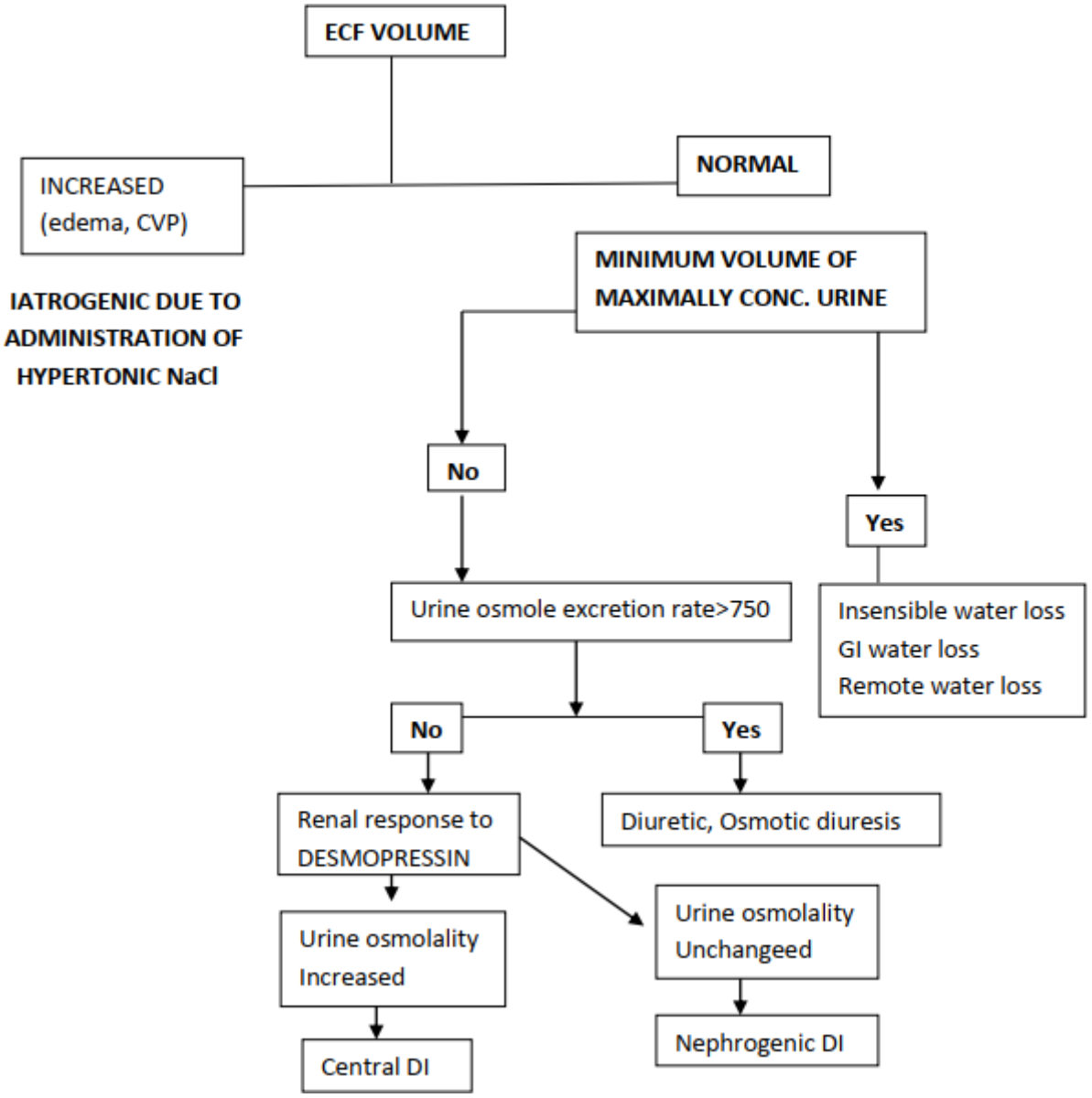
CNS mainly – alt sensorium

1 level tef = 5 gm Na = 68 M Eg of salt (Na Cl)of hyponatremia.

HYPERNATREMIA

(SERUM SODIUM>145meq/L)

Clinical approach to HYPERNATREMIAS



Investigations:- Ask for Na, K, Ca, Mg and Phosphorus

ALWAYS ASOCIATED WITH HYPEROSMOLALITY

CAUSE-IATROGENIC HYPERTONIC SALINE THERAPY, WATER DEFICITS

TREATMENT:-

- FLUID TO BE REPLACED IS CALCULATED BY-
- WATER DEFICIT=0.6 X WT X (OBSERVED Na- EXPECTED Na)/ EXPECTED Na
- TYPE OF FLUID ½ NS, ¼ NS, 5% DEXTROSE
- RATE OF LOWERING SODIUM SHOULD NOT BE > 2mmol/L/h

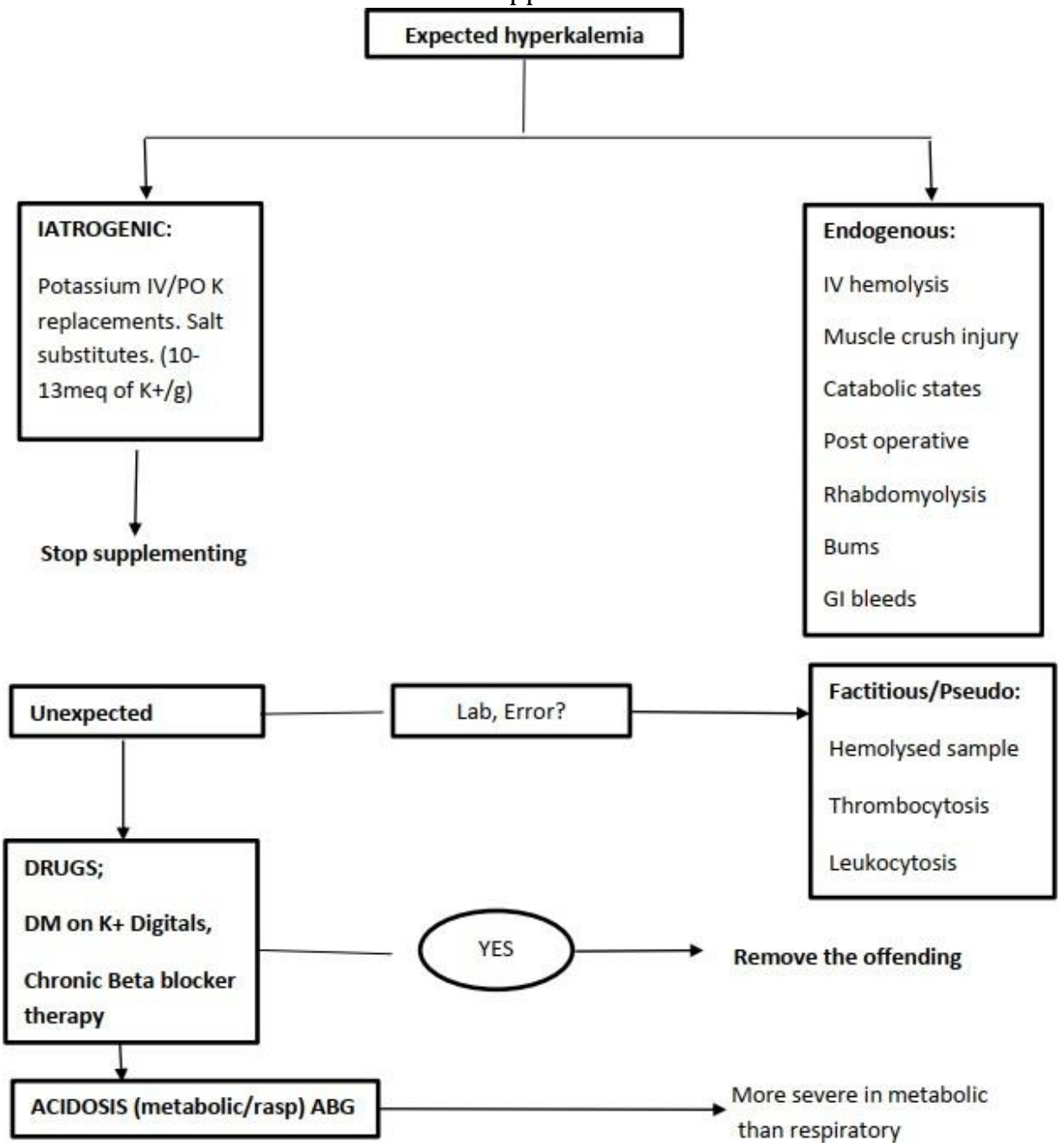
DISORDERS OF POTASSIUM BALANCE

- Predominantly intracellular ion (Only 2% is in ECF)
- Normal range 3.5-4.5 meq/L.
- Normal daily requirement = 60meq
- Normal K_1/K (potassium concentration intracellular/extracellular) ratio = 35-40 / 1
- 1g KCl = 13meq of K

HYPERKALEMIA

Defn: >5.0meq/L

An approach



CLINICAL FEATURES

Paresthesias, ascending muscle weakness, **Muscle weakness** is due to a rise in **the thresholdlevel** of stimulus.

ECG :- Changes in hyperkalemia.
In chronic cases, there maybe **no** ECG changes even at levels of 7.5meq/L.

Cardiac toxicity can therefore occur in them without any premonitory ECG changes, therebygoing into direct VF. **Thus treat all hyperkalemias > 6.0meq/L.**

ECG changes:	**Corresponding Se. K+ levels.**
Tenting 'T' waves, ST depression.	5.5-6.0meq/L
PR and QRS widening	6.0-7.0meq/L
'P' wave flattening	
Widening of QRS and PR	7.0-7.5meq/L
Atria conduction ceases, 'P', wave disappears	
QRS merges with 'T' wave, 'SINE' wave pattern.	>8.0meq/L
(Error of misdiagnosing VT)	
Ventricular asystole/fibrillation	>10meq/L

TREATMENT FOR ACUTE HYPERKALEMIA

(mostly by inter compartmental shifts)

EMERGENCY

Modality	Mech. Of action	Onset	Duration	Prescription
Calcium	Membrane stabilization (MS)	0-5mins	30-60min	10-20ml, 10% Ca Gluc/10-20ml, 5% Ca Chloride
Bicarbonate	MS + Redistribution	15-30mins	1-2h	Na Bicarb 100 meq IV
NaCl	MS	5-10min	2h	50-100 meq IV
Insulin	Redistribution	30min	4-6h	IV, 5-10U regular Insulin
Insulin-Glucose				50%, 25g (1amp) Gluc.
Terbutaline Salbutamol	Redistribution	15-30mins	2-4h	Nebulized 10-20mg in 4ml of NS, over 10mins.

- **Do not use** Calcium for Digitals induced cardiotoxicity with hyperkalemia

NON-EMERGENCY THERAPY

(mostly by getting rid of the potassium from the body)

Modality	Mech.Of action	Duration	Prescription
Loop diuretic	Renal Excretion	0.5-2h	Furosemide, 40-160mg, IV +/- NaBicarb
Kayexalate	Ion exchange	1-3h	PO 15-30g in 12.5% Sorbitol (50-100

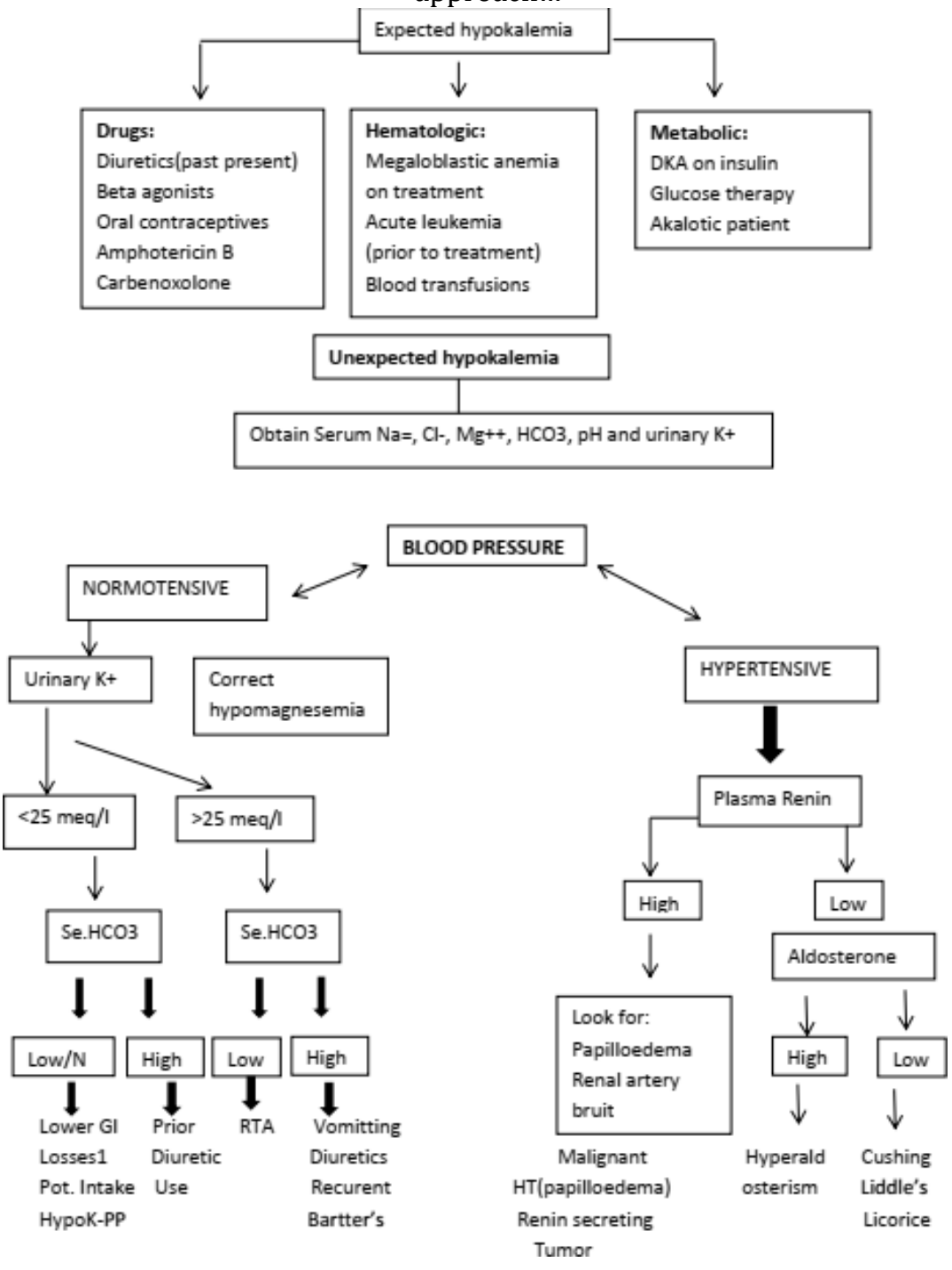
(Sodium polystyrene sulphonate)		ml). Rectal, 50g in 20% Sorbitol
Hemodialysis	48h	
Peritoneal dialysis	48h	Fast exchange, 3-4L/h

TREATMENT OF CHRONIC HYPERKALEMIA

- Low potassium diet (<60meq/d)
- High carbohydrate diet (if not diabetic)
- Avoid drugs causing hyperK+ e.g. ACEI, K+ sparing diuretics, Beta blockers,PG synthetase inhibitors, Heparin.
- Diuretic (Thiazides/ Furosemide in renal failure)
- Sodium bicarbonate orally to increase blood pH
- Exchange resins

HYPOKALEMIA

Defn:<36meq/L An approach…



Hypo kalemic PP (periodic paralysis) :- (Autosomal dominant)

Normotensive, Hypokalemia. NO kaliuresis.

RTA I and II :- Normotensive, Acidosis, hypokalemia, kaliuresis, hyperchloremic.

Bartters's :- Normotensive, Alkalosis, hypochloremic, hypokalemia, kaliuresis, Increased Renin and Aldosterone. Responsive to therapy with all potassium sparing diuretics.

Liddle's :- Hypertensive, decreased Renin and Aldosterone, Responsive only to Amiloride andTriamterene. Non-responsive to Spironolactone.

Potassium depletion without Hypokalemia :- Digitals
therapy
Untreated essential HT
Non-oliguric CRF

CLINICAL FEATURES

- No symptoms at >3.0 meq/L
- Muscle weakness :- Explained by the change in the Ke/Ki ratio due to intracellular potassium depietion, thereby making the resting membrane potential more negative.

This is in contrast to the mechanism of muscle weekness in Hyperkalemia.

Investigating a hypokalemia

- All other electrolytes as mentioned in the flowchart.
- ECG changes :-
    1. Flattening of 'T' waves
    2. 'ST' depression
    3. Emergence of 'U' waves
    4. 'PR' prolongation at <2.0.0meg/L
    5. Various arrhythmias :- APCs, VPCs, SVTs, high degree AV blocks,VT,VF.
- Chronic hypokalemia produces metabolic alkalosis.

TREATMENT OF HYPOKALEMIA

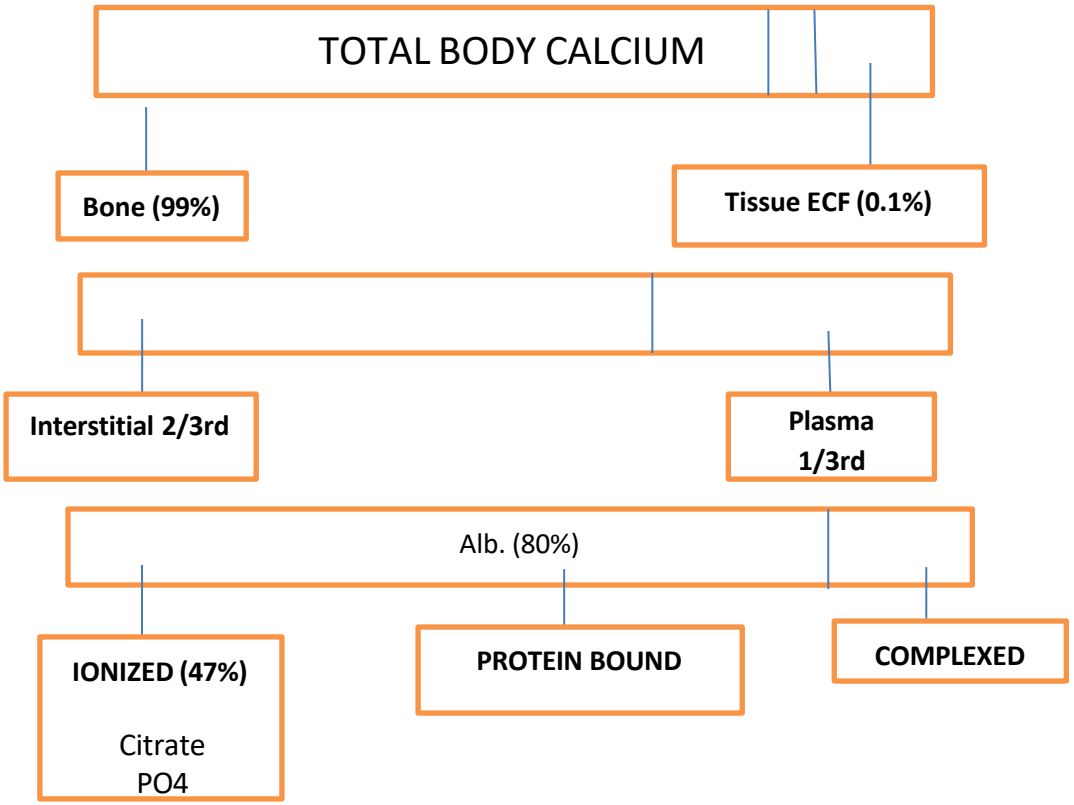
There is very poor correlation between total body potassium and the serum potassium. The following graph depicts the derived correlation between the two in a 70kg man.

- Total body potassium (3470)= intracellular potassium (3400 meq)+ extracellular potassium(70 meq).
- ACIDOSIS and ALKALOSIS shifts the curve in the above mentioned directions.
- Between 2.0 meq/L of serum concentrations, the potassium deficits is much more for asmall drop in serum concentrations.
- Above 4.5 an increase in serum potassium of 1 meq/L is associated with an increase of 100meq of total body potassium excess.
- In other words the potassium depletion has to be thrice as large than the potassium excess to give rise to a significant (1meq/L) change in the serum concentration of potassium.

- This difference is due to a large pool of intracellular potassium, which can replenish extraceilular stores when potassium is lost. The Ki/Ke(which is fairly constant between 2.3and 4.5, i.e. 35-40:1) ratio decreases whenever there is an excess of potassium. This ration of distribution also is low when potassium is given as a correction for hypokalemia. A decrease in ki/ke ratio increases the chance of cardiac arrest in asystole.
- Therefore, rates of replacement should never exceed 40 meq/h/l. A rate of replacement of 0.75 meq/kg over 1-2 hours increases the S. potassium by 1.0 to 1.5meq/L slowlydistributing at a ratio of Ki/Ke of 12-15:1.
- In case of REFRACTORY hypokalemia, correct magnesium (Intravenous magnesium SO4), change the infusing fluid to mannitol from dextrose/saline (to reduce the intracellular transport of the ion when given with dextrose and to prevent kaliuresis which proportionallyincreases with the distal tubular sodium load).
- THE BEST GUIDE TO REPLACEMENT THERAPY IS SERIAL MONITORING OF THE SERUMPOTASSIUM.

Preparations :- Oral syrup KCI (5ml=4meq of potassium)
InjectionKCI
For potassium chloride, 13meq=1g

DISORDERS OF CALCIUM



- LABORATORY VALUES ARE THAT OF *TOTAL SERUM CALCIUM* (IONIZED+COMPLEXED+ALBUMIN (80%)/ GLOBULIN (20%) BOUND) N=8.8-10.2 mg%
- % age of protein-bound Calcium= 80 x albumin (g/Dl) + 20X globulin (g/Dl) + 3
- Conversion for calcium values in **mg% = 2 x meq/L = 4x mmol/L**
- Out of these, only the **ionized component** is necessary for the **neuromuscular events,** the component which is acted upon influences the Parathormone activity and it is a deficiency/excess of this component solely which is **responsible for theclinical manifestations observed.**
- SERUM CALCIUM TO BE GIVEN AS A FASTING SAMPLE (Because of the falsely highlevels, postprandially due to prompt intestinal absorption.)
- SAMPLE SHOULD NOT BE COLLECTED WITH A TOURNIQUET IN PLACE. (Falsely highCalcium due to hypoxia-muscle cells extruding calcium from its sarcoplasmic reticulum- T tubule system into the ECF.)
- CHANGES IN Ph, (bicarbonate and citrates), being an acute event, change the **ionized component** of calcium **without changing the total serum calcium**, e.g.

In alkalosis, more albumin is ionized, more of calcium binds to it, and therefore, the ionized fraction falls resulting in a hypocalcemic manifestation in the presence of normaltotal serum calcium. The reverse takes place in acidosis.

In the physiological range, **ionized calcium decreases** by 0.5mmol/L for each 0.1 unit increases in **pH (i.e alkalosis)**

- CHANGES IN SERUM PROTEIN (mainly albumin) , being gradual event, changes the total serum calcium without changing the **ionized component, e.g**

In hypoalbuminemia, as the ionized fraction of the total calcium tends to rise due to less available albumin to bind to calcium, it is subject to counter regulatory influenceof the Parathormone, which brings it down. In effect, the measured total serum calcium falls through the ionized calcium is constant (which is our field of concern). Therefore we have to correct for hypo/hyper proteinemic states.

Formula to be used :-

Each fall/rise in serum albumin level by 1.0 g/dl (beyond the normal range of 4-5g/dl) is associated with a fall or rise of serum calcium concentration of approximately 0.8 mg/dl.

ACTUAL CALCIUM = MEASURED CALCIUM + (4 – SERUM ALBUMIN) x F where F= 0.8
for mg% 0.4 for meq/L, 0.2 for mmol/L

HYPOCALCEMIA

Definition :- Serum calcium (in the presence of normal proteins) < 8.8 mg% (2.2 mmol/L, 4.4 meq/L). This can be caused by either reduced ionized or protein bound calcium.

Causes :- with (lab. Interpretation of Calcium, Phosphates and Alkaline phosphatase)

- Hypoparathyroidism, pseudohypoparathyroidism, (L Ca, H p, N alkphos)
- Vitamin D deficiency/malabsorption. (L Ca, L p, N Alkphos)
- In acute care settings, excessive calcium sequestration (burns, toxic shock, septicemia, pancreatitis)
- Renal osteodystrophy (secondary hyperparathyroidism) (L Ca, H p, H Alkphos)
- **Reduced ionized calcium with normal total serum calcium** (citrate toxicity in massive transfusion, and respiratory alkalosis with PaCO2 < 21 mm of Hg, Hypomahnesemia.)
- Hypomagnesaemia
  (L= low, H= high, N=normal, Ca= calcium, p= phosphates.)

Clinical features :-

Tetany of muscles of the extremely and the larynx
TROSSEAUS sign :- inflate BP cuff above systolic and maintain at that for 3minutes- to look for carpo-pedal spasm.
CHVOSTEK'S contraction of ipsilateral facial muscle by tapping over the facialnerve at the angle of the jaw.
Physiology of all these tests depend on the fact that neuro-muscular tissue is irritable in the presence of hypocalcemia and using a BP cuff to occlude bloodsupply and make the muscles ischemic (TROUSSEAU'S) or to tap on the facial nerve (CHVOSTEK'S) are further insults or precipitating factors to elicit the tetany over a localized group of muscles.

Other features include circumoral paresthesiae, or fingertip tingling/burning, cramps, mental changes
(hallucination/confusion) areflexia, seizures and hypotension.

Chronic hypocalcemia can present with cataracts, papilloedema, basal ganglia calcification, alopecia, coarse dry skin.

Treatment :- for acute hypocalcemia

- IV calcium in symptomatic patients.
- CALCIUM CHLORIDE has 3 x the CALCIUM CONTENT in mmol. Than CALCIUM GLUCONATE for equal CONCENTRATIONS and VOLUMES.
- 10 ml of 10% Calcium gluconate IV over 3-5 mins.

- In severe cases, IV infusion of calcium gluconate 10%, 100 ml in 1000ml of 5% dextrose over 3-4 h. May need repetition of such supplements.
- In post parathyroidectomy hypocalcemia IV infusion of calcium gluconate 10%, 50 ml in 500 ml of 5% dextrose over Q12 hrly.

For chronic hypocalcemia

- Oral calcium supplements :- Tab.Sandocal (Calcium carbonate) 1 OD-TID.

HYPERCALCEMIA

Definition :- Total serum calcium > 10.2mg (2.55mmol/L, 5.1 meq/L) or increased ionized Calcium.

Causes :-

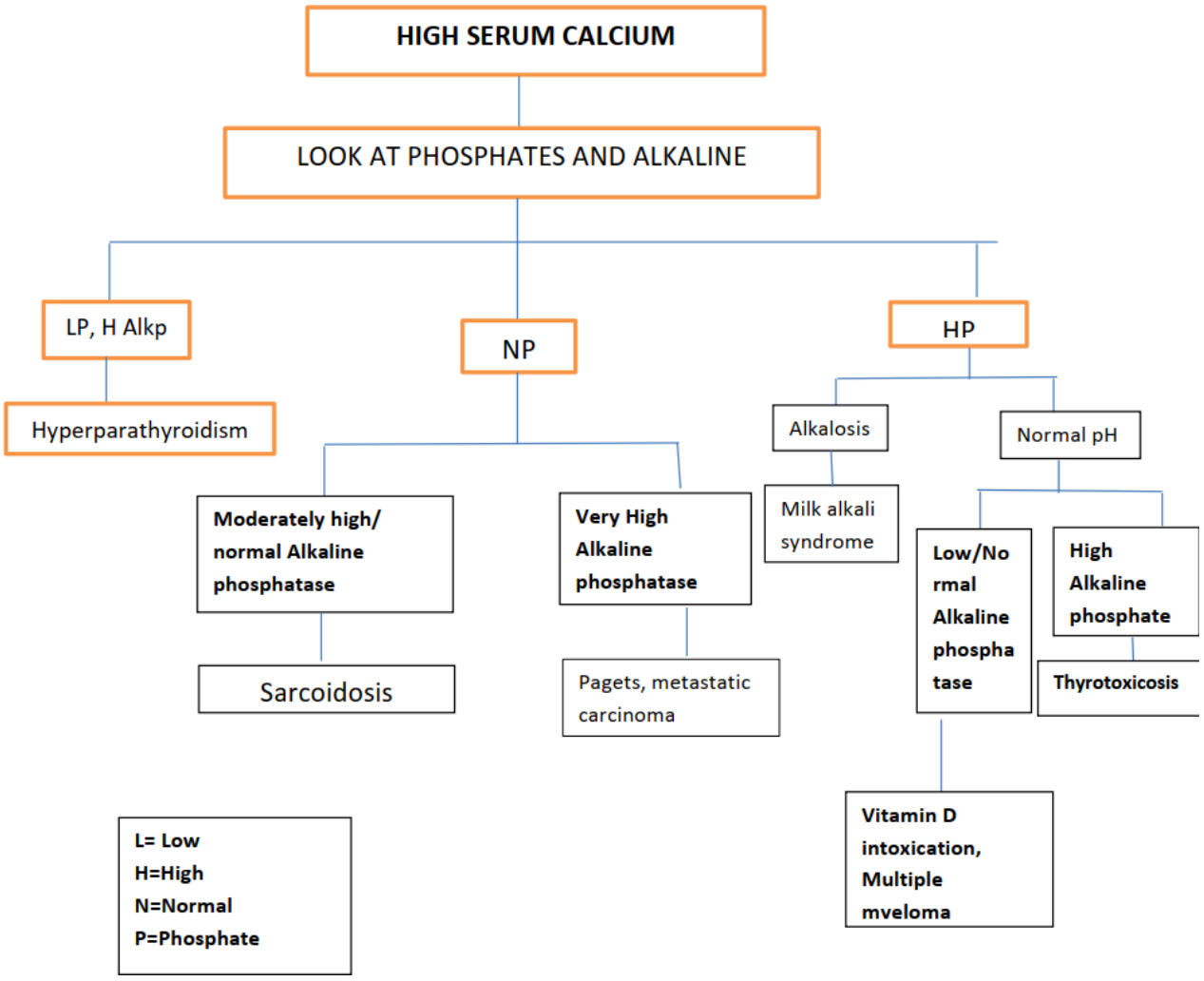
- A and D Vitamin toxicity, Alkali-milk syndrome.
- B one disease (Pagent's disese)
- Cancers (Lymphoma/lithium)
- Drugs (thiazides, lithium)
- Endocrine (thyrotoxicosis, phaeochromocytoma, Addison's disease, Acromegaly, VIPomas)
- Factitious (Haemoconcentration, postprandial)
- Granulomas (Tb, sarcoid)
- Hyperparathyroidism (primary, tertiary)

(Secondary hyperparathyroidism has a low serum calcium, i.e in Renalosteodystropy. A persisting secondary hyperparathyroidism can go on to tertiary hyperparathyroidism due to chronic stimulus resulting in autonomy.)

Clinical Features : usually with serum calcium >3.0mmol/L (12mg%, 6meq/L)

- GIT-anorexia, nausea, constipation, vomiting, pancreatitis, peptic ulcers
- URINARY- polyuria, polydipsia (Nephrogenic DI), nephrocalcinosis,
- CNS- depression, psychosis, apathy, somnolence, confusion, coma
- MUSCULOSKELETAL- arthralgia, myalgia, hypotonia, weakness.
  Popularly known as – '**BONES** (painful), **GROANS** (abdominal), **MOANS** (psychic)'
  Investigations :- Ca, Mg, HCO3, albumin, Phosphate, urinary hydroxyproline, Alkaline phosphatase, screening for malignancy with necessary lab investigations and imaging techniques.

Interpretation of lab. Results :-



Summarizing, single diagnosis to **be considered based on various combinations of the lab. Tests :-**

High Calcium-Low Phosphate-High Alkaline phosphatase….. Hyperparathyroidism

High Calcium- normal phosphates- High Alkaline phosphatase… metatastic carcinoma

High (mild) calcium-normal phosphates- Very Alkaline phosphatase… Pagets,

High calcium-normal phosphates- moderately high/normal Alkaline phosphatase…. Sarcoidosis.

High Calcium-High Phosphates-high bicarbonate… Milk-alkali syndrome,

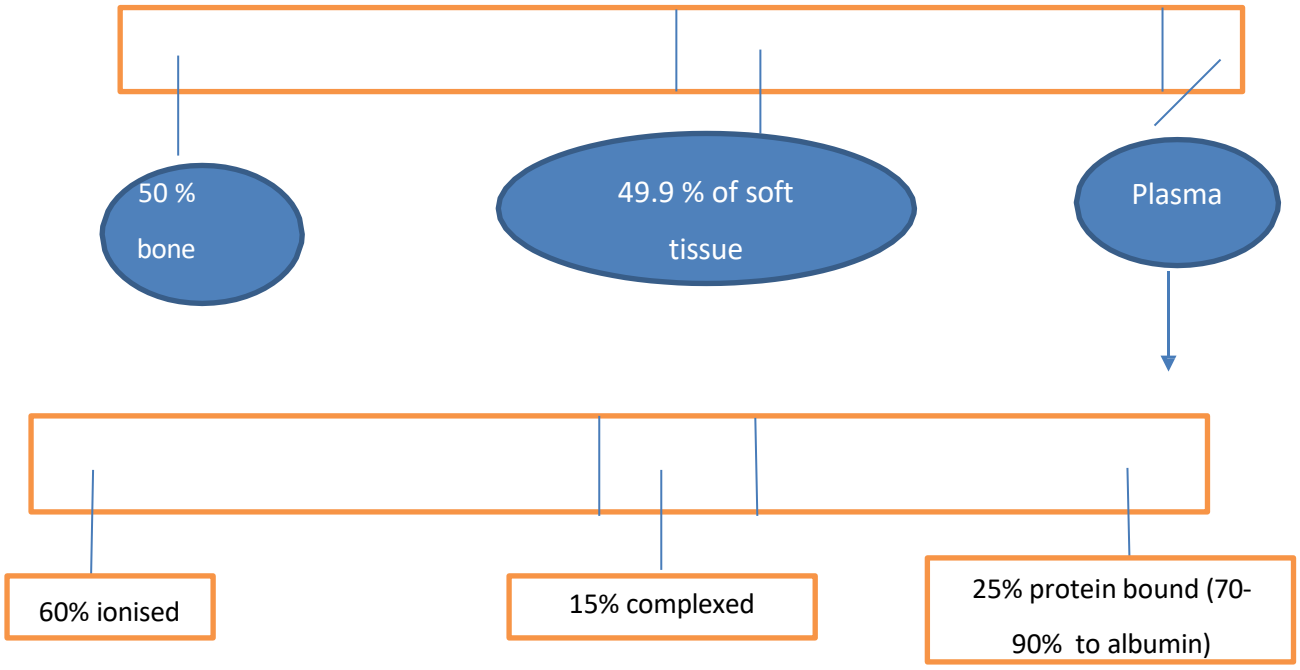
High Calcium :- High Phosphates- Low/Normal Alkaline phosphatase…. Vitamin D intoxication,Multiple myeloma

High calcium- High Phosphates- **High Alkaline phosphatase…**thyrotoxicosis.

Management :-

- **Correct dehydration** (which occurs due to NDI in hypercalcemia). A central line is preferred;monitor intake-output. Initially 2L of fluid is loaded (1L of 0.9%Nacl and 1L of 5% dextrose) followed by IV **Frusemide** 80 mg 2-4hly. The urine output is replaced by alternating 0.9% NS and 5%D upto 6L/day. Significant fall in calcium occurs in the first 4h. **DO NOT USE DIURESE WITHOUT ENSURING ADEQUATE VASCULAR FILLING.**
  Monitor Potassium, Calcium, Magnesium, Phosphate.
- **Corticosteroids** can be used at a dosage of 1-2mg/kg/day (PREDNISOLONE); effective only in sarcoidosis, melanoma, lymphoma, leukemia, Vitamin D intoxication (not useful in solid tumors). Desirable effect has a latency of 2-3 weeks to appear. Mechanism of action is by decreasing bone resorption and intestinal absorption of Vitamin D. IV 100-500mg of Hydrocortisone can be used for resistant hypercalcemias.
- **Mithramycin** 25 mcg/kg over 4-8h. as a SD. If hypercalcemia persists for24-48h after that, repeat the dose daily for 2-4 days. If necessary thereafter wkly.
- **Calcitonin** 100-200 U IV/IM, reduces calcium within 2-4h; short lived action, can be prolonged by addition of corticosteroids. Subcutaneous 4 U/kg q 12hly can be given.
- **Bisphosphonates** induce a slower but more sustained and complete response. IVPamidronate (upto 90mg), Etidronate (500mg) can be given.
- **Phosphate infusions no longer recommended because of vessel wall and metastaticcalcification.**

Disorders of Magnesium



- **Magnesium** is a predominantly intracellular ion.
- **Normal** range = 1.7-2.0 mg%
- **Complexed part** is with citrate, phosphates, bicarbonate, oxalates
- **Magnesium, Calcium and H+ ions** compete for the same site on the Albumin for binding.
- **Magnesium absorption** from the gut is load dependent, i.e irrespective of whether it isneeded or not therefore no protection against hyperMg++ as seen in antacid therapy.
- **Urinary loss of magnesium is** dependent on the intake (N=100mg/day; range 25-250).Fractional excretion of magnesium is halfway between Na and K. Sodium and Cl = 1%, potassium=10 , Magnesium =4%.

10 MALARIA
ANTIMALARIAL DRUGS

DL = Drug interaction
Cl = Contraindications
ADR = Adverse Drug reactions

Chloroquine

10 mg base/kg PO (max 600 mg), then 5 mg base/kg PO (max 300 mg) at 6h, 24h, and 48h (total 25mg/kg)

Same dose in children, as in adults.

ADR- Hypotension which given IV, impairs intradermal rabies vaccine, exacerbates psoriasis, causesretinopathy with 100 mg cumulative dose.

Dl- **Penicillamine and chloroquine** should not be used concurrently

Quinine sulfate
10 mg salt/kg PO q8h for 3-7d Same

dose in children, as in adults.

ADR- Allergy, hypoglycemia, myasthenia gravis, thrombocytopenic purpura, Blackwater fever, G6PDdeficiency, or history of cardiac dysrhythmias **(Stop if QTC is > 0.6/QRS is increased by >25% of the baseline).**ECG monitoring is therefore required.

Dl- Delays absorption of **digoxin** which can lead to increased serum concentration, antagonizeseffects of **antimyasthenics** and mefloquine increases risk of seizures.

Quinine dihydrochloride (for severe or complicated malaria)

20 mg salt/kg IV load 5% dextrose over 4h; then 10 mg salt/kg IV q8h until PO therapy possible (max,1800 mg/d); (EKG monitor in intensive care unit)

Same dose in children, as in adults.

ADR- Allergy, hypoglycemia, myasthenia gravis, thrombocytopenic purpura, Blackwater fever, G6PDdeficiency or history of cardiac dysarrhythmias (above are given the ECG indications to stop therapy).

Dl- Delays absorption of **digoxin** which can lead to increased serum concentration, antagonizeseffects of antimyasthenics and mefloquine increases risk of seizures.

Clindamycin
10 mg/kg PO (max 900 mg) q6h for 3-5d

Same dose in children, as in adults.

Cl- Risk-benefit should be considered with hypersensitivity of patient to lincomycins or doxorubicin, patients with history of severe gastrointestinal disease or severe liver dysfunction.

ADR- May cause pseudomembranous colitis and vomiting.

Primaquine(for prevention of relapse with P.Vivax and P.ovale)

Adults- 0.25 mg base/kg PO qd for 14d (usual dose 15 mg base [26.3 mg salt] PO qd for 14d) or 0.8mg base/kg PO once/wk for 6-8 wk (usual dose 45 mg base[79 mg salt] PO once/wk for 6-8 wk)

Chilldren – 0.25 mg base/kg PO qd for 14d or 0.8 mg base/kg PO once/wk 6-8 wk.

ADR- Hemolysis in G6PD deficiency, methemoglobinemia, rare agranulocytosis, and vomiting.

Dl- Quinacrine(may increase toxic effects of primaquine).

Pyrimethamine-sulpadoxine (Fansidar)

Pyrimethamine 1 mg/kg- sulfadoxine 20 mg/kg PO once (1 tablet=25 mg pyrimethamine -500 mgsulfadoxine; usual dose 3 tablets once on last day of quinine).

Cl- Allergy to sulfonamides, pyrimethamine, furosemide, thiazide diuretics, sulfonylureas, or carbonic anhydrase inhibitors preclude use of drug combination. Should not be used in patients withanemia, bone marrow depression, hepatic or renal dysfunction, porphyria or history of seizure disorders. May cause exfoliative dermatitis, Stevens-Johnson syndrome, hepatitis or pancytopenia.

Dl- In combination with **bone marrow depressants**, may cause increased leukopenia and/or thrombocytopenic effects. With **folateantagonists** combination can cause development of megaloblastic anemia.

Tetracycline
4 mg/kg PO (max 250 mg) q6h for 7d
Greater than 8y:kg PO (max 250 mg) q6h for 7d
Contraindicated for children 8y or under.

Unsafe in pregnancy.
Women who are breast-feeding should not use tetracycline.
Dl- Antacids, calcium supplements, salicylates, iron supplements,
Magnesium-containing laxatives, cholestyramine, colestipol or oral estrogen-containing contraceptives.

Doxycycline
100 mg PO bid 7d
Greater than 8 y of age: 2 mg/kg PO bid 7d
Cl- Pregnancy, children 8 years of age or younger Dl-
Barbiturates, carbamazepine, and phenytoin.

Mefloquine

15 mg base/kg PO, then 10 mg base/kg PO 6-8h later (max total dose 1250 mg: usually 750 mg PO,then 500 mg PO at 6-8h)

Safety and effectiveness have not been established in children.

Cl- Patients with seizure disorders, heart block, or psychiatric disorders. Toxic encephalopathy (in0.5-1.0% of Europeans and Africans and in 1% of Asians), seizures, prolonged Qtc syndrome, dysrhythmias, vomiting, dizziness, dysphoria, dissociation, ataxia and impairs fine spatial coordination.

Dl- Avoid use with **halofantrine**, addictive cardiotoxicity with **quinine,quinidine** and antiarrhythmics.

D- Unsafe in pregnancy.

Halofantrine

8 mg/kg PO (max 500 mg) q6h for 3 doses; repeat course in 1 week for the non-immune. Same

dose in children, as in adults.

Cl- Known congenital or family history of QT prolongation (risk of arrhythmia). Risk-benefit considered with AV conduction disorders, syncope, thiamine deficiency, ventricular dysrhythmias,cerebral malaria or other malarial complications.

Dl- With **Mefloquine**(increased risk of cardiac effects), **quinine,quinidine, anti- arrhythmics, tricyclicantidepressants, neuroleptics, terfenadine and astemizole.**

Artesunate

4 mg/kg PO qd for 3d (total dose 12 mg/kg) (1 tablet=50mg). For severe or complicated malaria usethe following dosages: 2.4 mg/kg IV load, then 1.2 mg/kg IV at 12h and 24h, then 1.2 mg/kg IV q24huntil PO therapy possible.

Same dose in children, as in adults.

ADR- Brainstem neurotoxicity and death in non-human primates, drug fever C-

Safety for use during pregnancy, has not been established.

Artemether(for severe or complicated malaria only) 1st

day-80 mg IM BD, 2nd, 3rd, 4th, 5th days 80 mg OD

TOTAL DOSE= 480 mg; CAPSULES 1st day 4 caps (160 mg) in 2 doses Q 12

hrly, thereafter 2 (80 mg) caps daily. TOTAL DOSE= 480Mg

3.2 mg/kg IM (anterior thigh), then 1.6 mg/kg IM q24h until PO therapy possible (never IV)

ADR- Brainstem neurotoxicity and death in non-human primates, drug fever.

Further Inpatient Care:

- The patient should have thick and thin **blood smears performedevery 12h until parasitemia falls below 1%** to ensure that the therapy instituted is clearing the infection. If paristemia does not fall by **75%within 48h** or if the blood is not cleared of parasites **after 7d,** a differenttherapeutic regimen should be initiated immediately.

Chemoprophylaxis is available in many different forms. The drug of choice is determined by the destination of the traveler and any medical conditions the traveler may have that contraindicatethe use of a specific drug.

Complications:

- Most complications are caused by P.Falciparum.
- **Coma (cerebral malaria) –** Defined as coma, altered mental status or multiple seizures withP.Falciparum in the blood. This complication is the most common cause of death in malariapatients. If untreated, cerebral malaria is lethal. Even with treatment, 15% of children and 20% of adults who develop cerebral malaria die. The symptoms of cerebral malaria die. Thesymptoms of cerebral malaria are similar to those of toxic encephalopathy.

MANAGEMENT:

- Meticulous nursing care
- Initiate IV chemotherapy
- Catheterize
- Input/output chart, rehydrate cautiously, correct electrolytes
- Q6 hrly blood glucose levels 10% dextrose 4-8 hr infusions.
- Monitor GCS, TPR, BP, maintain temp <38.5C
- Mannitol/glycerol to decrease oedema
- >15% parasitic index – exchange transfusion
- Treat convulsions with diazepam or paraldehyde

AVOID : Steroids, NSAIDS, Low mol. Wt dextran, Adrenaline, Heparin, Prostacyclin, Pentoxyphylline,Hyperbaric oxygen, Cyclosporin A.

- **Seizures**
- **Renal failure**- Up to 30% of nonimmune adults infected with P.falciparumsuffer acute renal failure. Consider peritoneal/hemodialysis if patient remains oliguric/anuric after adequate rehydration. Give isotonic saline till CVP is at 15cm of H2O
- **Hemoglobinuria** (blackwater fever)-Blaclkwater fever is the passage of dark, colored urine. This condition is caused by hemolysis, hemoglobinemia and the subsequent hemoglobinuriaand hemozoinuria.
- **Noncardiogenic pulmonary edema-** This affliction is most common in pregnant women andresults in death in 80% of cases.

Management :

- Keep patient upright, raise head end.
- High conc. Of oxygen, may even consider mechanical ventilation.
- Frusemide, 40 mg IV, can increase upto 200 mg, if no response.
- ICU care, O2 PEEP, ventilation, haemodynamic support.
- If due to overhydration stop all IV fluids, use Frusemide, withdraw 250 ml of blood byvenesection.

- **Profound hypoglycemia-** Hypoglycemia often occurs in young children and pregnant womenand is often difficult to diagnose since adrenergic signs are not always present and since stupor may already there.

- **Patients at risk for the above-** serious diseases, children, pregnant women and those onQuinine/Quinidine therapy.

Management : 50 ml 50% glucose at clinical suspicion followed by IV infusion of 5-10% dextroseclose monitoring of glucose

- **Lactic Acidosis-** This occurs when the microvasculature becomes clogged with P.falciparum.If venous lactate level reaches 45 mg/Dl, a poor prognosis is very likely.
- **Hemolysis** resulting in severe anemia and jaundice. If PCV<20% consider transfusing freshWB/packed cells.
- **Bleeding** (coagulopathy)

Prognosis:

- Most uncomplicated malaria patients show marked patients show marked improvementwithin 48h after the initiation of treatment and are fever-free after 96h. For pregnant women and children, see Special Concerns section below.

Special Concerns (WOMEN and CHILDREN)

- **PREGNANT women,** especially primagravid women, are up to **10 times**more likely to contract malaria than non-gravid women. Gravid women who contract malaria also have greater tendency to develop severe malaria. Unlike malarial infection in non-gravid individuals , pregnant women with P.vivax are at high risk for severe malaria and those and those with P.flaciparum have a greatly increased predisposition for severe malaria as well. Ifa pregnant woman becomes infected, she should know that many of the antimalarial and antiprotozoal drugs used to treat malaria are safe for use during pregnancy for both the mother and the fetus. Therefore, they should be used since the benefits of these drugs out- weigh the risks associated with leaving the infection untreated.

- **In children,** malaria has a **shorter course**, often **progressing rapidly** to severe malaria. Children are more likely to present with hypoglycemia, seizures, severe anemia and sudden death but much less frequently develop renal failure, pulmonary edema or jaundice. Cerebral malaria leaves between 9-26% of children with neurologic sequelae but of these about half resolve completely with time. Most antimalarials are very effective and safe in children, provided that the

proper dosage is administered and it is common for children to**recover from even severe malaria much faster than adults.**

INDICATIONS FOR PARENTERAL THERAPY

- Failure to retain drugs due to vomiting
- Cerebral Malaria
- Multiple complications
- Peripheral Asexual Parasitemia>/=5%

STEPS IN MANAGEMENT

- Hospitalize
- Parasitic Index-need for exchange transfusion
- Recheck daily

CONSIDER EXCHANGE TRANSFYUSION IF PARASITIC INDEX > 10%

RESISTANCE OF PARASITE TO DRUG ---------- WHEN TO SUSPECT/DIAGNOSE?

DRUG USED

Qunine Dl HCl 10 mg/kg (max. 600 mg) diluted in 300 ml dextrose over 1-2 hrs. Followed by

10 mg/kg/8 hrly

Switch over to oral quinine S04, 10 mg/kg TID x 3-7 days

CHLOROQUINE SENSITIVE P.FALCIPARUM

Rx:- Tab CQ. 150 mg base (250 mg each) 4 tabs stat followed by 2 of same 6 hours later followed by4 tabs (1 bdx2 days)

CHLOROQUINE RESISTANT

Quinine SO4 + Doxy 100 mg bd x 7d/ Clinda 900 mg TIDx 3 D/Metakelfin (Pyritmethamine 25 MG+ Sulfadiazine 500 MG) 4 TIDx5 D/Tetracycline 250-500 mg 4 times a day x 7D/ 3 tabs of Fansidar stat **OR**Mefloquine**OR**Halofantrine**OR**Artemether**OR**Artesunate

Px FOR P.VIVAX CQ+ Primaq.

ELIMINATION OF GAMETOCYTES

For P.Malariae, ovale, vivax -- Chloroquine
For P. Falciparum -- Primaquine

CHEMOPROPHYLAXIS

If chloroquine sensitive area :

- Chloroquine 500 mg/week --- Starting 1 wk before exposure and continuining 4 wks after

If Chloroquine resistant area :

- Mefloquin 25 mg/wk
  OR
- Doxy 100 mg/day Starting 2 days before, Cont. 4 weeks after Primaquine on returning home if significant exposure.
-

Pregnancy ---------- Chloroquine only.

11 TYPHOID

ESTABLISH DIAGNOSIS:-

INVESTIGATIONS:-

As for acute undifferentiated fever 80%
+ve Blood culture in 1st week

BM Cultures have **90% Sensitivity**, not changed by a previous course of chemotherapy. Bloos C/S are50-70% sensitive in the 2nd week, its sensitivity further being decreased by prior antibiotic therapy.

WIDAL 2nd week

INTERPRETATION OF WIDAL

1. High / rising titre of 0(>/=1:160)= active infection
2. High titre of H(>/=160)= Past immunization / infection
3. High titres of antibody to Vi Ag=carrier state

Stool + urine C/s in 3rd week

TREATMENT

ENTERIC FEVER or Salmonella Bacteremia (Systemic Febrile illness)

- Ciprofloxacin 500-750 mg bd x 10 Afebrile days OR
- Ceftriaxone 2.0 gm/D IV x 5 days OR
- Chloramphenicol 500mg QID PO/IV x 14 days
- Ofloxacin 15mg/kg /QD x 7days
- Other FQs can also be used

SALMONELLA DIARRHOEA (MILD ILLNESS)

- Any of the following for 3-7 days
- Ciprofloxacin 500-750mg Q12 hrly
- Norfloxacin 400mg Q 12 hrly
- Bactrim DS 1 BD
- Chloramphenicol 500mg QID

CARRIER STATE

DIAGNOSIS AND DEFINITION

Persistence of Salmonella in stools for a period exceeding 1 year.

LABORATORY INVESTIGATIONS

Vi antigen (high titres in carriers) Stool cultures positive for Salmonella.

TREATMENT REGIMENS

Ciprofloxacin 750mg BD x 4 weeks (Drug of choice) (Ref: CMDT 1999)
Norfloxacin 400mg BD x 4 weeks
Ampi/ amoxicillin 100mg/kg/day in divided doses x 6 weeks
Bactrim DS 2 BD x 6 weeks
OR Cholecystectomy (for those with abnormal gall bladder)

12 LEPTOSPIROSIS

Investigations:

- Se. Bilirubin (<2 usually)
- Alkaline PO4 ase (moderately elevated)
- SGOT/SGPT (rarely exceeds 100-200u)
- CPK (markedly elevated)
- Urine micro (preoteinuria, hematuria)
- Se. Creat. (can go upto 8mg/dl)
- Platelets decreased in DIC state.
- ECG showing changes consistent with myocarditis (ST-T changes)

Establish Diagnosis:

- MAT- Microscopic agglutination test, useful for serological grouping.
- Dipstick ELISA- more sensitive than MAT.

Treatment:

- **Severely ill patients**…..Penicillin G 1.5 ML. IU/Q6 Hrly or Ampicillin or Erythromycin
- **Less severe illness**….. Doxy 100mg/BD OR Ampi 500-750mg Q6 Hrly for 5-7 days
- Supportive Therapy for Hepatic/ renal impairments
- Treat Hypotension/ Major Haemorrhage

13 URINARY TRACT INFECTION

Presentations:-

Symptomatic-

- Frequency-dysuria syndrome (urgency, strangury, initial/ terminal haematuria, suprapubic discomfort.)
- Bacterial cystitis
- Abacterial cystitis (urethral syndrome)
- Acute pyelonephritis
- Acute prostatitis

Asymptomatic bacteriuria

Classification of UTIs:-

Uncomplicated-

- Normal renal functions
- Normal urinary tract
- Healthy females

Complicated-

- Abnormal tract (stones, VUReflux, indwelling catheter, Atonic bladder)
- Impaired host defences (Neutropenia, immunosuppressive therapy, Organ transplant recipient,Diabetes mellitus)
- Impaired renal function
- Virulent organisms (Staph., Proteus)
- All males

Clinical examination:-

Females:- Look for cystocele, vaginitis, cervicitis while doing a per-speculum and per-vaginalexamination

Males:- Look for phimosis, paraphimosis, feel for urethral strictures in the perineum, per-rectal tofeel the prostate

Diagnosis of UTI:-

Urine culture:-

A single MSU (mid stream urine) sample showing $>10^5$colony count has patient has a CI of more than95%

Routine screening for asymptomatic bacteriuria is recommended only in pregnancy (risk of pretermlabor)

A prostatitis should be diagnosed by collecting the first 5-10ml of urine after massaging the prostateper-rectally

Symptomatic females-10^3 colonies of a potential pathogen/ml of urine

Asymptomatic females – 105 colonies of a potential pathogen/ml of urine **Any male** – 10^3 colonies /ml of urine

Suprapubic sample/indwelling catheter-10^2 colonies /ml of urine is significant

Catheter in-and-out technique-10^3 colonies/ml of urine

INTERPRETING URINE MICROSCOPY AND CULTURE

Bacteria without pyuria:-

Asymptomatic bacteriuria
Contaminants (>5 epithelial cells/ HPF signifies contamination0

Pyuria without bacteria (sterilepyuria):-

Culture inhibited by antibiotics
Antiseptic contamination of specimenTuberculosis (typical/atypical) Fungal
Acute febrile episodes
Diabetes mellitus
Glucocorticoid therapy
Calculi (renal/ bladder)
Papillary necrosis
Chemical cystitis (Cyclophosphamide)
Analgesic nephropathy/ Interstitial nephritis
Non-bacterial (Chlamydia)
False pyuria (Cervicitis/ vaginitis)

Investigating an UTI:-

If complicated UTI is suspected from history and examination,

1. X-Ray KUB(stones, renal shadows)
2. Ultrasound abdomen (hydroureters, hydrnephrosis, structural anomalies)
3. Intravenous pyelogram(tract abnormalities, functional status)
4. Residual volume of urine (prostatic obstruction)
5. Uroflowmetry studies (Obstructive uropathy)
6. Cystometrogram (Detrusor instability/Atonic bladder / Stress incontinence
7. Cystoscopy (chronic cystitis, isolated haematuria)

Management of UTIs (use any one of the regimen)

Uncomplicated UTI-

Single dose regimen:-

- Cotrimoxazole DS 2 tabs
- Norfloxacin 800 mg
- Ciprofloxacin 500 mg
- Trimethoprim 600 mg

Short course (3day) regimen:-

- Cotrimoxazole DS 1 Bd
- Norfloxacin 400 mg BD

- Amoxycillin 250 mg q8hly
- Ciprofloxacin 250 mg BD
- Lomefloxacin 400 mg OD
- Augmentin(500mg mg max/ 125 mg Clav) q12hly
- Nalidixic acid- 500 mg/q8hly
- Nitrofurantoin 50 mg/q8hly

Complicated UTI:-
With poor renal function:-
3 day regimen-
Cephalexin 250mg q8hly
Cefaclor 250mg q8hly

With prostatitis:-
Duration of therapy 2-4 weeks-
DOC-Quinolones in the dosages mentioned above Repeat
C/S after 2 weeks of antibiotics

In pregnancy:-
DOC- Cotrimoxazole DS 1 BD x 3 days
Repeat C/S after 7-10 days

With pyelonephritis:-
Intravenous antibiotic therapy for 5 days
Gentamicin 3mg/kg loading, followed by 1mg/kg q8hly Amikacin
2.5-3.5 mg/kg Q12 hly (5.0-7.5 mg/kg/day) Ciprofloxacin 100mg
q12hly- switch over to oral after 48h Amoxycillin 1g Q8hly
Cefazolin 1g Q8hly Cephradine 1g Q8hly
Ceftriaxone 2g Q24hly (no need for dose adjustment in renal failure0 Imipenem
/ Cilastin 500mg /500mg Q8hly
Augmentin 1g amox /200mg Clav Q8hly

Recurrent UTI:-
Prophylaxis- Norfloxacin
200mg OD
Cotrimoxazole 1 tab OD
Nitrofurantoin 50mg OD
Cephalexin 125mg OD

Followup:-
Repeat C/S after 10-14 days

Urologic evaluation needed for all males, complicated UTIs and neonates for urinary tractabnormalities

Prophylactic antibiotics recommended for recurrent UTIs in women

In post-menopausal women, gynaecological to rule out a senile vaginitis/ urethral stricture/ carcinoma cervix . Vaginal oestrogen creams, +/- dilatation (in case of urethral stricture), +/- HRTmay be needed.

FEVER

DEFINITION:- Oral temperature at 6 a.m. > 98.9F (37.2C)

OR at 4 pm >99.9F (37.7C)

Is it a fever? Temperature

chart,

Document Fever by measurement.

HOW TO APPROACH? DURATION OF SYMPTOMS

Short (3 DAYS)

Look for localizing signs / symptoms ……order investigations accordingly

- Icterus…..LFT (Enzymes)
- Leptospirosis……send CK
- Throat/Teeth infection Pus Swab smear/C/S
- Sinus pain, Tenderness, Post nasal Drip X ray PNS
- Cough, Sputum, DyspnoeaX ray chest
- Abdomen Hepatosplenomegally MP, ECR, CBC, USG, Blood C/S
- Lymphadenopathy/Lymphangitis
- Dysuria .....urine Microscopy/C/S
- Skin abcess..... Look at the perianal area, Pus smear /C/S
- Genitalia ..... Discharge smear
- Fever with rash..... consider ricketsial fevers, typhoid, EBV infection, secondary syphilis, dengue, brucellosis, Viral exanthems, drug fevers.

No localizing signs/symptoms

- Malaria
- Enteric fever
- Viral / Anicteric Hepatitis
- UTI
- Viral fever – Dengue

Investigations

- TC,DC (If low, consider possibility of viral or enteric. If neutropenic, for differentials to beconsidered), MP/MF.
- LFT (enzymes, alkaline phosphatase)
- Urine Micro, C/S

- Blood C/S : Not to be done as a preliminary investigation in fevers of short duration unless there is strong clinical suspicion of Bacteremia/enteric fever in physical examination and lab investigations. For fevers persisting for > 1 wk..consider blood c/s.
- Widal (If fever > 1 wk)

Prolonged fevers

History

Travel, cardiac symptoms, H/o exposure, transfusions, joint pains, arthritis, Perianal Ulcers, drugsand medications.

How to use localizing signs?

Generalised Lymph Node Enlargement (LNE)

- Infections….EBV, CMV, Toxoplasma Brucella, Syphilis, PGL of HIV.
- Non-specific response to viral infection
- Leukemia, Lymphoma
- Tuberculosis
- Sarcoidosis
- Metastatic Disease

Hepatomegaly

MILD: Not useful, very non-specific

MODERATE: malaria, tuberculosis, amoebic abscess, hydatid.Splenomegaly

MILD/MODERATE : malaria, acute viral hepatitis, typhoid, military TB, septicaemia, infectiveendocarditis.

LARGE: CML, tropical splenomegaly, Kal-azar, myelofibrosis, lymphoma.

COMBIANTIONS OF :- FEVER+HEPATOSPLENOMEGALY+GENERALISED LYMPHADENOPATHY

- Disseminated Tuberculosis
- Leukemias, Lymphomas
- Immune-Stills, Systemic onset rheumatoid arthritis, SLE, MCTD, Polymyositis.
- HIV related (PGL- persistent glandular lymphadenopathy; Defined as Lymphnodes at >2extrainguinal sites, >3mo, > 1 cmsize)
- Metastatic Disease
- Secondary Syphilis

- EBV

Secondary investigations depending on the clinical findings :

DO NOT investigate too early in the phase of the disease. If already done, you may need to repeatthe first few e.g..Chest X-ray, USG, LFT, WIDAL.

Counts may need to be rechecked to look for a changing pattern. WIDAL should be repeated to lookfor increasing titres.

- CRP (C-reactive protein may give a clue as to whether it is infective inflammatory or neoplastic/drug related in etiology. Drug-related fevers should have normal CRP. Also usefulin the follow up of patients prior to start of empirical ATT – more sensitive marker of diseaseactivity and response to therapy than ESR.)
- Chest X-ray
- USG- always check for hidden abscesses. (Liver, subdiaphragmatic, perisplenic, pelvic, paraspinal, psoas, periappendiceal.) Ultrasound done too early may miss a liver abscesswhich in the process of breaking down.
- Bone marrow for routine C/S, AFB, fungal (In neutropenic and HIV +ves),trephine biopsy,smear, NNN medium culture. (If suspecting Kala-azar)
- Serology- ANA, LE, RF, HIV Elisa, Weil – Felix
- Lymphnode biopsy for AFB smear, C/S, histopathology, special stains for leukemia/lymphoma
- Thyroid function tests
- Liver biopsy
- Trans thoracic ECHO; if negative but strong suspicion of IE, transoesophageal can be done.Special tests :- (Only if localizing signs are present)

  UNSOLVED PUO
  If still undiagnosed……..
  THERAPEUTIC TRIAL
- Chloroquine and the, if no response
- A.T.T

TYPES OF PUOs

	CLASSICAL	NOSOMICAL	NEUTROPENIC	HIV-ASSOCIATED
DEF.	FEVER >38.0, >3 WKS. >2 VISITS/ 3DAYS AS INPATIENT	FEVER >38.0, >72 HRS NOT PRESENT/ INCUBATING ON ADMISSION	FEVER >38.0, >72 HRS <1000 PMNs	FEVER >38.0, >4WKS. FOR OPD/>3 DAYS FOR IP HIV INJECTION CONFIRMED
PATIENT LOCATION	COMMUNITY CLINIC/ HOSP.	ACUTE CARE HOSPITAL	HOSPITAL OR CLINIC	COMMUNITY/ CLINIC/ HOSPITAL
LEADING CAUSES	INFECTIONS INFLAMMATORY CONDITIONS LYMPHOMA MALIGNANCY	NOSOCOMIAL INFECTIONS (Respiratory, Bacterimia, Urinary, Wounds) SEPTIC THROMB - OPHLEBITIS	MAJORITY DUE TO INFECTIONS (candida aspergillosis, perianal) ETIOLOGY OFTEN NOT KNOWN	TYPICAL/ ATYPICAL MYCOBACTERIA TOXOPLASMA CMV

HISTORY TO BE EMPHASIZED

TRAVEL TO REGIONS ENDEMIC FOR CERTAIN INFECTIOUS DISEASES	OPERATIONS PROCEDURES DEVICES, CATHETERS , IV LINES DRUGS ET	DRUGS AND DOSAGE	DRUGS IVDU (IV DRUG USERS) RISK FACTORS

EXAMINATION EMPHASIS

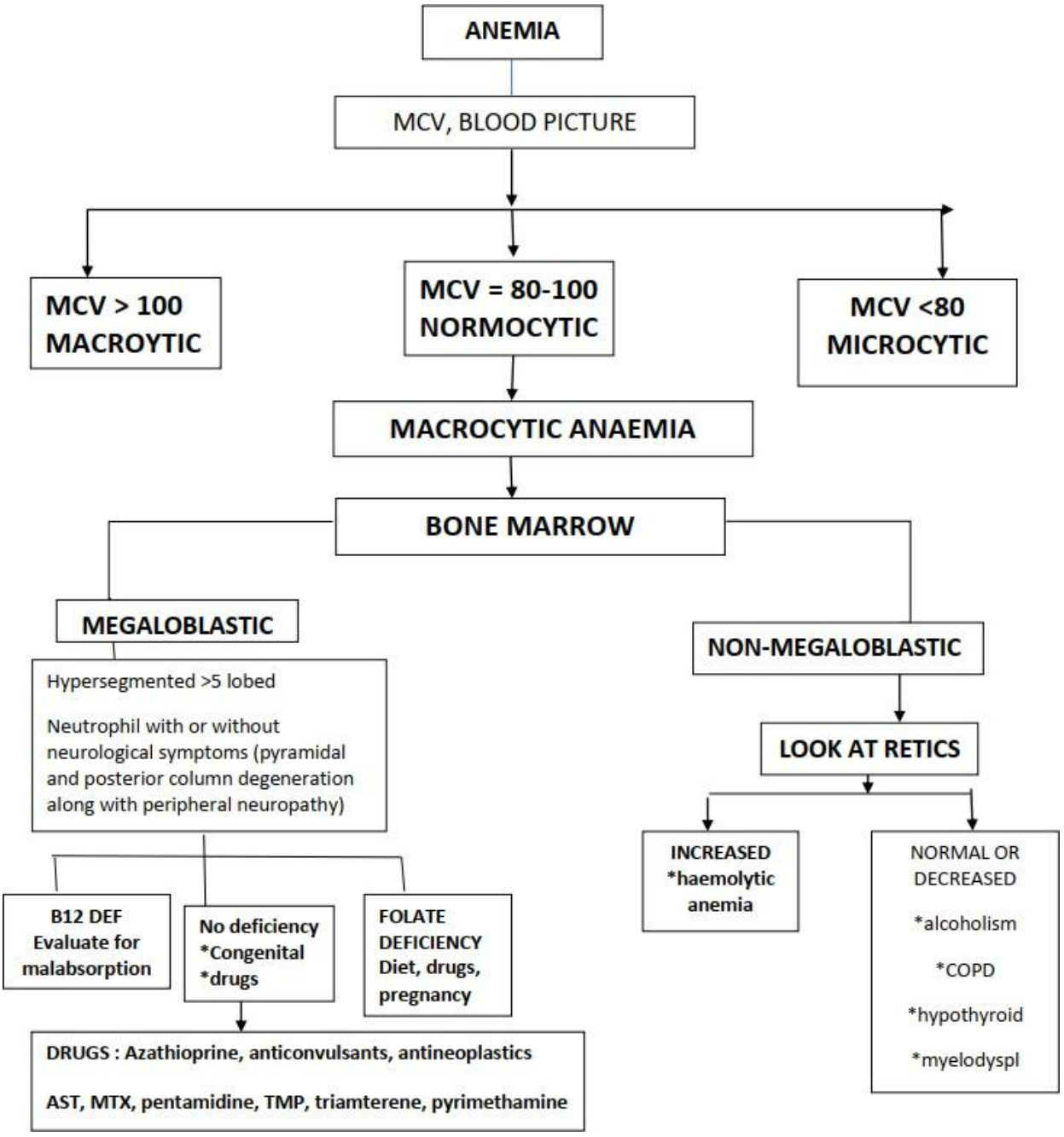
LYMPHNODES, JOINTS, ENT, NAILS, FUNDUS, DISTAL PULSES, ORAL CAVITY, ABDOMEN, PER-RECTAL, PV	WOUNDS DRAINS DEVICES SINUSES (NG TUBE) URINE BED SORES	SKIN FOODS IV SITES LUNGS PERIANAL AREA	SKIN FOLDS IV SITES LUNGS PERIANAL AREA

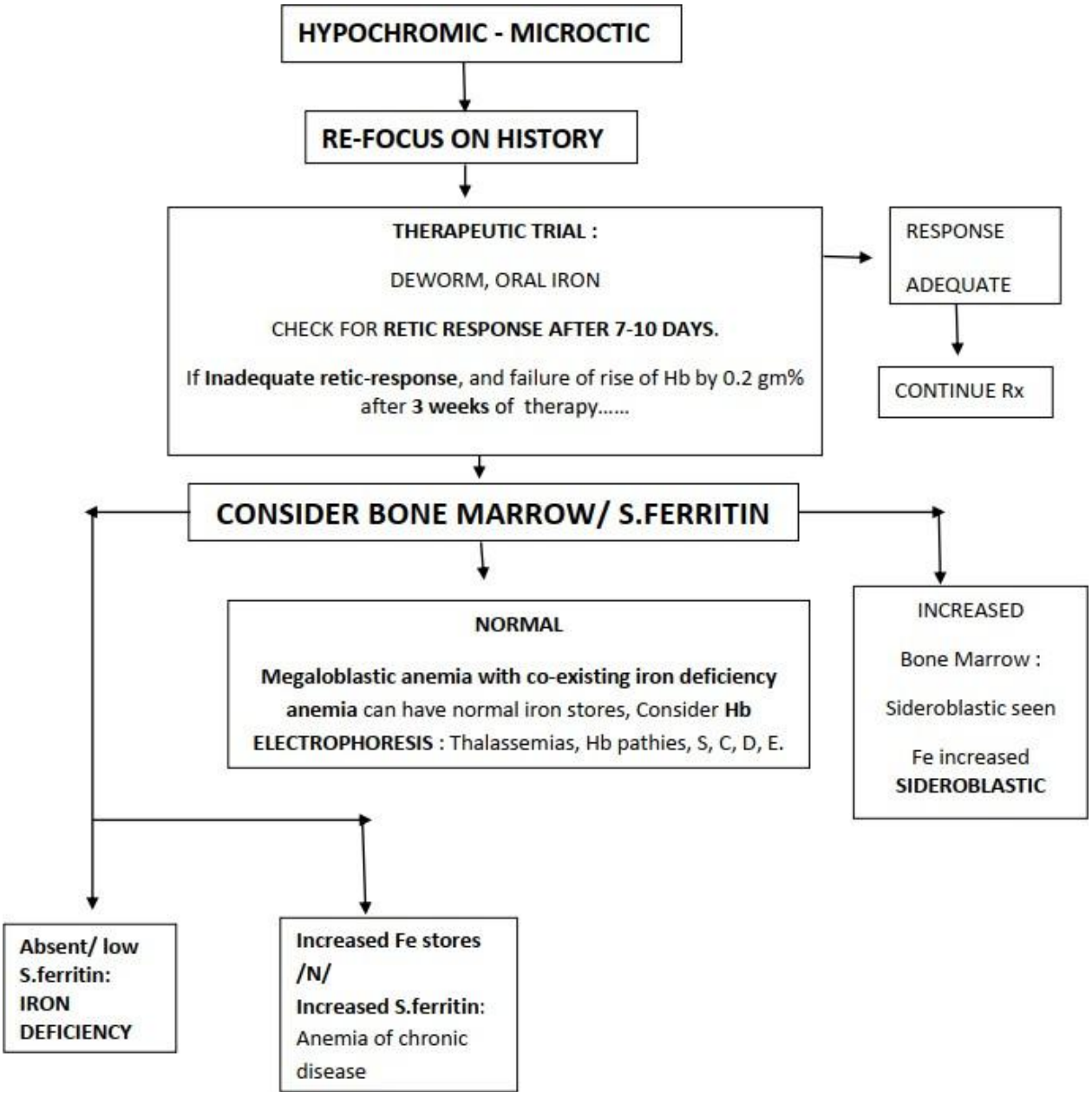
INVESTIGATIONS: AS DISCUSSED ABOVE

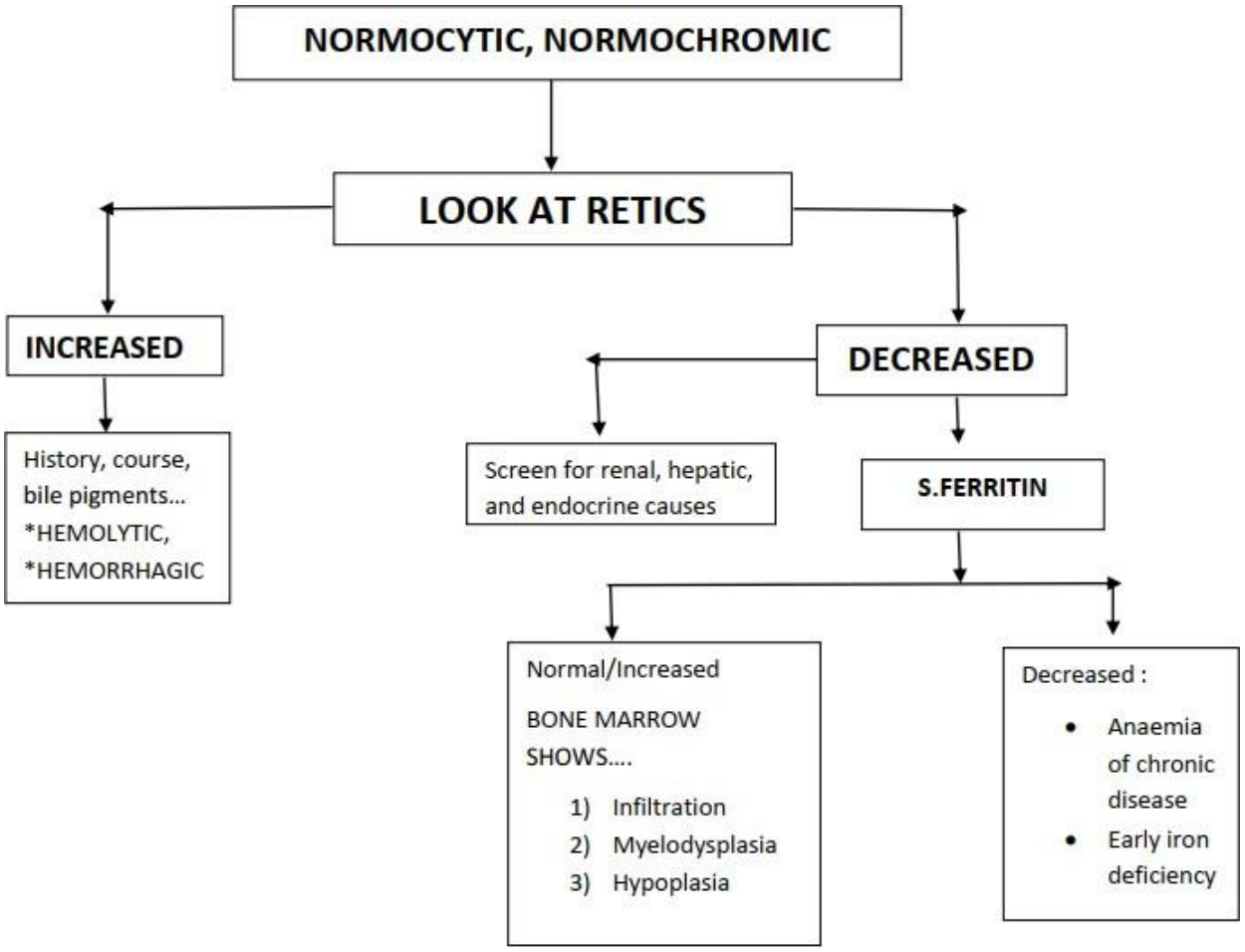
MANAGEMENT

TREATMENT AS PER DIAGNOSIS AVOID EMPERICAL DRUG THERAPY	DEPENDS ON SITUATION	Usually a combination of CEFOTAXIME + GENTAMICIN + FLUCONAZOLE/ AMPHOTERICIN-B	AZT ANTI-MICROBIAL AGENTS

HAEMATOLOGY
HOW TO INTERPRET THE TESTS AND APPROACH A CASE OF ANEMIA?




HYPOCHROMIC - MICROCTIC
RE-FOCUS ON HISTORY
THERAPEUTIC TRIAL :
DEWORM, ORAL IRON
CHECK FOR RETIC RESPONSE AFTER 7-10 DAYS.
If Inadequate retic-response, and failure of rise of Hb by 0.2 gm% after 3 weeks of therapy.....
RESPONSE
ADEQUATE
CONTINUE Rx
CONSIDER BONE MARROW/ S.FERRITIN
NORMAL
Megaloblastic anemia with co-existing iron deficiency anemia can have normal iron stores, Consider Hb ELECTROPHORESIS : Thalassemias, Hb pathies, S, C, D, E.
INCREASED
Bone Marrow :
Sideroblastic seen
Fe increased
SIDEROBLASTIC
Absent/ low S.ferritin: IRON DEFICIENCY
Increased Fe stores /N/
Increased S.ferritin:
Anemia of chronic disease



MANAGEMENT OF ANAEMIAS IRON

DEFICIENCY ANAEMIAS....

Remember, among premenopausal women, 60% of iron deficiency anemia is due to pregnancy and menstruation related reasons. Among men and post-menopausal women, 80% is due to GI blood loss. Out of which, 25% is a malignant cause, more in >50 yrs. Age group.

When B12 deficiency co-exists with iron deficiency, the bone marrow might show adequate iron stores because in the presence of megalo-blasticanemia, the marrow fails to use the iron stored.

- DIETARY
- DEWORM-MEBENDAZOLE 100 MG MD X 3 DAYS repeat after 2 wks. (safe in pregnancy)OR ALBENDAZOLE 400 MG STAT + REPEAT AFTER 2 WKS.

- ORAL IRON THERAPY:
  Tab. Ferrous SO4 200mg (60 mg elemental Fe) tid x 6months/ till adequate level of Hb. Is reached.continue for about same duration to replenish stores increased absorption with.C; do not take it with antacids, tetracyclines. Best absosrbed in empty stomach, but causes gastritis; therefore take at any time of the day. Check compliance by asking stool colour (black)
  B12/Folate deficiency…….
  Emperic therapy of megaloblastic anaemia with folic acid only is not recommended!----
  Neurological abnormalities might worsen
  FOLIC ACID:- 5mg / OD X 4mo; Maintenance -5 mg/ week
  VITAMIN B 12:- (Inj. Neurobion Forte 1 ampoule contains 1 mg of Vitamin B12) 1mg IM Dailyfor 1 week..followed by 1 mg IM/wk x 1-2 mo till normalization of Hb. Maintenance- 1 mg IM/ mo (Ref. Washington manual) to decrease the rate of relapse.

Dimorphic anemias…..mostly nutritional, MCV and bone-marrow iron stores might be normal.Combine both the therapies mentioned above.

FOLLOW UP OF ORAL REPLACEMENT THERAPY:-

MONITOR RETICS
IRON…….7-10 DAYS LATER (Adequate response is a rise of Hb. by 0.2g% at the end of 3 wks oftherapy).

B 12 .....4 DAYS, PEAKS AT 1 WEEK

FOLATE ..... 4 DAYS LATER

INDICATION FOR BLOOD TRANSFUSIONS:
Symptomatic: Heart failure
Elderly with angina Hypoxic encephalopathy Shock due to haemorrhage Pregnancy with the <5gm%

DISORDERS OF COAGULATION CASCADE (BLEEDING DISORDERS)

INTERPRETATION OF LABORATORY PARAMETERS

FACTOR DEFICIENT	BT	PTT	PT	TT
VII	N	N	A	N
VIII, IX, XI, XII	N	A	N	N
Von-Willebrand factor	A	A	N	N
Dysfibrinogenemia	N	A	A	A
II, V, X.	N	A	A	N
XIII	N	N	N	N

BT= Bleeding Time, PTT= Partial Thromboplastin Time, PT= Prothrombin Time, TT= Thromboplastin Time N=Normal, A= Abnormal.

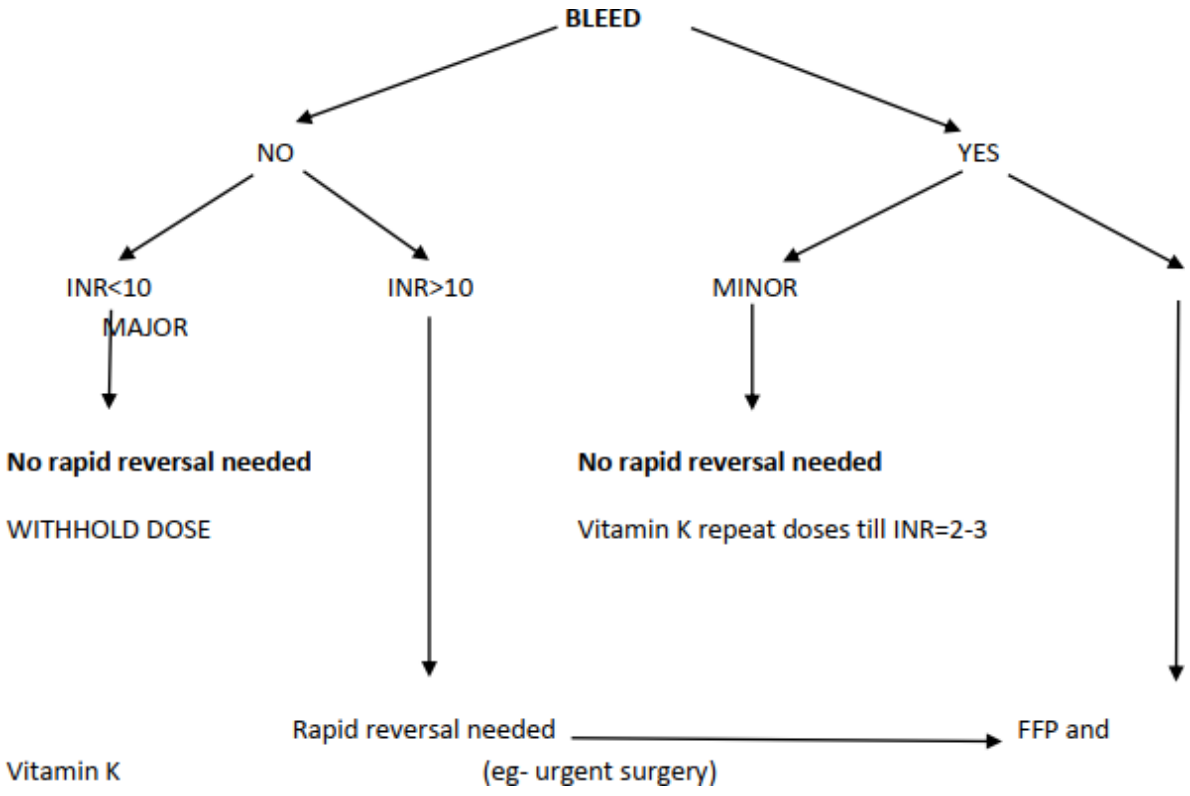
To differentiate between IX and VIII, a PTT should be repeated after trying correction with AGED and ADSORBED sera. If AGED serum corrects a PTT, it is a factor IX deficiency. If ADSORBED serum corrects a PTT, it is a factor VIII deficiency.

ANTICOAGULATION

Therapeutic goals for anticoagulation:

INDICATION	INR
Venous thromboembolism Pulmonary embolism Previous systemic embolismAtrial fibrillation Bioprosthetic heart valves	2-3
Mechanical heart valves	2.5-3.5
Antiphilosophical antibody with thrombosis	3-4

Correction of prolonged INR:



BLOOD AND BLOOD PRODUCTS

Guidelines to transfusion therapy RED

CELL PRODUCTS –

Whole blood- Changing concepts prefer need- specific individual component use; indications aremassive hemorrhage, Exchange transfusion etc.

Red cell concentrates – Indicated whenever there is impaired Oxygen carrying capacity, Chronic anaemias, Bone marrow failure, Transfusion of 'O' group RBC concentrate in emergency where ABOgrouping of blood is not feasible.

Leukocyte reduced RBCs- 85-90% depletion of leukocytes; Indicated in conditions where immunerelated post-transfusion febrile reactions are troublesome.

Washed RBCs- Plasma depleted, Leukocyte depleted, must use within 24 hours; Indicated in severeallergic reactions and in anaphylaxis in IgA deficiency.

Frozen RBCs- long term storage, plasma and Leukocyte depleted; to be used within 24 hours ofthawing- Indicated in autologous storage in postponed surgery. Rare donor unit.

Storage and problems associated with it

- **AIM-** To maintain RBC ATP concentration, which is essentials for red cell survival
- Temperature- 4-6^0 C. At this temperature the Na/K pump stops working. Therefore the intracellular and extracellular potassium concentrations equilibrate.
- **Hemolysis-** In prolonged storage due to ATP depletion in RBCs
- **Hyperkalemia-** in the supernatant plasma- Due to the above two factors – namely hemolysis and potassium redistribution and equalization; but rarely of any consequences unless A single unit of RBC concentrate has only 70 ml of plasma and a potassium of 5.5 meq/L at product expiry.
- **Citrate toxicity –** This is seen in large volume transfusions; more so in the setting of liver dysfunction. Hypocalcemia with its cardiovascular ill effects in the presence of a near normal total Serum Calcium is characteristically seen. IV Calcium gluconate in such settings can be considered where citrate toxicity is anticipated.
- **Microaggregates** – Stored blood has microaggregates made of platelets, fibrin and red cells. These are thought to cause post- transfusion hypoxia due to pulmonary microembolism called as TRALI (transfusion related acute lung injury). Therefore if >5U of blood are transfused at the same time, 20-40 um pore sized filters are to be used.

Guidelines to replacement therapy

- Should **target a Hb** of 8 g% for all medical conditions. **A higher Hb is not additionallybeneficial** to the patient.
- Earlier concept of **perioperative blood transfusion** as soon as the Hb falls to the 'magic' figure of 10gm/dl- (to cause faster wound healing and avoid the risks of general anaesthesia)has been thrown away. Now a Hb of 7gm% is used as a cut off instead.
- In case of **hemorrhage/ surgery with blood loss**,
  <10% of blood volume loss needs no transfusion;
  </= 20% requires crystalloids exclusively;
  >25% requires red cells along with crystalloids and colloids because the volume afterreplacement has a still poor oxygen carrying capacity and therefore tissue hypoxia.

PLATELET TRANSFUSION

Guidelines to replacement therapy-

- Platelet **lifespan** = 9.6 +/- 6 days.
- **Normal rate of platelet destruction**/ day is 3.6 x 10^{10} platelets
- Platelet concentration of **<10 x 10^9 /dL** increases chances of spontaneous intracerebralbleeds and can present as serious GI/GU bleeds
- At **<5 x 10^9** /dl can present as serious GI/GU bleeds
- If there is **major bleeding** of platelet concentrations of **more than 20 x 10^9 /dl, a vessel wallor platelet dysfunction should be looked for.**
- **One unit** of platelet rich concentrate contains **>5.5 x 10^{10}** platelets and at good centres, itmay even contain **7.0 x 10^{10}** platelets.
- Transfused platelets **distribute** as 1/3rd into a normal sized splenic pulp and 2/3rd in theintravascular compartment
- A single PRC increases the platelet concentration in a 75kg man by 6 x 10^9 or 8 x 10^9 plateletsdepending on the platelet concentration in the unit, apart from the normal splenic pooling.
- Therefore in **a production defect**, replacement dose would be **1U/ day**, providing at least **3.7 x 10^{10}** platelets in the intravascular compartment.
- **Hypersplenism** – It is unusual for this to present with platelets <40-50 x 10^9 platelets / dl. Therefore never calls for transfusion. If needed, another possible cause for bleeding shouldbe sought for.
- **Consumptive coagulopathy** – Needs supportive therapy and treatment of the cause. Mayneed platelet transfusion to stop troublesome bleeding.

Indications for platelet transfusion

- <5 x 10^9 platelets in a production disorder is an indication for prophylactic platelettransfusion.
- 6-10 x 10^9 platelets with fresh minor haemorrhages.
- 11-20 x 10^9 platelets with co-existing coagulation factor deficiency/ Heparin therapy/planned lumbar puncture.

PLASMA DERIVATIVES

FFPs – Fresh frozen plasma – frozen plasma within 6 hours of donation at a temperature of </= -18C. Should be used within 1 year. Should be used after 20-30 mins of thawing and before 24 hours;because factors V and VIII degrade to inadequate levels after 24 hours of thawing.

Indications –

- **Multiple coagulation factor defects** (e.g. CLD – just before a procedure only if PT > 16-18 secs, or in Massive transfusions – of 6-10 U of RBCs with post – transfusion bleeding)
- **DIC** (If bleeding or before any procedure)
- **Rapid oral anticoagulant reversal** (Stopping drug with normal GI absorption of Vitamin K takes 48h for normalization of coagulation parameters; With inj. Vitamin K it takes 12-18h;With FFP, immediate reversal occurs.)
- TTP/ HUS
- **Congenital coagulation defects**
- **C1 esterase deficiency** (life threatening angio- edema)

Not indicated:-

- Immunodeficiency
- Burns
- Volume expansion
- Source of nutrition
- Reconstitution of packed red cells

Composition-

1 U of FFP contains 200-280 ml of plasma.
0.7 -1.0 U/ml of each of the coagulation factors per ml of FFP. 1-2 mg of Fibrinogen per ml of FFP

Dosage - depends on the desired increment needed in the level of the clotting factors. Alternatively, **10-15 ml/kg** can be used as a general guideline along with monitoring the clinical response.

CRYOPRECIPITATE - Prepared by refreezing at < - 18C, the precipitate from a thawed FFP at 4C, afteraddition of 10-15 ml of plasma. It can be stored for 1 year at < - 18C.

Properties and composition – Richest source of factor VIII, vWF and fibrinogen, Contains 80-100 Uof factor VIII, 250mf of Fibrinogen, 50-60 ml of Fibronectin, 40-70% of the original vWF concentration (has no factor IX)

Indications –

- Hemophilia A/ vWD
- Fibrinogen deficiency (< 100 mg/dL)
- Dysfibrinogemia

Not inidicated –

- Uremic bleeding
- Sepsis

Goal is to maintain a **fibrinogen level of > 100mg/dL. 2-4**

U/10kg if the FFP is poorer in consumption.

U/10kg if it is rich in its factor and fibrinogen content.

ALBUMIN

Indications

- Large volume paracentesis
- Nephrotic syndrome with resistant ascites.
- Volume / fluid replacement
- Thermal injury
- Cerebral ischemia
- Plasmapheresis
- Support blood pressure during haemodialysis.
- **Caution -** Indiscriminate use of albumin is associated with a modest increase in the rates lofmortality in critically ill patients. (Ref : intensive Care Med 1999, Vol 25:321-324; Brit Med J 1998; 317: 235-240)

19 ACUTE BRONCHITIS

THIS IS A DIAGNOSIS BY EXCLUSION

TREATMENT …. MOSTLY SYMPTOMATIC

- Rest
- Antipyretics
- Beta agonist bronchodilators if ronchi are predominantly
- Deriphylline (R) 300 mg hsod
- Codeine preparations for cough suppression
- Expectorants
- Hydration : Steam inhalation, nebuliziers
- Antibiotics if fever persists, C XR shows Amoxycillin, azithromycin, sparfloxacin, ofloxacin,levofloxacin.

20 PNEUMONIAS

IN ADULTS:-

ESTABLISH DIAGNOSIS:-

Typical Pneumonia:- Cough, Sputum production, Fever (>38C/>104F) +/- Chest pain (pleuritic),Dyspnoea, Hemoptysis, O/E Tachypnoea, tachycardia, Pleural rub.

Atypical Pneumonia:- More often Subacute (slow onset, 3-4 days), Nonpulmonary symptoms, e.g.myalgias, joint pains, anorexia, fever, chills, headache, non-productive cough; disparity between chest findings and radiological features (more severe).

COMMUNITY ACQUIRED (NON-HOSPITALISED) SIX

PRIMARY PATHOGENS

S. Pneumoccus	Legionella
H. Influenzae	Mycoplasma
Moraxella	Chlamydia

RISK FACTOR FOR EXPOSURE	Disease causing organism.
COPD	S. Pneumoniae, H. Influenzae Legionella, GNB
>60y age	S. Pneumoniae, H. Influenzae

Smoker	H. Influenzae, M. Catarrhalis
Alcoholism	S. Pneumoniae, Klebsiella Anaerobes, MTb
<25y age	Mycoplasma, Chlamydia

THERAPY:-

Azithromycin 0.5gm POx1 followed by 0.5gm/d x7-10 d or Clarithromycin or Fluoroquinolones (drugs effective against highly resistant S.Pneumococcus) Sparfloxacin/levofloxacin.

CRITERIA FOR HOSPITAL ADMISSION:- (Only 20% of the Community acquired pneumonias requirehospital admission)

- AGE >65 YRS
- Other comorbidities e.g. DM, IHD, COPD, renal disease
- Unexplained leukopenia < 5000/cu mm.
- Staph, aureus, GNB or anaerobes
- Suppurative complications e.g. empyema, arthritis, meningitis
- Failure of OPD treatment
- Needing IV antibiotics
- Tachypnoea >30/mm, Tachycardia>140/min., PaO_2< 60mmof Hg., delirium
- SpO_2<90% by pulse oximetry.

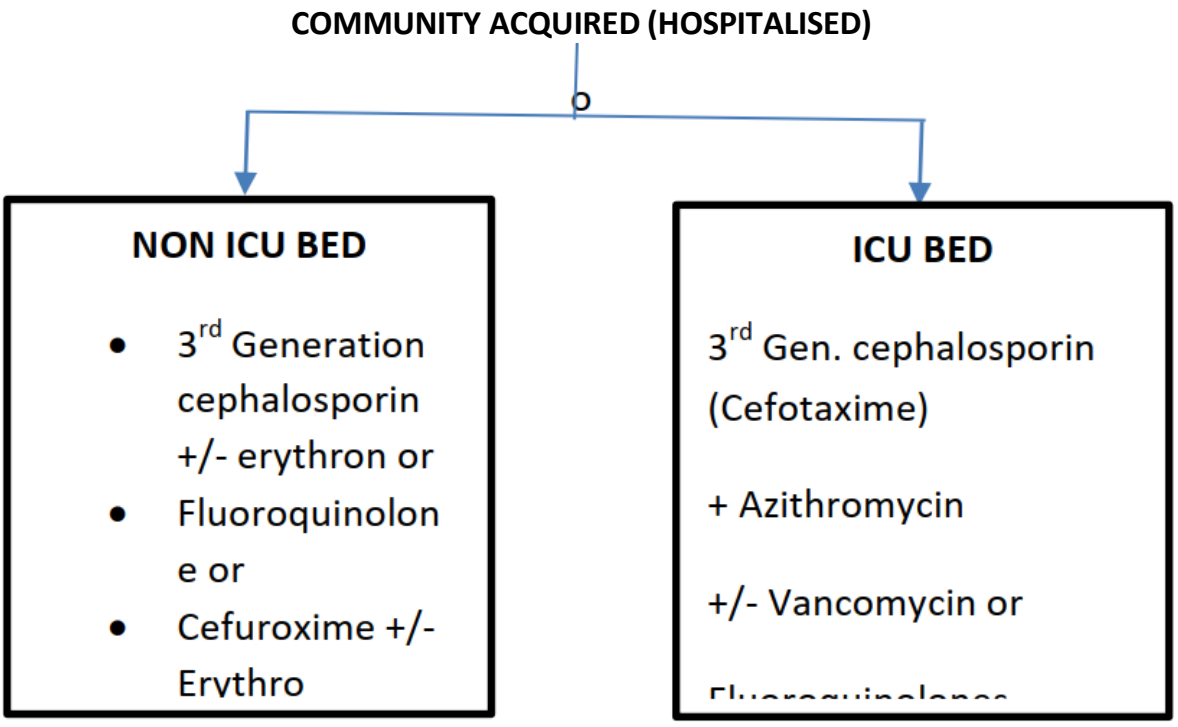
DOSAGES OF ANTIMICROBIALS IN TREATMENT OF PNEUMONIAS

Ampicillin	3gm IV q6 hrly
Azithromycin	500 mg IV od x3 days (in severe pneumonias, community acquired) Followed by oral 250 mg bd x 7-10 days
Cefotaoxime	1-2 gm IV q8-12 hrly
Ceftazidime	2 gm IV q 8 hrly
Ceftriaxone	2 gm IV q 12-24 hrly
Cefuroxime	750 mg IV 8 hrly
Ciprofloxacin	400 mg IV / 750 mg po q 12 hrly

Clindamycin	600-900 mg IV q 8 hrly / 300-450 mg q 6 hrly
Erythromycin	0.5-1.0 gm IV q 6 hrly

Single dose of ammoglycosides per day has less renal toxicity

Gentamicin	5 mg/kg/ day in 3 divided doses q 8 hrly
Metronidazole	500 mg q 8 hrly (po/IV same effect)
Sparfloxacin	400 mg x1d, followed by 200mg/d x 10 days
Levofloxacin	500 mg once daily
Vancomycin	1.0 gm IV q 12 hrly

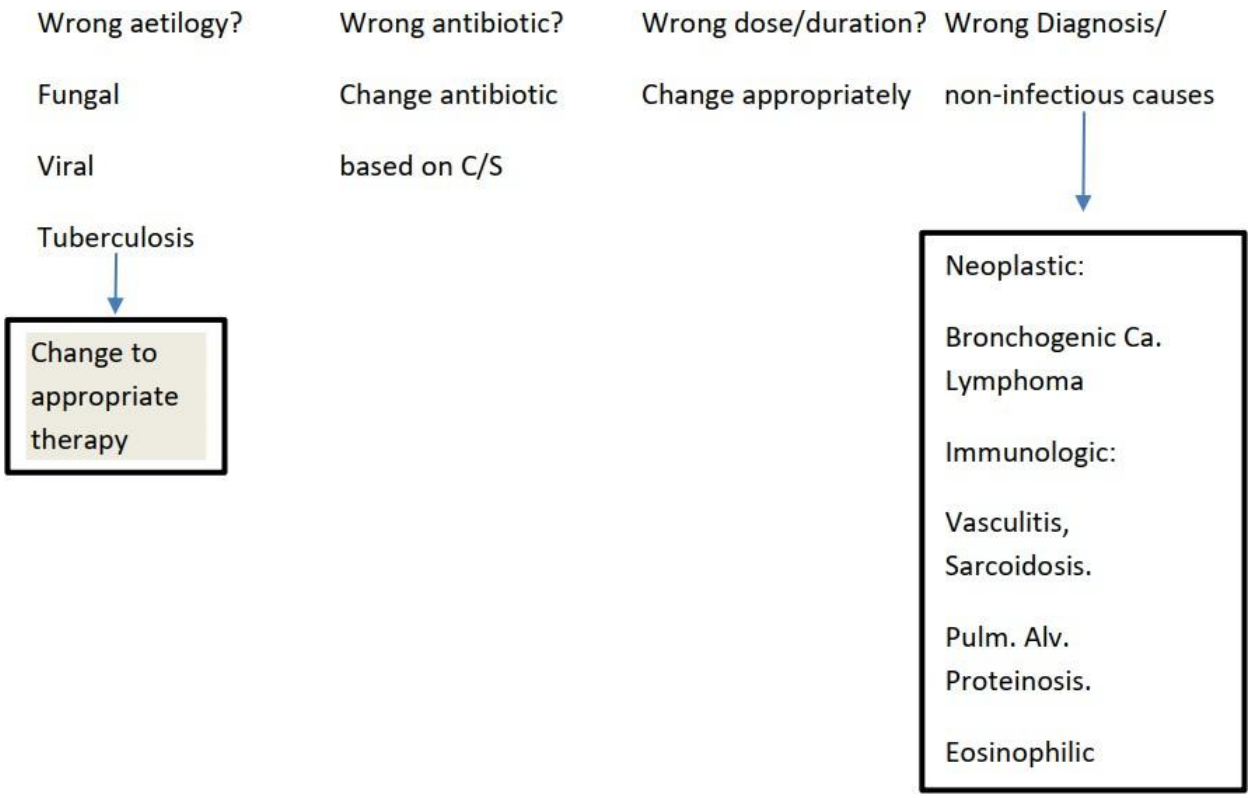


PNEUMONIA IN HIV- discussed under DISEASES RELATED TO HIV

GENERAL MEASURES:- EXPECTORANTS, HUMIDIFIED OXYGEN INDICATED BASED ON SpO_2,ANTIPYRETICS

FAILURE TO IMPROVE WITHIN 48-72h OF INITIATION OF ANTIBIOTIC THERAPY:-

Consider possible causes:-



21 PULMONARY EMBOLISM

SUSPECT DIAGNOSIS:-

- Anybody with sudden onset of breathlessness
- Hypoxia with clear chest X-ray
- Sinus tachycardia on ECG and
- Signs of right heart failure/strain on ECG (S1, Q3, T3)

PRESENTATIONS:

70-90% present with

Sudden onset of breathlessness

Pleuritic chest pain 70% Cough
40-50 %
Hemoplysis 20 %

Look for signs of tachycardia, tachypnoea and

Loud P2, raised JVP
Inspiratory crackles
Pleural rub
Pleural effusion (80% hgic and 20% Transudative)
DVT, Varicose veins

Look for stressors/predisposing factors to support diagnosis (their presence being, most specific)

Immobilization/stroke
Orthopedic surgery
Pelvic/lower limb fractures
Obesity OCP/Pregnancy/Postpartum
Indwelling central venous line
Occult carcinoma/chemotherapy for same

CONSIDER DIFFERENTIALS

- MI/Unstable angina… ECG changes, history, description of pain
- Pneumonia, COPD, pneumothorax X-ray chest
- CCF…… Usually no chest pain, more chronic, insiduous onset
- Asthma…… Improvement with bronchodilators
- Pericarditis… ECG, pain>dyspnoea
- Primary pulmonary HT
- Costochondritis…Tender costochondrial junction
- Psychogenic hyperventilation……Inconsistent history and findings

Preliminary investigations:-

Hb, TC/Dc, Platelets, ABG (PaO_2 <80 mm of hg with decreased $PaCO_2$ Baseline

PT/PTT for starting anticoagulation Rx

Chest X ray ECG

ECHO

Primary therapy:-

General … A-B-C, management of critically ill patients

- Pain relief
- Do not volume overload
- Dobutamine for heart failure/cardiogenic shock
- Parenteral anticoagulation

Start oral anticoagulation at the onset itself

22 CORONARY ARTERY DISEASE/ISCHAEMIC HEART DISEASE

Coronary artery narrowing causing myocardial ischemia can present as one of the following:

- Chronic stable angina
- Acute coronary syndromes
- Acute myocardial infarction

In all three presentations, the basic problem is myocardial ischemia. In chronic stable angina, there is fixed atherosclerotic narrowing of the coronary vessel(s). In acute coronary syndromes (of unstable angina and non Q myocardial infarct) and acute myocardial infarction, there is rupture of the atherosclerotic plaque with thrombus formation occluding the lumen of the coronary artery – in a Q wave infarct, there is complete occlusion of the lumen whereas in the acute coronary syndromes of unstable angina and non Q myocardial infract, the problem is that of a flow limiting thrombus. In Prinzmetal angina, the problem is that of vasospasm. The goal of therapy in the three presentations differ. In chronic stable angina it is to maintain exercise tolerance compatible with an adequate quality of life either using drugs or by revasularisation. In an acute myocardial infarct, the goal is to salvage myocardium by establishing reperfusion as soon as possible by drugs (aspirin, thrombolysis) or by catheter technique (PTCA). In unstable angina and non Q myocardial infarct, the goal is to stabilize the thrombus and use long term therapy to heal the lesion (control of metabolic and otherrisk factors).

Although acute myocardial infarction can be considered a form of acute coronary syndrome, it isconsidered separately as the management is different from other acute coronary syndromes.

Chronic stable angina:

In this presentation, there is chest pain precipitated by exertion or emotion (pain builds up within aminute) which is relieved promptly by rest in 5 – 15 minutes (even more rapidly with nitroglycerin).

Management:

1. Control risk factors: smoking, diabetes mellitus, hypertension, hyperlipidemia, obesity
2. Treat precipitating factors: anemia, hyperthyroidism
3. Drug therapy
    a) Antiplatelet drugs: aspirin 75-325 mg/day; clopidogrel 75 mg/day
    b) Beta blockers: propranolol 40-80 mg 2-4 times/day; metoprolol 50-200 mg twice daily;atenolol 50-200 mg/day
    c) Nitrates: nitroglycerin sublingually for immediate relief; isosorbide dinitrate 2.5-10 mg/day sublingually or 5-80 mg/day orally; isosorbide-5-

mononitrate 20mg twice dailyorally. A nitrate free interval of 12-16 hours is essential to prevent nitrate tolerance.

d) Calcium channel blockers are to be used if beta blockers are contraindicated –asthma/COPD; fatigue, sexual dysfunction or depression with beta blockers

4. Revasularisation either catheter technique (PTCA – Percutaneous Coronary Angioplasty) or surgically (CABG- Coronary Artery Bypass Graft) should be offered as an option as the symptoms may not correlate with the extent of the block. An aggressive approach need to be recommended as a routine to all patients. As a general approach, PTCA is advised in those with 1 or 2 vessel disease or in those with coronary disease and left ventricular dysfunction.

Acute Coronary Syndromes:

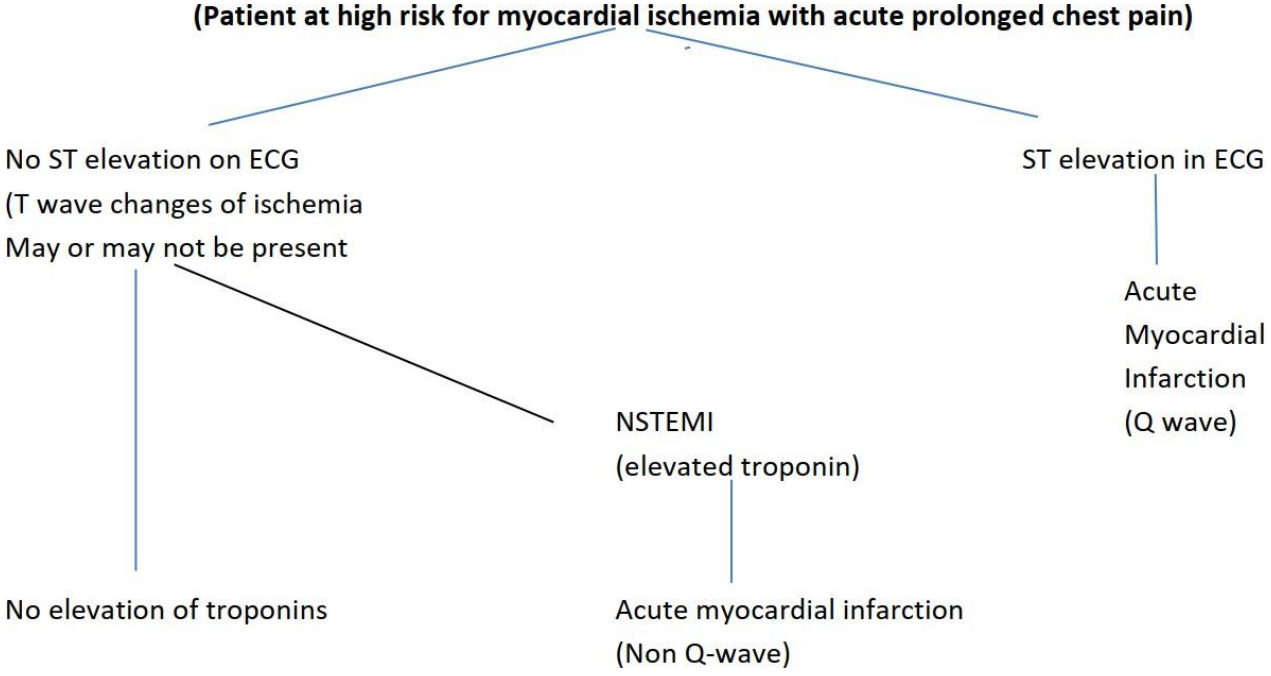
This group includes a number of syndromes involving acute chest pain of cardiac origin:

1. Unstable angina
2. Non-ST elevation myocardial infarction (NSTEMI)
3. ST elevation myocardial infarction (STEMI)

CLASSIFICATION

Acute coronary syndrome (ACS)

(Patient at high risk for myocardial ischemia with acute prolonged chest pain)



Unstable angina

Unstable angina refers to rapid aggravation of symptoms as manifested by more sever/more frequent/ more prolonged anginal pain (20 minutes), occurring at a reduced level of exercise or at rest.

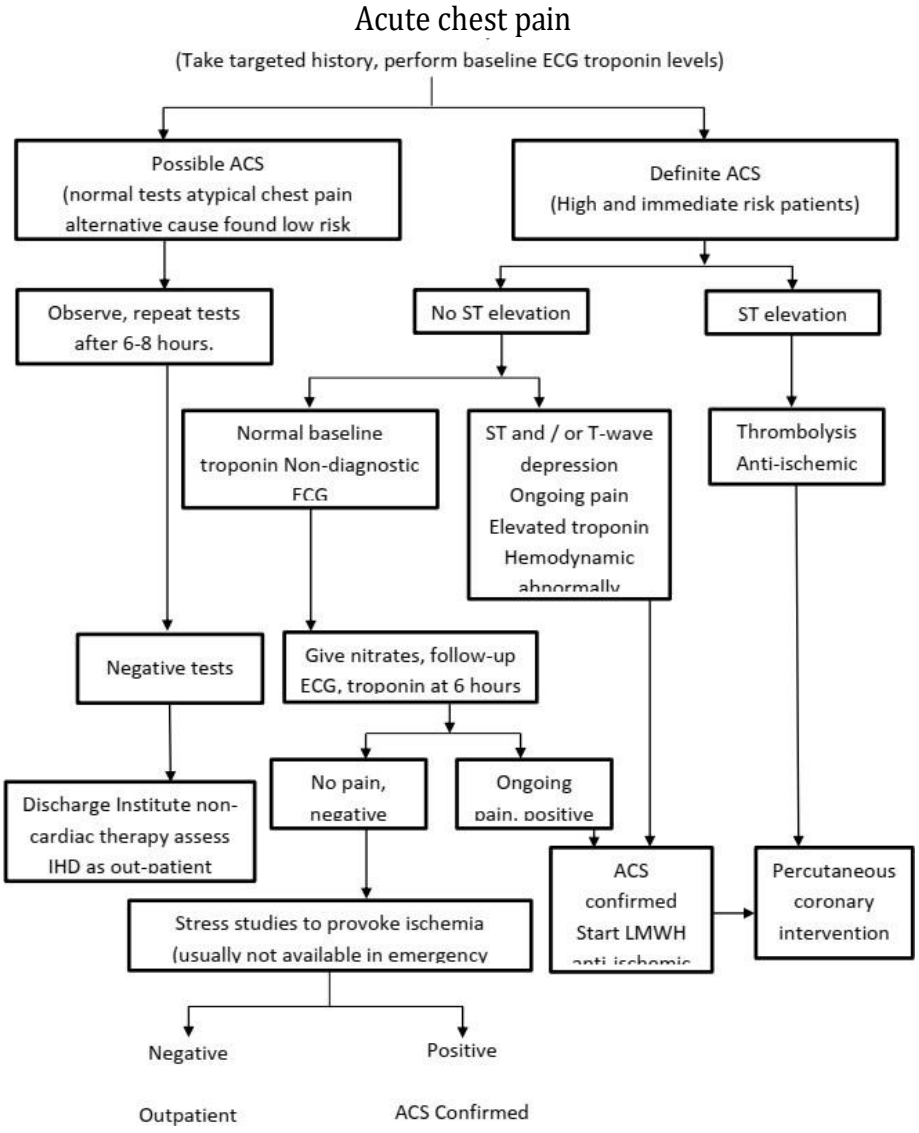
Non ST elevation MI is often in distinguishable from unstable angina clinically and by ECG; an elevated troponin level is the distinguishing feature. The reason for delineating the two states is that percutaneous coronary interventional procedures to improve myocardial perfusion, and newer anti-platelet drugs (glycoprotein llb/llla inhibitors) have been shown to be beneficial in those with elevated troponins. Such patients should be seen by a cardiologists for consideration of emergency intervention.

Risk stratification of chest pain patients

High risk of ACS:
Typical angina
Old MI, known CAD
CHF
New ECG changes, esp LBBB

Intermediate risk of ACS
Age>70 years
Male
Diabetes
Old PVD, CVA

CLINICAL APPROACH ACS



Follow up

LMWH : Low Moledular Weight Heparin

Management:

In contrast to Acute Myocardial Infarction, thrombolysis is NOT indicated in this group. The reasonwhy thrombolysis is not useful in this group of patients is not clear but may be related to the fact that thrombolysis activates platelets and can stimulate ongoing thrombus formation- these factorsmay be more important in this group of patients.

The management of these patients includes the use of:

1. Nitrates-intravenous (start at 10ug/mt and titrate) followed by oral

2. Beta blockers – intravenous followed by oral. The dose is to be titrated to reach a resting heart rate of 50-60/min. Contraindicated if there is bradycardia, hypotension, AV Block, leftventricular failure or significant bronchospasm.
3. Calcium channel blockers are useful primarily for patients with vasospastic angina. In the absence of beta blockers as it can increase the risk of a myocardial infarct. Dihydropyridine calcium channel blockers (DHPCC- nifedipine, amlodipine) if used without a beta blockers cause reflex tachycardia and can precipitate ischemia. The non DHPCC blockers (verapamil,ditiazem) can be used and should not be combined with a beta blocker.
4. Antiplatelet drugs- Aspirin/ Clopidogrel
5. Antithrombotic drugs

   Heparin- conventional or low molecular weight for 5-8 days

   Enoxaparin 1mg/kg twice daily

   Dalteparin 120 IU kg twice daily

   Conventional Heparin – to keep APTT 1.5-2.5 times the control

   These are to be continued till symptoms have stabilized for 55 days or till a revascularizationprocedure is done.

Acute Myocardial Infarction:

The conventional classification of myocardial infarcts into transmural and non-transmural based on the presence or absence of Q waves is obsolete as the Q waves do not correlate with a transmural infarct. Abnormal Q waves (at least 30ms wide, 0.2m Vdeep occurring in at least 2 contiguous leads)are specific for a myocardial infarct but are not sensitive as they are present in <50% of those with an acute myocardial infarction. The others have significant ST changes (elevation/depression of ≥0.10 mV measured 0.02ms after the J point in 2 contiguous leads), T wave changes or no ECG changes- these form the non Q wave myocardial infarcts. The pathophysiological difference betweenthe two is not well understood. The early events in a both types include plaque disruption, thrombusformation and vasospasm. It is postulated that in a non Q infarct, there is early spontaneous reperfusion due to lack of sustained vasoconstriction while in the Q wave type of infarct, sustained occlusion causes necrosis of the myocardium.

A summary of the differences of the 2 types is as follows:

	Q wave	**non Q wave**
Prevalence	47%	53%
% Coronary occlusion	80-90%	15-25%
ST depression	20%	75%
Treatment		

Thrombolysis	YES	NO
Beta blockers	YES	?
CCB		
Nifedipine	NO	?
Verapamil	YES	?
Ditiazem	NO	YES

The basic difference in the approach to thetwo patterns of presentation is that in a ST elevated MI(STEMI), thrombolysis or PTCA must be instituted as soon as possible in order to reperfuse rapidlyand salvage myocardium whereas in a non ST elevated MI, (non STEMI), as in unstable angina, thefocus is on antithrombotic therapy and plaque stabilization failing which percutaneous revascularization may be done.

It is to be noted that if cost, availability of technology and skilled personnel are not limiting factors, urgent percutaneous revascularization is the first therapeutic option in any myocardial injury even ifthe patient is in cardiogenic shock.

ACUTE MYOCARDIAL INFARCTION (AMI)

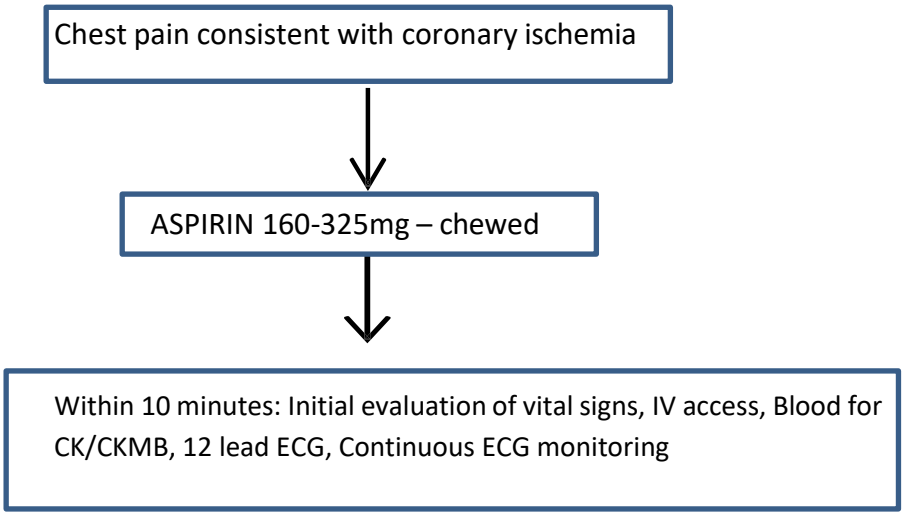
DIAGNOSTIC CRITERIA:- ANY 2 of following

- Typical chest pain
- ECG criteria
- Enzymatic criteria

Usually patient presents with "typical" chest pain.

- PLACE: Reosternal or precordial
- QUALITY: Pressure, aching, squeezing, discomfort, crushing, swelling, bursting
- RADIATION: May be skip areas with restrosternal pain, or pain only in the referral areas. e.g.

  LAD-L arm/both arms
  RC-jaw/neck
  CIRCUMFLEX- inbetween scapulae
- SYMPTOMS: Dysnoea, diaphoresis, nausea, vomiting, apprehension, syncope, agitation,palpitation (arrhythmias)
- TIME: 3-30 mins. Infarction > 15 mins (prolonged). Can suddenly decrease with reperfusion.



Clinical diagnosis

Right Ventricular Infarct :
High JVP with clear lung fields
Kussmaul's sign present

ECG evidence of inferoposterior

infarct, 'ST' depression in V1 and V2; elevation in V4, aVR

AVOID : Nitroglycerine and frusemide. Increase intravascular volume, Consider Dobutamine (2-10ug/kg/min).

Left ventricular Infarct :

Lung base crepitations
ECG evidence of anterolateral and anterior infarct, 'ST' depression in V4-V6

S/L Nitroglycerine 0.3-0.6 mg, repeat if BP (systolic) > 120 mm of Hg. Consider Beta-blocker (IV 2 doses followed by oral) therapy if HR> 100 with systolic BP> 110 mm of Hg. (Atenolol IV 5 mg over 5 mins, repeat, 10 mins later. 10 mins after the IV dose, start 50 mg/d of the

General measures: Propped up, Oxygen by mask, If in pulmonary edema, Morphine 5mg S/C or IV 3-5mg over 1 min, repeated at the rate of 2-5mg every 5-30 mins.

Therapeutic/diagnostic tracking according to 12 lead ECG +/- enzyme results

Normal/non-Diagnostic ECG

Consider Differentails

Keep in **casualty.**

Repeat **ECG 4-5h later.** (Usually within 4-12h ECG changes tend to appear) Review with **enzyme assays. Serial** serum marker levels.

ECG suggestive if ischemia-T wave inversion, ST depression.

Anti-ischemic therapy. Analgesia)Morphine S/C, IV) **CARDIOLOGY**Referral for expert care in **CCU**

ST segment elevation (>1mm in 2 cont.LL, or >2mm in 2 cont. chest or new BBB (more specific forAMI)

No **Contraindications**for thrombolysis (box no.4)

Thrombolysis<12h of onset/30 min of arrival at ED.

Primary PTCA if thrombolysis CI/in cardiogenic shock and possibly florid pulmonary edema.

ENZYMATIC CRITERIA FOR DIAGNOSIS OF ACUTE MI/NSTEMI

1. Troponin I > 1ng/ml check serial values at 4-6 hours
2. Serial increase and then decrease of CKMB values, with a change of >25% betweenany two values.
3. CKMB> 10-13 U/L (+/- 7 is considered as lab error, therefore > 21 is considered asdiagnostic in our lab) or >6% total CK activity.
4. Increase in CKMB values >50% between any two values separated by 4h.
5. If only a single sample, two fold increase over the normal value.

Suspect a false elevation if

1. Presence of a skeletal muscle disease/trauma
2. An atypical time course of increase and decrease of the plasma CKMB levels e.g more prolonged.
3. MBCK <5% of total CK activity (probable skeletal muscle source tongue, diaghragmetc. are relatively rich in CKMB)
4. A marked elevation of CK 20-30 folds.

Indications for thrombolytic therapy :

1. Patients irrespective of age seen within 12h of an AMI with ECG criteria for the same(i.e ST segment elevation > 1mm in 2 contiguous Limb Leads or >2mm in 2 contiguous chest Leads.)
2. New LBBB seen within 6h or chest pain.
3. New RBBB, proven acute infarction, associated heart failure.
4. Seen within 12h with ongoing ischemia, pain in sluttering episodes in the presence ofST segment elevation.

Contraindications to thrombolytic therapy

Absolute :

1. Altered consciousness.
2. Active internal bleeding.
3. Known CNS AV malformation/tumor.
4. Recent head trauma.
5. Known previous haemorrhagic stroke.
6. Intracranial/Intraspinal surgery within the past 2 months.
7. BP persistently higher than 200/120mm of Hg.
8. Known bleeding disorder.
9. Pregnancy
10. Suspected aortic dissection.
11. Previous allergy to Streptokinase.
12. Major trauma/surgery within 2 weeks that can bleed into a closed place.

Relative :

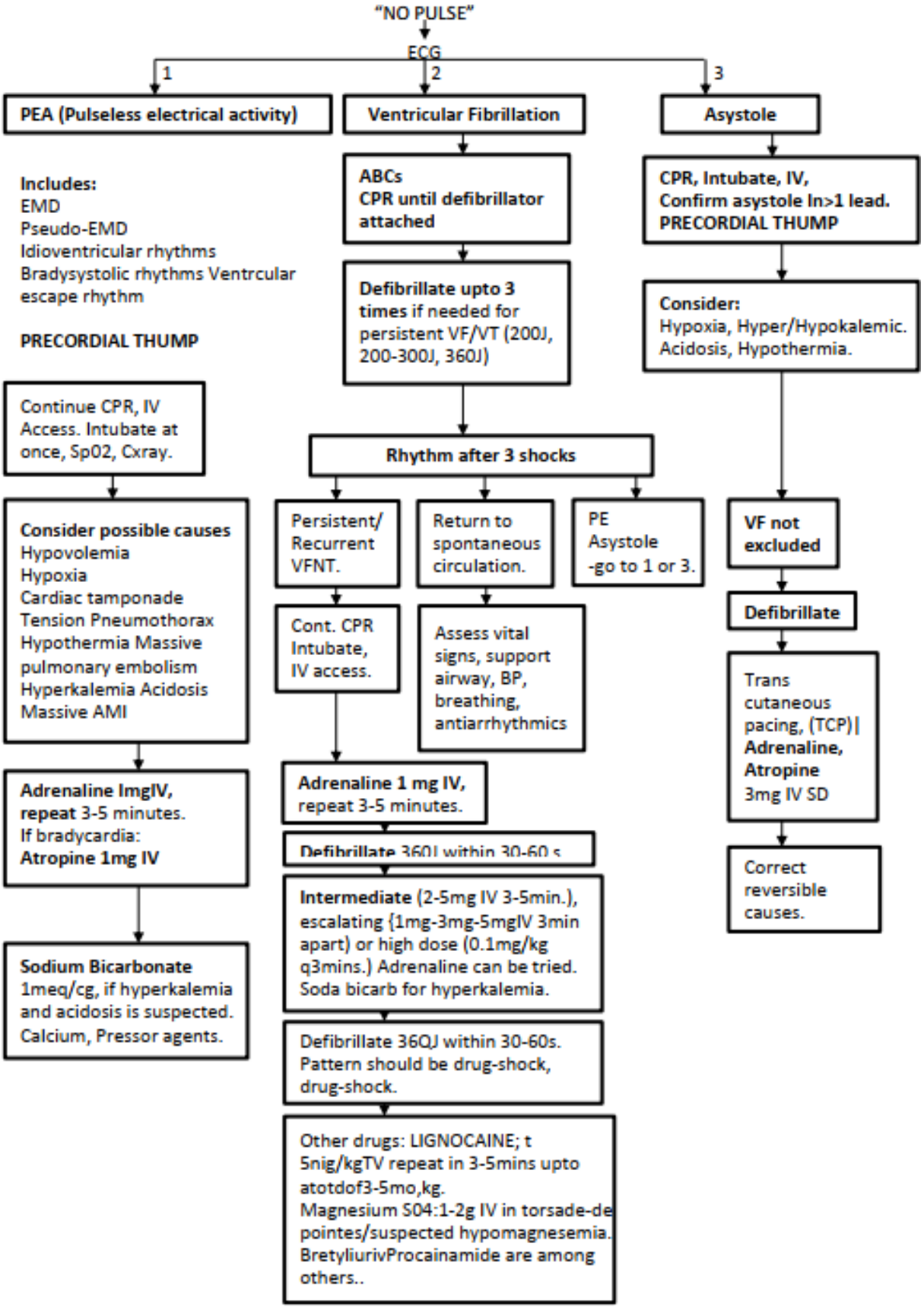
1. Active peptic ulcer disease.
2. H/o ischemic/embolic stroke.
3. Current use of anticoagulants.
4. Major trauma/surgery>2 weeks and <2months.
5. H/O chronic uncontrolled HT (treated/untreated)
6. Subclavian/IJV cannulation.

Thrombolysis regimens :
Streptokinase :1.5 million units in 500ml of 5% Dextrose/100ml of 0.9% Saline IV over 30-60 mins. (Cost=Rs. 2500-3500/=)

Anistreplase(APSAC) 30U in 5ml sterile water/saline by slow IV bolus over 2-5 mins.

tPA(Alteplase) : 15mg bolus; 0.75 mg/kg over 30 mins (max 50 mg); 0.5 mg/kg over 30 mins(max.35mg). Total dose </=100mg. (Cost =Rs 1.37.500/=)
The t-PA regimen quoted is called FRONT LOADING regimen(GUSTO TRIAL). Higher early patency rate.

23 CARDIAC ARREST



24 GASTROINTESTINAL & HEPATIC DISORDER

DIARRHOEAS

AN APPROACH ACUTE ONSET

< 3 DAYS DURATION

COMMONEST CAUSE :-VIRAL, VIBRIO

Management guidelines :-

- Stool hanging for Vibrio cholera
- Cap. Doxycycline 300mg stat.
- Fluid correction for Volume loss, Oral or IV.
- The **best guidelines for replacement therapy** in adults :- Mild hydration – THIRST
- Severe dehydration – CVP (Central Venous Pressure), Urine output
- Correction of electrolye imbalances.
- Urine output to be monitored and maintained .

Whatever the etiology, the investigations and management remains unaltered except if they arein HIGH RISK category e.g:-

1. Recent broad spectrum antibiotic use (suspect pseudomembranous colitis, performSigmoidoscopy, Rx. Metronidazole/Vancomycin oral)
2. HIV associated
3. Immunosupressed patient

OR

They have fever, bloody dysentery, tenesmus and abdominal pain, inflammatory type of diarrhea.(Stool routine shows WBCs, RBCs, C/S for Shigella, Salmonella, Campylobacter, Yersinia.)

DYSENTERY- BLOOD AND MUCUS DIARRHOEA

Investigations :-Stool C/S and stool for ova/ cyst/ parasites

Treatment :-

For Amoebic dysentery

Tab Metronidazole 400-800 mg tid x 7-10d/ Tinidazole 2g/daysx3 days with Diloxanidefuroate 500 mg q8hrly for 10 days to clear luminal cysts. Stool should be reexamined in 4 weeks time.

Amoebic liver abscess- Total duration of therapy with the above drugs for 3 weeks. Chloroquine can be added to Metronidazole at a dosage of 150 mg base q6hrly for 2days followed by 150 mg base q12hrly.

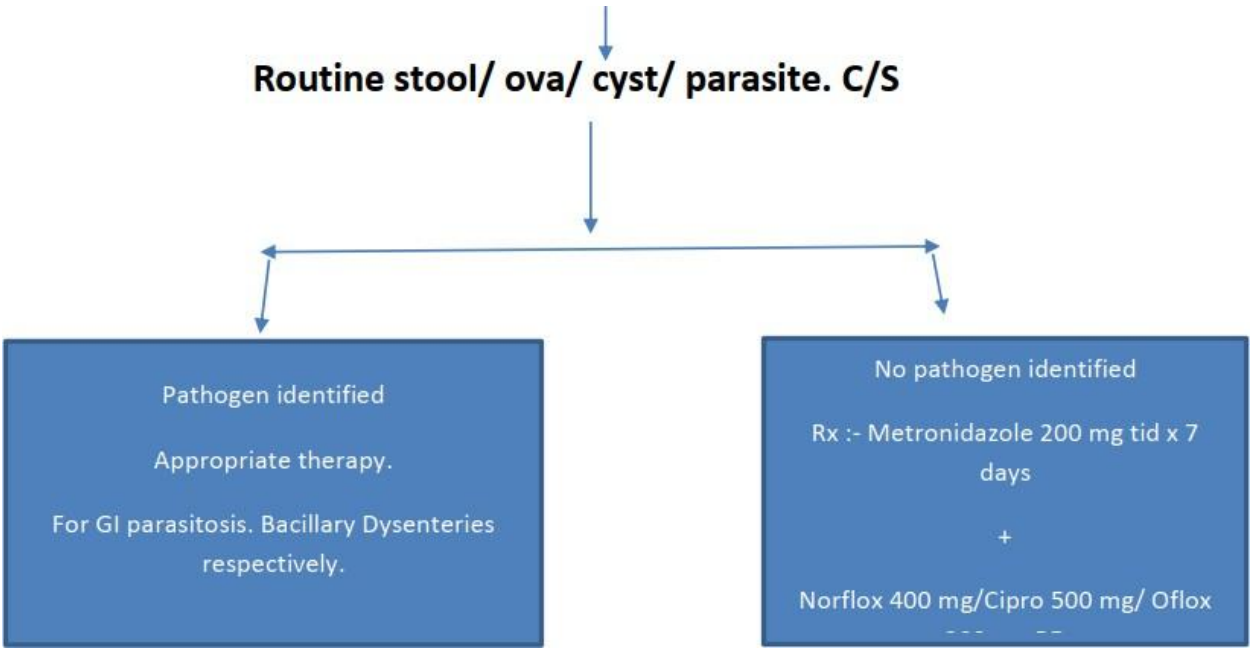
(Entamizole forte= 500 mg of Diloxanidefuroate and 400 mg of Metronidazole). Ittakes a few weeks of ultrasound findings in the liver to return back to normal.

Therefore, duration of therapy should be guided by clinical response and not radiological findings.

For bacillary dysentery

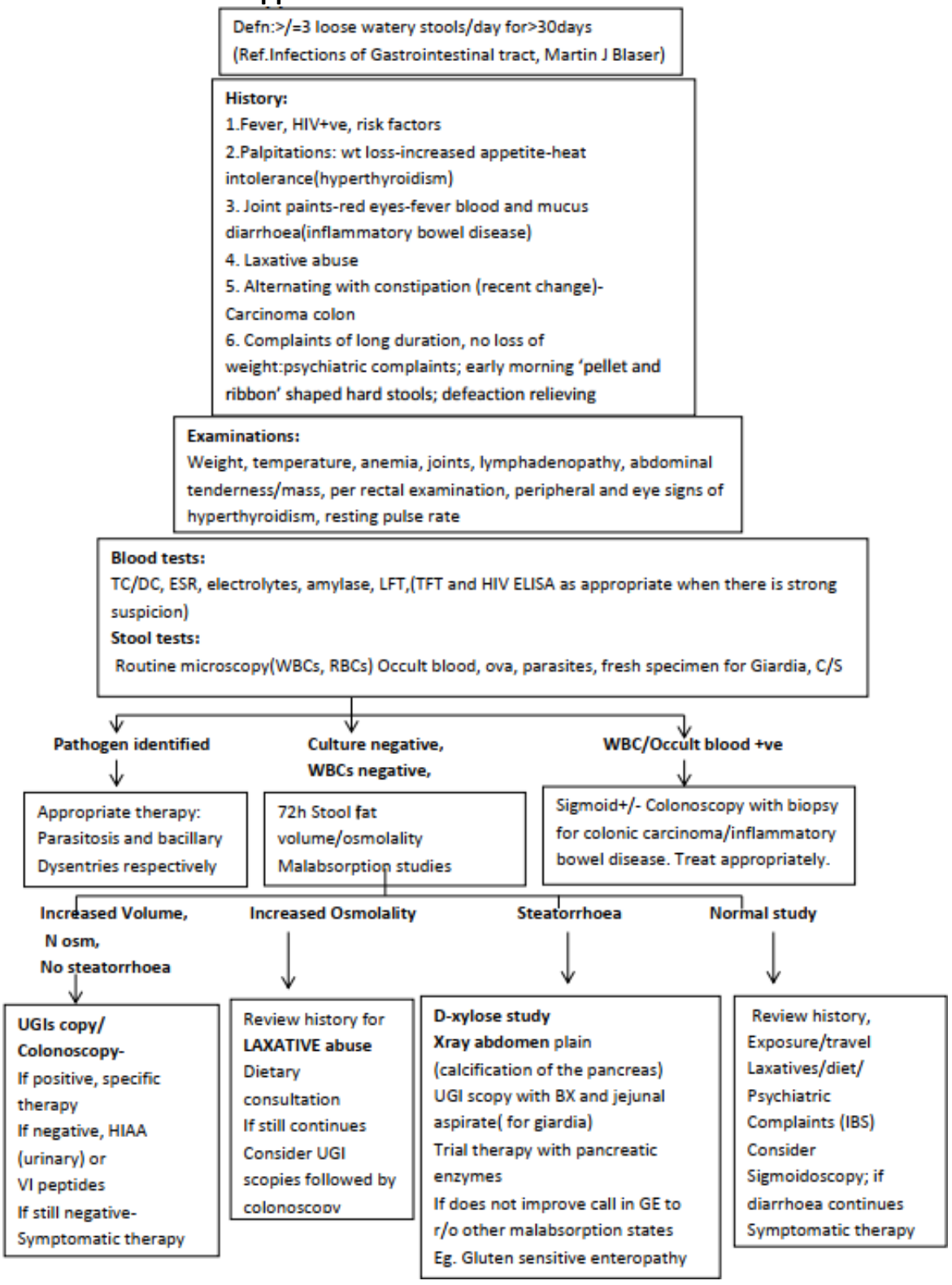
Ciprofloxacin 500 mg BD/ Norfloxacin 400 mg BD 1 week
Symptomatic therapy with syrup Codeine 30 ml tid or Tab. Loperamide 4mg initially followed by 2 mg stool after each stool (Maximum of 16 mg/day)

PROLONGED DIARRHOEAS



>3 Days but falling short of ' CHRONICITY'

Approach to CHRONIC Diarrhoeas.



***Stool fat in a 72h collection-** Patient has to be on a high(50gm) fat diet for three days prior to thestarting of collection and continue throughout the collection. Medications like pancreatic enzymesdigestives, etc should be withheld during the test.

25 **GASTROINTESTINAL BLEEDING OBVIOUS**

Hematemesis – Vomiting of blood.

Colour of vomitus depends on the concentration of HCl.

Hematemesis usually indicates bleeding **proximal to the ligament of Treitz,** because bleeding from the GI tract beyond that, rarely enters the stomach. While bleeding sufficiently enough to producehaematemesis, it is bound to produce melena; less than half of the patients with melena, have haematemesis.

Melena – Black tarry 'sticky' stools.

Usually denotes bleeding from esophagus, stomach and duodenum. However bleeding in the GItract down to the ascending colon can produce melena, provided the transit time has been adequate. Blood loss of upto 60 ml can cause a single episode of black stools. Less than 60 ml ofblood loss cause melena for 7 days.

History & Examination

- Ascesshaemodynamic status
- Aspirin, NSAID use
- Alcohol
- Retching, vomiting
- Other sites of bleed
- Telaglectasia – skin & oral mucosa

Estimating the amount of bleed – history and physical examination:-

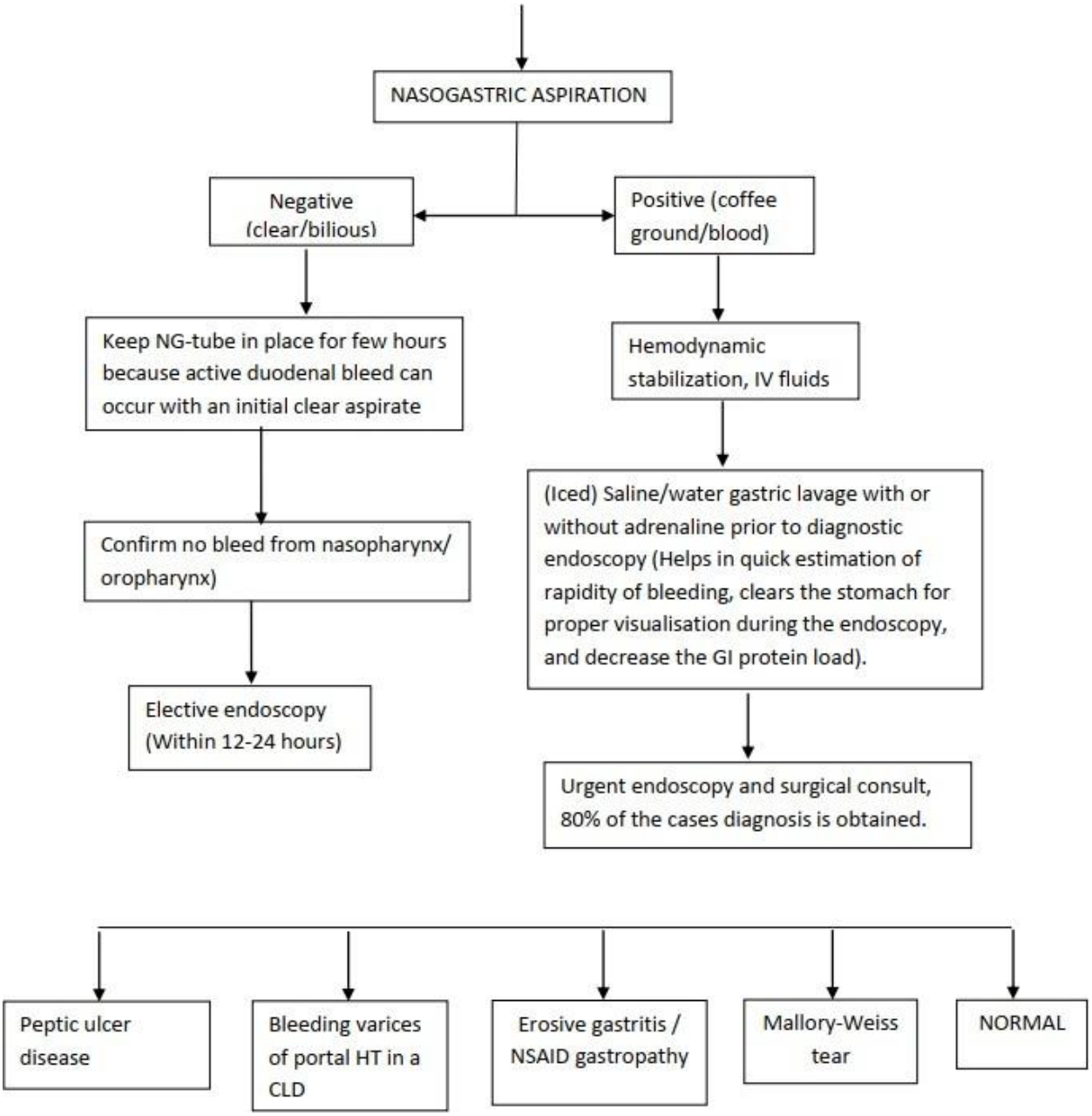
<500 ml (10% of blood volume) loss- Usually no signs or symptoms. Exceptions are elderly oranaemic patients.
20% blood loss- Gives rise to orthostatic hypotension of 10mm of Hg. Concomitant symptoms include nausea, syncope, light-headedness, sweating and thirst.
25-40% blood volume loss- Can cause profound shock with thready pulse, tachycardia, hypotension, prominent pallor and cold-clammy peripheries.

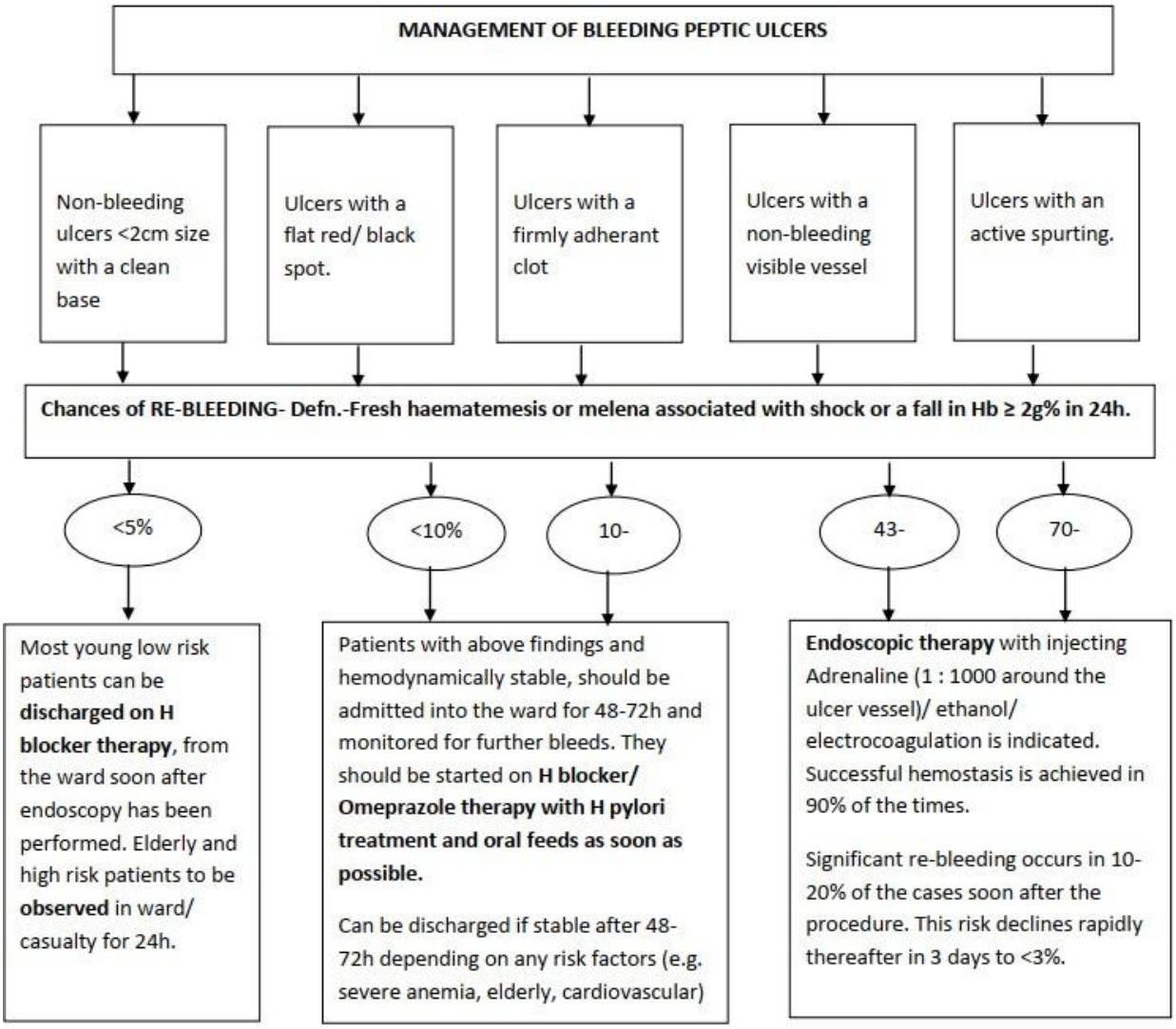
Investigating a GI bleed :-

Hematocrit- takes 8h to fall by dilutional process. Therefore may not be a good indicator of the amount of bleed. Leukocytosis and thrombocytosis occur with 6h of bleed. Urea rises disproportionately to the Creatinine (because of GI absorption of products of digested blood)

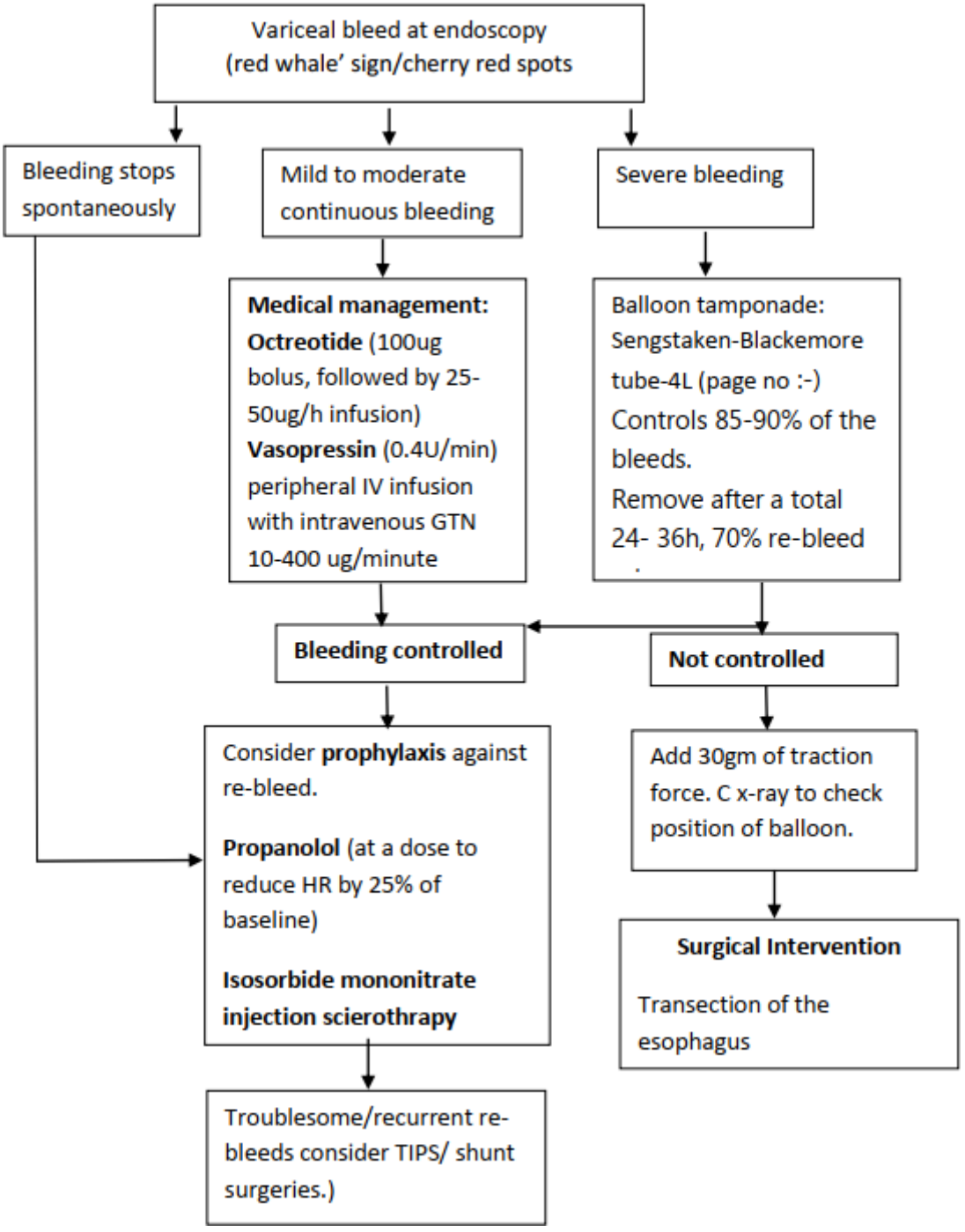
Blood for grouping and cross-matching two pints of blood. If stigmata of CLD are present, LFT, PT,PTT should also be done.

GUIDELINES TO MANAGEMENT OF ACUTE UPPER-GI BLEED




MANAGEMENT OF BLEEDING PEPTIC ULCERS
Non-bleeding ulcers <2cm size with a clean base
Ulcers with a flat red/ black spot.
Ulcers with a firmly adherant clot
Ulcers with a non-bleeding visible vessel
Ulcers with an active spurting.
Chances of RE-BLEEDING- Defn.-Fresh haematemesis or melena associated with shock or a fall in Hb ≥ 2g% in 24h.
<5%
<10%
10-
43-
70-
Most young low risk patients can be discharged on H blocker therapy, from the ward soon after endoscopy has been performed. Elderly and high risk patients to be observed in ward/ casualty for 24h.
Patients with above findings and hemodynamically stable, should be admitted into the ward for 48-72h and monitored for further bleeds. They should be started on H blocker/ Omeprazole therapy with H pylori treatment and oral feeds as soon as possible.
Can be discharged if stable after 48-72h depending on any risk factors (e.g. severe anemia, elderly, cardiovascular)
Endoscopic therapy with injecting Adrenaline (1 : 1000 around the ulcer vessel)/ ethanol/ electrocoagulation is indicated. Successful hemostasis is achieved in 90% of the times.
Significant re-bleeding occurs in 10-20% of the cases soon after the procedure. This risk declines rapidly thereafter in 3 days to <3%.
It is better to admit all patients with GI bleed as IP for evaluation.

MANAGEMENT OF BLEEDING ESOPHAGEAL/ GASTRIC VARICES



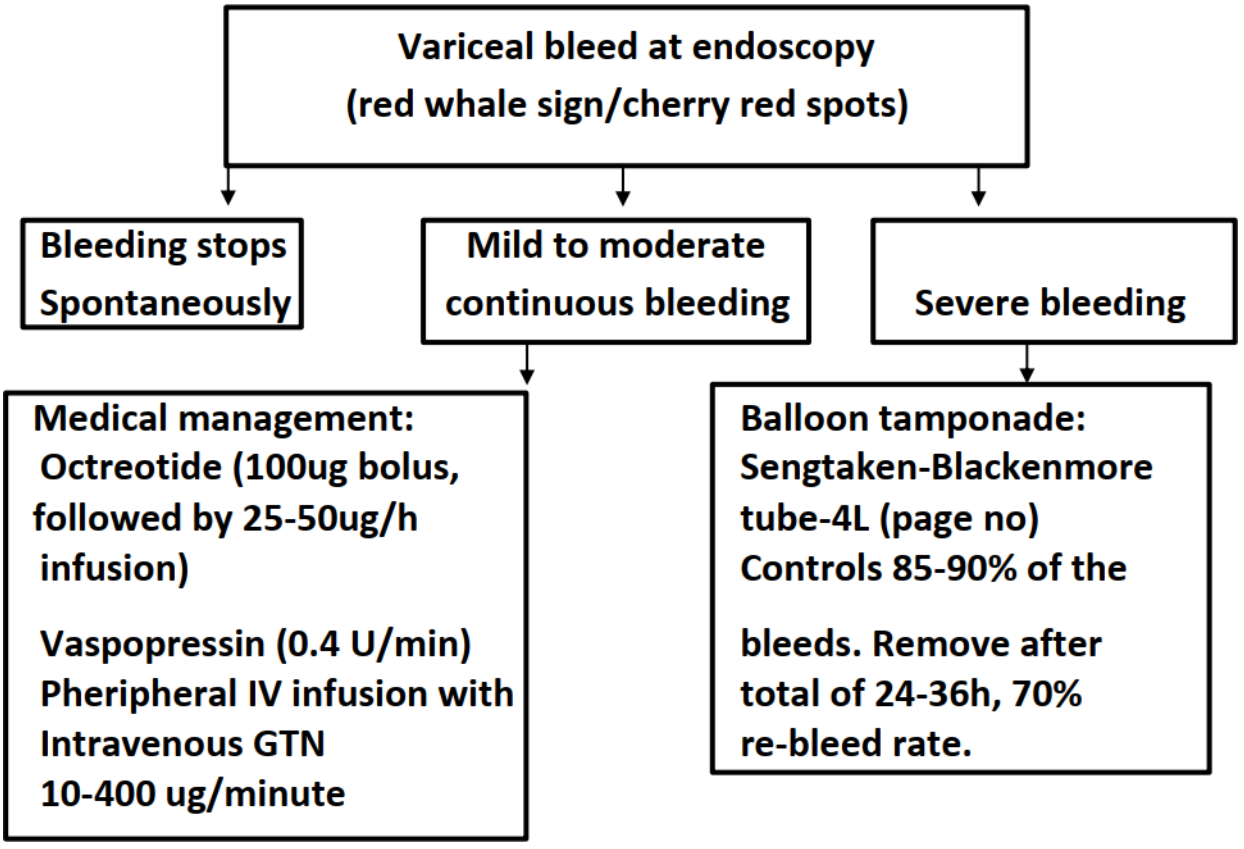
Remember :

Patients with varices and portal HT may be in 20-56% of the cases bleed from a non-varicealsource.

Independent Clinical/ Laboratory risk factors associated with non-variceal acute upper GI bleed

- Age more than 60 years
- Severe medical/ surgical co-morbid conditions
- Inpatient hemorrhage (admitted for some other medical/surgical condition)
- Persistanthaematemesis, hematochezia, red NG aspirate
- Persistant hypotension/shock
- Transfusion of >6U of RBCs required during one episode of bleeding
- Re-bleed from the same lesion
- Severe, co-existing coagulation/platelet disorder.

MANAGEEMENT OF BLEEDING ESOPHAGEAL/ GASTRIC VARICES



Method of insertion and removal of Sengstaken-Blakemore tube:

1. Empty stomach by suction at endoscopy.
2. Pass down a cooled, 4 lumen SB-tube (Cooling the tube stiffens it and allows easy passage ofthe tube without curling in the nasopharynx.
3. Maintain oral suction. Check position of tube in stomach by air injection and auscultationover the epigastrium.
4. Inject 500-200ml of air/saline into the gastric balloon. Withdraw till resistance felt againstthe gastric fundus. Should be 30-40 cm from the incisors. Tape the tube in place.
5. If bleeding continues, inflate the Esophageal balloon to 30-40 mm of Hg pressure with air.Double clamp the balloon tubes.
6. Maintain a constant low suction of 5-10mm of Hg at the esophageal luminal tube anddependant continuous drainage at the gastric luminal tube.
7. Check position of the tube with Chest X-Ray.
8. If bleeding continuous, question diagnosis, add a traction force of 30gm to the tube and takeX-Ray for confirming position of tube.
9. If at anytime respiratory distress arises, deflate both tubes completely and remove tube assoon as possible.
10. Remove tube after total of 24-36h or 12h after bleeding stops. Deflate the esophageal balloon first, if re-bleeding does not occur, remove tube after 12h, if re-bleed occurs, re-flatethe esophageal balloon. Chances of re-bleed after removal of SB-tube is very high (70%)

EROSIVE- GASTRITIS/ NSAID ABUSE

Most often settles with withdrawal of the offending drug and IV anti-secretory therapy withor without treatment for H.pylori. This is not amenable to endoscopic therapy.

MALLORY-WEISS tear

May or may not require endoscopic injection therapy.

NORMAL AT ENDOSCOPY

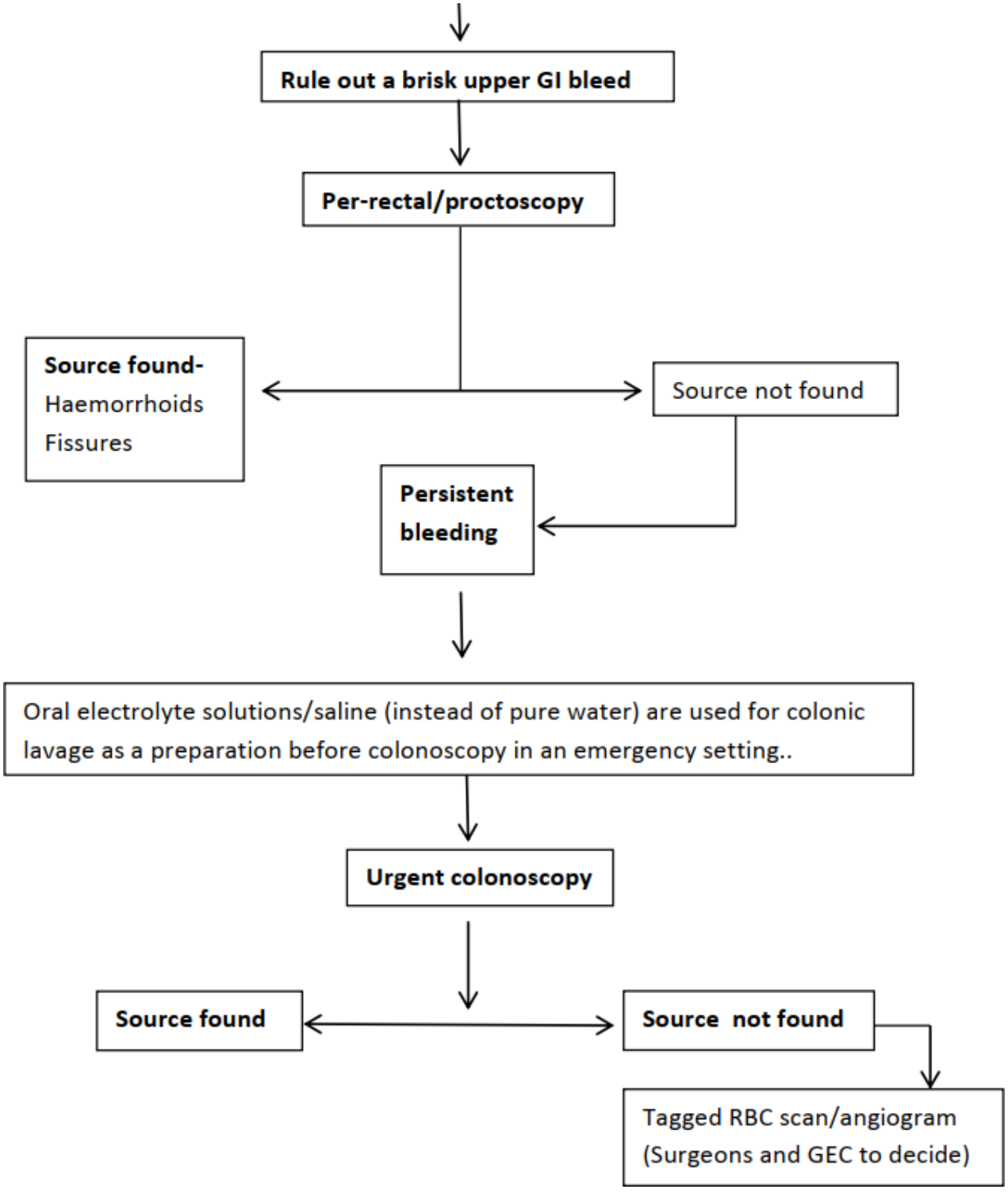
Visceral angiogram is indicated if there is active bleeding of >1ml/min in the absence of any structural lesion on endoscopy. For bleeding of lesser extent, colonoscopy is the best option;vascular malformations are the most common. For younger patients consider radio-nucleideTc99-sulphur scan for Meckels's diverticulum.

LOWER GI BLEEDING :-

Hematochezia – Fresh bleeding per rectum

Bleeding beyond the ligament of Treitzcan cause this. However brisk bleeding proximal tothis eg. Esophageal varices, can also give rise to hematochezia because of shortened GI transit time.

APPROACH TO BLEEDING FROM THE LOWER GI TRACT



OCCULT GI- BLEED

Occult GI bleeding :-Normal occult bleeding from GI tract is 0.5+/0.4 ml/day (maximum 1.5 ml/day).It does not vary with age or sex. GI bleedingupto 200ml/day can remain as occult.

Detected by 'hemospot/ 'GUAIAC' test in our laboratory which has a sensitivity of **0.5 ml/day**for colo-rectal bleeding and **10-20ml/day** for esphago-gastro-duodenal blood loss. The test detects hemoglobin peroxidase. False-negative test can arise due to ingestion of 500 mg of Vitamin C daily.Aspirin 80-325mg PO/day does not explain occult GI bleeding therefore is not a cause of false- positive result. Should be tested on a high fibre, low-meat die, off all NSAIDS and Vitamin C. Alwayssample 3 stools because of GI malignancies can bleed episodically. More than three sampling reduces the specificity of the test.

Stool for occult blood(SOB), being positive, is a matter of serious concern. It should be followed by an endoscopic evaluation for structural lesion in the GI tract. Patients on NSAIDS and Aspirin therapyare also not excluded from this because there are equal chances of GI malignancy in them and the general population if SOB is positive. (Daily Aspirin therapy is unlikely to cause SOB positive)

Other **investigations**to be carried out are, Haemoglobin, MCV, peripheral blood smear (tests foranemia evaluation). Stool for ova, cyst and parasites.

CAUSES OF OCCULT BLEEDING

AGE > 55 years :-

GI malignancy Inflammatory bowel disease Ischemic colitis Tuberculosis of the GI tract

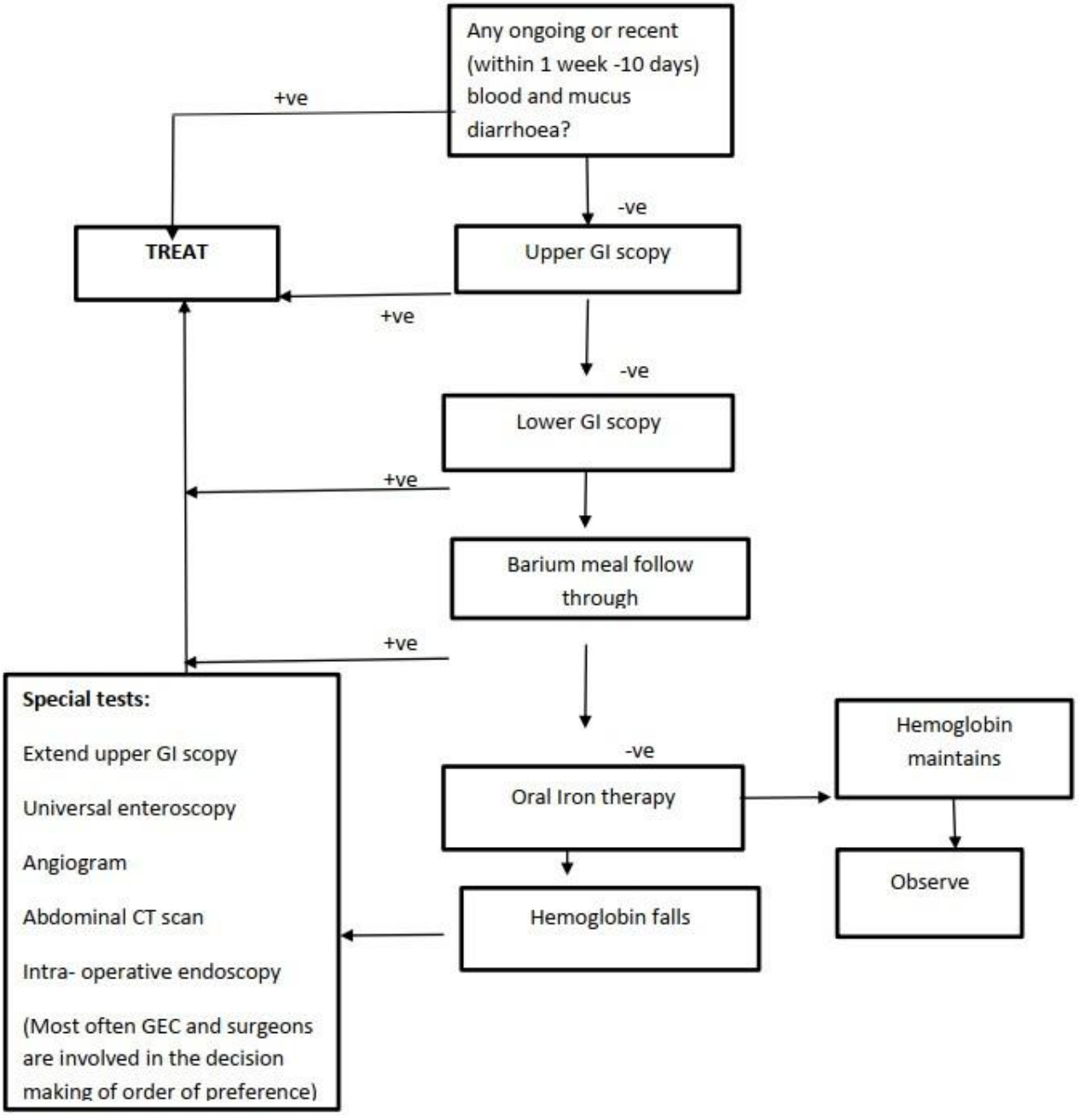
AGE < 55 years :-

Acid peptic disease
GI parasitosis (hookworms, ascaris)
Invasive diarrhoeas (for 1 week after the diarrhoea has settled clinically)

Inflammatory bowel disease (Ulcerative Colitis/ Crohn's)
Tuberculosis of the GI tract GI Malignancy/ polyps

Flow chart for investigating a case of occult GI bleed

(Please order investigations only after discussion with the consultant) Investigations can be discontinued at any stage when a possible cause for the SOB is detected.



SOB = stool occult blood

26 ACUTE RENAL FAILURE

Definition:- Abrupt onset decrease in urine output (<400 ml/day or <15 ml/h) with a concomitantincrease in serum creat. >1.5mg/dL.

Investigating an acute renal failure:- Urine

microscopy-

WBCs- UTI

RBCs- Glomerulonephritis

Eosinophils- Interstitial nephritis

S. Creatinine , Urea, S. electrolytes (potassium).

CVP line- to measure volume status

USG/KUB x-ray to look for kidney size/obstruction/hydronephrosis if symptoms are suggestive of thesame.

Pre-renal failure	Vs	Renal failure
Urea/ creat ratio > 40		Urea/creat ratio < 40
Urine spot sodium < 20 meq/L		>@) meq/L
Reversal of failure on treating		Absent
the pre-renal factor		
Urine Osmolality>600		Urine Osmolality <350

If POST-RENAL failure is suspected.....

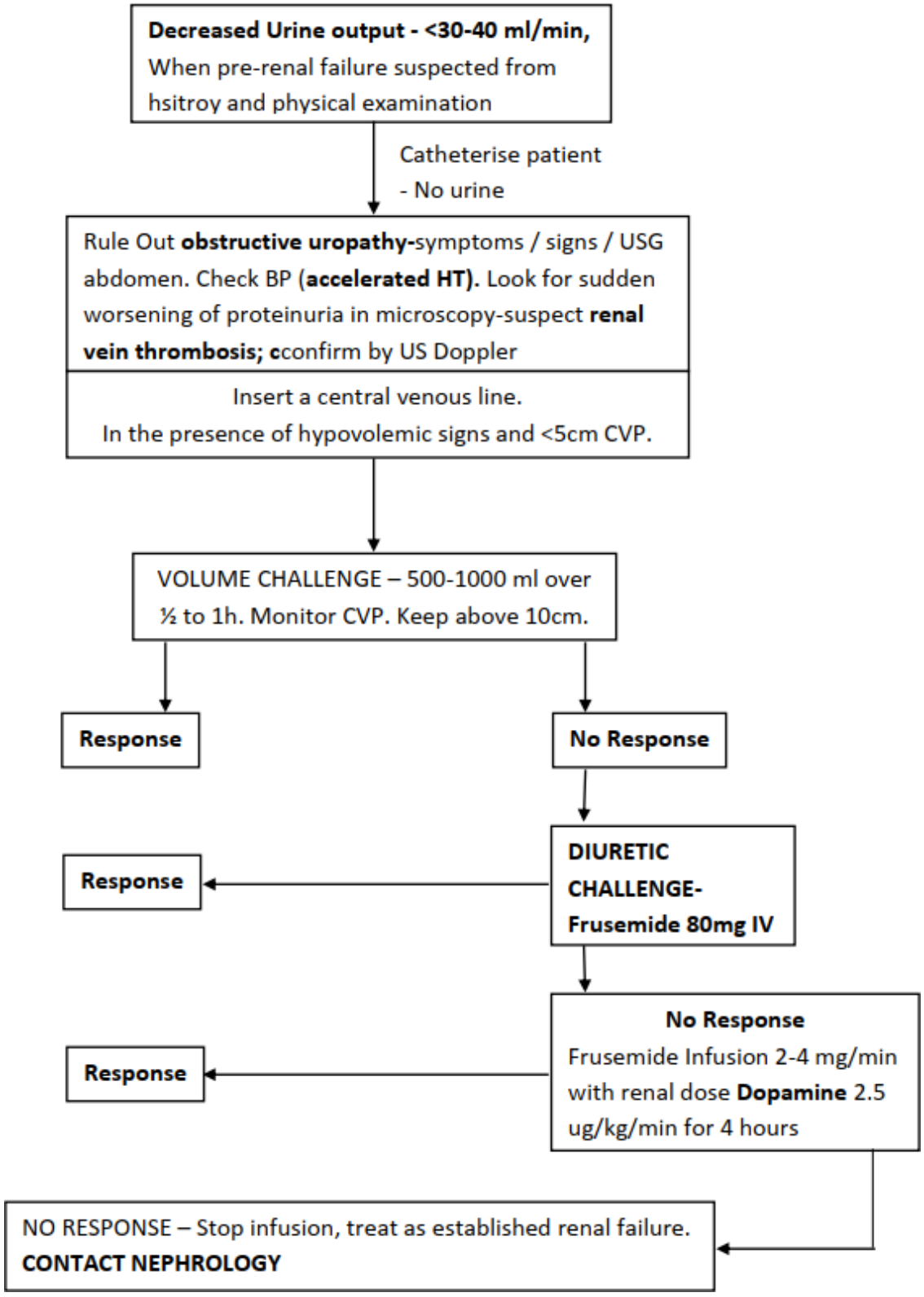
Catheterise distended bladder

Per-speculum and Per-vaginal examination to rule out carcinoma cervix in women. X-ray

KUB abdomen, USG abdomen to look for hydronephrosis and hydroureter.

Per-rectal examination to feel for prostatic enlargement.

Management of acute renal failure



Note : Low dose Dopamine has no renal protective action.
Lancet 2000; 356 : 2139 – 2143

Management of established renal failure :-

-Conservative

1. Monitoring – BP, HR, Urine output (catheterise), weight chart
2. Diet- No fruit juices, no added salt, restrict protein intake to 0.5g/kg.
3. Fluids- Restricted to Urine output + Insensible loss (500ml in winter, 750-1000ml insummer)
4. Frusemide to maintain urine output and control edema.
5. Control HT with CC Blockers and diuretics. (Avoid ACEIs)
6. Treat UTI vigorously modify drug dosage in renal failure.
7. Creatinine and electrolyte monitoring OD/alternate day.
8. Potassium and acid base correction.
9. Tab. Calcium carbonate 1g tid/ Tab. Sodium bicarbonate 650mg tid.
10. Look for absolute indications for initiation of dialysis & contact Nephrologist.

Acute-on-chronic renal failure

Rapid worsening of renal functions in a known case of CRF; causes same as ARF, but with underlyingCRF.

Common causes:-

Renal hypoperfusion:- Dehydration from diarrhoea and diuretics, CCF,Renal vascular disease,NSAIDs, ACEIs,systemic infections.

Obstruction and infection in the urinary tract- Papillary necrosis and sloughing, stones, cloth in theureter.

Metabolic and toxic- Hypercalcemia, hyperuricaemia, contrast media, Amino glycosides. Accelerated

phase of HT.

Renal vein thrombosis- Usually in a Nephrotic syndrome/Lupus nephritis.

Pregnancy- Reflux nephropathy.

In diabetes Mellitus- commonest are UTI (emphysematous pyelonephritis 40% mortality), Acutepapillary necrosis, Uncontrolled HT, Analgesic abuse/ radio contrast study/Aminoglycoside nephrotoxicity.

27. Cerebrovascular accident/Stroke Establish diagnosis

- Sudden Onset of focal neurological deficit, not due to seizure

DETERMINE THE CAUSE

EMBOLIC- source Artery to Artery/Cardia to Artery look for carotid bruit, cardiac murmer, signs of infective endocarditis, h/o MI (mural thrombus)H/O TIAs in the past (involving vascular territories)

THROMBOTIC --- H/O TIAs (involving the same territory should help usto rule in a diagnosis of Thrombotic stroke, but cannot rule out one in absence of the specific pattern.)

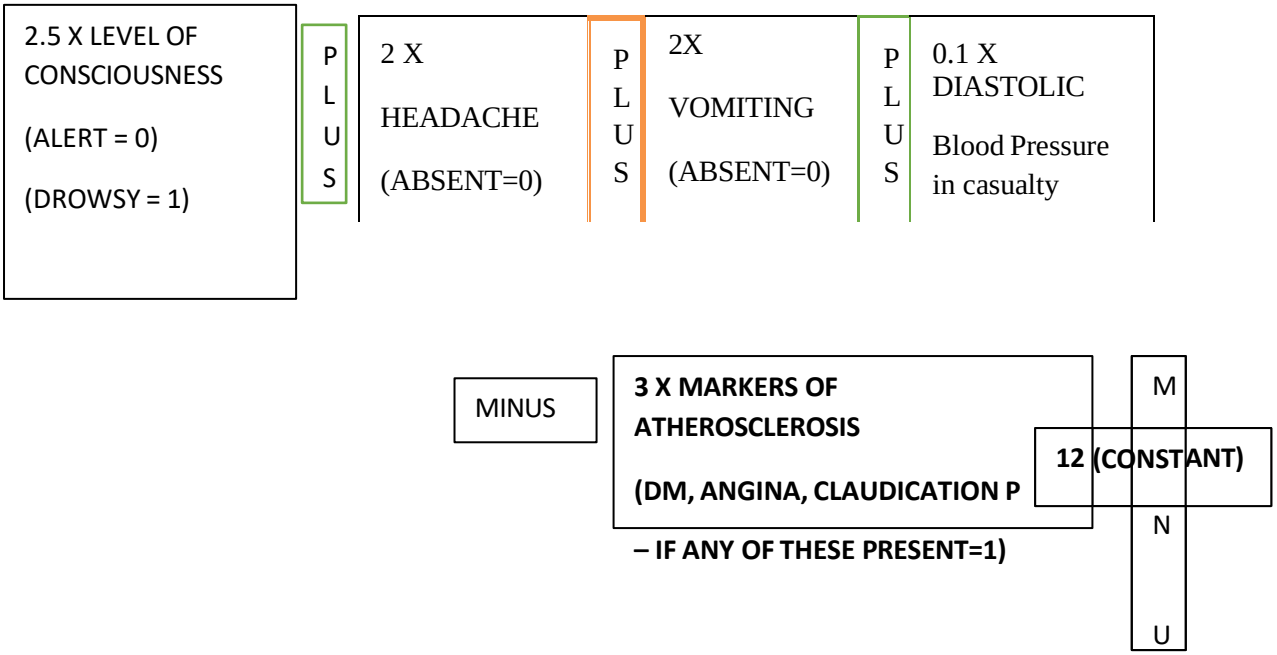
Step ladder progression of deficits (useful if present but absence doesnot rule in out) Look for risk factors DM, HT, Smoking, Hyperlipidemias
Look for other markers of atherosclerosis e.g., claudication pain, angina.

HAEMORRHAGIC :- SUB ARACHNOID OR INTRACEREBRAL :
Severe headache ("Thunderclap") , L.O.CSigns of rised I.C.T.
Neck Stiffness (if blood in ventricular system/subarachnoid space)

SIRIRAJ STROKE SCORE
ORIGINAL STROKE SCORE Adapted from : BMJ
1991 Jun 29 : 302 (6792) : 1665-7

Based on study done on south east Asian population : Diagnostic sensitivity for haemorrhagic stroke = 89.3 % Diagnostic sensitivity for non-hgic stroke = 93.3 % Overall predictive value = 90.3%
Studies done on **Indian population** show : Variable results (sensitivity of73-85)

Ref. JAPI – 1994 Apr; 42(4) : 302-3 ; 2000 June 48 (6) : 584-588



= TOTAL SCORE INTERPRETED AS

<-1= THROMBOTIC

>+1 HAEMORRHAGIC

+1 TO -1= EQUIVOCAL

The gold standard is still a CT scan.

IF YOUNG (<45 YRS) DIFFERENTIALS TO BE CONSIDERED

Rheumatic heart disease/Atrial fibrillation

Valve endocarditis

Vasculitis, Tb endarteritis

Subarachnoid haemorrhage (berry aneurysms associated with coarctation of aorta andpolycystic kidney disease)

AV malformations

Cortical venous thrombosis (pregnancy-postpartum)Bleeding diasthesis

INVESTIGATIONS

DO NOT GIVE ASPIRIN UNTIL HAEMORRHAGE IS RULED OUT OR IF IT IS AN EMBOLIC STROKE DUE TO SEPTIC EMBOLIC IN INFECTIVE ENDOCARDITIS.

IF CT SCAN NOT AVAILABLE…. GO BY SIRIRAJ STROKE SCORE. IF THE SCORE IS FAVOURING A THROMBOTIC PICTURE, GIVE FIRST DOSE OF ASPIRIN.

MANAGEMENT OF ACUTE STROKE :-

GENERAL- A-B-C approach to critically ill patients. Stop smoking/OCPs

SPECIFIC :- Ischemic- Infarct stroke

ANTIHYPERTENSIVES TO LOWER B.P

Do not bring down the B.P drastically… You may worsen cerebral ischemia bydecreasing the perfusion pressures.

Brain perfusion is completely dependent on Cerebral Perfusion Pressure (CCP= Mean Arterial Pressure- Intracranial Pressure). Raised BP in HT/ non HT, both fall sponstaneously and unpredictably within 24hrs/several days therefore, do not treat HF IF DBP<130mm oh Hg.

TIGHTER CONTROL OF SUGARS (hyperglycemia worsens ischemia)

A level of blood blood glucose around 150mg% (AC) and 200 mg% (PC) is acceptable. The need forInsulin/OHAs may decrease after the acute phase of stroke is over. Care should be taken not to cause hypoglycaemia- it will be an added insult to the ischaemic neurons!

LOWER BRAIN EDEMA/ ICT

How to determine the presence of raised ICT?

Look for HT/ bradycardia (Cushing's Reflex) in the presence of altered sensorium.

Dexamethasone, Antioxidants, Osmotic Diuretics are not of proven benefit. Can give I.V MANNITOL20% 100ml Q8 hly along with Oral Glycerol 30 ml Q6hly : slowly taper as sensorium improves.

ANTIPLATELET THERAPY IN ISCHEMIC (EMBOLIC/THROMBOTIC) STROKE ANTIPLATELET

DRUGS:

- ASPIRIN: 75-300 mg/day acts as prophylaxis against TIAs and reccurence of stroke.
- DIPYRIDAMOLE: 375 -400 mg/day in 3 divided doses. Has anti platelet action which isadditive to that of Aspirin.
- CLOPIDOEREL: 75 mg OD
- ANTICOAGULANTS are not indicated in acute stroke.
- THROMBOLYTICS are contraindicated in stroke.

Haemorrhagic

ANTIHYPERTENSIVES :-

Do not treat If SBP < 180mm of Hg/MAP < 110 mm of Hg Ref:

Sypnosis of intensive care medicine.)

AVOID ANTICOAGULANTS/ANTIPLATELET AGENTS.

REHABILITATIVE THERAPY :-

Good nursing care Position

change Q 2 Hrly Soft diets

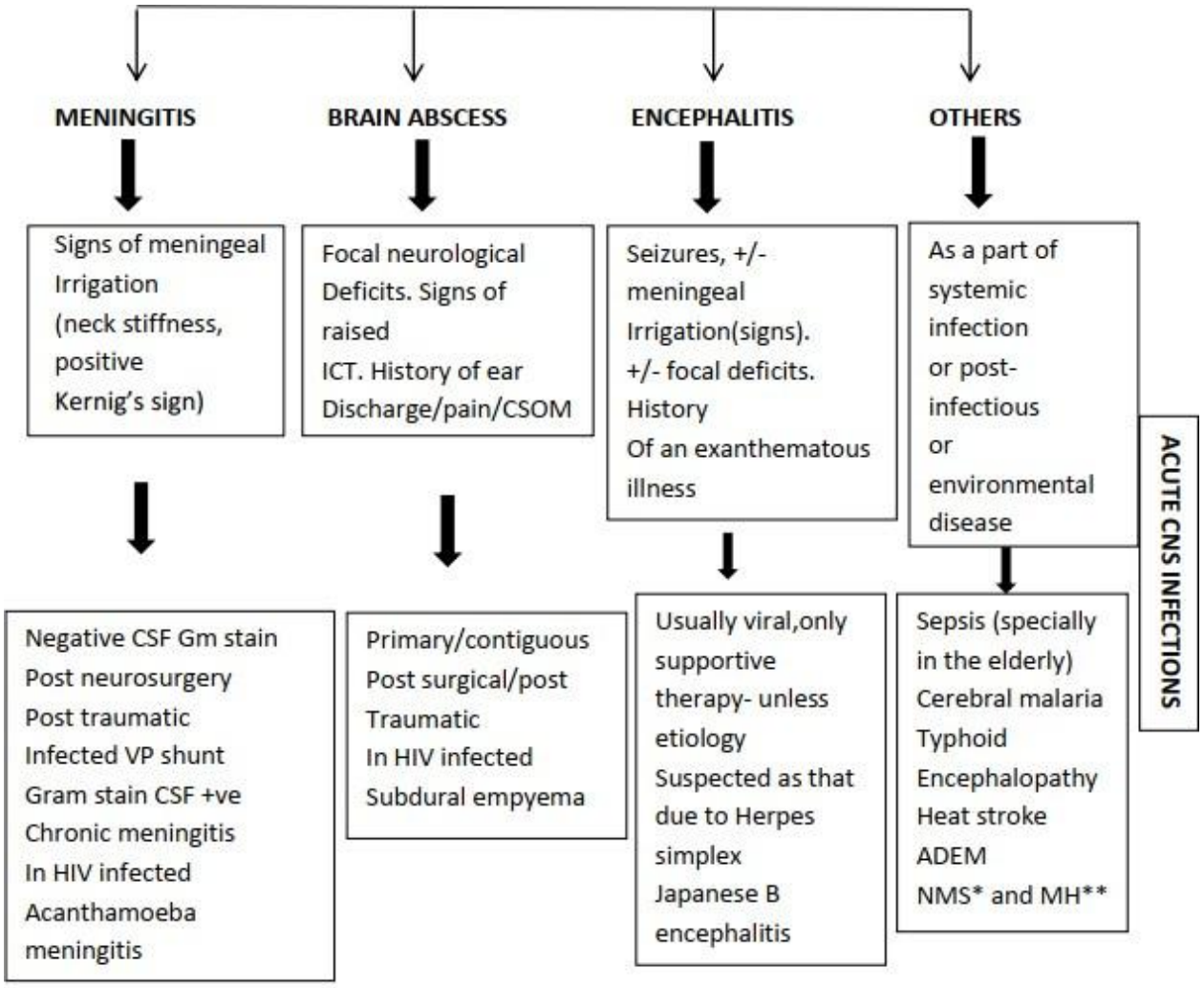
+/- Laxatives

Rehabilitative Therapy

Physio/Occupational Therapy wait for 2-3 days in case of haemorrhagic stroke; chances of re-bleed ishigh with strenuous activity.

28 Acute CNS infection

Differentials to be considered for a febrile patient with altered sensorium



Look up relevant sections for *NMS = Neuroleptic Malignant Syndrome
**MH = Malignant Hyperthermia

29 CONVULSIVE DISORDER (SEIZURES)

HISTORY TO EMPHASIZE UPON

Ask for EFFECTS of the seizures :- loss of consciousness, biting of tongue, injury to self, incontinence,postictal drowsiness/ confusion.

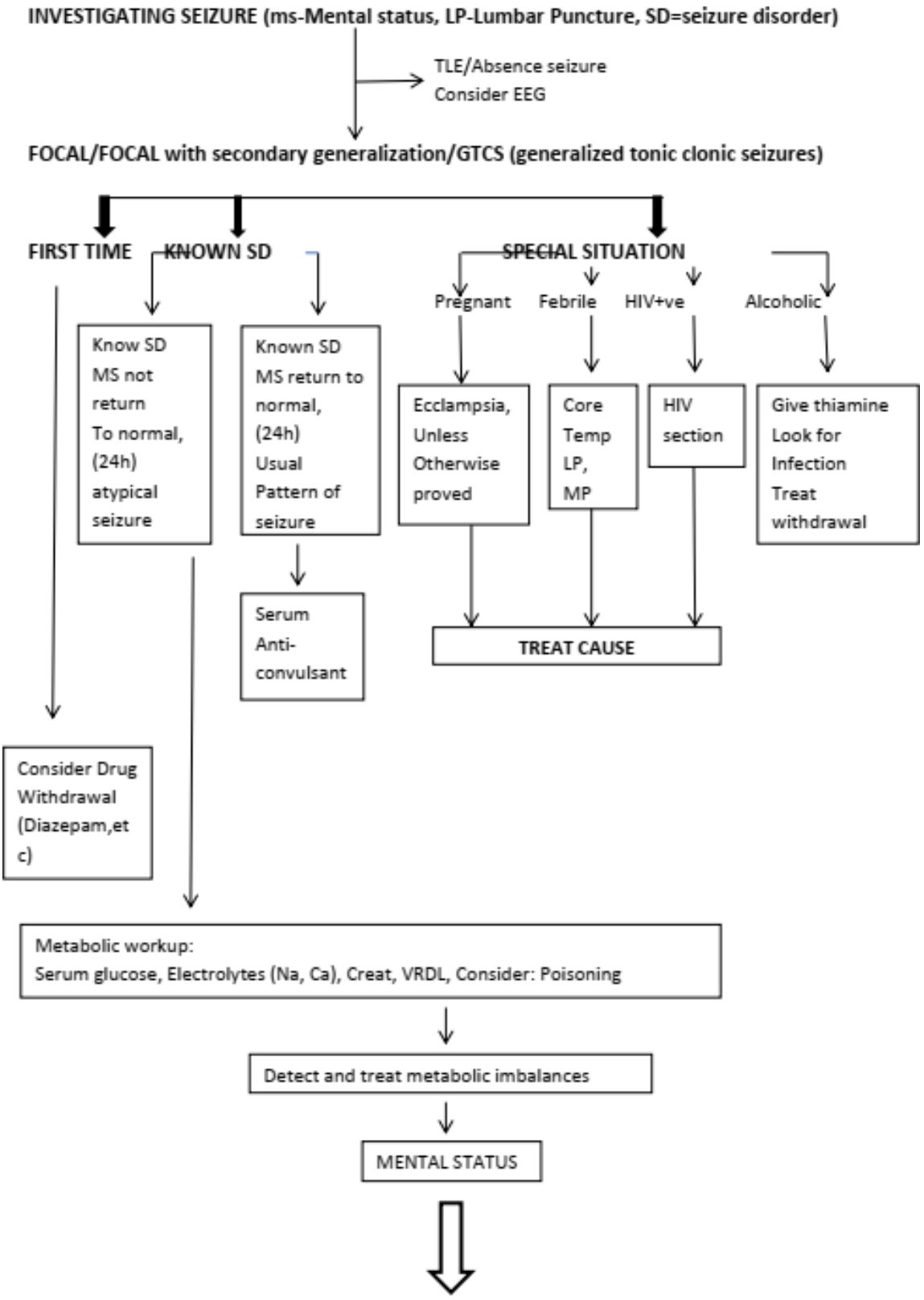
CAUSAL history:- Drug withdrawal, Alcoholism, Diabetic on insulin/OHAs; Antiepileptic therapy,Suicidal attempts? Poisoning

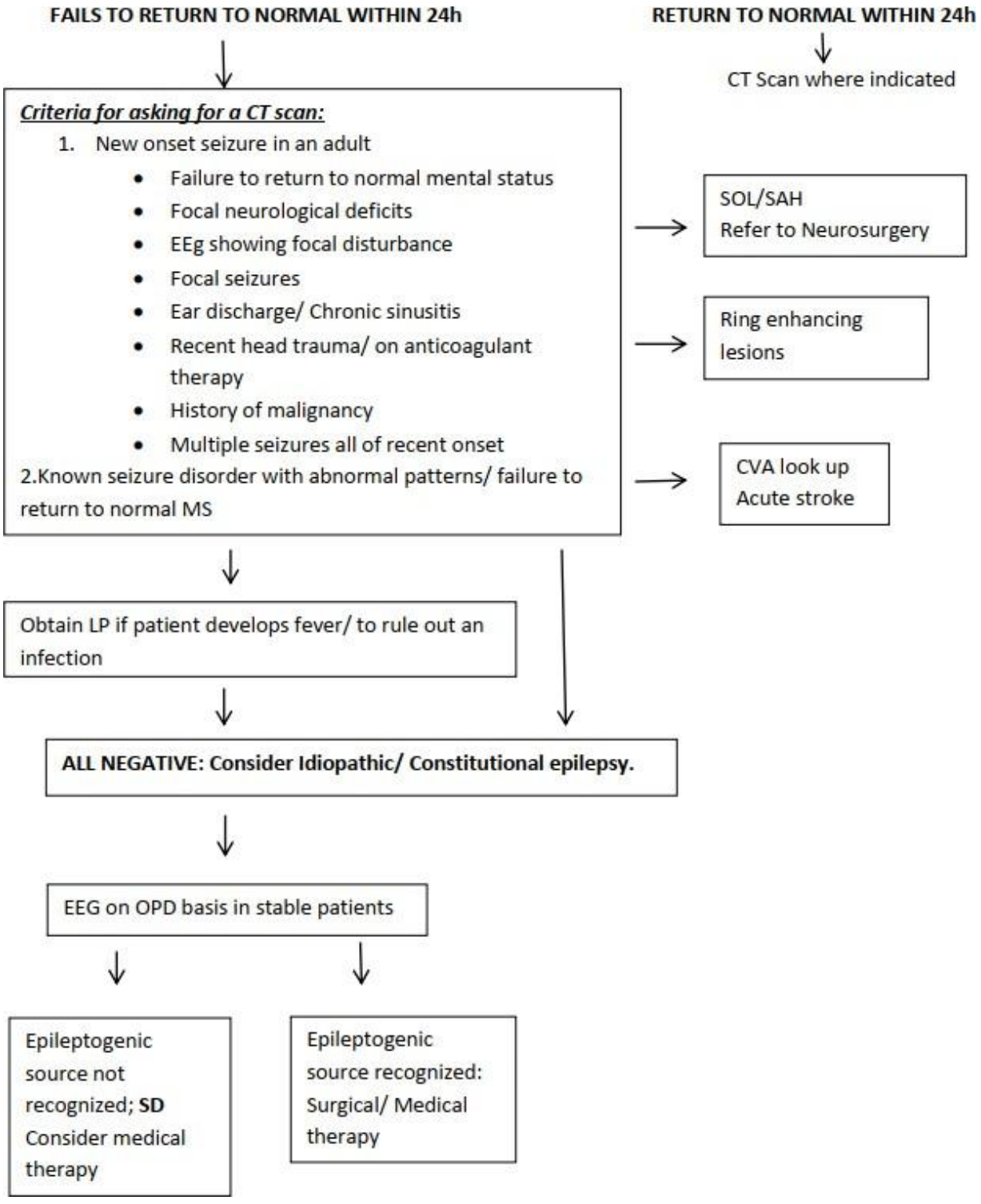
Demonstration of the spell can be asked by any member who has witnessed the episode.

Look for :- Signs of meningism, Focal neurological deficits, Pregnancy, Hypertension (HT encephalopathy)

Seizures in a pregnant lady, unless otherwise proved, isecclamsia. But do consider workup formeningitis if ferbile and / or signs of meningism are present.

SEIZURE DISORDER




FAILS TO RETURN TO NORMAL WITHIN 24h
RETURN TO NORMAL WITHIN 24h
CT Scan where indicated
Criteria for asking for a CT scan:
1. New onset seizure in an adult
Failure to return to normal mental status
Focal neurological deficits
EEg showing focal disturbance
Focal seizures
Ear discharge/ Chronic sinusitis
Recent head trauma/ on anticoagulant therapy
History of malignancy
Multiple seizures all of recent onset
2.Known seizure disorder with abnormal patterns/ failure to return to normal MS
SOL/SAH
Refer to Neurosurgery
Ring enhancing lesions
CVA look up
Acute stroke
Obtain LP if patient develops fever/ to rule out an infection
ALL NEGATIVE: Consider Idiopathic/ Constitutional epilepsy.
EEG on OPD basis in stable patients
Epileptogenic source not recognized; SD Consider medical therapy
Epileptogenic source recognized: Surgical/ Medical therapy

START ON ANTIEPILEPTIC THERAPY

POINTS TO CONSIDER :-

- Is there a need at all? (place of work, frequency of episodes)
- Choose drug based on seizure type, age, pregnancy status, DI, cost.

TAILORED ANTI-EPILEPTIC DRUGS

Assoc. condition	Drug of choice	Avoid using
Pregnancy	Phenobarb/Carbamz	Pheny/Valp/Clonazepam
Lactation	Valproate/(Phenyt/Carb)*	Clonaz/Lamotrigine
SLE	Carb/Phenobarb	Phenytoin/Ethosuximide
Liver disease	Phenobarb/Clonepam	Valp/Carbamzepine
Renal failure	Phenytoin	
Obstr. Sleep Apnoea	Valproate	
Cranial N neuralgia	Carbamazepine	
Manic	Carbamz/Clonazepam	
Diabetic neuropathy	Carbamazepine	
Alcohol withdrawal	Carbamazepine	Valproate
Porphyria	Valproate	Phenytoin/Carbmzepine Phenobarb/Primidone.

- With caution. Avoid single dose Phenobarb- sedates infant.
- Give only one drug (except in special circumstances when control is not achieved)

Drug	Dose/divided doses	Seizure type	Steady state	Cost (100 tabs)
>Phenytoin	200-400mg/1/d	GTCS/PS	5-10d	Rs.42-93(100mg)
$ Carbamaz	600-1200mg/2-3/d	GTCS/PS	3-4d	Rs.92-120(100mg)
#valporic acid	1.5-2.0g/3/d	GTCS/PS Petimal/Myoclonic S	2-4d	Rs.200
~Clonazepam	0.25-0.5mg/kg/2/d	Petimal ? Myoclonic S		Rs.90 (0.5mg)
**Phenobarb	100-200mg/1/d	GTCS/PS	14-21d	Rs.70 (30mg)

*PS= Partial seizures/GTCS=Generalized tonic clonic seizures.

>Start as 100mg tid, after stabilizing, can change onto SD.

$ Start 200mg BD, increments of 200mg/d/1/week upto 800mg BD.

Start as 600mg in divided dosage, increase at the rate of 200mg/3days till seizures controlled.

~ Start with 0.5-1.5mg/d, increase by 0.5mg/d/every 3-7 days upto a max.

** Start as a single dose at night, 60-120mg,go up by 30mg/d, 1/wk till max dose reached.

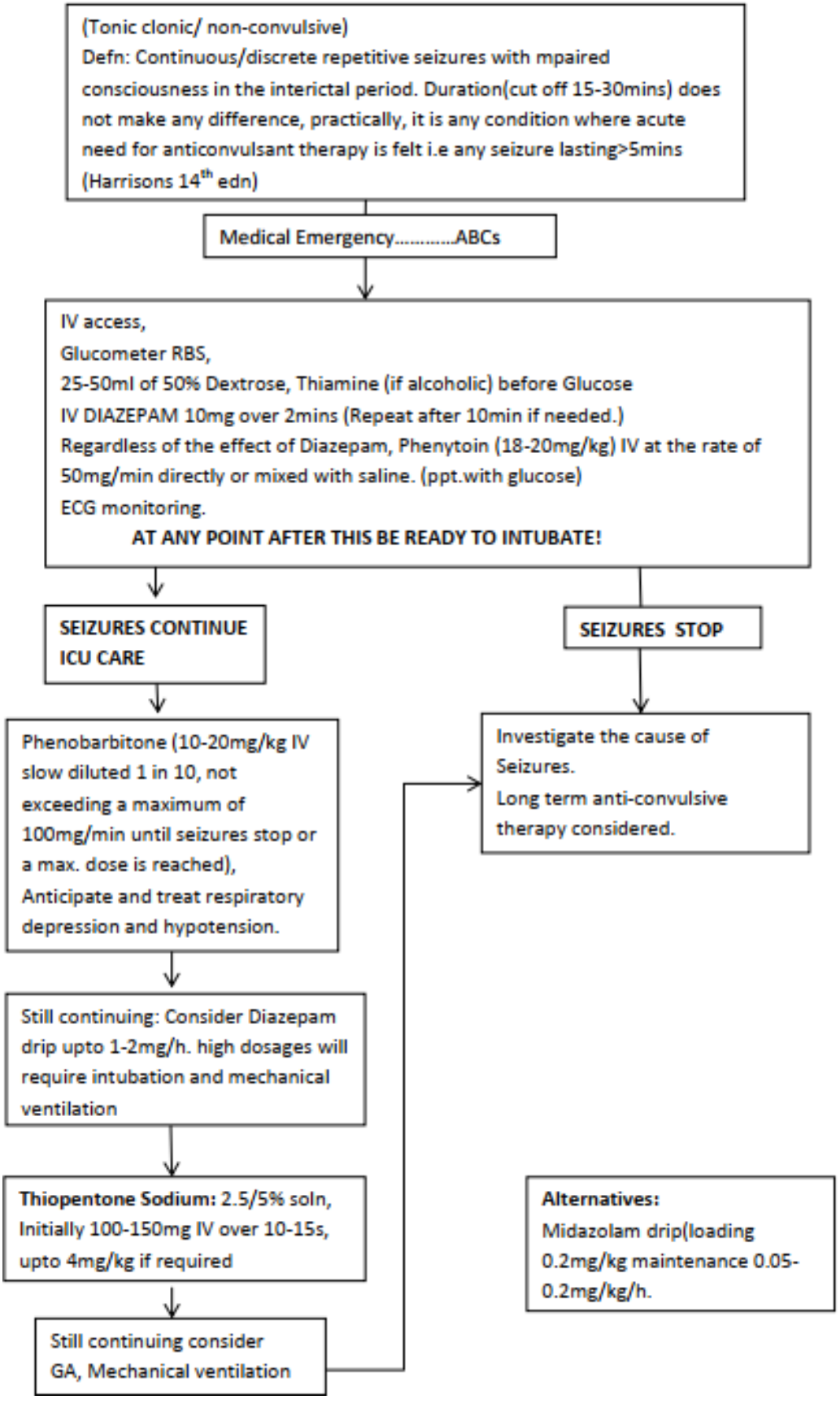
- Patient education about drug dosage, SE, and disease for compliance.
- Monitor- SE, seizure frequency, blood levels.

DRUGS THERAPEUTIC LEVEL

Phenobarb	20-40	ug/ml
Carbamazepin	5-12	ug/ml
Valporate	50-100	ug/ml
Phenytoin	10-20	ug/ml
Clonazepam	20-80	ug/ml

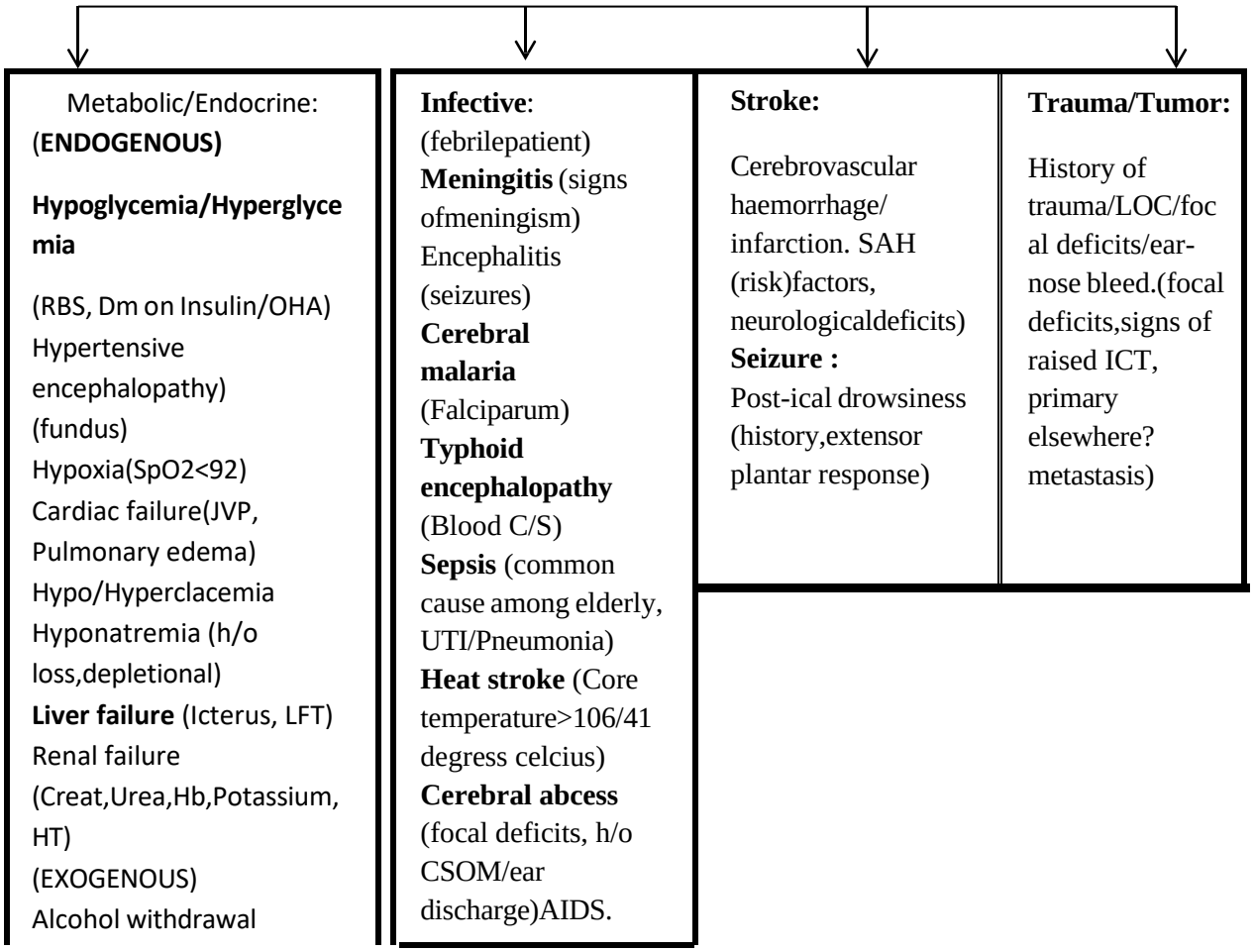
Determine policy for termination of therapy. Seizure free 3 years at least; more chances of recurrence in those with focal seizures/ continuing EEG abnormalities/initially failed to respond totherapy. Dose reduction should be gradual over months, one drug at a time. Restart therapy if recurrence occurs with same drug and regimen. A seizure is no more difficult to control after a recurrence than before. Do not stop in a mentally retarded patient.

STATUS EPILEPTICUS



30 ACUTE CONFUSIONAL STATE
(Delrium)

COMMON CAUSES Mnemonic- 'MIST' (within brackets pointers to diagnosis)



Treatment:-

Most often guided by the cause of the acute confusional state. Points to

remember:- Any acute confusional state-

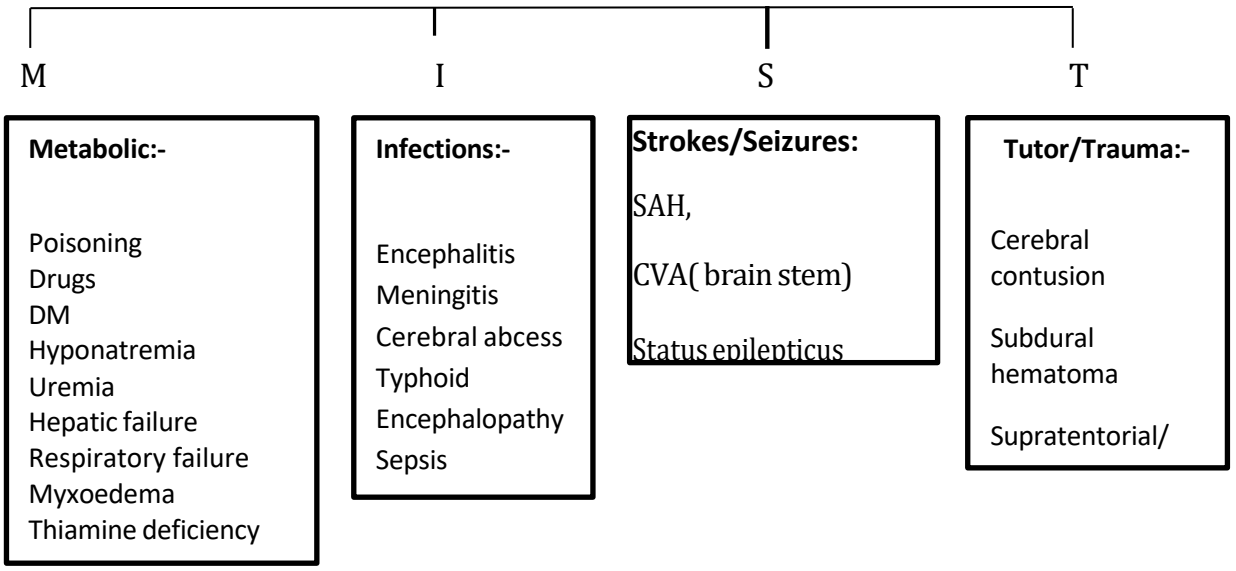
- ABC is first, start a line and collect blood for the routine investigations,
- Consider "Antidotes" = GOT FAN- Glucose, Oxygen, Thiamine, Flumazenil, Antropine, Naloxone
- Glucometer reading of whole blood glucose,
- IV bolus of 50% dextrose 25ml (in a non-alcoholic) or high dose Thiamine (100mg bolus)followed by any kind of glucose containing solutions in a known alcoholic.
- For Alcoholic delirium tremens add on Chlordiazepoxide (Librium) 50/100mg/day in divideddosage.
- If patient is restless and likely to cause harm to himself and others, use haloperidol 1mg-2.5mg as needed, IM.

31 COMA

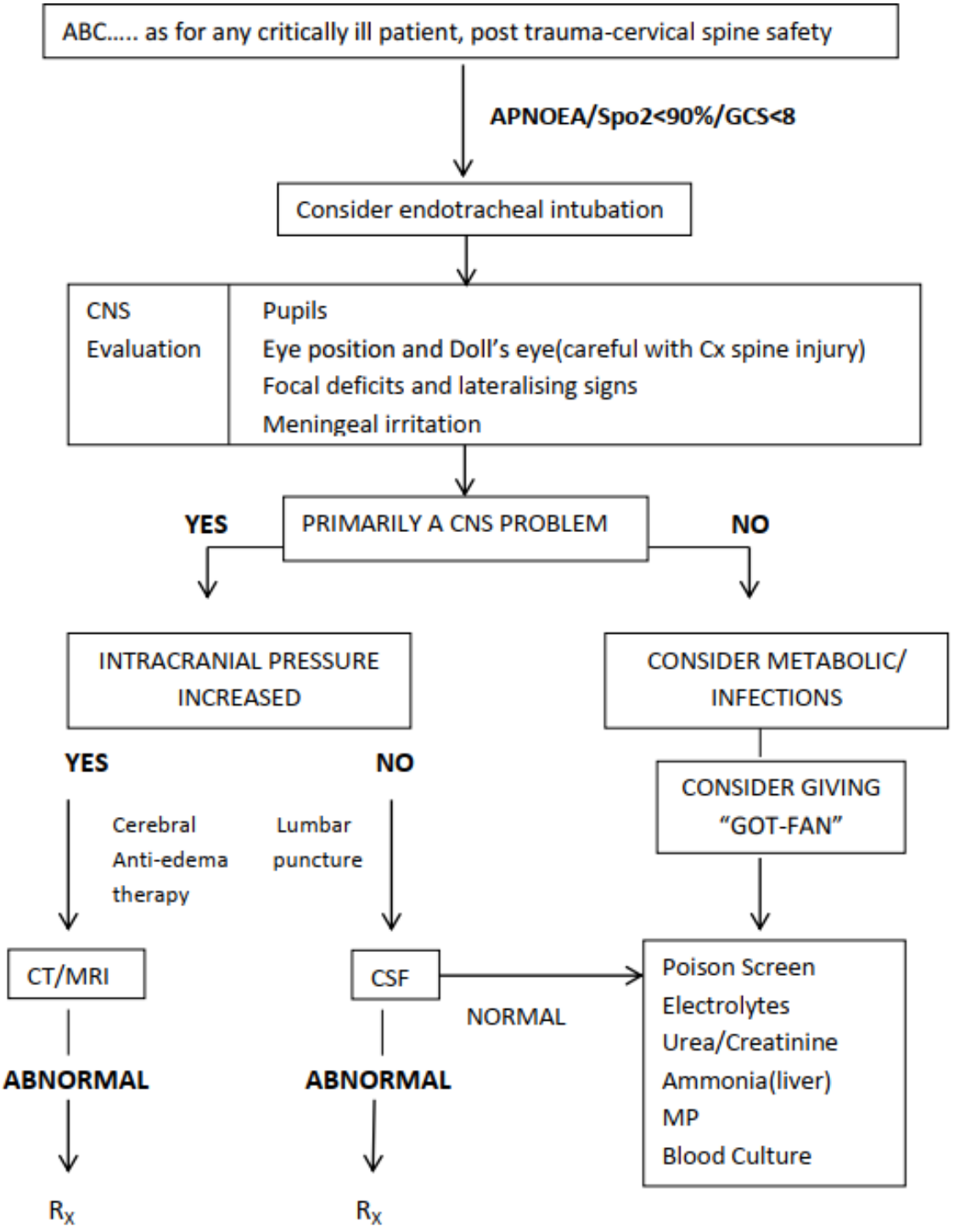
An approach…..

Definition:- Persistent loss of consciousness. COMMON

CAUSES :- Mnemonic 'MIST'



Approach to a Comatose Patient



GOT-FAN- Glucose, Oxygen, Thiamine Flumazenil, atropine, Naloxone

COMMUNITY MEDICINE STANDARD TREATMENT GUIDELINES

Standard Treatment Guidelines (Community Medicine)

List of diseases

1. Vaccine Preventable Diseases
2. Diarrheal diseases
3. Tuberculosis
4. Vector Borne Diseases - Malaria, Dengue, Chikungunya

1. VACCINE PREVENTABLE DISEASES

National Immunization Schedule

Age	Vaccines given
Birth	Bacillus Calmette Guerin (BCG), Oral Polio Vaccine (OPV)-0 dose, Hepatitis B birth dose
6 Weeks	OPV-1, Pentavalent-1, Rotavirus Vaccine (RVV)-1, Fractional dose of Inactivated Polio Vaccine (fIPV)-1, **Pneumococcal Conjugate Vaccine (PCV) -1***
10 weeks	OPV-2, Pentavalent-2, RVV-2
14 weeks	OPV-3, Pentavalent-3, fIPV-2, RVV-3, **PCV-2***
9-12 months	Measles & Rubella (MR)-1, **JE-1**** , **PCV-Booster***
16-24 months	MR-2, **JE-2****, Diphtheria, Pertussis & Tetanus (DPT)-Booster-1, OPV – Booster
5-6 years	DPT-Booster-2
10 years	Tetan us & adult Diphtheria (Td)
16 years	Td
Pregnant Mother	Td-1, Td-2 or Td-Booster***

* **PCV** in selected states/districts: Bihar, Himachal Pradesh, Madhya Pradesh, Uttar Pradesh (selected districts) and Rajasthan; in Haryana as state initiative

** JE in endemic districts only

*** One dose if previously vaccinated within 3 years

Vaccine	When to give	Dose	Route	Site
For Pregnant Women				
Tetanus & adult Diphtheria (Td)- 1	Early in pregnancy	0.5 ml	Intra-muscular	Upper Arm
Td-2	4 weeks after Td-1	0.5 ml	Intra-muscular	Upper Arm
Td-Booster	If received 2 TT/Td doses in a pregnancy within the last 3 years*	0.5 ml	Intra-muscular	Upper Arm
For Infants				
Bacillus Calmette Guerin (BCG)	At birth or as early as possible till one year of age	0.1ml (0.05ml until 1 month age)	Intra-dermal	Left Upper Arm
Hepatitis B - Birth dose	At birth or as early as possible within 24 hours	0.5 ml	Intra-muscular	Antero-lateral side of mid-thigh
Oral Polio	At birth or as early as	2 drops	Oral	Oral

Vaccine (OPV)-0	possible within the first 15 days			
OPV 1, 2 & 3	At 6 weeks, 10 weeks & 14 weeks (OPV can be given till 5 years of age)	2 drops	Oral	Oral
Pentavalent 1, 2 & 3	At 6 weeks, 10 weeks & 14 weeks (can be given till one year of age)	0.5 ml	Intra-muscular	Antero-lateral side of mid- thigh
Pneumococcal Conjugate Vaccine(PCV)	Two primary doses at 6 and 14 weeks followed by Booster dose at 9-12 months	0.5 ml	Intra-muscular	Antero-lateral side of mid- thigh
Rotavirus (RVV)	At 6 weeks, 10 weeks & 14 weeks (can be given till one year of age)	5 drops (liquid	Oral	Oral

		vaccine) 2.5 ml (lyophilized vaccine)		
Inactivated Polio Vaccine (IPV)	Two fractional dose at 6 and 14 weeks of age	0.1 ml	Intra dermal two fractional dose	Intra-dermal: Right upper arm
Measles Rubella (MR) 1st dose	9 completed months -12 months. (Measles can be given till 5 years of age)	0.5 ml	Sub-cutaneous	Right upper Arm

Vaccine	When to give	Dose	Route	Site
Japanese Encephalitis (JE) - 1	9 completed months -12 months.	0.5 ml	Sub-cutaneous (Live attenuated vaccine) Intramuscular(Killed vaccine)	Left upper Arm (Live attenuated vaccine) Anterolateral aspect of mid thigh

				(Killed vaccine)
Vitamin A (1st dose)	At 9 completed months with measles-Rubella	1 ml (1 lakh IU)	Oral	Oral
For Children				
Diphtheria, Pertussis & Tetanus (DPT) booster-1	16-24 months	0.5 ml	Intra-muscular	Antero-lateral side of mid-thigh
MR 2nd dose	16-24 months	0.5 ml	Sub-cutaneous	Right upper Arm
OPV Booster	16-24 months	2 drops	Oral	Oral
JE-2	16-24 months	0.5 ml	Sub-cutaneous (Live attenuated vaccine) Intramuscular(Killed vaccine)	Left upper Arm (Live attenuated vaccine) Anterolateral aspect of mid thigh (Killed vaccine)

Vitamin A (2nd to 9th dose)	16-18 months. Then one dose every 6 months up to the age of 5 years.	2 ml (2 lakh IU)	Oral	Oral
DPT Booster-2	5-6 years	0.5 ml.	Intra-muscular	Upper Arm
Td	10 years & 16 years	0.5 ml	Intra-muscular	Upper Arm

*One dose if previously vaccinated within 3 years Note:

- JE Vaccine is introduced in select endemic districts after the campaign.
- The 2nd to 9th doses of Vitamin A can be administered to children 1-5 years old during biannual rounds, in collaboration with ICDS.
- PCV in selected states/districts: Bihar, Himachal Pradesh, Madhya Pradesh, Rajasthan & Uttar Pradesh (selected districts), and in Haryana as state initiative

2. DIARRHEAL DISEASES

Diarrheal diseases include acute diarrhea, persistent diarrhea (diarrhea duration two weeks or more) and dysentery (blood-stained stools with fever). Diarrheal diseases are one of the most common causes of epidemic in our State. Most of the deaths in diarrheal diseases are due to dehydration which is preventable by timely and adequate replacement of fluids.

- Acute diarrhea – Cholera, Rota virus, Food poisoning, gastrointestinal disorders and medications (rare).
- Persistent diarrhea – Chronic bacterial infections, inflammatory bowel disorders, malabsorption syndrome.
- Dysentery – Amoebiasis, Giardiasis, Shigellosis.

Sign/symptom	Acute diarrhoea	Persistent diarrhoea	Dysentery
Frequency of stools/day	Three or more	Three or more	Three or more
Consistency of stools	Watery	Variable	Variable
Duration of diarrhoea	Less than 2 weeks	Two or more weeks	Less than 2 weeks
H/o fever	No	Variable	Yes
H/o blood stained mucus	No	Variable	Yes
Effect on appetite	No	Loss of appetite	Loss of appetite
Dehydration	Important, may lead to severe dehydration if not treated in time.	Patient may have some dehydration.	Patient may have some dehydration
Treatment principle	Management of dehydration is priority	Start management of dehydration. Simultaneously find cause of persistent diarrhoea and treat accordingly.	Start management of dehydration. Simultaneously start appropriate antibiotics.
Long term effects	No long term effect for occasional	If not treated correctly, child	Repeated attacks may lead to Protein

	episodes. Repeated attacks may lead to PEM.	may get severe Protein Energy Malnutrition	Energy Malnutrition

Sign/Symptom	Severity of symptoms and signs		
	No dehydration	**Some dehydration**	**Severe dehydration**
General condition of Patient	Patient well alert	Restless and irritable	Lethargic, unconscious, floppy
Presence of thirst	Normal/not thirsty	Thirsty, drinks water immediately when offered	Not able to drink
Dryness of mouth and tongue	Moist mouth and tongue	Mouth and tongue dry	Mouth and tongue very dry
Condition of eyes	Normal	Sunken	Very sunken, patient's face looks like old man's face.
Condition of tears	Tears appear while crying	Tears appear while crying	No tears, dry eyes even in crying child
Skin turgor	Normal. Pinch to skin immediately goes back to normal.	Pinch slowly goes back and takes some time to become flat.	Pinch remains as it is for 2-3 seconds and then slowly goes back.
Classification of dehydration	No dehydration	Some dehydration	Severe dehydration
Treatment of dehydration	Plan – A	Plan – B	Plan – C

Most important aspect in management of diarrheal diseases is correction of dehydration. Treatment of dehydration is divided into three plans as follows -

- **Plan-A**: For patients with no dehydration – principle is to prevent dehydration.

- **Plan-B:** For patients with some dehydration – principle is treatment of some dehydration and preventing patient from going into severe dehydration.
- **Plan-C:** For patients with severe dehydration - This is a lifesaving plan. Rehydrate patient as early as possible and prevent from going again into severe dehydration.

Description of treatment plans in details is as follows.

PLAN A

Principle of treatment

As diarrhea is continuing, there is continuous loss of water and electrolytes from body of patient which may lead to dehydration. Therefore, principle of Plan-A schedule is correction of whatever loss of water and electrolytes before the patient develops signs of dehydration. Plan-A can be advised at home to caretaker of patient. However, make sure that care taker has understood danger signs of dehydration (like thirst). Following steps are recommended in Plan-A.

a. Home available fluids

- Advise to give Home Available Fluids (HAF) e.g., sarbat, lassi, vegetable soup, kheer, buttermilk, tea, coconut water, etc. i.e., any liquid available at home to patient as much he/she can drink.
- Continue breast feeding and feeding – If child is being breastfed, then breast-feeding should be continued. Regular feeding of non-breast-fed child should also be continued.

b. ORS to prevent dehydration

If frequency and amount of diarrhea is not declining or amount of stool is large, then start ORS.

- Contents of WHO ORS are as follows – (New low osmolarity ORS).

Sodium chloride	-	2.6grams
Potassium Chloride	-	1.5 grams
Trisodium Citrate	-	2.9 grams
Glucose	-	13.5 grams

- Dissolve the packet in one litre of water to prepare ORS.
- Show caretaker how to prepare ORS. Following steps should be carried out for preparation of ORS -
  - Take clean pot of one and half litre capacity and one clean spoon.
  - Pour 1 litre of clean drinking water in the pot. (No need to boil water).
  - Add whole packet of ORS into one-litre of water and stir till all powder is dissolved. Now ORS is ready for use.

- Give ORS by cup or spoon to small children and by glass to bigger children and to adults as per indicated dose.
- If patient has vomiting, wait for 5 minutes and start again.
- Keep ORS covered. Once prepared ORS should be used within 24 hours. Do not use ORS beyond 24 hours, as there are chances of contamination.
- If child develops swelling on eyelids, stop ORS as it indicates overdose.
- Ask her to give ORS in following doses after passage of each liquid stool.

PLAN B

Start Plan-B treatment to patients showing signs and symptoms of some dehydration as per dehydration diagnosis chart. Aim of this plan is to correct dehydration and prevent patient from going into severe dehydration.

Dose of ORS: Dose of ORS is calculated preferably according to weight of patient. Give ORS in a dose of 100ml/kg in 4 hrs. If weighing is not possible, calculate age wise ORS requirement for four hours as follows –

Table -3: Age wise ORS requirement for four hours

Age	< 4 months	4–11 months	12–23 months	2 – 4 years	5 – 14 years	15 + years
Dose	200-400 ml	400 – 600 ml	600 – 800 ml	800 – 1200 ml	1200 – 2200 ml	2200-4000 ml

Continue breast feeding and feeding – If child is being breastfed, then breast- feeding should be continued.

Re-examination of patient

Re-examine patient after every four hours for status should also be continued.

Table-4: Management advice based on re-examination findings

Condition of patient on re-examination	Management advise
Patient improves, no signs of dehydration on examination and diarrhoea stops	Keep patient under observation for 24 hours. Continue HAF. Observe if diarrhoea and/or vomiting start again.
Patient improves, no signs of dehydration on examination but diarrhoea continues	Continue giving ORS in doses suggested in Plan-A, reexamine after four hours.
Dehydration status same	Continue with Plan-B. Check whether ORS is being given in correct dose. Re-examine after four hours.
Signs of severe dehydration appear	Switch on to Plan - C (start IV fluids). Continue to give ORS as much as possible.

If signs and symptoms of patient are suggestive of severe dehydration, start Plan – C. This is emergency plan. Incorrect or incomplete management of severely dehydrated patient may lead to death of patient. Medical Officer must personally examine patient and treat for severe dehydration.

Principles of management

Principle of management of severe dehydration is replacing fluid loss by giving rapid IV infusion. Only Ringer's lactate should be used as IV fluid and the dose is 100ml/kg body weight.

Age group	Intensive phase	Maintenance phase	Duration of treatment	Remarks
Infants (0-1 year)	30-ml/kg body wt. during first 1 hour.	70ml/kg body wt in next 5 hours.	6 hrs	Assess patient after every 6 hours
Older children and adults	30-ml/kg body wt. in first half hour.	70ml/kg body wt in next $2^1/_2$ hrs.	3 hrs	Assess patient after every 3 hours

Re-examine patient after every six hours in infants and three hours in adults for status of dehydration with the help of dehydration diagnosis chart and decide management plan as follows -

Table-5: Treatment advice based on condition of patient

Condition of patient	Treatment advise
Patient improves, no signs of dehydration on examination and diarrhoea stops	Keep patient under observation for 24 hours as patient may start diarrhoea/vomiting again
Patient improves, no signs of dehydration on examination but diarrhoea continues	Continue giving ORS (Plan-A)
Patient improves, signs of some dehydration on examination.	Stop IV fluids after required dose is administered. Continue giving ORS (Plan-B)
Dehydration status same	Continue with Plan-C. Check for any complications like anuria. If yes carefully examine the patient and decide for referral. Continue giving IV during transportation of patient.

Antibiotics are recommended only to suspected patients of cholera and dysentery. Other drugs like anti motility drugs, binding agents, anti-secretory agents and steroids are not of any use in management of diarrhea. They are harmful to patients and therefore not at all recommended for treatment.
Judicious use of antibiotics is appropriate in selected patients. Severely ill patients with febrile dysentery can be treated with ciprofloxacin 500mg bd for 3- 5 days.

Use of Zinc Tablets

Zinc Dosage Recommendation: Zinc is very safe drug and has a very large window of safety. Zinc dispersible tablets are to be given in each diarrheal episode along with low osmolality ORS or Oral rehydration therapy (in case ORS is not available), irrespective of type of dehydration.

Zinc administration as per age of child:

a) Children from 2-6 months: Children aged between 2-6 months should be given 10 mg of elemental zinc per day for a total period of 14 days from the day of onset of diarrhoea. A tablet of zinc contains 20 mg of elemental zinc. Therefore half tablet should be given to the children in this age group. Zinc when supplied in the form of dispersible tablets, easily dissolves in breast milk or water. Therefore, in infants below 6 months of age, the tablet

should be given by dissolving in breast milk and in infants above 6 months of age, it should be given by dissolving in breast milk or water.

b) Children above 6 months: One full tablet (20mg) should be given to all children with diarrhoea above 6 months of age. It should start from the day of onset of diarrhoea and continued for a total period of 14 days.

3. TUBERCULOSIS

Tuberculosis is an infectious disease caused predominantly by *Mycobacterium tuberculosis*. It is most commonly transmitted by inhalation of infected droplet nuclei which are discharged in the air when a patient with untreated pulmonary TB coughs or sneezes. A smear positive pulmonary TB can infect 10-15 persons in a year, and remain infectious for 2-3 years if left untreated. The chances of getting infected depend on the duration, frequency of exposure and the immune status of an individual. All those who get infected do not necessarily develop TB disease. The lifetime risk of breaking down to disease among those infected with TB is 10-15%, which gets increased to 10% per year among those PLHIV. Other determinants like Diabetes Mellitus, smoking, alcohol abuse and malnutrition also increase the risk of progression from infection to disease.

Burden of TB in India:

India accounts for one fourth of the global TB burden i.e 2.8 million out of 10.4 million new cases annually with the mortality rate of 36 per lakh population (source WHO Global TB Report 2016). In India, more than 40% of population is infected with *Mycobacterium tuberculosis*. It is estimated that there are approximately 5.1 million prevalent cases of all forms of TB disease.

India has the highest burden of both TB and MDRTB (1.3lakhs out of 5.8 lakhs globally). Based on sub-national drug resistant survey ~3% among new TB cases and 12-17% among previously treated TB cases have MDRTB

Symptoms of Tuberculosis

1. Pulmonary Tuberculosis (PTB)

The most common symptoms of PTB are cough with or without expectoration, chest pain, hemoptysis and shortness of breath. It may be accompanied by constitutional symptoms like fever, weight loss, night sweats, tiredness, loss of appetite etc.

2. Extrapulmonary TB (EPTB)

A person with EPTB may have symptoms related to the organs affected along with constitutional symptoms stated above.

Diagnosis: All efforts should be undertaken for microbiologically confirming the diagnosis in presumptive TB patients. The following are acceptable methods for diagnosis under NTEP and are available at free of cost-

1. Sputum smear microscopy (for AFB) – Zeihl-Neelson staining and fluorescence staining.
2. Culture- Solid and Liquid.
3. Line Probe Assay (LPA).
4. Nucleic Acid Amplification Test (CB-NAAT).

Other Diagnostic Tools:

1. Radiography.
2. Bio chemical.
3. Histopathological.
4. Tuberculin Skin Test (Only for supporting diagnosis as it indicates infection only NOT disease).
5. USG.

Treatment:

NTEP is now introducing for treatment of drug sensitive TB based on the principle of administering daily fixed dose combinations of 1st Line Anti TB drugs in appropriate weight bands.

Guidelines for treatment initiation:

1. Counseling of the patient and close family member about the disease, mode of infection, cough hygiene, treatment, side effect and importance of regular treatment.
2. Record of weight and height.
3. Look for co-morbidities (HIV, Diabetes, liver/Renal/neurological disorders) and substance abuse (Tobacco, drugs, alcohol).
4. Identify Treatment Supporter (erstwhile DOT provider).

The standard 6-month course of treatment consists of two phases-

Intensive phase (IP)	Continuation phase (CP)
The first phase lasts 2 months	The second phase lasts 4 months
HRZE	HRE

H- Isoniazid R- Rifampicin Z-Pyrazinamide E-Ethambutol S-Streptomycin

Extension of Continuation Phase: Extend CP by 3 to 6 months in special situations like Bone & Joint TB, Spinal TB with neurological involvement and neuro-tuberculosis.

Daily Dose Schedule for Adults (As per weight bands)

Weight band	Number of tablets (FDC)	
	Intensive phase (IP) (HRZE - 75/150/400/275)	Continuation phase (CP) (HRE - 75/150/275)
25 - 34 kg	2	2
35 - 49 kg	3	3

50 - 64 kg	4	4
65 - 75 kg	5	5
>= 75 kg	6	6

Drug dosage for paediatric TB

Weight category	**Number of tablets (dispersible FDCs)**		
	Intensive phase		**Continuation phase**
	HRZ	**E**	**HRE**
	50/75/150	**100**	**50/75/100**
4-7 kg	1	1	1
8-11 kg	2	2	2
12-15 kg	3	3	3
16-24 kg	4	4	4
25-29 kg	3 + 1A*	3	3+1A
30-39 kg	2 + 2A*	2	2+2A

A=Adult FDC (HRZE = 75/150/400/275; HRE = 75/150/275)

Follow up:

1. Clinical follow up: should be done monthly.
   - Improvement on chest symptoms, increase in weight etc. may indicate good prognosis
   - Control of co-morbid conditions like HIV and diabetes
   - Symptoms and signs of adverse reactions to drugs should be specifically asked .
2. Laboratory:
   - In case of pulmonary tuberculosis, sputum smear microscopy/ Culture should be done at the end of IP and end of treatment. A

negative sputum smear microscopy result at the end of IP may indicate good prognosis

- However, in the presence of clinical deterioration, the medical officer may consider repeating sputum smear microscopy even during CP. This will provide the patient an early opportunity to undergo drug susceptibility testing if s/he is found to be sputum smear positive
- Chest x-ray to be offered to drug sensitive pulmonary TB patients whenever required and available.
- Response to treatment in extra-pulmonary TB may be best assessed clinically. Help of radiological and other relevant investigations may be taken.

3. Long term follow up: After completion of treatment, the patients should be followed up at the end of 6, 12, 18 & 24 months.

Contact tracing: The following contacts of TB patients must be screened for TB.

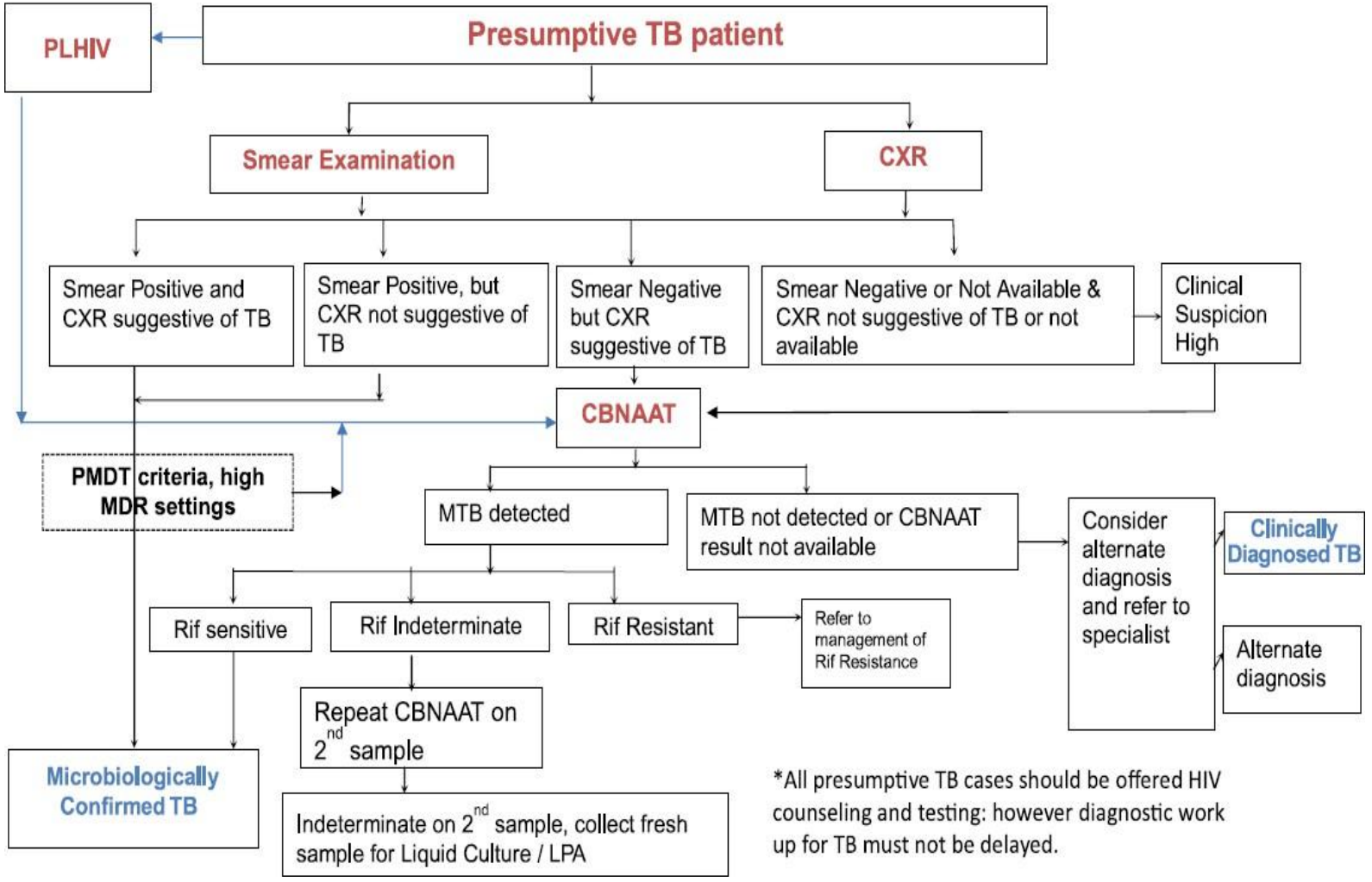
1. All close contacts like family members.
2. Reverse contact tracing in case of paediatric TB patients.
3. Children under 6 years.
4. Contacts with immune-compromised condition, diabetes, pregnancy, alcoholics etc.
5. Contacts with patient of DRTB.

Isoniazid Preventive Therapy (IPT)

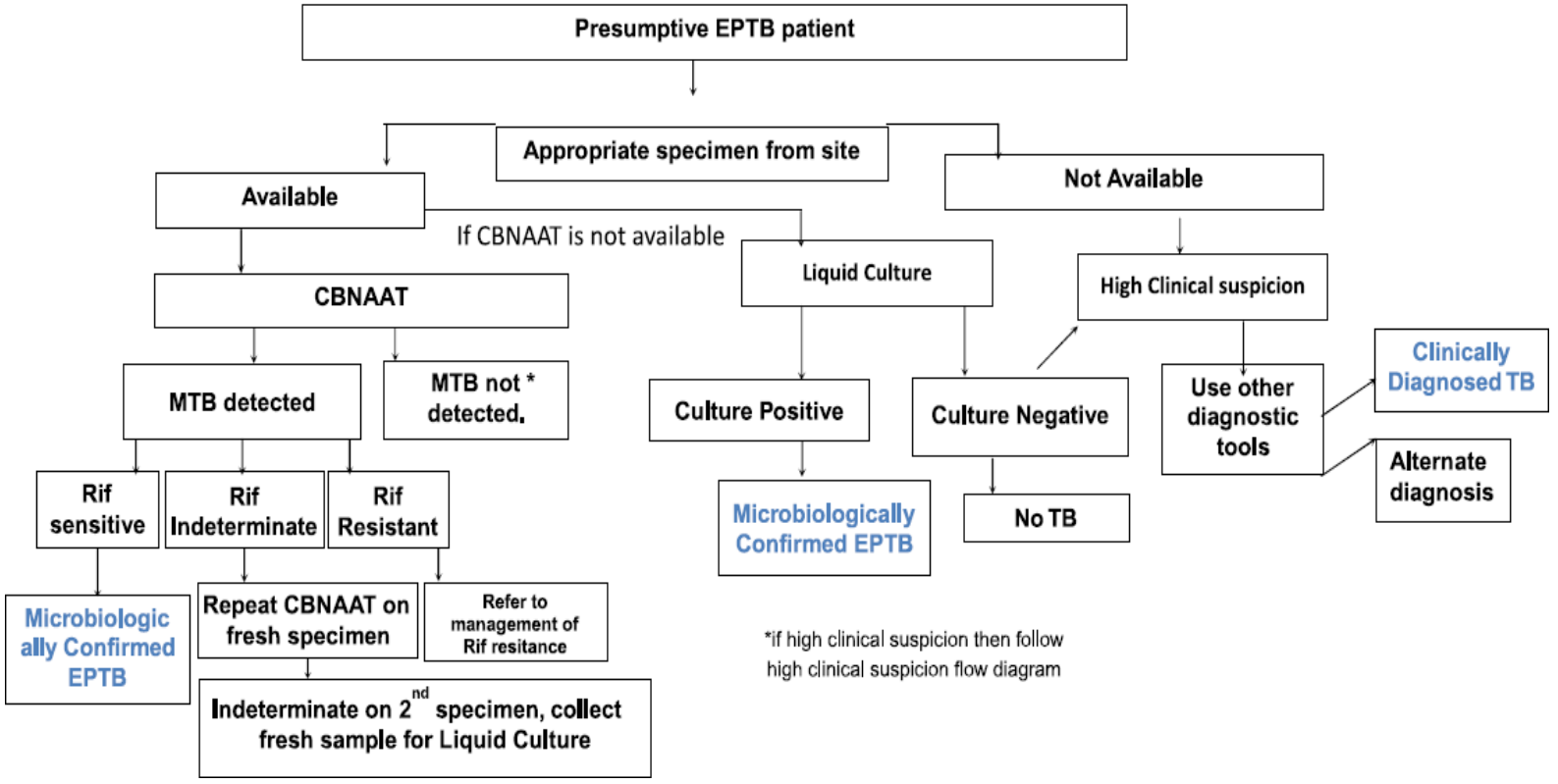
1. IPT should be given to PLHIV and children under 6 years who are close contact of TB patient after ruling out active disease irrespective of BCG/Nutritional status. The dose of Isoniazid is 10 mg/ kg body weight in pediatric and 300 mg in PLHIV (with pyridoxine 50 mg) daily for 6 months.

2. A child born to mother who was diagnosed to have TB in pregnancy should receive prophylaxis for 6 months, provided congenital TB has been ruled out. BCG vaccination can be given at birth even if INH preventive therapy is planned.

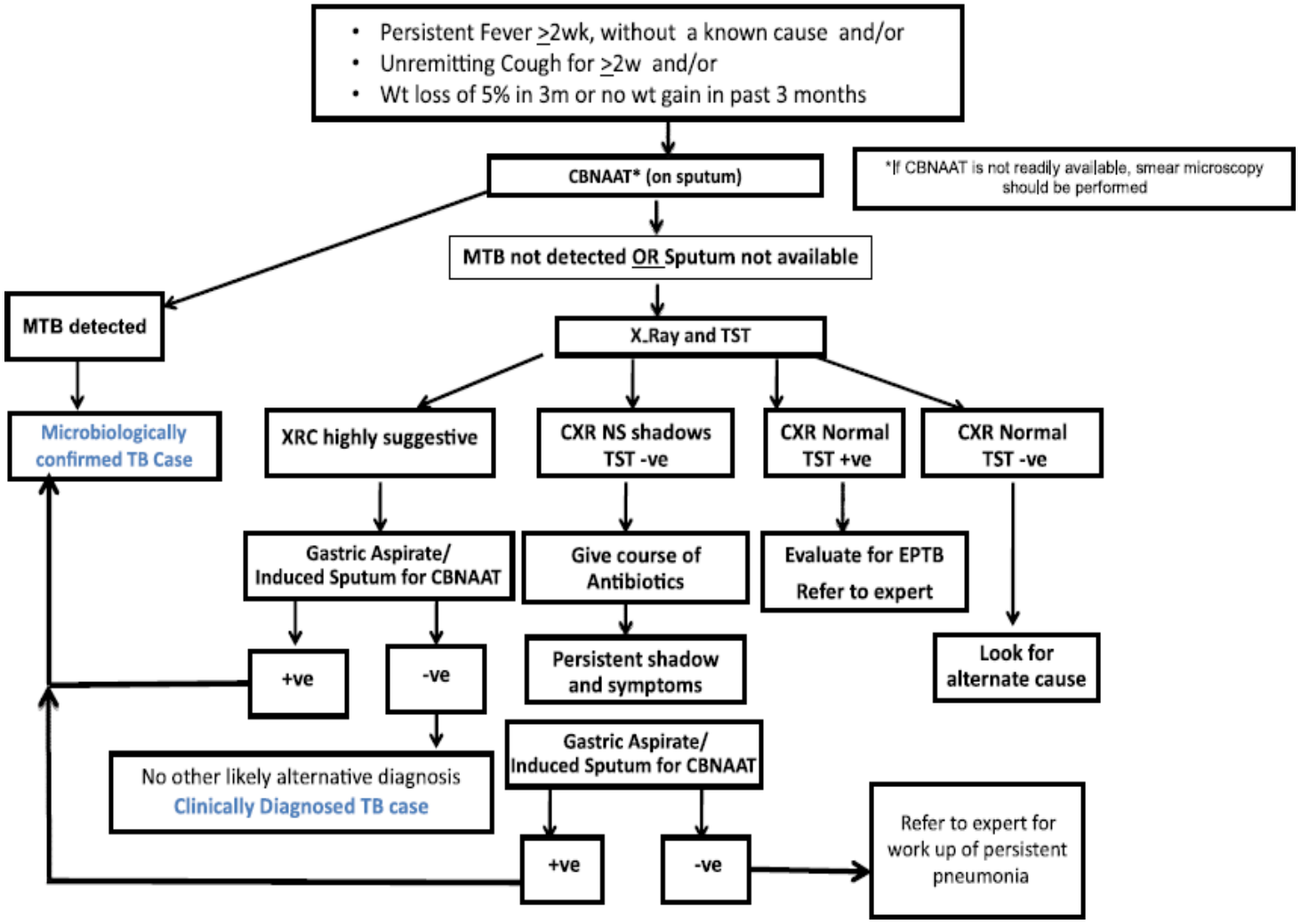
Diagnostic algorithm for pulmonary TB



Diagnostic Algorithm for Extra Pulmonary TB



Diagnostic algorithm for Pediatric Pulmonary TB



Programmatic management drug resistant TB (PMDT)

The term Programmatic management of drug resistant TB refers to programme based drug resistant diagnosis, management, and treatment.

Definitions:

- **Mono-resistance (MR):** A TB patient, whose biological specimen is resistant to one first-line anti-TB drug only.
- **Poly-Drug Resistance (PDR):** A TB patient, whose biological specimen is resistant to more than one first-line anti-TB drug, other than both INH and Rifampicin.
- **Multi Drug Resistance (MDR):** A TB patient, whose biological specimen is resistant to both isoniazid and rifampicin with or without resistance to other first line drugs, based on the results from a quality assured laboratory.
- **Rifampicin Resistance (RR):** resistance to rifampicin detected using phenotypic or genotypic methods, with or without resistance to other anti-TB drugs excluding INH. Patients, who have any Rifampicin resistance, should also be managed as MDR TB case.
- **Extensive Drug Resistance (XDR):** A MDR TB case whose biological specimen is additionally resistant to a fluoroquinolone (ofloxacin, levofloxacin, or moxifloxacin) and a second-line injectable anti TB drug (kanamycin, amikacin, or capreomycin) from a quality assured laboratory.

MDRTB suspect criteria:

1. **Criteria A:**
    - All failures of New TB cases
    - Smear positive previously treated cases who remain smear positive at 4th month
    - All pulmonary TB cases who are contacts of known MDRTB case
2. **Criteria B:** in addition to criteria A,
    - All smear positive previously treated pulmonary TB cases at diagnosis
    - Any follow up smear positive
3. **Criteria C:** In addition to criteria B,

- Smear negative previously treated pulmonary TB case at diagnosis
- TBHIV co infected cases.

Diagnostic Technology: Molecular Drug Susceptibility Testing (DST) by LPA and CBNAAT, Liquid culture, Solid culture for diagnosis. For follow up cultures, liquid culture is preferred over solid.

Sputum collection and transport: For diagnosis, two fresh samples early morning & spot or spot & spot (taken at least 1 hr apart) collected in Falcon tubes are transported to NTEP certified lab in cold chain within 72hrs. For follow up cultures one sample is sufficient.

Pretreatment evaluation:

1. Detail history (including screening for mental illness, seizer disorder, drug/alcohol abuse etc.) Weight
2. Height
3. Complete Blood Count with platelets count
4. Blood sugar to screen for Diabetes Mellitus
5. Liver Function Tests
6. Blood Urea and S. Creatinine to assess the Kidney function
7. TSH levels to assess the thyroid function (TSH levels alone are usually sufficient to assess the thyroid function of the patient)
8. Urine examination – Routine and Microscopic
9. Pregnancy test (for all women in the child bearing age group)
10. Chest X-Ray
11. ECG (if Moxifloxacin is to be used)
12. Serum electrolytes (if Capreomycin is to be used)
13. All DRTB cases will be offered HIV counseling and testing.

The results of pretreatment evaluation are communicated to DRTB centre committee and, if approved appropriate treatment regimen is initiated at DRTB centre or at the district level by the District TB Officer.

All services under PMDT are offered free of cost including patient and one attendant transportation cost.

Treatment Regimen:

Type of drug resistance	Intensive Phase	Continuation phase
Isoniazid mono-resistant	**(3-6 months) Km, Lfx, R, E, Z** Modify regimen based on baseline DST report to E, Z, Km, Lfx, Mfx	**(6months) Lfx, R, E, Z**
Rifampicin Resistant with Isoniazid sensitive or unknown	**(6-9 months) Km, Lfx, Eto, Cs, Z, E, H**	**(18months) Lfx, Eto, Cs, E, H**
MDRTB	**(6-9 months) Km, Lfx, Eto, Cs, Z, E** Modify based on the level of isoniazid resistance* and baseline second line DST result	**(18months) Lfx, Eto, Cs, E**
XDRTB	**(6-12months) Cm, PAS, Mfx, High dose-H, Cfz, Lzd, Amx/Clv**	**(18months) PAS, Mfx, High dose-H, Cfz, Lzd, Amx/Clv**

**For Isoniazid resistance, decision on use of Isoniazid in the regimen depends on following:*

- If High level resistance detected by Liquid culture - omit INH.
- If low level resistance detected by Liquid culture - add high dose INH.
- If LPA reports INH resistance by Kat G mutation- Omit INH If LPA reports INH resistance by INH A mutation- Use High dose INH. Ethionamide in the treatment regimen will be replaced with PAS.

Drug dosage for DRTB

S.No	Drugs	16-25 Kgs	26-45 Kgs	46-70 Kgs	>70 Kgs
1	Rifampicin*	300	450	600	600
2	Isoniazid$	200	200	300	450
3	Ethambutol	400 mg	800 mg	1200 mg	1600 mg
4	Pyrazinamide	500 mg	1250 mg	1500 mg	2000 mg
5	Kanamycin	500 mg	500 mg	750 mg	1000 mg
6	Levofloxacin	250 mg	750 mg	1000 mg	1000 mg
7	Ethionamide	375 mg	500 mg	750 mg	1000 mg
8	Cycloserine	250 mg	500 mg	750 mg	1000 mg
9	Na-PAS (80% weight/vol)[1]	7.5 gm	10 gm	12 gm	16 gm
10	Pyridoxine	50 mg	100 mg	100 mg	100 mg
11	Moxifloxacin (Mfx)	200 mg	400 mg	400 mg	400 mg
12	Capreomycin (Cm)	500 mg	750 mg	1000 mg	1000 mg
13	Amikacin (Am)	500 mg	500 mg	750 mg	1000 mg
14	High dose INH (High dose-H)	400 mg	600 mg	900 mg	900 mg
15	Clofazimine (Cfz)	100 mg	200 mg	200 mg	200 mg
16	Linezolid (Lzd)	300 mg	600 mg	600 mg	600 mg
17	Amoxyclav(Amx/Clv) (In child: WHO 80mg/Kg in 2 divided doses)	875/125 mg BD	875/125 mg BD	875/125 mg 2 morn +1 even	875/125 2 morn +1 even
18	Clarithromycin (Clr)	250 mg BD	500 mg BD	500 mg BD	750 mg BD

**For mono-H resistant TB; $For Rifampicin Resistant TB In case of PAS with 60% weight/volume the dose will be increased to 10 gm (16-25 Kg); 14 gm (26-45 Kg); 16 gm (46-70 Kg) and 22 gm (>70 Kg)*

Drug dosage for paediatric MDRTB

Drug	Daily Doses*
Kanamycin / Capreomycin	15-30 mg/kg (SM 20-40 mg/kg)
Levofloxacin / Moxifloxacin	Levo <5 yrs: 15-20 mg/kg split dose Levo >5 yrs: 10-15 mg/kg once day Moxi 7.5-10 mg/kg
Ethionamide	15-20 mg/kg
Cycloserine	10-20 mg/kg
Ethambutol	15-25 mg/kg
Pyrazinamide	30-40 mg/kg
(Na-PAS)	<30 kg: 200-300 mg/kg

Drug dosage for paediatric XDTB

Drugs	Daily doseas per WHO document 2014
Inj. Capreomycin (Cm)	15-30 mg/kg
PAS	<30 kg: 200-300 mg/kg
Moxifloxacin (Mfx)	7.5-10 mg/kg
High dose INH (High dose-H)	15-20 mg/kg*
Clofazimine (Cfz)	1 mg/kg (max. 200 mg / day) limited data
Linezolid (Lzd)	10 mg/kg TDS (max. 600mg /day) with pyridoxine
Amoxyclav(Amx/Clv)	80 mg/kg (based on the amoxicillin component) in two divided doses (max. 4gm amox+0.5gm clav)
Clarithromycin (Clr)	7.5 mg/kg every 12 hours

Follow up:

1. **Clinical-** monthly during intensive phase and every three months during Continuation phase. It includes recording of weight and management of adverse drug reaction.

2. **Laboratory-**

a. S.Creatine: every month for the first three months, then every three month during injectable phase.

b. Liver Function Test is to be done monthly during intensive phase and three monthly during continuation phase.

c. ECG once a month in intensive phase if Moxifloxacin is used.

d. Complete blood count with Platelets count weekly in the first month then monthly to rule out bone marrow suppression and anaemia as a side effect of Linezolid

e. X-ray: End of intensive phase, end of treatment and whenever clinically indicated.

f. Culture: MDRTB- every month (30days apart) from the 3rd month onwards during intensive phase, then every three months during Continuation phase. Mono resistant-2nd and 3rd month culture followed by 3 monthly culture till completion of treatment.

3. **Long term follow up-** After completion of treatment patient shloud be followed up with clinical and/or sputum examination at 6,12,18 and 24 months.

4. VECTOR BORNE DISEASES

MALARIA

Malaria is a protozoal disease caused by infection with parasites of the genus plasmodium and transmitted to man by female anopheles mosquito. It is a very important public health problem in India particularly due to *Plasmodium falciparum* which is prone to various complications.

Agent: Malaria in man is caused by P*lasmodium vivax, Plasmodium falciparum, Plasmodium ovale* and *Plasmodium malaria*. Out of these, *Plasmodium vivax* and *Plasmodium falciparum* are very common in India including Maharashtra.

Mode of transmission

Direct – through blood or plasma.
Vectors – bite of female anopheles' mosquito.

Incubation Period

The duration varies with species of parasite

12 (9-14) days for falciparum malaria.
14 (8-17) days for vivax malaria

When to suspect

The typical attack comprises three distinct stages viz. cold stage, hot stage and sweating stage.

Cold Stage:

The onset is with lassitude, headache, nausea and chilly sensation followed in an hour or so by rigors. The temperature rises rapidly to 39-41°C. Headache is often severe and commonly there is vomiting. In early part of this stage, skin feels cold; later it becomes hot. Parasites are usually demonstrable in the blood. The pulse is rapid and may be weak. This stage lasts for ¼-1 hour.

Hot Stage:

The patient feels burning hot and casts off his clothes. The skin is hot and dry to touch. Headache is intense but nausea commonly diminishes. The pulse is full and respiration rapid. This stage lasts for 2 to 6 hours.

Sweating Stage:

Fever comes down with profuse sweating. The temperature drops rapidly to normal and skin is cool and moist. The pulse rate becomes slower; patient feels relieved and often falls asleep. This stage lasts for 2-4 hours.

The febrile paroxysms occur with definite intermittent periodicity repeating every third or fourth day depending upon the species of the parasite involved. The classical 3 stages (cold, hot and sweating) may not always be

observed due to maturation of generations of parasite at different times. The disease has a tendency to relapse and is characterized by enlargement of the spleen and secondary anemia.

In patients with *P. falciparum* infection the primary fever in its first few days is usually irregular or even continuous and then the classical 48-hour periodicity becomes established or the fever may continue to be irregular and the hot and cold stages, so typical of other malarial infections are less clearly separated from one another, in persons with poor immunity. The paroxysms are associated with marked prostration. Headache, nausea and vomiting are usually more severe, and there is greater tendency towards the development of delirium, hemolytic jaundice and anemia. The mortality is much greater than in other forms of malaria.

With *P. vivax* infection, symptoms are same but are usually milder and more regularly divided into "hot" and "cold" stages than in *P. falciparum* infections.

Complications

The complications of *P. falciparum* malaria are cerebral malaria, acute renal failure, liver damage, gastro-intestinal symptoms, dehydration, collapse, anemia, black water fever etc. The complications of *P. vivax,* infection are anemia, splenomegaly, enlargement of liver, herpes, renal complications, ARDS etc.

Investigations

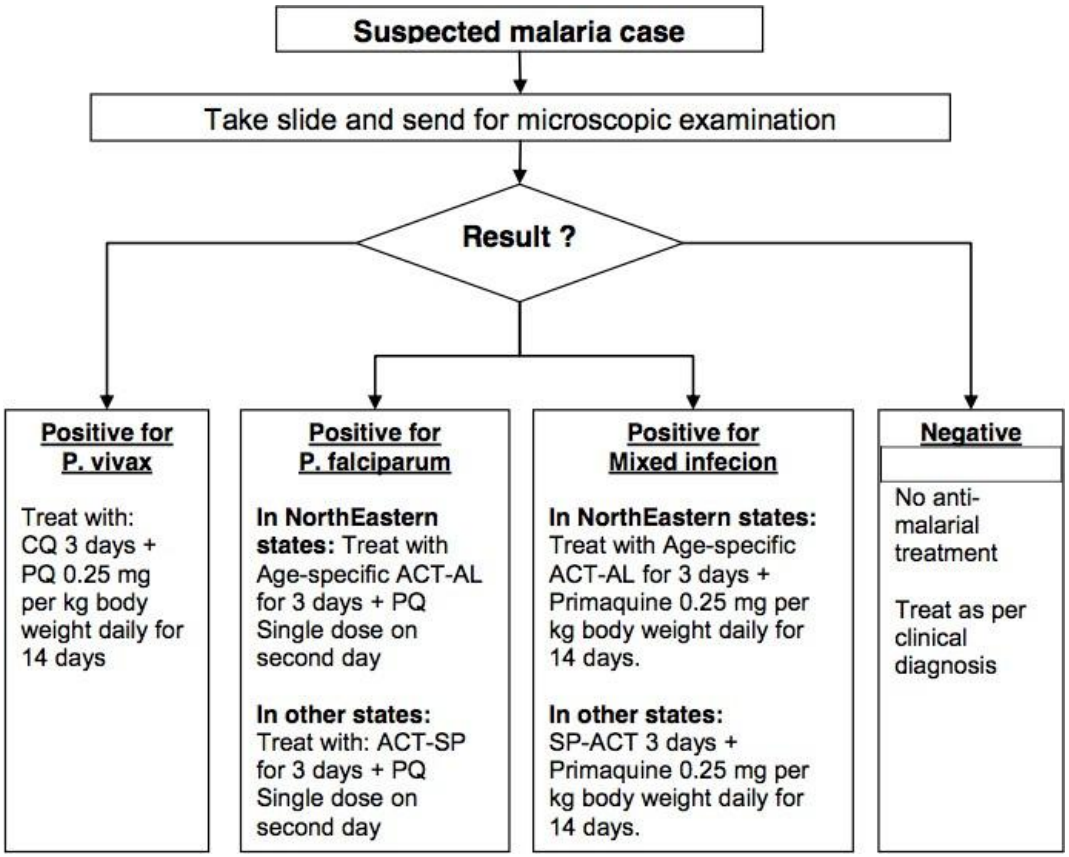
- Diagnosis of Malaria: One of the above clinical features, supported by blood smear examination for malarial parasites.
- Fever with splenomegaly in a patient with the above-mentioned clinical features make diagnosis of malaria more likely.
- Confirmation of diagnosis always depends on seeing the parasite in the blood. In all cases, thick and thin smears should be examined.
- Blood smears may be negative in severe and chronic forms and this would need repeated smears.

Diagnosis of Malaria

- It is stressed that all fever cases should be suspected of malaria after ruling out other common causes and should be investigated for confirmation of malaria by Microscopy or Rapid Diagnostic Kit (RDK) so as to ensure treatment with full therapeutic dose with appropriate drug to all confirmed cases. Presumptive treatment of malaria with a single dose of chloroquine has been stopped.

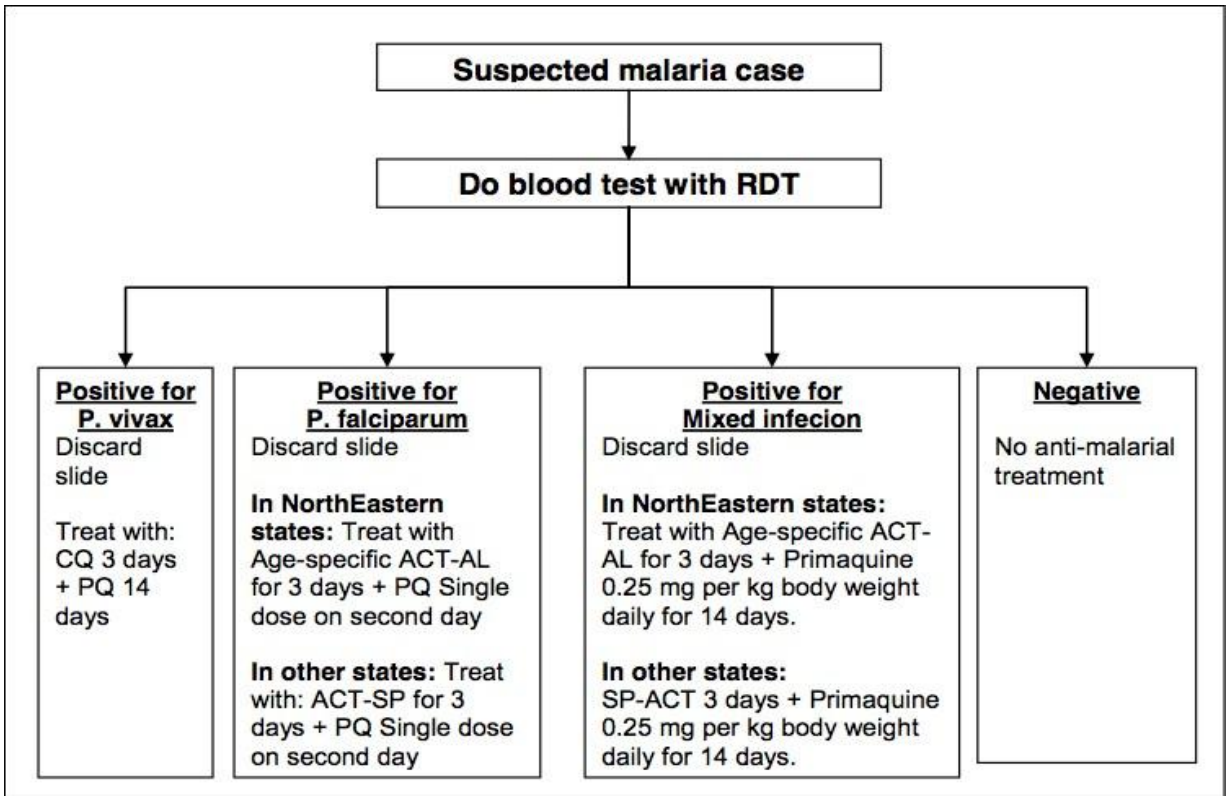
• All fever cases diagnosed as malaria by either RDT or microscopy should be promptly given effective treatment. The medicine chosen will depend upon whether the patient has vivax malaria or falciparum malaria as diagnosed by the blood test. The flow charts in different settings for diagnosis and drug selection for the treatment of malaria are mentioned below.

Where microscopy result is available within 24 hours



ACT-AL - Artemisinin-based Combination Therapy- Artemether - Lumefantrine
ACT-SP- Artemisinin-based Pyrimethamine)CQ - Chloroquine
PQ - Primaquine

Where microscopy result is not available within 24 hours and Bivalent RDT is used



Note: 1) However, if malaria is strongly suspected, prepare & send slide for microscopy

2) If a patient has severe symptoms at any stage, then immediately refer to a nearest PHC or other health facility with indoor patient management or a registered medical doctor.

3) PQ is contra-indicated in pregnancy and in children under 1 year (Infant).

Note: PQ is contra-indicated in pregnancy and in children under 1 year (Infant).

ACT-AL -Artemisinin-based Combination Therapy: Artemether - Lumefantrine ACT-SP-Artemisinin-based Combination Therapy (Artesunate+Sulfadoxine-Pyrimethamine)

CQ - Chloroquine PQ - Primaquine

TREATMENT OF MALARIA

Antimalarial drugs used in public health in India:

- Schizonticidal drugs: Chloroquine, quinine, sulfadoxine-pyrimethamine, artemisinin derivatives (artesunate, arte-mether and arte-ether).
- Gametocytocidal and anti-relapse drug: Primaquine.

All fever cases diagnosed as malaria by RDT or microscopy should promptly be given effective treatment. The treatment will depend upon the species of Plasmodium diagnosed.

A. Treatment of *P. falciparum* malaria

Artemisinin Combination Therapy (ACT) should be given to all the confirmed *P. falciparum* cases found positive by microscopy or RDT.

ACT consists of an artemisinin derivative combined with a long- acting antimalarial (amodiaquine, lumefantrine, mefloquine, piperaquine or sulfadoxine-pyrimethamine). The ACT recommended in the National Programme all over India **except northeastern states** is artesunate (4 mg/kg body weight) daily for 3 days and sulfadoxine (25 mg/kg body weight) -pyrimethamine (1.25 mg/kg body weight) [AS+SP] on Day 0.

In the **northeastern states (Arunachal Pradesh, Assam, Manipur, Meghalaya, Mizoram, Nagaland, and Tripura)**, due to the recent reports of late treatment failures to the current combination of AS+SP in *P. falciparum* malaria, the presently recommended ACT in national drug policy is fixed dose combination (FDC) of **Artemether-lumefantrine (AL)**.

In Other States (other than North-Eastern States):

1. Artemisinin based Combination Therapy (ACT-SP)*

   Artesunate 4 mg/kg body weight daily for 3 days Plus

   Sulfadoxine (25 mg/kg body weight) – Pyrimethamine (1.25 mg/kg body weight) on first day.

   * ACT is not to be given in 1st trimester of pregnancy.

2. Primaquine*: 0.75 mg/kg body weight on day 2.

With the introduction of different colored Blister Packs for different age groups, treatment by the field level staff has been made easy. The color code for different age groups for Packing of Tablet ACT+SP has been given as follows:

Dosage Chart for Treatment of *falciparum* Malaria with ACT-SP

Age group (years)	1st day		2nd day		3rd day
	AS	SP	AS	PQ	AS
0-1 Pink Blister	1 (25 mg)	1 (250mg+12.5mg)	1 (25 mg)	o	1 (25 mg)
1-4 Yellow Blister	1 (50 mg)	1 (500mg+25mg)	1 (50 mg)	1 (7.5mg base each)	1 (50 mg)
5 -8 Green Blister	1 (100 mg)	1 (750mg+37.5mg)	1 (100 mg)	2 (7.5mg base each)	1 (100 mg)
9-14 Red Blister	1 (150 mg)	2 (500mg+25mg)	1 (150 mg)	4 (7.5mg base each)	1 (150 mg)
15 & above White Blister	1 (200 mg)	2 (750mg+37.5mg)	1 (200 mg)	6 (7.5mg base each)	1 (200 mg)

* SP is not to be prescribed for children <5 months of age and should be treated with alternate ACT

In North-Eastern States (NE States):

1. ACT-AL Co-formulated tablet of: Artemether (20 mg) - Lumefantrine (120 mg) (Not recommended during the first trimester of pregnancy and for children weighing < 5 kg)
2. Primaquine*: 0.75 mg/kg body weight on day 2

Recommended regimen by weight and age group

The packing size for different age groups based on Kg bodyweight

Co-formulated tablet ACT-AL	5–14 kg (> 5 months to < 3 years)	15–24 kg (≥ 3 to 8 years)	25–34 kg (≥ 9 to14 years)	> 34 kg (> 14 years)
Total Dose of ACT-AL	20 mg/ 120 mg twice daily for 3 days	40 mg /240 mg twice daily for 3 days	60 mg /360 mg twice daily for 3 days	80 mg /480 mg twice daily for 3 days
	Pack size			
No. of tablets in the Packing	6	12	18	24
Give	1 Tablet twice daily for 3 days	2 Tablets twice daily for 3 days	3 Tablets Twice daily for 3 days	4 Tablets Twice daily for 3 days
Colour of the pack	Yellow	Green	Red	White

Treatment of uncomplicated *P. falciparum* cases in pregnancy 1st

Trimester: Quinine salt 10mg/kg 3 times daily for 7 days.

Quinine may induce hypoglycemia; pregnant women should not start taking quinine on an empty stomach and should eat regularly, while on quinine treatment.

2nd and 3rd Trimesters: Area-specific ACT as per dosage schedule given below. i.e.

ACT-AL in North Eastern States

ACT-SP in Other States

*Primaquine should not be given in pregnancy.

B. Treatment of *P. vivax* malaria

Confirmed *P. vivax* cases should be treated with chloroquine in full therapeutic dose

Chloroquine: 25 mg/kg body weight divided over three days i.e.

10 mg/kg on day 1,
10 mg/kg on day 2 and
5 mg/kg on day 3.

Dosage Chart for Treatment of Vivax Malaria

Primaquine: 0.25 mg/kg body weight daily for 14 days.

Primaquine is contraindicated in infants, pregnant women and individuals with G_6PD deficiency.

14 day regimen of Primaquine should be given under supervision.

Note: CQ 250mg tablet is having 150 mg base

C. Treatment of mixed infections (*P. vivax* + *P. falciparum*) cases:

All mixed infections should be treated with full course of ACT and Primaquine 0.25 mg per kg body weight daily for 14 days.

In North-Eastern States: Treat with: Age-specific ACT-AL for 3 days + Primaquine 0.25 mg per kg body weight daily for 14 days.

In Other States: SP-ACT 3 days + Primaquine 0.25 mg per kg body wt. daily for 14 days.

Dosage Chart for Treatment of mixed (vivax and falciparum) Malaria with ACT-SP

Age	Day 1		Day 2		Day 3		Day 4 to 14
	CQ (150 mg base)	PQ (2.5 mg)	CQ (150 mg base)	PQ (2.5 mg)	CQ (150 mg base)	PQ (2.5 mg)	PQ (2.5 mg)
Less than 1 year	½	0	½	0	¼	0	0
1 – 4 years	1	1	1	1	½	1	1
5 – 8 years	2	2	2	2	1	2	2
9 – 14 years	3	4	3	4	1½	4	4
15 years or more	4	6	4	6	2	6	6
Pregnancy	4	0	4	0	2	0	0

Age	Day 1			Day 2		Day 3		Days 4-14
	AS tablet (50 mg)	SP tablet	PQ (2.5 mg)	AS tablet (50 mg)	PQ (2.5 mg)	AS tablet (50 mg)	PQ (2.5 mg)	PQ (2.5 mg)
Less than 1 yr	½	½	0	½	0	½	0	0
1-4 years	1	1	1	1	1	1	1	1
5-8 years	2	1 ½	2	2	2	2	2	2
9-14 years	3	2	4	3	4	3	4	4
15 yrs or more	4	3	6	4	6	4	6	6

*All cases of mixed infection are to be treated as *Pf* as per the drug policy applicable in the area plus primaquine for 14 days.

D. Treatment of severe malaria cases

Severe malaria is an emergency and treatment should be given as per severity and associated complications, which can be best, decided by the treating physicians. Before admitting or referring patients, the attending doctor or health worker, whoever is able to do it, should do RDT and take blood smear; give a parenteral dose of artemisinin derivative or quinine in suspected cerebral malaria cases and send case sheet, details of treatment history and blood slide with patient. Parenteral artemisinin derivatives or quinine should be used irrespective of chloroquine resistance status of the area with one of the following options:

Chemotherapy of severe and complicated malaria

Initial parenteral treatment for at least 48 hours: **CHOOSE ONE of following four options**	**Follow-up treatment, when patient can take oral medication following parenteral treatment**
Quinine: 20mg quinine salt/kg body weight on admission (IV infusion or divided IM injection) followed by maintenance dose of 10 mg/kg 8 hourly; infusion rate should not exceed 5 mg/kg per hour. Loading dose of 20mg/kg should not be given, if the patient has already received quinine.	**Quinine** 10 mg/kg three times a day with: doxycycline 100 mg once a day **or** clindamycin in pregnant women and children under 8 years of age, - to complete 7 days of treatment.
Artesunate: 2.4 mg/kg i.v. or i.m. given on admission (time=0), then at 12 h and 24 h, then once a day. **or** **Artemether:** 3.2 mg/kg bw i.m. given on admission then 1.6 mg/kg per day. or **Arteether:** 150 mg daily i.m for 3 days in adults only (not recommended for children).	Full oral course of Area-specific ACT: **In NorthEastern states:** Age-specific ACT-AL for 3 days + PQ Single dose on second day **In other states:** Treat with: ACT-SP for 3 days + PQ Single dose on second day

Note: The parenteral treatment in severe malaria cases should be given for minimum of 24 hours once started (irrespective of the patient's ability to tolerate oral medication earlier than 24 hours).

After parenteral artemisinin therapy, patients will receive a full course of Area-specific oral ACT for 3 days. Those patients who received parenteral Quinine therapy should receive oral Quinine 10 mg/kg body weight three times a day for 7 days (including the days when parenteral Quinine was administered) plus Doxycycline 3 mg/kg body weight once a day or Clindamycin 10 mg/kg body weight 12-hourly for 7 days (Doxycycline is contraindicated in pregnant women and children under 8 years of age) or area-specific ACT as described.

Note:

- Pregnant women with severe malaria in any trimester can be treated with artemisinin derivatives, which, in contrast to quinine, do not risk aggravating hypoglycaemia.

- The parenteral treatment should be given for minimum of 48 hours
- Once the patient can take oral therapy, give:
  - ▶ Quinine 10 mg/kg three times a day with doxycycline 100 mg once a day or clindamycin in pregnant women and children under 8 years of age, to complete 7 days of treatment, in patients started on parenteral quinine.
  - ▶ Full course of ACT to patients started on artemisinin derivatives.
- Use of mefloquine should be avoided in cerebral malaria due to neuropsychiatric complications associated with it.

E. Chemoprophylaxis:

Chemoprophylaxis should be administered only in selective groups in high *P. falciparum* endemic areas. Use of personal protection measures including Insecticide Treated bed Nets (ITN) / Long Lasting Insecticidal Nets (LLIN) should be encouraged for pregnant women and other vulnerable population including travelers for longer stay. However, for longer stay of Military and Para-military forces in high *Pf* endemic areas, the practice of chemoprophylaxis should be followed wherever appropriate e.g., troops on night patrol duty and decisions of their Medical Administrative Authority should be followed.

e.1. Short term chemoprophylaxis (up to 6 weeks)

Doxycycline: 100 mg once daily for adults and

1.5 mg/kg once daily for children (contraindicated below 8 years)

The drug should be started 2 days before travel and continued for 4 weeks after leaving the malarious area.

Note: It is not recommended for pregnant women and children less than 8 years.

e.2. Chemoprophylaxis for longer stay (more than 6 weeks)

Mefloquine: 250 mg weekly for adults and should be administered two weeks before, during and four weeks after exposure.

Note: Mefloquine is contraindicated in individuals with history of convulsions, neuropsychiatric problems and cardiac conditions. Therefore, necessary precautions should be taken and all should undergo screening before prescription of the drug.

DENGUE

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, may lead to dengue fever (DF), dengue haemorrhagic fever (DHF) or when associated with plasma leakage may lead to hypovolemic shock and dengue shock syndrome (DSS).

Salient features:

Common Conditions

- 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.
- Haemorrhagic 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, melena
- Thrombocytopenia (platelets 100,000 cells/mm3 or less) and evidence of plasma leakage.

Dengue shock syndrome (DSS)

- All the above criteria of DHF plus signs of circulatory failure.

Notes: The tourniquet test is performed by inflating a blood pressure cuff to mid-way between the systolic and diastolic pressure.

Non pharmacological treatment

- Bed rest is advisable during the acute phase.
- Use cold sponging to keep temperature below

Pharmacological treatment

- Management of dengue fever is symptomatic and supportive
- Antipyretics may be used to lower the body temperature. Aspirin/NSAIDs like ibuprofen etc should be avoided since it may cause gastritis, vomiting, acidosis and platelet dysfunction.
- Paracetamol is preferable in the doses as follows:
    - 1-2 years: 60 –120 mg/dose
    - 3-6 years: 120 mg/dose
    - 7-12 years: 240 mg/dose
    - Adult : 500mg/dose

  Note: In children the dose is calculated as per 10mg/kg body weight per dose which can be repeated at the int
    - erval of 6 hrs.
- Oral fluid and electrolyte therapy are recommended for patients with excessive sweating or vomiting.
- Patients should be monitored in DHF endemic area until they become afebrile for one day without the use of antipyretics and after platelet and haematocrit determinations are stable, platelet count is more than 50,000/mm .

Management of Dengue Haemorrhagic Fever (Febrile Phase)

- The management of febrile phase is similar to that of DF.

- Paracetamol is recommended to keep the temperature below $39^{O}C$. Copious amount of fluid should be given orally, to the extent the patient tolerates, oral hydration solution (ORS), such as those used for the treatment of diarrhoeal diseases and/or fruit juices are preferable to plain water

- IV fluid may be administered if the patient is vomiting persistently or refusing to feed.

- Patients should be closely monitored for the initial signs of shock. The critical period is during the transition from the febrile to the afebrile stage and usually occurs after the third day of illness.

- Serial haematocrit determinations are essential guide for treatment, since they reflect the degree of plasma leakage and need for intravenous administration of fluids.

- Haematocrit should be determined daily from the third day until the temperature has remained normal for one or two days. If haematocrit determination is not possible, haemoglobin determination may be carried out as an alternative.

- The details of IV treatment when required for patients are given in Figure

Management of DHF Grade I and Grade II:

- Any person who has dengue fever with thrombocytopenia and haemoconcentration and presents with abdominal pain, black tarry stools, epistaxis, bleeding from the gums and infection etc needs to be hospitalized.

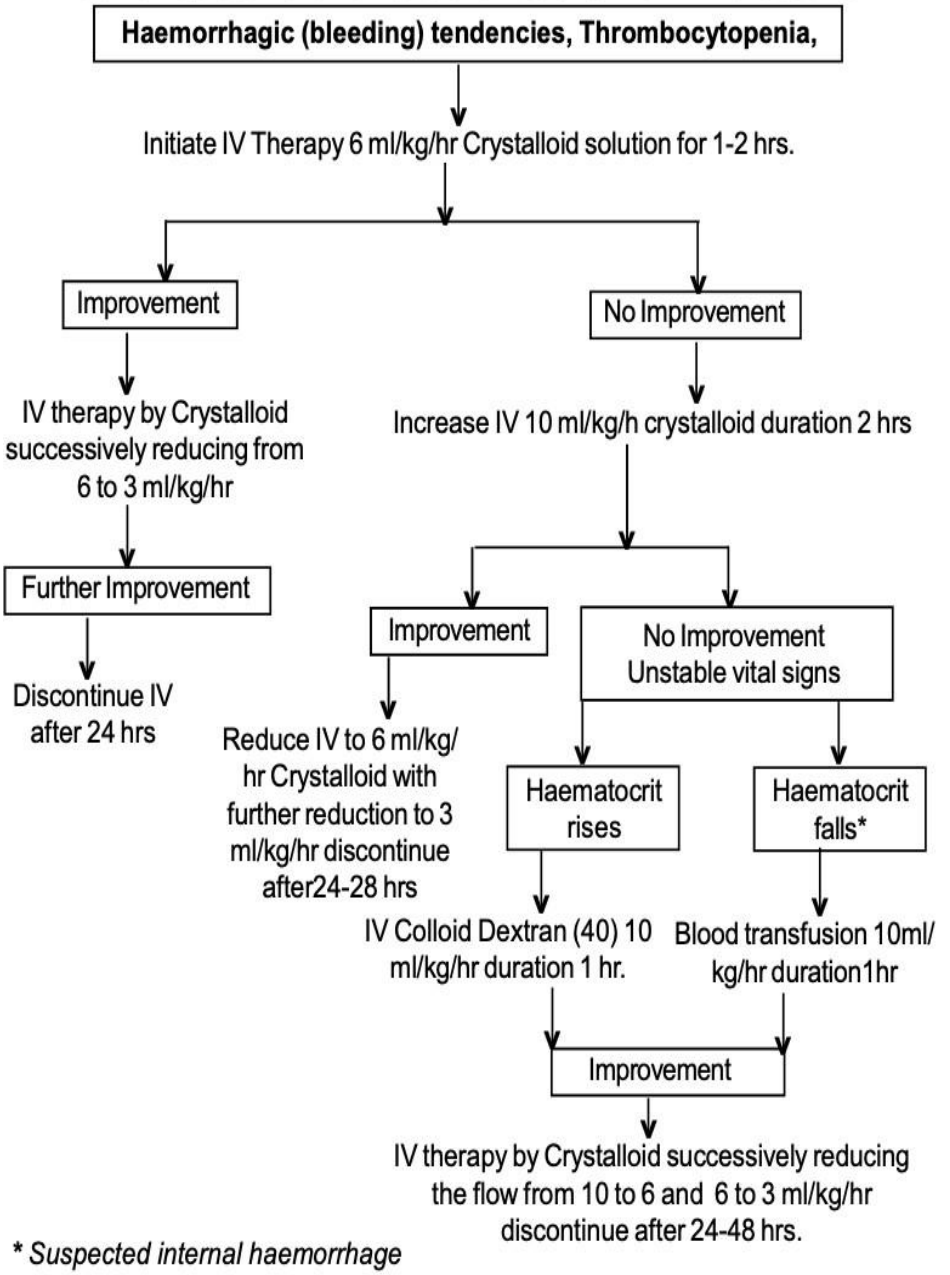
- All these patients should be observed for signs of shock. The critical period for development of shock is transition from febrile to afebrile phase of illness, which usually occurs after third day of illness.

- A rise of haemoconcentration indicates need for IV fluid therapy. If despite the treatment, the patient develops fall in BP, decrease in urine output or other features of shock, the management for Grade III/IV DHF/DSS should be instituted.

- Oral rehydration should be given along with antipyretics like paracetamol sponging, etc. as described above.

- The detailed treatment for patient with DHF Grade I and II is given at Figure 5. Common signs of complications are observed during the afebrile phase of DHF. Immediately after hospitalization, the haematocrit, platelet count and vital signs should be examined to assess the patient's condition and intravenous fluid therapy should be started. The patient requires regular and sustained monitoring.

Volume Replacement Flow Chart for Patients with DHF Grades I & II

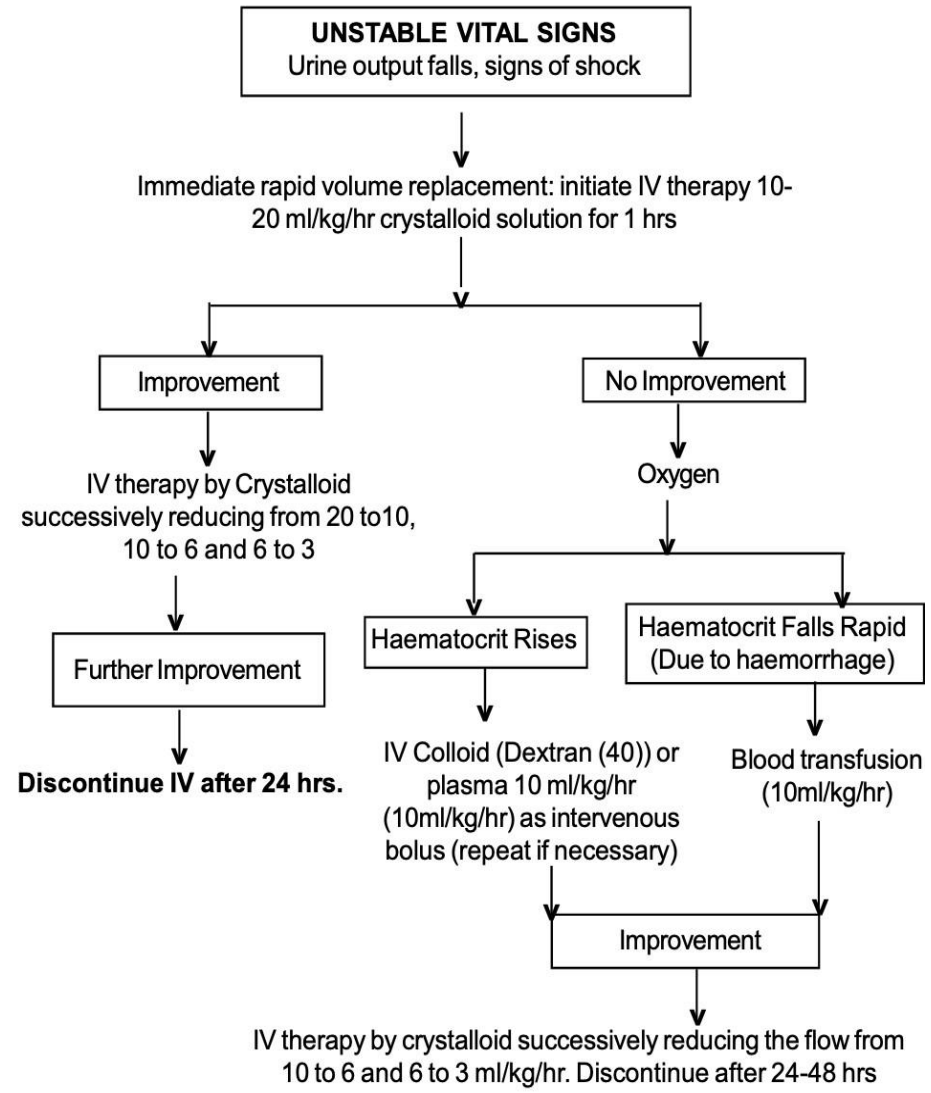


** Suspected internal haemorrhage*

Improvement : Haematocrit falls, pulse rate and blood pressure stable, urine output rises

No Improvement : Haematocrit or pulse rate rises, pulse pressure falls below 20 mmHg, Urine output falls

Unstable Vital signs : Urine output falls, signs of shock



- Serial platelet and haematocrit determinations: drop in platelets and rise in haematocrit are essential for early diagnosis of DHF.
- Cases of DHF should be observed every hour for vital signs and urinary output.

Fluid requirement

- The volume of fluid required to be replaced should be just sufficient to maintain effective circulation during the period of plasma leakage. To ensure adequate fluid replacement and avoid over-fluid infusion, the rate of intravenous fluid should be adjusted throughout the 24 to 48 hour period of plasma leakage by periodic haematocrit determinations and assessment of vital signs.

- The required regimen of fluid should be calculated on the basis of body weight and charted on a 1-3 hourly basis, or more frequently in the case of shock. The flow of fluid and the time of infusion are dependent on the severity of DHF. The schedule given below is a guideline and calculated for moderate dehydration of about 6% deficit (plus maintenance) (Table 5).

Table: Fluid requirement as per body weight of the patient

Weight on admission (kg)	Fluid requirement/kg body weight/day (ml/kg)
<7	220
7-11	162
12-18	130
>18	90

In older children who weigh more than 40 kg, the volume needed for 24 hours should be calculated as twice that required for maintenance (using the Holiday and Segar formula). The maintenance fluid should be calculated as follows:

Maintenance fluid requirement according to Holiday and Segar formula

Body weight in kg	Maintenance volume for 24 hours
<10kg	100 ml / kg
10-20kg	1000+50 ml / kg
>20kg	1500+20 ml / kg

For a child weighing 40 kgs, the maintenance is: 1500 + (20x20) = 1900 ml. This means that the child requires 3800 ml IV fluid during 24 hours.

Indications of red cell transfusion

- Loss of blood (overt blood) -10% or more of total blood volume
- Preferably whole blood/ component to be used
- Refractory shock despite adequate fluid administration and declining haematocrit - replacement volume should be 10 ml/kg body weight at a time and coagulogram should be done.
- If fluid overload is present, PCV is to be given
-

Indications of platelet transfusion

- Prophylactic platelet transfusion may be given at level of less than 10,000 cells/mm^3 in absence of bleeding manifestations.
- Prolonged shock; with coagulopathy and abnormal coagulogram.
- In case of systemic massive bleeding, platelet transfusion may be needed in addition to red cell transfusion.

CHIKUNGUNYA

Chikungunya is caused by an alpha virus closely related to O' nyong – nyong virus. The main vector is *Aedes aegypti* mosquito.

Salient features

- Acute self-limiting illness Incubation period is of 2 to 4 days. Abrupt onset presenting as fever with severe joint pain
- After 1 – 4 days, fever subsides, there will be a afebrile period 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 up to 4 months, in some cases even
- up to 5 years.

Non pharmacological treatment

- Mild exercises and physiotherapy may be suggested in recovering persons.
- Exposure to warm environment may be suggested
- Non weight bearing exercises
- Surgery in severely damaged joints

Pharmacological treatment

- Acute stage of illness
- Treat symptomatically - Tab. paracetamol 1 gm 3-4 times a day for fever, headache and pain
- Avoid aspirin and steroid because of risk of GI side effects and Reye's syndrome with aspirin.
- Tab. hydroxychloroquine 200mg orally once daily or Tab. chloroquine phosphate 300mg per day for a period of 4 weeks in cases where arthralgia is refractory to other drugs.
- Side effects of hydroxychloroquine include retinal damage and elevated liver enzymes.

CRITICAL CARE UNIT STANDARD TREATMENT GUIDELINES

Anaphylaxis definitions:

- Any acute onset illness with typical skin features (urticarial rash or erythema/flushing, and/or angioedema)
- With or without involvement of respiratory and/or cardiovascular and/or persistent severe gastrointestinal symptoms

or

- Any acute onset of hypotension or bronchospasm or upper airway obstruction where anaphylaxis is considered possible, even if typical skin features are not present.

The most common triggers of anaphylaxis (severe allergic reaction) are foods, insect stings and drugs (medications).

Signs and symptoms of allergic reactions: -

1) Mild or moderate reactions:

- Swelling of lips, face, eyes
- Hives or welts
- Tingling mouth
- Abdominal pain, vomiting (these are signs of anaphylaxis for insect sting or injected drug (medication) allergy

2) Anaphylaxis – Indicated by any one of the following signs:

- Difficult/noisy breathing
- Swelling of tongue
- Swelling/tightness in throat
- Difficulty talking and/or hoarse voice
- Wheeze or sudden persistent cough which is sudden onset unlike cough in asthma
- Persistent dizziness or collapse
- Pale and floppy (young children)
- Abdominal pain, vomiting (for insect sting or injected drug (medication) allergy).

3) Immediate actions to be taken:

- Remove allergen (if still present).
- Call for assistance.
- Lay patient flat. Do not allow patient to stand or walk. Do not hold infants upright. If breathing is difficult, allow the patient to sit.
- Give INTRAMUSCULAR (IM) INJECTION ADRENALINE (epinephrine) into outer mid-thigh without delay using an adrenaline autoinjector if available OR adrenaline ampoule and syringe.

- Give oxygen.
- ALWAYS give adrenaline FIRST, then asthma reliever if someone with known asthma and allergy to food, insects or medication has SUDDEN BREATHING DIFFICULTY (including wheeze, persistent cough* or hoarse voice) even if there are no skin symptoms.

*Unlike the cough in asthma, the onset of coughing during anaphylaxis is usually sudden.

• If required at any time, commence cardiopulmonary resuscitation (CPR).

Adrenaline administration and dosages according:

- Adrenaline (epinephrine) is the first line treatment of anaphylaxis and acts to reduce airway mucosal oedema, induce bronchodilation, induce vasoconstriction and increase strength of cardiac contraction.
- Give INTRAMUSCULAR (IM) INJECTION OF ADRENALINE (1:1000) into outer mid-thigh (0.01mg per kg up to 0.5mg per dose) without delay using an adrenaline autoinjector if available OR adrenaline ampoule and syringe, as follows.

Adrenaline (epinephrine) dosages chart

Age (years)	Weight (kg)	Vol. adrenaline 1:1000	Adrenaline autoinjector
~<1	<7.5kg	0.1 mL	Not available
~1-2	10	0.1 mL	7.5*-20 kg (~<5 yrs) 0.15mg device (e.g. EpiPen Jr)
~2-3	15	0.15 mL	
~4-6	20	0.2 mL	
~7-10	30	0.3 mL	>20kg (~>5yrs) 0.3mg device (e.g. EpiPen)
~10-12	40	0.4 mL	
~>12 and adults	>50	0.5 mL	

Note:

- If multiple doses are required for severe reactions (e.g. 2-3 doses administered at 5 minutes intervals), consider adrenaline infusion.
- For emergency treatment of anaphylaxis, ampoules of adrenaline 1:1000 should be used for both IM doses and infusion if required.
- IV adrenaline infusions should be used with a dedicated line, infusion pump and anti-reflux valves wherever possible
- Infants with anaphylaxis may retain pallor despite 2-3 doses of adrenaline, and this can resolve without further doses. More than 2-3 doses of adrenaline in infants may cause hypertension and tachycardia.
- Pregnant women experiencing anaphylaxis need to be treated without delay and there are no absolute contraindications to adrenaline use in anaphylaxis. If clinical judgement deems that there is a risk of maternal death or foetal compromise due to inadequately treated anaphylaxis, then in pregnant women weighing > 50kg, consider giving 500 mcg IM adrenaline.

Positioning of patients:

- Laying the patient flat will improve venous blood return to the heart.
- By contrast, placing the patient in an upright position, including holding infants upright over a shoulder, can impair blood returning to the heart, resulting in insufficient blood for the heart to circulate and low blood pressure.
- The left lateral position is recommended for patients who are pregnant to reduce the risk of compression of the inferior vena cava by the pregnant uterus and thus impairing venous return to the heart.
- Fatality can occur within minutes if a patient stands or sits suddenly. For mainly respiratory reactions, the patient may prefer to sit and this may help support breathing and improve ventilation. BEWARE that even sitting may trigger hypotension. Monitor closely. Immediately lay the patient flat again, if there is an alteration in conscious state or drop in blood pressure.
- If vomiting, lay the patient on their side (recovery position).
- Patients must not be walked to/from the ambulance, even if they appear to have recovered.

Supportive management:

- Check level of consciousness
- Check pulse, blood pressure, ECG, pulse oximetry
- Give high flow oxygen and airway support if needed
- Obtain IV access in adults and hypotensive children
- If hypotensive, give IV normal saline 20mL/kg rapidly and consider additional wide bore IV access

Additional measures to consider if IV adrenaline infusion is ineffective:

For Upper airway obstruction	• Nebulized adrenaline (5mL i.e. 5 ampoules of 1:1000). • Consider need for advanced airway management if skills and equipment are available.
For persistent hypotension/ shock	• Give normal saline (maximum of 50mL/kg in first 30 minutes). • Glucagon • In adults, selective vasoconstrictors only after advice from an emergency medicine/critical care specialist.
For persistent wheeze	Bronchodilators: Salbutamol 8 - 12 puffs of 100µg using a spacer OR 5mg salbutamol by nebulizer. Note: Bronchodilators do not prevent or relieve upper airway obstruction, hypotension or shock. Corticosteroids: Oral prednisolone 1 mg/kg (maximum of 50 mg) or intravenous hydrocortisone 5 mg/kg (maximum of 200 mg). Note: Steroids must not be used as a first line medication in place of adrenaline

Antihistamines and corticosteroids:

Antihistamines-

- Antihistamine therapy considered to be adjunctive to epinephrine so not to be administered alone
- Combination of H1 blocker and an H2 blocker considered to be superior to an H1 blocker alone in relieving the histamine-mediated symptoms.
- Diphenhydramine and ranitidine are an appropriate combination. IV administration ensures that effective dosing is not impaired by hemodynamic compromise, which adversely affects gastrointestinal (GI) or IM absorption. However, oral or IM administration of antihistamines may suffice for milder anaphylaxis.

Corticosteroids-

- Intravenous Corticosteroid recommended for all patients with anaphylaxis and to be administer early to prevent a potential late-phase reaction (biphasic anaphylaxis).
- Patients with asthma or other conditions recently treated with a corticosteroid may be at increased risk for severe or fatal anaphylaxis and may receive additional benefit if corticosteroids are administered to them during anaphylaxis.

Patient to be observed for at least 4 hours after last dose of adrenaline: Relapse, protracted and/or biphasic reactions may occur. Patients require overnight observation if they-

- Had a severe or protracted anaphylaxis (e.g. required repeated doses of adrenaline or IV fluid resuscitation), OR
- Have a history of asthma or severe/protracted anaphylaxis, OR
- Have other concomitant illness (e.g. asthma, history or arrhythmia), OR
- Live alone or are remote from medical care, OR
- Present for medical care late in the evening.

(True biphasic reactions are estimated to occur following 3-20% of anaphylactic reactions).

Preparation:

Equipments required for acute management of anaphylaxis-

- Adrenaline 1:1000 (consider adrenaline autoinjector availability, particularly in rural locations, for initial administration by nursing staff)
- 1mLor 2mL syringes
- Oxygen
- Airway equipment, including nebuliser and suction
- Defibrillator
- Manual blood pressure cuff
- IV access equipment (including large bore canulae)
- At least 3 litres of normal saline
- A hands-free phone in resuscitation room, to allow health care providers in remote locations to receive instructions by phone whilst keeping hands free for resuscitation.

Advanced Acute Management of Anaphylaxis:

Supportive management-

- Monitor pulse, blood pressure, respiratory rate, pulse oximetry, conscious state.
- Give high flow oxygen (6-8 L/min) and airway support if needed.
- Supplemental oxygen should be given to all patients with respiratory

distress, reduced conscious level and those requiring repeated doses of adrenaline.

- Supplemental oxygen should be considered in patients who have asthma, other chronic respiratory disease, or cardiovascular disease.
- Obtain intravenous (IV) access in adults and in hypotensive children.
- If hypotensive:
  - Give intravenous normal saline (20 mL/kg rapidly under pressure), and repeat bolus if hypotension persists.
  - Consider additional wide bore intravenous access.

(During severe anaphylaxis with hypotension, marked fluid extravasation into the tissues can occur so DO NOT FORGET FLUID RESUSCITATION)

Assess circulation to reduce risk of overtreatment:

- Monitor for signs of overtreatment (especially if respiratory distress or hypotension were absent initially) – including pulmonary oedema, hypertension.
- In this setting (anaphylaxis) it is recommended that, if possible, a simple palpable systolic blood pressure (SBP) should be measured:
  - Attach a manual BP cuff of an appropriate size and find the brachial or radial pulse.
  - Determine the pressure at which this pulse disappears/reappears (the "palpable" systolic BP).
  - This is a reliable measure of initial severity and response to treatment
  - Measurement of palpable SBP may be more difficult in children.

  (Note: If a patient is nauseous, shaky, vomiting, or tachycardic but has a normal or elevated SBP, this may be adrenaline toxicity (side effects) rather than worsening anaphylaxis.)

The protocol for 1000 mL normal saline is as follows:

- Mix 1 mL of 1:1000 adrenaline in 1000 mL of normal saline.
- Start infusion at ~5 mL/kg/hour (~0.1 microgram/kg/minute).
- If you do not have an infusion pump, a standard giving set administers

~20 drops per mL, therefore, start at ~2 drops per second for an adult.

- Titrate rate up or down according to response and side effects.
- Monitor continuously – ECG and pulse oximetry and frequent non- invasive blood pressure measurements as a minimum to maximise benefit and minimise risk of overtreatment and adrenaline toxicity.

Note:

- This protocol is intended for temporary use, when no infusion pump is available.
- Most anaphylactic reactions settle with only 1 mg adrenaline in 1 litre.
- Indefinite continuation of low concentration infusion increases risk of fluid overload.
- Caution - Intravenous boluses of adrenaline are NOT recommended due to risk of cardiac ischaemia or arrhythmia UNLESS the patient is in cardiac arrest.

Additional measures:

	Additional measures to consider if IV adrenaline infusion is ineffective
For persistent hypotension/shock	• Give normal saline (maximum of 50mL/kg in first 30 minutes). • In patients with cardiogenic shock (especially if taking beta blockers) consider an intravenous glucagon bolus of: - 1-2mg in adults - 20-30 microgram/kg up to 1mg in children This may be repeated or followed by an infusion of 1-2mg/hour in adults. • In adults, selective vasoconstrictors metaraminol (2-10mg) or vasopressin (10- 40 units) only after advice from an emergency medicine/critical care specialist. Beware of side effects including arrhythmias, severe hypotension and pulmonary oedema. • In children, metaraminol 10 micrograms/kg/dose can be used. Noradrenaline infusion may be used in critical care setting.

Advanced airway management:

- Oxygenation is more important than intubation per se.
- Always call for help from the most experienced person available.
- If airway support is required, first use the skills you are most familiar with (e.g. jaw thrust, Guedel or nasopharyngeal airway, bag-valve-mask with high flow oxygen attached). This will save most patients, even those with apparent airway swelling (these patients have often stopped breathing due to circulatory collapse rather than airway obstruction and can be adequately ventilated with basic life support procedures).
- DO NOT make prolonged attempts at intubation (since the patient is not getting any oxygen while intubation is being attempted)
- If unable to maintain an airway and the patient's oxygen saturation is falling further approaches to the airway (e.g. cricothyrotomy) should be considered in accordance with established difficult airway management protocols.

Special situation: Overwhelming anaphylaxis (cardiac arrest)- Key points:

- Massive vasodilatation and fluid extravasation.
- Unlikely that IM adrenaline will be absorbed in this situation due to poor peripheral circulation.
- Even if absorbed, IM adrenaline on its own may be insufficient to overcome vasodilatation and extravasation.
- Need both IV adrenaline bolus (cardiac arrest protocol, 1 mg every 2-3 minutes)
- Aggressive fluid resuscitation in addition to CPR (Normal Saline 20mL/kg stat, through a large bore IV under pressure, repeat if no response).
- Do not give up too soon - this is a situation when prolonged CPR should be considered, because the patient arrested rapidly with previously normal tissue oxygenation, and has a potentially reversible cause.
- Consider extracorporeal membrane oxygenation (ECMO) if resource is available.

DIABETIC KETOACIDOSIS

Diagnostic criteria:

- Serum glucose >250mg/dl
- Arterial pH <7.3
- Serum bicarbonate <18mEq/L
- Ketonuria or ketonemia

Management:

1. **Complete history and initial evaluation/physical examination/ Look for precipitating cause.**
2. **Investigations:**
   - Capillary glucose to confirm hyperglycemia
   - serum/urine ketones for presence of ketonemia/ketonuria
   - Arterial blood gas for metabolic profile before initiation of intravenous fluids
   - Complete blood count
   - Serum glucose level
   - BUN
   - Serum electrolytes
   - Chemistry profile
   - Creatinine level
   - Urinalysis
   - ECG
   - CXR if needed
   - Specimens for bacterial cultures
3. **Correction of Fluid Loss:**
   a) A crystalloid fluid is the initial fluid of choice for Initial correction of fluid loss either by isotonic sodium chloride solution or by lactated Ringer solution. The recommended schedule for restoring fluids is as follows:
      - Administer 1-1.5 L during the first hour.
      - Administer 1 L during the second hour.
      - Administer 1 L during the following 2 hours
      - Administer 1 L every 4 hours, depending on the degree of dehydration and central venous pressure readings

   b) When the patient becomes euvolemic, the physician may switch to half the isotonic sodium chloride solution, particularly if hypernatremia exists. Isotonic saline should be administered at a rate appropriate to maintain adequate blood pressure and pulse, urinary output, and mental status.

   c) If a patient is severely dehydrated and significant fluid resuscitation is needed, switching to a balanced electrolyte solution may help to avoid

the development of a hyperchloremic acidosis.

d) When blood sugar decreases to less than 180 mg/dL, isotonic sodium chloride solution is replaced with 5-10% dextrose with half isotonic sodium chloride solution.

e) After initial stabilization with isotonic saline, switch to half-normal saline at 200-1000 mL/h (half-normal saline matches losses due to osmotic diuresis).

Insulin Therapy:

- Start insulin therapy about an hour after IV fluid replacement and only if serum K >3.3 mmol/L
- The initial insulin dose is a continuous IV insulin infusion by infusion pump at a rate of 0.1 U/kg/h. A mix of 24 units of regular insulin in 60 mL of isotonic sodium chloride solution usually is infused at a rate of 15 mL/h (6 U/h) until the blood glucose level drops to less than 180 mg/dL; the rate of infusion then decreases to 5-7.5 mL/h (2-3 U/h) until the ketoacidotic state abates.
- When plasma glucose reaches 200–250 mg/dL, the insulin rate can be decreased by 50% or to the rate of 0.02–0.05 U/kg/h or
- In absence of infusion pump, larger volumes of an insulin and isotonic sodium chloride solution mixture can be used, providing that the infusion dose of insulin is similar (eg, 60 U of insulin in 500 mL of isotonic sodium chloride solution at a rate of 50 mL/h).
- The optimal rate of glucose decline is 100 mg/dL/h. Do not allow the blood glucose level to fall below 200 mg/dL during the first 4-5 hours of treatment. Hypoglycemia may develop rapidly with correction of ketoacidosis due to improved insulin sensitivity.
- Blood glucose level to be checked hourly if insulin is given by infusion.

Points to remember with insulin therapy:

- A low-dose insulin regimen has the advantage of not inducing the severe hypoglycemia or hypokalemia that may be observed with a high-dose insulin regimen.
- Only short-acting insulin to be used for correction of hyperglycemia.
- Subcutaneous absorption of insulin is reduced in DKA because of dehydration; therefore, using intravenous routes is preferable.

Electrolyte Correction:

- If the potassium level is greater than 6 mEq/L, do not administer potassium supplement.
- If the potassium level is 4.5-6 mEq/L, administer 10 mEq/h of potassium chloride.
- If the potassium level is 3-4.5 mEq/L, administer 20 mEq/h of potassium chloride.
- Monitor serum potassium levels 6-8 hourly (hourly monitoring is advocated if possible), and the infusion must be stopped if the potassium level is greater than 5 mEq/L. The monitoring of serum potassium must continue even after potassium infusion is stopped in case of (expected) recurrence of hypokalemia.
- In severe hypokalemia, not starting insulin therapy is advisable unless potassium replacement is under way; this is to avert potentially serious cardiac dysrhythmia that may result from hypokalemia.

Correction of Acid-Base Balance:

- Sodium bicarbonate is infused only if decompensated acidosis starts to threaten the patient's life, especially when associated with either sepsis or lactic acidosis.

Treatment of Concurrent Infection:

- Blood and urine culture and sensitivity
- Start empiric antibiotics on suspicion of infection until culture results are available.

DVT prophylaxis:

- DVT prevention either by low molecular weight heparin or conventional heparin OR
- By graduated compression stockings or sequential compression device in patient where heparin is contraindicated.

Management of Treatment-Related Complications:

Cerebral edema-

- 0.5-1 g/kg intravenous mannitol over 20 minutes.
- Repeat if no response is seen in 30-120 minutes.
- If no response to mannitol, give hypertonic saline (3%) at 5-10 mg/kg over 30 minutes.

Cardiac dysrhythmia-

Cardiac dysrhythmia may occur secondary to severe hypokalemia and/or acidosis either initially or as a result of therapy in patients with DKA. Usually,

- Correction of the cause sufficient to treat cardiac dysrhythmia,
- If it persists, consultation with a cardiologist mandatory.
- Important to continue cardiac monitoring on patients with DKA during correction of electrolytes.

Pulmonary edema-

- Although initial aggressive fluid replacement is necessary in all patients, particular care must be taken in those with comorbidities such renal failure or congestive heart failure.
- Diuretics and oxygen therapy often suffice for the management of pulmonary edema.

Myocardial injury-

- Myocardial biomarkers (troponin T and CK-MB) and
- ECG to be done

Diabetic retinopathy-

- Microvascular changes consistent with diabetic retinopathy have been reported prior to and after treatment of diabetic ketoacidosis.

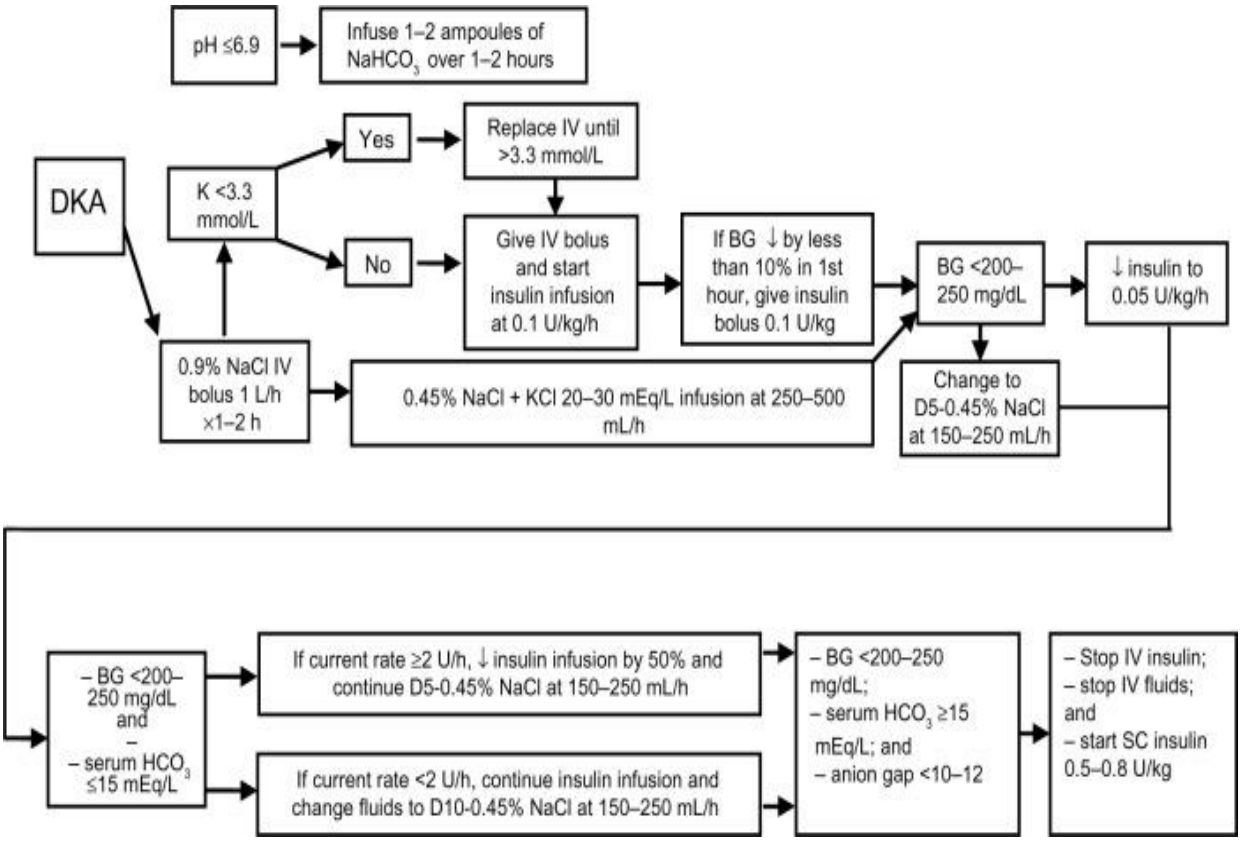
Hypoglycemia-

- In patients with diabetic ketoacidosis, hypoglycemia may result from inadequate monitoring of glucose levels during insulin therapy. Insulin sensitivity improves after clearance of ketones.

Hypokalemia-

- Hypokalemia is a complication that is precipitated by failing to rapidly address the total body potassium deficit brought out by rehydration and insulin treatment, which not only reduces acidosis but directly facilitates potassium re-entry into the cell.

WORKFLOW OF MANAGEMENT OF ADULT DKA



Checklist of DKA management milestones

Phase I (0–6 h)	Phase II (6–12 h)	Phase III (12–24 h)
Perform history and physical exam and order initial laboratory studies	Continue biochemical and clinical monitoring	Continue biochemical and clinical monitoring
Implement monitoring plan (biochemical and clinical)	Change isotonic fluids to hypotonic fluids if corrected Na normal/high	Adjust therapy to avoid complications
Give intravenous bolus of isotonic fluids	If glucose is <200–250 mg/dL, add dextrose to intravenous fluids	Address precipitating factors

	Start insulin therapy (after fluids started and only if K >3.3 mmol/L)	Adjust insulin infusion rate as needed	If DKA resolved, stop intravenous insulin and start subcutaneous insulin
	Consult diabetes team	Maintain K at 3.3–5.3 mmol/L range	Consult diabetes educator

INITIAL MANAGEMENT OF TRAUMA

Trauma:

- Is defined as a tissue injury that occurs more or less suddenly due to violence or accident and is accountable for initiating

  -hypothalami –pituitary –adrenal axis

  -immunologic and

  -metabolic responses that are responsible for restoring homeostasis.

- Can be

  -Penetrating trauma

  -Blunt trauma

  -Deceleration trauma

Organized approach to be used in clinical diagnosis, investigations & treatment as described below:

1. Triage:

- Triage is a process of determining the priority of patients' treatment based on the severity of their condition and the resources available to provide that treatment.

- In multiple casualty incidents, the number of patients and the severity of their injuries do not exceed the ability of the trauma care facility. The patients with life-threatening injuries are treated first.

- In mass casualty incidents, the number of patients and the severity of their injuries exceed the capacity of the trauma care facility. Here, the patients with the greatest chance of survival are treated first.

2. Primary Survey and Resuscitation:

Primary survey involves rapid early assessment of the patient. The life-threatening conditions are identified and treatment priorities are established based on their injuries, vital signs and injury mechanisms. During the primary survey, the following aspects are assessed and rapid corrective measures taken.

a) Airway maintenance with C-Spine Control

b) Breathing and Ventilation

c) Circulation and /haemorrhage Control
d) Disability/ Neurological Status
e) Exposure/ Environmental Control

3. Airway with C-spine control-

- The patency of the airway should be assessed with special attention to foreign body or maxillo- facial fractures that may result in airway obstruction.

- Chin-lift or Jaw-thrust manoeuvre may be used to achieve airway patency simultaneously protecting the cervical spine.

- A definitive airway (endotracheal intubation) is warranted in a patient with an altered level of consciousness or a Glasgow Coma Score of 8 or less.

- It is critical to protect the spine. Spinal injury should be assumed in any patient of trauma unless specifically excluded.

4. Breathing and Ventilation-

- The patient's chest should be exposed to adequately assess chest wall excursion.

- Auscultation to detect adequate air entry, percussion to exclude air or blood in chest and visual inspection and palpation to detect injuries to chest wall should be carried out.

- Specific life-threatening problems such as tension pneumothorax, massive haemorrhage, flail chest and cardiac tamponade should be identified immediately and addressed during the primary survey.

5. Circulation with Haemorrhage Control-

- Haemorrhage is the primary cause of shock in trauma patients. Rapid and accurate assessment of the patient's hemodynamic status and identification of the site of haemorrhage is therefore essential.

- It is critical to establish adequate intravenous access in a trauma patient. While the primary survey is going on, two intravenous lines should be established with short broad gauge cannula, preferably in the upper extremities, and resuscitation started with crystalloids.

- Central venous catheter insertion in case of haemodynamic instability requiring vasopressor support, inadequate peripheral IV access, and if there is need for hyperosmolar agent (mannitol)

6. Disability / Neurological Status-

- A rapid neurological evaluation is carried out at the end of primary survey after the resuscitation and before rapid sequence intubation.
- This assesses the patient's level of consciousness, papillary size and reaction and focal neurological deficit.
- The level of consciousness may be described in terms of Glasgow Coma Scale (GCS)
- GCS of 8 or less indicates a need for endotracheal intubation

7. Exposure / Environmental Control-

- The patient should be completely undressed to facilitate thorough examination and assessment
- At the same time care should be taken to prevent hypothermia to the patient

Adjuncts to Primary Survey and Resuscitation: -

a) ECG Monitoring:

- The appearance of dysrhythmias may indicate blunt cardiac injury.
- Pulseless electrical activity, the presence of cardiac rhythm without peripheral pulse may indicate cardiac tamponade, tension pneumothorax or profound hypovolemia.

b) Urinary Catheter:

- Urine Output is a sensitive indicator of the volume status of the patient and reflects renal perfusion.
- All trauma victims should be catheterized to enable monitoring of the urine output and plan intravenous fluid therapy.
- Transurethral catheterization is contraindicated in patients urethral transaction is suspected.

c) Gastric Catheter:

- A gastric tube is indicated to reduce stomach distension and decrease the risk of aspiration.
- It should be passed via the orogastric route in patients with head injury and suspected base skull fracture.

d) X-rays and Diagnostic Studies:

- The chest and pelvis x-ray help in the assessment of a trauma patient.
- Any trauma patient entering the red area of the emergency should undergo blood sampling. The blood should be sent for cross-match and arranging for packed cells, and important diagnostic parameters such as haemoglobin, renal parameters, ABG should be checked.
- Pulse oximetry is a valuable adjunct for monitoring oxygenation in injured patients.

e) FAST:

- Focused Assessment by ultra-sonography in Trauma is a rapid non-invasive tool used to assess free fluid in the abdomen, blunt abdominal injury and cardiac tamponade.

f) CT scan & MRI:

- For brain, spinal cord trauma and in injury to internal organs.

Secondary Survey: -

- Once the primary survey is accomplished, life- threatening conditions are managed and resuscitative efforts are underway, secondary survey is carried out.
- This is head to toe evaluation of trauma patient, which includes a complete history and physical examination and reassessment of all the vital signs.
- Each region of the body is completely examined. The care continues with regular re-evaluation of the patient for any deterioration and new findings, so that appropriate measures can be taken.

Re-evaluation: -

- After the completion of the secondary survey, the patient should be re-

evaluated beginning with the ABCs and thorough physical examination and examined for any missed injury such as fractures.

- Constant monitoring of the severely injured patient is required and
- May necessitate rapid transfer to the surgical intensive care unit, operating room or to another centre having better specialized facilities. The transfer to another centre should not be delayed for want of investigations.

Referral Criteria: -

- Patients should have basic control of airway, breathing and circulation, and bleeding to be stopped before contemplating transfer
- Referral if there is need for specialized surgery
- Referral if there is need for advanced intensive care after initial stabilization

RESPIRATORY FAILURE

TYPES OF RESPIRATORY FAILURE:

- Type 1- hypoxemic respiratory failure (oxygenation defect)
    - hypoxemia with PaO_2 <60 mm Hg on room air
    - eg. ARDS, Atelectasis, Pneumonia, ILD, Pulmonary oedema, Pulmonary embolism
- Type 2- hypercapnic respiratory failure (ventilatory defect)
    - hypercarbia with $PaCO_2$ > 50 mm Hg on room air
    - eg. COPD, Asthma, CNS depression, Neurological disease, Obesity hypoventilation syndrome
- Type 3- Postoperative
    - Eg. Thoraco-abdominal surgery, Inadequate analgesia postoperatively, Obesity, Diaphragmatic dysfunction
- Type 4- Increased metabolic demand
    - Eg. Shock (septic, hypovolemic, cardiogenic)
    - Hypermetabolic states

Approach to respiratory failure:

- A careful history
- Detailed examination of
    - the chest and upper airway
    - cardiovascular
    - neurologic
    - abdominal,
    - skin and
    - musculoskeletal system
- Pulse oximetry
- ABG to be done in all patients with respiratory distress
- Laboratory investigations
    - Complete blood count
    - Serum electrolytes

    - KFT
    - Cardiac enzymes
    - Microbiological evaluation
- CXR
- CT Chest
- Echocardiography
- Rapid Ultrasound for shock and hypotension (RUSH) recommended in rapid bedside evaluation of patients with ARF

Basic goals in management of Acute Respiratory Failure:

- To maintain airway, breathing, circulation
- To ensure adequate alveolar oxygenation and ventilation
- Treatment of the primary cause

All patients to be assessed for the following:

- **Airway**
    - First priority in any patient with poor sensorium is to secure the airways by clearing secretions, maintaining airway patency via oropharyngeal or nasopharyngeal airway, and endotracheal intubation in the worst scenario
- **Breathing**
    - Once airway is secured the patient should be assessed for breathing
    - If breathing is inadequate administer oxygen supplementation and assisted ventilation
- **Circulation**
    - An intravenous access to be secured
    - Intravenous fluids to be administered with the goal to restore normal volume status
- **Oxygenation**
    - Patients with ARF to be monitored closely using pulse oximetry with target SpO2 above 90%

Oxygen supplementation can be done by the following oxygen delivery system: -

1. **Nasal cannula:**
    - in patients who are hypoxemic without significant increased work of breathing and
    - require low to moderate FiO2 to achieve oxygenation goals.
2. **Simple face mask:**
    - for patients who are hypoxemic without significant increased work of breathing and requiring low to moderate FiO2 to achieve oxygenation goals.
    - May be a better option than nasal cannula in patients with nasal obstruction or epistaxis.
3. Face mask with reservoir (non-rebreather):
    - for patients who are hypoxemic with high inspiratory flow requirements.
    - The reservoir bag should be at least half distended throughout respiration
4. Venturi masks:
    - Are high-flow, fixed oxygen concentration devices
    - Can control the amount of air entrained to deliver a fixed FiO2 from 24 to 60%
    - Useful in COPD patients to titrate FiO2 without increasing the PaCO2 concentration
    - Relatively high flows of 100% O2 are required to achieve high FiO2.
5. High-flow nasal cannula (HFNC):
    - Basic components include a flow generator providing gas flow rates up to 60 liters per minute, an air-oxygen blender that achieves escalation of FIO2 from 21% to 100% irrespective of flow rates, and a humidifier that saturates the gas mixture at a temperature of 31 to 37 C. To minimize condensation, the heated humidified gas is delivered via heated tubings through a wide-bore nasal prong.

- Indicated in
    - Acute hypoxemic respiratory failure
    - Post-surgical respiratory failure
    - Acute heart failure/pulmonary edema
    - Hypercapnic respiratory failure, COPD
    - Pre and post-extubation oxygenation
    - Obstructive sleep apnea
    - Use in the emergency department
    - Do not intubate the patient

6. Non-invasive ventilation (NIV):
    - Non-invasive ventilation should be applied simultaneously to a patient in acute respiratory failure in addition to the rest of the treatment based on the clinical criteria, provided there is no contraindication
    - Non-invasive Positive Pressure Ventilation (NIPPV) is indicated in patients with appropriate diagnosis with potential reversibility and if patient any two of the following clinical criteria are fulfilled-

        - Moderate to severe respiratory distress
        - Tachypnoea (RR more than 25 / min)
        - Accessory muscle use or abdominal paradox
        - Blood gas derangement pH < 7.35, $PaCO_2$ > 45 mm Hg
        - PaO_2/FiO_2<300 or SPO_2<92% with FiO_2 0.5

Indications for NIV:

- Acute COPD with exacerbations
- Acute cardiogenic pulmonary oedema
- Obesity hypoventilation syndrome
- Weaning and post-extubation respiratory failure
- Mild ARDS

Contraindications to NIV:

- Cardiac or respiratory arrest
- Haemodynamic instability-patient on high vasopressor support
- Inability to protect airways or clear secretions
- Facial deformity, surgery
- High risk of aspiration-cerebrovascular accident (CVA)
- Recent oesophageal surgery
- Unco-operative patient

7. Invasive Mechanical Ventilation:

- All patients on NIV support should be closely monitored for sudden deterioration
- If not much improvement in gas exchange or relief in respiratory distress within a few hours invasive mode of ventilation to be started without delay
- Indications for endotracheal intubation in patients requiring invasive mechanical ventilation-
    - Impending cardio-respiratory arrest
    - Tachypnoea with signs of respiratory distress, use of accessory muscle
    - NIV failure/noncompliance
    - Severe refractory acidosis
    - Unable to protect airway/clear secretions
    - Hypoventilation with reduced ventilatory rate
    - Poor sensorium with GCS <8
    - Polytrauma

8. Tracheostomy:

Indications for tracheostomy-

- Long term mechanical ventilation
- Weaning failure
- Upper airway obstruction
- Airway protection

Ventilatory management for ARDS/ALI:

- Patients with ARDS to be ventilated using lung protective mechanism with low Vt (6ml/kg of ideal body weight)

- Permissive hypercapnia
- Optimum positive end expiratory pressure (PEEP)
- Maintenance of plateau pressures <30 cmH_2O
- Neuromuscular blockade, in spite of high level of sedation, may be required in severe ARDS to overcome desynchrony
- Prone positioning

Intensive Care Unit Ventilator Weaning Protocol: -

Criteria for readiness of weaning
Subjective assessment:
• Adequate cough
• No neuromuscular blocking agents
• Absence of excessive trachea-bronchial secretion
• Reversal of the underlying cause for respiratory failure
• No continuous sedation infusion or adequate mentation on sedation
Objective measurements:
• Stable cardiovascular status
• Heart rate ≤ 140 beat/minute
• No active myocardial ischemia • Adequate haemoglobin level (≥ 8 g/dl) • Systolic blood pressure 90–160 mmHg • Afebrile (36° C < temperature < 38° C)

No or minimal vasopressor or inotrope (< 5 µg/kg/minute dopamine or dobutamine)

Adequate oxygenation:

- Tidal volume > 5 mL/kg
- Vital capacity >10 mL/kg
- Proper inspiratory effort

- Respiratory rate ≤ 35/minute
- PaO2 ≥ 60 and PaCO2 ≤ 60 mmHg
- Positive end expiratory pressure ≤ 8 cmH_2O
- No significant respiratory acidosis (pH ≥ 7.30)
- Maximal inspiratory pressure (MIP) ≤ -20 – -25 cmH2O
- O_2 saturation > 90% on FIO2 ≤ 0.4 (or PaO2/FIO2 ≥ 200)
- Rapid Shallow Breathing Index (respiratory Frequency/Tidal Volume) < 105

Weaning procedure:

A weaning plan starts with assessing the ability of the patient for spontaneous breathing.

SBT Strategies-

- Continuous positive airway pressure (CPAP) trial using a CPAP level equal to the previous positive end-expiratory pressure (PEEP) level followed by
- T-piece trial, in which only supplemental oxygen is supplied through a T- piece connected to an endotracheal tube.

Criteria of successful spontaneous breathing trials:

- Respiratory rate < 35 breaths/minute
- Good tolerance to spontaneous breathing trials
- Heart rate < 140 /minute or heart rate variability of >20%
- Arterial oxygen saturation >90% or PaO2 > 60 mmHg on FiO2<0.4
- Systolic blood pressure < 180 mmHg or <20% change from baseline
- No signs of increased work of breathing or distress *

Extubation:

- If patient tolerates Spontaneous Breathing Trial for 2 h and
- All the above criteria are fulfilled patient is extubated
- If any of the criteria mentioned above is not satisfied mechanical

ventilation is continued and items in the checklist are re-checked the next day.

Supportive care:

1. Suctioning:
    - Maintains airway patency
    - Increases oxygenation and decreases work of breathing
    - Stimulates cough and prevents atelectasis.
2. Nebulisation:
    - Inline jet nebulizer / MDI
    - Delivery of bronchodilator drugs in aerosolised form.
3. Humidification: Prevents drying of secretions and maintains mucociliary function.
4. Physiotherapy: Prevents atelectasis, facilitates postural drainage, and prevents complication of mechanical ventilation.
5. Care of ETT: Proper fixing of the tube, measuring cuff pressure and maintaining it less than 25 mm of Hg.
6. Nutritional support: early enteral feeding, provide adequate calories, protein, electrolytes, vitamins and fluids, care of feeding tube.
7. Stress ulcer prevention: Early enteral feeding, H2 blockers or proton pump inhibitors for prophylaxis, minimise use of steroids and NSAIDS
8. DVT prevention: DVT prevention either by low molecular weight heparin or conventional heparin or by graduated compression stockings or sequential compression device in patient where heparin is contraindicated.
    - Head end elevation of 35-45°.
    - Bowel and bladder care
    - Care of eyes
    - Daily sedation interruption
9. Prevention of pressure sore: positioning, prevent soiling, use of air mattress, meticulous cleaning and good wound care.

- Adequate Analgesia for pain
- Infection control.
- chest radiographs and arterial blood gas measurement in patients undergoing mechanical ventilation best done when clinically indicated rather than routinely on a daily basis
- Bedside ultrasound/point of care ultrasound (POCUS) preferred to radiograph.

MANAGEMENT OF SEPSIS

INTRODUCTION

Sepsis is a life-threatening organ dysfunction caused by a dysregulated host response to infection. Sepsis and septic shock are major healthcare problems, impacting millions of people around the world each year and killing between one in three and one in six of those it affects. Early identification and appropriate management in the initial hours after the development of sepsis improve outcomes.

INITIAL RESUSCITATION:

1) For patients with sepsis induced hypoperfusion or septic shock, at least 30 mL/kg of IV crystalloid fluid should be given within the first 3 hours of resuscitation
2) Crystalloids are the first-line fluid for resuscitation and balanced crystalloids are preferred to normal saline for resuscitation.
3) Albumin can be used in patients who received large volumes of crystalloids over using crystalloids alone.
4) Colloids such as gelatins and starches are not recommended in septic shock

VASOACTIVE AGENTS:

1) Initial target mean arterial pressure (MAP) is 65 mm Hg
2) Norepinephrine is the first-line agent over other vasopressors
3) For adults with septic shock on norepinephrine with inadequate MAP levels, vasopressin can be added instead of escalating the dose of norepinephrine.
4) Vasopressin is usually started when the dose of norepinephrine is in the range of 0.25–0.5 μg/kg/ min.
5) If MAP is inadequate despite norepinephrine and vasopressin, epinephrine can be added
6) For adults with septic shock and cardiac dysfunction with persistent hypoperfusion despite adequate volume status and arterial blood pressure, dobutamine in addition to norepinephrine or epinephrine alone can be used
7) For adults with septic shock, vasopressors can be started peripherally to

restore mean arterial pressure rather than delaying initiation until a central venous access is secured.

INVESTIGATIONS

1) Complete blood count
2) Renal function test
3) Liver function test
4) Serum electrolytes
5) Serum lactate
6) Serum procalcitonin
7) C-reactive protein
8) Urine routine examination
9) Blood culture (2 sites)
10) Urine culture or any other culture as indicated
11) ABG (if clinically indicated) 12)
12) Ultrasound (if clinically indicated)
13) Chest Xray (if clinically indicated)

INFECTION CONTROL

1) For adults with possible septic shock or a high likelihood for sepsis, we should administer antimicrobials immediately, ideally within 1 hr of recognition.

2) For adults with suspected sepsis or septic shock but unconfirmed infection, we should continuously re-evaluate and search for alternative diagnosis and discontinue empiric antimicrobials if an alternative cause of illness is demonstrated or strongly suspected

3) For adults with possible sepsis without shock, a rapid assessment of the likelihood of infectious versus non-infectious causes of acute illness is recommended

4) For adults with possible sepsis without shock, a time-limited course of rapid investigation should be done and if concern for infection persists, antimicrobials can be administered within 3 hrs from the time when sepsis was first recognized.

5) For adults with a low likelihood of infection and without shock, antimicrobials can be deferred while continuing to closely monitor the patient

CHOICE OF ANTIBIOTICS

1) For adults with sepsis or septic shock and high risk for multidrug resistant (MDR) organisms, two antimicrobials with gram-negative coverage for

empiric treatment is preferred over one gram-negative agent

2) For adults with sepsis or septic shock and low risk for MDR organisms, one antimicrobial agent with gram-negative agent can be given
3) For adults with sepsis or septic shock at high risk of methicillin-resistant Staphylococcus aureus (MRSA), empiric antimicrobials with MRSA coverage is recommended
4) For adults with sepsis or septic shock, once the causative pathogen and the susceptibilities are known, one antimicrobial with gram negative coverage can be given
5) For adults with sepsis or septic shock, we **suggest** daily assessment for de-escalation of antimicrobials over using xed durations of therapy without daily re- assessment for de-escalation
6) For adults with an initial diagnosis of sepsis or septic shock and adequate source control where optimal duration of therapy is unclear, we **suggest** using procalcitonin AND clinical evaluation to decide when to discontinue antimicrobials over clinical evaluation alone.

SOURCE CONTROL

1) For adults with sepsis or septic shock, it is very important to rapidly identify or exclude a specific anatomical diagnosis of infection that requires emergent source control and implementing any required source control intervention as soon as medically and logistically practical.
2) This includes drainage of an abscess, debriding infected necrotic tissue, removal of a potentially infected device, or definitive control of a source of ongoing microbial contamination. Foci of infection readily amenable to source control include intra-abdominal abscesses, gastrointestinal perforation, ischemic bowel or volvulus, cholangitis, cholecystitis, pyelonephritis associated with obstruction or abscess, necrotizing so tissue infection, other deep space infection (e.g., empyema or septic arthritis), and implanted device infections.

ADDITIONAL THERAPIES

Corticosteroids

1) For adults with septic shock and an ongoing requirement for vasopressor therapy using IV corticosteroids is recommended

2) The typical corticosteroid used in adults with septic shock is IV hydrocortisone at a dose of 200 mg/d given as 50 mg intravenously every 6 hours or as a continuous infusion. It is suggested that this is commenced at a dose of norepinephrine or epinephrine ≥ 0.25

mcg/kg/min at least 4 hours after initiation.

Stress Ulcer prophylaxis

1) For adults with sepsis or septic shock, and who have risk factors for gastrointestinal (GI) bleeding, using stress ulcer prophylaxis is recommended

Venous Thromboembolism (VTE) Prophylaxis

1) For adults with sepsis or septic shock, using pharmacologic VTE prophylaxis is recommended unless a contraindication to such therapy exists.

2) Low molecular weight heparin (LMWH) is preferred over unfractionated heparin (UFH) for VTE prophylaxis.

3) For adults with sepsis or septic shock, using mechanical VTE prophylaxis in addition to pharmacological prophylaxis has no added advantage as compared to pharmacologic prophylaxis alone.

Glucose control

1) For adults with sepsis or septic shock, initiating insulin therapy at a glucose level of ≥ 180 mg/dL (10 mmol/L) is recommended

2) Following initiation of an insulin therapy, a typical target blood glucose range is 144–180mg/dL (8–10 mmol/L).

Nutrition

1) For adult patients with sepsis or septic shock who can be fed enterally, early (within 72 hours) initiation of enteral nutrition is recommended

Bicarbonate therapy

1) For adults with septic shock and hypoperfusion- induced lactic acidemia, sodium bicarbonate therapy should not be used to improve haemodynamics or to reduce vasopressor requirements
2) For adults with septic shock, severe metabolic acidemia (pH ≤ 7.2) and AKI (AKIN score 2 or 3), sodium bicarbonate therapy can be used

NUTRITION IN ICU

Nutrition is an essential part of the care for critically ill patients, but the optimal feeding strategy for patients in the intensive care unit (ICU) is still debated and often remains a challenge for the ICU team in clinical practice.

Medical nutrition therapy (MNT)is a term that encompasses oral nutritional supplements, enteral nutrition (EN) and parenteral nutrition (PN)

Medical nutrition therapy in the ICU aims at avoiding malnutrition in primarily well-nourished patients and at preventing further deterioration of previously malnourished patients.

Malnutrition is a significant prognostic risk factor for critically ill patients, influencing major outcomes such as mortality, length of stay, duration of mechanical ventilation, and infection rates.

There is no single "golden bullet" to diagnose malnutrition, but many helpful tools and criteria. All ICU patients should be regularly screened for risk of malnutrition.

WHEN TO START NUTRITION THERAPY

Medical nutrition therapy shall be considered for all patients staying in the ICU, mainly for more than 48 hours.

A general clinical assessment should be performed to assess malnutrition in the ICU, until a specific tool has been validated.

General clinical assessment could include anamnesis, report of unintentional weight loss or decrease in physical performance before ICU admission, physical examination, general assessment of body composition, and muscle mass and strength, if possible.

ENTERAL OR PARENTERAL?

Oral diet shall be preferred over EN or PN in critically ill patients who are able to eat.

If oral intake is not possible, early EN (within 48 h) in critically ill adult patients should be performed/initiated rather than delaying EN

If oral intake is not possible, early EN (within 48 h) shall be performed/initiated in critically ill adult patients rather than early PN

In case of contraindications to oral and EN, PN should be implemented within three to seven days

Early and progressive PN can be provided instead of no nutrition in case of contraindications for EN in severely malnourished patients.

HOW MUCH?

To avoid overfeeding, early full EN and PN shall not be used in critically ill patients but shall be prescribed within three to seven days.

Continuous rather than bolus EN should be used.

ROUTE

Gastric access should be used as the standard approach to initiate EN.

In patients with gastric feeding intolerance not solved with prokinetic agents, postpyloric feeding should be used.

In patients deemed to be at high risk for aspiration, post-pyloric, mainly jejunal feeding can be performed.

In critically ill patients with gastric feeding intolerance, intravenous erythromycin should be used as a first line prokinetic therapy.

Alternatively, intravenous metoclopramide or a combination of metoclopramide and erythromycin can be used as a prokinetic therapy.

ASSESSMENT OF ENERGY EXPENDITURE

In critically ill mechanically ventilated patients, EE should be determined by using indirect calorimetry.

If indirect calorimetry is used, isocaloric nutrition rather than hypocaloric nutrition can be progressively implemented after the early phase of acute illness

Hypocaloric nutrition (not exceeding 70% of EE) should be administered in the early phase of acute illness.

After day 3, caloric delivery can be increased up to 80-100% of measured EE. REQUIREMENT

The daily energy expenditure of each individual patient can be estimated or measured.

Daily energy expenditure based on sex, body weight (in kilograms), and height (in inches) is expressed as Harris–Benedict equations as shown in table below

TABLE: Methods for Determining Daily Energy Expenditure
Basal Energy Expenditure (BEE): Men: BEE (kcal/24hr) = 66 + (13.7 × wt) + (5.0 × ht) - (6.7 × age) Women: BEE (kcal/24hr) = 655 + (9.6 × wt) + (1.8 × ht) - (4.7 × age) (wt = weight in kilograms, ht = height in inches) *Resting Energy Expenditure (REE):* *REE (kcal/24hr) = [(3.9 × VO_2) + (1.1 × VCO_2) - 61] × 1440 †REE (kcal/24hr) = BEE × 1.2
*From Bursztein S, Saphar P, Singer P, et al. A mathematical analysis of indirect calorimetry measurements in 1440 is used to convert the time period to 24 hr. †REE is equivalent to the BEE plus the thermal effect of food.

Another more simplified predictive equation for the BEE is as follows:

BEE (kcal/day)=25 X Wt (in kg)

To allow for the thermal effect of food intake, the BEE is multiplied by 1.2 to derive the resting energy expenditure (REE), which is the energy expenditure of basal metabolism in the resting but not fasted state. Other adjustments in the BEE that allow for enhanced energy expenditure in hypermetabolic conditions are shown below:

- Fever: BEE X 1.1 (for each °C above the normal body temperature)
- Mild stress: BEE X 1.2

- Moderate stress: BEE X 1.4
- Severe stress: BEE X 1.6

The daily energy requirement should be provided by calories derived from carbohydrates and lipids, and protein intake should be used to maintain the stores of essential enzymatic and structural proteins.

Carbohydrates supply approximately 70% of the nonprotein calories Lipids should

provide approximately 30% of the daily energy needs.

Protein requirement: The goal of protein intake is to match the rate of protein catabolism in the individual patient

- Normal metabolism 0.8 to 1.0 g/kg
- Hypercatabolism 1.2 to 1.6 g/kg

The amount of glucose (PN) or carbohydrates (EN) administered to ICU patients should not exceed 5 mg/kg/min.

The administration of intravenous lipid emulsions should be generally a part of PN.

Intravenous lipid (including non-nutritional lipid sources) should not exceed 1.5 g lipids/kg/day and should be adapted to individual tolerance.

In patients with burns > 20% body surface area, additional enteral doses of GLN (0.3-0.5 g/kg/d) should be administered for 10-15 days as soon as EN is commenced.

In critically ill trauma, additional EN doses of GLN (0.2-0.3 g/ kg/d) can be administered for the first five days with EN. In case of complicated wound healing it can be administered for a longer period of ten to 15 days.

In ICU patients except burn and trauma patients, additional enteral GLN should not be administered.

High doses of omega-3-enriched EN formula should not be given by bolus administration.

EN enriched with omega-3 FA within nutritional doses can be administered.

High doses omega-3 enriched enteral formulas should not be given on a routine basis.

Parenteral lipid emulsions enriched with EPA + DHA (Fish oil dose 0.1-0.2 g/kg/d) can be provided in patients receiving PN

To enable substrate metabolism, micronutrients (i.e. trace elements and vitamins) should be provided daily with PN. Antioxidants as high dose monotherapy should not be administered without proven deficiency.

In critically ill patients with measured low plasma levels (25-hydroxy-vitamin D < 12.5 ng/ml, or 50 nmol/l) vitamin D3 can be supplemented.

In critically ill patients with measured low plasma levels (25-hydroxy-vitamin D < 12.5 ng/ml, or 50 nmol/l) a high dose of vitamin D3 (500,000 UI) as a single dose can be administered within a week after admission.

EN should be delayed

- if shock is uncontrolled and hemodynamic and tissue perfusion goals are not reached, whereas low dose EN can be started as soon as shock is controlled with fluids and vasopressors/inotropes, while remaining vigilant for signs of bowel ischemia;
- in case of uncontrolled life-threatening hypoxemia, hypercapnia or acidosis, whereas EN can be started in patients with stable hypoxemia, and compensated or permissive hypercapnia and acidosis;
- in patients suffering from active upper GI bleeding, whereas EN can be started when the bleeding has stopped and no signs of re- bleeding are observed;
- in patients with overt bowel ischemia; in patients with high-output intestinal fistula if reliable
- feeding access distal to the fistula is not achievable; in patients with abdominal compartment syndrome; and if gastric aspirate volume is above 500 ml/6 h.

Early EN should be performed

- in patients receiving ECMO
- in patients with traumatic brain injury
- in patients with stroke (ischemic or haemorrhagic)
- in patients with spinal cord injury

- in patients with severe acute pancreatitis

- in patients after GI surgery
- in patients after abdominal aortic surgery
- in patients with abdominal trauma when the continuity of the GI tract is confirmed/restored
- in patients receiving neuromuscular blocking agents
- in patients managed in prone position
- in patients with open abdomen
- regardless of the presence of bowel sounds unless bowel ischemia or obstruction is suspected in patients with diarrhoea.

SPECIAL CONSIDERATIONS

In non-intubated patients not reaching the energy target with an oral diet, oral nutritional supplements should be considered first and then EN.

In non-intubated patients with dysphagia, texture-adapted food can be considered. If swallowing is proven unsafe, EN should be administered.

In non-intubated patients with dysphagia and a very high aspiration risk, post- pyloric EN or, if not possible, temporary PN during swallowing training with removed naso-enteral tube can be performed.

In patients after abdominal or esophageal surgery, early EN can be preferred over delayed EN.

In critically ill patients with surgical complications after abdominal or esophageal surgery and unable to eat orally, EN (rather than PN) should be preferred unless discontinuity or obstruction of GI tract, or abdominal compartment syndrome is present.

In the case of an unrepaired anastomotic leak, internal or external fistula, a feeding access distal to the defect should be aimed for to administer EN.

In the case of an unrepaired anastomotic leak, internal or external fistula, or if distal feeding access is not achieved, EN should be withheld and PN may be commenced.

In case of high output stoma or fistula, the appropriateness of chyme reinfusion or enteroclysis should be evaluated and performed if adequate.

Trauma patients should preferentially receive early EN instead of early PN.

DERMATOLOGY STANDARD TREATMENT GUIDELINES

BACTERIAL SKIN INFECTION

Common bacterial infections of the skin are caused by pus producing organisms mainly Staphylococcus aureus and Streptococci pyogenes.

Predisposing factors

1. Poor Hygiene
2. Diabetes
3. In immunosuppressed conditions like HIV/AIDS
4. Patients on steroids & other immunosuppressive drugs.

FOLLICULITIS

- Inflammation of terminal part or ostium of hair follicle of infective or non-infective origin.
- Non-infective causes include contact with oils, adhesive tapes etc.
- Presents as small superficial pustules.

FURUNCULOSIS

- Extended involvement of the entire hair follicle including the perifollicular region in the dermis & subcutaneous tissues.
- Commonly known as Boil.
- Presents as painful nodule, later becomes pustular and necrotic.

CARBUNCLE

- Deep infection of two or more contiguous hair follicles.
- Commonly seen in diabetics at back of neck, shoulders
- Presents as extremely painful plaque with multiple pus points.
- Constitutional symptoms like fever, malaise usually present.

IMPETIGO

- Contagious, superficial infection of skin.
- 2 types - Bullous and non-bullous impetigo.
- Common in children, around mouth and nose and in limbs.
- Presents as discrete thin-walled vesicles that rapidly become pustular and then rupture leading to honey coloured (yellow brown) crusts.

CELLULITIS/ ERYSIPELAS

- Cellulitis – infection cause suppurative inflammation of the deeper dermis and subcutaneous tissue.
- Erysipelas - Infection of dermis and superficial lymphatics.
- Sites - scalp, face and limbs are more frequently involved.

- Presents as diffuse ill -defined swelling of affected part with warm, shiny surface. It may present with vesicles, bullae & erosions. Erysipelas have a typical raised, well defined edges.
- Constitutional symptoms like fever, malaise, generalised weakness, lymphadenopathy present.
- Complications - Fascitis, myositis, subcutaneous abscess, nephritis and septicemia.

Diagnosis

- Mainly clinical
- Gram staining
- Swab for Culture &Sensitivity.
- Patient should be investigated for diabetes and other predisposing conditions.

Non-pharmacological treatment

- Good personal hygiene and nutrition
- Proper washing with soap and water
- Avoid overcrowding

Pharmacological treatment

- Topical antibiotics for mild and localized disease –
  - Mupirocin cream, Fusidic Acid cream, framycetin cream
  - To apply twice a day for 1 week.

- Systemic antibiotics for widespread and severe disease-
  - Cap. Flucloxacillin 500 mg QID for 5-7 days. (50-100mg/kg/day in children).
  - Tab. Erythromycin 500mg QID for 5-7 days.
  - Cephalosporins 1-2gm daily (adult) and 30-50mg/kg/day (children).
  - In case of penicillin allergy: Clarithromycin 500 mg BD or Roxithromycin 50 mg BD.
  - Other drugs which are effective depending upon the causative organis in are amoxicillin /clavulanic acid, clindamycin, ampicillin /sulbactum, imipenem, vancomycin, piperacillin /tazobactum.
  - NSAIDS may be used to reduce constitutional symptoms.

Surgical treatment

- Surgical Drainage of pus if required.
- Debridement and regular aseptic dressings for deeper infections.

FUNGAL SKIN INFECTION

DERMATOPHYTE INFECTION

The fungi that infect skin / hair / nails are called "dermatophytes" and belong to the following genera: Microsporum, Trichophyton and Epiderimophyton.

Predisposing Factors

- Local or systemic immune suppression.
- Genetic susceptibility.
- Diabetes.
- Environmental factors.

Depending on the site of body involved, different names are given.

- Head : Tinea capitis
- Face : Tinea faciei
- Beard : Tinea barbae
- Trunk / body : Tinea corporis
- Groin / gluteal folds : Tinea cruris
- Palms : Tinea manuum
- Soles : Tinea pedis
- Nail : Tinea unguium

Clinical features

- Sharply marginated, annular or polycyclic lesions with erythematous papules, vesicles, scaly borders and central clearing.
- Tinea capitis – presents as area of partial hair loss, dull gray, lusterless hair that are easily pulled out or inflamed boggy, indurated swelling with pustules and crusting in the inflammatory type.
- Tinea unguium: may present as dirty, dull, dry, pitted, ridged, split, discoloured, thick, uneven nails with subungual hyperkeratosis.

Diagnosis & Investigations

- Mainly clinical
- Microscopy- KOH mount
- Culture – Sabouraud's dextrose agar
- Woods lamp examination – greenish yellow fluorescence

Non-pharmacological treatment

- Avoid tight fitting clothes and shoes
- Avoid prolonged working in wet area
- Keep the skin dry

Pharmacological treatment

Topical treatment:

- Clotrimazole, miconazole or Luliconazole as cream, ointment, lotion or powder formulation. To apply twice a day for 4-6 weeks.
- 5% Amorolfine or 8% Ciclopirox olamine nail lacquer. To apply twice or thrice a week for 2-3 months.

Systemic treatment:

- Tab Fluconazole 150mg/ week for 4-6 weeks (3-6 mg/kg in children)
- Cap Itraconazole 200 OD for 2-4 weeks (5mg/kg/day in children)
- Tab Terbinafine 250mg/day for 2-4 weeks (6mg/kg/day in children)
- Tab Griseofulvin 500mg OD for 6-8 weeks (10-20mg/kg/day in children)
- Treatment have to be continued for 3-6 months for tinea unguium.

CANDIDIASIS

It is a common infection caused by a yeast Candida. Most commonly caused by Candida albicans and sometimes by other species of candida.

Clinical features

- Flexural candidiasis – multiple diiscr etepustuIes on erythematous base with satellite lesions. Commonly seen between fingers, toes, axilla, groin and buttocks.
- Candidal paronychia – painful, swollen and red proximal and lateral nail folds with occasional discharge of pus.
- Oral candidiasis (Thrush) - friable greyish white membranous plaques in mouth.
- Candidal balanoposthitis – intense erythema with desquamation/ superficial erosions, fissuring, maculopapular rash over glans and foreskin.
- Candidal vulvovaginitis – vulvar pruritus, thick curd like vaginal discharge, dysuria and dyspareunia.

Diagnosis & Investigations

- Clinical
- Microscopy – 10% KOH exa mination to look for yeast cells and mycelia.
- Culture – Sabauraud,s Dextrose agar medium

Non- pharmacological Treatment

- Maintain personal hygiene
- Keep the affected area dry and clean
- Control of underlying co-morbidities

Pharmacological Treatment

Topical Therapy:

- Clotrimazole 1% mouth paint BD for 4 weeks
- Clotrimazole or miconazole cream BD for 2-4 weeks.
- Clotrimazole vaginal pessary – daily for 5-7 days

Systemic therapy:

- Tab. Fluconazole 150mg single dose (3-6 mg/kg in children)

PITYRASIS VERSICOLOR

It is a mild, superficial fungal infection of the skin caused by the mycelial form of malassezia furfur.

Predisposing conditions

Hot, humid and damp conditions.

Clinical features

- Macules which may be hypo or hyper pigmented covered with branny scales.
- Usually asymptomatic but sometimes mild irritation may be present.
- Sites : Mainly on upper trunk, neck and upper arms.

Diagnosis & Investigations

- Clinical
- Direct examination under woods lamp- yellow fluorescence
- 10% KOH examination of skin scrapings – mycelia and yeast form seen

Treatment

- Topical therapy:
  Lotions to be applied for 5-10 minutes before bath.
  - Selenium sulfide 2.5 % to be applied once daily x 14 days
  - Zinc pyrithione 1% to be applied once daily x 1 4 days
  - Ketoconozole 2% to be applied once daily x 2 weeks
  - Miconazole2 % to be applied once daily x 2 weeks

- Systemic therapy:
  - Tab Fluconazole 400 mg single dose
  - Ketoconazole 200 mg OD x 7 -1 0 days.
  - Itraconazole 200 mg OD x 3 -7 days.

VIRAL SKIN INFECTIONS

Viruses are obligatory intracellular parasites. The mode of transmission of these viruses varies from respiratory to faeco-oral route, arthropod borne, venereal and penetrating wound.

CHICKEN POX/ VARICELLA

An infectious viral disease caused by varicella zoster virus. Transmission mainly by droplet. 90% cases occur in children and teenagers.

Clinical feature

- Incubation Period: 4 -15 days.
- Rash often preceded by 2 -3 days of prodromal symptoms- fever, chills, malaise, headache, anorexia.
- Rash is polymorphic and is characterized by erythematous papules, vesicles "dew drops on rose petal", umbilicated pustules. Arranged in centripetal manner mainly over the trunk and proximal limbs.

Diagnosis & Investigations

- Mainly clinical diagnosis,
- Tzanck smear from blister – shows multinucleated giant cells.

Non-pharmacological Treatment

- Rest, cold compresses, tepid bath
- The patient to be kept in isolation.

Pharmacological Treatment

- Tab Acyclovir 800mg 5 times daily for 7 days (20mg/kg QID in children).
- Tab Valacyclovir 1gm TID for 7 days
- Tab Famciclovir 500mg TID for 7days

HERPES ZOSTER (SYN: SHINGLES)

It is a localized disease caused by reactivation of Varicella Zoster Virus lying dormant in the sensory ganglia.

Clinical features

- Rash appears following prodromol symptoms like fever, malaise, paraesthesia.
- Mostly unilateral, dermatomal and does not cross midline.
- Closely grouped vesicles, papules and pustules.
- Most common in thoracic region.
- Most common complication is Post herpetic neuralgia.

Diagnosis & Investigations

- Mainly clinical diagnosis.
- Tzanck smear – shows multinucleated giant cells.

Non-pharmacological Treatment

- Rest, cold compresses, tepid bath
- The patient to be kept in isolation.

Pharmacological Treatment

- Tab Acyclovir 800mg 5 times daily for 7 days (20mg/kg QID in children).
- Tab Valacyclovir 1gm TID for 7 days
- Tab Famciclovir 500mg TID for 7days
- Therapy to be continued for 7 days in immune competent persons and for 14 days in immuno suppressed individuals.
- Topical – Calamine lotion
- Non-steroidal anti-inflammatory drugs – for pain
- For post-herpetic neuralgia – amitriptyline, carbamazepine, phenytoin, gabapentin and pregabalin may be given.

HERPES LABIALIS

It is an infection of the lips by herpes simplex virus (HSV-1). It spread by close personal contact.

Clinical features

- Incubation Period: - 5-7 days.
- Mild prodrome of malaise and tingling sensation.
- Closely grouped vesicles seen around lips.
- Vesicles rupture to form shallow ulcers.

Diagnosis & Investigations

- Mainly clinical diagnosis.
- Tzanck smear- for multinucleated giant cell.
- IgG, IgM HSV 1 and 2 antibodies.

Non-pharmacological Treatment

- The patient should avoid contact until all the lesions are crusted.

Pharmacological Treatment

- Tab Acyclovir 400mg 3 times daily for 5 -7 days.
- Tab Valacyclovir 1gm bID for 7 days

- Tab Famciclovir 250mg TID for 7days

MOLLUSCUM CONTAGIOSUM

An infectious disease caused by pox virus, Molluscum contagiosum virus. Commonly seen in children.

Clinical features

- Incubation Period: 2 weeks-6 months.
- Dome shaped, pearly white, umbilicated waxy papules.
- Common sites - Children - axilla, side of trunk, lower abdomen, face. Young adults - genitalia

Diagnosis & Investigations

- Mostly clinical diagnosis.
- Giemsa stain – of expressed material to demonstrate HP bodies
- Skin biopsy – to show molluscum body.

Non-pharmacological Treatment

- Avoid scratching.
- Avoid sharing of towels, contact sports and communal bathing.

Pharmacological Treatment

- Chemical cauterization – by trichloroacetic acid 10-30%, phenol or salicylic acid. 10% potassium hydroxide in children.
- Cryotherapy using liquid nitrogen.
- Curettage or diathermy.
- Cidofovir 1-3% cream in immunocompromised patients.

WARTS

An infection of skin and contiguous mucous membrane by different groups of Human papilloma viruses.

Clinical Features

- Incubation Period - 3 weeks to 8 months.
- Common Sites: back of hands, palms, finger, knees, soles, anogenital area.
- Age: Peak age (l 2 -16 years), unusual in infancy and early childhood.
- Firm, papules with rough, horny surface.
- Types: common warts, flat warts, filliform warts, plantar warts, genital warts.

Diagnosis & Investigations

- Mostly clinical diagnosis.

- Skin biopsy

Non-pharmacological Treatment

- Avoid scratching.
- Avoid sharing of towels, contact sports and communal bathing.
- Safe sex practice in case of genital warts.

Pharmacological Treatment

- Wart paint (lactic acid 16% & salicylic acid 16%)
- Salicylic acid 10-40% under occlusion.
- Podophyllin 25% weely
- Trichloroacetic acid 20-100%
- Cryotherapy using liquid nitrogen.
- Curettage or diathermy.
- Tab levamisole 150mg twice a week.

PARASITIC INFESTATIONS

SCABIES

Scabies is caused by arthropod mite (sarcoptesscabiei). It is transmitted by close personal contact after an incubation period of 3-4 weeks

Clinical features:

- Nocturnal itching
- Excoriated papules, papulovesicles, burrows on inter digital clefts of hands, wrist, axillary folds, breasts, periumblical region, medial side of thigh and genitals.
- Face, palms, soles and scalp involved in infants

Diagnosis

- Clinical
- Inspection of mites from scabetic burrows.

Nonpharmacological treatment

- Maintenance of personal hygiene.
- Disinfection of bedding and clothing.
- All family members should be treated simultaneously.

Pharmacological treatment

- Gamma benzene hexachloride (GBHC) lotion 1%. Single overnight application below neck on entire body surface after a thorough scrub bath, to be washed off next morning.
- Permethrin 5% to be applied generously, after bath, at bed time, covering entire surface of the body below neck (except face).
- 25% Benzyl Benzoate emulsion overnight application for three consecutive days.
- Tab.ivermectin 200mcg/kg as asingle stat dose to be repeated after 2weeks.
- Antihistamines like Tab.cetirizine10mg daily. For children 0.3mg/kg/day.

PEDICULOSIS

It is commonly known as lice infestation. Transmission occurs by personal contact, sharing of combs, infected clothing and poor personal hygiene while transmission of pubic lice is by sexual-contact.

Types

1. Pediculosis capitis - by Head louse
2. Pediculosis corporis - by Body louse
3. Phthiriasis pubis - by Crab louse

Causative organism

- Pediculosis humanus var. capitis (head louse)

- Pediculosis capitis var. corporis (body louse)
- Phthirus pubis (pubic louse)

Clinical features
Presence of nits and louse associated with itching of the affected part. Excoriated papules and scratch marks may be seen.

Diagnosis
- Finding lice in occipital region in P.capitis, inseams of clothes in P.corporis and in pubic region in phthiriasis pubis.

Non-pharmacological treatment
- Maintenance of personal hygiene.
- Disinfection of bedding and clothing.
- All the family member should be treated simultaneously.

Pharmacological treatment
- Pediculosis capitis: Permethrin l% to be applied topically. Hair should be washed and towel dried followed by application of drug over entire scalp, hair and retro-auricular areas for 10-15 minutes and then should be rinsed with warm water.
- Pediculosis Corporis: Patient needs only to have a scrub bath and change of clothes. Laundering of clothes, especially underclothes, and bedding at high temperature and use of a hot iron with special attention to the seams of clothing.
- Phthiriasis pubis: Gamma-benzene hexachloride l% (lotion/cream/shampoo) should be applied to the entire body below the neck for 8-12 hours and then washed off.
- Tab Ivermectin: 200 mcg/kg single oral dose on an empty stomach.

LEPROSY

It is a chronic granulomatous disease caused by Mycobacterium leprae, principally affecting peripheral nerves and skin.

Clinical features (cardinal signs)
- Hypopigmented or erythematous skin lesion with or without loss/impairment of sensation, sweating and hair.
- Thickening of peripheral nerves with or without loss/impairment of function.
- Slit skin smear shows acid fast bacilli.

Classification of Leprosy

1. Ridley- Jopling classification: This classification is based on combination of clinical features, bacteriological index, immune response and histopathological features. Accordingly, it has been classified into 5 types: TT(TuberculoId), BT (Borderline tuberculoid), BB(Borderline), BL (Borderline lepromatous), LL(Lepromotous).

2. NLEP classification (for treatment purpose):

Criterion	Paucibacillary	Multibacillary
Skin lesions	1-5 lesions	6 and above
Peripheral nerve involvement	No nerve/ 1 nerve	More than one nerve irrespective of the number of lesions
Skin smear	Negative at all sites	Positive at any site

Diagnosis

Presence of at least 2 of the 3 cardinal signs

Investigations

Skin Smear for Acid Fast Bacilli, skin biopsy and rarely nerve biopsy is required in pure neural type of leprosy

Pharmacological treatment

Type of leprosy	Drugs	Supervised	Supervised	Duration
Multibacillary (adult)	Dapsone (100mg) daily	Rifampicin (600mg) monthly	Clofazimine (300mg) monthly + 50 mg daily	12 months
Paucibacillary (adult)	Dapsone (100mg) daily	Rifampicin (600mg) monthly	-	6 months
Multibacillary (paediatric)	Dapsone (2mg/kg) daily	Rifampicin (10mg/kg) monthly	Clofazimine (6mg/kg) monthly + 1mg/kg daily	12 months
Paucibacillary (paediatric))	Dapsone (2mg/kg) daily	Rifampicin (10mg/kg) monthly		6 months

Nonpharmacological treatment

- Care of insensitive hands and feet- emollient application, avoidance of trauma and burns, daily inspection and rest.
- Care of eyes – protective glasses.

- In case of deformity- use correct splints, micro-cellular rubber (MCR) shoes.

ECZEMA AND DERMATITIS

The word eczema and dermatitis are often used interchangeably. All eczemas are dermatitis but all dermatitis are not eczemas. They refer to a pattern of inflammatory response of the skin characterized by itching, redness, oedema, clustered papulo-vesicles, oozing during the acute stage, crusting and scaling in the sub-acute stage and lichenification during the chronic stage.

General Classification

1. Exogenous Eczema
   Irritant Dermatitis, Allergic contact dermatitis, Photo allergic dermatitis

2. Endogenous Eczema
   Atopic Dermatitis, Seborrheic Dermatitis, Asteatotic eczema, Pompholyx, Stasis eczema.

CONTACT DERMATITIS

Causes

Inflammatory response to an exogenous substance, immunologically or non-immunologically mediated. Common allergens are cement, metals, epoxy resins, rubber, plastics, drugs, plants, fertilizers insecticides etc.

Clinical features

- Sharp, well-defined erythema/ vesicles, erosions associated with itching and oozing.
- Lesions may or may not be confined to the site of contact.

Diagnosis & Investigations

- Mainly clinical based on thorough history taking.
- Patch testing and allergen specific IgE testing.

Treatment

Topical therapy:

- Compresses using saline, potassium permanganate, 0.25% silver nitrate solution.
- Topical corticosteroids - Mometasone furoate, 0.1%, Betamethasone dipropionate 0.05%, Betamethasone valerate 0.01%, Clobetasol 0.05%.

Systemic therapy:

- Antihistaminics - Hydroxyzine hydrochloride l0-25mg, tab cetirizine 10mg HS (0.3mg/kg/day in children)
- Tab prednisolone upto 1 mg/kg/day, tapered as soon as possible.

ATOPIC DERMATITIS

The word 'atopy' means "out of place' or 'strange" to signify the hereditary tendency to develop allergies to food, inhalant substances. Atopic Dermatitis applies to cutaneous manifestation of atopic diathesis.

Types

- Infantile Atopic dermatitis (2 months - 2 years)
- Childhood Atopic dermatitis (2 years - 12 years)
- Adult

Cause

Hereditary, environmentaI factors and infective causes.

Clinical features

- Pruritus.
- Flexural lichenification in adults. Facial and extensor involvement in infancy.
- Chronic relapsing dermatitis.
- Personal or family history of other atopic diseases as asthma, allergic rhinitis.

Treatment

- Emollients and humectants e.g. coconut oil, glycerine, etc.
- Topicul coritcosteroids of mild to moderate potency e.g.hydrocortisone valerate 1%, desonide 0.05%, fluticasone propionate 0.05%, betamethasone dipropionate 0.05%.
- Tacrolimus 0.03% and 0.1% in children above 2 years of age.
- Anti-histamines to relieve itching.

SEBORRHEIC DERMATITIS

Cause

Exact cause is unknown. Association with P Ovale /MaIassezia furfur, seborrhea

Clinical features

- Sites: Sites rich in sebaceous glands - scalp, face, upper trunk, axilla, groin.
- Erythematous patches with greasy scales.
- Mild form on the scalp is called dandruff.

Treatment

Topical therapy: in mild cases.

- Selenium sulphide shampoo 1%
- Coal tar shampoo 1%
- Topical mild steroid cream or lotions e.g. desonide 0.05%
- Anti-fungal preparations: ketoconazole 2% cream and lotion, ciclopirox olamine 1% cream and shampoo etc.

Systemic therapy: in moderate to severe cases.

- Oral anti-fungal: Ketoconazole 200 mg/day x 7 days or Terbinafine 250 mg/ day x 4 weeks or Itraconazole200mg/day x 7 days.

POMPHOLYX

It is a type of eczema affecting the palm/soles and is a nonspecific type of reaction to various provoking factors.

Causes

Direct contact with chemicals, metals and drugs, dermatophytid reaction, cigarette smoking and hyperhidrosis.

Clinical features

Deep seated itchy vesicles which looks like sago grains, appear in crops on palms and sides of fingers.

Treatment

- Potassium permanganate-(I :8000) compresses for 15 minutes 4 times/day.
- Burrows solution - aluminium acetote 1% compresses.
- Topical corticosteroids - Mometasone 0.1%, Betamethasone dipropionate 0.05%, Betamethasone valerate 0.01%, Clobetasol 0.05%.
- Topical tacrolimus 0.1%
- Antihistaminics - cetirizine 1 0 mg OD.

PITYRIASIS ALBA

It is a type of nonspecific dermatitis of unknown cause. Commonly seen in atopic individuals.

Clinical features

- Age: Predominantly in children.
- Presents as dry, scaly, irregular, rounded or oval, hypo-pigmented macules over face.

Treatment

- An emollient (coconut oil/olive oil/paraffin) to reduce scaling.

- If active signs of inflammation are present - mild topical steroid like hydrocortisone 1%, Desonide 0.05%.
- Tacrolimus ointment 0.1 % and pimecrolimus 1% cream.

ACNE VULGARIS

It is a chronic inflammatory disease of pilo-sebaceous glands of the face, neck and upper trunk. It usually affects adolescents and young adults. Occurs due to obstruction of pilo-seboceous ducts, increased sebum production, alteration in lipid composition, secondary infection with Propionibacterium acnes, and hormonal imbalance.

Clinical features

- Characterised by comedones (Blockheads (open) /white heads(closed)), papules, pustules, nodules, cysts and often scars depending upon the severity of disease.
- Sites: Predominantly face, neck, upper trunk

Assessment of severity

Grade 1 (mild)	–	Comedones, occasional papules
Grade 2 (moderate)	–	Comedones, many papules, few pustules
Grade 3 (severe)	–	Predominantly pustules, nodules and abscesses
Grade 4 (cystic)	–	Mainly cysts, abscesses, widespread scarring

Non- Pharmacological Treatment

- Avoid diet rich in sugar and milk products
- Avoid use of drugs causing acne like steroid, androgens, halogens
- Avoid use of oils, pomades and cosmetics
- Patient education about premenstrual flares and stress
- Washing face to keep skin clean and non-greasy
- Shampooing to keep scalp non-greasy

Pharmacological Treatment

Grade 1: Benzoyl peroxide gel or cream (2.5 - 5 %) or Azelaic acid 10- 20% to be applied at night.

Grade 2: Any of the following may be given:

- Topical retinoids - Tretinoin (0.025%, 0.05%) creams and gels, Adaplene (0.1 %) gel. Use only at night.
- Benzoyl peroxide gel or cream (2.5% - 10%) to be applied at night.
- Topical antibacterials like clindamycin 1%, clarithromycin 1%, Erythromycin 4%, Nadifloxocin 1% cream/ gel. To be applied twice daily.

Grade 3: Prolonged course of oral antibiotics should be added. Various antibiotic options are:

- Azithromycin 500 mg once daily for 3 days/week for 12 weeks.
- Roxithromycin 150 mg twice daily.
- Minocycline 100 mg once daily.
- Doxycycline 100 mg once daily.
- E rythromycin 500 mg twice daily.
- Isotretinoin and hormonal therapy in resistant cases.

Grade 4

- Isotretinoin therapy in nodulo-cystic acne.

URTICARIA

It is a vascular reaction of skin resulting in localized oedema of dermis and sometimes sub-cutis. Histamine is the most important mediator.

Causes

It occurs due to allergy to food such as shellfish, chocolate, peanut, meat etc. Natural food additives like yeast, citric acid, eggs or synthetic additives like azo dyes. Drugs like penicillin, NSAIDs and sulphonamides. Sometimes idiopathic.

Clinical features

Evanescent, erythematous wheals and plaques associated with itching.

Types

- Acute – less than 6 weeks
- Chronic – more than 6 weeks

Investigations

- Thoroughly investigate the patient for the cause
- Complete haemogram, AEC, Fasting Blood Sugar, urine examination, ESR, Liver Function Tests, Renal Function Tests, stool examination, urine culture/sensitivity, blood culture/sensitivity, thyroid profile, Antinucleor Antibody.

Non- pharmacological Treatment

- Avoid triggering factors
- Cold water sponging and soothing applications

Pharmacological Treatment

- Tab cetirizine 10mg at night or tab chlorpheniramine maleate 4mg twice a day
- Corticosteroids may be added if not controlled with antihistamines alone.
- Inj Epinephrine 0.5 to 1ml SC and Inj Hydrocortisone 100mg IV stat in case of laryngopharyngeal oedema.
- Cyclosporine, Azathioprine or Methotrexate may be added in resistant cases.

PSORIASIS

It is a chronic, recurrent, inflammatory, hyperproliferative disorder of skin characterized by circumscribed erythematous, scaly plaques.

Causes

Genetically mediated, association with HLA- DR3/4, drugs, alcohol, smoking, metabolic diseases. Driven by complex cascade of inflammatory mediators.

Clinical Features

- Erythematous papules and plaques with silvery white scales.
- Sites - elbows, knees, scalp, lower back, palms and soles, in a bilaterally symmetrical manner. Nails and joints may also be involved.
- Mild pruritus is present.
- Winter aggravation.
- Auspitz sign (bleeding points on removal of scales) is positive.

Types

1. Guttate Psoriasis: sudden onset of crops of erythematous scaly papules and plaques.
2. Inverse Psoriasis: involves folds, recesses of flexor surfaces e.g. axilloe, groin.
3. Nail Psoriasis: discoloration, nail pitting, subungual hyperkeratosis, 'oil drop' appearance of nail plate.
4. Psoriatic arthritis: asymmetrical oligoarthritis.

Diagnosis

Clinical and skin biopsy

Investigations

To be done in moderate to severe cases (when contemplating to start systemic drugs). Complete haemogram, liver profile, renal profile, viral markers, fasting blood sugar, urine examination, serum lipid profile, chest X- ray, skin biopsy.

Non-pharmacological treatment

- Identify and avoid triggering factors.
- Avoid stress, alcohol and smoking.
- Avoid beta-blockers and chloroquine.

Pharmacological Treatment

a) For mild cases, topical therapy is sufficient. These include

- Emollients (like coconut oil, liquid paraffin and white soft paraffin)
- Topical Corticosteroids (like mometasone furoate 0.1% cream, clobetasol propionate 0.05% cream).
- Keratolyics (like salicylic acid 3-6%, dithranol, tar preparations).
- Vitamin D analogues: Calcipotriol and calcitriol etc.

b) Moderate to severe cases :

- Tab Methotrexate 5-7.5 mg/wwek
- Tab Cyclosporine 50-100mg/day
- Cap Acitretin 25mg/day

SEXUALLY TRANSMITTED DISEASE

They are infection transmitted through sexual contact caused by bacteria, viruses or parasites. It involve the transmission of organism between sexual partners through different routes of sexual contact, either oral, anal or vaginal.

Symptoms and signs of RTIs/STIs in Men

- Urethral Discharge.
- Burning or pain during micturition or urination.
- Genital Itching.
- Inguinal swelling/scrotal Swelling / swollen and painful testicles.
- Blisters or ulcers on the genitals, anus or surrounding area, mouth, lip.
- Warts on genitals, anus or surrounding area.
- Fever, body-ache, muscle-ache, dark coloured urine, jaundice.

Symptoms and signs of RTIs/STIs in women

- Unusual Vaginal Discharge.
- Genital Itching.
- Abnormal and/or heavy vaginal bleeding.
- Dyspareunia
- Lower abdominal pain (pain below the belly button, pelvic pain).

Complications in Women

- Pelvic Inflammatory Disease (PID).
- Infertility.
- Ectopic pregnancy.
- Spontaneous abortions.
- Stillbirths.
- Low birth weight babies.
- Increased susceptibility to opportunistic infections.
- Cervical cancer.
- Chronic pelvic pain.

Complications in men

- Urethral stricture.
- Phimosis/paraphimosis.
- Disfigurement of genitals.
- Infertility.
- Cardiovascular complications (syphilis).
- Neurosyphilis.

Based on various presentations, seven major RTI/STI syndromes are incorporated in the National Guidelines on Management of RTI/STI, they are:

- Urethral Discharge
- Vaginal Discharge
- Genital Ulcers
- Inguinal Buboes
- Lower Abdominal Pain
- Acute scrotal pain and /or scrotal swelling
- Genital skin conditions

STI / RTI syndromes in men

Symptoms	Syndrome	RTIs/STIs
Urethral Discharge	Urethral / Vaginal discharge syndrome	Gonorrhoea, chlamydia, trichomonas
Genital ulcers	Genital ulcer syndrome,	Chancroid, syphilis, genital herpes
Inguinal buboes	Inguinal bubo syndrome	Lymphogranuloma venereum, Chancroid
Scrotal swelling	Painful Scrotal Swelling	Gonorrhoea, chlamydia
Genital skin conditions	Genital skin conditions	Genital warts. molluscum contagiosum, pediculosis pubis, scabies

STI Syndromes in Women

Symptoms	Syndrome	RTIs/STIs
Vaginal discharge	Vaginal discharge syndrome	Gonorrhoea, chlamydiu, trichomoniasis, herpes simplex, Candidiasis, bacterial vaginosis, cervicitis
Lower abdominal pain	Lower abdominal pain syndrome	Gonorrhoea, chlamydia, mycoplosma, gardnerella, anaerobic bacteria (bacteroids, e.g. gram positive cocci)
Genital Ulcers	Genital Ulcer syndrome	Syphilis, chancroid, genital herpes
Genital skin conditions	Genital skin conditions	Genital warts, Molluscum contagiosum, pediculosis pubis, scabies

Non-pharmacological treatment

- Educate and counsel the client and sexual partner regarding sexually transmitted infection/ reproductive tract infection, safer sex practices and importance of taking complete treatment.
- Treat partner. Advice sexual abstinence or condom use during the course of treatment. Provide condoms, educate about correct and consistent condom use.
- Refer all patients to ICTC.
- Follow up after 7 days for all sexually transmitted infections; on 3rd, 7th and 14th day for lower abdominal pain and on 7th, 14th and 21st day for inguinal bubo.
- If symptoms persist, re-assess for re-infection and advice prompt reference.
- Consider immunization against Hepatitis B.

Pharmacological treatment

- Various drug kits which are available as per Syndromic Management Protocol and provided by NACO at different health care levels in India.

Kit	Colour	Composition of kit	Syndrome/disease
	Grey	Tab. Azithromycin1g stat + TabCefixime 400 mg stat	Urethral discharge, cervical discharge, anorectal discharge, Painful scrotal swelling
	Green	Tab. Secnidazole2g stat + Cap. Fluconazole 150mg stat	Vaginal discharge
	White	Inj. Benzathine penicillin2.4mu IM Stat + Tab. Azithromycin 1g stat	Genital ulcerative disease (non-herpetic)
	Blue	Cap. Doxycycline100mg BD for 15 Days + Tab. Azithromycin 1g stat	Genital ulcerative disease (non-herpetic)
	Red	Tab. Acyclovir 400mg TDS for 7 days	Genital ulcerative disease (herpetic)

	Yellow	Tab. Cefixime 400 mg stat + Tab. Metronidazole 400mg BD for 15 days + Cap. Doxycycline 100mg BD for 14 days	Lower abdominal pain (LAP)
	Black	Cap. Doxycycline100mg BD for 21 Days + Tab. Azithromycin 1g stat	Inguinal bubo

DERMATOLOGICAL EMERGENCIES

Severe acute allergic reactions include urticaria, angioedema, anaphylaxis, Steven Johnson Syndrome, toxic epidermal necrolysis, life threatening stings and bites. Life threatening infections include necrotizing soft tissue infections, fungal and viral infections with complications. Severe dermatological conditions include: autoimmune vesiculobullous disorders, collagen vascular disorders, unstable psoriasis, cutaneous involvement in paraneoplastic disorders and systemic infections.

Treatment

Dermatological emergencies can only be managed at tertiary care centre.

Non-pharmacological treatment

- Treat the primary cause.
- Maintain CAB (circulation, airway and breathing).
- Careful monitoring of the vitals.
- Fluid and electrolyte management.
- Intake and output chart.

Pharmacological treatment (only at tertiary centres)

- Systemic corticosteroids for autoimmune blistering and collagen vascular disorders (dose depending on the severity of involvement and taper according to the response on follow up).
- Appropriate antibiotics like Tab. Azithromycin 500 mg OD for 3/5days, Tab. Erythromycin 500 mg QID for 5/7 days.
- Antiviral like Tab. Acyclovir 400 mg TDS for 7 days.
- Antifungals like Tab. Fluconazole 50-100 mg/day for 7-10 days (depending on severity).
- Immunosuppressant drugs used with caution and monitoring like cyclophosphamide 1- 1.5mg/kg/day, azathioprine 1-2mg/kg/day, cyclosporine (2mg/kg/day).
- IV Immunoglobulin and biological agents (rarely used).

ENT STANDARD TREATMENT GUIDELINES

ENT DISEASES

EAR WAX

Cerumen in the external ear canal is physiological and composed of sebaceous and ceruminous gland secretions, hair, desquamated epithelial debris, keratin and dirt.

Indications to remove cerumen

- Difficulty in examining the full tympanic membrane.
- Conductive deafness.
- Itching and earache.
- Occlusion of the external ear canal.

Management

1. Use wax solvents (paradichlorbenzene 2% solution) to soften hardened wax.
2. Removal by probe.
3. Syringing.

References

Wormald P.J, Scott Brown's Otorhinolaryngology Head & Neck Surgery, Great Britain, Hodder Arnold 2008.

COMMON COLD

- Commonly caused byviruses like Respiratory Syncytial Virus (RSV), Coronavirus, Adenovirus.
- Seen more commonly in children.
- Secondary bacterial infection: Streptococcus pneumonia, Staphylococcus aureus, Haemophilus influenza etc.

Clinical Features

- Malaise
- Nasal discharge-clear and watery or mucopurulent
- Sneezing
- Muscle aches and headache
- Loss of sense of smell
- Loss of appetite
- On anterior rhinoscopy, nasal mucosa congested and edematous. Pharynx appears slightly congested.
- Mildly enlarged, non-tender cervical lymph nodes.

Management -

A. Non pharmacological

- Washing hands to prevent transmission.
- Rest and plenty of fluids.

B. Pharmacological

1. Analgesics

- Paracetamol (Oral, IM)

Adults 0.5-1 gm every4-6 hours upto maximum of 4 gms

Children 10mgkg every 4-6 hours

- Ibuprofen (Oral)

Adults 400 mg q4-6 hrs

Children 10mg/kg 8 hourly (6-1 2yrs).

2. Decongestants: (Oral)

- Phenylephreine 5mg HS
- Pseudophedrine

Adults 60 mg P0 q4-6 hrs

Children 5-30 mg P0 q4-6 hrs

Syrup: 3mg/ml

3. Antihistamines

- Cetirizine (Oral)

Adults (6 yrs and older) 5-10mg once daily

Children (4-6yrs) 2.5 mg once daily to max of 5mg a day

- Chlorpheniramine (Oral)

Adults 4mg every 4-6 hrs as needed

Children (6-l2yrs) 2mg three to four times a day

4. Antibiotics-required when secondary infection supervenes

- Ampicillin

Adults 250 - 500mg 6 hourly

Children 50- 100 mg/kg/day P0 divided 6 hourly

- Amoxicillin

Adults 500mg P0 q12 hr

Children 25-30 mg/kg/day divided q 12 hr

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ACUTE VIRAL PAROTITIS (MUMPS)

Definition

Mumps (epidemic parotitis) is a disease of viral origin most commonly occurring in the pediatric age group.

Etiology

- Paramyxovirus
- Orthomyxovirus

Clinical Features

- Pain
- Swelling of the gland and overlying skin
- Anorexia
- Fever, Malaise
- Usually Bilateral involvement

Diagnosis

- Clinical picture is diagnostic.
- Serum and Urinary amylase
- CBC

Management

A. **Non-Pharmacological**

- Maintain proper hydration of the patient.
- Treat primary disease.

B. **Pharmacological**

1. Antipyretics and analgesics

- Paracetamol (Oral, IM)

Adults 0.5-1 gm q4-6 hours upto maximum of 4 gms

Children 10mg/kg q 4-6 hours

- lbuprofen(Oral)

Adult 400 mg 4-6 hrs

If secondary bacterial infection supervenes then appropriate antibiotics have to be started.

References

Porter S.R., Scott Brown's Otorhinolaryngology Head & Neck Surgery, Great Britain, Hodder Arnold 2008.

FACIAL PARALYSIS (BELL'S PALSY)

Definition

It is a unilateral paresis or paralysis of facial nerve with acute onset.

Etiology

No identifiable cause presents

Clinical Features

- Patient is unable to close eyes.
- On attempting, eye turns up and out.
- Tears flow from eyes.
- Dribbling of saliva from angle of mouth with drooping of angle of mouth.
- Face is asymmetrical.
- Noise intolerance of high intensity sounds on the affected side.
- Loss of taste.

It is a clinical diagnosis.

Management

A. Non-Pharmacological

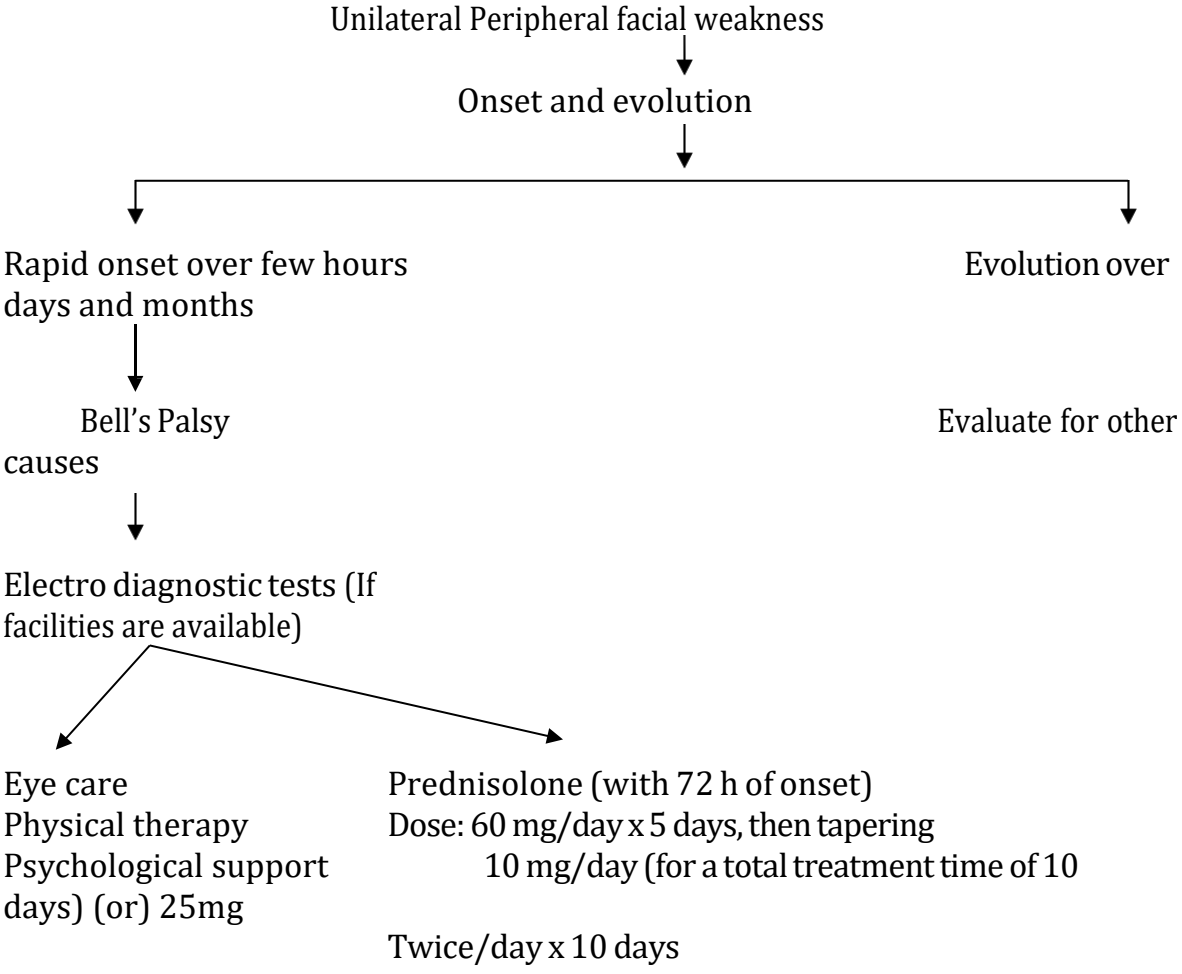
- Massage of facial muscles.
- Proper care of eye by covering the eye and using artificial tear drops to prevent keratitis.

B. Pharmacological

- Prednisolone: 1 mg/kg/day.
- For Bell's Palsy-Tab Acyclovir (200-400mg 5 times a day).

C. Surgical Treatment

Facial N. Decompression in cases which do not respond to medical therapy.



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b. Taylor DC, Bell Palsy, www.emedicine.com.

PHARYNGITIS

Definition

Inflammation of the pharynx.

Etiology

1. Bacterial pharyngitis

- Group A beta-hemolytic streptococci
- Staphylococcus aureus
- M.pneumonia
- Corynebacteriumdiptheriae

2. Viral pharyngitis

- Rhinovirus
- Corona virus
- Parainfluenza
- Influenza type A and B

3. Other causes

- Candida infection
- Dry air, allergy/postnasal drip, chemical injury, gastroesophageal reflux disease, smoking, neoplasia and endotracheal intubation. Clinical Features
- Malaise
- Sore throat
- Odynophagia
- Cough
- Fever- absent or low-grade in viral pharyngitis
- Reddish nodules on posterior pharyngeal wall.

Laboratory Diagnosis

- Throat culture
- Peripheral smear
- A complete blood counts.

Management

A. Pharmacological

1. Antibiotics 10- 14 Day Course

- Amoxicillin
  - Adults 500- 875 mg P0 TDS
  - Children 30-40 mg/kg/day in 3 divided doses
- Azithromycin
  - Adults 500mg once daily for 3 days orally
  - Children 10mg/kg once daily for 3 days

2. Analgesics

- Paracetamol (Oral, IM)
  - Adults 0.5-1 gm every 4-6 hours upto maximum of 4 gms
  - Children 10mg/kg every 4-6 hours
- lbuprofen(Oral)
  - Adult 400mg 4-6 hours

Children (6-12yrs) 10mg/kg8 hourly

3. Decongestants (Oral)

- Phenylephreine 5mg HS
- Pseudophedrine
  - Adults 60mg PO q4-6 hrs
  - Children 5-30mg PO q4-6hrs
    Syrup 3mg/ml

4. Antihistamines

- Cetirizine (Oral)
  - Adults L ⍰ 6 yrs) 5-10mg once daily
  - Children (4-6yrs) 2.5mg once daily to max of 5mg a day
- Chlorphenira mine (Oral)
  - Adults 4mg every 4-6hrs as needed
  - Children (6-1 2yrs) 2mg three to four times a day

References

1. Acerra JR, Pharyngitis, www.emedicine.com.
2. Nussenbaum B, Bradford CR, Cummings Otolaryngology Head & Neck Surgery, Philadelphia, Mosby Elsevier, 2010.

ACUTE OTITIS MEDIA

Definition

Bacterial or viral infection of the mucosal lining of the middle ear and mastoid air-cell system.

Etiology

1. Bacteria- Haemophilus influenza, Moraxella catarrhalis, Streptococcus pyogenes, Staphylococcus pyogenes, aureus
2. Environmental factors: Poor socio-economic status, Overcrowding.

Clinical Features

- Otalgia
- Hearing Loss
- Otorrhoea
- Fever
- Tympanic membrane is opaque, bulging, congested or red.

Lab Diagnosis

Bacterial swab for persistent otorrhoea and nasopharyngeal swab.

Management

A. Non-Pharmacological

- Improve socio-economic status
- Vaccination against viruses and bacteria

B. Pharmacological

1. Antibiotic

- Amoxicillin

Adults 500- 875 mg POTDS
Children 30-40 mg/kg/day in 3 divided doses

- For penicillin sensitive
  Erythromycin 30-50mg/kg In divided Oral doses

2. Decongestant And Antihistaminics
- Ephedrine nose drops
- Pseudoephedrine (Oral)
  Adults 60 mg P0 q4-6 hrs
  Children 5-30 mg P0 q4-6 hrs Syrup: 3mg/ml
- Oxymetazoline or Xylometazoline nose drops

3. Analgesics (Oral, IM)
- Paracetamol
  Adults 0.5-1 gm every 4-6 hours upto maximum of 4 gms
  Children 10mg/kg every 4-6 hours
- Ibuprofen(Oral)
  Adult 400 mg 4-6 hrs
  Children(6-l2yrs) 10mg/kg8 hourly

C. Surgical

Myringotomy in severe cases.

References

Rea P and Graham J Acute otitis media in children In Gleson Michael editor. Scott-Brown's otorhinolaryngology, Head and Neck Surgery. 7 thedn. Great Britain. Hodder Arnold; 2008.p.912-918.

CHRONIC OTITIS MEDIA

Definition

Otorrhoea of atleast 6 weeks duration in the presence of chronic tympanic membrane perforation (perforation is said to be chronic if present for 3 months).

Risk Factors

- More siblings under the age of 5
- Crowded accommodation
- Prolonged carriage rates of nasopharyngeal pathogens
- Poorer nutritional status
- Reduced exposure to medical services and supportive therapies
- Clinical syndromes like Down syndrome and cleft palate.
- Clinical Features
- Muco-purulent ear discharge in perforated TM is profuse.
- Mild to moderate hearing loss.
- Itiching and otalgia due to associated involvement of external auditory canal infection by continued and profuse ear discharge causing otitis externa.

- TM: thick opaque and perforation present in any quadrant.
- Middle ear mucosa inflammed.
- Tympanosclerosis and myrnigitis.
- Lab Investigations
- Ear swab for culture and sensitivity
- Microscopic examination
- Audiological assessment: Pure tone audiometry to asses type and degree of hearing loss Radiological Investigation
- CT Temporal bone if complications are suspected.

Management

A. Non Pharmacological

- Improvement in nutritional status.
- Improvement in living environment.
- Education about disease.
- Keepeardry.

B. Pharmacological

- Dry mopping of EAC
- Aural toilet with suction cleaning.
- Topical antibiotics- ciprofloxacin, ofloxacin, chioramphenical ear drops.

C. Surgical

- Myringoplasty.
- Tympanoplasty.
- Canal wall up mastoidectomy: CAT i.e. Combined Approach Tympanoplasty.
- Canal wall down mastoidectomy:radical or modified

References

Hamilton J. Chronic otitis media in childhood. In: Gleson Michael editor. Scott-Brown's otorhinolaryngology, Head and Neck Surgery.7 thedn. Great Britain.Hodder Arnold; 2008.
p.931-932.

OTITIS MEDIA WITH EFFUSION (OME)

Definition

Chronic accumulation of serous/mucus discharge within the middle ear cavity and sometimes the mastoid air cell system.

Etiology/ Risk Factors

- Recurrent upper respiratory infections including tonsillitis and adenoiditis
- Recurrent attacks of AOM.
- Eustachian tube dysfunction.
- Craniofacial abnormalities: cleft palate, Down or Turner syndrome
- Parental smoking

Clinical Features

- Blocking sensation of ears
- Deafness

- .Dull ear ache
- TM: dull lustreless with loss of landmarks
- Bulging of TM
- Evidence of fluid in the middle ear may be seen as air bubble
- Decreased TM mobility on valsalva and seigelisation
- Decreased school performance

Lab Investigations

- Tympanometry: Type B graph
- Pure tone Audiometry: Conductive hearing loss

Radiological

- X RAY Water's view of PNS to rule out nasal and sinus infection.
- Lateral view of nasopharynx to rule out hypertrophied adenoids.

Management

- Prevention of secretory otitis media by regular checkup and treatment of URTL
- Removal of the possible cause: Adenoidectomy and Tonsillectomy.

A. Pharmacological

Decongestants

- Phenyiephreine 5mg HS
- Pseudophedrine

  Adults 60 mg P0 q4-6 hrs

  Children 5-30 mg P0 q4-6 hrs

  Syrup: 3mg/ml

  Oxymetazoline orXylometazoline nasal drops

B. Surgery

Myringotomy and insertion of ventilation tubes in non responsive cases

References

Browning G.Otitis media wth effusion. In: Gleson Michael editor. Scott-Browns otorhinolaryngology, Head and Neck Surgery. 7 thedn. Great Britain. Hodder Arnold; 2008.p.877.

OTOMYCOSIS

Definition

Fungal infection of ear canal that often occurs due to Aspergillusniger, A. Fumigatus or Candida albicans.

Etiology

- Common in hot, humid climates.
- Secondary to prolonged treatment with topical antibiotics.
- Diabetes and immune compromised state.

Clinical Features

- Black, grey, green, yellow or white discharge with debris and musty odour
- Intense itching/discomfort

- Ear blockage
- EAC-Congested,ulcerated and the fungal mass seen.

Investigations

Blood sugar to rule out DM.

Management

- Toilet and removal of debris.
- Topical anti-fungal ear drops: Clotrimazole and Ketoconazole.
- 2% salicyclic acid in alcohol is a keratolytic agent.
- Keepeardry.

References

Amey AS. Otifsexferno and otomycosis. In: Gleson Michael editor. Scott-Brown's otorhinolaryngology, Head and Neck Surgery.7 thedn. Great Britain. Hodder Arnold; 2008.p.3351.

EXTERNAL EAR FURUNCULOSIS

Definition

It is a localised form of otitis externa resulting from infection of a single hair follicle in hair bearing area of external auditory canal.

Etiology

- Staphylococcus aureus is the most common organism.
- Diabeties mellitus is predisposing factor
- Clinical features
- Severe earache and tenderness
- Ear blockage
- Scanty and sero-sanguinous ear discharge
- Oedema and inflammation restricted to lateral segment of the canal
- If the infection is advanced abscess may be seen to be pointing into the canal
- Ear canal may be narrow

Management

A. Pharmacological

1. Analgecis
   - PAracetamol (Oral,IM)
     Adults 0.5-1gm every 4-6 hours upto maximum of 4 gms
     Children 10mg/kg 4-6 hours
   - Ibuprofen (Oral)
     Adult 400 mg 4-6 hrs
     Children 10mg/kg 8 hourly (6-1 2yrs)
2. Antibiotics
   - Oral Flucloxacillin 500mg óhrlyfor 14 days
   - Topical treatment Nasal mupirocin
3. Glycerol and ichthammol solution has a specific antistaphyloccal action.
4. Aluminium acetate solution is an astringent as well as hygroscopic agent.

B. Surgical

Incision and Drainage in case of abscess formation

References

Lalwani, A. K., 2012. Current diagnosis in otorhinolaryngology-head & neck surgery.S.I:McGraw Hill

ALLERGIC RHINITIS

Definition

It is an IgE mediated inflammation following exposure to allergen resulting in the running and blocking of nose, itching and sneezing.

Etiology/Risk Factors

- Genetics and family history
- Environment: developed urbanization, lifestyle changes, increased exposure to allergen, dietary modification.

Clinical Features

- Paroxysmal sneezing, rhinorrhoea and itching of nose.
- Perennial allergic inflammation -nasal obstruction, poor sense of smell.
- Allergic crease or allergic salute on nasal dorsum at junction of cartilaginous and bony part of nose.
- Allergic nasal mucosa bilaterally swollen, pale or bluish in colour, oedematous and covered with watery secretions.

Lab Diagnosis

- TLC, DLC
- Blood test for allergy (total IgE, specific IgE)

Management

A. Non - Pharmacological

- Allergen avoidance.
- Environmental control.
- Encase mattress and pillows in plastic covers or special allergen proof fabric.
- Remove objects that accumulate dust or place in a cabinet.
- Remove carpets, replace with washable rugs, install hardwood floor.

B. Pharmacological

1. Antihistamines

• Cetirizine (Oral)

Adults	5-10mg once daily
Children (4-6yrs)	2.5mg once daily to max of 5mg a day (6
yrs and older)	5-10mg once daily

• Fexofenadine (Oral)

Adults	180mg PO once daily
Children	30mg PO twice daily (not recommended in<2yrs of age)

• Loratidine (Oral)

Adult 10 mg P0 once daily
Children 5mg P0 once daily

2. Topical Steroids

Fluticasone, mometasone, beclomethasonedipropionate, budesonide, flunisolide acetate reduce inflammation and hyperreactivity, nasal symptoms, eye symptoms and improve sense of smell.

3. Sodium cromoglycate 2% solution for nasal drops or spray or as an aerosol powder.
4. Decongestants Xylometazoline nasal drops
5. Systemic corticosteroids for very severe symptoms during hayfeverfor short duration.

Can be combined with topical steroids.

6. Anti leukotrienes Montelukast
   Adult 10mg once a day
   Children 5mg once a day
7. Nasal douching improves quality of life.
8. Immunotherapy - updosing phase involves weekly injections for 8-16 weeks followed by maintenance injections at 4-8 weeks intervals for 3 to 5 years. **References**

1. Scadding G and Durham S .Allergic Rhniitis. In: Gleson Michael editor. Scott- Brown's otorhinolaryngology, Head and Neck Surgery.7 thedn. Great Britain. Hodder Arnold; 2008. p.1386
2. Dingra PL. disease of Ear, Nose and Throat. New Delhi: Elsevier;201 4. p.166- 169

ACUTE SINUSITIS

Definition

It is the inflammation of mucosa of nose and paranasal sinuses. It is said to be acute if duration is from 7 days to <4 weeks.

Etiology

- Mechanical obstruction of nose (Deviated nasal septum, nasal polyps, hypertrophied turbinates).
- Focal infection: nasal infection, adenotonsillitis, dental infection, trauma.
- Mucociliary clearance abnormality like cystic fibrosis.
- Allergy
- Immunodeficiency.
- Autonomic imbalance.
- Hormones: pregnancy, Oral contraceptive pills (OCP).
- Granulomatous conditions of nose.
- Idiopathic.

Clinical Features

- Facial pain or pressure over forehead or cheek(especially unilateral)
- Hyposmia/anosmia
- Nasal congestion

- Nasal discharge
- Postnasal drip
- Fever
- Cough
- Fatigue
- Maxillary dental pain
- Earfullness/pressure

Lab Diagnosis

- Diagnostic nasal endoscopy
- Culture and sensitivity test of nasal discharge

Radiological examination X Ray nose and PNS

Management

A. Non-Pharmacological

Steam inhalation & Hot fomentation

B. Pharmacological

1. Antibiotics
   - Amoxicillin
     Adults 500- 875 mg P0TDS
     Children 30-40mg /kg/day in 3 divided doses
   - Amoxillin with clavulanic acid
     Adults 625mgPOBD
     Children 375mg P0 BD
   - Erythromycin: given to those who are sensitive to penicillin.
     Adults 250-500mg PO q6-12 hrs
     Children 30-50 mg/kg/day P0 divided q6-12 hr
2. Nasal decongestants drops
   1% ephedrine oro.1%xylometazoline oxymetazoline
3. Antipyretics and analgesics
   Paracetamol, Ibuprofen

References

Beninger MS, Acute Rhinosinusitis, Scott-Brown's otorhinolaryngology, Head and Neck Surgery. 7 thedn. Great Britain. Hodder Arnold; 2008, pg 1439.

CHRONIC SINUSITIS

Definition

Inflammation of nose and paranasal sinuses that persists for 12 or more weeks characterized by

2 or more of the following symptoms

- Blockage of nose
- Discharge: anterior/posterior
- Facial pain/ pressure
- Reduction or loss of smell

Plus either

Diagnostic Endoscopy signs of

- Nasal Polyps
- Mucopurulent discharge from middle meatus
- Oedema /mucosal obstruction primarily in the middle meatus

And/ or

CT changes: Mucosal changes within OMC /sinuses

Etiology

- Mechanical obstruction of nose (Deviated nasal septum, nasal polyps, -hypertrophied turbinates)
- Focal infection: nasal infection, adenotonsillitis, dental infection, trauma
- Mucociliary clearance abnormality like cystic fibrosis
- Allergy
- Immunodeficiency
- Autonomic imbalance
- Hormones: pregnancy, Oral contraceptive pills (OCP)
- Granulomatous conditions of nose
- Idiopathic

Lab diagnosis

- Diagnostic nasal endoscopy (DNE)
- Culture and sensitivity test of nasal discharge

Radiological examination

NCCTPNS

Management

A. Non —pharmacological

- Reduce viral exposures by improved personal hygiene.
- Reduce exposure to dust, moulds, cigarette smoke; and other environmental chemical irritants.
- Smoking cessation

B. Pharmacological

- Amoxicillin plus clavulanic acid 625 mg BD in adults for 7 days
  375mg in children BD for 7 days.
- Steam inhalation and nasal saline irrigation.
- Topical Steroid Therapy: mometasone, budesonide orfluticasone spray

C. Surgical

- Caldwell luc surgery.
- Functional Endoscopic Sinus Surgery (FESS).

References

Scading G, Scott-Brown's otorhinolaryngology, Head and Neck Surgery.7 thedn. Great Britain. Hodder Arnold; 2008, pg 1469.

ACUTE TONSILLITIS

Definition

Inflammation of the pharyngeal tonsils.

Etiology/Risk Factors

- Viral or bacterial infections and immunologic factors.
- Overcrowded conditions and malnourishment.

Clinical Features

- Fever
- Sore throat
- Foul breath
- Dysphagia (difficulty swallowing)
- Odynophagia (painful swallowing)
- Tender cervical lymph nodes.

Lab Diagnosis

Throat cultures and Sensitivity test

Management

A. Non-Pharmacological

- Maintaining adequate hydration and caloric intake.
- Betadiene or Warm water gargles.

B. Pharmacological

1. Antibiotics
   - Amoxicillin with clavulanic acid
     Adults 625mgPOBD
     Children 375mg P0 BD
   - Amoxicillin
     Adults 500- 875 mg P0 TDS
     Children 30-40 mg/kg/day in 3 divided doses
   - Erythromycin: given to those who are sensitive to penicillin
     Adults 250-500mg PO q6-12 hrs
     Children 30-50 mg/kg/day P0 divided q6-12 hr

2. Analgesics:
   - Paracetamol (Oral, IM)
     Adults 0.5-1 gm every 4-6 hours upto maximum of 4 gms
     Children 10mg/kg every 4-6 hours
   - Ibuprofen
     Adults 400 mg 4-6 hrs
     Children 10mg/kg 8 hourly (6-12yrs)

References

McKerrow WS, Scott-Brown's oforhinolaryngology, Head and Neck Surgery.7 thedn. Great Britain. HodderArnold; 2008.

CHRONIC TONSILLITIS

Definition

It is defined as chronic inflammation of the palatine tonsil which occurs as a result of repeated attacks of acute tonsillitis or due to inadequately resolved acute tonsillitis.

Etiology/ risk factors

- Alpha- and beta-hemolytic streptococcal species
- S.aureus
- H.influenzae
- Local immunologic mechanisms are equally responsible in causing chronic tonsillitis.

Clinical Features

- Sore throat
- Cough
- Halitosis
- Bad taste in mouth due to pus in crypts
- Thick speech
- Difficulty in swallowing
- Sleep apneic episodes
- Persistent congestion of the anterior pillars
- Positive tonsillar squeeze
- Jugulo-digastric lymoh node enlargement

Lab Diagnosis

- CBC
- Throat swab for culture and sensitivity
- X Ray lateral view neck
- DNE to rule out co-existent adenoid hypertrophy.

Management

A. Non-Pharmacological

Maintaining adequate hydration and caloric intake.

B. Pharmacological

1. Antibiotics:

- Amoxicillin with clavulanic acid
  Adults 625mg P0 BD in adults
  Children 375mg P0 BD
- Amoxicillin
  Adults 500- 875 mg P0 TDS
  Children 30-40 mg/kg/day in 3 divided doses
- Erythromycin: given to those who are sensitive to penicillin

Adults 250-500 mg P0 q 6 hrs
Children 30-50 mg/kg/day P0 divided q 6 hrs

2. Analgesics
   - Paracetamol (Oral,IM)
     Adults 0.5-1 gm every 4-6 hours upto maximum of 4 gms
     Children 10mg/kg every 4-6 hours
   - lbuprofen
     Adult 400mg4-6hrs
     Children(6-l2yrs) 1Omg/kg8hourly

C. Surgical

Tonsillectomy

References

a) Macnamara M Acute and Chronic pharyngeal infection. In: Gleson Michael editor. Scoff- Brown's otorhinolaryngology, Head and Neck Surgery.7 thedn. Great Britain. Hodder Arnold; 2008.p.1981.
b) P1 Dhingra, S. D., 2014. Diseases of Ear,Nose and Throat & Head and NeckSurgery. New Delhi: Elsevier.

CHRONIC ADENOIDITIS

Definition

Chronic inflammation/enlargement of the adenoids causing obstruction to the nasopharyngeal airway and consequent recurrent nasosino infection, otitis media or mal-developement of the face.

Etiology/Risk Factors

Recurrent attacks of rhinitis or sinusitis.

Clinical Features

1. Nasal symptoms
   - Nasal obstruction
   - Anterior nasal discharge
   - Post nasal discharge
   - Obstructive sleep apnea
   - Hyponasal speech
2. Aural symptom
   - Recurrent otalgia
   - Deafness
   - Ear discharge
3. Throat symptoms
   - Recurrent sore throat
   - Dysphagia
   - Change in voice

Signs

- Discharge is usually seen on the floor of nasal cavity
- Cold spatula test shows decreased fogging
- Retraced or bulging TM

- Mucosal congestion of pharynx
- Granular posterior pharyngeal wall

Investigations

- Lateral neck radiograph
- Flexible or rigid nasopharyngoscopy.

Management

A. Pharmacological

- Systemic antibiotics have been used long-term (i.e. 6 wk) for lymphoid tissue infection.
- Topical nasal drops: OxymetazolineorXylometazoline

B. Surgical

Adenoidectomy

References

Lalwani, A. K., 2012. current diagnosis in otorhinolaryngology - head & neck surgery. s.I.:Mc Graw Hill.

FURUNCULOSIS OF NOSE

Definition

It is the localised inflammatory condition of the nasal vestibule involving the hair follicle caused by Staphylococcus aureus.

Etiology/Risk Factors

- Trauma
- Diabetes
- Immunodeficiency state
- Longterm steroids
- Agranulocytosis

Clinical Features

- Mild pain,redness and tenderness over the affected area.
- Swelling, which may involve tip of the nose depending on the site of affected hair follicle.
- In severe form,pain can be throbbing in nature if the area is touched.
- In more advanced form abscess formation may occur which can rupture.

Management

A. Non-Pharmacological

- Warm compresses

B. Pharmacological

- Antibiotics: Cloxacillin 500 mg P0 q6 hr
- Mupirocin ointment for 5 days in a month for 6 months
- Analgesic - Serratiopeptidase and acelofenac (100 mg BD) may reduce inflammation and pain.

Surgical

If abscess formation occurs, it can be incised and drained.

References

PL Dhingra, S. D., 2014. Diseases of Ear,Nose and Throat & Head and Neck Surgery. New Delhi:

EPISTAXIS

Definition

Bleeding from inside the nose.

Etiology

- Frequency is greatest in the autumn and winter months.
- NSAIDS intake
- Alcohol intake increases the bleeding time
- Hypertension
- Septal abnormalities
- Bleeding and Coagulation disorders
- Trauma
- Post surgery -
- Warfarin
- Liver and Kidney disorders
- Tumour

Clinical diagnosis

Bleeding from nose

Management

A. Precautions

- Avoid hard nose blowing or sneezing.
- Sneeze with the mouth open.
- Do not use nasal digital manipulation.
- Avoid aspirin and other NSAIDs.

B. Instructions for self-treatment for minor epistaxis:

- Apply firm digital pressure for 5-10 minutes by pinching the nose.
- Use an ice pack
- Practice deep, relaxed breathing.
- Use a topical vasoconstrictor.

C. Etamsylate 250-500mg P0 q4-6hour
Can be given by Oral & IV/IM route also.

D. Cauterisation

Useful in anterior epistaxis when bleeding point has been located.

Bleeding point cauterised with a bead of silver nitrate or coagulated with electrocautery.

E. Anterior and posterior nasal packing

References

McgarryGW,Scott-Brown's otorhinolaryngology, Head and Neck Surgery. 7 thedn. Great Britain. HodderArnold; 2008. Pg. 1596

OPTHALMOLOGY

STYE OR HORDEOLUM EXTERNUM

Definition

It is an acute suppurative inflammation of the glands of Zeis.

Etiology

Bacterial - Staphylococcus.

Symptoms

Acute Pain, Lid swelling, Watering.

Signs

- A painful swelling at the lid margin.
- A whitish, round, raised pus point at the affected eyelash root maybe present.

Treatment

- Hot compresses 2-3 times per day for 4-6 days.
- Topical Antibiotic eye drops like Ciprofloxacin 0.3%, Moxifloxacin, Gatifloxacin four to six times per day.
- Systemic antibiotics and anti-inflammatory medications for three or four days.
- When pus points out, it should be let out by pulling the affected lash i.e**. Epilation.**
- It should never be squeezed out.

Prevention of Recurrence

- Cleaning of lid margins.
- Application of antibiotic eye ointments at night for at least 2 months after the stye has subsided.
- Cap. Doxycycline 100 mg/day for 10 days but not in children below 8 years of age to avoid permanent colouring of teeth.
- Correction of refractive errors if any.

HORDEOLUM INTERNUM (INFECTED CHALAZION)

Definition

It is an acute suppurative inflammation of the Meibomian gland.

Symptoms

Pain, lid swelling and watering.

Signs

- A painful diffuse swelling away from the lid margin.
- Pus point is situated away from the lid margin and is on conjunctival side.

Treatment:

- Antibiotic eye drops 4-6 times per day.
- Systemic antibiotics and anti-inflammatory medication for three or four days.
- The pus should be drained by incision and curettage under systemic antibiotic cover.
- Hot fomentation.

CHALAZION

Definition

If is chronic granulomatous inflammation of the Meibomian gland.

Etiology

Obstruction of the duct of Meibomian gland due to sub-clinical infection.

Symptoms

Painless swelling of the lid, Heaviness of lid, Watering.

Signs

Painless nodular lid swelling.

Course

- Spontaneous resolution with or without hot fomentation.
- 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

- Hot fomentation.
- Incision and curettage under local anaesthesia.
- Intra-lesion injection of Depot steroids: 5- 10mg (0.1 to 0.2 ml) of Triamcinolone acetonide (40 mg/ml vial) may be helpful. Two to three injections at weekly interval may be needed.
- Correction of refractive errors, if any.

Patient education

If a Chalazion recurs soon after removal or is rapidly increasing in size or ulcerates, a biopsy must be done to rule out malignancy especially in adults above 40 years of age.

ERRORS OF REFRACTION OR AMETROPIA

Definition

Ametropia is a condition of the eye in which blurred image is formed upon the retina and vision remains sub-normal due to abnormal refractive status of the ocular optics.

Types of Refractive errors.

1. Myopia (Short sightedness)

Eye cannot see distant objects clearly. Image is focused in front of the retina and it happens when the eyeball is elongated.

2. Hypermetropia (Long sightedness)

Eye cannot see closer objects clearly. Image is focused behind the retina and happens when eye i is shorter in antero-posterior length.

3. Astigmatism

No single point focus is formed. Refraction is unequal in different meridians.

Sign/Symptoms

- Indistinct distant vision.
- Headache and pain in the eyes (Asthenopia).
- Eye strain and fatigue.
- Behavioural changes in children.

4. Presbyopia

It is a condition in which the reading distance recedes beyond convenient distance (30 cms) which usually manifests by the age of 40 years and it is a physio-pathological process which affects accommodation. Patient usually complains of eye strain on near work.

Diagnosis

- Status of decreased vision is ascertained by standardised Snellen's Charts for distant vision and Jaeger's Charts for near vision.
- The estimation of amount of refractive error for distant vision is done by Retinoscopy, Auto refractometry or by subjective verification (hit and trial method).

Treatment

a. If asymptomatic - No treatment is required.

b. If Symptomatic

- Correction by spectacles (Lenses)
- Contact lenses
- Refractive surgery

Spectacles (Lenses)

- Myopia - spherical concave lenses.
- Hypermetropia - spherical convex lenses.
- Astigmatism - cylindrical orsphero-cylindrical lenses.
- Presbyopia - spherical convex lenses depending on age and occupation of the patient.

Contact lenses

Commonly used lenses are soft contact lens, semi soft or gas permeable, soft toric lenses for astigmatism, bi-focal contact lenses for refractive errors.

Refractive Surgery

- LASIK: LaserAssistedIn Situ Keratomileusis.
- LASEK: LaserAssisted in Situ Epi-Keratomileusis.
- PRK: Photo refractive Keratotomy.
- Phakic IOL implantation.
- Conductive keratoplastyforpresbyopia using radio-frequency current.

Prevention

- Assessment of refractive errors should be done in Pre School Children (1 -3 years). The teachers should be trained for screening.
- School going children should be screened yearly.

ALLERGIC CONJUCTIVAL DISORDERS

It is inflammation of the conjunctiva due to allergic or hypersensitive reaction to local or systemic allergens, which may be immediate or delayed.

Common types

1. Simple allergic conjunctivitis

- Mild, nonspecific recurrent episodes occur with renewed contact with the allergen.

2. Vernal keratoconjunctivitis (Spring Catarrh)

- Recurrent, bilateral, self-limiting, allergic inflammation of conjunctiva.
- Periodic seasonal incidence in young patients, usually males between to 20 years.
- Common in summer and greater prevalence in tropics, less in temperate zones and nonexistent in cold climatic zones.
- In chronic stage cobble-stone appearance on the sub-tarsal conjunctiva is seen.

3. Phlyctenularkerato conjunctivitis

- Nodular allergic reaction by conjunctival and corneal epithelium to endogenous allergens.

ORTHOPEDICS STANDARD TREATMENT GUIDELINES

CONGENITAL ANOMALIES CTEV

(CLUB FOOT)

Introduction

Congenital club foot is a gross deformity of foot present at birth. Talipesequinovarus is the term most commonly used for clubfoot. Talipes is a generic term for any foot deformity that centers the talus. Equinus implies that the foot is flexed in the plantar direction. Varus means inwards turning of the foot.

The incidence of congenital clubfoot is approximately one in every 1000 live births. I deformities occur in 50% of patients. Several theories have been proposed regarding the cause of clubfoot. One is that a primary germ plasm defect in the talus causes continued plantar flegiii inversion of this bone, with subsequent soft- tissue changes in the joints and musculotendinous complexes.

Another theory is that primary soft-tissue abnormalities within the neuromuscular unit secondary bony change. Clubfoot should best be thought of as a spectrum of deformities. Clubfoot may occur as an isolated disorder or in combination with various syndromes and other anomalies, such as arthrogryposis, sacral agenesis, amniotic bands, Larsen syndrome, diastrophic dwarfism, Freeman-Sheldon syndrome, and myelodysplasia or spina bifida: with or without meningoceal / myelomeningoceal.

Clinical features

Clinically, the deformity is readily apparent at birth. The child presents with the foot in severe supination with a fixed equinus deformity, heel varus, forefoot and midfoot adduction, and varying amounts of cavus deformity. The involved foot is generally smaller than the opposite side with varying amount of calf atrophy.

The head of the talus is prominent and easily palpable on the dorsolateral aspect of the foot. Depending on the severity of the cavus deformity, there may be a deep skin crease across the planter medial aspect of the midfoot. The foot cannot be passively manipulated into the neutral position

Investigation

Though, the deformity is very much evident on examination, but still X-rays must be taken to evaluate the problem

Radiography:

Radiographs should be included as part of the evaluation of clubfoot, before, during, and after treatment. In a nonambulatory child, standard radiographs include anteroposterior and stress dorsiflexion lateral radiographs of both feet. Antero -posterior and lateral standing radiographs may be obtained for an older child.

Important angles to consider in the evaluation of clubfoot are the talocalcaneal angle on the anteroposterior radiograph, the talocalcaneal angle on the lateral radiograph, and the talus-first metatarsal angle

The anteroposterior talocalcaneal angle in normal children ranges from 30 to 55 degrees. In clubfoot, this angle progressively decreases with increasing heel varus. On the dorsiflexion lateral radiograph, the talocalcaneal angle in a normal foot varies from 25 to 50 degrees; in clubfoot, this angle progressively decreases with the severity of the deformity to an angle of 0 degrees. The tibiocalcaneal angle in a normal foot is 10 to 40 degrees on the stress lateral radiograph. In clubfoot, this angle generally is negative, indicating equinus of the calcaneus in relation to the tibia. Finally, the talus-first metatarsal angle is a radiographic measurement of forefoot adduction. This is useful in the treatment of metatarsus adducts alone, but is equally important in the treatment of clubfoot to evaluate the position of the forefoot. In a normal foot, this angle is 5 to 15 degrees on the anteroposterior view; in clubfoot, it usually is negative, indicating adduction of the forefoot.

Treatment

The goal of treatment in clubfoot deformity is to obtain and maintain the foot in plantargrade position: Treatment should be initiated immediately on diagnosis, preferably within the first week of life. Treatment for the newborn with clubfoot is by manipulation and then casting to maintain the correction obtained through manipulation. Corrections begun at a later age may be more difficult owing to ligamentous contracture and joint deformity. Toe-to-groin plaster casts are used to maintain the corrections obtained through manipulation. The equinus deformity is the last deformity corrected to prevent development of a rocker- bottom foot. Casts are changed at weekly intervals, and most deformities are

corrected in 5 to 7 casts. Successful treatment rates by casting regimens alone vary in the literature from 85 to 95%.

Treatment consists of weekly serial manipulation and casting during the first 6 weeks of life, followed by manipulation and casting every other week until the foot is clinically and radiographically corrected casting should be as follows: first, correction of forefoot adduction; next, correction of heel varus; and finally, correction of hindfoot equinus. Correction should be pursued in this order so that a rocker-bottom deformity would be prevented by dorsiflexing the foot through the hindfoot rather than the midfoot.

The Ponseti method consists of two phases: treatment and maintenance. The treatment phase should begin as early as possible, optimally within the first week of life. Gentle manipulation and casting are done weekly. Each cast holds the foot in the corrected position, allowing it to reshape gradually. Generally five to six casts are required to correct the alignment of the foot and ankle fully. At the time of the final cast, most infants (70%) require percutaneous Achilles tenotomy to gain adequate lengthening of the Achilles tendon.

The first cast application corrects the cavus deformity by aligning the forefoot with the hindfoot, supinating the forefoot to bring it in line with the heel, and elevating (dorsiflexing) the first metatarsal. It may be easier to apply the cast in two stages: first, a short leg cast to just below the knee, then extension above the knee when the plaster sets. Long leg casts are essential to maintain a strong external rotation force of the foot beneath the talus and to allow adequate stretching of the medial structures, especially the posterior tibial tendon. One week after application, the first cast is removed, and after about 1 minute of manipulation, the next toe-to-groin cast is applied. Manipulation and casting at this stage are focused on abducting the foot around the head of the talus, with care to maintain the supinated position of the forefoot and avoid any pronation. During these manipulations, the navicular can be felt reducing over the talar head by a thumb placed on the head of the talus (left thumb for a right clubfoot and right thumb for a left clubfoot). A crucial point in the Ponseti technique is that the heel is never directly manipulated. With reduction of the talar head beneath the navicular, correction of the talus, navicular, and cuboid causes the calcaneus to abduct and evert.

Manipulation and casting are continued weekly for the next 2 to 3 weeks to abduct the foot gradually around the head of the talus. The foot should never be actively pronated; however, the amount of supination is gradually decreased over

these several casts until the forefoot is in neutral position relative to the longitudinal axis of the foot.

The final cast is applied with the foot in the some maximally abducted position and dorsiflexed 15 degrees. In most children, a percutaneous Achilles tenotomy is done to prevent development of a rocker-bottom deformity. The foot is casted in the final position J approximately 70 degrees of abduction and 15 degrees of dorsiflexion for 3 weeks. Five or

casts usually are necessary to correct the clubfoot deformity.

Maintenance Phase

When the final cast is removed, the infant is placed in a brace that maintains the foot inift corrected position (abducted and dorsiflexed). The brace (foot abduction orthosis/Dennis Brown Splint) consists of shoes mounted to a bar in a position of 70 degrees of external rotation and 15 degrees of dorsiflexion. The distance between the shoes is set at about 1 inch wider than width of the infant's shoulders. This brace is worn 23 hours each day for the first 3 months casting and then while sleeping for 2 to 3 years.

If the patient comes late, then surgical intervention in the form of Postero-Medial release or application of JESS frame is required.

CONGENITAL DISLOCATION OF HIP

Introduction

This condition, also known as hip dysplasia or developmental dysplasia of the hip (DDH) has been diagnosed and treated for several hundred years. Developmental dysplasia of the hip ((DDH) describes a spectrum of conditions related to the development of the hip in infordsmWil young children. It encompasses abnormal development of the acetabulum and proximal femur and mechanical instability of the hip joint.

New born often have physiologic laxity of the hip and immaturity of the acetabulum during the first few weeks of life. In most cases, the laxity resolves, and the acetabulum proceeds to develop normally. With assessment of risk factors, serial physical examination of the hips, and appropriate use of imaging, most children with pathologic hips can be correctly diagnosed and treated without long- term sequel.

Definition:

Congenital dislocation of the hip generally includes subluxation of the femoral head, acetabular dysplasia and complete dislocation of the femoral head from the true acetabulum.

The incidence of DDH is higher in girls, perhaps because females are more susceptible than males to the maternal hormone relaxin, which may contribute to ligamentous laxity. The left hip is affected three times more often than the right hip, which may be related to the left occiput anterior position of most non breech infants.

Clinical features:

Asymmetry of the thigh or gluteal folds, limb length discrepancy and restricted motion (especially abduction) can be signs of a dislocated hip

Hip clicks or pops can sometimes suggest hip dysplasia but a snapping sound can occur in normal hip from developing ligaments in and around the hip joint.

Limited range of motion

Parents may have difficulty diapering because hips can't fully spread.

Pain is normally not present in infants and young children with hip dysplasia but is the most common symptom of hip dysplasia during adolescence and young adults.

A painless but exaggerated waddling limp or leg length discrepancy are the most common findings after learning to walk. If both hips are dislocated then limping with marked swayback may become noticeable after the child starts walking.

According to the AAP guideline, the most reliable sign in the three-month- old infant is limitation of abduction. Other features of DDH at this age include asymmetry of the thigh folds, relative shortness of the femur with the hips and knees flexed (called the Allis or Galeazzi sign) and a discrepancy of leg lengths.

Investigations:

One specific method, called the Ortolani test, begins with each of the examiner's hands around the infant's knees, with the second and third fingers

pointing down the child's thigh. With the legs abducted (moved apart), the examiner may be able to hear a distinct clicking sound, called a hip click, with motion. If symptoms are present with a noted increase in abduction, the test is considered positive for hip joint instability. It is important to note this test is only valid a few weeks after birth.

The Barlow method is another test performed with the infant's hip brought together with knees in full bent position. The examiner's middle finger is placed over the outside of the hip bone while the thumb is placed on the inner side of the knee. The hip is abducted to where it can be felt if the hip is sliding out and then back in the joint. In older babies, if there is a lack of range of motion in one hip or even both hips, it is possible that the movement is blocked because the hip has dislocated and the muscles have contracted in that position. Also in older infants, hip dislocation may be present if one leg looks shorter than the other.

By eight to 12 weeks of age, the Ortolani and Barlow tests are no longer useful, regardless of the status of the femoral head. At this age, capsule laxity decreases and muscle tightness increases.

The Galeazzi sign is a classic identifier of unilateral hip dislocation. This is performed with the patient lying supine and the hips and knees flexed. The examination should demonstrate that one leg appears shorter than the other. Although this finding is usually due to hip dislocation, it is important to realize that any limb-length discrepancy results in a positive Galeazzi sign.

X-ray films can be helpful in detecting abnormal findings of the hip joint. X rays may also be helpful in finding the proper positioning of the hip joint for treatment. Ultrasound has been noted as a safe and effective tool for the diagnosis of congenital hip dysplasia.

Ultrasound has advantages over x rays, as several positions are noted during the ultrasound procedure. This is in contrast to only one position observed during the x ray.

Radiographs are of limited value during the first few months of life but are more reliable in infants four to six months of age, when the ossification center develops in the femoral head. According to the guideline, ultrasonography and radiography are equally effective imaging studies for detecting DDH in infants four to six months of age.

Treatment:

The objective of treatment is to replace the head of the femur into the acetabulum and, by applying constant pressure, to enlarge and deepen the socket. In the past, stabilization was achieved by placing rolled cotton diapers or a pillow between the thighs. The child may be dressed in two or three diapers called double or triple diapering. Both these techniques keep the knees in a frog-like position. In the early 2000s, the Pavlik harness and von Rosen splint were commonly used in infants up to the age of six months to spread the legs apart and force the head of the femur into the acetabulum. A stiff shell cast, called a splint, may be also used to achieve the same purpose. In some cases, older children between six to 18 months old may need surgery to reposition the joint. Also at this age, the use of closed manipulation may be applied successfully, by moving the leg around manually to replace the joint. Operations are performed to reduce the dislocation of the hip and to repair a defect in the acetabulum. A cast is applied after the operation to hold the head of the femur in the correct position. As of 2004 the use of a home traction program was more common. However, after the child is eight years of age, surgical procedures are primarily done for pain reduction measures only. Total hip surgeries may be inevitable later in adulthood.

Non surgical treatment methods:

These methods are most common when baby is less than 6 months of age. They typically consist of bracing a baby in such a way so that his/her hips are kept in a better position hip joint development. The goal is to influence the natural growth process so a more stable hip joint is developed.

Pavlik Harness

The Pavlik Harness is one type of brace used to treat DDH. It has straps that are fastened around the babys legs and held up by shoulder and chest straps. This holds the hips and knees up with the legs apart. This is the best position for the hip joint to be in. It allows contact between the thigh and hip bones and helps strengthen the muscles and ligaments of the hip while it is developing.

Hip abduction braces

A brace can be used for infants to hold their hips in a properly aligned position to

Encourage normal hip joint development. Also called fixed abduction braces that hold the legs apart and are flexible like pavik harness.

Traction

Sometimes a few weeks of traction are used to stretch the ligaments before attempting a surgical treatment like a closed reduction. Traction is commonly used in Europe and Asia. The reason and benefits of traction remain controversial.

If not treated by these methods, then surgical intervention is needed.

SPINA BIFIDA

Introduction

Spina bifida literally means "split spine". Spina bifida happens when a baby is in the womb and the spinal column does not close all the way. Spina bifida, the most common neutral tube defect (NTD), is one of the most devastating of all birth defects. It results from the failure of the spine to close properly during the first month of pregnancy. In severe cases, the spinal cord protrudes through the back and maybe covered by skin and a thin membrane. Surgery to close a newborn's back is generally performed within 24 hours after birth to minimize the risk of infection and to preserve existing function in the spinal cord.

Because of the paralysis resulting from the damage to the spinal cord, children born with Spina bifida may need surgeries and other extensive medical care. The condition can also cause bowel and bladder complications. A large percentage of children born with Spina bifida also have hydrocephalus.

Types of Spina bifida:

1. Spina bifida Occulta : This is a mild form of Spina bifida which is very common. Estimates vary but between 5% and 10 % of people may have Spina bifida occulta. Ther is an opening in one or more of the vertebrae (bones) of the spinal column without apparent damage to the spinal cord.

2. Meningocele : The meninges, or protective covering around the spinalcord, have pushed out through the opening in the vertebrae in the sac called the "Meningocele". However, the spinal cord remains intact. This form can be repaired with little or no damage to the spinal cord.

3. Myelomeningocele : This is the most severe form of the spina bifida in which a portion of the spinal cord itself protrudes through the back. In some cases, sacs are covered with skin; in others, tissue and nerves are exposed.

Types and Clinical Features:

Spina Bifida Occulta

This mildest form results in a small separation or gap in one or more of the bones (vertebrae) of the spine. Because the spinal nerves usually aren't involved, most children with this form of spina bifida have no signs or symptoms and experience no neurological problems.

Visible indications of spina bifida occulta can sometimes be seen on the newborn's skin above the spinal defect, including:

- An abnormal tuft of hair
- A collection of fat
- A small dimple or a birthmark
- Skin discoloration

Many people who have spina bifida occulta don't even know it, unless the condition is discovered during an X-ray or other imaging test done for unrelated reasons.

Meningocele

In this rare form, the protective membranes around the spinal cord (meninges) push out through the opening in the vertebrae. Because the spinal cord develops normally, these membranes can be removed by surgery with little or no damage to nerve pathways.

Myelomeningocele

Also known as open spina bifida, myelomeningocele is the most severe form - and the form people usually mean when they use the term "spina bifida.'ln myelomeningocele, the baby's spinal canal remains open along several vertebrae in the lower or middle back. Because of this opening, both the membranes and the spinal cord protrude at birth, forming a sac on the baby's back. In some cases, skin covers the sac. Usually, however, tissues and nerves are exposed, making the baby prone to life-threatening infections.

Neurological impairment is common, including:

- Muscle weakness, sometimes involving paralysis
- Bowel and bladder problems
- Seizures, especially if the child requires a shunt
- Orthopedic problems - such as deformed feet, uneven hips and a curved spine (scoliosis)

Investigations Physical

Examination

The most obvious finding on physical examination is some degree of motor and sensory loss. Neurologic impairment is classified by traditional neurosegmental levels based on the clinically determined strength of specific muscle groups. The functional motor level does not always correspond to the anatomic level of the lesion.

In addition, it is important to realize that the motor paresis may be asymmetrical, that it may not correspond to the sensory level, and that it may result from a combination of upper and lower motor neuron lesions. Serial measurements and accurate documentation of the functional level of the lesion allow for early detection of progressive neurologic deterioration related to a variety of associated CNS problems.

In most cases, spina bifida is diagnosed prenatally, or before birth. However, some mild cases may go unnoticed until after birth (postnatal).Very mild forms (spinal bifida occulta), in which there are no symptoms, may never be detected.

Prenatal Diagnosis

The most common screening methods used to look for spina bifida during pregnancy are second trimester (16-18 weeks of gestation) maternal serum alpha fetoprotein (MSAFP) screening and fetal ultrasound. The MSAFP screen measures the level of a protein called alphafetoprotein(AFP), which is made naturally by the fetus and placenta. During pregnancy, a small amount of AFP normally crosses the placenta and enters the mother's blood stream. If abnormally high levels of this protein appear in the mother's bloodstream, it may indicate that the fetus has an "open" (not skin-covered) neural tube defect. The MSAFP test, however, is not specific for spina bifida and requires correct gestational dates to be most accurate; it cannot definitively determine that there is a problem with the fetus. If a high level of AFP is detected, the additional testing, such as an ultrasound or amniocentesis to help determine the cause.

The second trimester MSAFP screen described above may be performed alone or as part of a larger, multiple-marker screen. Multiple-marker screens look not only for neural tube defects, but also for other birth defects, including Down syndrome and other chromosomal abnormalities. First trimester screens for chromosomal abnormalities also exist but signs of spina bifida are not evident until the second trimester when the MSAFP screening is performed.

Amniocentesis—may also be used to diagnose spina bifida. Although amniocentesis cannot reveal the severity of spina bifida, finding high levels of AFP and other proteins may indicate that the disorder is present.

Postnatal Diagnosis

Mild cases of spina bifida (occulta, closed) not diagnosed during prenatal testing may be detected postnatally by plain film X-ray examination. Individuals with the more severe forms of spina bifida often have muscle weakness in their feet, hips, and legs that result in deformities that may be present at birth. Magnetic resonance imaging (MRI) or a computed tomography (CT) scan is done to get a clearer view of the spinal cord and vertebrae. If hydrocephalus is suspected, a CT scan and/orX-ray of the skull to look for extra cerebrospinal fluid inside the brain.

Treatment:

Spinabifida Occulta may not need any surgery, but just a watch on the patient is requiredto be vigilant that he/she does not develop any neurological symptoms. Other formsneed immideate surgical intervention, that too preferably from a paediatric neurosurgeon.

Physiotherapy

Physiotherapy is one of the most important ways of helping your child manage their condition so they're as independent as possible. For spina bifida, the main aim of physiotherapy is to promote movement and independent mobility to prevent the leg muscles from weakening.

Occupational therapy

An occupational therapist can identify problem areas in everyday life, such as getting dressed, and will help work out practical solutions. This can be by encouraging certain movements or providing equipment, such as handrails, to make the activity easier.

Assistive technology

Assistive technology can help children with spina bifida gain more independence and control over their symptoms.

Children with muscle weakness of the lower limbs will require a wheelchair. Electric wheelchairs are available, but using a manual wheelchair can help maintain good upper body strength. Leg braces and other walking aids can be used by children who have weakness to the muscles of the lower legs.

Computers are a good tool for children with learning disabilities. Software is available to help children organise their activities and plan their school work. There are also many educational programmes that use text and sound to help improve a child's reading ability.

NEUROLOGICAL DISORDERS

CEREBRAL PALSY

Introduction

It is Chronic disability of central nervous system origin characterised by aberrant control of movement of posture, appearing early in life and not the result of progressive neurological disease. Its an upper motor neurone lesion. Muscle tone is increased in the involved group of muscles. This is the reason, these children are also called as Spastic Children. Mental retardation is a usual accompaniment. Hence, usual diagnosis labelled is MRCP (mental retardation and cerebral palsy)

Types of Cerebral Palsy

Spastic: Hemiplegia, Diplegia, Paraplegia, Quadriplegia

Ataxic

Dyskinefic	Dystonic	Hypokinesia
		Hypertonia
	Chored-Athetoid	Hyperkinesia
		Hypotonia

Clinical Features

Usually history of late cry after birth can be elicited and sometimes there is history of some infection of the brain at or after birth

Early Signs of Cerebral Palsy:

1. Birth History

   - Prematurity.
   - Seizures.
   - Low apgars.
   - Intracranial haemorrhage.
   - Periventricular leucomalacia.

2. Delayed Milestones

3. Abnormal Motor Performance. Muscles are spastic. Reflexes' are increased.

- Handedness.
- Reptilian crawl.
- Toe waking.
- Scissoring gait

4. Altered Tone.

5. Persistence of primitive reflexes.

6. Abnormal posturing

7. Maternal fever> 38°c + Chorioamnionitis associated with increased risk of cerebral palsy.

Prevalence and incidence of cerebral palsy

- In Low Birth Weight Babies.
- More in Males (<58%).
- Incidence of Dyskinetic Cerebral Palsy.
- In Lowest Socio-economic Groups.
- Maternal Age and Parity.
- U Shaped Curve <20 Years -> 34 Years.
- 4 Children or >.
- Breach Delivery

Cerebral Palsy Associated Disabilities

- Mental retardation 1/3 N.1/2 I.Q. <55.
- Epilepsy 20-50 %> generalised.
- Speech disorders 50% delay/dysarthria.
- Vision and hearing 25%.
- Behaviour abnormalities.
- Learning difficulties.

Common Management Problems in Cerebral Palsy

- Feeding Problems: Failure to suck.
- Tongue trusting, gagging and choking.

- Vomiting and regurgitation.
- Dribbling.
- Constipation.
- Crying, screaming and sleep disturbances.
- Chilblains and cold injury.
- Growth.

Treatment of Cerebral Palsy

1. Parent guidance.
2. Physiotherapy
3. Orthopaedic correction of the deformities
4. Speech and Occupational Therapy.
5. Medical.
6. Psychiatric

Management of Spasticity in Cerebral Palsy

- Oral Medicines:
  - Baclofen
  - Diazepam
  - Tizanid
  - Dantrolene
- Intrathecal Baclofen.
- Botulinum Toxin.
- Selective Posterior Rhizotomy.

Prevention

1. Antenatal and Neonatal care.
2. Early detection and advice.
3. Drugs.
4. Immunization and screening
5. Genetic counselling.

6. Health education

POST POLIO RESIDUAL PARALYSIS (PPRP)

Introduction

Poliomyelitis is an acute viral infection caused by polio virus which attacks the anterior horn cells of the spinal cord. The patients coming to orthopaedic surgeons are actually showing the post polio residual paralysis(PPRP). The damaged anterior horn cells can never be recovered. During the acute phase, when the child is treated by the paediatric physician, care must be taken to splint the limbs in functional positions, so that the deformity formed is minimum.

The neurones affected are mostly of the lower limb and usually one limb is involved more than both the limbs. Upper limb involvement is uncommon.

For this condition, muscle power cannot be improved, but only deformities can be corrected. Muscle transfers only can improve the power in some muscle groups(Paralysed once).

Clinical Features

- It is a flaccid type of paralysis, where the muscle tone is decreased.
- Muscle power is decreased, may be from 0 to 4.
- Most common muscle group involved is Quadriceps Femoris and Teridoachillis, though other groups may also be involved.
- Patient has short limb with muscle wasting.
- Deformities at hip, knee and ankle can be seen, depending upon the severity of involvement.
- Mostly hand on knee gait is seen.
- Secondary changes may be seen in Hip and Spine.
- History is since child hood and not since birth.

Investigations

- Clinical examination and history is of prime importance.
- X-Rays of the deformed areas must be taken to assess the degree of severity which needs to be corrected.
- X-rays will show underdeveloped bones, in addition to various deformities at the joints.

Treatment:

- In the initial phases of acute infection, care must be taken to splint the joints in functional positions so as to avoid the severe contracture formations.
- In late stages, splintage and sometimes traction to rectify the deformities can be of some help.
- Mostly these people need various surgical procedures, depending upon the deformities present, to rectify the deformities or tendon transfer surgeries to improve the muscle power in a particular group of muscles or the limb lengthening procedures to equal both the limbs.
- Surgery may be needed at Hip, Knee or ankle alone or at a combination of these joints.

METABOLIC AND ENDOCRINE DISORDERS

OSTEOPOROSIS

Osteoporosis means weak bones. In this common disease, bones loose minerals like calcium and the amount and variety of bone propeins is altered. They become fragile and break easily. The compact woven structure of the bone becomes loosely woven. It is a skeletal systemic disease characterised by low bone mass and microarchitectural deterioration of bone tissue with consequent increase in bone fragility and susceptibility to fracture.

As per WHO, it is defined as . . .0 bone mineral density that is 2.5 strandard deviations or more below the mean peak bone mass (average of young, healthy adult) as measured by DXA.

The term" Established Osteoporosis" includes the presence of a fragility fracture. It is classified as:

Primarylype 1, (post menopausal osteoporosis)

Primarylype 2.(Senile Osteoporosis occurring after 75 yrs in both males and females.)
Secondary Osteoporosis.(Due to some disease or drugs).

Osteoporosis can lead to Kyphosis, Pain, Height loss or fractures.

Commonest fracture sites being Spine, hip and wrist.

It is also called The" Silent Disease" as the bone loss occurs without symptoms.

Bones are living organs and Calcium is deposited and withdrawn from bones daily. Bones build to about age 30. We need to build up a healthy bone account while young and continue to make deposits with age. After mid-30's, you begin to slowly lose bone mass. Women lose bone mass faster after menopause, but it happens to men too. Bonescan weaken early in life without a healthy diet and the right kinds of physical activity.

Risk factors:

Age, Prior fragility # after 50 yrs age, Underweight for height., Smokers, Female gender, Low BMI, Heredity, Early menopause, More than 2 drinks several times a week, Diet poor in calcium, Less activity, Medications like : Oral glucocorticoids (steroids),Cancer treatments (radiation, chemotherapy), Thyroid medicine, Antiepileptic medications, Gonadal hormone suppression, Immunosuppressive agents, Medical Conditions- like: Hyperthyroidism, Chronic lung disease, Cancer, Inflammatory bowel disease, Chronic liver or kidney disease, Hyperparathyroidism, Vitamin D deficiency, Cushing's disease, Multiple sclerosis, Rheumatoid arthritis.

Clinical Features

In itself, there are not much clinical symptoms. Generralised bone pains, feeling of loss of height, development of kyphosis in old age can be suggestive. Fracture with a trivial injury catches the diagnosis and the features will depend upon the site of fracture.

Investigations

- Plain X-ray of the spine will show Photo frame appearance.
- X-ray of the pelvis with both hip joints can help us grade the osteoporosis as per Singh's Index.
- DXA scan for BMD when T-score is less than 2.5
- Renal function tests
- Serum calcium, Albumen and phosphorus levels.
- Serum Vit-D level.
- Biochemiocal markers of bone resorption

Prevention

The National Osteoporosis Foundation (NOF) recommends FIVE simple steps to bone health and osteoporosis prevention

- Get your daily recommended amounts of calcium and vitamin D.
- Engage in regular weight- bearing exercise
- Avoid smoking and excessive alcohol

If this is your age	Then you need this much calcium each day (mg)
0 to 6 months	210
7 to 12 months	270

1 to 3 years	500
4 to 8 years	800
9 to18 years	1,300
19 to 50 years	1,000
Over 50 years	1,200

- Talk to your doctor about bone health
- Have a bone density test and take medication when appropriate.

How to get enough Calcium everyday'

Dietary Calcium Sources

- Dairy- low fat yogurt, skim milk, cheese, chocolate pudding, ice milk, ice cream or frozen yogurt.
- Protein- tofu, sardines, salmon
- Vegetables- turnip greens, Spinach, Broccoli, collard greens
- Other foods: cheese pizza, calcium fortified orange juice, vegetable lasagnia.
- An easy way to meet calcium needs is consuming 3 cups (8 oz.) each day of fat-free or low-fat milk or equivalent milk products in combination with a healthy diet.

Vitamin D

Main dietary sources of vitamin D are: Fortified milk (400 IU per quart) Some fortified cereals, Cold saltwater fish (Example: salmon, halibut, herring, tuna, oysters and shrimp) Some calcium and vitamin/mineral supplements

From sunlight exposure

Vitamin D is manufactured in your skin following direct exposure to sun. Amount varies with time of day, season, latitude and skin pigmentation. 10-15 minutes exposure of hands, arms and face 2-3 times/week may be sufficient (depending on skin sensitivity) Clothing, sunscreen, window glass and pollution reduce amount produced.

Drugs used for Post-menopausal Osteoporosis Bisphosphonates

Alendronate	70 mg/wk
Risendronate	35 mg/wk
Ibandronate	150mg/mo
Calcitoni n	100 U/d by inj. Or 1 Nasal spray/d
Raloxifene (non-hormonal)	60 mg/d
PTH (Teriparatide)	20 ugld byinl.

RICKETS

Introduction

Rickets is softening of bones in children and can be defined as a consequence of the vitamin D deficiency and may occur due to calcium and phosphorus metabolic disorders. Blood analysis shows hypocalcemia and hypophosphatemia. Histologically, there is Failure in mineralisation of the bone and cartilaginous tissues and clinically it manifests as skeletal growth disorder.

Risk factors

- Living in northern latitudes (>30°);
- Dark skinned children;
- Decreased exposure to sunlight (polluated geographical areas, humid climate)
- Maternal vita min, Ddeficiency;
- Diets low in calcium, phosphorus and vitamin D, e.g. exclusive breast-feeding into late infancy, toddlers on unsupervised "dairy-free" diets;
- Macrobiotic, strict vegan diets;
- High phytic acid diet, e.g. chapattis;
- Prolonged parenteral nutrition in infancy with an inadequate supply of intravenous calcium and phosphate;
- Intestinal malabsorption: defective production of 25 (OH) D3 - liver disease. Increased metabolism of 25 (OH) D3 - enzyme induction by anticonvulsants;

Defective production of 1,25 (OH) 2133

- Hereditary type I vitamin D-resistant (or dependent) rickets (mutation which abolishes activity of renal hydroxylase);
- Familial (X-linked) hypophosphataemic rickets - renal tubular defect in phosphate transport;
- Chronic renal disease;
- Fanconi syndrome (renal loss of phosphate)
- Target organ resistance to 1,25 (OH)2D3- hereditary vitamin D-dependent rickets type II (due to mutations in vitamin D receptor gene).

Calcium deficiency rickets can be classified in to 3 grades- I, II, Ill, Depending

on the duration, evolution and the complication:

1. Grade I, II, III; evolution acute, subacute, recidivant.

2. Depending on vitamin D insufficiency:

    - Diet
    - Infections
    - Food diversification
    - Habitual
    - No prophylaxis
    - Prophylaxis with low dose
    - Phenobarbital induced

Rickets may cause

- Rickets tetany
- Convulsions
- Respiratory disorders
- Cardiac disorders
- Skeletal deformation
- Frequent illness

Clinical manifestations:

Rickets may develop in any age of an infant, more frequent at 3-6mo, early in prematures.

- The first signs of hypocalcaemia are CNS changes- excitation, restlessness, excessive sweated during sleep and feeding, tremors of the chin and extremities.
- Skin and muscle changes- pallor, occipital alopecia, fragile nails and hair, muscular hypotony, motor retardation.
- Complications- apnoea, stridor, low calcium level with neuromuscular irritability (tetany).
- CNS changes are sometimes interpreted as CNS trauma and the administration of the Phenobarbital which activates the hepatic enzyme may deactivates Vit. D and within 1 - 2wk of the treatment with Phenobarbital the clinical stage worsens.

Acute Signs

Have acute and subacute clinical signs

Craniotabes - acute sign of rickets, osteolyses detected by pressing firmly over the occipital or poster parietal bones, ping-pong ball sensation will be felt. Large anterior fontanella, with hyperflexible borders, cranial deformation with asymmetric occipital flattening.

Subacute Signs

- Subacute signs are all the following: frontal and temporal bossing
- False closure of sutures (increase protein matrix), in the X-ray craniostenosis is absent.
- Maxilla in the form of trapezium, abnormal dentition.
- Late dental evolution, enamel defects in the temporary and permanent dentition.
- Enlargement of costo-chondral junctions -"rickets rosary"
- Thorax, sternum deformation, softened lower rib cage at the site of attachment of the diaphragm- Harrison groove.
- Spinal column- scoliosis, lordosis, kyphosis.
- Pelvis deformity, entrance is narrowed (add to cesarean section in females)

- Extremities- palpated wrist expansion from rickets, tibia anterior convexity, bowl knock kness legs.
- Deformities of the spine, pelvis and legs result in reduced stature, rachitic dwarfism
- Delayed psychomotor development (heat holding, sitting, standing due to hypotonia)

Investigations

- Serum calcium level (N=2.2-2.ómmol/l). At the level <2.0mmol/I convulsions sets in.
- Phosphorus normal (1 .5-1 .8mmol/l). Normal ratio of Ca: P= 2:1; in rickets become 3:1; 4:1.
- Serum 25 (OH) D3 (N=28+2.1 ng/ml) ; and 1,25 (OH) 2133 (N=0.035+0.003ng/ ml)
- Serum alkaline phosphatase is elevated >500mmol/l.
- Thyrocalcitonin can be appreciated (N =23.6+3 .3pM/l)
- Serum parathyroid hormone (N=598+5.0pM/l)
- In urine: Aminoaciduria >1 .0mg/kg/day

Urinary excretion of 35' cyclic AMP

Decreased calcium excretion (N =50-1 50mg/24h)

X- Rays

Only in difficult diagnostic cases.

- X-ray of the distal ulna and radius: concave (cupping) ends; normally sharply, Fraying rachitic metaphyses and a widened epiphyseal plate.

  Genu Varum deformity at knees.

- Osteoporosis of clavicle, costal bones, humerus. Rickety Rosary at chest. Pigeon chest.
- Green stick fractures.
- Thinning of the cortex, diaphysis and the cranial bones.

Prophylaxis in Rickets

Specific antenatal prophylactic dose administration : 500-1 000IU/day of vitamin D3 solution at the 28-th week of pregnancy. The total dose administered

is 135000-180000IU. In term infants prophylactic intake of vitamin D2 700IU/d started at 10 days of age during the first 2 years of life; in premature the dose may increase to 1 000IU/day.

WHO recommendation for rickets prophilaxis in a children coming from unfavorable conditions and who have difficult access to hospitals is 200000IU vitamin D2 intramuscular,

On the 7day, 2, 4, 6 month- total dose 800000IU. In case of the necessary prolongation 700IU/day till 24mo are given.

Treatment of Rickets

The treatment is with vitamin D3 depending on the grade.

- In grade I- 2000-4000IU/day for 4-óweeks, totally 120000-180000IU.
- In grade II- 4000-6000IU/day for 4-óweeks, totally 1 80000-230000IU.
- In grade III- 8000-1 2000IU/day for 6-8 weeks, totally 400000-700000IU.
- Along with vitamin D, calcium is also administered (40 mg/kg/day for a term baby,
- 80 mg/kg/dayfora premature baby) ; also indicate vitamin B&C preparations.
- From the 7-th day of the treatment massage can be started. Intramuscular administration of ATP solution in case of myotonia 1 ml/day is preferred.

Specific splints may be required to correct or to control the deformities in early stages. In We stages, when deformities have established, surgery may be required, especially for bow legs.

Patients must be encouraged for sun-bathing

AFFECTION OF BONES AND JOINTS

OSTEOARTHRITIS

Osteoarthritis is a progressive degenerative disorder of advancing age. However nowadays the term 'osteoarthrosis a degenerative joint disease is used because it is not associated with inflammation.

It may be:

1) Primary, which occurs in old age

2) Secondary to some preexisting disease in joint which can occur at any age It can involve many joints in the body, but the most common involved sites are

i. Knee

ii. Spine

iii. Hip

iv. Shoulder

Depending upon the stage of the disease, it may be:

- Mild
- Moderate or
- Severe

Diagnosis is made on the basis of history, examination and x-ray. Very rarely MRI or CT may be needed.

History:

Pain :- To start with it is mild and gradually increases in severity.

Difficulty in squatting, climbing stairs, sitting cross legged, etc

Examination:

Crepitus is most common finding. Varying

degree of deformity may be seen. Painful

restriction of movements..

X- ray will show decreased joint space with marginal osteophytes.

Treatment:

It varies with region involved, but there are certain common modes of non- surgical treatment which are applicable to every part of the body.

NON SURGICAL TREATMENT

1. Muscle strengthening exercises: This is applicable to every part of body because strong muscles reduce the stress on the joint.

2. Lifestyle modification. Patient should be advised to modify routine habits such squatting etc.

3. Education to the patient regarding decrease in weight.

4. Physical means in the form of

    i. Braces/orthosis / ambulatory aids

    ii. Heat and cold therapy

    iii. LASER therapy. This helps in improving the morning stiffness and it acts at both cellular and systemic level activating variety of mechanisms including cartilage regeneration

5. Pharmacological treatment in the form of NSAIDS:

    Any NSAID can be used such as diclofenac 50 mg tds, brufen 400 mg tds, etc.

    But for prolonged use paracetamol 1 5mg/kg tds and Etoricoxib 60- 120 mg in single or divided doses can be used.

6. Steroids (5-15 mg/kg) have been shown to help in relieving the symptoms but they are not recommended because of increase in the degenerative process

7. Diacerin : A new anti-inflammatory analgesic has modifying effect and is used very commonly. 50 mg OD to start then BD after 1 month and can be used minimum for 3 months or longer time depending upon the requirement.

8. Narcotic analgesics are usually reserved for patients with severe joint disease and intolerable suffering who are not candidate for other therapeutic interventions or in those where other therapeutic interventions have failed

9. Nutritional supplements such as glucosamine (1 gm daily) and chondroitin sulfate (750 mg daily) which are constituents of articular cartilage are also helpful.

10. Collagen peptides: They are also used because they increase the production of aggrecan, a special proteoglycan which is of central importance to cartilage function. Available in sachet 10.2 gm to be given once a day.

11. Rose hip extract: This extract is developed from blossoms of white rose and is a popular natural remedy. This has also been found to be useful in reducing the suffering of osteoarthrosis. -

12. Antidepressants because of continuous disabling pain, these patients usually go into depression so antidepressants help in decreasing pain but every patient should be evaluated before starting these drugs.

13. Viscosupplements : Hyaluronic acid given by intraarticular injection appear to provide some relief.

SURGICAL TREATMENT

Multiple surgical options are available and the patient should be referred to higher centre.

SUMMARY

Among all these option listed above, the following are most commonly used:

1. Muscle strengthening exercises.

2. Change in occupation and lifestyle

3. Analgesics.

4. Diacerin and nutritional substitutes such as glucosamine and chondroitin sulfate.

5. Surgery

SPINE

In addition to the above treatment sometimes these patients needs decompression when there is evidence of compressive myelopathy.

HIP

Routine treatment is same as described above, but refractory patients need arthroplasty.

SHOULDER

- In addition to the common schedule, intraarticular steroid injections can be used to relieve the symptoms.
- Local intraarticular injections : Hydrocortisone acetate 25 mg every week (3-5 injections)
- Methylprednisolone 80 mg biweekly (3-5 injections)
- Triamcinolone acetate 40 mg biweekly (3-5 injections)
- Patient not responding needs arthroplasty.

RHEUMATOID ARTHRITIS

- RA is a chronic multisystem disease of unknown etiology characterised by its pattern of diathrodial joint involvement.
- Its primary site of pathology is synovium in joints.
- It usually involves peripheral joints in symmetric distribution
- Joint changes probably represent autoimmune reaction

Pattern of onset

- Insidious onset:- 60-70% cases, over weeks to months with symmetrical joint involvement
- Acute onset :- 8-15 % cases. Less symmetrical, extremely painful joints, diffuse pain in surrounding tissues.
- Intermediate onset:- 15-20% cases develop symptoms over days to weeks.

Investigations: Inflammtory

Markers

- ESR: Is raised during acute phase and levels fall slowly-after 1 week fall is about 50%.

- CRP: Acute phase reactant. Level falls down quickly. It distinguishes between inflammtory and non inflammtory arthritis.

Antibodies

RA Factor: positive in 70% at the onset.

- It is a lgM subclass.
- With higher RA Factor titre patients develop more severe and eroding arthritis than RA Factor -ye patients

Anti CCP Antibody: It has high sensitivity and specificity.

- It is a predictor of erosive disease and joint damage
- New markers of diagnosis of RA is MMP-3
- Matrix metaloprotinase (MMP-3) play an important role in remodelling of extracellular matrix.
- MMP-3 is produced by articular synovial cells, fibroblasts and chondroblasts.
- Stromelysins (MMP-30 PRODUCTION IS INCREASED IN RA)

Hematological Parameters: Normocytic normochromic anemia

Plain radiography; describes bone erosions, joint space, bone alignment, soft tissue swelling, etc

Before proceeding towards treatment of RA, following points should be kept in mind

- Nothing is 100%
- Good history and physical examination along with knowledge of musculoskeletal system is important when evaluating a patient of RA
- Do not order lab tests unless you know why to order and what to do if it comes back abnormal.
- Patients with chronic inflammatory monoarticular arthritis of >8 weeks duration whose evaluation has failed to define an etiology needs synovial biopsy
- All patients with +ve RA Factor do not have RA.
- If associated with high grade fever, rule out infection.

Criteria

In 2010 the 2010 ACR / EULAR Rheumatoid Arthritis Classification Criteria were introduced. These new classification criteria overruled the "old" ACR criteria of 1987 and are adapted for early RA diagnosis. The "new" classification criteria, jointly published by the American College of Rheumatology (ACR) and the European League Against Rheumatism (EULAR) establish a point value between 0 and 10. Every patient with a point total of 6 or higher is unequivocally-classified as an RA patient, provided he has synovitis in at least one joint and is no other diagnosis better explaining the synovitis.

Four areas are covered in the diagnosis

- Joint involvement, designating the metacarpophalangeal joints, proximal interphalangeal joints, the interphalangeal joint of the thumb, second through third metatarsophalangeal joint and wrist as small joints, and elbows, hip joints and knees as large joints:
- Involvement of 1 large joint gives 0 points
- Involvement of 2-1 0 large joints gives 1 point
- Involvement of 1-3 small joints (with or without involvement of large joints) gives 2 points
- Involvement of 4-10 small joints (with or without involvement of large joints) gives 3 points
- Involvement of more than 10 joints (with involvement of at least 1 small joint) gives 5 points

Serological parameters -including the rheumatoid factor as well as ACPA - 'ACPA" stands for 'anti-citrullinated protein antibody":

- Negative RF and negative ACPA gives 0 points
- Low-positive RF or low-positive ACPA gives 2 points
- High-positive RF or high-positive ACPA gives 3 points

Acute phase reactants: 1 point for elevated erythrocyte sedimentation rate, ESR, or elevated RP value (c - reactive protein)

Duration of arthritis: 1 point for symptoms lasting six weeks or longer

Treatment:

Aims

- Relief of inflammation and pain
- Correction and control of systemic manifestations
- Prevention of deformity
- Correction of existing deformity
- Improvement of functional capacity

Supportive measures

- Patient education
- Behavioural modifications
- Rest
- Splints- orthotic devices
- Physical therapy

Pharmacological treatment

- Analgesics
- NSAIDs: Naproxen and etoricoxib can be used for a longer period
- Naproxen500mgBID
- Piroxicam20mgQlD
- Aceclofenac 100 mg BD
- Diclofenac sodium 50 mg TDS
- Etoricoxib 60-120 mg OD or BD
- Glucocorticoids: -are given for short time to-suppress the inflammatory process.
- DMARD's

DMARD - disease modifying antirheumatic drugs

D-penicillamine

125-250 mg OD then increase to 250 mg BD.

Hydroxychloroquine

6.5 mg/kg/day or 2 00 - 400 mg per day.

Leflunomide

Start with 100 mg /day as a loading dose for 3 days then 20mg per day

Methotrexate (Mtx)

15-25 mg weekly along with folic acid 1 mg/day to reduce the toxicifies.

Sulfasalazine (SSZ)

40mg/kg/day

In addition, less used DMARDS are Azafhioprine, Cyclosporine and gold salts.

These drugs can be used for a long time depending upon the response. However, time tested and reliable drug is methotrexate which should be given to every patient. For side effects CBC, creatinine, LFTs should be done every 2-3 months.

First line treatment

1. Any NSAIDs to subside the symptoms.
2. Methotrexate should be started along with to modify the disease process. In addition, if the symptoms are severe, any other DMARD can be added to the therapy because combination therapy has shown excellent safety and efficacy over methotrexate alone.
3. Steroid are not a preferred drug but in full flare up of the joint it should be used for a short period to suppress the inflammation and to avoid joint damage.

Combination DMARD therapy MTX +

SSZ + OH-Chloroquine MTX +

Leflunomide

Biologic Therapies, or Biologics

These are newer drugs that reduce inflammation in a more highly targeted manner than DMARDs. These are used when there is inadequate response with the DMARDS. These new drugs are very expensive and not easily available and full efficacy is still to be evaluated.

Surgical Treatment

Reserved for patientswith severely damaged joints It

includes

- Arth roplasty/ Total joint replacement
- Open/Arthroscopic synovectomy
- Reconstructive hand surgery

CERVICAL AND LUMBAR SPONDYLOSIS

Cervical Spondylosis

Cervical spondylosis is a chronic degenerative condition of the cervical spine that affects the vertebral bodies and intervertebral discs of the neck (in the form of, for example, disk herniation and spur formation), as well as the contents of the spinal canal (nerve roots and/or spinal cord).

Spondylotic changes can result in stenosis of the spinal canal, lateral recess, and foramina. Spinal canal stenosis can lead to myelopathy, whereas the latter 2 can cause radiculopathy.

Incidence

It is the most frequent cause of spinal cord dysfunction in patients older than 55 years. On the basis of radiologic findings, 90% of men older than 50 years and 90% of women older than 60 years have evidence of degenerative changes in the cervical spine.

Radiographically, the most frequently involved level is C5-C6 followed by C6-C7, and C4-05. Upper-level (occiput-C3) involvement is less common.

Clinical features-

Cervical pain

- Chronic suboccipital headache maybe present.
- Pain can be perceived locally, or if may radiate to the occiput, shoulder, scapula, or arm.
- Nerve roots may be directly compressed. Osteophytes, which develop as a reaction to the process of degenerative disc disease extending across the posterior and posterolateral aspect of the vertebral bodies, may cause direct compression. An inflammatory component of the neuroelements may be a more significant cause of pain than actual mechanical changes

Cervical radiculopathy

- Radiculopathy—root compression leads to ischemic changes. It is a lower motor neuron problem and is manifested by pain in the distribution of a nerve root. It can be associated with neck pain, sensory deficit, and motor

deficiency.

- The associated reflex may be diminished.
- The pain of cervical radiculopathy may be described as dull, aching, boring, to neck motion. It may or may not be related to sneezing or cough.

Cervical myelopathy

- Cervical spondylotic myelopathy is the most serious consequence of intervertebral disc degeneration.
- Myelopathy involves compression of the spinal cord, and thus, it can effect the upper and lower extremities with a mixture of upper and lower motor neuron lesions.
- Patients with myelopathy do not necessarily complain of pain.
- The hallmark is extremity dysfunction such as hand clumsiness with fine motor tasks and gait instability. Cervical myelopathy has an insidious onset, which typically becomes apparent in persons aged 50-60 years. Complete reversal is rare once myelopathy occurs.
- Anterior compression of the spinal cord results from posterior osteophytes. Posterior compression may result by infolding of the ligomenfum flavum particularly in extension Nutritional and vascular involvement with decreased blood supply through the spinal arteries resulting in ischemic changes to the spinal cord has been identified.
- Arthrosis of the facet joints in the spondylofic cervical spine may be a source of a dull, aching axial pain or radiating pain secondary to direct nerve root compression.

Physical findings

Findings at physical examination may include the following:

- Spurting sign - Radicular pain is exacerbated by extension and lateral bending of the neck toward the side of the lesion, causing additional for aminal compromise.
- Lhermitte sign - This generalized electrical shock sensation is associated with neck extension.
- Hoffman sign - Reflex contraction of the thumb and index finger occurs in response to nipping of the middle finger. This sign is evidence of an upper motor neuron lesion. A Hoffman sign may be insignificant if present bilaterally.
- Distal weakness
- Decreased ROM in the cervical spine, especially with neck extension

- Hand clumsiness
- Loss of sensation
- Increased reflexes in the lower extremities and in the upper extremities below the level of the lesion
- A characteristically broad-based, stooped, and spastic gait
- Extensor planter reflex in severe myelopathy

Differential Diagnoses

Shoulder soft tissue and articular pain syndromes

- Primary spinal cord tumors
- Syringomyelia
- Extramedullary lesions (tumors, thoracic disc herniation)
- Hereditary spastic paraplegia
- Normal pressure hydrocephalus
- Spinal cord infarction
- Spinal sepsis
- Whiplash syndrome (hyperextension- hyperilexion injury)
- Pancoast tumors
- Brachial plexopathy
- Thoracic Outlet
- Vascular Malformations

Work Up:

X Rays

Plain radiographsof the spine provide a clue to the level or levels of spine disease that may be responsible for the radicular syndrome in cervical spondylosis.

Studies can include AP, bilateral obliques, lateral, odontoid open mouth, and lateral flexion and extension views.

Look for evidence of foraminal encroachment, vertebral malalignment, sclerosis, facet joint subluxation, osteophyte protrusions, destructive changes within the disc or vertebral body, and ossification of the posterior longitudinal ligament.

The spondylotic spine may be hypermobile, resulting in instability. This can usually be identified on lateral flexion and extension radiographs. Anterior-

posterior movement of one vertebral body on another of 3.5 mm or greater in the adult is considered abnormal.

Computed tomography (CT) scanning

High-quality CT scans are extremely useful in assessing the size of the neuroforamina, which are normally 5 to 8 mm in vertical diameter.

A non contrast CT scan is also very useful in delineating bone from soft tissue in planning a decompressive procedure.

Myelography

Adds anatomic information in evaluating spondylosis and is useful in visualizing the nerve root takeoff.

Water-soluble contrast myelography in combination with CT scanning remains the securest way of defining root sleeve pathology. Myelography with flexion and extension views can demonstrate dynamic cord compression related to bulging of the posterior longitudinal ligament and lugamentumflavum, orto spinal instability.

MRI

MRI scanning has become the gold standard in evaluating the cervical spine. MRI is far superior in defining soft tissue anatomy. The disc material and nerve anatomy can be seen as well as demonstrating pathophysiolocal effects such as "gliosis" associated with chronic spinal cord compression. Infections, hematomas, and tumors are also much better visualized by MRI.

Direct imaging in multiple planes & Better definition of neural elements.

NCV & EMG (Nerve Conduction Velocity & Electromyography)

Electrodiagnostic studies may be useful in establishing the diagnosis particular by documenting the distribution of involvement, and distinguishing peripheral syndromes and generalized peripheral neuropathy, from radiculopathy.

Local Injection

In older patients with multiple levels of abnormality shown on radiologic and other imaging studies in whom cervical radiculopathy cannot be localized, injection of local anesthetic into the interspace under fluoroscopic control and

injections of local anesthetic into the facet joints maybe useful in localizing the pain syndrome.

Treatment:

Physical Therapy (Approximately 80% of radiculopathy patients can be successfull nonoperatively).

- Immobilization of the cervical spine is the mainstay of conservative treatment for patients with cervical spondylosis. Immobilization limits the motion of the neck reducing nerve irritation.
- Soft cervical collars are recommended for daytime use only, but they are unable to appreciably limit the motion of the cervical spine.
- More rigid orthoses (e.g. Philadelphia collar, Minerva body jacket) can significantly immobilize the cervical spine.
- A program of isometric cervical exercises may help to limit the loss of muscle tone that results from the use of more restrictive orthoses. Molded cervical pillows can better align the spine during sleep and provide symptomatic relief for some patients.
- Mechanical traction is a widely used technique. This form of treatment may be useful because it promotes immobilization of the cervical region and widens the foraminal openings. Gentle traction using 5 to 10 lb with a head halter and a neutral position of flexion-extension to open up the neuro foramina may be of value. Traction applied in either flexion or extension may aggravate the patient's pain problem.
- The use of cervical exercises has been advocated in patients with cervical spondylosis. Isometric exercises are often beneficial to maintain the strength of the neck muscles. Neck and upper back stretching exercises, as well as light aerobic activities, also are recommended. The exercise programs are best initiated and monitored by a physical therapist.
- Passive modalities generally involve the application of heat to the tissues in the cervical region, either by means of superficial devices (e.g. moist-heat packs) or mechanisms for deep-heat transfer (eg, ultrasound, diathermy).
- Manual therapy, such as massage, mobilization, and manipulation, may provide further relief for patients with cervical spondylosis.

Occupational Therapy

Disability can be improved with specific strengthening exercises of the upper extremities, special splinting to compensate for weakness, and the use of assistive devices that allow the patient to perform previously impossible activities.

Cervical spondylosis may result in complications including the following:

- Cervical myelopathy
- Paraplegia
- Tetraplegia
- Recurrent chest infection
- Pressure sores
- Recurrent urinary tract infection

Surgical Intervention

- Surgical treatment for rodiculopathy is usually indicated if there has been a documented failure of appropriate nonoperative treatment or if there is progressive neurologic deficit with a radiculopathy & Intractable pain. Options are Anterior or posterior decompression with or without fusion.
- Cervival myelopathy- Patients presenting with cervical myelopathy seldom improve with nonoperative management and roughly one-third will continue to deteriorate, sometimes suddenly with hyperextension. The intent of surgical decompression in myelopathy patients is to prevent progression with neurological improvement being secondary and unpredictable. Options are

    - Anterior decompression via corpectomy, discectomy, and fusion
    - Posterior complete laminectomy and decompression.
    - In the case of multilevel disease (3 or more levels), open door hinged laminoplasty, to expand the spinal canal, has been gaining favor. Open-door laminoplasty for multiple-level decompression seems to prevent postoperative swan-neck-type deformities, which sometimes occur after extensive multilevel posterior laminectomies.

Injections

Cervical epidural steroid injections can prove useful in treatment of radiculopathy especially if an inflammatory component is present.

Epidural and selective nerve root blocks can be diagnostically and therapeutically helpful in cases of radiculopathy. Trigger-point injections maybe helpful.

Cervical, zygapophyseal, intra-articular steroid injection can be helpful for active synovitis.

Mechanical facet pain is better evaluated with facet joint nerve blocks.

Medication

- NSAIDS- Add to Patient's comfort.
- Corticosteroids

Can be administered as-

- Pulse corticosteroid therapy with methylprednisolone or Dexamethasone - Intravenous pulse steroid therapy consists of administration of supraphysiological doses of glucocorticoids. It is useful in conditions where rapid antiinflammatory effect is deskW with less long-term systemic side effects associated.

  Prednisone (20-30mg/kg) or Dexamethasone (4-5mg/kg) per pulse usually repeated interval of 24-48hrs usuallyforó pulses.

- Long term Therapy- in form of tapering regimen started with Moderately high dosesood Tapered graduallylo prevent Adrenal suppression.

- Muscle relaxants

Lumbar spondylosis

Lumbar spondylosis, describes bony overgrowths (osteophyfes), predominantly those at the anterior, lateral and less commonly, posterior aspects of the superior and inferior margins of vertebral centra (bodies). This dynamic process increases with, and is perhaps an inevitable concomitant, of age.

Lumbar spondylosis usually produces no symptoms. When back or sciatic pains are symptoms, lumbar spondylosis is usually an unrelated finding.

Lumbar spondylosis appears to be a nonspecific aging phenomenon.

Pathophysiology

Lumbar spondylosis occurs as a result-of new bone formation in areas where the annular ligament is stressed.

Presentation

Lumbar spondylosis usually produces no symptoms (Due to wider spinal canal in lumbar region).

When back or sciatic pains are symptoms, lumbar spondylosis is usually an unrelared finding.

Lumbar spondylosis is usually not found unless a complication ensues.

Other problems to consider include the following:

- Spondyloarthropathy
- Spinal stenosis
- Diffuse idiopathic skeletal hyperostosis
- Fibromyalgia
- Postural disturbance
- Aortic aneurysm
- Ischial bursitis
- Trochanteric bursitis
- Hip arthritis
- Spondylolisthesis
- Osteoporosis
- Compression fracture
- Neoplasia
- Hemangioma
- Infectious spondylitis
- Endocarditis
- Disc disease

Imaging Studies

Radiographs, CT scans, and MRIs are used only in the event of complications.

Bone density scan (e.g. dual-energy x-ray absorptiometry scan [DEXA]) are used. Ensure that no osteophytes are in the area used for density assessment for spinal studies. Osteophytes produce the impression of increased bone mass, thus invalidating bone density tests if in the field of interest and masking osteoporosis..

Electromyography (EMG) and nerve conduction velocity (NCV) are used only in the event of complications.

Medical Therapy

- **Because back pain is an unrelated finding of lumbar spondylosis, seek the real cause of the patient's back or sciatica- type symptoms.**
- Do not assume that the patient's symptoms are related to osteophytosis.
- Look for an actual cause of a patient's symptoms.
- If actual symptomatic nerve root impingement occurs, 2 days of absolute bed rest is indicated.
- If that does not solve the problem, then surgical excision is indicated. Medication is not indicated in the absence of complications.
- Surgical excision is performed for impingement-documented sciatica that is unresponsive to 2 days of absolute bed rest.

Complications

- Nerve compression from posterior osteophytes is a possible complication only if a neuroforamen is reduced to less than 30% of normal.
- Posterior disc height reduction to less than 4 mm or foraminal height to less than 15 mm is compatible with diagnosis of osteophyte-induced nerve compression.
- If lumbar spondylosis projects into the spinal canal, spinal stenosis is a possible complication.

SPRAIN

A sprain is an acute traumatic injury to a ligament which is being stretched beyond its own capacity. A strain is an acute traumatic injury to the muscle-tendon junction. Ligaments are tough, fibrous tissues that connect bones to other bones. Sprains can occur in any joint most common in the ankle and wrist.

Symptoms

- Pain
- Swelling
- Bruising
- Decreased ability to move the limb
- If the ligament is ruptured, one may hear a popping sound
- Difficulty using the affected extremity

Classification

- First degree sprain -mild pain due tearing of less than one-third ligamentous fibers, with < 5mm joint laxity
- Second degree sprain - moderate pain and swelling, one-third to two-third fibers of ligament are torn, with 5-10 mm joint laxity
- Third degree sprain - severe pain arising from complete tear of the ligament causing joint instability

Diagnosis

- The diagnosis of a sprain injury is made by a physical examination.
- In most cases an x-ray (or stress x-ray) of the affected joint is obtained to rule associated fractures and joint laxity.
- If a tear in the ligament is suspected, then an MRI or arthroscopy is obtained.
- MRI is usually ordered after swelling has subsided and can readily identify the presence of a ligament injury.

Causes

Sprains typically occur when the joint is overextended. This can cause over stretching of the joints, tear or slipping of the ligament.

Joints involved

Although any joint can experience a sprain, some of the more common include:

- The ankle. It is the most common, and has been said that sprains such as serious ankle sprains are more painful and take longer to heal than actually breaking the bones in that area.
- The knee. Perhaps one of the more talked about sprains is that to the anterior cruciate ligament (ACL) of the knee. This is a disabling sprain common to athletes, playing basketball, pole vaulting, softball, baseball and some styles of martial arts.
- The fingers.
- The wrist.
- The toes.

Risk factors

There are certain factors which increases risk of sprains. Fatigue of muscles generally leads to sprains. When one suddenly starts to exercise after a sedentary lifestyle, sprains are quite common. While scientific studies are lacking, it is often thought that not warming-up is a common cause of sprains in athletes. Warming- up is thought to loosen the joint, increases blood flow and makes the joint more flexible.

Treatment

The treatment of sprains depends on the extent of injury and the joint involved. Medications like non-steroidal anti-inflammatory drugs can relieve pain. Weight bearing should be gradual and advanced as tolerated. The first modality for a sprain can be remembered using the acronym RICE.

- Rest: The sprain should be rested. No additional force should be applied onsite of the sprain. In case of, for example, a sprained ankle, walking should be kept to a minimum. Preferably a POP splint should be applied.
- Ice: Ice should be applied immediatelyto the sprain to reduce swelling and pain. It can be applied for 10-15 minutes at a time (longer application of ice

may cause damage instead of healing), 3-4 times a day. Ice can be combined with a wrapping to minimize swelling and provide support.

- Compression: Dressings, bandages, or ace-wraps should be used to immobilize the sprain and provide support. When wrapping the injury, more pressure should be applied at the far end of the injury and decrease in the direction of the heart; the reason for this is that it more easily causes unnecessary fluid to be flushed back up the blood stream in order to be recycled. Compression should not cut off the circulation of the limb.
- Elevation: Keeping the sprained joint elevated (in relation to the rest of the body) will also help minimize swelling.

The joint should be exercised again fairly soon, in milder cases from 1 to 3 days after injury. Special exercises are sometimes needed in order to regain strength and help reduce the risk of ongoing problems. The joint may need to be supported by taping or bracing, helping protect it from re-injury.

- NSAIDs : Will decrease swelling and edema and increase patient comfort.
- Diclofenac Sodium 50 mg tds in acute phase then 50 mg SOS.
- Aceclofenac, Ibuprofen, Coxibs etc can also be given.

Surgical treatment is generally required in cases with grade 3 tears.

Functional rehabilitation

Prolonged immobilization delays the healing of a sprain, as it usually leads to muscle atrophy and stiff joint. The components of an effective rehabilitation for all sprain injuries include increasing range of motion and progressive muscle strengthening exercise. These should be taken care of without delay.

ANKYLOSING SPONDYLITIS

Introduction

A form of spondyloarthritis, is a chronic, inflammatory arthritis and autoimmune It mainly affects joints in the spine and the sacroiliac joint in the pelvis, and can cause eventual fusion of the spine.

Definition

The typical patient is a young male, aged 20-40, however the condition also presents in females. The condition is known to be hereditary. Symptoms of the disease first appear, on average, at age 23 years. These first symptoms are typically chronic pain and stiffness in the middle part of the spine or sometimes the entire spine, often with pain referred to one other buttock or the back of thigh from the sacroiliac joint.

Incidence of condition in our country

Three men are diagnosed with AS for every one woman; the overall prevalence is 0.25%. Many rheumatologists believe the number of women with AS is underdiagnosed, as most women tend to experience milder symptoms.

Differential diagnosis

Differential diagnosis of Ankylosing Spondylitis include

- Rheumatoid Arthritis
- Other Spondylo arhtropathies

Prevention and counselling

As no direct cause for the disease has been identified the preventive measures could not be established.

Patient needs to be counselled regarding the chronic nature of the disease and need for regular treatment, possible complications and possible treatment options and chances of improvement.

SITUATION 1

- Clinical diagnosis: chronic pain and stiffness in the middle part of the spine or sometimes the entire spine, often with pain referred to one or other buttock or the back of thigh from the sacroiliac joint. Post inactivity stiffness and morning stiffness. In 40% of cases, ankylosing spondylitis is associated with an inflammation of the eye (iritis and uveitis), causing redness, eye pain, vision loss, floaters and photophobia. This is thought to be due to the association these two conditions have with inheritance of HLA-B27. Other common symptoms are generalized fatigue and sometimes nausea.
- Investigations:
    - XRay

- CT Scan
- MRI
- Complete Blood Picture
- ESR
- CRP
- Liver function test
- Renal function test
- HLA B2 7

- In a patient complaining of back pain of more than 12 weeks duration:
  - Morning stiffness of> 30 minutes
  - Improvement in back pain with exercise but not with rest.
  - Awakening because of back pain during the second half of the night only.
  - Alternating buttock pain
  - Peripheral assymetrical large joint involvement
  - Plain X Ray showing features of sacroilitis.
  - Absence of RA factor.

Any 2 out of first four criteria strongly indicate presence of Ankylosing Spondylitis even in the absence of xray and lab investigations.

Treatment:

Standard Operating Procedure

- In Patient:
  - Surgery (Joint Replacement for hip and knee)
- Out Patient:

- NSAIDS: First line therapy to relieve symptoms.

DMARDs

Such as cyclosporin, methotrexate, sulfasalazine, and corticosteroids, used to reduce the immune system response through immuriosuppression DMARDs are useful only for peripheral arthritis & not for axial skeleton involvement. -

"http://en.wikipedia.org/wiki/Tumor_necrosis_factor_alpha"

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Blockers (antagonists) such as etanercept, infliximab, golimumab and adalimumab (also known as biologics), are indicated for the treatment of and are effective immunosuppressants as in other autoimmune diseases.

- Physical Therapy - Patients to be encouraged to undertake active and passive range of motion exercises for all joints to maintain and prevent the progression of loss of mobility. Deep breathing exercises (Pranayaam) should be promoted to improve chest function.

- Day Care: Injectable medications

OSTEOARTICULAR TUBERCULOSIS

Introduction

For purposes of description osteoarticular tuberculosis can be discussed under the following heads:

- Tuberculosis of joints
- Bone tuberculosis
- Spine tuberculosis

Infection of a joint or bone with Mycobacterium tuberculosis is almost always secondary to a primary focus, in the lymphatic glands or lungs or mesentery, from where it disseminates by hematogenous route. Malnutrition or any debilitating disease, poor environment increase the incidence of the disease. Patients with immunodeficiency disease or HIV infection are more prone to develop tuberculosis. Primary bone tuberculosis is not uncommon.

Involvement of any bone or joint in the body can be affected by tuberculosis. Case definition the lesion in the joint can be:

- Extra-articular
- lntra-articular: It can originate in the bone (osseous lesion) or in the synovium (synovial disease).

Vertebral body involvement with tuberculosis is the most common and is nearly equal to tuberculosis of all other regions put together.

There may be a history of trauma, under the effect of which a small hematoma may form resulting in vascular stasis in that area. The hematoma may become a nidus for the tubercle bacilli to settle down and form a tuberculosis follicle with caseation, epitheloid cells, giant cells and fibrosis at the periphery.

The response to a tuberculous lesion is exudative and may form a cold abscess, which is nothing but a collection of necrotic material, caseous tissue and the exudative reaction. These cold abscesses than track through the fascial planes or the neurovascular bundles and may present at a distant site. Since the abscess is away from the area of inflammatory activity, it has no signs of inflammation in the skin overlying the abscess. A superficial abscess may burst and result into a sinus or an ulcer.

Granulation tissue is almost always present in the tuberculous lesion. lschemic necrosis of bone due to endarteritis and thromboembolic phenomenon in bone lead to formation of sequestra, which in osseous tuberculosis happen to be small. Isolated large sequestrate in osteroarticular tuberculosis are rare.

Differential Diagnosis

It can mimic almost any condition seen in bone like chronic osteomyelitis, osteoid osteoma, fibrous dysphasia, malignant/benign tremors.

Prevention and Counseling

In case of pain, swelling, night cries fever an orthopedics surgeon may be consulted.

Clinical Diagnosis

The tuberculosis of the joints mainly involves big joints. The common differential diagnosis includes pauciarticular juvenile chronic arthritis and septic arthritis. The involvement of joints may be osseous or synovial but if not treated,

one would infect the other. Tuberculous synovitis leads to effusion in the joint and synovial membrane becomes edematous. At this stage the joint would look swollen and movements may be present or limited due to muscle spasm. The radiological picture may show an increased joint space.

Clinical features

It is characteristically insidious in onset, and starts as monoarticular or mono-osseous involvement. The child complains of pain in the joint, aggravated by movement, and often wakes 'up at night because muscle spasm gets reduced and causes pain. It is classically called as "night cries". Low- grade fever, loss of weight and appetite are some of the symptoms of generalized toxemia usually seen. Joint movements are painful and elicit muscle spasm on attempted movement. In later stages when the cartilage gets eroded, all movements get restricted. Muscle atrophy around the joint is a predominant feature and occurs early. Sometimes an abscess forms, which bursts to form a sinus. It may get secondarily infected and may alter the radiological picture.

Investigations

- Blood- A low hemoglobin, relative lymphocyfosis and raised erythrocyte sedimentation rate (ESR) are often found in the active stage of the disease. The ESR is often used as aguide in monitoring the progress of the disease during treatment, though some people do not consider it a reliable investigation.
- Mantoux Test- A positive Mantoux test is seen in patients with active tuberculous lesion. A negative test may rarely be seen in severe or disseminated disease or in an immunocompromised patient.
- Radiographic Examination- It can be diagnostic in view of the typical radiological appearance of the tuberculous lesions. In early stage of the joint disease, capsular markings may become prominent. The earliest sign is widespread osteoporosis around a joint. Lytic lesions and periosteal reaction are seen, although latter is much more prominent in pyogenic infection.

  In case of joints, small bone erosions occur near the capsular reflection. Joint space decreases due to cartilage erosion and lytic lesions are seen in the epiphyseal area. The radiological signs of a healing lesion are absence of rarefaction and bony ankylosis.
- Smear and Culture- Tuberculous pus, joint aspirate, granulation tissue, sputum etc. may be examined by smear and culture for tubercu bus bacilli.
- FNAC (Fine Needle Aspiration Cytology)- Occasionally, even the most

modern methods of imaging may not help the clinician to reach to a final diagnosis, and therefore FNAC or biopsy may be undertaken to obtain tissue diagnosis. FNAC is now available for the cytological diagnosis of vertebral tuberculosis. 'Biopsy is a safe and a quick diagnostic procedure with high accuracy in the hands of trained cyfopathologists. It is recommended that it should be practiced in all diagnostic centres of our country, even for suspected vertebral tuberculosis.

Biopsy

Biopsy may have to be done in cases where there is doubt about the diagnosis, particularly in the early stages of the disease. Biopsy from the bone or synovium can provide on early diagnosis for starting the treatment in time and preventing damage to the joint. Biopsy from a cystic lesion in the bone or from synovium is more likely to be positive.

Treatment

The patient's response to treatment is as variable as anywhere else in the body and is dependent upon the host resistance, severity of infection, and the stage of the disease when the diagnosis is first made and treatment started. Eradication of the disease and preservation of function are important both in osseous and joint diseases.

General measures:

Good nutrition consisting of a high-calorie and high-protein diet is essential to be the resistance. General rest and local rest to the specific bone and joint are essential path treatment Local rest can be provided by means of splints or plaster casts However, in where the articular surface is not involved a judicious blend of rest and mobilization has to be resorted to for restoration of function.

Chemotherapy:

Most of osteoarticular lesions would respond to antituberculous drugs if the therapy is started early.

A standard drug regimen is given which includes rifampicin, pyrazinamide, ethanbutol, isoniazid, and in some cases even streptomycin. The latter is useful because it kills the rapidly multiplying extracellular tubercle bacilli in the lungs for the initial six months. After two clinically and radiologically clear intervals, pyrazinamide is stopped and isoniazed, rifampicin and ethambutol are continued

for one year. In some cases therapy may be required for 18 months for complete healing of the lesion. In case the infection is suspected to be with multidrug resistant ofloxacin, capreomycin, kanamycin, etc. may have to be given.

Surgical Treatment

Surgical treatment is an adjunct to the anti-tuberculosis drug therapy. The

surgical procedures generally perfomed in children are:

- Drainage of an abscess
- Excision of a focus
- Curettage of the lesion
- Synovectomy
- Costotransversectomy
- Anterolateral decompression

The general principle of surgery in tuberculosis demands that the abscess should be completely evacuated. In case of an osseous lesion, all sequestra, granulation tissue and caseous material should be removed till new bleeding bone is encountered, so that the antibiotics may reach the site of lesion better. The cavities so produced should be packed with autogenous bone grafts. Avoid dead spaces to prevent hematoma formation and close the wound primarily with or without suction. -

Tuberculosis can involve any bone or joint of the body but in children it has a special predilection for the hip and knee joints commonly, and for ankle and elbow joints rarely. Tuberculosis of spine with or without paraplegia is extremely common. Long bones are rarely involved but the short long bone involvement is somewhat common.

BONE TUMOURS, TUMOUROUS CONDITIONS & DEVELOPMENTAL DISORDERS MIMICKING BONE TUMOURS

IVORY OSTEOMA

Introduction: Benign bone tumour arising from osteoblasts, Bony (ivory) hard in consistency, 1 or 2 cm in size, over a flat bone, usually frontal bone asymptomatic unless the deeper extension is pressing on the brain which is very unusual

Incidence: Not worked out in our country, not very common

Differential Diagnosis: None

Prevention: Nil

Treatment: Asymptomatic lesions left alone, Excision of symptomatic ones or for cosmetic reasons

OSTEOID OSTEOMA

Introduction: Benign bone tumour, vascular and very painful, about 1 cm in size; elicits sclerotic reaction by the parent bone when the lesion is in the cortical bone; In cancellous bone the lesion is limited by a thin rim of sclerotic bone; in the spine it can cause scoliosis; if the lesion is in the metaphysis which is intraarticular can produce symptoms of arthritis; If the lesion is in the evolving stage it may not be seen routine plain radiography.

Incidence: Not uncommon; 10% of benign bone tumours

Differential Diagnosis: usually None in cortical bone, occasionally chronic osteomyelitis; In the cancellous bone - Brodie% abscess, arthritis, unusual form of tuberculosis

Prevention: Nil

Treatment: Excision in toto (enbloc in cortical bone); curettage in cancellous bone; In inaccessible locations - radiofrequency I laser ablation under CT guidance; curetted/excised material sent for histopathology

OSTEOBLASTOMA

Introduction: Benign bone tumour arising from osteoblasts Incidence: Very rare

Differential Diagnosis: Other tumourous conditions like Aneurysmal bone cyst, Fibrous dysplasia

Prevention: Nil

Treatment: Curettage and bone grafting; material sent for histopathology

Resources required:

Situation 1: Doctor, X-ray machine

Situation 2: Orthopedic surgeon, operating facilities, Histopathologist

OSTEOCHONDROMA

Introduction: Benign tumour arising from Chondroblasts of the growth plate; it can be of two types - pedunculated or sessile; the lesions grow as long as the skeleton grows and stop when the growth plates fuse

Incidence: most common benign tumour from bone; 30 to 50 % of benign bone tumours, 10 to 15% of all bone tumours

Differential diagnosis: Chondrosarcoma (secondary), parosteal sarcoma may be mistaken for osteochond ro ma

Prevention: Nil

Treatment: Differentiation between benign and malignant transformation; if benign excision in toto; If malignant -investigate for lung metastasis followed by wide excision/ amputation as the case may be

ENCHONDROMA

Introduction: Benign bone tumour arising from chondroblasts; usually present in the small bones of hands and feet and asymptomatic for long time; patient presents usually with a pathological fracture or sometimes pain.

Incidence: not uncommon; 25 % of all benign tumours, most common primary tumour in the hand

Differential Diagnosis: Aneurysmal bone cyst, Tubercular dactylitis, Giant cell tumour, clear cell chondrosarcoma & acrometastases. The latter two are extremely rare.

Prevention: Nil

Treatment: Curettage and auto/allo cancellous bone grafting, if there is a pathological fracture - needs fixation with plate and screws. (small fragment set)

BENIGN CHONDROBLASTOMA & CHONDROMYXOID FIBROMA

Introduction: Benign cartilaginous tumours; the former is also called "Codman's Tumour'. They may show specks of calcification signifying their cartilaginous origin. The latter can be aggressive; ends of bone are commonly involved especially tibia, proximal humerus and proximal femur

Incidence: rare tumours; less than 1 %of all primary bone tumours Differential Diagnosis: Aneurysmal bone cyst, GCT

Prevention: Nil

Treatment: Curettage and bone grafting (auto or allo)

BENIGN BONE TUMOUR - NON-OSSIFYING FIBROMA

Introduction: Benign bone tumour arising from fibroblasts. Usually seen in the metaphyseal region of immature skeleton and most of the times it is asymptomatic. It may be an incidental finding in a x-ray taken for some other

purpose or bigger lesions may present as pathological fractures. They have a characteristic radiological appearance of serpigenous margins which have pencil lined sclerotic borders.

Incidence: not uncommon

Differential Diagnosis: none

Prevention: nil

Treatment: Big lesions are curetted prophylactically to prevent pathological fractures and bone grafted. Once they present with a pathological fracture - either they are immobilized in plaster cast till the fracture unites and then curetted and bone grafted or the fracture is openly reduced and internallyfixed and at the same time the lesion is curetted and bone grafted.

BENIGN TUMOUROUS CONDITIONS - SIMPLE BONE CYST, ANEURYSMAL BONE CYST & FIBROUS DYDPLASIA

Introduction: These lesions mimic tumours and they look alike. Usually occur in adolescents. Lytic lesions with clear cut zones of transition. ABC may show fluid levels in MRI; Fibrous dysplasia has ground glass matrix They may present with pain and swelling or with pathological fracture.

Incidence: not very common

Differential Diagnosis: GCT, cartilaginous tumours

Prevention: Nil

Investigations: X-ray, CT scan! MRI, needle biopsy

Treatment: Curettage and bone grafting; if the patient has a pathological fracture, either the limb is immobilized in plaster cast till the fracture unites and then curettage and bone grafting carried out or the lesion is curetted and bone bone grafted and at the satime the fracture is internally fixed.

BENIGN TUMOUROUS CONDITION – FIBROMATOSIS

Introduction: A benign very slow growth of in the subcutaneous tissue or intermuscular connective tissue - does not metastasise but recurrence rate after excision is very high.

Incidence: rare

Differential Diagnosis: Other malignant soft tissue tumours like synovial sarcoma or fibrosarcoma.

Prevention: Nil

Investigations: Plain x-ray, CT scan, MRI, CT chest, Trucut needle biopsy

Treatment: Wide excision with a little surrounding soft tissue to prevent recurrence

BENIGN DEVELOPMENTAL DISORDERS MIMICKING BONE TUMOURS

(Multiple Osteochondromafosis, Muliple enchondromafosis, Oilier's disease)

Introduction: Freak outgrowths from the growth plates -multiple osteochondromatosis- which is familial and producing remodeling and growth abnormalities and ten times more potent for malignancy than its solitary counterpart. Freak inclusions of cartilaginous masses from growth plates into the metaphysic producing streaks of lucency is OIlier's disease - usually present in one side of the body and produces marked growth anomalies. Proliferation of cartilage in the medullary substance of small bones of hands and feet producing globular swellings is multiple enchondromatosis. This condition is not familial and is also associated with growth disturbances.

Incidence: The former is quite common whereas the other two are rare; multiple osteochondromatosis is 10 times less common than solitary osteochondroma.

Differential Diagnosis: usually none; One has to be vizilant to look for a malignant transformation in one of the lesions - the more proximal the lesion is to the axial skeleton more are the chances of malignancy.

Prevention: prevent consanguineous marriages

Investigation: Plain x-ray, CT scan, MRI, wide bore needle biopsy; Caution: Histology cannot be relied upon in diagnosing malignancy in cartilaginous lesions; One has to rely on clinical findings like pain and fast growth to diagnose malignancy. A cartilaginous cap of more than 1 cm thickness as seen in MRI is suggestive of malignancy.

Treatment: Lesions with complications should be excised. Lesions very proximal to the axial skeleton should be excised prophylactically

GIANT CELL TUMOUR (OSTEOCLASTOMA)

Introduction: A benign bone tumour arising from undifferentiated connective tissue cells of bone marrow. Occurs in the mature skeleton usually around knee and at wrist. It is an aggressive tumour and the chances of recurrence following curettage are very high. A small percentage might even metastasise to lungs.

Incidence: much more common in India, especially South India (4 to 6 times), than the western world.

Differential Diagnosis: Aneurysmal bone cyst, Benign Fibrous histiocytoma and aggressive chondromyxoid fibroma, Hyperparathyroidism

Prevention: Nil

Investigations: Plain x-ray, CT scan, MRI, wide bore needle biopsy

Treatment: Curettage with high speed burr, curettage with usage of adjuvants like phenol or liquid nitrogen (cryosurgery) have been in usage. However, extended curettage has the least chances of recurrence. Excision in toto is ideal if the bone involved is expendable (like lower ulna, proximal fibula). In instances like lower radius, excision and reconstruction using proximal fibula is practiced. In weight bearing bones like distal femur or proximal tibia, when the bone is totally destroyed, resection arthrodesis (Enneking procedure) is ideal if the patient

belongs lower socioeconomic group. But if the patient belongs to higher strata where the load demands are less, excision and custom mega prosthesis may be practiced. In fungating cases or after repeated recurrences, an amputation may be the last resort.

OSTEOSARCOMA & ITS VARIANTS

Introduction: occurs in second decade of life.

Incidence: most common malignant tumour in the immature skeleton.

Differential Diagnosis: Early lesions are difficult to diagnose unless one has high index of suspicion. Any pain in the metaphyseal region following a minor injury and disproportionate to the injury or if the pain is slowly increasing day by day after minor injury and especially without fever should lead the clinician to suspect this sinister disease. If it occurs after skeletal maturity, Giant cell tumour also must be thought off.

Investigations: Plain x-ray, x-ray chest, Serum alkaline phosphatase, MRI of lesion, CT Chest, needle biopsy

Treatment: In early cases - chemotherapy for 6 weeks followed by limb sparing surgery (excision of tumour in toto and replacement by custom mega prosthesis), after histological examination of the excised tumour for tumour necrosis, the chemotherapy may be suitably altered. Patient followed at frequent intervals for local recurrence and lung metastases. In cases of late presentation where tumour excision is not feasible a course of chemotherapy is followed by amputation of the limb - chemotherapy is continued. Where lung metastases are present amputation of the limb is palliative.

CHONDROSARCOMA

Introduction: Chondrosarcoma is a malignant bone tumor arising from chondroblasts. The lesion may arise de novo (primary chondrosarcoma) or there may be malignant transformation of an existing benign cartilaginous lesion -

osteochondroma / enchondroma (secondary chondrosarcoma). Thus the lesions may be central or peripheral The lesions are frequently calcified (C-shaped or 0- shaped calcifications)

Incidence: not rare; third most common bone malignancy; 25 % of all sarcomas Investigations: CT scan of part involved, CT scan Chest, MRI, Isotope bone scan, Needle Biopsy

Treatment: Chondrosarcomata are resistant to chemo and radiotheropies. Hence excision in toto, short of amputation, is the only alternative. Secondary and peripheral chondrosarcomata may be amenable for excision. Primary or secondary chondrosarcomoto of the limb girdle may be difficult to treat surgically but excision and reconstruction may be tried depending on the situation. Custom made prostheses may be tried for chondrosarcomata of proximal humerus and femur if the lesions are intracompartmental Inoperable tumours need disarticulation

EWING'S SARCOMA

Introduction: A malignant bone tumour where the cell of origin is uncertain; usually occurring in the diaphyses of children. It can also occur in the metaphyseal regions. When the tumour occurs in adults the ilium is more commonly involved. It is ci fast growing tumour with a lot of soft tissue swelling.

Incidence: Rare

Differential Diagnosis: Osteomyelitis is a major competitor for diagnosis not only clinically but also radiological and histological. Other round cell tumours of bone also should be thought of.

Investigations: X-ray, CBP & ESR, MRI of the part, CT scan of chest, wide bore needle aspiration - material must be sent for histopathology and culture of pyogenic organisms as well. The dictum "culture a tumour and biopsy an infection" holds good here.

Treatment: Though earlier on Radiotherapy was best suited for Ewing's sarcoma, present day sheet anchor is Chemotherapy. After an initial course of chemotherapy, wherever feasible, the tumour is resected and reconstruction done (appropriate surgery). For a recurrence of the tumour radiotherapy is preferred

since the recurrence is from cells which are resistant to chemotherapy given earlier. Where resection of the tumour is not possible amputation is performed.

PLASMACYTOMA/MULTIPLE MYELOMA

Introduction: It is a malignant tumour of the marrow elements where plaslymacytes multiply cancerously in an elderly individual. The condition must be suspectd when an elderly individual complains of vague pains all over the body and not responding to usual analgesics

Incidence: not rare; 10% of hematological malignancies, 1 % of all forms of cancer.

Differential Diagnosis: The main contender is osteoporosis with compression fractures; Multiple metastases especially from prostate in males and breast in females should be thought of; Osteomalacia in late adulthood and Fibromyalgia syndrome are also to be differentiated.

Investigations: CBP ESR, S.Calcium, Phosphorus, Alkaline phosphatase, Bone marrow examination (sterna puncture), serum protein electrophoresis.

Treatment: Chemotherapy and cortisones.

METASTATIC BONE TUMOURS

Introduction: Tumours metastatic to bone are usually from Prostate, Female genital organs, Breast, Lungs, Kidney, Thyroid and GIT. Patients usually complain of either localized or generalized pains. Patient may also present with a pathological fracture with no other symptoms. It is important to note that symptoms due to the primary disease are often lacking and only the secondary deposits cause symptoms. It is also interesting to note that quite often one fails to locate the primary lesion inspite of investigations

Incidence: Not uncommon

Differential Diagnosis: Multiple myeloma, osteoporosis, hyperparathyroidism etc

Investigations: CBP, ESR, x-ray chest, CT chest, Serum Ca, P. ALP, PTH, Serum protein electrophoresis, wide bore needle aspiration biopsy of the lyfic lesion detected on x-ray. Other blood investigations to detect cancers of the respective organs (like PSA, Ca 128 etc.) are to be carried out

Treatment: The primary depends on the cause. For a pathological fracture (or an impending fracture), curettage of the lesion, filling with bone cement and internal fixation preferably with an intramedullary nail is palliative and gives comfort for the rest of the life.

CLINICAL AND RADIOLOGICAL FEATURES OF FRACTURES

A fracture may be a complete break in the continuing of a bone or it may be incomplete break or crack.

Classification

- Fracture:- Caused by injury.
- Fatigue fracture:- Also known as stress fracture, because of repeated stress commonly occur in athletes or new military recruits. Commonly bones of lower limb is involved.
- Pathological fracture:- Fracture though a some already weakened by disease often gives awayfrom trivial trauma fracture maybe
  - Simple
  - Compound fracture: when it is a open fracture

Grean-stick fracture are peculiar to children, bones before 10 year are springy and resilient like branches of a tree.

A statement that the patient is unable to stand or walk after an injury or to use injured part must always arouse suspicion of a fracture. Immediate appearance of deformity is clearly diagnostic.

Clinical Examination:

The objective signs of fracture are so well known that only a brief summary is required here followed signs may be present:

- Visible or palpable deformity
- Local swelling
- Visible bruising (ecchymosis)

- Marked tenderness over injury
- Impairment of function

The following are cardinal signs in the fracture Abnormal mobility

- Crepitus when injured part is moved.
- Absence of transmitting movements clinical evidence; must always be confirmed by radiological examination

Additional Clinical Investigations

When fracture is diagnosed surgeon must see:

- Is there a wound communcally with fracture with fracture?
- Is there any impairment of circulation distal to fracture?
- Is there any evidence of nerve injury?
- Is there any evidence of visceral injury?

Skin Wound-The presence of a skin laceration does not necessarily mean that the fracture is an open one. It is very essential to know whether or not it communicate with bone at the site of fracture.

State of Circulation- The part of the limbs distal to the fracture must be examined for evidence of circulating impairment. Examination should be repeated frequently in the first 48 hours after fresh fracture that has been immobilized in plasters severe pain within the plaster or marked swelling of the digits should arouse swelling of the digits should arouse suspicion that all is not well. Observe the following things:

Colour- A pink colour is reassuring a blue, grey or white colour should arouse suspicion but in it say it does not necessarily by signify circulatory impairment.

Warmth- Warmth digits suggest a circulatory flow though it may be sluggish.

Arterial Pulses- The pulses if available for palpation are usually a reliable guide to the state of circulation, but when the limb is encased in plaster they are not readily accessible. If necessary plaster should be trimmed sufficiently to allow access to the pulses. It must be noted that is compartment syndromes (edema within faxial compartment) in leg or forearm builds up to such an extent that the viability of the contained tissue is unpaired. A very important feature is that impending muscle ischaemia causes severe pain when affected muscles are stretched passively.

Capillary Return- When the digital pulp or nail bed is compressed with a finger nail an area of blenching can be seen around the point of pressure. If on release of pressure the blood flows back briskly into the blanched area in a pink flush, the circulation in adequate of return is sluggish or absent obstruction to circulation should be suspected. Such cases should be referred to higher center immediately.

Nerve Conductivity- Ischaemic nerve quickly loses its ability to transmit impulses loss of sensibility in the digits, in absence of injury to nerves suggests ischemia. In ischaemic lesion of nerve all the nerve trunks are affected where as it is unusual for all trunks to be involved in a injury. Motor tests of nerve conductivity are less reliable.

Specific Test - If it should doubt about circulation give pin pick, if blood comes out it mean sluggish circulation is there otherwise send the patient to higher centre for Doppler study, ultrasonography. or anleviography.

State of spinal cord - There may be para or quadre plegia in spinal injury case. Bladder function must also be investigated in suspected injuries of spinal cord orcauda equina.

State of visera- In fracture of pelvis one must examine abdomen for bladder injury or urethral injury.

Radiological exam

Where facilities available suspected case or otherwise must be examine radiologically. It also tell the type of fracture.

Radiographic technique- The standard technique is to take two projections in plates at right angles to one another usually anterio prosterior and lateral views are taken. It must include the adjacent joint. In special situations additional oblique or tangential projection may be required especially in scophoid and head of radio fracture. If you are not satisfy with plain X-ray send the patient to higher centre for CT or MRI scan or ultrasound scanning. CT scan tells better about bones whereas MRI not only tells about bone but also give perfect image of soft tissues.

Clinical test of union

At a certain stage in the treatment of fracture it is necessary to ascertain whether or not sound union has occurred. The decision is made from clinical and radiological evidence.

There are three clinical test:

- Absence of mob.ility between the fragments.
- Absence of tenderness on firm palpation oversite of fracture.
- Absence of pain when angnlation stress is applied at the site of fracture.

Radiological criteria

There are two radiological features that indicate union

- Visible callus bridging the fracture.
- Continuity of bone trabecule across the fracture.

Principles of fracture treatment First

aid

The doctor who chances to be at the scene of an accident should attempts more than to ensure that the airway is clear, to control any external hemorrhage, to cover any wound with a clean dressing, to provide some form of immobilization for a fractured limb, and to make the patient comfortable while awaiting the arrival of the ambulance.

Temporary immobilization for the long bones of the lower limb is conveniently arranged by bandaging the two limbs together so that the sound limb forms a splint for the injured one. In the upper limb, support may be provided by bandaging the arm to the chest or, in the case of the forearm, by improvising a sling.

Treatment of uncomplicated closed fractures

The three fundamental principles of fracture treatment- reduction, immobilization and preservation of function-are well known, and there is still no better way of discussing the treatment of a fracture than under-these three headings.

Methods of reduction: When reduction is decided upon it may be carried out in three ways.

- by closed manipulation
- by mechanical traction with or without manipulation
- by open operation

Manipulative reduction: The technique is simply to grasp the fragments through the soft tissues to disimpact them if necessary them, and then to adjust them as nearly as possible to their correct position. -

Reduction by mechanical traction Indication

for immobilization

There are onlythree reasons for immobilizing a fracture.

1. to prevent displacement orangulation of the fragments
2. to prevent movement that might interfere with union.
3. to relieve pain

Methods of immobilization

When immobilization is deemed necessary there are four methods by which it may be affected:

- by a plaster of Paris cast or other external splint
- by continuous traction
- by external fixation
- by internal fixation
- Immobilisation by plaster, splint or brace

For most fractures the standard method of immobilization is by plaster of Paris cast. Also available are various proprietary substitutes for plaster, which offer the advantages of lighter weight, radiolucency and imperviousness to water, though at much greater cost. Most such products are also more difficult to apply: nevertheless they are being used on an increasing scale. For some fractures a splint made from metal, wood or plastic is more appropriate-for example, the Thomas's splint for fractures of the shaft of the femur, or a plastic collar for certain injuries of the cervical spine.

The plaster bandages are applied in two forms: round and round bandages and longitudinal strips or slabs to reinforce a particular area. Round and round bandages must be applied smoothly without tension, the material being drawn out to its full width at each turn. Slabs are prepared by unrolling a bandages to and fro

upon a table : an average slab consist of about 12 thicknesses. The slabs are placed at point of weakness or stress and are held in place by further turns of plaster bandage.

- Immobilisation by external fixation

By convention, however, the term external fixation is used to imply ridge anchorage of the bone fragments to an external device such as metal bar through the medium of pins inserted into the proximal and distal fragments of a long bone fracture.

Fixation is now by means of rigid bars of a frame-the fixator-to whichthe pins are attached by clamps with multiaxial joints. Two or three pins are inserted into each fragment and the producting ends of the pins surface parallel with the fractured bone.

- Immobilisation by internal fixation for internal fixation we refer the patient to higher centre

Rehabilitation

Active exercise - While a limb is immobilized in a plaster or splint; exercises must be directed mainly to the preservation of muscle function by static contractions. The ability to contract a muscle without moving a joint is soon acquired under proper supervision.

Continuous passive motion - When restrictive splints are no longer required, exercise should be directed to mobilizing the joints and building up the power of the muscles. Finally, when the fracture is soundly united, treatment may be intensified, movements being carried out against gradually increased resistance until normal power is regained. Engineers have designed machines that provide continuous to and fro movements at a joint without any effort on the part of the patient. The range of movement can be varied as required, being increased gradually as the joint becomes more mobile. This techniques of exercising joints passively has many applications.

Complications of fractures

Complications related to the fracture itself	**Complications attributable to sociated injury**
Infection	Injury to major blood vessels
Dalayed union	Injury to nerves

Non-Union	Injury to viscera
Avascular necrosis	Injury to tendons
Mal-Union	Injuries and post traumatic affections of joints
Shortening	Fat embolism

Infection

Wound infection occasionally remains superficial and the bone escapes, but more often the infection extends to the bone, giving rise to osteomyelitis This is a serious complication, because once a bone is infected with pyogenic organism the infection tends to become chronic.

Delayed Union

Union is deemed to be delayed if the fracture is still freely mobile 3 or 4 months after the injury.

Causes - Any of the following eight factors mayfavour non union.

1. Infection of the bone.

2. Inadequate blood supply to one or both fragments.

3. Excessive shearing movement between the fragments.

4. Interposition of soft tissue between the fragments.

5. Loss of apposition between the fragments (including over distraction by traction apparatus)

6. Dissolution of the fracture haematoma by synovial fluid (in fractures within joints).

7. The presence of corroding metal in the immediate vicinity of the fracture.

8. Destruction of bone, as by a tumour (in pathological fractures)

Shortening

Shortening of a bone after fracture may arise from three causes

1. Mal-union, the fragments being united with overlap or with marked angulation.

2. Crushing or actual loss of bone, as in severely comminuted compression fractures or in gunshot wounds when a piece of bone is shot away.

3. In children, interference with the growing epiphysial cartilage (growth plate)

Special features of fractures in children

Injuries involving the growth plate

The most obvious difference between the bones of children and those of adults is the presence in childhood of cartilaginous growth plates at each end of the major long bones but usually at only one end of the 'short' long bones (metacarpals and metatarsals). Here it may be recalled that the greater proportion of the growth of the bone, and later closure of the growth plate, occurs in the humerus at the proximal end, in the radius end, in the radius and ulna at the distal end, in the femur at the distal end, and in the tibia and fibula at the proximal end. In other words, the most growth occurs awayfrom the elbow and towards the knee.

Complications - Injury to brachial artery complicating supracondlylar fracture of the humerus is common especially tight bandage.

FRACTURE OF THE UPPER LIMB

CLAVICLE FRACTURE

Introduction- it is common in infants and young children and one of the common birth fractures. Most common site is middle 1 /3rd.

Etiology- Fall on out-stretched hand or point of shoulder. It may also occur during extraction in breech delivery.

Clinical Features-

- Pain
- Deformity- medial end is displaced upwards due to pull of sternocleidomastoid Lateral end is displaced downwards due to pull of pectoralis major and weight of upper limb.

Investigations- X-ray clavicle with shoulder joint (AP-View).

Treatment- Most of undisplaced fractures unite readily with conservative treatment with figure of eight bandage or clavicular brace along with arm sling pouch. Patients with associated neurovascular injury or in unstable fractures where facilities are not available for ideal internal fixation should be referred to higher centres.

FROZEN SHOULDER (Adhesive capsulitis/periarthritis shoulder)

It is characterized by pain and progressive limitation of some movements of shoulder joint occurring in past middle age.

Types-

- Primary idiopathic type- cause not known
- Secondary type- in patients with Diabetes mellitus, TB, cardiac ischemia

Clinical Features-

- Diffuse pain in shoulder radiating to middle of upper arm

- Tenderness in subacromial region & in anterior joint line
- Marked limitation of abduction & external rotation
- When condition involves whole rotator cuff results in total restriction of all movements of joint.

Investigations- Radiographs are normal

Treatment-

Analgesics & short term diathermy or wax bath followed by physiotherapy Resistant

cases-local infiltration of steroid &manipulation under anaesthesia

PAINFUL ARC SYNDROME

(Subacromial impingement syndrome/ shoulder impingement syndrome/ supraspinatus syndrome/ swimmer shoulder/ thrower shoulder)

Introduction

This is a clinical syndrome which is caused due to irritation and inflammation of tendons of rotator cuff muscles as they push through subacromian space beneath the acromian.

Etiology

- Any cause which leads to narrowing of the sub acromian space can result in painful arc syndrome
- Subacromian spur
- O/A spur on acromioclavicular joint
- variation in shape of acromion
- thickening or calcification of coracoacromial ligament
- thickening of subacromial bursa.

Clinical Features

- Pain

- weakness
- loss of movements
- grinding and popping sensation during shoulder movements
- decreased range of movements.

Investigations

- Xrays
- USG
- Arthrography
- MRI

Management

- Rest
- Physiotherapy
- NSAIDs
- Ice packs
- Local corticosteroids injections and local anaesthesia

If patient does not respond to the conservative treatment then refer to higher centre where all the facilities are available.

RECURRENT SHOULDER DISLOCATION

Introduction

Shoulder dislocation means the head of humerus is completely displaced out of the glenoid cavity. Shoulder joint is one of the most mobile and least stable joints of body. Recurrent dislocation implies having repeated dislocation of the shoulder and the patients having such tendency are known to have shoulder instability. It

may follow after traumatic dislocation, due to generalized laxity of ligaments, due to gradual stretching of joint capsule.

Clinical Features

- Repeated episodes of pain and discomfort on shoulder movements.
- Apprehension test.

Investigations

- Xrays- Hill Sachs lesion.
- MRI/MRarthrography-Bankart lesion, Hill Sachs lesion.
- CT/CT arthrography

Management

Depends on the functional demands of the patients and level of disability.

Conservative management:-

Patient having sedentary life style those who are not involved in active sports/strenous activities may be managed by lust avoiding the activities which are likely to cause dislocation. Reduction by the patients or by the relatives. Refer to higher centre for surgical procedure where all the facilities are available.

FRACTURE OF SHAFT OF THE HUMERUS

Incidence- Fall from a height or a direct impact as in vehicular accidents.

Diagnosis

- Swelling, pain and bruises are common features
- The arm may appear shortened and deformed if the fracture is significantly displaced.
- Inability to extend the wrist (wrist drop) and sensory deficit over the base of thumb on the dorsal aspect indicates an associated injury to radial nerve
- A thorough assessment of peripheral neurovascular status is essential in all humeral shaft fractures

- Associated injuries to the shoulder and elbow joints are not uncommon

Investigations

- X-rays (AP and Lateral) of the entire humerus should be taken to confirm the diagnosis
- CT is rarely indicated

Most humeral shaft fractures (>90%) will heal with non surgical management.

Twenty degrees of anterior angulation, 30 degrees of varus angulation and up to 3 cm of bayonet apposition are acceptable and will not compromise function or appearance.

Management

Conservative —for undisplaced fractures in the form of hanging castor U- slab

The patient must remain upright or semi upright most of the time with the cast in a dependent position for effectiveness

It is frequently exchanged for functional bracing 1 or 2 weeks after injury Union occurs in more than 90% of cases

Patients with associated neurovascular injury or in unstable fractures where facilities are not available for ideal internal fixation should be referred to higher centres.

DISTAL HUMERUS FRACTURE IN ADULTS

Incidence- 3% of all fractures

Diagnosis

- History-Accidental/Fall on hand sustaining injurylo elbow.
- Pain around elbow joint which increases on movement
- Examination-Swelling, deformity, bruising, tenderness, crepitus, instability
- Check for any signs of increased compartment pressure
- Check for distal pulses & neurological deficit of ulnar, radial & median

nerves

Investigations

- Xray-elbowAP/Lateral view
- CT scan— to delineate geometry of fracture & plan surgery especially in intraarticular fractures. compartment pressure monitoring in suspicious cases
- Ultrasound Doppler Study-to rule out vascular injury
- Angiogram- if vascular injury suspected
- MRI -rarely needed

Treatment

- Initial management - Splint the limb, elevation of the limb& ice packs application for anti-oedema measures.
- Conservative - in medically unfit patients and very comminuted fractures treated with casts (BAG OF BONES TREATMENT)

Patients with associated neurovascular injury or in unstable fractures where facilities are not available for ideal internal fixation should be referred to higher centres.

RADIAL HEAD FRACTURE

Introduction- Radial head fractures account for almost 33% of fractures of the elbow region. Radialhead plays an important role in providing smooth movements of forearm (Pronation and supinafion) and the elbow joint (flexion and extension at radiocapitellar joint)

History- Fall on an outstretched hand with a valgus strain

Diagnosis-

Clinical Features-Pain and swelling over elbow region

On examination-tenderness over the radial head with restriction of forearm and elbow movements

Imaging Techniques-Xray (AP, Lateral Oblique views) & Rarely CT or MRI

Management-

Conservative treatment:

Restricted to undisplaced fracture in the form of POP back splint. For displaced and comminuted fractures or associated neurovascular injuries refer to higher centre for operative procedures where facilities are available.

FRACTURE OF BOTH BONES FOREARM IN ADULTS

Incidence- 1%of all fractures

History

- Tenderness &swelling of forearm
- Deformity
- Abnormal mobility & crepitus at fracture site

Diagnosis

- Tenderness and swelling of forearm
- Deformity
- Abnormal mobility and crepitus at fracture site

Investigations

- Routine radiographs of forearm with elbow and wrist in AP and Lateral view are helpful in diagnosis and to rule out injuries of joints above and below
- Routine blood tests, chest x rays, ECG and ECHO performed for working up the patient for surgery
- Doppler study done in case of suspected vascular injury
- Thorough neurological examination done to rule out any nerve injury

Treatment

For undisplaced fractures

1. Closed reduction and cast immobilization
2. Functional bracing for weeks

For displaced fractures and nonunion or associated neurovascular injuries refer to higher centre for operative procedures where facilities are available

TENNIS ELBOW

Introduction- It is an inflammatory condition of common extensor origin over the lateral epicondyle of humerus following an injury or sudden contraction of the common extensor origin. Exact etiology is unknown.

Clinical Features-

- Pain over lateral aspect of elbow
- Sense of weakness when lifting even small object
- Localized tenderness

Investigation-

- Test - ask the patient to keep the elbow wrist & fingers in full flexion and forearm in full pronation. Jerky extension of elbow causes marked pain at lateral epicondyle.
- X-raysarenormal

Treatment-

- Rest to the elbow along with anti inflammatory drugs is very helpful
- Course of ultrasonic therapy is given
- Resistant cases will respond to local injection of steroid

If patient does not respond to the conservative treatment then refer to higher centre for operative procedures where all the facilities are available.

HAND INJURIES

CARPAL INJURIES

SCAPHOID FRACTURE

Fracture of the carpalscaphoid bone is the most common fracture of the carpus and frequently diagnosis is delayed

Incidence-Scaphoid fracture accounts for about 50-80% of'carpal injuries

Diagnosis Afld Investigations- Clinical evaluation +Xrays (PA Lateral, scaphoid & clenched fist view) MRI, CT, bone scan maybe used to diagnose occult fractures

Complications- Delayed union, non union, osteonecrosis, CRPS

Management-

Conservative : 6-8 weeks glass holding cast for undisplaced fractures. For displaced and nonunion fractures of scaphoid refer to higher centre-for operative procedures where facilities are available.

HAND FRACTURES

Metacarpal and phalangeal fractures are common comprising 10% of all fractures. There is high incidence of variation of mechanism of injury accounting for broad spectrum of patterns of fractures in hand.

Incidence and Classification - Distal phalanx fracture are most common of all hand fractures (45%) followed by metacarpal fractures (30%), proximal phalanx (15%) and middle phalanx (10%). -

Diagnosis and investigations - Clinical evaluation + X-rays (PA, Lateral & oblique radiographs). CT may be required to assess the intraarticular fractures

Complications - Delayed union, malunion, nonunion, CRPS, stiffness & loss of motion, Infection, post traumatic osfeoarthrifis etc.

Management -

Metacarpal fractures: Undisplaced stable fractures can be treated conservatively with MCP joint immobilized >70 degrees. Displaced fractures usually require ORIF with K-wires or mini-plates.

Proximal phalanx& middle phalanx:

- Intraarticular fractures-ORIF is preferred. For comminuted fractures, ligamentotaxis with external fixators or specialised reconstruction techniques can be used.
- Extra articular-Stable fractures: conservative
- Unstable-CRlF or ORIF (K wire or mini plate)

Distal phalanx:

- Intra-articular fracture (Mallet finger) - Extension block pinning for mallet finger. Extension splitting for soft mallet finger
- Extraarticular fractures - Usually treated as soft tissue injury. If displaced widely, CRlF is recommended.

Reasons- for referral to higher centre- Lack of expertise, lack of infrastructure, fracture dislocations.

Tendon injuries: Patient with tendon injuries should be referred to higher centres for surgical procedure where the facilities are available.

CARPAL TUNNEL SYNDROME

Introduction- It is a syndrome of compressive neuropathy of median nerve at wrist caused due to elevated pressure within carpal funnel. Carpal funnel is a fibrous tunnel formed by palmar hallow of the articulated carpal bones & roofed by flexor retinaculum.

Aetiology-

- Rheumatoid inflammation of flexor tendon sheath
- Compound palmargangliori
- Anterior dislocation of lunate

- Malunited Colle's fracture
- Myxoedema
- Amyloidosis
- DM
- Steroid use
- Pregnancy causing edema of tissues

Clinical Features-

- Pain
- Paraesthesia over palmar aspect of hand
- Hand numbness worsening at night
- Weakness and wasting of thinner muscles and sensory deficit

Treatment-

- Non operative methods such as NSAIDs and steroid injections
- If specific cause present, treat the cause

If patient does not respond to the conservative treatment then refer to higher centre where all the facilities are available.

FRACTURE OF THE LOWER LIMB FRACTURE

NECK OF FEMUR (INTRA-CAPSULAR)

Fracture neck of femur is still the unresolved fracture as evident by the number of procedures available and practiced, thus none is universally applicable and the surgeon has to select one which would be ideal in a given situation. The treatment varies with the age of the patient, the level of the fracture and the displacement of the fragments. Also the duration of the fracture is a major deciding factor. If union of the fracture Is not likely to be achieved then alternative method should be adopted which will suit the patient, keeping in mind his age, lifestyle, profession and economic status. Majority of our patients are not covered by health insurances, hence all the expenditure has to be borne by the patient himself. It is therefore desirable on the part of the treating orthopaedic surgeon to choose a method which these patients can afford.

Fracture neck femur is commonly seen in old people, but in our country quite a good number of patients are young adults. It is infrequent in children. Fracture neck femur whether intra-capsular or extra-capsular can be diagnosed and differentiated by clinical examination and confirmed by the roentgenograms. Any underlying pathologic condition like metastasis or osteoporosis if present can also be identified on roentgenograms.

Principles of management:

BELOW AGE 50

- Child:-fixation with
  - MOORES PIN
  - HAGES PIN
  - KNOWELS PIN
- Adults
  - Cannuated Iag screw
  - Subtroca nferic abduction osfeotomy
  - DHS

ABOVE AGE 50

1. THR
2. BIPOLAR hemiarthropasy

Fracture more than 3 weeks duration

BELOW 50- Osteosynthesis along with mcrnurray osteotomy

ABOVE 50-THR

If patient is poor afford ing wise:- girdle stone or bachelor procedure can be done

TROCHANTERIC FRACTURES (EXTRA-

CAPSULAR FRACTURE NECK FEMUR)

lntertrochanteric hip fractures account for approximately half of the hip fractures in the elderly; out of this more than 50% fractures are unstable. Unstable patterns occur more commonly with increased age and with low bone mineral density. The fracture commonly occurs through a bone affected by osteoporosis. The presence of osteoporosis in intertrochanteric fractures is important because fixation of the proximal fragment depends entirely on the quality of cancellous bone present, Unstable intertrochanteric fractures are those in which comminution of posteromedial buttress exceeds as implelesser trochanteric fragment or those with subtrochanteric extension. The results of fixation in unstable fractures are less reliable and have a high rate of failure (8%-25%).

Investigations- X-rays of the pelvis including both hips and knee joint and of other areas if required, General Investigations and specific if required according to the status of the health of the patient.

Examination- Pt should be carefully examined for distal vascular and neurological deficit, if any. If there is any pt should be referred to higher centre immediately.

Management- These pts need surgical treatment for early mobilization as they are generally elderly people. If the pt is kept in bed and immobalized he s bound to develop bed-sore and other complications. In case pt is unfit for surgery then pt should be put on skeletal traction in 30 deg of abduction for inter trochanteric fractures til radiological signs of union are visible on x- rays

FRACTURE SHAFT OF FEMUR

Fractures of the shaft of the femur are the result of high-energy trauma and therefore can be both life-threatening injuries and causes of severe permanent disability. Isolated injuries can occur with repetitive stress and may occur in the presence metabolic bone diseases, metastatic disease, or primary bone tumors.

The femur is very vascular and even a fracture can result in significant occult blood loss into the thigh. Up to 40% affected with isolated fracture shaft femur may require blood transfusion, as such injuries scan result in loss of up to 3 units of blood This factor is significant, especially in elderly patients who have less cardiac reserve.

Most femoral diaphyseal fractures are treated surgically with intramedullary nails or extra-medullary plate fixation. The goal of treatment is reliable anatomic stabilization, allowing mobilization as soon as possible. Surgical stabilization is also important for early extremity function, allowing both hip and knee motion and strengthening. Injuries and fractures of the femoral shaft may have significant short-and long-term effects on the hip and knee joints if alignment is not restored

Investigations- X-rays of the part including hip and knee and of other areas if required, x-ray of pelvis with both hips is must. General Investigations and specific if required, according to the status of the health of the patient.

Examination- Patient should be carefully examined for distal vascular and neurological deficit, if any. If there's any patient should be referred to higher Centre immediately.

Management- As these patients have profuse infernal bleeding, special attention to hemodynamic status should be given. Intravenous fluids such as Ringer, Dextrans (plasma expander) and blood transfusion should be used. Patient should be catherized to have a check on renal output. Leg traction should be applied to immobalize and align the fracture. Thomas splint should be used to transport the patient.

Femur should be fixed surgically with Nailing or Plating depending upon fracture site and pattern.

FRACTURE OF TIBIA/FIBULA

Introduction

- Lower leg fractures include fractures of the tibia and fibula. Of these two bones, the tibia is the main weight bearing bone: Fractures of the tibia generally are associated with fibula fracture, because the force is transmitted along the interosseous membrane to the fibula.
- The skin and subcutaneous tissue are very thin over the anterior and medial tibia and as a result of this; a significant number of fractures to the lower leg are open. Even in closed fractures, the thin, soft tissue can become compromised. In contrast, the fibula is well covered by soft tissue.
- Fractures of the tibia can involve the tibial plateau, tubercle, shaft, and plafond.

Incidence - Fractures of the tibia are the most common long bone fractures. The most common fracture of the lower limb occurs at the tibial diaphysis. Isolated mid-shaft or proximal fibula fractures are uncommon.

Clinical presentation

Unnatural mobility

crepitation

absence of transmitted movement

Investigations - Perform radiographs of the knee, tibia/fibula, and ankle as indicated and of other areas if required, General Investigations and specific if required according to the status of the health of the patient. In patients with tibial plateau fractures and tibial plafond fractures, computed tomography can help further reevaluate the extent of the fracture. In tibial plateau fractures, radiographs may under estimate the degree of articular depression when compared with computed tomography. This is important because articular depression of greaterthon 3 m may be considered for surgery.

Examination- Pt should be carefully examined for distal vascular and neurological deficit, if any. If there's any pt should be referred to higher center immediately.

Management- Most of tibial diaphyseal fractures can be managed conservatively with plaster of paris cast. But with the advent of image intensifier nailing can be done and patient made mobile next day if the fracture is stable. Trans articular

fractures and compound fractures need specialized care and should be ref to higher centre after splinting.

MENISCAL INJURIES

Meniscal tears are among the most common knee injuries. Athletes, particularly those who play contact sports, are at risk for meniscal fears. However, anyone at any age can get a meniscal tear. When people talk about torn cartilage in the knee, they are usually referring to a torn meniscus.

Three bones meet to form your knee joint: your thigh bone (femur), shin bone (tibia), and knee cap (patella).

Two wedge-shaped pieces of cartilage act as "shock absorbers" between your thigh bone and shin bone. These are called meniscus. They are tough and rubbery to help cushion the joint and keep it stable.

Menisci tear in different ways. Common tears include longitudinal, parrot- beak, flap, bucket handle, and mixed/complex.

Sports-related meniscal fears often occur along with other knee injuries, such as anterior cruciate ligament tears.

Causes- Sudden meniscal tears often happen during sports with rotational injury. Players may squat and twist the knee, causing a tear.

Older people are more likely to have degenerative meniscal tears. Cartilage weakens and wears over time. Aged, worn tissue is more prone to tears. Just an awkward twist when getting up from a chair may be enough to cause a tear, if the menisci have weakened with age.

Symptoms- You might feel a "pop" when you tear a meniscus. Most people can still walk on their injured knee. Many athletes keep playing with a tear. Over 2 to 3 days, your knee will gradually become more stiff and swollen.

The most common symptoms of meniscal tear are:

- Pain
- Stiffness and swelling
- Catching or locking of your knee
- The sensation of your knee "giving way"
- You are not able to move your knee through its full range of motion

Without treatment, a piece of meniscus may become loose and drift into the joint. This can cause your knee to slip, pop, or lock.

Examination - McMurray's Test-There are many tests to localize the lesion. Like anterior drawer test, posterior drawer test, Lachman test etc.

Nonsurgical Treatment

If your tear is small and on the outer edge of the meniscus, it may not require surgical repair. As long as your symptoms do not persist and your knee is stable, nonsurgical treatment may be all you need.

RICE - The RICE protocol is effective for most sports-related injuries. RICE stands for Rest, Ice, Compression, and Elevation.

- **Rest -** Take a break from the activity that caused the injury. Use crutches to avoid puffing weight on your leg.
- **Ice -** Use cold packs for 20 minutes at a time, several times a day. Do not apply ice directly to the skin.
- **Compression -** To prevent additional swelling and blood loss, wear an elastic compression bandage.
- **Elevation -** To reduce swelling, recline when you rest, and put your leg up higher than your heart.

- NSAIDs - Drugs like aspirin and ibuprofen reduce pain and swelling.

Surgical Treatment - If your symptoms persist with nonsurgical treatment, patient should be referred to higher center for arthroscopic or open surgery.

SPINAL INJURY

Introduction

With an estimated annual incidence of 300 per crore population, approximately 40,000 new spine injury cases are added every year in India. 40% of these are complete lesions i.e. tetra or paraplegia The socioeconomic impact of spinal injuries is huge with 85% of victims being male in the age group of 15 to 35 years. Management of patients who have sustained spinal cord injury requires careful assessment and management. In adequate assessment and management of these injuries may lead to worsening of existing spinal cord injury or the production of a new cord injury.

Case definition: For both situations of care (mentioned below*). Spinal cord injury (SCI) is an insult to the spinal cord resulting in a change, either temporary or permanent, in its normal motor, sensory, or autonomic function.

Incidence- In the absence of a national spinal cord injury registry in India, the exact incidence is not known. Approximately 30 cases per million population. Approximately 40% of these will be complete. Majority of the cases are due to road side accidents or fall from height.

Differential Diagnosis - All trauma patients should be assumed to have a spinal injury and treated as such till a detailed clinical examination and radiological investigation has been performed. Potential spinal cord injury should be suspected in following situations:

- Altered mental status.

- Evidence of intoxication.

-Associated head injury, extremity fracture

- Focal neurological deficit.

- Spinal pain or tenderness

Mechanism of injury e.g fall from height, fall on head, whiplash injuries, high energy injuries.

Clinical Diagnosis:

After the ABC have been taken care of, the patient is gently logrolled and whole of the spine is palpated for tenderness or a palpable step-off deformity.

Neurogenic shock, in continence of bowel, bladder and penile erection indicates ever spine injury. A careful and detailed neurological examination is then performed and meticulously documented.

Frankel's grades: Spinal Cord Injury is most commonly graded using Frankel's grades (A to E).

A Complete motor and sensory loss

B Sensation only present below lesion

C Sensations present and motor function is present but useless

D Motor useful but not normal

E Noneurological deficit.

After the motor and sensory examination, presence of sacral sparing may be noted by voluntary rectal sphincter tone and toe flex or contractions. Presence of sacral sparing indicates a better neurological prognosis.

Although spinal shock is over by 24 hours, rarely it may be prolonged. A positive bulbocavernous reflex or a positive anal wink indicates the end of spinal shock. If no motor or sensory function can be documented at this stage, a complete spinal cord injury is present.

Investigations:

X- Rays: All patients with suspected spinal injury should have radiographic evaluation.

- Initial screening can be done by conventional antero-posterior and lateral x-rays.
- The cervical spine radiographs must include the C7-T1 junction to be considered adequate
- Additional Open-mouth views should be done to evaluate odontoid injury. • Whole spine should be evaluated with a patient of spinal injury.
- The patient should be referred for advanced diagnostic modalities only when the patient is stable:

CT Scan: CT scan cervical spine in all cases of head injuries or intoxication at the same time as the brain CT. CT should be done when plain X-Ray is inadequate, particularly for upper cervical spine injuries and C7-T1 junction.

MRI: **MRI** is essential for evaluating injury to the soft tissues and ligaments, discs, intrinsic cord damage (oedema, hematoma, or contusion) and Para vertebral soft tissues. MRI is particularly useful in scenarios such as central cord syndrome where plain radiographs will not show any fractures or dislocations (SCIWORA). If possibIeMRl should be done before the cervical traction is applied. In patients with pre- injury morbidities such as Ankylosing Spondylitis, CT and MRI should be done to rule out occult instability even if x-rays are normal.

Management:

CONSERVATIVE - If no neurological deficit

SURGERY – Refer the patient to higher centre for specialized care.

Anterior decompression and fixation with plating or post decompression with pedical screws General care of the patient is the most commonly neglected part

1. Genitourinary Tract:

- Place an indwelling urinary catheter as part of the initial patient assessment unless contraindicated
- Leave indwelling urinary catheters in place atleast until the patient is haemodynamically stable
- Care of indwelling catheter should be taught to patient or the relatives as soon as the patient is stable

2. Gastrointestinal Tract

- Initiate stress ulcer prophylaxis.
- Evaluate swallowing function prior to oral feeding in any acute SCI patient.

3. Measures to prevent bed sores:

- Assess areas at risk for skin breakdown frequently.
- Place the patient on a pressure-reduction mattress or a mattress overlay depending on the patient's condition.
- Use a pressure-reducing cushion when the patient is mobilized out of bed to a sitting position.
- Reposition to provide pressure relief or turn at least every2 hours while maintaining spinal precautions.
- Keep the area under the patient clean and dry and avoid temperature

elevation.

- Assess nutritional status on admission and regularly thereafter.
- Inspect the skin under pressure garments and splints.
- Educate the patient and family on the importance of vigilance and early intervention in maintaining skin integrity

OPHTHALMOLOGY STANDARD TREATMENT GUIDELINES

STYE (EXTERNAL HORDEOLUM)

Stye is an acute suppurative inflammation of glands of Zeis due to staphylococci infection and is common in young adults and debilitated persons.

Clinical features:

- Acute pain and tenderness over inflamed Zeis gland, seen as a localised painful and hard swelling seen near the lid margin.
- The lid margin is red and oedematous.
- An abscess may form which points near the base of the lash.
- The pain subsides after evacuation of the pus.

Non- pharmacological treatment:

- Dry hot fomentation applied frequently in early stage is useful.

Pharmacological treatment:

1. Evacuation of the pus by pulling the involved lash or incising the abscess.
2. Antibiotic eye drops and ointment- to control and prevent spread of infection (fluoroquinolones e.g. moxifloxacin 0.5% eye drops 4 to 6 hourly until infection subsides)
3. Systemic antibiotics (e.g. Tab. amoxicillin with clavulanic acid 625 mg TDS for 5 days) For pain and inflammation e.g. Tab. ibuprofen 400 mg TDS till inflammation subsides.

Patient education:
Maintain ocular hygiene to prevent recurrence.

CHALAZION

A chalazion (meibomian cyst) is a chronic, sterile, granulomatous inflammatory lesion caused by retained sebaceous secretion leaking from the meibomian or other sebaceous glands into adjacent stroma. A chalazion secondarily infected is referred as an internal hordeolum.

Clinical features:

- A gradually enlarging painless nodule.
- Very rarely a large upper lid chalazion may press on the cornea, induce astigmatism and cause blurred vision.
- A 'marginal' chalazion is similar except that it involves a gland of Zeis and is therefore located not in the tarsal plate but on the anterior lid margin.
- Patients with meibomian gland disease or rosacea are at increased risk of chalazion formation which may be multiple and/or recurrent.

Non- pharmacological treatment:

- Treatment may not be required because at least a third of chalazia resolve spontaneously and an internal hordeolum may discharge and disappear. Persistent lesions may be treated as follows:
- Dry hot fomentation applied frequently in early stage is useful.

Pharmacological treatment:

1. Steroid injection into the lesion is preferable if close to the lacrimal punctum because of the risk of surgical damage.Between 0.2 and 2 ml of 5 mg/mL triamcinolone diacetate aqueous suspension diluted with lidocaine (or equivalent) to a concentration of 5mg/mL is injected through the conjunctiva into the tissue around the lesion with a 30-gauge needle.The success rate following one injection is about 80%. In unresponsive cases a second injection can be given 2 weeks later.
2. Cap. doxycycline (100 mg BD for 7 days) may be required as prophylaxis in patients with recurrent chalazia, particularly if associated with acne rosacea.
3. Incision and curettage, in case of large chalazia, if present for more than 3 – 4 months or if cosmetically unacceptable.

Patient education:
Recurrence may occur; common causes are uncorrected refractive error, blepharitis and diabetes.

VIRAL CONJUNCTIVITIS

Clinical features:

- Common symptoms include itching, foreign body sensation, watering of eyes.
- It can also present as pharyngoconjunctival fever and acute haemorrhagic conjunctivitis.
- Frequently a history of recent upper respiratory tract infection or contact with a patient. It often starts in one eye and involves the fellow eye a few days later.
- On examination, inferior palpebral conjunctival follicles and pinpoint subconjunctival haemorrhages are observed.
- The common organism responsible is Adenovirus.
- Conjunctival cultures/swabs are not needed unless the discharge is excessive or the condition becomes chronic.

Non- pharmacological treatment:

- Self-limiting condition that typically gets worse for the first 4 to 7 days after onset and may not resolve for 2 to 3 weeks or longer if there is corneal involvement.
- It is highly contagious, usually for 10 to 12 days from onset as long as the eyes are red (when not on steroids).
- Use dark goggles and avoid close contact with other persons for two weeks.
- Restrict work and school for patients with significant exposure to others.
- Frequent handwashing and cool compresses several times per day.

Pharmacological treatment:

1. Preservative-free artificial tears (carboxy methyl cellulose 0.5% eye drops) four to eight times per day for 1 to 3 weeks. Use single-use vials to limit tip contamination and spread of disease.
2. Antihistamine (e.g., epinastine 0.05% BD) if itching is severe.
3. Anti-inflammatory drops like ketorolac 0.5% may also be used if needed.
4. If a membrane / pseudomembrane are present, use a steroid in form of drops with or without ointment (e.g. loteprednol 0.5% QID, flurometholone 0.1% ointment QID or dexamethasone 0.3% ointment QID). Steroid treatment is maintained depending upon the response and then slowly tapered.Routine / unsupervised use of topical antibiotics or steroids should be discouraged.

Patient education:

- Patients should avoid touching their eyes, shaking hands, sharing handkerchiefs, towels with family members.
- Patient's drops or ointment must not be used by other members of the family.

ALLERGIC CONJUNCTIVITIS

It occurs as a result of hypersensitivity reaction due exposure to allergic substances.

Clinical features:

- The patient present with marked itching and watery discharge.
- History of allergy is usually present.
- On examination, conjunctival papillae and edema over eyelids is seen.

Non- pharmacological treatment:

- Cool compresses several times per day and avoid offending agents.

Pharmacological treatment:

1. Topical anti-inflammatory and anti-histaminic drops, depending on the severity (olopatadine 0.1% OD/BD, nedocromil 2% TDS, or ketotifen 0.025% BD, ketorolac 0.5% QID).
2. In severe cases, mild topical steroid (e.g., loteprednol 0.2% or fluorometholone 0.1%, QID for 1 to 2 weeks) may have to be added. Steroids are gradually tapered as patient responds to medications.
3. Preservative free artificial tears four to eight times per day.
4. Investigate for offending agents and avoidance of the same.
5. Oral antihistamine (e.g., diphenhydramine 25 mg TDS/QID or cetrizine /levocetrizine /montelukast /loratadine 10 mg OD) can be helpful in severe cases and in cases with associated systemic allergy.

VERNAL / ATOPIC CONJUNCTIVITIS

Vernal keratoconjunctivitis (VKC) is a recurrent bilateral disorder in which both IgE- and cell- mediated immune mechanisms play important roles. It primarily affects boys and onset is generally from about the age of 5 years onwards (mean age 7 years).

Clinical features:

- Common symptoms include marked itching, thick ropy discharge and seasonal occurrence (during spring/summer)
- History of atopy may be present. Usually seen in young patients, especially boys.
- Large conjunctival papillae seen under the upper eyelid or along the limbus (limbal vernal). Often, they are so large as to give a cobble stone appearance.
- Superior corneal "shield" ulcer (a well-delineated, sterile, gray–white infiltrate) maybe seen in some cases.

Treatment:

1. Treat as for allergic conjunctivitis.
2. Mast cell stabilizer (e.g. sodium chromoglycate QID) or a dual action mast cell stabilizer and antihistamine (e.g., olopatadine 0.1% OD/BD) or ketotifen 0.025%, or azelastine
3. 0.05% for 2 to 3 weeks, prophylactically, before the season starts).
4. If a shield ulcer is present, add topical steroid (e.g., loteprednol 0.5% or flurometholone 0.1%, prednisolone 1%, etc) four to six times per day. Taper steroids over 4 week as condition of patient improves. Shield ulcers may need to be scraped to remove superficial plaque-like material before re-epithelialization will occur.
5. Topical antibiotic (e.g., moxifloxacin 0.3% eye drop, tobramycin 1% QID) is given if associated infection is there or steroids are prescribed
6. Cycloplegic agent (e.g., cyclopentolate 1% BD drops or atropine 1% TDS in form of drops or ointment) if cornea is involved, when there is much pain due to ciliary spasm.
7. Consider cyclosporine 0.05% BD if not responding to the preceding treatment. Maximal effect may not be seen for several weeks.

- Follow-up should be done every 1 to 3 days in the presence of a shield ulcer; otherwise, every few weeks. Steroids are tapered slowly as improvement is noted.
- Anti-allergy drops are maintained for the duration of the season and are often reinitiate a few weeks before the next spring.

- Patients on topical steroids should be monitored regularly, including IOP monitoring, even if used only on the skin.
- Patients and relatives of children should be strictly instructed not to use or continue steroids, in any form, without medical advice.

Patient education:

Avoid exposure to substances causing atopy. Maintain hygiene to prevent secondary infection.

BACTERIAL CONJUNCTIVITIS (NON-GONOCOCCAL)

Acute bacterial conjunctivitis is a common and usually self-limiting condition caused by direct eye contact with infected secretions. The most common isolates are S. pneumoniae, S.aureus, H. influenzae and Moraxella catarrhalis.

Clinical features:

- The patient complains of redness, foreign body sensation, mucoid or mucopurulent
- discharge.
- Itching is much less prominent.
- On examination, purulent discharge, conjunctival papillae and chemosis are seen.
- If severe or recurrent, conjunctival swab for routine cultures and sensitivities and immediate Gram stain to evaluate for gonococcus.

Non- pharmacological treatment:

- Patients should avoid touching their eyes, shaking hands, sharing handkerchiefs, towels, etc.
- Restrict work and school for patients with significant exposure to others.
- Frequent hand washing.

Pharmacological treatment:

1. Use topical antibiotic therapy [moxifloxacin 0.5% drops QID or more frequent in severe cases] for 5 to 7 days.
2. H. influenzae conjunctivitis should be treated with oral amoxicillin/clavulanic acid (20 to
3. 40 mg/kg/day in three divided doses) because of occasional extra ocular involvement (e.g., otitis media, pneumonia, and meningitis).

Antibiotic therapy is adjusted according to culture and sensitivity results.

4. If associated with dacryocystitis, systemic antibiotics are necessary.

Patient education:

Follow up every 2 to 3 days initially, then every 5 to 7 days until resolved. Maintain ocular hygiene.

GONOCOCCAL CONJUNCTIVITIS

Clinical features:

- The patient complains of redness, foreign body sensation, mucoid or mucopurulent discharge.
- Itching is much less prominent.
- On examination, purulent discharge, conjunctival papillae and chemosis are seen.
- Clinical symptoms start within 12 to 24 hours and include severe purulent discharge, swelling of eyelids
- On examination, conjunctival papillae, marked chemosis and preauricular adenopathy is observed.

If severe or recurrent, conjunctival swab for routine cultures and sensitivities and immediate Gram stain to evaluate for gonococcus.

Examination of entire cornea for peripheral ulcers (especially superiorly) should be done because of the risk for rapid perforation.

Conjunctival scrapings for immediate Gram stain and for culture and sensitivities (e.g., blood agar and chocolate agar) should be taken.

Pharmacological treatment:

Initiated if the Gram stain shows gram negative intracellular diplococci or there is a high suspicion clinically of gonococcal conjunctivitis.

1. Inj. ceftriaxone 1 g intramuscularly in a single dose.
2. If corneal involvement exists, or cannot be excluded because of chemosis and eyelid swelling, hospitalize the patient and treat with Inj. ceftriaxone 1 g intravenously every 12 to 24 hours.
3. In penicillin-allergic patients, consider an oral fluoroquinolone (e.g., Tab. ciprofloxacin 500 mg, for 5 days) or a single oral dose of Tab. azithromycin 1 g.

4. Topical ciprofloxacin ointment QID or fluoroquinolone drops every two hours (e.g., gatifloxacin, moxifloxacin, or ciprofloxacin).
5. Saline irrigation QID until the discharge resolves.
6. Treat for possible chlamydial co-infection (e.g., Tab. azithromycin 1 g single dose or Tab. doxycycline 100 mg BD for 7 days).
7. Treat sexual partners with oral antibiotics for both gonorrhea and chlamydia as described previously.

Patient education:

- The patient should be followed daily until consistent improvement is noted, and then every 2 to 3 days until the condition resolves.
- The patient and sexual partners should be evaluated by their medical doctors for other sexually transmitted diseases.

TRACHOMA

Clinical features:

- Trachoma is an infectious disease caused by the Chlamydia trachomatis type A, type B, type C bacterium which produces a characteristic roughening of the inner surface of the eyelids.
- It is also called granular conjunctivitis and Egyptian ophthalmia and is the leading cause of infectious blindness in the world.
- Complaints of mild itching and irritation of the eyes and eyelids, purulent or mucoid discharge from the eyes, photophobia, blurred vision and eye pain are common.
- Diseases progresses slowly, and the more painful symptoms may not emerge until adulthood.
- Presence of at least two of the following signs: superior tarsal follicles, limbal follicles (Herbert's pit), typical conjunctival scarring and vascular pannus.
- Transmission of the disease occurs vial ocular and nasal secretions (direct spread, indirect via fomites, coughing/sneezing).

Grading of severity:

(Mac Callan Classification)

Stage 1: Superior tarsal follicles, mild superior SPK, and pannus, often proceeded by purulent discharge and tender preauricular nodes.

Stage 2: Florid superior tarsal follicular reaction (2a) or papillary hypertrophy (2b) associated with superior corneal SEIs, pannus, and limbal follicles.
Stage 3: Follicles and scarring of superior tarsal conjunctiva.
Stage 4: No follicles, extensive conjunctival scarring.
Late complications: Severe dry eyes, trichiasis, entropion, keratitis, corneal scarring, superficial fibrovascular pannus, Herbert pits (scarred limbal follicles), corneal bacterial superinfection, and ulceration.

World Health Organization (WHO) Classification

TF (Trachomatous inflammation: follicular): More than five follicles on the upper tarsus.

TI (Trachomatous inflammation: intense): Inflammation with thickening obscuring more than 50% of the tarsal vessels.

TS (Trachomatous scarring): Cicatrization of tarsal conjunctiva with fibrous white bands.

TT (Trachomatous trichiasis): Trichiasis of at least one eyelash.

CO (Corneal opacity): Corneal opacity involving at least part of the pupillary margin.

Treatment:

The **SAFE strategy** for trachoma management supported by the WHO and other agencies encompasses

S Surgery for trichiasis
A Antibiotics for active disease
F Facial hygiene and
E Environmental improvement

1. Antibiotics should be administered to those affected and to all family members. Single antibiotic course is not always effective in eliminating infection in an individual, and communities may need to receive annual treatment to suppress infection.
2. A single dose of azithromycin (20 mg/kg up to 1g) is the treatment of choice.Topical 1% tetracycline ointment is less effective than oral treatment; it should be given for 6 weeks.
3. Surgery is aimed at relieving entropion and trichiasis and maintaining complete lid closure
4. Facial cleanliness is a critical preventative measure. with bilamellar tarsal rotation.
5. Environmental improvement such as access to adequate water and sanitation, as well as control of flies, is important.

CORNEAL ULCER

- Defect in the integrity of corneal epithelium along with infiltration of underlying stroma is defined as corneal ulcer.
- It can be due to direct corneal trauma, chronic eyelid disease, tear film abnormalities, hypoxic trauma from contact lens wear, and deficient nutrition as in keratomalacia or neuroparalytic keratitis (Bell's palsy).

It is classified as;

1. Infective keratitis may be bacterial, fungal or viral.
2. Non infective like traumatic (chemical/thermal/radiation), neoplastic or immune mediated.

BACTERIAL KERATITIS

Clinical features:

- Source of infection can be exogenous or endogenous (from other ocular structures)
- Patient presents with history of trauma to the eye with pain and redness.
- Concomitant photophobia, discharge and dimness of vision can also be present.
- On examination, conjunctival and circumcorneal vessels may be engorged and inflamed with localized epithelium disintegrated and cast off.
- Ulcer is saucer shaped, walls project above the normal corneal surface.
- Surrounding area shows grey discoloration (cloudiness) suggestive of the progressive stage of the ulcer-microscopic tissue examination reveals leucocytic infiltration.
- In severe cases there is pronounced anterior chamber reaction, often with hypopyon.

Investigations:

- Conjunctival swab and ulcer scrapings can be tested with Gram stain, KOH preparation and culture to determine the etiology.
- Routine investigations like complete blood count, blood sugar, urine examination should be done. Septic foci should be found.

Non- pharmacological treatment:

- Maintain proper ocular hygiene by cleaning discharge twice a day.
- Removal of contributory factors like trichiasis, foreign body etc.
- The eye can be covered with a pad, unless the discharge is copious. In such case, shield the eye with dark goggles.
- Prevention and treatment of complications – secondary glaucoma.

Pharmacological treatment:

1. Control of infection with antibiotics-Start with empirical broad spectrum antibiotics, which can be changed according to sensitivity report
2. Use atropine 1% eye drops or ointment for controlling iridocyclitis, to relieve ciliary spasm and to prevent synechia formation.
3. Other measures like scraping and cauterization.
4. If the ulcer progresses in spite of the pharmacological treatment, the removal of the necrotic material may be hastened by scraping or ulcer may be cauterized with 100% carbolic acid, 10% trichloracetic acid 10% or 5% povidone iodine.
5. In case of non resolving ulcers, surgical methods like vasculoplasty, conjuctival hooding and patch graft, tectonic penetrating keratoplasty and tarsorraphy can be considered.

Patient education:

- Use dark goggles and avoid close contact with other persons for two weeks.
- Restrict work and school for patients with significant exposure to others.
- Frequent handwashing.

FUNGAL KERATITIS

Clinical features:

- Common organisms are aspergillus, penicillium, fusarium and candida.
- Risk factors are leukemia and diabetes, pre existing corneal disease like dry eye, vegetative injury and long term treatment with antibiotic and steroids.
- On examination, definite white ring in the mid periphery of the cornea with healthy cornea between the ring and ulcer margin is seen (Immune ring- diagnostic of fungal infection).
- Presence of hypopyon is the rule. Higher the hypopyon more ominous the sign and chances of perforation.

Non -pharmacological treatment:

- Use dark goggles and avoid close contact with other persons for two weeks.
- Restrict work and school for patients with significant exposure to others.
- Frequent handwashing.

Pharmacological treatment:

1. Treatment should be instituted promptly with topical antifungal drops,

initially every hour during the day tapered to 4 hrly interval for 3-4 days, then reduced to 4 times a day for at least 14-21 days or till there is resolution of active stage.

2. Subconjunctival injections also may be used in cases of severe keratitis, or when poor compliance exists.
3. An oral antifungal (eg, ketoconazole, fluconazole 100mg) should be considered for cases of deep stromal infection.
4. Fluconazole has been shown to penetrate better into the cornea after systemic administration compared to other azoles and is associated with fewer adverse effects.

5. Intra-cameral:

Inj. amphotericin-B (5 - 7.5% µg in 0.1ml in 5% dextrose).

6. Intracorneal:

Inj. amphotericin-B (5µg in 0.1ml) four to five places around the lesion intrastromaly, not in thin area.

Inj. voriconazole (50 µg in 0.1% in RL solution).

Systemic antifungal:

1. Oral fluconazole and ketoconazole are absorbed systemically with good levels in the anterior chamber and cornea - therefore they should be considered in the management of deep fungal keratitis fungal abscess, endothelial plaque.
2. Cycloplegics are mandatory and antiglaucoma drugs may be added to the treatment depending on the intraocular pressure etc.
3. Frequent corneal debridement with a spatula is helpful. It debulks fungal organisms and epithelium, and enhances penetration of the topical antifungal agent.
4. Surgery is considered in those patients who fail to respond to medical treatment and may result in corneal perforation.
5. The treatment of choice is therapeutic penetrating keratoplasty.

VIRAL KERATITS

Common viruses that cause corneal disease are Herpes simplex virus (HSV), Varicella zoster, Epstein Barr and Adenovirus.

Cytomegalovirus can also cause keratitis and is more commonly associated with AIDS.

HERPES SIMPLEX KERATITIS

Clinical features:

- Herpes simplex keratitis occurs in two forms – primary and recurrent.
- Primary infection of any of the 3 branches (ophthalmic, maxillary, mandibular) of cranial nerve V leads to latent infection of nerve cells in trigeminal ganglion.
- Interneuronal spread of HSV within ganglion allows patients to develop ocular disease without ever having had primary ocular HSV infection.
- Recurrent infection has been thought of as reactivation of virus in the sensory ganglion.
- The disease manifests as vesicular blepharoconjunctivitis occasionally with corneal involvement as punctate keratitis.
- The patient presents with irritation, photophobia, reduction in vision (when central cornea is affected) and corneal anaesthesia.
- Corneal ulceration and scarring can occasionally be the only sign of recurrent herpetic infections. Typical branching, linear pattern with feathery edges and terminal bulbs at ends are visualized by fluorescein staining.

Pharmacological treatment:

1. Antiviral drugs

- Trifluridine and acyclovir are much more effective in epithelial disease than others.
- Idoxuridine and trifluridine are frequently associated with toxic reactions.
- Oral acyclovir may be useful in treatment of severe herpetic eye disease particularly in atopic individuals.
- For active treatment 400 mg five times daily in non immunocompromised patients.
- 800 mg five times daily in compromised and atopic patients.
- Prophylactic dosage in recurrent disease is 400 mg twice daily.
- Famciclovir or valacyclovir may also be used.

2. Topical corticosteroids accelerate corneal thinning, increasing risk of corneal perforation

3. Epithelial debridement is an effective way to treat dendritic keratitis before acyclovir. Infected epithelium is easy to remove with tightly wound cotton tip applicator.

4. Adjunctive therapy with topical antiviral accelerates epithelial healing

5. Surgical management like penetrating keratoplasty is indicated for visual rehabilitation in patients with severe corneal scarring. It should not be undertaken until herpetic disease has been inactive for many months.

GLAUCOMA

The classical triad of increased intraocular pressure (IOP), optic nerve head cupping and visual field changes are always present and are signs of progress of the disease and bench mark for assessing response to therapy.

PRIMARY OPEN ANGLE GLAUCOMA

In angle glaucoma the structure of the trabecular meshwork appears normal but offers an increased resistance to the outflow of aqueous which results in an elevated ocular pressure.

Clinical features:

- The disease is insidious and usually asymptomatic, until it has caused a significant loss of visual field.
- Patients may experience mild headache, eye ache and visual field defect in late cases.
- Reading and close work often present increasing difficulties owing to accommodative failure due to constant pressure on the ciliary muscle and its nerve supply. Therefore, patients usually complain of frequent changes in presbyopic glasses.
- Ocular examination including slit-lamp biomicroscopy may reveal normal anterior segment.
- In late stages pupil reflex becomes sluggish and cornea may show slight haze.
- In the initial stages the IOP may not be raised permanently, but there is an exaggeration of the normal diurnal variation. Therefore, repeated observations of IOP (every 3-4hour), for 24 hours is required during this stage (Diurnal variation test). Applanation tonometry should be preferred over Schiotz tonometry.
- Gonioscopy reveals open angles.
- Optic disc changes are typically progressive, asymmetric and present a variety of characteristic clinical patterns. It is essential, therefore, to record the appearance of the nerve head in such a way that will accurately reveal subtle glaucomatous changes over the course of follow-up period.

Investigations:

- Perimetry to detect the visual field defects.
- Nerve fibre layer analysis and disc topography analysis to detect early damage and for progression analysis.

Pharmacological treatment:

1. Identification of target pressure. From the baseline evaluation data a 'target pressure' should be identified for each patient. Progression of disease is uncommon if IOP is maintained at less than 16 to 18 mm of Hg in patients having mild to moderate damage and 10 to 12 mmHg in patients with severe damage.
2. Single drug therapy. One topical antiglaucoma drug should be chosen after due consideration to the patient's personal and medical factors. If the initial drug chosen is ineffective or intolerable, it should be replaced by the drug of second choice.
3. Combination therapy. If one drug is not sufficient to control IOP then a combination therapy with two or more drugs should be tried.

Monitoring of therapy by disc changes, field changes and tonometry is most essential on regular follow-up. In the event of progression of glaucomatous damage, the target pressure should be reset at a lower level and any risk factors should be evaluated

3. Laser treatment.

Argon laser trabeculoplasty (ALT) or selective laser trabeculoplasty (SLT) are indicated in patients intolerant to topical medication, failure of medical therapy or non compliance to treatment. Since IOP reduction with laser is seldom greater than 30%, an IOP higher than 28 mmHg is unlikely to be adequately controlled by laser alone.

4. Surgical treatment

Uncontrolled glaucoma despite maximal medical therapy and laser trabeculoplasty. Non-compliance of medical therapy and

Intolerance to medical therapy

Eyes with advanced disease i.e., having very high IOP, advanced cupping and advanced field loss should be treated with filtration surgery as a primary line of management.

Types of surgeries- trabeculectomy, shunt & valve surgeries, non penetrating glaucoma.

Patient education:

Treatment of glaucoma whether medical or surgical is aimed to decrease further damage to optic nerve. Once damage has occurred, it is irreversible.

PRIMARY ANGLE-CLOSURE GLAUCOMA

The term 'angle-closure' refers to occlusion of the trabecular meshwork by the peripheral iris, obstructing aqueous outflow.

ACUTE ANGLE CLOSURE GLAUCOMA

Clinical features:

- Patient presents acutely (congestive glaucoma) with coloured halos around lights due to corneal oedema, ocular pain and headache.
- The IOP rise maybe often be so severe as to cause nausea and vomiting and mimic a case of acute abdomen.
- Precipitating factors include watching television or movie in a darken room, reading, pharmacological mydriasis or miosis, acute emotional stress and rarely systemic medications like parasympathetic antagonists or sympathetic agonists (e.g. inhalers, motion sickness patches and cold remedies) and topiramate.
- On examination, IOP is usually very high (> 40 - 50 mmHg) with conjunctival hyperaemia, corneal edema, unreactive mid dilated vertically oval pupil.
- Fellow eye generally shows an occludable angle.

Pharmacological treatment:

1. Treatment intensity should be individualized dependent on severity.
2. Hospital admission is usually required in an acute presentation.
3. The patient should assume a supine position to encourage the lens to shift posteriorly under the influence of gravity.
4. Inj. mannitol 20% 1–2 g/kg intravenously over half hour or glycerol 50% 1 g/kg orally, having checked for contraindications.
5. Pilocarpine 2–4% one drop to the affected eye, repeated after half an hour, and one drop of 1% as prophylaxis into the fellow eye. Some practitioners prefer to omit pilocarpine in an acutely presenting eye with very high IOP until a significant IOP fall has taken place, as the associated ischaemia will compromise the action of pilocarpine on the pupillary sphincter and it may also aggravate the pupillary block.
6. An additional oral dose of Tab. acetazolamide 500 mg may be given.
7. Topical timolol 0.5%, prednisolone 1% or dexamethasone 0.1% to the affected eye, leaving 5 minutes between each instillation.
8. Analgesia and an antiemetic may often be required. Subsequent medical treatment
9. Pilocarpine 2% QID to the affected eye and 1% QID to the fellow eye.

10. Topical steroid (prednisolone 1% or dexamethasone 0.1% QID) if the eye is acutely inflamed.

Any or all of the following should be continued as necessary according to response: timolol 0.5% BD and oral acetazolamide 250 mg QID may be required.

11. Surgical treatment:

Definitive treatment is laser iridotomy or iridoplasty after corneal oedema clears. Topical steroids and any necessary hypotensives can be continued for at least a week. Surgical options in resistant cases include surgical peripheral iridectomy, lens extraction, goniosynechialysis and trabeculectomy.

Patient education:

- Do not ignore headache or chronic ache in the eyes and report to the ophthalmologist if coloured halos around light appear.

CHRONIC ACUTE ANGLE CLOSURE GLAUCOMA

Clinical features:

- Many patients with angle-closure maybe asymptomatic, including those with intermittently or chronically elevated IOP.
- They may have intermittent milder symptoms of blurring ('smoke-filled room') unassociated with pain.

Pharmacological treatment:

1. Timolol 0.5% or betaxolol0.5% eye drops twice a day usually required lifelong.
2. Surgical treatment: Laser or surgical iridotomy is done to eliminate any element of pupillary block in affected as well as fellow eye.

LENS INDUCED GLAUCOMA

Lens induced glaucoma is classified into phacolytic, phacomorphic, phacoanaphylactic glaucoma, lens particle glaucoma and glaucoma due to dislocated lens. In addition to medically lowering the IOP, the cataractous lens needs to be removed, under steroid cover to suppress the inflammatory element.

ANTERIOR UVEITIS

It is the inflammation of the uveal tract i.e. iris, ciliary body and choroid. Inflammation of iris and ciliary body constitutes iridocyclitis and anterior uveitis.

Clinical features:

- Symptoms of acute uveitis include pain, redness, photophobia, consensual photophobia (pain in the affected eye when a light is shown in the fellow eye), excessive tearing and decreased vision.
- Symptoms of chronic uveitis include decreased vision (from vitreous debris, cystoid macular edema (CME), or cataract, periods of exacerbations and remissions with few acute symptoms [e.g. juvenile idiopathic (rheumatoid) arthritis].
- Cells and flare in the anterior chamber, ciliary flush and keratic precipitates (KP) are observed by slit lamp microscopy.

Complete ocular examination, including an IOP check and a dilated fundus examination. The vitreous should be evaluated for cells.

Non- pharmacological treatment:

- Wear dark glasses.

Pharmacological treatment:

1. Cycloplegic e.g., cyclopentolate 1% eye drops BD for mild to moderate inflammation; atropine ointment 1% TDS for severe inflammation.
2. Topical steroid like prednisolone acetate 1% one drop 1 to 6 hourly, depending on the severity. Most cases of moderate to severe acute uveitis require 1 to 2 hourly dosing initially. Consider FML (flurometholone) 1% ophthalmic ointment at night.
3. If the anterior uveitis is severe and is not responding to topical steroids, then consider periocular repository steroids (e.g., triamcinolone 20 to 40 mg subtenon injection).
4. If there is no improvement on maximal topical and repository steroids, or if the uveitis is bilateral and severe, consider systemic steroids (Tab. prednisolone 1mg/kg), or immunosuppressive therapy.
5. Treat secondary glaucoma with beta blockers, not with pilocarpine or prostaglandin analogues.

6. If an exact etiology for the anterior uveitis is determined (herpes, tuberculosis, ankylosing spondylitis etc.), then appropriate systemic management is required.
7. Follow-up is done every 1 to 7 days in the acute phase, depending on the severity and every 1 to 6 months when stable. At each visit, the anterior chamber reaction and IOP should be evaluated.

If the anterior chamber reaction is improving, then the steroid drops can be slowly tapered [usually one drop per day every 3 to 7 days (e.g., QID for 1 week, then TDS for 1 week, then BD for 1 week)].

Patient education:

- Recurrent nature of the disease which may interfere with vision should be explained and possible complications like cataract and glaucoma should be mentioned.
- Possible side effects of long term topical, periocular and systemic corticosteroid therapy should be explained.

ORBITAL CELLULITIS

Orbital cellulitis refers to an acute infection of the soft tissues of the orbit behind the orbital septum. The infection can be exogenous (trauma, foreign body), endogenous (septicemia) or extension of infection from neighbouring structures (paranasal sinuses, teeth, face, eyelids).

Clinical features:

- Common causes include Streptococcus pneumoniae, Staphylococcus aureus, Streptococcus pyogenes and Haemophilus influenzae.
- Symptoms include swelling of eyelids, severe pain which is increased by movements of eye or pressure and fever, nausea, vomiting and sometimes loss of vision.
- A marked swelling of lids with chemosis of conjunctiva is observed. Restriction of ocular movements is also seen.
- Fundus examination may show congestion of retinal veins and signs of papillitis or papilloedema
- Bacterial cultures should be performed from nasal and conjunctival swabs and blood samples, X-ray to identify associated sinusitis and orbital ultrasonography to detect intra- orbital abscess should be done.

Non- pharmacological treatment:

- Warm compresses.

Pharmacological treatment:

1. Intensive antibiotic therapy:

- After obtaining nasal, conjunctival and blood culture samples, intravenous higher broad spectrum antibiotics should be administered.
- Tab. amoxicillin and clavulanic acid 625mg TDS should be given OR
- Inj. cefotaxime 1-2 gm every 12 hours in adults and 100-150 mg/kg in 2 to 3 divided doses in children are given. Antibacterials are changed according to the culture sensitivity reports and continued till resolution of symptoms.
- Ciprofloxacin or vancomycin may be used an alternative.

2. Analgesic and anti-inflammatory drugs are helpful in controlling pain and fever.
3. Surgical intervention is needed when the patient is unresponsive to antibacterials, decrease in vision and presence of an orbital or subperiosteal abscess.

Patient education:

- Any ear, sinus or dental infection especially in children should be treated promptly
- Any patient presenting with unexplained lid edema or cellulitis should be immediately referred to an ophthalmologist.

ENDOPHTHALMITIS

It is an inflammation of the internal coats of the eye along with inflammatory conditions of intraocular cavities, aqueous and vitreous humour, retina and uvea.

Endophthalmitis can be endogenous (due to hematogenous spread of infective agents) and exogenous (direct inoculation of infecting agents through breach in continuity of ocular coats e.g. post traumatic).

Clinical features:

- Etiological agents include Staphylococcus aureus, coagulase negative Staphylococci, Streptococci, Pseudomonas aeruginosa, Hemophilus influenza and fungus like Candida, Aspergillus, Histoplasma etc.
- History of eye surgery, penetrating injury, fever, infection or predisposing

systemic diseases leading to metastatic endophthalmitis.

- Symptoms include visual loss, pain, discharge, photophobia, lid swelling. Signs include conjunctival congestion, hypopyon, vitrous exudates and loss of red fundus reflex.
- Smear of aqueous humour, vitreous humour can be taken for smear and culture testing. Antibiotic susceptibility testing can also be done.

Pharmacological treatment:

1. Treatment for Post operative Endophthalmitis:

-Intravitreal injection of antibiotics - Inj. vancomycin hydrochloride 1 mg in 0.1 ml plus Inj. ceftazidime 2 mg in 0.1 ml or Inj. amikacin sulfate 0.4 mg in 0.1 ml.

-Subconjunctival Inj. vancomycin 25 mg/0.5 ml plus Inj. ceftazidime 100 mg/0.5 ml plus dexamethasone 0.25 mg/0.5 ml.

-Vancomycin eye drops 50 mg/ml plus amikacin eye drops 15 mg/ml 1 drop every 6 hours.

-Homatropine 2% eye drops 3 times a day or atropine 1% eye ointment 2 times a day.

-Prednisolone acetate 1% eye drops or dexamethasone or betamethasone 0.1% eye drops every 6 hours.

-Tab. prednisolone 1 mg/kg/day in a single morning dose after 24 hours of antibiotic use and continue for 10-14 days 2 times a day for 5-10 days.

-Parenteral antibiotics are of questionable value and given only as a supportive therapy.

-Surgical treatment

Pars plana vitrectomy is indicated if visual acuity is limited to light perception or if there is poor response to above treatment in 30-36 hours.

Treatment for Traumatic Endophthalmitis:

-Hospitalize the patient and give immunization for tetanus.

-Inj. vancomycin 1 g IV infused over 1 hour, 12 hourly.

-Inj. gentamicin 2 mg/kg every 12 hour or Inj. ceftazidime 2 g IV every 12 hour or inj. ceftriaxone 2 g IV/day.

-Topical fortified eye drops, subconjunctival injection and intravitreal injection and cycloplegic drops as in cases of postoperative bacterial endophthalmitis.

Surgery

-Repair the ruptured eyeball at the earliest.

-Pars plana vitrectomy - indications are similar to that of postoperative bacterial endophthalmitis.

Treatment for Fungal Endophthalmitis:

Exogenous fungal infections may occur postoperatively or secondary to trauma. Endogenous bacterial endophthalmitis should be treated as an emergency treatment.

- Vitrectomy to debulk the vitreous of fungi.
- Intravitreal Inj. amphotericin B 5-10 mcg/0.1 ml or Inj. fluconazole 25 mcg/0.1 ml.
- Inj. amphotericin B 0.5-1.5 mg/kg/day slow infusion over 2-6 hours. (50 mg vial in powder form and is dissolved in 5% dextrose) for 10-14 days. Or
- Tab. fluconazole 400 mg loading dose followed by 200 mg daily, total dose should not exceed 600 mg/day. In children 12 mg/kg loading dose followed by 6 mg/kg/day. Or
- Tab. ketoconazole 200 mg orally 2 times a day or daily. In children above 2 years of age, 3.3-6.6 mg/kg/day.

-Homatropine 2% eye drops 4 times a day or atropine 1% eye ointment 2 times a day.

Patient education:

- All patients with open globe injury must contact an ophthalmologist after getting initial treatment.
- Cataract operated cases should never ignore pain, tearing and photophobia and decrease in vision in the operated eye and must consult the ophthalmologist at the earliest.

OPTIC NEURITIS

Optic neuritis includes inflammatory and demyelinating disorders of the optic nerve like papillitis (inflammation of the optic disc), retrobulbar neuritis (inflammation of retro-ocular portion of optic nerve) and neuroretinitis (when both optic nerve and retina are inflamed). Etiology may be hereditary, systemic bacterial/viral infections, endophthalmitis, orbital cellulitis, demyelinating disorders or idiopathic in nature.

Clinical features:

- Sudden, progressive and profound visual loss is the hallmark of acute optic neuritis.
- Decreased dark adaptation and impairment of colour vision, visual obscuration in bright light and episodic obscuration on physical exertion, hot bath, fatigue etc.
- Depth perception, particularly for the moving object may be impaired (Pulfrich's phenomenon). Patient may complain of mild dull eye ache. It is more marked in patients with
- retrobulbar neuritis than with papillitis. Pain is usually aggravated by ocular movements, especially in upward or downward directions due to attachment of some fibers of superior rectus to the dura mater.
- Marked abnormality in papillary responses to light reflex (sluggish or afferent pupil defect).
- Fundus examination reveals hyperaemia of the disc and blurring of the margins. Disc becomes oedematous and physiological cup is obliterated. Retinal veins are congested and
- tortuous. Splinter haemorrhages and fine exudates may be seen on the disc.
- Slit-lamp examination may reveal inflammatory cells in the vitreous. The most common field defect in optic neuritis is a relative central or centrocaecal scotoma.

Pharmacological treatment:

1. Efforts should be made to find out and treat the underlying cause. There is no effective treatment for idiopathic and hereditary optic neuritis and that associated with demyelinating disorders.
2. Corticosteroid therapy may shorten the period of visual loss, but will not influence the ultimate level of visual recovery in patients with optic neuritis.

Oral prednisolone therapy alone is contraindicated in the treatment of acute optic neuritis, since, it did not improve visual outcome and was associated with a significant increase in the risk of new attacks of optic neuritis.

- Indications for intravenous methylprednisolone in acute optic neuritis patients with a normal brain MRI scan are:

i. Visual loss in both eyes simultaneously or subsequently within hours or days of each other.
ii. When the only good eye is affected.
iii. When the slow progressive visual loss continues to occur.

3. Inj. methylprednisolone 1gm/day (or 15 mg/kg/day) intravenously in 2-4

divided doses for 3 days followed by Tab. prednisolone 1 mg/kg/day orally for 11 days tapered over the next week.

Patient education:

- Explain the recurrent nature of the disease and that permanent vision loss can occur.
- Avoid factors provoking transient visual obsurations like physical exertion, hot weather, stress, anger etc

DIABETIC RETINOPATHY

It is the microangiopathy of retinal vasculature occurring in long standing diabetes mellitus classified as non proliferative and proliferative. It is the sixth leading cause of blindness in the world. Risk factors include increased duration of diabetes, uncontrolled diabetes, abnormal lipid profile, smoking, renal disease etc.

Non- pharmacological treatment:

- Early diagnosis, proper diabetic control, regular follow up and fundus examination are important.
- In certain cases laser photocoagulation and vitrectomy surgery can help.

Pharmacological treatment:

No tested or proven pharmacological treatment exists which can delay, prevent or curdiabetic retinopathy.

Patient education:

- Explain the importance of yearly fundus examination. Laser treatment can prevent deterioration of vision but cannot correct existing visual defects.

RETINAL DETACHMENT

Retinal detachment (RD) is the separation of the sensory retina from retinal pigment epithelium. It may be localized or entire retina may be involved. Retinal detachment that includes the macula results in significant visual loss. The three types are Rhegmatogenous RD, exudative RD and tractional RD.

Clinical features:

- Rhegmatogenous RD is characterized by formation of a tear in the retina.

- Symptoms include flashes of light, presence of floaters in visual field and loss of central vision, if macula is involved.

The diagnosis is done by examination of the fundus with indirect ophthalmoscopy. The retina appears grey wit oscillating folds.

- Tractional RD is caused by gliotic bands on retina.
- Exudative RD is caused by collection of serous fluid between neurosensory retina and retinal pigment epithelium.

Non- pharmacological treatment:

- Treatment of choice is reattachment surgery involving identification of all retinal breaks areas of vitreous or retinal tractions and induction of aspectic chorioretinal inflammation around the breaks to seal them.
- Ensuring chorioretinal apposition by approximating choroid/retinal pigment epithelium to neurosensory retina by external tamponade, internal tamponade and drainage of subretinal fluid
- Adhesion between retinal pigment epithelium and retina can be achieved by cryotherapy, diathermy or photocoagulation.

Pharmacological treatment:

1. No pharmacological treatment which can prevent or cure rhegmatogenous RD.
2. Exudative RD due to inflammatory conditions like panuveitis can be treated with systemic corticosteroids as described in treatment of uveitis. The refractory patients can be treated with immunosuppresants.

Patient education:

- Patients with high myopia, family history of RD, post cataract surgery, past episodes of chorioretinal inflammation should be warned about the premonitory signs of impending RD (sudden onset of floaters, flashes of light and sudden obscuration of one part of visual field). In such cases, an opthalmologist should be contacted immediately.

VITAMIN A DEFICIENCY

The term xerophthalmia is now reserved (by a joint WHO and USAID Committee, 1976) to cover all the ocular manifestations of vitamin A deficiency, including not only the structural changes affecting the conjunctiva, cornea and

occasionally retina, but also the biophysical disorders of retinal rods and cones functions.

Clinical features:

- Vitamin A deficient diet is the main reason especially in young growing children of developing countries. It is usually associated with protein energy malnutrition.
- In some cases, decreased absorption of vitamin A due to gastro-intestinal disorders like lipid malabsorption or chronic alcoholism can occur.

Clinical Presentation can be as:

- Nyctalopia- night blindness is the earliest presenting symptom.
- Conjunctival xerosis
- Bitot spot: It is a metaplastic keratinisation of conjunctiva.
- Persistent epithelial defect (usually in chronic alcoholics) and corneal ulcer
- Keratomalacia: a diffuse corneal necrosis occurring in severe vitamin A deficiency.

Severe xerophthalmia is a medical emergency, so treatment should begin as early as possible. Treatment: Aimed at restoring vitamin A level to normal and addressing associated protein energy malnutrition.

1. Oral vitamin A:

0-6 month of age:50,000 IU (on 0 day, 1st day & after 4 weeks) 6-12 month of age: 1 lakh IU (on 0 day, 1st day & after 4 weeks)
More than 1 year of age : 2 lakh IU (on 0 day, 1st day & after 4 weeks)

2. **Intramuscular water** soluble vitamin A is given to children with persistent vomiting or severe malabsorption.
3. **Topical preservative free lubricating eye drops** and antibiotic drops (e.g. moxifloxacin eye drops 1 to 6 hourly) to prevent secondary infection can be given.

Patient education:

- Regular consumption of Vitamin A rich foods particularly fresh dark green leafy vegetables which constitute very rich and cheap sources.
- Pregnant women and lactating mothers should also consume Vitamin A rich diet regularly.
- Breast feeding including feeding of new born with rich colostrum.
- High dose universal distribution schedule for prevention of Vitamin A deficiency.

- Infants less than 6 months of age – 50,000 IU orally
- Infants 6-12 months of age - 100,000 IU orally.
- Children more than 12 months - 200,000 IU orally every 4-6 months till 5 years of

age.

Mothers - 200,000 IU orally within 8 weeks of delivery.

SENILE CATARACT

It is age related opacification of crystalline lens affecting persons of either sex usually above the age of 50 years. By 70 years, over 90% of the individuals develop senile cataract. The condition is usually bilateral, but almost always one eye is affected earlier than the other.

Clinical features:

- Gradual painless progressive diminution of vision in one or both eyes.
- Symptoms include glare, uniocular diplopia or polyopia, coloured haloes, distortion of images and misty vision and fixed black spots in visual field.
- Ocular examination reveals greyish white to white lenticular opacity on torch light examination which depends on the stage of cataract.
- Depending upon the location and maturation of cataract, the visual acuity may range from 6/9 to just perception of light.
- Slit-lamp examination should be performed with a fully-dilated pupil. The examination reveals complete morphology of opacity (site, size, shape, colour pattern and hardness of the nucleus).

Surgical treatment:

1. No medical management can halt the progression of cataract.
2. Surgery in the form of small incision cataract surgery or phacoemulsification is the preferred option.

Patient education:

- Do not wait for maturation of cataract for undergoing lens extraction.
- Secondary glaucoma or other complications can develop if total cataract remains unoperated for a long time.
- Visual rehabilitation in early post operative period is faster in small incision cataract surgery.

REFRACTIVE ERRORS

Refractive errors (ametropia) are the optical defects of eye in which the

parallel rays of light entering the eye do not come to focus on the fovea centralis. Ametropia includes myopia, hypermetropia and astigmatism.

Clinical features:

Hypermetropia (hyperopia) or long-sightedness is the refractive state of the eye wherein parallel rays of light coming from infinity are focused behind the retina with accommodation being at rest. Symptoms include astheopic symptoms (tiredness and watering of eyes, frontal headache) and defective vision, more for near vision.

Myopia or short-sightedness is a type of refractive error in which parallel rays of light coming from infinity are focused in front of the retina when accommodation is at rest.

Astigmatism - The optical power of the cornea in different planes is not equal. Parallel rays of light passing through these different planes are brought to different points of focus.

Surgical treatment:

1. No pharmacological treatment is available for ametropia.
2. Accurate retinoscopy and corrective spectacles or contact lens.
   Keratorefractive surgery

STRABISMUS (SQUINT)

Clinical features:

Any child presenting with strabismus should have the following conditions ruled out:

- Refractive error - refraction should be done under full cycloplegia i.e. Atropine Ointment 1% 3 times a day for 3 days prior to performing retinoscopy. If any refractive error is present, that should be fully corrected by spectacles for at least 3-6 months, before performing definitive surgical therapy for strabismus.
- Any opacity in the media e.g. cataract, corneal opacity, retinoblastoma etc.
- Amblyopia element whether induced by strabismus or vice versa should be treated with occlusion therapy or other modality before treating strabismus.

Surgical treatment:

1. Correct the refractive error or associated cataract, corneal opacity etc.
2. Fusion exercises for intermittent exotropia and other orthoptic exercises.
3. Definitive therapy is surgical realignment of axis once other associated features have been treated.

Patient education:

- Functional improvement in strabismus is best between 3-5 years of age. It is a misconception that squint is spontaneously corrected as the child grows.

COMPUTER VISION SYNDROME

Computer Vision Syndrome (CVS) is the complex of eye and vision problems related to near work which are experienced during or related to computer use.

Clinical features:

- Characterized by visual symptoms which result from interaction with a computer display or its environment.
- In most cases, symptoms occur because the visual demands of the task exceed the visual abilities of the individual to comfortably perform the task.
- Contributing factors: Decreased blinking reflex, prolonged near focusing efforts, repeated head posture change/fixation in a wrong posture, environmental factors of computer workstations like contrast and resolution of the display, viewing distances and angles, room lighting, sustained viewing etc.
- Symptoms include tiredness of eyes, headache, blurred near and distant vision, dry or irritated eyes, neck pain and/or backaches, diplopia (double vision), difficulty in re- focusing the eyes.
- Diagnostic tests include tear film break-up time and Schirmer test. Cervical spondylitis, anxiety and migraine are the differential diagnosis.

Non- pharmacological treatment:

Counselling regarding the syndrome and work environment modification Steps for relieving computer eye strain

- Get a computerized eye exam before start using computer. Repeat once a year.
- Use proper lighting. Eliminate exterior light by closing drapes, shades or blinds. Reduce interior lighting by using lower intensity bulbs and tubes.
- Minimize glare: To install an anti-glare screen on your monitor. Paint bright white walls a darker color with a matt finish.

- Upgrade your display. Use LCD monitor instead of a CRT monitor.
- Adjust the brightness and contrast of your computer screen.
- Blink more often.
- Exercise your eyes: Follow 20-20-20 rule i.e. after every 20 minutes, look at 20 feet distance for 20 seconds.
- Take frequent breaks - two 15-minute breaks -four additional five-minute "minibreaks" throughout the work day (6-8 hrs).
- Modify your workstation. Proper posture during computer work. Position computer screen 20 to 24 inches from your eyes. The center of your screen should be about 10 to 15 degrees below your eyes. Top of the screen tilted back slightly (10-20 degree) away from the operator.
- Consider customized eyeglasses specific for use during work on a computer screen. Anti- reflective coating in the lenses should be used and avoid contact lens use during computer work.

Pharmacological treatment:

1. Tear substitute should be considered if symptoms are aggravated during computer work. Commercially available tear substitutes include sodium carboxymethylcellulose and hydroxypropyl methylcellulose.

RADIODIAGNOSIS STANDARD TREATMENT GUIDELINES

STANDARD OPERATIVE PROCEDURE FOR RADIODIAGNOSIS NON-VASCULAR INTERVENTIONAL RADIOLOGY:

INTRODUCTION

Department of Radio-diagnosis provides comprehensive imaging solutions in the area of conventional radiology, cross-sectional imaging and emergency radiology services.

Radio-diagnosis department is located in the basement. All the equipments are located at functionally independent rooms and operated by qualified and trained technicians and doctors.

Standard Operating Procedure for Intervention Procedures In The Department of Radio- diagnosis

After due consultation with faculty members of the department, following protocol is being laid down as Standard Operating Procedure as regards Intervention Procedures (Ultrasound/CT guided procedures- diagnostic and therapeutic)

Inpatient and Outpatients are required to go to the Radiology Reception with the requisition slip filled by the referral doctors. The Radiographer / Receptionist should show the requisition slip to the Radiologist in-charge for screening of the patient and allocation of appointment dates.

At the time of giving the appointment, fitness of the patient is determined by overall general condition and checking serum Hb, platelet, PT-INR levels which is done within 1 month prior to the procedure. If the patient meets the fitness criteria, they are given appointment dates as per availability.

The procedures are to be undertaken by appointment and during working hours only. The procedures will be performed between 9:00am to 3:00pm only so as the sample to reach Pathology department before closure of their working hours.

The procedures will be performed under supervision and guidance of the Radiologists who is experienced and well conversant with the technique.

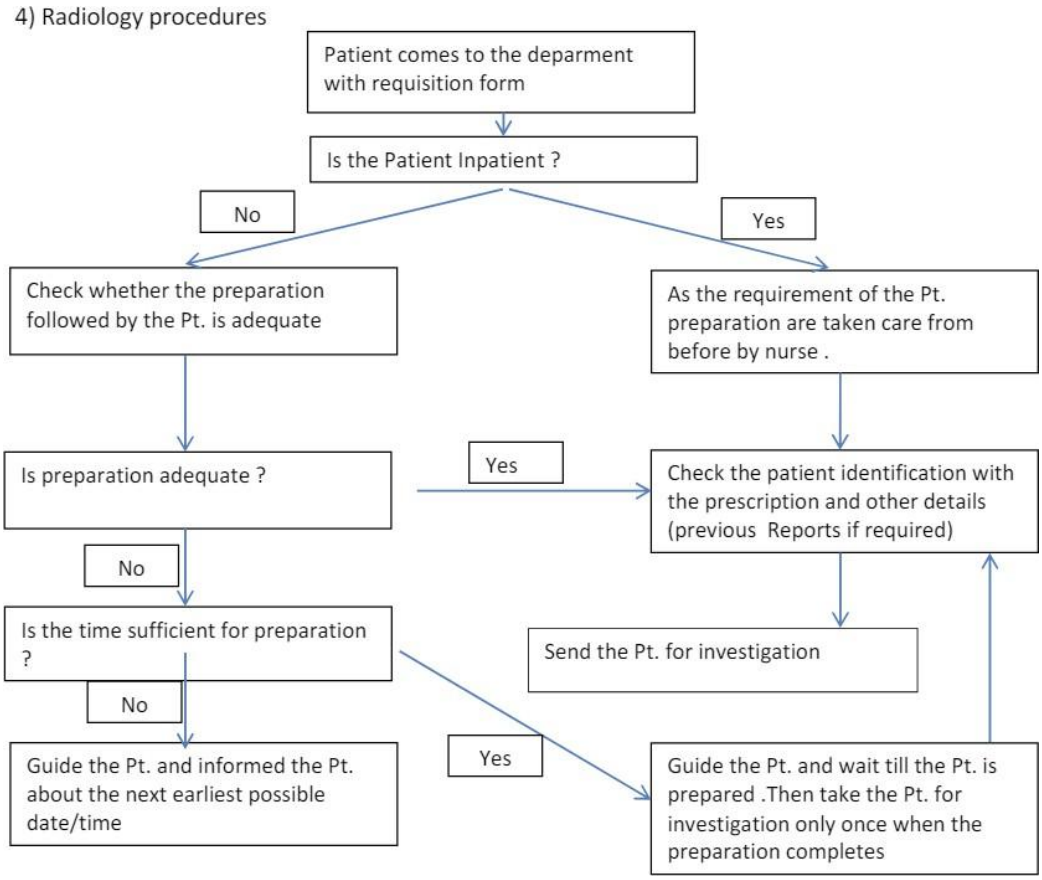
At the time of appointment, on duty JR/SR of Referring Department and Nursing Staff will assist the procedures after obtaining due consent.

A nursing assistant must be present during the procedure to assist the doctors. It will be his/her duty to keep emergency tray with all the necessary drugs ready. It is also imperative on the part of nursing staff on duty to keep emergency tray ready and see for expiry of drugs, on daily basis, which need to be discarded if expired.

The referring physicians/surgeons are requested to send their doctor-JR/SR who is competent to manage any complication which may arise during the procedure in the interest of patient care. It will be their duty to ensure that the sample obtained after the procedure, which is earmarked for pathological examination reaches immediately after the procedure for onward transmission.

The Radiologist will write procedural notes on a separate sheet of paper in duplicate and retain its copy in the department for record.

The referring the Department JR/SR will be responsible for the post-procedural follow-up and keep faculty member informed.



Pre-procedure preparation:

- Informed written consent.
- Coagulation profile to be checked.
- Blood group.

- Nil per oral for 4Hrs. prior to the procedure.
- Responsible Bystander/ Relative should accompany the patient to the department.

Post-procedure:

- IV cannula to be introduced on All Post-procedural care.
- Vital signs to be monitored for at least 3 hrs after the procedure.
- Additional X-ray/CT scan/ultrasound to be repeated if required after the procedure.

CONTRAST REACTION MANAGEMENT:

Radiopaque contrast agents are often used in radiography and fluoroscopy to help delineate borders between tissues with similar radiodensity. Most contrast agents are iodine based.

Iodinated contrast agents may be

- Ionic
- Nonionic

Ionic contrast agents, which are salts, are hyperosmolar to blood. These agents should not be used for myelography or in injections that may enter the spinal canal (because neurotoxicity is a risk) or the bronchial tree (because pulmonary edema is a risk).

Non-ionic contrast agents are low-osmolar (but still hyperosmolar relative to blood) or iso-osmolar (with the same osmolarity as blood). Newer nonionic contrast agents are now routinely used at nearly all institutions because they have fewer adverse effects.

The most serious contrast reactions are

- Allergic-type reactions
- Contrast nephropathy (renal damage after intravascular injection of a contrast agent)

Allergic-type contrast reactions

Reactions vary in severity:

- Mild (eg, cough, itching, nasal congestion)
- Moderate (eg, dyspnea, wheezing, slight changes in pulse or blood pressure)
- Severe (eg, respiratory distress, arrhythmias such as bradycardia, seizures, shock, cardiopulmonary arrest)

The mechanism is anaphylactoid; risk factors include the following:

- A previous reaction to injected contrast agents
- Asthma/Allergies

Treatment begins by stopping contrast infusion.

1. For **mild or moderate reactions,** diphenhydramine 25 to 50 mg IV is usually effective.
2. For **severe reactions,** treatment depends on the type of reaction and may include oxygen, epinephrine, IV fluids, and possibly atropine (for bradycardia).

In patients at high risk of contrast reactions, imaging tests that do not require iodinated contrast should be used. If contrast is necessary, a nonionic agent should be used, and patients should be premedicated with prednisone (50 mg orally 12 hours, 7 hours, and 1 hour before injection of contrast) and diphenhydramine (50 mg IV, IM, or orally 1 hour before contrast administration).

If patients require imaging immediately, they can be given diphenhydramine 50 mg IV, IM, or orally 1 hour before injection of contrast and hydrocortisone 200 mg IV every 4 hours until the study is performed, preferably deferring imaging, if possible, until at least 2 doses of hydrocortisone have been administered.

RESPIRATORY MEDICINE STANDARD TREATMENT GU-IDELINES

RESPIRATORY

ASTHMA

Definition:

It is defined by the history of respiratory symptoms, such as wheeze, shortness of breath, chest tightness and cough, that vary over time and in intensity, together with variable expiratory airflow limitation.

Symptoms:

Wheeze, shortness of breath, cough and/or chest tightness.

- Patients (especially adults) experience more than one of these types of symptoms.
- Symptoms are often worse at night or in the early morning.
- Symptoms vary over time and in intensity.
- Symptoms are triggered by viral infections (colds), exercise, allergen exposure, changes in weather, laughter or irritants such as car exhaust fumes, smoke or strong smells.

Diagnostic criteria:

- History of wheeze, shortness of breath, chest tightness and cough
- Post bronchodilator responsiveness – Increase in FEV1 > 12% or > 200ml

Treatment:

- **Using ICS-formoterol as the Reliever and Maintenance therapy:**

Symptoms	Treatment
Symptoms < 4-5 days a week	As needed low dose ICS-formoterol
Symptoms most days or waking with asthma once a week or more	Low dose maintenance ICS-formoterol
Daily symptoms or waking with asthma once a week or more and low lung function	Medium dose maintenance ICS-formoterol

Severe asthma not controlled with ICS-formoterol	Add on LAMA High dose ICS-formoterol Refer to Pulmonologist for phenotypic assessment and biologic therapy
RELIEVER: As needed low dose ICS-formoterol	

- **Alternative controller and reliever:**

Symptoms	**Treatment**
Symptoms < twice a month	Take ICS whenever SABA taken
Symptoms > twice a month but < 4-5 days a week	Low dose maintenance ICS
Symptoms most days or waking with asthma once a week or more	Low dose maintenance ICS-LABA
Daily symptoms or waking with asthma once a week or more with low lung function	Medium low dose maintenance ICS-LABA
Severe asthma	Add on LAMA High dose ICS-formoterol Refer to Pulmonologist for phenotypic assessment & biologic therapy
RELIEVER: As needed SABA or as needed ICS-SABA	

- **Vaccination:**
  a) Influenza vaccination

- **Non-pharmacological treatment:**
  a) Smoking cessation
  b) Physical activity
  c) Avoidance of occupational or domestic exposures to allergens or irritants
  d) Avoidance of medications that may make asthma worse
  e) Healthy diet
  f) Avoidance of indoor/ outdoor allergens
  g) Weight reduction
  h) Breathing exercises

INDICATIONS FOR REFERRAL

- Difficulty confirming the diagnosis of asthma
- Persistent or severely uncontrolled asthma or frequent exacerbations
- Any risk factors for asthma-related death
- Evidence of or risk of significant treatment side-effects

ASTHMA EXACERBATIONS:

Episodes characterized by a progressive increase in symptoms of shortness of breath, cough, wheezing or chest tightness and progressive decrease in lung function, i.e. they represent a change from the patient's usual status that is sufficient to require a change in treatment

Management of exacerbations:

Classify exacerbation into:

Mild or moderate:

- Talk in phrases
- Prefers sitting to lying
- Respiratory rate increased
- Accessory muscles of respiration not in used
- PR: 100-120bpm
- SPO2: 90-95% at room air

Severe:

- Talks in words
- Sits hunched forward
- Agitated
- Respiratory rate > 30/min
- Accessory muscles of respiration in used
- Pulse rate > 120bpm
- SPO2< 90% at room air

Life threatening exacerbation:

- Drowsy
- Confused

- Silent chest

Treatment:

Mild/moderate exacerbation:

- SABA (Levosalbutamol) 4-10puffs stat and repeat every 20 minutes for 1 hour
- Prednisolone: 40-50mg
- O_2 inhalation – target SPO_2: 93 – 95%
- Review response at 1 hour:
  a) If symptoms improve and SPO_2 > 94% at room air – advice discharge with reliever medication and continue prednisolone for 5-7 days
  b) If symptoms worsen – Add ipratropium bromide, O_2 inhalation and systemic corticosteroids and refer to higher centre

Severe/ life threatening exacerbation:

- SABA + SAMA (Duolin, Salbair I, Combimist L)
- O_2 inhalation
- Systemic corticosteroids
- Refer to Higher Centre

PLEURAL EFFUSION

Approach to Pleural Effusion

Indications for thoracocentesis

- Diagnostic (new effusion of unclear etiology) for pleural effusion
- In patients with CHF the following features should prompt thorcocentesis
  1. Features suggestive of an alternate etilogy - eg BL effusion of significantly disparate sizes
  2. Features suggestive of Pleurisy or fever
  3. Features suggestive of infection or cancer
  4. Echocardiogram not consistent with CHF
  5. Disproportionately wide alveolar arterial O2 gradient
  6. Lack of resolution with effective CHF therapy

- Therapeutic (symptom relief)
- Prevention of complications

Contraindications for thoracocentesis

- Insufficient pleural fluid
- Skin infection or wound at needle insertion site
- Severe bleeding diathesis

Following thoracocentesis, the following biomarkers should be sent for most pleural effusions

- Cell counts and differential counts
- Total protein
- ADA
- LDH
- Glucose
- Culture
- Grams stain
- Cholesterol
- AFB stain/ CBNAAT
- Malignant cells

Concurrent testing from serum for the following is required

- Total protein
- LDH
- RBC counts in suspected hemothorax
- Amylase (rupture of esophagus)
- Creatinine (urinothorax)
- Bilirubin (bilothorax)

The following gross observations may be helpful

Colour of fluid	
Pale yellow	Transudates, some exudates
Red (bloody)	Malignancy, pulmonary infarct, postcardiac injury syndrome, trauma
White (milky)	Chylothorax or cholesterol effusion
Brown	Long standing effusion, ruptiure f amoebic abscess
Black	Aspergillus spp. Pancreaticopleural fistula, crack cocaine use, bronchogenic adenocarcinoma, chronic hemothorax
Yellow green	Rheumatoid pleurisy
Dark green	Bilothorax
Character of fluid	
Pus	Empyemna
Viscous	Mesothelioma
Debris	Rheumatoid pleurisy
Turbid	Inflammatory exudate
Anchovy paste	Amoebic liver abscess
Odour of fluid	
Putrid	Anaerobic empyema
Ammonia	Urinothorax

Classify pleural fluid into transudates and exudates on the basis of Light's criteria

- Pleural fluid to serum ration > 0.5
- Pleural fluid to serum LDH ratio > 0.6
- Pleural fluid LDH >).67 i.e >2/3rd the upper limit of normal serum LDH

In cases of

1. suspected heart failure related pleural effusion on diuretics, 25 – 30% may be misclassified as exudates by Light's criteria. In such cases, measure
    - Albumin or total protein gradient
    - Pleural fluid or serum NT-proBNP
2. Suspected esophageal or pancreatic related effusion
    - Salivary amylase
    - Pleural fluid pH
3. Suspected chylous or cholesterol effusion
    - Cholesterol and triglyceride levels from plural effusion
    - Rarely, lipoprotein analysis for chylomicrons
4. Suspected hemothorax
    - Perform hematocrit on fresh sample of pleural fluid and compare with blood hematocrit
5. Suspected urinothorax
    - Compare pleural fluid and serum creatinine
6. Suspected ventriculoperitoneal shunt related effusion
    - Beta 2 transferrin
7. Suspected glycinothorax
    - Compare pleural fluid and serum glycine
8. Suspected bilothorax
    - Compare pleural fluid and serum bilirubin

In case of unclear etiology, the following steps should be taken

- Retake history and examination
- Reanalyze pleural fluid
- Consider additional imaging like PET/CT etc If

etiology is still unclear

- Do a pleural biopsy
    - Diffuse pleural involvement : closed pleural biopsy
    - Pleural mass or thickening : Image guided pleural biopsy
    - Patchy pleural involvement : thoracoscopic pleural biopsy

Approach to Pulmonary Embolism

The following signs and symptoms may be present in a patient with pulmonary embolism

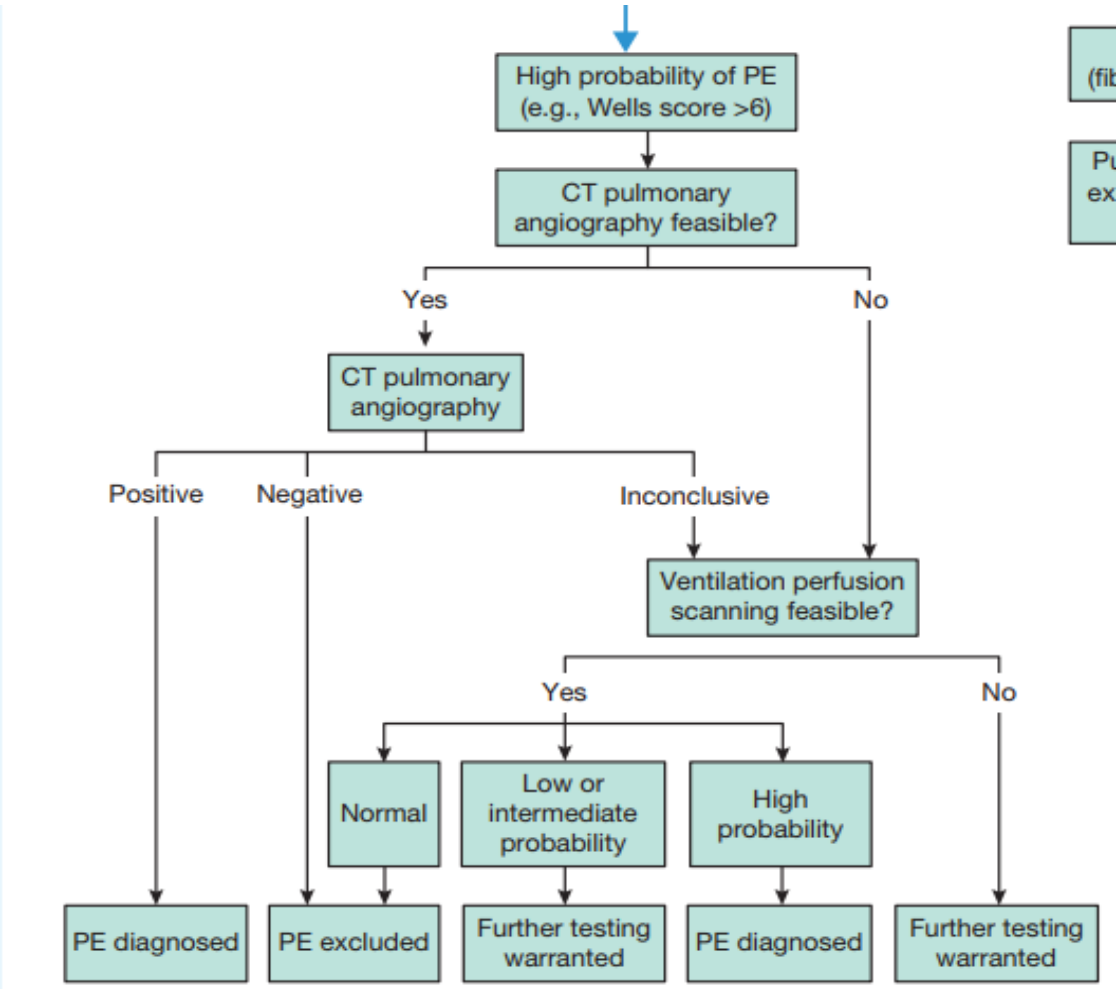
1. Sudden onset dyspnea - over minutes to hours and in approx occurs over days (most common)
2. Pleuritic chest pain
3. Cough
4. Leg swelling or pain
5. Hemoptysis
6. Unexplained tachypnea with RR > 20/min is found in approx 70%
7. Rales on chest auscultation
8. Tachycardia
9. Increased pulmonic component of second heart sound
10. Fever

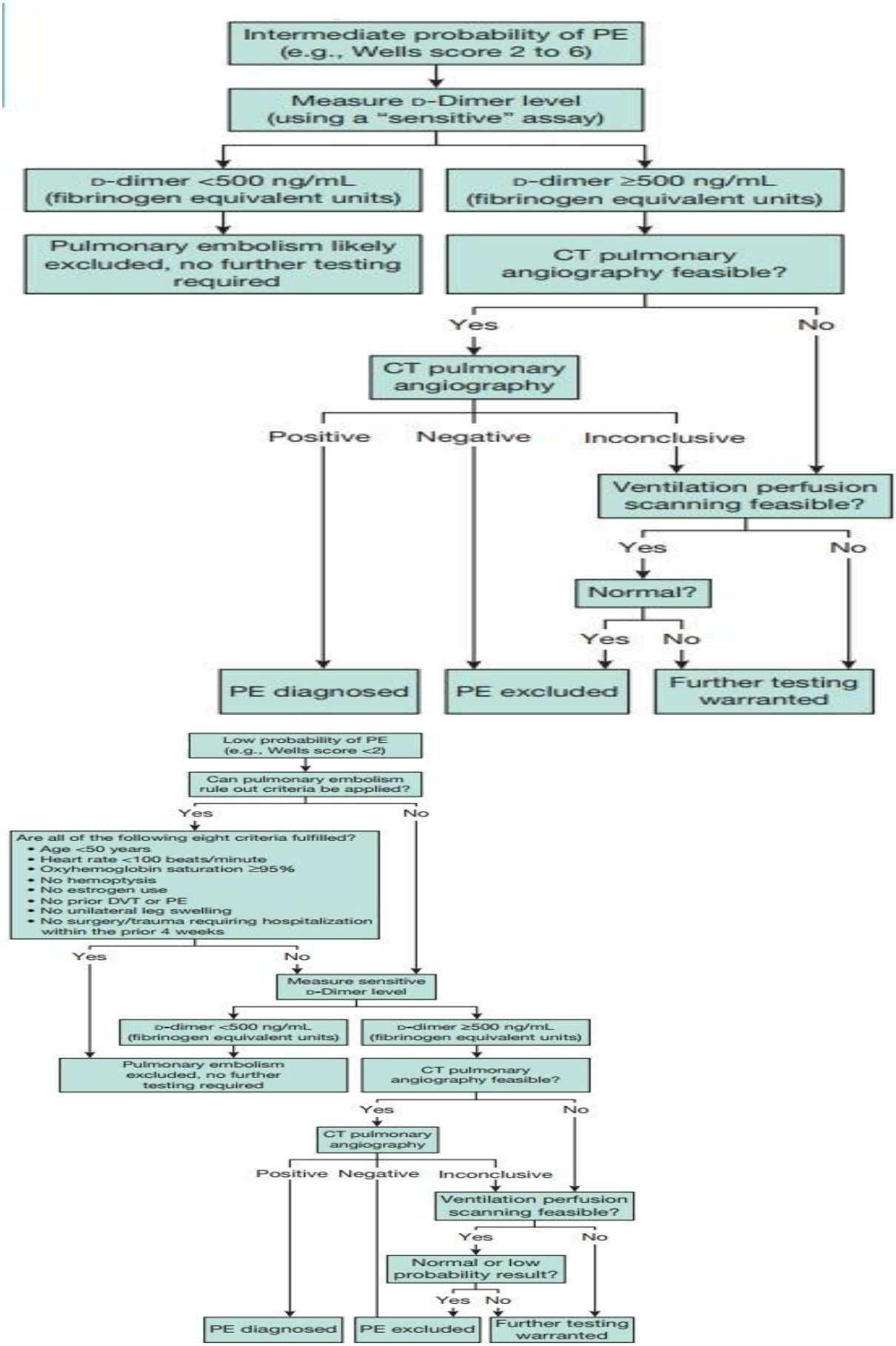
- Classify risk using Well's clinical prediction score

Variable	Points
DVT signs and symptoms	3
PE likely or more likely than alternative diagnosis	3
HR > 100	1.5
Immobilization/ surgery previous 4 weeks	1.5
Previous DVT or PE	1.5
Hemoptysis	1
Malignancy	1

Total Score	Pretest probablilty of PE
<2	Low
2 – 6	Moderate
>6	High
Dichotomized score	
≤ 4	PE unlikely
> 4	PE likely

- Use the following diagrams for further evaluation




Intermediate probability of PE (e.g., Wells score 2 to 6)
Measure D-Dimer level (using a "sensitive" assay)
D-dimer <500 ng/mL (fibrinogen equivalent units)
D-dimer ≥500 ng/mL (fibrinogen equivalent units)
Pulmonary embolism likely excluded, no further testing required
CT pulmonary angiography feasible?
Yes
No
CT pulmonary angiography
Positive
Negative
Inconclusive
Ventilation perfusion scanning feasible?
Yes
No
Normal?
Yes
No
PE diagnosed
PE excluded
Further testing warranted
Low probability of PE (e.g., Wells score <2)
Can pulmonary embolism rule out criteria be applied?
Yes
No
Are all of the following eight criteria fulfilled?
• Age <50 years
• Heart rate <100 beats/minute
• Oxyhemoglobin saturation ≥95%
• No hemoptysis
• No estrogen use
• No prior DVT or PE
• No unilateral leg swelling
• No surgery/trauma requiring hospitalization within the prior 4 weeks
Yes
No
Measure sensitive D-Dimer level
D-dimer <500 ng/mL (fibrinogen equivalent units)
D-dimer ≥500 ng/mL (fibrinogen equivalent units)
Pulmonary embolism excluded, no further testing required
CT pulmonary angiography feasible?
Yes
No
CT pulmonary angiography
Positive
Negative
Inconclusive
Ventilation perfusion scanning feasible?
Yes
No
Normal or low probability result?
Yes
No
PE diagnosed
PE excluded
Further testing warranted

- Treatment of hemodynamically stable patients
    - For those in whom risk of bleeding is low, anticoagulant therapy is indicated
    - For those with contraindications to anticoagulation or those with high risk of bleeding placement of Inferior vena cava filter is indicated
        - Thrombolytic therapy is not recommended
- Treatment of hemodynamically unstable patients
    - Thrombolytic therapy is indicated in most patients, provided there is no contraindication
    - Embolectomy is appropriate in whom thrombolysis is not possible or failed
- Initial anticoagulation
    - To be administered as soon as possible to quickly achieve therapeutic anticoagulation
- Long term anticoagulation
    - All patients are anticoagulated for a minimum of 3 months

Factors that influence agent selection for anticoagulation in patients with acute venous thromboembolism

Factor	Preferred anticoagulant	Qualifying remarks
Cancer	LMWH, factor Xa inhibitors	More so if: Just diagnosed, extensive VTE, metastatic cancer, very symptomatic; vomiting; on cancer chemotherapy.
Initial parenteral therapy to be avoided	Rivaroxaban; apixaban	VKA, dabigatran, and edoxaban require initial parenteral therapy.
Once daily oral therapy preferred	Rivaroxaban; edoxaban; VKA	
Liver disease and coagulopathy	LMWH	DOACs contraindicated if INR raised because of liver disease; VKA difficult to control and INR may not reflect antithrombotic effect.
Renal disease and creatinine clearance <30 mL/min	VKA	DOACs and LMWH contraindicated with severe renal impairment. However, dosing of some DOACs can be renally adjusted, although adjustment varies with different levels of renal impairment depending on the DOAC.
Coronary artery disease	VKA, rivaroxaban, apixaban, edoxaban	Coronary artery events appear to occur more often with dabigatran than with VKA. This has not been seen with the other DOACs, and they have demonstrated efficacy for coronary artery disease. Antiplatelet therapy should be avoided if possible in patients on anticoagulants because of increased bleeding.
Dyspepsia or history of GI bleeding	VKA, apixaban	Dabigatran increased dyspepsia. Dabigatran, rivaroxaban, and edoxaban may be associated with more GI bleeding than VKA.
Poor compliance	VKA	INR monitoring can help to detect problems. However, some patients may be more compliant with a DOAC because it is less complex.
Thrombolytic therapy use	UFH infusion	Greater experience with its use in patients treated with thrombolytic therapy.
Reversal agent needed	VKA, UFH, DOACs	Reversal agents for DOACs may not be universally readily available.
Pregnancy or pregnancy risk	LMWH	Potential for other agents to cross the placenta.
Cost, coverage, licensing	Varies among regions and with individual circumstances	

LMWH: low molecular weight heparin; VTE: venous thromboembolism; VKA: vitamin K-dependent antagonist (ie, warfarin); DOACs: direct oral anticoagulants; INR: international normalized ratio; GI: gastrointestinal; UFH: unfractionated heparin.

STG OF HOSPIRAL-ACQUIRED AND VENTILATOR-ASSOSCIATED PNEUMONIA

Hospital-acquired (or nosocomial) pneumonia (HAP) and ventilator-associated pneumonia (VAP) remain important causes of morbidity and mortality despite improvements in prevention, antimicrobial therapy, and supportive care

Pneumonia types — Pneumonia is frequently categorized based on site of acquisition

- **Hospital-acquired (or nosocomial) pneumonia (HAP)** is pneumonia that occurs 48 hours or more after admission to the hospital and did not appear to be incubating at the time of admission.
- **Ventilator-associated pneumonia (VAP)** is a type of HAP that develops in intubated patients on mechanical ventilation for more than 48 hours. VAP also includes HAP that occurs within 48 hours of extubation.
- **Non-ventilator-associated HAP (NV-HAP)** refers to HAP that develops in hospitalized patients who are not on mechanical ventilation nor underwent extubation within 48 hours before pneumonia developed. NV-HAP can be divided into patients that ultimately require mechanical ventilation (VHAP) due to the pneumonia versus those that do not. VHAP is associated with particularly poor clinical outcomes.

➢ PATHOGENESIS:

The pathogenesis of HAP and VAP is related to the number and virulence of micro-organisms entering the lower respiratory tract and the response of the host (eg, mechanical, humoral, and cellular host defenses).

The primary route of infection of the lungs is through microaspiration of organisms that have colonized the oropharyngeal tract (or, to a lesser extent, the gastrointestinal tract) . Approx 45 percent of healthy subjects aspirate during sleep and an even higher proportion of severely ill patients aspirate routinely. Although frequently regarded as partially protective, the presence of an endotracheal tube facilitates aspiration of oropharyngeal secretions and bacteria into the lungs.

Hospitalized patients often become colonized with microorganisms acquired from the hospital environment, and as many as 75 percent of severely ill patients will be colonized within 48 hours.

An additional mechanism of inoculation in mechanically ventilated patients is direct contact with environmental reservoirs, including respiratory devices and

contaminated water reservoirs. Disposable tubing used in respiratory circuits or tracheostomy or endotracheal tubes may become contaminated in the process of routine nursing care or via the (contaminated) hands of hospital personnel. Such contamination can occur despite rigorous cleaning of ventilator equipment.

In addition, the near sterility of the stomach and upper gastrointestinal tract may be disrupted by alterations in gastric pH due to illness, medications, or enteric feedings. For this reason, much attention has been paid to the possible adverse effect of ulcer prophylaxis regimens that raise the gastric pH.

Less frequently, pneumonia results from inhalation of infectious aerosols or from bacteremia originating in a distant focus.

➢ MICROBIOLOGY:

The most common organisms are *Staphylococcus aureus* (including methicillin-resistant *S. aureus*) and *Pseudomonas aeruginosa*.

Other common causes - aerobic gram-negative bacilli (eg, *Escherichia coli*, *Klebsiella pneumoniae*, *Enterobacter* spp, *Acinetobacter* spp) and gram-positive cocci (eg, *Streptococcus* spp).

Also may be due to viruses in general medical and surgical patients and both viruses and fungi in immunocompromised patients.

➢ RISK FACTORS:

The most significant risk factor for HAP is intubation. Other risk factors include:

- Older age
- Chronic lung disease
- Depressed consciousness
- Aspiration
- Chest or upper abdominal surgery
- Agents that increase gastric pH (H_2 blockers, antacids, proton pump inhibitors [PPIs])
- Previous antibiotic exposure, especially broad spectrum
- Reintubation or prolonged intubation
- Mechanical ventilation for acute respiratory distress syndrome
- Frequent ventilator circuit changes
- Total opioid exposure
- Multiple trauma

- Paralysis
- Number of central venous catheter placements and surgeries
- Use of muscle relaxants or glucocorticoids
- The presence of an intracranial pressure monitor
- Malnutrition, chronic renal failure, anemia, Charlson Comorbidity Index, previous hospitalization

Role of gastric pH — *There is an increased incidence of HAP when the gastric pH is increased with the use of H_2 blockers, antacids, or PPIs. Avoid agents that raise gastric pH in patients who are not at high risk of developing a stress ulcer or stress gastritis.*

➢ DIAGNOSIS :

The clinical diagnosis of HAP and VAP is difficult in part because the clinical findings are nonspecific. The 2016 IDSA/ATS guidelines for the management of HAP and VAP recommend a clinical diagnosis based upon a new lung infiltrate plus clinical evidence that the infiltrate is of infectious origin, which includes the new onset of fever, purulent sputum, leukocytosis, and decline in oxygenation.

While the clinical features described above support the diagnosis of HAP or VAP, no individual sign or symptoms nor any combination of signs and symptoms have been found to be highly sensitive or specific for diagnosis. *As an example, the presence of a new or progressive radiographic infiltrate plus at least two of three clinical features (fever >38ºC, leukocytosis or leukopenia, and purulent secretions) has a 69 percent sensitivity and 75 percent specificity for VAP.*

Cultures of pulmonary secretions (sputum, endotracheal aspirates, bronchoalveolar lavage) are also prone to false positives and false negatives. When compared with histology, quantitative endotracheal aspirate cultures had a pooled sensitivity of 48 percent and positive predictive value of 81 percent ; quantitative bronchoalveolar lavage cultures had a sensitivity of 75 percent and positive predictive value of 77 percent.

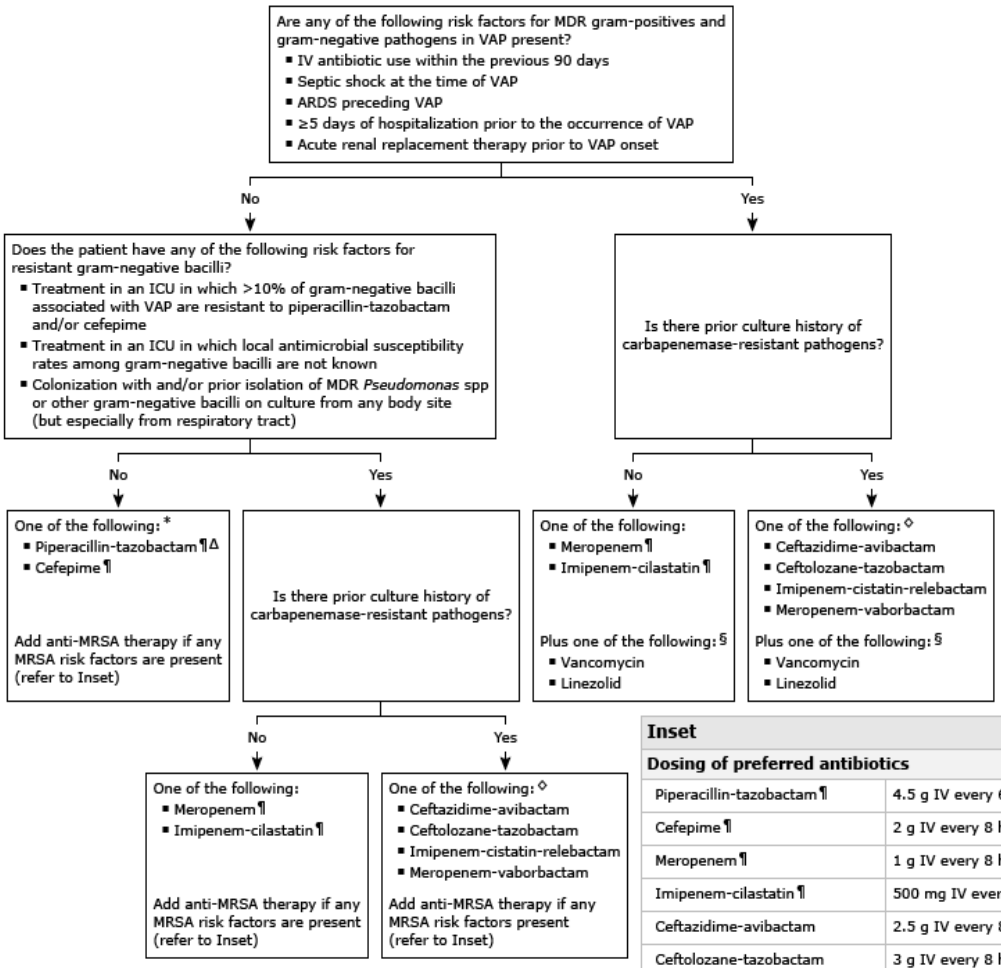
➢ TREATMENT :

EMPIRIC THERAPY-

Timing of antibiotics — Once nvHAP or VAP is suspected clinically, microbiological specimens should be obtained as soon as possible in all patients. In patients with signs of septic shock or rapidly progressive organ dysfunction,

antimicrobial therapy should be started as soon as possible. If the diagnosis is uncertain and the patient is not in sepsis or septic shock, then it appears to be safe and potentially beneficial to gather more data and await culture results before treating.

Empiric treatment of ventilator-associated pneumonia (VAP) in adults with normal kidney function



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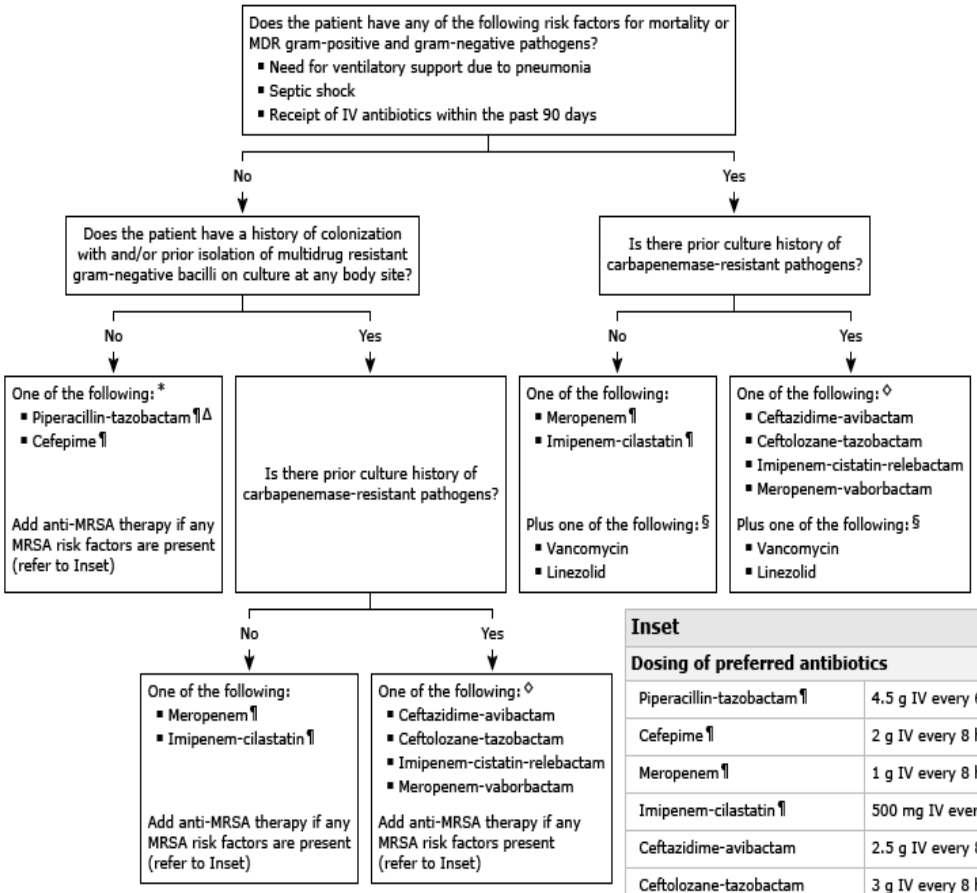
Dosing of preferred antibiotics	
Piperacillin-tazobactam ¶	4.5 g IV every 6 hours
Cefepime ¶	2 g IV every 8 hours
Meropenem ¶	1 g IV every 8 hours
Imipenem-cilastatin ¶	500 mg IV every 6 hours
Ceftazidime-avibactam	2.5 g IV every 8 hours
Ceftolozane-tazobactam	3 g IV every 8 hours
Imipenem-cilastatin-relebactam	1.25 g IV every 6 hours
Meropenem-vaborbactam	4 g IV every 8 hours
Add anti-MRSA therapy if patient has one of the following risk factors for MRSA: ▪ Treatment in a unit in which >10 to 20% of *S. aureus* isolates associated with VAP are methicillin resistant ▪ Treatment in a unit in which the prevalence of MRSA is not known ▪ Colonization with and/or prior isolation of MRSA on culture from any body site (but especially the respiratory tract)	
Anti-MRSA therapy consists of one of the following: §	
Vancomycin	Generally 15 to 20 mg/kg every 8 to 12 hours for most patients with normal kidney function. Interval adjustments should be based on AUC-guided (preferred) or trough-guided serum concentration monitoring. The vancomycin loading dose is based on actual body weight; the dose is rounded to the nearest 250 mg increment and not exceeding 2000 mg. Within this range, we use a higher dose for critically ill patients.
Linezolid	600 mg IV every 12 hours

Empiric treatment of nonventilator hospital-associated pneumonia in adults with normal kidney function



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Imipenem-cilastatin-relebactam	1.25 g IV every 6 hours
Meropenem-vaborbactam	4 g IV every 8 hours
Add anti-MRSA therapy if patient has one of the following risk factors for MRSA: ▪ Treatment in a unit in which >10 to 20% of *S. aureus* isolates associated with VAP are methicillin resistant ▪ Treatment in a unit in which the prevalence of MRSA is not known ▪ Colonization with and/or prior isolation of MRSA on culture from any body site (but especially the respiratory tract)	
Anti-MRSA therapy consists of one of the following: §	
Vancomycin	Generally 15 to 20 mg/kg every 8 to 12 hours for most patients with normal kidney function. Interval adjustments should be based on AUC-guided (preferred) or trough-guided serum concentration monitoring. The vancomycin loading dose is based on actual body weight; the dose is rounded to the nearest 250 mg increment and not exceeding 2000 mg. Within this range, we use a higher dose for critically ill patients.
Linezolid	600 mg IV every 12 hours

Regimens — Dosing will need to be adjusted in those with renal dysfunction. When available, choice of antibiotics should be guided based on local antibiogram resistance rates.

- **No risk factors present** — suggest a regimen that has activity against *Pseudomonas*, other gram-negative bacilli, and methicillin- susceptible *S. aureus* (MSSA). Preferred intravenous empiric antibiotic regimens include one of the following:
-
  - *Piperacillin-tazobactam 4.5 g IV every 6 hours*
  - *Cefepime 2 g IV every 8 hours*

However, levofloxacin 750 mg IV daily may be preferred if there is a high suspicion for *Legionella* spp infection and local resistance rates of *S. aureus*, *P. aeruginosa*, and other gram-negative bacilli to fluoroquinolones are low . The ATS/IDSA guidelines also include imipenem and meropenem as options, but we generally reserve these agents for patients with a high likelihood of infection with an extended-spectrum beta-lactamase (ESBL)-producing gram-negative bacillus or for patients in units where local antibiograms favor these agents over other broad-spectrum beta-lactams.

- **Risk factors for MDR gram-negative bacilli**

No history of carbapenem-resistant pathogens — suggest **one of the following :**

- *Meropenem 1 g IV every eight hours*
- *Imipenem 500 mg IV every six hours*

Patients at risk of seizures from imipenem (eg, renal insufficiency, underlying central nervous system [CNS] disease) should be closely monitored or alternative beta-lactams should be used.

History of carbapenem-resistant pathogens — suggest empiric monotherapy with one of the following agents:

- *Ceftazidime-avibactam 2.5 g IV every eight hours*
- *Ceftolozane-tazobactam 3 g IV every eight hours*
- *Imipenem-cilastatin-relebactam 1.5 g IV every six hours*
- *Meropenem-vaborbactam 4 g every eight hours*

If the beta-lactam beta-lactamase agents listed above are not available and there is a high suspicion for carbapenem-resistant gram-negative bacilli, then the addition of one of the following agents **along with** a carbapenem is a reasonable alternative:

- *Tobramycin 5 to 7 mg/kg IV daily*
- *Colistin 300mg colistin base activity (CBA) loading dose followed by 150 mg CBA every 12 hours*
- *Aztreonam 2 g IV every eight hours*
- *Levofloxacin 750 mg IV or orally daily*
- *Ciprofloxacin 750 mg IV every 12 hours or 400mg orally every eight hours*

- **Risk factors for MRSA** — recommend either linezolid or vancomycin for infections suspected or proven to be due to MRSA .

Preferred agents — For patients with VAP or nvHAP who have risk factors for MRSA, we suggest adding one of the following agents to the patient's empiric therapy regimen:

- Linezolid 600 mg IV every 12 hours, which may be administered orally when the patient is able to take oral medications.
- Vancomycin dosing as summarized in the following table

Approach to vancomycin dosing for adults with normal kidney function*

Loading dose (for patients with known or suspected severe *Staphylococcus aureus* infection)¶	Load 20 to 35 mg/kg (based on actual body weight, rounded to the nearest 250 mg increment; not to exceed 3000 mg). Within this range, we use a higher dose for critically ill patients; we use a lower dose for patients who are obese and/or are receiving vancomycin via continuous infusion.
Initial maintenance dose and interval	Typically 15 to 20 mg/kg every 8 to 12 hours for most patients (based on actual body weight, rounded to the nearest 250 mg increment). In general, the approach to establishing the vancomycin dose/interval is guided by a nomogram.Δ
Subsequent dose and interval adjustments	Based on AUC-guided (preferred for severe infection)[1] or trough-guided serum concentration monitoring.◇

DURATION OF THERAPY: Suggest treating most patients with nvHAP or VAP for seven days, in agreement with the 2016 ATS/IDSA guidelines and the combined 2017 European and Latin American guidelines on HAP and VAP . Seven days appears to be

as effective as longer durations in most circumstances and may reduce the emergence of resistant organisms.

For selected patients with metastatic infection, gram-positive bacteremia, slow response to therapy, immunocompromise, and pyogenic complications such as empyema or lung abscess, the duration of therapy should be individualized and courses longer than seven days may be warranted.

Monitoring serial procalcitonin levels can help guide the decision to discontinue antibiotics. While the optimal approach to using procalcitonin in patients with HAP or VAP has not been determined, a low or declining procalcitonin level (eg, <0.25 ng/mL or ≥80 percent decrease from peak) in a patient who has clinically improved on antibiotics provides additional reassurance that antibiotics can be safely stopped.

CONVERSION TO ORAL ANTIBIOTICS:

Switch to oral therapy when they are hemodynamically stable, clinically improving, and able to tolerate oral medications. If a pathogen has been identified, the choice of antibiotic for oral therapy should be based on the organism's susceptibility pattern. If a pathogen has not been identified, the oral antibiotic selected should be based on an appropriate de-escalation approach and generally can exclude coverage for MRSA and *P. aeruginosa*. All oral antibiotics selected for the treatment of pneumonia should have good lung penetration

PREVENTION

The Society for Healthcare Epidemiology of America (SHEA) and the Infectious Diseases Society of America (IDSA) issue updated practice recommendations to reduce the risk of VAP and NV-HAP (table 2).

Strategies to prevent ventilator-associated pneumonia

Essential practices that should be provided whenever possible to all patients to prevent VAP
▪ Use high-flow nasal oxygen or NIPPV, when appropriate, to avoid intubation, facilitate early extubation, and prevent reintubation ▪ Provide enteral instead of parenteral nutrition, when possible ▪ Avoid changing the ventilator circuit except in the following circumstances: • The ventilator circuit is visibly soiled; • The ventilator circuit is malfunctioning; or • A ventilator circuit change is recommended after a fixed number of days by the manufacturer ▪ Minimize sedation ▪ Maintain and improve physical conditioning through active and passive exercises ▪ Provide oral care, including toothbrushing (do not use chlorhexidine) ▪ Set the head of the patient's bed to an elevation between 30 and 45 degrees
If VAP rates remain high despite implementing the preceding practices, the following additional practices can be considered:
▪ Tracheostomy after 1 to 2 weeks of sustained invasive mechanical ventilation, taking into account patient trajectory and preferences* ▪ Using endotracheal tubes with subglottic secretion drainage ports for patients expected to require more than 48 to 72 hours of mechanical ventilation* ▪ Postpyloric feeding (instead of gastric feeding) for patients at high risk of aspiration or with gastric intolerance* ▪ Using selective oral or digestive decontamination in ICUs with low antibiotic utilization and low prevalence of antibiotic-resistant organisms¶

Essential practices that are recommended by SHEA/IDSA for preventing VAP and NV-HAP in all acute care hospitals include avoiding intubation and preventing reintubation when possible (eg, using noninvasive ventilation or high-flow oxygen by nasal cannula instead), minimizing sedation through the use of sedative protocols, implementing ventilator liberation protocols, maintaining and improving physical conditioning, elevating the head of the bed, providing oral care with toothbrushing but without chlorhexidine, and changing ventilator circuits only if visibly soiled or malfunctioning.

The following discussion will review some of the modalities that have been evaluated for preventing VAP.

Preventing aspiration — Aspiration is a major predisposing mechanism for both HAP and VAP. Elevating the head of the bed, minimizing sedation, draining subglottic secretions in ventilated patients, maintaining endotracheal tube airway

cuff pressure (20 to 30 cm H_2O), and application of positive end-expiratory pressure are measures that have been proposed to minimize aspiration.

Patient positioning — Supine positioning appears to predispose to aspiration and the development of HAP, particularly in patients receiving enteral nutrition. The head of the bed should therefore be elevated to 30 to 45° .While no effect of positioning on duration of mechanical ventilation or mortality has been demonstrated, it seems prudent to preferentially place intubated patients in the semirecumbent position unless contraindicated.

Subglottic drainage — Drainage of subglottic secretions that pool above the endotracheal tube cuff may lessen the risk of aspiration of secretions around the cuff and thereby decrease the incidence of VAP.

Gastric volume monitoring — It has long been standard clinical practice to monitor patients' gastric residual volume at regular intervals and/or prior to increasing the infusion rate of gastric tube feeding, with the hope of minimizing the risk of unrecognized gastric fluid accumulation and vomiting resulting in pneumonia. However, several studies have shown that measurement of gastric residuals correlates poorly with aspiration risk and is associated with a decrease in calorie delivery.

Decontamination of the oropharynx and digestive tract — Decontamination of the oropharynx and/or digestive tract may reduce the incidence of pneumonia in critically ill patients by decreasing colonization of the upper respiratory tract. Potential methods used include antiseptics in the oropharynx, selective decontamination of the oropharyngeal tract (SOD) with nonabsorbable antibiotics applied in the oropharynx, and selective decontamination of the digestive tract (SDD) with nonabsorbable antibiotics applied to the oropharynx and administered orally, with or without intravenous antibiotics.

Selective decontamination of the digestive tract — Selective decontamination of the digestive tract (SDD) refers to use of nonabsorbable antibiotics applied to the oropharynx and administered orally, with or without four days of intravenous antibiotics. Examples of antimicrobials included in oral and gastric tube regimens include colistin, tobramycin, and nystatin. The intravenous agent is typically a third- generation cephalosporin or a quinolone.

GENERAL SURGERY STANDARD TREATMENT GUIDELINES

PREOPERATIVE ASSESSMENT OF THE PATIENT

For achieving the desired optimum results in a surgical patient, apart from evaluating the nature and extent of the diseases and choice of surgery from available options, the assessment of the patient for his ability to withstand the stress of surgery and anaesthesia is very important. The factors that must be considered in preoperative assessment are:

- The disease (and its extent) for which the surgery is planned.
- The condition of the patient and his organ systems.
- The relative urgency of the surgery.
- The type of surgery and its alternatives.
- The relative morbidity and mortality of the disease.
- The relative morbidity and mortality of the surgical procedure.

All these factors are interdependent and this assessment is the most fundamental task to be performed in a surgical patient. The best person to undertake this task is the surgeon himself. Surgeon and other specialists may, at times, need the help of a physician, cardiologist, paediatrician or anaesthetist, to take the right decision. The preoperative assessment should also include discussion on drugs being taken by the patient and documentation of known allergies.

1. Informed consent

Informed consent on the presented format should be taken after a detailed discussion by the surgeon (or his responsible assistant) with the patient and his close relatives, informing them about the nature of the procedure planned, benefits expected, risks involved and possible alternatives, giving full opportunity to them to ask questions and clear doubts.

2. Preoperative preparation

- Routine investigations- Hb, TLC. DLC, CT, BT, RBS, LFT, KFT, HBsAg, HCV, HIV test, Urine routine examination, and in patients over 30 years, Chest X-ray and ECG.
- Special and specific investigations depending upon the nature of the procedure planned and the physical condition of the patient for evaluating fitness for surgery.
- Lipstick, nail polish and other cosmetics which may mask cyanosis and interfere with pulse oximetry should be removed.
- Dentures, spectacles, contact lenses, artificial limbs, artificial eyes, hearing aid and jewellery, cash and mobile phones should be removed before shifting the patient to operation theatre and instructions should be given to the patient.

- Withholding feeds before surgery depending upon age of the patient and nature of anaesthesia and surgery planned.
- Bathing. If possible, patient should take a proper bath on the day prior and on the morning of surgery giving special attention to the operative area. Patient should wear only clean hospital clothing.
- Hair removal should be done as close to the time of surgery as possible. If the skin is to be shaved, it is best done immediately before surgery. These are best removed with a clipper or chemical depilator. Shaving results in damage to skin and leads to abrasions that may not be visible.
- One should ask for history of allergic reactions to any chemical solutions.
- While scrubbing, one should work away from the operative site. Visibly soiled skin should be washed with soap and water before using surgical scrub.
- Pre anaesthetic medication as per policy.

Skin preparation for surgery

Preoperative surgical antisepsis aims at blocking infection into surgical wound and consists of washing, gloving along with application of antiseptic to surgical site. Different surgical scrubs available for skin preparation before surgical procedure include any of the following:

- **Povidone iodine**

The mechanism of action of povidone-iodine is the release of free iodine that binds to bacteria. Agent has excellent activity against gram-positive bacteria and good activity against gram-negative bacteria, viruses and fungi. Povidone iodine's free iodine attracts and binds with organic substances, modifying or decreasing its antiseptic effectiveness in the presence of blood. Removing organic lances such as blood, pus, or fat from the surgical site yields optimal results with use of a povidone e agent. Disadvantage is minimal persistent and residual activity and decreased effectiveness in the presence of blood and organic material

- **Chlorhexidine gluconate**

The mechanism of action for this broad-spectrum agent is disruption of the cell membranes by cytologic and physiologic changes that lead to cell death. It is effective against all categories of microbes, namely bacteria, viruses and yeasts. Chlorhexidine gluconate is classified as moderate in relation to the rapidity of action and has excellent persistent and residual activity. Chlorhexidine gluconate has been shown to remain effective in the presence of serum and protein- rich biomaterial, is blood. Disadvantage is that it is contraindicated for use on eyes, ears, brain and spinal tissue,

ha, mucus membranes and it is inactivated in the presence of saline solution, has drying effect on n. Accumulative benefit of chlorhexidine when repeatedly applied.

Use a combination of a rapid-acting agent (alcohol) and a longer lasting agent (chlorhexidine) is recommended.

- **Alcohol**

(70% ethyl or isopropyl alcohol), it is 95% effective against gram negative and gram-positive bacteria, mycobacteria, fungi and viruses It is not completely effective against spores All traces of alcohol should be dry before drapes are put. Alcohol is never used on mucus membranes and open wounds as it may cause desiccation

POSTOPERATIVE CARE

1. POSTOPERATIVE PAIN RELIEF

Postoperative pain is associated with all surgical procedures. This varies according to the surgical procedure. Severe pain can prolong gastrointestinal ileus, urinary retention, impair respiratory movements producing atelectasis and predisposes to deep vein thrombosis due to immobilization. Various methods to alleviate postoperative pain are NSAIDs, opioids (intramuscular, transdermal or transmucosal), patient-controlled analgesia, local infiltration of anaesthetic drugs, epidural analgesia and intrapleural analgesia. The method used depends upon the site and the magnitude of surgery done, severity of pain, whether the patient is allowed orally, facilities and expertise available. It is necessary to give analgesics by intramuscular or intravenous route in the immediate postoperative period and till the patient is able to accept orally. Commonly used agents are:

lnj. Diclofenac sodium 75 mg 8 hourly or

Inj. Pentazocine (30 mg/ml) 30-60 mg IM/IV repeated 3-4 hourly or lnj.

Tramadol (50 mg/ml) IM/1V4-6 hourly or

lnj. Morphine (15 mg/ml) 4-15 mg, can be repeated 4-6 times.

In tertiary care centres, epidural analgesia, intravenous patient- controlled analgesia, intrapleural analgesia can be used under expert care.

When patient is able to accept orally-

Tab. Paracetamol 500 mg 3-4 times a day or Tab.

Ibuprofen 400-600 mg 8 hourly or Tab.

Diclofenac 50 mg three times in a day.

Tab. Aceclofenac 100mg + Paracetamol 325mg, BD, 12 hourly

2. POSTOPERATIVE NAUSEA AND VOMITING

Postoperative nausea and vomiting leads to significant morbidity and prolonged hospitalization. It has an incidence of 20-30% after abdominal surgery. Predisposing factors are diabetes mellitus, pregnancy, dehydration, electrolyte imbalance, gastroesophageal reflux, use of certain anaesthetic drugs and opioids. Treatment- Bowel obstruction (mechanical or paralytic ileus) should be ruled out as a cause of vomiting by proper examination and investigations if it is associated with abdominal distension, fever and occurs beyond 3rd postoperative day. Nausea and vomiting are managed with intravenous fluids, analgesics to relieve postoperative pain, nasogastric decompression.

lnj. Metoclopramide (5 mg/mi) 10mg l/M or slow I/V 1-3 times daily or lnj.

Ondansetron (2 mg/ml) 4 mg slow IV or IM.

In children: 100 mcg/kg (max 4mg/day) by slow IV or IM. Or inj.

Promethazine (25 mg/ml) 2 ml I/V slowly

3. POSTOPERATIVE PNEUMONIA

Pulmonary disorders remain the most frequent post-operative problem and (10-15 percent) of patients are considered to have clinically significant chest complication after surgery under general anaesthesia.

Factors predisposing to increased chest complications are smoking, obesity, chronic restrictive obstructive lung disease, prolonged general anaesthesia and presence of nasogastric tube.

Postoperative pneumonia is caused by pathogens such as Pseudomonas, Serratia, Proteus and streptococcus.

Salient features

- Fever, productive cough, dyspnoea, Chest pain.
- Bronchial breathing and presence of rales.
- Chest X- ray shows areas of consolidation.

Treatment

1. Antibiotics: depending upon sputum culture and sensitivity. Initial treatment can be started with aminoglycoside and antipseudomonas Cephalosporins.
2. lnj. Ketorolac 30 mg every 6-8 hours lV or lM/lnj. Diclofenac 75 mg 1M every 6-8 hours.
3. Chest physiotherapy
4. Nebulization with bronchodilators may be used if bronchospasm is present.

5. HANDLING OF MEDICO-LEGAL CASES

First aid has to be provided in all cases who report in an emergency state. After stabilizing the patient, patient should be properly guided and helped in shifting to the appropriate centre. In case that we decide to treat, we must:

- Send information, in duplicate, to the police.
- Prepare a medico legal report.
- Preserve and seal clothes etc. preserve fluid and stain samples where indicated.
- Respond to information sought by the police.
- Arrange to take dying declaration, where indicated.
- Preserve all X-rays and patient records.
- Respond to court summons.
- In case of death, hand over the body to the police.
- In case of discharge/referral, police need to be informed.
- All medico-legal work should be carried out as per the existing state guidelines.

5. CARE DURING TRANSFER

This would depend on a number of factors like nature, patient's condition, referral, readiness of the referral centre to accept the patient and whether the transfer or elective.

Emergency transfer

- Identify the degree of emergency.
- Resuscitative measures to be adopted in serious patients with management etc.
- Transfer in a well-equipped ambulance.
- Transfer to a referral center with prior intimation and confirmation of the referral centre.

- Doctor (if required) or paramedical staff to accompany the sick patient.

Referral slip Should contain information on:

- Condition of the patient when first seen.
- Diagnosis and resuscitative measures taken.
- Reasons for referral.
- Where referred.
- Precautions advised during transportation.
- Any other information (e.g. any staff or equipment sent along with, any referral centre or specialist concerned).

6. ACUTE WOUND CARE

Irrigate gently with copious quantities of water or normal saline. If it is incised primary suturing is done. In lacerated wound margins are freshened, devitalized tissue is primary suturing is done. In crushed or devitalized wound there will be edema and t wound, after excising all the devitalized tissues the edema is allowed to subside for 2-61 delayed primary suturing is done. Wound debridement involves excision of all the devitalized regular intervals. If the wounds are fresh and less than 12 hours old, they can be closed with sutures staples. Any wound which is more than 24 hours old should be suspected to be contaminated and not closed completely. Only the deeper tissues can be approximated and the skin should be Left open.

- **Primary suturing** means suturing the wound immediately within 6 hours. It is done incised wounds. Delayed primary suturing involves suturing the wound in 48 hours to 10 days. It is done in lacerated wounds. This time is allowed for edema to subside.

- **Secondary suturing** implies suturing the wound in 10-14 days or later. It is done wounds. After control of infection, secondary suturing is done.

Associated injuries to deeper structures like vessels, nerves, tendons should be looked for before closure of the wounds. Any foreign body in the wound should be removed. Antibiotics and analgesics are required. Sutures are removed after seven days

Acute Appendicitis

Introduction

Appendicitis remains one of the most common diseases faced by the surgeon in practice. Despite improving diagnostic technology, there is still no single test or

clinical finding that is 100% reliable. The cause of acute appendicitis is unknown but is probably multifactorial; luminal obstruction with an appendicolith is quite common. Lymphoid hyperplasia, parasitic infections, and neoplasm are less common causes

In 1886, Reginald Fitz's seminal publication, 'Perforating Inflammation of the Vermiform Appendix' advocated early appendectomy for appendicitis. Appendicectomy is traditionally the treatment of choice and is increasingly done as a laparoscopic procedure.

Classification: Classified as uncomplicated or complicated based on clinical, radiologic, intra-operative, and/or histologic findings

Uncomplicated appendicitis (simple or non-perforated appendicitis) Inflamed appendix without any evidence of gangrene, perforation, purulent intraperitoneal fluid, appendicular phlegmon, or intra-abdominal abscess.

Complicated appendicitis (includes perforated appendicitis) Gangrenous inflamed appendix, formation of appendicular phlegmon, perforation, purulent-intraperitoneal fluid, , or intra-abdominal abscess. The appendiceal wall has been compromised due to pressure and inflammation and the intraluminal contents have leaked out into the peritoneal cavity.

Incidence

Western data estimates that as much as 6% to 7% of the general population will develop appendicitis during their lifetime, with the incidence peaking in the second decade of life. Indian data is lacking.

Symptoms

1. Patients typically complain of anorexia followed by epigastric or periumbilical abdominal pain. Usually, the pain localizes to its classic location in the right lower quadrant, so referred to as 'migrating pain'.
2. Pain gets worse after oral ingestion or movements
3. Feeling of nausea and vomiting (usually after onset of pain)
4. Low grade fever (absence of fever does not rule out appendicitis)
5. Weakness

Signs

1. Typical appendicitis patient appears ill and prefer to lie still because of the presence of localized peritonitis, which makes any movement painful.

2. Mild to moderate tachycardia.

3. Normal to low grade elevation of temperature.

4. Abdominal examination typically reveals tenderness and guarding on palpation of the right lower quadrant. The location of the tenderness is classically over Mc Burney's point (located one-third the distance between the anterior superior iliac spine and the umbilicus)

5. Rebound tenderness is also commonly elicited.

6. Occasionally if perforation occurs, diffuse peritonitis is seen.

A number of clinical tests and signs have been described to help in the diagnosis of acute appendicitis, however they are all indicators of peritoneal irritation and may differ depending on the location of the appendix.

Investigations

The diagnosis of acute appendicitis is predominantly a clinical one; many patients present with a typical history and examination findings. However, in atypical presentation diagnosis may be clinched with the help of imaging modalities like ultrasound scan and / or CT scan. The diagnosis of acute appendicitis can be challenging. Scoring systems devised are not definitive. A high index of suspicion– clinical, biochemical and radiological findings put together, may help a better decision making in the management of the patient with suspected appendicitis.

Diagnostic aides:

Laboratory tests are non-specific and need to be correlated clinically.

- White cell count - usually elevated
- C-Reactive Protein - usually elevated
- Urine examination including pregnancy test (for female patient in child bearing age) – to be done to rule out ectopic pregnancy in atypical presentation.

Imaging aides

1. USG abdomen/pelvis –

- preferred initial test in most patients

- risk of radiation – Nil
- usually seen is a distended, non-compressible appendix with an at least 6 mm diameter

2. CT scan abdomen /pelvis:

- for selected patients especially with diagnostic dilemma, perforation and obese patients
- Risk of radiation exposure – present
- Plain or with IV contrast is recommended (Oral and rectal administration of contrast material is not routinely required)
- Appearance of a thickened, inflamed appendix with surrounding fat "stranding" indicative of inflammation, is usually suggestive
- High sensitivity and high negative predictive value –may help reduce the rate of negative appendicectomy
- Not preferred as first line modality during pregnancy

3. MRI abdomen/pelvis

- Reserved for suspected appendicitis during pregnancy
- Recommended without use of contrast agents
- Risk of radiation exposure– Nil
- Appendiceal enlargement (>7 mm), thickening (>2 mm), and the presence of inflammation are usually suggestive of appendicitis
- Low availability and expertise during non-working hours

For fitness for Surgery & Anaesthesia

- Complete Hemogram
- Blood sugar – if diabetic
- Serum Electrolytes – in selected patients with profound vomiting
- Blood Urea, Serum creatinine – in selected patients
- Bleeding time, clotting time and /or prothrombin time
- X-ray chest – in elderly and with history of pulmonary diseases
- ECG & 2D echo – in selected patients when cardiac compromise is suspected or evident
- PFT/ ABG –optional in high-risk cases
- Other patient profile specific tests – as per pre-anaesthetic check up

Management of Acute appendicitis:

The following comorbidities are associated with poorer outcomes following treatment of appendicitis:

- Diabetes
- Immunocompromised state
- Obesity
- Crohn's disease
- HIV infection
- End-stage renal disease

I. Management of Acute Uncomplicated Appendicitis

A. Operative management of Uncomplicated appendicitis The appropriate treatment of acute uncomplicated appendicitis is prompt appendectomy.

1. **Timing of Surgery**: (Urgent, not emergent) The timing of surgery has been categorized as urgent intervention and not as emergent and can be performed within 6-24 hours of diagnosis without any statistically significant difference in length of hospital stay, operative time or rates of complications.

2. **Resuscitation, optimization & Antibiotics**: Patient should be fluid resuscitated as many presents with mild dehydration. Appendicitis is considered polymicrobial infections and IV broad spectrum antibiotics need to be administered to cover Gram- ve bacteria and anaerobes (as per institutional policy).

3. **Surgical approach**

Open Appendicectomy: Traditional approach

Position: supine

Anaesthesia: Spinal/General / Regional / Even local

Incision: The choice of incision is surgeon's preference, Commonly

used incisions are:

- Oblique muscle-splitting (Grid iron) incision (Mc Burney),
- Transverse incision (Rockey-Davis),
- Conservative midline incision, as indicated

- Lane's crease incision

Common Steps:

- The cecum is grasped by the taeniae and delivered into the wound
- Allows delivery of the appendiceal tip & visualization of the base of the appendix.
- The mesoappendix is divided sequentially.
- Appendix is crushed just above the base, ligated with an absorbable ligature at the site of crush, and divided.
- The stump is then either cauterized or inverted by a purse-string or Z suture technique.
- Abdomen is thoroughly irrigated if indicated, haemostasis noted and the wound closed in layers

Laparoscopic Appendicectomy

Laparoscopic appendicectomy is to be offered only when facilities and expertise is available and only when the charges are acceptable to the patient and not a procedure to be recommended as the only option to the patient.

Position: supine (The bladder is emptied by a straight catheter or by having the patient void immediately before the procedure). Both the surgeon and assistant stand to the left side of the patient with the left arm tucked. Patient strapped to table to prevent sliding off during table tilts.

Anaesthesia: General

Access & Port placements:

- Open or Veress needle access
- Ports: Supra/infra/trans umbilical camera port and 2 working ports (5mm) typically in the left / right lower quadrant and one in either suprapubic, supra umbilical or right upper quadrant as per surgeons preference to allow optimum triangulation. Common Steps:
- 30 degree telescope is used routinely used, a four-quadrant exploration is performed quickly and diagnosis confirmed.
- The patient is placed in reverse Trendelenburg position with right side up to gain exposure to the appendix and caecum.

• In a retrocecal appendix, sometimes the lateral peritoneal attachments, the white line of Toldt, must be divided to mobilize the cecum and expose the appendix.

• Atraumatic bowel graspers are used to elevate the appendix and inspect the base.

• Mesoappendix is carefully divided using the cautery (preferably bipolar) or harmonic scalpel and scissors after clipping or ligation.

• The base is then secured with Endoloops and the appendix divided.

• Alternatively, the appendix may be divided with an endoscopic stapler (prefer to use a blue loadstapler in cases in which the entire appendix is friable to get a healthy base stapled close to the caecum)

• Appendix is usually retrieved in an endo-bag.

• The pelvis is irrigated, haemostasis noted, trocars are removed under vision, and the wounds are closed.

• Laparoscopic appendectomy may also be performed with single-site laparoscopic surgical techniques as well in expert hands.

Although the laparoscopic approach is increasingly becoming the procedure of choice in most patients, open appendectomy still remains the choice of treatment on a global basis.

Post Operative Care: No further need for antibiotics. Patient is usually discharged after passage of flatus and tolerating oral diet.

Common Complications of Appendicectomy

• Bleeding • Ileus • Bowel obstruction • Stump leakage • Stump appendicitis

• Intra-abdominal abscess • Wound infection

B. Nonoperative Management of Uncomplicated Appendicitis

• Current best practice at this time for uncomplicated appendicitis is prompt appendectomy either by open or by laparoscopic approach. This concept has been challenged with non-operative management of acute appendicitis and is evolving.

• If non-operative management is chosen for some reason, the surgeon must remain extremely vigilant.

• Serial examinations and imaging are necessary to monitor for treatment failure especially if there's an appendicolith.

• The rate of recurrence of appendicitis treated with antibiotics alone is shown to be 7% to 14% at 1 year from the indexed episode especially in children.

• Treatment is by IV antibiotics, analgesics, fluids and serially initiating enteral feeds depending on abdominal findings.

• In future, the algorithm may move towards less invasive approaches, however, at this time appendicitis is still a surgical disease

II. Management of Acute Complicated appendicitis:

- Delayed Presentation with septicemia
- Appendicular perforation
- Appendicular phlegmon or Mass
- Appendicular abscess

Patients with diffuse peritonitis are challenging to manage with a high morbidity and sometimes mortality as well. These patients can be managed with multidisciplinary approach

e.g: • interventional radiologists inserting a percutaneous drain for an abscess or collection

• may require ICU admission

• In these cases, the treatment should be individualized on the basis of the nature of the presentation.

• In general, treatment for these patients is initially accomplished non-operatively. Fluid resuscitation is initiated, and broad-spectrum antibiotic therapy is initiated. A CT scan is obtained, and perforated appendicitis with a localized abscess or phlegmon is confirmed.

• Due to the extreme induration and friability of the involved tissues, Immediate exploration and attempted appendectomy in these patients may result in substantial morbidity, including

- failure to identify the appendix,
- postoperative abscess or fistula, and
- unnecessary extension of the operation to include ileocecectomy.

If a localized abscess is identified,

- Antibiotic therapy for 5-7 days
- Drainage of abscess
- CT-guided percutaneous drainage is performed for source control. The drainage catheter is typically left in place for 4 to 7 days
- Laparoscopic drainage is another option that can be exceptionally useful. This technique is performed by visualizing the inflammatory mass with the laparoscope and then entering the abscess with a laparoscopic suction tip, evacuating the purulent material, and placing a drain within the residual abscess cavity.
- Postoperative management is identical to that of patients who are successfully drained percutaneously.

• If an appendiceal phlegmon is present or if the amount of fluid present is not sufficient to drain, the patient may be treated with antibiotics alone, typically for 4 to 7 days also, as recommended by institutional guidelines for treatment of intra-abdominal infection.

• Interval appendicectomy can be performed based on risk assessment and for patients harbouring appendicolith. The current data is inclined towards an interval appendicectomy in children, however in adults the evidence in favour of interval appendicectomy is declining.

Referral Criteria:

• All patients with suspected appendicitis need to be referred for a Surgical Consultant and be managed by him. ICU admission criteria: o usually for complicated appendicitis

o ICU care may be needed in patients who present late with shock, septicemia or with perforation peritonitis and / or have other systemic illnesses.

o Patients with appendicitis who are not improving in-spite of intra-venous antibiotics and/or require tertiary care including ventilator support.

MDT approach: • Patients with complicated appendicitis requiring interventional radiologists support for abscess drainage or fecal fistula, intensivists for nutrition and sepsis management or experienced surgical team for further management.

• Pregnancy related management of acute appendicitis including need for MRI abdomen/pelvis.

Who does what?

Doctor: Surgeon: - (Surgical team –PG's, Registrar's, Consultants)

- Diagnosis & work up
- Pre operative planning
- Operative procedure
- Peri operative care in conjunction with Anaesthetist / Intensivist
- Post operative follow up

Anaesthetist: - Pre-Anaesthesia Check-up Part of resuscitation and stabilization Performing anaesthesia Post op ICU management in conjunction with Surgeon

Nurse:-(OT, ICU, ward & OPD) Pre/Intra/Postop comprehensive care Dressing of the wound

OT Technician: - Pre op equipment and drugs to be checked and kept ready Assist anaesthetist in the OT Assist the surgeon, positioning of the patient Resources required for one patient / procedure (Patient weight -approx. 60 Kgs)

Human Resources Drugs/Consumables Equipment

1. Surgeon – 1
2. Medical Officer / Assistant Surgeon – 1
3. Anaesthetist – 1
4. Pathologist – 1----- Services from outside can be availed
5. Staff Nurse – 1
6. Technician – 1
7. Nursing Orderly – 1
8. Cleaning staff-1

Investigations

1. Haemogram 2. Blood Sugar 3. Renal Function Test in selected cases 4. LFT in selected cases 5. S. Electrolytes in selected cases 6. USG in selected cases 7. ECG-if justifiable clinically 8. X- Ray – Chest- if justifiable clinically 9. Histopathology

Drugs & Consumables 1. OT Table & lights 2. Instrument trolley 3. Anesthetic Machine, instruments including endotracheal tubes & drugs 4. Monitor 5. Set of surgical Instruments (open and/or Laparoscopic) 6. Suction (open and/or laparoscopic) 7. Sutures / endoloops / stapler 8. Drains 9. Catheters 10. Cautery – a basic set (monopolar and/or bipolar) or harmonic shears 11. Antibiotics 12. Analgesics 13. I.V. Fluids 14. Dressings.

If the centre has facilities for Laparoscopic Surgery, the procedure can be done laparoscopically or open surgery as deemed appropriate by the Surgeon.

APPENDICITIS

Appendicitis is the commonest cause of acute abdomen and may appear as catarrhal appendicitis or as obstructive appendicitis and sometimes it may present as an appendicular lump or appendicular abscess or as burst appendix with peritonitis.

Salient features

- Acute central abdominal pain, followed by nausea, vomiting and fever, with the pain after a variable period, shifting to right lower abdomen localized tenderness maximum at the Mc Burney's point, rebound tenderness and guarding in the right iliac fossa,
- An inflammatory lump in the right lower abdomen or signs of peritonitis.
- A polymorph nuclear leukocytosis and ultrasonography appearances may help to corroborate the clinical diagnosis.
- Investigations are primarily undertaken to exclude other conditions like ectopic gestation or ureteric calculus.

Management

A. Definitive treatment

- Appendectomy within 48 hours before the lump formation.
- An interval appendicectomy may be performed where a lump has formed or when attack has already resolved or circumstances make surgery not feasible.
- Laparotomy is to be done in cases of generalized peritonitis.
- Stop oral feeding.

B. Pharmacological (Expectant management)

1. Intravenous fluids (R.L/N.S/Dextrose) to maintain hydration. Requirement of fluids would be more if the patient has peritonitis and septicemia.
2. Inj Ceftriaxone 1gm + Salbactum 500mg, iv twice daily or
3. Inj. Ciprofloxacin infusion (100mg/50 ml) 100 ml twice a day for 5 days or
4. Inj. Gentamicin (40 mg/ml), 80 mg IV 8 hourly. Or
5. lnj. Amikacin (500 mg/2 ml), 2 ml lVfwice a day.
6. Inj. Metronidazole infusion (500 mg/i 00 ml) 100 ml IV 8 hourly. lnj. Diclofenac sodium (25 mg/ml) 50 ml IM SOS.
   (Caution: Purgation and enema are contraindicated)

Pain subsides first, followed by relaxation of the abdomen and control of fever. Tenderness disappears later. Polymorph nuclear leukocytosis tends to settle down.

Failure of signs and symptoms to subside or the appearance of new signs and symptoms during expectant treatment, calls for surgical intervention.

C. Postoperative management

- Oral feeding is started when abdomen is soft, the patient has passed flatus/ stools and bowel sounds have appeared. Start with liquids, gradually permitting semi solid and solid diet over a period of 2-3 days.
- Antibiotics should continue for 5 days or more if the condition demands.
- Initially antibiotics are given by parenteral route and later switched to oral route when the patient starts tolerating semi solid diet.
- Patient is discharged usually between 3rd and 5th postoperative day, if comfortable, ambulatory, tolerating semi solid or solid food, afebrile and has a healthy wound.
- Sutures are removed around 7th postoperative day.

D. Patient education

- Normal routine physical work can be permitted in 10-15 days (5-7 days after laparoscopic appendectomy).
- Moderate physical work is permitted after 4-6 weeks (2 weeks after laparoscopic appendectomy).
- Heavy physical work is permitted after 2-3 months (4-6 weeks after laparoscopic appendectomy).

Acute Cholecystitis

Acute Cholecystitis is the acute inflammation of the gall bladder and is associated in majority of cases with gall stones (90%) and without gall stone in 10% of the cases

Pathogenesis

Calculous - Stone causing obstruction of the Hartmann's pouch or cystic duct.

Acalculous – Common in patients who have undergone major trauma, burn, sepsis. Causative bacteria- E. Coli (most common), Klebsiella, Pseudomonas, Proteus, Streptococcus Faecalis, Salmonella, Clostridium welchii.

Symptoms

- Right upper quadrant or epigastric pain which may radiate to the back
- Pain is often dull and constant, may be colicky

- There may be dyspepsia, flatulence, intolerance to fatty food

- Biliary colic presents as severe right upper quadrant pain that is associated with nausea and vomiting and may radiate to the chest, may last for a few minutes to several hours. Pain starts at night, awakening the patient.

Signs

- Fever (may be present)

- Tenderness – Right upper quadrant. Positive Murphy's sign

- Palpable tender mass in right upper quadrant of abdomen (may be present)

- Tachycardia

- Jaundice (may be present)

Investigations

- Haemogram– Leucocytosis, Raised CRP

- Liver function tests - deranged

- Blood sugar

- Urea / Creatinine- if needed

- Prothrombin Time

- Chest X-ray

- ECG

- Echocardiogram – In selected cases

- USG examination of abdomen – Gall bladder distended, wall thickened, oedematous, with or without pericholecystic fluid. Gall stones present in 90% of the cases. Pooled sensitivity and specificity of USG in the diagnosis of gallstones were 84 % and 99 %

- MRCP – In case of concomitant jaundice

- CECT abdomen – In case of complications

- Hepatobiliaryiminodiacetic acid scan (HIDA scan) has the highest sensitivity and specificity for acute Cholecystitis, although its scarce availability, long time required to perform the test, and exposure to ionizing radiation limit its use (LoE 2

GoRB) Diagnosis Combining clinical, laboratory and imaging investigations is recommended, although the best combination is not yet known (LoE 4 GoRC) Differential

Diagnosis

Common

- Acute appendicitis
- Perforated peptic ulcer
- Acute pancreatitis

Uncommon

- Acute pyelonephritis
- Acute myocardial infarction
- Pneumonia – Right lower lobe

Complications

- Mucocele
- Empyema of the gall bladder
- Perforation
- Obstructive Jaundice
- Acute Pancreatitis
- Acute Cholangitis
- Intestinal Obstruction due to Gall Stone Ileus

Risk Stratification

Patient's age above 80 in ACC is a risk factor for worse clinical behaviour, morbidity and mortality (LoE 3 GoR B) The co-existence of diabetes mellitus does not contraindicate urgent surgery but must be reconsidered as a part of the overall patient comorbidity (LoE 3 GoR C) Currently, there is no evidence of any scores in identifying patient's risk in surgery for ACC

Treatment Conservative

treatment

Nil per mouth, IV fluid, analgesics, broad spectrum antibiotics – effective against gram negative aerobes (E.g.- Cephazolin, Cefuroxime, Gentamicin), regular monitoring of the temperature, pulse and other physical signs of the patient to assess the response to the conservative treatment.

Once the pain has subsided, and the temperature and pulse have become normal, the patient can be fed orally and be sent home and kept on regular follow up, and taken up for cholecystectomy after 3 to 6 weeks (Delayed laparoscopic cholecystectomy)

Types of Surgical Approach- -In ACC, a laparoscopic approach should initially be attempted except in case of absolute anaesthesiology contraindications or septic shock (LoE 2 GoR B) –

Laparoscopic cholecystectomy for ACC is safe, feasible, with a low complication rate and associated with shortened hospital stay (LoE 1 GoR A)

- Among high-risk patients, in those with Child A & B cirrhosis, advanced age >80, or pregnant women, laparoscopic cholecystectomy for ACC is feasible and safe (LoE 3 GoR C)

- Laparoscopic or open subtotal cholecystectomy is a valid option for advanced inflammation, gangrenous gallbladder, or any setting of the "difficult gallbladder" where anatomy is difficult to recognize and main bile duct injuries are more likely (LoE 2 GoR A)

- In case of local severe inflammation, adhesions, bleeding in Calot's triangle or suspected bile duct injury, conversion to open surgery should be strongly considered. (LoE 3 GoR B)

Timing of Surgery

- Early laparoscopic cholecystectomy is preferable to delayed laparoscopic cholecystectomy in patients with Acute Cholecystitis as long as it is completed within 3 days of onset of symptoms (Level 1 Evidence; Grade A recommendation)

- Laparoscopic cholecystectomy should not be offered for patients beyond 10 days from the onset of symptoms unless symptoms suggestive of worsening peritonitis or sepsis warrant an emergency surgical intervention. In people with more than 10 days of symptoms, delaying cholecystectomy for 45 days is better than immediate surgery (LoE 2 GoR B)

Associated common bile duct stone: suspicion and diagnosis at the presentation

- Elevation of liver biochemical enzymes and/or bilirubin levels is not sufficient to identify ACC patients with choledocholithiasis and further diagnostic tests are needed. (LoE 2 GoR B)

- At AUS, the visualization of CBDS is a very strong predictor of choledocholithiasis. (LoE 5 GoR D). Indirect signs of stone presence such as increased diameter of common bile duct are not sufficient to identify ACC patients with choledocholithiasis and further diagnostic tests are needed. (LoE 1 GoR A)

- Liver biochemical tests, including ALT, AST bilirubin, ALP, gamma glutamyltransferase (GGT), AUS should be performed in all patients with ACC to assess the risk for CBS. (LoE 2 GoR B)

- Common bile duct stone risk should be stratified according to the proposed classification, modified from the American Society of Gastrointestinal Endoscopy and the Society of American Gastrointestinal Endoscopic Surgeon Guidelines (LoE 5 GoR D)

- Patients with moderate risk for choledocholithiasis should undergo preoperative MRCP, EUS, intraoperative cholangiography, or Laparoscopic ultrasound depending on the local expertise and availability. (LoE 1 GoR A)

- Patients with high risk for choledocholithiasis should undergo preoperative ERCP, intraoperative cholangiography, Laparoscopic ultrasound, depending on the local expertise and the availability of the technique. (LoE 1 GoR A)

- CBDS could be removed preoperatively, intraoperatively, or postoperatively according to the local expertise and the availability of the technique. (LoE 1 GoR A)

Alternative treatments for high-risk patients

- Gallbladder drainage, together with antibiotics, converts a septic Cholecystitis into a non-septic condition; however, the level of evidence is poor (LoE 4, GoR C)

- Among standardized gallbladder drainage techniques percutaneous trans hepatic gallbladder drainage (PTGBD) is generally recognized as the preferred technique due to the ease and the reduced costs. (LoE 4, GoR C)

- PC could be considered as a possible alternative to surgery after the failure of conservative treatment in a small subset of patients unfit for emergency surgery due to their severe comorbidities (LoE 2 GoR B)

• Delayed laparoscopic cholecystectomy could be offered to patients after reduction of operative and anaesthesiology related risks to reduce further hospitalization (LoE 5 GoR D) Post-

Operative Care

Antibiotic Therapy- - Patients with uncomplicated Cholecystitis can be treated without post-operative antibiotics when the focus of infection is controlled by cholecystectomy. (LoE 1 GoR B)

- In complicated acute Cholecystitis, the antimicrobial regimens depend on presumed pathogens involved and risk factors for major resistance patterns. (LoE 3 GoR B)

- The results of microbiological analysis are helpful in designing targeted therapeutic strategies for individual patients to customize antibiotic treatment and ensure adequate antimicrobial coverage in patients with complicated Cholecystitis and at high risk for antimicrobial resistance. (LoE 3 GoR C)

Referral Criteria

ICU care may be needed in patients who present late with severe sepsis and have other systemic illnesses. Medico legal Issues

• Failure to diagnose and institute immediate proper treatment

Who does what?

Surgeon

- Establishing the diagnosis and working up the patient

- Follow the abovementioned treatment algorithm

- Post-operative care and follow up

Anaesthetist-Pre-Anaesthetic work up

- Anesthetizing patient during surgery and post op management in critical patients

Nurse - Pre, Intra and Post op care

Technician

-Pre op equipment and drugs to be checked and kept ready

- Assist the anaesthetist in the OT

- Assist the surgeon, positioning of patient

Human Resources Drugs/Consumables Equipment

1. Surgeon – 1
2. Medical Officer / Assistant Surgeon – 1
3. Anaesthetist – 1
4. Pathologist – 1 ----- Services from outside can be availed
5. Staff Nurse – 1
6. Technician – 1
7. Nursing Orderly – 1
8. Cleaning staff-1

Investigations

1. Hemogram
2. Blood Sugar
3. Renal Function Test - in selected cases
4. LFT
5. S. Electrolytes in selected cases
6. USG 7. ECG
8. Echocardiogram – in selected cases
9. X- Ray – Chest
10. MRCP – in selected cases
11. CECT – in selected cases
12. HIDA scan – selected cases
13. Histopathology- following surgery

Drugs & Consumables

1. OT Table & lights
2. Instrument trolley
3. Anaesthetic Machine, instruments including endotracheal tubes & drugs
4. Monitor
5. Set of surgical Instruments
6. Suction
7. Sutures
8. Drains
9. Catheters
10. Cautery – a basic set
11. Antibiotics
12. Analgesic

13. I.V. Fluids
14. Dressings
15. If the centre has facilities for Laparoscopic Surgery, the procedure can be done laparoscopically as decided by the Surgeon.

Abbreviations

CBDS - common bile duct stones

GoR - Grade of Recommendation

IOC - Intraoperative cholangiography LC-

Laparoscopic cholecystectomy LoE -

Level of Evidence

LUS - Laparoscopic ultrasound;

PC - Percutaneous Cholecystostomy

CHOLECYSTITIS

It is the inflammation of gall bladder, which may occur most commonly due to obstruction of - Cystic duct with gall stones. Females are twice as likely to develop cholecystitis.

Salient features

- Acute pain in right hypochondrium or epigastrium. Initially pain is intermittent but later presents as constant and severe.
- Murphy's sign is generally positive.
- Boas sign maybe present in some cases.
- Ultrasound is paramount in diagnosis.

Management

A. Low fat diet. ln concurrent APD, spices are to be avoided.

B. **Definitive treatment** is cholecystectomy in symptomatic and asymptomatic patients with diabetes or a solitary large stone or multiple small stones with wide cystic duct or porcelain gall bladder or anxious patients. If the patient comes after 48 hours manage conservatively and cholecystectomy after 6-8 weeks.

C. **Expectant management**

In case of acute cholecystitis, empyema gall bladder.

- Maintenance of IV fluids
- Inj Ceftriaxone 1gm + Inj Salbactum 500mg iv twice daily
- Inj. Ciprofioxacin (infusion 100 mg/50 ml) 100 ml IV twice a day. lnj. Gentamicin (40 mg/mI) 2 ml IV 8 hourly.

Or

- inj. Ampiciliin (500 mg/mi) 1 ml IV 6 hourly. Inj. Cloxacillin (500 mg/ml) I ml IV 6 hourly.

Or

- Inj. Ciprofloxacin (infusion (100 mg/50 ml) 100 ml IV twice a day. inj. Amikacin (500 mg/2 ml) 2 ml twice day.
- In case anaerobic bacterial infection is suspected or anticipated, give Inj. Metronidazole (500 mg/100 ml) 100 ml IV 8 hourly.
- Inj. Diclofenac sodium (25 mg/ml) 2 -3 ml IM SOS or 6 hourly. Or
- Inj. Pentazocine lactate (30 mg/ml) 1 ml IM SOS
- Inj. Hyoscine butylbromide (20 mg/ml) I ml IV SOS.
- In patients having obstructive jaundice, add Inj. Vitamin K (10 mg/ml) 1 ml IM once a day for 3 days. In case of no improvement, liver functions are to be assessed. Antibiotics are usually stopped after 5-7 days unless the patient has evidence of persistent infection.

Patient education

- To avoid fatty and fried meals for 3 months.
- Although ambulation is encouraged as early as possible, heavy physical exertion should be avoided for 2 weeks (after laparoscopic cholecystectomy) and for 3 months after conventiorxi cholecystectomy.
- If T-tube has been placed, it should be removed after 2-3 weeks, after ensuring that the CBD is patent and there is free flow of contrast into the duodenum during 1-tube cholangiography.

FISSURE-IN-ANO

Acute fissure

Linear defect in the epithelial lining of the anal canal distal to the dentate line

Chronic fissure

- When failed to heal within 6-8 weeks
- Exposed internal anal sphincter muscle fibre
- Skin tag
- Internal hypertrophied anal papilla

It most commonly occurs in the midline posteriorly. Most cases are idiopathic and may be due to trauma, inflammatory bowel diseases and sexually transmitted disease.

Salient features

- There is severe pain on defecation.
- Bleeding is usually small and occurs as a streak by the side of stools.
- On examination, a longitudinal ulcer is seen in the midline posteriorly that may be covered by a skin tag. There is local inflammation and induration.

Treatment

The aim of the treatment is to obtain complete relaxation of the sphincter and provide relief from pain.

A. Medical therapy

Breaking the cycle of pain, spasm, ischemia.

- Warm sitz bath
- Stool- bulking agents
- 200-500mg of 0.2% Nitroglycerin paste locally (34% headache, 60% heals at 8 wks)
- Calcium channel blocker locally— Diltiazem (2%), Nifedipine
- Botulinum toxin -temporary muscle paralysis (73% heals at 8 wks)
- Steroids/ topical creams are of limited utility.

B. Surgery

Lateral anal sphincterotomy.

Anal dilatation - Discouraged (incontinence rate -12.5% to 24.3%).

C. Patient education

Local care of the region and sitz bath should be regularly taken.

Avoid constipation by the use of high fiber diet and use of purgatives.

FISTULA-IN-ANO

- Fistula-in-ano is a tract lined by granulation that connects superficially the skin around the anus and deeply the anal canal or the rectum.
- Low level fistula opens into the anal canal below the anorectal ring.
- The high-level fistula opens into the canal at or above the anorectal ring.
- It is important to know the level of fistula since a low-level fistula can be laid open without fear of incontinence.

Salient features

- Persistent seropurulent discharge that may be blood stained.
- Pain and sometimes a history of a perianal abscess that has been drained.
- Fistula-in-ano maybe associated with tuberculosis, Crohn's disease, carcinoma, bilharziasis.
- There is usually an opening within 3-4 cm of the anal orifice with granulation tissue. The fistula heals only to recur later on.
- Digital examination may reveal the internal opening.

Treatment

A. Nonpharmacological

Local hygiene and sitz bath. Diet modification to avoid constipation.

B. Pharmacological

- Cap. Ampicillin 500 mg every 6 hours.
- Tab. Metronidazole 400 mg every 8 hours.
- Bulk laxative to relieve and avoid constipation.

C. Definitive treatment

Fistulotomy (laying open of the fistula tract), fistulectomy (excision of the fistula tract) and use of Seton. Secondary fistula needs treatment of primary disease. High level fistula may need proximal colostomy for treatment.

D. Patient education

- Do not take treatment for anal disorders like abscess and fistula from unqualified persons.
- Avoid constipation and take bulk laxatives. Maintain local hygiene.

HEMORRHOIDS

- Haemorrhoids (commonly called Piles) are the dilated tortuous veins occurring in relation to the anus.
- These can be primary or secondary to some other disease like carcinoma of rectum, pregnancy, straining at micturition, or constipation due to any cause.
- These can be classified into external, internal or mixed (externo-internal) depending on their position in relation to anal orifice.
- Internal haemorrhoids are classified as -
  1. First degree- Bleed, but do not prolapse
  2. Second degree -Spontaneous prolapsing and reducing with or without bleeding
  3. Third Degree- Prolapsing, that requires manual reduction
  4. Fourth Degree - Prolapsed, cannot reduce

Salient Features

- Patients can present with bleeding - it is bright red in colour, occurs as splash in pan during defecation, mass per rectum, mucus discharge, pruritus, pain, anaemia.
- On digital rectal examination only thrombosed pile can be felt. If there is hard mass on rectal examination than it is further investigated.
- On proctoscopy- exact position can be made out as bulge into the proctoscope.
- Complications of haemorrhoids include strangulation, thrombosis, ulceration, gangrene, fibrosis, suppuration and pyelophlebitis.

Treatment

- Asymptomatic haemorrhoids do not need any treatment
- Secondary haemorrhoids due to concomitant disease also tend to resolve once the underlying disease is cured.
- First-degree haemorrhoids- Dietary fibre, avoid straining during defecation, sitz baths. Rubber band ligation, sclerosis, and thermotherapy by using infrared beam, electric current, CO2 laser, or ultrasonic energy is used in first- or second-degree haemorrhoids.
- Operative haemorrhoidectomy is reserved large third and fourth-degree haemorrhoids, mixed haemorrhoids with a prominent external component.
- The complications of surgery include pain, acute retention of urine, reactive bleeding and later on secondary haemorrhage and anal stricture.

Patient education

- Avoid constipation and use laxative, if required. Use high fiber diet that produces high roughage. Sitz bath to reduce pain and spasm.
- Haemorrhoids that prolapsed should be reposed gently and not forced back. Take treatment for any disease that promotes straining at micturition like benign hypertrophy of prostate.

RETENTION OF URINE

Retention of urine is inability to pass urine. It can be either acute or chronic.

Causes

1. Mechanical causes of retention are: posterior urethral valves, foreign bodies, tumours, blood clot and stones, phimosis, paraphimosis, trauma (rupture of urethra or bladder), urethral stricture, urethritis, meatal ulcer, tumours, prostatic enlargement-benign or malignant, retroverted gravid uterus, fibroid, ovarian cyst, faecal impaction.

2. Neurogenic causes are- postoperative retention, neurogenic bladder, spinal cord injuries, hysteria, drugs- anticholinergics, antihistaminics, and smooth muscle relaxants.

Salient Features

- Acute retention of urine is characterized by inability to pass urine despite urge, suprapubic discomfort or severe agonizing pain. There may be previous such episodes or history of trauma, instrumentation or surgery.
- Chronic retention is an enlarged painless bladder whether or not the patient is having difficulty with micturition. Sometimes acute episode can be precipitated in cases of chronic retention of urine.
- There may be symptoms suggestive of prostatic enlargement in elderly male.
- On examination, there is suprapubic swelling arising out of pelvis in the midline in the hypogastric region that is dull to percussion and cystic in nature. This helps to differentiate from anuria where urinary bladder is not palpable.
- Rectal examination will help to confirm the prostatic pathology in elderly patients.
- Spinal defects or neurological findings suggest presence of neurogenic bladder.

Treatment

A. General measures

- Sedation, adequate hydration and antibiotics if sepsis is present.
- If there is history of trauma, urethral injury should be ruled out before attempting catheterization.
- If urethra is patent, a catheter is passed in to the bladder under strict aseptic precautions and connected to a sterile closed collecting system. The catheter is chosen according to the size of the external meatus. In cases of acute retention, single catheterization is adequate or an indwelling self-retaining catheter is inserted if deemed necessary.

B. Surgical Management

- If urethral pathology is present or there is inability to pass the catheter, a suprapublic puncture or cystostomy is performed to relieve the retention.
- In case of chronic retention, decompression should be performed intermittently (300-400 ml volume) to avoid haematuria that can occur after sudden decompression.
- That patient should be kept under observation after admission for investigation to elucidate the cause of retention. The investigations include urine examination, renal functions, plain and contrast radiological studies; ultrasound, CT scan or MRI. Urodynamic studies are required to diagnose neurogenic bladder. Cystoscopy can help to diagnose and treat many conditions of the urethra and urinary bladder.

C. Definitive treatment of the aetiology is done after proper investigations.

D. Pharmacological

1. Tab. Cotrimoxazole (960mg) 2 times a day Or

   Tab. Norfloxacin 400mg 2 times a day for 5-7 days.

   This may be changed according to urine culture and sensitivity reports.

E. Patient education

- Explain catheter care-measures - tip of the urethra should be cleaned with antiseptic solution regularly.
- Watch for blood in urine.

UNDESCENDED TESTIS

Undescended testis is defined as the testis, which cannot be brought to the base of the scrotum without undue tension on the spermatic cord.

This anomaly is often diagnosed early but the treatment is delayed due to misconceptions leading to various complications.

Salient features

- The testis can be located in the superficial inguinal pouch, inguinal canal or intra-abdominal site. Truly ectopic testis can be present in perineum, femoral region, pubopenile site or contralateral hemiscrotum.
- Differentiate from retractile testis which is occasionally pulled up due to reflex contraction of cremasteric muscle. The retractile testis is normal in size, can be

brought down into scrotum where it stays for some time and the scrotum is normally developed.

- Complications of undescended testis include temperature effects on testis, endocrine effects, germ cell alteration, lower fertility; higher incidence of malignancy, increased incidence of torsion, increased chances of-trauma and psychological trauma.

Treatment

1. If the newborn child is seen with unilateral undescended testis, follow up the patient at intervals to see the descent.
   - If testis fails to descend by the age of 12 months, orchiopexy is advised.
   - If seen after first birthday the operation of orchiopexy should be done before the age of two years.
   - The operation entails mobilizing the testis and cord structures and fixing it in the subdartos pouch in the scrotum with unabsorbable sutures.
2. If the newborn child has bilateral undescended testes with hypospadias, it should be investigated for intersex disorder.
3. If a child has undescended testis with clinically visible hernia, orchiopexy can be done at an earlier age along with herniotomy.

Patient education

The parents should be informed about the anomaly if detected at birth and advised to monitor the descent of testis and to get it operated by the age of 1 year.

SCROTAL SWELLINGS

Scrotal swellings can be either congenital or acquired. The acquired scrotal swellings could be further classified as inflammatory, traumatic or malignant. Important diagnoses include hydrocele, epididymo-orchitis, torsion of testis and tumors.

HYDROCELE

This is a collection of fluid in some part of processus vaginalis usually tunica. It can occur in children and adults. Hydrocoele could be primary or secondary to testicular diseases like inflammation, infections or malignancy. It can be unilateral or bilateral.

Salient features

- Cystic swelling usually translucent, it is possible to reach above the swelling and it is not possible to feel the testis distinct from the swelling.
- Although there is history of reduction of size in children, it is not reducible

- If the hydrocele is lax and of moderate size, it may be due to epididymo - orchitis, torsion, or testicular tumors.

Treatment

- In infants, it is advised to wait till the age of two years to allow spontaneous resolution.
- Beyond the age of two years, the surgical treatment entails herniotomy by the inguinal approach.
- In adults, definitive treatment requires drainage of the fluid along with eversion of the sac with or without excision of the same. This can be done under local or regional anaesthesia.

EPIDIDYMO-ORCHITIS

- Epididymo-orchitis is inflammation of the epididymis and the testis due to various causes.
- It can be acute or chronic. Infection reaches the epididymis via the vas deferens from the urinary tract.
- A history of urinary tract infection is usually available.
- The condition has to be differentiated from torsion of testis.

Salient features

- The epididymis and the testis show swelling with shiny oedematous skin and tenderness. It may be possible to feel the epididymis and testis separately. The pain is relieved by rest elevation of testis.
- Urine examination may shows pus cells Complications include secondary hydrocele with clear fluid, abscess formation and pus discharge from sinus formation.

Treatment

- Bed rest and scrotal support.
- Cap. Doxycycline 100mg once daily for 8-10 days. It may be change according to urine culture and sensitivity.
- Analgesic and antipyretics may be required.

TORSION OF TESTIS

Torsion of testis is an emergency condition where testis rotates in its axis compromising its blood supply. Testicular torsion is most common between 10 and 25 years of age.

Clinical features

1. Patient presents with sudden onset pain in the scrotum, groin and lower abdomen.

2. On physical examination, the testis will be swollen, tender, and high-riding, with an abnormal transverse lie.

- The testis seems high and the tender twisted cord can be palpated above it.
- The most sensitive physical finding in testicular torsion is the absence of the cremasteric reflex.
- This reflex is elicited by stroking or pinching the medial thigh, causing contraction of the cremaster muscle, which elevates the testis.
- The cremasteric reflex is considered positive if the testicle moves at least 0.5 cm.
- A negative Prehn sign (relief of pain with elevation of the testicle) is classically thought to be a predictor of torsion, this is unreliable for diagnosis

Differential Diagnosis

1. Epididymo-orchitis
- In epididymo-orchitis there will usually be dysuria associated with the accompanying urinary infection.
- Elevation of the testis reduces the pain in epididymo-orchitis and makes it worse in torsion.
2. Torsion of Appendage of testis

Investigation

Color Doppler Ultrasound scan will confirm the absence of the blood supply to the affected testis.

- Non-surgical correction can sometimes be accomplished by manually rotating the testicle in the opposite direction (i.e., outward, towards the thigh) It is based on the assumption that twisting of the testis occurs in a medial direction.
- The resolution of symptoms after manual detorsion did not correlate with the presence or absence of persistent torsion. This maneuver maybe attempted, but it should not delay transfer to the operating room for definitive detorsion and fixation of the testis.

Management

- Testicular torsion is a surgical emergency that requires immediate intervention to restore the flow of blood.
- If treated either manually or surgically within six hours, there is a high chance of preserving the testicle.
- If there is any doubt about the diagnosis, the scrotum should be explored

without delay. Immediately refers to a higher center if facilities are not available.

- Treatment of torsion of testis requires immediate correction by surgical exploration through scrotal incision, untwisting of the cord and orchiopexy. It is important to fix the opposite testis at the same time.

Patient education

- Any scrotal swelling should be brought to notice of your doctor.
- Any sudden onset swelling of the testis merits immediate attention of the surgeon and delay in diagnosis or treatment even for few hours can be harmful.

INGUINAL HERNIA

- Hernia is an area of weakness or disruption of the fibro muscular tissues of the body wall
- It occurs due to raised intro-abdominal pressure due to various causes or weakness of wall due to any disease.
- A hernia consists of the sac, the coverings and the contents of the sac that could be o intestine, ovary or Meckel's diverticulum.
- Most common type of the external hernia is the inguinal and incisional hernia, less being femoral and umbilical.

Clinical Features

- Patient presents with swelling in the groin region.
- There will be history of decrease in size of swelling on lying down.
- Cough impulse and reducibility is present in uncomplicated hernia. But hernia can co and present with irreducibility, obstruction or strangulation.

Differential Diagnosis

- In the male include vaginal hydrocele, encysted hydrocele of the cord, spermatocele, hernia, incompletely descended testis in the inguinal canal, lipoma of the cord.
- In the female include hydrocele of the canal of Nuck, femoral hernia.

Management

A. Surgical

- The treatment of choice for hernia is surgical repair.
- Any predisposing factors need to be treated first before hernia repair else recurrence is possible.
- The hernia with complications needs to be operated in emergency.
- Treatment in children entails herniotomy while in adult's repair of the

posterior wall of the inguinal canal without (herniorrhaphy) or with prosthesis (hernioplasty) after high ligation and the sac is done. This can be done by open repair or laparoscopic repair by the experts. Day care surgery under local anaesthesia is practiced at many centres.

- Complications of herniorrhaphy include infection, haematoma formation, injury to viscera like urinary bladder, injury to vas and recurrence.

B. Nonsurgical treatment

This is not advocated for the treatment of hernia except in the extremely frail patients unfit for surgery or where surgery is refused by the patient. Application of external pressure causes skin and may cause injury to the contents.

C. Patient education

- Reduce weight and quit smoking before surgery.
- Treatment of any predisposing factors like chronic cough, prostatic enlargement and constipation is necessary.
- The surgery should not be delayed since complications of hernia are frequent and can be serious.
- After surgery, avoid lifting heavyweights, cycling etc. for three months.

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CARCINOMA BREAST

Breast cancers is a common cancer in Indian women and most common cancer in some Indian metropolitan cities in women.

Symptoms and Sign

- Breast lump which is seen in 85% of cases
- Lump in axilla
- Change in size/shape of breast
- Dimpling, puckering of breast
- Skin induration
- Redness
- Nipple discharge
- Nipple retraction
- Found on investigation for breast pain
- No symptoms

Diagnosis

- Breast self-examination
- Clinical suspicion
- Clinical Examination
- FNAC
- Biopsy
- Mammography
- Breast ultrasound
- Newer techniques: MR[, nuclear scan.
- Metastatic workup

The most important, practical and time-tested method of using the available gadgets to come to clinical diagnosis is TRIPLE ASSESSMENT

Triple Assessment- The main components of triple assessment are

- Clinical examination,
- Radiological imaging of breasts (mammography and ultrasound breast)
- Pathological examination of tumor (core biopsy, FNAC)

1. Breast examination

Early detection of breast cancer significantly reduces the risk of death.

Every woman between the ages of 20 and 49 should have a clinical breast examination by a health professional every one to two years. Those over 50 should be examined annually.

2. Monthly self-examination

- Self-examination once a month just after the menstruation, as during this time breast are less engorged.
- Stand in front of a mirror. Check for changes or redness in the nipple area. Look for changes in the appearance of the skin. With hands on the hips, push the pelvis forward and pull the shoulders back and observe the breasts for irregularities. Repeat the observation with hands behind the head.
- Lie down on the back with a rolled towel under one shoulder. Palpate the breast with the flat of the finger of the hand for any lump.
- Examine the nipple area, by gently lifting and squeezing it and checking for discharge.
- Repeat step 3 in on upright position.

Note: A lump can be any size or shape and can move around or remain fixed. Of special concern are specific or unusual lumps that appear to be different from the normal varying thicknesses in the breast.

3. Mammography

- A mammogram is a radiographic examination of the breast, either displayed on a film or on a computer monitor.
- Images of each breast in the CC (craniocaudal) and MLO (mediolateral oblique) projections are taken.
- High probability of malignancy occurs in fine linear, branching, pleomorphic, and heterogeneous microcalcifications.
- Lobulated mass and mass with spiculated margins is also suggestive of carcinoma.

4. Ultrasound Breast

Malignant sonographic features of solid masses include an irregular or angular shape, more than three lobulations, ill-defined, spiculated or microlobulated margins, width greater than anteroposterior diameter (orientation not parallel to the skin surface or "taller than wide"), markedly hypo echoic (dark) echogenicity; a surrounding thick, echogenic (white) halo, posterior shadowing (black

shadows posterior to the mass), duct extension and associated calcifications.

5. Biopsy The various methods of biopsy techniques are

- FNAC
- Core needle biopsy
- Stereotactic biopsy
- Incisional biopsy
- Excisional biopsy

Core biopsy is the investigation of choice in the evaluation of breast malignancy. ER/PR/Her 2/Neu tumor status should be determined for all samples of invasive cancer

Treatment

Treatment modalities available are Surgery, Radiation, and combination drug therapy. combination therapy is virtually always required.

The choice is determined by many factors, including the age of the patient and (among women) menopausal status, the kind of cancer (e.g., ductal vs. lobular), its stage, and the tumor hormone receptors status

A. Surgery

Most commonly performed operation is Modified radical mastectomy (MRM). The entire tissue is removed along with the axillary contents (fatty tissue and lymph nodes). In contrast to a radical mastectomy, the pectoral major muscles are spared.

Breast conservative surgery (BCT) - In this form the patient is left with native breast. Wide local excision is followed by radiotherapy

- Advantages of BCT
    - Acceptable cosmetic appearance
    - Lower level of psychological morbidity
    - Equivalence in terms of disease outcome for BCT and mastectomy in selected patients
- Indications of BCT
    - Ti ,T2(<5cm), NO, Nl, MO
    - T2 >5cm in large breasts. More important, however, than the true size of the tumor is the ratio of the tumor size to the breast size.
    - Single clinical and mammographic lesion
- Absolute contraindications
    - Multicentric disease (tumors in more than one quadrant of the breast),
    - Diffuse malignant-appearing calcifications,
    - Inflammatory breast cancer,
    - Prior radiation to the chest or breast or inability to receive radiation,
    - Persistent positive margins despite appropriate attempts for breast-conserving surgery,
    - Need for radiation during pregnancy.

B. Chemotherapy

- Neo-adjuvant chemotherapy is when chemotherapy given prior to surgery. It converts a previously unresectable, locally advanced breast cancer to an operable tumor. Down staging a primarily operable breast cancer results in

small increase (7% to 12%) in breast conservation rates.

- Adjuvant chemotherapy is when chemotherapy is given after surgery. Same regimens can be used in, the neo-adjuvant and adjuvant therapeutically settings. Commonly used chemotherapy regimens are CAF, CEF, CMF and taxanes based in node positive patients.

C. Adjuvant hormone therapy

- In hormone positive tumors defined by ER/PR expression on IHC, adjuvant hormone therapy is considered in all positive patients
- Tamoxifen and aromatase inhibitors (letrozole, anastrazole, exemestane) are used.
- In HER 2/neu positive tumors **trastuzumab** is used.

D. Radiotherapy

Indication for post-mastectomy radiation-

Four or greater than 4 lymph nodes involvement, T3 and 14 tumors, extra capsular invasion, lymph vascular space invasion, close surgical margins, presence of 1-3 lymph node is relative indication.

Radiotherapy is also used after conservative breast surgery, loco-regional recurrence following mastectomy, palliation of bone or brain secondaries including spinal cord compression, fungating and bleeding advanced tumors.

Breast cancer management requires multi-disciplinary approach.

BREAST ABSCESS AND MASTITIS

- Mastitis is a cellulitis of the interlobular connective tissue within the mammary gland, which can result in abscess formation and septicaemia.
- Mastitis is a complication often encountered in primiparous women and develops in 1% to 24% of breastfeeding women. A breast abscess develops as a complication of mastitis in 5% to 11 % of cases. The most common bacteria is Staphylococcus aureus.
- Differentiating between mastitis and abscess can be difficult; when there is suspicion for abscess, the woman should be referred for ultrasound evaluation.
- Mastitis on ultrasound will appear as an ill-defined area of altered echotexture with increased echogenicity in the infiltrated and inflamed fat lobules
- The diagnosis of abscess requires identification of a hypoechoic collection, often with a thick echogenic periphery.
- Common clinical symptoms of breast infection include pain, redness, and increased temperature.
- Ultrasound is the first-line investigation because it is relatively pain less and,

provides guidance for percutaneous drainage.

Treatment

A. Mastitis Phase

- Tab. Erythromycin 500 mg 3 times a day for 7 days.
- Oral cephalosporins or clindamycin hydrochloride are excellent choices to cover the most common organisms
- Tab. Diclofenac 50 mg BD

B. Where an abscess has formed, aspiration of the pus, preferably under ultrasound control, has now supplanted open surgery as the first line of treatment. Antibiotics should be continued to reduce systemic infection and local cellulitis.

C. Open surgical drainage may be necessary for patients with loculated collections or for those who have failed conservative management with antibiotic therapy and percutaneous drainage.

After open surgical drainage of an abscess, suckling may be difficult for a few days mechanical reasons on the affected side, but the mother should be encouraged to feed on unaffected side. The infected breast, however, should be emptied either by manual expression or by pump until such time as feeding can be recommenced. The mothers with breast abscesses should be encouraged to continue breastfeeding.

If lactation is to be suppressed cabergoline is preferred over bromocriptine.

D. Patient education

Good hygiene and avoidance of breast engorgement or cracked nipple are important.

During pregnancy, daily washing will remove the dried secretions that will otherwise collect on the nipple.

After feeding the infant, the nipples should be dried and any segments of the breast that have not been adequately emptied during feeding expressed

NIPPLE DISCHAGE

Nipple discharge is a presenting complaint of between 3-7.4% of women seeking medical care for breast problems. Differentiating between physiologic and pathologic nipple discharge is important for management.

- Physiologic nipple discharge is from bilateral breast, from multiple duds, non-spontaneous, multi- coloured (milky, grey, green, brown, yellow) with thick sticky consistency.
- Pathological nipple discharge is usually from unilateral breast, from single duct, spontaneous colour is bloody, serous or clear and consistency is watery and copious.

Causes of pathologic nipple discharge papilloma, papillomatosis, ductal carcinoma in situ, invasive ductal carcinoma.

Management

- Check for character of discharge and any lump in the breast.
- Galactorrhoea in non- lactating or without pregnancy then get serum prolactin and thyroid profile
- If discharge is from multiple ducts, bilateral, expressible then reassure the patient and mammogram if over 40 years of age.
- If profuse multiple duct in post - menopausal lady consider Hadfields surgery.
- If discharge is multi-coloured, from single duct, unilateral, spontaneous, serous, bloody then get mammogram and ultrasound.
- If the discharge is from singe duct, profuse, spontaneous, blood stain, RBC on cytology then duct excision.

VARICOSE VEINS

They are dilated, torturous, elongated veins in the leg.

There are three categories of venous insufficiency

1. Congenital venous insufficiency is comprised of predominantly anatomic variants that are present at birth. Examples of congenital venous anomalies include venous ectasias, absence of venous valves, and syndromes such as Klippel-Trenaunay syndrome.

2. Primary venous insufficiency is an acquired idiopathic entity. This is the largest clinical category and represents most of the superficial venous insufficiency encountered in the office.

3. Secondary venous insufficiency arises from a post-thrombotic

Salient features

- Heaviness, discomfort, and extremity fatigue legs.
- Pain is characteristically dull, does not usually occur during recumbency or early in the morning, and is exacerbated in the afternoon, especially after periods of prolonged standing.

- Swelling
- The discomforts of aching, heaviness, and/or fatigue are usually relieved by leg elevation or elastic support.
- Cutaneous burning, termed venous neuropathy, can also occur in patients with advanced venous insufficiency.
- Pruritus occurs from excess hemosiderin deposition and tends to be located at the distal calf or in areas of phlebitic varicose branch segments.
- Discoloration, ulceration in the feet

Duplex ultrasonography helps in localization of perforators, saphenous-femoral, saphenous-popliteal incompetence, to rule out DVT.

Management

Indications for treating varicose veins are: Cosmesis, symptoms refractory to conservative therapy, bleeding from a varix, superficial thrombophlebitis lipodermatosclerosis, venous stasis ulcer.

A. Conservative

- Compression treatment- to use gradient compression stockings that provide 30-40- or 40-50-mm Hg of compression at the ankle, with gradually decreasing compression at more proximal levels of the leg
- Elevation of limbs- relives edema.
- Drugs like Cacium dobsilate (500 mg BD), Diosmin (450 mg BD). Benefits of all these drugs are doubtful.

Surgical

- Sclerotherapy
- Trendelenburg operation -High ligation, division, stripping of the GSV
- Endovenous thermal ablations- Endovenous LASER oblations (EVLA) or Radiofrequency ablation (RFA)

Postoperative management

1. Compression bandaging immediately following stripping or avulsion of veins. i bandages by compression stocking after 2 days.
2. Limb elevation and encourage the patient to walk with compression stockings after fire' of dressing 48 hours after operation.
3. Postoperative pain is controlled with dextropropoxyphene or NSAIDs.

C. Patient education

- Certain Do's are leg exercise, leg elevation, wear stockings and drinking 4-5 L of fluids in a day.
- Certain Don'ts are hot bath, exposure to extremes of temperature, pregnancy, contraceptivepil oestrogens, long journeys (flight).
- Teach the patient leg exercises -frequent movements of toes and heels,

sarvangasan or shirshasan, and elevation of foot end of the bed about 6 inches by putting a block of wood or 2 bricks under foot end of be and to avoid prolonged standing or dangling legs down.

ACUTE ABDOMEN

Abdominal pain can occur due to variety of medical and surgical causes. It is important to elicit a detailed clinical history and perform abdominal examination to determine the cause of pain. Severe cases, it may be necessary to give treatment before proper history can be obtained or examination is allowed by the patient.

Causes of acute abdomen

1. Abdominal causes

1. Inflammation of peritoneum due to bacterial or chemical contamination. - Perforation of appendix or bowel, ulcer, pancreatitis or pelvic inflammatory disease.

2. Mechanical obstruction of hollow viscera-intestinal obstruction, ureteric obstruction due to stone or other causes, and obstruction of the biliary tree.

1. Vascular disturbances - vascular rupture, embolism or thrombosis, torsion of pedicle

2. Other causes

Diabetic-ketoacidosis, Tetany, Angina, Sickle cell anemia and viral infections.

PANCREATITIS

Is an inflammation of the pancreas and requires immediate medical attention. It may be acute or chronic.

Salient features

- Severe abdominal pain radiating to back, nausea and vomiting.
- Abdomen is tender but to lesser degree than the pain itself.
- 80% of cases of pancreatitis are caused by alcohol and gall stones. Gall stones

is single most common aetiology of acute pancreatitis, alcohol intake is single most common aetiology of chronic pancreatitis.

- Blood amylase or lipase will be 4-6 times higher than normal.
- Ultrasound/ CT shows an inflamed pancreas.

Management

1. Mild acute pancreatitis

- Mild cases are without complications. Patient is kept NPO till abdomen becomes soft, which usually takes up to 5 days.
- Dehydration may occur, adequate rehydration is required intravenously
- Pain killers are required.
- Antibiotics may be required in moderate and severe cases.

2. Severe acute pancreatitis

- Severe acute pancreatitis is associated with organ failure, necrosis, pseudocyst of pancreas and abscess formation.
- Patient needs ICU care in a tertiary care health centre.
- Hypovolemia is generally present; oxygen inhalation and antibiotics are required.
- Laparotomy, Pancreatic necrosectomy or surgery for pseudocyst may be required.

THYROID SWELLING

Thyroid swelling forms one of the most important differentials for swelling in front of the neck. The differential diagnoses of thyroid swelling are benign goitre, intrathyroid cysts, thyroditis benign and malignant tumours.

- Simple goitre is enlarged thyroid gland and occurs commonly around puberty in girls iodine deficiency.
- Malignancy should be suspected in case of extremes of age, male sex, rapidly growing swelling, persisting pain, dysphagia, recurrent laryngeal nerve palsy, hardness and fixity of the gland and presence of one or more palpable neck nodes.
- Thyroid function test, Fine needle aspiration cytology, ultrasonography are helpful in differentiating the causes of thyroid swelling.

Treatment

1. Simple diffuse hyperplastic goitre is preventable by using iodized salt.

2. Treatment with L-thyroxine may reverse the swelling at this stage. Simple nodular goiter is treated by subtotal thyroidectomy

3. Thyroidectomy

Preoperative care - Indirect laryngoscopy (IDL) is performed to identify compensated or unsuspected recurrent laryngeal nerve palsy. Before operation, thyrotoxic patients should be made euthyroid with antithyroid drugs (carbimazole 10-15 mg 4 times a day and prop 20 mg 3 times a day). Fully discuss the potential complications with the patients- mentioning risk to parathyroid gland and recurrent laryngeal nerve.

Postoperative care - Place patient in a slightly propped up position. Carefully observe for respiratory insufficiency, haemorrhage from the wound, irritability to the facial nerve and carpopedal spasm (parathyroid injury). Monitor drain output daily and remove if 24 output becomes lesser than 10 ml. Check wound site for infection and sutures are removed on the 5th day.

Complications

- The most immediate life-threatening complication is haemorrhage under deep cervical fascia which can lead to acute asphyxia. Management includes reopening of the suture line, to drainage of the hematoma and re-exploration for control of bleeders.
- Damage to recurrent laryngeal nerve can lead to respiratory distress (bilateral recurrent laryngeal nerve) and hoarseness of voice.
- Parathyroid damage leads to hypocalcaemia. Symptomatic hypocalcaemia (positive Chvostek's or Trousseau's signs or corrected serum calcium level <8 g/dl) is treated with 10% calcium gluconate intravenously. If hypocalcaemia persists, oral Calcium supplement and synthetic Vitamin D is necessary.
- Late complications include recurrent thyrotoxicosis, hypothyroidism, and recurrence of malignancy at the local site or in the lymph nodes in the neck.

4. Radio-iodine therapy

- Radio-iodine therapy is indicated in follicular, papillary and mixed carcinoma.
- Following total thyroidectomy; a total body radioactive isotope scan should be arranged four weeks after the operation.
- During this period L-thyroxine therapy should be withheld.
- If radioactive scan shows residual thyroid tissue or metastatic deposit then further dose of radioiodine should be given to ablate these.
- Following isotope scan, high dose L-thyroxin (0.2-0.3 mg) should be started and continued for life.
- Radioactive iodine has no role in residual/metastatic medullary carcinoma.
- Treatment approach to Hurthle cell neoplasm is similar to follicular neoplasm.

5. Follow up

Patients should be followed at three monthly intervals for the initial 2 years and 6 monthly for three years and then at yearly interval for life.

On each follow up visit patient should be examined for any local or nodal recurrence in the neck, a chest X-ray should be done to exclude pulmonary deposit and clinical features of thyroid toxicity noted and dose of L-thyroxine regulated

CERVICAL LYMPHADENOPATHY

An enlarged cervical lymph node is the commonest cause of lump in the neck. Cervical lymph des may become enlarged as a result of inflammation or neoplastic process. Tuberculosis is one of common causes of cervical lymphadenopathy.

Causes of lymphadenopathy and clinical features

Condition	Cause	Features
Acute inflammation tender	Infection of the upper Aero- digestive tract, Head and neck or Other infections	Fever, sore throat, firm, nodes 1-2 cm in diameter swelling in the neck and fever cough may or may not be present
Chronic Inflammation	Tuberculosis, sarcoidosis Histiocytosis X	Variable on presentation depending on the stage of the disease: multiple matted lymph Nodes/ cold abscess
Lymphomas/	Hodgkin Non- Hodgin Lymphoma	painless rubbery lymph nodes. symptoms related to primary disease

Malignancy	Metastatic Carcinomas of The upper aero- digestive tract Squamous Cell Carcinoma Melanoma- firm to hard lymph nodes

Management

- Detailed history and examination are essential to pinpoint specific aetiology. Majority of the lymph nodes are reactive to viral infections of upper respiratory tract, therefore, do not require any treatment.
- In case of acute suppurative lymphadenopathy secondary to any focus of bacterial infection in the drainage area:

  Cap. Cephalexin 250-500 mg every 6 hours for 7 days, or Cap.

  Amoxicillin 250-500 mg every 8 hours for 7 days.

- If lymph nodes persist, perform fine needle aspiration cytology (FNAC) and treat accordingly. If FNAC is inconclusive take a biopsy from the enlarged lymph node and treat accordingly.
- In case of chronic lymphadenopathy perform FNAC and treat accordingly, If FNAC is inconclusive, perform biopsy and treat accordingly.
- Tubercular lymphadenopathy

Start anti-tubercular therapy. Reassess the patient after 6 months. If lymph nodes are either not present or less 1 cm size keep the patient under follow – up and continue treatment. However, if lymph nodes are palpable and more than 1 cm take a biopsy of the node and accordingly and consider second line anti-tubercular drugs.

CIRCUMSION

INTRODUCTION:

Religious male circumcision is considered a commandment from God in Judaism widely practiced in Islam and customary in Christian churches in Africa. Virtually all the current policy statements from specialty societies and medical organizations do not recommend routine neonatal circumcision The opponents to circumcision consider it a violation of human rights

CASE DEFINITION: The words "circumcision" is derived from the Latin circum (meaning "around") and coedre (meaning "to cut"). Male circumcision is the removal of some or the entire foreskin (prepuce) from the penis

MEDICAL INDICATIONS:

- In infants and young boys – true phimosis caused by BXO (Balanitis xerotica obliterans)
- Recurrent balanoposthitis
- Recurrent UTI's with an abnormal upper urinary tract
- Phimosis may result from misguided attempt by parents to expose the glans forcibly
- In adult – inability to retract prepuce for intercourse
- Splitting of an abnormally tight frenulum
- Balanitis
- Before radiotherapy for carcinoma penis
- Paraphimosis
- Diabetes mellitus with recurrent balanoposthitis
- HIV
- UTI INCIDENCE: Proportion of males circumcised worldwide vary from one sixth to a third Circumcision is most prevalent in the Muslim countries of the world In India too, it is nearly 100% among Muslims 15

PREVENTION AND COUNSELLING: Physiological adhesion between the foreskin and glans penis may persist until 6 years of age and be mistaken for phimosis. Forcible retraction of the skin is not recommended in physiological phimosis. At 4-5 years of age, topical corticosteroid cream may be used for 6 weeks if phimosis continues to exist.

Circumcision – is done if it is

- Resistant to topical steroid therapy
- If patient requires treatment for balanitis
- When there is urinary obstruction due to very high prepuce Carcinoma penis should be ruled out. When confined to prepuce, circumcision may be adequate

treatment but regular follow up is necessary Similarly chancre which may present as phimosis should be ruled out Balanitis xerotica obliterans – normal foreskin becomes thickened and does not retract Has increased susceptibility to carcinoma and hence requires early treatment

Treatment is circumcision

OPTIMAL DIAGNOSTIC CRITERIA: Phimosis is diagnosed by inability to retract the prepucial skin

SITUATION 1:

I. PHIMOSIS: clinical features

- Inability to retract the prepuce
- Ballooning of prepuce (second bladder) in children
- Balanoposthitis because of inability to clean the glans

ii. PARAPHIMOSIS: clinical features Retracted prepuce cannot be pulled forward; forms a tight ring and acts as constriction. Venous congestion increases with swelling of glans and can result in ulceration and gangrene of the glans iii. History of diabetes with recurrent attacks of balanophosthitits iv. History of bleeding and short duration of lack of retractibility would suggest carcinoma v. History of STD; sexual history to r/o chancre 16

DIFFERENTIAL DIAGNOSIS: 1. Chancre 2. Cancer 3. Meatal stenosis (masked by prepuce)

INVESTIGATION: Simple phimosis is a clinical diagnosis and requires no investigation for confirmation Routine investigation before surgery such as Blood sugar Haemogram Urine r/m X Ray and ECG may be done as per anaesthetic indication Biopsy of underlying lesion if any USG of the abdomen and pelvis to evaluate the entire urinary tract in cases of

UTI TREATMENT: Medical treatment in children 5-6 years with congenital phimosis
– topical steroid cream Surgical treatment –

circumcision

PROCEDURE: In infant: Applying a clamp (or bone forcepts) across the prepuce distal to the glans with blind division of the foreskin is to be condemned Perform a proper circumcision under direct vision as in an adult

ANAESTHESIA – GA – in children, infants and neonates Dorsal penile nerve block, Ring block and / or EMLA (lidocaine/prilocaine) topical cream may be used in adults 17 Razmus et al reported that newborns circumcised with the dorsal block and ring block in combination with oral cucrose had lowest pain scores Wg et al found EMLA cream in addition to local anaesthetic effectively reduces the sharp pain induced by needle puncture In adults frenular stretch must to avoid bleeding from frenular artery Histopathology: should be done when there is suspicion of malignancy or other associated conditions

POST OP: Analgesic Antibiotic: perioperative dose Abstinence for 4-6 weeks in adults The patient should be reviewed 5-7 days post op Retract and clean any skin covering the glans to prevent adhesion

COMPLICATINS OF CIRCUMCISION:

- Bleeding most common
- Infection
- Scar
- Meatal stenosis
- Phimosis in later life – if insufficient skin is removed in a child during the first surgery
- Skin bridge formation in infants SOP: Day care REFERAL CRITERIA: The patient should be referred to a higher centre for treatment of associated conditions if any, such as malignancy Patient with bleeding disorders and co morbidities may be safely operated in a higher centre SITUATION 2: DIAGNOSIS: Clinical as in situation

1 INVESTIGATIONS: as in situation 1

18 HbA1C Coagulation profile if bleeding disorder is suspected

TREATMENT: As in situation 1 Additional procedures: Devices are available for infant circumcision – Plastibell, Gomco clamp, or Mogen clamp used together with a restraining device 1. Frenulum may need to be broken or crushed and cut from the corona near the urethra to ensure that the glans can be freely and completely exposed SOP: Day care WHO DOES WHAT? AND TIMELINES

a. Doctors:

- Clinical examination

- Diagnosis
- Planning surgery
- Surgery
- Post op care
- Anesthesia

b. Nurse: • Pre & post operative care • Assisting during surgery

c. Technician: • Pre op equipment and drugs to be cechked and kept ready • Assist anaesthetist in the OT • Assist the surgeon

COLECTOMY

INTRODUCTION: Sir William Arbuthnot was one of the early proponents of the usefulness of total colectomies. Colectomy is commonly performed for the treatment of colon cancer.

DEFINITION: Colectomy implies the surgical resection of any extent of the large intestine (colon). Based on the segment of colon removed colectomies are termed as

1. Right hemicolectomy.
2. Extended right hemicolectomy
3. Transverse colectomy
4. V resection
5. Left hemicolectomy
6. Extended left hemicolectomy
7. Sigmoidectomy
8. Proctosigmoidectomy
9. Total colectomy
10. Total proctocolectomy
11. Subtotal colectomy

DIFFERENTIAL DIAGNOSIS:

- Polyps
- Inflammatory bowel disease-ulcerative colitis, Crohn's disease
- Tuberculous stricture of the large bowel with obstruction 21
- Vascular malformations with lower gastro intestinal bleeding
- Amoebiasis

PREVENTION: In familiar situations like FAP & HNPCC early colectomy is advised. It is important to understand the carcinogenesis in colorectal cancer & the associated molecular events.

ENVIRONMENTAL FACTORS also pay an important role, particularly dietary factors & estrogen replacement. Association between hyperplastic polyposis & colorectal cancer & adenomas called sporadic MIS tumours Colorectal cancers: are Sporadic in 75% cases & Genetic in 25% (younger age at diagnosis) Positive Familial history is present in 15%-20%. HNPCC (5%)-80% risk FAP (less than 1%)-100% risk of development of CRC – prophylactic total colectomy/proctocolectomy COUNSELLING:

GENETIC COUNSELLING PREDISPOSITION SHOULD BE COUNSELLED & SCREENED FOR COLON CANCER. Screening colonoscopy and polypectomy – reduces colon cancer mortality.

1. OPTIMAL DIAGNOSTIC CRITERIA: Situation 1 Clinical Diagnosis Anatomical locations and clinical manifestations of colon cancer Distribution % Ascending / Caecum Transverse Descending/Sigmoid Manifestations Bleeding Anaemia Malena Abdominal pain Mass obstruction Abdominal pain Obstruction Mass Changing bowel habit Obstruction Mass Abdominal pain Mass 22 Diarrhoea obstruction Perforation Low back pain
2. INVESTIGATIONS:
   - ♣ Hemogram
   - ♣ Colonoscopy – investigation of choice
   - ♣ - Biopsy & HPE - Brush cytology if biopsy is not possible
   - X-ray abdomen – if patient presents with features of large bowel obstruction
   - Double contrast barium enema : - When colonoscopy is contra indicated or not available - Findings – constant irregular filling defect - Detects associated lesions - Small ulcerative lesions can be diagnosed
   - USG abdomen
   - Endoluminal ultrasound – if available

- CECT – if available is used in large palpable abdominal masses = To determine local invasion
- Urograms – when evidence of hydronephrosis on USG/ CT in left sided tumours

3. TREATMENT:

1. Pre op evaluation of staging, respectability, patient's operative risks are mandatory.
2. Accurate localization of tumour – of particular importance.

a. Sometimes known cancer may not be apparent on serosal aspect.
b. Localization by tattooing during colonoscopy, Barium enema.
c. Pre op CT, USG assessment of iver metastasis should be done

PRE-OP PREPARATION: Mechanical bowel preparation Prophylactic antibiotics 23 Blood grouping and cross matching Thromboembolism prophylaxis

OPERATIVE TECHNIQUES: Resection should follow Standard oncological principles:

- Proximal ligation of primary arterial supply at its margins
- Adequate proximal & distal margins (5 cm) determined by area supplied by the primary feeder artery
- Appropriate lymphadenectomy – harvesting of minimum 12 nodes
- Extent of resection is an important prognostic factor (SAGES guidelines 2000)
- Any tumour not removed intraoperatively strongly influences prognosis & therapy

Ro – absence of residual tumour, margins free histologically
R1 – no gross residual tumour but margins histologically positive R2 – residual gross disease remains unresected

RADIAL MARGIN:
T4 lesions are a complex group & should be considered separate from other T groups Radial tumour free margins should be resected. Radial margin should be histologically free of disease for resection to be curative. Specimen labelling, marking are important for a good pathological report R1 & R2 resection – incomplete resection for cure affects curability though TNM stage remains same

LATERAL CIRCUMFERENTIAL MARGIN: In addition to radial, proximal & distal margins, circumferential margins should also be pathologically assessed. Positive margins are associated with increased rate of local and distal failure. Disease free survival and mortality significantly related to margin involvement after

TME ADJUVANT Ro stage: Adjuvant therapies require complete resection A case is not Ro if it is 24

- ♣ Non enbloc resection
- ♣ Radial margins positive for disease
- ♣ Bowel margin positive for disease
- ♣ Residual lymph node disease present or
- ♣ Nx (incomplete staging)

LYMPHADENECTOMY: Should be radical (up to the level of origin of primary feeding artery) Apical nodes positive for disease may have prognostic significance in addition to number of positive lymph nodes

ENBLOC RESECTION of adherent tumours: En bloc removal of adjacent organs locally invaded by cancer colon can achieve survival rates similar to patients with tumour that do not invade an adjacent organ, provided negative resection margins are achieved.

PERFORATION OF TUMOUR SHOULD BE AVOIDED (SAGES GUIDELINE) Inadvertent full thickness perforation of rectum would probably classify tumour as T4 and resection as R1 Perforation at the site of cancer, as opposed to an area remote from the tumour has a greater impact on survival & local recurrence. Inadvertent local perforation predisposes to local recurrence and warrants post-operative radiotherapy.

INTRAOPERATIVE SPILLAGE: HAS AN INDEPENDENTT EFFECT ON PROGNOSIS Adjuvant radiotherapy may be considered to decrease rates of local recurrence NO TOUCH TECHNIQUE: Value inconclusive 25

SURGICAL PROCEDURES: Anatomical Resection of Colon Cancer Tumour location Vascular Ligation Colon resection Anastamosis Caecum, ascending colon ileo-colic, right colic Right hemicolectomy ileotransverse colostomy Hepatic flexure, Proximal transverse colon ileocolic right, middle colic Extended right hemicolectomy with omentectomy iIeodescending colostomy

Distal transverse colon splenic flexure ileocolic right, middle or left branch of middle colic, left colic Extended right hemicolectomy with omentectomy or Left hemicolectomy ileosigmoid colostomy or Transverse sigmoid colostomy Descending colon Inferior mesenteric or left colic Left hemicolectomy Transverse colorectal anastamosis Sigmoid colon Inferior mesenteric or sigmoid Left colectomy or Sigmoid resection Transverse colorectal anastamosis or descending colorectal anastamosis Colectomy may be performed by the i) Conventional open technique

REFERRAL CRITERIA: Patients suspected of colon cancer & biopsy proven should be referred to a higher centre for further evaluation and treatment when
1) Adequate surgical facilities are not available / surgeon does not have sufficient experience in colon cancer surgery.
2) Competent pathologist to report on malignant lesions as per standard oncological guidelines is not available.
3) For adjuvant / neo-adjuvant radio and chemo therapy

TREATMENT: Patient requiring colectomy for biopsy proven cancer are best referred to a super specialty centre In view of the need for multi-modality treatment.

SITUATION 2: All investigations as in situation 1
- Spiral CT in elderly patients more than 80 years
- CT colonoscopy also called virtual colonoscopy – 6 mm polyps may be picked up effectively
- CEA – fetal glycoprotein - Increased pre op CEA in node positive Ca – indication for chemotherapy
- MRI :
- PET : detection of metastasis
- SPECT – if single photon emission is studied, such as technetium or thallium
- FDG-PET – useful in evaluation of recurrent colorectal cancer -Differentiates post op changes from recurrent / residual disease -Useful diagnostic tool but prohibitive cost
- CT-PET – fusion tests provide the most powerful integrated images
- NUCLEAR MEDICINE IMAGING: -Using 131 I , 111 In, 99m Tc bound to monoclonal antibodies, leucocytes & erythrocytes.

TREATMENT: As outlined in situation 1. Laparoscopic resection is gaining popularity. However, it is not freely available & performed as per protocols.

SPECIAL CONSIDERATIONS

1. Synchronous malignancies or polyps Patients with synchronous malignancies should be considered for subtotal colectomy depending on the distance between lesions Colonic cancer with multiple adenomatous polyps – subtotal colectomy 27 (Due to increased risk of metachronous lesion and to facilitate surveillance of the remaining colon) Factors that influence the decision to perform prophylactic subtotal colectomy -number -location -size of accompanying polyps -age -compliance of patient

2. Cancer is a polyp Complete endoscopic removal of polyp with cancer in situ – no further treatment Histoplathology shows invasive carcinoma: Ensure that endoscopic polypectomy was complete Specimen was submitted with proper orientation to the pathologist for histopathology Carcinoma at margin of resection requires formal resection Carcinoma with free margins – a. thorough pathological review, b. identification of adverse histological features i. poor differentiation, ii lymphatic or venous invasion iii invasion into the stock of the polyp – formal resection It is difficult to locate the previous polypectomy site during surgery Even if polyp is not removed it may be soft and difficult to palpate through the colon wall 28 Endoscopic distance (from anal verge or dentate line) misleading Polypectomy site should be videotaped for later review and marked with vital dye that can be seen serosally at the time of surgery

3. Obstructing Cancers- 2% of colorectal cancers Partial obstruction – Gentle bowel preparation over several days-Elective surgery Total obstruction - Rt colon cancers – Rt Hemi colectomy – immediate ileocolostomy - Lt colon cancers

1) Endoscopic decompression by laser passed beyond the obstructed Segment – This allows mechanical preparation and elective resection. - This is possible only when the narrowed lumen can be traversed by the endoscope. - It is not possible when obstruction is complete
2) Primary resection and immediate anastamosis with ontable colonic washout with or without proximal colostomy.
3) Primary resection with colostomy. Anastamosis at second stage.
4) Subtotal colectomy with primary anastamosis
5) Decompressive colostomy followed by formal colonic resection

4. Adjacent organ involvement- 10% Locally advanced tumours are potentially curable with multi organ resection.-Do not necessarily Portend a dismal prognosis. -A non metastasizing variant of colon cancer grows to a large size without spreading to regional nodes -Separation of adhesions adjacent to a malignancy can lead to dissemination of tumour cells. 29 -Enbloc resection of these tumours, depending on location can lead to five year survivals of 70% Hepatic metastases – 10% at the time of exploration. -Solitary metastasis amenable to –wedge resection with clear margins can be removed concomitantly. -Formal hepatic lobectomy done as a second stage procedure.

5. Ovarian metastasis – 7% at the time of colon resection Oophorectomy: at the time of colorectal surgery Indications i) Large ovarian metastasis (Krukenbergt's tumour) which are symptomatic (prevents second surgery for the metastasis, benefit of preventing primary ovarian cancer) ii) Direct ovarian involvement iii) Post menopausal women – prophylactic oophorectomy 6. Inadvertent Perforation -Predisposes to local recurrence -Warrants post op radiotherapy Follow up Aim: Early detection of recurrence or metachronous lesion History Physical examination Faecal occult blood CBC } every 3 months- first 3 years LFT every 6 months additional 2 years 30 Tumour markers (CEA)

- monthly – 3 years, 3 monthly-next 2 years Colonoscopy – first colonoscopy within 6-12 months of surgery, yearly-next 2 years, 2-3 yearly thereafter. CXR CT abdomen and pelvis – if primary loco regionally advanced -LFT ↑ -CEA ↑ 80- 90% of recurrence of colon cancers occurs in the first two years. SOP All patients should be admitted when a colectomy is planned

WHO DOES WHAT?

Doctor: c) Surgeon: diagnosis & work up Pre operative planning Operative procedure Post operative follow up

d) Radiotherapist : radiotherapy – neoadjuvant & adjuvant

e) Medical oncologist : Chemotherapy

f) Anesthetist: PAC, anesthesia, post op ICU management

NURSE: • Siting of colostomy when required by some nurse • Care of stoma • Dressing of the wound • Pre & post operative care 31 TECHNICIAN: • Pre op equipment and drugs to be checked and kept ready • Assist anesthetist in the OT • Assist the surgeon, positioning of the patient

PEPTIC ULCER PERFORATION

INTRODUCTION: Lau and Leow have indicated that perforated peptic ulcer was clinically recognized by 1799, but the first successful surgical management of gastric ulcer was by Ludwig Heusner in Germany in 1892. In 1894, Henry Percy Dean from London was the first surgeon to report successful repair of a perforated duodenal ulcer. Wangensteen et al reported that in a patient with perforation but without evidence of pneumoperitoneum, one can safely assume that perforation has sealed off on its own. They advocated a nonoperative approach for such patients. However, they too supported operative treatment in patients with perforated ulcer and evidence of pneumoperitoneum. Berne and Donovan emphasized the use of a water- soluble upper GI study to demonstrate spontaneous sealing of the perforation. They demonstrated that as many as 40% of perforated peptic ulcers had no evidence of leak on upper GI contrast studies. Berne and Donovan concluded that these patients can be observed safely as long as peritonitis does not develop. Mortality rates were 6% and 3% in the operative and nonoperative groups, respectively. Donovan et al proposed dividing patients based on their Helicobacter pylori infection status and recommended nonoperative treatment in all patients except those without H pylori infection and those in whom prior treatment of H pylori infection had failed. Despite strong arguments favouring nonoperative treatment of patients with perforated PUD, delaying the initiation of surgery more than 12 hours after presentation was demonstrated to worsen the outcome. Therefore, when definitely indicated, a laparotomy should be performed as soon as possible..

SYMPTOMS

- Sudden, sharp and severe pain in upper abdomen
- Spreading of pain to rest of abdomen
- Pain gets worse after oral ingestion or movements
- Feeling of giddiness and fainting
- Fever
- Weakness

SIGNS

Tachycardia, Fever, Pallor, Reduced abdominal wall movements

INVESTIGATIONS

- Haemogram

- Liver Function Tests
- Blood sugar
- Serum creatinine
- Bleeding time, clotting time and prothrombin time
- Xray chest • ECG • USG abdomen • Upper GI endoscopy

MANAGEMENT

Resuscitation Fluid resuscitation should be initiated as soon as the diagnosis of peptic ulcer disease (PUD) is made. Essential steps include insertion of a nasogastric tube to decompress the stomach and a Foley catheter to monitor urine output. Intravenous infusion of fluids is begun, and broad-spectrum antibiotics are administered. In select cases, insertion of a central venous line or a Swan-Ganz catheter may be necessary for accurate fluid resuscitation and monitoring.

As soon as the patient has been adequately resuscitated, emergent exploratory laparotomy should be performed. Conservative Treatment Wangensteen et al reported that in a patient with perforation but without evidence of pneumoperitoneum, one can safely assume that perforation has sealed off on its own. They advocated a nonoperative approach for such patients. However, they too supported operative treatment in patients with perforated ulcer and evidence of pneumoperitoneum. Berne and Donovan emphasized the use of a water-soluble upper GI study to demonstrate spontaneous sealing of the perforation. They demonstrated that as many as 40% of perforated peptic ulcers had no evidence of leak on upper GI contrast studies. Berne and Donovan concluded that these patients can be observed safely as long as peritonitis does not develop. Mortality rates were 6% and 3% in the operative and nonoperative groups, respectively. Donovan et al proposed dividing patients based on their Helicobacter pylori infection status and recommended nonoperative treatment in all patients except those without H pylori infection and those in whom prior treatment of H pylori infection had failed. 55 Despite strong arguments favouring nonoperative treatment of patients with perforated PUD, delaying the initiation of surgery more than 12 hours after presentation was demonstrated to worsen the outcome.

Therefore, when definitely indicated, a laparotomy should be performed as soon as possible Surgical Treatment The appropriate surgical procedure depends on the location and nature of the ulcer. Many authorities recommend simple oversewing of the ulcer, with treatment of the underlying H pylori infection or cessation of nonsteroidal anti-inflammatory drugs (NSAIDs) for bleeding PUD.

Additional surgical options for refractory or complicated PUD include vagotomy and pyloroplasty, vagotomy and antrectomy with gastroduodenal reconstruction (Billroth I) or gastrojejunal reconstruction (Billroth II), or a highly selective vagotomy.

The patient is placed in the supine position. A midline incision provides the most expeditious entry into the abdominal cavity. The incision can be extended to the symphysis pubis if necessary. Once the abdomen is entered, the stomach and duodenum are carefully examined to determine the site of perforation. If the anterior surfaces of the stomach and duodenum show no abnormalities, the gastrocolic ligament is serially divided between clamps to allow entrance into the lesser sac and inspection of the posterior surface of the stomach. The choice of operative procedure depends on variables such as the presence of shock, the presence of life-threatening comorbid conditions, the degree of contamination of the upper abdomen, the amount and duration of perforation, and whether the patient has a history of, or currently has intraoperative evidence of, chronic peptic ulceration. In the presence of life- threatening comorbid conditions and severe intra-abdominal contamination, the safest technique for an acute anterior duodenal perforation is a simple closure with a Graham patch, using omentum. Several full-thickness simple sutures are placed across the perforation, using 2-0 or 3-0 silk sutures. A segment of omentum is placed over the perforation. The silk sutures are secured. If contamination of the upper abdomen is minimal and the patient is stable, a definitive ulcer procedure can be performed.

For a perforated duodenal ulcer, this may include a highly selective vagotomy, a truncal vagotomy and pyloroplasty, or vagotomy and antrectomy. For a perforated gastric ulcer, the procedure performed depends on the patient's condition. If the patient is moribund, the ulcer is best excised by grasping it with multiple Allis clamps and using a linear stapler. Alternatively, the ulcer can be excised with electrocautery; the defect is approximated with a 2- layer closure with inner continuous 3-0 absorbable sutures and outer interrupted Lambert sutures using 2-0 or 3-0 silk sutures. In a stable patient, the ulcer is excised and sent for frozen section analysis to exclude malignancy. For a benign gastric ulcer, a distal gastrectomy with either a Billroth I gastroduodenostomy or a Billroth II gastroduodenostomy is performed.

Post Operative Care & Complications

The nasogastric tube can be discontinued on postoperative day 2 or 3, depending on the return of GI function, and diet can be slowly advanced. Patients who are found to have H pylori infection should receive the appropriate antibiotic regimen. Patients

with high serum gastrin levels should undergo an evaluation for Zollinger-Ellison syndrome.

Patients should undergo upper endoscopy to evaluate the area of ulcer and healing of the perforation site 4-6 weeks after surgery. Surgical complications include pneumonia (30%), wound infection, abdominal abscess (15%), cardiac problems (especially in those >70 y), diarrhea (30% after vagotomy), and dumping syndromes (10% after vagotomy and drainage procedures).

REFERRAL CRITERIA:

- ICU care may be needed in patients who present late with severe sepsis and have other systemic illnesses.
- Patients with recurrence of perforation few days after surgery may need ICU care, parenteral nutrition, investigations for gastrinoma and further surgery.

MEDICOLEGAL:

- Failure to detect / investigate or refer a patient of suspected peptic ulcer perforation.
- Delay in treatment.
- Delay in diagnosing complications and taking corrective action. WHO

DOES WHAT?

Doctor: g) Surgeon: diagnosis & work up Pre operative planning Operative procedure Post operative follow up h) Anesthetist: PAC, anesthesia, post op ICU management

NURSE: • Dressing of the wound • Pre & post operative care 57

TECHNICIAN: • Pre op equipment and drugs to be checked and kept ready • Assist anesthetist in the OT • Assist the surgeon, positioning of the patient

SMALL BOWEL PERFORATIONS

I. Introduction: Small perforation is breach in seromuscular continuity of small intestine ie from D-J junction to ileocaecal junction. It can be single or multiple and of varying sizes depending on nature and stage of pathology causing it. It may even be associated with gangrenous segment of variable length of small intestine.

II. **Incidence** of the condition in our country In India, the commonest cause of small bowel perforation is enteric fever and tuberculosis. Rapidly increasing incidence of

vehicular trauma contributes to another category of perforation called traumatic perforation. Penetrating injury caused by knife, gunshot etc also adds to the etiology of these perforations. Rarely these perforations can be associated with long standing small intestinal volvulus or near the site of band compressing the gut causing ischemia and perforation. Iatrogenic perforations too can occur during conduct of various other abdominal operations and even gynecological operations.

III. **Differential diagnosis** The common conditions that should be considered in any patient presenting with features of peritonitis (apart from small bowel perforations):-

1. Acute Pancreatitis

2. Duodenal perforation

3. Appendicular perforation with peritonitis

4. Mesenteric vascular ischemia

IV. Prevention and counseling Timely medical advice and treatment for conditions like enteric fever and tuberculosis. In case of injury whether blunt or penetrating, seek hospitalization without any delay. Using of seat belts during travel (wherever possible) is also a good preventive step V. Optimal diagnostic criteria, Investigations, Treatment & Referral Criteria(Situation 1)

a. Clinical diagnosis

¬ Small bowel perforation is suspected clinically in any patient presenting with history of fever, trauma, abdominal pain, vomiting, distension of tummy, inability to pass flatus and feces of variable duration depending on type and duration of pathology. ¬ Clinical examination will reveal features of peritonitis which is mostly generalized but rarely may be localized also. Hippcratic facies will be present. The patient may be in shock (hypovolemic or septic) or may even be having septicemia at the time of presentation. 64

b. Investigations

¬ Plain X-ray abdomen in erect posture shows gas under one or both domes of diaphragm

¬ USG evidence of fluid collection showing internal echoes

¬ Abdominal paracentesis , not routinely but only where X-ray provides some doubt

¬ Additional serological tests like Widal may be carried out.

c. Treatment (Standard operating procedure)

i. In Patient ¬ Hospitalization followed by resuscitation followed by investigation followed by optimization for surgery which essentially consists of

- Laparotomy
- Closure of perforation/Ileostomy/resection and anastomosis depending on condition of the patient, condition of the bowel, location and multiplicity of pathology.(If single perforation with healthy bowel and condition of the patient is not bad , primary closure; otherwise ileostomy. In the event of multiple perforations with healthy bowel and good condition of the patient , resection and anastomosis ; otherwise ileostomy is advised . Once a while exteriorization may be considered if the condition of bowel and patient demands this procedure)
- Thorough peritoneal lavage
- Closure after providing adequate drainage tubes

Postoperative Care in ward and involves - I/V fluids, antibiotics, pain killers and monitoring - Oral allowance after bowel movements - Stitch removal at appropriate time

ii. Outpatient None

iii. Day Care None

d. Referral criteria ¬ If any of the facilities, infrastructure or expertise to carry out any of the above step is not available either at diagnostic level or treatment level or at the level of postoperative care, then the case must be referred to higher centre.

Optimal diagnostic criteria, Investigations, Treatment & Referral Criteria (Situation 2)

a. Clinical diagnosis ¬ Same as in situation 2

b. Investigations ¬ Same as in situation 1 plus CT scan and diagnostic laparoscopy if any equivocality is involved despite already mentioned investigations.

c. Treatment (Standard operating procedures)

i. In Patient ¬ Same as in Situation 1 with addition of Laparoscopic procedure in following situations: • Facilities and infrastructure available 65 • Expertise is available ¬ Post-operative care of the patient in HDU or ICU if the patient is unstable

.

ii. Out patient – None

iii. Day care – None

d. Referral criteria – None from situation 2

VI. Who does what and Timeliness?

a. Doctor ¬ The job of diagnosis, treatment including surgery, post-operative care and follow up.

b. Nurse ¬ Pre-operative care, operative assistance, post-operative care, administration of treatment instructed by the doctor and monitoring as instructed.

c. Technician ¬ Keeps all machines and equipments in order and assist the anesthetist during operation.

VII. Further reading a. Bailey & Love's Short Practice of Surgery b. Schwartz's Textbook of Surgery c. Abdominal Operations by Maingot

BLUNT ABDOMINAL TRAUMA

1. Name of the condition: Blunt abdominal trauma

2. When to suspect/ recognize?

a. Introduction: Blunt abdominal trauma (BAT) is an increasingly common problem encountered in the emergency department. The usual causes of BAT include vehicular accident, assault, falls, sports injuries and natural disasters.

b. Case definition: BAT is suspected in any patient involved in above situations and presents with abdominal pain, distention or shock. It should be looked for in patients of polytrauma.

3. Incidence of the condition: One study has reported 2.1% incidence of BAT amongst all surgical patients admitted to a tertiary hospital during 1 year.

4. Differential diagnosis: Abdominal trauma forms a differential diagnosis of any patient presenting with acute abdomen.

5. Prevention and counseling: Use of appropriate safety measures during various activities associated with BAT can significantly reduce its incidence.

6. Optimal diagnostic criteria, investigations, treatment and referral criteria:

I. Clinical diagnosis: This is based on

a. High level of suspicion of intra-abdominal injury

b. Presence of wounds/ bruising on the abdomen

c. Abdominal guarding/ tenderness

d. Presence of free gas/ fluid in the peritoneal cavity

e. Presence of fracture of lower ribs and/ or pelvis increases the likelihood of intraabdominal injury

f. Note should be made of altered mental state, drug or alcohol intoxication and distracting injuries which may mask the features of BAT

g. Repeated examination increases the accuracy of diagnosis

II. Investigations:

a. All hemodynamically stable patients with suspected BAT should undergo Focused Abdominal Sonography in Trauma (FAST)or Diagnostic Peritoneal Lavage (DPL)

b. Urgent laparotomy is indicated in patients with evidence of BAT who remain hemodynamically unstable despite initial resuscitation

III. Treatment (Standard operating procedure):

a. Inpatient:

i. All patients should have initial cervical stabilization and resuscitation, if required

ii. Initial fluid resuscitation should be done with 2L warmed Ringer Lactate solution infused rapidly through 2 peripheral lines

iii. A nasogastric tube and a Foley catheter should be put

iv. Laparotomy should be done, if indicated on the basis of clinical features, FAST or DPL

v. Laparotomy should be done through a long midline incision

vi. Bleeding should be controlled by clamping/ packing till definitive control is possible

vii. Hollow viscus should be repaired

viii. In case the intra-abdominal injuries are extensive, patient is very sick and OT facilities/ surgeon's experience is suboptimal, Damage Control Surgery may be done. Definitive surgery should be done subsequently under improved circumstances or at a higher center.

b. Outpatient: Not indicated

c. Day care: Not indicated

IV. Referral criteria: After Damage Control Surgery if the local facilities are inadequate.

OBSTETRICS & GYNAECOLOGY
STANDARD TREATMENT GU- IDELINES

OBSTETRICS & GYNAECOLOGY RECOMMENDATIONS

FOR ROUTINE ANTENATAL CARE

These recommendations have been developed with the following aims.

- They cover the clinical antenatal care that all healthy women with an uncomplicated singleton pregnancy should receive and baseline care for all pregnancies.
- It does not cover the additional care that women at increased risk of complications should be offered.

The Good Clinical Practice Recommendation does not, however, override the individual responsibility of health professionals to make decisions appropriate to the circumstances of the individual patient, in consultation with the patient and/or guardian.

The good clinical practice recommendations can be divided into two parts forcare of a routine antenatal patient

a) Basic essential care recommended for all pregnant women

b) Additional care and investigations to be preferably offered if available for routine antenatal care of normal healthy woman

The advantages of hospital delivery should be stressed upon. Ideally, all pregnant women must at least have a trained birth attendant.

Basic essential care recommended for all pregnant womenat all levels

- All pregnant women must be counseled for regular Antenatal visits minimum one visit in first trimester, monthly visits till 30weeks , every 2 weekly till 36 weeks and weekly visits till delivery
- Blood investigations for Hb, Blood grouping and Rh Typing,VDRL, Blood sugar -R, and a Routine Urine examination withalbumin & sugar should be done.
- A repeat Hb and Urine Sugar to be done in third trimester
- Immunization with 2 doses of Td/TT
- Iron, Folic Acid and Calcium Supplements
- At least one Ultrasound for congenital anomalies should bedone before 20 weeks of pregnancy. Delivery by a doctor or a trained birth attendant
- Education on nutrition, diet and hygiene
- Education in breast feeding and birth spacing andcontraception methods.

Additional care and investigations to be preferably offered if available for routine ante-natal care of normal healthy woman. Besides the basic essential ANC the following should be preferably offeredif easily available:

- Preconception counseling and care
- Counseling for HIV, HbsAg and HCV testing
- Counseling and screening for Thalassemia, Down'ssyndrome
- Repeat blood for Hb, Blood sugar screening and UrineEvoluation in each trimester
- Ultrasound evaluation once in each trimester and Institutional delivery recommended
- Additional screening for infections, growth retardation, thyroid dysfunctions

The care should be woman-centered care and informed decision making.

1. **Provision and organization of care**
   Who provides care?
   - Auxiliary Nurse Midwife (ANM) and I or doctor.
   - There should be continuityof care throughout theanfenatal period.
   - A system of clear referral should be there
   - A system of clear referral should be there
   - The antenatal record should be meticulous and systematic.
   - A schedule of antenatal appointments should be determined. -
2. **Gestational age assessment: IMP and ultrasound**
   - Pregnant women should be offered an early ultrasound scan to determinegestational age (in lieu of last menstrual period [LMP] for all cases) and to detectmultiple pregnancies whenever possible. This will ensure consistency ofgestafional age assessments, improve the performance of serum screening forDown's syndrome and reduce the need for induction of labour after 41 weeks.
3. **Lifestyle considerations**

- **Working during pregnancy**
  - Pregnant women should be informed of their maternity rights and benefits.
  - The majority of women can be reassured that it is safe to continue working duringpregnancy provided there are no medical or obstetrical complications.
- **Nutritional supplements**
  - Pregnant women (and those intending to become pregnant) should be

informed that dietary supplementation with folic acid, before conception and up to l2 weeks' gestation, reduces the risk of having a baby with neural tube defects(anencephaly, spina bifida). The recommended dose is 400 micrograms per day.
    - Iron, protein and calcium supplementation should be offered routinely to all pregnant women. This is because there is high incidence of anemia, hypoproteinemia and osteopenia in the Indian population due to poor diet and repeated pregnancies.
- **Prescribed medicines**
    - Prescription medicines during pregnancy should be limited to circumstances where the benefit outweighs the risk.
- **Exercise in pregnancy**
    - Pregnant women should be informed that a moderate course of exerciseduring pregnancy is not associated with adverse outcomes.
- **Sexual intercourse in pregnancy**
    - Pregnant woman should be informed that sexual intercourse in pregnancyis not known to be associated with any adverse outcomes.
- **Alcohol and smoking in pregnancy**
    - Due to increased fetal risks. It is suggested that women should avoidalcohol consumption when pregnant.
    - Pregnant women should be informed about the specific risks of smoking/tobacco use during pregnancy (such as the risk of having a baby with low birthweight, IUGR and preterm) and should be encouraged to quit.

- **Travel during pregnancy**
    - Travel is safe. However patient should be counseled regarding risks of longdistance travel, especially the risk of DVI with long flights.
    - Pregnant women should be informed that, if they are planning to travelabroad, they should discuss considerations such as flying, vaccinations and travelinsurance with their doctor.

4. Clinical examination of pregnant women

- **Measurement of weight**
    - Maternal weight and height should be measured at the first antenatalappointment, and the woman's BMI calculated.
    - Regular weight check during pregnancy should be done with everyANCvisit.
- **Blood Pressure Measurements**
    - Routine evaluation of blood pressure at every ANC visit and

moreimportantly, look for even minimal rise in BP or fluctuations. These are predictivefor the development of IUGR and PIH.
    - Large double cuff for obese women should be used for accurate readings.
- Pelvic examination
    - Routine antenatal pelvic examination does not accurately assessgestational age, nor does it accurately predict preterm birth or cephalopelvicdisproportion. It is recommended when ultrasound facilities for gestational age arenot available.
- **Abdominal examination**
    - Every ANC visit after the first trimester should include abdominalexamination for checking that thèuterine size is corresponding with period ofgestations. Early IUGR can be detected by inappropriate growth. Multiplepregnancy, hydramnios etc can be suspected if there is excessive growth.
- **Domestic violence**
    - In our country, there is a high incidence of domestic violence, even whenthe woman is pregnant. Healthcare professionals need to be alert to the symptomsor signs of domestic violence and women should be given the opportunity todisclose domestic violence in an environment in which they feel secure.

5. Screening for hematological conditions

- **Anaemia**
    - Pregnant women should be offered screening for anaemia.Screening should take place early in pregnancy (at the first appointment) and at 28weeks when other blood screening tests are being performed. This allows enoughtime for treatment if anaemia is detected. Haemoglobin levels outside the normalrange for pregnancy (that is, 10 g/dl at first contact) should be investigated.
    - In our country there are ethnic groups who are at risk for Thalessemia.Whenever possible testing for it and evaluation of the fetus if needed should beoffered
- **Blood grouping**
    - Women should be offered testing for blood group and RhD status in earlypregnancy.
    - It is recommended that post partum anti-D prophylaxis is offered to all nonsensitizedpregnant women who are RhD negative.
    - Women should be screened for Rh antibodies at first visit and again at 28weeks and if positive they should be offered referral to a specialist centre forfurther investigation and advice on subsequent ANC.

6. Screening for fetal anomalies

- **Screening for structural anomalies**
    - Pregnant women should be offered an ultrasound scan to screen

forstructural anomalies, preferably between 18 and 20 weeks' gestation. Thereshould be care taken to adhere to all aspects of the PC-PNDT Act and under nocircumstances should the pregnant lady be informed about the sex of the child.

- **Screening for Down's syndrome**
  - Pregnant women may be offered screening for Down's syndrome with atest which provides the current standard of a detection rate above 60% and afalse-positive rate of less than 5%. The following tests meet this standard:
    - From lito 14 weeks
      - Nuchal translucency (NT)
      - The combined test (NT, hCG and PAPP-A)
    - From l4to20weeks
      - The triple test (hCG, AFP and uE3)
      - The quadruple test (hCG, AFP, uE3, inhibin A)
    - From 11 tol4weeksandl4to20weeks
      - The integrated test (NT, PAPP-A + hCG, AFP, uE3, inhibin A)
      - The serum integrated test (PAPP-A + hCG, AFP, uE3, inhibin A).

These tests are recommended wherever possible, and not mandatory as theremay financial and logistic problems in these tests being made availableeverywhere, especially in remote rural areas.

7. Screening for infections

- Asymptomatic bacteriuria
  - Pregnant women should be offered routine screening for asymptomatic bacteriuria by midstream routine urine examination. Urine culture should be askedwhere indicated. Identification and treatment of asymptomatic bacteriuria reduces the risk of preterm birth.
- HBsAg
- HIV
- VDRL

- **Bacterial Vaginosis**

- Recurrent vaginal infections and high incidence of preterm labour are interlinked, and hence whenever possible and feasible pregnant women should have a vaginal smear to r/o possibility of bacterial vaginosis.

8. Screening for clinical conditions

- **Pre-eclampsia**

  - At first contact a woman's level of risk for pre-eclampsia should be evaluated so that a plan for her subsequent schedule of antenatal appointments can be formulated. The likelihood of developing pre-eclampsia during a pregnancy is increased in women who:
    - are nulliparous
    - are aged 35 or older
    - have a family history of pre-eclampsia - have a prior history of pre-eclampsia - obese women
    - have multiple pregnancy or pre-existing vascular disease (e.g. hypertension or diabetes).

  - With every ANC visit, blood pressure is measured in pregnancy and if possible, urine sample should be tested at the same time for proteinuria.
  - Pregnant women should be informed of the symptoms of advanced preeclampsia because these may be associated with poorer pregnancy outcomes for the mother and/or baby or both. Symptoms include headache; problems with vision, such as blurring or flashing before the eyes; abdominal pain just below the ribs; vomiting and sudden swelling of face, hands or feet.

- **Gestational diabetes mellitus**

  - Ideally, every pregnant woman must be offered routine screening for gestational diabetes mellitus by blood sugar estimations in every pregnant woman. However, financial and logistic problems may not be able to support this on routine basis. Hence, a urine sugar examination during ANC visit will help in identifying normal women at risk. Also, identify women with risk factors and these women should be screened thoroughly.
  - Whenever possible, a glucose challenge test using 75gm glucose load, is the ideal method of screening for gestational diabetes.

- **Thyroid deficiency**
  - In our country thyroid deficiency is endemic in many areas, especially in the northern regions. Thyroid screening should preferably -be done at least once, especially in all pregnant women hailing from these areas.

9. Immunization during pregnancy

- All pregnant women should be immunized against tetanus and diphtheria (Id Vaccine) as per the recent WHO guidelines. However, places where Td is not available, immunization should be with IF.
- 2 doses of Td/ TT should be given 4-6 weeks apart to all pregnant women during the ANC period after 16 weeks onwards.
- Rubella, Yellow Fever and all vaccines with live virus should be avoided.

10. Diet and hygiene during Pregnancy

- Adequate information of a balanced diet should be provided to all pregnant women.
- Pictorial charts and if possible suggested nutrients should- be given to all pregnant women with advice on improvements in daily diet needs.
- Care should be taken to see that there is enough, proteins, carbohydrates, calcium, iron and fats in the daily diet and if not, the woman should be advised appropriately by the ANM's or the doctor.
- Pregnant women should be informed of primary
- infection prevention measures, such as:
    - Washing hands before handling food
    - Thoroughly washing all fruit and vegetables before eating
    - Thoroughly cooking raw meats and fish.
    - Wearing gloves and thoroughly washing hands after handling soil and farming
    - Avoiding cat/cow faeces in litter or in soil.

11. Fetal growth and well-being

5. Abdominal palpation for fetal presentation
6. Measurement of symphysis-fundal distance
7. Auscultation of fetal heart
8. Routine monitoring of fetal movements

- All pregnant women should be fold about the importance of fetal movements. They should be advised to report to the ANM or the doctor if they donot feel any movements for 12 hours or more.

12. Management of common symptoms of pregnancy

- **Nausea and vomiting in early pregnancy**
    - Women should be informed that most cases of nausea and vomiting in pregnancy will resolve spontaneously by end of first trimester and that nausea and vomiting are not usually associated with a poor pregnancy outcome. If a woman requests or would like to consider treatment, non-pharmacological

agents or safe anti-emetics. In hyper-emesis hospital admission may be needed.

- **Heartburn**
  - Women who present with symptoms of heartburn in pregnancy should be offered information regarding lifestyle and diet modification.
  - Antacids may be offered to women whose heartburn remains troublesome despite lifestyle and diet modification.
- **Constipation**
  - Women with constipation in pregnancy should be offered information regarding diet modification, such as. is a pgol (psylium husk) supplementation and medication if needed.
- **Hemorrhoids**
  - In the absence of evidence of the effectiveness of treatments for hemorrhoids in pregnancy, women should be offered information concerning diet modification. If clinical symptoms remain troublesome, standard hemorrhoid creams should be considered.

I. **Varicose veins**
  - Women should be informed that varicose veins are a common symptom of pregnancy that will not cause harm and that compression stockings can improve the symptoms but will not prevent varicose veins from emerging.
- **Vaginal discharge**
  - Women should be informed that an increase in vaginal discharge is a common physiological change that occurs during pregnancy. If this is associated with itch, soreness, offensive smell or pain on passing urine there may be an infective cause. In these cases, evaluation and treatment should be considered.
  - A 7 day course of a topical co-trimazole is an effective treatment and should be considered for vaginal Candidal infections in pregnant women.
- **Backache**
  - Backache is a common problem which only increases as the pregnancy advances. Women should be informed that exercising in wafer, massage therapy and group or individual back care classes might help to ease backache during pregnancy.

13. Education on breast feeding and infant care

- When ever possible all pregnant women should be taken around the post-natal ward and allowed to interact with just delivered women to under stand and be prepared for normal labour.
- When ever possible the pregnant women should be taught how to breast feed their babies and to look after the hygiene.
- The proper care to be followed after breast feeding, burping the in fantand how to position the infant when sleeping should all be taught to the women during

the ANC period itself.

- Pre-pregnancy classes on labour and infant care can be offered whenever possible.

14. Contraception and birth spacing

- The importance of birth spacing should be stressed and they should be informed about all the methods that can be safely used during the post-partum period when they are breast feeding their babies. Effective contraception with risks, advantages and benefits must be explained. They should also be explained about the difference between spacing and permanent methods.
- At the first post-natal visit, IUCD / Injectable contraceptive / POP/Implant / preference for IL should all be offered as the basket of choices. All these should be offered with adequate counseling and proper selection according to the WHO criteria for each method. Importance of LAM should be stressed for all women and breast feeding should be encouraged.

Schedule and content of Antenatal appointments

The schedule below, which has been determined by the purpose of each appointment, presents the recommended number of antenatal care appointments for women who are healthy and whose pregnancies remain uncomplicated in the antenatal period

Focused antenatal care (ANC):The four-visit ANC model outlined in WHO clinical guidelines

Goals

First Visit 8-12 weeks	**Second Visit 24-26 Weeks**	**Third Visit 32 Weeks**	**Fourth Visit 36-38 Weeks**
Comfirm pregnancy and EDD, Classify women for	**Assess** Maternal and fetal well -being Exclude PIH and anaemia.	**Assess**maternal And fetal well-being. Exclude PIH anaemia, multiple	**Assess** maternal and fetal well-being Exclude PIH,

basic ANC (four visits) or More specialized care. Screen, treatand give preventive measures Develop a birth and emergency plan. Advise and Counsel.	Give preventive measures Review and Modify birth and emergency plan. Advice and counsel.	pregnancies Give preventive measures. Review and modify birth and emergency plan. Advice and counsel.	anaemia, multiple pregnancy, malpresentation. Give preventive measures. Review and modify birth and emergency plan. Advise and counsel.

Activities

Rapid assessment and management for emergency signs, give appropriate treatment, and refer to hospital if needed,

History (ask, Check records)	Assess significant symptoms.	Assess significant symptoms.	Assess significant symptoms.	Assess significant symptoms.
	Take psychosocial medical and obstetric history. Comfirm pregnancy and calculate EDD. Classifyall	Check record for previous complications and treatments during the pregnancy. Re- Classification If needed	Check record for previous complications during the pregnancy. Re-classification If needed	Check record for previous complications and treatments during the pregnancy. Re-Classification If needed

	Women (in some cases after test Result)			
Examination (look,listen,feel)	Complete general, and obstetrical examination BP	Anaemia,BP, fetalgrowth, and movements	Anaemia,BP, fetalgrowth,multiple pregnancy	Anaemia, BP, fetal growth and movements, multiple pregnancy, malpresent-ation
Screenning and tests	Haemoglobin Syphilis HIV Proteinuria Blood/Rh group* Bateriuria*	Bateriuria*	Bateriuria*	Bateriuria*
Treatments	Syphilis ARV if eligible treat bateriuriaif indicated*	Anithelmin Thic**, ARV if eligible Treat bacteriuria if indicated*	ARV ifeligible Treat bacteriuria if indicated*	ARV if eligible if breech, ECV or referral for ECV Treat bateriuria if indicated.
Preventive measure	Tetanus toxoid Iron and folate +	Tetanus toxoid, Iron and folate IPTp	Iron and folate IPTp ARV	Iron and folate ARV
Health eduction advice and counseling	Self-care, Alcohol and tobacco use, nutrition, safe sex, rest, sleeping under ITN, birth and	Birth and emergency plan, reinforcement of previous advice	Birth and emergency plan, infant feeding, postpartum /postnatal care, pregnancy spacing,	Birth and emergency plan, infant feeding, postpartum/ postnatal care, pregnancy

	emergency plan		reinforcement of previous advice	spacing, reinforcement of previous advice

The patients with high risk pregnancy should be referred to LEVEL 3 or LEVEL 4 for better management and care;

- Elderly primi (30 yrs and over)
- Short stature (< 140 cms)
- Malpresentation (breech, transverse lie)
- APH, Threatened abortion
- Pre-eclampsia, Eclampsia, IUGR
- Anaemia
- Twins, Hydramnios
- Previous still birth, IUD, MROP
- Grand multipara/Rh incompatibility
- Postdate pregnancy
- Previous caesarean or instrumental delivery
- Pregnancy with heart disease, renal disease, diabetes, tuberculosis

Investigations to be done in high risk pregnancies

- In Rh -v e mother (Rh +ve father)
- ICTat 1st visit, 28 wks and 34 wks
- In BOH TORCH - 1gM and lgG
- Anti phospholipid antibody— 1gM and IgG
- Down syndrome: Triple test at 15 wks of pregnancy (MSAFP, B-HCG & urinary 03)
- CVS, amniocentesis & cordocentesis for various chromosomal fetal malformations

Nausea and Vomiting of Pregnancy

- Morning sickness in pregnancy is common medical condition where there is mild nausea and vomiting.
- Excessive and severe form of morning sickness during pregnancy is called as Hyperemesis Gravidarum.

CAUSES

- Hormones Excess of HCG and Oestrogens
- Psychogenic Emotional factors.
- Dietary deficiency BI BóVitamins,

- Decreased gastric motility are the more common theories.
- "HG" Hyperemesis Gravidarum is more common in first pregnancy, first trimester, and with positive family history, multiple gestation and, H Mole,
- Gynaecological Twisted Ovarian cyst Red degeneration of fibroid uterus, Fatty liver of pregnancy.

TREATMENT OF NAUSEA AND VOMITING, (Valid for all levels of health care)

- Most cases of nausea and vomiting are mild and do not require any treatment. It usually resolves with sixteen to twenty week and is not associated with a poor pregnancy outcome. However persistent vomiting and severe nausea can progress to Hyperemesis Gravidarum, if the woman is unable to maintain adequate hydration, fluid and electrolyte balance as nutritional status may be jeopardized. Hospital admission becomes necessity and intravenous fluid administration is urgently required.
- Aim of treatment in Nausea and Vomiting
- Dietary intake,
- Life style modifications and
- Reassurance should be encouraged and women should be counselled to eat whatever they like to take.
- To feel better, eat and drink enough so that she does not loose weight. • Treatment may not eliminate nausea and vomiting completely. • Dietary changes
- Avoiding food and not eating at all, actually makes the nausea worse.
- Try eating before or as soon as you feel hungry, to avoid any empty stomach which aggravates nausea.
- Eat snacks frequently and have small meals six small meals a day. Rich in proteins, and carbohydrates, and low in fat. Brushing of teeth after eating may help preventing symptoms.
- Drink cold, clear and carbonated or sour fluids, Ginger and lemon fluids.
- Avoid triggers
- Avoid spicy foods, High sugar foods, perfumes, chemicals and coffee and smoke. Visual and physical motions flickering lights and driving,

MEDICATIONS

- Medications that reduce the nausea and vomiting are definitely effective in some women and are safe to take during pregnancy.
- Doxylamine Succinate 10 mgs is an Hi receptor antagonist found to be effective in treatment of NV of pregnancy. Pyridoxine 10 mgs vitamin B6 can reduce the symptoms of mild to moderate nausea.
- Anti-histaminesare safe for nausea and vomitingcommoniy used but causes drowsiness.

- Promethazine (Phenergan) 25mgs injectable 2-3 times intramuscularly thrice a day. May cause. drowsiness and dry mouth.
- Metoclopramide (Region) It speeds of emptying of stomach and thus may help to reduce nausea and vomiting.
- Vitaminsvit Bi, B6, B12, vit C as nutritional supporti 00 mgs i/v in drip. • Intravenous fluids
- Fluids and Nutrition If unable to hold down food or liquids, treatment will have to start with intravenous fluids.
- Level 3(100 bedded Community Health Center)
- If the patient's symptoms are not relieved or diagnosis is not confirmed and the patient is sick refer to level no. 3 to get the ultrasound of pelvic organs and whole abdomen and treatment of the medical causes like gastroenteritis and cholecystitis.
- Level 4(100 or more bedded District Hospital)
- All patients with complications or patients having surgical causes should be referred to level 4 for treatment of complications like diabetic ketoacidosis or electrolyte imbalance and ureamia and other surgical treatment.

Complications

- Neurological serious complications like Wernick's encephalopathy, peripheral neuritis, central pontine myeiinolysis, rupture of oesophagus and jaundice can occur.

Prevention

- Early, energetic and effective management of simple vomiting of pregnancy can reduce the further development of Hyperemesis Gravidarum.

THREATENED ABORTION

It is a clinical entity where the process of Abortion (Less than 20 weeks period of gestation) has started but further progression can be averted and pregnancy can be continued.

Clinical Features

Symptoms

1) Amenorrhea depending upon the duration of pregnancy.
2) Bleeding per vaginum usually slight and bright red in colour. It usually stops spontaneously.
3) Pain may appear after bleeding. Dull pain in lower abdomen or mild back ache.

Management at level I To Ill

Level 1: Solo Physician Clinic

Level 11:6-10 Bedded PHC.

Level Ill: 100 Bedded Community Health Centre.

Per vaginal examination

Gentle p/v is performed to see the state of cervix, uterine size, adenexal tenderness or any mass. The external or is closed in threatened abortion.

Per speculum examination

- Insped vagina and cervix for amount and source of bleeding.
- Any local lesion of cervix orvagina.
- Any product of conception coming out through the external os
- Any discharge.

General physical examination

- General condition of patient and her nutritional status should be assessed.
- Pallor, temperature, pulse, bp should be assessed.
- Systemic examination for cardiovascular, respiratory or other systems should be done.
- Per abdominal examination for any distension, mass, tenderness, size of uterus whether corresponding to the period of amenorrhea or not.

Laboratory investigations

- Urine for pregnancy test - positive
- Hemoglobin estimation, BT, CT, TLC/DLC
- HIC, HCV, HBsAg status
- Blood group - ABO and Rh typing,
- Serum progesterone levels- option test A level of more than 25ng/ml usually indicates a viable pregnancy.

Ultrasonography

In early pregnancy Transvaginal sonography is better than transabominal sonography. It confirms the diagnosis of pregnancy, localization of pregnancy (Intrauterine I Extrauterine) and cardiac activity depending upon the period of gestation. It demonstrates subchorionic haemorhage if any and position of the placenta. It also rules out molar pregnancy. If cardiac activity and yolk sac present in ultrasound - 98% of cases of pregnancy continue till term.

Differential Diagnosis

1) Ectopic gestation - In ectopic gestation there may or may not be history of amenorrhea, with occasional attacks of colicky pain in lower abdomen and vaginal bleeding. There is tenderness or a tender mass in adenexa. Ultrasound examination confirms the diagnosis. UPT may be negative or positive.
2) Ovarian torsion - Here UPT is negative.
3) In evitable abortion - The as is open.
4) In incomplete abortion - The os open with products of conception lying in uterine cavity or cervical canal.
5) Functional menstrual disturbance. UPT is negative bimanual examination reveals absence of signs of pregnancy.

Management

- Hospitalize the patient.
- Patient should be counselled that prognosis is excellent with majority of pregnancies i.e. 80-90% cases.
- Advice rest to patient and limit the activities. Ask the patient to save her pads to know about amount and character of bleeding or any products of conception and clots.
- Sedation with Injection 25mg promethazine l/M4-6 hourly.

There is no conclusive evidence of the role of progestogenis but they are given on emerica I basis.

1) Natural Micronized progesterone 200mg BD Oraly OR 1 00m BDvaginaly.
2) Tab Dydrogestrone 10mg BD.
3) Tab Allyl Strenol 5mg TDS.
4) injection 17-alpha hydroxyprogesterone caproate (PROLUTON DEPOT) 250-500mg I/M weekly for V'half of pregnancy.

5) Inj. HCG-2000IU l/Mtwiceaweektill3 months.
6) Anti-D prophylaxis if patient is Rh negative.
   In Gestational Age <1 2weeks Inj. Anti-D 50g IM In

   Gestational Age >1 2weeks ml. Anti-D 300g IM

7) Hematinics and calcium to be added along with folic acid if gestational age is >12 weeks.
8) If there is no bleeding for 48 hours, patient can be discharged with advice to take rest at home.

Advice on discharge and follow up

- Patient should limit her activities for at least 2 weeks, avoid coitus and heavy work.
- Follow up after 1 month to assess the growth of fetus by ultrasound.

Late complications

fetal - increased incidence of prematurity, fetal growth restriction and perinatal death in fetus.

Maternal-increased maternal risk of antepartum haemorrhage, manual removal of placenta and placenta previa and caesarean section in women with threatened abortion.

Management of Level IV: 100 or more bedded district hospital.

- Management of threatened abortion is same as mentioned before
  Following patients are referred to level - IV Health Institution :-
- In cases of continued bleeding inspife of giving adequate management of threatened abortion.
- Inevitable abortion - In complications like inevitable abortion. P/V is done the clots and products can be seen protruding through the cervix along with bleeding.
- If ultrasonography is not possible at lower levels.
- Patients complaining of acute colicy pain in lower abdomen and features of shock to rule out ectopic pregnancy.
- If patients is severely anaemic and needs blood transfusion. Anaemia is corrected with packed red blood cells to a haemoglobin of 8gm/dI.
- If patient is in state of shock due to excessive haemorrhage. Adequate volume resuscitation should be done. Vasopressors may also be added (e.g. Dopamine and nor epinephrine).

SEPTIC ABORTION

Septic abortion, an infected abortion complicated by fever, endometritis, and paramefritis, remains one of the most serious threats to women's health worldwide. Although morbidity and mortality from septic abortion are infrequent in countries in which induced abortion is legal, suffering and death from this process are widespread in many developing countries in which abortion is either illegal or inaccessible. Septic abortion is a paradigm of preventive medicine.

The World Health Organization (WHO) estimates that 21.6 million unsafe abortions occur each year and that 47,000 deaths from unsafe abortion occur in the world every year.

Diagnostic criteria, investigations, treatment and referral criteria: -

AT LEVEL 1 (SOLO PHYSICIAN CLINIC) & LEVEL 2(6-10 BEDDED PHC)

After suspecting a case of septic abortion, the following investigations are done and treatment started simultaneously.

Investigations include the following: -

- Routine investigations are mandatory to know the status of patients
- Haemoglobin, blood group& Rh status, BT,CT.
- Urine complete and microscopic examination.
- Temperature monitoring
- Renal function tests(blood urea, serum creatinine)
- Blood sugar levels.
- HIV, HBsAg, HCV status.
- Total leucocyte count & differential leucocyte count(TLC&DLC)
- Ultrasonography abdomen & pelvic organs.
- X-ray chest & X-ray abdomen.

Treatment:-

- Confirm diagnosis of pregnancy with urine pregnancy test.
- Perform physical examination.
- Perform pelvic examination with attention to uterine size and position, other pelvic pathology.
- Obtain ultrasound examination.
- IN line access to be maintained. Provide prophylactic antibiotics:

REGIMEN A:-

i. Injection aqueous penicillin 5 million units i/v 6 hourly after sensitivity test or injection ampicillin 500 mg to 1gm i/v 6 hourly(to cover gram positive bacteria)
ii. Injection gentamicin 60-80 mg i/v 8 hourly after ruling out renal failure(to cover gram negative bacilli)
iii. Injection metronidazole 500mg i/v 8 hourly(to cover anaerobes)

REGIMEN B:-

I. Injection ciprofloxacin 500mg i/v 12 hourly
II. Injection metronidazole 500mg i/v 8 hourly

REGIMEN C: FOR MORE SEVERE INFECTIONS

I. Injection cefotaxime 1 gm i/v 12 hourly or injection ceftriaxone 1 g i/v 12 hourly.
II. Injection metronidazole 500mg i/v 8 hourly.
III. Injection gentamicin 60-80 mg i/v or i/rn 12 hourly.

- **Psychological support.**
- **Refer to higher center in severe cases for further management.**

LEVEL 3: 100 BEDDED COMMUNITY HEALTH CENTRE:

In addition to investigations already mentioned, following investigations to be added:

- Liver function teste.
- Platelet count & PTI
- Serum electrolytes
- Urine & vaginal cultures.
- Blood culture
- Input output charting

Treatment :-

In addition to the treatment outlined above for level 1 & level 2, following measures can be instituted:

- Prophylactic anti gas gangrene serum (8000 units) and anti tetanus serum (3000 units) intramuscularly are given if there is history of interference.
- Oxygenation by face mask in severe cases.
- Uterine curettage: if patient's condition is stable, within 1 hour of antibiotic therapy, evacuation of the uterus by gentle curettage to remove infected products. If general condition is low at admission, curettage after 6-8 hours of antibiotic therapy and treatment of hypovolemia is done.

Treatment of complications: -

- A rapid initial assessment is needed to determine the severity of the problem. If the patient has been symptomatic for several days, more generalized, serious illness may be present.
- with more advanced gestations, there is greater risk of perforation and of retained tissue. Perforation markedly increases the risk of serious sepsis. the abdominal and pelvic examinations merit special attention.
- Ideal management is immediate re-evacuation in the ambulatory clinic or the emergency room.
- outpatient management of pelvic inflammatory disease is appropriate for patients with early postabortal infection limited to the uterine cavity in addition to uterine evacuation. One such regimen is ceftriaxone 250 mg by intramuscular injection (or other third generation cephalosporin such as cefoxitin, ceftizoxime, or cefotaxime) plus doxycycline 100 mg orally twice a day for 14 days, with or without metronidazole 500 mg orally twice a day for l4 days.

Level 4: 100 or more bedded district hospital:-

Along with management mentioned above, we have to avoid serious consequences of infection, including hysterectomy and death.

- One time-honored regimen for severe pelvic sepsis is penicillin (5 million units intravenously [iv] every 6 hours) or ampicillin (2 g iv every 6 hours) combined with gentamicin (2 mg/kg loading dose, followed by 1.5 mg/kg every 8 hours or 5 mg/kg every 24 hours depending on blood levels and renal status).
- Laparotomy will be needed if the patient does not respond to uterine evacuation and adequate medical therapy. Other indications are uterine perforation with suspected bowel injury, pelvic abscess, and clostridial myometritis. Although ultrasound-guided percutaneous needle aspiration of

pelvic abscesses is practiced, in critically ill women with severe postabortal sepsis, hysterectomy will likely be needed in addition to drainage of any abscess.

- Patients with severe sepsis and septic shock should be managed in intensive care settings in collaboration with physicians and nurses trained in critical care medicine. Principles of management are aggressive source control with antibiotics and early hemodynamic resuscitation.
- Vasopressors are added if the patient remains hypotensive despite adequate volume resuscitation or in patients who develop cardiogenic pulmonary edema. Norepinephrine should be the first line agent used for the management of refractory hypotension in the setting of septic shock followed by dopamine.
- Anemia is corrected with packed red blood cells to hemoglobin of 8 g/dl. Fresh frozen plasma, platelets or cryoprecipitate should be used only when there is clinical or laboratory evidence of coagulopathy.

Conclusion:-

Death and serious complications from abortion-related infection are almost entirely avoidable.

ANTE - PARTUM HAEMORRHAGE

Definition--All cases of bleeding from any part of the genital tract after 20 weeks (24 weeks for developing world) of pregnancy but before the birth of the baby.

Causes

Mainly 3 categories:

- Placental cause(more common 70%incldes placenta previa and abruption placenta)
- Extra-placental causes(cervical polyp, cacervix, varicosevein, local trauma)
- Unexplained orindeterminate

Placenta previa

Clinical types:mild degree (type 1, 2)major degree (type 3,4)

Type 2 posterior placenta previa is dangerous because the major thickness of placenta overlies the sacral promontory, so decreases the a.p diameter of inlet and prevents engagement of presenting part.

Symptoms

Recurrent, painless and causeless vaginal bleeding is the hallmark of placenta previa.

Signs:

- The size of uterus corresponds to pog.
- The uterus in normally relaxed and soft, so the fetal parts are easily palpable and no area of tenderness is found
- In about 1/3 rd cases malpresentations are observed, more commonly breech & transverse. Twin pregnancy is frequent.
- The presenting part is usually high or floating in major degree & posterior placenta previa.
- If the placenta is lateral or marginal, the presenting part may be just at the pelvic brim or even get fixed in early labour.
- Fhs is present if bleeding is small. However if major amount of placenta is separated & the patient is in shock fhs may not be present.

- In case of posterior placenta previa, fetal heart may become slow or irregular if fetal head is forced into the pelvis due to compression of placenta. (stallworthy sign)..

Vulval examination- only inspection is to be done to note the amount of bleeding & color of blood. The blood is bright red in placenta previa.

***refer patients of aph to tertiary care hospital for further management.**

Vaginal examination should never be done

Diagnosis ultrasound localisation of placenta is done as a routine usg in the second trimester of pregnancy or may be done as a diganostic procedure in a case of aph, this is the most accurate method to localise the placenta.

Colour-doppler prominent venous flow in the hypoechoic areas near the cervix indicates placenta previa.

Per speculum examintation should be done.

Management

- These days the localisation of placenta on routine usg has made the management easy. If placenta is in lower segment the patient is advised bed rest ,avoid travelling,to avoid intercourse and to avoid heavy work
- Gentle abdominal examination to note the height of uterus, area of uterine tenderness and f.h.s. Diagnosis is made by history, physical examination and confirmed by ultrasound
- Management protocol mainly depends on the maternal condition and gestation is divided into two groups
  -conservative or expectant

  -definitive or active

Conservative

- also known as johnson and macafee protocol
- this comprises judicious non interference and extensive monitoring
- it is based on the understanding that

1) in most cases the first bleeding occurs when the fetus is premature
2) the first bleeding is seldom fatal to the mother and fetus
3) the bleeding often stops on its own, to recur later at indefinite intervals

- The main objective is to allow the pregnancy to continue at least for 37 weeks to improve fetal prognosis
- Approximately threedays after all bleeding has stopped a gentle speculum examination should be performed to rule out any local cause of bleeding.
- 2 doses of betamethasone 12 mg j/m 12 hours apart are given for fetal lung maturation.
-

- the expectant treatment should be discontinued

-37 weeks of pregnancy are completed

-severe bout of bleeding occurs

-the patient goes into labour

-if the fetus is dead or congenitally malformed

Definitive management

Comprises prompt delivery:

- the patient has her first episode of bleeding very severe

-the first bout of bleeding is at or after 37 weeks of pregnancy

- successful conservative treatment brings the patient upto 37 weeks

-the patient is in labour

-the fetus is dead or congenitally malformed

Usg localises the placenta and also defines the extent to which the placenta covers the as thereby predicting the likelihood of vaginal delivery or c.s. Vaginal delivery in type

1 and type 2 anterior placenta previa Ceasearen section: major degree placenta previa (3 and 4) and type 2 posterior, the lscs is the treatment of choice

Accidental hemorrhage

Defined as separation of normally situated placenta after 20 weeks of pregnancy (24 weeks in developing world) but before the birth of the child

3 varieties

- Revealed: the hemorrhage is external and is most common, occurs in 80% cases.
- Concealed: hemorrhage is intrauterine in which no hemorrhage occurs at the vulva. it is rare.
- Mixed: some blood is concealed and some is revealed at the vulva.

The patient may present with variable degrees of shock and acidosis.

Lab tests -

Bleeding time: 2-4 mins

Clotting time (3-8 min if the clotting time is prolonged, it indicates deficiency of clotting factors. Absence of clotting indicates fibrinogen levels less than 50 microgm/dl.

Clot retraction time: normal is 30 mins. A weak friable clot indicates hypofibrinogenemia and early dissolution indicates enhanced fibrinolysis. Peripheral smear and platelet count: peripheral smear shows thrombocytopenia, leucocytosis and evidence of haemolysis like schistocytes, platelet count

Special investigations (for tertiary care)

Prothrombin time: normal is 11 -1 7 seconds. It tests the integrity of extrinsic and common pathway. It is prolonged in deficiency of factors i, ii, v, vii orx .the test is most sensitive to a fall in factor vii which is one of the vitamin k dependent factors.

Partial thromboplastin time: normal 25-35 seconds. It tests the integrity of extrinsic and common pathway.

Thrombin time: normal is 10- 15 sec

Fibrinogen degradation products (fdp's): in normal pregnancy usually they are absent. They are elevated and can be detected in dic.

D-dimer assay: it is specific component of fibrinogen breakdown. Levels more than 200 mg/I can be found in dic.

Coagulation inhibitors: low plasma levels of coagulation inhibitors like antithrombin iii, protein c may help in diagnosis and to determine the prognosis.

Clinical features

Clinical features are dependent on the degree of placental abruption.

- the uterus is hard and eventually tender to touch, that the patient resents even gentle palpation.

- uterine height is more than period of gestation. - thefetus is most often dead and fhs is inaudible.

- the systolic BP is at shock levels below 80mm of Hg and pulse very rapid.

- sometimes in patients with hypertension the systolic pressure may be normal but they could be in a state of shock as the pressure would have been much above the normal before abruption. This factor has to be borne in mind.

- coagulation failure and renal failure are more often associated with this type.

- all cases of concealed accidental hemorrhage fall under this category.

Diagnosis

Ultrasonography for diagnosing placental abruption

Management of abruptio placentae

- Treatment of abruption depends on maternal and fetal condition, gestational age and cervical status.
- These patients should be managed in a well equipped hospital with medical team and resources capable of delivery with all the complications of abruptioplacentae like coagulopathy.
- A quick assessment of the general condition of the patient is made by checking the vitals,
- The two important criteria are-
- to keep the hoematocrit at least 30% • urinary output at least 30 mt/hour

- Unless there is malpresentation every effort should be made to deliver the patients with abruptio placentae, and fetal death vaginally with careful attention paid to coagulation status during labour induction.
- Third stage must be managed activelyand preparation must be made to deal with PPH if it occurs.
- Measure urinary output .Watch for renal failure and clotting defects which in some appear after delivery for the first time.

Expectant management:

- In mild cases of abruption which present before 34 weeks with stable maternal and fetal condition and normal lab findings - a conservative approach is adopted. The main objective is to achieve fetal maturity. The patient is kept in the hospital.
- Regular assessment of maternal condition.
- Frequent assessment of maternal hematocrit and coagulation profile.
- Antepartum fetalsurvillence with non stress test and biophysical profile.
- Administration of betamethasone
- Anti D if needed.

Management of abruptio with live fetus:

There are 2 main subgroups of such patients:

- those with hypertonic uterus
- those with soft uterus

If the fetus is alive and uterus is rigid the abruption is probably large, **LSCS should be done.**

If the uterus is soft and fetus alive —the abruption is not greater than 25% and the chances of coagulopathy are extremely low and the prospects for vaginal delivery and favourable outcome are excellent. **Induction of labour should be done by ARM and oxytocin infusion.**

- If during labourthe uterus becomes hypertonic or fetal distress occurs Non progress of labourinduction of labour fails, ceasearen should be done

Management of coagulopathy (In tertiary center)

- Plasma fibrinogen levels may be 100- 150 mg /dldecreased.PTF(partial thromboplastin time) and PT (prothrombin time) increasedD-dimer concentration increased Platelet count decreased
- One unit of packed cells increases the hematocrit by 3-5 %. One unit of FFP

(200-250 ml) increases fibrinogen by 10 mg/dl.

- Alternatively fibrinogen orcryoprecipitate may be given. Platelets are given if there is thrombocytopenia.
- Heparin should be avoided in these patients.
- ArdifiwombinIII is preferred over Heparin.
- Muman recombinant factor 'Ill a is a remarkable drug for the control of obstetric bleeding.
- It is given as a bolus injection of 60-100 ugm/kg. The results are obtained after 10 units.
- -Any type of operative interference should be avoided. –

Extra placental causes of bleeding

Rare causes includes cervicitis,cervica I erosions,endocervica I polyps,cancer of cervix,vagina I, valvar and cervical varicosities,vagina I infections,Foreign bodies,Genital lacerations, Excessive show,Vasa previaandMarginal separation of placenta

- A direct cervical examination with a speculum should be done. Delivery at term is done accordingly.

ECTOPIC PREGNANCY

DEFINITION: An ectopic pregnancy is one in which the fertilized ovum is implanted and develops outside the normal uterine cavity.

IMPLANTATION SITES

A. Exfra uterine

1) Tubal (commonest 97%)

- Ampulla (55%)
- Isthmus (25%)
- Infundibulum (18%)
- Interstitial (20%)

2) Ovarian (0.5%)

3) Abdominal (1%)

B Uterine (1.5%)

- Cervical
- Angular ne)
- Cornual

TUBAL PREGNANCY

Risk factors for ectopic pregnancy:

Related to genital tract:

- History of PID (pelvic inflammatory disease); present salpingitis.
- Contraceptive failure (progesterone containing pills ,copper containing IUCD)
- Previous H/O ectopic pregnancy.
- History of infertility treatment
- ART (artificial reconstruction technology)
- Development defect of the tube.
- Transperifonal migration of the ovum.
- Previous induced abortion. Salpingitis isthmica nodosa.

Unrelated to genital tract:

- Current cigarette smoking.
- Multiple partners
- Intercourse before 18 years.
- In utero DES exposure.
- Age >4oyears.

Surgical:

- Tubal constructive surgeries.
- Tubal sterilization.

Acute ectopic:

LESS COMMON (30%)

It is associated with cases of tubal rupture or tubal abortion with massive intraperitonal haemorrhage

Symptoms

Classic triad of disturbed tubal pregnancy are: amenorrhea (75%), followed by abdominal pain (10) and vaginal bleeding (70%)

The above triad may be accompanied by nausea, vomiting, fainting attacks (even syncope in 1 cases).

Examination:

General examination:

- Patient lies quiet and conscious, perspires and looks blanched.
- Pallor present.
- Features of shock: rapid and feeble pulse, fall of blood pressure, cold clammy extremities.

Per abdomen -

- Abdomen is tense, tumid and tender.
- Shifting dullness may be present.
- Rigidity/ guarding +1-
- Cullen's sign - bluish discolouration around the umbilicus may be present.

On Bimanual examination:

- Vaginal mucosa appears blanched.
- Uterus- normal size/ slightly bulky.
- Extreme tenderness on cervical movement.
- Forn ices -tender.
- Unilateral adnexal mass: is palpable in one third to half of patients.

Culdocentesis:

It is a simple technique used to identify haemoperitoneum. Fluid is aspirated from cul-de-sac via posterior fornix with the help of a needle. If non-clotted is obtained, it is indicative of an intraperitoneal bleed. (Probably a ruptured ectopic)

On USG:

- Fluid in cul-de-sac.
- Empty gestational sac.

Diagnosis: Classic history of acute abdominal catastrophe with fainting attack and collapse associated with features of intra-abdominal haemorrhage in woman of child bearing age points to a certain diagnosis of acute ectopic.

lnvestigations:ABO-Rh,Hb, BT, CT, Urine C/E.

Management:

- The principle in management of acute ectopic is resuscitation and laparotomy.
- Antishock treatment started simultaneously with preparation for urgent laparotomy. Ringer's solution (crystalloid) is started.
- Arrangement is made for blood transfusion.
- Colloid administration after withdrawing samples for grouping and cross matching. Urgent laparotomy: principle is 'quick in quick out'.
- Indications for laparofomy: 1) Patient is haemodynamically unstable.
    1. Laproscopy is contraindicated.
    2. Evidence of rupture.
- Salpingectomy is the definite surgery. The excised tube should be sent for hisfopathological examination.
- The ipsilateral ovary and its vascular supply arepreserved.

Unruptered tubal ectopic:

High suspicion is required in sexually active female with abnormal bleeding and/or abdominal pain.

Symptoms:

- Presence of delayed periods or spotting with features suggestive of pregnancy.
- Uneasiness in one of the flanks.

Signs: On bimanual examination-

- Uterus is slightly smaller than the period of amenorrhea.
- Small, well circumscribed tender mass may be felt through one fornix, separate from the uterus.

Diagnostic modality:

- **Blood test:** aborh and Hb, TIC and DLC, ESR.
- Serum progesterone—level >25ng/ml is suggestive of viable intrauterine pregnancy. Whereas level <5ng/ml suggests an ectopic or abnormal

intrauterine pregnancy.

- Estimation of ß HCG: UPT(urine pregnancy test)will be positive in 95% of cases but single estimation of hcg either in the serum or in urine confirm pregnancy but cannot determine its location
  Suspicious finding in serum ßhcg measurement are:

  1) Abnormally low level of ßhcg for gestational age.
  2) Doubling time in plasma fails to occur in 2 days(48 hrs).
- **Sonograpy:** TVS is more informative.
  Diagnostic features are:

  1) Absence of intrauterine pregnancywith positive pregnancytest.
  2) Fluid in pouch of Douglas.
  3) Adnexal mass clearly separated from the uterus.
  4) Rarely cardiac motion may be seen in an unruptured tubal ectopic pregnancy.
- **Doppler sonography:** Gestational sac in the adnexa surrounded by a hyper echoic ring (tubal ring sign).
- **Combination of ß HCG and sonography-** ß HCG levels above the discriminatory zone (>1500 miu/ ml) and no intrauterine gestational sac suggestive of ectopic pregnancy.
  Rise in HCG <66% in 48 hrs.
- **Laparoscopy-** Gold standard for identification of ectopic pregnancy. But only feasible in hemodynamically stable patient. Therapeutic management can also be done at the same time.
- **Culdocentesis-** Unfortunately negative culdocentesis does not rule out on ectopic pregnancy neither a positive result is very specific. Management of unruptured tubal pregnancy:
  *Refer patient to tertiary care hospital for management after diagnosis.

Expectant management: only observation is done in hope of spontaneous resolution i.e. Falling serial HCG titres.

Indications:

1) Diameter of ectopic mass <4 cm.
2) No evidence of bleeding or rupture assessed by vaginal sonography.

Conservative management:

A .Medical management: No. Of chemotherapeutic agents have been used either systemic or direct local.

Drugs used are - methotrexate, potassium chloride, prostaglandin (PGF 2 a), hyperosmolar glucose or actinomycin.

Requirements:

The patient must be

1) Hemodynamically stable.
2) Tubal diameter <4 cm without any fetal cardiac activity.
3) No intraabdominal haemorrhage.
4) Reliable commitment to complywith required follow up care.

Most commonly used drug is methotrexate (systemic therapy).

- Protocol of administration and follow up:
- For systemic therapy, a single dose of methotrexate 50mg/$m^2$is given intramuscular. Monitoring: done by measuring serum ß hcg on day 4 and 7.
- If decline in HCG between day 4 and 7 is >15 %., patient is followed up weekly with serum HCG
- until HCG <10 iu/ml. If decline is <15% a second dose of methotrexate is given on day 7.

B. Conservative surgery:

- It is done in patients who do not fulfil the criteria laid down for medical management.
- The procedure is either done laparoscopically or by microsurgical laporotomy.

1) Linear salpingostomy
2) Linear salpingotomy
3) Segmental resection
4) Plucking out of distal tube. (Fimbrial expression)

Radical surgery: salpingectomy

Indications:

- The patient has completed her family;
- Tubes grossly damaged;
- Ectopic pregnancy has recurred in a tube already treated conservatively.

Chronic ectopic:

Symptoms: amenorrhea of 6-8 weeks, lower abdominal pain. Vaginal bleeding.

On examination: patient looks ill, pallor present, high pulse rate, no features of shock.

Management: all cases of chronic or suspected ectopic are to be admitted as an emergency. Patient is kept under observation, all required investigation are done and patient planned for laparotomy. Usually pelvic haematocele is found. Blood clots are removed. The affected tube is identified and salpingectomy is commonly done.

References- DC Dutta textbook of obstetrics, Williams's obstetrics

POSTPARTUM HEMORRHAGE (PPH)

Definition

Postpartum hemorrhage has been defined as blood loss in excess of 500 ml in a vaginal birth and in excess of 1 L (1000 ml) in a cesarean delivery

- Haemoglobin: 10% fall from antenatal level
- For clinical purposes, any blood loss that has the potential to produce hemodynamic instability should be considered a PPH

Incidence

1 to 5% of deliveries (14 million cases / year)

Single most imp. Cause of maternal deaths worldwide 88%

deaths due to PPH occur within 4 hours

Deaths from haemorrhage could often beavoided

Thus the need for set protocols and emergency drills for PPH management

Types

1. Primary PPH (immediate)
2. Secondary PPH (late)

Primary Postpartum Hemorrhage -

Primary (immediate) PPH occurs within the first 24 hours after delivery. Approximately 70% of immediate PPH are due to uterine atony.

Atony of uterus is defined as failure of the uterus to retract afterthe child is born It is

of 2 types

- Third stage hemorrhage- Bleeding occurs before expulsion of the placenta
- True PPH -Bleeding occurs subsequent to expulsion of placenta

Secondary Postpartum Hemorrhage –

Secondary (late) PPH occurs between 24 hours after delivery of infant and 6 weeks postpartum. Most late PPH is due to retained products of conception, infection or both.

Aetiology

- Retained placenta
- Failure to progress in 2nd stage
- Placenta accreta
- Lacerations
- Instrumental delivery
- Large for gest. age baby (>4000 g)
- Hypertensive disorders
- Induction of labor
- Augmentation of labor with oxytocin
- Placenta praevia.
- Previous history of PPH
- Obesity
- High parity
- Asian or Hispanic race
- Precipitous labour
- Pre-eclampsia
- Only a fewwomen with riskfactors develop PPH
- Manywomen without risk factors have PPH

Prevention of PPH

The incidence of PPH has shown a falling trend with the routine use of active management of third stage of labor (AMTSL)

AMTSL has 3 main components:

- Uterotonic(most important component)
  - Oxytocin 10 IU I/M (First choice drug -recommended by WHO)
  - Ergometrine/Methylergometrine 0.2 mg l/M
  - Misoprostol 600 µg orally
- Controlled cord traction (CCI)
- Uterine massage after delivery of the placenta

AMTSL reduces:

- Incidence of PPH by 60%
- Quantity of blood loss—thereby decreasing incidence & severity of anemia (100-150 ml)
- Emergencies & related cost
- The use of blood transfusion

PRIMARY POSTPARTUM HEMORRHAGE

Incidence

- Primary PPH occurs in 2-3% of all deliveries. About 15% of these are associated with retained placenta requiring manual removal

Aetiology

4Ts:	Cause	Incidence(%age)
Tone	Atonic uterus	70
Trauma	Perineal, vaginal, cervical lacerations	20
	Haematoma, Rupture uterus	
Tissue	Retained placenta or retained clots	10
Thrombin	Coagulation disorder	01

Diagnosis.

- PPH is usually external. It may partly be concealed from distention of uterus or vagina with blood clots. Concealed hemorrhage is confirmed by squeezing the uterus firmly, when the blood will be forced out with gush

- Blood loss is underestimated because in pregnancy signs of hypovolaemia do not show until the losses are large and visual quantification is difficult
- S Mother can lose up to 30-35% of circulating blood volume (2000 ml) before showing signs of hypovolaemia
- Blood is mixed with other fluids (amniotic fluid, urine) and therefore underestimated
- Bleeding may occur slowly over several hours and condition may not be recognized until woman suddenly enters shock

We mostly have to depend on clinical competence to assess blood loss

Degree of blood loss & features

Blood loss in ml (%)	Systolic Blood Pressure	Signs & Symptoms
500-1000(10-15) Tachycardia	Normal	Palpitation, Dizziness, or None
1000-1500(15-25) Tachycardia	Slightly low	Weakness, sweating,
1500-2000(25-35)	70-80	Restless, Pallor, Oliguria
2000-3000(35-45) Anuria	50-70	Collapse, Air hunger,

Atonic uterus:

Most common cause of Primary PPH is uterine atony Causes of

uterine atony are:

- Grand multipara
- Overdistension of uterus as in hydramnios, twins
- Malnutrition and anaemia
- APH
- Prolonged labor
- Anesthesia
- Initiation or augmentation of delivery by oxytocin

- Malformation of uterus
- Uterine fibroid
- Mismanaged third stage of labor
- Precipitate labor
- Full bladder

Initial Assessment and Management (If at periphery ,refer if bleeding is not controlled after giving uterotonic drugs and uterine massage)

- Shout for help—mobilize personnel
- Evaluate woman's condition including vital signs
- Place the woman on a flat surface, such as delivery table or birthing bed, with her feet higher than her head
- Massage uterus to expel clots and feel to see that it is contracted—recheck intermittently
- Start oxygen
- Give uterotonics
- Infuse IV fluids
- Catheterize bladder, if needed
- Check to see that placenta has been expelled—examine for completeness
- Examine the cervix, vagina and perineum for tears
- After bleeding is controlled, check for anemia

Management of Atonic Uterus

- Continue IVfluids
- Continue to massage uterus (per abdomen)
- Continue oxytocic drugs
- Perform bimanual compression or perform aortic compression
- Hydrostatic intrauterine ballontamponade
- Arterial embolization
- Laparotomy
  - B-Lynch suture
  - Consider stepwise devascularisation
  - Hysterectomy

Uterotonic Drugs

- OXYTOCIN –FIRST CHOICE
  - 101Ul/M(if not already given)
  - IV: Infuse 20 units in 1 L(nOrmal saline) at 60 drop/mm.

- Continuimg Dose: Infuse 20 units in 1 1 at 40 drop/mm.
- Maximum Dose: Not more than 3 LoflVfluids
- Precautions/Contraindications: Do not give as IV bolus

ERGOMETRINE or METHYLERGOMETRINE (used if oxytocin is not available or bleeding continues despite having used oxytocin)

  - Repeat 0.2 mg IM every 2-4 hours for a maximum of 5 doses
  - Max. Dose: 5 doses or 1 mg
  - Precautions/Contraindications: Pre-eclampsia, hypertension, heart disease
- MISOPROSTOL
  - 800 □g sublingually (4x 200 □g tablets)
- PGF2 alpha
  - IM: 0.25mg
  - Continuing Dose: IM: 0.25 mg every 15 mm
  - Max. Dose: 8 doses or 2 mg -
  - Precautions/Contraindications: Asthma

If bleeding persists after administration of uterotonics, these life saving measures are to be considered: **Bimanual Compression of Uterus**

- Wearing sterile gloves, insert hand into vagina; form fist
- Place fist into anterior fornix and apply pressure against anterior wall of uterus
- With other hand, press deeply into abdomen behind uterus, applying pressure against posterior wall of uterus
- Maintain compression until bleeding is controlled and uterus contracts
  - Apply downward pressure with closed fist over abdominal aorta directly through abdominal wall just above the umbilicus slightly to be save
  - With other hand, palpate femoral pulse to check adequacy of compression

Pulse palpable = inadequate Pulse

not palpable = adequate

Maintain compression until bleeding is controlled

1 LAPAROTOMY

1. B-Lynch sutures

Haemostatic sutures applied with No. 2 Chromic catgut as shown in diagram

Anterior and posterior wall of uterus are compressed to see if bleeding stops, that means the sutures will be beneficial

1. Stepwise pelvic devascularization

- Ligation of ascending branch of uterine artery
- Ligation of uterine and ovarian artery anastomosis
- Ligation of ant. Division of internal iliac a tier

2. Hysterectomy (Subtotal or Total)

- If conservative measures have failed, then hysterectomy is the final option.
- Subtotal hysterectomy may not beeffective when source of bleeding is in lower segment, cervix or vaginal fornices.
- There is belief that both operating time and blood loss are significantly lower with the subtotal technique but some studies have reported no such differences.

Continued care of the woman

- Once bleeding is controlled and woman is stable careful monitoring for next 24-48
- hours is needed -
- A rising BP and stabilizing heart rate is reassuring
- Keep checking that uterus is well retracted &remains so
- Carefully estimate blood loss
- Assess vital signs
- Ensure proper fluid intake
- Monitor blood transfusion
- Monitor urinary output

Finally

Before discharging the woman from health facility:

- Check the hemoglobin
- Give iron and folate supplementation as indicated by her condition

RETAINED PLACENTA

Retained placenta is generally defined as a placenta that has not undergone expulsion within 30 minutes of the baby's birth

Retention of placenta takes place under 2 different set of circumstances:

1. The placenta though completely detached, is not expelled. This maybe due to Uterine inertia or
   - Formation of a contraction ring
2. Adherent placenta
   - Simple adhesion
   - Morbid adhesion

1) Adherent Placenta

- **Simple adhesion** - The placenta remains in union with the uterine wall although its attachments are not abnormal. The condition tends to recur in the same patient.
- **Morbid adhesion -** The placental attachments are abnormal (pathological). There is no line of cleavage between the placenta and the uterine wall. It is a rare condition. May be of following types;
  - Placenta accreta- Chorionic villi are anchored to myometrium without intervening decidua
  - Placenta increta- Chorionic villi invade the myometrium but not beyond
  - Placenta percreta- Chorionic villi penetrate the whole uterine wall up to the serosal layer

Management of Retained Placenta(To be done at FRU)

- If placenta is felt in the vagina, ask woman to push, remove
- Ensure bladder is empty, catheterize if necessary
- Start oxytocun drip
- Attempt controlled cord traction If not successful proceed with
- Manual removal of placenta

Manual removal of placenta (MROP)

- Done under GA
- Pt is placed in luthotomy position and bladder catheterized with aseptic measures.
- One hand is introduced in cone-shaped manner into the uterus following the cord.
- When placental margin is reached, fingers are insinuated between placenta and uterine wall with the back of hand in contact with the

wall.

- Support the fundus of uterus while detaching the placenta
- When placenta is completely separated, extracted by traction on cord.
- Uterine hand explores the cavity to be sure nothing is left behind.
- I/V methergin 0.2 mg is given and massage done.
- Inspect placenta and membranes for completeness.

Note: If plane of cleavage is not found no attempt should be made to deliver placenta and patient referred to higher center as this can provoke massive hemorrhage

Post procedure care:

- Give antibiotics.
- Observe the woman closely until the effect of IV sedation has worn off.
- Monitor the vital signs (pulse, blood pressure, respiration) every 30 minutes for the next 6 hours or until stable.
- Palpate the uterine fundus to ensure that the uterus remains contracted.
- Check for bleeding P/V.
- Continue infusion of IV fluids. - Transfuse as necessary.

Complications

- Shock
- Postpartum hemorrhage
- Puerperal Sepsis
- Subinvolution of the uterus

Morbid adhesion of the placenta (placenta accreta)

- The incidence of placenta accreta has increased 10-fold in the past 50 years, to a current frequency of 1 per 2,500 deliveries, largely as a result of the increase in the number of cesarean sections
- Because of the fact that many of these cases become evident only at the first attempt to separate the placenta at delivery, it is essential to attempt to identify antenatally both placenta accreta and its attendant risk factors, the most common of which is concurrent placenta praevia & previous CS.
- Such cases should be referred timely to higher center for management

Management of Retained Placental Fragments

- Under GA
- Feel inside uterus for placental fragments.
- Remove placental fragments by hand, ovum forceps or large curette
- Give oxytocics, do uterine massage and observe for bleeding P/V
- Transfuse as the need be

PRE-ECLAMPSIA AND ECLAMPSIA

Introduction

- Pre-eclampsia is the new onset hypertension and eitherproteinuria or end organ dysfunction after 20 weeks of gestation in a previously normotensive woman.
- It is a multisystem progressive disease and delivery results in resolution of the disease.
- It contributes significantly to maternal and perinatal mortality and is responsible for 13% maternal deaths worldwide.

Incidence

Varies from 5 to 10%

Definitions

- Hypertension is defined as a bp measurement of more than or equal to 140/90 mm of hg or an increase in mean arterial bp { 1/3 systolic bp + 2/3 diastolic bp } of 20 mm hg taken on two occasions atleast 6 hours apart.
- Bp is measured in sifting or lateral lying down position with manometer at the level of heart and korotkoff phase v (disappearance of sounds) is used.
- Proteinuria is the presence of a total proteins in 24 hours urine of 300 mg or more or 1 + or more on dipstick random samples.
- Edema has been abandoned as a diagnostic criterion as it occurs in many normal pregnant women.

Classification

There are 5 types of hypertensive diseases that complicate pregnancy rate

1) **Gestational Hypertension**
   - BP ≥140/90 mm Hg for first time during pregnancy No proteinuria
   - BP returns to normal before 12 wks post partum
   - Final diagnosis made only post partum
   - May have other features of preeclampsia like epigastric discomfort or thrombocytopenia
2) **Pre eclampsia**
   - BP >- 140/90 mm Hg after 20 wks gestation
   - Profeinuria > 300 mg/24 hrs or> 1 + dipstick
   - Definitive criteria are
   - BP>160/llO mm Hg
   - Proteinuria 2.0 mg/24 hrs or > 2+ dipstick
   - Serum creatinine ≥1.2 mg/dl unless known to be previously elevated
   - Platelets <1 00000/p1
   - Micro angiopathic hemolysis - ↑ LDH
   - Elevated serum ALT or AST
   - Persistent headache or cerebral or visual disturbances
   - Persistent epigastric pain
3) **Eclampsio**

   Convulsions in a case of pre eclampsia in the absence of other causes of convulsions

4) **Superimposed pre eclampsia on chronic hypertension**
   - New onset proteinuria ≥ 300 mg/24 hrs in hypertensive woman but no proteinuria before 20 wks gestation
   - A sudden rise in proteinuria or BP or platelet count < 1 00000/ l in a woman with hypertension and proteinuria before 20 wks gestation
5) **Chronic hypertension**
   - BP ≥140/90 mm Hg before pregnancy or diagnosed before 20 wks gestation not attributable to gestational trophpoblastic disease
   - Hypertension first diagnosed after 20 wks but persistent 12 wks post partum

Risk factors

- Nulliparify
- Extremes of age
- Multiple pregnancy
- Chronic hypertension
- Obesity
- Hydafidiform mole
- New partner/ pregnancy with doner semen
- Anti phospholipids antibodies
- Diabetes or renal disease
- Hydrops fetalis

- Genetic predisposition
- Previous h/o pre eclampsia

Pathophysiology

- Basic pathophysiology is vasospasm in almost all organs which causes t peripheral resistance, ? bp, endothelial damage with leakage of blood constituents, platelet and fibrin deposition in subendothelial layers leading to hemorrhage, necrosis and damage to endorgans & collection of fluid in extra vascular space.
- It affects all organs including placenta, liver, kidney, brain, retina and cardiovascular system resulting in hypoxia and iugr.

Severity of disease

- Can be either mild or severe form
- Indicators of severe disease are
- Systolic bp ??160 mm hg
- Diastolic bp ? 110 mm hg
- Proteinuria ??3+ dipstick
- Presence of headache , visual disturbances, epigastric or upper abdominal pain, convulsions, oliguria, pulmonary edema, obvious iugr and elevated serum creatinine, markedly elevated liver enzymes, platelet count <100000/ ?1 and evidence of hemolysis are the criteria of severe pre eclampsia.
- An apparently mild disease may progress to severe disease very rapidly.
- HelIp syndrome characterized by hemolysis, elevated liver enzymes and low platelets is a feature of severe pre eclampsia.

Clinical features and diagnosis

Symptoms

- May be asymptomatic or mild swelling over ankles in the mornings or the swelling may involve face, abdominal wall and rest of the body.
- Serious symptoms like headache, sleep disturbances, epigastric or right hypochondric pain or visual disturbances are associated with acute onset disease.

Signs

- Rapid weight gain of more than ½ kg a week or 2 kg a month in later months of pregnancy
- Sudden or massive generalized edema usually indicates imminent eclampsia.
- Rise in BP
- Basal crepts in lungs- pulmonary edema
- Brisk tendon reflexes
- Oligohydramnios and IUGR
- Oliguria

Investigations

- Complete hemogram with platelet count. The hematocrit may be raised due to hemoconcetration.
- Coagulation profile
- Serum uric acid – Levels > 4.5 mg/dl indicate pre eclampsia but not very useful as severe disease can occur with normal uric acid levels.
- Blood urea maybe normal or slightly raised
- Serum creatinine > 1 mg/dl
- IFT - ALT, AST or LDH may be raised
- Urine examination for proteins in 24 hrs urine and for casts
- Fundus examination - may show constriction of arterioles or retinal edema or hemorrhages
-

Fetal monitoring

- Daily fetal movement count (dfmc) there should be at least 10 movements per day.
- Clinical examination for fetal growth, presentation and well being
- Nst (non stress test) twice a week
- Cardiotocography
- Ultrasound examination should be done every 2 weeks for fetal growth, well being and amount of amnioticfluid. Nsf twice a week and bpp (biophysical profile) once a week. Doppler studies for umbilical artery blood flow should also be done.

Complications of pre eclampsia

Maternal complications

- Eclampsia in upto 2% cases
- Abrupfio placentae
- Renal failure
- Diminution of vision and blindness
- Cerebral hemorrhage
- Coagulation failure and dic
- Hellp syndrome
- Adult respiratory distress syndrome (ards)
- Preferm labor
- Increased operative delivery

- Death
- Remote complications include recurrent pre eclampsia in next pregnancy (25%), residual hypertension and chronic nephritis post partum hemorrhage (pph) and shock
- Infection
- Hepatic rupture

These complications can lead to maternalfetal complications

- Iugr due to chronic placental insufficiency
- Intra uterine asphyxia
- Intro uterine death (iud)
- Prematurity due to preterm labor, accidental hemorrhage or induced labor
- Oligohydramnios
- Placental infarction
- Perinatal mortality is around 20%

Management Prevention

- Low dose aspirin - it may prevent pre eclampsia by suppressing the production of thromboxane a from platelets. 75 mg daily started as early as 12 wks, .given upto 34 wks. Its role is still controvercial.
- Bed rest - absolute bed rest is not recommended but restricted mobility with adequate rest does help in lowering bp
- Salt restricted diet - no role
- Fish oil supplementation - no role
- Antioxidents - no role
- High dose calcium - no proven role but may be beneficial in calcium deficient women

Treatment

Management depends upon the following:

1. Severity of disease
2. Period of gestation
3. Maternal condition
4. Fetal condition

Mild Pre eclampsia

General

- Women with mild pre eclampsia <37 wks gestation are managed expectantly with close maternal and fetal monitoring.
- Ideally all patients with newly diagnosed or persistent or worsening disease should be admitted to hospital for initial evaluation and stabilization.

- A detailed history and examination including signs & symptoms of severe disease should be recorded.
- Maternal weight should be checked on admission and regularly thereafter.
- BP record 4 hourly (in Eclampsia Chart)
- Urine for proteins twice a week
- Weekly RFT, LFT, uric acid, platelet count & hematocrit
- Weekly fundus examination
- Ultrasonographic evaluation of fetal size, amniotic fluid and NST and BPP on admission and every 1-2 weeks
- Daily fetal movement count is recorded

Drugs

- Sedation - Sedatives and tranquilizers are not prescribed routinely
- Diuretics - Should be avoided as they can be harmful to the fetus. May decrease the placental perfusion causing IUGR, can cause rise in blood urea, uric acid and can cause neonatal thrombocytopenia.
- Diuretics are to be used only in Pulmonary edema, Cardiac failure and in some cases of severe generalized anasarca.
- Anti hypertensive drugs are generally not recommended as they may lower the BP but do not decrease the complications or the perinatal mortality.
- Corticosteroids - Antenatal corticosferoids (betamethasone) to promote fetal lung maturity should be administered to women < 34 wks gestation as they are at increased risk of progressing to severe disease and pre term delivery.

Management of labour

Mild and stable cases are generally allowed to go upto term. Indications for

early delivery are:

1. Worsening of hypertension, proteinuria or vital organ involvement

2. IUGR or fetal distress

- Generally the labor is induced at 37 -38 wks.
- Method of induction depends on Bishop score. If cervix is favourable (Bishop score > 6) low rupture of membranes followed by oxytocin infusion is used. If cervix is unfavourable (Bishop score < 6), prostaglandin E2 gel vaginally or intracervically is used to ripen the cervix.
- During labor strict fetal & maternal monitoring is done. Aim is to deliver vaginally.
- LSCS is done only for obstetric indications

- Third stage to be managed actively by 10 units oxyfocin tIM
- Fluid balance should be monitored closely to avoid overload. Maintenance fluid of 80 ml/hour is adequate in the absence of excessive loss.

Severe pre eclompsia management -

- Hospitalization in a tertiary hospital is required
- Bed rest
- Daily monitoring of vitals, DFMC, NST, weight record
- Urine protein testing twice a week
- Weekly ultrasound for fetal monitoring, Doppler study every 2 weeks or earlier if required
- Look for signs and symptoms of impending eclampsia
- Anti hypertensive treatment: May be started when systolic BP is 160 mm Hg or diastolic BP is 105 or 110 mm Hg. The diastolic BP should be kept between 90 and 100 mm Hg to prevent complications like cereBPal hemorrhage
- Methyldopa 250 mg 8 hourly is started initially, can be increased to a maximum of 500 mg 6 hourly.Or
- Labetalol 100-200 mg 8 hourly can be given.
- Nifedipine 10 mg 4-6 hourly can also be given.
- Corticosteroids - If < 34 wks gestation, Betamethsone 12 mg I/M 2 doses 24 hours apart should be given for pulmonary maturity.

Indications for delivery

1. Worsening of maternal condition
2. Fetal compromise
3. Completion of 34 weeks of gestation
4. Premature labor

- In cases of severe pre eclampsia with gestation< viability or 34 weeks, the pregnancy should be terminated.
- Prophylactic Anti convulsant treatment using Magnesium sulphate should be given to severe pre eclampsia cases with impending eclampsia and during labor & 24 hours post partum as it reduces the development of eclampsia.
- Urine output is monitored using Foleys catheter.
- Fluid overload is avoided.
- Blood, platelet concentrates and fresh frozen plasma to be given if there is coagulopathy.
- Labor to be induced by oxytocin or prostaglandin E2 gel & amniotomy depending upon Bishop score.
- LSCS to be done for obstetric indication. Epidural can be given if coagulation profile is normal.
- For third stage active management 5-10 units of oxytocin can be given I/M

Post natal care

- Patient is sent home after improvement in general condition and control of BP.
- BP & proteinuria is checked 6 weeks post partum.
- OCP & IUCD can be advised, postpartum ligation is avoided for fear of thromboembolism.

ECLAMPSIA

Pre eclampsia complicated by generalized tonic clonic convulsions is termed Eclampsia.

Eclampsia is characterized by

- Hypertension , proteinuria and convulsions
- The convulsions may occur in the antepartum, intrapartum or postpartum period.

Imminent Eclampsia

-When an eclamptic fit is likely to occur very soon

Symptoms:

Severe headache

Drowsiness

Mental confusion

Visual disturbances (e.g. blurred vision, flashes of light, doublevision) Epigastric pain

Nausea, vomiting Decreased urinary output

Signs:

A sharp rise in the BP

Increased proteinuria

Exaggerated knee jerk

High risk factors for Eclampsia

Risk factors for eclampsia are same as that of preeclampsia

Status eclampticus -

Refers to a state in which convulsions or eclamptic fits continue incessantly one after the other.

It is dangerous for both mother and foetus and can lead to maternal and foetal mortality.

Effects of eclampsia on mother

* Respiratory (asphyxia, aspiration of vomitus, pulmonary oedema, bronchopneumonia)

* Cardiac (heart failure)

* BPain (haemorrhage, thrombosis, oedema)

* Renal (acute kidney failure)

* Hepatic (liver necrosis)

* HELLP syndrome (haemolysis, elevated liver enzymes,low platelet count)

* Haemorrhage due to coagulation defect, i.e. DIC

* Visual problems (temporary blindness: due to oedema of the retina)

* Injuries (fractures, tongue bite)

The most common causes of maternal death in eclampsia:

- aspiration of vomitus
- kidney failure,
- intracereBPal haemorrhage
- multi-organ failure.

Effects on the foetus

Placental insufficiency leads to:

Hypoxia: This may lead to permanent BPain damage, which may result in

- physical handicap
- cereBPal palsy
- mental retardation

IUGR

- Prematurity

Management of eclampsia and severe pre-eclampsia

1. Making sure that the woman can Breathe

Place the woman on her left side so that mucus or saliva can drain out in a dark room. Clean the mouth and nostrils by gentle suction

Give oxygen

Instruct the nursing staff to make sure that:

- the patient's airway remains clear;
- injury, especially to the tongue (tongue bite), is prevented
- by placing padded tongue blades between her teeth (Do NOT attempt this during a convulsion) also use bed with padded railing for the patient.
- Catheterise the patient

2. Controlling the fits

Magnesium sulphate is the drug of choice.

Loading dose: lnj. Magnesium sulphate 4 g (20 ml of 20% solution), slow IV, over 5- 10 minute

Thereafter administer Inj. Magnesium sulphate 5 g (10 ml of 50% solution), deep IM, with 1 ml of 2% Lignocaine in the same syringe in each gluteus (a total dose of 1 Og)

If convulsions recur: After 15 minutes, give an additional 2 g of Magnesium sulphate (10 ml of 20% solution) IV slowly. If the convulsions still continue, give Diazepam 5mg, IV slowly.

If referral is delayed for long, or woman is in late stage of labour, continue treatment as below:

- Give 5 g of 50% Magnesium sulphate solution IM with 1 ml of 2% Lignocaine every 4 hours alternately in each buttock.
- Before giving the next dose of Magnesium sulphate, ensure that:
  * The urine output is at least 100 ml per 4 hours;
  * Knee lerk reflexes are present;
  * The RR is at least 16 BPeaths per minute.
- Postpone the next dose if the above criteria are not met.
- Precautions: Do NOT give 50% Magnesium sulphate solution IV without diluting it to 20%.
- Do NOT give a rapid IV infusion of Magnesium sulphate as it can cause respiratory failure or death.
- If respiratory depression occurs (RR <16 BPeaths/minute) after giving

Magnesium sulphate, discontinue the drug.

- Give the antidote; Calcium gluconate 10 mg IV (10 ml of 10% solution) over a period of 10 minutes.

3. Controlling the blood pressure

- Anti hypertensive therapy: If the diastolic BP is 110 mmHg or more antihypertensives are recommended.
- The goal of treatment is to keep the diastolic pressure between 90 and 100 mmHg to prevent cerebral haemorrhage.
- There is no good evidence that any one antihypertensive is better than another for reducing the BP

Nifedipine

- Dose and administration : The dose of Nifedipine is 10 mg orally.
- To avoid sudden hypotension, After 10 minutes, monitor the BP
- If the BP is still not BPought under control, another 10 mg of the drug can be repeated similarly.
- Disadvantage: Nifedipine may cause a sudden and massive fall in BP Hence, it should be used with caution,and the dose delivered slowly.
- Precaution: Nifedipine, when used in conjunction with Magnesium sulphate, can cause a dangerous fall in BP
- Hence, when Nifedipine and Magnesium sulphate are used together, the BP should be monitored carefully

Labetalol- is given IV

Dose- 20mg IV slowly. If response is adequate, 40 mg may be given, after 20 minutes. The dose can be further increased to 80 mg after another 20 minutes.

Hydralazine-

Dose of 5-10 mg IV slowly every 15-20 mins until blood pressure is lowered. It should be given only in ICU setting.

4. Controlling the fluid balance

- Insert indwelling urinary catheter to measure the urinary output.
- Record the urine output every 4 hours.
- Suspect kidney failure if urine output less than 100 ml per 4 hours.
- Record fluid intake. Give all the necessary fluids slow IV.
- The patient should receive sodium lactate or5% dextrose @ 60 ml (maximum) per hour unless there is an unusual fluid loss from vomiting, diarrhoea, orexcessive blood loss at delivery.

5. Delivering the baby

- Decide on the method of delivery depending on whether or not the woman

has gone into labour, and the stage and progress of labour.

- In severe pre-eclampsia, delivery should occur within 24hours of the onset of symptoms;
- In eclampsia, delivery should occur within 12 hours of the onset of convulsions.

6. Giving care after delivery

- It is important to realize that fits can occur for the first time after delivery, especially during the immediate postpartum period.
- Fits, if they have occurred before delivery, can also recur after delivery.
- Therefore, the patient must be carefully observed during the immediate postpartum period.
- Refer the woman to an FRU one hour after delivery, after ruling out immediate PPH, and ensure that woman's condition is stable.
- If the patient has fits after delivery, continue to observe and manage her for 48 hours after the last fit.
- Monitor the BP every hour. Continue giving anti hypertensives as and when required, until the diastolicBP drops below 110 mmHg.
- Monitor the urinary output
- Do not give too much fluid intravenously during this period.
- Advise the woman to have her BP checked regularly.

References- Williams Obstetrics, textbook of obstetrics by Dr J B Sharma, national guidelines for BEmOC

TRAUMA - GENITAL TRACT TEARS

- Perineal tears and lacerations
- Vaginal tears
- Cervical tears
- Para-vaginal & VuIval hematoma
- Pelvic or Broad ligament hematoma
- Uterine rupture

Management of Genital Tract Tears

- Inspect cervix, vagina and perineum

- Repair tears that are:

- Bleeding
- More than first degree
- Away from urethra

- Place catheter if necessary

All the while:

- Transfuse blood as needed
- Consider concurrent diagnoses if bleeding still heavy

Perineal Tear

- Should be repaired immediately
- Define the limits properly
- The suture used is catgut or polydioxanone(PDS) - VICRYL
- As accurate approximation possible should be done
- Vaginal tear is repaired first
- In complete perineal tear, repair rectal mucosa first followed by anal sphincter, vaginal mucosa, perineal body and finally skin
- We need GA for 3rd and 4th degree perineal tears
- Give antibiotics
- The bowels are not encouraged to act for a few days
- Thus a low residual diet is started from 2nd day onwards

Vaginal tears

- Vaginal lacerations are more common in upper and lower thirds
- They bleed profusely, so should be inspected and properly sutured
- A good light source is necessary for proper visibility
- Packing maybe done and removed after 6-8 hours

Cervical tears

- Cervical tear is the commonest cause of traumatic PPH
- Left lateral tear is the commonest.
- A very good light source is needed for diagnosis and a successful repair
- Proper assistance should be there
- Repair is done under G.A.
- Ant and post margins of torn cervix are grasped by sponge holding forceps. Apex is identified and 1st mattress suture is applied just above the apex using catgut.
- Bleeding stops and rest of the tear is repaired by similar mattress sutures.

HAEMATOMAS

Vulval hematoma

- In vulval hematomas bleeding is limited to the vulval tissues superficial to the anterior urogenital diaphragm. The hematoma will be evident on the vulva.
- Vulvovaginal hematomas are also evident on the vulva but they extend into the paravaginal tissues.
- Both types arise from injury to the branches of the pudendal artery (the posterior rectal, transverse perineal and posterior labial arteries)

Vaginal hematoma

- Vaginal or Paravaginal hematomas arise from damage to the descending branch of the uterine artery.
- The hematoma is confined to the paravaginal tissues in the space bounded inferiorly by the pelvic diaphragm and superiorly by the cardinal ligament.
- Rectal pain, vague lower abdominal pain but hematoma will not be obvious
  externally but can be diagnosed by vaginal examination.
- The mass often occludes the vaginal canal and extends into the ischiorectal fossa.

Diagnosis of hematomas

- severe perineal pain and usually rapid appearance of a tense, fluctuant, and sensitive tumor of varying size covered by discolored skin
- Symptoms of pressure, if not pain or inability to void, should prompt a vaginal examination with discovery of a round, fluctuant tumor encroaching on the lumen

Management of hematomas (Vulval& Vaginal)

- Smaller vulvar hematomas(≤ 5cm) maybe treated expectantly with analgesics, observation and icepacks.
- If the pain is severe or the hematoma continues to enlarge, the best treatment is prompt incision
- done at the point of maximal distention along with evacuation of blood and clots and ligation of bleeding points
- The cavity may then be obliterated with mattress sutures. Often, no sites of bleeding are identified after the hematoma has been drained.

- In such cases, the vagina, not the hematoma cavity, is packed for 12 to 24 hours
- With hematomasof the genital tract, blood loss is nearly always considerably more than the clinical estimate
- Hypovolemia and severe anemia should be prevented by adequate blood replacement

Broad ligament and retroperitoneal hematoma

- These occur when a vessel ruptures above the urogenital diaphragm
- The bleeding extends into the supravaginal space between the leaves of broad ligament and maytrack retroperitoneally even as high as the kidneys.
- They occur most commonly following operative delivery, trauma, or surgery, but if may also occur following spontaneous vaginal delivery.
- These can be dangerous as they may be silent and not cause obvious vaginal bleeding.
- Most patients report back pain, fullness or pressure in the rectoanal area, or an urge to push, or they complain of dizziness
- Large broad ligament haematomas may be felt on bimanual examination and push the uterus to one side
- Extensive broad ligament and retroperitnealhaemafomas may cause profound hypovolumic shock and may rupture into peritoneal cavity
- Diagnosis may be aided by USG or MRI if available
- Broad ligament hemafoma may be treated either conservatively with blood transfusion, fluid resuscitation, and observation
- Or it may be successfully treated by uterine artery embolization if facilities are available
- Or with surgical exploration and evacuation followed by ligature of bleeding points
- A careful check should be made to confirm or deny uterine rupture as source of haematoma

References:

1. Fogsi focus January 2007 Post-Partum hemorrhage by Federation of Obstetric and Gynaecological Societies of India
2. FIGO GUIDELINES - Prevention and Treatment of postpartum heamorrhage in low-resource settings. International Journal of Gynecology and Obstetrics 117 (2012) 108-118

PRETERM LABOUR

It is the presence of contractions of sufficient strength and frequency to effect progressive effacement and dilation of cervix between 20 to 37 weeks of gestation

Early preterm labour

Cervix is dilated > 1 cm but <3 cm, >80 % effaced Documented

uterine contractions with no cervical change

Advanced preterm labour

If the cervix is >80% effaced and cervical dilatation is 3 cm or more Prematurity is

classified in three groups according to gestational age:

- **Severe prematurity** when birth occurs <30 weeks
- **Intermediate prematurity** when birth occurs between 30-34 weeks
- **Late or mild prematurity** when birth occurs between 34-37 weeks

Symptoms

1. Uterine activity (painful or painless uterine contractions)
2. Pelvic pressure
3. Menstrual-like cramps
4. Watery vaginal discharge

Signs

1. Regular uterine contractions with or without pain at least one in every 10 mm
2. Dilation >2cm and effacement (80%) of the cervix
3. Length of cervix (measured by TVS<2.5CM
4. Show
5. Bulging membrane
6. Rupture of membrane

Prevention of preterm birth

Primary prevention

1. Prevent pregnancy in teenagers
2. Prevent smoking and illicit drug use
3. Prevent RTI/STI; treatasymtomaticbacteriuria
4. Access of family planning method to prevent unwanted and frequent delivery
5. Pre-conceptional counselling
6. Improve nutrition and general health of woman

7. Decrease factor causing stress and give adequate rest

SECONDARY PREVENTION

It includes identification of women who are at PTB and their close surveillance it includes screening tests for early defection and their treatment

ACOG guidelines

Major recommendations

- There are no clear 1st line tocolytic drugs to manage preterm labour
- Circumstances & physician's preference should dictate the treatment
- Antibiotics do not appear to prolong the gestation & should be reserved for gpB streptococcal prophylaxis in patients in whom delivery is imminent.
- Neither maintainance treatment with tocolytic nor repeated acute tocolysis improve the perinatal outcome
- **Tocolytics may prolong pregnancy for 2 to 7 days, which allow steroid administration & transfer to tertiary care centre with good NICU**

LEVEL B recommendations

- Cervical USG Metal fibronectin have good negative predictive value, thus either approach or combined maybe helpful in determining patients who need tocolyfics
- Amniocentesis may be used in women in preterm labour to assess fetal lung maturity & intraamniotic infection.
- Bed rest and hydration do not appear to improve the rate of preterm birth & should not be routinely recommended

Investigations

- Full blood count
- Urine for routine analysis, culture and sensitivity
- Cervicovaginal swab for culture and fibronectin
- Ultrasonography for fetal well being, cervical length and placental localisation
- Serum electrolyte and glucose levels when tocolytic agents are to be used

Management

The following regime may be used to arrest preterm labour -

- Bed rest
- Adequate hydration
- In utero transfer

- Tocolyfic agents

1. Bed rest

Patient to lie on left lateral position though the benefits are doubtful

2. Hydration and sedation/bed rest

In a study woman received 500 ml of crystalloid over 30 min and 8-10 mg of morphine i/rn had outcome similar to bed rest. So it gives no added advantage

3. ln utero transfer

If local facilities are inadequate to treat preterm labour an inuter transfer is better than exutero transfer as the uterus is better transfer incubator.

5. Short Course of Tocolytic therapy
   Indications
   a) gestation <34 weeks
   b) no fetal ormaternal compromise
   c) in utero transfer

Contraindications to tocolysis

a) Fetal demise or anomalies incompatible with life
b) Fetal distress
c) Severe bleeding or abruptio placentae
d) Severe IUGR
e) Chorioarnnionitis
f) Cervix > 3cm. Dilated
g) Fetal maturity
h) Maternal hemodynamic instability
i) PPROM

Tocolysis has not be shown to improve perinatal outcomes. It prolongs pregnancy by at least 48 hrs allowing administration of betamethasone and shifting the patient to a centre equipped with better neonatal facility

Regimes for Tocolysis

- Calcium channel blockers
- B-sympathomimetics
- Non steroidalanti inflammatory agent
- Oxytocin receptor antagonist

- Nitric oxide donors

Calcium channel blockers (nifedipine)

It is calcium channel blocker that causes smooth muscle relaxation and is used for the t/t of chronic hypertension

- The loading dose is 20-30mg and maintenance dose is 10-20mg every 6 hr (max dose is 160 mg)
- It is best 1st line tocolytk agent available in market because of easy availability, cheaper cost, ease of administration and fewer side effects than b-sympathomimetics

Side effect

Headache, tachycardia, palpitation, flushing, fatigue, dizziness, nausea, constipation and edema

Maternal contraindication of use of nifedipine

- Hypotension (SBP<90mm of Hg)
- Known allergy to nifedipine
- Cardiac dis (CCF,aortic stenosis)
- Concurrent use of salbutamol, glycerol trinitrate, other anti hypertensive use, hepatic dysfunction
- Caution with usage withMgSO4 because significant hypotension with neuromuscular blockage can occurs

Beta-adrenergic agonists

Ritodrin

For intravenous administration the initial dose is 1 OOug/min The

dose is increased by 50 ug/min until the contractions stop Max

dose is 350ug/min

Once labour is inhibited ,maintenance dose is for 12 hr

Fluid is restricted to 2.51/24 h Infusion of B agonist resulted in frequent, and at times serious and fatal side effects

Pulmonary edema is a special concern.lt causes increased capillary permeability, disturbance of cardiac rhythm and Ml.

Terbutaline

B agonist commonly used to forestall labour

5 mg of terbutaliné is dissolved in 500 ml of RL and started at 5ug/min

Dose is increased gradually by 5ug/min every 10 to 20 min until uterine contractions stop

The max dose is 30ug/min.

Then s/c admn 0.25-0.5mg for every 2 to 4hr for 12 hr.

A maintenance dose of 2.5-5mg orally given 4-6 times daily Side

Effects of B Mimetics

Headache, Palpitations, Tachycardia, Pulmonary edema, Hypotension, Cardiacfailure,

Hyperglycemia, ARDS, Hyperinsulinemia, Lactic acidosis, Hypokalemia Contraindication to

betamimetic agents

Maternal cardiac rhythm disturbance, Poorly controlled DM, Thyrotoxicosis, Sickle cell ds, chorioamniotis

MAGNESIUM SULPHATE

Loading dose 4 g over 15-20 mm

Followed by infusion at 1-2 gm/hr

Serum levels of 8 to 10meq/l required for tocolysis

Causes sedation, ↓ analgesic requirements

Modest prolongation of bleeding time due to effect on platelet aggregation by antagonizing the effects of Ca^{++}

SIDE EFFECTS OF MgSO4

Flushing, maternalhypothermia, Perspiration, Headache, paralytic ileu, Muscle weakness

Contraindications to Mg SO4

Hypocalcemia, Renal failure Myasthenia gravis INDOMETHACIN

50 mg PO/PR followed by 25 mg 6 hrly for 48 hrs

Limit course of therapy to less than 72 hr and administer only before 32 week gestation to minimize neonatal side effects

No cardiovascular side effects like other agents

Indomethacin can be used as second stage tocolytic agent in early gestational age PTL

It may be 1st line tocolytic in associated polyhydramnios (to have renal effects of indomethacin)

Side Effects

GI bleeding, Asthma, thrombocytopenia, cause premature closure of ductusarteriosus in utero

ATOSIBAN

Given in IN infusion (300ug/min)

CVS effects are much less than b mimetics It is

expensive and not yet available in India

NITROGLYCERIN

Preferred way is to give by transdermal patch, manufactured to release a specific amount of medication b/w 0.1 -0.8mg/hr. Minimal side effects include hypotension and headache

Steroids to Accelerate Fetal Lung Maturity

Betamethasone:12 mg. IM in 24hx2 dose

Dexamethasone: 6 mg, IM in 12h x4 doses Effect of glucocorticoids on fetal lungs lasts no longer than 1 week

Rescue weekly repeat doses of betamethasone should not be given because of neonatal side effects

Repeat doses interfere with CNS myelination, decrease birth weight, decrease head circumference with increase in risk of cerebral palsy

Contraindication tocorticosferoids

- fetal, neonatal deaths
- Chorioamnoitis
- Maternal tuberculosis
- Porphyria
- Pregnancy> 34 weeks
- Maternal or fetal infection

*Refer Patient to higher centre for further management.

ROLE OF PROGESTERONES

PROGESTERONE maintains uterine quiescence and blocks the labour initiation

Benefit is primarily in reduction of birth before 34 weeks

- FDA of USA has recently approved administration of weekly injections of 17-hydroxyprogesterone acetate for prevention of recurrent preterm birth

- ACOG2008c has concluded that progestrone therapy should be limited to women with documented h/o previous spontaneous birth at <37 weeks

Strategies for prevention of PTL and PTB -

Use of tocolyfic as maintenance therapy after primary treatment Till date available evidence does not support the use of oral B-mimetic drugs and other focolyfic drugs for maintenance therapy after threatened PTL

Management of women presenting with threatened or actual preterm labour

Once diagnosis is confirmed clinical exam with appropriate investigation of maternal and fetal condition should be done:

- Ultrasound is done to know about fetal number, estimated fetal weight, fetal morphology along with presentation ,liquor vol and placental sitefwb ,along with umblical vessel doppler assessment and fetal activity along with fetal breathing movement which are suppressed in women with PlL

Follow up

Search for cause /precipitating factors

Establish plan for future pregnancy Provide

long term follow up for neonate

Key Message

1. Corticosteroids should be given to the mother to reduce the risk of neonatal respiratory distress syndrome.
2. In-utero transfer of the mother for delivery in a unit where appropriate neonatal care can be provided
3. Tocolyfic drug can be used for a short period unless contraindicated
4. Antibiotic in cases with infection
5. Careful intrapartum monitoring, minimal trauma & involvement of neonatologist during delivery are essential
6. Vaginal delivery is preferred unless caesarean is indicated for obstetric reasons

ANAEMIA IN PREGNANCY

Definition: according to who, anaemia in pregnancy is present when the haemoglobin concentration in the peripheral blood is 11gm/100ml or less

Classification:

A- physiological anaemia of pregnancy
B- pathological

Pathological

1 -deficiency anaemia

- Iron deficiency
- Folic acid deficiency
- Vitamin B12 deficiency
- Dimorphic (Both iron and folic/vit-131 2 deficiency)
- Protein deficiency. 2

-Haemorrhagic

- Acute- Following bleeding in early months or APH
- Chronic- Hookworm infestation, bleeding piles etc.

3. Hereditary

- Thalassemias
- Sickle cell haemoglobinopathies
- Hereditary haemolytic anaemias

4. Anaemia of chronic infection - HIV, malaria and tuberculosis

5. Chronic renal disease or neoplasm

6. Bone marrow insufficiency- Aplasia or hypoplasia due to malignancy, radiation and drugs

Degree of anaemia

- Mild - hb% level between 9-11 gm%
- Moderate- between 7-9 gm%
- Severe - less than 7gm%

Iron deficiency anaemia is the most common anemia in pregnancy.

Clinical features:

- **Symptoms:-**
  1. Lassitude and feeling of exhaustion or weakness.
  2. Anorexia and indigestion
  3. Palpitation caused by ectopic beats and dyspnoea.
  4. Giddiness and
  5. Swelling of legs
- **On examination:-**
  1. Pallor of varying degrees.
  2. Glossitis and somatitis
  3. Edema of legs
  4. Koilonychia
  5. Soft systolic murmur 'haemic murmer' may be heared.
  6. Crepitations at the base of lungs may be heared due to congestion.

Investigation

The following investigations are to be done-

1. Haematological indices- hb%, total red cell count, pcv,mcv,mch,mchc.

2. Peripheral blood film/smear

3. Serum iron, total iron binding capacity, saturation percentage and serum ferritin.

4. Examination of stool.

5. Complete examination of urine and culture

6. Estimation of serum protein in hypoproteinaemia

7. Hba2 (in chronic cases)

Treatment

- Prophylactic
- Curative

Prophylactic treatment in cludes:

1. Avoidance of frequent child-births:-minimum interval between pregnancies should be atleast 2 years.
2. Supplementary iron therapy: -daily administration of 335mg of ferrous sulphate containing 100 mg of elemental iron along with 0.5 mg folic acid daily for 100 days is quite effective.
3. Dietary prescription:- a balanced diet rich in iron, vitamins, minerals and protein should be prescribed.
4. Adequate treatment should be done to eradicate hookworm infestation, dysentery, malaria, bleeding piles and uti.
5. Early detection of falling hb % level should be made, hb % level should be estimated at first anc visit, at 28^{th} -30^{th} and 36^{th} week

Curative treatment:

1. **Hospitalisation:**

All patients with hb% level of 7 or less should be hospitalised for investigations and treatment.

2. **Specific treatment:**

Choice of treatment depends on severity of anaemia, duration of pregnancy and associated complicating factors.

- **Iron therapy**: oral and parenteral therapy
- **Blood transfusion:**

Iron therapy:

Oral route:

- fersolate tab containing 100 mg of elemental iron is given, once/twice/thrice a day according to severity of anaemia.
- Response of therapy is evidenced by sense of well being, increased appetite, improved outlook of the pt and rise in Hb%.

- Reticulocytosis within 7-10 days of therapy.
- Improvement should be evident within three weeks of therapy. The haemoglobin level is expected to rise atthe rate of about 0.7 gm/dl perweek

-parenteral route:

- Intravenous
- Intramuscular

-repeated injections and total dose infusion(tdi)

- Indications of parenteral therapy are- contraindications of oral therapy, patient seen for the first time during the last 8-10 weeks and non-complaint patients to take oral iron.
- The expected rise in haemoglobin concentration after parenteral therapy is 0.7-1gm/dl per week

Iintravenous -total dose infusion (tdi)-

-the deficit of iron is first calculated and the total amount of iron required is administered by a single intravenous infusion. The compound used is iron sucrose (acog-2008).

- formula used for iron sucose:

Total iron dose(mg)= 2.3 x w x d+500 {w=wt in kg , d= hb% deficit,(target -actual), 500mg for body store}.

-example, the total elemental iron required for an anaemic patient having hb 8gm% weighing 50 kg is calculated as- 2.3 x 50 x 3 + 500= 845 mg. It is given iv, 100mg in 100ml normal saline over 15 minutes.

Intramuscular therapy:

-iron dextran and iron-sorbitol-citric acid complex are used.

-total dose to be administered is calculated as in i.v therapy and given repeated injections i.m in buttocks using "z" technique.

Blood transfusion

- Blood transfusion in anaemia of pregnancy is very much limited.
- Less than 36 weeks of pregnancy.
- Indications are-to correct anaemia due to bloodloss like in aph and to combat post-partum haemorrhage.
- Patient with severe anaemia seen beyond 36 weeks of pregnancy. -

- Refractory anaemia..
- Only packed cells are to be transfused.

Management during labour First

stage:

- The patient should be on bed and should lie in position comfortable to her.
- Arrangements for oxygen inhalation-are to be kept ready.
- Strict asepsis is to be maintained to minimize puerperal infection.

Second stage:

- Asepsis is maintained.
- Prophylactic low forceps or vacuum delivery may be done to shorten the duration of second stage of labour.
- Restricted fluid to be given

Third stage:

- one should be very vigilant during the third stage.
- oxygen inhalation to be given accordingly.
- restricted i.v fluids.
- active management of third stage must be done by giving 10 units of oxytocin intramuscularly (inj. methergin should be avoided)
- significant amount of blood loss should be replenished by giving packed cell transfusion. **Puerperium:**

-prophylactic antibiotics are given to prevent infections.

-iron therapy should be continued for at least 3 months following delivery;

-patient should be warned of the danger of recurrence in subsequent pregnancies.

Govt of india is providing iron tablet free of cost to all antenatal women containing, 100mg of elemental iron and 0.5 mg of folic acid.

Mohfw goi 2013

Guidelines for control of iron deficiency anaemia

Pregnant women and lactating mothers

Iron and folic acid tablets are being distributed through sub-centres, primary health centres (phcs), community health centres (chcs) and district hospitals (dhs) to all pregnant women and lactating mothers.

Implementation

Provision of IFA tablets to pregnant women will be during routine antenatal visits at subcentre/ PHC/CHC/DH. ASHA to ensure provision of IFA supplements to pregnant women who are not able to come for regular antenatal checkups through home visits. She will also monitor compliance of IFA tablets consumption through weekly house visits.

Pregnant and Lactating Women

Screening of all pregnant women for anaemia at sub-centre/VHND/outreach/PHC level can be done by Sahli's haemoglobinometer or by Standard Hb Colour Scale. Therapeutic dose of oral IFA supplementation can be initiated even on clinical signs and symptoms, however, such cases must be referred for confirmation of degree of anaemia through Hb testing and for further management as per Table.

Haemoglobin Level	Level of facility	Therapeutic regimen
Hb level between 9-11 gm/dl		
9-11 gm/dl (at least the	Signs and symptoms (generalised weakness, giddiness, breathlessness, etc.) Clinical examination (pallor eyelids, tongue, beds, palm, etc.) Confirmation by testing	• 2 IFA tablets (1 in the morning and 1 in evening) per day for at least 100 days 200 tablets of IFA). • Hb levels should preferably be reassessed at monthly intervals. If on testing, Hb has come up to normal level, discontinue the treatment. nail • If it does not rise in spite of the administration of 2 tablets of IFA daily and dietary laboratory supplementation, refer the woman to the next higher health facility for further management.

7-9 gm/di	PHC/CHC	Hb level between 8-9 gm/dl
	Signs and symptoms woman	• Before starting the treatment, the
	(generalised	should be investigated to defect the cause of
	weakness, giddiness,	anaemia. -
	BPeathlessness, etc.) level	• Oral IFA supplementation as for Hb
	Clinical examination	9-11 gm/dl. Hb testing to be done every
	(pallor of eyelids,	month.
	tongue, nail beds,	•Depending on the response to treatment,
	palm, etc.)	same course of action as prescribed for Hb
	Confirmation by	level between 9-11 gm/dl.
	laboratory testing	• Hb level between 7-8 gm/dl
		• Before starting the treatment, the woman should be investigated to diagnose the cause of anaemia.
		• Injectable IM iron preparations (parenteral iron) should be given if iron deficiency is found to be the cause of anaemia.
		• IM iron therapy in divided doses along with oral folic acid daily if women do not have any obstetric or systemic complication; repeat Hb after 8 weeks: If the woman has become non-anaemic, no further medication is required: if Hb level is between 9-11 gm/dl, same regimen of oral IFA prescribed for this range
		• If woman with Hb between 7-8 gm/dl comes to PHC/CHC in the third trimester of pregnancy, refer to FRU/MC for management.
		Multiple dose regime

Intramuscular (IM) - Test dose of 0.5 ml given deep IM and woman observed for 1 hour. Iron dexfran or iron sorbitol citrate complex given

as 100 mg (2 ml) deep IM in gluteal region daily. Recommended dose is 1500-2000 mg

(IM in divided doses) depending upon the body weight and Hb level. If parental iron therapy is contradicted e.g. in CHF, H/O allergy, asthma,eczema; Haemochromatosis, liver cirrhosis, rheumatoid arthritis and acute liver cirrhosis, rheumatoid arthritis and acute renal failure etc, refer the woman to FRU/MC

<7 gm/dl	FRU/DH/MC	Hb level between 5-7 gm/dl
	Signs and symptoms for Hb	• Continue parenteral iron therapy as
	(generalized,	level between 7-8 gm/dl. Hb testing to be
	weakness, giddiness	done after 8 weeks
	breathlessness, etc.)	• If the woman becomes non-anaemic, no
	Clinical examination	further medication is required.: if Hb level is
	(pallor eyelids, tongue,	between 9-11 gm/dl, same regimen of oral
	nail beds, palm, etc.)	IFA prescribed for this range
	Confirmation by	• Depending on the further response to
	laboratory testing	treatment, same course of action as
		prescribed for Hb level between 9-11 gm/dl Hb
		levels less than 5 gm/dl
		• Refer patient to higher center for further
		management

preparation:

- Evidence for injectable IV sucrose under Randomised Control Trial of GOI
- Immediate hospitalization irrespective of period of gestation in hospitals where round-the-clock specialist care is available for intensive personalized care and decision for blood transfusion (packed cell transfusion)

Pre-requisites for parenteral therapy

- Should be given under proper supervision
- After test dose only
- Close monitoring required
- Inj. Adrenaline, Hydrocortisone and oxygen to be available for management of anaphylactic reactions.
- Cardiopulmonary resuscitation facility to be available.
- Other indications for parenteral iron therapy are poor compliance or intolerance to oral iron therapy.

Post-partum/post-ncital period

If the woman is non-anaemic in post-partum period, prophylactic regime (1 tablet per day for 100 days) should be given.

Precautions for oral therapy

- Intake of doses as per regime, should be taken regularly and must complete the treatment
- Ideally, tablets should be taken on empty stomach for better absorption. In case of gastritis, nausea, vomiting etc., advise to take one hour after meal or at night
- If constipation occurs, advise to drink more water and add roughage to diet
- IFA tablets should not be consumed with tea, coffee, milk or calcium tablets
- IFA treatment should always supplemented with diet rich in iron, vitamins

(particularly Vitamin C), protein, minerals and other nutrients e.g. Green leafy vegetables, whole pulses, jaggery, meat, poultry and fish, fruits and black gram, groundnuts, ragi, whole grains, milk, eggs, meat and nuts, etc.

Megaloblcistic anaemia

Megaloblastic anaemia is characterized by blood and bone marrow abnormalities from impaired DNA synthesis in which there is derangement in red cell maturation and production of abnormal precusors cells known as megaloblasts.

It is caused by deficiency of vitamin B-12, folic acid or both.

- Vitamin B-12deficiency is rare in pregnancy so megaloblastic anaemia is mainly due to deficiency of folic acid.
- Incidence varies from 0.5 to 3%.
- More common in multiparae, 5 times more than in primigravidae and in multiple pregnancy (8 fold increase than in singleton).

Causes: Common causes of Vit B-12 are:

1. Strict vegetarian diet
2. Gastritis
3. Gastrectomy
4. Heal bypass
5. Crohn's disease
6. Drugs- metformin and proton pump inhibitors
7. Addisonian pernicious anaemia
8. Megaloblastic anaemia of malabsorption syndrome.

Causes of folic acid deficiency are:

1. Inadequate intake
2. Increased demand as in pregnancy
3. Abnormal demand in case of infection and haemorrhage
4. Diminished absorption in malabsorption syndrome
5. Failure of utilization- pt on anticonvulsant therapy
6. Diminished storage- hepatic disorders
7. Associated with iron deficiency anaemia.

- When anaemia fails to improve with iron therapy addition of folic acid should be tried before proceeding for a detailed investigation.

Clinical features:

Specific to megaloblastic anaemia are:

1. The onset is usually insidious and is first revealed in the last trimester or acutely in early puerperium

2. Anorexia or protracted vomiting
3. Occasional diarrhea
4. Neurological symptoms like paraesthesias and numbness of extrimities.
5. Constitutional symptom like unexplained fever is often associated.
6. Haemorrhagic patches under the skin and conjunctiva
7. Hepatomegaly and spleenomegaly

Haematological findings:

1. Hyper-segmentation of the neutrophils 5 or more lobes, megaloblasts, howell- jolly bodies, giant polymorphs, macrocytosis and anisocyfosis.
2. Mcv is more than 1000, mch is high > 33pg, but mchc is normal
3. Serum iron is normal or highand iron binding capacity is low.
4. Associated leucopenia and thrombocytopenia.
5. Serum folate is below 3ng/ml (n-3-8)
6. Serum vitb-l2 level is below 9opg/rnl(n-300)
7. Bone marrow shows megaloblasticerythropoisis

Complications:

1. Abortion
2. Dysmaturity
3. Prematurity
4. APH
5. Fetal malformation like cleft lip, harelip, neural tube defects.

Prophylactic therapy:

All woman of reproductive age should be given 400 μg of folic acid and additional amount 4mg should be given in situations where demand is high.

Women with previous history of having babies with neural tube defects should be given 4mg of folic acid daily 1 month before conception till 12th week of pregnancy.

Curative:

1. Daily administration of 4mg folic acid orally and continued forfour weeks following delivery.
2. Supplementation of 1 mg of folic acid with iron and nutritious diet.
3. Supplementary intramuscular injection of vit B daily or alternate day may be added when response to folic acid alone is not adequate.

THE INDIAN MTP ACT

To avoid the misuse of induced abortions, most countries have enacted laws whereby only qualified Gynaecologists under conditions laid down can do abortions in clinics/hospitals that have been approved. The Medical Termination of Pregnancy Act was enacted by the Indian Parliament in 1971 and came into force from 01 April, 1972. The MTP act was again revised in 1975.

The act was amended in 2003 to include the medical methods of abortion with two drugs Mifepristone (RU 486) and Misoprostol

Under the act pregnancy can be terminated upto 20 weeks of gestation.

The MTP Act lays down the condition under which a pregnancy can be terminated, the persons and the place to perform it.

The reasons, for which MTP is done, as interpreted from the Indian MTP Act, are:

(i) where a pregnant woman has a serious medical disease and continuation of pregnancy could endanger her life like:

- Heart diseases.
- Severe rise in blood pressure.
- Uncontrolled vomiting during pregnancy
- Cervical/ Breast cancer.
- Diabetes mellitus with eye complication (retinopathy).
- Epilepsy.
- Psychiatric illness.

(ii) Where the continuation of pregnancy could lead to substantial risk to the newborn leading to serious physical/ mental handicaps examples like

- Chromosomal abnormalities.
- Rubella (German measles) viral infection to mother in first three months.
- If previous children have congenital abnormalities.
- Rh iso-immunisation.
- Exposure of the fetus to irradiation.

(iii) Pregnancy resulting of rape.

(iv) Conditions where the socio-economic status of the mother (family) hampers the progress of a healthy pregnancy and the birth of a healthy child.

Failure of Contraceptive Device irrespective of the method used (natural methods/ barrier methods/ hormonal methods).

This condition is a unique feature of the Indian Law. All the pregnancies can be terminated using this criterion.

Consent:

- If married--- her own written consent. Husband's consent not required.
- If unmarried and above 1 8years ---her own written consent.
- If below 18 years ---written consent of her guardian
- If mentally unstable --- written consent of her guardian.
  Consent assures the clinician performing the abortion that she has been informed of all her options and has been counseled about the procedure, its risks and how to care for herself after she has chosen the abortion of her own freewill.

Person or persons who can perform MTP

Physicians qualified to do MTP are:

- Any qualified registered medical practitioner who has assisted in 25 MTPs.
- A house surgeon who has done six months post in Obstetrics and Gynecology.
- A person who has a diploma /degree in Obstetrics and Gynecology.
- 3 years of practice in Obstetrics and Gynecology for those doctors registered before the 1971 MTP Act was passed.
- 1 year of practice in Obstetrics and Gynecology for those doctors registered on or after the date of commencement of the Act.
- Whenever the pregnancy exceeds 12 weeks but is below 20 weeks opinion of two registered medical practitioners is necessary.

Place where MTP can be performed:

Any institutions licensed by the Government to perform MTP**. The certificate issued by the Government should be conspicuously displayed at a place easily visible to persons visiting the place.**

DIAGNOSIS:

A. Clinical diagnosis

- A thorough history including date of LMP
- An internal examination to confirm the duration of pregnancy.
- GPE including blood pressure and weight

B. Laboratory Diagnosis

- Urine test for confirmation of pregnancy.
- Routine urine analysis.
- Routine blood counts including hemoglobin estimation.
- Blood group and Rh factor.

- Fasting Blood Sugar
- HIV, HbsAg, HCV
- For 2nd Trimester Abortions with Ethacridine - Renal function

C. Radiological Diagnosis

- Ultrasound may be required, If the client does not remember her LMP or there is disparity between period of amenorrhea and clinical examination

Methods of Induced Abortion:

Abortion can be induced by different methods depending on the weeks of pregnancy completed.

Methods of MTP

- Upto 12 weeks of pregnancy
  1. MEDICAL METHOD
     - Miférpristone and Misoprostol
  2. SURGICAL METHOD
     - Suction evacuation/Vacuum aspiration!

Suction curettage

-Dilatation and Curettage(D&C)

1. MEDICAL METHOD - By Mifepristone and Misoprostol

The termination of pregnancy by medical methods is allowed upto 7 weeks period of gestation (49 days from the first day of last menstrual period in a woman with a regular cycle of 28 days)

Mode of Action:

MIFEPRISTONE (RU-486)- It is a derivative of norethindrone and has antiprogestin action

-it binds to progesterone receptors at endometrium and decidua so leads to necrosis of placenta and detachment

- it softens cervix

- it causes uterine contractions

MISOPROSTOL - it binds to myometrial cells causing strong myometrial contractions and causes cervical softening and dilatation. This leads to expulsion of conceptus from the uterus

Contra indications:

Absolute:

1. Inherited porphyria
2. Allergy to drugs

Relative:

1. Anemia (Hb<8gm%)
2. Suspected/confirmed ectopic pregnancy
3. Female on anticoagulant therapy
4. Chronic adrenal failure
5. Current use of systemic corticosteroids
6. Uncontrolled Hypertension with BP 160/100
7. Cardiovascular disease as angina, valvular disease
8. Severe renal, liver or respiratory distress
9. Uncontrolled seizure disorder

Medical abortion need to be done cautiously in:

- Pregnancy with IUCD in situ (remove IUCD first)
- Pregnancy with fiBPoids
- Pregnancy with scarred uterus
- Bronchial asthma

Pre-abortion counseling:

- Patient should be told that she will have to come for minimum three follow-up visits
- Family support
- Side effects of drugs
- Easy access to health care facility
- D & C will be needed in case of failure
- Risk of congenital malformation in case of continuation of pregnancy
- Contraceptive advice

Consent: Is must (Form C)

Dosage Schedule:

DAY 1 -Mifepristone 200m stat is to be given

- Give her lnj Anti-D, if pregnancy> 6 weeks
- Back-up facility address and phone numbers should be given where she can contact in case of emergency

- She must return to clinic after 24-48 hrs
- She should be told that bleeding may occur with mifepristone alone also

DAY 2-3 - Misoprosfol 400µg (2 tablets of 200pgecich) to be given orally or kept in posterior vaginal fornix

- Ask for H/O bleeding or side-effects if any
- Observe the woman in clinic/hospital for 4 hours
- Give pain relief if required (Paracetamol 500 mg)
- No need to give antibiotics routinely
- Advise her adequate rest and to avoid going out of station
- She should report in case of excessive bleeding/pain(she should be told that soaking two pads per hour for 2 hours in a row is all right at the time of peak cramping which is often the case when the pregnancy expulsion occurs)
- She should avoid intercourse or use condoms or oral pill fill next visit

DAY15-

- Ask for clinical history (regarding the expulsion of products of conception and bleeding) to ensure abortion is complete
- P/V exam (to confirm complete abortion)
- USG-if needed. The clinician must understand that during medical abortion, once the

  gestational sac is expelled, the uterus will normally contain blood, blood clots and decidua, which appear as hyper echoic tissue on USG and may be interpreted as incomplete abortion. In the absence of excessive bleeding, these patients should be followed conservatively
- Contraceptive advice (very important)
- Inform her that next period may be delayed

A written statement signed by the woman must be kept on record if surgical treatment is refused in case of failure

Side-effects:

- Pain
- Bleeding (average 9-11days)
- Nausea
- Vomiting
- Diarrhea
- Headache

- Chills
- Feeling of warmth
- Dizziness & fatigue

These will subside with passage of time and rarely need medication Efficacy:

- A combination of Mifepristone and misoprostol has a success rate of 95-99% for pregnancies up to 7 weeks
- Approximately 1 % woman may require surgical evacuation for heavy bleeding
  • 1 % may fail to abort
- Abortion may be incomplete in 2-3% requiring surgical evacuation

Failure:

- Failure with medical abortion is a term when a surgical curettage is performed for any reason
- True drug failure is defined as presence of cardiac activity 2 weeks following mifeprestone and misoprostol administration

Prevention and Patient Education

- Encourage to come for follow-up when periods resume
- Offer contraception to all seeking medical abortion
- Oral pills/DMPA- stated on day 15 if abortion complete
- Oral pill may be started on day of misoprostal administration as it does not interfere with abortion process
- Cu T – after next periods
- Condoms – preferably intercourse should be avoided till day 15 but can be used if woman has intercourse before that
- Tubal ligation- prefer surgical termination or after next cycle
- Inform about emergency contraception
- Counsel regarding HIV/AIDS

Advantages:

- Abortion possible at early gestation
- Feasible with minimal assistance
- Less complication rate
- More privacy
- Less invasive
- No instruments used
- No anesthesia needed
- No effect on future fertility

Disadvantages:

- Patient needs at least 3 visits
- Bleeding is prolonged (9-11 days)
- Drug side-effect may occur
- Failure in small percentage i.e. 2-3%
- Potential risk of congenital malformation it fails to cause abortion

Record keeping

- All medical practitioners or institution heads should maintain admission register for medical abortion (a combined register can be maintained for medical & surgical abortion)
- The register should include serial no., date of registration, name of patient and name of her husband/father/guardian, address, education, age, religion, marital status, reason for termination, obstetrical history, any significant past/surgical history, findings of GPE and pelvic examination and relevant investigations done
- The prescribed drug protocol should be written clearly, indicating date & time, dose and route of administration
- The follow-up visits should be recorded with comments
- Admission register is a secret document and the particulars of the pregnant woman should not be disclosed to any person except appropriate authority

- Entries made in case sheet, OT register, follow-up card or any other document should not disclose indentify of pregnant woman in any way
- Destination of admission register and other papers: as per MTP regulations 1975 are to be followed

Record of complications and failures

A record of complications especially pertaining to heavy bleeding necessitating I/V fluids, blood transfusion or curettage, sepsis, incomplete abortion, continuation of pregnancy, adverse drug reactions should be maintained

Reporting to authorities

- Every Head of hospital/institution or practitioner should send a monthly record of abortions with Mifepristone & Misoprostol to CMO of the state as per MYP Act
- This report should include the name and address of the practitioner/ Head of institution who provided medical abortion with completed consent forms and any drug reactions noted
- Confidentiality should be maintained

2. SURGICAL METHOD-

A) Vacuum Aspiration (Also k.a. Suction Evacuation/Suction Curettage)

B) Dilatation & Curettage (D&C)

A) Vacuum Aspiration

- Vacuum aspiration, manual or electric is the preferred method of choice for the first trimester surgical abortion
- Manual vacuum aspiration and electrical vacuum aspiration are both equally effective
- According to FIGO Suction Evacuation is the most effective method of 1st trimester abortion
- Counseling is very important, regarding method, follow-up and contraceptive advice

Cervical ripening: (if preg. >8wks)

1 Misoprostal sublingual or intravaginal 400µg is given3-4 hours before the procedure)

Anesthesia

Paracervical block General

Anesthesia

Pre-op procedure:

History/GPE

Routine investigations

Fasting- 6 hrs

Mild sedation

Procedure:

1. Evacuate bladder
2. Lithotomy position
3. Clean and drape (sponge holding forceps)
4. P/V exam (confirm findings)
5. Retract the posterior vaginal wall (Sims speculum)
6. Hold anterior lip of cervix (Volsellum)
7. Clean cervix (antiseptic- Povidone iodine)
8. Give paracervical block (if not under GA)
9. Do cervical dilatation (fully not reqd) Cervical dilator

10. Karman's cannula inserted (by no touch technique)
11. Electronic suction machine or MVA syringe (Manual Vacuum Aspiration) is used to do suction evacuation
12. Pressure created is 600mm of Hg
13. Routine curettage is done in the end to check completion of procedure
14. lnj. Anti-D is given in case of Rh-ye mother

Indications for completion of procedure:

- No more uterine contents coming
- Blood stained froth starts coming
- There is grating feeling
- There is gripping of cannula at the level of internal as
    - Methergin 0.2mg given IN slowly (may not be needed if priming already done with misoprostol)
    - Check the material obtained
    - Clean the vagina and instill povidone-iodine If

no tissue obtained: the reason may be

- Failure to interrupt pregnancy
- Incomplete abortion
- Ectopic pregnancy
- Patient may not have been pregnant at all

Follow-up:

- Observe patient for 2-4 hrs
- Give antibiotics and pain killers
- Contraceptive advice
- Abstinence for 10-14 days
- Immediately report to the doctor in case of acute pain lower abdomen, excessive bleeding, raised temperature or if symptoms of pregnancy persist

Complications: occur in <2% of cases

- Uterine hemorrhage
- Pelvic infection
- Uterine perforation
- Retained products of conception

- Continuation of pregnancy
- Failure rate- <1%
- Mortality rate is < 2/lac

Late sequelae or long term complications:

- Infertility caused by tubal blockade
- Incompetent as following trauma to cervix
- Asherman's syndrome
- Ectopic pregnancy
- Rh-isoimmunisation
- Psychiatric disorders

B) Dilatation & Curettage (D & C) - FOGSI recommends against the routine use of D &C in first trimester terminations

- As in suction evacuation dilatation is done by increasing number of cervical dilators
- I/V Methergin 0.2 mg is given as a routine
- The contents of the uterine cavity are removed with ovum forceps
- Curettage is done to check that the cavity is empty
- The rest of the steps are same as in suction evacuation The

disadvantages are:

- bleeding is more
- It takes more time
- Chances of incomplete evacuation are more
- Complication rate is more between 12 - 20 weeks of pregnancy

1. MEDICAL METHODS

A) Extra-amniotic instillation of Ethacridine lactate
B) lntra-amniotic instillation of hypertonic saline
C) Mifepristone and Misoprostol

2. SURGICAL METHODS

A) Dilatation and Evacuation (D&E)

B) Aspirotomy

C) Hysterotomy

MEDICAL METHODS

A) Extra-amniotic instillation of Ethacridine lactate

Procedure: Involves instillation of 0.1% ethacridine lactate, on oxyfocic, through a Foley's catheter inserted transcervically, in an extra-amniotic space

Steps:

a) After holding cervix with sponge holding forceps, No 16 Foley's catheter is introduced via cervix into uterus, in extra-amniotic space

b) Bulb is inflated with 30cc of normal saline and pulled down so that it snugly fits over internal as

c) Ethacridine is instilled through other channel, either by syringe or drip method

d) Volume to be instilled is POG in weeks X 10 but not more than 150 cc

e) Catheter is wrapped to medial aspect of thigh

f) To expedite the procedure, ethacridine can be combined with prostaglandins or oxytocin

g) Induction abortion interval ranges between 10-30 hours

h) Gentle curettage may be required after expulsion of products

B) Intra-amniotic instillation of hypertonic saline

- Procedure involves trans-abdominal instillation of 20% hypertonic saline by needle introduced in intra-amniotic space, either blindly with prior USG or under USG guidance
- Few deaths because of hypernatremia have been reported so not used these days
- Apart from saline, urea and prostaglandins can be instilled intra-amniotically

C) Mifepristone and Misoprostol

- With the advent of mifepristone and misoprostol use of induction methods have declined. But it must be remembered that use of medical method using mifepristone and misoprostol is not approved in India for second trimester abortions
- Recommended Dose: Mifeprisfone 200 mg stat followed after 36-48 hours by 400 micrograms of oral, sublingual or vaginal misoprostol every 3-6 hours up to doses

2. SURGICAL METHODS

a) Dilatation and Evacuation

- It is mainly employed between 13-16 weeks
- Prior priming is done by using either laminaria tents or misoprostol (preferred)
- This is followed by suction evacuation by larger cannula or evacuation by ovum forceps of the products
- May be associated with many complications like failure of complete evacuation, uterine perforation, cervical lacerations etc.
- Not preferred

B) Aspirotomy

- The procedure is performed to undertake termination of pregnancy beyond 14-20 weeks
- The procedure involves initial slow dilatation with either laminaria tents or misoprostol followed by rapid dilatation during procedure by metal dilators up to no. 14-16
- Then by introducing 14-16 no cannula, membranes are ruptured and amniotic fluid aspirated
- Aspirotomy forceps is introduced and fetal parts are grasped crushed and extracted bit by bit and arranged outside to make sure that no fetal parts are left behind
- An oxytocin drip is started to ensure good uterine contractions, ensure complete evacuation and reduce bleeding
- It is technically difficult, requires proper set up, training and skill and has much higher complication rate, thus not a preferred method for second trimester abortions

C) Hysterotomy

- Abdominal Hysterotomy or mini caesarean section maybe required as method of termination in the following cases:

  1. Women with previous two caesarean sections or other uterine scars
  2. Placenta praevia with risk of severe bleeding by trans-vaginal methods
  3. Where other methods of medical termination have failed or contraindicated
  4. Rarely women have vascular cervical lesion or cancer cervix with risk of massive hemorrhage with vaginal methods

Summary:

Though literature has proved efficacy of misoprostol with or without mifepristone as a safe and effective method of second trimester abortion, it is not an approved method in India. Extra-amniotic instillation of ethacridine lactate remains the method of choice in most cases of second trimester abortions

References:

1. The medical termination of pregnancy act, 1971 (act no. 34 of 1971) (10th August, 1971)
2. Amendment to MTP Rules: (Ref. GSR 485(E) notified on 30th June, 2003)
3. Guidelines for Early Medical Abortion in India using Mifepristone and Misoprostol. Prepared by WHO-CCR in Human Reproduction, Department of Obstetrics & Gynecology, AIIMS New Delhi in collaboration with Ministry of Health and Family Welfare GOI 2007
4. FOGSI FOCUS January 2012 by the Federation of Obstetric and Gynecological Societies of India

FORM C

(See rule 8)

I _____daughter/wife of

_____aged about _____years

(here state the permanent address) at present residing

at _____do hereby give my consent to the termination of pregnancy at _____

_____ (State the name of place where

the pregnancy is to be terminated)

Place:

Date: Signature

(To be filled in by guardian where the woman is a mentally ill person or minor)

I_____son/daughter/wife of _____aged about _____years of _____ at present residing at (Permanent address)

_____

_____do hereby give my consent to the termination of the pregnancy of my ward _____

who is a minor/lunatic at_____ (place of termination of my pregnancy)

Place:

Date: Signature

PRENATAL DIAGNOSIS

Prenatal diagnosis is the science of identifying structural or functional abnormalities - birth by defects in the fetus

Etiology of Birth Defects

1. **Malformation-** it is most common type of structural fetal abnormality. It is an intrinsic abnormality programmed in development regardless of whether a precise genetic etiology is known. e.g. Spina bifida
2. **Deformation-** when a genetically normal fetus develops abnormally because of mechanical forces imposed by the uterine environment. e.g. an otherwise normal limb that develops contractures because of prolonged

oligohydraminos.

3. **Disruption-** it is more severe change in form or function that occurs when genetically normal tissue is modified as the result of specific insult.e.g. Damage from an amniotic band causing a cephalocele or limb reduction abnormality.

4. **Syndrome -** a cluster of several abnormalities of defects with having same cause. e.g. Trisomy 18

5. **Sequence -** all anomalies developed sequentially as a result of one initial insult.
   e.g. Oligohydraminos leading to pulmonary hypoplasa, limb contractures and facial deformities.

6. **Association** - in which particular anomalies occur together frequently but do not seem to be linked etiologically. E.g. VATER association of vertebral defects, anal atresia, tracheoesophageal fistula with esophageal atresia, and radial dysplasia.

Risk involved

Baseline risk for having a child with a serious birth defect is 2-3%

Purpose of prenatal diagnosis:

- To diagnose fetal anomalies and termination of pregnancy.
- Provide a range of informed choice to the couple who are at high risk of having child with congenital abnormality
- Reassurance and reduce anxiety, especially among high risk groups
- Allow the high risk couples to know that the presence or absence of the disorder could be confirmed by testing
- Allow the couples the option of appropriate management (psychological, pregnancy I delivery and postnatal)
- To enable prenatal treatment of the affected fetus

Indications for Prenatal Diagnosis:

- Singleton pregnancy and Advanced maternal age > 35 yrs (most important) - 1 in 385 risk of down syndrome and 1 in 178 risks of chromosomal abnormalities at term.
- Previous pregnancy with chromosomal abnormality.
- Chromosomal abnormality in either parent.
- Family H/O chromosomal abnormality.
- Bad obstetric history (H/O recurrent abortions).
- Family H/O NTD.
- Previous child birth having multiple malformations.
- Couples at risk of inborn error of metabolism or NTD or sickle cell anemia.

- One or both parents' carrier of sex linked or autosomal trait.
- A mentally retarded child present.
- Mother with H/O viral infections like rubella orcytomegalovirus.
- Fetal sex determination in pregnancies at risk of a serious X linked recessive disorders. Parental consanguinity leading to hereditary or congenital abnormalities.
- Maternal illnesses like poorly controlled diabetes mellitus and maternal epilepsy treated with antiepileptic drugs.
- When mother have been exposed to high grade radiations.
- Dizygotic twin pregnancy and maternal age older than 31 at delivery.

Role of Obstetrician in Prenatal Diagnosis:

- Screening of all the pregnant women during the first or second trimester of pregnancy for chromosomal abnormalities and refer all the patients with positive screening to adequate facilities for counselling and further testing. (* Refer patient to higher centre for further management)
- Screening of all pregnant women for fetal anomalies by ultrasound examination at 18- 20 weeks of gestation and refer all women with positive findings to adequate facilities for counselling and further testing.

Methods of Prenatal Diagnosis:

Non invasive	Invasive methods
• Maternal serum markers	• Chronic villous sampling
• **Double screen:** Free β- hCG, PAPP-A, 11-14wks.	• Amniocentetis
• **Triple screen:** MSAFP + Free β-hCG+ unconjugated estriol 15-18 weeks.	• Fetal sampling
	• Fetal biopsy
• **Quadriple screen:** MSAFP + Free β-hCG + unconjugated estriol + inhibin A, 15_20 weeks	• Fetoscopy

First Trimester Screening of Fetal Abnormalities

The 11 to 14 week window of gestational age is the best period to assess gestational age, to screen for fetal aneuploidy based on nuchal translucency (NT) and, to obtain a potentially detailed anatomic survey. Increased NT >3.5mm may be seen in trisomy 21, 18, 13, cardiac defects, diaphragmatic hernia, exomphalos and Noonan syndrome. Early termination of pregnancy has proven its medical and psychological benefits for women than with late termination.

Down syndrome is the most common chromosomal abnormality in liveborn children. It is caused by nondisjunction during meiosis, translocation, and inversion. A woman's risk of having fetus with Down syndrome depends on her age, the gestational age and her history of chromosomal defects. A 33yr old female at 10wks GA has 1 in 352 risk of having such fetus and it decreases with advancing gestation because of spontaneous death of fetuses with chromosomal anomalies, the risk decreases to **1 in 547 at 40wks**.

Neural Tube Defects:

- Second most common major congenital defect (1-2/1000)
- Not a chromosomal anomaly
- Routinely tested and screened for in pregnancy
- Failure of neural tube to close at 28 days of gestation
- 20% are closed lesions and difficult to detect prenatally

Risk Factors of NTDs

- Family history
- MTHFR - methylene tetrahyd rofolate reductase gene mutation.
- Aneuploidy – T13, 18, triploidy
- Diabetes
- Hyperthermia - hot tub, fever (controversial)
- Medications-valproic acid, carbamazepine, coumarin, aminopterin, thalidomide, efavirenz
- UK, India, China, Egypt, Mexico
- Syndromes associated like - Meckel Gubler syndrome, Roberts, HARDE

*Most common structural defects diagnosed prenatally during screening at 11 -1 4wks

- Atrioventricular defects and hypoplastic left heart syndrome
- Acrania
- Holoprosencephaly

- Omphalocoele and gastroschisis
- Megacystitis
- Lethal skeletal defects

BIOCHEMICAL MARKERS

Free Beta Human Chorionic Gonadotropin

Only free beta subunit is used for screening. It increases at the end of first trimester and continues during second trimester, making it useful for both trimester screening. Maternal serum free beta hCG levels are increased in Trisomy 21 (Down Syndrome) and decreased in trisomy 13 and 18. Its levels are normal in fetus with sex chromosomal abnormality. The mean level of free beta hCG in first trimester Down syndrome pregnancies is elevated to 1.98 MoM

Pregnancy Associated Plasma Protein A

Level increases with gestational age and is lower in pregnancies with aneuploidy (trisomy 2 13,18). The median MoM of PAPP-A is 0.5 in aneuploid pregnancies. When used alone, it detects 40% of fetuses with aneuploidy, increases to 48% when combined with maternal age. Mean level of PAPP-A. is reduced to approx. 0.43 MoM in Down syndrome (DS) pregnancies.

Combined Screening Test

It is most effective method of screening of trisomy 21, achieved by a combination of matern age, fetal NT and maternal serum free beta hCG and PAPP-A concentrations at 11 to 13 wks. 6 days. It detects 90% Trisomy21 with false positive rate of 5%.

Dual Test	11-l4weeks	Free beta HCG, PAPP-A
Triple test	15-20 weeks	Free beta HCG, MDAPL, U-estriol
Quadruple test	15-20 weeks	Tripe test + insulin A

Sonography 18-20 weeks

ADVANTAGES OF FIRST TRIMESTER SCREENING

Detection rate is better than second trimester screening. Majority of the patients can be reassured early in gestation of the normalcy of pregnancy and those found to have an affected fetus and who choose to terminate the pregnancy could have it done by a procedure much safer than that used in later gestation.

Screening for fetal aneuploidy Detection of

structural abnormalities:

- Some major structural anomalies are detectable as early as 12 weeks. These include anencephaly, holoprosencephaly, abdominal wall defects, and major limb defects.
- Detection rates are dependent on experience in obstetric ultrasound.

Multiple pregnancies:

- Chorionicity is optimally determined by either visualization or absence of the lambda sign.
- Twin-twin transfusion syndrome is more likely in monochorionic pregnancies with increased NT thickness.

After undergoing combined fetal NT and maternal serum free beta hCG and PAPP-A screening, patients are assigned to high risk category with a risk of 1 in 100 or more, a low risk category with a risk estimate of less than 1 in 1000 or an intermediate risk category with a risk estimate between 1 in 101 and 1 in 1000. Those in intermediate risk category, has further assessment of risk by first trimester US to determine the presence/absence of Nasal Bone, normal/abnormal Doppler velocity waveform in Ductus Venosus, or the presence/ absence of TR. CVS is offered if their adjusted risk become 1 in 100 or more.

SECOND TRIMESTER SCREENING OF FETAL ABNORMALITIES

- Maternal biochemical analytes like MSAFP, free beta hCG, free unconjugated estriol (uE_3) and Inhibin A.
- Ultrasound screening method is called as comprehensive method or genetic ultrasound.

AFP

MSAFP screening is done between 15 to 20 wks. Using maternal serum AFP level of 2.0 to 2.5 MoM as the upper limit of normal.

Factors influencing the MSAFP levels-

- Maternal weight
- Gestational age - MSAFP increases by 15% per week during second trimester, so accurate gestation is essential.
- Race - African - American women have 10 times higher MSAFP despite having a lower NTD risk.
- Diabetes
- Multifetal gestation-a higher screening threshold value is used in twin pregnancies.

- **Elevated Levels Of Maternal Serum AFP (MSAFP) was seen in** Underestimated gestational age, Multifetal gestation, Fetal death, Neural tube defects, Omphalocoele and gastroschisis, Low maternal weight, Renal agenesis, oligohydraminos, Placental abruption, chorioangioma of placenta, Low birth weight, Maternal hepatoma or teratoma, Preeclampsia, Esophageal or intestinal obstruction, Cystic hygroma, sacrococcygeal teratomd, Urinary obstruction,
- **Low levels of AFP seen** in Obesity, Diabetes; Chromosomal trisomies (T21), Gestational trophoblastic disease, Overestimated gestational age.
- **In Neural Tube Defects (NTDs),** after attempting amniocentesis we determine amniotic fluid level of AFP and it is raised. Then an assay of acetylcholinesterase is performed, if it is also positive, it is diagnostic of NTDs

Free Beta hCG levels increased to twice the normal value during the second trimester in fetuses with aneuploidy.

Unconjugated Estriol (UE_3)

The concentration of free estriol in the second trimester is decreased about 25% in aneuploid pregnancies, making it valuable for screening.

Inhibin A: inha levels are not discriminatory in first trimester between fetuses with trisomy 21 and normal ones, but in second trimester affected fetuses show higher concentrations than normal ones.

Triple test: it includes msafp, beta hhcg and ue3. The three variable are independent predictors of genetic risk and in combination with maternal age generate a patient specific risk of having fetus with down syndrome. The gestational age to perform it is between 15 to 21 weeks.

Quadruple test: it includes msafp, beta - hcg, free estriol and inhubin a to assess the risk for down syndrome. The addition of inhibin a improves the detection rate of down syndrome.

Genetic sonogram: ultrasound markers play important role in aneuploidy screening & ideally between 18-20wks. The most powerful markers of aneuploidy are the absence of nasal bone, increased nuchal fold, and cardiac abnormalities. The prior risk is based on serum screening results/maternal age.

Aneuploidy soft markers

1. Structural anomalies, including cardiac (four-chamber and outflow tracts)
2. Short femur (observed to expected <10th percentile)
3. Short humerus (observed to expected <10th percentile)
4. Pyelectasis (anteroposterior diameter of renal pelvis ≥4 mm)
5. Nuchal fold thickening (≥6 mm)
6. Echogenic bowel (similar to echogenicity of iliac bones)
7. Choroid plexus cysts (>10 mm)
8. Hypoplastic middle phalanx of the fifth digit
9. Wide space between the first and second toes (sandal gap)
10. Two-vessel umbilical cord
11. Echogenic intracardiac focus, short tibia, short fibula, short ear
12. Absent nasal bone

Second trimester sonoraphic signs in NTDs

- Lemon sign -frontal bone scalloping
- Banana sign- bowing of cerebellum with effacement of cisterna magna
- Ventriculomegaly
- Small biparietal diameter

Screening Test Sensitivity for Down Syndrome (%)

Age, triple-screen results	60%-70%
Age, quad-screen results	67%-75%

Integrated screening	94%-96%
Stepwise sequential screening	90%-95%
Contingent sequential screening	88%-94%

Invasive procedures for prenatal diagnosis

- Chorion Villous Sampling (CVS), Fetal Blood Sampling (FBS), Amniocentesis, Fetal Biopsy allow testing of fetal materials for chromosomal, genetic, and biochemical abnormalities. All invasive procedures should be done after informed consent of the couple by practitioner with high level of training in obstetric USG under complete asepsis. 300g Anti-D injection intramuscular after invasive procedures is recommended to prevent alloimmunization in Rh negative mothers.

CHORIONIC VILLUS SAMPLING (CVS)

- CVS is performed from 11weeks onwards for the diagnosis of chromosomal and genetic conditions.
- It is associated with more than 50% of diagnosis of chromosomal abnormality because of first trimester screening.
- It can be done transabdominally (preferred) or transcervically

Indications

- Early detection of chromosomal disorders
- Rapid diagnosis
- Fetal karyotype is decided by qf-pcr (quantitative fluorescent polymerase chain reaction) of chromosomes 13, 18, 21 and y or by fish (fluorescent in situ hybridisation)

Complications of cvs

Fetal loss rate - 1 %, **Severe limb defects -** seen with first trimester CVS (done as early as 6-7wk). These defects are not seen after 11 wks gestation.

FETAL BLOOD SAMPLING

Cordocentesis / Percutaneous umbilical blood sampling (PUBS) Indications

- Chromosomal abnormalities
- Single gene defects
- Fetal anemia
- Prior to intrauterine transfusion

- Thrombocytopenia
- Hypoxia and acidosis
- Infection -viral/ bacterial culture
- Monitoring of transplacental therapy
- Pcr/ metabolic and hematological studies -

Complications and risks

- Fetal Bradycardia is most common complication of cordocentesis but is usually transient.
- Fetal Loss 1.4%.
- Hemorrhage or hematoma
- Chorioamnionitis
- PROM
- Placental abruption
- Infection transmission (hepatitis, HIV)
- Maternalisoimmunization in rhesus negative mothers
- Maternal intra-abdominal infection and bleeding
- Needle injury to maternal organs like bowel or vessels.

Fetoscopy: Fetoscopy is performed during the second trimester (after 16 weeks' gestation). In this technique, a fine-caliber endoscope is inserted into the amniotic cavity through a small maternal abdominal incision, under sterile conditions and ultrasound guidance, for the visualization of the embryo to detect the presence of subtle structural abnormalities.

- It is also used for fetal blood and tissue sampling. Fetoscopy is associated with a 3-5% risk of miscarriage, therefore, it is superseded by detailed ultrasound scanning.

FETAL TISSUE BIOPSY

FETAL LIVER BIOPSY

FETAL MUSCLE BIOPSY

Preimplantation biopsy of blastocysts obtained by In vitro fertilization:- These techniques will be helpful for selective transfer and implantation of those pregnancies into the uterus that are not affected by a specific genetic disorder. Place and referral for different intervention:

Place and Referral for different interventions

Level -1 (Sub-center, Community Health Centre, and PHC): Screening of all pregnant women at risk for chromosomal and structural fetal malformation.

Level - II (Civil Hospital): Ultrasound at 11 -13 weeks for structure defects/Genetic ultrasound or level -Ill ultrasound at 18-20 weeks.

Level -III (Medical Colleges/ Institutes / Tertiary Care): Aneuploidy Screening: - First Trimester/Double Screen at 11-14 weeks, Free β-hCG and PAPP-A test.

Second Trimester Screen: Triple test (15-18 weeks) MSAF-P, Free β-hCG, uncogugated oestriol.

Quadruple screen (15-20 weeks) - MSAFP, free βhCG, UE_3, inhibin test.

Level -IV: Advanced centres for fetal medicine with expert ultrasonologist. All invasive procedures CVS, Cordocentesis, Amniocentesis, Fetal Biopsy.

PRE-CONCEPTION AND PRENATAL DIAGNOSTIC TECHNIQUES (PROHIBITION OF Sex Selection) ACT, 1994

Objective of this law

- Prohibit sex selection (before or after conception)
- Regulate pre-natal diagnostic techniques for detecting genetic or metabolic disorders or chromosomal abnormalities or certain congenital mal-formations or sex linked disorders
- Prevent misuse of such techniques for the purpose of sex determination of female foeticide

Genetic Counseling Centre - According to Section 2 (c) it means: an institute, hospital, nursing home or any place which provides genetic counseling to patients

Genetic Clinic - According to Section 2 (d) it means: a clinic, institute, hospital, nursing home or any place which is used for conducting pre-natal diagnostic Procedures. Genetic Clinic includes a **vehicle** where ultrasound machine or imaging machine or scanner or other equipment or a portable equipment is used for detection of sex during pregnancy or selection of sex before conception

Genetic Laboratory

According to Section 2 (e) it means: Laboratory, includes a place where facilities are provided for conducting analysis or tests of samples received from Genetic Clinic for pre-natal diagnostic test. It usually have instruments like

It includes a place where: ultrasound machine, imaging machine, scanner or other equipment which may be used for detection of sex during pregnancy or selection of sex before conception.

Pre-natal diagnostic procedure - According to Section 2 (i) if means all gynecological or obstetrical or medical procedures such as: Ultrasonography, Fetoscopy, taking or removing samples of: amniotic fluid, chorionic villi, blood, any tissue or fluid of a man or a woman before or after conception which is sent to a Genetic Laboratory or Clinic for conducting any type of analysis or pre-natal diagnostic tests for selection of sex before or after conception.

Pre-natal diagnostic test - According to Section 2 (k) if means: Ultrasonography and or test or analysis of: amniotic fluid, chorionic villi, blood, any tissue or fluid of any pregnant woman or conceptus conducted to defect: genetic disorders, metabolic disorders, chromosomal abnormalities, congenital anomalies, haemoglobinopathies or sex-linked diseases.

Sex selection - According to Section 2 (o) Sex selection includes: Procedure, technique, test, administration, prescription or provision for the purpose of ensuring or increasing the probability that an embryo will be of a particular sex.

REGULATION OF GCC, LABORATORIES & CLINICS

As per Section 3 no Genetic Counseling Centre, Laboratory or Clinic:

(1) Unless registered can conduct, associate or help in conducting activities relating to pre-natal diagnostic techniques

(2) Can employ or take services of any person (honorary or payment) who does not possess qualifications

Point to Remember:

No medical geneticist, gynecologist, pediatrician, doctor or any other person can conduct or cause to be conducted or aid in conducting by himself or through any other person any pre-natal diagnostic techniques

Does the law allow the conduct of PNDT?

Yes. As per Section 4(1) conduct of pre-natal diagnostic techniques is allowed only for the detection of:

a. Chromosomal abnormalities
b. Genetic metabolic diseases
c. Haemoglobinopathies
d. Sex-linked genetic diseases
e. Inborn anomalies
f. Other defects or diseases specified by the Central Supervisory Board

Q. When can PNDT be conducted?

Techniques can be used or conducted only when any of the following Conditions

exist:

a. Pregnant woman is above 35 years

b. Pregnant woman has undergone 2 or more spontaneous abortions or foetal loss

c. Pregnant woman has been exposed to potentially teratogenic agents such as drugs

d. Radiation, infection or chemicals

e. Pregnant woman or her spouse has a family history of mental retardation or

f. Physical deformities such as spasticity or any other genetic disease

g. Any other condition specified by Central Supervisory Board

Points to Remember:

- It is mandatory that the person conducting ultrasonography on a pregnant woman to keep complete record in the clinic.
- Any deficiency or inaccuracy found in the records will amount to contravention section 5 or section 6

Q. When can a person conduct of PND procedures?

According to Section 5 No person will conduct the pre-natal diagnostic procedures unless:

a. explained all known side and after effects of the procedures to the pregnant woman

b. obtained her written consent to undergo the procedures in the language which she understands

c. copy of her written consent is given to the pregnant woman

Point to Remember:

Written consent will be taken as provided in **Form G**

- According to Rule 10 (lÀ) any person conducting ultrasonography/image scanning on a pregnant woman will give a declaration on each report that he/she has neither detected nor disclosed the sex of foetus to anybody.
- The pregnant woman before undergoing ultrasonography/image scanning must declare that she does not want to know the sex of her foetus.

What are the conditions for analysis or test of PND procedures?

According to Rule 14 conditions are:

- A Genetic Laboratory can conduct analysis or test of any sample only when referred by Genetic Clinic
- Before every pre-natal diagnostic procedure through ultrasonography the foetus and placenta has to be located
- Pre-natal diagnostic procedure has to be done under direct ultrasonographic monitoring to prevent any damage to the foetus and placenta

PROHIBITIONS: ON PLACES

Section 6 (a) No genetic counseling centre or clinic or laboratory will:

- Conduct pre-natal diagnostic technique including an ultrasonography for the purpose of determining the sex of the foetus.
- According to Rule 17 (1) every genetic counseling centre or clinic or laboratory is required to display prominently a notice in English and in the local language or languages that conduct of sex-determination tests/disclosure of sex of the foetus is prohibited.
- According to Section 19(4) the Registration certificate has to be prominently displayed

PROHIBITIONS: ON PERSONS

Section 18 (1) No person will:

- Open any genetic counseling centre, clinic or laboratory including clinic,

laboratory or center having ultrasound or imaging machine or scanner or any other technology capable of undertaking determination of sex of foetus and sex selection unless such centre, clinic or laboratory is duly registered separately or jointly.

- Render any services to any facility unless such facility is duly registered

o As per Section 4 (4) No person including a relative or husband of the pregnant woman will seek or encourage the conduct of any pre-natal diagnostic techniques on her except for medical reasons

o As per Section 4 (5) No person including a relative or husband of the pregnant woman will seek or encourage the conduct of any sex-selection technique on her or him or both.

o As per Section 6 (b) No person will conduct or cause to be conducted any pre- natal diagnostic technique including ultrasonography for purpose of sex determination

o As per Section 6 (c) No person will allow selection of sex before or after conception.

o As per Section 3 A No person, including a specialist or a team of specialists in the field of infertility will conduct or aid in conducting by himself or by any other person, sex selection on a woman or a man or on both or on any tissue, embryo, conceptus, fluid or gametes derived from either or both of them.

o As per Rule 3 B No person shall sell any ultrasound machine or imaging machine or scanner or any other equipment capable of detecting sex of foetus to any Genetic Counseling Centre, Laboratory, Clinic or any other person not registered under the Act

o According to Section 5 (2) No person including the person conducting a pre- natal diagnostic procedures will communicate to the pregnant woman or her relatives or any other person the sex of the foetus bywords, signs or in any other manner

Prohibition of advertisement

As per Section 22 (1) No person, organization, Genetic Counselling Centre, Laboratory or Clinic including clinic, laboratory or center having ultrasound machine or imaging machine or scanner or any other technology capable of undertaking determination of sex of foetus or sex selection will:

- Issue

- Publish
- Distribute
- Communicate

Any advertisement in any form including internet regarding facilities of pre-natal determination of sex or sex selection before Conception available at such center, laboratory, clinic or at any other place

As per Section 22 (2) No person, organization including Genetic Counseling Centre, Laboratory or Clinic will:

- Issue
- Publish
- Distribute
- Communicate any advertisement regarding pre-natal determination or pre-conception sex selection by any means whatsoever even if its scientific

As per Section 3B and Rule 3A No person will:

- Sell
- Distribute
- Supply
- Rent
- Allow or authorize

The use of any ultrasound machine or imaging machine or scanner or any other equipment capable of detecting sex of foetus whether on payment or otherwise to any Genetic Counseling Centre, Laboratory, Clinic or any other person or body which is not registered

OFFENCES: By Persons

I. If any person acts contrary to the prohibitions listed above he will be liable to be punished with:

- Up to 3 years imprisonment and -
- Up toRs.10,000 fine

Any subsequent conviction entails:

- Up to5 years imprisonment and
- Up to Rs.50, 000 fine

II. In case of a person seeking the aid of the bodies or persons referred to above for sex selection or for conducting pre-natal diagnostic techniques on any pregnant woman for the purposes other than those specified in Section 4(2), he shall be liable to be punished with:

- Imprisonment up to 3 years; and
- Fine which may extend to Rs.50, 000/-

Any subsequent conviction entails:

- Imprisonment which may extend to 5 years and
- Fine which may extend to Rs.1 lakh

Ill. In case of a doctor his name will be reported by the Appropriate Authority to the State Medical Council for taking necessary action:

- Suspension of the registration if charges are framed by the court and till the case is disposed of and
- Removal of his name from the register of the council on conviction for the period of:
  - Five years for the first offence
  - Permanently for the subsequent offence

IV. Husband and relatives of the pregnant woman who undergoes a pre-natal diagnostic technique is presumed to have compelled the woman to undergo the pre- natal diagnostic technique

- Liable for abetment of offence under Section 23(3)
- Punishable for the offence under Section 23 (3)

V. If any person contravenes any provision of the Act or the Rules where no penalty has been specified, he will be liable to be punished with:

- Up to 3 months i**mprisonment** or
- Upto**Rs.1000 fine** or
- Both (imprisonment and fine)

Any subsequent contravention will have **additional fine up** to Rs.500/- for everyday during which such contravention continues after conviction for the 1st contravention

OFFENCES: By Company

In case of offence by company:

- Every person in charge and
- Every person responsible to the company for the conduct of the business of the company at the time the offence was committed

The company shall all be deemed to be guilty and accordingly proceeded against and punished

If consent, connivance of or that it was attributable to any neglect on the part of:

- Director and in relation to a firm, a partner in the firm
- Manager
- Secretary
- Other officer

They shall also be deemed to be guilty and accordingly proceeded against and punished

Q. When will the Court take cognizance of offences?

According to Section 28 Court will take cognizance of offences on a complaint made by—

a. The Appropriate Authority or any officer authorized in this behalf by the Central Government or State Government or
b. a person who has given not less than 15 days' notice to the Appropriate Authority of the alleged offence and his intention to make a complaint to the court.

Which Court can try these offences?

- Metropolitan Magistrate or
- 1st class Judicial Magistrate will try any offence punishable under this law

According to Section 27 every offence is cognizable, non-bailable and non-compoundable Amendment in 2003

Pre-Natal Diagnostic Techniques (Regulation and Prevention of Misuse) Act, 1994 (PNDT), was amended in 2003 to The Pre-Conception and Pre-Natal Diagnostic. Techniques (Prohibition Of Sex Selection) Act (PCPNDT Act) to improve the regulation of the technology used in sex selection.

Implications of the amendment are

- Amendment of the act mainly covered BPinging the technique of pre conception sex selection within the ambit of the ad
- Bringing ultrasound within its ambit
- Empowering the central supervisory board, constitution of state level supervisory board
- Provision for more stringent punishments

- Empowering appropriate authorities with the power of civil court for search, seizure and sealing the machines and equipments of the violators
- Regulating the sale of the ultrasound machines only to registered bodies

Abnormal uterine bleeding

- Definition
- Abnormal bleeding from the uterus in the absence of organic disease of the genital tract is known as dysfunctional uterine bleeding.
- DUB is a group of disorders with dysfunction of the uterus, ovary, pituitary, hypothalamus or other parts of the reproductive system.

Criteria for Normal Menstrual Cycle

Cycle length--- 23-39.4 days (5^{th} & 95^{th} percentile) mean 29.6 days.

Duration --2-7 days Average 5 days

- Average MBL - 40 ml, Menorrhagia > 80 ml
- No significant correlation between MBL and duration of menstruation
- 90% of MBL occurs in first 2-3 days.

Abnormal Menstruation

- Menorrhagia Days> 7, amount> 80ml, cycle remains constant.
- Poly menorrhoea. Cycle duration < 21 days
- Poly menorrhagia. Cycle duration< 21 days. Days> 7, amount> 80ml
- Metrorrhagia. Irregular bleeding
- Meno-Metrorrhagia. Irregular and excessive bleeding Basic classification system.

FIGO Classification for causes of abnormal bleeding per vaginum

- The basic system comprises 4 categories that are defined by visually objective structural criteria
- (PALM: polyp; adenomyosis; leiomyoma; and malignancy and hyperplasia),
- 4 that are unrelated to structural anomalies
- (COEI: coagulopathy; ovulatory dysfunction; endometrial; iatrogenic), and
- 1 reserved for entities that are not yet classified (N).
- The leiomyoma category (L) is subdivided into patients with at least 1 submucosal myoma (LSM) and those with myomas that do not impact the endometrial cavity (LO).

Polyp	Submucosal	Coagulopathy
Adennomyosis	Other	Ovulatory dysfunction
Leiomyoma		Endometrial
Malignancy & hyperplasia		Latrogenic
		Not yet classified

Anovulatory

Puberty Menorrhagia

Premenopausal menorrhagia

Polycystic Ovarian syndrome

Secondary DUB - latrogenic Steroid hormones (oral progesterones, injectable depot prog, IUCDs)

- Low dose prog. - Underdevelopment of spiral arterioles & degenerative lesions of venules
- Depot injections - large superficial dilated venules with atrophy of endometrium
- IUCD - increased MBL
  Superficial ulceration, increased vascularity, interstitial extravasation of RBCs, absence of platelet thrombosis, increase no. of mast cells (heparin), increase no. of macrophages (PGs).

Systemic Causes

- Bleeding disorders - menorrhagia may be first manifestation
- Thrombocytopenia, vWD, afibrinogenemia, factor II, V, VII, X XI deficiency
- Keep possibility of undetected bleeding disorder in all cases of persistent or recurrent menorrhagia -- Get complete blood count done
- Thyroid diseases
- Hypothyroidism - 32-80% incidence of menorrhagia
- Hyperthyroidism - oligomenorrhoea or amenorrhoea
- Iron Deficiency anaemia
- Liver Disorder
- Vascular Disease
- Endocrinopathies

Work up of a case of AUB

Aim to Evaluate about blood loss -- by Hb% Evaluate causes

- Pregnancy
- Gestational trophoblastic disease
- Genital tract Malignancies
- Endometritis

- Myoma
- Endometrial polyp
- Endometriosis or Adenomyosis
- Cervical errosion or polyp

Iatrogenic factors--

- IUCD, OCP, COC, POP, tamoxifen, metoclopamide, digitalis, ginseng, tranquilizers, anticonvusants.

Systemic Disease-

- Haemophilia, A,B,C, Von willebrand's Disease
- Platelet deficiency in; - Leukemia, Severe Sepsis, lIP, Hypersplenism,
- Thrombocytopenia absent radius syndrome TRAS or Glanzmann's thromoasthenia Functional abnormality of platelets mainly seen in Puberty MH.
- Cirrhosis of liver- Reduced Estrogen Metabolism and thrombocyfopenia

Endocrinological Disorders

- Hypothyroidism, Hyperprolactinemia, Diabetes Mellitus, Cushing Syndrome, Late onset CAH, Obesity
- Anaemia
- Hypothalamic causes
- Chronic or systemic illness
- Stress, Athletics, Eating Disorder, Obesity, Drugs

Pituitary Causes

- Hypopituita rism ,Tumor, Infiltration, Infarction
- Good history taking, physical and local exam., Full blood count (S. Ferritin Level is not required)
- DUB in adolescent Girls Coagulation Disorder in 20% (Hospitalized) in 1/4 of Girls with <l0gm%.
- Platelet count, BT, CT, PT, APT1 Factor VIII or IX deficiency, Von wilibrand Disease, Haemophilia.

- ITP --- P1 count, Bone marrow, Anti p1. antibody assay
- Thyroid- T3, T4, TSH, TPO.
- Prolactin., CT Brain
- DHEAS {E2, FSH, LH} not be done in MH

  Non-Invasive ultrasound-- TAS, TVS, Sonohysterogram, uterine size. ET, Endo m cavity, Adnexal.

  Invasive --- Hysteroscopy.Guided endometrial biopsy,
- Dilation and Curettage
- Sometimes Laparoscopy, Colour Doppler, Angiography is requiredafter 35 years female with AUB requires D&C and HPE

Treatment Objectives

1. To control bleeding
2. Prevent recurrence
3. Preserve fertility if required
4. Induce ovulation if conception desired

Management is dependent on the following points

- Age of the patient
- Fertility requirement
- Severity of the bleeding
- The type of dysfunctional uterine bleeding
- Endometrial histology.
- Expectation of the patient.
- **To control bleeding**

  1. HORMONAL
  2. NSAIDS
  3. ANTI FIBRINOLYTIC

4. Gnarl
5. Surgical

1. Hormonal

High dose of Hormones

1. Estrogen --- IN 25 mg of CCE
2. Progesteron-- Nor -Testosteron group 3040 mg / day
3. Combined
4. Androgens

Outpatient Regimen for Treatment of Abnormal Vaginal Bleeding with Oral Contraceptives

One pill QID x4 days

One pill lID x 3 days

One pill BID x7 days

One pill QD x 7-14 days*

Stop all pills for 7 days and then begin cycling on low dose OCP

*length of therapy depends on level of anemia and amount of time required to reach an adequate hemoglobin level for resumption of menstruation

NSAIDs& Antifibrinolytic

Mefanimic Acid 2gm/day

Transexamic, Ethamsylate -2- 6gm/day

Surgical ---

Dilatation and Curettage or Tamponade

Medical Curettage -- High Dose of Harmones for 3-4 days then with withdrawal of drugs.

The progestogen especially nor ethisterone as cyclical therapy is effective for menorrhagia

Dose:

Nor - ethisterone (Norgest, Primok,t-N, DUB, Sysron) 5mg

thrice a day from day 5 to 26 of a cycle

Initially for 3-6 months

Reassess the patient - if she is helped, it can easily be continued for 2-3 years The dose

should be same for Ovulatory and anovulatory DUB.

Medroxy Progesteron Acetate (Modus, Deviry, Meprate) 10

mg thrice a day from day 5 to 26 of a cycle

Initially for 3-6 months

Reassess the patient - if she is helped, it can easily be continued for 2-3 years The

dose should be same for Ovulatory and anovulatory DUB. Combination Oral

Contraceptive PillsFor women with menorrhagia requiring contraception COC are effective

i Reduce menstrual bleeding

ii. Control cycle irregularities

iii. Relieve menstrual pain

Caution:

i. Elderly women

ii. Obese women

iii. Smokers

iv. Contraindications to COC (H/o thromboembolism)

- Evidence suggests that these hormonal contraceptives are effective in the treatment of DUB, and when used long term, reduce flow by 40 to 70 percent.
- Advantages include the additional benefits of reducing dysmenorrhea and providing contraception.
- Method of action - Endometrial atrophy, diminished prostaglandin synthesis and decreased endometrial fibrinolysis.
- COC-Unpopular due to arterial & thromboembolism.

- Low-Dose can be given after ruling h/o smoking, obesity, and hyperlipidaemia & thrombo philia.
- Levonorgestrel impregnated intrauterine contraceptive device: it contains 52 mg of Levonorgestrel mixed with poly-dimethylsiloxane, which releases about 20 □g of progesterone every day.
- Amenorrhoea caused by Levonorgestrel devices is not due to altered ovarian function but due to local suppressive effect.
- Contraceptive benefits are for 5 years and supressive effects for 8 years.
- It has the additional benefit of being a contraceptive. The Pearl index is 0.14 making it compatible to sterilization with a full reversal.

Androgens (Diazole and Gastronome)

- Diazole is an isoxazole derivative of the synthetic steroid 1 7-ethinyl testosterone.
- Diazole creates a hypoestrogenic and hyperandrogenic environment, which induces endometrial atrophy.
- Menstrual loss is reduced by approximately half, and it may even induce amenorrhea in some women.
- For heavy menstrual bleeding, suggested dosing is 100 to 200 mg taken orally every day.
- Has significant androgenic side effects that include weight gain, oily skin, and acne.

GnRH agonists

- It is thus usually reserved as a second-line drug for short-term use prior to surgery. GnRH agonists have significant costs, risks, and side effects.
- Side effects result from a profound drop in serum estrogen levels & include vasomotor symptoms, libido changes, and vaginal epithelium dryness and dyspareunia.
- Importantly, 6 months of agonist therapy can result in a 6 percent loss in trabecular bone, not all of which may be recouped following discontinuation.
- As a result, these agents are not recommended for use longer than 6 months.
- Available are Leuoprolide, triptorelin, gosrelin, naferelin **ORMELOXIFENE**

Easy to administer - 60mg tablet twice a week (Sunday & Wednesday) for 3 months followed by one tablet of 60mg once weekly for next 3 months.

Surgical method

- Surgical D and C
- Endometrial ablation where no tissue is obtained for histopathology, endometrial resection where histopathology is possible. The global or blind technique or second-generation technique which is common these days and hysteroscopy guided technique which is becoming obsolete these days or also known as first-generation techniques.
- Hysterectomy- abdominal, vaginal, LAVH.

I treatment

Minimally invasive surgery

Method	Advantages	Disadvantages
Cryoablation	Not blind	No outcome data
	Less pain	In intracavitary
	No anesthesia	lesions
Thermal balloon	Globally approved	Not in abnormal Ut
Hydrothermal	Hot water circulates	Not in uterus>l0cm
	Direct visualization8mm hysteroscope	
		Stimulates pain
		Risk of burns
Bipolar radiofreq		
	Short procedure	Not in enlarged or
	No pretreatment	Abnormal Ut cavity
Microwave	Large cavity	>1cm myometrium
	Small myomas	Not in prior CCS

- Hysterectomy is curative.

- Although it is excessive surgery if the diseased organ is endometrium.
- Indication for hysterectomy in cases of DUB are:
- Failure of medical management.
- Failure following repeat D and C.
- Failure following ablation or resection methods.
- Patient wants definitive treatment.
- Patient not very compliant for follow up.
- Premalignant conditions.
- History of carcinoma endometrium in the family.
- Associated pathologies requiring surgical intervention

CARCINOMA CERVIX

Cervical cancer is the most common malignancy in females. About 3,70,000 cases diagnosed annually; 78% of the cases in developing countries. It ranks the first carcinoma and breast carcinoma ranks second. Ratio between cervix and breast cancer is 3:1. But in developed countries the ratio between and cervical CA and breast CA is 1:3 where cervical CA ranks the second.lnvasive cancer of the cervix is considered a preventable disease because it has a long preinvasive state, cervical cytology screening program and effective treatment of preinvasive lesion As a result cervical cancer is being diagnosed earlier which leads to better survival rate. This is why the world-wide incidence of cervical cancer is decreasing. Commonest cancer among women.

- Burden of Ca cervix in India
- New cases = 130,000/year
- Deaths = 70,000/yearProjected at 100,000 deaths by 2010
- > 200 females die each day
- One women is dying every seven minutes
- Eight women die each hour
- It has a bimodal distribution with peak: 35-39 years, 60-64 years

- In India the prevalence is higher amongst the comparatively younger age group.
- Major factors influences the prevalence of CA cervix in a population are economic factor, sexual behavior and degree of effective mass screening
- **Risk factors for Ca Cervix**
- Sexual activity at a young age
- High number of pregnancies
- Cigarette smoking
- Long-term use of oral contraceptives
- Other sexually transmitted infections (e.g., HIV and chiamydia
- malnutrition,
- Unhygienic genital health
- lndiviJual's immune status

1. Early age at first sexual intercourse (<1 óyears) has already been discussed that at menarche the SCJ is at active metaplastic state which is more vulnerable to oncogenic agents like sperms, seminal fluids, histones, infections with Trichomonas, chlamydia, HSV-11, H PV- 1 6,18.
2. Early age at first pregnancy.
3. Multiple sexual partners.
4. High parity.
5. Sexually transmitted diseases - syphilis prevails 3.5 times more commonly in CA cervix.

   Chlamydia trichomatis and CMV are strongly associated with cervical CA. Trichomonasis and candidiasis are found in CIN but not in invasive cancer.
6. Smoking It increases incidence by 2 folds.

Diminishes the immune function secondary to systemic effect of cigarette smoke and its biproducts or locally effect of tobacco specific carcinogens

7. Low socio-economic status

Incidence is higher due to poor diet, low hygiene and risk of STDs.

8. IMMUNOSUPPRESSION

- Women who are infected with HIV infection have an elevated prevalence of human papilloma virus infection and persistence of HPV and cervical dysplasia.
- Incidence also occurs more in organ transplant patients.

9. RACE

- Highest incidence seen in black women as compared to white (3 times more) & Low incidence in Muslims (due to circumcision).
- Less in Jewish. Diet
- Low intake of Vit.A, deficiency of Vit.0 and folic acid shown to increase the risk.
- Oral contraceptives - Otherwise controversial but females using OCPs are relatively more sexually active, OCPs also act as steroids -decreases the immunity
- suppresses the P53 gene - increases self-proliferation for cervical dysplasia and leads to adeno - carcinoma.
- Husband whose previous wife died because of CA cervix.

Human Papilloma Virus (HPV) lnfection:

- HPV infection has a central causative role in the etiology of cervical neoplasia. HPV are members of a large family of virus Papova viridae.
- Papilloma virus have a tightly coiled, circular double standard DNA molecules about 8000 base pairs in length The complete virion consists of a DNA core and surrounding proteins.
- HPV infects all surface epithelium including skin and mucus membrane.
- Approx. 100 types of HPV identified. More than 30 of which can affect the lower genital tract of female and there are 14 high risk HPV subtypes.

These are further subdivided into three subtypes according to risk for oncogenecity

1. Low oncogenic risk - 6, 11, 42, 43, 44 - causes low grade CIN and flat condylomata.

2. Inter mediate oncogenic risk - 31, 33, 35, 51, 52 - causes low grade and high grade CIN.

3. High oncogenic risk- 16, 18, 45, 46-invasive CA

- Two of high risk subtypes 16 & 18 are found in up to 62% of cervical carcinomas. HPV 18 is the most specific and is associated with tumour with a more aggressive clinical behavior but HPV 16 is the most commonly found and causes large cell Keratinizing squamous cell CA.Virus enters the genital tract by sexual contact and affects any part of the female genital tract.
- Virus have incubation period 1-6 months.
- After that the first lesion appears and there is a rapid and active growth of the virus, as well as immune response occurs in the host.
- After about 9 months of this activity two outcomes are expected

a) Immune response good - sustain clinical remission.

Immune response is poor - persistent and recurrence disease

Clinical Presentation:

HPV infection may not cause any symptom hence diagnosis is difficult to make. But it is possible by

- Clinical study.

- Cytology.

- Colposcopy.

- Histopathology.

- Molecular biology.

- Clinical diagnosis is based upon the warts on external genitalia, usually due to low oncogenic virus.

Detection of HPV Infection

Cytology

HPV- DNA can be detected by

- PCR

- Southern blotting

- In Situ Hybridisation

- Dot Blot Hybridisation

- Hybrid Capture Technique

Target Population for HPV Screening

- A sexually active woman above the age of 30 years.

VACCINES AGAINST HPV

- Bivalent vaccine - Against 16 and 18 viruses but also give cross protection against others. It is called Cervarix Quadrivalent vaccine against 6, 11, 16 and 18. (Gardasil or Silgard) give protection against genital warts

- Prophylactic vaccine is given as I/M in three doses at 0, 1 and 6 months. It is recommended only for girls up to the age of 26 years. But can be given to males or females age 10-12 years. Preferably before starting sexual activity

Available vaccine 1. Quadrivalent (against 6, 11, 16, 18) Gardasil dose 1st dose, 2nd dose at 2 months, 3rd dose 6th month

2. Bivalent(against 16, 18) Ceryarix dose i' dose, 2nd dose at 1 month, 3rd dose at 6 months

- Cervical Cancer Prevention Primary Prevention

HPV information and prevention

Screening

Behavioral modification Sexual

precautions Prophylactic

vaccines

- **Secondary Prevention**

CIN Detection and treatment

SCREENING

Do a good per speculum examination in good light

VIA_ Apply 3-5 % acetic acid to note any areas becoming white after acetic acid

application.

VILI_Apply lugos iodine to check dark mahgony or light mahgony area Paps smear should be taken once acute massive vaginal discharge is clear. Do not take smear during menstrual phase.

- No P/V before Pap smear
- No lubricant on speculum
- 3600 sweep with Ayre's spatula - smeared on slide

Screening is to be started at 21 years if married not before 21 years even if married for 3years. Co-testing means HPV with pap's smears.

21 years to 29 years ------- do only pap's smear. HPV NOT TO BE DONE

30years to 65 years --- only pap's every 3 yearly, if co-testing is done then every. 5 years.

After 65 years -- Stop if previous 3 negative smears.

After total hysterectomy -- no need if done for benign disesase

In case of abnormal pops smear refer the patient for colposcopy and biopsy if required.

DIAGNOSTIC METHODS

1. Colposcopy - All changes of CIN are exaggerated.

- Abnormal vessels - Loop, branching, reticular cork screw or 1i' shaped pattern.
- Irregular surface contour
- colour tone is yellow-orange but in intact sq. epithelium if is pink and in endocervical

  epithelium is red.

Adeno CA does not have specific colposcopic appearance; abnormal blood vessels maybe seen.

2. Endocervical cureftings to rule out adeno CA. -

Biopsy In presence of obvious lesion

Punch biopsy

Wedge biopsy

Ring biopsy (Ring of tissue from SCJ)Cone biopsy for adeno CA when there is no obvious growth.

Colposcopic directed biopsy - lesion can be delineated before taking biopsy by painting the cervix with Schiller1s iodine (5% iodine in 100% Kl) - stains normal cell mahogany brown and leaves abnormal cells unstained.

Tumour markers - sq. cell CA antigen <2ng/ml is normal, when raised - CA cervix.

PROCEDURES FOR STAGING

- Physical examination - P/V and P/R.examination
- Procedures:

- If there is obvious growth then cervical biopsy.
- If no obvious growth - then colposcopic examination with cervical biopsy and endocervical curettings.
- If diagnosis cannot be established with colposcopy and directed biopsies then cervical conization to be done.
- Cystoscopy - If histopathology positive.
- Proctoscopy.
- Radiological Studies:
- Intravenous pylogram. Barium enema. Chest X-ray. Skeletal X-ray.
- Optional Studies: Not allowed by FIGO
- CT Scan / MRI, Ultrasonography, ,Lymphangiography, PET Scan

Stage	Descrition
I	The Carsinom is Stricly confirmed to the cervix (extension to the uterin corpus should be disregarded)

	IA	Invasive cancer identified only microscopically. (All gross lesions even with superficial inveasion are Stage IB cancers.) Invasion is limited to be measured stronal invasion with a maximum depth of 5 mm[b] and no wider than 7 mm. IA1 Measured Invasion of stroma <_ 3 mm in depth and <_ 7 mm width. IA2 Measure invasion od stroma > 3 mm and <5 mm in depthband <_ mm width.
	IB	Clinical lesions confirmed to the cervix, or preclinical lesions greater than stage IA. IB 1 : Clinical lessons no greater than 4 cm in size. IB 2 : Clicnical lessons > 4 cm in size
II		The carcinoma extends boy and the uterus, but has not extended on the pelvic wall or to the lower third of vagina.
	IIA	Involvement of up to the upper 2/3 of the vargina. No obvious parametrial involvement. IIA 1 : Clinically visible lesion <_ cm IIA 2 : Clinically visible lesion > 4 cm
	IIB	Obvious parametrial involvement but not onto the pelvic sidewall.
III		The carcinomas has extendended on t the pelvic sidewall. On rectal examination there is no cancer-free space between the tumor and pelvic sidewall. The tumor involves the lower third of the vagina. All cases of hydronephrsis or non-functioning kidney should be included unless they are known to due to other causes.
	IIIA	Involvement of the lower vagina but no extension onto pelvic sidewall.
	IIIB	Extension onto the pelvic sidewall, or hydronephrosis non-functioning kidney.
IV		The carcinoma has extended beyond the true pelvic or has clinically involved the mucosa of the bladder and or rectum.
	IVA	Spread to adjascent pelvic organs
	IVB	Spread to distant organs.

* Adapted from FIGO Committee on Gynocologic Oncology [2]

* The depth of ivasion should not be more than 5 mm taken from the base of the epithelium, either surface of glandular, from which it originates. Vascular space invasion should not alert the staging.

Ca Cervix can be managed

* Refer patient to higher center for further management.

- SURGERY
- RADIATION
- CHEMOTHERAPY
- A COMBINATION OF ABOVE

General Treatment Scheme for Invasive Cervical Carcinoma

- Stage IA1 Simple hysterectomy, abdominal or vaginal, or cervical conization Stages
- 1A2,b hA, I131, and nonbulky hA Radical (class Ill) hysterectomy or trachelectomy, bilateral pelvic lymphadenectomy with postoperative irradiation, plus or minus concurrent chemotherapy in selected high-risk patients
- Stages 1132 and bulky hA Full external and intracavitary pelvic irradiation with concurrent chemotherapy (extrafascial hysterectomy) or radical abdominal hysterectomy and pelvic

  (periaortic lymphadenectomy)
- Stages IIB to IVA Full external and intracavitary pelvic irradiation with concurrent chemotherapy Stage IVB Palliative chemotherapy

MENOPAUSE

Menopause means permanent cessation of menstruation at the end of reproductive life due to loss of ovarian follicular activity. Average age is47.5 yrs. Clinical diag. is confirmed after stoppage of menses for 12 consecutive months without any other pathology Therefore menopause is a retrospective diagnosis.

Premature menopause- occurs before 40 yrs.

Natural or spontaneous menopause is after 12 months of amenorrhea for which there are no obvious pathological and physiological causes.

Pre-menopause refers to the entire reproductive period, up to the final menstrual period.

Pen-menopause: It is the period immediately prior to and up to 1 year after the final menstrual period. If may last for 3-5 yrs.

Post-menopause: span of time from the final menstrual period.

Delayed menopause is two SDs above from the natural average age of menopause in a given population ie.> 54 yrs.

Induced menopause is that follows bilateral oophorectomy or iatrogenic ablation of ovarian function.

Post-menopausal bleeding: vaginal bleeding following a woman's final menstrual cycle and not on cyclical hormone therapy. Bleeding that occurs 6 months after amenorrhea warrants investigation.

Diagnosis of Menopause

- Cessation of menses for consecutive 12 months during climacteric.
- Menopausal symptoms , hotilush and night sweats.
- Vaginal cytology: Maturation index of atleast 10/85/5
- Serum Oestradiol < 20 pg / ml
- Serum FSH and LH more than 40 mlu / ml
- (3 values at a week's interval' required)

Risk factors

- Surgical menopause
- Radiation menopause
- Chemotherapy.
- Smoking, caffeine, alcohol
- Family history of menopausal diseases
- Drug: gnrh, heparin,corficosteroids,clomiphene.

Risk factors for osteoporosis

1) NON-MODIFIABLE RISK FACTORS:

   Family history,Age, Race, Body weight (low BMI, small body frame)& estrogen deficiency.

2) MODIFIABLE RISK FACTORS:

- SEDENTARY HABITS
- DIETARY: Decreased calcium&VIT D INTAKE Excessive intake of caffeine products,alcohal & smoking.
- DISEASES: Thyroid disorders, Hyperparathyroidism,Chronic renal disease,Conditions requiring corticosteroids.
- MENOPAUSAL SYMPTOMS

In majority, no symptoms but in some symptoms appear.

1).VAS O-MOTOR SYMPTOMS

- Grading:

Mild - feeling of heat without sweating.

Moderate - feeling of heat with sweating.

Severe - feeling of heat, sweating and palpitation that disrupts usual act

- Hot flushes which last for 2-5 minutes each. Sudden feeling of heat followed by profuse sweating
- Palpitation, anginal pains, fatigue and weakness, depression, lack of conc. and not irritability may be present.

Thyroid function test should be done if vasomotor symptoms are Atypical or resistant to therapy.

2).NEAUROLOGICAL SYMPTOMS:

- VMS
- Feeling of Pins and needles on extremities.

3).URINARY TRACT SYMPTONS:

Atrophic changes may cause: Urethral caruncle, Dysuria with or without infection,Urge/stress incontinence

4) .GENITAL SYMPTOMS

- Atrophic, dry vagina can cause dysparunia.
- Loss of libido, vaginal bleeding. infection, Pruritis & leucorrhea.

5).SEXUAL DYSFUNCTION

- Decreased libido

TREATMENT-

- Treatment of anxiet depression.
- Counselling.
- sex therapist and exercises.
- lubricants, androgens, combination of estrogens & androgens, transdermal patch, testosterone ointment 1% arm, lower abdomen or vagina).

6). PSYCHOLOGICAL CHANGES

- Anxiety.
- Headache.
- Insomnia.
- Irritability.
- Dysphasia.
- Depression.
- Dementia.
- Mood swings.
- Inability to concentrate.
- Crying spells.

7).SKIN:

- Wrinkles
- Purse-String (around mouth) & crow-feet (around eyes).
- Oestrogen cream - Feminine forever cream delays skin changes, but beneficial temporarily & only for initial stages.
- Pricking & itching skin sensation.
- Crawling skin (feels as tiny insects marching along your body). It disappears on its own.

8).HAIR

Some loss of pubic & axillary hair and slight balding.

9).DEMENTIA is more common in post-menopause.

10). ENDOCRINE SYSTEM

- Mild virilisation& obesity.
- Hypothyroidism, DM.

11).PYOMETRA may develop by cervical stenosis. Needs drainage

12). Others: Co nsti pation, Weight gain,Prolapse & stroke. 13).CARDIOVASCULAR DISEASES:

Oestrogen deficiency, can cause atherosclerosis, IHD and MI.

14): OSTEOPOROSIS & FRACTURES

- At 40 years - Total bone calcium amounts to 1200 gms.
- At critical level of 750 gms, female is susceptible to fracture.
- Vertebral bone compression leads to:
    - Dowgers Hump
    - Decreased height
    - Back Pain
- Fracture of neck of femur, wrist &vertebrae

Investigations

1. Complete blood picture, ESR,Urine C/E
2. RBS
3. Serum calcium,
4. Lipidprofile
5. Serum TSH.
6. Stool for occult blood.
7. PAP smear
8. TVS
9. Mammogram/ultrasound.

10. Eye checkup
11. Preferably fasting serum phosphorus
12. Serum creatininé, Serum albumin.
13. Alkaline phosphatase
14. 25hydroxyvitaminD
15. X-ray of thoracolumbar spine (lateral view)
16. PTH (Based on clinical iudgmenf)

Perimenopausal bleeding

1. Sonohysferography is superior to TVS to defect intra-cavitary lesions.
2. Perform Endomefrial tissue sampling in patients with AUB older than 40 yrs.
3. TVS is primary screening test for AUB, consider MRI when diagnosis is inconclusive.

Post menopausal bleeding.

1. Initially do TVS & endometrial biopsy.
2. Women with PMB with ET of < 4 mm in TVS do not require endometrial sampling unless high-risk for endometrial carcinoma or bleeding is episodic.
3. If ET is > 4 mm in TVS, consider endometrial sampling.

In women with homogeneous and normal morphology, women on HI and hypertensive medication, the acceptable combined thickness is 6 mm.

4. D&C and fractional curettage are useful in low resource settings.
5. Saline infusion sonography and 3D USG play a limited role in PMB evaluation.

Osteoporosis

Diagnosis of an osfeoporotic fracture is by the presence of fragility fracture and or by BMD. BMD can be studied by

1. CT
2. DUAL energy X-Ray absorptiometery (DEXA or DXA) DEXA of

the hip & spine is primary technique for BMD assessment.

WHO BMD(t-score) based diagnosis of osteoporosis for postmenopausal women

- Normal 1-score above (i.e., better than) -1.0
- Osteopenia or low bone mass 1-score between -1.0 and -2.5
- Osteoporosis 1-score below (i.e., worse than) or equal to -2.5

Severe osteoporosis 1-score below -2.5 with fragility fracture. WHEN

HRT IS CONSIDERED, DOCUMENT:

1 - Base-line pelvic ultrasoundwhich includes

-OVARIAN SIZE

- ENDOMETRIAL THICKNESS

2-Mamography.

3-E 2, FSH levels

Management/Prevention

1. Spontaneus menopause is unavoidable.
2. Artificial menopause can be delayed or prevented.

- HOLISTIC APPROACH

1. Adopt a holistic approach & selectively prescribe HRT.
2. Use Minimal dose of HRT required, it avoids risks while giving the beneficial effect.

Counselling

- Family members should be sympathetic.
- Explain about physiologic events.
- Counselling will minimise fear, depression & insomnia
- Advice on contraceptives.

Life-style changes

- Active detachment plays constructive role in family affairs.
- Watch I V, read books and do .prayers.
- Wear light, loose, layered clothes.
- Comfortable Room temperature.

- Avoid alcohal, smoking, caffeine.
- Develop hobbies.

Tranquilisers & antidepressants

Mild tranquilisers relieve anxiety, sleeplessness and depression.

Vasomotor symptoms

1. Life-style modifications.
2. Most effective is HI
3. Low dose OCP can be used in transition phase.
4. Non-hormonal agents may relieve VMS, but have side-effects. Can be considered when HI is contraindicated or not desired.

Non-Hormonal Treatment-

1- NUTRITIOUS DIET

- Low in fat, sugar, salt & animal products.
- Adequate intake of water.
- Non-fattening diet with calcium & protein
- Soyabeans.

2- SUPPLEMENTARY CALCIUM

- Diet should include at least 1.2 gms calcium.
- Vit. A,C,E
- Daily intake of 1 to 1.5 gms. of calcium reduce osteoporosis & fractures.

3- EXERCISE:

- Weight-bearing exercises prevent or delay osteoporosis.
- Yoga, meditation, social work reduce mental stress.
- Acupuncture.

4- VITAMIN D:

- D Vitamin D3 (400-8001U/day) with calcium reduce osteoporosis and fractures.
- Sunlight enhances synthesis of cholecalciferol (VII. D3) in the skin. D

5-.FAMILYFEUDs: Should be attended & members counselled for cordial relations.

• BIPHOSPHONAIES:

1). Commonly used drugs:

-Alendronate

-Ibandronate (150 mg/ month FOGSI

-Risedronate (35 mg/ week or 5 mg /day FOGSI)

-Pamidronate & Clodronate can be given IN 2).

2). SIDE EFFECTS

- Oesophageal ulcers.

- GIT distress.

-Arthralgia, myalgia.

3). Are taken empty stomach.

4). Alendronate (5 mg/day or 35 mg/week) & Risedronate (5 mg/day or 35 mg/week has been approved by FDA for prevention of osteoporosis.

- Alendronate is 1000 times more potent than efidronate with no side effects.
- It is marketed as OSTEO : FOS-1 0.
- Dose : 35 to 70 mg /week (NOVAK)

  10 mgl day (FOGSI).
- Etidronafe
- 10 mg/Kg (Appx. 400 mg /orally daily) is given for two weeks followed by a gap -of two weeks x 3 months(3 months course).
- Course is repeated for 10 such cycles.
- Don't give with calcium, tea, coffee or juice.
- Calcium should be taken in the morning and etidronate swallowed (NOT CHEWED) in the afternoon on an empty stomach with water in the upright position. Nothing to be taken orally at least for 30 minfues.
- To remain upright for 30 mm.. This reduces oesophageal irritation.

- Milk & antacids can reduce gastric irritation.
- Overdose causes hypocalcaemia.
- FLUORIDE:
- prevents osteoporosis & increases bone matrix.
- 1 mg /kg for short term only.
- Calcium to be continued.
- Long term therapy induces side effects(brittle bones). The

  role of Calcitonin & Fluoride are not established.
- CALCITONIN
- injection is costly.
- Simultaneous calcium (ig) & vitamin D3 (8001U) should be given daily.
- Not first line drug but recommended in patients with substantial pain of osteoporotic fracture because of its Analgesic action.
- Once substantial pain subsided or not subsided in 4 weeks, give other therapy.
- CLONIDINE THERAPY
- To treat HOT FLUSHES.
- In hypertensive patients, not responding to oestrogen therapy (SHAW) or if oestrogens are contraindicated.
- 0.2 to 0.4 mg/ daily.
- NOVAK: - Orally: 0.1 to 0.2 mg / daily or

  - weekly transdermal patch 0.1 mg/day.
- Side effects: Orthostatic hypotenfion & drowsiness
- PAROXETINE
- Is a selective serotonin reuptake inhibitor (SSRI's).
- Reduce hot flushes.
- PAROXETINE CR (PAXIL): 12.5 and 25 mg/ day

Side effects: headache, nausea & insomnia.

- Other drug is FLUOXETINE: 20 mg / day.
- PHYTOESTROGENS

  Phytoestrogens, containing isoflavones lower incidence of VMS, osteoporosis & CVDs

- Soy beans contain isoflavone which is strongly oestrogenic,
- 45 to 60 mg soy protein daily is protective
- It also decreases Cholesterol, LDL & Triglycerides with a marginal increase in HDL.
- It has : Antiviral,Antifungal,-Anticarcinogenic effects
- SELECTIVE OESTROGEN RECEPTOR MODULATORS (SERMS)

  Out of many SERMS, RALOXIFENE increases BMD, reduces serum LDL & raises HDL- 2 level.

  RALOXIFENE (EVISTA) is approved by FDA

- It is one of the first line drugs for prevention of osteoporosis.
- Has very low risk of endometrial & breast cancer.
- It is given 60 mg daily with Ca and Vit D.

SIDE EFFECTS

1. Hot flushes
2. Cramps
3. Venous thrombosis.
4. Retinopathy
5. Haemopfysis
6. Headache, migrane
7. Loss or change in speech
8. Vision problems
9. Pain or numbness in arms, chest, legs

10. Shotness of breath

CONTRAINDICATIONS:

1. Venous thrombosis.
2. Not given with oestrogens, lndomethacin, Naproxen, lbuprofen, Diazepam
3. Hepatic dysfunction.
4. STOP THE. DRUG 72 HRS BEFORE SURGERY

- BELLEGRAL.
- Reduces hot flushes.
- Bellergal- S - 0.2 mg BD (FOGSI)
- Surgery/medical managemen for:

- Arthritis, - UTI, SUI
- cataract , - Urinary fistula
- osteoporosis, - Rectovaginal fistula

DENTAL AND ORAL CONDITIONS STANDARD TREATMENT GU- IDELINES

PREOPERATIVE ASSESSMENT OF THE PATIENT

- A thorough assessment of a patient's medical status is standard practice when dental care is provided.
- Although this is true for procedures performed under local anesthesia alone, the information gathered may be viewed somewhat differently if the dentist is planning to use sedation or general anesthesia as an adjunct to dental treatment. It is a 2-part sequence and the first part will address general principles and cardiovascular considerations. The second part will address pulmonary, metabolic, and miscellaneous disorders.

RECORDING THE MEDICAL HISTORY

Anesthetic History

The patient should be questioned carefully regarding past experiences with local and general anesthetics. Most patients vividly recall any unpleasant experiences, regardless of their true significance. For those who have had little or no experience with any form of anesthesia, questions regarding other family members may be helpful, because the patient may be genetically or psychosocially predisposed to an adverse anesthetic outcome. This is especially true for general anesthetics. Finally, information regarding past hospitalizations will enlighten the examiner regarding the patient's status.

Current Medications

Information regarding a patient's medications not only provides insight regarding his or her medical status, but may alert the dentist to possible drug interactions. Careful attention should be paid to any prescribed medications the patient is taking currently or has taken within the past month. In the case of corticosteroids, extended use (ie, greater than 2 weeks) within the previous month or 2 presents a risk for adrenal atrophy that may indicate a need for glucocorticoid prophylaxis. This is especially true if extensive treatment is planned, or a stormy postoperative course is anticipated. Finally, questioning should be directed to include any medications prescribed but not taken by the patient.

With only a few exceptions, there is little reason to discontinue any medication prescribed for cardiovascular disease. Diuretics can be withheld until after the appointment to minimize need for micturition. Their long-term use may be associated with hypokalemia and risk for cardiac arrhythmias; any irregularity in the patient's baseline pulse should be viewed with suspicion. It may be wise to order a serum

potassium level if a general anesthetic is planned.

Antihypertensive drugs can potentiate the hypotensive influences of sedatives and anesthetics, but there is even greater risk for acute rebound hypertensive episodes if long- term medications other than diuretics are withheld. With the possible exception of angiotensin-converting enzyme (ACE) inhibitors and angiotensin-receptor blockers (ARBs), it is better to continue these medications and give particular attention to intraoperative monitoring and cautious ambulation following postural change. A thorough review of considerations regarding cardiovascular medications was presented in previous continuing education articles in this journal.

All chronically prescribed psychoactive agents should be continued. In most cases, their therapeutic influence requires a steady state serum concentration that has taken several weeks to establish. Furthermore, interrupting sedative/anxiolytics such as benzodiazepines may result in bothersome signs and symptoms of withdrawal. No major interactions with psychoactive drugs are known in anesthetic practice, other than monoamine oxidase inhibitors. The use of meperidine is contraindicated for patients taking this category of antidepressant because the interaction can precipitate seizure and a hypertensive crisis. Putative interactions regarding vasopressors and antidepressants have been overstated. Although indirect-acting sympathomimetics should be avoided, the judicious use of epinephrine or levonordefrin is not contraindicated for patients medicated with this or any of the remaining categories of antidepressants, including tricyclic antidepressants and selective serotonin reuptake inhibitors.

Often we forget to question patients regarding use of nonprescription drugs, recreational drugs, and homeopathic supplements. Cough, cold, and allergy medications may have a direct impact on vital sign assessment, and they introduce risk for cardiac arrhythmias and drug interactions. The same can be said for appetite suppressants, decongestants, and cocaine, because all are sympathomimetic amines. Recommendations have been provided for managing asymptomatic cocaine abusers,6 and they are reasonable suggestions if there is evidence of excessive use of any sympathomimetic agent. The dentist may wish to avoid outpatient general anesthesia, opting instead for sedation and local anesthesia and, along with monitoring for blood pressure and pulse, electrocardiographic (ECG) monitoring of such patients is encouraged.

Patients who smoke are prone toward hyperactive airways and bothersome episodes of coughing, in addition to well- established compromise of cardiovascular and respiratory function. Heavy smokers have a reduction in oxygen binding sites on hemoglobin due the presence of carbon monoxide, which also impairs unloading of oxygen from the hemoglobin. This shifts the hemoglobin dissociation curve to the left and negates the accuracy of pulse oximetry to accurately reflect the patient's actual

partial pressure of oxygen in arterial blood (PaO2). Whereas a hemoglobin saturation of 95 by pulse oximetry normally reflects a PaO2 of ~80 mm Hg, oxygen tension will be lower in a patient with elevated carbon monoxide levels. It is wise to oxygenate heavy smokers and to maintain pulse oximeter readings >95. It may also be useful to administer a bronchodilator such as albuterol preoperatively, especially when a general anesthetic is planned.

The long-term use of opioids or other CNS depressants, either prescribed or abused, should alert the clinician to possible dependence. In such cases, it is advisable to maintain the patient's current level of use. For those suspected of opioid dependence, it is wise to avoid the use of nalbuphine, pentazocine, or butorphanol, because these particular opioids have mixed agonist-antagonist action that may precipitate a withdrawal syndrome.

All NSAIDs inhibit cyclooxygenases from converting arachidonic acid to prostaglandins. This shifts the arachidonic acid pathway toward another group of enzymes, lipoxygenases, which are not inhibited by the NSAIDs. These enzymes convert arachidonic acid into leukotrienes, and even subtle increases in these autacoids may lead to adverse reactions in atopic patients. It is wise to substitute acetaminophen for patients who claim allergy to any of the NSAIDs.

Penicillin have been confirmed as producing both IgE- and non–IgE-mediated reactions. Patients rarely have serologic confirmation that their previous reaction was IgE mediated, leaving the clinician little recourse but to avoid all penicillins when allergy is suspected. Erythromycin and clindamycin are the most conventional alternatives, but if necessary, cephalosporins can be prescribed, provided the reaction to penicillin was only pruritic or maculopapular in nature. A history of urticaria (hives) or anaphylactoid symptoms is more convincing evidence that the patient's reaction to penicillin was truly IgE mediated, and in this case, one should refrain from prescribing any beta lactam derivative, including cephalosporin.

Recent emphasis on infection control has contributed to an increased number of reports regarding latex allergy. Latex is a milky-white sap obtained from rubber trees (Hevea brasiliensis) and is used in more than 40,000 medical products. IgE- mediated reactions to latex have been confirmed, and a surprising number of allergic incidents have been reported, including anaphylaxis and death. All standard medical history forms should be revised to include an inquiry regarding adverse reactions to rubber products.

PHYSICAL ASSESSMENT

Along with a complete medical history, baseline information regarding vital signs is an essential component of the medical record. It not only aids in assessing the

patient's medical status, but also provides essential reference data during intraoperative monitoring. Ideally, the information is gathered during an interview appointment, when the patient is less likely to be apprehensive concerning eminent treatment. Along with the patient's age, weight, and height, essential vital signs should include blood pressure, pulse rate, and hemoglobin saturation by pulse oximetry.

The airway should be examined and the Mallampati classification noted. Airway management is the most important aspect of patient care during sedation or general anesthesia, and examination of the patient's airway is an essential component of the preoperative assessment. Documentation of a Mallampati airway classification has become a standard of care when any level of sedation is provided . Patients who have Class III and IV airways are more difficult to intubate, more likely to obstruct, and more difficult to artificially ventilate, should this become necessary. Unless one has training and significant experience in advanced airway management, deeper levels of sedation should be avoided in these patients. Patients with additional factors that warn of difficulty in attaining a good mask seal for positive-pressure ventilation include those who are fully bearded or edentulous, or who have a short thyromental distance, and those with a large neck circumference.

Patients should be questioned regarding their physical stamina, including exertional or postural dyspnea and any history of light-headedness or syncopal events. The dentist should note any visual impressions of cardiovascular, respiratory, or neurologic compromise. These might include evidence of distended jugular veins, edema of extremities, or elevated nail beds. For elderly patients, a notation regarding any evidence of dementia will be useful in evaluating any concerns regarding residual influences of sedative or anesthetic agents during the subsequent day or two. A system for assessing functional capacity was introduced by Hlatky et aland was modified as a useful adjunct when patients with questionable ASA status are assessed.

ASSESSMENT OF PREEXISTING MEDICAL CONDITIONS

The proper assessment of preexisting medical conditions requires a basic understanding of the pathophysiologic features of the condition. This will enable the provider to comprehend the patient's status and to properly design a plan for appropriate management while providing dental care. Cardiovascular diseases represent the most widespread preexisting medical conditions among patients presenting for dental treatment. By convention, the most common disorders will be addressed separately, but patients frequently suffer from combinations of these conditions.

Ischemic Heart Disease

` Ischemic heart disease is a condition whereby the myocardium is inadequately supplied with oxygenated blood. The pathogenesis reflects an imbalance between coronary artery supply and myocardial oxygen demand. Atherosclerosis is generally the primary coronary lesion and compromises perfusion through vessel stenosis. However, as this condition progresses, plaque rupture, thrombosis, and/or vasospasm may all be superimposed. If a vessel becomes totally occluded, the normally perfused myocardial cells undergo necrosis (ie, myocardial infarction). Myocardial oxygen demand is determined primarily by heart rate and systolic wall tension (afterload).

Chest pain (angina) is generally the first symptom of coronary artery disease. However, a patient may tolerate varying degrees of coronary stenosis without anginal pain provided heart rate and blood pressure do not impose excessive demand. In fact, some patients remain completely asymptomatic, and their disease is discovered during treadmill or other screening examinations. In this case, the condition is labeled silent ischemia. For example, severe diabetes can result in peripheral sensory neuropathy, and these patients may not experience anginal pain during periods of myocardial ischemia or infarction.

The nature and frequency of anginal episodes are indicative of the coronary lesions. When chest pain occurs only during exercise or emotional stress, the condition is called stable or classic angina. Because the pain is triggered by an increase in cardiac strain or myocardial oxygen demand, it is presumed that the coronary lesion is relatively stable and reflects uncomplicated atherosclerosis. It is believed that pain occurs because the sudden increase in demand outstrips the ability of the stenosed vessel to provide adequate oxygenated blood to the heart muscle. In rare cases, chest pain occurs only at rest. In this case, brief periods of coronary spasm are suspected and may be due to heightened excitability of coronary smooth muscle cells. Presumably, the underlying atherosclerosis contributes to this irritability, and the condition is described as Prinzmetal's or vasospastic angina. When chest pain becomes more frequent and occurs during exercise or rest, it is assumed that atherosclerotic lesions are becoming unstable and are exhibiting plaque rupture and thrombotic events. This is called unstable or preinfarction angina.

Myocardial infarction is the death of cardiac muscle cells attributed to complete occlusion of a primary coronary artery or one of its branches. This occlusion is the result of thrombosis following rupture of an atherosclerotic plaque. Although atherosclerosis produces stenosis of coronary arteries, the actual degree of stenosis does not alwayspredict imminent myocardial infarction. Indeed, some patients who have 80 to 90% stenosis merely present episodes of stable angina and never experience myocardial infarction. This occurs because total occlusion requires thrombosis. The vulnerability of atherosclerotic plaques to rupture is not predicated on the degree of stenosis, but on the portion that comprises cholesterol-rich lipids. At times, this fragile appearance is noticeable on angiographic

studies, but otherwise it is presumed on the basis of an unstable pattern of anginal episodes. These episodes are believed to follow embolization of plaque fragments or small thrombi that lodge in small, distal branches and dissolve. They do not occlude coronary perfusion long enough to cause necrosis and, in this regard, resemble the process observed in transient ischemic attacks (TIAs) of the cerebral circulation. Nevertheless, the risk for myocardial infarction certainly increases as atherosclerosis evolves to produce greater degrees of stenosis. The sequelae that follow infarction depend on the size and location of the necrotic zone.

Occlusion of a terminal branch to a small area in the right ventricular wall may result in chest pain only and may heal uneventfully. Necrosis of an isolated papillary muscle may lead to prolapse of the related valve. However, if a large vessel supplying the left ventricle is occluded, necrosis may be so extensive that the ventricle fails to eject adequately or ruptures. In either of these cases, the patient may experience a cardiac arrest, which is the absence of a palpable pulse. Most often, however, cardiac arrest is attributed to an arrhythmia such as ventricular fibrillation that is precipitated by the anoxic insult to neural conduction pathways within the heart.

The nature and frequency of anginal episodes, prescribed medications, and the effectiveness of these medications are key items of information when one is assessing severity and stability of the patient's ischemic disease. Medications generally include antiplatelet agents, beta blockers, and vasodilators. A system for classification of angina, which was introduced by the Canadian Cardiovascular Society, is useful for patient assessment. Class 1 or 2 angina reflects a stable pattern and present little cardiovascular risk during dental procedures, provided standard stress reduction protocols are followed. Class 3 or 4 angina reflects severe or unstable patterns, respectively, and presents significant risk for an acute episode of angina or plaque rupture leading to myocardial infarction. For these patients, consultation with the patient's physician should precede dental treatment. It has been accepted as standard care to postpone elective dental procedures for any patient who has suffered myocardial infarction during the previous 6 months. It has also been reasonable to expand this convention to include those who have undergone any invasive cardiac procedure (eg, angioplasty, stent insertion, coronary artery bypass grafting [CABG]), even if infarction did not occur. However, a recent joint report of the American College of Cardiology and the American Heart Association suggests that after 30 days, in the absence of unstable angina, noncardiac surgery may proceed without further evaluation or testing in the following cases: (1) recent myocardial infarction, (2) balloon angioplasty, and (3) bare-metal stent placement. Continued risk for rethrombosis following drug-eluting stent placement is believed to persist for at least 1 year, and consultation with the cardiologist is wise. Consultation is also encouraged when there is any evidence of decompensated congestive heart failure or significant cardiac dysrhythmias such as advanced heart block or symptomatic tachycardic or bradycardic

dysrhythmias. If a patient can manage to do 4 METS of physical activity, routine dental care and even more invasive procedures are likely to be tolerated.

Obviously, the dentist cannot influence the severity of coronary lesions, but it is generally agreed that stress reduction and proper use of sedation can have very beneficial effects by reducing myocardial oxygen demand. Conversely, inadequate anesthesia or the indiscriminate use of vasopressors in local anesthetics will increase heart rate, blood pressure, and subsequent myocardial oxygen demand.

Heart Failure

Heart failure is the inability of the heart to deliver a supply of oxygenated blood sufficient to meet the metabolic needs of peripheral tissues. Frequently, it is due to a defect in myocardial contractility that can be attributed to a variety of causes, including ischemia from coronary artery disease, primary cardiomyopathies, excessive programmed cell death (apoptosis), and excessive hemodynamic burden imposed by chronic hypertension. In some cases of heart failure, contractility is not impaired and ejection fractions are normal. The defect is related to diminished compliance of the ventricular wall, which prevents it from stretching to accept an adequate filling volume during diastole. This form of heart failure is described as "diastolic failure" to distinguish it from "systolic failure," attributed to a diminished contractile strength. (More recently, these terms have been revised to heart failure with or without normal ejection fractions.)

Regardless of the fundamental defect, diminished contractility, and/or compliance, heart failure results in 2 groups of consequences that can be categorized as forward and backward failure. Forward failure is due to diminished cardiac output leading to inadequate perfusion of vital organs. Early clinical manifestations of forward failure include weakness, fatigue, reduced exercise tolerance, and other symptoms of hypoperfusion. Eventually, kidneys begin to fail, vision becomes impaired, and other consequences of end-organ damage begin to appear.

Backward failure relates to consequences of venous backup and congestion due to the inability of the heart to accommodate adequate filling volumes. Clinicians find it useful to describe backward consequences of heart failure as left-sided failure, right-sided failure, or biventricular failure.

Elevation of the left atrial pressure results in symptoms and signs of pulmonary congestion, including dyspnea and orthopnea. Patients with orthopnea must elevate their heads on several pillows at night and frequently awaken short of breath or coughing (the so-called nocturnal cough) if their heads slip off the pillows. The patient's sense of breathlessness is often relieved if he or she sits upright because this

position reduces venous return. In advanced cases, orthopnea may become so severe that patients cannot lie down at all and must spend the entire night in a sitting position. Consequences of right-sided failure include symptoms and signs of systemic venous congestion. Pitting edema of the ankles, congestive hepatomegaly, and ascites are prominent. Late in the course of their disease, patients generally present signs and symptoms of biventricular failure, and attempts at characterizing them as right or left sided become artificial.

Drugs used to manage these patients include digoxin, which provides a positive inotropic influence; diuretics, which counter edema; and vasodilators which reduce preload and afterload on the compromised myocardium. Currently, angiotensin-converting enzyme (ACE) inhibitors (eg, captopril [Capoten], lisinopril [Zestril], enalapril [Vasotec]) are regarded as vasodilators of choice. The routine use of digoxin and diuretics has lost favor. In recent years, beta blockers have been found useful for managing heart failure. This is a dramatic shift in paradigm, because blocking beta receptors would appear detrimental in that they decrease myocardial contractility. However, the pathogenesis of heart failure has been found to include excessive sympathetic stimulation of the heart leading to hypertrophy, weakening, and loss of compliance. Although initiating beta blockade in these patients is initially met with a mild decline in ejection fraction, over time cardiac function actually improves. In addition, some of the newer beta blockers have mild alpha-blocking action, which produces vasodilation and subsequent reduction of afterload on the failing heart.

The patient with heart failure should be assessed for signs of peripheral edema and symptoms of pulmonary congestion, especially when reclined to a supine position. Careful attention should be given to monitoring blood pressure because sudden elevations can lead to venous backup and pulmonary congestion.

Hypertension

Hypertension is the most common cardiovascular disorder in developed countries. A myriad of conditions confound an understanding of this disorder, and attempts at classification based on etiology are artificial (eg, primary [essential] vs secondary hypertension). Although exceptions have been reported, most untreated adults with hypertension will develop additional increases in arterial pressure over time. Furthermore, it has been demonstrated that untreated hypertension is associated with shortening of life by 10 to 20 years. Morbidity and mortality associated with hypertension are attributed to its deleterious influences on vital organ systems. These influences may be secondary to acceleration in atherosclerosis and subsequent ischemia, or to direct damage caused by sustained pressure on organs. Coronary artery disease, heart failure, renal failure, retinopathy, and stroke are the typical end- organ consequences of chronic hypertension. Of these,

cardiovascular consequences are the leading cause of death.

Medical management of hypertensive patients includes the use of diuretics, beta blockers, and vasodilators. The most recent guidelines for diagnosis of hypertension are summarized in Table 4.29,30 Formerly, the severity of hypertension was based on diastolic pressure, but current guidelines use whichever reading is highest—systolic or diastolic. The use of "stages" has replaced dated adjectives such as mild, moderate, and severe. There are no absolute guidelines regarding acceptable limits for elective dental treatment. However, Fleisher31 assessed the association of preoperative stages of hypertension with adverse outcomes during elective noncardiac surgery and found no increase in risk with preoperative blood pressure readings up to 180 systolic blood pressure (SBP) or 110 diastolic blood pressure (DBP). An increase in adverse events was associated with values of 181 to 210 SBP and/or 111 to 120 DBP if patients were also afflicted with coronary artery disease, heart failure, or significant diabetes. SBP >210 and DBP>120 were associated with increased risk, regardless of comorbidities. Based on experience and the results of this study, it would appear reasonable to avoid elective procedures in patients having SBP readings >180 or DBP readings >110.

This suggestion does not obviate the requirement for additional judgment, however. Whereas a general anesthetic may aggravate any degree of hypertension, especially during induction and emergence, sedation may actually prove beneficial. Keeping in mind that baseline readings in the dental office are most likely elevated due to stress, it would be reasonable to administer an anxiolytic dose of a sedative and reassess blood pressure before postponing treatment.

Valvular Heart Disease

Valvular heart disease can produce turbulence in blood flow through the heart that is audible on auscultation. This sound is described as a "murmur." At times, turbulence can be heard under normal conditions, and the murmur is described as "functional" or "benign," to distinguish it from those attributed to structural or "organic" damage. It is important to understand that a murmur is merely a sign, not a disease.

Three primary conditions affect the heart valves: regurgitation, stenosis, and prolapse. A regurgitant valve allows reflux into the proximal chamber when it closes; stenosis or narrowing of a valve opening impedes flow into the distal chamber. In either case, a murmur can be heard during auscultation. Valve prolapse occurs when leaflets bulge into the proximal chamber upon closing. It does not alter blood flow that results in a murmur, but it may produce a "clicking" sound on closure. Regurgitation may occur alone, or it may accompany valves that are stenotic or prolapsed (eg, mitral prolapse with regurgitation).

There are many causes of valve disease. Lesions may follow injury to supporting structures such as chordae tendineae or papillary muscle, or to the valve leaflets

themselves. Most often, valve disease is attributed to congenital defects, excessive strain from chronic hypertension, myocardial infarction, or inflammatory damage following rheumatic fever. Stenosis and regurgitation result in hemodynamic changes that lead to impaired circulation through the heart and strain on muscular chambers. The aortic and mitral valves are most commonly affected and will be examined more closely.

Mitral valve prolapse is a common condition in which the mitral valve leaflets are displaced into the left atrium during ventricular systole. It is more common in women than in men, and, in most cases, mitral valve prolapse represents a benign abnormality unless regurgitation develops.

During acute mitral regurgitation (such as can occur with endocarditis or chordal rupture following ischemia), left atrial and pulmonary venous pressures increase quickly, giving rise to dyspnea and pulmonary edema. In more chronic forms of mitral regurgitation, an increase in left atrial pressure is offset by a concomitant increase in atrial compliance, and symptoms appear late in the course of the disease. Left atrial enlargement predisposes the patient to atrial fibrillation and atrial thromboembolism. In long-standing mitral regurgitation, pulmonary hypertension can develop, which, in turn, leads to tricuspid regurgitation and right-sided heart failure.

Aortic stenosis causes concentric left ventricular hypertrophy as a compensatory mechanism that maintains cardiac output despite the increased pressure gradient across the valve. Eventually, this compensatory mechanism is overcome, causing the left ventricle to dilate and cardiac output to decline. Aortic regurgitation causes a volume overload of the left ventricle. In chronic aortic regurgitation, the volume overload is well tolerated for years. The left ventricle dilates to accommodate the increased volume load and thereby maintains a normal resting cardiac output. Compared with that observed with mitral regurgitation, enlargement of the left ventricle is far more severe.

Eventually, compensatory mechanisms fail, and dyspnea follows elevated left atrial and pulmonary venous pressures.

Unless it is injured, endothelium is resistant to thrombus formation and to infection by most bacteria. However, endothelium of the heart can be injured by high-velocity jets produced by stenotic or regurgitant valves. These areas, as well as fibrotic valve leaflets, allow direct infection by virulent organisms or the development of an uninfected platelet-fibrin thrombus called nonbacterial thrombotic endocarditis (NBTE). This vegetation provides a site for bacterial attachment during transient bacteremia and may result in infective endocarditis.

Patients at greatest risk for infective endocarditis are those who have previously experienced endocarditis, which leaves vegetations after healing, and those who have had valve replacements, which may allow vegetations at suture sites. For these patients, antibiotics are administered in conjunction with selected procedures considered to entail a risk for bacteremia and subsequent endocarditis. The benefits

of antibiotic prophylaxis have not been proven and in fact may be modest if one considers that most cases of endocarditis do not occur after a procedure. Dental treatments, the procedures most widely accepted as predisposing to endocarditis, are no more frequent during the 3 months preceding this diagnosis than in uninfected matched controls.32 Nevertheless, an expert committee of the American Heart Association has identified procedures that may precipitate bacteremia and suggests antibiotic prophylaxis for those patients at greatest risk for developing endocarditis.

Valvular heart disease does not have a great impact on sedation and anesthesia, except in the most severe cases. Stenosis compromises filling of the distal chamber, and this may be further diminished with shortened filling time. For this reason, tachycardias should be avoided if patients have significant aortic stenosis, because blood pressure could drop precipitously. Conversely, regurgitant flow is accentuated by slow heart rates and high pressure within the distal chamber. These fundamental pathophysiologic principles spawn 2 pragmatic caveats: (1) Rapid heart rates should be avoided if patients have aortic or mitral stenosis, and (2) slow rates and hypertension should be avoided if patients have prolapsed or regurgitant valves. A cardiology consult should be obtained when there is any evidence of symptomatic aortic or mitral stenosis. Cardiac ArrhythmiasThe minute output of the heart (cardiac output) is the paramount cardiovascular event required to sustain blood flow throughout the body. The heart must sustain a regular cycle of relaxation and contraction if it is to fulfill this objective. Any abnormality in rate or rhythm is labeled an arrhythmia. A complete analysis of the many supraventricular and ventricular arrhythmias is not within the scope of this article and is not always essential for adequate preoperative assessment. The primary consideration is whether the rhythm is stable enough to allow a cardiac output sufficient to sustain arterial pressure. A history of syncope or dizziness and light-headedness are signs that the rhythm is unstable and a medical consultation is indicated. For procedures using moderate to deep sedation or general anesthesia, monitoring should include continuous ECG tracing and blood pressure assessment every 5 to 10 minutes. Hypotensive episodes may follow both bradycardias and tachycardias. Slow rates may not produce enough cycles to sustain minute output, and tachycardias may reduce time for ventricular filling, which reduces stroke volumes.

For patients with concurrent coronary disease, tachyarrhythmias may precipitate episodes of angina even when blood pressure remains stable. This is due not only to an increase in myocardial oxygen demand, but also to a reduction in coronary perfusion. Coronary perfusion is greatest during diastole, when the myocardium is relaxed and is not compressing the intramyocardial vessels. Tachycardias shorten diastolic periods and compromise time required for adequate flow through the coronary vessels.

Although most arrhythmias are managed with medication, pacemakers are required in refractory cases. Cardiac pacemakers are implanted in more than a million people

in the United States each year. They are used most often in patients suffering symptomatic supraventricular arrhythmias and heart blocks unresponsive to drug therapy.

Pacemakers have 2 essential components: (1) a pulse generator powered by a lithium-iodine battery, and (2) pacing leads. These leads are stainless steel wires that most often are introduced via the subclavian or external jugular veins and guided to the cardiac chamber(s), where they are attached to an electrode anchored in the right atrium and/or ventricle. Asynchronous pacemakers generate impulses at a predetermined fixed rate. Synchronous or "on-demand" pacemakers are most common and are activated when the patient's natural rate falls above or below a preset level.

Pacemakers are classified using a 3-letter code that indicates the chamber paced, the chamber sensed, and the response delivered. The most versatile and widely used pacemaker uses a DDD code. Examination of this table will reveal that this particular device paces and senses both atria and ventricles and may trigger or inhibit activity, depending on what is sensed.

Preanesthetic evaluation of patients with pacemakers should include the indication for pacing and a comment regarding its quality of performance. The type, date, and location of the pacemaker implanted should also be recorded. Any history of vertigo or syncope indicates dubious pacemaker function, and the patient should be referred to his or her physician. Monitoring during sedation or anesthesia should reflect the function and integrity of the pacemaker. Ideally, this is provided by continuous electrocardiography and continuous monitoring of pulse by plethysmography using a pulse oximeter. It is reasonable to consult with the cardiologist regarding any need to temporarily alter the pacing mode.

Modern pacemakers have been improved to resist electromagnetic interference, but the dental office environment may still present some hazard. Ultrasonic scalers, ultrasonic bath cleaners, and electrosurgery units are documented sources of electromagnetic interference.36 Influence from electrocautery units can be minimized by placing the grounding plate as far away from the implanted generator as possible and making sure that the generator is never located between the grounding plate and the electrode tip of the cautery unit. Also, keep the current as low as possible, and limit the pulse duration (1 s) and frequency (1 pulse/10 s).

Pharmacologic considerations and indications for sedation and anesthesia should not be changed solely because of the presence of a pacemaker. Agents to counter bradyarrhythmias (eg, atropine, epinephrine) should be readily available for use, should pacemaker failure occur. In the unlikely event that defibrillation or cardioversion should become necessary, paddles should not be placed near the implanted generator.

Considerations regarding pacemakers are identical for patients who have

implantable cardioverter-defibrillators. Consultation with the cardiologist is encouraged, and a decision can be made to program off of any tachyarrhythmia treatment algorithms during treatment to prevent unwanted shocks due to spurious signals.

There are several simple tests that may assist in diagnosis of dental pain.

Pulp sensitivity test.

Dry ice on a cotton bud, or an ordinary ice stick (made in a plastic or glass tube), is placed on the cervical third (neck region) of the tooth crown. A response (pain is the only sensory response from the dental pulp) to the stimulus indicates that the pulpal tissue is capable of transmitting nerve impulses and is vital. No response may indicate pulp necrosis.

Percussion test.

Using an instrument handle, the tooth is tapped in the longitudinal axis. A painful response suggests possible periapical inflammation due to inflammatory sensitivity of the mechanosensory receptors in the periodontal membrane surrounding the tooth.

Probing.

Placing a fine, blunt probe gently into the gingival sulcus surrounding the tooth enables the health of the gingival tissues to be assessed. Bleeding and/or sulcus depths greater than 3–4 mm indicate gum disease due to inflammation.

Mobility test.

Holding a tooth firmly on the buccal (cheek) and lingual sides between the fingers enables mobility to be assessed. All teeth have a small amount of mobility (<0.5 mm), but visible movement suggests loss of bone support around the root of the tooth.

Palpation.

Careful palpation around the area of concern may reveal tenderness and the type and extent of swelling. Sinus formation. Chronic dental abscesses tend to drain buccally through the mucosa causing mucosal sinuses.

Rarely lower mandibular teeth with chronic abscesses may drain buccally (below the buccinator muscle attachment) or inferiorly below the mylohyoid muscle resulting in dermal sinuses that are often mistaken for skin lesions remaining resistant to routine dermatological remedies.

Radiographic examination.

If it is possible to obtain a screening radiograph, such as an orthopantomograph , this may assist in the diagnosis and localisation of the cause of the pain. The radiograph should show clearly the apical and periapical structures of teeth and associated tissues. The relationship of the maxillary molars and premolars to the floor of the maxillary sinus can be examined, and radiographs may reveal

recurrent caries or periapical radiolucencies associated with an established infection

PAEDIATRIC DENTAL AND ORAL PROBLEMS

Teething

The eruption of the primary teeth (around 6 months old) is usually accompanied by inflamed and sore gingiva. There may be irritability, disturbed sleep and drooling. Teething does not cause high fever or convulsion.

Treatment

Analgesic/anti-inflammatory such as elixir paracetamol "Teething ring" or something hard to chew on, like hard biscuits

Trauma to Soft Tissue and Primary (milk) Teeth . Small superficial oral lacerations heal spontaneously and no antibiotic is indicated. Dirty lacerations need surgical debridement and antibiotic if infected.

Antibiotics used are:

- Phenoxy methyl penicillin 12.5mg/kg qid for 5-7 days OR
- Amoxicillin 25mg/kg tid for 5-7 days OR
- Benzyl penicillin, 15-30mg/kg IV every six hours If hypersensitive to penicillin, use:

- Erythromycin 10-20mg/kg orally bd OR
- Cephalexin 6.25mg/kg orally every 6 hours OR
- Clindamycin (child: 5mg/kg up to) 300mg orally, q8H for 5 days

Alveolar bone in a child is elastic and rarely fractures.

Injuries to the primary teeth are usually loosening with/without displacement. Fractures to crown or root can happen.

Treatment includes elixir paracetamol for pain/fever, oral penicillin or amoxicillin if infected and referral for dental assessment.

Trauma to Secondary / Permanent Teeth

Permanent teeth start to erupt into the oral cavity at 5-6 years and continue up to the age of 21.

After the initial eruption, root formation/development continues for a period of 18-30 months. Injuries during this phase have the potential to interrupt root development.

Injuries involved are mostly fractures of the root or crown and

displacement (luxation, intrusion, extrusion or avulsion).

Treatment includes oral paracetamol for pain and immediate dental referral. Successful outcome depends on timely re-establishment of a normal periodontium (supporting structures around tooth).

Toothache

Toothache in a child is usually caused by either caries impacted with food, abscess, root infection or an erupting tooth.
Treatment includes paracetamol for pain/fever, phenoxy methyl penicillin or amoxicillin for infection and referral for further dental treatment.

INFECTIONS

Bacterial Infections

Causative organisms are usually a mixture of aerobic and anaerobic oral flora. All cases should ideally be referred to a dentist or dental therapist for appropriate treatment.

Gingivitis

Presents as red swollen gums, that easily bleed on brushing teeth Antibiotic is normally not indicated in most cases
Local dental care such as regular tooth brushing to control bacterial plaque is usually sufficient

Acute Necrotising Ulcerative Gingivitis (ANUG)

This is a painful yellowish-white ulcer of the interdental papillae and gingival margins which bleeds easily. Causative bacteria are a mixture of the anaerobes: Borrelia vincentii, Fusobacterium fusiform, Bacteroidis and Treponema species. The appearance of ANUG in an otherwise healthy individual may be the presenting sign of HIV infection.

Treatment

Advise

Adequate oral hygiene 0.2% chlorhexidine gluconate mouthwashes (if available), adjunct to tooth brushing
Metronidazole (10mg/kg up to) 400mg tds for 5 days Refer for dental debridement

Periodontal Abscess

Localised collection of pus in a periodontal pocket of a tooth There is pain on lateral movement of the tooth and it may be quite mobile Treatment

Oral Amoxicillin 500mg tds and metronidazole 400mg orally tds for 5 days Refer for dental treatment

Chronic Periodontitis

This is usually caused by gram negative anaerobes which are also prominent in active disease. Teeth involved are usually mobile and painful.

Treatment

0.2 % chlorhexidine gluconate mouthwash (if available) bd Doxycycline 100mg orally bd for 5 days

OR

Phenoxy methyl penicillin (child 12.5mg/kg) up to 500mg orally 6- hourly for 5 days,

PLUS

Metronidazole 400mg orally tds for 5 days (in moderately severe cases)

Use erythromycin (child: 10 mg/kg up to) 500 mg orally, q6H for 5 days in place of penicillin in penicillin allergy

Pocket dental treatment for localised pus formation

Pericoronitis

This is an inflammation / infection of a gum flap (operculum) overlying a partially erupted tooth, usually a lower wisdom tooth (or lower three molars) - often traumatised by an overerupted upper wisdom tooth (or upper three molars)

Treatment

Removal of the opposing upper third molar Adequate oral hygiene

Chlorhexidine gluconate 0.2% mouth wash Irrigation with 3% hydrogen peroxide

Phenoxy methyl penicillin 12.5mg/kg four times daily for 5-7 days OR Amoxicillin 25mg/kg three times daily for 5-7days If penicillin hypersensitive- Erythromycin 10-20mg/kg orally twice daily OR

Cephalexin 6.25 mg/kg orally 6-hourly

Facial Swelling and Infection

Facial swelling can either be due to odontogenic causes (e.g. caries, retained roots, periodontitis) or non-odontogenic causes (e.g. soft tissue infection, fractures, osteomyelitis, sialoadenitis, foreign body).

Infections can spread to the soft tissue around jaws, neck and cause cellulitis and suppuration.

This can easily be life-threatening.

In the absence of systemic signs and symptoms, odontogenic causes can be usually treated by local dental care, such as removal of the infected pulp tissue.

If accompanying systemic signs and symptoms are present, the following treatment should be given:

Oral amoxicillin and metronidazole for 5 days

Patients hypersensitive to penicillin should be given either erythromycin or cephalexin.

If progressive trismus arises and airway is compromised, admit case and give: Penicillin G

1.2-2.4g IV qid OR

Ampicillin 1-2g IV qid PLUS

Metronidazole 1-2g IV tds PLUS

Gentamicin 3-5mg/kg/day IV (ideally not exceeding 48 hours; in patients with poor renal function serious damage to the vestibular apparatus may result from gentamicin. Get an estimate of renal function before prescribing)

Pus must be drained surgically by the dentist

Be careful of poorly controlled diabetic and hypertensive patients, who may need antibiotic cover

Septicaemia

Septicaemia due to skin infection or cellulitis is usually caused by Staphylococcus aureus or Streptococcus pyogenes.

Treatment is with IV cloxacillin 1-2 g, 4 to 6-hourly.

Patients hypersensitive to penicillin, give Clindamycin (child:10 mg/kg up to) 450- 900 mg IM/IV, every 6-8 hours

In children, facial or periorbital cellulitis may be caused by Haemophilus influenzae or Streptococcus pneumonia in addition to the above pathogens, add one of the following to the above:

Ceftriaxone 100mg/kg (max. 2g/day) IV once daily Children hypersensitive to penicillins or cephalosporins, give

Chloramphenicol 100mg/kg/day (max 3g/day) IV in 3 or 4 divided doses Viral

Infections

Primary Herpetic Stomatitis

Causative agent is Herpes Simplex Virus 1 (HSV 1). It presents with multiple oral ulcers accompanied by fever, malaise, anorexia and irritability. In children, they may have drooling of saliva.

Treatment

For symptomatic relief; soft diet and adequate fluid intake, since this is a self limiting illness

Antipyretic such as paracetamol

Local antiseptic mouthwashes such as chlorhexidine 0.2% solution Aciclovir, 10mg/kg qid orally for 7-10 days

Herpes simplex labialis (cold sore)

Causative agent is HSV 1. The virus is latent in the trigeminal ganglia and is reactivated as herpes labialis. It is precipitated by sunlight, trauma, systemic disease or stress. Papules are followed by blisters then pustules.

Treatment

Aciclovir cream (5%) applied qid early, before blisters appear.

Herpes zoster (shingles)

Causative agent is Varicella Zoster Virus (VZV), the same one that causes chicken pox. It presents as an acute painful, vesicular rash along the dermatomal distribution of the sensory nerves; commonly of the trigeminal or the intercostal nerves.

Treatment

Aciclovir 800mg oral 5 times daily OR valaciclovir 1g oral, q8H for 7 days; beneficial only if started within 72 hours from the onset of the vesicles.

Ophthalmic herpes zoster should be referred to the Eye clinic. FUNGAL

ORAL CANDIDIASIS

A white creamy plaque which leaves a red base when wiped off. Causative agent is usually Candida albicans, when triggered off by the use of antibiotics, steroids, unhygienic dentures, smoking and in immunocompromised hosts. It can be seen in neonates too.

Treatment

Eliminate predisposing factors

Nystatin 100,000 U/mL suspension 1mL oral, q6H for 7-14 days. For severe cases in immunocompromised hosts, give itraconazole 100-200 mg oral, daily for 14 days OR nystatin suspension 2mL orally qid.

ODONTOGENIC PAIN

Odontogenic pain refers to pain initiating from the teeth or their supporting structures, the mucosa, gingivae, maxilla, mandible or periodontal membrane.

> 'A toothache, or a violent passion, is not necessarily diminished by our knowledge of its causes, its character, its importance or insignificance.' TS Eliot

Toothache is caused by inflammation of the dental pulp, most commonly as a result of dental caries (tooth decay), the most common human infective disease worldwide, affecting 60–90% of school children worldwide. Periodontal disease (gum disease) is the second most common infection, and similar to chronic mycobacteria infections, for example Leprosy, is painless. The two bacteria appear to be particularly likely to cause aggressive periodontal disease. Both P gingivalis and A. actinomycetemcomitans, along with multiple deep pockets in the gum, are associated with resistance to standard treatments for gum disease. Other risk factors include smoking and there is very likely a genetic predisposition to developing this silent painless disease, which is the leading cause of tooth loss, and is found in 5–400% of middle-aged adults. The diagnosis and management of this condition remain outwith this article's remit.

The role of all medical personnel in improving oral health in children is being recognised. Caries is preventable using fluoride toothpaste and simple dietary advice such as reducing the frequency of sugar intake. Despite this, the numbers of children undergoing general anaesthetic for dental extractions due to caries continues to increase.

PROTOCOL FOR PAINFUL TOOTH/TEETH

Where possible, refer case to the dental department for identification and treatment of cause of pain. Variation in an individual's response to pain is affected by fatigue, anxiety and sometimes depression.
While one is waiting for definitive dental treatment, the following analgesics could be given:

Mild Pain

Paracetamol 500mg-1g orally 4 to 6-hourly OR

Aspirin® 300-600mg 4 to 6-hourly (avoid in children, breast feeding mothers, people with gastric diseases and those with bleeding tendencies)

OR

Other NSAIDS such as ibuprofen 400mg-1.6g bd Moderate Pain

ADD codeine 15-60mg qid oral to the above medications Severe Pain Pethidine 25mg-100mg SC/IMI 2 to 3-hourly PRN OR

Morphine 2.5mg-10mg SC/IMI 2 to 3-hourly PRN

BONE PROBLEMS

Alveolar Osteitis (Dry Socket)

Severe dull pain post dental extraction, two-three days later. Tooth socket appears 'dry' with exposed bone and no blood clots, gingiva is inflamed.

Treatment

Analgesics

Dental referral for LA debridement and curettage to initiate socket healing Facial Fractures Mandibular fracture (broken jaw)

For simple, undisplaced fractures, advise soft diet, PRN analgesia is sufficient. (No surgical intervention required).

For compound displaced fractures: Amoxicillin 500mg tds orally OR Penicillin G 1.2g IV qid PLUS

Metronidazole 500mg IV bd if infected;

Check tetanus toxoid status and give it if not covered Refer for dental surgery

In children, closed condyle or TMJ fractures, encourage early jaw movement (to prevent ankylosis), soft diet and paracetamol as analgesics. (Do not use aspirin®).

Midface fractures

Le Fort types I, II and III or isolated midface fractures, refer for dental surgery.

If compound fracture, initiate benzyl penicillin 1.2g qid IV and metronidazole 1g qid IV for 5-7 days while awaiting transfer for surgery.

Cerebrospinal fluid leaks

Fractures of the facial middle third and skull, which injure the dura, can cause CSF leaks and present as otorrhoea or rhinorrhoea. They predispose to meningitis and must be covered with antibiotics until the leak stops.

Recommended treatment: Rifampicin 600mg oral daily OR Chloramphenicol 500mg oral qid OR Ceftriaxone IMI 250mg daily.

Continue for two days after CSF leak stops Osteomyelitis

NEUROLOGICAL PROBLEMS

Trigeminal Neuralgia

Characterised by an unilateral, sharp, stabbing and intermittent pain in division of the trigeminal nerve but no sensory loss. Diagnosed by relief following nerve block using bupivacaine 0.5%.

Treatment

Carbamazepine 100-200mg oral once or bd

Dose can be increased to 400-600mg and even up to 1.2g/day.

Bell's Palsy

Acute unilateral, lower motor neurone type of facial palsy of unknown aetiology (maybe viral). Most recover spontaneously.

It is advisable to protect the eye with pad or artificial tears to prevent corneal damage when eyelids cannot close properly.

Steroids are of unproven value but still, certain authorities advise giving oral prednisolone 5-10mg bd for 5 days, early during the disease to aid recovery.

ULCERS AND OTHER ORAL CONDITIONS

Oral Ulcers

Oral ulceration is probably the most common oral mucosal disease seen. It can potentially be the most serious too. There are many causes and one must make careful history and examination to help diagnosis.

Antibiotic use is rarely indicated. A corticosteroid cream application may help but if the ulcer does not heal in 2-3 week's time, refer immediately for dental assessment. Human and

Animal Bites Oral

Burns

A common chemical burn seen in adults is caused by putting aspirin® in the buccal sulcus to relieve headache. Treatment is to treat cause of headache and don't put aspirin® in the buccal sulcus!

Burnt mucosa heals itself quite quickly.

Chemical burns in children are usually due to ingestion of caustic liquids. Regular saline mouthwash should be done.

In severe burns, admit case for IV fluids and antibiotics (usually penicillin). Periodic follow-up is needed to check for scarring and adhesions.

Sedatives for Dental Procedures

To be used in anxious patients such as those who are phobic to needles, or in children.

Use diazepam 5-30mg 0.5-1 hour before procedure. This can also be given in divided doses such as 5mg nocte, 5mg in the morning and 5mg at 0.5-1 hour before dental procedure.

In children, diazepam 2mg (or according to age), is given either orally, IMI or PR at 0.5-1 hour before the procedure

OR

Midazolam 0.1mg/kg orally, SC, IMI or IV

TOOTH AVULSION

One of the commonest sequelae of facial trauma is tooth avulsion, exfoliation or articulation.

Salient features

- History of fall, interpersonal violence, sports injury, assault or accident.
- Children are more common.
- Central incisors and developing teeth are more frequently avulsed.
- Patient presents extreme pain with a bleeding socket, clot in the socket and a raw wound with or without Lip & Labial mucosa injury.

Treatment

Immediately refer to a dentist. Best result is observed if tooth is reimplanted within 5-20 minutes. Fixation of implanted tooth with periodontal wiring, arch bar wiring or composite resin; fixation period 6 to 8 weeks; root canal treatment done after reimplantation only (to avoid desiccation of periodontal ligament).

Interim storage

Best method is to place back the tooth in the socket immediately. Other storage media are saliva, milk (placed in ice since this minimizes the adverse effects on the periodontal ligament) and saline.

Pharmacological treatment

Cap. amoxycillin 250-500 mg 3 times a day for 5 days Or Tab. ciprofloxacin 250- 500 mg twice a day for 5 days.

Tab. diclofenac Potassium 50 mg b.d. or Tab. ibuprofen 400 mg 3 times for 3-5 days.

DENTAL CARIES

This is a multifactorial infectious disease of hard tissues of teeth characterized by demineralization of inorganic and destruction of organic part of the tooth. (Enamel & Dentin)

Salient features

- *Usually asymptomatic in early stages.*
- *Patient presents with tooth sensitivity and tooth ache only on cold & sweet intakes which may disappear on removal of stimuli.*

Treatment

- Examine for stage of caries and treat accordingly.
- Careful assessment of oral cavity for presence of any white/ brown or black spot.

Non-pharmacological treatment

- In non-cavitated lesion and low risk patient with good oral hygiene practices, no treatment is given.
- In cavitated lesion, restoration is done.

Pharmacological treatment

- Patients with caries is likely to progress (in high risk patient) pit and fissure sealant.
- Mouth wash with 0.2% chlorhexidine twice a day.
- Advise fluorinated toothpaste or fluorinated mouthwashes.

Assessment of response to therapy

- For caries active patient - follow up visit every 3 months and to check the progression of white / brown or black spot on the teeth.
- For normal patients - follow up every 6 months to 1 year to check the development of the white spot cavitation.

Patient education/prevention

- For caries active/high risk patient preferably.
- Diet control and avoidance of sugar containing food.
- Frequent ingestion of food containing sucrose should be substituted by sugar free foods.
- Oral hygiene: (a) brushing of teeth twice a day (b) flossing (c) thorough rinsing after every meal.
- Fluoride application using Topical 2% sodium fluoride (by dentist) 4 applications at weekly intervals at the age of 3, 7, 11 and 13 years.
- 0.05% sodium fluoride daily rinse (should not be swallowed).
- 0.2% sodium fluoride supervised weekly rinse in school (age of children >7 years) only if these children have been identified as caries active patients.

DENTAL ABSCESS

Patient presents with pain and swelling. The most common types of dental abscesses are periapical abscess and lateral periodontal abscess.

I. PERIAPICAL ABSCESS

Salient features

- *Presence of caries or trauma. Severe throbbing pain, disturbed sleep*
- *Tooth is tender to touch & extruded from socket. Tooth may be mobile and associated with localized or diffuse swelling.*

Immediate treatment

- To give antibiotics as given below and refer to a dentist.
-

Pharmacological treatment

- Cap. amoxycillin 250 -500 mg 3 times a day for 5 days. Or Tab. Ciprofloxacin 250-500 mg two times a day for 5 days.
- Tab. diclofenac potassium 50 mg b.d. or Tab. ibuprofen 400 mg 3 times a day for 3-5
- days.

Surgical treatment

- Drainage of pus by entering the pulp chamber (pulpectomy) & relieve occlusion.
- If fluctuant swelling of soft tissue is present drain by incision.
- Extraction or root canal treatment should be done when acute symptoms subside.
- Spread of infection should be closely observed to prevent complications like Ludwig's angina or osteomyelitis.

Patient education

- Maintenance of oral hygiene.
- Control of diabetes mellitus, if present.
- No hot fomentation over the skin.
-

II. LATERAL PERIDONTAL ABSCESS

Salient features

- Same as in acute periapical abscess, often associated with bad taste.
- Tooth is usually mobile and tender on percussion, associated with localized or diffuse swelling of the adjacent periodontium.
- Vitality test usually positive if no associated pulpal problem.
- Radiograph shows vertical or horizontal bone loss in relation to the tooth.
-

Pharmacological treatment

- Cap. amoxycillin 250-500 mg 3 times a day for 5 days.
- Tab. metronidazole 400 mg 3 times a day for 5 days.
- Tab. diclofenac potassium 50 mg b.d. or Tab. ibuprofen 400 mg 3 times/day for 3-5 days.

Surgical treatment

- Debridement of pocket and drainage of pus and irrigation with chlorhexidine.
- Spread of infection to be closely observed to prevent complications like Ludwig's angina.

Patient education

- Maintenance of oral hygiene.
- No hot fomentation over the skin.
- Control of diabetes mellitus if present.

ADULT TYPE PERIODONTITIS

Most common dental disease of the gums & periodontium. Salient features

- Swollen gums, bleeding from gums either spontaneously or while brushing.
- Pain on eating something hard, difficulty in chewing food, dull pain in the gums, pus discharge from gum on pressing, loosening of teeth, recession of gums.
- There is slowly progressive destruction of periodontitum, loss of periodontal attachment and presence of periodontal pocket.
- Bad breath (Halitosis), sensitivity to hot & Cold intakes

Non-pharmacological treatment

- Refer to a dentist for oral prophylaxis in form of thorough scaling and root planning.
- Advise brushing twice daily once after breakfast and once after dinner with super soft tooth brush for at least 3 minutes.

Pharmacological treatment Local therapy

- Advise antiplaque toothpast containing antibiotics.
- Gel metronidazole to be massaged on the gums twice daily.
- Rinsing with 0.2% chlorhexidine mouthwash twice daily which is effective after scaling.

Systemic therapy

- In adults, Cap. tetracycline 250 mg 4 times a day for 5-7 days.
- In very deep pockets: Combination of drugs i.e., Tab. ciprofloxacin 500 mg twice daily & Tab. tinidazole 300 mg twice daily for 5-7 days.
- Recheck the depth of periodontal pockets, if it persists, refer to dentist for further management.

JUVENILE PERIODONTITIS

It is characterized by rapid destruction of periodontal tissues. Salient features

- Common in the age group of 13-25 years.
- Mobility in incisors and molars, spacing in upper incisors, distolabial migration of upper incisors, arc shaped bone loss extending from distal surface of second premolar to mesial surface of second molar.

Pharmacological treatment

- Cap. tetracycline 250 mg 4 times a day for 14 days.

Surgical treatment

- Extraction of badly involved teeth. Refer the patient to periodontist for further periodontical management at the earlies
- Patient education
- Proper brushing twice daily with super soft tooth brush.

INFLAMMATORY GINGIVAL ENLARGEMENTS

The gingival enlargement can be acute which is very painful or they can be chronic which may be painless. There are many causes for gingival enlargements. Before giving treatment, etiological factor must be evaluated.

Salient features

- Acute enlargements may be localized or generalized, very painful, deep red in color, soft friable with shiny surface.
- Chronic type may be localized or generalized, often painless and slowly progressive.

Pharmacological treatment

- Tab. ciprofloxacin 500 mg 2 times a day for 3-5 days.
- Tab. diclofenac potassium 50 mg b.d. or Tab. ibuprofen 400 mg 3 times a day for 3-5 days.
- Rinsing with 0.2% chlorhexidine mouthwash twice daily.
- Refer to a periodontist for surgical management and drainage of pus.

Patient education

- Proper brushing twice daily with super soft tooth brush.

DENTAL FLUOROSIS

Salient features

- Mainly seen in patients who are drinking fluorinated water (> 3PPM) during tooth development time.

- White chalky/ brown spots on crowns of teeth
- Brownish discoloration on enamel mainly, involves anterior teeth & first molars

Treatment

- Oral hygiene maintainance as patients are more caries susceptible
- Bleaching of teeth (Whitening of teeth)
- Veneers or laminates for better esthetics

TRIGEMINAL NEURALGIA

It is sudden, sharp, severe or short duration, like electric shock like pain. Salient features

- Presence of trigger zones involving trigeminal nerve areas
- Unilateral sharp, shooting, lancinating type of pain may provoke on touch, washing face, shaving, on cold wind exposure.
- Pain will be in recurrent bouts of attacks, may persist for second to minute

Treatment

- Diagnostic dose of carbamazepine given to establish diagnosis
- Evaluation of any primary lesion in brain by MRI
- Tab. carbamazepine 200 mg. t.d.s as per intensity & frequency of pain & attacks.
- Refer to Dentists for further evaluation & treatment.

ORAL CANCER (HABIT RELATED DISEASE)

Oral cancer is most common and associated with smoking and tobacco containing habit. Adults are more commonly involved. Salient features

- Long standing (> 2 weeks) non-healing chronic ulcer in oral cavity.
- Bleeding & non painful ulcer or growth
- Regional lymphadenopathy, mobility or exfoliation of teeth.
- Decreased appetite, reduced weight, Poor Oral Hygiene, history of tobacco habit since long time. Altered speech, difficulty in opening of mouth (Trismus)

Treatment

- Refer to Oncologist after thorough clinical and histopathological examination.
- Advice symptomatic treatment if required.

ORAL SUBMUCUS FIBROSIS (OSMF)

Salient features

- Most common disease related with tobacco, lime and Betelnut
- Gradually reduced mouth opening (Trismus)
- Blanching (White opaque) oral mucosa
- Recurrent ulcers & burning sensations on taking spicy food.
- Change of voice, difficulty in deglutition.
- Poor oral hygiene with gingivitis & periodontitis
- Small shrunken uvula and decreased mobility of soft palate

Treatment

- Quit habit as soon as possible, mouth opening exercises (physical exercises)
- Oral Hygiene prophylaxis
- Local medicaments in form of steroids

CYST OR TUMOR OF JAW

Salient features

- Facial asymmetry, Non tender swelling or growth, Paresthesia of jaw
- Displacement of teeth, improper occlusion
- May be having discharge, Discoloration of teeth, Absence of teeth in arch

Treatment

Refer to oral maxilla facial surgeon for bone fractures

GENERAL MEASURES FOR GOOD ORAL HYGIENE

Select the right quality of tooth brush which should be short, soft and have uniformly trimmed bristles. Change the brush at least after every 3 months

Brush teeth at least twice a day for 2-3 minutes particularly at night before going to sleep. Use right technique of teeth brushing. Never use force while brushing. Use flossing at least once a day.

Avoid too much sugar and aerated drinks.

Avoid eating in between meals, if cannot be avoided rinse your mouth or preferably brush your teeth. Do not ingest or swallow fluorinated toothpaste while brushing.

Ensure regular dental checkup at 6 monthly interval.

ANTIBIOTIC PROPHYLAXIS IN DENTAL PROCEDURE

If patient is in the high or moderate risk groups, then antibiotic prophylaxis is recommended for the following dental procedures:

Dental extractions.
Periodontal procedures including surgery, scaling and root planing, probing, and recall maintenance.
Dental implant placement and reimplantation of avulsed teeth. Endodontic (root canal) instrumentation or surgery only beyond the apex. Sub gingival placement of antibiotic fibers or strips.
Prophylactic cleaning of teeth or implants where bleeding is anticipated. Antibiotic prophylaxis not recommended for the following dental procedures Restorative dentistry (operative and prosthodontic) with or without retraction cord. Local anesthetic injections (nonintraligamentary).
Intracanal endodontic treatment; postplacement and buildup.
Placement of rubber dams, postoperative suture removal, taking of oral impressions, and fluoride treatments.
Placement of removable prosthodontic or orthodontic appliances and orthodontic appliance adjustment.
Taking of oral radiographs. Shedding
of primary teeth.

Management of potentially premalignant oral epithelial lesions

oral premalignant lesion is defined as any lesion or condition of the oral mucosa that has the potential for malignant transformation. A new term **potentially premalignant oral epithelial lesions (PPOELs)** has recently been used as a broad term to define both histologic and clinical lesions that have malignant potential

This encompasses a number of oral lesions, such as

- leukoplakia
- erythroplakia
- erythroleukoplakia
- lichen planus
- oral submucous fibrosis (OSF)
- oral dysplasia

DETECTION AND DIAGNOSIS

To date, there have been no reliable and validated in vivo chairside adjuncts that have sufficient sensitivity and specificity to be more superior than clinical examination and tissue biopsy

Adjunctive aids include:

- photodynamic detection- including autofluorescence

- vital staining (toluidine blue, Lugol's iodine),
- and brush cytology.

TREATMENT PPOELs

can be managed conservatively by observation alone.

Surgical excision is the invasive management of choice for this group of lesions-include

- traditional excision,
- cryosurgery,
- and carbon dioxide (CO_2) laser ablation.

Nonsurgical treatment

falls into the category of chemoprevention or observation.

- Chemoprevention is the use of naturally or synthetically fabricated compounds designed to halt malignant transformation of PPOELs.
- they may cause regression or eradication of PPOELs and increase the threshold of malignant transformation.
- It uses the same concept of field cancerization but for treatment purposes

MANDIBULAR FRACTURES

Initial Assessment - History

The diagnostic work-up of mandible fractures begins with a thorough primary survey as outlined by the Advanced Trauma Life Support (ATLS) protocols.

Life-threatening injuries, when present, need to be recognized and managed early before fracture assessment can begin (eg. Bucket Handle Fractures which may require tracheostomy)

A thorough history of present illness and past medical and surgical history will highlight any relevant medical conditions, previous trauma, bone disease, nutritional and metabolic disorders, and psychiatric conditions that may influence timing and

management of the fracture.In addition, the patient's premorbid dental history and occlusion needs should be accounted for. When available, photographs can aid in reduction of the patient's fracture to re-establish the premorbid occlusion.

Initial Assessment - Clinical Examination

Focused evaluation of the head and neck is a part of the secondary survey outlined by ATLS protocols

Examination should begin with inspection and palpation. The classical signs of inflammation, pain, swelling, and erythema will help guide the physician in thorough identification of potential injuries. After examining for any lacerations or sources bleeding that needs to be addressed urgently, the clinician should perform an in- depth fracture assessment.

Extra- and intra-oral findings, in addition to a neurosensory examination, will help the physician in identification of fractures or fractures patterns that may be present.

Extra-oral Examination:

1. An extra-oral assessment should begin by examining the face and mandible for any abnormal contours or step defects.
2. Changes to the patient's facial profile and mandibular movements will cue the physician for types of fractures. For instance, a flattened facial profile may be due to a fractured mandibular body, angle, or ramus. A retruded chin may be caused by bilateral parasymphyseal fractures. An elongated face may be the result of bilateral subcondylar, angle, or body fractures.
3. Trismus or limited mouth opening, and deviation on opening may be due to guarding of the muscles of mastication, non-functioning of muscles, or bony impingements. Deviation upon opening may signify a mandibular condylar fracture due to unopposed contraction of the contralateral lateral pterygoid muscle. Inability to fully open may be due to impingement of the coronoid process on the zygomatic arch when fractures of the ramus and coronoid process or depression of the zygomatic arch is present. On the other hand, inability to fully close may signify dentoalveolar process, angle, ramus, or symphysis fractures. Inability to fully bring one's teeth together may be due to an open bite that was present pre-injury; the presence of mammelons on the

incisal edges of the anterior dentition may be a clue to determining a premorbid anterior open bite

Intra-oral Examination:

1. This includes assessment of the mandibular arch form and occlusion and identification of gingival lacerations, hematomas, or ecchymosis, and injuries to the teeth. The mandible is unique in that it is a continuous, U-shaped bone that crosses midline; deviations from this arch form may indicate a fracture. Any change in occlusion is highly suggestive of a mandible fracture. The mandible should be palpated bimanually to assess for fracture mobility.
2. The patient should be asked if their bite feels different. This can identify injuries to the teeth, dentoalveolar process, mandible, or temporomandibular joint (TMJ). Premature posterior contacts between the maxillary and mandibular dentition can result from bilateral mandible fractures of the angles or ramus-condyle units or signify the presence of a displaced maxillary fracture. Asking for premorbid photographs of the patient's premorbid occlusion can help to ensure accurate reduction of fractures based upon the occlusion.
3. Gingival lacerations, hematomas, or ecchymosis may indicate injury to the mandible For instance, sublingual ecchymosis is a pathognomonic sign for symphysial, parasymphyseal, or body fractures. In addition, retromolar trigone ecchymosis can signify angle fractures. Segments of fractured teeth may indicate fractures to the dentoalveolar process or mandible itself. Fractured teeth, mobile teeth, and any grossly carious teeth in the line of fracture may require extraction for reduction and to prevent aspiration. Missing teeth should that have not been accounted for should be considered swallowed, aspirated, or displaced into soft tissue. Radiography and operative exploration may help identify lost teeth and may require removal to prevent infection or airway issues.

Neurosensory Deficits should be accurately documented. Paresthesia, dysesthesia, or anesthesia of the lower lip indicates injury to the inferior alveolar nerve distal to the mandibular foramen. Most injuries are neuropraxic in nature and related to transient ischemia, inflammation, and traction.

Initial Assessment - Radiographic Examination

Most patients with mandible fractures, undergo radiographic assessment with computed tomographic (CT) imaging of the head and cervical spine (C-spine). Although CT imaging is now considered the gold standard, various other imaging studies can be helpful when CT is not available. These include plain films with Reverse Towne, Caldwell posteroanterior, lateral oblique, or occlusal views or panoramic radiograph (panorex). Panorex (OPG) is advantageous in allowing visualization of the entire mandible including the subcondylar unit/TMJ unit; however, its availability may be limited in the acute setting. In addition, certain fracture patterns, particularly in the posterior mandible, may be missed on single-view panoramic radiography. When evaluating a patient with mandibular injury with only plain film imaging it is prudent to obtain atleast two views

Surgical Anatomy

The osteology of the mandible, various muscle attachments and their influence, and presence of developing or permanent dentition, or lack of dentition, need to be understood for accurate treatment of mandible fractures. A full description of a mandibular fracture should include an assessment of its relationship to the external environment (ie, simple/closed, compound/open), type (ie, incomplete, greenstick, complete, comminuted), dentition (ie, primary, mixed, permanent, or lack of dentition), displacement, favorability, and location.

Bony Anatomy of the Mandible

The mandible is a U-shaped bone that crosses anatomical midline. The mandible has thirteen muscle attachments. Organized by function, these are jaw closers (temporalis, masseter, medial pterygoid), openers (digastric, lateral pterygoid), and glottic attachments (genioglossus and geniohyoid). The remaining muscles can influence displacement of fractures and may be involved in soft tissue closure (buccinator, platysma, mentalis, mylohyoid, depressor labii inferiorius, and depressor anguli oris).

The major vascular supply to the mandible during development is from the inferior alveolar artery, but transitions to the involved periosteum and muscle attachments as the body ages. During fixation of comminuted or atrophic mandible fractures, areas with poor blood supply, such as the body, careful soft tissue management is mandatory, as the blood supply to these regions is periosteal, rather than endosteal. Periosteal stripping in these areas should be minimized and done only to the extent necessary to apply fixation. Supra-periosteal placement of hardware has been studied in this context, but bears no discernable advantage for healing. The course of the facial artery and vein around the mandible in the antegonial notch should also be appreciated when treatment requires a transcervical approach.

The mandibular branch of the trigeminal nerve includes the auriculotemporal, buccal, inferior alveolar, and lingual nerves. Innervation of the mandible is supplied by the inferior alveolar nerve (IAN) and its branches, the mylohyoid, dental, incisive, and mental branches. The IAN enters the mandibular foramen on the medial aspect of the ramus behind the lingula, travels within a bony canal, and exits the mental foramen of the mandible near the apex of the first and second premolars. IAN injuries in the setting of mandible fractures have been reported to range from 5.7 to 58.5%.

Definition of Sites of Fracture

As defined by Dingman and Natvig, the mandible can be subdivided as follows symphysis, bounded by vertical lines distal to canine; parasymphysis, distal aspect of the roots of lateral incisors to distal of roots of canine teeth; body, from distal roots of canine to line corresponding to anterior border of the masseter muscle (usually coincides with third molar); angle, bounded by anterior and posterior borders of the masseter muscle; ramus, bounded by superior aspect of the angle to sigmoid notch; condylar process, area superior to ramus; coronoid process, and alveolar process, the area that normally contains teeth.

Favorable versus Unfavorable Fractures

Mandibular angle and body fractures can be classified as vertically favorable or unfavorable or horizontally favorable or unfavorable. Favorability is determined by the direction of a fracture line and its relationship to muscle action on the fracture segments. Vertically favorable fractures resist the medial pull of the medial pterygoid muscle on the proximal segment in the vertical plane. Horizontally favorable fractures resist upward the vertical pull of the masseter, temporalis, and medial pterygoid muscles on the proximal segment in the horizontal plane. The more

forward a fracture occurs in the body the more the upward displacement is counteracted by the downward pull of the mylohyoid muscles.

Fracture Fixation Principles

The mandible is the only moveable, load bearing bone of the skull. To properly treat mandible fractures, one must first understand basic fracture fixation principles. These can be grouped into tension versus compression and load-bearing versus load- sharing principles

Tension versus Compression

At any time, there are counteracting forces of tension and compression on the mandible influenced by muscular attachments and loading. At rest, these forces are equal. While an oversimplification, forces of tension generally separate a fracture and forces of compression bring a fracture together. Under compression, fractures generally undergo rapid healing and a greater resistance to separation. However, without addressing tension forces, overcompression can compromise ideal bony healing leading to nonunion.

Studies have shown that in the region of the mandibular body tension exists along the alveolar border while compression exists along the inferior border of the mandible. Moving toward the symphysis and parasymphysis, these two opposing forces become mixed or even inverted due to the introduction of torsional, or rotational, forces.

Biomechanically, it is most advantageous to apply bicortical rigid fixation along the zone of tension. Bicortical rigid fixation along the alveolar border is not feasible due to the presence of tooth roots, thin cortical bone, and thin gingival tissue. The inferior border of the mandible is not constrained by these limitations, with the notable exception of pediatric patients in the primary or mixed dentition. Bicortical screw fixation in this region is extremely stable and then only requires placement of a tension band at the alveolar level (either a continuous arch bar at the dentition or a small plate with monocortical screws) to resist tensile forces.

Load Bearing versus Load Sharing

Fracture fixation can be divided into either load bearing or load sharing. Choosing which type depends on the bone quality, location of the fracture, comminution, or bone loss. With load bearing osteosynthesis, the plate bears 100% of all of the forces of function at the fracture site. Load bearing osteosynthesis is indicated in comminuted mandible fractures, segmental defects, complex fracture patterns, or fractures with compromised bone such as atrophic mandibles or patients with metabolic or endocrine disorders. Fixation is accomplished with 2.3mm-2.7mm diameter locking reconstruction plates

Various methods exist for obtaining rigid fixation of mandibular fractures. In high energy injuries, such as this comminuted mandibular fracture secondary to a gunshot wound , interfragmentary fixation use can be used to align the different segments of the mandibular and rigid fixation was achieved with the addition of a locking plate along the inferior border. In edentulous patients, reconstruction plates with bicortical screws should be placed as laterally and inferiorly as possible, to avoid interference with denture fabrication.

When using load sharing osteosynthesis, stability at the fracture site is shared between the plate and well-buttressed bone. Depending on the location, the functional load is either shared equally between bone and plate (e.g., angle fractures), or in more ideal situations the bone assumes a greater share of the functional load than the plate (e.g., body fractures in dentate mandibles). Here, fixation can be accomplished with 2.0mm diameter miniplate systems. Examples of load-sharing fixation include a single miniplate along the oblique ridge for angle fractures (ie, Champy technique), or a single miniplate and an arch bar (providing tension) for body or symphysial fractures, and lag screw fixation.

Lag Screw Fixation

The use of lag screws was popularized by Niederdellmann et al. in 1976.Lag screws can be use in simple fractures where there is well-buttressed bone such as in symphysis or parasymphysis fractures. A lag screw has threads on only half the shaft so that the portion below the screw head is smooth and will not engage bone. Thus, the threads only engage the inner segment of bone and compress it against the outer segment. Typically, the two screws are placed, with minimal divergence between their long axes.

Rigid versus Non-rigid Fixation

Fixation can be grouped into rigid fixation, nonrigid fixation, or semirigid fixation. With rigid fixation, no bony callus if formed during healing and fracture segments are completely immobilized. In nonrigid fixation, micro-mobility of the fracture segments occurs and the fracture cap undergoes callus formation. Rigid fixation techniques include the use of plates and screws (miniplate and tension band with two screws on each side of the fracture), two lag screws, or reconstruction plates with three screws on each side of the fracture.

Fracture Management Techniques by Anatomic Site

Symphysis/Parasymphysis

Fractures of the anterior mandible can be addressed using either closed reduction, open reduction and internal fixation (ORIF), lag screws, or a combination of lag screws and miniplates. Closed reduction of fractures is most commonly achieved by applying Erich arch bars with circumdental stainless steel wires or hybrid arch bars. The patient is then placed into maxillomandibular fixation using stainless steel wires loops or heavy elastics. While still acceptable for simple fractures, ORIF is the standard of care for management of most displaced mandibular injuries.

ORIF can be achieved using a variety of techniques. Semi-rigid fixation using application of transoral mini-plates can be applied in simple, non-displaced fractures. Rigid fixation with the use of a reconstruction plate and bicortical screws may be required in severely comminuted or displaced fractures. In severely comminuted fractures, a transcervical approach may be required for adequate reduction of fracture segments; simultaneous bone grafting may be considered for fractures with segmental bone loss.

Body

As with anterior mandible fractures, body fractures can be treated with either a closed approach using MMF or ORIF. MMF can only be used in the presence of reliable, reproducible occlusion, using either the patient's own teeth or a mandibular prosthesis. It may be indicated in nondisplaced, favorable fractures, comminuted fractures, fractures in children with mixed dentition, or edentulous fractures (e.g., using dentures or gunning splints). This technique is generally contraindicated in

displaced, unfavorable fractures, multiple fractures, or instances of malunion. In addition, certain systemic conditions such as psychiatric disease, neurologic problems like seizures disorders, pulmonary disease, or gastrointestinal disorders where aspiration of emesis is a concern preclude prolonged intermaxillary fixation.

In most clinical circumstances, management of displaced mandibular injuries necessitates. Fixation can be achieved solely via an intraoral approach with a single reconstruction plate (2.3 to 2.5mm at the inferior border of the mandible or two miniplates (superior and inferior border plate) along zones of tension and compression. A transbuccal puncture for screw fixation may be required to access more posterior body fractures. A review by Ellis of 682 patients treated with ORIF of body and/or symphysial fractures with two miniplates was associated with more postoperative complications (wound dehiscence, plate exposure, need for plate removal, and tooth root damage) compared with the use of a single, larger diameter plate.

Angle

Management of angle fractures is especially complex given the distracting forces of the muscles of mastication, lack of tooth-bearing segments, and possible presence of the third molar. If the fracture is unfavorable, treatment with only MMF would be contraindicated as the proximal segment may rotate.

Ramus-Condyle Unit

Classification and management of condylar fractures is a controversial topic in craniomaxillofacial trauma. The two commonly used classification system are Lindahl and Spiessl. Lindahl's classification is based on three factors: level of fracture, amount of displacement, and relationship of the condylar head to the glenoid fossa. The three levels of fracture are condylar head (located within the joint capsule), condylar neck (inferior to the joint capsule and inferior attachment of the lateral pterygoid muscle), and subcondylar fracture (between the sigmoid notch and posterior aspect of the ramus). Spiessl's classification is based on the degree of displacement of fracture segments and dislocation of the condylar head from the fossa.

Treatment includes observation, closed reduction, ORIF by either transfacial or intraoral approaches. Current absolute indications include bilateral fractures,

severe dislocation, cases where closed reduction doesn't re-establish occlusion, concomitant fractures of other areas of the face that compromise occlusion, foreign bodies, or dislocation of the condyle into the middle cranial fossa.

Regardless of treatment choice early mobilization and physical therapy has been shown to decrease risk of ankylosis and trismus.Decision-making for condylar injuries should prioritize early mobilization. In patients with intracapsular injuries and associated malocclusion, a short course of intermaxillary fixation (no longer than 7 days) can be considered, followed by aggressive mobilization to prevent ankylosis.

In patients with displaced condylar or subcondylar injuries, treatment with open or closed approaches is valid .When considering closed management, our approach is to re-establish occlusion with closed reduction and placement of intermaxillary fixation. This is followed by a period of tight intermaxillary fixation for 2 weeks, then 2 weeks of elastic (4–6 oz) fixation with jaw stretching exercises at meal times (3–5 minutes), and then 2 weeks of elastics use only at night, with jaw stretching exercises at meal times. The use of oral muscle relaxants during the subacute healing period may help patients with jaw stretching exercises.

Special Populations

Atrophic Mandible

Atrophic, edentulous mandibular fractures represent 1% of all facial fractures. Although thought to be associated with advanced age, mandibular atrophy is more related to the duration of edentulism. The presence of tooth roots and forces of mastication send signals to inhibit bony resorption. To be classified atrophic, the remaining bone height must be 15mm or less. Severe atrophy is classified as 10mm in height or less. Decreased bony height correlates to increased incidence of nonunion, malunion, recurrent fractures, hardware failure, postoperative infection, and osteomyelitis. The incidence of these complications is between 10–20%.

ORIF has largely replaced other reduction techniques such as gunning splints, circum-mandibular wires around dentures, and external fixators. Load bearing osteosynthesis with a reconstruction plate for anatomic reduction provides a solid construct for atrophic mandible fractures. In some cases, a large inferior border plate on the lateral border, coupled with a shallow vestibule may make wearing a denture

less feasible. In these circumstances, placement of the reconstruction plate on the underside of the mandible, with screws oriented vertically or near vertically, may allow a patient to continue to wear a denture. This technique may also avoid potential intraoral hardware dehiscence. With any open approach, periosteal detachment should be limited to the buccal tissues to minimize devascularization. Autologous grafting can also be undertaken concurrently to aid in fracture healing and anticipation of dental rehabilitation.

Mandibular Injuries in Children

Mandible fractures in children follow different patterns than those observed in adults. In children, up to 50 to 80% of mandible fractures involve the condyle, subcondylar, or angle. The next most common are symphysis and parasymphysis fractures. Body fractures are relatively rare. Management must take into account eventual mandibular growth, presence of tooth buds, and eruption of permanent teeth. The main goals of treatment of pediatric mandible fractures are to obtain bony union, restore occlusion, and prevent growth disturbances.

Treatment modalities for pediatric mandible fractures include physical therapy without MMF, a short period of MMF (7–14 days) followed by physical therapy, and ORIF. When considering intermaxillary fixation in children in the primary or mixed dentition, screw-retained devices should not be used, due to the risk of injury to the developing permanent dentition. Ivy loops, Risdon cables, Erich arch bars, sutures, and dental splints are all potential options for achieving intermaxillary fixation in children. Similarly, placement of internal fixation should account for the presence of the developing permanent teeth .In the dentate regions of the mandible, plates should be placed at or near the inferior border, with monocortical screws. If titanium fixation is used, it is prudent to consider removal at 2–3 months post-operatively in growing patients. Resorbable fixation in this population remains under investigation, but appears to result in stable healing when appropriately utilized.

Similar to management in adults, treatment of pediatric condylar fractures remains controversial. Long-term favorable facial growth outcomes have been described with closed treatment of condylar fractures. Early mobilization is crucial to prevent hemarthrosis and ankylosis. TMJ ankylosis can be very difficult to treat and can children can result in profound facial deformities, particularly in growing children.

Management of odontogenic infections and sepsis:

Odontogenic infections can lead to sepsis, a potentially life-threatening condition caused by the body's immune system responding abnormally. This can lead to tissue damage, organ failure and death. A patient with non-odontogenic-related infection could also present with sepsis at a dental practice.

Odontogenic infections pass through three key stages:

- Stage 1: 1-3 days; soft and mildly tender swelling
- Stage 2: 2-5 days; hard, red and severely sore swelling
- Stage 3: 5-7 days; abscess formation.

Seven principles have been proposed to achieve the best outcome in managing odontogenic infections

- Establish the severity of the infection
- Assess host defences
- Elect the setting of care
- Surgical intervention
- Medical support
- Antibiotic therapy
- Frequently evaluate the patient.

Establish the severity of infection

A careful history and thorough clinical examinations are essential to determine the severity of any infection. History-taking will highlight factors like immune system competence and the level of systemic reserves to fight infections. A physical examination can identify clinical observations outside normal limits. C-reactive protein (CRP), fever and anatomical locations have been investigated for the assessment of the extent of odontogenic infections and presumed duration of hospital stay.

Additional factors must be considered to establish the infection severity:

- Anatomical location

- Airway compromise.
-

Ludwig's angina

A compromised airway is synonymous with Ludwig's angina and the initial assessment of a patient with Ludwig's angina should follow the familiar 'Airway, Breathing, Circulation, Disability, Exposure' (ABCDE) approach. Signs of a compromised airway in these patients could include noisy (gurgling) breathing with drooling saliva, stridor, dyspnoea, tachypnoea, tachycardia, dysphagia and trismus. The initial immediate management usually includes positioning the patient in an upright position and administering oxygen 15 litres/minute

Assess host defences

A healthy immune system is essential to the maintenance of host defence against infection

- Factors that can compromise the immune system

1. Diabetes steroid therapy
2. Organ transplants
3. Malignancy
4. Chemotherapy
5. Chronic renal disease malnutrition
6. Alcoholism.

Elect the setting of care

Severe neck infection in an immunocompromised elderly person warrants treatment in a secondary care setting.

Criteria for referral to secondary care

1. Difficulty in swallowing and dehydration
2. Threat to the airway or vital structures
3. Infection in moderate- or high-severity anatomic spaces
4. Involvement of orbital contents
5. Need for general anaesthesia

6. Need for inpatient control of systemic disease.

Surgical intervention

Five principles must be followed:

- Elimination of the source of the infection. This can be achieved by either removing the tooth or commencing root canal treatment
- Incisions to be made on healthy skin or mucosa
- Blunt dissection to explore the abscess cavity without damaging vital structures A microbiology swab should be obtained
- Copious irrigations will ensure the dilution of the bacterial load
- Drainage is maintained by placement of a drain to keep the abscess cavity open.

Blunt dissection is achieved by inserting a closed haemostat, then open it at a depth of penetration and remove the instrument while it is still open. A haemostat should never be closed while it is inside the wound. Different surgical drains are available to use and should be removed when the drainage ceases, usually between 48-72 hours.

Although abscess formation takes place between the fifth and seventh days, early elimination of the infection source and surgical intervention will decompress the involved anatomical spaces. Relying on antibiotics only in relieving dental infection is likely to be less effective and can cause antimicrobial resistance

Two of the challenges to performing adequate drainage of any odontogenic infection in dental practice are:

- Achieving adequate local anaesthesia
- Risk of spreading the infection to other anatomical spaces.

The ability to deliver safe, adequate local anaesthesia is essential for any dental procedure. The mechanism of action of the local anaesthetic solution depends on the tissue pH. In the presence of infection, tissue pH becomes more acidic, which slows down the degree of ionisation, resulting in less optimal or failed anaesthesia.

To overcome this problem, the injection of the anaesthetic solution at a distance from the inflammatory site is required (nerve blocks). It will also avoid infection spread to different tissue spaces.

Medical support

Medical support has a critical role in controlling the disease. Adequate hydration, nutrition and control of fever are essential to optimise the medical care for patients presenting with odontogenic infections. Stabilisation of any underlying systemic disease (for example, uncontrolled diabetes) is extremely important.

Antibiotic therapy

Odontogenic infections are multi-microbial with a combination of facultative and anaerobes species. Facultative *Streptococcus viridans* group are commensal Gram-positive bacteria and include *S. anginosus*, *S. intermedius* and *S. constellatus*. These organisms are abundant in the mouth and most frequently associated with orofacial cellulitis and abscess. After a few days, the anaerobes (*Prevotella* and *Porphyromonas*) predominate. The majority of the facultative *streptococci* that cause odontogenic infections are sensitive to penicillin. Approximately a quarter of strains of *Prevotella* and *Porphyromonas* are penicillin- resistant.

Penicillin-based antibiotics remain the first line for the treatment of odontogenic infections. Metronidazole is effective against anaerobic bacteria.

Recommended antibiotics and doses

Issue to treat	Antibiotic dose
First-line antibiotics for dental abscess in dental practices (adults and children more than 12 years)	Amoxicillin 500 mg TDS for five days (the dose can be doubled in severe infection) Phenoxymethylpenicillin 500 mg QDS for five days (the dose can be doubled in severe infection)

Issue to treat	Antibiotic dose
	Metronidazole 400 mg TDS should be used as an alternative if the patient is allergic to penicillin or as adjunct to the above antibiotics in spreading infection
Second-line antibiotics for dental abscess (if a patient has not responded to the first-line treatment)	Clindamycin 150 mg QDS for five days Co-amoxiclav 375 mg TDS for five days Clarithromycin 250 mg BD for five days

Frequently evaluate the patient

The last principle, but as vital as the previous ones, is the periodic re-evaluation of these patients. In outpatient settings, the recommended follow-up is after two days. Forty-eight hours will allow the drainage to cease and the immune system to overcome the initial insult from the infection. If no improvement or deterioration of symptoms is noted, further escalation in care must be provided. The review interval, however, depends on the clinical course of the infection. A patient with a rapidly developing swelling and mild temperature may need review within 24 hours, but a patient with a chronic abscess and no systemic symptoms will need to be reviewed at the end of the antibiotic treatment.

Causes of treatment failure include:

- Failure to remove the source of infection
- Underlying systemic disease; for example, uncontrolled diabetes
- Antibiotic-related factors - patient non-compliance, drug not reaching site secondary to inadequate drainage, wrong antibiotic choice or incorrect dose.

MANAGEMENT OF SEPSIS

Incidence of sepsis

Although deaths from sepsis due to odontogenic infection are very rare, they have been reported. The incidence of sepsis is on the increase, possibly due to:

- A growing elderly population
- An increased use of invasive surgery
- An increased incidence of bacterial resistance
- An increased number of immunocompromised patients

Causes of sepsis

- A localised infection which progresses into an uncontrolled systemic response is usually the cause of sepsis. Progression to acute physiological deterioration with the risk of multiple organ failure and death can be swift.

- In normal circumstances, the body's immune system will prevent or fight infection (bacteria, viruses, fungi). However, the immune system can sometimes go into overdrive, resulting in vital organs and other tissues being targeted. This can result from any injury or infection in the body.
- Although a wide variety of different microorganisms (for example, *Streptococcus*, *E. coli*, MRSA or *Clostridium difficile*) can cause sepsis, it is usually caused by common bacteria that don't normally make patients ill.

- Any infection can lead to sepsis though pneumonia (commonly referred to as chest sepsis) is the cause in half of the cases.

- **Sources of sepsis infection (approximate percentages)**
    1. Pneumonia: 50%
    2. Urinary tract: 20%
    3. Abdomen: 15%
    4. Skin, soft tissue, bone and joint: 10%
    5. Endocarditis: 1%
    6. Device-related infection: 1%
    7. Meningitis: 1%
    8. Others: 2%.

Risk factors for developing sepsis

The National Institute for Health and Care Excellence (NICE)has highlighted the following risk factors for sepsis:

- Children under one year of age and people >75 years old
- Frailty
- Impaired immune systems because of illness or drugs, including patients:
- On chemotherapy for cancer
- Taking long-term steroids
- Taking immunosuppressant drugs to treat non-malignant disorders such as rheumatoid arthritis
- With an impaired immune function; for example, diabetics, previous splenectomy and sickle cell disease
- Recent surgery, or other invasive procedures, in the past six weeks
- Breach of skin integrity; for example, cuts, burns, blisters or skin infections
- Intravenous drug users
- Existing indwelling venous line or urinary catheter
- Pregnancy and within six weeks following birth, termination of pregnancy or miscarriage.

General Principles and Outline of Management

1. All patients with suspected carcinoma of head and neck should be evaluated by a head and neck surgical oncologist and should record the following:

A. History

- Disease related information
- Detailed history of habits and addictions
- Medical and Family history, including any prior malignancy
- Comorbidity

B. Clinical Examination

- Performance and Nutrition status assessment
- Histological diagnosis – FNAC/Biopsy/ Slide review
- Imaging for extent of disease and assessment of operability
- Clinical staging and documentation of the subsite(s) involvement

C. Investigations

- X-Ray
- Chest CT Scan / MRI for extent of disease
- EUA / Endoscopy for mapping of disease
- USG for N0 neck in select cases
- Ba swallow + PC film
- PET - CT whenever indicated.

Treatment decisions for all patients should be made in a multidisciplinary joint clinic with the goal for maximizing survival and preservation of form and function.

General guidelines for selecting a treatment modality:

- Stage I / II disease - Single modality (Surgery or Radiotherapy)
- Stage III & IV disease - Combined modality

1. Surgery + Radiotherapy ± chemotherapy
2. Chemotherapy + radiotherapy

Selection of modality depends on the subsite of cancer.

- When different modalities are available, the modality that gives maximum chance of cure should be used.
- When different modalities have similar results, a modality that gives better quality of life, with organ / function preservation is preferred.

Surgery is preferred over radiotherapy as a single modality in

1. Sites where surgery is not morbid (cosmetically and functionally)
2. Lesions involving or close to bone - to prevent radionecrosis.
3. Young patients – possibility of a subsequent second primary
4. Presence of sub mucous fibrosis (SMF).

Radiotherapy is preferred over surgery as a single modality, were

1. Severe impairment of function / cosmesis with surgery, e.g. base tongue, glottis.

2. Surgery is technically difficult with high morbidity and poor results e.g. nasopharyngeal carcinoma.

3. Patient refuses surgery

4. High risk of surgery

For patients undergoing planned surgery,

- A plan should be developed for a tumour free resection margin and appropriate reconstruction for restoration of form and function
- No modification of this plan should be done based on response to any prior chemotherapy
- Modify plan for wider resection, if there is disease progression while waiting.
-

Assessment of resectability

A. Tumour involvement of the following structures are considered technically unresectable:
    - Erosion of pterygoid plates, sphenoid bone, widening of foramen ovale
    - Extension to superior nasopharynx or deep extension into Eustachian tube or lateral nasopharyngeal wall
    - Encasement of internal carotid artery, defined radiologically as tumor surrounding the carotids > 270 degrees.
    - Involvement of mediastinal structures
    - Involvement of prevertebral fascia or cervical vertebrae

Principles of resection

1. En bloc resection of primary tumor whenever feasible

2. In continuity neck dissection when direct extension of primary into neck

3. Third dimension (the base) should be taken carefully into account before excision

4. Adequate margin: 1.5 – 2 cm

5. Clear margin: > 0.5 cm

6. Close margin < 0.5 cm

7. Frozen section confirmation for margins may be done if the facility is available

8. Contralateral neck should be addressed when the probability of bilateral / contralateral metastases is high. Eg. Tumours crossing the midline / midline tumours.

Reconstruction options:

1. Mucosal defects:

- Small defect –Primary closure/local flap / SSG / leave raw according to the site involved
- Large defect –Try to replace tissue loss with similar kind of tissue

2. Soft tissue loss: (Pedicled Flaps Eg. PMMC) or Free tissue transfer

- Skeletal defects +/- Soft tissue and Skin loss
  - Anterior or Midline:
  - Free fibula / Deep Circumflex Illiac Artery (+/- Skin paddle
  - Regional osteo myocutaneous flaps
  - Plate
  - Posterior Segment
    - PMMC
    - Free Fibula
- Skin defects can be covered with
- Local flaps /forehead flap
- Deltopectoral flap / PMMC Free flaps

Indications for postoperative radiotherapy

1.Primary:

- Large primary – T3/T4
- Deep infiltrative tumour
- High grade tumour
- Lymphovascular and perineural invasion

2. Lymph nodes:

- Bulky nodal disease N2/N3
- Extra nodal extension
- Multiple level involvement
- Multiple nodes

3. Chemo-radiotherapy

- Positive or close margin after curative resection
- Nodes with perinodal extension

4. Role of Brachytherapy (BRT)

- Accessible lesions
- Small (preferable < 3 cm) tumours
- Lesions away from bone
- N0 nodal status
- Superficial lesions

Dose for radical radiotherapy

Tumours suitable for brachytherapy

- **T1-2 N0:-** Radical BRT: 60-70Gy low dose rate 192Iridium or equivalent doses with fractionated high dose rate.
- **T1-3 N0-1**- External RT: 56 -60Gy/28-30#/6wks Boost BRT: Low dose rate 192Iridium: 15-20 Gy or High Dose rate: 14Gy in 4 fractions over 2 days (4-3- 3-4 Gy)

Tumours not suitable for brachytherapy

- **T1-4 N0-2** -Concomitant chemoradiation: 66-70Gy/33-35#/ 6-7 wks + Sconcomitant Cisplatin, 30mg/m2 for 6-7 wks or 3 weekly Cisplatinum, 100mg /m2 x 3 cycles

Or

- **External RT**: 66-70GY/33-35#/6-7weeks (reducing fields)

Doses and Volumes in adjuvant setting

- Primary and involved nodal disease: 56-60 Gy/ 28-30#/6 weeks, using reducing fields.
- Site of residual disease, positive cut margins: 4-10 Gy Boost
- Uninvolved nodal stations: 45 -50 Gy

Dose of chemotherapy in the adjuvant setting in combination with radiotherapy: 30mg/m2 weekly with hydration and antiemetic prophylaxis

Rehabilitation

- Abstinence from tobacco/alcohol
- Oral hygiene
- Shoulder physiotherapy in all cases of neck dissections
- Bite guide prosthesis following mandibulectomy
- Jaw stretching exercises to prevent post-operative trismus
- Swallowing and speech rehabilitation

Follow up :

- Every 2-3 months in first 2 years
- Six monthly for next 3 years
- Annually thereafter
- On every follow up thorough head and neck examination for loco-regional control, second primary tumour and late sequelae of treatment. Investigation only if indicated by symptoms and positive clinical findings.
- Serum T3, T4 & TSH annually for all patients ssreceiving RT.

MANAGEMENT OF PRIMARY: (Lip/Buccal Mucosa/Oral Tongue/Floor of mouth/Lower alveolus /Retro Molar Trigone)

LIP

- **T1, T2 Tumors:** Surgery or RT Surgery: Wide excision Radiotherapy: Radical Radiotherapy / Brachytherapy.
- **T3, T4 Tumors:** Surgery + Post operative RT/ CT-RT Surgery: Wide excision with marginal/ segmental / hemimandible resection with appropriate reconstruction.

BUCCAL MUCOSA

- **T1, T2 Tumors:** Surgery or RT Surgery: wide excision +/- marginal mandibulectomy with appropriate reconstruction. Radiotherapy: Radical RT/ Brachytherapy
- **T3, T4 Tumors**:Surgery + Post operative RT/ CT-RT Surgery: Composite resection of the buccal mucosa with mandible or upper alveolus or overlying skin with reconstruction.

ORAL TONGUE & FLOOR OF MOUTH

- **T1, T2 Tumors:** Surgery or RT Surgery: Wide excision Glossectomy / Hemiglossectomy with appropriate reconstruction. Radiotherapy: Radical RT/ Brachytherapy.
- **T3, T4 Tumors**:Surgery + Post operative Radiotherapy/ CTRT

Surgery: Appropriate wide excision glossectomy with mandibular swing or pull through along with lingual plate / segmental / hemimandibular resection, if required (based on extent of involvement) with reconstruction.

LOWER ALVEOLUS & RETRO MOLAR TRIGONE

- Mandible uninvolved or minimally involved

Surgery: Wide Excision with marginal mandibulectomy (avoided in RMT disease, edentulous mandible, paramandibular disease, post radiotherapy) if required with reconstruction.

- Indication for Marginal Mandibulectomy:

- Whenever tumor is close to the mandible to achieve adequate margin (5 mm-10mm)
- Limited superficial bony erosion
- Limited periosteal invasion

Mandible grossly involved

Surgery + Post operative/ CT-RT

Surgery: Wide Excision (cheek flap) with segmental/ hemimandible resection with reconstruction.

Indication for Segmental Mandibulectomy:

- Gross tumor invading the mandible
- Prior radiotherapy s
- Edentulous mandible
- Gross paramandibular disease
- Whenever inferior soft tissue and bony margin of 1 cm is not possible (Eg. Retro Molar Trigone,gross periosteal invasion)

MANAGEMENT OF NECK NODES:

(Lip/Buccal Mucosa/Oral Tongue/Floor of mouth/Lower alveolus /Retro Molar Trigone)

- **T1, T2 Tumors N0**: Observe or SOHD (if cheek flap is raised, USG suspicious, thick tumor>3-4mm ,high grade tumor or poor follow up expected) followed by FS, if positive nodes MND is required
- **N+: MND / RND** :Post op RT as per earlier guidelines.
- **T3, T4 Tumors N0:** SOHD followed by FS, if positive nodes MND is required
- **N+: MND / RND** : Bilateral neck needs to be addressed if the primary disease is in midline or extending across midline (including middle third mandible). Post op RT/CT-RT as per earlier guidelines.

Primary:

Maxillary antrum not involved

- Surgery: Upper alveolectomy / Partial maxillectomy
- Radiotherapy: Radical RT / Brachytherapy for selected early T1-2 Hard palate lesions

Maxillary antrum involved

- Surgery: Orbital floor preserving total maxillectomy with reconstruction.

Nodes: Neck needs to be addressed if the neck is clinically positive, if there is extension of the primary disease to the buccal mucosa or there is soft tissue infiltration or radiological suspicion of metastatic node. Post operative RT/ CT-RT as per guidelines mentioned earlier.

Reconstructive options for oral cavity

Objectives:

- Achieve primary healing
- Maintain oral competence
- Facilitate swallowing
- Prevent aspiration
- Preserve speech
- Cosmesis

Based on the size and composition of defect, the options are:

Mucosal defects

- Leave raw
- Primary closure
- Split thickness skin graft (STSG)
- Mucosal grafts

Full thickness defects

- Local Flaps: Abbe-Estlander's flap, Gille's Flap (for lip)
- Regional flaps: Tongue flap, Nasolabial flap, Facial artery myomucosal flap, Masseter flap, Platysmal flap, , Forehead flap
- Distant Flaps: Pectoralis major myocutaneous flap, Deltopectoral flap, Latissimus dorsi myocutaneous flap
- Free Flaps: Radial forearm flap, Lateral arm flap, Antero-lateral thigh flap

Mandibular Defects

Anterior mandibular defect needs to be reconstructed by

- Free osteocutaneous flapsFibular osteocutaneous flap (preferred because of long bone length, easy contouring and dual blood supply), Radial osteo- cutaneous flap, Scapular osteocutaneous flap
- Distant flapsPectoralis major myocutaneous flap, Latissimus dorsi osteocutaneous flap, Trapezius osteocutaneous flap

Lateral mandibular defects may be reconstructed with

- adequate soft tissue replacement, complemented by proper use of guide bite prosthesis and appropriate postoperative isometric exercises.

Criteria for Inoperabilitsy:

Primary disease: Adequate surgical clearance is not achievable.

- Extensive Infratemporal Fossa involvement
- Extensive involvement of base skull.
- Extensive induration /soft tissue disease till zygoma or hyoid.

Nodal Disease: s

- Clinically fixed nodes.
- Infiltration of Internal /Common carotid artery.
- Extensive infiltration of prevertebral muscles, skull base.

These patients are usually treated with palliative intent with chemotherapy or radiotherapy. If general condition is good, then concurrent chemo radiotherapy can be offered. If general condition is poor, then only best supportive care.

Prognosis: Oral cavity stage 5 year relative survival (95% CI)

- Stage I 69.5% - 73.5%
- Stage II 55.5% - 60.4%
- Stage III 41.8% – 47.3%
- Stage IV 40.3% – 33.6%

Lip stage 5 year relative survival (95% CI)

- Stage I 86.5% - 92.7%
- Stage II 75.5% - 91.5%
- Stage III 39.8% - 69.8%

Stage IV : 34.2% - 60.1%

PEADIATRICS STANDARD TREATMENT GUIDELINES

ENTERIC FEVER

INTRODUCTION

Enteric fever is acute generalized infection of reticuloendothelial system with predilection for intestinal lymphoid tissue and gallbladder.

The term includes typhoid fever caused by Salmonella typhi (around 80% of all cases worldwide) and paratyphoid fever caused by Salmonella paratyphi A or B (20% of all cases). The bacterium is gram negative and nonlactose fermenting.

DIAGNOSIS

Mainly clinical: The most common cause of fever without focus.

- Infant to children up to 5 years: Fever, vomiting, and diarrhea.
- Older children: Fever in increasing trend (step ladder pattern) over 5–7 days, anorexia, abdominal pain, cough followed by toxic look, lethargy, tender abdomen, soft splenomegaly, hepatomegaly, and relative bradycardia.
- Rash: Rose spots described in Western textbooks is almost never seen in Indian children.

LABORATORY DIAGNOSIS:

- Hemogram: Total leukocyte count: normal or low, with neutrophilia and thrombocytopenia. Eosinopenia is remarkably consistent with typhoid fever. There may be mild elevation of transaminases.
- Culture and sensitivity: It is the gold standard and the most important investigation for diagnosis. Automated blood culture systems like BACTEC have improved recovery and are cost-effective in the long run. Salmonella is an easy organism to culture and antimicrobial sensitivity results are important for treatment.
    - Blood culture: 90% yield in first week and up to 40% in the fourth week of illness. Send paired cultures with total volume of blood to be sent as 5–10 mL with a blood: broth ratio of 1:5.
    - Bone marrow culture is an important investigation in pyrexia of unknown origin (PUO) in later stages of the illness as it remains positive even after antibiotic therapy.

- Stool and urine cultures are not recommended due to poor yield.

- Serology: These tests are not diagnostic, may be supportive and should not be relied upon for patient management decisions.

  - Widal test: It detects presence of immunoglobulin M (IgM) and IgG antibodies against H (flagellar antigen) and O (somatic antigen) of S. typhi and paratyphi A and B in the second week of illness. Tube method is better than the slide method.

  Antibody titer of both O and H in range of 1:160 dilution or more is taken as a positive test. Fourfold rise in titer in paired samples 1 week apart is the conventional method, however, it is less practical.
  As sensitivity and specificity are low, widal may come false positive in malaria, rickettsial infection, or infection with other Enterobacteriaceae.

  It may come false negative in patients treated with prior antibiotics.

  - Typhidot/enzymeimmuneassay(EIA)test: It detects IgM and IgG antibodies against 50 kd outer membrane protein antigen which is specific for S.typhi. Specificity is only 37% and anamnestic reactions may be seen in other infections. A Cochrane database review in 2017 concluded that the rapid diagnostic serologic tests need further robust evaluation.

Choice of empirical therapy for typhoid fever is shown in Table 1.

TABLE 1: Choice of empirical therapy for typhoid fever.

Patient's condition	**First-line choice**	**Second-line choice**
Severe illness Indoor patient Any complications	Ceftriaxone	Cefotaxime (concomitant hepatitis) Aztreonam (penicillin allergy)
Outpatient department	Cefixime	Azithromycin (penicillin allergy)

Drug dosage guideline is presented in **Table 2**.

TABLE 2: Drug dosage guideline.

Drug	**Dose (mg/kg/day)**	**Maximum dose (per day)**
Ceftriaxone 100 4 g	Ceftriaxone 100 4 g	Ceftriaxone 100 4 g

Cefotaxime 150–200 8 g	Cefotaxime 150–200 8 g	Cefotaxime 150–200 8 g
Cefixime 20 1,200 mg	Cefixime 20 1,200 mg	Cefixime 20 1,200 mg
Azithromycin 20 1 g	Azithromycin 20 1 g	Azithromycin 20 1 g
Aztreonam 50–100 8 g	Aztreonam 50–100 8 g	Aztreonam 50–100 8 g

DURATION OF TREATMENT

Treat for at least 7 days after defervescence or a total of 14 days, whichever is later. Azithromycin is used for a total of 7 days.

ROLE OF STEROIDS

Steroids are indicated only in severe illness. If the patient presents with shock, coma, or in altered sensorium, dexamethasone in the dose of 3 mg/kg followed by 1 mg/kg every 6 hours for 2 days may be given. Prolonged use of steroids can increase the relapse rate and cause adverse effects, hence use judiciously.

DELAYED DEFERVESCENCE

If by day 7 of antibiotics, defervescence has not occurred but child looks less toxic, there is increase in duration between fever spikes or quick response to antipyretics, same antibiotics can be continued till day 10.

Clinical Failure

No defervescence and child looks toxic with increase in fever spikes after 7 days of starting
optimal treatment is clinical failure. In
such a scenario, rule out:

- Complications such as abscess formation and infection-associated hemophagocytic lymphohistiocytosis (HLH)
- Coinfections such as malaria and hepatitis A
- Drug fever or thrombophlebitis
- If the child was culture negative, review the diagnosis with careful history, physical examination, and repeat investigations.

Special Therapeutic Concerns

- Culture positive report empowers the clinician, gives confidence even in late defervescence and prevents unnecessary use of azithromycin which must be kept as a reserve drug.

- Quinolones are contraindicated in pediatric age group and should not be used for treatment of
- enteric fever.

- Aminoglycosides like amikacin have no role in management as their site of action is extracellular while Salmonella is an intracellular organism.

- In the era of antimicrobial resistance (AMR), it is very important that laboratories give drug sensitivity reports with minimum inhibitory concentrations (MICs) and its interpretation. In enteric fever, MIC ≤1 for ceftriaxone is associated with excellent clinical outcome. Rising ceftriaxone MICs are being reported from India. MIC breakpoints for resistance to ceftriaxone are [≥4 as per Clinical and Laboratory Standards Institute (CLSI)] and [>2 as per European Committee on Antimicrobial Susceptibility Testing (EUCAST)]. Azithromycin is the drug of choice in ceftriaxone resistant isolates. If such a child is hemodynamically unstable or the disease is severe, then meropenem may be used.

RELAPSE

Even after adequate treatment, enteric fever has a relapse rate of 5–20%. Recurrence of fever 2–3 weeks after its initial resolution is called relapse. It is usually milder. Treatment of relapse is with the same drug used for initial therapy. Relapse can be differentiated from reinfection only by molecular typing.

CARRIER STATE

It is defined as an asymptomatic person who sheds Salmonella in stool or urine beyond 3 months of an episode of enteric fever. It is uncommon in pediatric age group hence post illness screening for S. typhi carriage is not recommended. If detected treat with trimethoprim-sulfamethoxazole (10 mg/kg/day for 6–12 weeks) or high dose amoxicillin (75–100 mg/kg/day for 4–6 weeks) to decrease the risk to close contacts.

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CROUP IN CHILDREN

DEFINITION

Croup or viral laryngotracheobronchitis (LTB) is one of the frequent causes of stridor. Stridor is a high pitched, harsh sound which occurs during inspiration. Wheeze is a musical sound that occurs during expiration, due to lower airway obstruction.

EPIDEMIOLOGY

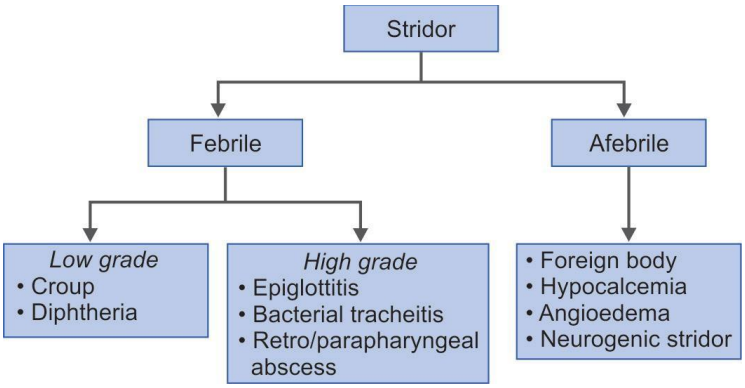
Highest incidence among the preschool children (6 month to 3 year of age), occurs during the autumn and winter months. Less than 5% of children with croup, require hospitalization
and among those hospitalized only 1–2% require intensive care. Mortality rate in croup is
usually <0.5% even for intubated patients.

ETIOLOGY OF CROUP

Viral infections: Parainfluenza types 1 and 3 accounts for >70% of viral LTB cases. Other viruses— influenza A, influenza B, adenovirus, respiratory syncytial virus, and metapneumovirus.

Though croup is the most common cause, there are many other causes of stridor which are given in the Flowchart 1.

Flowchart 1: Various causes of stridor and clinical approach.



CLINICAL

- Sudden onset of a distinctive barky cough
- Usually preceded by upper respiratory infection (URI) symptoms
- Accompanied by stridor and respiratory distress
- Hoarse voice

LABORATORY

A complete blood count (neutrophilic leukocytosis) and high C-reactive protein (CRP) may help distinguish croup from bacterial etiologies of stridor (e.g., bacterial tracheitis, epiglottitis, peritonsillar abscess, and retropharyngeal abscess), but it is nonspecific.

X- RAY CHEST

It may show classical steeple sign secondary to glottic and subglottic narrowing (Fig. 1). However, this finding is neither specific nor sensitive for croup. X-ray neck lateral view will be useful in the diagnosis of epiglottitis and retropharyngeal abscess. Identification of organism by doing antigen test or culture can be useful to identify bacterial cause other than croup.

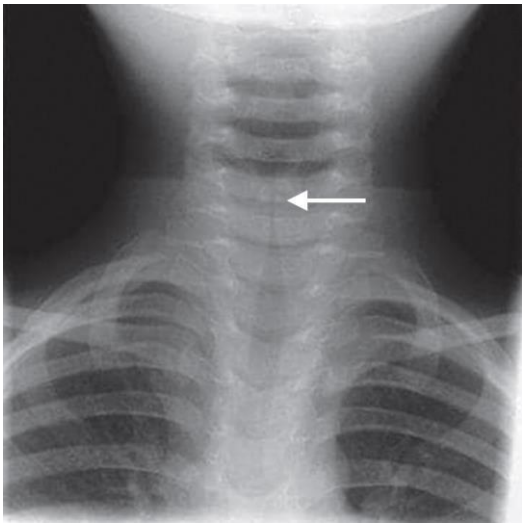


Fig. 1: Church steeple sign in croup (arrow). Picture Courtesy: Dr Paramarth C

DIAGNOSIS

TABLE 1: Severity assessment.			
Signs Mild-to-moderate Severe Life-threatening	**Signs Mild-to-moderate Severe Life-threatening**	**Signs Mild-to-moderate Severe Life-threatening**	**Signs Mild-to-moderate Severe Life-threatening**
Sensorium Alert (A) Lethargic	Sensorium Alert (A) Lethargic	Sensorium Alert (A) Lethargic	Sensorium Alert (A) Lethargic
arousable (V)	arousable (V)	arousable (V)	arousable (V)
Agitated, pain responsive, or unresponsive (in	Agitated, pain responsive, or unresponsive (in	Agitated, pain responsive, or unresponsive (in	Agitated, pain responsive, or unresponsive (in
AVPU scale)	AVPU scale)	AVPU scale)	AVPU scale)
Respiratory	Respiratory	Respiratory	Respiratory

(AVPU: alert, verbal, pain, unresponsive; ICR: intercostal recession; SCR: subcostal recession; SpO2: oxygen saturation; SSR: suprasternal recession)

Source: Modified from Bjornson CL, Johnson DW. Croup in children. CMAJ. 2013;185(15):1317-23.

MANAGEMENT

Initial Management

- Baby should be kept on mother's lap. Separation and crying may worsen stridor.
- Oxygen should be administered in a nonthreatening manner to maintain oxygen saturation (SpO2) > 95%.

- Postpone intravenous access attempt or blood tests, unless it is absolutely needed.
- Do not insert tongue depressor. If essential, can be done later after stabilization.
- Do not sedate the child until airway is secured.
- Never shift the child for X-rays before stabilization.

Specific Management (Flowchart 2)

Mild

- Oral dexamethasone at 0.6 mg/kg or nebulized budesonide 2 mg.
- If stable, send home instructing the parents about the natural course and likelihood of recovery in 48–72 hours.
- Explain the warning signs of worsening (worsening of stridor, poor feeding, or change in level of consciousness) and instruct to come to emergency department (ED), if worsening happens.
- There is no role for antibiotics or beta-agonist nebulization in viral croup.

Moderate-to-severe

It is preferable to hospitalize the child.

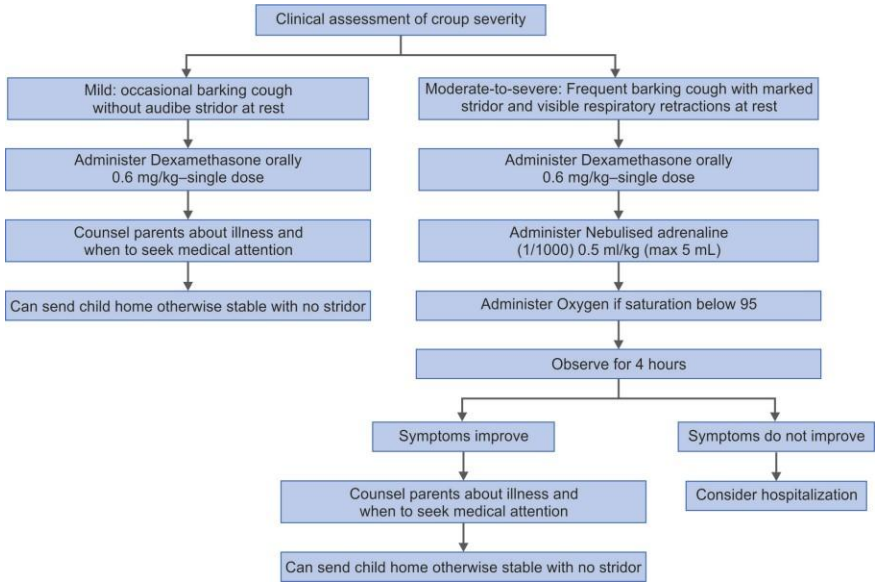
❖ Adrenaline nebulization:
  - Undiluted 1:1,000 adrenaline 0.5 mL/kg is mixed with normal saline (NS) to a maximum dose of 5 mL. For example, in a child with body weight 8 kg, adrenaline 4 mL and 1 mL of NS is used. In a child with 10 kg and beyond undiluted adrenaline can be used without NS.
  - Second dose can be repeated after 2 hours, if needed.
  - Caution: Here epinephrine is used as nebulization and not as parenteral route.

❖ Steroids:
  - It is indicated even in children who recovers after adrenaline nebulization as the effect of adrenaline will wane after 2 hours.
  - Dexamethasone—0.6 mg/kg (maximum—8 mg) oral or IV or IM
  - Nebulized budesonide 2 mg (dose same at all ages)
  - Majority may require single dose of steroids, but in severe cases frequent doses may be needed for 48 hours.

INDICATION FOR IMMEDIATE REFERRAL TO HOSPITAL

- If the child is pain responsive or unresponsive, having reduced respiratory effort, and saturation <94%.
- Accompany the child, simultaneously supporting with bag valve ventilation with oxygen.

Flowchart 2: Algorithm for management of croup.



Source: Zoorob R, Sidani M, Murray J. Croup: an overview. Am Fam Physician. 2011;83(9):1071.

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COMMUNITY-ACQUIRED PNEUMONIA

DEFINITION

Acute infection of lung parenchyma in previously healthy child, acquired outside of the hospital settings, and not hospitalized within 14 days prior to onset of symptoms. This excludes children with immunodeficiency, severe malnutrition, and postmeasles state.

ETIOLOGICAL TYPES

TABLE 1: Etiological types and characteristic differentiating features.

Viral pneumonia Streptococcal	**Viral pneumonia Streptococcal**	**Viral pneumonia Streptococcal**	**Viral pneumonia Streptococcal**
• Follows short upper respiratory tract infection (URTI) • Gradual onset cough • Less toxic look • Wheeze may be associated (bronchiolitis like features) • Usually, bilateral affecting all lobes • Lasts 3–5 days and resolves spontaneously	• More toxic • Rapid progression • Lobar pneumonia • Gastrointestinal manifestations (lower lobe pneumonia)	• Empyema • Cellulitis/ abscess • Necrotizing pneumonia • Pneumatocele formation	• More like viral pneumonia • Wheezing • May not be sick (walking pneumonia) • Diffuse lung involvement

CLASSIFICATION

TABLE 2: Revised World Health Organization (WHO) classification (2014) in children aged 2–59 months.

Classification	**Clinical findings**
No pneumonia	Cough and cold
Pneumonia	Fast breathing: ≥ 50/min (2 months to 1 year) ≥ 40/min (>1–5 years) ≥ 30/min (>5 years)

	and/or Chest indrawing
Severe/very severe pneumonia	General danger signs • Not able to drink/feed • Persistent vomiting • Convulsions, cyanosis • Lethargy/Unconscious • Stridor in a calm child • Severe malnutrition

Persistent single cardinal clinical sign which is very sensitive and specific to diagnose pneumonia is rapid breathing or tachypnea. Auscultatory features are not sensitive.

TRIAGING

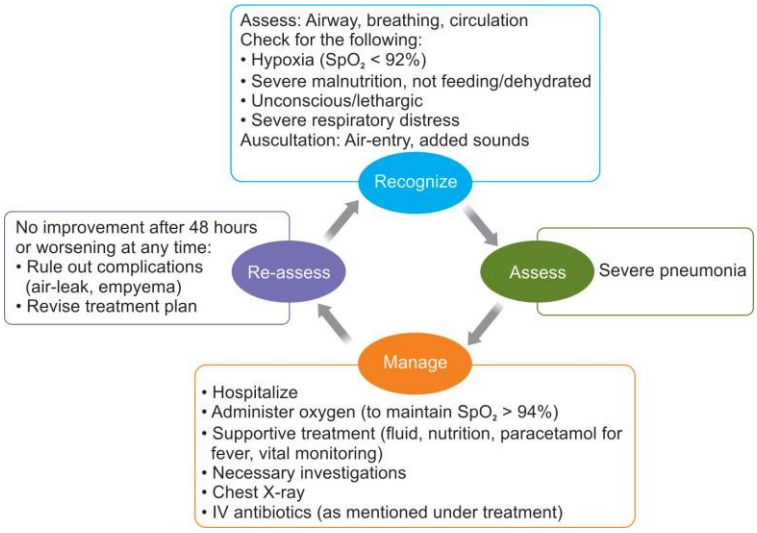

Fig. 1: Triaging of a pneumonia case (2–59 months) in the emergency. (IV: intravenous; SpO2: oxygen saturation)

Source: Pneumonia in Children (PIC) Module of IAP Respiratory Chapter, 2021.

INITIAL APPROACH

- Community-acquired pneumonia (CAP) is a clinical diagnosis and no investigations are required in outpatient department (OPD) setting.
- Investigations required in hospitalized children—complete blood count (CBC), blood culture, chest X-ray, inflammatory markers [C-reactive protein (CRP) and procalcitonin], and molecular methods [multiplex reverse transcription–polymerase chain reaction (RTPCR) and BioFire).

- A combination of CRP, procalcitonin, and CBC—better understanding the response to the treatment.
- Isotype enzyme-linked immunosorbent assay (ELISA) for antibody detection against Mycoplasma—better than cold agglutinins.
- Pulse oximetry is helpful in assessing the severity and monitoring response to treatment in hospitalized children or those with severe disease.

INDICATIONS FOR ADMISSION OR REFERRAL

TABLE 3: Indications for admission or referral.		
Age < 3 months Oxygen saturation (SpO2)	Age < 3 months Oxygen saturation (SpO2)	Age < 3 months Oxygen saturation (SpO2)
< 92%	< 92%	< 92%

TREATMENT

TABLE 4: Outpatient treatment (oral therapy).			
Age	**First line**	**Second**	**If Staphylococcus aureus suspected**
<3 months Always admit and treat in the hospital			
3 months to 5 years	Amoxicillin (80 mg/ kg/d), BD for 5 days (in India, 40–50 mg/ kg/d is sufficient as penicillin-resistant pneumococci prevalence is <10%)	Co-amoxiclav (dose schedule same as that of amoxicillin) Or Cefpodoxime (10 mg/kg/d), BD for 5 days Or Cefuroxime (30 mg/kg/d), BD for 5 days	Co-amoxiclav (dose schedule same as that of Amoxicillin) Or Cefuroxime (30 mg/kg/d), BD for 5 days Or Linezolid* (10 mg/kg/d), TID for 5 days
>5 years	Same as above	Co-amoxiclav or cefpodoxime (as above) Or Azithromycin (10 mg/kg/d),	Same as above

		OD for 5 days (empty stomach	

* Linezolid is a reserve drug for tuberculosis (TB), so the National Tuberculosis Elimination Programme (NTEP) has advised to use it with caution.

INPATIENT TREATMENT AND SWITCH TO ORAL THERAPY

TABLE 5: Inpatient treatment (parenteral therapy).

Age	**First line**	**Second**	**If Staphylococcus aureus suspected**
<3 months	Cefotaxime ± gentamicin (5–7 mg/kg/d, OD) Or Amikacin (15 mg/kg/d, OD) Or Ceftriaxone (75–100 mg/kg/d), BD	Piperacillin-tazobactam ± gentamicin or amikacin Or Cefoperazone-sulbactam ± gentamicin or amikacin	Ceftriaxone + cloxacillin (50–100 mg/kg/d, QID) Or Cefuroxime/or co-amoxiclav* + gentamicin or amikacin Second line Ceftriaxone + vancomycin (40–60 mg/kg/d, QID) or linezolid** (same as oral dose)
3 months to 5 years	Ampicillin (100 mg/ kg/d, TID or QID)***	Co-amoxiclav* Or Cefotaxime Or Ceftriaxone	Ceftriaxone + Cloxacillin Or Cefuroxime or Co-amoxiclav or cefazolin (50 mg/kg/d, BD or TID) Second line Ceftriaxone + vancomycin or clindamycin (20 mg/kg/d, TID or QID)

			or linezolid** (same as oral dose)
>5 years	Ampicillin (dose same as above)	Co-amoxiclav* Or Cefotaxime (150 mg/kg/d, TID) Or Ceftriaxone Or Azithromycin	Same as above

* Co-amoxiclav injectable dose: 100 mg/kg/d, TID.
** Linezolid is a reserve drug for tuberculosis (TB), so the National Tuberculosis Elimination Programme (NTEP) has advised to use it with caution.
***Ampicillin dose in severe infection: 200 mg/kg/d, TID or QID.

TABLE 6: Oral therapy in hospitalized children.

Etiological agents	Parenteral therapy	Oral therapy	Total duration
Bacteria other than Staphylococcus aureus	b-lactam antibiotics	Amoxicillin OR cefpodoxime OR cefdinir (14 mg/kg/d, BD)	7–10 days
Methicillin-susceptible Staphylococcus aureus (MSSA)	b-lactam antibiotics	Cephalexin (50 mg/kg/d, BD or TID) Or Co-amoxiclav	7–10 days
Methicillin-resistant Staphylococcus aureus (MRSA)	b-lactam antibiotics + vancomycin/ clindamycin	Linezolid* Or Clindamycin	14 days (if no complications) Or 4–6 weeks (if complications)

* Linezolid is a reserve drug for tuberculosis (TB), so the National Tuberculosis Elimination Programme (NTEP) has advised to use it with caution.

TREATMENT

Macrolides in CAP (used in following situations):

- In a child immunized against Hemophilus influenzae type b (Hib)/pneumococcal conjugate vaccine (PCV): If no response to first-line antibiotics or suppurative complications of CAP are absent.

- Persistence of the following: Low-grade fever, cough, few clinical signs, and chest X-ray showing bilateral perihilar streaky infiltrates.
- Extrapulmonary manifestations not suggestive of Staphylococcus aureus or no response to antistaphylococcal antibiotics.

For Viral Pneumonia

- Only symptomatic and supportive treatment
- Oseltamivir can be given if H1N1 infection is suspected but that should be initiated within 3 days of symptoms. The details of dose schedule are provided in Table 7 [recommended by the American Academy of Pediatrics (AAP) and Centers for Disease Control and Prevention CDC)].

TABLE 7: Indication and dose schedule.

Indications	**Dose schedule**
Treatment	Infants (<1 year old): 3 mg/kg/dose twice daily Children (≥1 year old): ≤15 kg: 30 mg twice daily >15–23 kg: 45 mg twice daily >23–40 kg: 60 mg twice daily >40 mg: 75 mg twice daily
Prophylaxis (7 days)	Not indicated in infants <3 months of age (limited data) Infants ≥3 months and <1 year of age: 3 mg/kg/dose once daily Children (≥1 year old): The doses mentioned above under different weight band should be given as once daily dosing

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ACUTE WATERY DIARRHEA

DEFINITION

- Acute watery diarrhea is defined as a change in the consistency of stool leading to loose or liquid stools and/or an increase in the frequency of evacuations to three or more in 24 hours, with or without fever or vomiting lasting 7 days or less.
- Frequent passing of formed stools is not diarrhea. Babies fed only on breast milk often pass loose, "pasty" stools; this also is not diarrhea.

ETIOPATHOGENESIS

- Viruses are the most common agents accounting for >60% of cases, followed by bacteria and parasites. Globally, Rotavirus infection remains the leading cause of diarrhea in children < 5 years.
- Water and electrolytes (sodium, chloride, potassium, and bicarbonate) are lost through liquid stools, vomit, sweat, urine, and breathing. Dehydration occurs when these losses are not replaced.

ASSESSMENT OF DEHYDRATION

- The degree of dehydration is rated on a scale of three.

Severe dehydration (at least two of the following signs):
- Lethargy/unconsciousness
- Sunken eyes
- Unable to drink or drink poorly
- Skin pinch goes back very slowly (≥2 seconds)

Some dehydration (two or more of the following signs):
- Restlessness and irritability
- Sunken eyes
- Drinks eagerly and thirsty

No dehydration

- Not enough signs to classify as some or severe dehydration.

In most cases, children with acute watery diarrhea do not require any diagnostic workup.

- In severe conditions and/or in the hospital setting, investigations may be appropriate in individual cases.
  - Microbiological investigations should be considered in the following:
  - Children with underlying chronic conditions (e.g., oncologic diseases, inflammatory bowel
  - disease, and immunodeficiency)
  - Extremely severe clinical conditions (e.g., sepsis)
  - Prolonged symptoms (>7 days)
  - During outbreaks (childcare, school, and hospital)
  - Children with high fever
  - History of travel to at-risk areas
  - In children with severe dehydration, renal function test, serum electrolytes, and blood glucose should be done..

MANAGEMENT

To prevent potentially fatal complications including dehydration, metabolic acidosis, electrolyte disturbances, and sepsis.

REHYDRATION

Oral rehydration solution (ORS) (low osmolarity 75 mmol/L Na) is the first line of treatment for acute watery diarrhea (Table 1).

MANAGEMENT

TABLE 1: Rehydration therapy in acute diarrhea.

Treatment plan	Plan–A	Plan–B	Plan–C
State of hydration	No dehydration	Some dehydration	Severe dehydration
Percentage of body weight loss	<5	5–10	>10
Estimated fluid deficit (mL/kg)	<50	50–100	>100
Goals of management	Replacement of ongoing losses of fluid and electrolytes	Correction of existing deficits of fluid and electrolytes	Urgent replacement of existing deficits of fluid and electrolytes

Fluid therapy	Maintenance (oral)	Rehydration (oral)	Rehydration [intravenous (IV)]
Treatment facility	Home	Health facility	Health facility
Rehydration fluid	Oral rehydration solution (ORS)/homemade solutions	ORS	RL*
Amount of rehydrating fluid	For every loose stool: 10 mL/kg Age up to 2 months—5 teaspoons/purge 2 months to < 2 years → 50–100 mL Age 2–10 years → 100–200 mL Older child: As much as desired Plus Free access to drinking water	75 mL/kg Over 4 hours Plus Non-breastfed infants <6 months—100–200 mL of clean drinking water Older children and adults: Free access to plain water in addition to ORS	IV fluid Infants 30 mL/kg Over 1 hour 70 mL/kg Over 5 hours Age > 1 year 30 mL/kg Over ½ hour 70 mL/kg Over 2½ hours Plus ORS (5 mL/kg/h) start orally as soon as child is able to drink
Monitoring	Watch for vomiting, early signs of dehydration, blood in stools, etc.	Monitor every hour and reassess after 4 hours • If still in plan B,repeat as above • If rehydrated, shift to plan A	Monitor ½ hourly and reassess after 6 hours (infants) 3 hours (older children) • If still in plan C, repeat as above • If rehydrated, shift to plan B/A

*Normal saline (0.9% NaCl) or half strength Darrow's solution may be used if Ringer Lactate (RL) is not available. Severely malnourished children rehydrated slowly over 6–12 hours.

In children who fail on oral rehydration, administration of rehydration fluids either by nasogastric (NG) tube or intravenously (IV) is effective and recommended.

NUTRITIONAL MANAGEMENT

Infants younger than 6 months to continue breastfeeding and for non-breastfed, not to introduce diluted or modified formula.

- Regular oral feeding to be reintroduced no later than 4–6 hours after the onset of rehydration.
- In children with severe acute malnutrition (SAM), food offered during rehydration
  phase.
- Home available fluids can be given such as rice or pulses-based drink (rice water and dal water); vegetable soup; yogurt drink with salt (salted Lassi); lemon drink
- (Shikanji with added salt and less sugar), and coconut water. Plain water can be given in between.
- Elimination diet is usually not indicated
- An extra meal a day with energy rich foods for at least a week or two, after the diarrhea stops or until the child is back on its original weight.

ZINC

- It helps in reducing the duration, severity of diarrhea, and in preventing further episodes of diarrhea for next 3 months.
- Dose: 6 months to 5 years of age: 20 mg/day × 14 days 2–6 months: 10 mg/day × 14 days

PROBIOTICS

- Effective in reducing the duration and intensity of symptoms
- Selected probiotic strains (including Lactobacillus rhamnosus GG, Saccharomyces boulardii, and also L. reuteri DSM 17938) can be considered as an adjunct to ORS.

ANTIEMETICS

- Ondansetron administered either orally or intravenously (0.15 mg/kg/dose, maximum: 8 mg) is effective in reducing vomiting.

INDICATIONS FOR ANTIBIOTICS

Routine use of antibiotics is not recommended for the treatment of acute watery diarrhea. The use of antibiotics may be considered in:

- Infants < 3 months
- Children with underlying chronic conditions or immunodeficiency
- Children with SAM
- Infections with Shigella, enterotoxigenic Escherichia coli (ETEC) (not Shiga- like toxin producing), Vibrio cholerae, and Yersinia enterocolitica
- Invasive bacterial infection.

The routinely used antibiotics are given in Table 2.

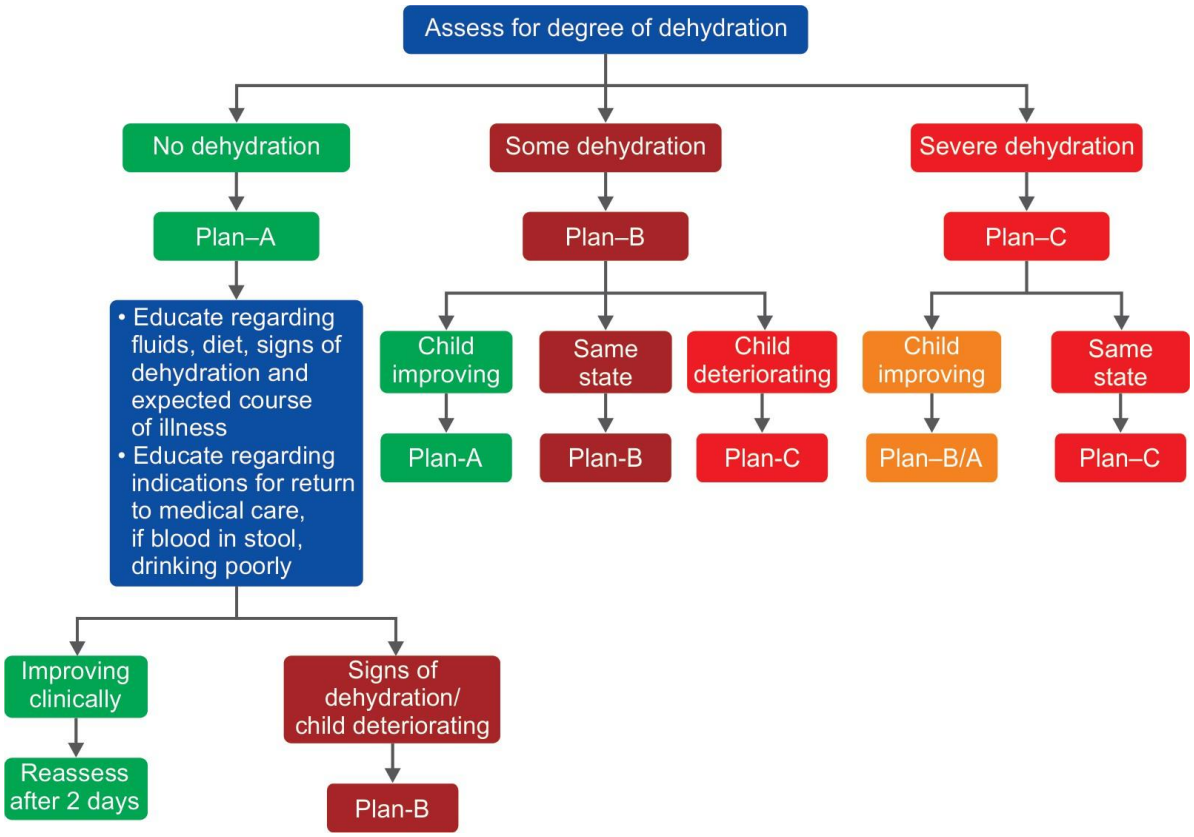
TABLE 2: Antibiotics recommended for the treatment of acute watery diarrhea.

Pathogen	Drug of choice	Alternative
Shigella	Parenteral, IV, IM: Ceftriaxone (50 mg/kg for 2–5 days)	Cefixime PO (8 mg/kg/day); ciprofloxacin PO (20–30 mg/kg/day)
Salmonella (non-typhi) Only in high-risk children	Parenteral ceftriaxone (50–100 mg/kg/day)	Azithromycin PO (10 mg/kg/day); ciprofloxacin PO (20–30 mg/kg/day)
Enterotoxigenic Escherichia coli	Azithromycin PO (10 mg/kg/day) for 3 days	Cefixime (8 mg/kg/day) for 5 days
Vibrio cholerae	Single dose of doxycycline (>2 years) 2–4 mg/kg	Single dose of azithromycin/ ciprofloxacin 20 mg/kg

VACCINATION

Up-to-date immunization, especially for Rotavirus and measles, helps in preventing diarrhea.

Flowchart 1: Approach to management of acute watery diarrhea.



DIARRHEA IN CHILDREN WITH SEVERE ACUTE MALNUTRITION

Assessment and management of dehydration in an SAM child differs from children without malnutrition. Diagnosis is by history:

- Definite history of diarrhea of sudden onset within few hours or days
- Recent change in the child's appearance
- Mother says the eyes have changed to become sunken since the diarrhea started
- Eagerness to drink

SAM child in shock:

- Cold hands with
- Slow capillary refill >3 seconds and
- Weak and fast pulse.

DIARRHEA IN CHILDREN WITH SEVERE ACUTE MALNUTRITION

- Managed in a health facility.
- Oral rehydration. NG tube used for children who drink poorly.
- IV fluids used only for the treatment of shock, due to risk of overhydration and heart failure.

- Oral rehydration 70–100 mL/kg over 12 hours. Start 10 mL/kg/hour in the first
  2 hours. Then alternate hours give starter diet.
- Continue at this rate or a lower rate based on the child's thirst and ongoing stool
  losses.
- Increasing edema is evidence of overhydration.
- Full-strength ORS solution should not be used for oral or NG rehydration. It provides too much sodium and too little potassium.
- When using the new ORS solution containing 75 mmol/L of sodium:
  - Dissolve one ORS packet into 2 L of clean water (to make 2 L instead of 1 L);
  - Add 45 mL of potassium chloride solution (from stock solution containing 100 g KCl/L)
  - Add and dissolve 50 g sucrose
- Rehydration solution for malnourished (ReSoMal) can be used, dilute one sachet in 2 L water. It has high potassium and low sodium.

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BRONCHIOLITIS

INTRODUCTION

Bronchiolitis is an acute inflammatory condition of the bronchioles that is a result of virusinduced injury.

ETIOLOGY

Respiratory syncytial virus (RSV) is the most common viral agent isolated in about 75% (30–70% in Indian studies).

Other viruses: Rhinovirus, parainfluenza, adenovirus, human metapneumovirus, and bocavirus are the other viruses commonly causing the condition. Mycoplasma is more frequently implicated in older children with bronchiolitis.

DIAGNOSIS

- Persistent cough, following a prodrome of coryza lasting 1–3 days, with tachypnea with or without chest recessions and wheeze and/or crackles occurring in a child <2 years of age (usually below 1 year of age, with a peak between 3 and 6 months).
- Associated fever, usually below 39°C, in around 30% cases and poor feeding, vomiting usually after 3–5 days of illness.
- Apnea may be the only presenting feature, particularly below 6 weeks of age.
- The chest may appear hyperexpanded and may be hyper-resonant to percussion. Wheezes and fine crackles may be heard throughout the lungs.

INDICATIONS FOR HOSPITALIZATION

- Persistent tachypnea >60 breaths/minute or respiratory distress in form of grunting, recessions
- Inadequate oral intake, inability to feed, dehydration, and inadequate fluid intake (50–75% of usual volume)
- Oxygen saturation (SpO2) <92% in room air
- Child appears seriously unwell to the healthcare provider
- Skill and confidence of the caregiver to look after the child at home and distance from the hospital

SIGNS OF SEVERE BRONCHIOLITIS

- Apnea, observed/reported
- Marked respiratory distress (severe grunting/chest indrawing/tachypnea >70/minute)
- Central cyanosis, or SpO2 below 90% (age > 6 weeks) or below 92% (age < 6 weeks, or any age with underlying health conditions)

RISK FACTORS FOR SEVERE BRONCHIOLITIS

Predictors of severe bronchiolitis are presented in Table 1.

TABLE 1: Predictors of severe bronchiolitis.

A. Host-related risk factors	B. Environmental risk factors	C. Clinical predictors
• Prematurity, especially <32 weeks of gestation • Low birth weight • Age <6–12 weeks • Chronic lung disease including BPD • Hemodynamically significant congenital heart disease (e.g., moderate-to-severe pulmonary hypertension, cyanotic heart disease, or congenital heart disease that requires medication to control heart failure) • Immunodeficiency • Neuromuscular disorders	• Having older siblings • Passive smoke • Household crowding • Child care attendance • Lower socioeconomic status	• Toxic or ill appearance • Oxygen saturation <95% by pulse oximetry while breathing room air • Respiratory rate 70 breaths per minute • Moderate/severe chest retractions • Atelectasis on chest radiograph

Differential Diagnosis

- Pneumonia: Fever >39°C with persistent focal crackles
- Episodic viral wheeze: Persistent wheeze without crackles, or recurrent episodes with or without a family history of atopy

INVESTIGATIONS

- Is a clinical diagnosis based on age, seasonal occurrence, typical clinical presentation, and physical examination?
- Blood investigations and radiology is routinely not indicated.
- A pulse oximetry reading helps to identify hypoxia and need for admission.
- Investigations in admitted patients to rule out alternate diagnosis such as bacterial pneumonia, congenital heart disease with failure, or sepsis might occasionally be indicated.

- Admitted babies may need an arterial blood gas (ABG) analysis, complete blood count, C-reactive protein (CRP), serum electrolytes, and chest radiography for managing the more serious patients.
- Measurement of lactate dehydrogenase (LDH) concentration in the nasal-wash fluid has been proposed as an objective indicator of bronchiolitis severity (Table 2).
- Identification of viral agents does not affect management in the majority of patients. However, in the hospital setting, to avoid antibiotic abuse and prevent nosocomial transmission may be done by:
- Antigen detection, immunofluorescence, polymerase chain reaction (PCR), and culture of respiratory secretions obtained by nasal wash or nasal aspirate.
- New techniques such as real-time PCR, nested PCR, and multiplex PCR have improved the virologic diagnosis of bronchiolitis immensely.

TABLE 2: Severity of bronchiolitis.

	Mild	**Moderate**	**Severe**
Feeding ability	Normal ability to feed	Appear short of breath during feeding	May be reluctant or unable to feed
Respiratory distress	Little or no respiratory distress	Moderate distress with some chest wall retractions and nasal flaring	*Severe distress with marked chest wall retractions, nasal flaring and grunting *Can have frequent and prolonged apnea
Saturation	Saturation >92%	Saturation <92%, correctable with O_2	Saturation <92%, may or may not be correctable with O_2

MANAGEMENT

- Treatment is focused on symptomatic relief and maintaining hydration and oxygenation.
- Fever should be controlled with paracetamol.
- Nose block should be cleared with saline nasal drops and gentle suctioning.
- Child should be made to lie in a propped up or head end elevated positioning.

- Orogastric tube feeding may be indicated in admitted patients. Intravenous (IV) fluids in children with impending respiratory failure or who do not tolerate orogastric/nasogastric (OG/NG) fluids.
- Suctioning of the upper airway in children with apnea, respiratory secretions, and feeding difficulties due to upper airway secretions
- Supplemental oxygen in children with SpO2 below 90% (>6 weeks) or below 92% (<6 weeks or with underlying health issues)
- Continuous positive airway pressure (CPAP) in babies with impending respiratory failure (limited low-quality evidence)
- High-flow nasal cannula (HFNC) oxygen may have a role as a rescue therapy to reduce proportion of those requiring intensive care
- Drugs with questionable value might reduce need for admission or length of hospital stay, but broad consensus is lacking.
    - Nebulized hypertonic saline: In children hospitalized for >3 days
    - Nebulized adrenaline: 0.1–0.3 mL/kg/dose of 1:1,000 as a potential rescue medication; however inconsistent and short-lived improvement
    - Beta-agonists: Optional single trial; may be continued if there is clinical response (a trial of bronchodilator therapy may be initiated, but should be discontinued if there is no objective improvement)
- No role of:
    - Chest physiotherapy
    - Antibiotics
    - Antivirals
    - Montelukast
    - Ipratropium bromide
    - Systemic or inhaled steroids
    - Steam inhalation
    - RSV polyclonal immunoglobulin/palivizumab (no roll in acute management but useful in prophylaxis)
    - Inhaled furosemide/inhaled interferon alfa-2a/inhaled recombinant human deoxyribonuclease (DNase)
- Interventions which are possibly effective for most severe cases:
    - CPAP
    - Surfactant
    - Heliox
    - Aerosolized ribavirin

CRITERIA FOR DISCHARGE

- Clinically stable
- Taking adequate oral feeds, at least 75% of usual

- Maintaining SpO2 above 90% (>6 weeks) and 92% (<6 weeks or with health issues) in room air
- Ability of the caregiver to look after at home and distance from the hospital, and have understood the "red flag" signs Red flag signs for the caregiver at home:
- Increased work of breathing (e.g., grunting, nasal flaring, and chest retractions)
- Fluid intake <50–75% of normal or no urine for 12 hours
- Apnea or cyanosis
- Exhaustion (i.e., not responding normally to social cues and responds only with prolonged stimulation)

PREVENTION

- Breastfeeding: Three-fold greater risk in non-breastfed infant
- Hand hygiene
- Avoid passive smoking
- Immune prophylaxis:
    - •• Palivizumab: Monoclonal antibody, monthly injections during seasonal epidemics

      Indications: Infants <12 months with prematurity <29 weeks; CLD of prematurity; hemodynamically significant heart disease

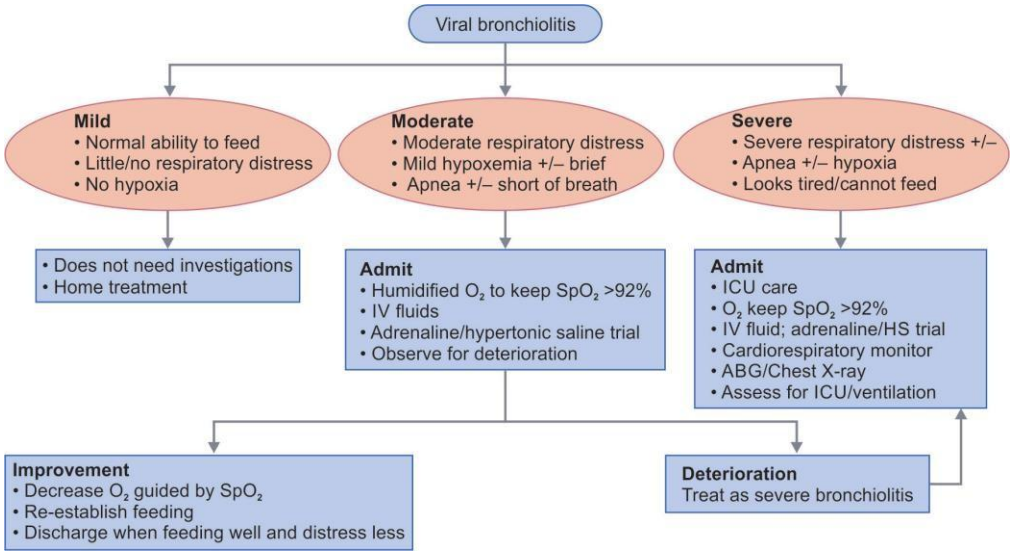
      Palivizumab is administered intramuscularly at a dose of 15 mg/kg monthly (every 30 days) during the RSV season. A maximum of five doses is generally sufficient prophylaxis during one season.
    - •• Nirsevimab: On trial; single dose for 5 months
    - •• Motavizumab, a second-generation mAb, and Numax-YTE, a third-generation mAb—under trial

COMPLICATIONS

- Acute respiratory distress syndrome (ARDS)
- Myocarditis
- Congestive heart failure
- Arrhythmias
- Bronchiolitis obliterans
- Secondary bacterial infection
- Predisposition to childhood asthma

SUMMARY

Flowchart 1: Summary of viral bronchiolitis.



(ABG: arterial blood gas; ICU: intensive care unit; IV: intravenous; SpO2: oxygen saturation)

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PERSISTENT DIARRHEA

INTRODUCTION

Diarrhea is the second most common cause of death in children. There are about 6 billion episodes of diarrhea every year in the world with 2 million deaths. Diarrhea is a condition characterized by a change in the consistency and frequency of stoolscompared to the normal bowel habit of the child. The vast majority of cases of diarrhea subside within 7 days—this is
known as acute diarrhea.

PERSISTENT DIARRHEA (PD)

- An episode of diarrhea of presumed infectious etiology, which starts acutely but lasts for more than 14 days, and excludes chronic or recurrent diarrheal disorders such as celiac disease, tropical sprue, or other congenital, biochemical or metabolic disorders, leading to a deterioration in nutritional status and a substantial risk of death.
- About 60% of PD occurs before 6 months and 90% below 1 year of age. According to WHO, PD accounts for only 10% of diarrheal episodes, but as much as 35% of diarrheal deaths in children below 5 years are due to it.

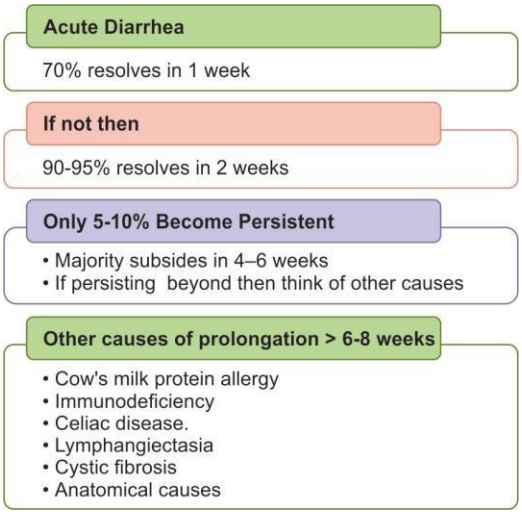
CHRONIC DIARRHEA

Chronic diarrhea has an insidious onset unlike PD, lasts more than 2 weeks and is usually due to non-infectious causes. These children need a complete work up for underlying malabsorption.

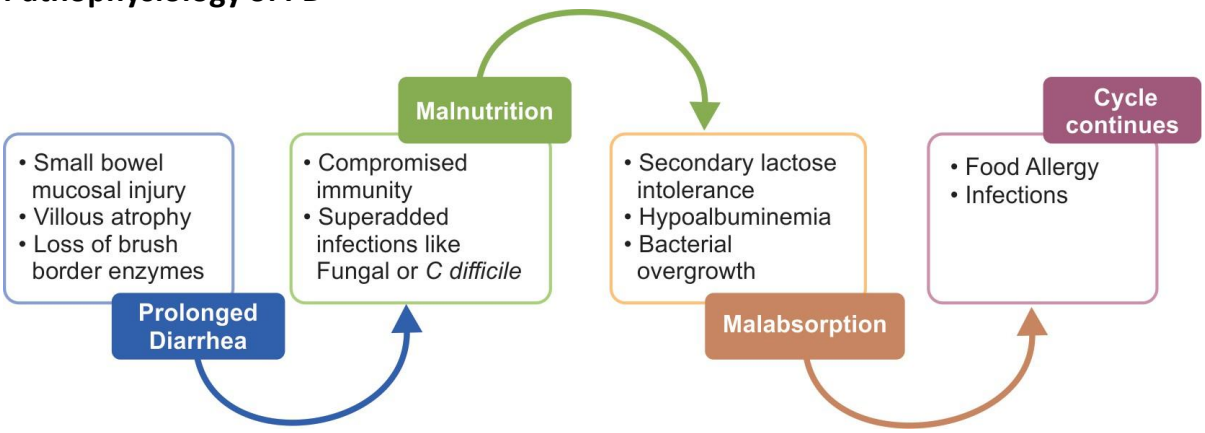
Factors that contribute to PD:

- Secondary lactose intolerance
- Fungal super infection
- Primary malnutrition leading to enteropathy
- Other bacterial infections such as urinary tract infection, otitis media, etc.
- Antibiotic associated diarrhea

NATURAL HISTORY OF DIARRHEA



Pathophysiology of PD



MANAGEMENT OF PD

The common features in all children with PD are small bowel mucosal damage which affects absorption of nutrients, and infections including small intestinal bacterial overgrowth (SIBO).

The cornerstone of management of PD is to break the above chain as mentioned in the pathophysiology.

There are three principles of management:
1. Control of diarrhea and its consequences
2. Treatment of infections, if any
3. Nutritional rehabilitation and correction of malnutrition

STEPS IN MANAGEMENT

- Fluid resuscitation:

•• Low osmolality ORS is effective; IV fluids may be necessary if child has severe dehydration
•• Correction of electrolyte imbalance
•• Correction of hypoglycemia

- Identify infections and treat with antimicrobials or antifungals as appropriate:
  •• A thorough clinical examination for chest infection , otitis media or signs of sepsis
  •• Examination of perineum and oral cavity for superadded fungal infection
  •• CBC, CRP, total protein and albumin, blood sugar, electrolytes, blood and urine cultures, and chest radiograph
  •• Stool routine examination and culture usually have no role in management
  •• Stool for opportunistic infections such as fungal hyphae, cryptosporidium, and assay of Clostridium difficile toxin A and B in appropriate clinical setting are advisable.
  •• Start antibiotics: Quinolones/oral third generation cephalosporins in the presence of gross blood in stools; parenteral ampicillin and aminoglycosides in sepsis, children <3 months of age or associated extra-gut infections, and HIV
  •• Start antifungals flucazole 6 mg/kg/dose for 4–6 weeks if there is oral thrush, or perineum showed fungal infection
- Dietary management (Tables 1 and 2)
- Home-made, culturally acceptable and age—appropriate diet to be started.
- Total calories should be approximately 100 cal/kg/day.
- If child has features of lactose intolerance, then diet should be modified as mentioned below. Lactose intolerance should be considered when child has explosive stools with perianal redness and stool pH<6.5. Keeping child fasting for 24 hours and diarrhea will be reduced significantly which implies towards osmotic diarrhea.

TABLE 1: Diets for PD.

Type A Diet		Type B Diet	
Ingredient	Amount	Ingredient	Amount
Milk	40 ml	Puffed rice	13.5 g
Sugar	2.25 g	Egg white	11 g
Puffed rice powder	12.5 g	Sugar/glucose	3.5 g
Oil	2 g	Oil	3.5 ml
Water	Make it 100 mL	Water	Make it 100 ml
Total calories	96 kcal/100 g	Total calories	92.2 Kcal/100 g
Protein	10%	Protein	9.5%
Fat	33%		33 %

Puffed rice is ground and mixed with sugar and oil. Boiled water is then added to make a thick gruel. This has a shelf life of 3 hours.		Egg white is added to the mixture of weighed rice, sugar and oil. Boiled water is added to make a thick gruel weighing 100 g.	

TABLE 2: Nutritional Supplements in PD.

Supplement	Dose	Duration
Multivitamins	Twice RDA	2–4 weeks
Iron	After cessation of diarrhea—3 mg/kg	
Folic acid	1 mg/day	2 weeks
Vitamin A	<6 months 50,000 IU; 6–12 months one lakh IU; >12 months 2 lakh IU	One Stat Dose
Potassium	5-6 mEq/kg/day	2 weeks
Magnesium Sulphate	0.2 ml/kg/day IM	2–3 days
Elemental zinc	10 mg<6 months; 20 mg> 6 months OD	2 weeks
Vitamin D	200–400 IU/d	

TREATMENT OF LACTOSE INTOLERANCE

- Always start with Diet A (low lactose diet) for 7 days. This is based on the fact that secondary lactose intolerance is common in children with PD and malnutrition. To reduce lactose concentration in animal milk, it should be mixed with cereals, but not diluted with water as that reduces the caloric content.
- If there is no response after a week, then start Diet B—milk (lactose) free and provides carbohydrates as a mixture of cereals and glucose. Milk protein is replaced by chicken, egg or protein hydrolysate. The starch content is reduced and partially substituted by glucose.
- A small number of children with PD who do not respond to diet A/B is given diet C for 7 days. It contains only glucose and a protein source as egg white or chicken or commercially available protein hydrolysates. Energy density is increased by adding oil to the diet.

- If there is no response after a week, then elemental diet such as hydrolyzed or amino acid formula may be started. If there is still no response after a week, child may require total parenteral nutrition, and hence he may be referred to a pediatric gastroenterologist.

GREEN BANANA DIET

- Green (unripe) banana diet is gaining acceptance for treatment of PD. The amylase resistant starch present in this is not digested in small intestine and reaches colon. Colonic bacteria ferment this to short chain fatty acids which have trophic effects for colon and increase the absorption of salt and water.
- A dose of parenteral Vitamin K should be given at admission. After the infant has begun to improve and is gaining weight, 3 mg/kg/day of iron is added.
- There are no published data to recommend the use of empiric antibiotics directed against enteric pathogens, probiotics, racecadotril, steroids or drugs that alter intestinal motility, including loperamide, codeine, and paregoric.
- The failure to respond to treatment may be due to severe systemic infections, unusual enteropathogens, sucrase/isomaltase deficiency, and severe glucose malabsorption.
- Strategies to prevent PD include promotion of exclusive breastfeeding, safe complementary feeding practices, promotion of safe drinking water, low osmolality ORS, zinc supplementation, avoiding unnecessary antibiotics, and continued feeding during diarrhea.

CONCERNS IN THE MANAGEMENT OF PD

- The management of infants below 6 months of age continues to be a problem as they cannot be given most food recommended for older children. They may need extensively hydrolysed 100% bovine casein infant formulas and elemental amino acid formulas which are very expensive and are not freely available in our country.
- Many hospitals are not able to manage children with PD who do not respond to the usual treatments described above. Hence, IAP should take up the initiative to set up regional centres which can manage such refractory cases.
- Parent education and uniform protocols of management should be followed by all pediatricians in India.

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EVALUATION AND MANAGEMENT OF STATUS EPILEPTICUS IN CHILDREN

INTRODUCTION

Status epilepticus (SE) is the most common childhood neurological emergency. Practically, SE is defined as any child presenting convulsing to a healthcare facility or having repeated seizures without regaining of consciousness in between. The International League Against Epilepsy (ILAE) defines SE in terms of time points (t1 and t2) (Table 1).

TABLE 1: Status epilepticus (SE) in terms of time points (t1 and t2).

Type of SE	Time beyond which if seizures persist, patient is considered in SE (t1)	Time after which persistent seizures have long-term consequences (t2)
Generalized convulsive SE	5 minutes	30 minutes
Focal status with impaired consciousness	10 minutes	>60 minutes

ETIOLOGY

Etiology may be known or unknown:

- Known (symptomatic):

•• Acute (stroke, toxicity, derangements in serum electrolytes and blood glucose, trauma, hypoxia, febrile seizures, neuroinfections, and inborn errors of metabolism)

•• Remote (brain scars due to above causes, genetic, brain malformations, etc.)

•• Progressive (neurodegenerative disorders and tumors)

•• Known cases of epilepsy: Poor drug compliance or by nature drug-resistant epilepsies such as Lennox–Gastaut syndrome and Dravet syndrome

- Unknown cause: Entities like new-onset refractory status epilepticus (NORSE), a subset of which is febrile infection-related epilepsy syndrome (FIRES)

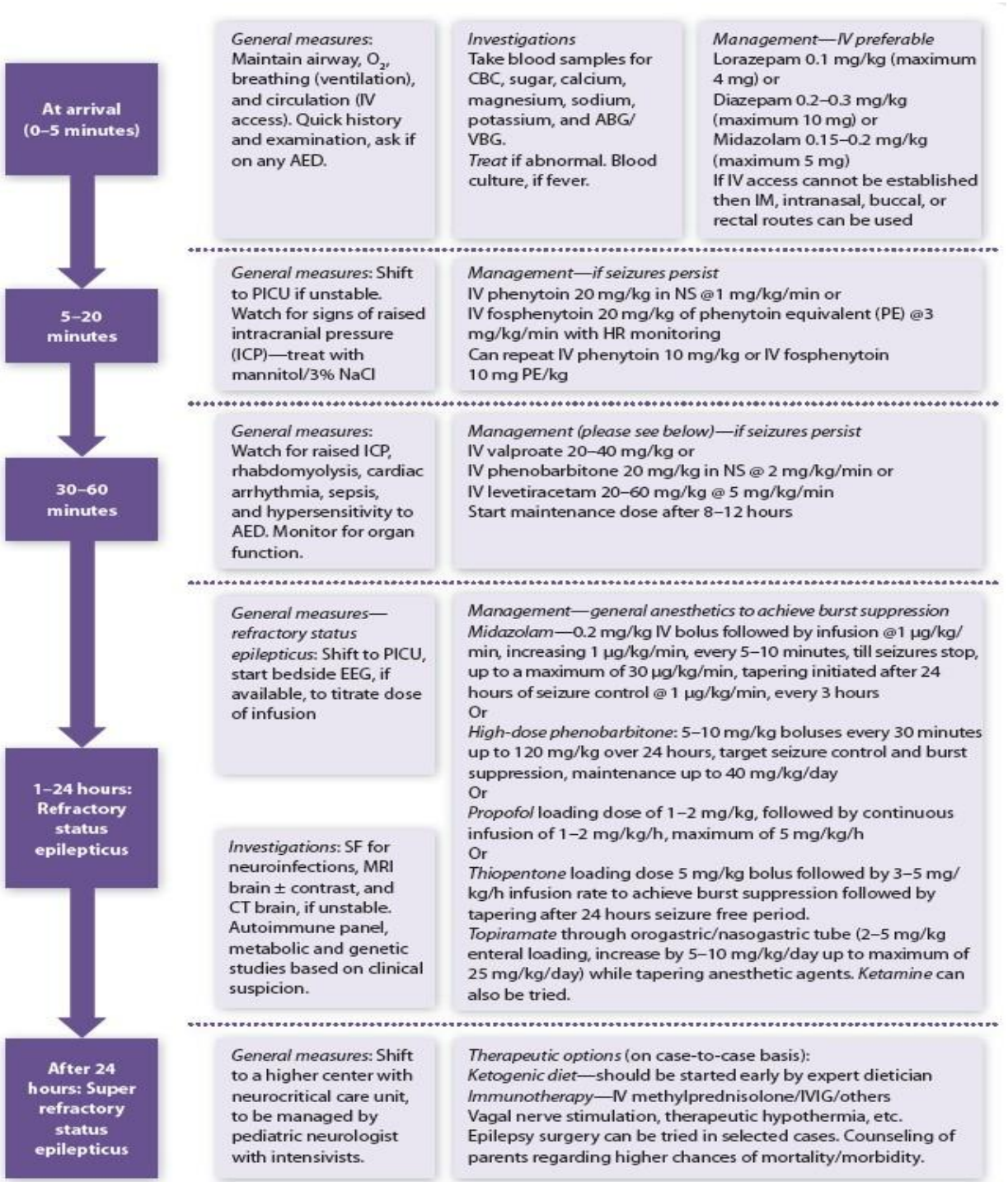
MANAGEMENT

Status epilepticus is a life-threatening emergency. To improve outcomes, each unit should have a fixed protocol and team members should be familiar with their roles. Diagnosis and management should proceed together. A quick focused history and examination help to search for etiology, which helps in streamlining diagnostic work- up.

PREHOSPITAL MANAGEMENT (HOME OR CLINIC)

1. The child should be put in *recovery position* (to prevent aspiration).
2. *Rescue medication*: Any of the following can be kept handy in the clinic:
   - *Midazolam* (buccal/nasal)—*2 puffs/5 kg weight* (0.1–0.2 mg/kg/dose) or
   - *Lorazepam* (intramuscular or intranasal)—0.1–0.2 mg/ kg/dose or
   - *Diazepam* (intramuscular or rectal)—0.5 mg/kg/dose
   - *Blood sugar* can be checked and glucose started, if low
3. Airway, breathing, and circulation should be maintained.
4. Shift the child to a hospital, preferably with oxygen in an ambulance.

IN-HOSPITAL MANAGEMENT



(ABG: arterial blood gas; AED: antiepileptic drug; CBC: complete blood count; EEG; electroencephalogram; IV: intravenous; IVIG: intravenous immunoglobulin; NS: normal saline; PICU: pediatric intensive care unit; VBG: venous blood gas)

Special Points

- All caregivers of children with seizures should be taught first aid including recovery position and use of rescue medications.
- These three drugs are not preferred in a particular sequence. Avoid phenobarbitone if facility for mechanical ventilation is not available; avoid valproate if suspected inborn error of metabolism or liver dysfunction (levetiracetam is the preferred drug in such cases).
- In known cases of epilepsy with breakthrough seizures (on phenytoin ≤6 mg/kg/day, phenobarbitone ≤5 mg/kg/day, valproate ≤30 mg/kg/day or levetiracetam ≤30 mg/kg/day), give half the maintenance dose. For larger doses avoid loading and give the maintenance dose.

Outcome

Mortality in acute phase is seen in 10–20 %. Around 15–56% show long-term cognitive and
motor disability.

Conclusion

A structured and systematic approach should be followed for treatment of SE, with the role of
individual team members well-specified beforehand, as .... Time is Brain!

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UNDER 5 WHEEZE

What is a Wheeze?

High pitched, musical, whistling sound, monophonic, or polyphonic, that occurs when intrathoracic medium and small airways are narrowed and vibrate due to increased resistance to the movement of air.

Wheezing occurs in large proportion of children under 5 years of age. It is commonly
associated with viral respiratory tract infections.

First Time Wheezing

When wheezing occurs for the first time in an infant it is usually due to acute viral bronchiolitis. The diagnosis is mainly clinical. X-ray chest may show hyperinflation with air trapping or sometimes it may be normal.

Management of first time wheezer (usually acute bronchiolitis):

- To maintain oxygen saturation and adequate hydration. Oxygen by nasal cannula or hood is given whenever saturation falls below 92%.
- Bronchodilators like salbutamol may be given through nebulizer as a trial initially to assess response in infants who wheeze for the first time, triggered by viral respiratory tract infections. However, in acute bronchiolitis per se it is not effective and not indicated.

Recurrent Wheezing Details

of the Symptoms

In a child with recurrent wheezing, detailed history and examination is of paramount importance. The following questions need to be asked to ascertain a diagnosis of asthma.

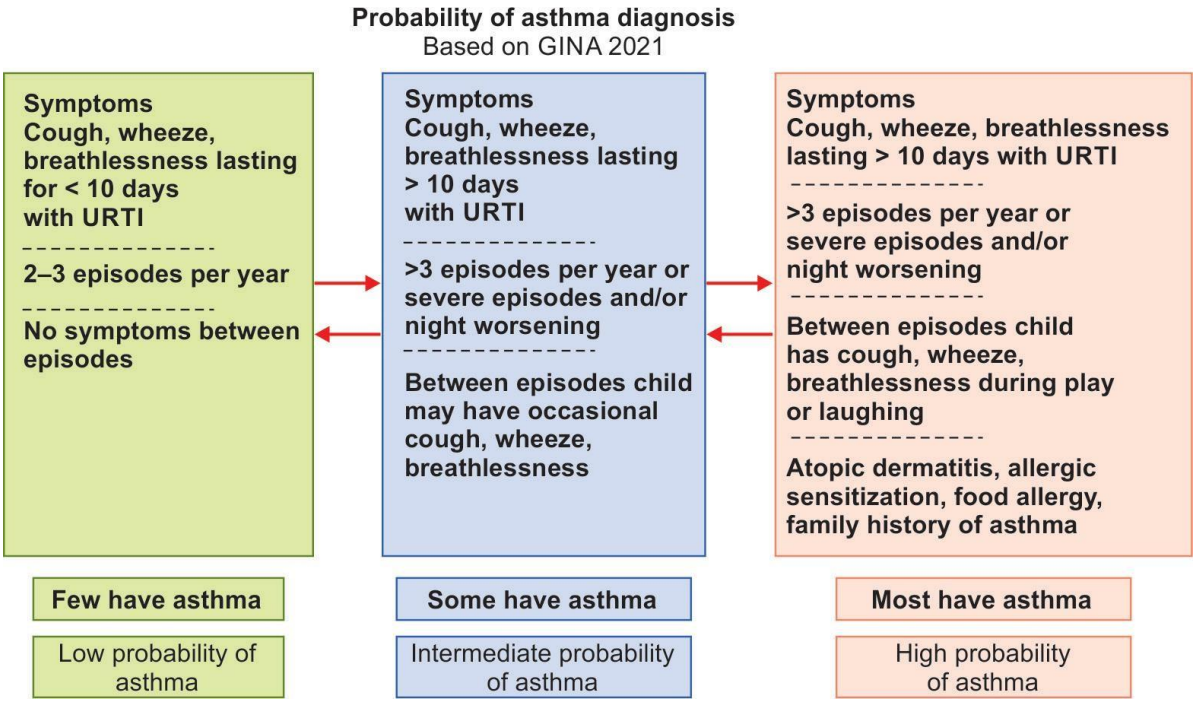
- Onset of cough/wheezing—insidious/sudden
- Duration of cough—how many days
- Timing of cough/wheezing—daytime/nocturnal
- Type of cough—dry/wet
- Frequency of cough/wheezing—number of days per month
- Perennial or seasonal
- Symptoms—variable or persistent, whether associated with fever?
- Interval symptoms—symptoms in between the episodes
- Activity limitation—school absenteeism and restricted play
- Triggers—viral infections, smoke, exposure to dust, mold, cat, grass/tree pollen, food, in response to activity/exercise, laughing, crying, etc.

- History of allergy (atopic dermatitis and allergic rhinitis)—in the child/asthma in parents

Documenting airflow limitation: The presence of wheeze one or more times in the past, documented by a physician

Documenting reversibility: Response to beta2-agonist inhalation or documented response to a trial of inhaled corticosteroid (ICS)

The Global Initiative for Asthma (GINA) guideline proposes grouping of recurrently wheezing patients based on probability of asthma.

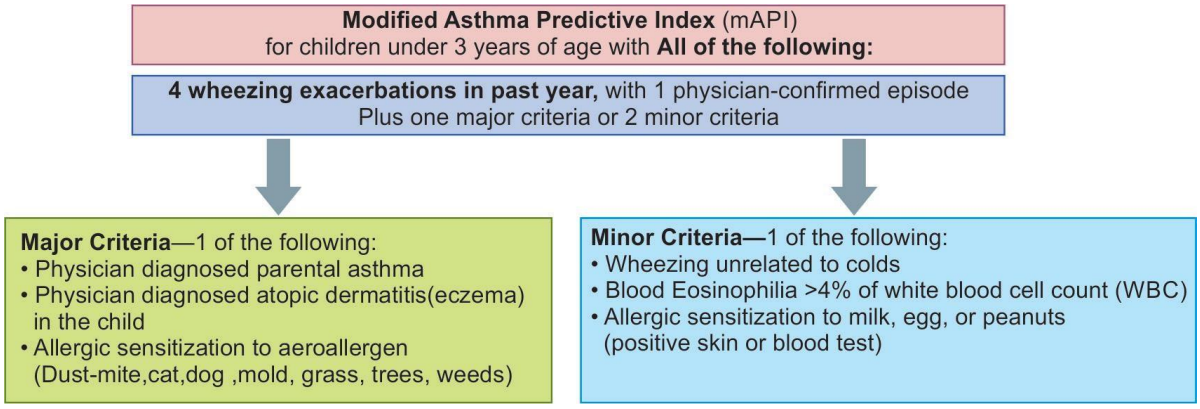


Modified Asthma Predictive Index

Modified Asthma Predictive Index is an objective tool used to predict the occurrence of asthma in preschool children.

It is used in children who have 4 or more wheezing episodes in a year. It is said to be positive when at least one major or two minor criteria are present (Flowchart 1.)

Flowchart 1



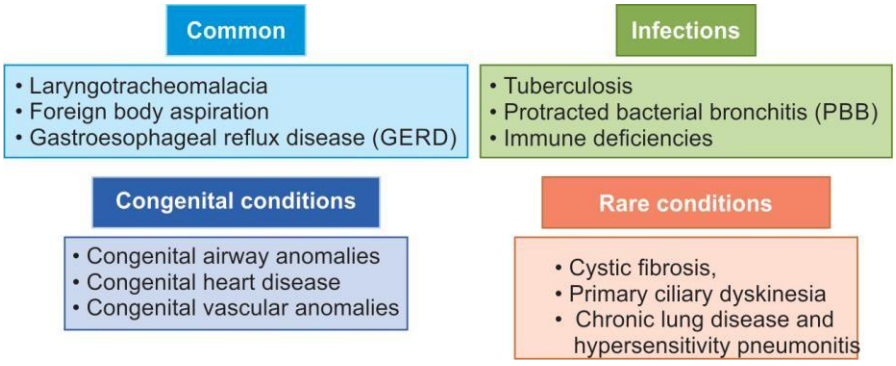
The mAPI has moderate positive predictive value but good negative predictive value (95%) owever, before confirming asthma diagnosis in any child, particularly in this age group it is important to exclude alternative causes of cough and wheezing.

Clinical features where alternative conditions or asthma mimics are more likely are as following:

- Onset of wheezing in the neonatal period
- Wheezing in infants below 6 months of age
- Nonvariable fixed or persistent signs or symptoms
- Unilateral signs and localized signs
- Wet productive cough, presence of high fever
- Failure to thrive, steatorrhea, and clubbing
- Multiple system involvement and persistent hypoxia
- Poor response to bronchodilators
- Wheezing with dependent edema and/or cardiac murmurs
- Wheezing accompanied by stridor

Differential Diagnosis in Wheezers below 5 Years of Age—Asthma Mimics

All the conditions mentioned in the figure should be excluded by history, examination, and, if necessary, by appropriate investigations.



Investigations in Recurrent Wheezers

If the history is suggestive of typical viral-induced wheezing or that of asthma no investigations are necessary. However, in ambiguous cases with suspicion of some alternative condition certain investigations may be required.

- Complete Hemogram

Eosinophilia may be detected. However, its absence does not rule out possibility of asthma.

- X-ray Chest

To rule out alternative conditions in differential diagnosis.

- Allergy Tests

These may be useful to document allergen sensitization in those who report allergic symptoms on exposure to a particular allergen.

Investigations in Recurrent Wheezers

- Fraction of Nitric Oxide

Fraction of nitric oxide (FeNO) in exhaled air may be used to identify those with eosinophilic inflammation to predict response to corticosteroids.

- Spirometry

Spirometry usually cannot be performed reliably by children under the age of 5 years; other techniques such as forced oscillation technique (FOT) and impulse oscillometry (IOS) are now available at a few centers which can be used in doubtful cases.

Treatment of Recurrent Wheezers

Inhalation treatment is the mainstay in the treatment of wheezing. Inhaled short- acting betaagonist (SABA), usually salbutamol, is used as initial reliever treatment until symptoms subside.

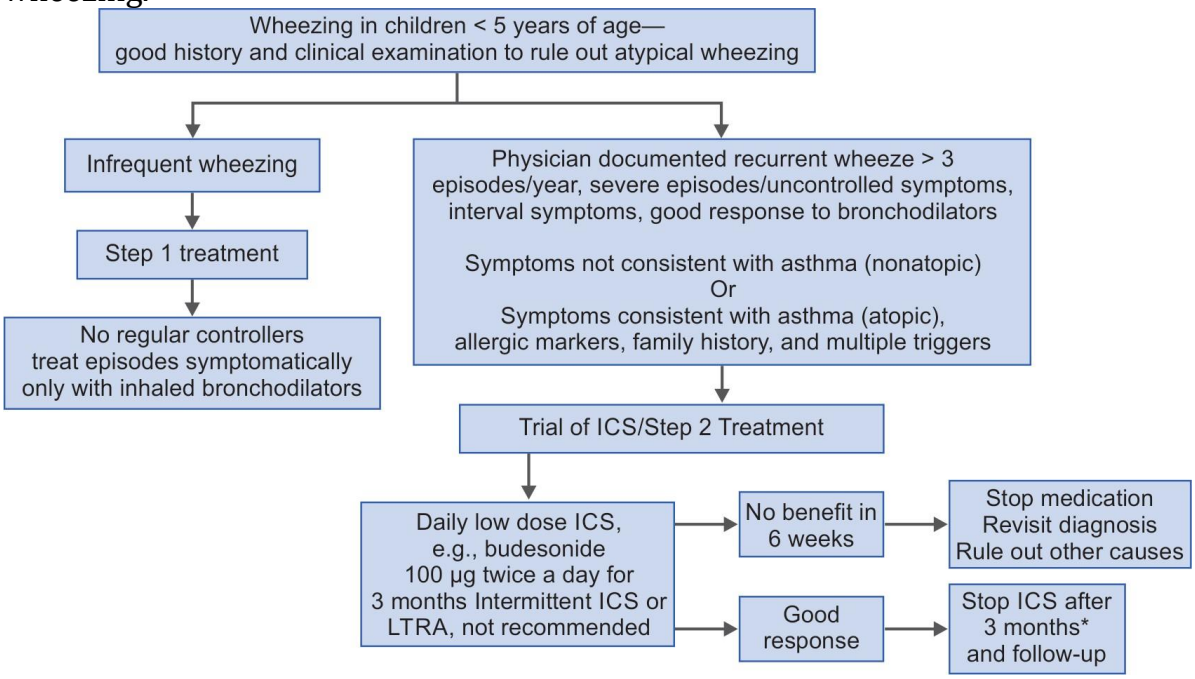
Choice of Inhaler Device

- Till 4 years: Metered-dose inhaler (MDI) + spacer + mask or nebulizer in hospital setting
- Above 4 years: MDI + spacer with mouth piece. Mask should be removed as soon as child can breathe through mouthpiece.

Treatment Plan

In children under 5 years of age with recurrent wheezing, the following treatment plan is recommended (Flowchart 2).

Flowchart 2: Treatment plan for children under 5 years of age with recurrent wheezing.



*In children with severe episode/life-threatening exacerbation in the past, it is important to continue inhaled corticosteroid (ICS) for a longer period at a minimum dose that can prevent further exacerbation with periodic monitoring until the child outgrows the symptoms. If no risk factors for exacerbation are present, ICS should be stopped and child followed up for symptom recurrence. ICS can be restarted if symptoms recur.

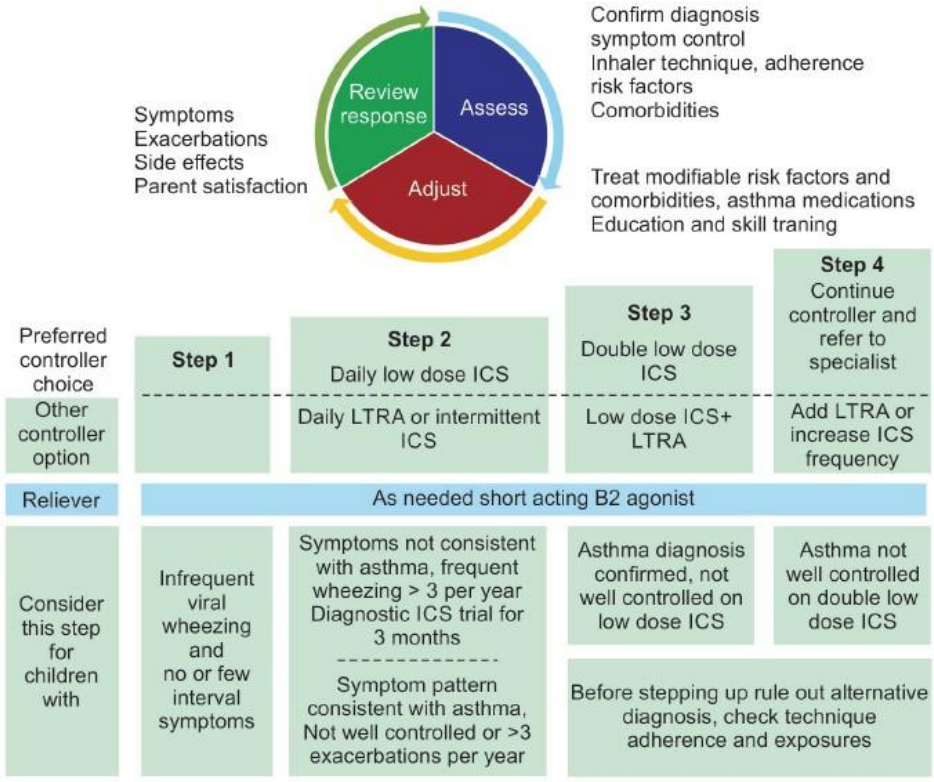
Step 3 and Step 4 Treatment

In some children, if symptoms are not well controlled after 3 months of low dose budesonide and if diagnosis of asthma is certain then higher step of treatment may be necessary. But before proceeding to higher dose of ICS, the following should be checked:

- Adherence with inhaled medications
- Technique of device use
- Comorbidity
- Ongoing exposure to allergen

If even after correcting all of the above, symptoms persist, then treatment is stepped up to step 3. ICS dose is doubled, e.g., 200 µg of budesonide twice a day for next 3 months.

If child is not well-controlled on this double low dose regime after removing all modifiable risk factors, treatment can be stepped up to step 4. However, such a child should be referred to asthma specialist for further management.



Stepwise management in children 5 years and younger—GINA 2021. (GINA: Global Initiative for Asthma; ICS: inhaled corticosteroid; LTRA: leukotriene receptor antagonist)

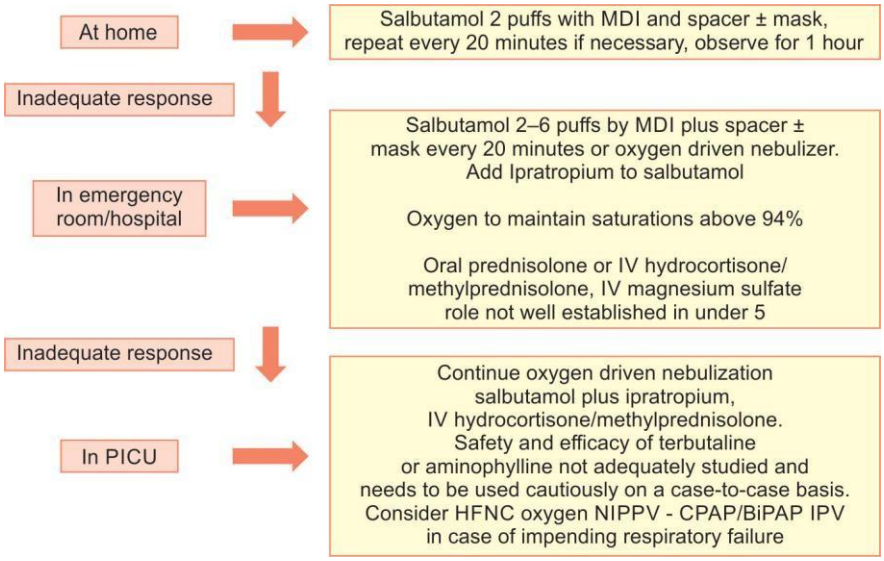
Treatment of Acute Exacerbation (Flare up or Asthma attack)

Exacerbations (flare-ups) of asthma are episodes characterized by progressive increase in symptoms of shortness of breath, cough, wheezing or chest tightness, and progressive decrease in lung function. They are classified as mild, moderate, or severe attack. A scoring system is used to classify and monitor severity of attack.

Doses

- For nebulization—salbutamol 0.15 mg/kg/dose, ipratropium-250-500 µg/dose
- Prednisolone-1 mg/kg/day for 3-5 days
- Injection hydrocortisone 2-5 mg/kg intravenous (IV) 6 hourly or injection methyl-prednisolone 1-2 mg/kg/6 hourly
- IV magnesium sulfate 25-50 mg/kg/dose as slow infusion over 30 minutes

Management of Acute exacerbations



Summary of management of flare-up. (BiPAP: bilevel positive airway pressure; CPAP: continuous positive airway pressure; HFNC: high-flow nasal cannula; IV: intravenous; MDI: metered-dose inhaler; NIPPV: noninvasive positive-pressure ventilation; PICU: pediatric intensive care unit)

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FEBRILE SEIZURES

DEFINITIONS

Febrile Seizure

Seizure accompanied by fever without central nervous system infection, metabolic or electrolyte disturbances, or a history of afebrile seizure or any acute neurological insult/head trauma in children aged 6 months to 6 years. Few guidelines include younger children up to 3 months [National Institutes of Health (NIH)] and even 1 month [International League against Epilepsy (ILAE)] after ruling out causes of provoked seizures. Fever can occur anytime during or after a seizure and the majority of febrile seizures (FSs) occur within 24 hours of fever onset.

Simple Febrile Seizures

Fever with isolated, generalized tonic clonic seizures, which last <15 minutes, and do not recur within 24 hours.

Complex Febrile Seizures

Fever with seizures with any one of the following features: focal and/or prolonged for >15 minutes and/or recur within 24 hours and/or have incomplete recovery within 1 hour.

Febrile Status Epilepticus

Febrile seizure lasting for 30 minutes or more and/or series of seizures without full recovery in between that.

Afebrile Febrile Seizure

Seizures in an acute infectious illness (particularly gastroenteritis) without documented fever and features consistent with simple FS.

Febrile Seizure Plus

Febrile seizures that continue past the usual age where they are expected to resolve (6 years) and/or accompanied by afebrile generalized or focal seizures.

Genetic Epilepsy with Febrile Seizure Plus

Febrile seizures plus with a family history of FSs, FSs plus, or afebrile generalized or focal seizures.

Pathophysiology of Febrile Seizures

Several hypotheses have been proposed to explain causation of FSs. It is believed that in genetically predisposed children, increase in brain temperature leads to

perturbation of temperature sensitive ion channels that in turn causes increased neuronal firing. Moreover, interleukin-1β acts as both a pyrogen and seizure provocator, acting at glutamate pathway. Interleukin-1β is also an NMDA agonist. Last, but not the least, hyperthermia-induced brain alkalosis is also believed to result in neuronal excitability.

Diagnosis of Febrile Seizure

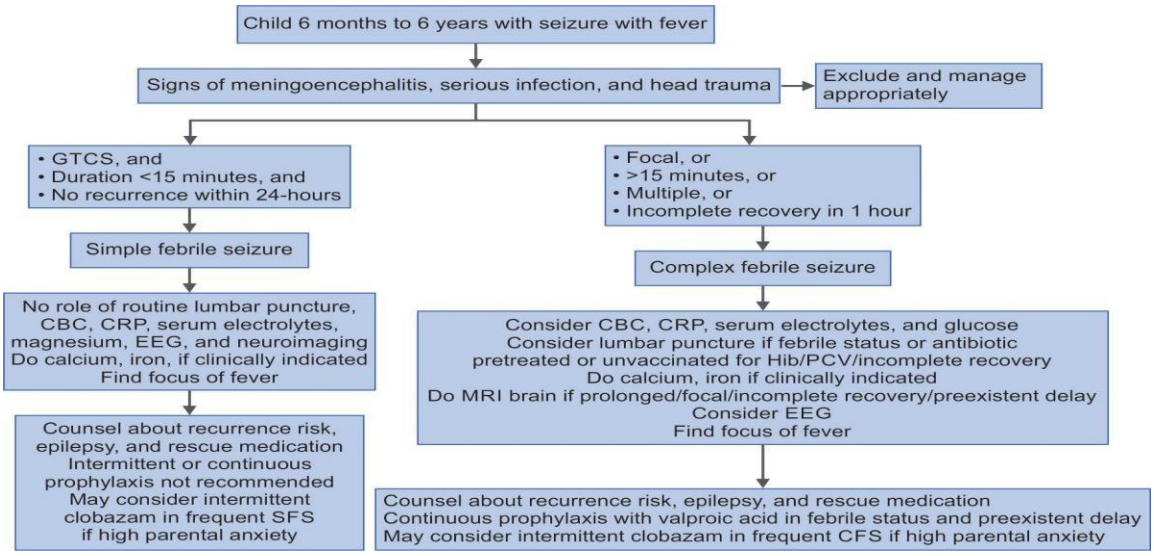
TABLE 1: Diagnosis of febrile seizure (FS).

History	Clinical examination	Red flag signs
☑ Nature and duration of the convulsions and postictal phase ☑ Recent fever/ear discharge/dysuria ☑ Recent antibiotic therapy/ antipyretics/rescue anticonvulsants Immunization history ☑ Past history of previous episodesof FS, a diagnosis of epilepsy, andother neurologic conditions and diseases ☑ Family history of FS, epilepsy, orneurologic diseases ☑ History of neonatal intensive care unit stay or developmental delay,if any	☑ Vitals ☑ Obvious focus of infection ☑ Anthropometry ☑ Features of raised intracranial pressure ☑ Features of meningitis like bulging fontanelle, neck retraction, or meningeal signs ☑ Neurocutane ousmarkers ☑ Dysmorphism ☑ Focal neurological signs ☑ Meningeal signs	☑ Focal neurological signs ☑ Persistent altered sensoriumafter 1 hour of seizure ☑ Features of raised intracranial pressure such as headache, vomiting, papilledema, brisk deep tendon reflexes, Cushing's triad of bradycardia, irregular respiration, and hypertension ☑ Features of meningoencephalitis/non-blanching rash in an unwellchild ☑ Features of sepsis/shock/ respiratory distress

Diagnosis of febrile seizure is depicted in Table 1.

Evaluation of a Child with First Episode of Febrile Seizure

Flowchart 1: Algorithm for evaluation and management of febrile seizure.



(CBC: complete blood count; CFS: complex febrile seizure; CRP: C-reactive protein; EEG: electroencephalography; GTCS: generalized tonic-clonic seizure; Hib: Haemophilus influenzae type B; PCV: pneumococcal conjugate vaccine)

Evaluation of a Child with First Episode Febrile Seizure

TABLE 2: Evaluation of a child with first episode febrile seizure.

	Simple febrile seizure (SFS)	Complex febrile seizure (CFS)/ febrile status epilepticus (FSE)
Basic history, examination,and red flags **(Table 1)**	Yes	Yes
Lumbar puncture	If features of meningitis; children <12 months of age who have not received the Hib or pneumococcalvaccines, children pretreated with antibiotics or children with severe protein energy malnutrition	Consider in children who remainobtunded after 1 hour of seizure

Neuroimaging	Neuroimaging is routinely not recommended. Consider neuroimaging prior to lumbar puncture in children with focalneurological deficits or clinicalsymptoms and signs of raised intracranial pressure	MRI brain should be preferably considered within 72 hours for features of viral encephalitis, acute disseminated encephalomyelitis, intracranial space-occupying lesions, cortical malformations, andfor hippocampal abnormalities Also do if preexistent developmental delay
Electroencephalography(EEG)	No	Yes, prognostic significance ofthe abnormalities to predict future epilepsy is unclear. Perform preferably after 48 hours and within 1 week of febrile seizure
Complete blood countwith CRP	Not routinely, but may be considered to find cause of fever	Yes
Serum electrolytes	No	Yes
Serum calcium	If <1 year	Yes
Serum magnesium	No	Yes
Blood sugar	No	Yes
Serum iron	If clinical pallor	
Urine analysis	If <18 months or if clinical features suggestive of urinary tract infection(dysuria, frequency, and urgency), if >18 months of age	
Genetic testing for Dravet syndrome (SCN1A, SCN1B, GABRG2, and SCN2A)	Recurrent febrile status epilepticus, onset of prolonged hemiconvulsive seizures below 1 year age, vaccine- associated encephalopathy, or GEFS+	

(CRP: C-reactive protein; GEFS+: genetic epilepsy with febrile seizure plus; Hib: Haemophilus influenza type B)

Acute Management during Seizure

- The drug of choice for rescue management at home is intranasal midazolam (0.2 mg/kg; maximum: 5 mg). Other effective drugs are intramuscular/buccal midazolam, buccal lorazepam, and per rectal diazepam. Maximum two doses, 5 minutes apart.
- Management of febrile status epilepticus at hospital is similar to management of convulsive status epilepticus.
- Stabilize with ABCDE approach (airway, breathing, circulation, disability, and exposure/ examination).
- If diagnosed with Dravet syndrome, FS+, GEFS+, sodium channel blockers (phenytoin) may be avoided.
- In young children, in case of clinical suspicion of meningitis and febrile status start thirdgeneration cephalosporin till lumbar puncture results.

Points to Ponder before Institution of Prophylactic Therapy

- Children with FSs are prone to recurrence.
- Slightly increased risk of epilepsy in this population is the result of genetic predisposition.
- No difference in learning has been identified in children with simple FSs, except in those children who had neurologic abnormalities before their first seizure.
- There is a negligible risk of a child dying during a simple FS due to injury, aspiration, or cardiac arrhythmia.
- Successful treatment of simple FSs can neither prevent later development of epilepsy nor simple FSs cause any structural damage to the brain.
- Possible adverse effects of prophylactic therapy include rare fatal hepatotoxicity (especially in children younger than 2 years who are also at greatest risk of FSs), thrombocytopenia, weight loss and gain, gastrointestinal disturbances, and pancreatitis with valproic acid and hyperactivity, irritability, lethargy, sleep disturbances, and hypersensitivity reactions with phenobarbital; lethargy, drowsiness, and ataxia for intermittent diazepam as well as the risk of masking an evolving central nervous system infection.
- Antipyretics and tepid sponging, if properly done, may improve the comfort of the child, they do not prevent fever or FSs, e.g., paracetamol 15 mg/kg/dose SOS up to 6 hourly.

Prophylactic Regimens

- Neither continuous nor intermittent anticonvulsant therapy is recommended for children with one or more simple FSs as there is preponderance of harm over benefit with therapy.
- Intermittent prophylaxis with oral benzodiazepine may be considered among children with frequent recurrent simple FSs with parental anxiety and

residence far from medical facilities or complex FS who have not been started on continuous prophylaxis.

- Continuous prophylaxis with anticonvulsants may be considered among children with febrile status epilepticus, FSs in children with neurodevelopmental delay like cerebral palsy, global developmental delay or autism spectrum disorder, frequent complex FSs, and children with FS+/GEFS+ with afebrile seizures.

Prophylactic Drugs

The drug of choice for intermittent prophylaxis is clobazam (0.5–1 mg/kg/day in two divided doses for 3 days without tapering; maximum dose 20 mg/day).

Drug of choice for continuous prophylaxis is sodium valproate (20–40 mg/kg/day), others are phenobarbital (3–5 mg/kg/day); primidone (15–20 mg/kg/day). Carbamazepine and phenytoin are ineffective.

Duration

The anticonvulsants should be considered for 2 years seizure freedom period or guided individually based on primary syndrome (GEFS+/Dravet syndrome).

TABLE 3: Future prognosis.

Future risk of epilepsy	Recurrence of febrile seizures
Children with simple febrile seizures have 1% risk ofdeveloping epilepsy by the age of 7 years (same as the general population) and 2.4% by 25 years of age	32% children have recurrent seizures17% have one recurrence 9% have two recurrences 6% have three or more recurrences
Children who have had multiple simple febrile seizures are younger than 12 months at the time oftheir first febrile seizure and have a family history ofepilepsy are at higher risk	Recurrence rate within 1 year and 2-year of firstfebrile seizure is 75% and 90%, respectively
Risk of epilepsy after complex febrile seizuresdepends on number of complex features: ☑ With 1 complex feature: 6–8% ☑ With 2 complex features: 17–22% ☑ With 3 and more features: 49%	Risk factors for recurrent febrile seizures ☑ Early onset <18 months of age ☑ Family history of febrile seizures or epilepsy ina first-degree relative ☑ Low grade fever associated with seizure onset(<39°C) ☑ Short duration of fever before the seizure(<1 hour) ☑ Complex febrile seizure ☑ Attendance at a day care nursery—presumedincreased viral exposure
Risk factors for developing subsequent epilepsyinclude: family history of epilepsy, any neuro- developmental problem, prolonged or focal febrile seizures, and febrile status epilepticus	

Family History of Febrile Seizure

- One-third of children with FS have a positive family history.
- Prevalence risk increase to one in five if one sibling affected and one in three if both parents and sibling have been affected.
- Concordance rate of FS are higher in monozygotic (35–69%) than dizygotic twins (14–20%).

Further Reading

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ATYPICAL BACTERIAL PNEUMONIA

INTRODUCTION

Community-acquired pneumonia (CAP) or infection of the lungs is a common cause of morbidity and mortality in children. In India, the typical organisms causing pneumonia are viruses [respiratory syncytial virus (RSV), human metapneumovirus (hMPV), influenza, and parainfluenza) and bacteria (gram-negative bacilli, Staphylococcus aureus, Streptococcus pneumoniae, and others). Pneumonia caused by atypical organisms (loosely referred to as "atypical pneumonia") may be caused by Mycoplasma pneumoniae, Chlamydophila pneumoniae, and Legionella pneumophila.

EPIDEMIOLOGY

- In general, M. pneumoniae and C. pneumoniae pneumonia are more common in children aged >3 years, Chlamydia trachomatis pneumonia is more frequent in infants, and L. pneumophila pneumonia is very rare in children aged <19 years.
- As per a recent study from India, M. pneumoniae and C. pneumoniae serology was positive in 4.3% and 1.1% of CAP in children, respectively. Polymerase chain reaction (PCR)-based analyses of pneumonia etiology report prevalence <1%. L. pneumophila is relatively rare in children, accounting for <0.01% of pneumonia cases.
- M. pneumoniae and C. pneumoniae are droplet infections caused by contact with an infected person. L. pneumophila spreads via aerosolization from humidifiers and hot water heaters. Human spread is not common.

Clinical Features

- Pneumonia and tracheobronchitis are the main manifestations of M. pneumoniae infection.
- Children present with fever, malaise, sore throat, followed by cough that can last for 2–3 weeks.
- Extrapulmonary disease can affect the skin, central nervous system (CNS), blood, heart, gastrointestinal tract, and joints.
- Although, M. pneumoniae infection is self-limiting, it can lead to complicated pneumonia, parapneumonic effusions, necrotizing pneumonia, and bronchiolitis obliterans.
- Radiological findings are consistent with bronchopneumonia involving the perihilar areas and lower lobes with hilar lymphadenopathy. The degree of consolidation in chest X-ray may be more than that expected for the severity of clinical manifestations.
- C. pneumoniae and M. pneumoniae have similar clinical manifestations. Pneumonia is usually unilateral and involves the lower lobes.

- Legionella presents with high-grade fever and productive cough, chest pain, and quickly progresses to alveolar disease with cavitation. It causes more extensive extrapulmonary organ dysfunctions such as dyselectrolytemia and renal failure, liver failure, and rhabdomyolysis.

Diagnosis

- PCR from an appropriate respiratory specimen is the best way of diagnosis. It should be remembered that nasopharyngeal specimens are only surrogates for appropriate specimens.
- A four-fold or greater rise in Mycoplasma immunoglobulin M (IgM) titer in the convalescent versus acute period suggests acute infection. However, Mycoplasma IgM can remain positive for a year after infection, therefore, a combination of IgM and PCR may help differentiate carrier state from acute infection. Serology alone is not reliable for accurate diagnosis of Mycoplasma or Chlamydophila.
- There is no standardized, validated test for diagnosis of Chlamydophila. As mentioned earlier, positive PCR can suggest Chlamydophila infection. Acute chlamydial infections are defined by a four-fold increase in the IgG titer or an IgM titer ≥16; and prior exposure is defined as an IgG titer ≥16.
- The most common method for Legionella detection is the urinary antigen assay.

Treatment

- Atypical pathogens do not have a peptidoglycan cell wall, hence they do not respond to β-lactam antibiotics. Instead, they show good responses to protein synthesis inhibitors (macrolides and tetracyclines) or deoxyribonucleic acid (DNA) synthesis inhibitors (fluoroquinolones).
- Macrolides are the treatment of choice for atypical pneumonia because of their low minimum inhibitory concentration (MIC) and high safety profile in children. However, macrolide antibiotics should not be used indiscriminately and should be used only in confirmed Mycoplasma or Chlamydophila infections. This is especially true in settings where macrolides are reserved for multidrug-resistant Salmonella typhi infection.
- There are reports of increasing incidence of macrolide-resistant M. pneumoniae pneumonia (MRMP) in some settings, although not in India. MRMP may be considered in patients with proven Mycoplasma pneumoniae who show no response to macrolide treatment for 72 hours.
- Levofloxacin and doxycycline are alternative second-line antibiotics for MRMP and their use should be restricted because of the risk of side effects. Tetracycline can induce permanent teeth discoloration and there are reports of tendinopathy with fluoroquinolones.

TABLE 1: Treatment of atypical bacterial pneumonia.		
Age group and pathogens	**Empirical antibiotic**	**Comments**
1–6 months		
Chlamydia trachomatis	Azithromycin PO, 20 mg/kgonce daily for 3 days	Conjunctivitis and staccato cough
≥6 months		
Mycoplasma pneumoniae or Chlamydia pneumoniae	Azithromycin PO, 10 mg/ kg on day 1, followed by 5 mg/kg once daily fromdays 2–5	Alternative agents in case of macrolideresistance in an individual patient: ☑ Levofloxacin - <5 years: PO/IV 8–10 mg/kg twicedaily - ≥5 years: PO/IV 10 mg/kg once daily ☑ Doxycycline PO/IV 2.2 mg/kg every 12th hourly

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RESPIRATORY DISTRESS IN THE TERM NEWBORN

Definition

Respiratory distress (RD) in newborn is characterized by increased work of breathing (WOB)
in the form of tachypnea, grunting, chest retractions, and often associated with reduced air entry and cyanosis.

Incidence

Respiratory distress is common in the neonatal period. Incidence of RD is around 5% in term,
15% in late preterm, and >30% in infants with gestation <34 weeks.

Assessment of Severity

Downes and Silverman Anderson Score (SAS) on the clinical evaluation, oxygen saturation (SpO2) and fraction of inspired oxygen (FiO2) requirement, oxygen saturation index (OSI), alveolar-arterial diffusion gradient of oxygen (A-aDO2), oxygenation index (OI), and arterial blood gas parameters are useful in the assessment of severity of RD in a term infant.

There are various clinical scoring systems for assessing the severity of RD objectively, out of which Downes scoring (Tables 1) and Silverman Anderson (Tables 2) scoring systems are widely used. Downes scoring system is used for term neonates whereas SAS score is often used in preterm neonates.

A total score of 0 suggests no distress, score of 1–4 mild RD, score of 5–7 moderate RD, and score of >7 severe distress or impending respiratory failure.

TABLE 1: Downes score.

Score	*Respiratory rate*	*Cyanosis*	*Air entry*	*Grunt*	*Retraction*
0	<60 breaths/minute	Nil	Normal	None	Nil
1	60–80 breaths/minute	In room air	Mild decrease	Audible with stethoscope	Mild
2	>80 breaths/minute or apnea	In >40% oxygen	Marked decrease	Audible without stethoscope	Moderate-to-severe

TABLE 2: Silverman Anderson score (SAS).

Score	*Upper chest**	*Lower chest#*	*Xiphoid retractions*	*Nares dilatation*	*Grunting*
0	Synchronized	No retractions	None	None	None

1	Lag on inspiration	Just visible	Just visible	Minimal	Heard with stethoscope
2	Seesaw	Marked	Marked	Marked	Heard without stethoscope

*Part of the chest anterior to mid-axillary line. #Part of the chest posterior to mid-axillary line.

Pulse Oximetry

- Noninvasive saturation monitoring by pulse oximetry helps in assessing the severity.
- Saturation level <95% indicates the need for intervention.
- Preductal saturation target for a sick newborn on respiratory support is 90– 95%.
- Oxygen saturation index can be calculated for any neonate on invasive respiratory support. OSI value of <7 suggests mild hypoxic respiratory failure (HRF), 7–15 moderate HRF, and >15 severe HRF.
- Pulse oximetry screening is useful in early detection of critical congenital heart disease (CHD). All neonates must undergo preductal (right upper limb) and postductal (one of the lower limb) saturation check around or after 24 hours of life and saturation <95% or saturation difference between preductal and postductal of >3% is considered as screen positive and should undergo echocardiography.

Other Assessment Tools Calculation

of various formula

Saturation index = (MAP × FiO2)/SpO2

A-aDO2 = (700 × FiO2) – (PaCO2 + PaO2) or

= (760* – Water vapor pressure × FiO2) – (PaCO2/0.8#) – PaO2

PF ratio = PaO2/FiO2

Oxygenation index = (MAP × FiO2)/PaO2

*760 denotes the atmospheric pressure at sea level

#0.8 denotes respiratory quotient

(MAP: mean airway pressure; FiO2: fraction of inspired oxygen; PaO2 and PaCO2: calculated from arterial blood gas; SpO2: saturation from pulse oximeter)

Alveolar–Arterial Diffusion Gradient of Oxygen

- A-aDO2 is the difference between amount of oxygen in alveoli and the amount of oxygen dissolved in plasma (arterial oxygenation).
- A-aDO2 values could reach up to 200–400 in severe RD syndrome, persistent pulmonary hypertension (PPHN) and severe meconium aspiration syndrome (MAS).

PF Ratio (PaO2/FiO2)

- This is one of the measures used in ventilated neonates. Ratio of <300 mm Hg indicates abnormal gas exchange.

Oxygenation Index

- This is commonly used in neonates to assess the severity and to guide on the timing of intervention.
- OI value of <15 suggest mild HRF; 15–25 suggests moderate HRF; values >25 suggest severe HRF, and the need for inhaled nitric oxide (iNO) therapy.
- A persistent value above 40 is an indication for extracorporeal membrane oxygenation (ECMO).

Blood Gas Analysis

- Arterial or capillary blood gas analysis helps in assessing the severity of RD and guiding the management.
- Normal range of blood gas values in neonates are:
    - pH 7.35–7.45, PaCO2 35–45 mm Hg, PaO2 45–80 mm Hg, bicarbonate 20–24 mEq/L, and base deficit 3–7 mEq/L.

Etiology

Causes of RD in term neonates are depicted in Table 3.

TABLE 3: Causes of respiratory distress.

Common causes	*Uncommon causes*
☑ Transient tachypnea of the newborn (TTN) ☑ Meconium aspiration syndrome (MAS) ☑ Respiratory distress syndrome (RDS) ☑ Congenital pneumonia/sepsis ☑ Persistent pulmonary hypertension (PPHN) ☑ Perinatal asphyxia ☑ Critical congenital heart disease ☑ Congenital diaphragmatic hernia (CDH) ☑ Air leak syndrome: Pneumothorax	☑ Inborn errors of metabolism ☑ Anemia/high output failure ☑ Acidosis, hypoglycemia, hypothermia, and hyperthermia ☑ *Cardiac*: Arrhythmias and cardiomyopathy ☑ *Upper airway anomaly*: Choanal atresia, micrognathia, Pierre Robin sequence, laryngeal web, tracheal atresia, and vascular rings ☑ *Respiratory*: Alveolar capillary dysplasia, surfactant protein deficiency, pulmonary lymphangiectasis, and pulmonary alveolar proteinosis ☑ *Thoracic*: Chest wall deformities, skeletal dysplasia, hydrops fetalis, and phrenic nerve palsy

- ☑ *Neuromuscular*: Neuromuscular disorders, cerebral malformations, maternal sedation, and birth injury

History, onset of RD, and clinical evaluation are useful in identifying the etiology of RD in term infant (Tables 4 and 5).

TABLE 4: Etiology according to the onset of respiratory distress.

Onset	*Etiology*
<6 hours of life	☑ Transient tachypnea of newborn (TTN) ☑ Early-onset pneumonia/sepsis ☑ Meconium aspiration syndrome (MAS) ☑ Perinatal asphyxia ☑ Congenital diaphragmatic hernia (CDH)
>6–12 hours of life	☑ Sepsis ☑ Pneumonia ☑ Critical congenital heart disease (duct dependent systemic andpulmonary) ☑ Hypothermia and hypoglycemia ☑ Inborn error of metabolism ☑ Congenital pulmonary airway malformation (CPAM)

(CDH: congenital diaphragmatic hernia; CPAM: congenital pulmonary airway malformation; CTG: cardio- tocography; MAS: meconium aspiration syndrome; PPROM: preterm remature rupture of membrane; RDS: respiratory distress syndrome; TTN: transient tachypnea of newborn)

Diagnosis

Chest X-ray, ultrasound lungs, and echocardiography helps in differentiating various etiology of RD in neonates apart from history and clinical examination (Table 6).

TABLE 6: Chest X-ray and ultrasound findings in various conditions.

Condition	*Chest X-ray*	*Ultrasound lung*
TTN	Sun burst appearance; fluid in minor fissure	Thickened pleural lines, B lines, double lung point
MAS	Hyperinflation with bilateral patchy lungopacities	Disappearance of A lines,scattered B lines

RDS	Reticulogranular opacities/ground glassappearance	B lines, white lungs
Pneumonia	Asymmetrical parenchymal infilterates	Nonspecific changes
Pneumothorax	Collapsed lung border with air in pleural spacewith mediastinal shift	Absence of sliding sign; Bar code sign

(RDS: respiratory distress syndrome; TTN: transient tachypnea of newborn; MAS: meconium aspiration syndrome)

Differential Diagnosis

Table 7 elucidates the differences between congenital heart disease (CHD) and pulmonary disease.

TABLE 7: Differences between CHD and pulmonary disease.

	Pulmonary disease	*Cyanotic heart disease*
Onset of respiratorydistress	Since birth or within 6 hours of life	Usually after 24 hours, when the ductus arteriosus closes
History	Risk factors such as maternal fever, prolonged rupture of membranes, andmeconium-stained amniotic fluid couldbe elicited	Family history of congenital heart disease may be seen
Antenatal scans	Could detect congenital malformations such as CDH, CPAM, tracheoesophagealfistula	Structural heart conditions could havebeen detected in antenatal scans
Respiratory distress	Usually moderate to severe distress associated with chest retractions	Silent tachypnea in cardiac conditionswith reduced pulmonary flow; mild- to-moderate distress in conditions with increased pulmonary blood flow
Other signs	Scaphoid abdomen and hyperinflatedchest in CDH; copious secretion in TEF; septic shock can be present in pneumonia; barrel-shaped chest in MAS; labile saturations; and hypoxia during handling in PPHN	Cyanosis, murmur, signs of cardiac failure (gallop rhythm, and hepatomegaly), prominent precordial pulsations, single second heart sound,and feeble femoral pulses
Pulse oximetry screenin	Occasionally positive (false positive in PPHN and certain respiratory conditions)	Positive with greater accuracy

g		
Arterial blood gas	Hypoxia (PaO_2 low) Hypercapnia ($PaCO_2$ high)	Hypoxia (PaO_2 low) Hypocarbia or normocarbia ($PaCO_2$ normal or low)
*Hyperoxia test**	PaO_2 > 150 mm Hg	PaO_2 < 150 mm Hg
Chest X-ray	No cardiomegaly Patchy consolidation in pneumonia; bilateral patchy infiltrates with hyperinflation-MAS; prominent bronchovascular marking and fluid in minor fissure TTN; normal lungs in PPHN;ground glass appearance in RDS	Egg on side appearance in TGA; normal or small heart with pulmonaryedema in obstructive TAPVC; box- shaped heart in Ebstein anomaly
Echocardiography	Structurally normal heart; could show features of PPHN	Confirms the diagnosis

*Limited value with advent of echocardiography; also it carries risk of oxygen toxicity. (CDH: congenital diaphragmatic hernia; CHD: congenital heart disease; CPAM: congenital pulmonary airway malformation; MAS: meconium aspiration syndrome; PPHN: persistent pulmonary hypertension; RDS: respiratory distress syndrome; TAPVC: total anomalous pulmonary venous connection; TEF: tracheoesophageal fistula; TGA: transposition of the great arteries; TTN: transient tachypnea of the newborn)

Other Differentials

Metabolic Acidosis

Metabolic acidosis causes deep and high rate of breathing as a compensatory mechanism to wash out the partial pressure of carbon dioxide (pCO2). Air entry would remain good and characterized by absence of associated finding such as grunting or cyanosis. SpO2 is normal and above 95%.

Anemia

Anemia results in tachypnea as a part of high output cardiac failure. History of antepartum hemorrhage or Rh incompatibility along with clinical examination showing pallor should rise the suspicion of anemia and appropriate evaluation should be carried out.

Treatment

General Therapy

- TABC: Maintain thermoneutral zone, clear the airway, and ensuring adequate breathing and circulation. Maintain skin temperature between 36°C and 37°C. RDS and PPHN are aggravated by hypothermia.
- Continuous clinical and pulse oximeter monitoring to be done to determine the requirement for respiratory support (including escalation and de-escalation of support and type).
- Maintain euglycemia, normal fluid and electrolyte balance. Ensure a minimum glucose infusion rate of about 4 mg/kg/min for adequate glucose homeostasis. In at risk newborns, 6–8 mL/kg/day of calcium gluconate to be added to the fluid. Enteral feeding should be started as soon as the infant is clinically stable and escalated to full feeds.
- Maintenance of adequate and age-appropriate hematocrit.
- Antibiotics are usually not required. Decision to start antibiotics would depend on the clinical situation, but the threshold should be low.
- Warm, humidified oxygen should be given with soft nasal cannula preferably with an FiO2 meter and pulse oximeter monitoring to titrate the concentration of oxygen needed. Avoid using hood oxygen. When on any respiratory support maintain SpO2 between 90 and 95%.
- When low flow nasal oxygen (<2 L/min) with nasal cannula fail to maintain target oxygen saturation (just above 94%) and PaO2 of 50–80 mm Hg, heated humidified high-flow nasal cannula (HHHFNC) may be tried first. Begin with a flow of 4–6 L/min and increase @ 0.5–1 L/min as required [suggested by increasing respiratory rate (RR), WOB, and FiO2 requirement) till a maximum of 8 L/min.
- If target saturation (90–95%) are not maintained or in those with increased WOB, noninvasive respiratory support by either continuous positive airway pressure (CPAP) or nasal intermittent positive pressure ventilation (NIPPV) should be started. Indications for starting CPAP are a Downes or Silverman score of ≥5 or an FiO2 requirement of >0.3 to maintain an acceptable saturation on pulse oximeter. CPAP is started with a positive end-expiratory pressure (PEEP) of 5 cmH2O, FiO2 of 0.3 and titrated to maximum of 8 cmH2O and 0.6 FiO2, respectively. NIPPV is started with initial settings of PEEP of 5 cmH2O, peak inspiratory pressure (PIP) of 14 cmH2O, rate of 30 bpm, Ti of 0.50 second, FiO2 of 0.3 and titrated to a maximum PEEP of 8 cmH2O, PIP 25 cmH2O, rate 50 bpm, Ti 0.5 second and FiO2 0.6, respectively.
- When noninvasive ventilation (NIV) fails, intubate and switch to invasive mechanical ventilation (IMV). Ventilation mode should depend on infant's clinical condition, type of ventilator, and clinician's preference. Patient triggered ventilation with volume guarantee (4 mL/kg) is considered the best.

For best outcomes this should be given to babies in impending respiratory failure or failed CPAP rather than in complete respiratory failure. Indications for IMV are FiO2 requirement >0.6 to maintain target SpO2, respiratory acidosis (PaCO2 > 60 mm Hg), pH <7.2, or recurrent apnea. CPAP is said to have failed when the FiO2 requirement is >0.6 or the CPAP required to maintain oxygenation exceeds 8 cmH2O. Respiratory failure is defined a PaCO2 > 60 mm Hg or PaO2 < 50 mm Hg, or saturation < 85% in 100% O2 with or without a pH of <7.25. If conventional IMV fails, especially in MAS with PPHN, consider switching to high-frequency oscillatory ventilation (HFOV), if available. Indications for HFOV are requirement of PIP > 28 cmH2O, FiO2 > 0.6, respiratory acidosis with pH < 7.2 on conventional IMV.

- ECMO: ECMO is a life-saving therapy in neonates with severe hypoxic failure not responding to conventional therapy and acts as a bridge to recovery.

Specific Therapy

Surfactants

- Surfactant is the drug of choice in babies with RDS/hyaline membrane disease (HMD) (term born babies >37 weeks account for 7.8% of total RDS cases in newborns, more common among early term infants of 37–38 weeks and in infants of diabetic mother).
- It is given as early rescue therapy within the first few hours of birth when newborn is on CPAP or NIV or MV and has FiO2 requirement of >0.40 and chest X-ray is suggestive of RDS.
- Natural surfactants are preferred. Poractant alpha at an initial dose of 200 mg/kg or beractant at a dose of 100 mg/kg administered by intubation-surfactantextubation(INSURE), less invasive surfactant administration (LISA), or minimally invasive surfactant therapy (MIST) methods of surfactant administration.
- Surfactants are also beneficial in MAS and/or PPHN management.

Antibiotics

- Antibiotics should be started early in case of congenital pneumonia after collecting blood for culture and sensitivity.
- Usually, unit-specific first-line antibiotics are started and adjusted later as per culture and sensitivity report. In certain situations, MAS and RDS may mimic pneumonia. In such situations, antibiotics are started and can be stopped early when clinical and laboratory parameters points toward alternative diagnoses.

Inotropes

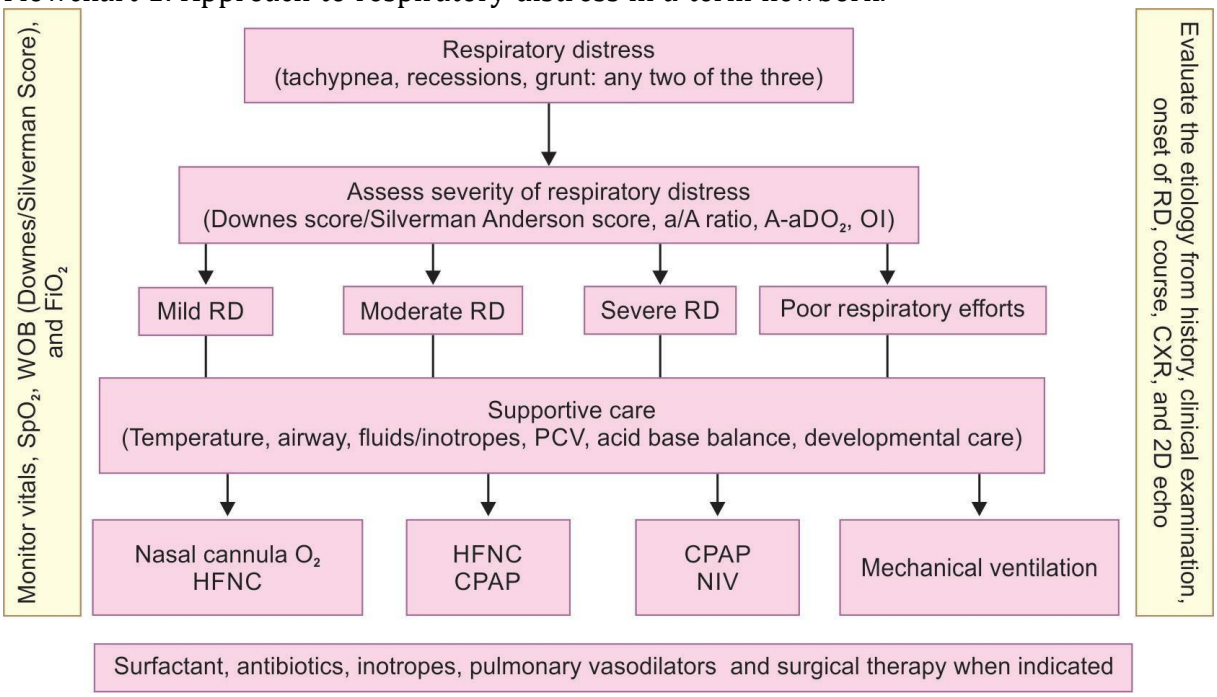
- Ensure adequate perfusion of organs with fluid boluses [0.9% normal saline (NS) 10 mL/kg over 20–30 minutes] and then add inotropes, if required.
- Choice of inotropes, if needed, depends on etiology and evaluation of circulation, blood pressures (systolic/diastolic/MAP) assisted by functional

echocardiography. Dobutamine is preferred in conditions associated with left ventricular (LV) dysfunction for its inotropic action. Milrinone has inotropic and lusitropic actions and also is a pulmonary vasodilator, so preferred in PPHN. Low-dose epinephrine has inotropic effects. Vasopressors such as dopamine, vasopressin, epinephrine (high dose), or norepinephrine are used to maintain normal blood pressure.

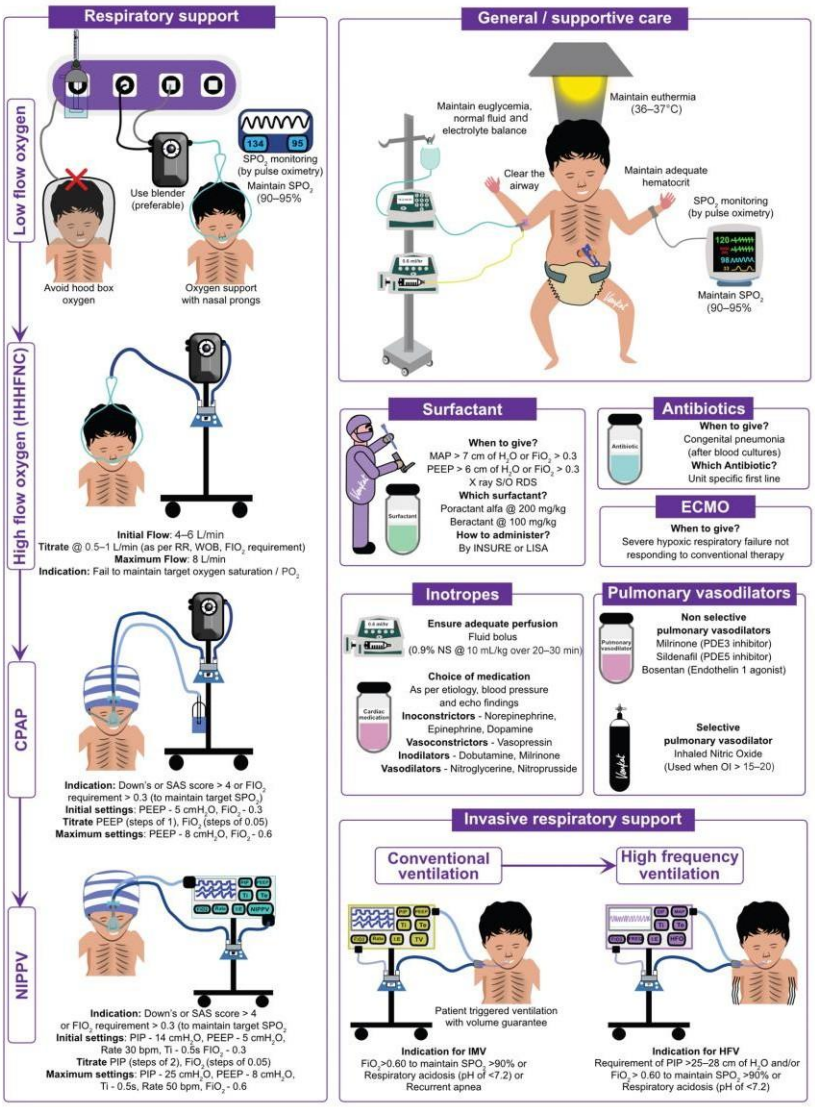
Pulmonary Vasodilators

- In case of PPHN, after supportive care and lung recruitment strategies such as ventilation/surfactant, selective pulmonary vasodilator, and iNO are used when OI is >15–20. If iNO is not available or is ineffective, alternative nonselective pulmonary vasodilators, sildenafil [phosphodiesterase-5 (PDE5) inhibitor] or bosentan (endothelin-1 agonist) may be tried with an aim to reduce pulmonary pressure and improve oxygenation.

Flowchart 1: Approach to respiratory distress in a term newborn.



(A-aDO2: alveolar-arterial diffusion gradient of oxygen; CPAP: continuous positive airway pressure; CXR: chest X-ray; FiO2: fraction of inspired oxygen; HFNC: high-flow nasal cannula; NIV: noninvasive ventilation; OI: oxygenation index; PCV: packed cell volume; RD: respiratory distress; SpO2: oxygen saturation; WOB: work of breathing)



Infographics Courtesy: Dr Venkat Reddy Kallem.

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NEONATAL JAUNDICE

Introduction

Physiological Jaundice

- Neonatal jaundice is common, occurring in 60% in term and 80% in preterm infants.
- Appears after 24 hours of life, decreases after 5–6 days, and undetectable after 14 days.
- Maximum values seldom exceed 15 mg/dL.

Severe Jaundice— Hyperbilirubinemia

- Any jaundice visible in first 24 hours of life
- Yellow staining of palms and soles or deep yellow appearance (measure bilirubin values using transcutaneous bilirubinometer or laboratory testing of serum sample, when in doubt)
- Bilirubin values >95 centile for gestation/weight/age in hours, evaluated on standard charts like the American Academy of Pediatrics (AAP) or National Institute for health and Care Excellence (NICE), UK) charts
- Warning signs of encephalopathy such as poor feeding and lethargy

Evaluation for Risk of Hyperbilirubinemia

Before discharge 24-72 hrs from birth All babies must be evaluated clinically for bilirubin levels while in hospital and before discharge; and confirmed objectively when in doubt, by a transcutaneous bilirubinometer or serum bilirubin plotted on hour specific nomograms. Kramer's Criteria is helpful in clinical assessment of the severity of the jaundice. The clinical
assessment requires natural light (can be faulty in hospital lighting). It also depends on experience of personnel and subjectivity of assessment) (Fig. 1).

Visual Assessment by Kramer Criteria		
1.	Face	4–8 mg/dL
2.	Upper trunk	5–12 mg/dL
3.	Lower trunk and thighs	8–16 mg/dL
4.	Arms and lower legs	11–18 mg/dL
5.	Palms and soles	>15 mg/dL

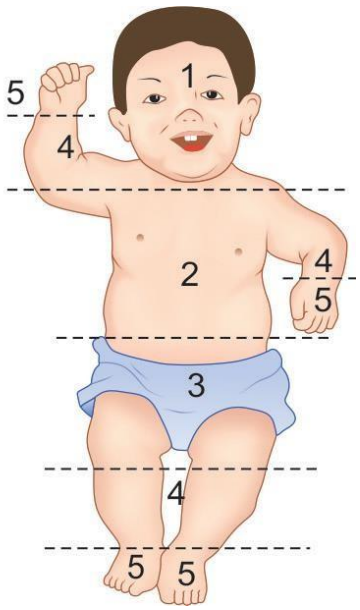


Fig. 1: Kramer's criteria.

Use the hour-specific nomogram to evaluate risk before discharge from birth admission. Babies with values in high-risk zone must be re-evaluated within 24 hours (Fig. 2).

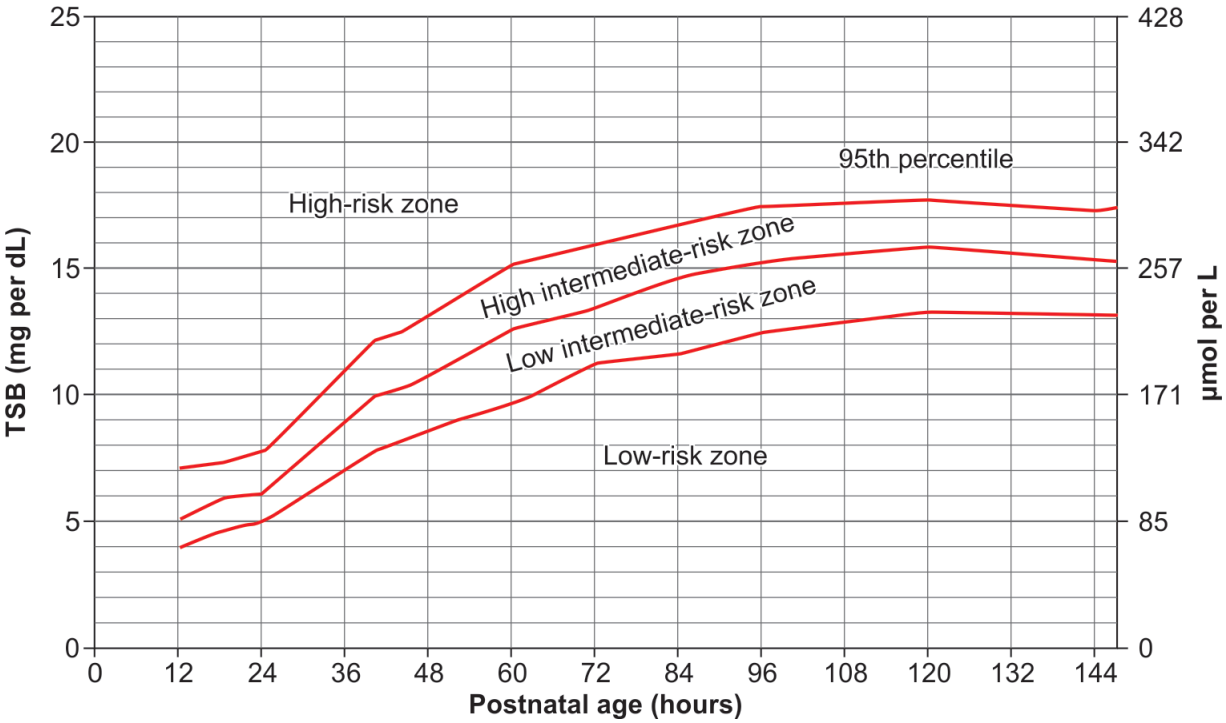


Fig. 2: Hour-specific nomogram. (TSB: total serum bilirubin)

Source: American Academy of Pediatrics Subcommittee on Hyperbilirubinemia. Management of hyperbilirubinemia in the newborn infant 35 or more weeks of gestation. Pediatrics. 2004;114(1):297-316.

Evaluation of a Baby with Hyperbilirubinemia

- After discharge (until day 5–6 of life) from hospital

•• All babies reviewed within 48 hours and babies with higher risk within 24 hours of discharge for yellow staining of palms and soles or deep yellow appearance (measure values using transcutaneous Bilirubinometer or laboratory testing of serum sample, when in doubt, use specific charts such as AAP or NICE charts to evaluate need for treatment).

•• Look for lactation problems (excess weight loss and delayed transition of stool to yellow color), infrequent stool, and urine.

•• Exclude early signs of encephalopathy (poor feeding and lethargy)

Close follow-up (within 24 hours of discharge) is warranted in risk groups (Box 1).

BOX 1: Risk groups: Need close attention.

- Mother Rh-negative or O group
- Gestation of baby <38 completed weeks
- Lactation not established
- Predischarge bilirubin in high-risk zone (transcutaneous bilirubin >13 mg/dL)
- Cephalohematoma
- Previous baby with jaundice
- Glucose-6-phosphate dehydrogenase (G6PD) deficiency

History

- Gestational age and postnatal age (in hours)
- Birth weight and current weight
- Mode and adequacy of feeding
- Urine color and number of wet nappies
- Passage of meconium and color of stool
- Activity and behavior during sleep/waking up
- Any abnormal cry or body movements
- Any bleeding/bruising
- Mother's blood group and baby's blood group
- Previous baby with severe neonatal jaundice

Evaluation of a Baby with Hyperbilirubinemia

Examination

Feature of acute bilirubin encephalopathy (hypotonia and hypertonia, lethargy, highpitched
cry, poor suck, irritability, seizure, and opisthotonos posture)

Investigations

- Total serum bilirubin (TSB)
- Mother and baby's blood group (collect cord blood/venous sample immediately after birth, if mother's blood group is Rh-ve)
- Suspected hemolysis: Complete blood count, reticulocyte count, peripheral blood smear, and direct Coombs' test
- In areas of high prevalence: Screen for glucose-6-phosphate dehydrogenase (G6PD) deficiency
- For prolonged jaundice*: Total and direct bilirubin, thyroid function test, urine reducing substances, and culture. Ultrasound abdomen to exclude biliary atresia.

*Prolonged jaundice: Visibly detectable jaundice beyond 2 weeks of age in a term and beyond 3 weeks of age in a preterm infant. Ask for pale stool or yellow urine, check for adequacy of weight gain. Do total and direct bilirubin test.

Management

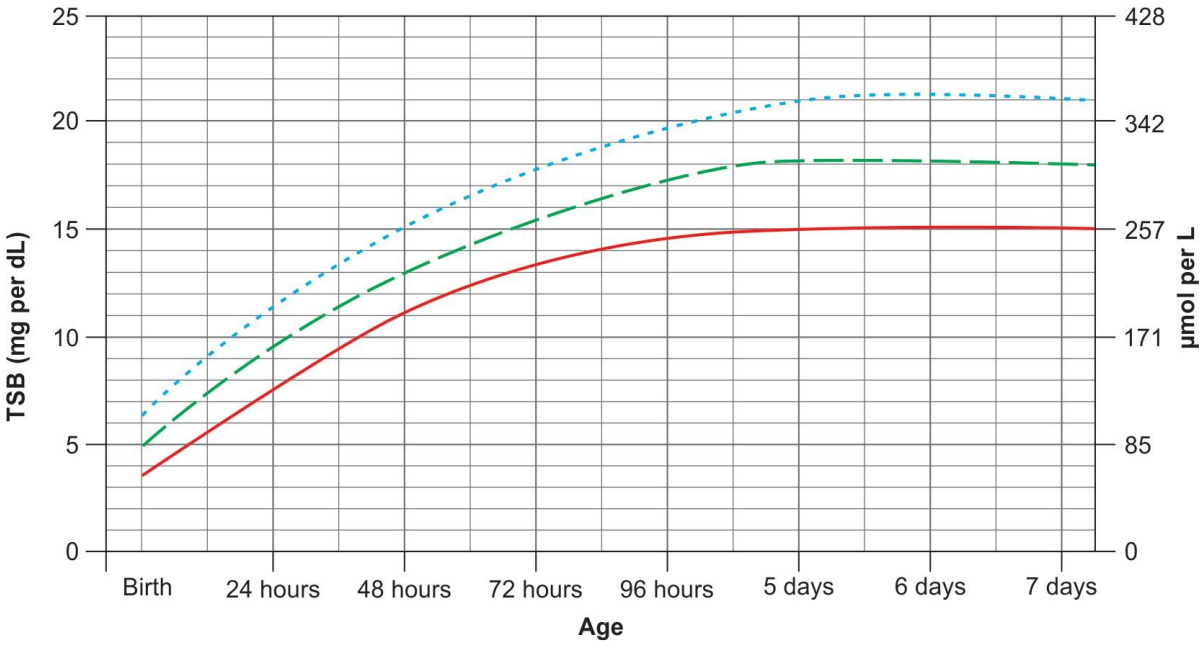
Hyperbilirubinemia is a potentially treatable condition. It may cause long-term neurodevelopmental impairment, if not treated timely and appropriately.

Prevention

Early initiation and frequent breastfeeding and/or early use of mother's own milk (MOM) in neonatal intensive care unit (NICU).

Treatment

Reducing the level of serum bilirubin by intensive phototherapy and/or exchange transfusion Phototherapy is a noninvasive, cost effective, safe, and easy to use method; it is available at all levels of neonatal healthcare. It should be started (after sending TSB) when jaundice appears within 24 hours and/or involving palm and soles; and if TSB is in range of phototherapy as per AAP or NICE charts. Stop phototherapy, if serum bilirubin level is 2–3 mg/dL lower than the phototherapy range (Fig. 3).



- Use TSB. Do not subtract direct reacting or conjugated bilirubin
- Risk factors include isoimmune hemolytic disease, glucose-6-phosphate dehydrogenase deficiency, asphyxia, significant lethargy, temperature instability, sepsis, acidosis, or albumin <3.0 g per dL (30 g per L, if measured).
- For well infants delivered at 35 to 3T⅙ weeks' gestation, TSB levels for intervention can be adjusted around the medium-risk line. Intervention at lower TSB levels is an option for infants delivered closer to 35 weeks' gestation, and at higher TSB levels for those delivered closer to 37⅙ weeks.
- Conventional phototherapy in the hospital or at home is an option for infants with TSB levels 2 to 3 mg per dL (35 to 50 mmol per L) less than those shown, but home phototherapy should not be used in any infant with risk factors.

Fig. 3: Guidelines for phototherapy in hospitalized infants delivered at 35 or more weeks' gestation. (TSB: total serum bilirubin)

Source: American Academy of Pediatrics Subcommittee on Hyperbilirubinemia. Management of hyperbilirubinemia in the newborn infant 35 or more weeks of gestation. Pediatrics. 2004;114(1):297-316.

Optimizing phototherapy:

- Use blue light and appropriate intensity of phototherapy (>30 µW/cm2 per nm)
  - •• Light-emitting diode (LED) and compact fluorescent lamps (CFL) most often deliver the required intensity for a long duration. A periodic check of the intensity must be done to ensure efficacy (once in 6 months).
- Place phototherapy as close to baby as possible without causing hyperthermia
- Expose maximum area of body

- Ensure optimal breastfeeding and stool output

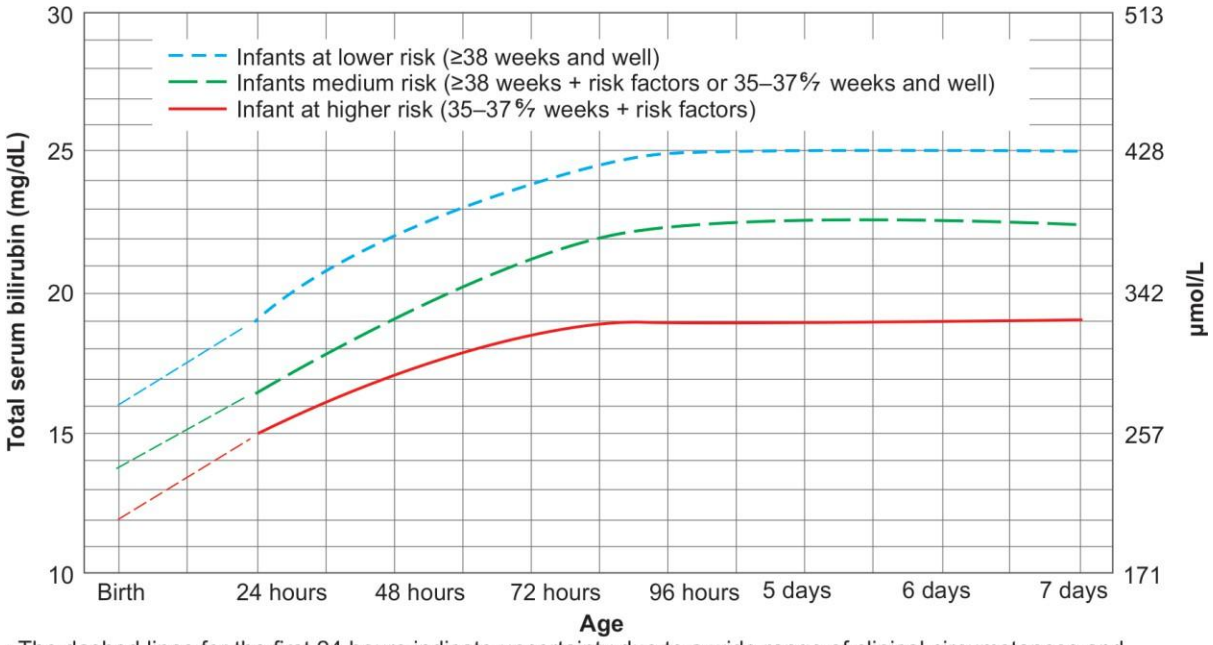
Indications for referral for potential exchange transfusion:

- Hyperbilirubinemia (as per AAP or NICE charts) not responding to intense phototherapy
- Any signs of early encephalopathy (poor feeding/lethargy) in babies with hyperbilirubinemia
- Babies with hyperbilirubinemia noted within 24 hours of life, preterm babies, and previous child requiring exchange transfusion or sick babies (sepsis) with hyperbilirubinemia are at risk of developing bilirubin associated neurologic damage at values less than that indicated on standard chart. They must be referred early to centers with facilities for exchange transfusion.

Exchange transfusion is a rapid, invasive, and effective method to reduce serum bilirubin. It is a specialized procedure, performed where facilities and skills are available. If facilities are not available, refer the baby along with mother's blood sample (if mother is not accompanying) (Fig. 4).

Types of blood used for exchange transfusion:

- Blood being used must be crossmatched with mother's blood.
- For Rh-isoimmunization: O-negative packed cells suspended in AB plasma or O-negative whole blood or Rh-negative baby's ABO group after crossmatch.
- For ABO isoimmunization: O group (Rh-compatible) packed cell suspended in AB plasma or O group whole blood (Rh-compatible with baby) after crossmatch.
- In other situation, baby's blood group should be used.



- The dashed lines for the first 24 hours indicate uncertainty due to a wide range of clinical circumstances and a range of responses to phototherapy.
- Immediate exchange transfusion is recommended if infant shows signs of acute bilirubin encephalopathy (hypertonia, arching, retrocollis, opisthotonos, fever, high-pitched cry) or if TSB is ≥5 mg/dL (85 μmol/L) above these lines
- Risk factors—isoimmune hemolytic disease, G6PD deficiency, asphyxia, significant lethargy, temperature instability, sepsis, acidosis.
- Measure serum albumin and calculate B/A ratio
- Use total bilirubin. Do not subtract direct reacting or conjugated bilirubin
- If infant is well and 35–37⁶⁄₇ week (median risk) can individualize TSB levels for exchange based on actual gestational age.

Fig. 4: Guidelines for exchange transfusion in infants 35 or more weeks' gestation.

Source: American Academy of Pediatrics Subcommittee on Hyperbilirubinemia. Management of hyperbilirubinemia in the newborn infant 35 or more weeks of gestation. Pediatrics. 2004;114(1):297-316.

Follow-up and Long-term Neurodevelopmental Outcome

Babies who had hyperbilirubinemia must be followed up periodically using developmentscreening tools until school age. The assessments should include early language milestones. Babies who had signs of encephalopathy or required exchange transfusion must have a hearing evaluation for sensorineural hearing impairment by brainstemevoked audiometry before 6 months age.

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UNPROVOKED SEIZURES

Introduction

- First seizure is a dramatic and frightening event. For parents whose child has had a first-time seizure, and for the physicians who treat them, the most salient concern after stabilizing the child is the risk of recurrence.
- Unprovoked seizure: Seizure or seizure clusters occurring within 24 hours in a child >1 month age occurring in the absence of precipitating factors, such as fever, hypoglycemia, dyselectrolytemia, meningitis, drug/toxin overdose, trauma, stroke, and intracranial hemorrhage.
- Incidence rates of single unprovoked seizure: 23–64.1/lakh/person-years.
- 10–12% of children and adults with a first unprovoked seizure will present with a seizure lasting ≥30 minutes (status epilepticus) as their first seizure.
- First seizure in a child can be febrile seizure (seizure associated with febrile illness but not related to neuroinfection), nonfebrile illness seizure (seizure associated with an infection but without fever like upper respiratory tract infection or gastrointestinal illness) and unprovoked seizure.
- Acute symptomatic seizures (reactive or provoked seizures or situation related seizures): These are associated with acute brain insult, which may be due to infectious, toxic, metabolic, or traumatic cause.
- Remote symptomatic seizures: These occur without immediate cause but with a prior identifiable major brain insult such as severe trauma or accompanying a condition such as cerebral palsy or mental retardation.
- Genetic/idiopathic etiology: Not associated with a known central nervous system (CNS) disorder and are of suspected genetic etiology.
- Unknown/cryptogenic etiology: Causes not yet known but these children also have developmental delay or other neurological deficits.

Investigations

- Serum calcium should be done in all children <2 years especially those <1 year with unprovoked seizure.
- Serum electrolytes, random blood sugar (RBS) can be obtained if the child is drowsy post seizure at presentation.
- Electrocardiogram (ECG): It is considered in children with seizures associated with syncope or breath-holding spell and when the event was not observed by an adult or when the event occurred while playing.
- Electroencephalogram (EEG): It can give clue to underlying structural lesion and guide further tests. Inclusion of both an awake and a sleep tracing, as well

as hyperventilation and photic stimulation is recommended to increase the yield.

•• Emergent EEG (<24 hours): It is used to confirm the occurrence of seizure and differentiate from a seizure mimic. In case of true seizures, EEG may show slowing of background (focal/generalized) and/or interictal epileptiform discharges. It also helps to diagnose nonconvulsive or subtle convulsive status epilepticus that may be mistaken for a prolonged postictal state and difficult to diagnose clinically. One-fourth of patients with treated status epilepticus who appear to be seizure-free continue to have electrographic seizure activity detectable with EEG.

•• Nonemergent EEG after 1–2 weeks can help in identifying the seizure type and epileptic syndrome.

- An EEG helps to predict recurrence, particularly if there is epileptiform activity and to help make a diagnosis of epilepsy or epileptic syndrome.
- Neuroimaging: Abnormal neuroimaging has been considered as a risk factor for recurrence after a first seizure.
- Indications:
  •• Acute head trauma
  •• Fever
  •• History of anticoagulation/bleeding diathesis
  •• History of malignancy
  •• New onset or postictal focal neurologic deficit not resolving in a few hours
  •• Focal seizure
  •• Persistent altered mental status
  •• Persistent headache

- Neuroimaging is important to exclude an epileptogenic lesion that may confer an elevated recurrence risk and in providing accurate diagnosis.
  •• Emergent: CT has radiation risk and is inferior to MRI in identifying lesions. Migration defects and mesial temporal sclerosis, both of which are known to cause childhood onset seizures are not readily seen on CT. Hence, MRI is the preferred imaging modality.
  •• Follow-up: Nonurgent MRI is indicated in:
    •• Infants
    •• Significant cognitive or motor impairment of unknown etiology
    •• Unexplained abnormalities on neurologic examination
    •• Focal onset of seizure
    •• No evidence of benign partial epilepsy of childhood or primary generalized epilepsy on EEG.

- The International League Against Epilepsy (ILAE) recommended MRI when feasible for all epileptic patients, who do not have a clearly identifiable epilepsy syndrome. However, the decision is less clear after a first unprovoked seizure which may turn out to be an isolated event without any recurrence.
- Lumbar puncture (LP) is not routinely recommended in afebrile children. It can be considered in:
    - Children <12 months
    - Persistently altered mental status
    - Meningeal signs
    - Immunocompromised status.

If increased intracranial pressure is suspected, the LP should be preceded by neuroimaging.

Treatment

- Rescue medications: Intranasal midazolam spray 0.2 mg/kg/dose (one puff for every 5 kg—maximum 10 mg/dose), buccal midazolam (0.5 mg/kg), rectal diazepam 0.2–0.5 mg/kg (maximum 20 mg) to abort the ongoing seizure.
- If seizure persists for >5 minutes or if the child is seizing at presentation to emergency room, status epilepticus management guidelines can be followed: maintain airway, breathing, and circulation. Measure RBS, electrolytes, and calcium. Benzodiazepines: Injection lorazepam 0.1 mg/kg/dose or injection midazolam 0.1 mg/kg IV/IM or injection diazepam 0.2–0.3 mg/kg intravenous (IV). If seizure persists, loading with levetiracetam 40–60 mg/kg or valproate 30–40 mg/kg or phenytoin/fosphenytoin at 20 PE/kg can be given.
- Risks of chronic daily treatment with antiseizure medication (ASM) could be more detrimental to the child's health or development than seizure recurrence. Therefore, the risks and benefits of treatment must be individualized.
- The risk of recurrence depends on several clinical, etiological, EEG, and neuroimaging findings that should be approached on an individual basis.
- Treatment with ASM after a first seizure reduces the risk of recurrence, but does not prevent the development of epilepsy. There is no evidence of difference between starting treatment after the first seizure versus after a second seizure in achieving a 1- or 2-year seizure remission.
- Adverse effects of ASM include rash, hirsutism, weight gain, effects on behavior, and higher cortical function, which are often dose related and may be under-recognized. Severe reactions such as hepatic toxicity, bone marrow toxicity, and Stevens–Johnson syndrome cannot be

anticipated and require early recognition of symptoms. Side effects occurred at a rate of 9% for phenytoin, 4% each for carbamazepine and valproate.

•• Initiating ASM is not generally advised, but shall be considered in individual situations where the benefits of reducing the risk of a second seizure outweigh the risks of pharmacologic and psychosocial side effects.

•• The ILAE Task Force on Sports for People With Epilepsy (PWE) based on potential risk of injury or death should a seizure occur:

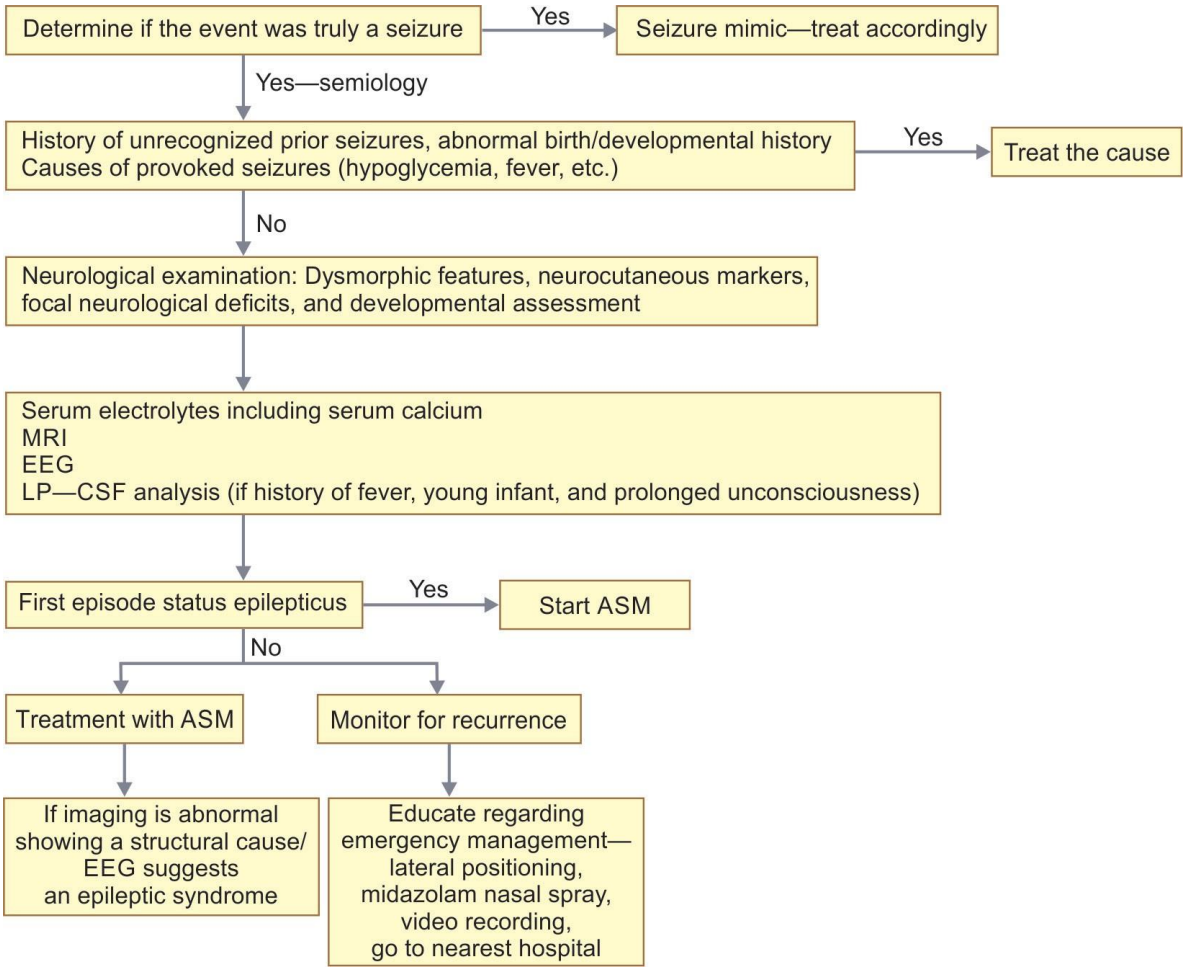
- *Group I*: Sports with no significant additional risk (athletics, bowling, most collective
sports in the ground, etc.)
- *Group II*: Sports with moderate risk to PWEs, but no risk to bystanders (gymnastics, fencing, cycling, karate, etc.)
- *Group III*: Sports with major risk for PWE and bystanders (aviation, climbing, diving, horse riding, etc.).

Prognosis

- Most children with a first unprovoked seizure will have few or no recurrences, and about 10% will have ≥10 seizures despite therapy.
- Seizures that can occur in clusters in 24 hours associated with acute diarrheal illness with/ without fever have less chance of recurrence and hence do not usually warrant long-term ASMs.
- Serious injury from a seizure in a child is a rare event, usually from a fall with loss of consciousness. To reduce that risk, restrictions are recommended that would apply to any young child, such as bicycling with a helmet on a sidewalk rather than the street and swimming only with a guardian.
- 46–54% children experience a recurrence.
- The recurrence rate by 2 years is higher in individuals who have a remote symptomatic etiology (50–80%). In those with an idiopathic/cryptogenic etiology, it is significantly lower (30–50%).
- None of the children studied prospectively had residual motor or cognitive disability.
- If a child with an initial prolonged seizure does experience a seizure recurrence, it is more likely to be prolonged.
- 10% will develop difficult-to-control and protracted epilepsy.
- Identifying genetic, immune, or imaging markers may improve prognostication.
- *Seizure recurrence rate*: 16, 21, 27, and 34% at 12, 24, 36, and 60 months, respectively.

- The majority of patients experiencing new episodes do so in the first 3 months after the initial event.
- Focal seizures have a higher risk of recurrence than generalized seizures because they are mostly associated with a structural etiology, abnormal EEG, and neuroimaging findings.

Flowchart 1: Algorithm to approach of seizures in children.



(ASM: antiseizure medication; CSF: cerebrospinal fluid; EEG: electroencephalogram; LP: lumbar puncture)

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ACUTE DYSENTERY

Definition

A syndrome characterized by frequent small stools containing visible blood, often accompanied by fever, tenesmus, and abdominal cramps. Visible blood is essential for diagnosis. The definition does not include blood streaks on surface of formed stool or blood detected only by microscopic examination or biochemical tests.

Causative Agents

- Bacterial: Shigella accounts for the highest incidence of dysentery in the age group of 1–4 years. Salmonella, Campylobacter jejuni, Escherichia coli, and Yersinia enterocolitica are some of the other bacterial agents.
- Parasites: Parasites are uncommon and Entamoeba histolytica, accounts for only 3–7% of cases.

Pathogenesis

Pathogenesis mainly includes feco-oral transmission, but also person-to-person contact (in organisms requiring small inoculums for transmission). Pathogens usually invade and multiply within the intestinal epithelial cells and induce extensive destruction and inflammation producing ulcers and microabscesses that manifest with diarrheal stools containing blood, mucus, and pus.

Clinical Course

- Day 1: High fever, crampy abdominal pain, emesis, toxicity, anorexia and malaise, watery diarrhea, and tenesmus. Abdominal distention and tenderness, hyperactive bowel sounds, and tender rectum.
- Day 2: Blood and mucus appears.

- Day 2/3 onward: In 50%, stool volume decreases with frequent passage of scanty blood and mucus. Untreated diarrhea may last up to 2 weeks. Bacteremia is an uncommon presentation. It is important to keep these differentials in mind while diagnosing dysentery (Table 1) and be aware of the complications (Box 1). Flowchart 1 shows an algorithm for approach to a case of acute dysentery.

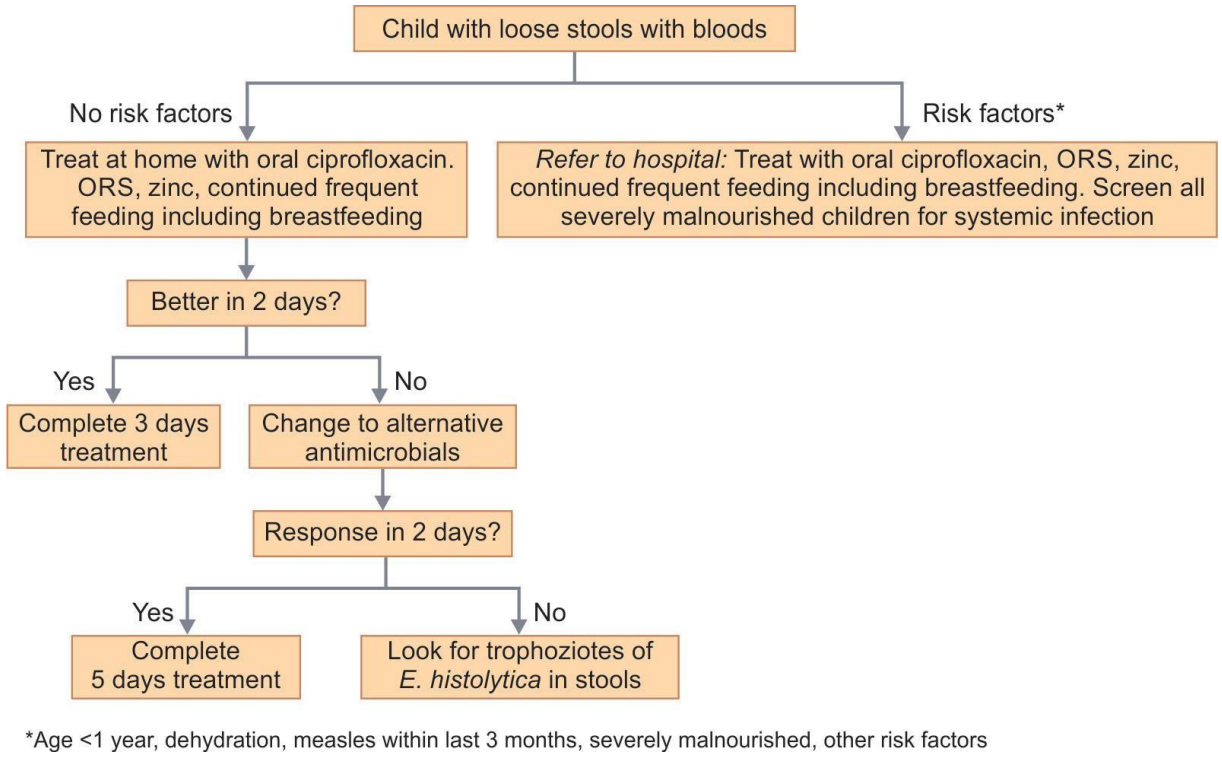
TABLE 1: Differential diagnosis in a child with bloody diarrhea.

Intussusception	Severe paroxysms of pain, with child normal in between episodes
Ulcerative colitis	*Onset:* Adolescence and young adult extraintestinal manifestations
Meckel's diverticulitis	2 years of life with painless bleeding
Eosinophilic gastroenteritis	Atopic features, elevated serum IgE, peripheral eosinophilia (AEC >660/mm^3)
Pseudomembranous colitis	Patient on antibiotics; recovers once offending drug is discontinued
Allergic colitis	Age <6 months, healthy baby, improves on removing allergen (usually milk) from diet and recurs on challenge

(AEC: absolute eosinophil count; IgE: immunoglobulin E) BOX 1: Complications of acute dysentery.

- Neurological complications
- Seizures
- Hemolytic uremic syndrome
- Dehydration and dyselectrolytemia
- Hypocalcemia, hypoglycemia, and hypothermia
- Reiter's syndrome (with HLA B27)
- Nonsuppurative arthritis
- Protein losing enteropathy
- Toxic dilatation and colonic perforation
- Rectal prolapse
- Leukemoid reaction

Flowchart 1: Algorithm for approach to a case of acute dysentery.



(ORS: Oral rehydration therapy)

Management

- Management of dehydration: Chances of dehydration are more in infants and children with severe diarrhea and vomiting. They require intravenous fluids or oral rehydration therapy (ORT). Normal diet should be continued. Breastfeeding is necessary if the baby is <6 months.
- Nutrition: High protein and high caloric diet. Amylase resistant starches that mainly found in green bananas significantly decrease the stool volume.
- Zinc: Elemental zinc should be given for 14 days. It significantly decreases the duration of diarrhea and enhances the recovery.
- Vitamin A: A large single dose of vitamin A (200,000 unit) is recommended.
- Antimicrobial therapy: Antimicrobial therapy for shigellosis should be initiated as most cases of dysentery are caused by it and almost all severe cases are caused by it. Antimicrobials also decrease the probability of feco-oral transmission to the members of the household neighbors and friends. Currently treatment using ciprofloxacin or second-line antibiotic medication (i.e., azithromycin or third-generation cephalosporin) is recommended. Shigella is now mostly resistant to ampicillin and trimethoprim- sulfamethoxazole.

Multidrug-resistant (MDR) Shigella (resistant to more than two first-line antibiotics including ciprofloxacin, cotrimoxazole, and ampicillin) are becoming a major global concern. Ceftriaxone is considered the best option for severe infections caused by MDR strains and azithromycin can be used as empirical treatment for severe dysentery before the culture sensitivity tests. Choice of antibiotics should be according to the antimicrobial resistance pattern of the community and the locality (Table 2).

- Amoebic dysentery: Being rare, it is to be considered if two different antibiotics have been used without any sign of improvement or if microscopic examination of fresh stool specimen shows trophozoites of E. histolytica containing red blood cells (RBCs). Metronidazole is the drug of choice.

*If susceptibility known or likely based on local data.

TABLE 2: Dose of commonly used agents in acute dysentery.

Ciprofloxacin	15 mg/kg po bid × 3 days
Cefixime	8 mg/kg once daily × 3 days
Ceftriaxone	50–100 mg/kg/day IV × 3 days
Azithromycin	12 mg/kg on day 1, followed by 6 mg/kg once daily for next 3 days
Cotrimoxazole	4 mg/kg/day of trimethoprim (TMP)*
Metronidazole	10 mg/kg/dose thrice a day po × 5 days

Precautions

There is no role for diphenoxylate hydrochloride with atropine (Lomotil) or loperamide and it is best to avoid fizzy drinks and fruit juices.

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NEONATAL SEIZURE

Introduction

- Seizures in the newborn represent the most distinctive common manifestations of neurological disease in the neonatal period. The incidence increase with lower gestational age and birth weight and is most common in the very low birth weight (VLBW) infant.
- Estimated incidences are up to 58/100 live births in the VLBW infant and 1 to 3.5/1000 live births in the term infant.
- Most seizures during the newborn period are acute symptomatic seizures due to cerebral injury or dysfunction due to varied etiology.
- Important manifestation to alert the clinician to underlying neurological disorders.
- Seizure onset and semiology will guide in identifying possible etiology and determining appropriate management.
- Amplitude-integrated electroencephalography (aEEG) can pick up 70–80% of seizures in monitoring.

Clinical Versus Electrical Seizures

- As per the World Health Organization (WHO) recommendations, all clinically apparent seizures lasting for >3 minutes or brief serial seizures are to be treated.
- If continuous electroencephalogram (EEG) monitoring is available, all electrical seizures should be treated even in absence of clinically apparent seizures, especially if babies are paralyzed.

Types of Neonatal Seizures

- Electrical activity that evolves over time and meets criteria
- Electroclinical seizure: Electrographic seizure with associated clinical signs
- EEG-only (subclinical, nonconvulsive, and occult) seizures: Electrographic seizures without clinical signs

Electrographic Criteria for Neonatal Seizures

- Sudden change in EEG
- Repetitive waveforms that evolve in morphology, frequency, and/or location
- Amplitude: At least 2 μV
- Duration: At least 10 seconds

- Seizures must be separated by at least 10 seconds to be considered separate
- Clinical signs may or may not be present
- EEG seizures can be as follows:
    - Unifocal—seizures arise from a single region
    - Multifocal—seizures originate from at least three independent foci with at least one in each hemisphere
    - Lateralized—seizures propagate within a single hemisphere
    - Bilateral independent—seizures occur simultaneously in two regions and begin, evolve, and behave independently
    - Bilateral—both hemispheres involved
    - Migrating—the seizure moves sequentially from one hemisphere to another
    - Diffuse—asynchronous involvement of all brain regions

Nonepileptic Phenomenon

- Roving eye movement, nystagmoid jerks, sucking, and other limb movement during sleep or in drowsy state
- May be mistaken as seizure activity
- Movement stops with gentle restrain, and no autonomic phenomena occurs
- Causes could be mild hypoxic-ischemic encephalopathy (HIE)/metabolic/drug withdrawal

Etiology

Etiologies of neonatal seizure are given in Box 1.
BOX 1: Common underlying etiologies (responsible for 80–85% of seizure).

- Hypoxic-ischemic encephalopathy (38%)
- Stroke (18%)
- Intracranial hemorrhage (11%)
- Intracranial infections (5%)
- Cerebral dysgenesis (4%)
- Metabolic and genetic (12–15%)

Classification of Neonatal Seizure

- Subtle seizure
- Clonic seizure
- Tonic seizure
- Spasm
- Myoclonic seizure

- Term versus preterm (Table 1)

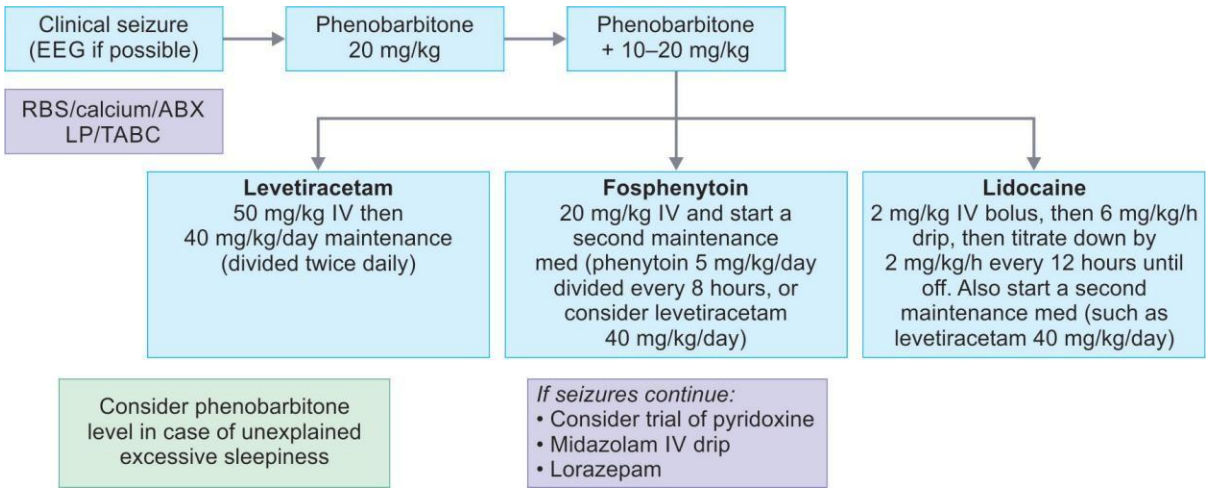
TABLE 1: Term versus preterm neonatal seizure.

Cause	*Preterm*	*Full term*
Hypoxic-ischemic encephalopathy (HIE)	+++	+++
Intracranial hemorrhage	++	+
Intracranial infection	++	++
Development defect	++	++
Hypoglycemia	+	+
Hypocalcemia	+	+
Epilepsy syndrome	—	+

Management

Management of neonatal seizure is depicted in Flowchart 1.

Flowchart 1: Management of neonatal seizure



(ABX: antibiotics; EEG: electroencephalogram; LP: lumbar puncture; RBS: random blood sugar; TABC: temperature/airway/breathing/circulation)

How to Plan to Wean the Anticonvulsants if no Clinical Seizure or EEG Awaited

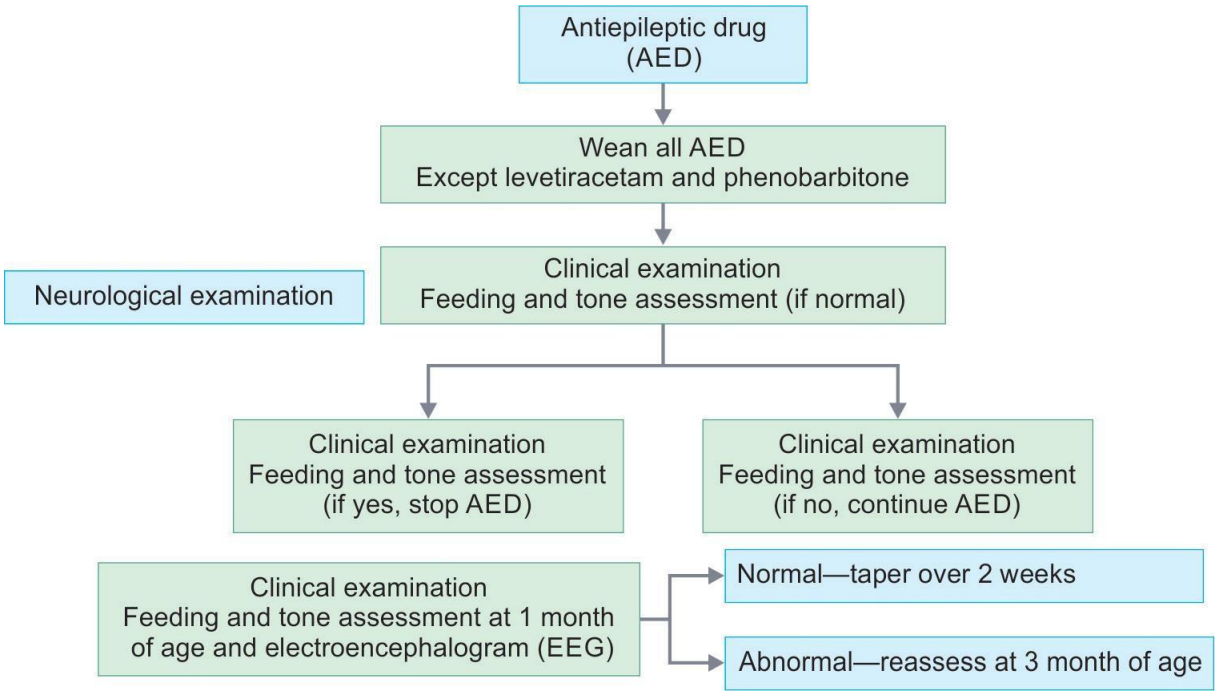
- Monitor EEG (if available)
- If on phenobarbitone (PB) maintenance (get trough level, if available)

- Get MRI brain/lumbar puncture and other relevant investigations to rule out the cause of seizure
- Always attempt to wean to one drug for maintenance
- Try to stop all medication before discharge if seizure free for 48–72 hours

Determinants of Duration and When to Stop Anticonvulsant Therapy (Flowchart 2)

It is mainly depends on three factors:
1. Neurological examination at the time of discharge—examine for feeding pattern and tone assessment
2. Cause of neonatal seizure
3. EEG pattern

Flowchart 2: Weaning of all AEDs.



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MANAGEMENT OF FEVER WITHOUT FOCUS IN OFFICE PRACTICE

Introduction

Fever without focus: Fever without a source (FWS), defined as acute fever for <7 days without a clear focus of infection after a complete examination, is challenging for physicians as children often present with nonspecific symptoms and the initial clinical presentation can vary widely.

Why important?

Most infants with fever without focus (FWF) have a good prognosis, and most of the cases are self-limited.2 However, between 1–30% of the patients may have some severe bacterial infection (SBI) such as urinary tract infection, bacteremia, pneumonia, or meningitis.

Risk Factors for Serious Bacterial Infection

No single risk factor can be used in isolation, but certain children are at a greater risk of SBI.

- Age <3 months3
- Those with Ill appearance
- Children with comorbidities such as chronic medical conditions and intellectual disability
- Immunocompromised children

Causes

Although the differential diagnosis of fever is quite broad and includes both infectious and noninfectious causes, many febrile children have underlying infectious causes of fever. The etiology might vary with age and the risk of invasive bacterial infection is more common in children <3 months of age.

- Most children between 3–36 months of age, who are well-appearing and have no identifiable source of infection have a self-limited viral illness.
- Other common causes are UTI, occult bacteremia, and clinically occult pneumonia.
- Postvaccination fever is common, with a typical onset within 24 hours of immunization and duration up to 2–3 days however, in an unwell child, fever should not be attributed to vaccination alone.
- Urinary tract infection: With a prevalence of about 8–10% in young children with a fever ≥39°C (102.2°F), urinary tract infection (UTI) is the most common occult bacterial infection among febrile infants and young children.4 In children <2–3 years, UTI may present with nonspecific symptoms fever may be the sole manifestation.

- Bacteremia: Occult bacteremia is defined as the isolation of a bacterial pathogen in a blood culture taken from an otherwise well-appearing febrile child. The risk of occult bacteremia in these patients depends upon their immunization status. The incidence of occult bacteremia in completely immunized children who have FWS is <1%.3 However, the frequency of occult bacteremia in well-appearing 3–36-month-old children with temperatures >39°C (102.2°F) prior to the availability of PCV7 or PCV13 and Hib conjugate vaccines was 3–11%.5
- Pneumonia: Most children with fever and pneumonia have some abnormality on physical examination. However, a reliable physical examination in a young child can be a challenge, and pneumonia may not be apparent.
- Rare and uncommon causes: Septic arthritis or osteomyelitis is often missed.

Evaluation

The goal of the evaluation of the young, well-appearing, febrile child without an apparent source of infection on examination is to determine the risk of a clinically occult bacterial infection and the need for further investigation and/or antibiotic therapy Table 1.

Historical Findings

These findings suggest an occult source of infection may be subtle.

- Was there documented fever? If so, how was it documented? "Fever Phobia" has to be ruled out.
- A response (or lack thereof) to antipyretic medications does not predict whether the underlying cause is bacterial or viral.
- The provider should ask about the child's functional status, including oral intake, presence of irritability or lethargy, change in activity, and associated symptoms and immunization history. Decreased walking, crawling, or movement of an extremity may indicate a deep soft tissue or bone infection. In older children, dysuria, foulsmelling urine, and frequency may point to a UTI.
- The clinician should also identify any underlying medical condition that increases the child's risk for serious infection such as the presence of underlying immunosuppressed state.

Physical Examination

On careful evaluation, some children initially felt to have fever without a source may demonstrate subtle findings that suggest an infectious focus. Specific features to note include:

- Lesions in the oropharynx that may identify a recognizable viral illness, such as herpetic gingivostomatitis (anterior ulcers) or Coxsackie virus infection (pharyngeal vesicles).

- Pain with bone palpation or passive joint range of motion
- Skin findings, such as petechiae, cellulitis, or viral exanthem
- Tachypnea

TABLE 1: NICE traffic light system for assessment of children with fever.

	Green—Low-risk	***Amber—Intermediate***	***Red—High-risk***
Color	Normal color	Pallor reported by parent	Pale/mottled/blue
Activity	☑ Responds normally to social cue ☑ Stays awake or quickly awakes ☑ Strong normal cry/not crying	☑ Not responding normally to social cue ☑ Wakes only with prolonged stimulation ☑ Decreased activity	☑ No response to social cue ☑ Does not wake or stay roused if awaken ☑ Weak cry or continuous cry
Respiratory		☑ Nasal flare ☑ *Tachypnea:* Respiratory rate >50/min; 6–12 months >40/min; >12 months SpO_2 ≤95% in air ☑ Crackles in chest	☑ Grunting ☑ *Tachypnea:* Respiratory rate ☑ >60/min ☑ Moderate-to-severe chest indrawing
Circulation and hydration	☑ Normal skin and eyes ☑ Moist mucous membranes	☑ *Tachycardia:* Heart rate >160/min; <12 months >150/min; 12–24 months >140/min; 2–5 years CRT ≥3s ☑ Dry mucous membrane ☑ Poor feeding in infants ☑ Reduced urine output	Reduced skin turgor
Others	None of amber or red symptoms	☑ Age 3–6 months; Temperature ≥39°F ☑ Fever ≥5 days ☑ Rigors ☑ Swelling of a limb or joint ☑ Non weight-bearing limb/ not using a extremity	☑ Age <3 months Temperature ≥ 38°F ☑ Non-blanching rash ☑ Bulging fontanelles ☑ Neck stiffness ☑ Status epilepticus ☑ Focal neurological signs ☑ Focal seizures

Source: NICE, National Institute for Health and Care Excellence 2019.

Investigations (Table 2)

- Blood investigations: Blood tests in well children early in their febrile illness play a limited role as they have not been shown to be reliable predictors of SBI.
- An elevated total peripheral white cell count (WCC), or leukocytosis, can occur with infection but also with other conditions such as chronic inflammation, medications, and malignancy.
- A "left shift" refers to the release of immature neutrophils into the circulation and the subsequent rise in absolute neutrophil count (ANC), but this is usually delayed until approximately 24 hours after clinical infection.
- Andreola et al.7 report that CRP and procalcitonin are useful markers in predicting SBI in children with fever without source and that they perform better than WCC and ANC.
- Procalcitonin is the most cost-effective strategy for the detection of SBI in infants with FWS.8 However, these results should be interpreted within the clinical context of the patient and not as a single method for therapeutic decision-making.
- Urine test: The risk of a UTI remains substantial in some fully immunized children and supports rapid testing by urine dipstick or microscopic urinalysis and a urine culture in selected patients. In children who are not toilet-trained, urine should be collected by catheterization. Bag specimens should not be sent for culture because they are frequently contaminated. A clean-voided urine specimen is preferred in toilet-trained children.
- Chest X-ray: The decision to obtain a chest radiograph must be balanced against the potential harms, such as radiation exposure and cost. In a well- child, the high likelihood of benign viral illnesses producing respiratory symptoms must be considered and chest X-ray is often unnecessary. However, in an ill-appearing child where no focus can be identified, a chest X-ray is often beneficial.

TABLE 2: Management based on NICE traffic light system.

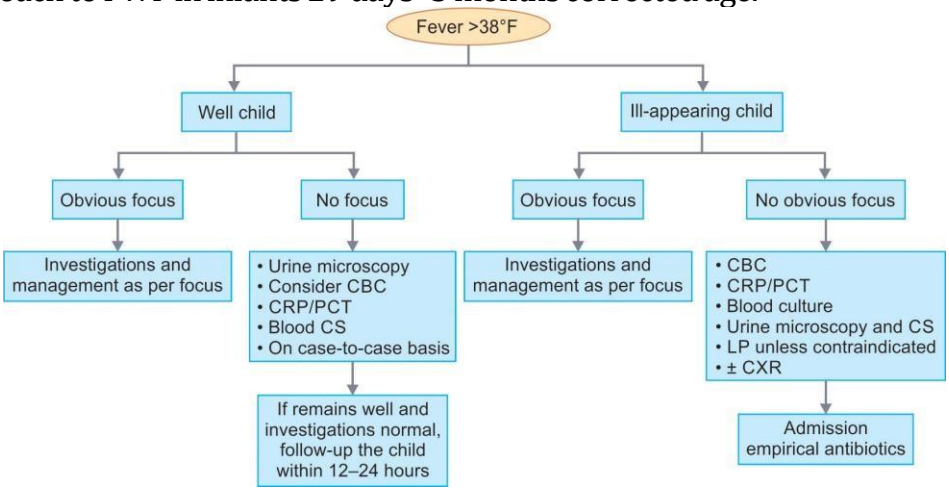
Red	*Amber*	*Green*
☑ CBC ☑ Blood culture ☑ C-reactive protein or Procalcitonin	All the investigations under "red" should be organized unless considered to be unnecessary byan experienced pediatrician	Urine testing
Urine testing	Lumbar puncture should be considered for children <1 year	
Following investigation guided by clinical assessment:	Chest X-ray for a child with fever >39°F and WBC >20 × 10^9/L	

☑ Lumbar puncture ☑ Chest X-ray ☑ Serum electrolytes ☑ Blood gas	even in absence of respiratory symptoms	

(CBC: complete blood count)

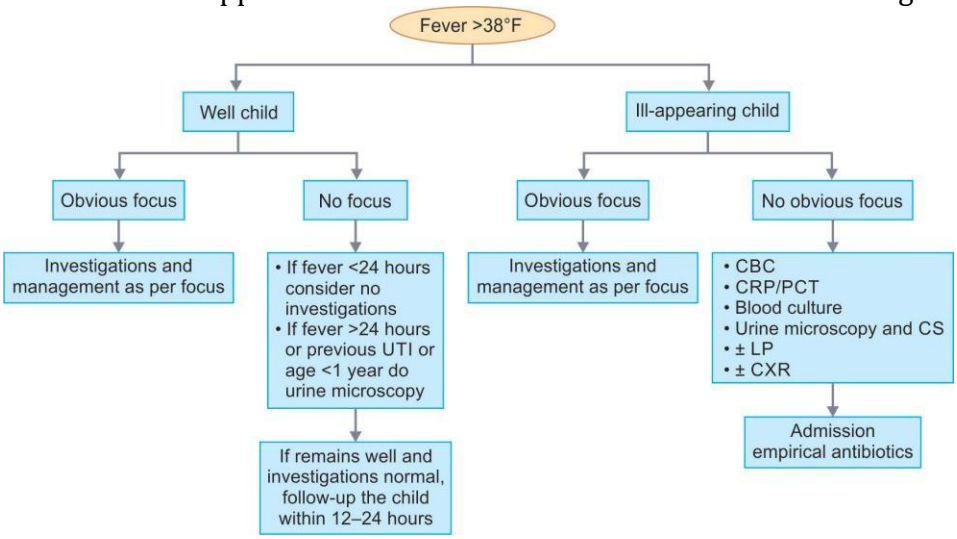
The management algorithm based on age group is shown in Flowcharts 1 and 2: Flowchart 1:

Approach to FWF in infants 29 days–3 months corrected age.



(CBC: complete blood count; CRP: C-reactive protein; PCT: procalcitonin).

Flowchart 2: Approach to FWF in children >3 months corrected age.



(CBC: complete blood count; CRP: C-reactive protein; PCT: procalcitonin).

Red Flags for Serious Illness

- Pale, mottled, ashen or blue skin, lips or tongue
- Ill appearance
- No response to social cues

- Respiratory rate >60 breaths per minute
- Does not wake or, if roused, does not stay awake
- Grunting
- Weak, high-pitched or continuous cry
- Moderate or severe chest indrawing
- Reduced skin turgor
- Bulging fontanelle

Dos and Don'ts

- Do not use the oral or rectal routes routinely to measure the body temperature in children aged 0–5 years.
- Do not use duration of fever to predict the likelihood of a serious illness.
- Response to antipyretic therapy should not be used as a clinical decision-making parameter to differentiate between a serious and non-serious illness.
- Do not prescribe oral antibiotics to children with fever without apparent source.

Key Points

- Presence of fever should be confirmed and documented.
- Every effort must be made to obtain a detailed history and to perform careful physical examination to identify hidden localizing clue to etiology.
- Even if febrile infant 1–3 months of age is assessed to be otherwise "well", screening tests to rule out a serious bacterial infection are ideal beginning with urinalysis.
- In older infants and young children, a methodical clinical approach is often sufficient to guide decision making on further management.
- Counseling on further follow-up is essential until fever subsides or a definite diagnosis is made.

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FEVER: GENERAL MANAGEMENT

What is fever? Is it a friend or a foe?

Normal body temperature is around 37°C (98.6°F), plus or minus about 0.6°. When the body detects any infection or inflammation, the brain responds by raising the body temperature to help fight the condition. So, fever is sort of body's defense mechanism against various types of insult. A rectal temperature of over 38°C (100.4°F) is considered as fever. A high body temperature is beneficial to us in two ways—firstly, the raised temperature helps in controlling the disease process, and secondly, it is an important sign which tells us that "all is not well" in the body, and hence prompts us to look for the underlying cause. However, fever does make a child uncomfortable and increases the metabolic needs of the body. Fever itself is neither a friend nor a foe; rather, it is a messenger that brings you notification whenever your body is responding to an insult.

My baby's forehead always feels warm when touched. Is it fever?

Touching is a crude and unreliable method of temperature measurement. A digital thermometer is best for taking temperatures. Do not use a mercury thermometer, as it is toxic and could break. Although the most accurate way to take a temperature is through rectum, it needs some expertise in performing and may not be comfortable for child. The axillary (armpit) method is fairly precise and most commonly used in children. It is however important to realize that axillary temperature is 0.5–1.0°F lower than oral temperature while rectal temperature is 0.5–1.0°F higher than the oral temperature.

Age	*Recommended technique*
Birth to 6 months	1. Rectal (Definitive) 2. Axillary (Screening)
6 months to 5 years	1. Rectal (Definitive) 2. Axillary, tympanic or temporal artery (Screening)
>5 years	1. Oral (Definitive) 2. Axillary, tympanic or temporal artery (Screening)

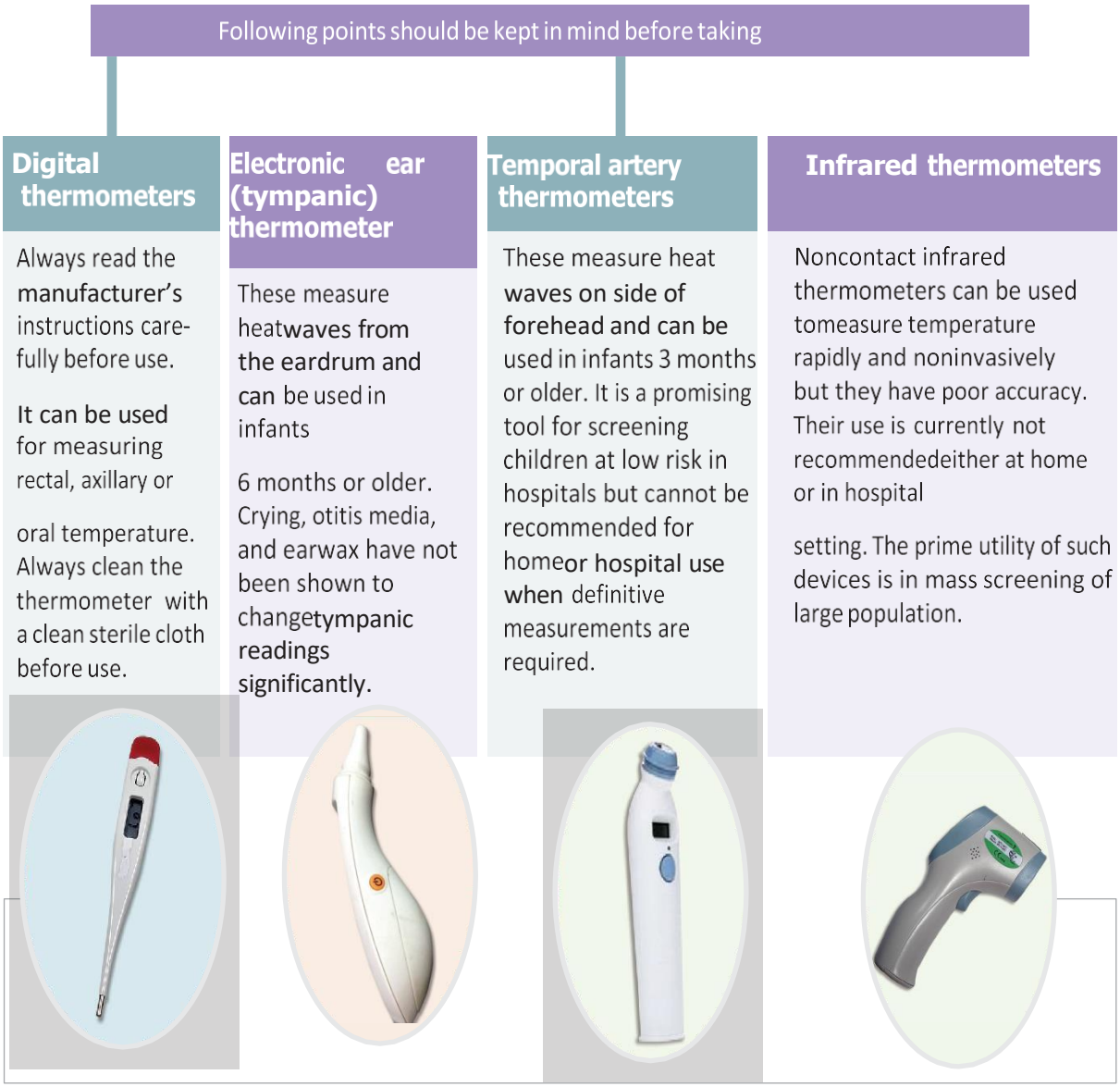


Fig. 1: Different types of thermometers.

Why my child develops fever?

As discussed above, fever is body's defense mechanism and can occur in response to following conditions:

- •• Infections (viral, bacterial, protozoal, and fungal)
- •• Dehydration
- •• Autoimmune diseases
- •• Drugs

- •• Postvaccination
- •• Cancers

Most causes of fever in children are benign and self-limiting. Viral infections are by far the most common cause of fever in children. Your pediatrician may decide to do some tests to know the underlying cause. Fever can also occur postvaccination. Vaccination prevents diseases from infecting the body. The material in vaccine is made up of organisms (viruses/bacteria) causing an infection against which protection is required. These organism's ability to cause illness in the recipient is toned down. When the vaccine enters in the body, it activates immunity cells in the body, which in response to invasion by the organisms produce inflammatory markers, which in turn cause swelling and pain at the site of injection and fever in the body. In most cases, a postvaccination fever will resolve on its own, but your pediatrician may prescribe medicines to reduce the discomfort of child.

What should I do when my child develops fever? Can I send him school if fever is mild?

Whenever a child develops fever, the focus should be on finding the underlying cause. Fever is only a symptom, and its etiology must be established. The main objective of treating fever is to reduce the discomfort of child, rather than to just reduce the temperature.

Do the following to make the child comfortable:

- Do not overdress the child. Have him wear loose clothes, as per season.
- Keep the child well hydrated. Children may lose more fluids during fever.
- Medications (acetaminophen, ibuprofen) can be used in doses recommended by a doctor.
- Tepid sponging with water at 28–30°C can be done to reduce the temperature after medication is administered. Use of icecold water for sponging is not recommended in fever.

Sponging should be done by continuous wiping of the body with tepid water from head to toe for 15–20 minutes. Sponging action ensures that water film is constantly moving thus maximizing heat conduction. Studies indicate that use of hydrotherapy alone is clearly inferior for reduction of fever for periods longer than 30 minutes. However, external cooling could still be of value, if it potentiates activity of antipyretics. Child can also be given a bath with tepid water during fever. Bathing can actually help bring down your fever. Young infants (<3 months old) should not be kept exposed for long due to the risk of hypothermia and sponging may be preferred in this age group.

Mild fever with no other symptoms is usually not a reason enough for a child to stay at home. But many schools or childcare centers request a child to not return until at least 24 hours after a fever has subsided. It is preferred to not send a child to school during febrile illness for following reasons:

- Child may feel weak, uncomfortable, and dehydrated during fever episode making it difficult to sit in school.
- Child may have some contagious underlying infection (most viral infections are highly contagious), which may spread to other children.

Should I give antibiotics for fever?

Fever in children is most commonly caused by viral infections. Antibiotics are drugs used specifically against bacterial infections. They are not effective against viruses, and their indiscriminate use for every febrile illness is not warranted. In a febrile illness, your doctor will decide whether the child needs antibiotics, depending on if he/she is suspecting a bacterial infection.

My child does not want to eat anything during fever. What should I do?
The discomfort and muscle-aches associated with fever may make a child fussy and dull. It is important to maintain hydration during fever, and the child should be encouraged to take small sips of water and light meal during illness. Complete inability to take anything (even fluids and breast milk) from mouth is an indication to visit your doctor.

When should I be worried for fever?

Most febrile illnesses are not serious. However, you should visit the pediatrician immediately if your child is younger than 3 months old or has any of the following:

- •• Extreme lethargy, drowsiness, excessive cry or irritability
- •• Vomiting everything and/or not able to accept feeds orally
- •• Headache, neck stiffness or breathing difficulty
- •• Abnormal body movements or abnormal behavior
- •• Temperature above 104°F
- •• Fever persisting for more than 5 days

Similarly, in a febrile child following signs should make you relaxed:

- •• Playful and active during interfebrile period
- •• Acts like himself/herself during most of the day
- •• Feeding well
- •• Passing urine normally

Is it true that high fever can lead to seizures and brain damage in children?

Febrile seizures are convulsions that can occur in a child with fever. These seizures usually occur in kids 6 months to 5 years old. Children are more likely to have a febrile seizure if they have a family history of same, or if they have already had one in the past. Most children outgrow having febrile seizures by the time, they are 5 years old. There is no evidence that intensity of fever is linked to probability of having febrile seizures or brain damage in children. Also, these seizures do not increase the risk of further epilepsy.

Doctor prescribed paracetamol for my elder son. Can I use the same for my younger child?

Drugs in children are administered according to their body weight. Wrong dosage may lead to toxicity and unwanted side effects. You should always consult your pediatrician before administering any drug to your child. Different brands may have different formulation and strength of medicine. This should be confirmed with pharmacist before buying the drug.

My baby's fever does not come down to normal even after giving paracetamol. Can I use other medicines?

Paracetamol (acetaminophen) is the safest drug for fever to be used in children. If given in correct dose (15 mg/kg body weight), it brings symptomatic relief. Remember, the purpose of fever medicine is not to bring down the temperature to normal level, but to provide symptomatic relief to child by reducing pain and discomfort. If the initial fever was high, say 104°F, administration of paracetamol may bring it down to 101°F and not make the child afebrile. In children who are unable to take orally, your doctor may decide to give paracetamol suppository through rectal route.

Avoid overdosing if the fever does not normalize. You can repeat the next dose after 4–6 hours if required. Other medicines (ibuprofen, mefenamic acid) are also available, and should be used only in consultation with your pediatrician. Though ibuprofen at a dose of 10 mg/kg has similar efficacy as paracetamol in reducing fever, it has more side effects. Sometimes combination of paracetamol and ibuprofen is prescribed to have a rapid response, but it has not been proven to affect the overall outcome. Also, combination drugs have more side effects than individual drugs. Mefenamic acid is not recommended to be used in children owing to its serious side effects. Do not use aspirin or nimesulide for relief of fever in children.

What not to do in fever?

- Do not overclothe the child. Keep him/her in a cool airy environment.
- Do not sponge the child with cold water or ice. Always use tepid water (28–30°C), if needed.
- Do not use aspirin or nimesulide for control of fever. Paracetamol is safest.
- Do not keep treating fever at home or by yourself. Consult a pediatrician at earliest.

NEONATAL SEPSIS

DEFINITIONS AND NOMENCLATURE OF NEONATAL SEPSIS

- Probable sepsis: Is clinical and laboratory findings consistent with bacterial infection
  without a positive culture.
- Clinical sepsis: When the screen and blood/cerebrospinal fluid (CSF) culture is negative, but there is a suggestive history with a high clinical suspicion.
- Culture proven or definitive sepsis: Culture positive sepsis.
- Neonatal sepsis: Best defined as presence of systemic features associated with pure growth of bacteria from one or more sites.
- At risk (ruling out sepsis): Often we "suspect" sepsis based on risk factors and clinical features, but the clinical course (rapid recovery within few hours) and "screening tests" are not suggestive; we should not label these as "suspected sepsis". They are more like "rule out sepsis".

ROUTES OF TRANSMISSION

- Vertical transmission: This transmission is in utero, either hematogenous or through amniotic fluid or during birth. The symptoms usually manifest within the first 72 hours of life.
- Horizontal transmission: These are hospital-acquired infections, majority being lateonset sepsis (>72 hours of life). However, breach in asepsis during resuscitation, immediately after delivery, is also horizontal transmission, but the symptoms may manifest within the first 72 hours of life.

APPROACH TO MANAGEMENT OF NEONATAL SEPSIS

Perinatal Risk Factors

- Rupture of membranes > 24 hours
- Spontaneous preterm labor

- Chorioamnionitis (intra-amniotic infection)
- Prolonged labor
- Unclean per vaginal examinations
- Perinatal asphyxia

Extreme Risk Factors

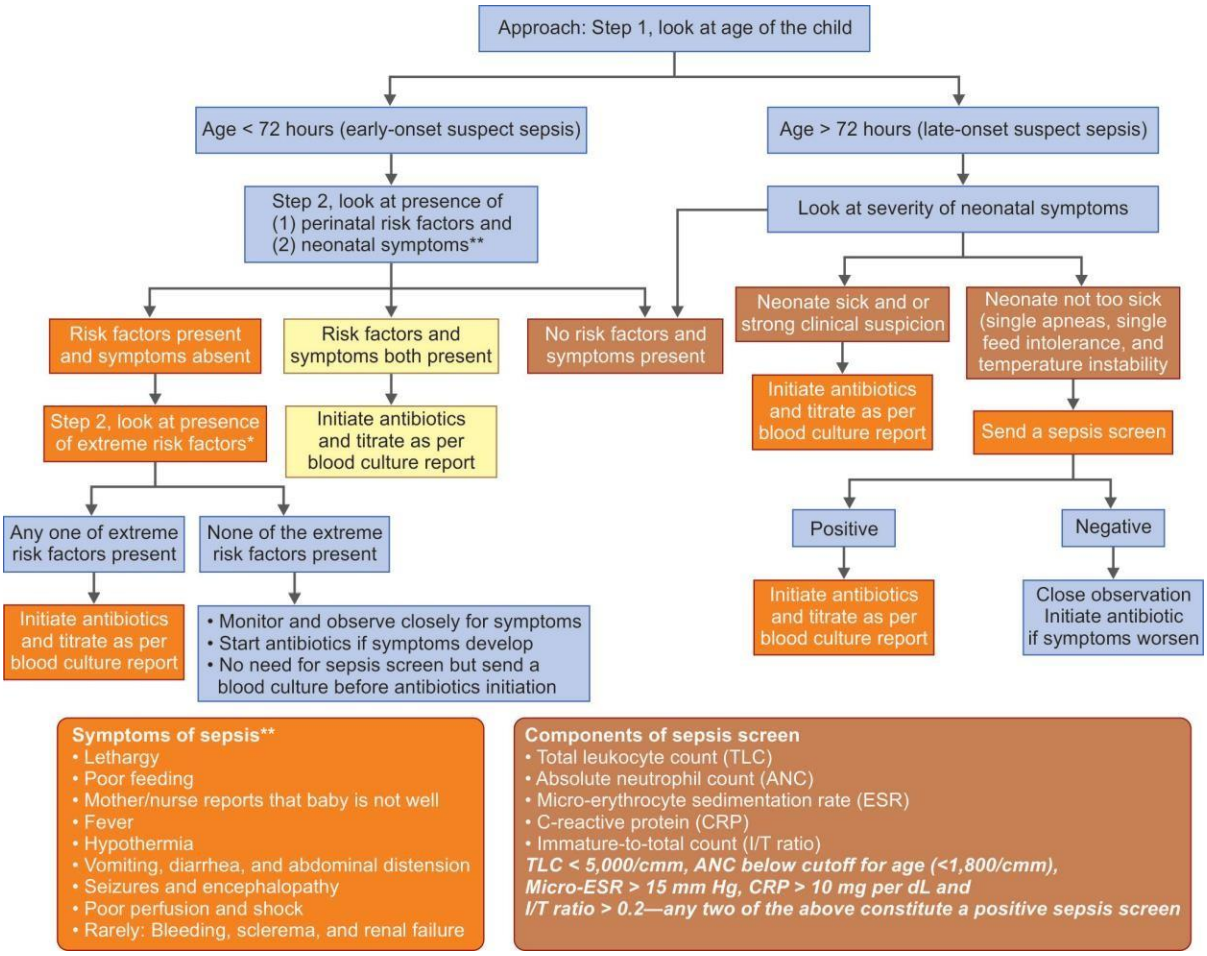
- Rupture of membranes > 72 hours
- Chorioamnionitis (intra-amniotic infection)
- Foul smelling liquor

Definition of Intra-amniotic Infection Or Inflammation Or Both (Triple I) Maternal fever: Maternal oral temperature ≥ 39°C on any one occasion. If oral temperature is 38 (100.4) to 38.9 (102°F), repeat after 30 minutes, if repeat value remains at least 38°C (100.4) it is defined as fever

Suspected triple I: Fever without a clear source plus any of: (1) baseline fetal tachycardia (>160 bpm for >10 minutes)

Maternal total leukocyte count (TLC) > 15,000/cmm in absence of corticosteroids
Definite purulent fluid from cervical os
*Confirmed triple I: All the above plus laboratory findings such as:

- Positive amniotic fluid Gram stain/culture
- Histopathological evidence of infection or inflammation or both in placenta



MANAGEMENT

- It is not possible to recommend a single antibiotic policy for use in all newborn units.
- It should be based on local culture and sensitivity data and profile of organisms for last 6-12 months. If not available, use data from nearby units or National Neonatal Perinatal Database (NNPD).
- Individual antibiotics and rational combinations of antibiotics must be evaluated for the percentage of organisms that they cover. The simplest and cheapest rational combination of antibiotics must be selected for each line
    - •• First line: Must cover approximately 75–80% of isolates
    - •• Second line: Must cover approximately 90–95% of isolates
    - •• Third line: Must cover approximately 95–100% of isolates
- Initial combination should cover both gram-negative and gram-positive organisms. One should use the lowest generation antibiotic combination which would cover about 70% of organisms. This is to ensure you have something to fall back on.

- Avoid cephalosporins as first-line antibiotics—proven harm [high risk of extended- spectrum beta-lactamases (ESBL) organisms, increased Candida infections, necrotizing enterocolitis (NEC), and mortality]—units who do not use have shown multiple benefits.
- Have a written unit/departmental/institutional antibiotic policy and practice antibiotic stewardship (reasons for starting the antibiotics, review the plan for antibiotics at 48 hours and again at 5 days based on culture reports and clinical course, have an exit plan, do not use reserve drugs without consultation)

A broad guide to initial antibiotics is mentioned below:

I. Septicemia or Pneumonia

Birth weight < 2 kg

Antibiotic	*Each dose*	*Frequency*		*Route*	*Duration (Days)*
	0–14 days age	*>14 days age*			
Injection ampicillin* or	50 mg/kg/dose	12 hourly	8 hourly	IV	7–10
Injection cloxacillin#	50 mg/kg/ dose	12 hourly	8 hourly	IV	7–10
AND					
Injection gentamicin	5 mg/kg/dose	24 hourly	24 hourly	IV	7–10

Birth weight ≥ 2 kg

Antibiotic	*Each dose*	*Frequency*		*Route*	*Duration (Days)*
	0–7days age	*>7 days age*			
Injection ampicillin* or	50 mg/kg/dose	12 hourly	8 hourly	IV	7–10
Injection cloxacillin#	50 mg/kg/ dose	12 hourly	8 hourly	IV	7–10
AND					
Injection gentamicin	5 mg/kg/dose	24 hourly	24 hourly	IV	7–10

II. Septicemia Second-Line Drugs

Birth weight < 2 kg

Antibiotic	*Each dose*	*Frequency*		*Route*	*Duration (Days)*
	0–14 days age	*>14 days age*			
Injection piperacillin#					

Tazobactam***	50 mg/kg/dose	12 hourly	8 hourly	IV	7–10
Injection amikacin**	15 mg/kg/ dose	24 hourly	24 hourly	IV	7–10

Birth weight ≥ 2 kg

Antibiotic	*Each dose*	*Frequency*		*Route*	*Duration (Days)*
	0–7 days age	*>7 days age*			
Injection piperacillin#					
Tazobactam***	50 mg/kg/dose	12 hourly	8 hourly	IV	7–10
Injection amikacin**	15 mg/kg/ dose	24 hourly	24 hourly	IV	7–10

III. Meningitis (for Confirmed Meningitis)

Birth weight < 2 kg

Antibiotic	*Each dose*	*Frequency*		*Route*	*Duration (Weeks)*
	0–7 days age	*>7 days age*			
Injection cefotaxime*	50 mg/kg/dose	12 hourly	8 hourly	IV	3
Injection amikacin**	15 mg/kg/ dose	24 hourly	24 hourly	IV	3

Birth weight ≥ 2 kg

Antibiotic	*Each dose*	*Frequency*		*Route*	*Duration (Weeks)*
	0–7 days age	*>7 days age*			
Injection cefotaxime*	50 mg/kg/dose	8 hourly	6 hourly	IV	3
Injection amikacin**	15 mg/kg/ dose	24 hourly	24 hourly	IV	3

IV. Meningitis—Second-line Drugs

Antibiotic	*Each dose*	*Frequency*		*Route*	*Duration (Weeks)*
	0–7 days age	*>7 days age*			
Injection meropenem****	40 mg/kg/dose	8 hourly	8 hourly	IV	3
Injection amikacin**	15 mg/kg/ dose	24 hourly	24 hourly	IV	3

Start if pustules/umbilical sepsis.
* Infuse as an IV infusion using syringe infusion pump over 30 minutes or longer. Use a concentration not >100 mg/mL for infusion.
** Infuse as an IV infusion using syringe infusion pump over 30 minutes or longer. Use a concentration not >5 mg/mL for infusion.

*** Infuse as an IV infusion using syringe infusion pump over 30 minutes or longer. Use a concentration not >50 mg/mL for infusion.
**** Infuse as an IV infusion using syringe infusion pump over 30 minutes or longer.

POINTS TO REMEMBER

- As neonatal sepsis is a dynamic, complex, and heterogeneous condition, intense monitoring (subjective and objective) of the baby is warranted. A repeat sepsis screen within 12 hours may be adopted if initial screen is negative and suspicion of sepsis is strong.
- Process of taking blood culture: Take all aseptic barrier precaution [local disinfection of site with 70% alcohol, povidone iodine (avoid in extremely low birth weight), then alcohol), 1 mL of blood to be put containing at least 5 mL of broth for culture, take blood from fresh puncture site (preferably from freshly inserted intravenous cannula.

FURTHER READING

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PSYCHIATRY STANDARD TREATMENT GUIDELINES

GENERAL TREATMENT GUIDELINES ABOUT DEMENTIA

1. General guidelines about Dementia Annexure 1

a. Thorough history taking and examination including MSE

b. Ask and note down _____(mention ICD/DSM criteria)

1. Consciousness: Conscious, confused, clouding, delirium, stupor, coma.
2. Attention: Digit Forward/Digit Backward (Digit Span test) Ask 3/5 item to remember & ask again
   Write a sentence and ask to do it e.g. pick up the ball pen.
3. Concentration: Serial subtraction 100 - 7, 40 - 3
   - Ask to say days of week] in reverse order, month of year
4. Orientation: Time, Place, Person
5. Memory: Immediate, Recent, Remote
6. Judgment: Personal Judgment, Social Judgment, Test Judgment

2. Investigations required

a. Psychometric investigations: Mini Mental State Examination (MMSE)

b. Radiological: CT or MRI of brain

c. Laboratory: (if physical complications are present)

3. Treatment

a. If patient is in early stage

i) Donepezil 10 mg

ii) Memantine 10 mg

iii) Rivastigmine 1.5 mg

iv) Symptomatic and supportive treatment including nutrition to be given as per the need of the patient

v) If the patient has anxiety or depression or psychotic symptoms, he/she may be treated accordingly as per STG in the relevant section.

Early identification and treatment may treatment may delay or arrest the process of cognitive decline. Treatment of dementia is based on the cholinergic hypopthesis of memory. Cholinesterase inhibitors like donepezil, rivastigmine and galantamine are indicated for patients with dementia. Donepezil should be started with dosage of 5mg/day which may titrate to 10mg/day in 2 weeks. Antipsychotic may be indicated for controlling aggressive behavior, psychotic symptoms and severe agitation. Resperisone (1 to 3 mg) and quetiapine (25 to

400mg) may be used.
b. If patient is in mid or late stage
i) Medication written in 3 (a) may be given as per clinical judgment
ii) Special care may be given to oral, physical and sleep hygiene

4. Follow-up

a. Physical check-up at OPD
b. Through telephonic conversation/TeleManas
c. Follow-up by Targeted Intervention (ARDSI-Mizoram Chapter) in Aizawl

5. Referrals

a. Depending upon the severity and complications of the patient, he/she may need to be referred to medicine/surgery department.
b. To TeleManas
c. To the nearest DMHP
d. To psychiatry OPD at Kulikawn Hospital
e. To higher centers outside Mizoram through Referral Board.

GENERAL TREATMENT GUIDELINES ABOUT ALCOHOL USE DISORDER

1. General guidelines about Alcohol Use Disorder

A chronic disease in which a person craves drinks that contain alcohol and is unable to control his or her drinking

- If a person has 3 or more of the following characteristics for a period of 1 year, then he/she has **Alcohol Use Disorder**

1. Strong desire or compulsion to take the substance.
2. Difficulties in controlling substance – taking behaviour in terms of onset, termination, or levels of use.
3. Physiological withdrawal state when substance use has ceased or been reduced (Anxiety, tremors, headache, nausea, vomiting, insomnia, sweating).
4. Evidence of tolerance - need to increase alcohol dosage to gain the desired effect
5. A lot of time is spent on drinking alcohol; Recreational, social activities are given up because of alcohol use
6. Alcohol used despite knowledge that it created physical and psychological problems.

History Taking and Mental Status Examination
Annexure1

Special emphasis -

- Daily dosage
- length of use and last dosage
- multiple substance use

2. a. Investigations required

1. Psychometric assessment: AUDIT, CAGE, MMSE
2. Laboratory: (if physical complications are present)
    - ✓ Routine CBC
    - ✓ Peptic ulcer
    - ✓ Liver disease
    - ✓ Kidney
    - ✓ Others
    - ✓ LFT
    - ✓ KFT
    - ✓ Urine
    - ✓ Blood sugar
    - ✓ Lipid profile
    - ✓ S. Electrolytes

b. Co morbidity

1. Anxiety Disorder
2. Mood Disorder
3. Somatoform disorder
4. Other substances

c. Differential Diagnosis

- Consider acute head injury and hypoglycaemia. Consider also the possibility of intoxication as the result of substance use. The following five-character codes may be used to indicate whether the acute intoxication was associated with any complications.
- Uncomplicated (Symptoms of varying severity, usually dose-dependent, particularly at high dose levels.
- With trauma or other bodily injury
- With other medical complications, such as haematemesis, inhalation of vomitus.
- With Delerium
- With convulsions
- Intoxication of/with other substance

3. Treatment

a) Psychopharmacology

- Immediate detoxification with Benzodiazepine (preferably long acting)
- Symptomatic and supportive
- Long term use of Disulfiram (if patient desire and is motivated)

Treatment for substance disorder

1. This guideline should be used for substance addiction only.
2. Drug addiction should be diagnosed by MBBS doctors based on ICD 10 and DSM 5 criteria. (E.g. drug addiction, alcohol abuse, withdrawal symptoms, prioritizing drugs over anything else, using substance to the point of incontinence and to the point of physical harm etc)
3. The diagnosis should be made by taking history from a reliable source or informant (personal interviews, telephone interviews, medications and doses, and other important information are important for self-care)
4. Patients history should include: a) What type of drugs, b) Last dose of substance intake, c) Daily dose of drug used, d) Whether the patient take OST or not, e) Does the patient has any other illness?
5. Drug use should be stopped immediately upon entry into the Centre
6. Qualified nurses under the guidance of MBBS doctor should treat the patient. MBBS Doctor should treat the patient with understanding while taking into consideration the mental, physical and

characteristics of the patient.

7. The following are ways to tell if the patient needs to be referred to a more advanced facility:
   - The patient is physically tired and weak
   - The patient is confused and dangerous to others and himself
   - Covid Care Center in a buaipui theih bak chin a thleng tawh ni a ngaihin.

I. PHARMACOLOGICAL DETOXIFICATION

1. Treatment for alcoholics

After entering the centre, the alcoholic must stop drinking alcohol. If he does not need alcohol anymore, he must take his medicine from his daily intake. For example, if the daily dose is Local 500 ml or a bottle Sap Zu, the following medicines should be given considering the effects on the body:

i. Lorazepam 2 mg/ Chlordiazepoxide 10 mg/ Clonazepan 0.5 ng tablets should be given twice a day, i.e. Two (2) tablets in the morning, two (2) tablets in the afternoon and three (3) tablets in the evening. The dose should be gradually reduced daily or weekly over a period of seven to ten days (7-10 days)

ii. Thiarmine 100 mg (Bl -e.g. Beplex Forte) should be given for three days. Thiarnine should be reduced to 10 mg for one month.

iii. If the drug addict is not able to control himself or if he is not able to control his mother, Inj. Diazepan 1 tablet IV or Inj. Lorazepam 1 ampoule each IV/IM.

iv. If Darnlo chå is mentally ill or has a mental illness, Inj. Haloperidol and Inj. Prornethazine 1 ampoule each IM.

v. Treatment should be given according to the patient's illness and severity

II. FOLLOW UP

1. Patient should be advised to revisit Psychiatry OPDs/ DMHP OPDs- district towns after discharge from Centres

2. Patients and their caregivers can call Psychiatrists, Clinical Psychologists and DMHP Mental Health Professionals on State helpline 102 (toll free) if necessary.

b) Psychosocial Intervention

Psychoeducation – nature of illness, etiology, progression, consequences, prognosis, treatment.

For patient	For Caregiver/Guardian

After 3 weeks from consultation/admission	At the time of patient's consultation/admission

i. *Relapse Prevention Therapy -*
a) Identifying the triggers
b) Strategies for coping with triggers (5 D's) delayed, drink, divert,discuss,deep breath

ii. *Motivation Enhancement Therapy*-**Motivational enhancement therapy (MET)** is a directive, person-centered approach to therapy that focuses on improving an individual's motivation to change.

- ✓ Pre contemplation- not interested/not even thinking about behaviour change
- ✓ Contemplation- ambivalent/ considering change
- ✓ Preparation- ready for action/change is necessary and possible
- ✓ Action- initiating action/actively working toward behaviour change
- ✓ Maintenance/recovery- already acting/sustaining new behaviour

iii. *Family counselling*

iv. *Physical Activity / Activity Scheduling*-Activity scheduling (AS), also called behavioral activation (BA), is a therapeutic technique based on the premise that regularly engaging in pleasant activities may help alleviate depression and elevate mood.

- ✓ **Exercising:** Physical Exercises, calisthenics exercises, stretching, brisk walking Playing basketball, going to the gym, or getting out for a hike.
- ✓ **Nurturing relationships:** Going out to dinner, seeing a movie, or attending a play with friends or family
- ✓ **Self-education:** Taking a class, going to the library, reading more
- ✓ **Participating in hobbies:** Taking a cooking class, learning how to knit or paint, learning to play an instrument, learning new languages, new game, carpentry or clay modelling etc
- ✓ **Expanding self-care**: Learning mindfulness techniques, practicing relaxation therapy, visualizing, or doing yoga

a) Combined treatment

4. Follow-up

a. Psychiatry opd/ DMHP
b. Physical check-up at OPD
c. Through telephonic conversation TeleManas14416/18008914416

5. Referrals

Depending upon the severity and complications of the patient, he/she may need to be referred to Psychiatry department.

a) To TeleManas
b) To the nearest DMHP
c) To psychiatry OPD at Kulikawn Hospital or ZMC
d) To higher centers outside Mizoram through Referral Board.

GENERAL TREATMENT GUIDELINES ABOUT OPIOIDS USE DISORDER

1. General guidelines about Opioids Use Disorder

Opioid use disorder (OUD) is a complex illness characterized by compulsive use of opioid drugs even when the person wants to stop, or when using the drugs negatively affects the person's physical and emotional well-being.

- 3 or more of the following characteristics for a period of 1 year:

1. Strong desire or compulsion to take the substance
2. Difficulties in controlling substance – taking behaviour in terms of onset, termination, or levels of use
3. Physiological withdrawal state when substance use has ceased or been reduced (Anxiety, tremors, headache, nausea, vomiting, insomnia, sweating)
4. Evidence of tolerance - need to increase alcohol dosage to gain the desired effect
5. A lot of time is spent on drinking alcohol; Recreational, social activities are given up because of alcohol use
6. Alcohol used despite knowledge that it creates physical and psychological problems

History taking and examination including MSE Annexure 1

Special emphasis -

- Daily dosage
- length of use and last dosage
- also multiple substance use

2. a) Investigations required

i. Psychometric investigations: Alcohol, Smoking and Substance Involvement Screening Test (WHO-ASSIST)
ii. Laboratory: (if physical complications are present)

b) Comorbidity

i. Anxiety Disorder
ii. Mood Disorder

c) Differential Diagnosis

- Consider acute head injury and hypoglycaemia. Consider also the possibility of intoxication as the result of substance use. The following five-character codes may be used to indicate whether the acute intoxication was associated with any complications.
- Uncomplicated (Symptoms of varying severity, usually dose-dependent, particularly at high dose levels.
- With trauma or other bodily injury
- With other medical complications, such as haematemesis, inhalation of

vomitus.

- With Delerium
- Intoxication with other substance

3. Treatment

a) Psychopharmacology
   - Acute intoxification - Naloxone
   - Detoxification with sofer opiates
   - Buprenorphine
   - Symptomatic and supportive

2. Opioid use disorders (Heroin-No 4)

Herion-No 4 dose should be calculated from the average daily dose. for example, if the daily dose is 3 caps (insulin syringe cap), taking this into account the effects of the drug, the following drugs should be given

i. Tramadol tablets 100 mg should be given three times daily, i.e. Two tablets in the morning, two tablets in the afternoon and three tablets in the evening. The dose should be decreased daily or weekly within 7-10 days. If the treatment is still slow, continue for 3 to 5 days.

ii. Nitrazepam 10 rng/Lorazeparn 2 ng/Zolpiden 10 mg mum should be given if the patient cannot take it.

iii. Inj Haloperidol and Inj Promethazine lampule IM, darnlo should be given if the problem or symptoms appear

iv. Treatment should be given according to the patient's condition

3. Multiple Substance Use Disorder

Point No II (1) and (2) above should be used to reduced or remove the drug from the body.

II. FOLLOW UP

1. Patient should be advised to revisit Psychiatry OPDs/ DMHP OPDs- district towns after discharge from Centres
2. Patients and their caregivers can call Psychiatrists, Clinical Psychologists and DMHP Mental Health Professionals on State helpline 102 (toll free) if necessary.

b) Psychosocial Intervention
Psychoeducation – nature of illness, etiology, progression, consequences, prognosis, treatment.

For patient	For caregiver/guardian
After 3 weeks from consultation/admission	At the time of patient's consultation/admission

c) Psychotherapy

i. Relapse Prevention Therapy -
- Identifying the triggers
- Strategies for coping with triggers (5 D's) delayed, drink, divert,discuss,deep breath

ii. Motivation Enhancement Therapy-**Motivational enhancement therapy (MET)** is a directive, person-centered approach to therapy that focuses on improving an individual's motivation to change.
- ✓ pre contemplation- not interested/not even thinking about behaviour change
- ✓ Contemplation- ambivalent/ considering change
- ✓ Preparation- ready for action/change is necessary and possible
- ✓ Action- initiating action/actively working toward behaviour change
- ✓ Maintenance/recovery- already acting/sustaining new behaviour

iii. Family therapy

iv. Physical Activity / Activity Scheduling
- ✓ **Exercising:** Physical Exercises, calisthenics exercises, stretching, brisk walking Playing basketball, going to the gym, or getting out for a hike.
- ✓ **Nurturing relationships:** Going out to dinner, seeing a movie, or attending a play with friends or family
- ✓ **Self-education:** Taking a class, going to the library, reading more
- ✓ **Participating in hobbies:** Taking a cooking class, learning how to knit or paint, learning to play an instrument, learning new languages, new game, carpentry or clay modelling etc
- ✓ **Expanding self-care:** Learning mindfulness techniques, practicing relaxation therapy, visualizing, or doing yoga.

a) Combined Treatment

4. Follow-up

a. Psychiatry opd/ DMHP
b. Physical check-up at OPD

c. Through telephonic conversation TeleManas14416/18008914416

5. Referrals

a) Depending upon the severity and complications of the patient, he/she may need to be referred to Psychiatry/medicine department.
b) To TeleManas
c) To the nearest DMHP
d) To psychiatry OPD at Kulikawn Hospital and ZMC
e) To higher centers outside Mizoram through Referral Board.

GENERAL TREATMENT GUIDELINES ABOUT SCHIZOPHERNIA

1. General guidelines about Schizophrenia

- A major psychotic disorder characterized by abnormalities in the perception or expression of reality. It affects the cognitive and psychomotor functions. Common clinical signs and symptoms include delusions, hallucinations, disorganized thinking, and retreat from reality.
- A severe emotional disorder of psychotic depth characteristically marked by a retreat from reality with delusion formation, hallucinations, emotional disharmony, and regressive behavior.
- A disorder that affects a person's ability to think, feel and behave clearly.
- People may experience
  - **Behavioural:** social isolation, disorganized behaviour, aggression, agitation, compulsive behaviour, excitability, hostility, repetitive movements, self-harm, or lack of restraint
  - **Cognitive:** thought disorder, delusion, amnesia, belief that an ordinary event has special and personal meaning, belief that thoughts aren't one's own, disorientation, mental confusion, slowness in activity, or false belief of superiority
  - **Mood**: anger, anxiety, apathy, feeling detached from self, general discontent, loss of interest or pleasure in activities, elevated mood, or inappropriate emotional response
  - **Psychological**: hallucination, paranoia, hearing voices, depression, fear, persecutory delusion, or religious delusion
  - **Speech:** circumstantial speech, incoherent speech, rapid and frenzied speaking, or speech disorder
  - **Also common:** fatigue, impaired motor coordination, lack of emotional response, or memory loss

Positive Symptoms	Negative Symptoms
Delusions of Grandiose, Reference, Suspiciousness, persecution, Control.	Little/no drive to do things
Hallucination – Auditory, thoughtwithdrawal, Insertion and Interruption, Thought broadcasting, Somatic Hallucination	Lack of energy and intertest
Disorganized speech	Lack of display of feelings
Grossly disorganized or catatonic behavior	Speaking very less

History taking and examination including MSE Annexure 1

2.a) Investigations required

a. Psychometric investigations: Positive and Negative Symptom Scale (PANNS)
b. Laboratory: (if physical complications are present)

b) Comorbidity: Major depression, alcohol and drug induced psychoses, obsessive compulsive disorder and anxiety disorder.

c) Differential Diagnosis: Substance-induced psychotic disorder, Mood disorders with psychotic features, Sleep-related disorders, Delusional disorder, Paranoid personality disorder, Schizotypal personality disorder, Pervasive developmental disorder, Psychosis secondary to organic causes.

3. Treatment

a) Psychopharmacology

Investigations

Though no specific test is diagnostic, but depending on the possible list of differentials for causes of organic psychosis, investigations can be advised. Look for concurrent conditions e.g. alcohol use, signs or symptoms suggestive of stroke/ diabetes/ hypertension/ HIV or AIDS/ cerebral malaria/ medications usage (e.g. steroids, ATT).

Treatment

A comprehensive treatment program includes:

1. Antipsychotic medication, which forms the cornerstone of treatment of psychosis
2. Education of the individual about his/her illness and treatment
3. Family education and support
4. Support groups and social skills training
5. Rehabilitation to improve the activities of daily living
6. Vocational and recreational support

Pharmacological management

After identification of the case, antipsychotic medications should be started depending on the clinical status. The treatment can be broadly divided into two phases: acute and maintenance. The goals of acute phase of treatment are to reduce symptoms, to prevent harm to self/others and improve biological functions. The goal of maintenance phase of treatment is to prevent relapse and to help patient improve one's level of functioning.

Selection of antipsychotic drugs: Two classes of antipsychotic

drugs are available, typical antipsychotics (haloperidol, chlorpromazine, trifluoperazine) and atypical antipsychotics (risperidone, olanzepine, quetiapine). Both the groups are equally effective, but differ in their side effect profiles. In typical antipsychotics, high potency drugs (e.g. haloperidol) have more extra pyramidal side (EPS) effects and low potency drugs (e.g. chlorpromazine, fluphenazine, trifluoperazine) have more anticholinergic side effects (e.g. dryness, urinary retention, constipation) and cardiovascular side effects (e.g. tachycardia, postural hypotension)

Managing side effects

Monitoring for common acute side effects is essential

1. Extrapyramidal side effects (drooling of saliva, rigidity, fine tremors in hands), acute dystinia (sudden sustained contraction of a group of muscles, most commonly neck and oral musculatures are affected), oculogyuric crisis (sudden up rolling of eyeballs), rabbit syndrome (fine perioral tremors). Manage extra pyramidal side effects by reducing antipsychotic dosage or addition of oral anticholinergic drug e.g. trihexyphenidyl or acutely by giving injection promethazine.
2. Cardiovascular side effects (hypotension, bradycardia, QTc prolongation in ECG): These side effects require reducing dosage or switching to other agents.

Antipsychotic	Adult dose range (mg/day)		Side effects
	Acute	Maintenance phase	
Chlorpromazine	100-1600 mg oral; 25-400 mg IM	50-40 mg oral	Sedation, postural hypotension
Trifluoperazine	4-40 mg oral	5-20 mg oral	Sedation, extra pyramidal side effects
Fluphenazine		12.5-50 mg IM (deaconate, fortnightly)	

Haloperidol	5-20 mg oral;	1-5 mg oral;	Sedation, extra pyramidal side effects, dystonia, akathisia, amenorrhea
	5-20 mg IM	25-200 mg IM (deconate, monthly)	
Risperidone	4-6 mg oral		Sedation, extra pyramidal side effects, amenorrhea
Olanzapine	7.5-30 mg oral		Sedation, postural hypotension, weight gain
Quetiapine	300-800 mg oral		Sedation, postural hypotension, dizziness

Non pharmacological management

Psycho-education: Discuss with the patient and family regarding: The person's ability to recover;

- The importance of continuing regular social, educational and occupational activities as far as possible
- The suffering and problems can be reduced with treatment
- The importance of taking medication regularly;
- The right of the person to be involved in every decision that concerns his or her treatment
- Importance of staying healthy (e.g. following healthy diet, staying physically active, maintaining personal hygiene).
- Additional messages to family members of people with psychosis
- The persons with psychosis may hear voices or may firmly believe things that are untrue
- The person with psychosis often does not agree that he or she is ill and may sometimes be hostile
- The importance of recognizing the return/worsening of symptoms and of coming back for re-assessment should be

stressed

- The importance of including the person in family and other social activities should be stressed
- Family members should avoid expressing constant or severe criticism or hostility towards the person with psychosis.
- Person with psychosis may have difficulties recovering or functioning in high-stress working or living environments.
- It is best for the person to have a job or to be otherwise meaningfully occupied.

Follow up care

People with psychosis require regular follow-up. In this phase, particularly general physicians can be of great help. Once a patient is in remission, or behaviourally stable, can continue to follow up locally with nearest general physician. Subsequently, patient can be referred to specialised mental health services only on need basis.

Follow up frequency:

Acute phase: Follow up one or twice

weekly. Maintenance phase: Follow up every

one to three months.

Follow up assessment: During follow up visits, assess for the following:

- Level of symptoms
- Side-effects of medications
- Treatment adherence: Treatment non-adherence is common, address it
- Assess for and manage concurrent medical conditions
- Assess for the need to psychosocial interventions at each follow-up
- Maintain realistic hope and optimism during treatment
- Involvement of carers is critical during such periods

b) Psychosocial Interventions

i) Psychoeducation: Nature of illness, etiology, progression, consequences, prognosis, treatment.

For patient	For caregiver/guardian
After the positive symptoms subside psychoeducation should be given	At the time of consultation

ii) Family Therapy

iii) Support Group and social skills training

b. If patient is in mid or late stage
i) Rehabilitation – *to improve the activities of daily living*
ii) Cognitive Behavioural Therapy (CBT)
iii) Vocational and recreational support

4. Follow-up

a. Psychiatry OPD/ DMHP
- Initially once in two weeks for 2 months
- Monthly check up for 1 year.

b. Physical check-up at OPD
c. Through telephonic conversation/TeleManas 14416/18008914416

5. Referrals

a. Depending upon the severity and complications of the patient, he/she may need to be referred to Psychiatry department.
b. To TeleManas
c. To the nearest DMHP
d. To psychiatry OPD at Kulikawn Hospital or ZMC
e. To higher centers outside Mizoram through Referral Board.

GENERAL TREATMENT GUIDELINES ABOUT DEPRESSION

1. General guidelines about Depression

Depressive disorder (also known as depression) is a common mental disorder which involves a depressed mood or loss of pleasure or loss of interest in activities for long periods of time.

- Following symptoms should have been present for at least 2 weeks.

Symptoms	
• Depressed mood • Loss of interest and enjoyment • Reduced energy leading to fatiguability and diminished activity • Reduced concentration and attention • Reduced self-esteem and self- confidence	• Ideas of guilt • Feeling of worthlessness, helplessness, hopelessness • Bleak and pessimistic views of the future • Ideas of acts of self-harm or suicide • Disturbed sleep • Diminished appetite

History taking and examination including MSE Annexure 1

Special emphasis -

- History of metabolic diseases
- Autoimmune diseases

2. a) Investigations required

i. Psychometric investigations: Becks Depression Inventory-II (BDI-II)

ii. Laboratory: (if physical complications are present)

c) Comorbidity - anxiety disorders,substance use disorders.

d) Differential Diagnosis – Neurological causes such as cerebrovascular accident, multiple sclerosis, subdural hematoma, epilepsy, Parkinsons disease, Alzheimer disease.

3. Treatment

a) Psychopharmacology

- Treatment with Benzodiazepines and SSRI's
- Anti psychotic (if severe)
- Symptomatic and supportive

For a depressive episode: diagnostic criteria

For at least 2 weeks, has the person had at least 2 of the following **Core symptoms:**

Depressed mood (most of the day, almost every day), (for children and adolescents – irritability or depressed mood)

Loss of interest or pleasure in activities that are normally pleasurable

Decreased energy or easily fatigued

Symptoms should be causing significant socio occupational **dysfunction**

Rule out **bereavement** in past 2 months

Rule out the possibility of **bipolar** depression, (Ask about prior episode of manic symptoms such as elevated, expansive or irritable mood, increased activity and talkativeness, flight of ideas, decreased need for sleep, grandiosity, distractibility, loss of social inhibitions and extreme optimism leading to reckless behaviour.

During the last 2 weeks has the person had at least 2 **other features** of depression

Reduced concentration and

attention Reduced self-esteem and

self-confidence Ideas of guilt and

unworthiness

Bleak and pessimistic view of the future

Ideas or acts of self-harm or suicide

Disturbed sleep

Diminished appetite

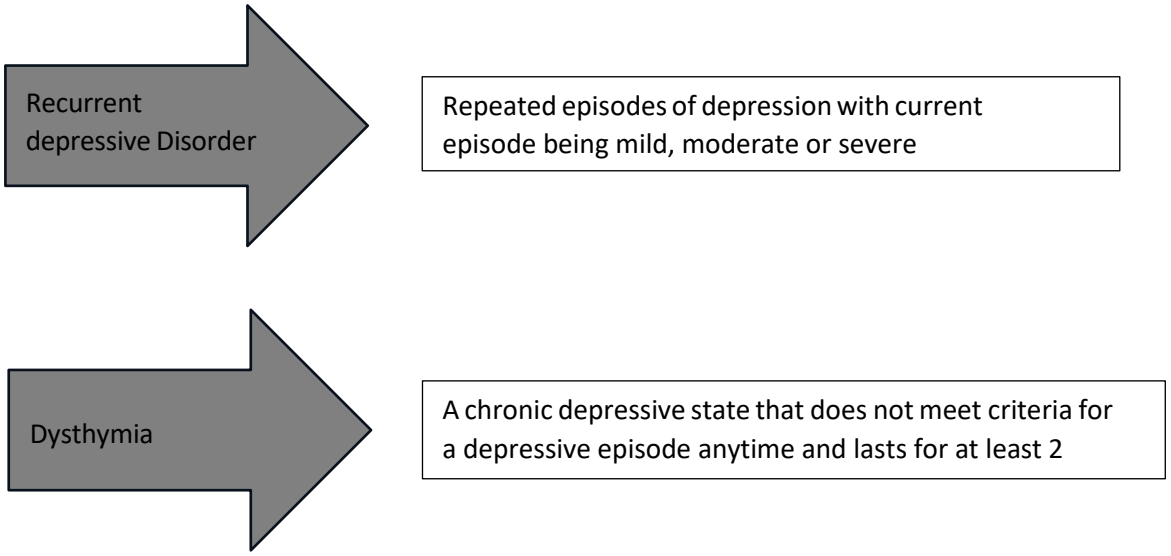
If **2** other features **Mild depressive episode**

If **3** other features **Moderate depressive episode**

If **3** of the core features and **4** other symptoms **Severe depressive episode** it may be **with or without psychotic features**

An episode can be with or without **Somatic syndrome** (4 or more of the following)

loss of interest in activities and lack of emotional reactivity, Loss of appetite, Weight loss, Insomnia mainly as early morning awakening, depression worse in morning. Low libido, Psychomotor retardation or agitation



b) Psychosocial Treatment

i. Psychoeducation – nature of illness, etiology, progression, consequences, prognosis, treatment.

For patient	For Caregiver/guardian
At the time of consultation	At the time of consultation

ii. Psychotherapy

- *Cognitive Behavioural Therapy (CBT)*- It helps you to change unhelpful or unhealthy ways of thinking, feeling and behaving.

- *Physical Activity / Activity Scheduling*

✓ **Exercising:** Playing tennis, golf or basketball, going to the gym, or getting out for a hike

✓ **Nurturing relationships:** Going out to dinner, seeing a movie, or attending a play with friends or family

✓ **Self-education:** Taking a class, going to the library, reading more

✓ **Participating in hobbies:** Taking a cooking class, learning how to knit or paint, or learning to play an instrument

✓ Expanding self-care: Learning mindfulness techniques, practicing relaxation therapy, visualizing, or doing yoga

i. Family counselling

c) Combined Treatment

4. Follow-up

a. Psychiatry opd/ DMHP
b. Physical check-up at OPD
c. Through telephonic conversation TeleManas14416/18008914416

5. Referrals

a) Depending upon the severity and complications of the patient, he/she may need to be referred to Psychiatry department.
b) To TeleManas
c) To the nearest DMHP
d) To psychiatry OPD at Kulikawn Hospital an ZMC
e) To higher centers outside Mizoram through Referral Board.

GENERAL TREATMENT GUIDELINES ABOUT BIPOLAR AFFECTIVE DISORDER

1. General guidelines about Bipolar Affective Disorder

The fundamental disturbance is a change in mood or affect, usually to depression (with or without associated anxiety) or to elation. This disorder is characterized by repeated (i.e.at least two) episodes in which the patient's mood and activity levels are significantly disturbed, this disturbance consisting on some occasions of an elevation of mood and increased energy and activity (mania or hypomania), and on others of a lowering of mood and decreased energy and activity (depression).

Manic episodes usually begin abruptly and last for between 2 weeks and 4-5 months (median duration about 4 months). Depressions tend to last longer (median length about 6 months), though rarely for more than a year, except in the elderly. Episodes of both kinds often follow stressful life events or other mental trauma, but the presence of such stress is not essential for the diagnosis. The first episode may occur at any age from childhood to old age. The frequency of episodes and the pattern of remissions and relapses are both very variable, though remissions tend to get shorter as time goes on and depressions to become commoner and longer lasting after middle age.

Although the original concept of "manic-depressive psychosis" also included patients who suffered only from depression, the term "manic-depressive disorder or psychosis" is now used mainly as a synonym for bipolar disorder.

Manic phase	Depressive phase
Inflated self-esteem or grandiosity	Depressed mood most of the day, nearly every day
Decreased need for sleep	Loss of interest or pleasure in all, or almost all, activities
Increased talkativeness	Significant weight loss or decrease or increase in appetite
Racing thoughts	Engaging in purposeless movements, such as pacing the room
Distracted easily	Fatigue or loss of energy
Increase in goal-directed activity or	Feelings of worthlessness or guilt

Psychomotor agitation	Diminished ability to think or concentrate, or indecisiveness
Engaging in activities that hold the potential for painful consequences, e.g., unrestrained buying sprees	Recurrent thoughts of death, recurrent suicidal ideation without a specific plan, or a suicide attempt

2. a) Investigations required

a. Psychometric investigations: Mood Disorder Questionnaire (MDQ), YBOCS

b) Laboratory: (if physical complications are present)

c) Comorbidity: Anxiety, substance use disorder and conduct disorder.

d) Differential Diagnosis: Personality disorder, Cyclothymia, Psychotic disorder, ADHD.

3. Treatment

a) Psychopharmacology

If present episode is mania/hypomania	Begin treatment of acute mania stabilizer with mood stabilizer (lithium, valporate and carbamazepine) or with antipsychotics Consider short term benzodiazepine such as diazepam for behavioural disturbances or agitation. Discontinue any depressants
If present episode is mania/hypomania	Begin treatment with mood stabilizer Consider antidepressants combined with mood stabilizer for moderate /severe depression with monitoring of switching to mania. However, referral to a specialist should be done. Consider non pharmacological management of mild bipolar depression along with mood stabilizer

National Formulary of India. Antipsychotics for acute mania (Olanzapine, Haloperidol, Chlorpromazine, Fluphenazine) and short term Benzodiazepines (Diazepam, Lorazepam, Alprazolam, Nitrazepam)

When to refer

- If acute episode is severe and not being controlled at primary health care setting
- A patient with bipolar depression
- Medical emergencies comorbid with Bipolar mood disorder
- Special population like elderly, pregnant women and children and adolescent
- Toxicity with drugs

Lithium	Valproate	Carbamazepine
Starting dose: 300 **Usual dose:** 600-1200 **Levels:** 0.6 – 1.0 mEq / litre **Mania:** 0.8 – 1.0 mEq / litre **Maintenance:** 0.6 – 0.8 mEq / litre. Rehular serum level	**Starting dose:** 500 **Usual dose:** 1000-2000	**Starting dose:** 200 **Usual dose:** 400-600

Avoid in pregnancy and advice contraception to woman of child bearing age along with folate supplementation. Lithium also associated with birth defects (mainly cardiac) though at a lower rate than antiepileptic drugs

monitoring critical, Monitor TSH and S. creatinine levels also **Side effects:** Tremors, sedation, weight gain Impaired coordination, polyuria, polydipsia, cognitive problems, cardiac arrhythmias, diabetes insipidus, hypothyroidism START only if clinical and laboratory monitoring is available. Effective against the relapse of both mania and depression Educate about lithium toxicity	Tremors, sedation, weight gain, hepatotoxicity, leukopenia. Watch for hepatitis, pancreatitis, thrombocytopenia, No level monitoring required. Get hepatic status before initiating. Explain the signs and symptoms of blood and liver disorders. Monitor hepatic and blood indices.	Tremors, sedation, weight gain, leukopenia, hepatotoxicity. Watch for diplopia, impaired coordination, rash, liver enzyme Elevations, Stevens – Johnson syndrome, aplastic anaemia. START if both lithium and valproate ineffective or not tolerated. Monitor blood and hepatic parameters.

Primary mood stabiliser can be lithium, valproate or carbamazepine as shown in the table below.

- Take plenty of fluids (summers) with no salt restriction
- Reduce or stop lithium temporarily on developing fever, loose motions, vomiting or any condition leading to fluid loss
- ACE inhibitors, diuretics and NSAIDs along with Lithium increase risk of toxicity
- Gastrointestinal upset, dysarthria, ataxia, coarse tremor followed by impaired consciousness, fasciculation, myoclonus, seizures, and coma are signs of toxicity
- Monitoring for features of lithium toxicity should be routinely done and appropriate referral done accordingly.

b) Psychosocial Intervention

a. If patient is in early stage

i) Psychoeducation:Nature of illness, etiology, progression, consequences, prognosis, treatment.

For patient	For caregiver/guardian
After the manic episode subside psychoeducation should be given	At the time of consultation

ii) Address current psychosocial stressors

iii) Activity scheduling

iv) Physical activities

b. If patient is in mid or late stage

i) Cognitive Behavioural Therapy (CBT)

4. Follow-up

a. Psychiatry OPD/ DMHP

- Initially once in two weeks for 2 months
- Monthly check up for 1 year.

b. Physical check-up at OPD

c. Through telephonic conversation/TeleManas 14416/18008914416

5. Referrals

a. Depending upon the severity and complications of the patient, he/she may need to be referred to Psychiatry department.

b. To TeleManas

c. To the nearest DMHP

d. To psychiatry OPD at Kulikawn Hospital or ZMC

e. To higher centers outside Mizoram through Referral Board.

GENERAL TREATMENT GUIDELINES ABOUT GENERALIZED ANXIETY DISORDER

1. General guidelines about Generalized Anxiety Disorder

Generalized anxiety disorder is a mental health disorder *that produces fear, worry, and a constant feeling of being overwhelmed*causing significant distress/functional impairment.

- Symptoms should be present for most days for at least several weeks at a time, and usually for several months.

Apprehension:	**Motor tension/Physical Symptoms**	**Autonomic Overactivity**
• Worries about future misfortunes. • Fears that suffer or relative will shortly become ill or have an accident. • Feeling "on edge". • Difficulty in concentrating.	• Restless fidgeting. • Tension headaches. • Trembling. • Inability to relax. • Breathing difficulty. • Lump in throat sensation. • Chest pain. • Nausea. • Muscle aches.	• Light Headedness • Sweating. • Palpitation. • Dry Month. • Dizziness. • Epigastric discomfort. • Tachycardia. • Tachypnoea

History taking and examination including MSE Annexure 1

Special emphasis -

- History of thyroid problems and metabolic diseases
- History of substance use

2 a) Investigations required Psychometric assessment: Becks Anxiety Inventory (BAI), GAD- 7 Laboratory: (if physical complications are present)

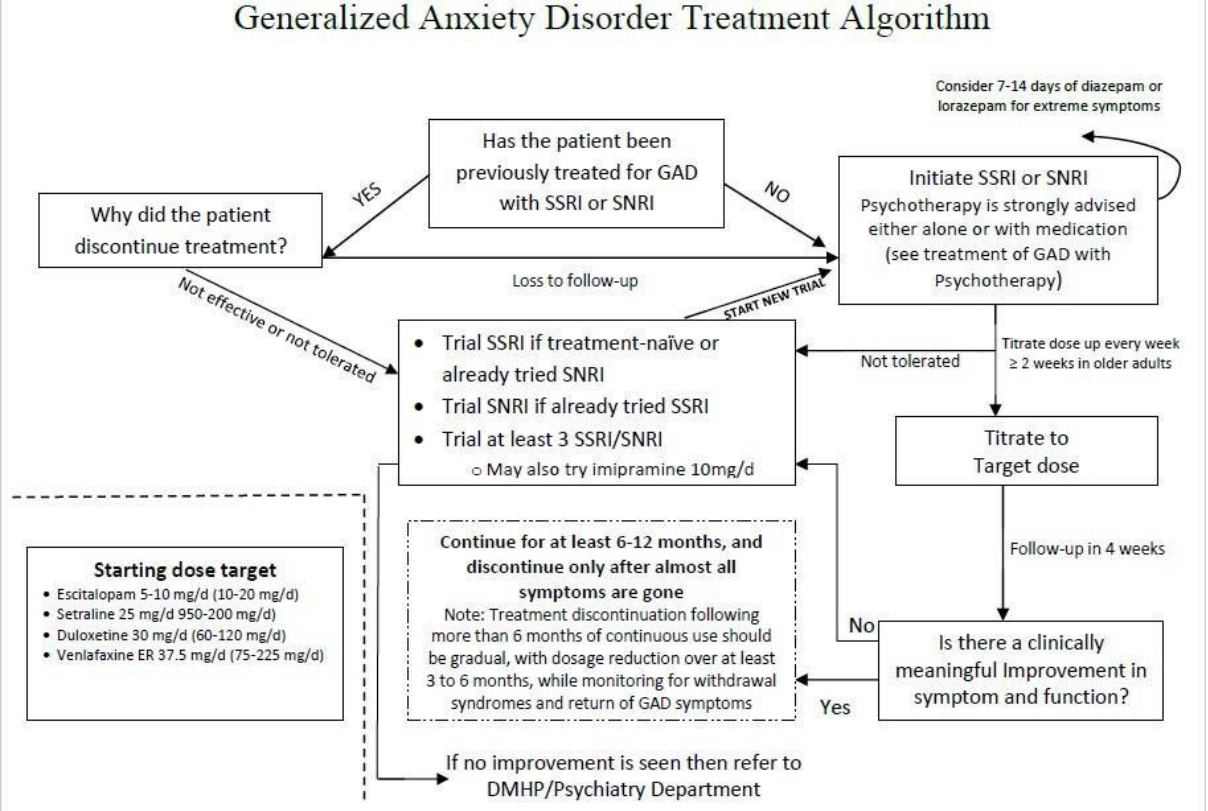
b) Comorbidity – Major depressive disorder, other anxiety disorders especially panic disorder.

c) Differential Diagnosis – drug induced conditions, hyper/hypo thyroidism, Substance abuse,

3. Treatment

a) Psychopharmacology

- Treatment with Benzodiazepines and SSRI's
- Symptomatic and supportive



MANAGEMENT MODALITIES

Pharmacological	Non Pharmacological
❖ Short term treatment with benzodiazepines (BZD's) ❖ Withdraw BZD's after initial 2-4 weeks. ❖ Always taper BZD's ❖ Long term treatment with antidepressants preferably Selective Serotonin Reuptake Inhibitors (SSRI's) followed by tricyclic antidepressants and MAO inhibitors	❖ Psycho education about the disorder ❖ Exploring the concomitant stressors and addressing the worried in a reassuring, emphatic and neutral manner ❖ Cognitive behavioural therapy (CBT) (include relaxation, biofeedback and addressing cognitive distortions) ❖ Supportive psychotherapy ❖ Insight oriented psychotherapy ❖ Regular exercises, yoga, breathing exercises may be advised

SPECIAL CONSIDERATIONS DISORDER WISE

Phobic anxiety disorder: pharmacotherapy with SSRI's, beta blockers (performance anxiety) or CBT (systematic desensitization)

Panic disorder: pharmacotherapy with SSRI's, beta blockers or CBT involving relaxation training and cognitive remodelling

Generalized anxiety disorder: short term management with BZD's + long term CBT or SRRI's

Obsessive compulsive disorder: Either CBT as Exposure and Response Prevention (ERP) or antidepressants (SRRI's or Clomipramine) or a combination of both

Acute stress disorder: short term BZD's + Debriefing in groups post trauma or pharmacotherapy

Post-traumatic stress disorder: CBT (systematic desensitization), eye movement desensitization and reprocessing (EMDR) or pharmacotherapy (SSRI's or imipramine, amitriptyline

COMORBID CONDITOINS WITH ANXEITY DISORDERS

General medical conditions	**Other psychiatric disorders**
Endocrine Hyperthyroidism, hypoparathyroidism, Cushing's syndrome, hypoglycaemia, Pheochromocytoma, Addison's disease **Pulmonary** Asthma, hyperventilation **Cardiovascular** Anaemia, cardiac failures, mitral valve prolapse, hypertension, angina, myocardial infraction **Neurological** Infarcts, haemorrhage, epilepsy, migraine, Wilson's disease, basal ganglia disease like Sydenham's chorea and Huntington's disease (OCD) **Metabolic** Dyselectrolytemia, renal/hepatic failures **Nutritional** Vitamin B 12, Folate deficiency **Tumours** Cerebral/systemic	Substance use disorders Schizophrenia Mood disorders **Drugs Withdrawal of** Alcohol, opiates, sedatives, hypnotics **Intoxication** Nicotine, amphetamine, cocaine, theophylline, amyl nitrite, hallucinogens

The following table is an outline for the use of drugs for treating **anxiety disorders**.

Group of drug	Dosages (mg/day)	Adverse effects
Selective serotonin reuptake Inhibitors (SSRI's) Sertraline Escitalopram* Paroxetine Citalopram Fluoxetine*	50-200 10-20 20-50 20-40 20-60 100-300	Sleep disturbance, gastrointestinal side effects, headache anxiety, prolonged bleeding time, hyponatremia, sexual dysfuntion, discontinuation syndrome
Tricyclic antidepressants (TCA) Clomipramine Imipramine* Amitriptyline	75-300 75-300 75-200	Sedation, hypotension, cardiac side effects mainly prolonged QT interval, anticholinergic effects, weight gain
Benzodiazepines (BZD's) **Clonazepam** **Alprazolam*** **Lorazepam***	0.5-2 0.5-4 1-4 5-15	On long term use-Tolerance, dependence and withdrawal, ataxia, dizziness, daytime drowsiness, amnesia, in overdose respiratory depression
Beta-blockers Propranolol	10-20 mg bid or tds	Hypotension, bradycardia, worsening of asthma and hypoglycaemia, GI side effects

*In National formulary of India

WHEN TO REFER

- If 2 adequate trial of treatments {antidepressant (usually 8-16 weeks) or psychotherapy or combination} fails
- Need for specialized psychological intervention and lack of resources
- Severe or chronic disorders or those with comorbid psychiatric conditions that need multidimensional approach
- Secondary depressive features are severe enough to warrant independent

b) Psychosocial Interventions
Psychoeducation – nature of illness, etiology, progression, consequences, prognosis, treatment.

For patient	For caregiver/guardian
At the time of consultation	At the time of consultation

c) Psychotherapy
   i. *Relaxation Techniques- Breathing exercise*
   ii. *Flooding*
   iii. *Systematic desensitization*
   iv. *Cognitive Behavioural Therapy (CBT)*-It help you to change unhelpful or unhealthy ways of thinking, feeling and behaving.
   v. *Family counselling*

d) Combined treatment

4. Follow-up

a. Psychiatry opd/ DMHP
b. Physical check-up at OPD
c. Through telephonic conversation TeleManas14416/18008914416

5. Referrals

a) Depending upon the severity and complications of the patient, he/she may need to be referred to Psychiatry department.
b) To TeleManas
c) To the nearest DMHP
d) To psychiatry OPD at Kulikawn Hospital or ZMC
e) To higher centers outside Mizoram through Referral Board.

GENERAL TREATMENT GUIDELINES ABOUT PANIC DISORDER, PTSD

1. General guidelines about Panic Disorder, PTSD

Panic attack is the sudden, abrupt, unpredictable, recurrent attacks of severe anxiety, that lasts only for minutes, with the persistent fear of having another attack.

- For a definite diagnosis, several severe attacks of autonomic anxiety should have occurred within a period of about 1 month

Physical Symptoms	Psychological Symptoms
• Palpitations/ chest pain • Choking sensation • Breathing difficulty • Dizziness • Light headedness • Dry mouth	• Feelings of unreality (depersonalization & derealization) • Fear of dying • Fear of losing control • Fear of going mad • Crescendo of fears

History taking and examination including MSE Annexure 1

Special emphasis -

- History of thyroid problems and metabolic diseases
- History of substance use

2. a) Investigations required

i. Psychometric investigations: Becks Anxiety Inventory, HAM-A

ii. Laboratory: (if physical complications are present)

b) Comorbidity

For Panic Disorder	For PTSD
Other anxiety disorders especially GAD, Agoraphobia, Adjustment Disorders, Acute stress reaction, OCD	Depression, Substance abuse

c) Differential Diagnosis – It may be secondary to depressive disorders, particularly in men, and if the criteria for the depressive disorders are fulfilled at the same time, the panic disorder should not be given as the main diagnosis.

3. Treatment

a) Psychopharmacology

- Treatment with Benzodiazepines and SSRI's
- Symptomatic and supportive

b) Psychotherapy

i. *Relaxation Techniques*

ii. *Crisis management*

iii. *Cognitive Behavioural Therapy (CBT)* -It help you to change unhelpful or unhealthy ways of thinking, feeling and behaving.

iv. *Family counselling*

v. *Play therapy* – It is a form of therapy used primarily for children. That is because children may not be able to process their own emotions or articulate problems to parents or other adults. It can also help the child explore emotions and deal with unresolved trauma.

- ✓ Creative visualization
- ✓ Story telling
- ✓ Role play
- ✓ Arts and crafts
- ✓ Toy phones, blocks, construction toys
- ✓ Dance and creative movement
- ✓ Musical play

c) Combined Treatment

4. Follow-up

a. Psychiatry opd/ DMHP
b. Physical check-up at OPD
c. Through telephonic conversation TeleManas14416/18008914416

5. Referrals

a) Depending upon the severity and complications of the patient, he/she may need to be referred to Psychiatry department.
b) To TeleManas
c) To the nearest DMHP
d) To psychiatry OPD at Kulikawn Hospital an ZMC
e) To higher centers outside Mizoram through Referral Board.

GENERAL TREATMENT GUIDELINES ABOUT SOMATOFORM DISORDER

1. General guidelines about somatoform disorder

Repeated presentation of physical symptoms, together with persistent requests for medical investigations, in spite of repeated negative findings and reassurances by doctors that the symptoms have no physical basis.

- If a person has one or more distressing symptoms for more than 6 months, then he/she has somatoform disorder. (DSM V)
- Disproportionate & persistent thought about physical symptoms.
- Persistently high level of anxiety about health or symptoms.
- Excessive time & energy devoted to these symptoms or health concerns.

History taking and examination including MSE Annexure 1

Special emphasis

- Medical history

2. a) Investigations required

i. Psychometric investigations:
ii. Laboratory: (if physical complications are present)

b) Comorbidity – Major depressive disorder, generalized anxiety disorder and panic disorder.

c) Differential Diagnosis – Hypochondriacal delusions. In addition, the presence of unpleasant and frightening physical sensations can be regarded as a culturally acceptable explanation for the development and persistence of a conviction of physical illness.

3. Treatment

a) Psychopharmacology

- Treatment with SSRI's and if accompanied with anxiety - Benzodiazepine
- Symptomatic and supportive

Treatment of Somatoform Disorders

Somatoform disorders are difficult to treat as patients often cling to the belief that their symptoms have an underlying physical cause. Reassurance by a doctor does not usually help as they feel their doctors cannot find the cause for their symptoms.

A strong doctor-patient relationship is a key to getting help in patients with somatoform disorders. Seeing a single health care provider with experience managing somatoform disorders can help cut

down on unnecessary tests and treatment (Box-2).

The focus of treatment is on improving daily functioning, not on managing symptoms. Stress reduction is often an important part of getting better. Stress management techniques, Counseling for family and friends, Promotion of self-care activities, relaxation and breathing exercises, Lifestyles change and occupational counseling may be useful and can be done at primary level. Medications do not have much of a role except if the disorder is associated with underlying mental illnesses such as depression, anxiety or substance abuse. Still, antidepressants such as amitriptyline, imipramine and selective serotonin reuptake inhibitors (SSRIs) along with short-term anti- anxiety agents such as benzodiazepines have been found effective.31

Specific treatments for somatoform disorders

- ***General advice***

Lifestyle change
Relaxation

- ***Drug treatments***

Antidepressants

Anti-Anxiety drugs (short duration)

- ***Occupational and social***

Occupational counseling

Problem solving for social problems

- ***Psychological treatment***

Cognitive-behavioral and other therapies

b) Psychosocial Intervention – nature of illness, etiology, progression, consequences, prognosis, treatment.
c) Psychoeducation – nature of illness, etiology, progression, consequences, prognosis, treatment.

For patient	For Caregiver/guardian
At the time of consultation	At the time of consultation

Psychotherapy

i. *Relaxation Techniques*
ii. *Cognitive Behavioural Therapy (CBT)* -It help you to change unhelpful or unhealthy ways of thinking, feeling and behaving.

ii. *Family counselling*
iii. *Physical Activity / Activity Scheduling*

- ✓ **Exercising:** Playing tennis, golf or basketball, going to the gym, or getting out for a hike

- ✓ **Nurturing relationships:** Going out to dinner, seeing a movie, or attending a play with friends or family
- ✓ **Self-education:** going to the library, reading more
- ✓ **Participating in hobbies:** Taking a cooking class, learning how to knit or paint, or learning to play an instrument
- ✓ Expanding self-care: Learning mindfulness techniques, practicing relaxation therapy, visualizing, or doing yoga

d) Combined Treatment

4. Follow-up

a. Psychiatry opd/ DMHP
b. Physical check-up at OPD
c. Through telephonic conversation TeleManas14416/18008914416

5. Referrals

a) Depending upon the severity and complications of the patient, he/she may need to be referred to Psychiatry department.
b) To TeleManas
c) To the nearest DMHP
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e) To higher centers outside Mizoram through Referral Board.

GENERAL TREATMENT GUIDELINES ABOUT DISSOCIATIVE DISORDER

1. General guidelines about Dissociative Disorder

- A disorder characterized by the presence of two or more identities with distinct patterns of perception and personality which recurrently take control of the person's behavior; this is accompanied by a retrospective gap in memory of important personal information that far exceeds ordinary forgetfulness. The changes in identity are not due to substance use or to a general medical condition.
- A dissociative disorder in which the individual adopts two or more distinct personalities. Each personality is a fully integrated and complex unit with memories, behavior patterns and social friendships. Transition from one personality to another is sudden.
- At least two distinct and relatively enduring personality states, recurrent episodes of dissociative amnesia, inexplicable intrusions into consciousness (e.g., voices, sense of self, depersonalization and derealization, intermittent functional neurological symptoms, emotion and behavior dysregulation
- Feeling of disconnected from yourself and the world around
- Forgetting about certain time periods, events and personal information
- Feeling uncertain about who you are
- Having multiple distinct identities
- Feeling little or no physical pain

History taking and examination including MSE Annexure 1

Special emphasis

- Medical history

2. a) Investigations required

a. Psychometric investigations: to confirm the diagnosis and to rule out Differential Diagnosis

b. Laboratory: (if physical complications are present)

b) Comorbidity: Post Traumatic Stress Disorder and Depressive disorder, Somatization disorder and borderline personality disorder.

c) Differential Diagnosis: Personality Disorder and Psychotic Disorder.

3. Treatment

a) Psychopharmacology

- SSRI's
- Symptomatic and supportive

b) Psychosocial Intervention

a. If patient is in early stage
i) Psychoeducation– Nature of illness, etiology, progression, consequences, prognosis, treatment.

For patient	For caregiver/guardian
At the time of consultation.	At the time of consultation

ii) Talk therapy

b. If patient is in mid or late stage
i) Cognitive Behaviour Therapy
ii) Support group

4. Follow-up

a. Psychiatry OPD/ DMHP
- Initially once in two weeks for 2 months
- Monthly check up for 1 year.

b. Physical check-up at OPD
c. Through telephonic conversation/TeleManas 14416/18008914416

d. Follow-up by Targeted Intervention (ARDSI-Mizoram Chapter) in Aizawl.

5. Referrals

a. Depending upon the severity and complications of the patient, he/she may need to be referred to Psychiatry/medicine department.
b. To TeleManas
c. To the nearest DMHP
d. To psychiatry OPD at Kulikawn Hospital and ZMC
e. To higher centers outside Mizoram through Referral Board.

GENERAL TREATMENT GUIDELINES ABOUT SLEEP DISORDER

1. General guidelines about Sleep Disorder

- Conditions characterized by disturbances of usual sleep patterns or behaviors; divided into three major categories: dyssomnias (i.e. Disorders characterized by insomnia or hypersomnia), parasomnias (abnormal sleep behaviors), and sleep disorders secondary to medical or psychiatric disorders.
- The most common kinds are
  - Insomnia - a hard time falling or staying asleep
  - sleep apnea - breathing interruptions during sleep
  - restless legs syndrome- a tingling or prickly sensation in the legs
  - narcolepsy - daytime "sleep attacks"

Nightmares, night terrors, sleepwalking, sleep talking, head banging, wetting the bed and grinding your teeth are kinds of sleep problems called parasomnias.

Rapid eye movement (REM) sleep behavior disorder (RBD) is a parasomnia characterized by dream-enactment behaviors that emerge during a loss of REM sleep atonia. RBD dream enactment ranges in severity from benign hand gestures to violent thrashing, punching, and kicking. Patients typically present to medical attention with a concern related to injurious or potentially injurious actions to themselves and/or their bed partner.

Non rapid eye movement (NREM) parasomnias are abnormal behaviors arising primarily but not exclusively during non-REM stage three (N3) sleep. Phenotypes include sleepwalking, sleep terrors, confusional arousals, sexsomnia, and sleep-related eating disorder (SRED)

People may experience

- Excessive daytime sleeping
- Irregular breathing or increased movement during sleep
- Irregular sleep and wake cycle
- Difficulty falling sleep
- Restless
- Feeling an uncomfortable urge to move while trying to fall asleep
- Behavioural changes like difficulty focusing or paying attention
- Mood changes like irritability and trouble managing emotions

History taking and examination including MSE Annexure 1

Special emphasis

- Medical history

2. a) Investigations required

a. Psychometric investigations: Insomnia Severity Index (ISI)
b. Laboratory: (if physical complications are present)

b) Comorbidity: Depression, anxiety and ADHD
c) Differential Diagnosis: PTSD, Depression, Anxiety Disorder, Substance abuse and Bipolar Disorder

3. Treatment

a) Psychopharmacology

Refer to benzodiazepines and SSRIs given for treatment in General Anxiety Disorder

b) Psychosocial Intervention

a. If patient is in early stage
i) Psychoeducation: Nature of illness, etiology, progression, consequences, prognosis, treatment.

For patient	For caregiver/guardian
At the time of consultation.	At the time of consultation

ii) Sleep hygiene – changing sleep routine to promote regular sleep schedule and proper sleep hygiene

- 10 Points in Sleep Hygiene
  - Maintain a consistent sleep timing going to bed and waking including the weekend
  - Bedroom should be comfortable, quiet, dark, relaxing and pleasant temperature
  - Avoid/Reduce screen time
  - Avoid/Reduce large heavy dinner
  - Avoid/Reduce caffeine, tea, alcohol
  - Be physically active during the day, exercises/meditation
  - Avoid heavy exercises at night
  - Avoid napping during the day
  - Associate bed with sleeping alone

iii) Creating comfortable sleep environment

b. If patient is in mid or late stage
i) Cognitive Behaviour Therapy

4. Follow-up

a. Physical check-up at OPD
- Initially once in two weeks for 2 months
- Monthly check up for 1 year.

b. Physical check-up at OPD
c. Through telephonic conversation/TeleManas 14416/18008914416

5. Referrals

a. Depending upon the severity and complications of the patient, he/she may need to be referred to Psychiatry/medicine department.
b. To TeleManas
c. To the nearest DMHP
d. To psychiatry OPD at Kulikawn Hospital and ZMC
e. To higher centers outside Mizoram through Referral Board.

GENERAL TREATMENT GUIDELINES ABOUT EATING AND SEXUAL DISORDER

1. General guidelines about Eating and Sexual Disorder Eating Disorder

- A broad group of psychological disorders with abnormal eating behaviors leading to physiological effects from overeating or insufficient food intake.
- A group of disorders characterized by physiological and psychological disturbances in appetite or food intake.
- Eating disorders are serious behavior problems. They include
  - anorexia nervosa, in which you become too thin, but you don't eat enough because you think you are fat
  - bulimia nervosa, involving periods of overeating followed by purging, sometimes through self-induced vomiting or using laxatives
  - binge-eating, which is out-of-control eating

  women are more likely than men to have eating disorders. They usually start in the teenage years and often occur along with depression, anxiety disorders and substance abuse.

Sexual Disorder

Sexual Dysfunctions are syndromes that comprise the various ways in which adult people may have difficulty experiencing personally satisfying, non-coercive sexual activities. Sexual response is a complex interaction of psychological, interpersonal, social, cultural and physiological processes and one or more of these factors may affect any stage of the sexual response. In order to be considered a sexual dysfunction, the dysfunction must:

1) occur frequently, although it may be absent on some occasions;
2) have been present for at least several months; and
3) be associated with clinically significant distress.

Approximate Synonyms

- Abnormal sexual function
- Sexual dysfunction Clinical Information
- Change in sexual function that is viewed as unsatisfying, unrewarding, inadequate
- Deleterious change in sex response

Eating Disorder		**Sexual Disorder**
Anorexia nervosa	Bulimia nervosa	
Extremely restricted eating	Chronically inflamed and sore	Absent or delayed ejaculation despite enough sexual stimulation

	throat	
Extreme thinness (emaciation)	Swollen salivary glands in the neck and jaw area	Inability to control the timing of ejaculation (early or premature)
A relentless pursuit of thinness and unwillingness to maintain a normal or healthy weight	Worn tooth enamel and increasingly sensitive and decaying teeth as a result of exposure to stomach acid	Inability to achieve orgasm
Intense fear of gaining weight	Acid reflux disorder and other gastrointestinal problems	Lack of interest in or desire for sex
Distorted body image, a self-esteem that is heavily influenced by perceptions ofbody weight and shape, or a denial of the seriousness of low body weight	Intestinal distress and irritation from laxative abuse	Inability to become aroused
	Severe dehydration from purging of fluids	Pain with intercourse
	Electrolyte imbalance (too low or too high levels of sodium, calcium, potassium, and other minerals) which can lead to stroke or heart attack	Inability to achieve or maintain erection

History taking and examination including MSE **Annexure 1**

Special emphasis

- Medical history

2.a) Investigations required

a. Psychometric investigations: Sexual Health History taking perfoma
b. Laboratory: (if physical complications are present)

b) Comorbidity: Eating Disorder – Mood disorder, Anxiety Disorder, Post Traumatic Stress disorder, substance use disorder and

Personality Disorder.
Sexual Disorder- Hypertension, High cholesterol, Depression, Anxiety and Diabetes.

c) Differential Diagnosis: Eating Disorder- Bipolar Affective disorder, Depressive Disorders and Borderline Personality Disorder. Sexual Disorder- Depression

3. Treatment

a) Psychopharmacology

- Only if anxiety and depression is comorbid
- Symptomatic and supportive (if necessary)

b) Psychosocial Intervention

a. If patient is in early stage

i) Psychoeducation – Nature of illness, etiology, progression, consequences, prognosis, treatment.

For patient	For caregiver/guardian
At the time of consultation.	At the time of consultation

ii) Family-based therapy –to help parents or individuals with anorexia nervosa assume responsibility for feeding their child, appears to be very effective in helping people gain weight and improve eating habits and moods.

iii) Behavioural treatments -*These involve various techniques, including insights into harmful behaviors in the relationship, or techniques such as self-stimulation for treatment of problems with arousal and/or orgasm.*

b. If patient is in mid or late stage

– Cognitive Behaviour Therapy *to help address sexual trauma from the past, feelings of anxiety, fear, guilt and poor body image. All of these factors may affect sexual function.*

– *helps a person learn how to identify distorted or unhelpful thinking patterns and recognize and change inaccurate beliefs.*

4. Follow-up

a. Psychiatry OPD/ DMHP

- Initially once in two weeks for 2 months
- Monthly check up for 1 year.

b. Physical check-up at OPD

c. Through telephonic conversation/TeleManas 14416/18008914416

5. Referrals

a. Depending upon the severity and complications of the patient, he/she may need to be referred to Psychiatry department.

b. To TeleManas

c. To the nearest DMHP

d. To psychiatry OPD at Kulikawn Hospital an ZMC
e. To higher centers outside Mizoram through Referral Board.

GENERAL TREATMENT GUIDELINES ABOUT CHILDHOOD AUTISM

1. General guidelines about Childhood Autism The onset is usually before the age of 3 years

Pervasive developmental disorder defined by the presence of abnormal and/or impaired development in areas of social interaction, communication and restricted, repetitive behaviour.

Social interaction and reciprocity	Patterns of Communication	Restricted, Stereotyped, Repetitive Behaviour
• Lack of responses to other people's emotions • Lack of modulation of behaviour according to social context • Poor use of social signals • Poor social communication (lack of eye-contact, lack of social smile, etc.)	• Lack of use of language • Impairment in make- believe and social initiative play • Lack of reciprocity in conversational interchange • Poor flexibility in language expression	• Rigidity in routine, interests, activities incl. play patterns • Attachment to unusual, typically non-soft objects • Motor stereotypes

History taking and examination including MSE Annexure 1

Special emphasis

- Medical history

2. a) Investigations required

i. Psychometric investigations: Indian Scale for Assessment of Autism (ISAA), Childhood Autism Rating Scale (CARS)

ii. Laboratory: (if physical complications are present)

b) Comorbidity

c) Differential Diagnosis – specific developmental disorder of receptive language with secondary socio-emotional problems; reactive attachment disorders or disinhibited attachment disorders; mental retardation with some associated emotional or

behavioural disorders; schizophrenia of unusually early onset; and Rett's syndrome

3. Treatment

a) Psychopharmacology
- Only if anxiety or depression is comorbid

b) Psychosocial Intervention

Psychoeducation – nature of illness, etiology, progression, consequences, prognosis, treatment.

For patient	For Caregiver/guardian
At the time of consultation	At the time of consultation

Psychotherapy

i. *Play therapy* – Play therapy is a form of therapy used primarily for children. That is because children may not be able to process their own emotions or articulate problems to parents or other adults. It can also help the child explore emotions and deal with unresolved trauma.
- ✓ Creative visualization
- ✓ Story telling
- ✓ Role play
- ✓ Arts and crafts
- ✓ Toy phones, blocks, construction toys
- ✓ Dance and creative movement
- ✓ Musical play

ii. *Behaviour Therapy* -Behavioral therapy is a term that describes a broad range of techniques used to change maladaptive behaviors. Behavior techniques use reinforcement , punishment, shaping, modelling, and related technique to alter behavior

iii. *Occupational Therapy*-Occupational therapy is a healthcare profession that involves the use of assessment and intervention to develop, recover, or maintain the meaningful activities, or occupations, of individuals, groups, or communities. It also can help them regain independence in all areas of their lives.

iv. Family counselling

c) Combined Treatment

4. Follow-up

a. Psychiatry OPD/ DMHP
b. Physical check-up at OPD

c. Through telephonic conversation TeleManas14416/18008914416

5. Referrals

a) Depending upon the severity and complications of the patient, he/she may need to be referred to Psychiatry/medicine department.
b) To TeleManas
c) To the nearest DMHP
d) To psychiatry OPD at Kulikawn Hospital and ZMC
e) To higher centers outside Mizoram through Referral Board.

GENERAL TREATMENT GUIDELINES ABOUT ATTENTION DEFICIT HYPERACTIVITY DISORDER (ADHD)

1. General guidelines about ADHD

Attention-deficit/hyperactivity disorder (ADHD) is marked by an ongoing pattern of inattention and/or hyperactivity-impulsivity that interferes with functioning or development. People with ADHD experience an ongoing pattern of the following types of symptoms:

- **Inattention** means a person may have difficulty staying on task, sustaining focus, and staying organized, and these problems are not due to defiance or lack of comprehension.
- **Hyperactivity** means a person may seem to move about constantly, including in situations when it is not appropriate, or excessively fidgets, taps, or talks. In adults, hyperactivity may mean extreme restlessness or talking too much.
- **Impulsivity** means a person may act without thinking or have difficulty with self- control. Impulsivity could also include a desire for immediate rewards or the inability to delay gratification. An impulsive person may interrupt others or make important decisions without considering long-term consequences.

Inattention	Hyperactivity- Impulsivity
Overlook or miss details and make seemingly careless mistakes in schoolwork, at work, or during other activities	Fidget and squirm while seated
Have difficulty sustaining attention during play or tasks, such as conversations, lectures, or lengthy reading	Leave their seats in situations when staying seated is expected, such as in the classroom or the office
Not seem to listen when spoken to directly	Run, dash around, or climb at inappropriate times or, in teens and adults, often feel restless
Find it hard to follow through on instructions or finish schoolwork, chores, or duties in the workplace, or may start tasks but lose focus and get easily sidetracked	Be unable to play or engage in hobbies quietly
Have difficulty organizing tasks and activities, doing tasks in sequence, keeping materials and belongings in order, managing time, and meeting deadlines	Be constantly in motion or on the go, or act as if driven by a motor

Avoid tasks that require sustained mental effort, such as homework, or for teens and older adults, preparing reports, completing forms, or reviewing lengthy papers	Talk excessively
Lose things necessary for tasks or activities, such as school supplies, pencils, books, tools, wallets, keys, paperwork, eyeglasses, and cell phone	Answer questions before they are fully asked, finish other people's sentences, or speak without waiting for a turn in a conversation
Be easily distracted by unrelated thoughts or stimuli. Be forgetful in daily activities, such as chores, errands, returning calls, and keeping appointments	Have difficulty waiting one's turn Interrupt or intrude on others, for example in conversations, games, or activities

History taking and examination including MSE Annexure 1

Special emphasis

- Medical history

2. a) Investigations required

a. Psychometric investigations: Vanderbilt ADHD Diagnostic Rating Scale

b. Laboratory: (if physical complications are present)

b) Comorbidity: In children autism spectrum disorder and in Adolescents Anxiety Disorder.

c) Differential Diagnosis: Anxiety Disorder, Bipolar Disorder, depression, Dysthymic disorder, Posttraumatic Stress Disorder, Sleep wake disorder.

3. Treatment

a) Psychopharmacology

- Methylp henidate
  - Amphetamine
  - Antidepressants

MANAGEMENT

Pharmacotherapy

The CNS stimulants. These drugs reduce hyperactivity and improve attention span. Dextroamphetamine and Methylphenidate are the drug of choice. They are to be given in the morning and at noon because nighttime dose may produce sleep difficulty.

Dextroamphetamine is given in a dose of 5-10 mg/day. Methylphenidate in

doses of 0.25 - 1 mg/kg/day is effective. Non-stimulant: Atomoxetine (1-1.4 mg/kg qd)

Psychological Treatment

The parents and teachers are advised not to retaliate against the child but their hyperactivity could be channelized into outdoors sports and their poor attention can be improved by appropriate educational technology.

b) Psychosocial Intervention

a. If patient is in early stage

i) Psychoeducation: Nature of illness, etiology, progression, consequences, prognosis, treatment.

For patient	For caregiver/guardian
At the time of consultation.	At the time of consultation

ii) Parental counselling

iii) Hyperactivity could be channelized into outdoor sports and poor attention can be improved by appropriate educational technology

b. If patient is in mid or late stage

i) Cognitive Enhancement Therapy

ii) Cognitive Behaviour Therapy

4. Follow-up

a. Psychiatry OPD/ DMHP

- Initially once in two weeks for 2 months
- Monthly check up for 1 year.

b. Physical check-up at OPD

c. Through telephonic conversation/TeleManas 14416/18008914416

5. Referrals

a. Depending upon the severity and complications of the patient, he/she may need to be referred to Psychiatry department.

b. To TeleManas

c. To the nearest DMHP

d. To psychiatry OPD at Kulikawn Hospital an ZMC

e. To higher centers outside Mizoram through Referral Board.

GENERAL TREATMENT GUIDELINES ABOUT CONDUCT DISORDER

1. General guidelines about Conduct Disorder

- A disorder diagnosed in childhood or adolescence age group characterized by aggressive behavior, deceitfulness, destruction of property or violation of rules that is persistent and repetitive, and within a one year period.
- A repetitive and persistent pattern of behavior in which the basic rights of others or major age-appropriate societal norms or rules are violated. These behaviors include aggressive conduct that causes or threatens physical harm to other people or animals, nonaggressive conduct that causes property loss or damage, deceitfulness or theft, and serious violations of rules. The onset is before age 18.
- Disorders characterized by a repetitive and persistent pattern of dissocial, aggressive, or defiant conduct. Such behaviour should amount to major violations of age- appropriate social expectations; it should therefore be more severe than ordinary childish mischief or adolescent rebelliousness and should imply an enduring pattern of behaviour (six months or longer). Features of conduct disorder can also be symptomatic of other psychiatric conditions, in which case the underlying diagnosis should be preferred.
- Examples of the behaviours on which the diagnosis is based include excessive levels of fighting or bullying, cruelty to other people or animals, severe destructiveness to property, fire-setting, stealing, repeated lying, truancy from school and running away from home, unusually frequent and severe temper tantrums, and disobedience. Any one of these behaviours, if marked, is sufficient for the diagnosis, but isolated dissocial acts are not.

These children may show any one or more of the following

- Excessive level of fighting or bullying
- Cruelty to animals or other people
- Fire setting
- Stealing
- Repeated lying
- Truancy from school and running away from home
- Frequent and severe temper tantrums, defiant proactive behaviour and persistent severe disobedience

History taking and examination including MSE Annexure 1

Special emphasis

- Medical history

2. a) Investigations required

a. Psychometric investigations: Delinquent Activities Scale (DAS)/ Problem Behaviour Check List (PBCL)
b. Laboratory: (if physical complications are present)

b) Comorbidity: Depression. Attention deficit hyperactivity disorder (ADHD) and Learning Disorder.
c) Differential Diagnosis: Mood disorder, psychotic disorder that precipitate excessive indulgence in negative behaviors and hostility towards other.

3. Treatment

a) Psychopharmacology

- Only if anxiety and depression is comorbid
- Symptomatic and supportive (if necessary)

b) Psychosocial Intervention

a. If patient is in early stage
i) Psychoeducation – Nature of illness, etiology, progression, consequences, prognosis, treatment.

For patient	For caregiver/guardian
At the time of consultation.	At the time of consultation

ii) Family Therapy – *family should always be involved and attempt should be made to help the family to provide consistent upbringing*
iii) Behaviour modification with positive reinforcement
iv) Children may be involved with a group of normal children which may provide them proper role models to emulate

b. If patient is in mid or late stage
i) Cognitive Behaviour Therapy
ii) Aversion Therapy

4. Follow-up

a. Psychiatry OPD/ DMHP

- Initially once in two weeks for 2 months
- Monthly check up for 1 year.

b. Physical check-up at OPD
c. Through telephonic conversation/TeleManas 14416/18008914416

5. Referrals

a. Depending upon the severity and complications of the patient, he/she may need to be referred to Psychiatry department.

b. To TeleManas
c. To the nearest DMHP
d. To psychiatry OPD at Kulikawn Hospital an ZMC
e. To higher centers outside Mizoram through Referral Boar

Annexure - 1

HISTORY TAKING SCHEME IN PSYCHIATRY

1. Name, age, sex, address, marital status, religion, occupation, income.
2. Information obtained from– name, relationship (duration of relationship).
3. History – adequacy, reliability.
4. Chief Complaints mention – Duration of illness
   - i) – mode of onset
   - ii) – episode
   - iii) – precipitating factor (if any)
5. History of present illness

   (include negative history in the later part)
6. History of past illness – psychiatric
   - Medical
7. Family history:

   Parents, sibling, age, marital status,

   relationship. Family income, social

   status.

   Family history of mental illness, substance use disorder.
8. Personal history: Early development (pre-natal)

   Childhood – Physical illness

   Mental retardation Psychiatric disorder Schooling –

   when started & ended

   Performanc
   e, peer
   relation,
   Behavior
   problems

   Marital status Occupation Income

   Substance use disorder

Relationship with spouse,
workmate, others Sexual history,
menstrual history

Number children & related history

9. Premorbid Personality

To assess ask about – relationship, leisure (hobbies)

Predominant character, habits, Attitude & standards

(moral).

A. **MENTAL STATUS EXAMINATION**

i) **Appearance and behaviour** :

General appearance, rapport, eye to eye contact.

Attitude towards interviewer – Co-operativeness, guarding, hostility, suspiciousness.

Facies (non verbal facial
expression) Posture,
movement.

Personal grooming
& hygiene. Motor
activity.

Hallucinatory behaviour.

ii) **speech:** Rate, Volume, tone, spontaneity,

reaction time, quantity

(stammering, stuttering,

dysprosody include here)

iii) **Thought:**

A. Form & Stream.

Flight of ideas, paucity of ideas, over abundance of ideas, spontaneity,
hesitant thinking Irrelevant, loosening of

association, circumstantiality, tangenciality, perseveration, distractibility, blocking, rambling, evative.

Incoherence, clang association, neologism, word salad. (quotation or samples on verbation – to be noted)

B. Content & Posession.

a) Pre – occupations - phobias, obsessions, hypochondriasis, illness, overvalued ideas, antisocial urge.
b) Delusions - persecution, reference, guilt, grandeur, jealousy, sins etc.
c) Ideas - Reference & influence.

iv) **Mood** – Subjective, Objective.
- Consistency, reactivity, appropriateness.

Mood – Prevailing emotional state.

Description – depth, intensity, duration fluctuation

examples of mood – depressed, angry, fearful, anxious, guilt feeling, expansive, euphoric, proud etc.

Affect – expression & expressibility of patient's emotion.

Affect may be described as – normal range, constricted blunt or flat.

v) **Perception**

a) Hallucinations & illusions
b) Depersonalisation & Derealisation
c) Dreams and fantasies

vi) **Cognitive function**

1) Consciousness – Conscious, confused, clouding, delirium, stupor, coma.
2) Orientation – time – day, date, month, week etc.
Place – home, hospital, office, school.
Person – children, spouse, ward staff.
3) Attention – DF/DB (Digit Span test)
Ask 3/5 item to remember & ask again

Write a sentence and ask to do it e.g. pick up the ball pen.

4) Concentration – Serial subtraction 100 - 7
40 - 3

- Ask to say days of week] in reverse order month of year

5) memory – Immediate – DF/DB
ask to memorize

3 different item and ask to repeat of some gap. Spell of a word backwards.

Recent – Breakfast/ lunch/ dinner recent

big news

Remote – date of marriage, birthday of children year of graduation.

6) Abstract thinking – Proverb test 2-3
similarity/Dissimilarity 2-3 Conceptual series completion

vii) **Judgement**

i) Personal Judgement (Aim in life/future plans)
ii) Social Judgement (Relation with others/staff/inmates)
iii) Test Judgement – letter on the road, house on fire, injured child on the road.

viii) **Intelligence** – General information
Calculation Psychometry

ix) **Insight** – Grade.

B. **General Physical Examination**

1. appearance, built, anaemia, jaundice, cyanosis, oedema, lymphadenopathy, deformities, signs of injury, injection marks etc.
2. C.V.S
3. Chest
4. Abdomen
5. Skeletal system
6. CNS

C. **Investigation** – Laboratory

Psychometry

D. **Summary**

E. **Provisional Diagnosis**.

F. **Differential Diagnosis**.

G. **Final Diagnosis**.

H. **Management**.