National Board of Examinations

Considering the variations in the level of standards of post graduate and post doctoral examinations in our country and based on the recommendations of an Expert Group set up for maintaining uniform standards, the Ministry of Health & Family Welfare, Government of India, established the National Board of Examinations (NBE) in 1975, with its headquarters at New Delhi.

Objectives of NBE

Conduct postgraduate examinations in the disciplines of modern medicine at the national level.

Maintain a high standard of examination, so as to ensure that candidates have received adequate training and are competent in every way to practice as specialitists, in their respective fields.

Constitute Speciality Boards in which the examinations are to be conducted.

Formulate basic training requirements for eligibility to appear for the respective examinations.

Prescribe course curricula for postgraduate studies.

Organize postgraduate courses, workshops, seminars, symposia and training programmes of specialised nature.

Institute professorships, other faculty positions, fellowships, research cadre positions and scholarships etc. for realising the objectives of the Board.

Constitute an Accreditation Committee to approve centers for DNB courses.

Co-ordinate with national and international bodies, agencies, universities for the furtherance of the objectives of the Board.

NATIONAL BOARD OF EXAMINATIONS

MODULE FOR CONTINUING MEDICAL EDUCATION FOR DNB CANDIDATES

NATIONAL BOARD OF EXAMINATIONS
(Ministry of Health & Family Welfare, Government of India)
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NATIONAL BOARD OF EXAMINATIONS

Module for

Continuing Medical Education

For DNB candidates

NATIONAL BOARD OF EXAMINATIONS
(Ministry of Health & Family Welfare)
Ansari Nagar, New Delhi-110029

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Introduction

The National Board of Examination was established in 1975 with the primary objective of improving the quality of the Medical Education by elevating the level and establishing standards of post graduate examinations in modern medicine, on an all India basis. There are more than 650 accredited hospitals/ institutions, imparting DNB courses in 58 medical specialties. In order to improve the competencies of DNB candidates in medical research (as envisaged in the requirement of thesis submission by them) it has been suggested to conduct short term workshops on regional basis. The present CME programme module has been designed to prepare the candidates for DNB practical examination.

General Objective

To enhance the basic clinical and communication skills of the candidates so that they are able to make better presentation of long and short cases etc. and are well prepared for their practical examinations

Specific Objectives

The candidates should be able to

- Discuss the relevance of history taking, eliciting clinical signs for the selected short and long cases
- Discuss with the resource persons interpretation of relevant investigations, x-rays, ECG,CT scans, equipments and drugs etc.
- Discuss the important issues related to OSCE in selected specialties

Main Content Areas

History, clinical signs and symptoms, differential diagnosis, investigation and management of common cases; Interpretation of common investigations, spots, specimen, equipments, case scenario in ward setting etc.

Duration

3 days

Methodology

Demonstration and return demonstration of basic clinical and communication skills for presentation of common short and long surgical cases. Discussion on the key issues related to anaesthetic administration and management of common cases.

Category & Number of participants

The DNB candidates who are in final year and are preparing for their examination, those who have cleared their theory examination and are due for their practical examination and the candidates who have failed in their practical examination from NBE accredited hospitals/ institutions.

The number of the participants per workshop will be 35-40.

Evaluation of Workshop

i. Feed back to the participants on their clinical and communication skills by the resource persons during the case presentations.

ii. Feed back from the participants on given format.

Tentative programme and guidelines for organization of CME

Day-I, 8.00 to 9.00 AM -Allocation of long clinical cases to students (Please ensure that each case is allotted to two candidates, one from final year and one from first or second year. Please ask one of them to work out history and the other to do clinical examination). In each batch there would be 25 to 30 candidates thus there would be 12 to 15 groups of two students each for case allocation. The list of important cases is given in the CME module you may shortlist the cases from the list and may also add more in case you desire so.

9.00 to 9.30 AM- Tea/ snacks/ inauguration

*9.30 to 1.30 PM - Long case discussions***-**All the 25-30 students will sit in one room and all the experts would sit as one panel. The students would present cases and the entire faculty/consultants would discuss these highlighting important points in history or clinical signs, investigations, differential diagnosis, management etc. from clinical and examination point of view. The faculty should ensure that it becomes a learning session for all the students some important point be summarized frequently and some important signs be demonstrated to all the students. Nearly 10 minutes should be spent on case presentation and more time should be spent on case discussions. Thus nearly 30-40 minutes would be available for each case.

1.30 to 2.00 PM **-**Lunch

2.00 to 5.00 PM, Long case discussions continue

Day-II, 8.00 to 8.30 AM, Allocation of short clinical cases to students (Please ensure that each case is allotted to two candidates, one from final year and one from first or second year. Please ask one of them to work out history and the other to do clinical examination). In each batch there would be 25 to 30 candidates thus there would be 12 to 15 groups of two students each for case allocation. The list of important cases is given in the CME module you may shortlist the cases from the list and may also add more in case you desire so.

8.30 to 9.00 AM, Tea/ snacks

*9.00 to 1.30 PM, Short case discussions***-**All the 25-30 students will sit in one room and all the experts would sit as one panel. The students would present cases and the entire faculty/consultants would discuss these highlighting important points in history or clinical signs, investigations, differential diagnosis, management etc. from clinical and examination point of view. The faculty should ensure that it becomes a learning session for all the students some important point be *summarized frequently and some important signs be demonstrated to all the students. Nearly 5 minutes should be spent* on case presentation and more time should be spent on case discussions. Thus nearly 15 to 20 minutes would be available for each case.

1.30 PM to 2.00 PM, Lunch

2.00 to 5.00 PM, Ward Rounds-The candidates in smaller groups of 6-8 will be taken for the ward rounds in the specialty where they would be shown cases and discussions would be done about prognosis, treatment modalities, dietary, electrolyte and fluid requirements, clinical and diagnostic procedures etc.

Day-III, 8.00 to 11.30 AM, Instruments for clinical/surgical procedures-The students would be seated in one room and the facilitators would sit in a panel and carry out discussions on common instruments for various clinical and surgical procedures, about their use, precautions, complications of procedures

11.30 to 1.30 PM, Investigation modalities-All the 25-30 students will sit in one room and all the experts would sit as one panel. And carry out discussions on common investigation modalities as X-rays, CT scans, MRI, Ultra sounds etc. ECG, EEG, biochemical and pathological reports, slides, specimens etc. with focus on their interpretation and drawing meaningful clinical conclusions for treatment and follow up.

1.30 PM to 2.00 PM, Lunch

2.00 to 5.00 PM, OSCE/ Preparing for theory examination-In the specialties of ENT, Ophthalmology and Pediatrics the students would also be given exposure to OSCE stations and mock OSCE with 10-15 stations be planned and later the response of the students be discussed with them. In other specialties where there is no OSCE the students be given instructions how to plan a theory question in the final examination and how to score more by properly attempting the question in the allocated time.

Tentative programme and guidelines for organization of for CME in Family Medicine

Day-I, Placement in Medicine Department

8.00 to 9.00 AM, Allocation of clinical cases in Medicine to students (Please ensure that each case is allotted to three candidates, one from final year and one from first and one second year. Please ask one of them to work out history and the others to do clinical examination). In each batch there would be 25 to 30 candidates thus there would be 10 groups of three students each for case allocation. The list of important cases is given in the CME module you may shortlist the cases from the list and may also add more in case you desire so.

*9.00 to 9.30 AM,*Tea/ snacks/ inauguration

*9.30 to 1.30 PM, Case discussions***-**All the 25-30 students will sit in one room and all the experts would sit as one panel. The students would present cases and the entire faculty/consultants would discuss these highlighting important points in history or clinical signs, investigations, differential diagnosis, management etc. from clinical and examination point of view. The faculty should ensure that it becomes a learning session for all the students some important point be summarized frequently and some important signs be demonstrated to all the students. Nearly 10 minutes should be spent on case presentation and more time should be spent on case discussions. Thus nearly 30 minutes would be available for each case.

1.30 to 2.00 PM, Lunch

2.00 to 4.00 PM, Case discussions continue

4.00 to 6.00 PM, Ward Rounds-The candidates in smaller groups of 6-8 will be taken for the ward rounds in the specialty of Medicine where they would be shown cases and discussions would be done about prognosis, treatment modalities, dietary, electrolyte and fluid requirements, clinical and diagnostic procedures etc.

Day-II- Placment in OBG Department

8.00 to 9.00 AM, Allocation of clinical cases to students (Please ensure that each case is allotted to three candidates, one from final year and one from first and one second year. Please ask one of them to work out history and the others to do clinical examination). In each batch there would be 25 to 30 candidates thus there would be 10 groups of three students each for case allocation. The list of important cases is given in the CME module you may shortlist the cases from the list and may also add more in case you desire so.

8.30 to 9.00 AM, Tea/ snacks

*9.00 to 1.30 PM, Case discussions***-**All the 25-30 students will sit in one room and all the experts would sit as one panel. The students would present cases and the entire faculty/consultants would discuss these highlighting important points in history or clinical signs, investigations, differential diagnosis, management etc. from clinical and examination point of view. The faculty should ensure that it becomes a learning session for all the students some important point be summarized frequently and some important signs be demonstrated to all the students. Nearly 10 minutes should be spent on case presentation and more time should be spent on case discussions. Thus nearly 30 minutes would be available for each case.

1.30 PM to 2.00 PM, Lunch

2.00 to 4.00 PM, Case discussions continue

4.00 to 6.00 PM, Ward Rounds-The candidates in smaller groups of 6-8 will be taken for the ward rounds in the specialty of OBG where they would be shown cases and discussions would be done about prognosis, treatment modalities, dietary, electrolyte and fluid requirements, clinical and diagnostic procedures etc.

Day-III- Placement in Pediatrics

8.00 to 9.00 AM, Allocation of clinical cases in Pediatrics to students (Please ensure that each case is allotted to three candidates, one from final year and one from first and one second year. Please ask one of them to work out history and the others to do clinical examination). In each batch there would be 25 to 30 candidates thus there would be 10 groups of three students each for case allocation. The list of important cases is given in the CME module you may shortlist the cases from the list and may also add more in case you desire so.

9.00 to 9.30 AM, Tea/ snacks

*9.30 to 1.30 PM, Case discussions***-**All the 25-30 students will sit in one room and all the experts would sit as one panel. The students would present cases and the entire faculty/consultants would discuss these highlighting important points in history or clinical signs, investigations, differential diagnosis, management etc. from clinical and examination point of view. The faculty should ensure that it becomes a learning session for all the students some important point be summarized frequently and some important signs be demonstrated to all the students. Nearly 10 minutes should be spent on case presentation and more time should be spent on case discussions. Thus nearly 30 minutes would be available for each case.

1.30 to 2.00 PM, Lunch

2.00 to 4.00 PM, Case discussions continue

*4.00 to 6.00 PM, Ward Rounds***-**The candidates in smaller groups of 6-8 will be taken for the ward rounds in the specialty of Pediatrics where they would be shown cases and discussions would be done about prognosis, treatment modalities, dietary, electrolyte and fluid requirements, clinical and diagnostic procedures etc

SAMPLE CASES FOR PRESENTATION AND DISCUSSION
ANAESTEHSIA

Long cases

- Cyanotic Heart Disease (TOF, ASD, VSD, PDA)
- Patient on pacemaker
- Valvular Heart Disease (RHD with Mitral stenosis, Mitral regurgitation, Aortic stenosis, Tricuspid regurgitation)
- Patient of Scoliosis and kyphoscoliosis for anesthesia
- Difficult airway (TM Joint ankylosis)
- Hypertension
- Coronary Artery Diseases
- Bronchial Asthma
- Chronic Obstructive Pulmonary Disease (COPD)
- Diabetes Mellitus
- Thyroid Swelling with hyper & hypo function
- Obstructive Jaundice
- Portal Hypertension
- Chronic Renal Failure
- Severe Anaemia
- Benign Hypoertrophy of Prostate for TURP
- Paraplegia/GBS/Motor Neuron Disease
- Pregnancy related disease- Normal & related disease

Short cases

- Cyanotic Heart Disease (TOF, ASD, VSD, PDA)
- Buerger's disease
- Cleft lip and cleft palate
- Meningocele or Meningo-myelocele-
- Hydrocephalus
- Tracheostomy
- Burn Contracture
- Congenital Telipes Equnio Varus(CTEV)
- Diabetic Foot Ulcer
- Squint

Spots

Electrocardiogram (ECG)

- Arrhythmia's (Atrial Fibrillation,Ventricular Fibrillation,,Atrial flutter, Ventricular Tachycardia, Supraventricular tachycardia, Junction rhythm)
- Pacemaker
- Coronary Artery Disease with Myocardial Ischaemia and infarction
- Left Ventricular Hypertrophy and Right Ventricular Hypertrophy
- Right Bundle Branch Block
- Left Bundle Branch Block

X-ray Chest

- Pneumothorax,
- Malignancy,
- Pleural Effusion,
- Bronchiectasis,
- Emphysema,
- Hydropneumothorax,
- Consolidation,
- Collapse of lung,
- Pericardial effusion,
- Cardiomegaly,
- Mitral Stenosis,
- Plethoric lung field (PDA),
- Pulmonary Edema,
- Adult Respiratory Distress Syndrome
- Oligaemic lung field (TOF)
- Fractured ribs
- Foreign body Bronchus
- Foreign body Oesophagus
- Pulmonary Koch's Miliary TB
- Inter costal drain, Pacemaker
- Diaphragmatic hernia
- Eventration of diaphragm
- Lung Cyst-Congenital & adult,
- Hydatid cyst

Equipments

- Anesthesia Machine
- Anesthesia Circuits
- Anesthesia vaporizers-all old and new-
- Oxygen analyzer
- Airways-Oropharyngeal and ,Nasopharyngeal
- Identification , Indications, and Method of use of-

Stylet,Magill Forceps,Gum elastic bougie,LMA-classic, flexible, ILMA, proseal LMA, Endotracheal tubes-PVC, PVC reinforced, Red rubber, RAE-northand south pole, Cole tube,Light wand, Track light, Laryngoscopes-Macintosh, Miller Mccoy, Face masks-anatomical, AMBU, Rendall - Baker Souzeck, Ventimask, Polymask, T-

piece, Nasal Prongs, Nasal Catheter, NIV-BiPAP, Ventilator-ICU, Anesthesia, Needle-spinal, epidural/CSE sets, Bronchial , Blocker, Central Venous catherters-single lumen (70cm), triple lumen,Neuromuscular junction Monitor, EKG monitor, EtCO2 monitor and tracing, Arterial blood gas cards- Hypoxia, hypercarbia, acidosis-Metabolic and respiratory, alkalosis-respiratory and metabolic, Flow Volume Loops

Resuscitation- CPR dummy
Skeleton for blocks

ENT
Sample of long Cases

- Safe CSOM
- Chronic SOM (unsafe type)
- Serous otitis media
- Adhesive otitis media
- Carcinoma larynx & laryngopharnx
- Facial palsy
- Growth maxilla
- Complications of chronic sinusitis
- Cleft palate
- Nasopharyngeal angio fibroma
- Carcinoma larynx
- Secondaries neck with unknown primary
- Carcinoma nasopharynx
- Tumours of Parotid/submandibular salivary gland
- Laryngeal web
- Unsafe CSOM
- Complicated CSOM
- Nasal Polyps
- Angiofibroma
- DNS
- Nasal Tumors
- Maxillary Tumors
- Vocal Cord Paralysis
- Vocal Nodules
- Vocal Polyps
- Laryngeal Popilloma
- Malignancies of Larynx, Hypopharynx, Nasopharyns, Oropharynx
- Swellings of the neck
- Thyroid

Sample of Short Cases

- Acute suppurative otitis media
- Thyroglossal cyst
- Thyroglossal fistula
- Bronchial sinus
- Facial Palsy
- Osteoma of PNS
- # Nasal bones
- Blow out fracture of orbit
- Cleft lip
- Cervical lymphadenitis
- Dysphagia
- Haemangioma nasal cavity
- Otosclerosis
- Meniere 's disease
- CSF Rhinorrhoea
- Ramsay Hunt syndrome
- Retropharyngeal abscess
- Solitary nodule Thyroid
- Goitre
- Submandibular Rialadenitis
- Stone in Rubmandibular gland duct
- Laryngococle

OBG
Long Cases
Obstetrics

- Normal Term Pregnancy
- Pregnancy Induced Hypertension / Preeclampsia
- Diabetes
- Anaemia
- Heart Disease
- Antepartum Hemorrhage
- Jaundice
- Malpresentations
- Previous Caesarean section
- Bad Obstetrical History (Recurrent Preterm)**/** (Recurrent abortions**)**
- Intrauterine growth restriction
- Pregnancy with Hypothyroidism
- Post dated pregnancy
- Rh negative pregnancy
- Multiple pregnancy
- Breech
- Preterm Labor
- Hydramnios
- Intra Uterine Fetal Death(IUFD)
- Malformed / Contracted Pelvis
- Pregnancy with Asthma

- Pregnancy with UTI
- Pregnancy with TB

Gynaecology

- Fibroid
- Abnormal Uterine Bleeding
- Malignancies
- Gestational trophoblastic Disease
- Infertility, PCOD
- Prolapse
- Post Menopausal Bleeding(PMB)
- Ovarian tumour
- Fistulae
- Adnexal masses
- Amenorrhoea – primary/secondary
- Stress Urinary Incontinence

Short Cases

Obstetrics

- Abortions
- Ectopic pregnancy (post operative)
- Cervical incompetence
- Puerperal Cases
- Post LSCS (Caesarean Section)
- Early pregnancy
- Molar Pregnancy
- Adolescent Problem
- Preterm labor
- Pregnancy with UTI

Gynaecology

- Vaginal discharge
- Pruritis vulvae
- Cervical erosion
- 3^0 Perineal tear
- Vulvar ulcer
- White lesions of vulva
- Obesity / Hirsutism
- Abnormal Utrine Bleeding
- Fibroid Uterus
- Ovarian Cyst / Adnexal Mass

ORTHOPEDICS
Long cases

- Non union intra capsular fracture neck femur
- Malunited Trochantric fracture
- Un reduced neglected dislocation of hip
- Perthe's disease
- Slipped capital femoral epiphysis
- TB hip with deformities
- Osteonecrosis of hip with secondary OA
- Pott's spine with paraplegia
- Prolapsed intervetebral disc
- Lumbar canal stenosis
- Ankylosing Spondylitis with hip involvement
- Ankylosed hip
- Traumatic paraplegia
- Cervical spine tuberculosis with quadriplegia

Short cases

- Cubitis Varus
- Cubitis Valgus
- Un reduced dislocation of elbow
- Recurrent dislocation of shoulder
- Post polio residual paralysis with Equinovarus foot
- Equinovalgus foot
- Calcaneovalgus foot
- Cerebral palsy with spastic equines
- Knee Instabilities
- Osteosarcoma of a long bone
- Ewings sarcoma of a long bone
- Diaphyseal aclasis
- Tuberculosis of elbow
- Tuberculosis of knee with triple deformity
- TB Ankle
- TB wrist
- Median nerve injury at wrist and above elbow
- High and low radial nerve palsy
- High and low ulnar nerve palsy
- Brachial plexus injuries _ Erb's paralysis
- stiff elbow
- Neglected Club foot
- Club hand
- Maidling deformity
- Non Union and delayed union of long bones
- Rickets and angular deformities like genu valgum and varum
- Chronic Synovitis of knee
- Flail shoulder
- Upper limb poliomyelitis
- GCT lower end radius or upper end of tibia

Spot slides

- ❖ Giant Cells
- ❖ Malignant Bone Tumors like Osteo sarcoma, GCT, Ewings, Chondrosarcoma

MEDICINE

- ➢ Cerebro-vascular disease
- ➢ Multi-valvular heart disease
- ➢ Cerebellar disease
- ➢ Congenital Heart Disease
- ➢ Myopathy
- ➢ Extrapyramidal disease/ Movement disorders
- ➢ Fibro-cavitory lung disease
- ➢ Pleural disease
- ➢ Pulmonary mass / consolidation
- ➢ Cranial Nerve Palsies
- ➢ Ascites
- ➢ Diabetes Mellitus with complications
- ➢ Spinal Mellitus with complications
- ➢ Spinal cord disease
- ➢ Chronic obstructive airway disease
- ➢ Hepatosplenomegaly
- ➢ Rheumatological Diseases
- ➢ Thyrotoxicosis
- ➢ Hypothyroidism
- ➢ Nephrotic Syndrome
- ➢ Jaundice

PEDIATRICS

CNS

- ➢ Floppy infant
- ➢ Hemiplegia / Quadriplegia / Paraplegia
- ➢ Developmental Delay
- ➢ Mental Retardation with Cerebral Palsy
- ➢ Neurodegenerative Disorders
- ➢ TBM
- ➢ Muscle Dystrophy
- ➢ ICSOL
- ➢ Potts Spine
- ➢ Large Head
- ➢ Microcephaly
- ➢ Abdomen—
- ➢ Hepatosplenomegaly
- ➢ Chronic Diarrhea & Malabsorption

CVS

- ➢ RHD with Multi Valvular Involvement Congenital Heart Disease
- ➢ Aortic Arteritis
- ➢ Dilated Cardio Myopathy

Respiratory

- ➢ Pleural Effusion Consolidation / Collapse Chronic Lung Disease Lung Abscess

Others

- ➢ Anemia
- ➢ Lymphadenopathy with Anemia Short Stature
- ➢ Petichiae / Purpura
- ➢ Rickets / Skeletal Dysplasia Arthritis
- ➢ PEM

Neonatal Cases

- ➢ Low Birth Weight
- ➢ Preterm
- ➢ A child with Jaundice
- ➢ Normal Neonate
- ➢ A baby with Tachypnea
- ➢ Neonate with hepatospleenomegly
- ➢ A baby with dysmorphic features (common ones)
- ➢ Common Neonatal problems (Cephlohemotoma, Skin problems)

SURGERY

- ➢ Solitary Thyroid Nodule/Thyroid Carcinoma MNG
- ➢ Cervical Lymphadenopathy
- ➢ Salivary Gland tumour
- ➢ Hepato splenomegaly
- ➢ retroperitoneal tumours
- ➢ Obstructive Jaundice
- ➢ Pancreatic mass
- ➢ Peripheral Vascular Disease/ Varicose Veins
- ➢ Breast nodule
- ➢ Inguino-scrotal Swellings
- ➢ External Genitalia (Ulcer/nodule)
- ➢ Soft tissue tumours limb
- ➢ Soft tissue tumours trunk
- ➢ Oral ulcer/growth

OPTHALMOLOGY

Short Cases

Anterior Segment

- ➢ Ptosis

- Proptosis
- Squint
- Complicated Cataract
- Ocular surface disorders including cornea / PK
- Poly Ocular Trauma
- Endophthalmitis
- Secondary Glaucoma
- Facial Palsy
- Herpes Zoster Ophthalmicus
- Ocular Manifestations of Leprosy
- Congenital Anomalies
- Concurrent Surgical Procedures
- Bvphthalmos
- Trachoma
- Congenital Anomalies of anterior segment
- Traumatic Cataract
- Duanes Retraction Syndrome
- Dermoid
- Lid Tumors / Chalazion
- Uveitis
- Corneal Opacities / Scars
- OSD, Symblepharon
- Entropion / Ectropion
- Pterygium
- Psuedophakia, Multifocal / Scleral Fixated / Accommodative IOL
- Cranio Facial Disorders
- Angle Closure Glaucoma
- Staphyloma, Atrophic, Phthisis Bulbi

Posterior Segment

- Myopia
- Retinal detachment
- Eale's Disease
- Maculopathies
- ARMD
- CRVO, BRVO
- Hypertensive / Diabetic Retinopathy
- Posterior Segment Colobomas
- Optic Atrophy
- Glaucoma
- Papilloedema / Papillitis

OSCE

- ERG / VEP, OCT, HRT, FFA, Visual Fields, Specular Microscopy, Topography, CT, MRI, USG (a-b scan)
- Drugs
- Clinical Photographs (Anterior segment / Adnexa / Injuries)
- Surgical Videos
- Microscopic / Gross Specimens
- Histology Microphotographs - Retino blastima, Malignant Malanoma, Lid Carcinoma, Lacrimal Sac, Cornea, Lens, Ciliary body, Trabecular meshwork, extra ocular muscles
- Embryology models
- Diagnostic appliances / Dark Room procedures
- Surgical Instruments
- Clinical Situations
- Lab procedures, staining, culture medial
- IOLs, drainage devices, and other artificial appliances
- Ophthalmic History / Medical Ethics / Communication skill with patients

GENERAL GUIDELINES FOR CASE PRESENTATION AND DISCUSSION

History-Correct sequence of chief complaints, present history, past history, family history, birth history, development history, socioeconomic history. All headings to be covered even if they are normal. However relative emphasis may be on relevant history e.g. dietary history in detail is important in malnutrition and development history in cerebral palsy, family history in genetic disorders and socioeconomic history in rheumatic fever etc. Emphasize on clarity of presentation and avoid unnecessary repetition.

History of Present illness-Cover points in symptoms to find etiology of disease e.g. in failure to thrive, whether it is a chronic illness, malabsorption, nutritional deficiency etc. Progress of the disease eg static, improving or worsening. Secondary effects of the disease e.g. vit deficiencies in chronic liver disease.Treatment history should be covered in detail regarding nature of medicines e.g. tablets, injections, syrups etc and their effect on the illness. Patient may be able to tell actual name of medicine and it should not be disregarded. Just saying that patient has taken treatment from outside is not enough and analysis of treatment should be done. Course of the patient during hospital stay should be also asked.

Past history -Relevant past history e.g. sore throat in Rheumatic fever etc and also generally of common chronic diseases egg TB etc should be covered .Any prolonged illness and hospitalization should be recorded.

SE history-Per capita income. Education of parents, type of house and other relevant details .

Dietary history-Must tell actual caloric intake .Calories of foods eaten once in 2-3 days may be equally divided over the week egg if a banana is eaten twice a week then calories may be of 2/7 banana a day and foods eaten very occasionally may be ignored from calculation of dietary intake. Premorbid as well as morbid caloric intake may be asked.Try to check if anthropometry corresponds to caloric intake as if caloric intake calculated is half of required but wt and ht are normal ,then recheck the calculation of caloric intake and try to explain the reason.

Development history- Details of development should be asked as relevant to the case e.g. a case of Kalazaar in 12 yr old one may ask gross mile stones only but in dev delay in 1 yr old all mile stones in minute details may be asked.

Examination-Detailed examination of vitals, anthropometry, general physical and systemic examination should be done. Canditate should present the involved system first. Various techniques of examination should be checked and demonstrated during the CME.

Diagnosis and differential diagnosis-First most likely diagnosis should be told.Then diagnosis which can not be ruled out by clinical examination but require investigations for the same should be given.Then the similar conditions which have been clinically ruled out.

Investigations-Should be relevant to the case. Ask interpretation of investigations. Discuss x ray, ECG , CT, ECHO findings etc.

Treatment-Discuss specific treatment. Supportive treatment. Problems in treatment regarding side effects etc. Cost of treatment.

Prognosis-Prognosis should be discussed**.**

Counseling-Counseling in each case should be discussed.

Routine care –Immunization; Family welfare; Psychological aspects.

SPECIFIC GUIDELINES FOR CASE PRESENTATIONS IN ANAESTHESIA

Long cases

Cyanotic Heart Disease (TOF, ASD, VSD, PDA)

History-To bring out the onset of cyanosis (congenital cyanotic vs Eisenmenger syndrome); To elicit history of cyanotic spells; To elicit history of chest infections/failure to thrive/cardiac failure/any interventions in the past.
Clinical examination-To bring out the physical/developmental milestones in case of pediatric age group;To recognize cyanosis and other signs of chronic hypoxemia; To identify features of associated congenital anomalies.
Investigations-Typical ECG, X-ray, echocardiography, cardiac catheterization, blood gas findings.
Differential Diagnosis-To be able to enumerate other congenital cyanotic heart diseases/ to discuss Eisenmenger syndrome.
Management-To be able to discuss the rationale of anesthetic management based on the patho-physiology (To discuss various induction/volatile agents/opioids and their effects on PVR, SVR and heart rate) of basic cyanotic heart disease; Discussion of anesthetic management of incidental surgery for routine and emergency

Patient on pacemaker
History-Reason or indication for pacemaker insertion; Type of pacemaker; History pertaining to pacemaker malfunction; Last time pacemaker function was evaluated.
Clinical examination-Routine CVS examination; Examination pertaining to co-morbidities; Tests for demand pacemaker; Quality, rate, rhythm of pulse
Investigations-ECG, X-ray chest; Pertaining to specific co-morbidities; Pertaining to pacemaker function ECG with/without magnet.
Management-How would the presence of single or dual chamber pacemaker alter your anaesthesia planning ?;What is the role of the magnet intraoperatively; Discuss electromagnetic interference with pacemaker function; How do you minimize electromagnetic interference?; Types of pacemaker and their terminology; VVI pacemaker and implications of its use in a patient with complete heart block; Electromagnetic interference with pacemaker function in the Operation theatre setting; Modalities to minimize preoperative pacemaker dysfunction; Acceptable values of pacing threshold sensing R wave and resistance; Temporary pacemakers/AICD and their utility; Patient with pacemaker for MRI, ESWL and electroconvulsive therapy-implications

Valvular Heart Disease (RHD with Mitral stenosis, Mitral regurgitation, Aortic stenosis, Tricuspid regurgitation)
History-To differentiate between various valvular lesion based on history; Aortic stenosis – typical type of angina, syncope and CHF; To elicit history related to etiology – rheumatic fever.
Clinical examination-To identify typical murmur pertaining to various lesion; To elicit arrhythmias, narrations of pulse volume, signs of failure.
Investigations-To be able to identify typical ECG, x-ray, echocardiography, cardiac cath findings etc.
Differential Diagnosis- To arrive at a diagnosis based on history & clinical findings; Students should be able to present points in favour and against each probable diagnosis.
Management-To be discussed if the patient were presenting for cardiac/non-cardiac surgery; Emphasis on hemodynamic goals for each individual lesion; Commonly encountered perioperative events associated with particular lesions and their management; To be able to discuss pharmacology and indications of various inotropes/vasoactive agents/ antiarrhythmic drugs.

Patient of Scoliosis and kyphoscoliosis for anesthesia

History-Onset and progress of disease; Associated congenital anomalies; Functional status and neurologic deficits; Previous surgery for scoliosis of any; H/O – T.B., Poliomyelitis, Neurofibromatosis, Dysautonomia, Duchenne Muscular Dystrophy ; FAMILY H/O – Malignant Hyperpyrexia as it is more common in patients with KYPHOSEOLIOIS.

Clinical examination-General physical examination including room air saturation/bedside pulmonary functiontests; Relevant examination for associated syndromes and cardiovascular effects; Implications of Halo traction; LOOK FOR SIGNS & SYMPTOMS OF- Restrictive Lung Disease, Pulmonary Hypertension, Congenital Heart Disease, Airway Abnormalities, Preexisting Neurological Deficit

Investigations-X-ray spine for Cobb's angle and implications; Chest x-ray; Pulmonary function tests; Echocardiography

*Management***-**Treatment of underlying infection; Discuss how different surgical approaches –anterior, posterior, transthoracic (open/endoscopic) would alter anaesthetic managements; Chest physiotherapy and breathing exercises; Implications and techniques of intraoperative neurological monitoring; Intraoperative and postoperative pain management strategies; Minimize blood loss, intraoperative positioning, spinal cord monitoring, Wake up test.

Difficult airway (TM Joint ankylosis)

*History***-**Course of the restriction and any previous surgery; History of truma

Clinical examinations-To be able to perform a detailed airway examination –Mouth opening, neck movements, patency of nostrils; To elicit associated co-morbidities

Investigations-Relevant radiological examination; Investigations pertaining to co-morbidities

Management-Special emphasis on consent taking for awake intubation/tracheostomy; Modalities of securing airway blind nasal, fiberoptic guided etc.; To discuss awake versu asleep modalities of securing the airway; Techniques of airway topicalization; Extubation strategy; Modalities for post-operative pain management

Hypertension

History-History of rise in blood pressure, headache, visual disturbances, nose-bleeding, fatigue, nausea, anxiety, nervousness; History to evaluate end organ damage- CVS, CNS, eyes and renal; History of diabetes, obesity, family history of hypertension,history of exclusion of secondary causes of hypertension- Coarctation of the aorta, Chronic renal disease, Renovascular disease, Glomerulonephritis, Pheochromocytoma, Primary adosteronism, Cushing's syndrome, Intracranial lesions, Estrogens/oral contraceptives, steroidal & NSAIDS, nasal decongestants, appetite suppressants, and the tricyclic antidepressants, Excessive alcohol intake; Treatment history.

Clinical signs- Accurate measurement of blood pressure with proper size cuff over a week; Palpation of all peripheral pulses should be performed; Look for renal artery bruit over the upper abdomen; the presence of a unilateral bruit with both a systolic and diastolic component suggests renal artery Stenosis; Examination of neck for thyroid enlargement, bruits, distended veins; Examination of the lungs; Examination of the heart for increased rate, size, precordial heaves, rhythm, gallops, murmurs; Examination of abdomen for enlarged kidneys, masses, aortic dilatation, bruits; Examination of extremities for edema; Neurological assessment; Careful cardiac examination to evaluate signs of LVH; Displacement of apex; A sustained and enlarged apical impulse presence of an S_4, Tambour S_2 occasionally (with aortic root dilatation)

Differential diagnosis-Adrenal Adenoma, Aortic Coarctation, Aortic Dissection, Apnea, Sleep, Atherosclerosis, Atherosclerotic Disease of the Carotid Artery, Cardiomyopathy, Cocaine, Cardiomyopathy, Hypertrophic Hyperaldosteronism, PrimaryHypertension, Hypertension and Pregnancy, Hypertension, Malignant Hypertensive Heart Disease, Hyperthyroidism, Obstructive Sleep Apnea – Hypopnea Syndrome, Pheochromocytoma, Renal Artery Stenosis.

*Investigations***-**ECG, X-ray chest, ECHO; Renal function tests; Blood glucose, lipid profile; Investigations for secondary hypertension depending upon history and clinical examination.

Management-To bring the patient to normotensive level, antihypertensive drugs; Anaesthetic Management - Techniques of choice, Deciding factors; Regional – spinal /epidural, Local, General Anaesthesia, TIVA
Discussion of case **-**Classification of Hypertension; Classification of antihypertensive drugs, dosages indications / contraindications; Pathophysiology of Hypertension; Pathogenesis and regulation of blood pressure; Pathogenesis of target organ damage.

Coronary Artery Diseases
*History***-**Pertaining to angina, myocardial infarction, current activity level (Functional capacity in terms of METS); Any intervention/drug therapy pertaining to IHD, hypertension, diabetes, renal diseases etc.; History of congenital hyperplipidemic states.
*Clinical signs***-**Cardiovascular system in details as describes in Hutchison's methods
Investigations-Haematological, renal function, blood sugar and their implications; ECG- to be able to discuss features of related to ischaemia and relationship between various lead and myocardial locations; Invasive/non-invasive stress tests and their implications.
Management-ACC/AHA guidelines for risk stritifiaction; Preoperative optimization – Goals and interventions (pharmacological/non-pharmacological); Anticoagulants/ statins (pharmacology/ implications on anaesthetic techniques); Perioperative haemodynamic goals and how to achieve them; Perioperative monitoring – ECG, CVP, invasive blood pressure, TEE; Post-operative pain management.

Bronchial Asthma
History-History of breathlessness, duration and frequency, precipitating factors; History of hospital admission, ICU admission, ventilator management; History of medications, steroids.
*Clinical signs***-**Wheezing, respiratory system examination; Differential diagnosis-Aspirated foreign bodies, Viral tracheobronchitis, Restrictive pulmonary diseases (Sarcoidosis), Rheumatoid arthritis and associated bronchiolitis, Extrinsic compression like mediastinal neoplasm, thoracic aneurysm, Intrinsic compression like croup, epiglottitis, Recent history of trauma, surgery or tracheal intubation, Congestive heart failure, Pulmonary embolism, Gastroesophageal reflux and aspiration.
*Investigations***-**Breath holding and pulmonary function tests; X-ray chest, blood count, ABG
*Management***-**Bronchodiltors; Corticosteroids; Cromolyns; Anticholinergic; Chest physiotherapy, systemic hydration, appropriate antibiotics and bronchodilator therapy during the preoperative period improves reversible components of asthma; Technique of anaesthesia- Regional anaesthesia by avoiding upper airway instrumentation is an attractive proposition. However, accidental high levels of sensory block, spontaneous and uncontrollable coughing, inability to lie flat for a long time can all prove problematic. *General anaesthesia*
Any other-Intraoperative bronchospasm- Diagnosis and treatment; Pregnancy and asthma

Chronic Obstructive Pulmonary Disease (COPD)
History-Candidate should be able to bring out typical history of chronic bronchitis or emphysema; History of recent exacerbations, hospitalization, therapy, activity level; History of any domiciliary oxygen therapy should be brought out.
*Clinical signs***-**Examination of respiratory system as described in Hutichison's Hunter; Simple bedside pulmonary function tests; Signs of respiratory failure
Differential diagnosis-To be able to discuss the differences between reactive airway disease and COPD
Investigations-Should be able to discuss, Chest x-ray, Pulmonary function tests, flow volume loops, arterial blood gases, and their implications
Management-Preoperative risk stratification and optimization of the patient for lung surgery and other non-thoracic surgery; To be able to discuss lung isolation and implications of one lung anaesthesia; Modalities of post-operative pain management available and their implications

Diabetes Mellitus

History-Type, duration of DM, involvement of other organs, treatment history; Ischaemic heart disease, work up, latest recommendation of drugs; History to evaluate end organ damage

Clinical signs- Evaluate vitals and signs of organ involvement; Autonomic, peripheral neuropathy

Investigations-Haemoglobin and relevance; Glycosylated haemoglobin and its relevance; ECG, X-ray chest; Blood chemistry

Management-Drugs used for glycemic control; Drugs used for other co-morbid situations; Anaesthetic implications including perioperative control of blood sugar; Diabetes coming for emergency surgery; Management of diabetic coma; Glycemic control in critically ill patients

Thyroid Swelling with hyper & hypo function

History-Residence— endemic areas; Swelling—onset, duration, whether associated with pain, any sudden increase in size with pain; Sleep disturbance-like complaints of sleeplessness as in primary thyrotoxicosis; Pain, Pressure Effects- dyspnoea, dysphagia, hoarseness of voice, stridor; Symptoms of Primary Thyrotoxicosis-loss of weight, preference for cold and intolerance to heat and excessive sweating; CNS involvement - nervous excitability, irritability, insomnia, tremor of hands and weakness of muscles; Cardiovascular Symptoms-palpitation, tachycardia.and dyspnoea on exertion; Exophthalmos-staring or protruding eyes, double vision or diplopia, any edema or swelling of the conjunctiva (chemosis); Menstrual disturbances-usually amenorrhoea; Symptoms of Secondary Thyrotoxicosis— more of cardiovascular system; involvement-palpitations, irregular heart beats (ectopics), dyspnoea on exertion, chest pain, signs of CCF like ankle swelling, oliguria; Symptoms of myxoedema (Hypothyroidism) -increase of weight in spite of poor appetite, intolerance of cold weather, dryness of skin, facial puffiness, dull expression, loss of hair, muscle fatigue and lethargy, failing memory and hoarseness of voice; Constipation; Oligomenorrhoea; Past history— Course of treatment and its effect on the swelling; H/O ingestion of goitrogenic drug eg.. PAS or sulphonylureas or antithyroid drugs, which are goitrogenic ; Personal History - Dietary habits ,Goitrogenic Vegetables—; Family History - H/O similar occurrences in family, endemic goiter.

Clinical signs and symptoms-Build and state of Nutrition; Facies, Mental state and Intelligence; Pulse Rate- rate, rhythm, regularity, Sleeping Pulse Rate ; Skin — hot and moist or dry and inelastic; Tremor; Local Examination- Inspection, palpation, percussion, auscultation; General Examination; Primary toxic manifestations in case of goiters affecting the young ; Secondary toxic manifestations in nodular goiter; Metastasis in case of malignant thyroid disease; Search for Metastasis- - Cervical nodes; Distant metastasis-bones such as skull, spine, end of long bones etc.; Lung metastasis

Differential diagnosis-Non-toxic goiter-Diffuse parenchymatous, Colloid, Multinodular, Solitary nodular; Toxic goiter-Diffuse (Grave's Disease), Multinodular, Toxic nodule (solitary nodular) ; Neoplastic —Benign or Malignant; Thyroiditis -Acute bacterial, Granulomatous, Autoimmune, Riedel,s, ; Chronic bacterial; Retrostemal goiter -knowledge of the diagnostic features.

Investigations and Management- Thyroid Function Tests; Serum Protein Bound Iodine; Serum Thyroxine (T4) ; Total SerumTri-iodothyronine (T3) ; T3 Resin Uptake Test; Serum Thyroid Stimulating Honnone (TSH); T3 Suppression Test; Thyroid Scan; Miscellaneous Test-BMR, Serum Creatinine, Serum cholesterol, Measurement of tendon reflexes, ECG; Radiography-to diagnose position of trachea; Both AP and Lateral view of neck; Bone Scan; FNAC; **U**ltrasound

Management-Medical Management; Surgical Management- Anaesthetic management including perioperative complication

Hypothyroidism

Increased sensitivity to depressant drugs; Hypodynamic cardiovascular system; Decreased cardiac output; Slowed drug metabolism; Unresponsive baroreceptor reflexes; Impaired ventilatory responses to arterial hypoxemia or hypercarbia ; Hypovolemia; Delayed gastric emptying time; Hyponatremia, anaemia, hypoglycemia; Adrenal insufficiency; Anaesthetic management including pre operative medication, choice of drugs for induction and maintenance of anaesthesia, monitoring with emphasis on temperature monitoring, concerns about recovery from anaesthesia; Role of regional anaesthesia.

Obstructive Jaundice

*History***-**Onset, duration, progress of jaundice; Relevant history to elicit features of hepatic failure
*Clinical signs***-**General physical examination and detailed examination of GIT to elicit signs of hepatocellular failure; To demonstrate sites of eliciting jaundice; Features of portal hypertension
Differential diagnosis-To be able to differentiate on the basis of history/examination/available investigations the etiologies of obstructive jaundice examples Malignancy, CBD stone, Congenital
Investigations-Relevance of each component of liver function tests; Risk stratification scores
Management-To be able to discuss special consideration for Whipple's procedure, hepatic resection-Major/minor, CBD exploration; To prevent hepatorenal syndrome; Pain management strategies

Portal Hypertension

History-Presenting complaint with duration (Haematmesis), malena, ascitis; Weight loss, Nausea, vomiting, anorexia, malaise, abdominal discomfort; Arthralgia ,myalgia- acute viral hepatitis; H/o drug exposure-CCl_4,vinyl chloride, acetaminophen ingestion, INH, methyldopa; H/o contact with jaundiced patient, blood transfusion, injections; H/o Alcohol consumption, variceal bleeds, flapping tremors, ascites, weight loss, loss of libido, menstrual disturbances—-*suggestive of cirrhosis;* H/o travel to hepatitis endemic area; Jaundice
Clinical signs-Pallor, dry papery skin, scratch marks; Cachexia, clubbing – Biliary cirrhosis; Pedal edema- bilateral in cirrhosis; Lymphadenopathy- Supraclavicular fossa- malignancy; Stigmata of Chronic liver disease; Abdominal Examination; Ascites (distended abdomen, bulging flanks), Umbilicus stretched – transversely in ascites and vertically in ovarian cyst. Skin over abdomen-tense, shiny and transparent; Hepatomegaly, splenomegaly, pleural effusion; Right upper quadrant tenderness, palpable gall bladder; *Signs of liver cell failure*- parotid swelling, Dupuytrens contracture, gynaecomastia, fetor hepaticus, asterixis, testicular atrophy etc.; Cardiovascular system-Signs of hyperdynamic circulations may be present; Respiratory System**-** Signs of Hepatopulmonary syndrome; Central Nervous System- Look for signs of Hepatic encephalopathy.
Investigations-Basic- Haematological, Liver function test, kidney function tests
Management- Medical management of varices; Surgical management- Shunt; Liver failure- liver transplant

Chronic Renal Failure

*History***-**Pertaining to precipitating etiological factor, duration of disease, renal replacement therapy if any; History pertaining to renal status i.e., urine output, swelling of the body, breathlessness, activity level; History of antihypertensive, hypoglycemic agents, history of haematocrit improving measures (erythropoietin, blood transfusion); Past surgery/ dialysis schedule; History of immunization (hepatitis B)
*Clinical signs***-**General physical examination – pallor, blood pressure, oedema, nutritional states, presence of fistula; Respiratory system – infections/ effusion; Cardiovascular system- cardiomyopathy, effusion
Differential Diagnosis-To differentiate between acute/chronic renal failures and end stage renal disease.
Investigations-Haematocrit, urea, creatinine, electrolytes, total proteins, chest x-ray, ECG, Echocardiography, DPTA scan and their implications
Management-Implications of CRF on various body systems and their interactions with anaesthetic agents; Perioperative management of a patient for live related renal transplant/cadaveric transplant; Management of a patient with CRF for incidental surgery.

Severe Anaemia

History-Depend upon age, severity and speed with which it appears; Mild anaemia (8-10gm/dL)-loss of stamina, shortness of breath with exercise, palpitation; Moderate anaemia (6-8gm/dL)- Exercise capacity markedly reduced, exertion associated with headache, rapid exhaustion, dyspnoea, palpitation; Worsening of angina, claudication, heart failure; For type of anaemia-Sore mouth, difficulty in swallowing, craving for ice/dirt (pica) – Iron deficiency anemia;Life long h/o periodic severe bone

and joint pain – sickle cell anemia ;Jaundice, leg ulcers, gall stones – suggestions of hemolytic anemia; H/o prior transfusion; Drug/alcohol intake, Nutritional habits; Family h/o anemia, Racial background; H/o increased or acute blood loss from GIT, female genital tract (menorrhagia); Worms in stool; H/o chronic disease which may be associated with anemia i.e CRF, connective tissue disorders, infections, malignancy, diabetes, AIDS, alcoholic liver disease and even pregnancy; H/o conditions which will worsen O_2 delivery to tissues i.e. COPD, restrictive lung disease; H/o conditions which will predispose patients to increased risk from anemia. Eg. IHD
*Clinical signs***-**Pallor, koilonychias; Apical impulse forceful; Strong peripheral pulses with wide pulse pressure; Mild systolic/ holosystolic murmur secondary to increased blood flow and turbulence; Paresthesia, spleenomegaly
*Investigations***-**Hb, blood count, reticulocyte count, Haematocrit, MCV, MCHC, Platelet count, peripheral blood smear (Cell Morphology- cell size, Hb content, Anisocytosis, Poikilocytosis, Polychromasia); Reticulocyte count; Iron supply studies; Serum Iron; Total iron binding capacity; Serum ferritin; Marrow iron stain; Bone marrow examination; Aspirate; Erythroid/granulocyte precursor ratio; Cell Morphology; Iron stain; Biopsy; Cellularity; Morphology
Management-Blood transfusion; Iron- oral or intravenous; Vitamin B12

Benign Hypertrophy of Prostate for TURP
History-To bring out feature of obstructive uropathy; As these patients are usually elderly to bring out associated fractures and co-morbidities; Drug history
*Clinical signs***-**General physical examination including examination of spine; To bring out features of associated co-morbidities
Differential diagnosis-Benign and malignant conditions
*Investigations***-**Haematocrit; Pertaining to co-morbidities
Management-Type of anesthesia- advantages and disadvantages; TURP syndrome- diagnosis and treatment; Bladder perforation; Pacemaker and TURP; Radical prostectomy- Robotic surgery.

Paraplegia/GBS/Motor Neuron Disease
History-Evolution of the disease highlighting the main relevant complaint; History of any associated medical diseases; Past history- Similar problem at any time and what was done? Any previous anaesthesia exposure? Any member in the family had complication because of anaesthesia.
Clinical Examination- Whether the concerned organ system has been focused on candidate. He must examine the cases from head to toe. Hemust examine all the systems as a routine followed by specific system examination.

Investigation- Must mention the basic investigations and should be able to Recognize abnormality and normal range .No battery of unnecessary tests since cost factor is also important. Special and advanced investigations must be mentioned by the candidate to arrive at a diagnosis.
Differential Diagnosis-Important because many of the signs and symptoms may be found in other diseases also. This will complete the probable provisional diagnosis and the candidates clinical knowledge will be known.
Non Surgical Management- Knowledge about the optimization of the patient. Anaesthetic management includes ASA grading ,premedication, choice of anaesthesia technique and why the proposed technique was chosen. Monitoring and post operative management regarding pain ,any special orders to the Post operative nurse .Oxygen, any post op. mechanical elective ventilation of lungs.
Any other- Candidates may be asked to tell about the technique and the various drugs available for the specific case in the institution where they got trained. In the anaesthetic management the candidate must be able to say why he/she is managing the case particular way and the justification for doing so.

Pregnancy - Normal & related disease
*History***-** Pertaining to the pregnancy i.e., weeks of amenorrhoea, nausea, vomiting, gastritis, etc.; Pertaining to associated co-morbidities if any

*Clinical signs***-**Emphasis on eliciting anemia; Features pertaining to pregnancy induced hypertension; Airway examination; Fundal height and fetal heart sound
Differential diagnosis-Hydatidform mole, Ovarian cyst
Investigations- Implications of various physiological changes of pregnancy
Management-To be able to discuss the various physiologic changes in pregnancy and their implications on anaesthesia; Labour analgesia- method and modalities; Failed intubation drill

Short cases

Cyanotic Heart Disease (TOF, ASD, VSD, PDA)
History-To bring out the onset of cyanosis (congenital cyanotic vs Eisenmenger syndrome); To elicit history of cyanotic spells; To elicit history of chest infections/failure to thrive/cardiac failure/any interventions in the past.
Clinical examination- To bring out the physical/developmental milestones in case of pediatric age group; To recognize cyanosis and other signs of chronic hypoxemia; To identify features of associated congenital anomalies.
Investigations-Typical ECG, X-ray, echocardiography, cardiac catheterization, blood gas findings.
Differential Diagnosis-To be able to enumerate other congenital cyanotic heart diseases/ to discuss Eisenmenger syndrome.
Management-To be able to discuss the rationale of anesthetic management based on the patho-physiology (To discuss various induction/volatile agents/opioids and their effects on PVR, SVR and heart rate)

Buerger's disease
*History***-**History of smoking, hypertension, cough, pain in calf muscle or Foot, intermittent claudication.
Clinical signs -Check for pulses- peripheral, low volume; Skin colour, blackish to brownish; BP may be raised if uncontrolled; Local examination, specially lower limbs
*Investigations***-**Haemoglobulin; X-ray Chest; Doppler; ECG
Management-Abstain from smoking; Vasodilator; Amputation; Neuraxial block for sympathectomy and Anaesthetic Management; Antiplatelets or anticoagulants; Calcium channel blockers

Cleft lip and cleft palate
*History***-**History of feeding; Voice change
Clinical signs **-**Cardiovascular for associated anomalies; Other congenital anomalies; Local examination
Investigations-Haemoglobulin; Investigations for associated disorders
Management- Surgery; Induction- Ideal Method; Intubation- types of endotracheal tube; Anaesthetic Management; Pain-relief- Infraorbital nerve block

Meningocele or Meningo-myelocele
History-History of trauma; Any history of neurological deficit
Clinical signs- Local Examination; Other associated congenital anomalies; Full neurological examination
*Differential diagnosis***-**Lipoma
*Investigations***-**CT scan; MRI; Associated congenital annomalies
*Management***-**Surgical excision; Prone position; Care during induction- Position of head; Choice of endotracheal tube; Post-operative neurological assessment
*Any other***-**Development problem

Hydrocephalus
History-H/o vomiting; History of neurological problems; History of tuberculosis
Clinical signs- Signs of raised intracranial pressure (Bulging of Anterior fontenalle); Pulse rate; Cushing triad; Sun setting sign; Neurological examination
Differential diagnosis-Macrocephaly
Investigations-CT head; Fundoscopy; Serum electrolytes

Management-Medical; Surgical- Shunts, types of shunt; Anaesthesia problems- Induction; Developmental problems; Causes of hydrocephalus; Peri-operative problems

Tracheostomy

*History***-**Questions pertaining to indications of tracheostomy; History of respiratory problems; History of change of voice; History of loss of weight or appetite; History of trauma; History of prolonged ventilation

Clinical signs-Vitals- Pulse, blood pressure; CVS examination; Respiratory system examination; Associated injury in trauma patient

*Differential diagnosis***-**Ca larynx; Ca thyroid

*Investigations***-**Pertaining to primary cause; X-ray Chest, neck

Management-Types of tracheostomy; Percutaneous tracheostomy (PCT); Steps of PCT; Complications of tracheostomy; Indications of tracheostomy

Burn Contracture

*History***-**Type of burns-flame burns, steam burns, chemical or electric burns; History suggestive of inhalational smoke injury as it could cause respiratory impairment; Period elapsed since the injury, as it would dictate choice of muscle relaxants; Associated comorbid conditions

*Clinical signs***-**General physical and systemic examination; Examination of the contracture; Contractures of the face, head and neck—can result in a difficult airway ; Chest contractures can lead to a restrictive pulmonary disorder; Contractures involving major joints can produce difficulty in positioning & placement of monitoring modalities as well as IV access

Exam of the airway is specially important- Patency of the nares (may be obliterated by contractures); Mouth opening-at least 2 finger breaths between the incisors is desirable in adults (maybe reduced due to contractures) ; Teeth- prominent upper incisors, loose teeth ; TM Joint- Movement (maybe restricted in facial contractures); Submental space- hyometal/thromental distance should be > 6 cm (reduced in neck contractures); Neck movements- may be reduced in neck contractures; Hoarseness/ stridor/previous teacheostomy suggests Stenosis of trachea; Distorted face anatomy makes mask ventilation difficult.

*Differential diagnosis***-**Different type of burns can have different consequences; Flame burns-commonest They maybe associated with smoke injury ; Inhalational smoke injury- causes more airway damage; Steam burns-cause airway damage even below the cords ; Electric burns-produce far more destruction than the surface indicates

Investigations-Hb, TLC, DLC, PCV, Coagulation profile; Urine RE; LFT; Serum electrolytes; Blood sugar; ECG ; Xray chest,PFTs ; Xray cervical spine (in severe neck contractures)

Management-Resusucitation and management of burns patient; Surgical management of acute and chronic burns patient; Anesthetic management

Congenital Telipes Equino Varus (CTEV)-It is a congenital deformity of foot in three planes involving several joints and occurs in the background of constant change due to growth. It is also known as 'Club foot'

History-Presenting complaints- Though, the history dates back to birth, a child with CTEV may present sometime after birth, often as late as adulthood with deformity of foot; Family history- The risk of CTEV is increased 20 times if a 1st degree relative has the condition. It may be associated with some genetically inheritance; Perinatal history- Increased intra-uterine pressure may predispose towards development of CTEV. The child may be suffering from cerebral palsy; Post conceptional age if relevant; Developmental history- History of inability to bear weight on lower limbs /walk.

*Clinical signs***-**Small stature; Generalised myopathy; Kyphoscoliosis; Neurological deficit due to neurological cause- Cerebral palsy, Spina bifida; Deformities at other joints (Arthrogryposis multiplex congenita); Congenital hip dislocation

*Investigations***-**X-ray Foot- AP & Lateral views with foot in whatever corrected position possible; X-ray spine- In cases of spinal deformities such as spina bifida; If child has Cerebral palsy- EEG,

Psychometric and sensory evaluation, Assessment of language and learning disabilities, Chromatography of plasma and urine for inborn errors of metabolism, CT/MRI brain- To rule out hydrocephalus/ICSOL; If child has Muscle dystrophies- EMG, Muscle biopsy, Serum Creatine phosphokinase.

Management-Non-operative treatment-Manipulation alone, Manipulation and strapping or corrective plaster, Maintenance devices, Dennis brown splint, CTEV shoes.

Operative Methods- Soft tissue release- May be sufficient in younger children (<3 years); Posteromedial soft tissue release on posterior, medial and plantar sides; Limited soft tissue release- - For equines alone- Posterior release; - For adduction alone- Medial release; - For cavus alone- Plantar release; Tendon transfers- Minimum age- 5 years- Tibialis anterior transferred to outer side of foot; Osteotomy- Minimum age- 3 years; Open-wedge osteotomy of calcaneum to correct varus; Ilizarov's technique- Different components of deformity are corrected by gradual stretching using an external fixator.

Any Other-Anaesthetic management-Manipulation, strapping and corrective plasters can be done under sedation; Procedures like soft tissue release may require short GA; Corrective osteotomies can be done under GA with Caudal block; Any other problem or drug interaction

Diabetic Foot Ulcer

History-Mode of onset – How has the ulcer developed- Spontaneously or following Trauma; Durations – How long is the ulcer present there?- Acute Onset or Chronic Onset; Pain – Is the ulcer painful? Discharge – Does the ulcer discharges or not? Nervous Disease – Tabes dorsalis, syringomyelia, transverse myelitis and peripheral neuritis; Chronic Disease – Syphilis, TB, Diabetes; History of diabetes and its treatment; History of Retinopathy, Nephropathy, Neuropathy,Autonomic Neuropathy, History of – Sleep apnoea, Postural Hypotension, Regurgitaion, vomiting, diarrhea; CVS involvement – History of HTN, IHD and patient may complaints of chest pain and dyspnoea; Management of diabetes; Past History. History of hospitalization in the past; Drug history and ingestion of toxic metabolites, history of infection in the childhood like mumps, Food habits; Family History of Diabetes.

Clinical signs-Examination from head to toe- Nourishment, Built – Thin, Obese; Vitals- PR – Resting Tachycardia , Beat to beat Variation – absent with deep breath valsalva maneuver. Arrhythmias- BP –Monitoring of blood pressure- Including testing for orthostatic changes- RR -Altered regulation of breathing; Temperature – increased during infection; Local Examination; Systemic examination ; Respiratory System – Look for respiratory infection; Assess the air way (mobility of A-O joint); Per Abdomen – NAD; CNS – Superficial and deep tendon reflexes

Differential diagnosis-Traumatic, Ischaemic, venous, trophic, Tropical, Tuberculous

Investigations-Blood sugar, glycosylated (HbA1C), depending upon the organ involvement

Management-Medical Management - TREATMENT OF DIABETES; Surgical and anaesthetic Management

Squint

History-History of deviation of eyes; History of long sightedness; History of measles, viral fever, meningitis etc.; History of muscle weakness elsewhere (squint can be manifestation of underlying myopathy); History of any surgery under general anaesthesia - Complications like convulsions rigidity, hyperthermia to rule out malignant hyperthermia

Clinical signs-Detailed general and systemic examination to rule out associated systemic diseases, congenital abnormalities and muscle dystrophies; Airway Assessment - Patient with squint may have associated congenital syndromes which affect airway (Downs syndrome, Marfan's syndrome)

Investigations-Routine investigations like Hb, TLC, DLC, Urine - examination are needed in most of the patients as per history and examination

Management-Anaesthetic Management of Strabismus or squint surgery is guided by age &. co-operation of the patients. Strabismus surgery in adult can be performed under local anaesthesia with or without sedation; Adult Un-coperative patient can be managed with total intravenous anaesthetic

technique with sedative and narcotic drugs; Children will always require general anaesthesia for corrective surgery. Strabismus is the commonest eye operation in children; Before discussing the anaesthetic management, the special anaesthetic problems associated with Strabismus surgery are to be considered; Oculocardiac reflex; Strabismus & Malignant hyperthermia; Antiemetic prophylaxis can be provided with Ondansetron 0.1 mg/kg IV to prevent postoperative vomiting.

Spots

Electrocardiogram-The candidates should be able to diagnose the following ECGs- ECG – Arrhythmia's (Atrial Fibrillation,Ventricular Fibrillation,,Atrial flutter, Ventricular Tachycardia, Supravntricular tachycardia, Junction rhythm); Pacemaker; Coronary Artery Disease with Myocardial Ischaemia and infarction; Left Ventricular Hypertrophy and Right Ventricular Hypertrophy; Right Bundle Branch Block; Left Bundle Branch Block

X-ray Chest-Description of the x-ray; Whether AP or PA? How will you differentiate? What are the characteristics or findings of the following conditions- Pneumothorax, Malignancy, Pleural Effusion, Bronchietasis, Emphysema, Hydropneumothorax, Consolidation, Collapse of lung, Pericardial effusion, Cardiomegaly, Mitral Stenosis, Plethoric lung field (PDA), Pulmonary Edema, Adult Respiratory Distress Syndrome, Oligaemic lung field, Fractured ribs, Foreign Body in Bronchus, Foreign Body Oesophagus, Pulmonary Koch's Miliary TB, Inter costal drain, Pacemaker, Diaphragmatic hernia, Eventration of diaphragm, Lung Cyst-Congenital & adult, Hydatid cyst; What are the clinical findings in the above conditions? Management and treatment of the above conditions?

CT-Chest-The candidates and teachers should know the interpretations of CT-Chest of the above conditions

Equipments

*Anesthesia Machine***-**Safety Mechanisms; Physics of pressure reducing valves; Rotameter; Physics about the vaporizers

Anesthesia Circuits-Different types of circuits; Mapleson's circuit; Functional anaylysis of different anaesthesia circuits; Testing of circuits; Methods and flow rate for preventing rebreathing.

Anesthesia vaporizers-all old and new-Basic principle, classification, description, pressurizing and pumping effect; Functioning under low flows; Functioning at high altitutde

*Oxygen analyzer***-** Principle; Limitations; Masimo technology; SpO2 monitor/oxi meter/tracings

Airways-Oropharyngeal and ,Nasopharyngeal-Sizes, Method of insertion, problems; Peter-Safer-Description and indication

*Identification , Indications, and Method of use***-**Stylet; Magill Forceps; Gum elastic bougie; LMA-classic, flexible, ILMA, proseal LMA; Endotracheal tubes-PVC, PVC reinforced, Red Rubber, RAE-north pole, Cole tube; Light wand; Track light; Laryngoscopes-Macintosh, Miller, McCoy; Face masks-anatomical, AMBU, Rendall -Baker Souzeck; Ventimask; Polymask; T-piece; Nasal Prongs; Nasal Catheter; NIV-BiPAP; Ventilator-ICU, Anesthesia; Needle-spinal, epidural/CSE sets; Bronchial Blocker; Central Venous catherters-single lumen (70cm), triple lumen; NMJ Monitor; EKG monitor; EtCO2 monitor and tracing; ABG cards- Hypoxia, hypercarbia, acidosis- metabolic and respiratory, alkalosis- respiratory and metabolic; Flow Volume Loops.

Resuscitation- *CPR dummy*-Demonstration of resuscitation under different emergency scenario

Skeleton for blocks-The candidate should be able to demonstrate all types of blocks for acute or chronic pain relief

Problems cards on short clinical situations-Thyrotoxicosis, Hypothyroidism, intraoperative bronchospasm, intraoperative pneumothorax, anaphylaxis; The candidate should be able to diagnose the scenario and should briefly know the management.

IV-fluids Crytalloids-Normal Saline, Dextrose –Saline, Ringer Lactate, 5% Dextrose , 10% Dextrose ; Colloids-hetastarch, pentastarch ,dextran, Haemaccel and albumin; The candidate should know-The composition, Osmolarity, pH, Tonicity, Indications and contraindications of these

Drugs Anaesthetic-Opioids-morphine, pethidine, fentanyl, pentazocine; Muscle relaxantas-vecuronium /pancuronium .d-tubocurarine, Gallamine, anticholinesterases, Pyridostigmine, Neostigmine ; Induction agents- -Thiopentone, Propofol, Etomidate, Ketamine; Benzodiazepines-Midaxolam, Diazepam; Vasopressors and inotropes-Vasopressin, Dopamine, Dobutamine, adrenaline/ noradrenaline/isoprenalie ; Vasodilators—Nitroglycerine, sodium nitroprusside ; Beta-blockers-Esmolol, propanolol ; Potassium chloride, Calcium gluconate, Calcium chloride, Magnesium sulphate; Local Anaesthetic agents- Cocaine, lignocaine, bupivacaine, tetracaine, prilocaine, procaine, cinchocaine ; Nasal Decongestants- Otrivin; Inhalational agents- Isoflurane, Halothane, sevoflurane, ether, trilene, chloroform; SAIDS-paracetamol-suppository; Inj.Ramtac/perinorm/Na citrate; Dilantin sodium- Malignant hyperthermia; Phenytoin; The candidate should know-The type and group of the drug, Indications and contraindications, Toxicity and complications, Dosage.

ENT

Nasopharyngeal carcinoma

History **-**duration of symptoms; Unilateral hearing loss from a middle ear effusion; Neck mass; Nasal; obstruction or epistaxis; Headache; Double vision; Dryness of eyes; Facial pain

Clinical examination **-**Nasal cavity for mass, Diplopia, Opthalmoplegia, Horner's syndrome, Neck for neck nodes, Examination of the nasopharynx

*Investigations***-**Contrast CT with bone and soft tissue windows, MRI, Bone scan, Positron emission tomography (PET) to access questionable neck nodes, Chest x-ray, Routine CBC, chemistry profiles and liver function test, Anti-EBC serologic test for detecting and determining the prognosis of nasopharyngeal, Carcinoma, Audiogram, Antibody – dependent cellular cytotoxicity (ADCC) assay. High titers of this antibody are related to better long – term survival.

Differential diagnosis- Nasal polyp, Angiofibroma, Pailloma, Scwannoma.

Non surgical management -External beam radiation therapy- directed at the primary lesion and the upper echelon lymph nodes; Chemotherapy - as palliative therapy

Surgical management **-**Biopsy for histologic examination and for EBV testing; Surgical resection in this region - Rarely indicated as a primary treatment

Surgical approaches **-**The infra temporal fossa and transported temporal bone approaches, The transpalatal approach, Transmaxillary and transmandibular approaches, Radical neck dissection in cases of successfully treated primary tumor with regional failure, Myringotomy with ventilation tube placement before radiation therapy.

Chronic suppurative otitis media

These are broad guidelines for the resource person to highlight the various 'must know' areas for a post-graduate resident. The emphasis is equally on concepts, facts and skills. It must be reinforced that any of the areas mentioned could well be a take off point. For an elaborate discussion.

History- a. Symptomatology- Elucidate with clarity the temporal evolution of various symptoms from onset to presentation; Relevance of negative history. b. Otorrhea-i. Explanation for foul smelling otorrhoea in attico-antral disease; ii. Explanation for persistent discharge in in attico-antral disease; iii. Explanation for blood stained discharge in attico-antral disease - Other causes of blood stained otorrhoea; iv. Old concept of classifying otitis media as active, quiescent and inactive; its significance in relation to Middle Ear Risk Index (MERI); v. Explain common microbes involved in otorrhoea in Indian context and the role of microbiological tests in assessing pathogen . When and how will you take an ear swab? c. Hearing Impairment-i. Assessment of severity of hearing loss from history; ii. Assessment of type of hearing loss from history; iii. Assessment of social disability from hearing loss. d. Tinnitus- i. Clinical significance of tinnitus in otitis media. e. Vertigo- i. Clinical significance of vertigo in otitis media. f. Otalgia- i. Clinical significance of otalgia in otitis media; Awareness of common problems like associated otomycosis and external otitis and uncommon ones like subdural abscess. g. Other relevant points-i. Elicit and explant the pathognomonic symptoms like persistent fever, headache, vertigo, tinnitus and facial paresis which herald complications; ii. Antibiotic (systemic & topical) usage history in persistent otorrhoea, Role of topical antibiotics, Risks associated with long

term use of antibacterial agents especially gentamycin in discharging ears; iii. Significance of evaluating common co-morbidities in otitis media- Diabetes mellitus, Tuberculosis, Hypertension

Clinical Examination -a. Ability to explain a procedure to the patient before doing it; b. Demonstrate technical knowledge of common examination equipment; i. Bull's eye lamp and head mirror; ii. Otoscope and Siegel's speculum; iii. Tuning Forks; c. Demonstrate the skill in-i. Otoscopy; ii. Pneumatic otoscopy; iii. Fistula Test; iv. Routine tuning fork tests especially masking with Barany's noise box for bone conduction assessment ; v. Free Field testing for Conversational Voice and Forced Whisper ; vi. Ataxia test battery; vii. Assessment of nystagmus; viii. Fistula Test ; ix. Dix Hallpike manouvre ; x. Testing facial nerve function; xi. Aural toilet; d. Demonstrate the otoscopic findings in the case (preferably on a videotoscope). He should be able to conclude at the end of otoscopy- i. Whether tympanic membrane is normal or abnormal; ii. If abnormal, whether it is intact or perforated; iii. If intact, the degree and scale of retraction or atelectasis; Define the position, extent of retraction pockets; - Demonstrate presence, location and extent of cholesteatoma, granulations and polypi; Demonstrate associated bone destruction especially of outer attic wall and postero- superior bony canal; iv. If perforated, differentiate between central and marginal perforation- If central, position, size and other characteristics of perforation, If marginaL in addition to the characteristics of perforation, demonstrate epithelial ingress into middle ear, if any ; Demonstrate the presence of TM and ME tympanosclerosis; - Assess the status of middle ear mucosa; - Visualise other middle ear structures; - ossicles, Eustachian tube, hypotympanic air cells; e. Explain the basis and interpretation of -i. Pneumatic otoscopy; ii. Free Field assessment of hearing; iii. Tuning Fork tests, iv. Spontaneous & induced nystagmus - Basis ofVOR v. Fistula test, vi. Ataxia test battery - Romberg's, Unterberger's, Straight Line Walking, Tandem Walking etc

Investigations-a. Otomicroscopy and otoendoscopy , Indications and relative merits of each; b. Ear swab for Gram/Ziel Nielson Staining and culture/ sensitivity- Basics of staining techniques, Ideal method of taking ear swabs; c. Patch test - its relevance in today's practice; d. Pure Tone Audiometry- Basics of clinical audiology, Explanation of terms - Pure tones, Frequency, dB, SPL, HL, SL, Speech frequencies, Threshold, Averaging Thresholds, AB Gap; - Identification of legends and notations in an audiogram, - Principle and practice of masking in PTA; e. Immittance; Principle and interpretation of tympanogram 'Principle and interpretation of Acoustic reflex; f. X'ray mastoid Schuller's view ; . Indications-ii. Degree of pneumatisation and its relevance in CSOM , Other plain skiagrams for imaging of temporal bone - Law's, Towne's and per -orbital views; g. Current role of CT scans/ MRI for evaluating middle ear

Differential Diagnosis-Differentiate between Tubotympanic Vs Atticoantral disease; In attico-antral disease, differentiate between- Congenital, Primary or secondary acquired cholesteatoma, Epitympanic vs meso tympanic cholesteatoma and gradations thereof ; Concept of persistent mucosal disease; Differentiate between complicated vs uncomplicated CSOM

Non-surgical Management-Principles of medical management in an active tubotympanic CSOM; Practice of chemical cauterization;Practice of office myringoplasty;Principles of conservative management of retraction pockets! small localized cholesteatoma

Surgical Management- Prognostication of outcome in middle ear surgery -Concept of Glasgow benefit plot, Middle Ear Risk Index etc.; Soft tissue approaches in middle ear surgery - i. Postaural, ii. Endaural, iii. Endomeatal, iv. Variations of the above; Conceptual difference between myringoplasty and tympanoplasty; Conceptual difference between onlay and underlay - i. Indications for onlay and underlay technique, ii. Complications of onlay and underlay technique; Types of tympanoplasty - i. Wullstein's original classification, ii. Current modifications; f. Middle ear ossicular chain reconstruction - i. Use of homologous tissues especially incus, ii. Middle ear prosthesis - various materials, design, results and complications.

Any other- Knowledge of medico-legal aspects especially provisions of Consumer Protection Act; Ability to take a proper informed consent of various surgical procedures; Ability to inform a bad outcome with honesty, dignity and empathy; Knowledge of assessment of disability with regard to hearing loss according to the current labour and company laws; Awareness of recent advances in

the areas of - Advanced audio-vestibular evaluation BERA, CE~ CNG. OAE, Newer Waging techniques like virtual endoscopy, 3D Waging, fMRI(functional MRI) etc.,Technology like lasers in middle ear surgery

Cleft lip and cleft palate

History- Incidence 1 in 800; Females are more affected by cleft lisp; A cleft lip is either unilateral or bilateral; Asians are most commonly affected; A history of maternal smoking, phenytoin use or use of diazepam is predisposing factor; Feeding difficulties in newborn leads to anaemia. Surgical repair si performed within the first few days; How the mother feeds the baby.

Clinical Examination-Condition of the newborn for fitness; Unilateral or bilateral defect; Extent of detect; Any other congenital defects

*Investigations***-**Haemoglobin; Complete haemogram; Routine preoperative check up in the frist year of life; X-ray chest to find out any infection/atelectasis/aspiration; To check for suitable endotracheal tube and laryngoscope; Veins for IV route; If the child is irritable suitable premedication; Differential Diagnosis not required

Non-Surgical Management-Corrrect the anaemia; Suitable antibiotics for chest infection; If the defect is small two edges of the cleft are brought to gether. Very few patients are benefited; Proper feeding

Surgical Management-Many anaesthetic techniques are used; Small defect-Rectal sedative + Local; A mask induction with Inhalational agents like Halothane, Sevoflurane; Endotracheal tube- RAE, Nylon reinforce, Oxford ; Modified Jackson Rees circuit, Paediatric Bains circuit; Nitrous oxide, Oxygen, Halothane, or Sevoflurane or short acting muscle relaxant; Proper premedication; Precordial stethoscope and monitors

Complications-Endotracheal tube may come out; If excessive blood loss then shock occur; If proper anaesthetic circuit is not used then rebreathing and CO2 retention; If not properly covered then fall in temperature and shock; Proper fixation of the endotracheal tube; Soft arm restrains are used to prevent infant handling the repair; Calculated dose of local anaesthetic with epinephrine should be used as per body weight.

Cleft palate

Family studies show siblings of patients with cleft lip have increased incidence of cleft palate; Males are more affected by cleft palate; Smoking, Phenytoin treatment or Diazepam consumption may predispose; Asians are more affected Cleft palate occurs when hard palate and soft palate has not joined; Feeding difficulty and airway com[promise occurs. Because of regurgiatioan then chest complications occur; Anaemia and general weakness occurs; Same investigations like cleft lip; Intubation is more difficult in cleft palate; There is more blood loss in cleft palate surgical resection; Surgical correction by 12 months of age, speech should not get affected; If cleft lip and cleft palate occurs then lip repaired first; Check for other congenital defects; Anxiolytic premedication is essential; Secure intravenous line; Keep rolled gauze ready to put in to defect before laryngoscopy; Inhalational induction by Halothane or Sevoflurane; If surgeon wants to use adrenaline then do not use halothane; Cleft palate is exposed by placement of Dingmans' mouth gag; A throat pack is often placed to prevent passage of blood in to the larynx; Anaesthetric techniques are the same like cleft lip, only problem is maintenance of airway hence proper fixation of the endotracheal tube is must; Prior to tracheal extubation thorough suction of the oro-pharynx without displacing he pack is essential; Some surgeons place a suture through the anterior portion of the tongue to avoid airway obstruction; Post operatively maintain airway and provide analgesia

Vocal cord nodule

History of present illness- change in voice; Nature- Hoarseness; Aphonia, Breathiness, Harshness – high pitch/ cracking, Feeble/whispered voice; Easy fatigability; Duration - Days/weeks/months/ years; Onset - Sudden/insidious; Progressive – Worsening , Diurnal variation-Static;Aggravatingand relieving factorsSmoking/ Drinking/ lectures/ singing; Associated symptoms Dysphagia, Dysphonea, Noisy breathing, Lump in the neck, Sinonasal symptoms, GERD symptoms, H/o repeated throat cleaning, Cough with expectoration, Pain in throat/ear

History of Past illness-H/o similar complaints; Any previous treatment; Any other surgery/medical illness; Previous intubation (prolonged)
Personal history-Voice abuse; Profession / Nature of job; Habits - Smoking / pan / Alcohol / tobacco; Food habits - Excessive Tea/coffee, Spicy food; Home Environment
Clinical examination-General examination; Routine ENT head & neck examination; IDL scopy Mobility of cords, phonatory gap; Patient is seated opposite with sit erect with the head and chest leaning slightly towards the examiner. Patient is asked to protrude his tongue, which wrapped in gauze between the thumb and middle finger. Index finger is used to push the upper lip out of the way. Gauze piece is used to get a firm grip of the tongue and to protect it against injury by the lower incisors; Laryngeal mirror, which has been warmed and tested on the back of hand, is introduced into the mouth and held firmly against the uvula and soft palate. Light is focused on the laryngeal mirror and patient is asked to breathe quietly. To see movements of the cords, patient is asked to take deep inspiration (abduction of cords), say "Aa" and "Eee". Movements of both the cords are compared; Structures examined by indirect laryngoscopy - Oropharynx - Base of tongue, ligual tonsils, valleculae, medial and lateral glossoepiglottic folds; Larynx- Epiglottis, aryepiglottic folds, arytenoids, cuneiform and corniculate cartilages, ventricular bands, ventricles, true cords, anterior commissure, posterior commissure, subglottis and rings of trachea; Laryngopharynx- Both pyriform fossae, postcricoid region, posterior wall of laryngopharynx; Description of the lesion- Site of mass, Colour, Size, Shape, Surface, Single or multiple, Pedunculated or sessile, Rigid video laryngoscopy.
Investigations-Fibreoptic RPL scopy; Strobosopy; Routine pre-op evaluation
Differential diagnosis (vocal nodules/vocal polyps)- Papilloma, Angioma, Fibroma, Circumscribed reinkie's oedema, Vocal cord cyst, Varcies, Haematoma, Polyposis mucosal thickening in elderly, Amyloidosis for vocal polyps
Non-surgical for nodule only- Voice rest, Speech therapy, Avoidance of irritants and habitual throat cleaning, Improve hydration, GERD treatment, Sinonasal symptoms
Surgical-ML scopy and biopsy – Conventional, Laser, Radio frequency, Coblation

Otosclerosis
History- H/o Present Illness - H/o Hearing Loss(Paracusis willissi, Tinnitus/Vertige, change of speech); Personal/Family History - H/o Similar complaints in family, Age of one set & Progress; Associated Factors- Pregnancy / menopause / Any surgeries, Relation of disease with surgeries, Association with osteogenesis imperfacta, Presence of Vander-Hoeve syndrome
Examination- Appearance of Tympanic memberane; Schwartze's sign – present / absent; Eust. Tube Function; T.F. Tests-
Investigation- Pure Tone audiometery – Presence / absence; Impedance qudiometry presence or absence of Carhart's notch.
Differential diagnosis- OM with effusion; Conductive deafness, Tympanosclerosis, Fixed Head of Malleus, Cong, stapes fixation, Ossicular discontinuity
Management- Non surgical – Role of sod. Fluoride; Surgical – stapedectomy – Procedure; Prosthesis - Indications, Contraindications.

Aural polyp
History- Patient may present with all features of CSOM(otorrhoea, otalgia, Aural bleed, Hearing loss/ Deafness, Itching in the Ear, Mass in the Ear; Complication of CSOM / Mastoid (Rare) - Brain abscess, Facial Palsy, Meningitis, Osteomyelitis
Clinical examination- Aural Polyps are the result of chronic inflammation of the middle ear or mastoid (can be manifestation of cholesteatoma). They are benign fleshy growth from the skin or glands of External Auditory canal or from the surface of tympanic membrane. Clinically polyps represent granulation tissue or oedematous mucosa arising from the mucous membrane of the middle ear protruding through a perforation in the tympanic membrane. Polyp is pedunculated while granulations are multiple and sessile. They usually arise from attic, Posterior superior margin of the tympanic membrane, Promontory, Eustachian tube orifice, Aditus ad antrum. Granulation tissue polyps in the

forming stage are soft, red and bleed readily when touched. Later polyps become more fibrous and the surface may be covered with metaplastic squamous epithelium. Clinical examination may or may not give evidence of cholesteatoma / CSOM. Inflammatory polyps are soft while neoplastic polyps are firm.

Probing- A probe can not be passed all around the polyp, if the polyp arises from the external auditory canal. The probe can be passed all around the polyp arising from the middle ear.

Investigaions- Discharge is sent for bacteriological and histological examination, Audiometry may show conductive deafness; Radiographs of the mastoid are normal in cases having polyps arising from the external auditory canal. A polyp associated with benign chronic otitis media may show a sclerotic mastoid while a polyp due to dangerous chronic otitis media shows a sclerotic mastoid bone with translucent areas of erosion caused by cholesteatoma. Patient having a neoplastic polyp may reveal erosion of the temporal bone; CT Scan of the ear will show better detail than the radiodgraph; Biopsy can settle the diagnosis.

Differential diagnosis- Granulomatous diseases or fungal infections, Neoplastic proliferation (Jugulo-tympanic paraganglioma), Glomus tumour, Langerhan's Cell histiocytosis (LHC), Foreign bodies of external auditory canal, Exostosis, Ceruminoma

*Non-surgical management***-** Antibiotics given systemically and locally may help early inflammatory polyps. In elderly individuals and who refuses operations, there is some place for conservative treatment like suction clearance and attic irrigation with white venigar solution and cauterization of granulations with 10% Silver Nitrate or with Trichloro Acetic Acid.

Surgical management- Polypectomy is performed with the help of an aural snare or a punch forceps. Polyps arising from the middle ear should not be avulsed to prevent damaged to the middle ear structures. Polypectomy helps the drainage of the middle ear and permits visualization of the ear drum for proper diagnosis. Causative factors should be treated like modified radical mastoidectomy for cholesteatoma, tympanoplasties for hearing, Radiotherapy / Chemotherapy / Radical operations for neoplasm.

*Any other***-** AIDS, Tuberulosis.

Fibroius dysplasia

History- Benign Fibroosseous Lesion; Slowly Progressive Disorder; Site of Involvement in Decreasing Order-Maxila, Mandible, Frontal Bone; Types Monostotic 70%; Polyostotic 27%; Involvement of Bones of Skull 10% in Monostotic; Involvement of Cranio Facial Bones 50% in Polyostotic; Condition Diagnosed Before The Age of 20 Years; Lesion Burns Out After Puberty; Malignant Transformation Uncommon (<1%)

Clinical features- Insidiou, Painless, Asymetric Enlargement of Affected Bone; May Involve Maxillary Sinus Zygoma, Sphenoid, Orbital Floor; Presents As Unilateral Facial Swelling, Deformity of Alvelar Margins, Loosening of Teeth; Swelling Bony Hard, Diffuse Painless

Investigatons-radiological - Ground Glass Appearance, Illdefined Margins; histological - Mixture of Fibrous And Osseous Tissue, Trabecular of Woven Bone Dispersed in Cellular Fibrous Tissue

Differential diagnosis- From Other Fibro Osseous Lesions- Ossifying Fibroma, Chronic Osteomyelitis, Pagets Disease, Osteo Blastoma, Florid Osseous Dysplasia

Treatment- Non Surgical - Observation Until Puberty; Surgical – always delayed **(**Curettage, Partial And Radical Excision, Gross Deformity, Proptosis, Loss of Visual And Auditory Acuity May Force Early Surgical Intervention.

Nasopharyngeal angiofibroma

History-Unprovoked torrential bleeding from nose; Nasal obstruction/Snoring/Mouth breathing; Anosmia/Hyposmia; Nasal intonation of voice; Headache denote coexisting sinusitis or dural compression; Blocked ears due to ET orifice block; Rarely diplopia & proptosisor or failing vision due to tenting of optic nerve; Recurrence after earlier incomplete surgery; H/O earlier surgery or radiotherapy

Clinical examination - Adolescent male; Red or pink or purplish smooth, lobulated/nodular mass pushin the septum to opposite side in the midst of secretion sin the nasal cavity & nasopharynx; Soft palate may be bulged with restricted movement; Fullness of cheek/temple or frog faced deformity; Proptosis; features of Sec' otitis media & conductive deafness; Trismus; Anaemia; Palpation can be done using soft palate as a curtain- Fibroangioma if very frim/Angiofibroma if softer; Intraoral palpation in the interval between the ascending ramus & the side of maxilla reveals disease extension to pterygopalatine fossa

Investigations - X'Ray-Paranasal sinuses, Lat' view nasopharynx & Base of skull; Tomogram; CT; MRI; DSA; Complete hemogram & Blood grouping with work uo for G.A.; Ophthalmologist's opinion; Neurologist's opinion(SOS); Excision biopsy

Differential diagnosis- Antrochoanal polyp with squamous metaplsia; Hypertrophic turbinate & adenoids; rathke's pouch or Thomwaldt's cyst; Nasonasopharyngeal rhinosporidiosis; Other tumours of nasopharynx such as- Chordomas/Fibromas/Teratomas/Hemangiomas/Condromas/Gliomas/ Rhabdomyomas/Fibrous dysplasia/Lymphomas ***NPC***

Management - Surgery - Prior embolisation, Approaches- Endoscopic approach for small tumors(Stage I) - Transfacial approach- Moure's lateral rhinotomy, Medial maxillectomy, Midfacial soft tissue degloving, Weber Fergusons incision transnasoantral approach, Transmaxillary, Maxillary swing; Oral approach to nasopharynx- Transoral, Transpalatal; Hormone therapy - Primary or adjunctive treatment(Diethylstilbestrol & flutamide)- To reduce vascularity; T. (30-40Gy) – reduces vascularity- Primarily for intracranial extensions & inoperable tumours

Cancer Larynx

History- Hoarseness of voice; Difficulty in breathing; Difficulty in swallowing; Pain, radiating pain to ear; Selling in the neck; H/O aspiration & cough; H/o smoking & alcohol consumption

Clinical examination–General-Comfortable/ in stridor;Cachexia; Throat-Dental/oral hygiene; IDL- mas description, cord mobility, airway adequacy; Neck-Lymphadenopathy – level; Laryngeal contour; Laryngeal crepitus; Laryngeal tenderness; Thyroid gland; Trachea; Abdomen; Respiratory system; Spine; Provisional diagnosis-TNM staging.

Investigations **–**DL scopy & biopsy; X-ray soft tisdsue neck-AP & lateral; X-ray chest; CT scan neck; Blood investigations; USG abdomen; Whole body bone scan

Differential Diagnosis-Tuberculosis of larynx; Squamous cell ca; Sarcoidosis; Scleroma of larynx; Basloid sq. cell ca; Verrucous ca; Scarcomatod ca

*Non surgical management***-**Palliative/curative radiotherapy; Cobalt-60/linear accelerator; Irradiation field perimetes; Dose 60Gy-70Gy; Duration 6-7 weeks; Complications of radiotherapy; Preparation of patient for RT; Role of chemotherapy

Surgical management-Micro laryngeal laser surgery; Conservation laryngectomy; Subtotal laryngectomy; Near total laryngectomy; Total laryngectomy; Extended total laryngectomy

Carcinoma laryngopharynx

History- Difficulty in swallowing; C/o stickin of food in the throat on swallowing; Pain radiatin pain to ear; Hoarseness of voice; Difficulty in breathing; Swelling in the neck; H/o aspiration & cough; Hemoptysis; Wight loss; H/o smoking & alcohol consumtion

*Clinical examination***-**General-Comfortable/in stridor,Cachexia/pallor; Throat-Dental/oral hygiene,IDL-mass description, cord mobility,. Airway adequacy, pooling os saliva; Neck-Lymphadenopathy-level, Laryngeal contour, Laryngeal crepitus, Laryngeal tenderness, Thyroid gland, Trachea; Abdomen; Respiratory system; Spine; Provisional diagnosis-TNM staging

Investigations-DL scopy & biopsy; X-ray soft tissue neck-AP & lateral; X-ray chest; Barium swallow; CT scan neck; Blood investigations; USG abdomen; Whole body bone scan

Differential Diagnosis-Squamous cell ca; Adeno ca; Lymphoma; Basaloid sq. cell ca; Adeno sq. cell ca; Lipa sarcoma; Lympho epithelioma

Non surgical management **-**Palliative/Curative radiotherapy; Cobalt-60/linear accelerator; Irradiation field perimeters; Dose 60Gy-70Gy; Duration 6-7 weeks; Complications of radiotherapy; Preparation of patient for RT; Role of chemotherapy

Surgical management **-**Lateral pharyngotomy; Total pharyngolaryngectomy; Total Pharyngolaryngoesophagectomy with gastric pull up(ONG's procedure)

OBG

Long cases

Primary amenorrhea- Student should know the following in a case of primary amenorrhea - Normal puberty changes, Definition of primary and secondary amenorrhea, Various underlying causes of primary amenorrhea - Presence of secondary sexual characters, Absence of secondary sexual characters; History; Examination - General weight, height, BP; Presence and extent of Secondary sexual characteristics; Perineal examination – Introitus, Hymen, Anal opening; Investigations - General – Haemogram, Hormonal profile (Thyroid, prolactin, LH,FSH, if indicated), Ultrasound – Pelvic organs, Kidney & Ureter; Management - Surgery for imperforate hymen, If lower tract normal - 1st withdrawal with progestecone, 2nd withdrawal with estrogen- progesterone; Psychosocial support

Vaginal discharge- The student should know the following in a case of vaginal discharge - Anatomy and physiology of vagina -Changes from puberty to reproductive age and to old age, pH of vagina, lactobacillus; Various causes of abnormal vaginal discharge and classical s/s of each; History including - Drug intake, Medical history, Recurrences, Sexual history / contraceptive use; Examination - Perspeculum examination, Bimanual pelvic examination; Investigations and diagnosis - Clinical diagnosis, KOH mount, gram stain microscopy, Pap's smear; Treatment - a. Medical treatment - Metronidazole, - Fluconazole, b. Vaginal pessaries – clotrimazole for candida, c. Cryosurgery for cervical erosion; For discussion should know cervical erosion, PID

Infertility- The student should know the following in a case of infertility - Definition of primary / secondary infertility; Various causes of infertility – Male factors/ Female factors; Normal semen perameters; Normal menstrual cycle, tests for ovulation; Tests for tubal patency – Hysterosalpingography, Sono salpingo infusion, Laproscopic evaluation; History (coital, menstrual, medical or surgical history etc.); Examination (Breasts for galactorrhea, pelvic infections etc.); Investigations for finding cause and evaluating treatment - Husband semen analysis, USG (uterus, follicle monitoring , PCOS), Role of hormonal evaluation e.g. LH,FSH, TSH, prolacting levels, Day 22 progesterone, Role of post coital test, Hysterosalpingography, Role hystero laparoscopy; Counselling for - Rubella Screen and vaccination, Hepatitis B virus & vaccination, Diet and weight control in obese, Smoking check, Psychological support, Folic and supplementation; Treatment - Drugs for ovulation induction – Clomiphene, letrozole, Gonotrophines, What is IUI and procedure of IVF, Other drugs- Metformin, d. When to do IVF/ICSI- indications, risks, success

Post menopausal bleeding- The student should know the following in a case of postmenopausal bleeding - What is menopause, physiological changes of menopause, definition of postmenopausal bleeding; Various causes of postmenopausal bleeding; History in detail – Drug intake – HRT, Medical history – DM, Hypertension, Family History – of cancer; Examination – General - P, BP, Catchexia, Lymphnodes, Breasts, Perspeculum – vaginal / cervical status, Bimanual examination – fornix palpation for extent of disease; Diagnosis and investigations – Clinical diagnosis, Biopsy of growth – HPE, Ultrasound – adenexal status, Role of MRI / CT, Staging investigations of cancer cervix, Role of cystoscopy, sigmoidoscopy, IVP, General investigations – Blood test, CxR etc. to check for fitness of extended / radical surgery; Etiology of cervical cancer; FIGO staging of cervical cancer; Treatment options - Radical hysterectomy -Indications, Procedure, Results in survival, Complications, Radio therapy – in which cases - When to give preoperative/ postoperative, Survival results, Short term/ long term complications, Role of adjuvant chemotherapy and drugs used; Post operative case – Immediate; For discussion should know about Pap's Smear, CIN – Colposcopy and management options for CIN.

Fibroid uterus- The student should know the following in a case of fibroid uterus - Causes and definition of menorrhagia, polymenorrhia, polymenorrhagia, .metromenorrhagia; Etiology of fibroids – Associated factors; Pathophysiology of fibroids – Gross and microscopic appearance, Dependance

on hormones; History in detail – Duration, amount of bleeding, Affect on health, social well being, Urinary or bowel problems, Desire for surgery; Examination – General – pallor, abdominal lump, Pelvic examination – size of uterus, adenexal pathology; Investigations and diagnosis – Clincal differential diagnosis, Ultrasound, Role; Treatment – Role of medical therapy – GnRH agonists, Role of levonorgestial UCD, Hysterectomy, When to do TAH with BSO or without BSO, Role of myomectomy - Risk of procedure, Long term consequences – eg LSCD, rupture uterus, Role of uterus arteryemboligation - Advantages and disadvantages, Clinical evidence, Role of laparoscopic removal of fibroids, Role of hysteroscopic removal of fibroids; For discussion should know definition, eteology and treatment of adenomyosis

Prolapse uterus- Student should know the following in a case of prolapse - Anatomical supports of uterus and vagina; Definition and degrees of prolapse – Anterior vaginal wall prolapse – cystocele, Uterine/ Vault prolapse, Posterior vaginal wall prolapse / enteroclle / rectocele - Ist, IInd and IIIrd degree, procidentia; Etiology of prolapse –Attenuation of supports due to child births, difficult labours, menopause etc., Congenital, Aggravating factors – obesity etc.; History in detail with special emphasis on - Urinary symptoms – stress/ urge incartinence, voiding difficulties, Bowel symptoms, Coital problems, Discharge, bleeding; Examination –General condition – arenxia, other debilitatring conditions like diabetes in elderly as surgical fitness needs to be evaluated) etc., Pelvic examination P/S and P/V; Investigations - Diagnosis is primarily clinical, routine blood and urine tests for general status and urine infection respectively; Treatment – It depends on - Age and parity, Sexual and fertility desires, Severity of signs and symptoms, Type of prolapse, Associated urinary symptoms eg. SUI, Medical risk for surgery, Conservative treatment –Lifestyle advice- diet, weight, excuses, Pelvic floor (Keigel) excuses, Ring pessaries; Surgical treatment - Anterior colporapphy, Colpo perineo rapphy, Manchester repair, Vaginal hysterectomy, Sacrospirosis colpopesy/ sacrocolpopexy, Abdominal shing surgeries, Copoclerisis - Indications, contra-indications, complications of each; Decubitus ulcer, Role of hormone replacement therapy, Prevention of prolapse – Care during labour and delivery, Postnatal exercises, Surgical technique to prevent prolapse during pelvic surgeries like TAH, vaginal hysterectomy for other gyneacological conditions.

Pregnancy induced hypertension- The student should know the following about PIH - Changes in blood pressure during normal pregnancy; When to label a case as hypertension during pregnancy; Classification of hypertensive disorders in pregnancy; Various factors responsible for PIH (Pathophysiology of PIH) – Vasospasm, Cardiovascular changes, Haematological changes, Kidney and live changes, Brain and retinal changes; Diagnosis of PIH – BP, Edema, Proteinuria, Signs and symptoms of impending eclampsia (Headache, epigastric pain, oligourra etc.) ; Diagnosis of severity of PIH –Clinical, Investigations – Hematocrit, Proteinuria, Electrolytes, Renal function tests, Fundus examination of eye, USG and Doppler evaluation; Treatment of PIH – When to admit in hospital, When to start oral medications,Various antihypertensive medications used in pregnancy, Role of diuretics (when and how to use safe diuretics); Termination of pregnancy –When to terminate pregnancy, Mode of termination of pregnancy; Prevention of PIH - Role of aspirin; Treatment of impending eclampsia or eclampsia –Various anticonvulsants used in pregnancy, b) Various regimes used; Care during delivery – PIH, Eclampsia / eminent eclampsia; Post partum counseling

Anemia in pregnancy - The student should now the following about anemia in pregnancy - Diet; Calorie intake; Iron and other dietary factor requirement in pregnancy; Iron metabolism; Types of anemia; Effect of anemia on mother and fetus; Investigations in a case of anemia with pregnancy; Routine investigation (common causes); Investigations related to uncommon causes (Thalassemia, sickle cell anemia); Treatment of case of anemia; Iron supplementation –Oral iron preparation, Injectable iron preparation; Folic acid supplementation; Place of blood transfusion; Care during labour in this case; Post partum counseling.

Antepartum haemorrhage- The student should know the following in a case of pregnancy with APH - Definition of APH; Causes of APH; Diagnosis of APH (causes) – History – in detail including

assessment of blood loss, Clinical examination, to confirm extent of blood loss, P/A examination, Investigations – Haemogram / Ultrasound; How to transfer a case of APH from remote area to referral centre, what all precautions to be taken; Management of placenta previa; Role of expectant management; When to terminate pregnancy; Mode of termination of pregnancy; Care during delivery; Case during cesarean sectron; Prevention of postpartum haemmorrhage in such cases; Treatment of post partum haemorrhage in case of placenta previa; Perinatal morbidity and mortality in such a case; Maternal morbidity and mortality in such a case; Followup case; For discussion student should know about accidental abruption placenta and other causes of bleeding.

Pregnancy with previous cesarian section - Detailed history – regarding previous cesarean section, its indication, time of cesarean (electric or emergency), post operative complications if any. History of present pregnancy; Examination – P/A - Scar tenderness, Presentation and position of fetus; Investigations – Routine USG for Scar thickness; Decision making for trial of vaginal delivery or repeat cesarean section; Care during trial for vaginal delivery – Prerequisites, Contraindications, Guidelines for management of trial, When to cutshort thetrial, Exploration of scar after delivery; Decision for repeat cesarean –Timing of CS if electric, Care during cesarean, Various problems one can encounter in case of repeat cesarean at the time of operation and how to tackle them for eg. Difficulty in opening of abdomen, adherent bladder or injury to bladder etc.; Post operative care; Various indications for cesarean section –absolute, relative; Types of cesarean sections; Morbidity and mortality associated with cesarean section; Tibial ligation with cesarean section.

Heart disease in pregnancy- Physiological changes in cardiovascular system in pregnancy; Various types of cardiac diseases in pregnancy; Classification of heart diseases in pregnancy; History in detail; Clinical examination to put into type of functional impairement according to New York Heart Association; Investigations – Routine – Haemogram/ Urine examination for bacteria, ECG, Echo cardiography, Obstetric USG; Management during pregnancy especially –prevention of anemia, Prophylaxis for infection, frequency of antenatal visists and 28 riequisite check up (wt. BP), Advice on activity, diet, weight gain andmedication, When to admit, Complications during pregnancy, Place and time of cardiac surgery during pregnancy, Place of therapeutic MTP in case of heart disease; Mode of delivery - Vaginal delivery - Care during 1st stage, Care during 2nd stage, Care during 3rd stage, Place of cesarean; Puerperal care; Contreceptive advice; Perinatal and maternal morbidity and mortality.

Gynaec & Obstetric Viva

Obsteterics – Pelvis, Foetal Head, Doll, Episiotomy set, Koekrforceps, National Health Schems, Population Policy, Mortality Rate- Mortality / infant, Goals;Students should know –Normal labour, All presentation – Vertex, Occipito-posterior, Face, Brow, Breech, Oblique & transvers; All minor obstetrics Op. - Suction Evacualtion, ARH, Episiotomy, Stiching of tears - Parival/ Carvial/ Vaginal, Induction of labour – Medical/ Surgical, Ventose, Forceps, Caesarian Section, Manual Removal of Placenta, Tubal ligation; Management of case of - septic shock, Haemorrhagic shock, Congestive Cardiac failure, Ac. Endocarditis; Laproscopy – Diagnostic, Lap. Tubal ligation, Medicolegal aspect, Consumer protection Act Imp. Of consent and counseling

Family planning- Condom; I.U.C.D. – Cu-T, Cu-7, Cu-350, Multi load; Lippes loop; Oral pills - MalaD, Mala N, Saheli, Lynoral; Injection Depoprovera; Today; Cervical Cap; Laproscopic rings 9laproscopii set);Students should know - Various types of contraceptive device – indication/ contraindication; MTP – Act, Procedure, Complication, Contraceptive quidence; Faliure rates of various contraceptives – Sterlisation- Female- Tubal ligation, Prmsoy's/ Laps, Couple protection rate, Fertility Rate- Total population- Vasectomy, Contraceptive prevalence, Failure rates of various methods & legal implications

Gynaec- Specimen - Fibroid uterus, Cancer cervix, Vascular Mole, Ectopic pregancny, Adenomyosis, Cervical fibroid, Cancer Endometorium, Ovarian cyst

X-ray – Hysterosalpingography, Breech presentation, Twins/ Multiple pregnancy, Anencephaly, Hydrocephaly, Conjoined Twins; U/S - Vascular mole

Students should know - Minor operation – Dilatation & curretlage, E.B., Cervical Dilatation, Cervical Biopsy – Type, Fraction curettage, Pap smear/ Colposcopy, Vulvar biopsy, Wet smear; Major operations - Various types abdominal incision, opening & closing of abdomen wound, Types of hysterectomy – Indications, Route; Ovarian Cystectomy; Oviriotomy; Salpigo-opherectomy; Salpingotomy – Partial/ total; Operation for - Cervical Malignancy, Uterine Malignancy Ovarian Malignancy, Vulval Malignancy ; Chemotherapy in gynaec malignancy

Bad obstetrical history

History- Age of the patient at conception, education status of husband and wife; Husband's occupation; Last Menstrual period; Period of gestation

Presenting complaints- Bleeding per vaginum; Gestational age at the time of its occurrence; Amount of bleed, (fresh , old, clloted blood); Any passage of clots, products of conception or grape like vesicles pain abdomen; Nature of pain and duration; History of any intercourse or trauma; Syncopial attacks; Duration and recurrence of bleeding; History of nausea and vomiting or its sudden cessation; H/o of fever with rash or genital ulcers; H/o urinary frequency, urgency or dysuria; H/o UPT done or documentation of USG examination and appearance of cardiac activity.

Eig2nd trimester pregnancy - Period of gestation; H/o quickening; H/o pelvic pressure; H/o bleeding P/V or leaking P/v; H/o recurrent lower abdominal pain; History suggestive & thyroid disorder (constipation, w ht gain, heat and cold intolerance); History S/o diabetes mellitus (polyuria, polyphagia, polydypsia); History S/o APLA (arterial or venous thrombosis, swelling of limbs, transient ischaemic attack); H/o S/o cardiac disease (breathlessness, palpitation, chest pain, swelling of feet)

Abnormal uterine bleeding

History **-** Age; obstetric history pertaining to abortions, ectopic, gestational trophoblastic disease, last child birth, any complication causing endometritis; MENSTRUAL HISTORY – excessive bleeding, content (clots or POCs), amount, cydical / acyclical, post – coital or intermenstrual; H/O D&C or instrumentation; H/O Hormonal Contraception or irregular use of hormonal preparations, IUCDs; H/O sexually transmitted diseases; H/O bleeding menstrual history **-** Age of menarche; Regularity of the cycles; History of prolonged cycles, duration of flow, amount of bleeding; Passage of clots; History s/o dysmenorrheal

Obstetric history **-** Age at marriage and first conception; History regarding early fetal losses; Gestational age at fetal loss; History of blighted ovum (UPT + USG documentation); Pattern of expulsion; Any pain abdomen and bleeding PV; Any operative intervention (D & C procedure)

2ND trimester foetal losses **-** Any history of bleeding or leaking per vaginum and pain abdomen; Duration of expulsion of foetus; Any bleeding P/v; Pattern of fetal (with increasing or decreasing gestational age of the losses); Any congenital abnormality in the fetus; H/o IUGR; Early PIH or feature suggestive of arteriovenous thrombosis

Past history of thyroid disorder **-** Obesity, DM, HT(hypertension), Hyperprolactinemia (galactorrhoea), Cardiac, renal or hematological illness, H/o any surgery or trauma to the cervix, History s/o leiomyoma, adenomyosis PCOD (treatment taken for infertility), History s/o autoimmune disease (fever, joint and muscular pain)

*Family history***-** DM, Hypertension, Recurrent pregnancy loss, Congenital and chromosomal defects in the babies

*Personal history of***-** Cigarette smoking, Drug abuse, Alcohol intake

Examination specific to recurrent pregnancy loss **-** Height, Weight, BMI, Hirsutism, Acanthosis, Body habitus, Pulse, BP, Thyroid examination, Breast examination (for sign of pregnancy)

Per abdominal (2nd trimester pregnancy) **-** Uterine fundal height, Estimated baby weight, Presentation, Liquor volume, Fetal heart sound

Per speculum **-** Cervix (blue or not), Length of the cervix, Internal os open or closed, Bleeding or product of conception coming through os

Per vaginum **-** os closed or open, funneling of the cervix, cervical length, Uterine size corresponding to POG or not, Adnexal mass and tenderness.
Investigations **-** Blood group+ Rh typing, Complete blood cont (haemoglobin with platelet count), Blood sugar (fasting and postprandial), VDRL, TORCH, Lateral vaginal wall smear (to rule out progesterone deficiency)
*USG examination for***-** Foetal biometry, GCA, Liquor volume, Placental localization, Congenital malformation of uterus, associated fibroid, Cervical length/dilataion of funneling cervix coning of the membrane, Parental peripheral karyotyping, ACA, LAC, ANA levels, Fetal karyotyping by CVS (if indicated)
Management **-** Management of etiology found if any; Management of current pregnancy - Complete bed rest during the critical period, Diet, Folic acid supplementation, Luteal phase support, Heg injection (1500-5000iu biweekly following LH surge for 8 weeks), Inj. Proulation depot 500mg i/m weekly till 14 weeks, Natural micronized progesterone by oral and vaginal route, 100-200mg BD till 14 weeks, Symptomatic treatment of nausea and vomiting, Reassurance to the patient; Treatment of etiological factors - Syphilis, toxoplasmosis, thyroid disorder, disbetes mellitus, Immunotherapy (APLA syndrome) from 5 weeks of pregnancy, Low dose aspirin – 80mg daily, Heparin 10000 IU s/c BD, Prednisolone 40mg to 80mg OD, Other trials regarding immunomodulation are going on, Limitation of physical activity during critical peiod

Preterm labour
History **-** Age of the patient at the time of conception; Education status and occupation of the patient and socioeconomic status; Period of gestation
Presenting complaints **-** H/o pain abdomen, nature and severity of pain, duraiotn; Whether pain associated with hardening of uterus; H/o any urinary frequency or urgency, burning; H/o fever; H/o thyroid disorder/PIH/heart disease
Treatment history **-** 1st trimester - exact last menstrual period and UPT, USG confirmation for exact dating, H/o fever/rash/drug exposure; 2nd trimester - H/o of quickening, H/o of leading p/v discharge p/v, bleeding p/v or pain abdomen, H/o early onset PIH
Menstrual history **-** Menarche, LMP, Regularity of cycles
Obstetrics history-Duration of marriage; H/o any treatement taken for infertility; H/o previous preterm birth; H/o any D & C done in past; H/o any surgery done on the cervix or any encirclage procedure done in the past; Sequence of events of pregnancy loss – leaking P/v, Bleeding P/v, abdominal pain; Any significant postpartum events (H/o puerperal sepsis/fever or foul smelling discharge); Past history of DM/jaundice, asthma, thyroid disease any surgery done on cervix
Personal history - Addiction to drugs, alcohol, cigarette smoking, Socioeconomic status, Nutritional adequacy; Family History - H/o DES exposure in the mother of the patient, H/o DM/HT/thyroid disorder, H/o recurrent preterm births in family, H/o of GCA in the baby in the family.
Examination **-** Height, Weight, BMI, Pulse, BP, Thyroid examination, Breast (pregnancy change of breast), Per Abdomen - Uterine fundal height, whether corresponding to gestational age or not, Any uterine activity (whether any contractions present or not), Presentation of the fetus, Estimated baby weight, Liquor volume, Fetal heart rate auscultation, Per speculum - Any discharge P/V, Leaking and bleeding P/V, Cervical os dilation/funneling of the cervix, Per vaginum, Cervical length, dilatation of os, funneling of the cervix
Investigations **-** BG, RH typing, Complete blood count (TLC, DLC), Sugar – fasting and post prandial, Urine – routine and microscopy + culture, HVS-C/S or cervical smear – culture and sensitivity; USG - Fetal maturity, viability and fetal growth biometric parameters, To rule out GCA, Liquor volume, Placental localization and grading, Uterine congenital anomaly, Cervical parameters- length, dilatation or funneling of the cervix
Management of preterm labour- Management of patient with warning symptom of preterm labour, Management of patients in established preterm labour
Management of patient with warning symptoms- Bed rest, Reassurance, Treatment of any risk factors for preterm labour – treatment of vaginal infection, urinary tract infection, fever or medical disorder

like PIH, renal disease; Sedation may be considered; Glucorticoids may be considered; Role of tocolysis in inhibiting preterm labour – only when uterine contraction are present and associated cervical dilation is there, under 2 conditions-For the effect of – gluocorticoids can set in 24 hours, tocolysis can be given, For the transfer to a tertiary care unit, where good nursery care facilities are available; Identification of patient in whom the preterm labour need not be inhibited - Advanced preterm labour, Choriomnionitis, Fetal congeniatl anomaly, Severe fetal growth restriction, Disease detrimental to meternal health - Uncontrolled DM, Severe preeclampsia, eclampsia.
Management of established preterm labour - Glucorticoids (inj. Betamethasone 12mg I/M repeat 12mg after 24 hrs.); Tocolysis may be considered if no contraindication to its use are present and till the time glucorticoids action sets in - Beta-adrenrgic agents, Isoxurpine, Ritodrine, Terbutaline, Calcium channel blockers, Magnesium sulpate, Indomethacin; Nursery and pediatrician to be informed; Intensive labour monitoring and aseptic precautions at the time of delivery.

Abnormal uterine bleeding
History-age;Obstetric history pertaining to abortions, ectopic, gestational trophoblastic disease, last child birth, any complication causing endometritis;Menstrual history – excessive bleeding, content (clots or POCs), amount, cydical / acyclical, post – coital or intermenstrual; H/O D&C or instrumentation; H/O Hormonal Contraception or irregular use of hormonal preparations, IUCDs; H/O sexually transmitted diseases;disorders, aspirin or any anticoagulant use; H/O abnormal weight gain, cold or heat intolerance, S&S of diabetes, hirsutism (excessive hair growth), glactorrhoea
*Examination***-** Appearance, obese/excessively cachetic, weight, height; Enlargement of thyroid, petechiae or ecchymosis on skin ; Discharge from breasts, hair distribution; Secondary sexual characterstics; Any mass or lump in abdomen, hepatosplenomegaly; Any abnormal discharge, growth or ulcer in per speculum examination
Investigations-CBC with differential count and platelet count; Coagulation profile, APTT where required; Blood Grouping and RH typing; Urine albumin, sugar and microscopy; Blood sugar – fasting and psot-prandial; Liver function tests; Pap's smear; Endomaterial sampling or Hysteroscopy (particularly in > 40 year age group); Thyroid profile, prolocation (wherever indicated); Ultrasonography – transvaginal
*Differential diagnosis***-**Pregnancy complications; Anovulation associated; Submucous fibroids; Endometrial Polyps; Medication related; Endometrial hyperplasia; Endometrial cancer; Infection associated; Coagulation abnormalities; Mullerian abnormalities; IUD complications; Adenomyosis
*Management***-**Hormonal – Combined pills, progestogens, danazol, GnRH Agonists, Mirena; Tranexamic acid, mefenamic acid; D & C, Hysteroscopy; Conservative Surgery – TCRE, Endometrial ablation by Various methods (LASER, thermal, radiofrequency); Hysteroscopy – abdominal, non-descent vaginal, laparoscopic; Treatment of basic pathology.

Pruritus vulvae
*History***-**Mode of onset, its duration, intensity, relation to menstruation; Associated gynecological symptoms like type of white discharge- Mucoid, Frothy,curdy white, etc.; Associated abnormal vaginal bleeding; Any urinary or fecal incontinence, history of passing worms in stool; Any vulval sore or growth; Contraceptive history- use of any spermicides, condoms, etc.; Generalized skin diseases-Eczema, psoriasis, etc.; Any allergy to medications, soaps, deodorants; History of diabetes mellitus; History of dyspareunia
Clinical examination- Look for evidence of dermatological, metabolic or deficiency diseases; Condition of vulva, vagina, urethra, anus; Type of discharge
Investigations **-**Haemogram; Urine for sugar to exclude glycosuria; Urine for C/S to exclude urinary tract infection; Blood sugar to exclude Diabetes Mellitus; Wet saline vaginal smear to look for Trichomonas vaginalis; KOH smear to diagnose Moniliasis; Stools for ova and cyst; Biopsy of vulval lesion-to confirm the diagnosis and to exclude carcinoma in-situ.
*Diifferential diagnosis***-** Eczema, psoriasis; Various types of vulval dystrophies; Contact Dermatitis; Allergic dermatitis; Diabetic Vulvitis; Monilial & Trichomonas vaginitis and vulvitis

Management- Treatment of specific cause if found; Control of diabetes; Control of Trichomonal infection and monilial infection with specific drugs. Treatment of husband also to prevent re-infection; If Vulval dystrophy is localizied, local excision/partial vulvectomy; If extensive and atypical epithelial activity present advise simple vulvectomy and follow up; Apart from specific measures general measures like cleanliness, antihistamines, sedatives and hydrocortisone ointments prevent itching and provides adequate night sleep. Avoidance of allergens and antigens.

Fibroid utreus

History-*age* (peak between 35-45 years);patients are usually nulliparous or history of long period of secondary infertility; menstrual irregularities- menorrhagia or hypermenorrhoea commonest. Metrorrhagia can be due to ulceration of submucous fibroid or fibroid polyp; symptoms of anaemia like shortness of breath, palpitation may be present; Heaviness or lower abdominal lump. Progress of the Lump(static, slowly increasing, rapidly increasing or initially slowly increasing but later rapidly.); pain is not a common symptom.It may be due to cervical dilation induced by submucosal myoma, torsion of a pedunculated myoma or red degenration associated with pregnancy. Congestive variety of dysmenorrhoea may be present due to associated endometriosis or adenomyosis; Pressure symptoms- More common with cervical fibroids. When the uterine size is increased, pressure is exerted on adjacent organs. Urinary manifestations include frequency, urinary incontinence or difficulty in urination. Posterior wall myoma exerting pressure on the rectosigmoid may cause constipation or tenesmus. Rectal pressure can occur due to incarceration of the myoma in the culde-sac; infertility *Clinical examination*-General examination-Usually various degrees of pallor; Abdominal examination-Inspections- number, site, size, shape, surface, margin, Consistency, mobility, temperature, tenderness; Fibroid uterus is usually firm to hard in consistency, maybe cystic in cystic degeneration. Margins are well-defined except the lower pole which cannot be reached. Surface nodular or uniformly enlarged. Mobile from side to side but not from above downwards; Rising test to confirm intra-abdominal or parietal swelling. Knee-elbow position and examination of the swelling again to decide whether the swelling is intraperitoneal or retroperitoneal.Hernial sites should be examined.Percussion-fibroid is dull (shifting dullness and fluid thrill should be examined in any lump abdomen); Pelvic examination-Uterus is not felt separated from the swelling. No groove is felt between uterus and mass. Cervix moves with the movement of the tumor.
Investigations-To confirm diagnosis; Preoperative assessment-USG-TVS is the gold standard. Mapping accuracy of USG decreases in larger uteri containing multiple fibroids. Smaller fibroid and subserosal fibroid may not be detected by TVS. Addition of sonohysterography to TVS generally improves the sensitivity in detecting submucosal fibroids. USG assessment should include examination of ovaries, presence peritoneal fluid also; CT scan has no role in work up of fibroid; MRI-It is the most accurate imaging modality for the diagnosis, mapping and characterization of myoma. MRI finding can suggest further investigation in the line of leiomyosarcoma; Diagnostic laparoscopy-when uterus is less than 12 weeks gestation and associated with pelvic pain and infertility, it may reveal coincidental endometriosis, pelvic adhensions or tubal pathology. It differentiated between ovarian neoplasm and pedunculated fibroid if it is unclear on the basis of clinical findings or USG; Measurement of depth of uterine cavity with a sound can give an idea whether the mass is uterine or ovarian; Complete hemogram, sugar, urea, creatinine, x-ray chest, ECG, urine and stool examinations; Urinary tract study if urinary symptoms present - cystoscopy may be required to rule out intrinsic bladder lesion, IVU to demonstrate ureteral deviation, compression or dilatation in case of laterally located fibroid, broad ligament fibroid or cervical fibroid; PAP smear to rule out associated cervical malignancy; Endometrial sampling to rule out endometrial hyperplasia or carcinoma which may coexist.
Differential diagnosis-Full Bladder- Strictly suprapubic, cystic or tense cystic, ill defined margin, tendency of urge on pressure, disappear after catheterization; Pregnancy- Recent history, amenorrhoea, soft elastic feelings, Braxton- Hick's contraction, External ballotment present, USG confirmed; Ovarian tumor- Slow growing, no menstrual abnormality, cystic, tense cystic or sometimes solid in feel, well defined margin including lower one, usually freely mobile, uterus is separated from

lump, USG confirmed; Adenomyosis- Along with menorrhagia, congestive Dysmenorrhoea is most prominent symptoms, lump is rarely more than 14-16weeks pregnant uterus, uniform swelling , USG confirm; Encysted peritonitis- History of Koch's infection , usually associated with amenorrhoea, cystic lump with ill defined margin, uterus is separated, USG; Dysfunctional uterine bleeding-uterine lump usually is not more than 12-14 weeks pregnant uterus. USG to rule out fibroid.; Pelvic inflammatory disease- Women may present with pelvic pain, tender pelvic mass, abnormal uterine bleeding. USG and laparoscopy to confirm; Endometriosis- Presented with chronic pelvic pain or congestive dysmenorrhoea, endometriotic cyst of ovaries may mimic myomas. Laparoscopy is the gold standard of diagnosis.

Treatment-No treatment is required for asymptomatic fibroids which are less than 12 weaks size after clinical examination or found incidentally in USG; Non- surgical treatment;_improvement of general condition and anemia; 2.Estrogen and progesterone therapy in combination or progresteronealone, ofteb are the first line of treatment for patient with uterine myoma and abnormal uterine bleeding. It brings endometrial atrophy and stabilization but as it is a temporary measure and they have not been shown to reduce myoma size; 3. GnRH agonist causes amenorrhea and rapid decline of uterine and myoma size. Mean uterine size decreases 30-64% after3-6 months of therapy. Maximum response occurs by 3 month. After stoppage of treatment menses return in 4-6 weeks and myoma and uterine size return to pretreatment level in 3-4 months; Indications of GnRH agonist—Severe pre-operative anemia, minimizing the need for transfusion,Preoperative shrinkage of large and awkwardly situated fibroid to reduce blood loss and tissue injury, Preoperative shrinkage also facilitate vaginal hysterectomy, hysteroscopic resection or ablation and laparoscopic destruction possible, Preservation of fertility in women with large leiomyoma before attempting conception or preoperative treatment before myomectomy, Treatment of women approaching menopause to avoid surgery; Medical contraindications to surgery.It should be commenced in the midluteal phase for most rapid pituitary gonadotrophin down regulation no sexual intercourse that month to rule out pregnancy. Estrogen progesterone add-back can be given back to prevent osteoporosis, but it reduces the effectiveness of GnRH agonist. Use of GnRH agonist in the preoperative phase may make surgical plane less distinct and myomectomy more difficult. A small fibroid becomes a tiny one and may be missed during operation There is delay in diagnosis of leiomyosarcoma.GnRH antagonist directly inhibits action of GnRh on the pituitary resulting in immediate gonadal suppression and avoidance of stimulatory phase seen with agonist; Aromatase inhibitors directly inhibit ovarian estrogen synthesis and serum estrogen levels decrease after one day of treatment.Myomas are known to over express aromatase, an estrogen synthetase, which suggests that myoma may produce their own estrogen and aromatase inhibitors can target the local source of estrogen and thus decrease myomavolume. Mifepristone- antiprogestin, a progresterone receptor modulator with primarily antagonist action. There are reports of shrinkage of uterine fibroids in response to continuous therapy with mifepristone. A daily doses of 5-50 mg of mifepristone were used in various studies for a periods of 3-6months, highlights that mifepristone therapy effectively regress myoma size while maintaining stable bone density; however endometrial hyperplasia may limits the long term therapy. Danazole and gestrinone have been studied for treatment uterine myoma. Both the drugs lead to significant reduction in myoma volume. Androgen side effects of these drugs are there most prominent disadvantages. Levonorgesteral containing intrauterine device has proven, effective, reversible treatment for menorrhagia by inducing endometrial atrophy. Enlarged uterus with distorted uterine cavity or a submucosal fibroid is a contraindication for LNG-IUS use.

Surgical management-Indications for surgery- Abnormal uterine bleeding with resultant anaemia unresponsive to hormonal management; Chronic pain with severe dismenorrhea, dyspareunia, lower abdominal pressure or pain; Acute pain as in torsion of a peduculated leiomyoma or prolapsing submucosal fibroids; Urinary symptoms or signs such as hydronephrosis after complete evaluation; Infertility with leiomyoma as the only abnormal finding; Markedly enlarged uterine size with compression symptoms or discomfort; Rapid enlargement of the uterus during premenopausal years or any increase ; in uterine size in postmenopausal women because of the inability to exclude uterine sarcoma.

Types of surgery-Hysterectomy-Routes depend upon the size of uterus, the situation of fibroids, history of previous surgical procedures. Hysterectomy can be done by abdominal route, vaginal route or laparoscopic assisted vaginal route. Shrinkage of myoma with GnRH can be done prior to LAVH or vaginal hysterectomy for large fibroids will be rendered easier by prior enucleation of fibroid. Ureters maybe vulnerable during removal of a broad ligament fibroid or lateral fibroid and their pathways almost be identified. Incase of large cervical fibroid hemisectionof the uterus followed by enucleation of the fibroid is an alternative approach in order to gain access to uterine artery and cervix;

Myomectomy-Indications- Patient is in the reproductive period, desirous of having a baby. In case of recurrent pregnancy loss due to fibroid.Infertility evaluation, hysteroscopy to detect a fibroid encroaching uterine cavity or tubal block and endometrial carcinoma should be done before myomectomy. Myomectomy can be done by hysteroscopy incase of submucosal fibroid, or laparotomy.Patients should be counseled about the recurrence of myoma which is as high as 50% and about one third patient requires repeat surgery. She may requires hysterectomy at any time during operation. It is better to remove as many fibroids as possible through a single incision to prevent postoperative adhesion. Incision on the posterior wall be better avoided to prevent adenexal adhesion. Prevention of blood loss during myomectomy can be done by Bonney's clamp and Rubin's tourniquet. Now these procedures are replaced by use of vaso-occlusive injection Vasopressin used either intramurally, or perivascularly in the broad ligament at the junction of anastomosis of ovarian and uterine blood supply. Myoma greater than 4-5 cm and not having greater than 50% protrusion into the uterine cavity are not good candidates for hyteroscopic removal.

Uterine artery embolization (UAE)-It is a minimally invasive technique for any symptomatic fibroid except pedunculated subserous fibroid. Other contradiction is pelvic inflammation. The disadvantage is no pathological confirmation of uterine fibroid is obtained and uterine sarcoma could be missed. UAE may be an ideal conservative treatment for leiomyoma to eliminate symptoms, reduce the size of myoma, limit recurrence of future myoma and preserve fertility. But UAE is not without complications like pain, nausea and vomiting and general malasia, (Post embolization syndrome). Expulsion or sloughing f fibroid occur in few case particularly common in submucous fibroid.Myolysis-It can be done laparoscopically by electrosurgical heat, laser energy or cryotherapy. This procedure termed myolysis is accomplished by destruction of tumour tissue or obliteration of vascular supply of fibroid.

Other points to remember-Ascites may present along with myoma. Myoma may attach to omentum and get blood supply from omental vessels (floating myoma) , If there is torsion and obstruction of these vessels there may be transduction of fluid and development of ascites; Polycythemia may be present due to elevated level of erythropoietin; Benign metastasizing uterine myoma characterizied by myoma like lesion in lung; Intravenous leiomyomatosis is a hormonally responsive disease that manisfests as vermiform extensions originating in the uterus that can extend as far as the heart; Shrinkage of myoma with GnRH against can be done before. Performing vaginal hysterectomy or LAVH; It is difficult to assess pelvis during hysterectomy for large fibroid. It can be made easy by prior enucleating the fibroid; Ureter may be vulnerable during operation of broad ligament fibroid, so their pathways may be identified; There is no contraindication to oral contraceptive in women known to have fibroids provided uterine volume is monitored and there is no increase in size of fibroids. In women with out fibroid long term oral contraceptives may have a protective against tumor development; Pregnancy related manifestation-Increase in size, mostly in the early pregnancy, myoma less than in 5 cm diameter increased in size and larger myomas decrease in size during the second trimester; Red degeneration; Miscarriage, PROM, placental abruption , malpresentation, prolong labor, increase operative interference.

Heart disease complications in pregnancy

History- H/O -Rheumatic Fever in Childhood ; H/O Heart Disease - Surgery done for Heart Disease; Previous Pregnancy and outcome ; H/O taking anticoagulants and antibiotics; H/O Congenital Heart disease in her mother

Clinical Examination **-**On Inspection- Dyspnoea, Orthopnoea, Neck vein pulsations, Clubbing of fingers, Cynosis Edema legs; Symptoms **-** Progressive dyspnoea, Orthopnoea, Nocturnal cough, Hemoptysis, Syncope, Chest pain, Palpitations; Signs - Murmur, S2 Split&Other Sounds, Dilated Neck Veins, Arrythmias, Clubbing, Cyanosis, Hyper dynamic circulation of pregnancy causes alteration in CVS that mimics heart disease, Premature atrial &Ventricular ectopic beats, of peripheral pulse volume, d pulsation of neck vein, forceful apex beat due to 1"d CO suggest cardiomegaly. which occurs in normal pregnancy should be differentiated from those occuring in heart disease; Dyspnoea- Most frequent symptom; Can occur in normal pregnancy; Differentiated from heart disease by history whether present prior to pregnancy or not Chest pain **-** . Occurs due to ischemic heart disease,AS,HOCM; Pulmonary hypertension MI-Arrythmias, VF-sudden death;Hemoptysis **-** Seen in MS; Orthopnoea & nocturnal **-**COUGH are other presentations of heart disease; Palpitations **-** Occurs commonly in pregnancy; . No correlation between dysrrythmia & symptoms; A palpitation often short&does not necessarily requires treatment; Seen in HOCM; Syncope **-** Occurs in ll Trimester, Peripheral resistance.,Cardiac output, Occurs in AS, HOCM, TOF, Eisenmingers syndrome; Palpitations **-** Occurs commonly in pregnancy; No correlation between dysrrythmia & symptoms; . Palpitations often short&does not necessarily requires treatment ; Seen in HOCM; syncope **-** Occurs in ill Trimester; Peripheral resistance cardiac output~s; Occurs in AS. HOCM, TOF, Eisenmingers syndrome; edema **-** Non specific for heart disease; Occurs due to - Pressure of gravid uterus; in intravascular colloid osmotic pressure exacerbated injudicious use of crystalloid solutions; dilated veins ,apex beat **-** Significant If heart beat is shifted >2cm outside the MCL ca ;

NYHA classification

CLASS - I - No limitation of physical activities,Ordinary physical activities does not cause palpitations, dyspnoea or anginal pain.

CLASS - ll-Slight limitation of physical activity. Pt is Comfortable at rest.

CLASS - ill - Marked limitation of physical activity ,less than ordinary activity causes fati gue, pal pitati on, dyspnoea&angina

CLASS - IV - Inability to cany out any physical activity with out discomfort,syrnptoms of cardiac insufficiency or of anginal syndrome may be present even at rest. Any physical activity causes discomfort

*Murmurs***-**Can occur in normal pregnancy ; Early to midsystolic murmur which is soft & of grade I & II is normal; ESM heard over Rt & Lt II ICS about 2cm from steral edge systolic or continuous modified by pressure of stethoscope occurs due to ot flow in mammary vessels; Venous hum ,Murmurs should be attached clinical significance if systolic murmur is loud or long like; PSM(VSD,MR, TR); Late SM (MR,MVP); ESM louder than Gr3/6(AS)or that varying with respiration (PS);. Or associated with other abnormalities like ejection click (pSorAS) . Munnur associated with thrill, Diastolic murmur MURMUR -Mid to late systolic murmur associated with or without mid systolic click - MVP Postural maneuvers are used to aid in *As.;* ctivities that Lt ventricular volume degree of MVP;

Arrhythmias-Sinus bradycardia, tachycardia, atrial&ventIjcular premature contractions,SVT occurs In pregnancy ; VT ,multiform premature ventricular ; complexes are less common in normal pregnancy. AF suggests heart disease; ASD can present with supra ventricular arrhythmias;Cyanosis,clubbing,pulse deficit peripheral signs of endocarditic are other signs

Investigations- Chest X-Ray - Radiation to abdomen &fetus is minimal; Use of pelvic & abdominal shield is mandatory . CXR is not specific ; Pulmonary venous congestion,cardiomegaly, valve calcifications can be made out ECG - ST depression,flattening ofT wave in It side precordicalleads occurs in 14o/00fnorrnal pregnancy ;T wave inversion,Q wave in L III in seen in healthy pregnant women; ECG is used in diagnosing dysrrythmias &rare causes of cardiomyopathy than in demonstrating structural abnormalities of heart; Ischemic changes in ECG is important if associated with biochemical changes; Blood Chemistry -. Haemoglobin , TC, ESR,& LDH occurs even in normal pregnancy; serum glutamic acid transarninases & CPKwith appropriate clinical setting is used in diagnosing MI; During puerperium interpretation of enzyme should be cautious because they are liberated in tissue destruction of involuting uterus; MB isoenzyme of CPK is specific to cardiac

muscle;ECHO -. Used to demonstrate structural changes in heart ; Bacterial vegetations. prosthetic valwlar dysfunction can be made out better by Transesophageal echo.done by attaching the transducer to a flexible endoscope. In Marfan' s echo showing significant aortic root dilatation.pregnancy should be discouraged; DOPPLER -Used to study flow patterns. But prevalence of regurgitant flow across Rt sided valve is significantly greater in nonnal pregnant woman than in non pregnant state. Hence it should be considered;USG - Important in establishing gestational age early ; Early obstetrical scan is important for confirmation of due date; Diagnosing multiple pregnancies or fetal anomalies; USG done in 16-20 wks is used to study fetal heart & major vessels; Those at risk for intrauterine growth retardation occurring in Eisenminger's should be subjected to serial USG every 2-4 wks in III Trimester
Differential Diagnosis **-** Normal Physiological changes during pregnancy; Anaemia ; Hypoproteinemia
Non - Surgical Management **-** Class I & II ,Admission 2 weeks earlier to EDD; Class Ill&IV -Immediate Hospitalization, AN Care; Prevent Anemia, Prevent Infections - RI . UTI. Limitation of activities; JP Care - Hospitalization in tertiary care centre
Stage 1 - Propped up position, oxygen, sedation.
Stage 2 - Prophylactic outlet forceps or vacuum ; Withheld prophylactic methergin
Stage 3- Ifbleeding, oxytocin to be used.
Stage 4- If patient is in failure, oxygen, Digoxin, IV , Oral Digoxin, Diuretics - Injection - Furesimid 40 to 80 mg IV. Sedation - Injection - Morphia 15 mg 1M
Antibiotics -Injection - Ambicillin 1.5 mg and Inj Garamycin 60 mg eighth hourly. - SHE Prophylaxis;Arrhythmia - Quinidine, Electro version.
Surgical Management **-**Because of the assumed risk to both the mother and fetus, surgery has been confined to the person who is refractory to medical management with either intractable heart failure (or) intolerable symptoms.
Indication for pregnancy termination -Absolute -PHT; Dilated Cardiomyopathy; Marfans with carillo vascular involvement -Pulmonary A VF; Relative- Parous women with ill,IV cardiac lesions; **I** & II patients with history of cardiac failure in early pregnancy in between pregnancy -Done < 12 weeks by suction evacuation or dilatation and evacuation -Dilated cardiomyopathy
Induction of Labour can be done with adequate monitoring; Use of anticoagulants - Inj Heparin (fractionated) during 6 to 12 weeks and after 36 weeks and Puerperium; Tab. Warfarin from 12 to 36 weeks; LSCS for Obstetric indication, coarctation of Aorta, pulmonary hyper tension -Contraception - Barrier method, Tubectomy or Vasectomy for Husband. -Breast feeding - Not contra indicated. Peripartum Cardio Myopathies Heart Transplant

Obesity and hirsutism

History- Age of Onset at menarche suggests PCOS, idiopathic hirsutism or 21-hydroxylase deficiency. Onset distinct from menarche suggests tumor; Progression - Rapid progression of hirsutism or other symptoms suggests tumor; Menstrual disorder; Drug intake like Phenytoin, Minoxidil, Cyclosporin
*Clinical findings and lab tests***-**Hirsutism, Acne - PCOS or idiopathic hirsutism; Virilisation - Ovarian or adrenal tumor, hyperthecosis ; Evidence of ovulation; Ovarian ultrasound - Confirm polycystic ovaries, rule out ovarian tumor; Total and free Testosterone and LH levels ; Evidence of insulin resistance; 17 OH progesterone levels; Lipid profile; Prolactin, TSH; CT of adrenal glands - If ovarian ultrasound is normal and tumor is suspected; Dexamethasone suppression test
*Differential Diagnosis***-**PCOS, Ovarian hyperthecosis, Idiopathic hirsutism , Adrenal/Ovarian steroidogenic enzyme deficiency , Late onset 21 Hydroxylase deficiency, 3 â Hydroxy steroid dehydrogenase or 11 Hydroxy deficiency, Adrenal/ovarian tumors, Drug induced
Non Surgical Treatment **-**Aim of medical therapy - suppress androgen production, block androgen receptors or decrease the conversion of testosterone to dihydrotestosterone by inhibition of the enzyme 5a-reductase ; Oral Contraceptive Pills; Gonadotropin Releasing Hormone Agonists; Glucocorticoids - Hirsutism secondary to late-onset congenital adrenal hyperplasia or functional adrenal hyperandrogenism; Androgen Receptor Antagonists - cyproterone acetate ; Spironolactone - OCP is often used in conjunction; Flutarnide is a nonsteroidal anti androgen - works at the androgen receptor ; 5a-reductase Inhibitors – Finasteride; Insulin Sensitizing Agents – Metformin, Eflornithine

HCL(difluriomethyl ornithine); Non-Medical Therapy - different means of mechanical hair removal have been employed in combination with medical therapy- Shaving, Epilation, Chemical depilatories Electrolysis Lasers ; Photodynamic therapy - aminolevulinic acid

*Treatment of Insulin Resistance and Other Metabolic Abnormalities***-** Weight loss; Insulin-sensitizing agent - Thiazolodinediones . rosiglitazone and pioglitazone, Metformin ; OCPs and anti antiandrogens - spironolactone flutamide and GnRH agonist

Treatment of Anovulation **-** Clomiphene citrate; Aduvant therapy like Metformin, Dexamethasone and dopamine agonists Gonadotropins

Surgical Treatment **-** Bilateral ovarian wedge resection; Laparoscopic drilling; Surgical removal of tumors

Post dated pregnancy

History- In order to determine whether the size of the baby is correct for the gestation, the gestational age needs to be accurately known; Prediction of the expected date of confinement; Patient's statement - last normal menstrual period (LNMP or LMP); Naegles rule- first add 7 days to 1st day of LMP, then go forward 9 months; Cycles- regular preferably 28 days in length / irregular / prolonged / short - if the interval of cycle is longer, add extra days; if the interval is shorter, subtract lesser days ; Accurate maternal recall of the date of LMP; Period occurred at the expected time and was of the usual duration; There had been no vaginal bleeding in the immediate subsequent month; That the woman had not been using hormonal methods of contraception within the last 3 months of LMP; Clomiphene induced- ovulation generally occurs 48 - 72 hrs after the last dose of CC; Date of insemination- fertilization usually occurs within 24 hrs after insemination; add 265 days from day insemination (fertilization - delivery interval = 266 days); Date of embryo replacement- 2 cell- 30 hrs, 4 cell- 48 hrs; add 264 days to day of transfer ; Day of fruitful coitus- client may remember in relation with some social event . Date of quickening- Primi - add 22 wks, Multi - add -4 wks

Previous records- he required weeks are to be added to make it 40-Size of uterus prior to 12 wks generally corresponds with period of amenorrhoea; 8 wks - cricket ball size, 12 wks - fetal head (term size) i.e. fills pelvis completely; Palpation of fetal parts (external ballotment) - earliest 20th wk; FHS - stethoscope 18 to 20th wk, Doptone – lO th wk; Preg color test - 5th to 6th wk ; USG - gestation sac with yolk sac- 5th wk, gestation ring with internal echoes- 6th wk; CRL in centimeters + 6.5 = approximate wks of gestation

Clinical examination-Fundal height -Principle- the height of uterus depends on the duration of pregnancy; tell the client what you are going to do; ask for a chaperone if you belong to male gender; ensure that the client has voided; client lies on her back with legs semi-flexed; always keep eye contact; correct dextro-rotation; the position of the fundus is palpated using the fITst Leopold grip; at 16 wks- 1 -2 fmgerbreadths above the symphysis; at 20 wks- 2 fingerbreadths below the umbilicus; at 24 wks- at the umbilicus; at 28 wks- 2 - 3 fingerbreadths above the umbilicus; at 32 wks- midway between the umbilicus and the sternal xiphoid; at 36 wks- at the costal margin; at 40 wks- 1 - 2 fingerbreadths below the costal margin; early in the 3Th wk the fundus descends and regains the same height as at 32 wks. One can reckon on an expected date of delivery 3 - 4 wks after this time

Symphisio-fundal height (SFH)-serial SFH measurement has a sensitivity of 76% at specificity 79%;with a positive predictive value (PPV) of 36% (Reference- Lindhard trial) ; determine fundus first ;the distance from the upper margin of the symphysis and the middle of the upper edge of the fundus is measured in centimeters; keep the tape blinded (i.e. non-marked side up) othef"!-ise biasness; SFH is a conditional measurement of the size of the uterus and an indirect measure of the size of the gestation. It corresponds in a characteristic manner with the wks of pregnancy. As a rule of thumb, from the 16th to 35th wk the SFH in cm will be the same figure as the duration in wks; Used alone SFH identifies 28% of antenatal population as being risk of small for gestational age (SGA), and would detect 78% SGA babies.

Abdominal circumference- A simple though *very inaccurate,* method of estimation of gestational age. May be used in in conjunction with SFH; Measured at the level of umbilicus; Measured at each

antenatal visit from 20th wk - only regular consecutive measurements are of value; The average normal girth at term is 100 - 105cm. Much lower values (less by 6cm)may indicate growth retardation or incorrect dating. Larger values (greater than 6cm) may occur with large baby, multifetal gestation, hydramnios, and obesity.
Leopold's palpation-Four consecutive steps-First Leopold - height of fundus, two hands; Second Leopold – back, two hands;Third Leopold - two hand pelvic grip (reference- Leopold's grips in Williams Obstetrics 2002); Fourth Leopold - one hand pelvic grip; Amount of liquor - cystic, versus uterus full of fetus ; Feel of head - hard
Vaginal examination-Bishop's score - value span- 0 – 13; bishop's score increased by one point for each previous vaginal delivery, at the initiation of labour, in pre-eclampsia ; Bishop's score decreased by one point in post dated pregnancy, premature rupture of membranes, primigravidae (reference- Nisswander . Cervical scoring systems in Danforths Obstetrics, 2002)
Investigations- Ultrasonography- Confinnation of date- BPD,FL; Growth - HC / AC; Liquor – AFI; Fetal well-being - biophysical score, components, fetal tone(1imb movement), trunk movement, breathing movement, FHR, amniotic fluid volume (at least one pocket> lcm); Cardiotocography - 2 x wkly / daily ; Pre-induction scoring- cervical scoring systems
Differential diagnosis **-** Mistaken dates; IUGR - SGA, Preterm; Oligohydramnios

Management **-** Placental ageing leads to impaired gas transfer - fetal hypoxia & distress. Hypoxic state is aggravated in elderely clients, hypertensive disorders, bleeding during pregnancy.During labour, there is increased incidence of asphyxia and intracranial damage due to Aggravation of pre-existing hypoxia, Increased incidence of difficult labour due to big size baby, non moulding of head, Shoulder dystocia, Increased incidence of cord compression. After birth, greater chance of meconium aspiration syndrome and atelectasis, hypoglycaemia, Polycythaemia,Perinatal mortality increased x 1.5 at 42 wks, x 2 at 43 wks, x 4 at 44 wks Therefore IOL at 10 days past expected date; 25 - 30% clients start on their own by 10 days

Non-surgical management **-** Cervical ripening methods and agents- Prostaglandin E2 gel, PGE2 pessary, misoprostol 25~g, foley's catheter; Stripping of membranes digitally

Surgical management **-** ARM; Planned Caesarean section - post-caesarean pregnancy, malpresentation, elderely primi;

Any other Accoucher should be conversant with management of shoulder dystocia.

Jaundice in pregnancy
History-_Period of ammenorrhoea; Duration of Jaundice; Pruritis/Itching; Abdominal pain/distension; Pale/clay wloured stools; History of Blood transfusion, I/V drugs, fluids, hepatoxtoxic drug, needle prick; Nausea(mild/moderate/severe); History of malaise, anorexia, weight loss; H/o fever, rash; Any History suggestive of — coagnlopathy(bleeding diathesis),encephalopathy(altered sensorium, conrulsions),nephropathy(renal failure); Family H/o Jaundice; H/o Jaundice in previous preganancies
Clinical examintation- Weight of the patient; Icterus-eyes, palm, oral mucosa & skin; Signs of pruritis(scratch marks); Bleeding spots(if any); Oedema; Sensorium; Temperature; Blood Pressure; Liver- Surface, span, margin, consistency, tenderness; Spleen; Ascites; Any evidence of dhydration; Obstertrical examination- Height of uterus Presentation Fetal Heart; Any evidence of IUGR< Fetal distress; Patients of Pre-eclampsia with HELLP syndicrome are vry ill with hypertension, protecnuria, hemolysis,Jaundice, hemoglobinopathies, thrombocytopenia with activation of coagulation cascade
Investigation **-** Complete hemogram including ESR; Serum Bilirubin – Total Direct, Indirect; SGOT, SGPT; Serum alhaline phosphalase; Urine-Bile salts, bile pcpinen ts, urobilingogen, ketone; Serum proteins- Total Differential(A/G Ratio); PTI, PTTK; Viral markers.hepatitis serology; Blood sugar; Serum creatinine; Ultrasound of abdomen for - Liver echotexture, Splenoportal axis, Collaterals in portal hypertension, Ascites, Space occupying lesion, Biliary stone, Fetus & placenta, IUGR/IUD
Differential diagnosis- Causes not specific to pregnancy - Viral hepatitis, Cholelithiasis, Autoimmune hepatitis, Neoplasia, Gilberts syndrome

Specific to pregnancy- Hyperemesis gravidarum (Ist trimester), Severe pre-eclampsia/HELLP (2nd half of pregnancy), Acute fatty liver of pregnancy (2nd half of pregnancy), Intrahepatic cholestasis (2nd half of pregnancy)

Management **-**Management will be as per aetiological cause
Hepatitis- Antenatal(Prenatal) - Supportive care, Dietary advice, Monitor liver function tests, Testing for HbsA*f* , Asess fetal growth & health, Vigilance for uterine activity, Infection control measures
Labor/delivery and postnatal - Infection control measure; Watch for PPH; In HbsA*f* positive cases, newborn should receive passive(HBIG) and active immunization;
Intrahepatic-Cholestasis of Pregnancy-Prenatal- Local antipruritic measures; Consider cholestyramine, ursodeoxycholic acid; Vit K supplement; Monitor fetal well being; Consider elective deliver; Biliary tract ultrasonography
Labor/delivery-Anticipate preterm delivery; Increased risk of PPH
Postnatal— Monitor biochemical resolution; Vit K supplement for baby
"Acute Fatty Liver of Pregnancy" Prenatal- Estabilish diagnosis, resuscilate; Intensive care; Supportive therapy; Plan delivery; Labor/Delivery - Maternal resuscitation by correction of – Hypoglycemia, Fluid imabalance, Coagulopathy; Treatment of liver failure; Intensive fetal monitoring; Urgent delivery when maternal condition is stabilized, vaginal delivery preferable for mother; Meticulous hemostasis, including adequate wound drainage;Postnatal-Continue intensive care management; Watch for postpartum wound hematoma formation and spses, PPH; Supportive contraceptive measure
"Severe Pre eclampsea-Prenatal & Labor - Control of hypertension; Control coagulation disturbance; Consider anticonvulasant prophylaxis; Close fetal monitoring; Watch for fall in hemoglobing from hemolysis; Fluid and electrolyte management; Initiate deliver process;Postnatal - Anticipate delayed postnatal recovery; Continue monitoring plalelets and renal function; Monitor for hemolysis; Control severe hypertension
"Hyperemesis Gravidarum"-Intravenous fluid and electrolyte therapy; Diet-Discuss with dietitian; Antremetic regimen; Nutrient and Vitamin supplement; Antressphageal reflerex measures; Psychological and social support; Steroids-controversial
*Surgical Management***-**No role of surgery

Urinary fistula
History-Details of Incontinence - true or false, associated presence of urinary stream Previous obstretric history including-details of labour; mode of delivery; interval between delivery and leakage Previous gynae operative history Type of surgery performed; Interval between surgery and leakage
Clinical examination-Inspection of Vulva with special reference to excoriation; *PIS* to demonstrate site and size of Fistula; *PN* to confirm above and to comment on extent of scaring *I* fibrosis; Further information on *PN* to assess regarding uterine and adnexal pathology ; Urinary examination for microscopy and culture Methylene blue test Cystoscopy; IVP in selected cases; Examination under anaesthesia especially if clinical examination is not infonnative enough; Must distinguish between ureterovaginal and vesicovaginal fistulae
*Management***-**Non - Surgical management - Role of prolonged catheterization Pre Operative management - Elimination of urinary tract infection; Treatment of external excoriations; Surgical management -Various techniques of repair; Special reference to postoperative management - Prophylactic measures for both obstetric and gynae urinary fistulae

Faecal fistulae
Histroy- Similar to urinary fistulae. Special importance to intermittent incontinence with reference to consistency of faecal *matter*
*Vulval examination***-** PIS and P/V to determine details of fistula and especially to assess distance of fistula from anal verge and introitus; Tone of external anal sphincter;
*Investigation***s –** Routine; Special - proctoscopy *I* sigmoidoscopy ; Examination under anesthesia if required - Fistulogram

Differential diagnosis- Distinguish between RVF and 3rd degree perineal tear
Management-- Local hygiene; Various kinds of repair and their approaches; Prophylactic measures during episiotomy, Perineorrhaphy suturing and hysterectomy.

ORTHOPEDICS

Management of neglected fracture neck of femur

History

Age of the patient- it concerns the line of management. Treatment line may be different in children, young
adults and elderly people.

Sex - Child bearing age in females has a relationship with treatment

Profession- employed, unemployed, farmer / labourer, office going person / quality of life. This too concerns the line of treatment.

Mode of trauma - Was the Trauma high velocity? or trivial, as is generally seen in osteoporotic elderly patients. High energy trauma has worse prognosis for fracture healing.

Duration of injury-neglected fractures of longer duration may have more shortening and may require traction to achieve limb length equalization.

Ability - o stand and bear weight after the injury (it is very important point- Studentsgenerally forget to note in examination)

Neurological status- Was patient able to move his toes just after the injury?

History of pain - preceding the fracture ? Presence of hip sympomatology before injury could be crucial, because presence of degenerative disease or associated pathology has implication on treatment options. Like wise metabolic diseases causing a traumatic fracture may have implications on fracture healing.

Treatment - The details of treatment taken by the patient should be enquired into. History of massage or tight bandages by local quacks/osteopaths should always be asked and their complications be looked into if so. A number of femoral neck fractures are actually posterior dislocation of hip before they were managed by local osteopaths. Thus the patient may have fracture neck of femur secondary to posterior dislocation of hip consequent upon manipulation done by osteopath.

Clinical examination -Examination in supine; and standing particularly if patient comes walking should be known to the candidate. Heal of the ground, knee flexion, exaggerated lumber lordosis require discussion.

Attitude **-** Student must express about the attitude of the limb, shortening on inspection, level of anterior superior iliac spines, level of two medial malleoli, wasting of muscles, and extent of external rotation. A mention of attitude of hip in adduction to should be looked into.

Palpation **-** Local temperature in scarpas triangle,, Tenderness in scarpas triangle, trochanteric region, gluteal region. Femoral pulsations, their volume on two sides. Resistance in femoral triangle in an attempt to know the status of femoral head. Plapation of greater trochanter is equally important to rule out associated pathology if any besides fracture itself. Gluteal region too msut be palpated for any kind of abnormal mass. A mention of shortening above trochanter by palpatory Briant's method is equally important before it is finally ascertained on drawing Briant's triangle and performing true and apparent measurements. The candidate must look for status of inguinal lymph nodes in local examination as well.

Measurements - Both true and apparent measurements must be discussed.Briants,Triangle, shortening above / below trichanter should be discussed.

Deformity - Methods to detect and measure a deformity like flexion deformity of the hip (fixed or otherwise) must be lookedinto in a neglected fracture neck of femur. Methods to measure the deformities should be discussed with the candidate.

Movements - The range of movements both active and passive, pain free or painful should be discussed.

Straight leg

Raising test**-**Both active and passive.

*Telescopic test***-** How to perform and its importance?
Gait of the patient should be looked into both from front and . If the patient caries stick/lathi in hand, this too should be discussed. How to look for Trenddenienberg gait?
Differential diagnosis - Is the fracture extracapsular or intracapsular? Posterior dislocation of the hip? Infective pathology like tuberculosis of hip?
Investigations- X-Rays **-** Both antero-posterior view and Frog leg view or lateral view of the hip must be taken. On antero-posterior radiograph the candidate should comment upon shenton's line, prominence of lesser trochanter in terms of external rotation and proximal migration, prominence of greater trochanter vis a vis flexion deformity of hip, proximal displacement of distal fragment, type of fracture is terms of Pauwel's and Garden's classification. Status of head of femur in terms of its viability and joint line must be discussed besides Singh's index to grade osteoporosis. Rotation of head of femur like varus position, absorption of neck of femur and saggital relationship of proximal and distal fragments should be looked into.
MRI **-** To discuss viability of head femur.
Computed Axial Tomography- It is effective in evaluating non union.
Treatment - Patients age, morphological features of the fracture, femoral head viability, extent of femoral neck resorption, shortening bone quality and status of auricular cartilage are some important factors that need to be discussed.
The candidate must know biological procedures which could be taken up in younger patients.Ostocotmy of hip eg. Mcmurray and Valgus osteotomies and their pricinple.Fibular grafting and osteosynthesis.Excision arthroplasty in ederly poor females unfit for major surgery, fusion of hip in poor young labourers with femoral heads; and hemi & total hip arthroplasty in elderly patients with poor bone stock or avasular necrosis of femoral head should be discussed vis a vis priciples of treatment.

PIVD
History -Importance of age- Most disc prolapses occur between the age 20 to 40 when the nucleus is juicy and fleshy; Relatively sudden onset of pain with radiation to one or both lower limbs; Relief of pain on rest and increase of pain on activity; Definite increase of pain on coughing and sneezing which raises epidural venous pressure; History of remissions and exacerbations should be elicited; History of weakness and numbness in the lower limbs;Hesitancy of micturition and retention indicating impending cauda equinqa syndrome; History of neurogemic claudication; past history of similar episodes of pain;Profession e.g. heavy manual labourer.
Clinical examination **-**Spinal asymmetry. Compare the levels of shoulders, rib cage on both sides; Ability to differentiate between spinal list and scoliosis. Relation of the type of list to the anatomical location of the prolapse whether shoulder or axillary type; Spinal mobility. Schober's test. If selective restriction of flexion and lateral flexion with normal extension could be demonstrated, it is highly suggestive of a prolapse; Muscular spasm; Spinal tenderness.
Root tension signs viz. SLR- Lasegue's sign Braggad's sign;Crossed / Contra lateral SLR; Bowstring sign;Reverse SLR or Femoral stretch sign for higher lumbar prolapses; False positive or false negative SLR; Neurological examination of lower limbs; Muscular wasting indicating root involvement; EHL weakness in L5 roots involvement; Quadriceps wasting in L3 root involvement; Gluteal wasting / weakness in S;Check dermatomal sensory loss and detailed dermatomal mapping should be demonstrated; Perianal / saddle anesthesia to be particularly looked for in cases of Impending cauda equina compression.
Inappropriate signs to check malingering -Flip Test; Burn's Test
Always check SI joints;Peripheral pulse to rule out vascular occlusive disorders
Investigations - Plain X-ray L-Spine; Does not prove the diagnosis of a disc prolapse but may be helpful in ruling out certain conditions which can mimic a prolapse.
CT – Usually needs contrast, as CT is good to delineate bony structures rather than tissues.
MRI – gold standard but a strong clinical correlation is required to to diagnose. Up to 30% of normal individuals can have prolapses detected by MRI with no clinical relevance.

Differential diagnosis-Lumbosacral strains; Ankylosing spondylitis; Tumors like osteoid osteoma;Major vascular occlusions / thrombosis like Leriche Syndrome.
Non surgical management -It should be highlighted that approximately 90% of the prolapses respond to non operative treatment like -Bed rest with or without L-S traction; Anti – inflammatory drugs; Epidural steroids; Local heat.
Surgical management -Indications - Absolute(Acute cauda equine compression with bladder bowel involvement;Major neurological deficit like foot drop); Relative (Pain not responding after 6 weeks of non-operative treatment;chronic PIVD with debilitating attacks of pain many times during a year); Surgical procedures -Laminectomy;Hemilaminectomy; Fenestration; Microdiscectomy; Chemonucleolysis.

Lateral condyle fracture
*Surgical aspects***-**Elbow is a complex hinge joint. It has three different articulations namely humeroulnar joint where flexion and extension take place ; Humero radial joint a Pivot joint ;superior radio ulnar joint. Superior radio ulnar joint along with humeroulnar joint rotates the forearm internally and externally. It has two major muscle group origins namely common forearm flexors from medial epicondyle and common extensor origins from lateral epicondyle.
History **-**Presenting complaints-Pain in the elbow joint; Pain around elbow joint; Swelling; Deformity; Restriction of elbow movements; Loss of elbow movements (stiffness); Exaggerated/ abnormal movements; Locking of elbow.History of present illness**-**Mode of onset duration and progress;Pain-exact location, characters, radiation aggravating factors, relieving factors, relation with movement of elbow, night cries, exacerbation and remission.Swelling- Location, progressive regressive history of exacerbation, variation (more prominent, less prominent) on movement of the joint.Deformity-bony deformity or joint deformity (varus or valgus), undue bony prominences, variation (obliteration or appearance) of the deformity with movement of the elbow joint.Restriction of movements-Flexion or extension; Supination or pronation; Associated with spasm or crepitus or pain;Loss of movements-Total loss or flicker of movements associated with pain, spasm or crepitus; Locking of elbow-At what degree, whether associated with pain; Exaggerated/ abnormal movements-Hyper extension, valgus or varus;Movements Associated with creptus; History of trauma-Direct or indirect injury;H/o massage after the injury; H/o treatment by an osteopath.Treatment history-Details of the treatment take in respect of each complaint whether there was relief from the symptoms (partial or complete) or no improvement.

Clinical examination
Inspection-Attitude;Carrying angle measurement of angle;Deformity (flexion deformity, cubitus varus, cubitus valgus); Measurement of the angle of deformity; Swelling – position, number, extent, size, shape, surface, skin over the swelling, obliteration of hollow and depression around elbow, fullness of cubital fossa; Sinuses and scars, discharge from the sinus, bluish discolouration of the skin, engorged veins, visible pulsation
*Palpation***-**Local rise of temperatue;Local bony tenderness (anatomical location);Joint line tenderness;Tenderness over the swelling crepitations;Swelling consistency, fluctuation, boggy feeling;Sinus – mobility (fixity to under lying bone), any discharge on palpation, mouth of the sinus;Palpation of bony prominences;Tip of olecranon, lateral epicondyle, supracondylar ridges and posterior surface of humerus; Tip of medial epicondyle, supracondylarride, capitulum, radial head (rotating forearm); Three bony relations in flexion (90^0) a triangle and on extension, straight line between lateral epicondyle, medial epicondyle and tip of olecranon; Interacondylar distance altered in intercondylar fractures, condylar fractures and epicondylar fractures.
Movements-Flexion-extension;Whether restricted or exaggerated, associated with pain spasm or crepitus;Abnormal mobility (Whether joint is unstable) anterior or posterior block;Supination – pronation;Loss of functional ability;Estimation of muscle strength, instability of superior, Radio ulnar joint.

Measurements-Length of the arm and forearm (arm short in supracondylar fracture, forearm short in posterior dislocation of elbow);In old fractures of the lateral condyle or intercondylar fractures intercondylar distance is increased;Measurement of the triangle;Measurement of the carrying angle, varus or valgus deformity;Wasting of muscles.
Diagnostic points in nonunion of lateralcondyle of humerus-Patient is commonly a child; History of injury (commonly fall); Inadequate treatment; H/o massaging may be present;Swelling, more pronounced on the lateral condylar region;Local warmth may be present; Bony tenderness over lateral condyle;Bony thickening or loss of contour of lateral condylar ridge; Abnormal mobility of lateral condyle; Lateral instability may be present; Restriction of flexion or extension; Intercondylar distance is increased; Weakness of extensor group of muscles.
Investigations-X-ray, AP, lateral, with valgus angle with varus angle
Differential diagnosis-Intercondylar fracture- Common in adults with history of fall on point of Elbow, tenderness over intercondylar area and posterior part of humerus enlarged and thickened, intercondyle length increased. Movements of the elbow restricted and painful.
Management-Very small fragment conservative in a POP slab; Undisplaced – Plaster immobilization or percutaneous 'K' wire fixation; Displaced-open reduction and fixation with "K" wire or cancellous screw fixation engaging the medial condyle with or without bone grafting (precaution to be taken to maintain the soft tissue attachment)
Classification of lateral condyle fractures- **C**lassified not based on morphological trauma or by a mechanism of trauma, but by amount of displacement-Undisplaced (stable centrally) – Incomplete articular fractures; Displaced fractures – displacement of more than 2 mm in the central part – complete articular fractures.

SURGERY

Thyroid swelling

History - Duration and pattern of progress; Recent increase in size;Change in voice / difficulty in swallowing; H/O exposure to radiation; Goitrogenic drugs / Living in endemic area; History of features of hypo/hyperthyroidism

Clinical examination - Gen Physical exam – routine with emphasis on Pulse rate / Pedal oedema; Local (Neck) exam – should include; Thyroid exam – complete evaluation specially unilateral or bilateral , consistency, surface , mobility , position of trachea and retrosternal extension (by percussion); Evaluation of surrounding structures eg Trachea, Recurrent laryngeal nerve and Carotids; Neck nodes – complete evaluation; Systemic exam – includes evaluation of thyroid status through CVS, CNS and eye signs . Respiratory exam must also be done

Investigations-Ultrasound;Thyroid function tests (TSH, T3 , T4);FNAC;If further needed , Thyroid scan ; CT Neck for node evaluation , finding retrosternal extension;X-ray Chest

Differential diagnosis- For Diffuse thyroid enlargement (Physiological;Diffuse colloid; MNG (Endemic); Autoimmune Thyroiditis; Thyrotoxicosis – Primary & Secondary; Malignancy – Papillary , Follicular, MCT, Anaplastic & Lymphoma);STN (Dominant nodule of MNG; Cyst; Adenoma – Follicular & Toxic; Carcinoma; Focal Thyroiditis)

Non- surgical treatment- Thyroxine – for suppressing TSH in colloid adenomas (curative intent), After Surgery of malignancy for suppressing TSH in Papillary ca. , as replacement after Near Total thyroidectomy and to prevent recurrence after subtotal excision in MNG; Needle aspiration under USG guidance for all cyst; Anti – thyroid drugs and Propranolol for thyrotoxic; Lugol's / Collosal iodine for preparing thyrotoxic for surgery

Surgical management— Some kind of thyroid resection for all except Anaplastic and Lymphoma; Either Lobectomy or Subtotal thyroidectomy or Near Total or Total thyroidectomy; If malignancy – Modified (conservative) block dissection depending upon the Lymph node group involvement; Any other(External Beam Radiation for Anaplastic / Lymphoma;Radio-iodine for select group of Thyrotoxic cases)

Cervical lymphadenopathy

History-(Duration and the pattern of progress of swelling; Fever – details; Constitutional symptoms; H/O respiratory difficulty , dysphagia , any change in voice; Associated symptoms pertaining to Oral cavity/Abdomen/ Chest viz cough , abd. Lump., oral ulcer / salivation etc.; H/O testicular swelling , Parotid swelling)

Clinical examination- General physical exam – Besides routine specially look for jaundice, clubbing , pedal edema etc; Local (Neck) Exam – Complete evaluation of all groups of cervical nodes eg size, consistency, matting , mobility , fluctuations, any evidence of collar-stud abscess etc; If any associated thyroid swelling . it's detailed clinical evaluation; If any Parotid swelling , it's detailed evaluation; Exam of oral cavity (lip, tongue , cheek, floor of mouth , gums, palate and inner Waldeyer's ring) including oropharynx; Examination of Head & face for evidence of melanoma, Squamous cell carcinoma etc; Clinical evaluation of Ear , Nose and Throat; Systemic Examination should include – exam of abdomen for any malignancy, Examination of Testis , Chest for clinical evidence of ca. Lung and Breast ca. and P/R and /or P/V examination

Investigations - Pertaining to Lymph nodes(Hb, TLC, DLC , ESR; Chest X-ray; FNAC; Excision/ Incision biopsy depending upon site if lymphoma); If Primary is obvious-complete evaluation of primary by USG/CT/MRI etc & FNAC; If Primary is not obvious-Battery of investigations consisting of endoscopic evaluation of nasopharynx / laryngopharynx; Plain X-rays and radiocontrast studies; USG/CT/MRI; Upper / Lower GI endoscopy; Exfoliative cytology etc.

Differential Diagnosis - Reactive hyperplasia (to infection); Tuberculosis; Lymphoma; Secondaries form abdomen, Chest , Breast , Thyroid , oral cavity etc; Rare surgical causes to be enlisted in last (if at all)

Non- Surgical Treatment-Antibiotics for infections; Anti-tubercular treatment for tubercular nodes; Chemotherapy for appropriate set of cases of lymphoma / Secondaries.

Surgical Treatment - (Excision/Incision biopsy - has diagnostic role and not therapeutic); Antigravity aspiration for cold abscess; Excision for tubercular sinus; Block dissection / modified block dissection for secondaries depending upon primary and secondary itself. Any Other-Radiotherapy for selected set of cases of secondaries and lymphoma.

Salivary gland tumor

History -Duration and pattern of progress of swelling;Any recent increase in size , or ulceration; History of variation in size / pain with meals; History s/o facial nerve involvement eg food bolus collecting in cheek, inability to close eyes etc.; Inability to open mouth; Constitutional features.

Clinical examination- Gen Physical exam – all routine; Local exam (of Parotid/ submandibular)- Obliteration of fossa between angle of mandible and tip of mastoid s/o swelling being parotid; Raising of ear lobule – definite sign of parotid enlargement; Complete evaluation of swelling with regard to surface , consistency , mobility/fixity to massetter (parotid) to mandible (submandibular) and facial / hypoglossal nerve examination; Intra-oral examination (for lateral oropharyngeal wall and tonsillar bed (for deep lobe of parotid); Stenson's and Wharton's duct ; Bigital palpation to differentiate submandibular gld from node; Cervical lymph node evaluation);

Investigations - FNAC (Trucut contra-indicated); Incisional biopsy if ulcer present ; CT/MRI for assessment of extent and depth if malignant; Chest X-ray

Differential hiagnosis - Lymph node enlargement; Sialoadenitis;Soft tissue tumor.

Non- Surgical management-Virtually no role except broad spectrum antibiotics and anti inflammatory in acute sialoadenitis.

Surgical management- Stone removal and /or gland excision in Sialoadenitis depending on the presentation; For Benign – Excision of gland amounting to Superficial parotidectomy (in Parotid) , Submandibular excision (for submandibular) and complete excision of sublingual/minor salivary gland; For deep lobe involvement of parotid (benign) – Total conservative parotidectomy (preserve facial nerve); Radical Surgery for malignant lesions sacrificing nerves encountered along with gland ,facia and modified neck dissection; Any Other-Radiotherapy for Recurrent pleomorphic adenoma; Malignant tumor having extra-glandular disease, Perineural invasion, Regional metastasis , high grade malignancy etc.; Complications like Frey's syndrome must be known in details

Obstructive Jaundice

History-Name,Age, Sex, Occupation

Sequence of events preceding onset of jaundice & presence or absence of pain must be included.

*Chief complaints with duration***-** Pain right side upper abdomen / epigastrium; Yellowish discoloration of eye, skin, mucosa, nails; Loss of weight & poor appetite, nausea, anorexia; Itching of skin, swelling & heaviness upper part of abdomen; Fever chills / rigor; Colour of stools & urine

History of presenting illness- Pain(Onset, Nature, Radiation, Relation with food & posture); Jaundice(Relation with pain, Progressive / waxing waning)

Past history - Cholecystitis, cholecystectomy, pancreatitits, jaundice, blood transfusion.

Personal history - Alcohol, smoking, drug ingestion / allergy.

Family hisotry - Haemoglobinopathy like sickle cell, hereditary spherocytosis.

Clinical Examination- Nutrition status(wasting), anemia, jaundice & its depth, palpable neck, nodes, scratch marks; Pulse, BP; Heart, Lung, Edema legs

Per Abdomen – Inspections(Shape, movement with respiration, visible swelling moving with; Respiration or not)

Palpation / Percussion - Soft / rigid, tender / nontender; Presence of absence of ascies – methods to elicit shifting dullness/fluid thrill; Hepatosplenogmegally & its characteristics; Lump Intraabdominal / parietal / retroperitoneal; Site, Extent, Consistency; Murphy"s test, Moynihan"s test; Gall bladder palpable or not; Courvoisier's law & its exceptions.

Investigations - Urine for bile salts & bile pigments

LFT- Serum Bilirubin, ALP, SGOT, SFPT, PT & INR, AG Ratio; Normal Value, Significance of rise ALP; Liver dependent clotting factors; Reason of deranged PT in jaundice, how to correct it; When PT not corrected by vitamin K injection – reason.

Radio Imaging - What are imaging test done to arrive at diagnosis in chronology, screening tests; Benefits of MRCP over ERCP; Indication & limitations of USG abdomen, CECT, ERCP, MRCP.

Differential Diagnosis **-** How to differentiate surgical from non surgical jaundice; Common causes of obstructive jaundice in chronology.

Treatment- Cholelithiasis with CBD stone-Different options are Open choledochotomy & cholecystectomy; ERCP removal of stone followed by lap / opne cholecystectomy; Gold standard for gall stones; Steps of open cholecystectomy with DO's & DONOT's; Anatomy & anomalies of biliary tract; During Cholecystectomy what to do in case of Bile duct injury; Hepatic artery injury; Why do remove CBD stone before cholecystectomy.

CBE stone alone-Non Surgical treatment(How to prevent & treat cholangitis, Hepatorenal syndrome; Correct fluid & electrolyte imbalance; Correct cogulopathy & treat sepsis; When to stop conservative treatment & go for surgery.

Surgical treatment– Choledochotomy(Method & Interpretation of POCG; Absolute & relative indications of choledochotomy; Steps of choledochotomy; What to do if stone is impacted at lower end of CBD; Role of transduodenal sphincterotomy), Choledochoduodenostomy(Methods, indications & contraindications, pre-requisite; Why not preferred in young patients.)

ERCP removal & sphincterotomy-Suitable candidates; Technique & difficulties; Contradictions & complications.

Residual CBD Stone - With T tube(Burnhenn's technique; Different treatment options), Without T tube(Plan of action)

Carcinoma head of parncreas- History & Clinical Examination same; Triad of palpable gall bladder, weight loss & jaundice; Stool for occult blood; Benefits of CT over USG abdomen; How to confirm in periampullary carcinoma; Most useful investigation in suspected Ca head pancreas; Difference of pectability & operability

When curative / palliative; Criteria for declaring inoperable; Guidelines for preoperative confirmation of diagnosis, Gross? Frozen section?; Different palliative procedures & steps of roux en Y; Basic steps of Whipple's; Current data related morbidity, mortality & survival benefits of Whipple"s

For better performance

History & clinical examination - The student should know the relevance of all positive and negative history & be able to express only relevant things, When to ask leading questions.

Investigations - Gold standard for gall stone, CBD stone & Ca head of pancreas; Fallacies & short comings of different imaging investigations; Benefits of one over the other; Out of several investigations arts of ordering useful investigation only/

Treatment - Treatment plan (ideal in each case); Feasibility n give set up; Criteria of operability, respectability;Basic steps of operation should be very clear to satisfy perform the procedure; Care of T tube; T-tube cholangiogram & interpretation; Should have knowledge of complications & their management; Idea of difficult situation like Mirrizzi"s syndrome, anomalous hepatobiliary; tee Bismuth classification of bile duct injury & management; Post cholecystectomy syndrome; Waltman-walker syndrome

Hepato splenomegaly - History (prolonged / unexplained fever; early fatigability , easy bruisability , prolonged bleeding from minor wounds; Yellow discoloration of skin , sclera; Pain abdomen; Haemetemesis; Symptoms due to mass effect specially very large spleen; Lump abdomen.

Clinical examination - On general physical exam – look for altered mental status(end stage liver disease); Pallor, icterus, Ecchymotic patches, lymphadenopathy, pedal oedema, clubbing; Local exam (abdomen) – Dilated veins (with direction of flow in them); Contour of abdomen – ascitis (confirmed by Fluid thrill and shifting dullness) or abdominal lump (detailed evaluation of abdominal lump); Dipping method to be employed for palpation if huge ascitis; Differentiation between Spleen and left renal lump; And Liver and right renal lump; Liver span MUST be mentioned if liver is enlarged; Per Rectal examination (colour of fingerstall); Systemic exam – Respiratory; CVS and CNS.

Investigations - Complete hemogram; Peripheral blood film; Bone marrow aspiration/biopsy; Ascitic fluid examination – biochemical and cytology; Liver functions; X-ray Chest; ECG; USG evaluation of abdomen and if any equivocality , CECT; Upper GI endoscopy; Diagnostic laparoscopy.

Differential Diagnosis - (Single pathology which can explain both hepatosplenomegaly ; Otherwise incidental mild splenomegaly may be associated with even Secodaries liver); Malaria , Kala Azar; Lymphoma; Chronic hemolytic anemias; Portal Hypertension; Other rare causes at the end

Non- surgical treatment - Antimalarial for malaria. Pentamidine for kalaazar; Antitubercular treatment for TB; Supportive measures like blood transfusion etc.

Surgical Management - Splenectomy (in ITP, Heriditary spherocytosis); Splenectomy may be useful in hypersplenism; Splenectomy done sometimes for relieving pain and mass effect.; Splenectomy for staging in stage I & II; For Portal hypertension(Endoscopic therapy; Decompressive shunts and TIPS; Devascularisation procedures; Liver transpalnatation for end stage liver diasease; Any Other - Child classification slould be known.

Peripheral vascular disease – History(Pain – Intermittent claudication / Rest pain; Uni or bilateral; Mode of onset; Paraesthesia; Attacks of fainting, Black out , chest pain , weakness , abdominal pain , impotence etc.; Past history of IHD, Diabetes; Excessive smoking.

Clinical Examination - Gen physical exam – Routine with emphasis on peripheral pulses; Local exam; Gangrene – extent , line of demarcation and type (Dry or wet); Pre-gangrene; Evidence of chronic ischemia – thin shiny skin with scanty hair, atrophic limbs , Brttle nails; Any previous surgery scar viz Lum.sympecthectomy / Amputations; Burger's test , Capillary filling time; Raynaud's phenomena; Adson test, Elevated arm stress test , Allen test for Thoracic outlet syndrome; Systemic exam; CVS particularly.

Investigations - Complete blood evaluation; B Sugar/ S Cholesterol; Angiography; DSA; Doppler; Ultrasound; Duplex scan; Plethysmography.

Differential Diagnosis - Burger's Disease; Atherosclerosis; Raynaud's; Other rare cause at the end

Non – Surgical treatment - Apart from supportive treatment for pain; Burger's exercise; Stop smoking; Obesity reduction; Avoid injuries; Vasodilators; Chemical sympethectomy; Surgical Management; Lumbar sympecthectomy (Contraindicated in Intermittent caludication stage); Omento pexy (& other revasularisation procedures); Amputation; By pass grafts (for Atherosclerosis); Aorto-ileal endarterectomy; Baloon angioplasty;

Breast nodule

History-Patient's Name; Age; Duration of lump; H/o – Pain – Nature, Severity, whether associated with menstruation; Fever; Increase in size; Cough, expectoration; Whether received any treatment.
Family history – H/o – Such nodule in mother or sister, treatment received.
Past history – H/o – Previous operation.
Personal history – Menstrual history.
Clinical examination - *General examination - Pallor(Jaundice),* Local - Location – quadrant of the breast(Size; Overbuying skin; Prominent veins; Tethering; Peau de arange; Uleration; Nodule on the skin; Nipple(wheter retracted, Whether recent retraction, Areola); Surface of the lump(Smooth; Irregular; Local temp; Tenderness; Consistency); Fixity to - Overbuying skin and Underlying muscle; Lymph nodes – Ipsilateral - Axillary, Supraclaviulan, Contrateral - Axillary; Examination of other breast
Systemic examination- Chest; Abdomen – Hepatomegaly; Ascites.
Investigations – FNAC; Biopsy if FNAC in not conclusive; If Bemign nodule – Pre operative investigations; If Malignant(X Ray chest; Liver function tests;USG – abdomen – Liver; Ascites; Pelvis; ovaries; X-Ray of bones, if bony pains; Bone scan if suspicion of Secondaries; ER and PR receptor study
Pre operative investigations, if operable - Urine; Hb%; Blood sugar; ECG; Blood urea; Sr. Creatinine
Differential Diagnosis - Fibroadenoma; Carcinoma of Breast; Simple Cyst; Tuberculosis of breast; Chronic breast abscess;
Treatment - Depending upon the final diagnosis**.**

Inguino-scrotal swelling
History-Duration and pattern of progress of swelling; Fever , Chills (for filarial infection); H/O Trauma(If swelling present from birth; Any H/O irreducibility of swelling; Gepgraphical area of residence; Any H/O increased intra-abdominal pressure viz cough, constipation, urinary difficulty; Constitutional symptoms; Married and No of children (for varicocele)
Clinical Examination - General physical examination – Routine specially pedal edema/edema leg; Local examination - Routine exam of swelling laying emphasis on can or can't get above swelling; can testis be palpated separately; cough impulse; Transillumination; Fluctuations; Movement of swelling with traction on cord; Testicular sensation; Feeling of bag of worm; Ring Occlusion test etc; Systemic examination – to evaluate any sec . from testicular tumor into abdomen / renal lump on left side in varicocele cases; Abdominal muscle tone/ malgagnian bulges ; Neck for supraclavicular nodes.
Investigations - No specific diagnostic investigations for Hernia (for these case evaluation of Chest by Chest X-ray and USG prostate adv); No specific for hydrocele (but if non-transilluminant with positive fluctuations, then a formal ultrasound may be considered); For testicular swelling - FNAC; Tumor makers; Semen analysis for all cases of varicocele
Differential Diagnosis - Inguinal Hernia (Direct & Indirect); Femoral hernia; Hydrocele; Testicular tumors; Encysted hydrocele of cord; Epididymal cyst / Spermatocele; Lipoma of cord; Varicocele
Non-Surgical Management - Advisable for mild varicocele, mild hydrocele and aymptomatic bubonocele in form of scrotal support; Hetrazone therapy for filarial eradication
Surgical Management - Herniotomy, herniorrhaphy, hernioplasty depending on case; Hydrocele – must know all type of operations with relevant indications; Excision for epididymal cyst, spermatocele, lipoma of cord; Varicocele – Ligation (Open and laparoscopic); Testicular tumor – High inguinal orchidectomy followed by Chemotherapy /Radiotherapy/RPLND depending on stage and response; Any other -Follow up and tumor markers for testicular tumors

External Genitalia (Ulcer/nodule)
History-Mode of onset and progress pattern; H/O unprotected sexual activity; Bleeding , pain or discharge from the lesion; Associated diseases eg TB, peripheral neuritis etc; Religion).

Clinical examination- Gen physical exam – Apart from routine ,stigmata of syphilis , cachexia; Local examination – Exam of glans and penile shaft alongwith scrotum and inguinal area looking for; Ulcer / nodule – detailed exam; Exam of external meatus including any discharge;Inguinal adenopathy
Investigations - Complete hematology; Urine exam – complete and Culture & Sensitivity; Serology-VDRL, ELISA and CFT; Microscopic examination of discharge;;Swab culture; Wedge biopsy; FNAC of lymph nodes / nodule(s)
Differential Diagnosis - Trauma; Tumor; Syphilis; Chancroid; Lympho-granuloma Inguinale; Herpes; HIV; Carcinoma Penis; Condyloma; Premalignant lesions eg Leukoplakia, Paget's disease etc; Non-surgical management; For benign ulcers – antibiotic according to causative agent and supportive therapy
Surgical Management - Generally for – malignant disease and consist of partial or complete amputation ; Inguinal block dissection depending upon requirement
Radiotherapy - For malignant cases (Proper indication and ways to give radiotherapy must be known)

Soft tissue swellings/ tumors

History-Duration and pattern of progress of swelling eg if history of sudden increase recently or became more painful; Associated fever; Painful or pain free; Associated edema / motor dysfunction / sensory loss; Any ulceration of swelling
Clinical examination - General physical exam – all routine; Local exam – Complete evaluation of swelling specially anatomical plane of swelling , mobility , consistency, evidence of involvement of deeper structures viz muscles , nerves or vessels or even bone; Relevant pulsations; Movement of joint (if relevant to location of swelling); Multiplicity of swellings over body; Systemic exam – all routine
Investigations - Apart from routine; Ultrasonic evaluation of swelling; If deeper infiltration , CT/MRI; Chest X-ray for sarcomas for knowing secondaries; FNAC / Trucut needle biopsy
Differential diagnosis - Sebaceous cyst, Lipoma, Neurofibroma; Ganglion; Parasitic cyst; Papilloma; Hamartomas; Fibrous histiocytomas; Fibrosarcoma; Other rare causes may be mentioned in last
Non- Surgical management - By and large no role; Inj Hyaluronidase for Ganglion if small and simple
Surgical management - Enucleation/ Excision for swellings like lipoma, Seb cyst etc; Wide excision for locally recurrent tumors viz histiocytoma , hamartomas etc; Three dimensional excision / disarticulation for sarcomas, Any other - Nil

Soft tissue tumors of trunk

History-Swelling – onset and progress pattern; Any h/o recent rapid increase in size or pain; Previous h/o surgery for same site swelling; Symptoms pertaining to local structures involvement ; Constitutional symptoms
Clinical examination - General physical examination – Routine; Local examination - Venous prominences; Visible pulsations; Complete examination of swelling specially to know clinically the anatomical plane of origin , consistency, fixity/mobility, pulsatility, surface and margins; Complete examination of affected surrounding structures viz peripheral nerves / vessels / joint movements if relevant to that particular case-Bruit if any audible; Detailed evaluation of regional lymphadenopathy
Investigations - Plain radiography; Chest X-ray for evidence of metastasis; Ultrasonic assessment , if relevant to the site - CT/MRI evaluation for extent and depth / involvement of surrounding structures or relations with vascular & neural structures; FNAC; Core needle biopsy
Differential Diagnosis - Benign viz Lipoma , Solitary neurofibroma , Nerve sheath tumor; Desmoids; Hamartomas; Fibrous histiocytoma; Dermatofibroma protuberns; Sarcoma like Liposarco. Fibrosarco. etc; Other rare causes at the end
Non- Surgical Management - No primary role; Chemotherapy for malignant tumors mostly as adjuvant or sometimes as neo-adjuvant
Surgical Management - Wide excision is the mainstay of therapy and that may sometimes involve removal of muscles from origin to insertion; Followed by primary closure / grafting / use of flaps for closure depending upon individual case, Any other – Radiotherapy as adjuvant

Oral ulcer / growth

History-Pain – local or referred; Excessive salivation; Foul smell; Difficulty in protruding tongue / opening mouth; Alteration of voice; H/O smoking , Tobacco and alcohol; Spicy food consumption; Betalnut chewing; Dental plate / Sharp tooth

Clinical Examination - General physical exam – routine; Local – complete evaluation of Lip, Cheek, Gums, Palate and floor of mouth; Tongue for size ,colour, oral cracks , fissure , ulcer , mobility and induration; Angle of mouth- Fauces / Tonsils / Post third of tongue; Dental plate/Sharp edge of tooth; (A ggod bidigital palpation); Complete examination of all cervical group of nodes; Systemic exam for Tuberculosis & Syphlis (Although rare)

Investigations - Wedge biopsy; Exam of oropharynx; Chest X-ray; FNAC of Lymph node(s)

Differential Diagnosis - For growth – Squamous cell carcinoma; Tumor from minor salivary gland; For Ulcer – Trauma; Malignancy; Other rare causes at the end

Non-Surgical management – Chemotherapy; Radiotherapy

Surgical management - Wide excision; Wide excision with or without bone removal (Composite resection) with reconstruction; With or without modified Radical neck dissection; Any Other - Nil

Generalised lymphadenopathy

History-Primary focus of the node & drainage; Loss of appetite, weight and fever; Pressure effects-Local; Infiltration- Neuro vascular; Personal History - Tobacco, smoking, alcohol, eating habits

Clinical examination - General Exam - Nutritional Status; Performance status in malignancy. Local exam- Examination from behind, front & sides; Sequence of nodal examination; All nodes are deep to deep fascia; Group of nodes involved and its character ; Involvement of adjacent structures(Nerves – Cranial, Cervical Sympathetic; Vascular) ; Other-Trachea, Esophagus(Drainage area for focal site; Examination of oral cavity, ENT, Salivary glands; Cervical spine and thyroid if need be) 3).Others-Examination of other lymph nodes groups(generalized); Breast – if need be; Abdomen for spleen, liver, nodes, lump, free fluid PV & PR for pelvic deposits & nodes.; Respiratory system for Tb, secondaries, mediastinum

Differential diagnosis- Pathological causes of nodal swelling – Infective - Especially tuberculosis; Malignancy - Primary ; Secondary Swelling other than the nodes (if need be) - Carotid body tumor; Branchial cyst; Neurofibroma; Lipoma – subfascial; Schwanoma; Salivary gland swelling

Investigations-Routine baseline blood investigations; For tuberculous nodes (Chest X-ray; ; USG abdomen; FNAC; Biopsy), For lymphomas(Chest x-ray and CT chest; USG abdomen and CT abdomen ; Biopsy; BM Biopsy for stage IIA onwards, For secondaries with unknown primary(Pan endoscopy to visualize upper aerodigestive tracts and biopsy if lesion is visualized.Colonoscopy and biopsy if lesion found for lower neck (Lt) & Supraclavicular (Lt) nodes – if need be.; If lesion not found – FNAC of the node -Positive for squamous cell ca (SCC). Further CT of head & Neck for evaluation; FNAC positive for adeno carcinoma(CT Head & Neck for thyroid and salivary glands; Chest for bronchus; Abdomen & Pelvis for GI tract), FNAC Negative for malignancy, do biopsy of the node, Secondaries with known primary(Biopsy of focal lesion (Primary); CT head and neck; CXR (Ct scan chest-SOS)

Management - Non surgical (Acute lymphadenitis; appropriate antibiotics & drainage SOS; Tb adenitis –ATT & Drainage SOS; Lymphoma-Stage la, IIa- Radiotherapy (RT); Secondaries with unknown primary; Stage. N1, N2a, 2b – curative comprehensive RT; Stage. N2C and N3 – SCC – Palliative RT; Adeno Ca. Palliative Chemo; Secondaries with known primary-Early disease -Comprehensive Curative RT to Both local & Nodal disease; Advanced disease-Palliative RT/Chemo 2). Surgical Neck secondaries with known primary; Early diseae – aggressive surgery for local & node; RND (equally effective is RT)- (Node with unknown (occult) primary; N1, N2, a, b – RND (Consider post op RT)

Others- Anatomy of neck- triangles; Oral cavity; Drainage sites; Tuberculosis- a) Cold abscess stages (Pathology); b) Microbiology (Ziehl Neelsons); c) Collar stud abscess (Tb & Pyogenic); Lymphoma - Clinical staging (Ann Arbor);Principles in cervical node biopsy- Anaesthesia; Incision; Node - Incision Biopsy; Excision Biopsy; Imprint cytology

Neck Mass

History-Age Group, When examining a patient with a neck mass the first consideration should be the patients age group pediatric (upto 15 yrs), young adult (16 to 45 yrs) or old adult (more than 40 yrs). Within each group, the incidence of congenital, inflammatory and neoplastic diseases fit into one of these three categories. Pediatric patient generally exhibits inflammatory neck masses more frequently than congenital ones and developmental more than neoplastic masses. This incidence is similar to that found in younger adults. In contrast, the first consideration in older adults should always be neoplasia with a smaller emphasis on inflammatory masses and even less emphasis on congenital masses.; Location of Mass - The next consideration should be the location of the neck mass. This is particularly important in the differentiation of congenital and development masses because they usually occur in consistent locations. The location of a mass is both diagnostically and prognostically significant. The spread of head and neck carcinoma is similar to inflammatory diseases, generally following an orderly lymphatic spread. The appearance and location of a metastatic neck mass may be the key to identifying the primary tumour or source of infection. As age group and location, duration, consistency, associated complaints, progressive status of neck mass, all these add to reach on provisional diagnosis.

Clinical examination-The most important diagnostic step is the physical examination of head and neck. Visualization and palpation are the most important components of the clinical evaluation. There help determine the location of mass according to anatomic lymphatic drainage area, the size of lesions and its relationship (fixation or displacement) to surrounding structures, the consistency of mass, presume of any pulsations or thrills. The physician should perform an indirect mirror of flexible endoscopics examination of all mucosal surface of the upper aerodigestive tract. These areas should also be palpated even when no lesion can be seen specifically the primarily site for lymphatic drainage to the location area of the mass is question. Often, even the most thorough physical examination only gives a general impression of the derivation of mass – vascular, salivary, nodal; inflammatory, congenital or neoplastic and not a firm diagnosis. At this point a battery of test are available for help.

Investigation- Ultrasonography- To differentiate solid from cystic masses, especially useful in congenital and development cysts useful non-invasive technique for vascular lesions; Needle Biopsy- Gold standard in diagnosis of a neck mass; use small gauge needle; obtain flow cytometry of lymphoid population; Endoscopy and Biopsy- To identify primary tumor as source of metastatic node, use in all patients suspected of having neck neoplasia.

Open Biopsy- Use only after workup is complete and if diagnosis is not evident; specimen for histologic frozen section, be prepared to do simultaneous neck dissection. *CT & MRI*- Single most informative test; differentiates cyst from solid lesions; locates mass within or outside the gland or within a nodal chain mucosal disease enhancement, provides anatomic relationships. *Arteriography*-For vascular lesions and tumor fixed to the carotid artery. *Sialography*- To diagnosis diffuse sialoadenopathies or to locate mass within or outside a salivery gland. *Radionucleotide scanning*- Obtain in lesions of ant. neck compartment, helpful in thyroid lesions and in localizing a lesion to be within a salivary gland, PET scan may be helpful in differentiating tumor from post irradiation changes, and identifying distant metastasis. *X-Ray, Plain*- Rarely of help in differentiating neck masses. *Culture and Sensitivites*- Inflammatory tissue at open biopsy.*Skin Tests*- Used when chronic and granulomatous inflammatory lesion is suspected. *Antibiotic Course*- Clinical test for suspected inflammatory bacterial lymaphadenopathy, must pursue workup if unsoloved after course of antibiotics. Micro amounts of tissue obtained by fine needle aspiration have been studied by flow cytometry for lymphoma diagnosis and polymerase chain reaction (PCR) to identify the Epstein Barr Virus (EBV) diagnosis of primary nasopharyngeal carcinoma. FNA biopsy is needed for staging and planning purpose in a patient of distant metastasis, to make a tissue diagnosis to initiate non-surgical therapy and in a patient with unknown mass. If the nature of mass or the source of metastasis identified by FNA remains elusive, the aerodigestive tract should be examined endoscopically especially in the area from which primary lymphatic drainage to mass occurs. An obvious lesion should be biopsied; when no lesion is seen or palpated, guided (not blind) biopsies should be performed of the most logical areas for the silent

primary tumor based on known lymphatic drainage. These areas are usually the nasopharynx around. Roseonmuller's fossa, the tonsil, the base of tongue and pyriform sinus. The rationale for the guided biopsy when an obvious lesion is not present is that the primary tumor is often submucosal or arises deep in the cysts of the palative tonsil or the fold of the lingual lymphoid tissue. Main indication for open excisional biopsy or incisional biopsy are - Progressively enlarging nodes A single asymmetric nodal mass; A persistant nodal moss without antecedent active signs of infection; Actively infectious conditions that do not respond to conventional antibiotics. Asymmetric enlargement of one or more cervical lymphnodes in an adult is almost always cancerous and usually is due to metastasis from a primary lesions from upper aerodigestive tract. Another principle regarding unknown primary lesions is that the immediate removal of an enlarged lymphnode for diagnosis is disservice to the patient with metastatic cervical carcinoma. Distant metastasis and late regional recurrences and wound necrosis are more frequent in patients who have pretreatment biopsies than in those with same stage of disease who have not. These findings suggests that disruption of lymphatic drainage and manipulation of a metastasis decreases the chances for clean surgical excision and cure. In a patient who present with a neck mass and in whom prior routine physical examination of head and neck is negative, an independent second survey of the less visible areas of upper aerodiagnostic tracts is the most cost effective diagnostic tool. Direct endoscopic examination should be performed after T2-weighted MRI because of its better delineation of submucosal disease. Enlarged node high in the neck or in post triangle suggest a nasopharyngeal lesion, whereas enlarged jugulodiagastric node points to the tonsil, base of tongue and the supraglottic larynx. The ipsilateral tonsil should be removed and examined for upper jugular adenopathy. When the enlarged nodes are in the supraclavicular area or the lower third of the neck, the surgeon must consider the whole length of the digestive tract, tracheobronchial tree, breast, genitourinary tract, thyroid gland as potential site for lesions.

Non surgical V/s surgical managements - For a patient with an unknown primary metastatic squamous cell carcinoma, postoperative irradiation of the nasopharynx, ipsilateral tonsil, base of tongue and the contralateral side of neck is frequently advocated after radical neck dissection. The best candidates for postoperative irradiation to control recurrence in the neck are patient whose nodal mass is staged N_1, with nodal capsular penetration, N_2 or N_3. Controversy exists over how to manage patient with N_1 disease not extending through the nodal capsule. These are advocates for surgery to the neck only, for local nodal excision plus irradiation and for complete neck dissection plus radiotherapy. Regardless of whether postoperative irradiation therapy is used, patient with malignant metastatic cervical nodes and unknown primary lesions, must be re-examined frequently. The neck mass in a patient with a known primary neoplasm of the head and neck should be managed according to the principles for each primary site. In general clinically positive cervical lymphnode metastasis are present, a complete cervical lymphadenectomy should be done alongwith removal of primary tumour.

Management of individual cases of neck masses **-** Thyroid neoplasm, both benign and malignant, are a leading cause of anterior compartment neck masses in all age groups. In the pediatric group, thyroid-neoplasm frequently show a male predominance as well as increased incidence of malignant disease. In contrast, the young adult and older groups show a greater incidence of benign conditions and a female preponderance. Ultrasound, thyroid function tests, thyroid scans can be considered for patients having an anterior compartment neck mass. Cystic lesions of thyroid found on ultrasound should be aspirated. Solid lesion should be managed according to there activity on nuclear scan. Functioning nodules should be managed by suppression and all non-functioning cold nodules should be explored with appropriate concomitant therapeutic measure being taken on the basis of histology and extent of disease. Lymphomas, Hodgkin's disease and lymphosarcoma occurs commonly in pediatric and young adult age groups. Except for progressive enlargement of lymphnode tissue, local head and neck symptoms are usually absent. Lymphomas are usually discrete, rubbery and non tender. Extranodal lymphomas can be associated with gastrointestinal or central nervous system involvement and require appropriate evaluation. Salivary neoplasm must be considered whenever an enlarging solid mass lies in front of and below the ear, at the angle of mandible, submandibular

triangle. Pain, rapid growth, cranial nerve (VII) symptoms or skin fixation suggest malignancy. The diagnostic test of preference is open biopsy in the form of complete submandibular gland removal of superficial parotidectomy. The surgeon planning to a open node biopsy must be prepared to perform an immediate neck dissection, the surgeon approaching masses in and around the ear should be prepared to do a total partidectomy and facial nerve dissection. Carotid body tumours or glomus tumors or paraganglioma classically occur in the upper anterior triangle around the carotid bifurcation and are pulsatile, compressible masses that rapidly refill on the release of pressure. Carotid body tumor can be moved side to side but not up or down a "Positive Fontaine Sign". Both a bruit and thrill are present and with glomus vagale tumor, the ipsilateral tonsil may pulsate and be deviated towards midline. The diagnosis is made angiographically. Small tumor in young patient should be resected. In elderly patient or for extensive tumors in patients who are at high risk for functional disability from cranial nerve damage by resection, management by irradiation to arrest the growth with a good long term outcome is permissible. Schwannomas or neurilemomas are solid, neurogenic tumors that occur most commonly in parapharyngeal space and will usually cause medical tonsillar displacement. There origion from vagus nerve can cause hoarseness, or Horner's syndrome when arise from the sympathetic chain. Surgical exploration and excision is indicated after routine evaluatio. Lipomas are ill defined soft masses that occurs in various neck locations in patient older than 35 yrs. These are asymptomatic and on CT scan a lipoma appears as a fat air density. Surgical excision is advised. Branchial cleft cyst most commonly occur in late childhood or early adulthood. They frequently follow an upper respiratory tract infection, and they persist as soft, doughy, variable size masses in anterior triangle of neck, after a course of antibiotic therapy. Ultrasound scan can be helpful in identifying the lesions as cystic. Aspiration of the content yields a milky, mucoid, brownish fluid which often contains cholesterol crystals. Management involves initial control of local infection followed by surgical excision of cyst and its entire tract. Thyroglossal duct cyst are anterior neck, midline structure often appear after an upper respiratory tract infection. USG can be used to differentiate the persistant mass from a lymphnode, a dermoid cyst, or thyroid tissue. A pathognomic sign is vertical motion of the mass with swallowing and tongue protrusism. Radionucleotide scanning being reserved for cyst in the tongue base, which must be differentiated from undescended lingual thyroid tissue. The cyst tract should be completely removed, alongwith the mid portion of the hyoid bone. Lymphangiomas usually occur at birth or evident within first year of life, located commonly in posterior triangle of neck. The cervical lymphangioma is a fluctuant, diffuse, soft, spongy mass, often having indiscrete margin. Its extent is often much greater than apparent. Transillumination is diagnostic. The lesion should be excised if it is easily accessible or is affecting vital functions. Sclerotherapy represents as option in extensive lesions with a high risk of recurrence or complication. Hemangiomas are usually considered congenital are bluish purple colouration, increased warmth compressibility followed by refilling, bruit and thrill help to distinguish them. Traditional management of hemangioma has consisted of observation only unless – rapid growth, thrombocytopenia or involvement of vital structures. Most of these resolve spontaneously. Newer Pulse dye lasers management is being advocated. Local resection of some lesions is also advocated for better end result cosmesis. Dermoid cysts occurs most commonly in pediatric patient and young adults, slowly enlarge because of accumulation of sebaceous content unlike sebaceoun cyst they lie deep to cervical facia and skin moves freely over them. These cysts are curved by simple complete excision. Lymphadenitis, occurs in nearly every person at some point in life, especially during the first decade of life. Lymphadenopathy caused by bacterial or viral infection of the upper respiratory tract, is so common that it is an expected sign. Granulomatous inflammatory diseases affects specific age group and locations like tuberculosis, atypical tuberculosis and actinomycosis common in pediatric group. Excisional biopsy is usually diagnostic and curative. Incisional biopsy should be avoided due to the sequalate of a chronic draining fistula. Cervical lymphnode hyperplasia is ubiquitous in human immunodeficiency virus(HIV) positive patient. Tender enlarging nodes should make one suspicious of tuberculosis or nocardia species – infection, whereas non tender enlarging head and neck nodes often indicates 'Kaposi's' sarcoma or Burkitt's lymphoma. Sequelae of trauma occasionally present as a neck mass. In pediatric patients.

Haematoma, due to forcep delivery, can result a mass in anterior neck within the sternomasthoid muscle, which organized later on. Heat massage and observation are often associated with resolution. Continued growth or increasing torticollis indicate surgical explorations. Pseudoaneurysm of major vessels are occasionally associated with blunt trauma neck. Neuromas are small neck mass that found after surgery, especially radical neck dissection commonly in post triangle of neck. They occur from sensory nerve ending commonly from great anvicular nerve (C2 – 3). Neuromas are tender, associated with sharp shooting pain on palpation, quite slow in growth and require excision.

OPHTHALMOLOGY

Ptosis

History - Taking-Mode and Age of Onset / Progression / Duration / Diurnal Variation; Diplopia; Decrease in Vision; Painful / Painless / Headache ; Trauma / Surgery; Difficulty in speaking and swallowing
Examination- Visual acuity / Aided / Unaided / amblyopia; Head Posture and its significance; Facial features Telecanthus, Epicanthus; Ptosis assessment(Mild / Moderate / Severe) ; Measurement of Vertical fissure height in both eyes; Measurement of MRD1 , MRD2, MRD3 wherever applicable; Measurement and Grading of Levator function ; Ocular movements to R/O Cranial Nerve Palsy / Paresis; Bells phenomenon; Associated Squint; Pupillary Reactions ; Fundus
Investigations - Tensilon Test Indications, Technique, Precautions, Interpretation; EMG; CT /MRI / MR Angio (3ed Nerve palsy) ; Tests for Myasthenia Gravis; Any other
Management / Discussion - Type of Ptosis; Myasthenia Gravis; III N palsy; Blephrophimosis Syndrome; Operative Procedure of choice; Farsenella Servat Surgery Indications surgical steps; Levator Resection Which procedure and how much resection Surgical Steps; Frontalis Sling Indications Technique Materials that can be used follow up; Management of Blehrophimosis syndrome; Management of amblyopia / Use of Crutch Glasses / Temporary management of Ptosis; Management of associated squint

Proptosis

History-Taking- Onset / Progression / Constant / Intermittent ; Painful / painless / Laterality ; Diplopia / Loss / dimness of vision /; Trauma; Thyroid disease; Similar complaints in Past / Remission with steroids / Treatment History; Headache
Examination-Visual Acuity; Facial Features; Unilateral / Bilateral / Axial / Non axial Proptosis; Measurement of Proptosis; Signs of Inflammation; Pulsatile / Non Pulsatile / Reducible / Non Reducible ; Signs of TRO Demonstration ; Ocular Movements / Squint evaluation / Other cranial nerves palsy ; Test Cranial Nerves ; cornea including corneal sensations Exposure Keratitis; IOP; Fundus evaluation
Investigations - Thyroid work up Normal Values; CT Scan; MRI Angio; Any Other
Management - Causes of Proptosis; Management Plan; Management of TRO / IODD; Role of steroids / Immuno drugs; Role of Orbital Decompression; Squint surgery; Any Other

Squint

History-Onset / Progression ; Intermittent / Constant / Association with fatigue / ; Headache / Vomiting / Giddiness; Trauma;Using Glasses / Occlusion therapy / Treatment History / Prisms therapy; Complaints about vision; Head Posture if told; Nystagmus; Diabetes / Hypertension
Examination-Visual Acuity; Refraction / Cycloplegic Refraction acc to age ; Head posture / Nystagmus; Rule out Pseudosquint; Ocular Movements Ductions and Vergence; Pupillary Reactions; COVER TESTS and its interpretations; Hirschberg test; ; Amount of Squint by PRISMS with / without glasses / Distance / Near / Upgaze / Down Gaze / either Eye Fixing; Primary and Secondary Deviation; PRISM BAR COVER TESTS ; Synaptophore examination Status of Binocularity and the confirmation of amount of squint and state of Retinal correspondence; Measurement of AC/A ratio ; Diplopia charting ; Examination of other Cranial nerves; Order ENT Exam in 6th Nerve Paralysis; Worth 4 D Test and other sensory tests; Look out for special squint syndromes like Duane's / Brown; Fundus Examination

Management - Paralytic / Non Paralytic Squint; Role of Refractive error / Accommodation in Squint / Management of Accommodative / Non Accommodative Esotropia ? Role of AC/A Ratio; Sensory Adaptations in squint; Management of paralytic Squint / Optical / surgical Management; Principles of Squint surgery / Muscle weakening / strengthening procedures / role of releasable sutures; Surgical steps of resection and Recession surgeries; Management of Nystagmus

Complicated Cataract

History- Symptoms Duration; Redness of Eyes / Pain in eyes / ; Use of drugs; Any other systemic disease / Diabetes / Myotonia ; High Myopia ; Intraocular surgery; Any Other

Examination- BCVA; Cornea Any Corneal Lesion ; Anterior Chamber depth and activity including Flare and cells; Iris Iris atrophy patches, Tran illumination defects; Posterior Synechie; Pupillary reaction; Ring / annular synechie/ Total Posterior synechie; Lens / polychromatic Lusture / snow flake cataract / Posterior sub capsular cataract; Vitreous; Detailed fundus Evaluation to look for lesions in retina and Choroid; IOP; USG; Examination of other eye

Management- Causes; Problems during Surgery and how to handle them; Which IOL to implant; Post Operative complications / problems and how to handle them

Pterygium

History- Duration / symptoms / any previous pterygium surgery / occupation;Injury / Redness

Examination **-** True Pterygium / Pseudo Pterygium; Progressive / Regressive; Any other

Management **-** Pterygium excision techniques;Use of MMC / Autograft Advantages and Disadvantages operative steps

Post Penetrating Keratoplasty

History **-** Dimness of Vision; INDICATION Injury / corneal Ulcer / Keratoconus; Post operative course; Treatment History

Examination **-** Visual Acuity / BCVA; Cornea Clarity; Graft Host Junction / Endothelial Status / Epithelial Edema; Anterior chamber Reaction; Lens Status / Pseudophakia / Aphakia / Triple Procedure; IOP; Fundus; Look in other eye the indication of P K / Keratoconus/ Corneal dystrophy; Whether corneal graft is Successful / Failure

Discussion - P K / lamellar Graft Indications; Corneal Storage Medium / Eye banking / Donor Tissue Assessment; Causes of corneal decompensation; Procedure of PK /LK; Complications; Management of Astigmatism following PK; any Other

Poly Ocular Trauma

History- Nature of Injury Suffered; Occupation; Medico Legal Aspect

Examination- Assessment of Adnexeal Injuries Fracture Floor of the Orbit; Visual Acuity at presentation PR status; Corneal Tear / Scleral tear / Importance of dangerous zone / ; whether Retained IOFB or not / Siderosis / Chalcosis; Lens status / Dislocation / Subluxation / Cataract Changes; Presence / Absence of RAPD Importance; Vitreous disturbance; Retinal Tear / Detachment; Importance of IOP

Management and discussion- Investigations including Gonioscopy / USG / CT Scan / MRI Indications; Management of Adnexal Injuries / Lid Repair Principles / Cannicular Repair / ; Management of Corneal Scleral tear / Importance of Suture Placement / Importance of prophylactic Cryo or Endolaser; Management of Infections; Traumatic Endophthalmitis; Role of IOFB removal at first / second surgery / VR SURGERY

Endophthalmitis

History - Intraocular Surgery; Trauma / Injury; Corneal Ulcer; Systemic Illness

Examination - Visual acuity especially Projection; Wound status, Suture abscess ; Anterior Chamber ; Reaction and Hypopyon; IOL Type / Aphakia / Vitreous in AC; Red Glow

Discussion - Etiology; Investigations including Vitreous tap. Emphasis on microbiological staining and culture methods; Management including preparation and choice of Intravitreal therapy , Pharmacology of the drugs used; Surgical Management

Facial paralysis
History - Recent Illness / URI; Trauma; Ear Infection ; Leprosy; Other Infections
Clinical Examination - Type of Facial palsy UMN / LMN; Bells phenomenon / Exposure Keratitis / Lagophthalmos; Test other cranial nerves; Test Corneal sensations; ENT examination; Epiphora
Investigations - CT Scans; MRI Scan; E M G
Discussion and management **-** Medical Management; Role of Tarrsorraphy; Facial Blocks

Herpes Zoster Ophthalmicus
History **-** History and development of lesions; HIV / Immunodeficiency
Examination- Involvement of branches; Immunocomprimised lesions; Lesion Characteristics / Staining Techniques; Multiple Cranial Nerve involvement; Uveal Involvement
Discussion - Management / Management in Immunocomprimised patient; Complications

Congenital anomalies of Anterior / Posterior Segment
Points to be discussed - Uveal Lesions Complete / Incomplete / Grading of choroidal coloboma / Clinical Associations / Management / Complications / Genetics; Cornea Dystrophies / Presentations / Investigations and Management; Retina / Clinical Associations / Genetics / Investigations / Management

Traumatic Cataract
History - Nature of Injury / Occupation; Visual Disturbances; Pain / Redness ☹(secondary Gl)
Examination - Visual acuity; Corneal wound if Any (Perforating Injury); Lens status (Rosette Cataract / Subluxated / dislocated); Vitreous status (Loss / In AC / Disturbed and its importance); Retina status . role of USG
Discussion - Timing and Type of the surgery; Use of CTR in Subluxated cataracts / Zonnular Dehiscence; Vitrectomy ; Choice of IOL

Dermoid
Points- Should be able to describe the lesion adequately; Able to give differential diagnosis of dermoid; Systemic Associations of dermoid; Pathlogy of dermoid; Management of dermoid / Excision in toto; Need for Lamellar graft in limbal dermoids

Lid Tumors
Points-Exposure to U V Light / long sun exposure; Premalignant lesions of skin;Should be able to describe the lesion adequately; Palpation of Lymph nodes / should know the lymphatic drainage of the lid / metastasis; Clinical Diagnosis of BCC / SCC / MCC; Histological features of BCC /SCC/ MCC / ; Management options / Frozen Section ; Management of Lid defects / Large and small / name and technique of various lid sharing procedures / skin Grafting

Uveitis

History- Visual disturbances; Pain / redness; Macropsia / Micropsia / Metamorpopsia and its Clinical Correlation; Flashes of Light; Scotoma

Examination- Visual Acuity; Demonstration of Aqueous Flare / Cells; Posterior Synechie; Lens Status / cataract; I O P; Examination of Fundus / Indirect Ophthalmoscopy and Goldman 3 mirror for Pars planitis; Any Other

Discussion - Acute / Chronic / Healed Uveitis; Appropriate investigations; Management(Pharmacotherapy of Uveitis; Details of steroids use / indications / dosage / route and complications; Immunosuppressive drugs / Indications / Routes / Complications) ; Surgical Management; Any Other

Corneal Opacity / Scars
Points to be discussed - Coloured diagram of the findings; Cause of the lesion / Traumatic / Degenerative / Dystrophy; Test Ocular Surface; Management including L K / Amniotic Membrane Graft / P K ; Limbal stem cell transplant / Amniotic membrane graft / Lamellar Keratoplasty

Entropion / Ectropion
Points to be discussed - Cause; Management including surgeries

Pseudophakia
Discussion - Type of IOL Used Advantages and disadvantages of the IOL material; Special IOLS like Phakic I O L, Accommodative I O L, Scleral Fixated I O L, Multifocal; Indications, Properties of the I O L stated as above; Patient Selection Which I O L to use in Multifocals ; Problems with the above I O L'S; I O L Calculation Which Formula to use

Cranio Facial Disorders
Examination and discussion-Know various cranio facial disorders and the etiology in relation with sutures; Know about the various skull sutures ; Complications

Angle Closure Glaucoma
History - Pain / Redness/ Blurred vision; Colored haloes and its significance; Previous attacks; Using Glasses; Headache / Eye ache
Examination with special reference to **-** IOP recording; Anterior chamber depth / Von Herrick's Classification; Gonioscopy findings / Grading of the angle / Occudable / Non occludable; Anterior chamber activity ; Nanophthalmos; Examination of the other eye too; Fundus examination Diagrams of Cup Disc ratio. Marking of the NRR, and Examination of the Nerve fibre layer by red free light . Importance of documentation of findings should be stressed; Visual field examination At least by confrontation Procedure should be explained and tested
Discussion - Management of A C G; Role of Iridotomy / Iridoplasty ; Malignant / Inverse glaucoma

Diabetic Retinopathy
Points- Master 90 D examination Draw Retinal Diagram with color coding; Confirmation of the findings; How to Differentiate MA with Blot Hges; CSME present or not. Criteria for CSME; High Risk Characteristics Staging of DR according to latest criteria; Look out for NVI , NVA, NVD, NVE; Management plan of the patient including Metabolic control , Parameters; Indications / Procedure / details of Focal / Grid and PRP/ Type of laser used / Settings of the laser / No of settings / quadrant selection; VR surgery / Indications / Vitrectomy / Immediate / Late; Associated Hypertensive Retinopathy; Role and Indications of FA Findings therein; Any other

Retinal Vascular Disease
Points - Master 90 D examination Draw Retinal Diagram with color coding; Confirmation of the findings; Ischemic / Non Ischemic; Role of F A and its findings; Systemic examination check up; Treatment options Medical / Whether to do laser treatment or not; Indications / Procedure / Details of Focal / Grid and PRP/ Type of laser used / Settings of the laser / No of settings / quadrant selection; Surgical Management. AV Sheathotomy; Associated Hypertensive Retinopathy

Retinal Detachment
History-Dimness of vision; Loss of field of vision; Floaters / Flashes; Using Glasses HIGH MYOPIA; Trauma / Intraocular surgery; Diabetes; Systemic Conditions
Examination- Visual acuity; Pupillary Reactions; IOP; Indirect Ophthalmoscopy / Goldman 3 mirror / 90 D . Look for Retinal Breaks/ RD Configuration / Traction Bands / Retinal degenerations / Previous RD Surgery bands / Drawing of color coded retinal diagram; Any other
Discussion- Confirmation of Retinal diagram; Lincoff Role; location of retinal break; Management / Whether to treat or not / ; Surgical procedure; V R Surgery

Macular Lesions

Points - Micropsia / Macropsia / Metamorphopsia; Smoking; Examine both eyes; Amsler Chart Ask For; 90 D/ 78D / Goldman 3 mirror mandatory MASTER SLIT LAMP BIOMICROSCOPY; Note the size of Lesion in Disc Diameters; Note other Fundus findings; Whether fresh lesion / old lesion; You may be asked which further investigations you would like to do;

Angle Closure Glaucoma

History- Age (40 – 50 yrs) ; Sex (Female Predisposition) ; C/o episodes of redness, visual blurring, colored halos formation, mild severe pain, nausea/vomiting; H/o precipitating or relieving factors, if any reading in dim illumination, watching movie in cinema hall, psychological factors, and anxious personality; Timing of attacks; evening or morning (Attacks of ACG occur in evenings/times of dim ligh); Frequency & duration of episodes; Spontaneous resolution of such attacks or not; H/o use of hypermetropic glasses; Differential diagnosis of other causes of colored halos in history; Family history.

Clinical Examination-Latent angle closure glaucoma/prod oval stage Pt is a symptomatic; Shallow anterior chamber; Convex lens iris diaphragm; Gonioscopy show occludable angles in at least three of four quadrants; Sub acute angle closure glaucomaSigns & symptoms similar to acute ACG, but mild and papillary reactions are present; Resolution is spontaneous. Acute angle closure glaucoma Redness & marked pain in eye, colored halos; Visual acuity – severe diminution of visual acuity (may be reduced to light perception or hand motions) ; Marked ciliary congestion; Pupillary reactions – fixed & mid-dilated pupil; IOP measurement – marked & acute rise of IOP; Examination of cornea – corneal edema; Examination of AC – shallow anterior chamber; Gonioscopy – closed angles on gonioscopy; Fundas optic disc edema in acute stage; Examination of fellow eye – the fellow eye shows narrow, occludabel angles or other forms of ACG; Signs of past attacks of ACG patchy iris atrophy (mainly around sphincter area, pigment on the posterior surface of cornea (Krukenberg spindle), lens glaucomflecken (Vogt's triad), wide areas of synedchial or appositional closure. Chronk angle closure Progressive diminution of vision; Few symptoms; Mild ciliary congestion; Increased IOP; Glaucomatous cupping of optic disc; Gonioscopy show appositional or synechial closure; Fellow eye show similar gonioscopic pictures or narrow angles; Absolute glancoma Painful blind eye; Fixed dilated pupil; Corneal edema; Iris atrophy; Markedly raised IOP; May be associated with leticular changes or neovascularisation of iris; Advanced glaucomatous cupping.

Investigation- Refraction usually hypermetropic. Gonioscopy the following must be observed Grading of angle; Level of iris insertion, both true and apparent; Shape of peripheral iris – convex, concave, flat; Degree of trabecular pigmentation; Areas of iridotrabecular apposition or synechiae; Dynamic indentation gonioscpy to differentiate between appositional or syechial closure; Slit lamp grading of peripheral anterior chamber depth – Van Herick method; Ultrasound biomicroscopy of the anterior segment – visualization of iris, iris root, corneoscleral junction, ciliary body andlens. Provocative tests Dark room test; Prone test; Dark room-prone test; (Ensure patient does not go to sleep during above tests); Mydiratic test (homatropine 2%); Combined mydriatic miotic test(One eye to be done at a time for the fear of precipitation of attack of ACG).

Differential Diagnosis- It mainly requires to differentiate other causes of acute of IOP. Phacolytic glaucoma open angles, mature lens, cells & flare in AC; Phacomorphic glaucoma intumescent cataractous lens with widely dilated pupil; inflammatory glaucoma cells, flare, KPs, open angles; Ghost cell glaucoma H/O vitreous hemorrhage, open angles, khaki cored cells in AC; Neovascular glaucoma closed angles, dilated pupil with ectropin uveae, NVI; Differentiate other causes of colored haloes early cataract, mucopurulent conjunctivitis; Differentiate other causes of red eye acute conjunctivitis, acute uveitis.

Non- Surgical Management Acute angle closure The aim is to break the attack; Lower IOP; Safeguard the fellow eye by appropriate treatment- Intravenous hyperosmotic agent (mannitol 20%), oral acetazolamide and glycerol 50% (consider side effects and contraindications of these when using) ; Intensive topical pilocarpine 2%. Topical corticosteroid are given to reduce inflammation; Depending

upon IOP control & corneal clarity, laser iridectomy is done; Prophylactic iridectomy in the fellow eye; Peripheral argon laser iridoplasty may help breaking an attack; Sub acute ACG Laser irideectomy; Chronic ACG Laser iridectomy with medical treatment / filtering surgery.
Argon laser trabeculoplasty Indications- Plateau iris configuration; Acute attack of ACG. ; Discuss limitations of Argon Laser Trabeculoplasty;
Surgical treatment- Surgical iridectomy when peripheral anterior synechiae are present in less than 50% of angle or when IOP can be controlled by use of miotics only.
Trabeculectomy & its modification- when peripheral anterior synechiae are present more than 50% of angle / when miotics fail to control IOP; Goniosynechiolysis.
Treatment of absolute glaucoma- (when painful eye) Cyclocrytherapy or cydophotocoagulation; Retrobulbar injection of alcohol; Enucleation as last resort.
Always remember to manage fellow eye.

Pseudoexfoliation (Pex)
History-Usually Asymptomatic; Loss of vision If the disease is very far advanced when diagnosed, the patient may be aware of the loss of vision; Many patients with PEX also develop cataracts and may present with loss of vision on that basis.
Clinical examination- Deposition of whitish exfoliative material . There is deposition of whitish flake like material on the anterior lens capsule that looks as though it is arising from the surface of the lens; typically there is a disc shaped opacity centrally, a mid-peripheral clear zone and additional material deposited on the peripheral portion of the lens; the clear zone corresponds to the area of the lens in contact with the papillary margin of the iris; additional deposits of this whitish material may be seen on the papillary margin, anterior iris stroma, corneal endothelium and the trabecular meshwork. *Pigment Dispersion* Some degree of pigment dispersion in the anterior segment is usually seen with corneal endothelial pigment pigment dotting of the anterior iris stroma, and quite commonly heavy pigment in the trabecular meshwork more marked inferiorly than superiorly; there is a pigment line seen anterior to the schwalbe's line on gonioscopy (Sampaolesi's line). *Iris Trans-Illumination* Atrophy and trans-illumination defects of the peripupillary iris are often seen. *Elevated intra-ocular pressure (IOP)* Usually signinficantly elevated and often markedly asymmetric even without overt signs of glaucoma; often very labile in exfoliative glaucoma; about 20% of patients with newly diagnosed PEX have glaucoma or elevated IOP; about 50% of patients with PEX will ultimately develop exfoliative glaucoms. *Shallow Anterior Chamber* The anterior chamber is shallower, and the angle is more narrow than average in many patients with exfoliation; however angle closure glaucoma is unusual. *Optic Disc Cupping* Indistinguishable from other forms of open-angle glaucoma. *Visual Field Loss* As with other forms of open-angle glaucoma.
Investigations - Diagnosis is based on slit lamp examination; examination under mydriasis is mandatory.
Differential diagnosis - Few things are easily confused with PEX - *Pigmentary Glaucoma* The pigmentation is diffuse darker "mascara" type and bilateral. The iris trans-illumination defects are elongated radial spokes in the iris periphery contrary to peripupillary location in PEX. *Synechiae, Fibrin or Cyclitic Membrane* May involve the anterior lens capsule as debris but may lack the homogenous granular appearance and characteristics flakes of PEX. *True Exfoliation (Capsular Delamination*) Trauma, exposure to intense heat (e.g., glass blowers) or severe uveitis can cause a thin membrane to peel off the anterior less capsule. This usually presents as diaphanous membrane with a free edge floating in the aqueous; very rare. *Systemic Amyloidosis* May produce deposition of flake like material in the anterior segment and may produce glaucoma also; however it is very rare and there are systemic manifestations.
Non-surgical management - Pharmacological -This is similar to that for open angle glaucoma. It is often less effective and labile and poorly controlled IOP persists. The IOP control also becomes more difficult to control with time. *Non-Pharmacological* Laser trabeculoplasty is especially successful in PEX glaucoma; initial success rate is about 80%. However success rates decrease with time as

with open angle glaucoma, averaging 50% or less by 5 years. There have been less reports of success with re-treatment.
Surgical treatment - Indications for filtration surgery and the outcomes are as with open angle glaucoma.
Points to remember - Whitish Material in the papillary area; Non-Dilating pupil;Lenticular Subluxation; Unilateral Glaucoma-*All these call for an evaluation of PEX.*

Amblyopia
History- Mother's pregnancy history, gestational age at time of birth, birth weight & neonatal history; Ask about developmental milestones as developmental delay of the child, positive family history, prematurity increases a child's chance of developing amblyopia; If the child has strabismus – enquire about the following – is it associated with psychological or physical stress, unilateral or alternating, constant or intermittent for distance or near or both? ; Previous treatment, if any, be reviewed such as occlusion therapy, spectacle correction orthoptic therapy any eye muscle surgery; Past & present medication should be recorded, along with drug sensitivities and/or allergic responses, surgical history, anaesthetic methods used & their results; Family history of strabismus or any other eye disorders.
Clinical Examination-Visual acuity assessment(Distance & Near)- Verbal Children (above 3 years) Distance acuity by snellen letters. Often isolated letters can be used, that may lead to measurement underestimating amblyopia. 'Crowding bars' may help to alleviate this problem; In preverbal & non verbal children – CSM method. (C – Corneal light reflex location as the child fixates examiner's light under monocular conditions i.e., opposite eye covered., S – Steadiness of fixation on examiner's light. M – Ability of the child to maintain alignment first with one eye, then with the other, as opposite eye is uncovered. Maintenance if fixation is evaluated under binocular conditions.); Corrected near acuity is recorded with a near card at 14 inches (35cm). *Other Aspects of visual function***-** That may be affected in amblyopia should be tested. Such as – Binocular vision and stereo acuity – by worth four-dot test. Some other functions that are affected e.g. contrast sensitivity, motion perception & processing and spatial localization can be done if facilities are provided. *Assessment of ocular movements. Tests of ocular alignment*- Cover test, corneal light reflex test.
Investigations - To rule out any organic cause of visual loss and identify the underlying cause of amblyopia; Rule out 'malingering' in teens & adults.
Management-The principles are—Elimination of any obstacle to vision such as cataract, central corneal opacity, severe ptosis occurring during infancy or in childhood; Correction of refractive error; In most unilatcral or asymmetric cases forcing use of the poorer eye by limiting use of the better eye.
Refrective Correction **-** Full refractive error as determined under cycloplegia should be correctd; Refractive correction following cataract surgery/keratoplasty/correction of ptosis/ correction of squint must be provided promptly.
Occlusion therapy & optical degradation- Amblyopia Treatment Study (ATS)-Demonstrated equal efficacy of Penalization (Atropine treatment 1-3 days a week) & Occlusion in their early reports. Long term follow up data not yet available.
Factors affecting success of abmblyopia treatment - Age of onset; Depth of amblyopia; Compliance with treatment regime; Presence of associated anomalies
Criteria for Visual Improvement - Isoacuity between the good and the previously amblyopia eye; Equal speed of reading for both the eyes; Free alteration of fixation ensures that a cure has been obtained
When to stop amblyopia treatment? - Isoacuity between the good and the previously amblyopia eye; When there is persistent deficit in visual acuity – if poor compliance can be ruled out continue treatment until no further improvement is noted on three consecutive follow up visits separated by at least 3-4 weeks; Once treatment is stopped, the child must be checked periodically until 9 years of age to detect recurrence of amblyopia.

Recurrence may occur in about 50% of patients when amblyopia treatment is discontinued after fully or partially successful completion of occlusion therapy. However, this can nearly always be reversed with renewed therapeutic effort. Repeated backsliding can be prevented by an acuity maintenance regimen, typically patching for 1-3 hours a day or equivalent. Once the need for maintenance occlusion is established, it should be continued until 8-10 years of age.
Surgical Treatment of Treatable Cases to Prevent Amblyopia - If dense cataract in one eye, Cataract Surgery should be done at the earliest; If unilateral myopia/hypermetropia, refractive corneal surgery may be done; If unilateral corneal opacity, corneal transplantation should be done at the earliest; If squint with or without nystagmus, correction of squint should be done at the earliest.
*Refractive Correction following a froe said surgeries must be given promptly***.**

Adherent Leucoma
History-Onset, duration and progression of following symptoms should be asked - Diminished vision for near and distance; Cosmetic disfigurement; Redness and pain present or not; Presence of squint.Past History - Regarding trauma should be asked, if it is since childhood then age of onset should be asked - Regarding exanthematous fever; Regarding vitamin A deficiency – opacity usually bilateral; Regarding any keratitis – bacterial, fungal, viral.
Clinical Examination - Vision status for distance and near **;** Torch Light Examination - To localize site and size of the opacity; Pupillary reaction – very unpredictable as a part of the iris is incarcerated in the wound; Anterior chamber depth; Cover test to detect phoria and tropia; Projection and perception of rays of lights. Slit lamp Examination - For site, size, vasculature and extent of adherent leucoma; AC Depth; Distance from limbus - Assessment of Cataract; Intra Ocular Pressure (IOP) – ideally should be taken with pneumotonometer / digitally.
Investigations- B Scan; Fluorescein Stain; Dilatation and refraction; Post mydriatic test for correction if possible; Corneal Topography & Specular, Microscopy, Pachymetry, AC Depth assessment.
Differential Diagnosis- Active Ulcer; Abscess; Limbal Dermoid; Band Shaped Keratopathy; Corneal Scar due to injury.
Non-Surgical Management - Contact Lens; Cosmetic Contact Lens; Tattooing
Surgical Options-Optical Iridectomy; Cosmetic Keratoplasty; PK with synechiolysis with AC reconstruction; Cataract and IOL SOS; Squint operations for cosmetic reasons
Other relevant points- Recent advance in keratoplasty, eye banking and various newer medias available for better preservation of donor cornea, keratoprosthesis – newer modalities, suture materials, suturing techniques.

Post Blunt Trauma
History- Mechanism (Divinities & Force) ; Size of Object; Time of Injuring
Examination- Visual Acuity; Pupilly Reaction; Slit Lamp Examination; Funds Examination - Direct Opthalmology; Indirect Opthalmology.
Investigations- Ultrasound Examination; CT Scan; VEP in opth. Neuro Surgery
Differntial diagnosis - In finding a fresh injury; thing is diabetic eye or recently objective eye
Management- Factors treatment are IOP Level; Hyphrame Level; Co meal storing; Any associated disease

CSR
History- Sudden onset of blurred/distorted vision in one eye associated with a relative positive scotoma, micropsia & metamorphopsia
Clinical Examination - *Moderate reduction of vision which is often correctable to 6/6 or so with a weak ‘plus lens’.* Slit Lamp Biomicroscopy & Indirect Ophthalmoscopy —Shallow round or oval elevation of the sensory retina at the posterior pole; Evidence of small precipitates on the posterior surface of the detached neuro sensory retina; A pinpoint whitish area within the elevated detached neuro sesory retina denoting the area of leakage; Evidence of subretinal fluid which could be clear or turbid.

Investigations **-** Amsler Grid- Distortion of straight lines with scotoma; FFA – Smoke stack/ Ink Blot appearance- confirms the diagnosis; Sometimes associated with RPE detachment; OCT – helpful in identifying subtle, subclinical, neurosensory macular detachment
Differential Diagnosis - ARMD (older pts, evidence of Drusen, CNVM, & is often bilateral) ; Optic pit associated with serous detachment; PED – Margins of PED more distinct; Choroidal Tumor involving the
posterior pole; Macular Detachment as a result of Rhegmatogenous Detachment.
Non Surgical Management **-** Wait & watch for spontaneous absorption of fluid & recovery of vision.
Surgical Management - Laser Photocoagulation to the site of leakage in cases of ; Recurrent episodes of CSR; Vision of fellow eye impaired from previous attacks; Duration of symptoms greater than 3-4 mnths; Occupational demands; Very, very rarely in case of bilateral bullous serous detachment not responding to either conservative wait & watch policy or after Laser Photocoagulation.

Pars Planitis
Parsplanitis is described as the type of uveitis in which there is idiopathic accumulation of inflammatory materials in the region of vitreous base and parsplana.Intermediate uveitis (IUSG) is a diagnosis based on anatomic location of ocular inflammation. The intermediate zone of the eye includes ciliary body, pars plana, choroids and peripheral retina as posteriorly as the exit of the vortex vein. Intermediate uveitis is one in which inflammation involve this area of the eye.
History-H/O Dimness of vision or blurring of vision; H/O Floaters; H/O Shortness of breath/ cough/ swelling elsewhere in the body/ weight loss to rule out sarcoidosis/ Tuberculosis/ lymphoma ; H/O Vertigo, ataxia, paresthesia, sphincter dysfunction is taken to rule out Multiple sclerosis; H/O unusual sexual exposure to rule out Syphilis and HIV; H/O skin lesion suggestive of erythema migrans or joint pain like migratory arthralgia or travel to endemic areas of Lyme disease; H/O irregular bowel habits and abdominal symptoms to rule out Inflammatory bowel disease; H/O contact with pets particularly puppies for suspected Toxocariasis
Clinical Features- Most common presenting feature is painless blurring of vision accompanied by floaters.
Rarely patient may present with unilateral blurring of vision with anterior uveitis like pain, redness, photophobia etc. ; Vision is usually 6/12 on presentation and most patients don't have systemic illness
signs - Quiet anterior segment, uncommonly mild anterior segment inflammation may be present; *Vitreous cells*- can range from being occasional to 3+ depending on the severity and chronicity of disease process; The classic finding is "*Snows bank"* where in the exudative aggregate's deposited in the inferior pars plana and anterior retina. These are seen in *indirect ophthalmoscopy and scleral depression*; Cystoid macular oedema (CME) is present in 28-64% of patients at presentation and is usually cause for decreased vision; Focal areas of phlebitis in retinal periphery can occasionally be seen. Disc oedema is present in 3-20% of the eyes; Although patients are symptomatic in only one eye, the other eye shows signs characteristic of involvement. Hence *examination with scleral indentation of the fellow eye is very important.* ; In some cases only vitreous cells can be present without snow bank or CME. This termed as "Pars plana variant" which could be an early form of pars planitis; In case of intraocular lymphoma, vitreous cells will be large, fluffy and the course; of disease is resistant to steroid treatment
Investigation **-** Investigation is guided by result of assessment and review of systems. *Angiography (FFA)* may be useful in patients with vision of *6/12 or less should document macular edema* and justify therapy. The aim of each investigation is to rule in or rule out specific diagnosis. Few of the tests are- *Complete blood count* with differential (to rule out Myelo-proliferative and infectious disease) ; *Angiotensin converting enzyme***,** serum calcium, Lysozyme, Gallium scan-for sarcoidosis; *Chest X-Ray; Mantgoux (110, 000), ESR***-** for Tuberculosis; *VDRL* or TPHA or FTA – ABS To rule out syphilis; ELISA for *HIV;* ANA rarely for collagen vascular disease; *Toxocara antibody testing in children; HLA – DR* full panel (Common haplotypes – A3, B7, DR2, DW2) in-patient with h/o Multiple

Sclerosis if MRI shows definite plaques and other evidence in Myelography or CSF studies; Rarely *ECOGRAPHY-In* case of typical Pars Planitis or Intermediate Uveitis, ecography (B-USG) adds no information. However in severe vitreous inflammation, RD, Toxocara canis granuloma, complicated cataract it can be useful. Using UBM, it is possible to demonstrate Pars Plana exudates and even inflammatory cell aggregates in the vitreous.

Differential Diagnosis **-** Sarcoidosis; Multiple sclerosis; Tuberculosis; Syphilis; Lyme disease (In

Endemic areas); Human T-Cell leukemia Virus Type I; Cat-Scratch Disease; Epstein Barr Virus;

Intraocular Lymphoma; Toxocariasis; Inflammatory Bowel Disease etc.

Management- Management in Pars Planitis is individualized and based on parameters like – Presence of unilateral or bilateral disease; Visual acuity in each eye; Severity of inflammation, presence of CME and threat to vision; Management decisions are clearly based on the level of visual acuity and presence of macular edema; Patients with amblyopia of unrelated causes of one eye are managed as patients with only one eye.

Treatment -Disease observation; Medical Therapy; Surgical Therapy

The four step therapy by HJ Kaplan as a protocol consisting of -1st step was to use peri-ocular or oral steroids. If this fails 2nd step was cryo therapy to area of snow bank, if this fails 3rd step was therapeutic pars plana vitrectomy and finally use of immune suppressive as 4th step (This 4th step approach has been modified and the use of other immune suppressive agent or anti-metabolites is being tried before cryotherapy and therapeutic vitrectomy).

Peri-ocular steroid consider In unilateral conditions- In bilateral conditions and patient having other

systemic disease like diabetes, where oral steroids are relatively contraindicated; Periocular steroids of Inj.

Triamcinolone acetonide (Tricot) 40 mg is given by, subtenon's route every 3 weeks. IOP needs to be monitored to rule out steroid induced glaucoma; Dexamethasone Injection (4mg/ml vial, 20mg/ml vial)

(Decadron, Decdan, Dexona) can be given as periocular steroid.

Oral steroid consider Patient has bilateral affection - Peri-ocular steroid cannot be given or tolerated. ; Patient is a steroid responder; Oral steroids are started in high doses at 1 mg per kg body weight for atleast

2 weeks and then tapered slowly depending on response over few months. Oral calcium (1000 to 1500 mg

daily) and antacids are supplemented; Very rarely, high dose intravenous corticosteroids (Injection IV methyl prednisolne) 1 gm IV for 3 consecutive days may be used in patients who fail to respond to oral and periocular corticosteroids and who have vision threatening lesions; If steroid fail or patients cannot tolerate steroids alternative immunosuppressive can be considered. Drugs like tab. Azathioprine 1.5-2 mg per/kg/day. (Available as 50mg tablet), Tablet Methotrexate – 0.1 – 0.5mg/kg/ week taken orally once in a week, (available as 2.5 mg tablet). Cyclosporin (2-5mg kg/day in one dose or 2 divided dose daily, (available as 25,50,100mg capsule); If intermediate uveitis is still recalcitrant and severe then cryotherapy (single freeze-thaw treatment described by Aarberg)/ Laser therapy is tried in the areas of snow banking to disrupt the neovascularisation; Therapeutic vitrectomy is needed in severe cases with a hope of – reducing

CME, Decreasing the visual morbidity, to decrease the dose of oral steroids or antimetabolite.

Surgical Therapy- Cryotherapy and scatter Laser photocoagulation of the peripheral retina as well as pars

plana vitrectomy with or without pars plana lensectomy have been shown to be effective in the treatment of pars planitis. Likewise, visual rehabilitation through cataract extraction with IOL implanatation in complicated cataract following pars planitis is a safe and effective procedure, provided vigilant perioperative control of inflammation is achieved; Pars Plana Vitrectomy with or without Pars Plana lensectomy is not only the modality of choice to treat certain complication of pars planitis which include dense vitreous opacification, tractional or rhegmatogenous RD, vitreous haemorrhage, epiretinal membrance formation but it may also have salutary effect on active disease refractive to medical treatment & on CME and may ultimately alter the natural history of the disease.

Course & Prognosis Pars Planitis may become inactive and quiescent after a protracted course. Tight control of inflammation and close monitoring of the disease as well as its complications may result in good visual recovery.

In long term follow up study of 182 eyes Smith et al suggested that pars planitis has 3 patterns Self limited benign course – where there is gradual improvement with only rare recurrences (10% of cases fall in this group) ; Prolonged course without any exacerbation was seen in 59% of cases; A chronic smoldering course with one or more episodes of exacerbation of inflammation 30% of eyes were seen in this category.

Regardless of the pattern of the disease, most eyes were seen to improve or remain unchanged in terms of visual acuity.

Follow up In the acute phase, patients are reevaluated every 1 to 4 weeks, depending on the severity of the condition. In the chronic phase, reexamination is performed at 3 to 6 months interval.

Congenital Ptosis

History- Age of onset; Family history; Fluctuating ptosis with strabismus; Allergic/ drug reaction; Trauma; Anti coagulant use/ bleeding disorder; Complete medical history; Family history of malignant hyperthermia; Head position.

Clinical Examination- Head position; Visual acuity, refractive error Cycloplegic refraction; Dialated fundus examination;Strabismus evaluation; Absent upper lid crease; Tear function; Corneal sensitivity; Exophthalmometry; Pupillary size; Iris colour difference; Lid height (palpebral fissure distance) ; Lid position in down gaze; Levator function; MRD1, MRD2; Bells phenomenon; Palpation of eyelids and the orbital rim to rule out mass lesion; Marcus Gunn jaw – winking phenomenon

Investigation - Photographs for documentation; Neuro imaging of orbit and brain; EMG, Tensilon test, ice test; ECG if mitochondrial disorder is suspected; Muscle biopsy and mitochondrial is suspected

Differential Diagnosis - CPEO; Myasthenia gravis; Myotonic dystrophy; Pseudoptosis

Non Surgical Management - Observation in mild cases if no sign of amblyopia, strabismus and abnormal head posture; Treatment of amblyopia, refractive error; Eyelid crutches

Surgical Management - Levator muscle resection; Frontalis muscle suspension; Fasanella – servant procedure

Keratoconus

History - Multiple unsatisfactory attempts in obtaining optimum spectacle correction; Decreasing Vision; Distortion, Monocular diplopia or ghost images; Sudden loss of vision with some ocular discomfort; Intolerance to contact Lens; Frequent eye rubbing; Allergic tendencies and Atopy; H/O associated systemic Problem like Down Syndrome, Connective tissue disorders; H/O associated ocular problem like Retinitis Pigmentosa, VKC, Leber's Congential amaurosis, aniridia, ROP, CL wearing

Clinical Examination - General Examination; To R/O Systemic association
Ocular Examination - Lid – Sign of Ocular Allergy; Conjunctiva – Sign of Ocular allergy; Cornea - Fleischer ring; Vogts Striae; ; Corneal Thinning; Eccentrically located ectatic protrusion (cone) ; Breaks in Bowmans Layer; Stromal Scars; Enhanced appearance of Corneal nerves; Rizzuti Sign; Munson's Sign; Scar at the level of DM; Oil drop sign in distant direct Ophthalmoscopy; Scissoring reflex in Retinoscopy
Investigation - Placido disc; Keratometry; Computer assisted Videokerato graphy; Ultrasonic Pachymetry
Differential Diagnosis - Pellicuid Marginal degeneration; Terrien's Marginal degeneration; Kerato Globus
Non Surgical Management - Spectacles; Contact Lens-Soft, RGP, Piggy back lenses;Special lenses for advanced KC; Sclera Lenses; Medical management of Hydrops
Surgical Management - Intra Stromal rings; Deep anterior Lamellar Keratoplasty; Penetrating Keratoplasty; Epikeratoplasty; Keratectomy to remove the nodular scar

Pseudoexfoliation (Pex)
History - Usually Asymptomatic; Loss of vision - If the disease is very far advanced when diagnosed, the patient may be aware of the loss of vision; Many patients with PEX also develop cataracts and may present with loss of vision on that basis.
Clinical Examination Deposition of whitish exfoliative material - There is deposition of whitish flake like material on the anterior lens capsule that looks as though it is arising from the surface of the lens; typically there is a disc shaped opacity centrally, a mid-peripheral clear zone and additional material deposited on the peripheral portion of the lens; the clear zone corresponds to the area of the lens in contact with the papillary margin of the iris; additional deposits of this whitish material may be seen on the papillary margin, anterior iris stroma, corneal endothelium and the trabecular meshwork. *Pigment Dispersion* Some degree of pigment dispersion in the anterior segment is usually seen with corneal endothelial pigment pigment dotting of the anterior iris stroma, and quite commonly heavy pigment in the trabecular meshwork more marked inferiorly than superiorly; there is a pigment line seen anterior to the schwalbe's line on gonioscopy (Sampaolesi's line). *Iris Trans-Illumination* Atrophy and trans-illumination defects of the peripupillary iris are often seen. *Elevated intra-ocular pressure (IOP)* Usually signinficantly elevated and often markedly asymmetric even without overt signs of glaucoma; often very labile in exfoliative glaucoma; about 20% of patients with newly diagnosed PEX have glaucoma or elevated IOP; about 50% of patients with PEX will ultimately develop exfoliative glaucoms. *Shallow Anterior Chamber* The anterior chamber is shallower, and the angle is more narrow than average in many patients with exfoliation; however angle closure glaucoma is unusual. *Optic Disc Cupping* Indistinguishable from other forms of open-angle glaucoma. *Visual Field Loss* As with other forms of open-angle glaucoma.
Investigations - Diagnosis is based on slit lamp examination; examination under mydriasis is mandatory.
Differential Diagnosis - Few things are easily confused with PEX- *Pigmentary Glaucoma* The pigmentation is diffuse darker "mascara" type and bilateral. The iris trans-illumination defects are elongated radial spokes in the iris periphery contrary to peripupillary location in PEX; *Synechiae, Fibrin or Cyclitic Membrane* May involve the anterior lens capsule as debris but may lack the homogenous granular appearance and characteristics flakes of PEX; *True Exfoliation (Capsular Delamination)* Trauma, exposure to intense heat (e.g., glass blowers) or severe uveitis can cause a thin membrane to peel off the anterior less capsule. This usually presents as diaphanous membrane with a free edge floating in the aqueous; very rare; *Systemic Amyloidosis* May produce deposition of flake like material in the anterior segment and may produce glaucoma also; however it is very rare and there are systemic manifestations.
Non-Surgical Management - Pharmacological This is similar to that for open angle glaucoma. It is often less effective and labile and poorly controlled IOP persists. The IOP control also becomes more difficult to control with time*; Non-Pharmacological* Laser trabeculoplasty is especially successful in

PEX glaucoma; initial success rate is about 80%. However success rates decrease with time as with open angle glaucoma, averaging 50% or less by 5 years. There have been less reports of success with re-treatment.
Surgical Treatment - Indications for filtration surgery and the outcomes are as with open angle glaucoma.
Points to remember- Whitish Material in the papillary area; Non-Dilating pupil; Lenticular Subluxation; Unilateral Glaucoma — All these call for an evaluation of PEX.

Sellar Tumours
The most important point to note in the case of sellar tumours is that the main entity is a pituitary tumour and pituitary tumours usually give ocular symptoms till they grow to a macroadenoma
History - Visual Symptoms - common with non-functional adenomas - Painless asymmetric visual loss and mono-ocular blindness This is the predominant ocular symptom; almost 2/3 have visual symptoms and 2/3 of them have loss of vision as the presenting complaint; the great majority of the visual symptoms consist of visual loss in one or both eyes; Many patients are unaware of their visual loss; The visual loss precedes other symptoms of headache and endocrinopathy in almost half the cases; The duration between the visual disturbances and the other symptoms would vary between less than a month to more than ten years; Attacks of amaurosis some lasting as long as an hour may precede permanent visual loss; Occasionally acute mono-ocular visual loss in a previously healthy individual is the first evidence of a pitpitary tumour(confused with retro-bulbar neuritis); Central vision may be reduced in both eyes acutely, though still asymmetric (one should suspect acute pre-chiasmal expansion); Vision decomposition in patient with known bitemporal hemianopia is suggestive of pituitary apoplexy. *Hemifield Slide Phenomena* Intact nasal fields when overlap produce a sensory *diplopia(non-paraetic diplopia),* or separate to where objects seem to disappear from the central vision. *Postfixation Blindness* In a complete bitemporal hemianopia when a patient converges to a point, his blind hemianopic visual fields align behind fixation; when the patient attempts to thread a needle, for example, the thread enters the eye of the needle, but does not appear to exit on the other side.
Non-visual - Headache fairly common; bursying headache is considered characteristic of pituitary adenoma; frequently frontal, bitemporal or retro-orbital; Decreased libido, impotence; Amenorrhoea; Galactorrhoea, oligomenorrhea; Infertility Asthenia; Acromegaly in adults and gigantism in young; Cushing's syndrome; Pituitary Dwarfism (delayed body growth); rohlich's syndrome (adiposogenital dystrophy); immond's disease (reduced hair growth, intolerance to cold temperatures, weight gain, cachexia)
Clinical Examination - isual field defects with respect for the vertical border- Bitemporal hemianopia that is *greater near fIXation* is the pathognomonic of visual field defect of an intra-sellar tumour; seen in 70% of the cases; Mono-ocular central scotoma with supero-temporal defect in the other eye (junctional scotoma of Traquair) is as diagnostic of a chiasmallesion as is Bitemporal hemianopia; Mono-ocular field loss alone in the form of central scotoma or temporal anopia or superor or inferior temporal depression may occur; Nerve fibre bundle defects; Homonymous hemianopia with a prefixed chiasm as the growth compresses the tracts. *Optic nerve head defect A late sign of chiasmal affection, weakly correlated with duration of the symptoms and strongly correlated to persistent decreased post-operative visual acuity;* visual acuity loss, relative afferent papillary deficit, dyschromatopsia, pallor of the neuro-retinal rim of the optic disc and dropout of the nerve fibre layer (Disc pallor was once seen in about 2/3 of the cases; with improved endocrinological diagnosis this has come down to about 2% only)
Investigations Perimetry any type of perimeter will produce the typical field defects of a sellar tumour; the kinetic perimetry programme by the computerised perimeters will give a field with which the neuro-surgeons are conversant with *Neuro-iD.1aging* the hall mark of the investigation for a chiasmallesion; CT picks up calcification seen in 90% of craniopharyngiomas and in meningiomas of this region; but MRI will be the investigation of choice *Endocrinological evaluation Must*

Differential Diagnosis of Visual Fields Bilateral optic nerve lesions - Primary Open Angle Glaucoma (POAG), Disc anomalies; Testing artifacts; Psychogenic visual loss; 4. An empty sella syndrome should be entertained particularly in patients with pseudo- tumour cerebri and in patients who have undergone pituitary tumour resection. *Of Optic nerve deficit - POAG* The field defects would be different; however in doubtful cases neuro- imaging is a must; in a few cases both could co-exist*; Retro Bulbar Neuropathy (RBN)* monocular vision loss with a normal looking optic nerve-head would give a diagnosis of RBN and steroid therapy has been instituted with improvement in the condition only to deteriorate with the cessation of the same; MRI would be diagnostic for both.
Non-Surgical Management - Bromocriptine/ octreotide for functional adenomas and in macro adenomas prior to surgery for shrinking the mass
Surgical Treatment Trans-sphenoidal/ trans-ethmoidal for miro-adenomas; Trans-frontal for macro-adenomas. Post -surgical radiation is necessary for prevention of re-growth
Points to Remember In any case with pale optic disc, a chiasmallesion should be kept in mind. The following are some pointers - A pale optic disc with normal Intra-Ocular Pressure, Non-glaucomatous fields with a pale disc, Rapid deterioration of visual parameters in a patient on anti-glaucoma treatment. A working classification of sellar turnours Primary intracranial turnours - A. Intrasellar (Pituitary adenoma, Pituitary carcinoma), B. Extrasellar (Craniopharyngioma, Sphenoid ridge meningioma, Tuberculum sella meningioma, Dysgeminoma, Optic, chiasmal and hypothalamic gliomas, Cavernous sinus turnour) ; Primary cranial turnours (Sarcoma, Chondroma, Multiple myeloma, Giant cell turnour), Metastatic (Nasopharyngeal (by direct spread), TumoUTS of the breast, lung, stomach, kidney and colon(mainly by hematogenous spread))

Adherent Leucoma

Symptoms- Onset, duration and progression of following symptoms should be asked – Diminished vision for near and distance; Cosmetic disfigurement; Redness and pain present or not; Presence of squint
Past History - Regarding trauma should be asked, if it is since childhood then age of onset should be asked-Regarding exanthematous fever, Regarding vitamin A deficiency – opacity usually bilateral, Regarding any keratitis – bacterial, fungal, viral
Clinical Examination - Vision status for distance and near –Torch Light Examination-To localize site and size of the opacity; Pupillary reaction – very unpredictable as a part of the iris is incarcerated in the wound; Anterior chamber depth; Cover test to detect phoria and tropia; Projection and perception of rays of lights. Slit lamp Examination-For site, size, vasculature and extent of adherent leucoma; AC Depth; Distance from limbus; Assessment of Cataract; Intra Ocular Pressure (IOP) – ideally should be taken with pneumotonometer / digitally.
Investigations- B Scan; Fluorescein Stain; Dilatation and refraction; Post mydriatic test for correction if possible; Corneal Topography & Specular, Microscopy, Pachymetry, AC Depth assessment
Differential Diagnosis- Active Ulcer; Abscess; Limbal Dermoid; Band Shaped Keratopathy; Corneal Scar due to injury;
Non-Surgical Management - Contact Lens; Cosmetic Contact Lens; Tattooing
Surgical Options-Optical Iridectomy; Cosmetic Keratoplasty; PK with synechiolysis with AC reconstruction; Cataract and IOL SOS; Squint operations for cosmetic reasons
Other relevant points- Recent advance in keratoplasty, eye banking and various newer medias available for better preservation of donor cornea, keratoprosthesis – newer modalities, suture materials, suturing techniques.

Amblyopia

History- Mother's pregnancy history, gestational age at time of birth, birth weight & neonatal history; Ask about developmental milestones as developmental delay of the child, positive family history, prematurity increases a child's chance of developing amblyopia; If the child has strabismus – enquire about the following – is it associated with psychological or physical stress, unilateral or alternating, constant or intermittent for distance or near or both?; Previous treatment, if any, be reviewed such

as occlusion therapy, spectacle correction orthoptic therapy any eye muscle surgery; Past & present medication should be recorded, along with drug sensitivities and/or allergic responses, surgical history, anaesthetic methods used & their results; Family history of strabismus or any other eye disorders.

Clinical Examination -Visual acuity assessment (Distance & Near) – Verbal Children (above 3 years) Distance acuity by snellen letters. Often isolated letters can be used, that may lead to measurement underestimating amblyopia. 'Crowding bars' may help to alleviate this problem; In preverbal & non verbal children – CSM method. (C – Corneal light reflex location as the child fixates examiner's light under monocular conditions i.e., opposite eye covered., S – Steadiness of fixation on examiner's light. M – Ability of the child to maintain alignment first with one eye, then with the other, as opposite eye is uncovered. Maintenance if fixation is evaluated under binocular conditions.) ; Corrected near acuity is recorded with a near card at 14 inches (35cm).*Other Aspects of visual function* That may be affected in amblyopia should be tested. Such as – Binocular vision and stereo acuity – by worth four-dot test. Some other functions that are affected e.g. contrast sensitivity, motion perception & processing and spatial localization can be done if facilities are provided.*Assessment of ocular movement. Tests of ocular*

alignment Cover test, corneal light reflex test.

Investigations **-** To rule out any organic cause of visual loss and identify the underlying cause of amblyopia. Rule out 'malingering' in teens & adults.

Management- Principles are— Elimination of any obstacle to vision such as cataract, central corneal opacity, severe ptosis occurring during infancy or in childhood; Correction of refractive error; In most unilatcral or asymmetric cases forcing use of the poorer eye by limiting use of the better eye.

*Refrective Correction***-** Full refractive error as determined under cycloplegia should be correctd; Refractive correction following cataract surgery/keratoplasty/correction of ptosis/ correction of squint must be provided promptly.

Occlusion therapy & optical degradation Amblyopia Treatment Study (ATS) Demonstrated equal efficacy of Penalization (Atropine treatment 1-3 days a week) & Occlusion in their early reports. Long term follow up data not yet available.

Factors affecting success of abmblyopia treatment - Age of onset; Depth of amblyopia; Compliance with treatment regime; Presence of associated anomalies

Criteria for Visual Improvement- Isoacuity between the good and the previously amblyopia eye; Equal speed of reading for both the eyes; Free alteration of fixation ensures that a cure has been obtained

When to stop amblyopia treatment - Isoacuity between the good and the previously amblyopia eye; When there is persistent deficit in visual acuity – if poor compliance can be ruled out continue treatment until no further improvement is noted on three consecutive follow up visits separated by at least 3-4 weeks; Once treatment is stopped, the child must be checked periodically until 9 years of age to detect recurrence of amblyopia. Recurrence may occur in about 50% of patients when amblyopia treatment is discontinued after fully or partially successful completion of occlusion therapy. However, this can nearly always be reversed with renewed therapeutic effort. Repeated backsliding can be prevented by an acuity maintenance regimen, typically patching for 1-3 hours a day or equivalent. Once the need for maintenance occlusion is established, it should be continued until 8-10 years of age.

Surgical Treatment of Treatable Cases to Prevent Amblyopia If dense cataract in one eye, Cataract Surgery should be done at the earliest; If unilateral myopia/hypermetropia, refractive corneal surgery may be done; If unilateral corneal opacity, corneal transplantation should be done at the earliest; If squint with or without nystagmus, correction of squint should be done at the earliest.

Refractive Correction following a froe said surgeries must be given prompthy.

Acute Dacryocystitis

History – Previous episodes? Concomitant ENT infection; External Examination – Gentle compression of the lacrimal sac with cotton tipped swab in an attempt to express discharge from the punctum. This is done bilaterally to uncover subtle contralateral dacryocystits; Ocular Examination – Check extraocular movements and look for proptosis (Hertels exopthalmometry); Gram stain, blood agar culture chocolate agar culture in children) f discharge expressed from the punctum; CT scan (axial and coronal) of orbital and PNS in atypical or severe cases or cases unresponsive / worsening to antibiotics. Probing/Irrigation is contraindicated during the acute stage.

Etiology- NLD obstruction; lacrimal sac diverticula; dacryolith; nasal or sinus surgery; trauma or; lacrimal sac tumor (rare)

Symtoms- Pain, Redness, Swelling over the innermost aspect of lower eyelid (over lacrimal Sac) tearing, Discharge; Fever; may be recurrent.

Signs- Erythematous, tender swelling centered over the nasal aspect of the lower eyelid and extending over the periorbital area nasally; Mucoid/purlent discharge expressed from the punctum when pressure is applied over the lacrimal etc.; Dacryocystitis has swelling below the medical central tendon; Lacrimal sac tumor (rare) if swelling is above the medical canthal tendon should be considered; Fistulas after emerging from skin below the medical canthal tendon; Rarely orbital or facial cellulites

Microbiology- Staphyiococci, Streptococci, Diphtheroids.

Differential Diagnosis- Facial Cellulits involving the medical canthus (discharge cannot be expressed from the punctum by placing pressure over the lacrimal sac and radiographically patient lacrimal drainage system); Acute Ethmoid sinusitis (Pain, tenderness, erythema over the nasal bone medical to the inner canthus) Frontal headache and nasal obstruction are common. Patients are often febrile; Acute frontal sinusitis (Inflammation predominantly involves upper eyelid. The forehead is tender on palpation).

Treatment Children- Afebrile, systemically well, mild case, reliable patient-Amoxycillin/Clavulanate or Cefaclor 20-40 mg/kg/d in 3 divided doses Febrile, acutely ill, moderate to severe care, Cefuroxime 50-100 mg./kg/d IV in 3 divided doses. *Adults*- Cephalexin 500 mg. PO Q 6H IV Cefazolin 1g Q 8H; Topical antibiotic drops; Warm compress and gentle massage to the inner canthal region qid; I & D of pointing abscess; DCR with silicone intubation after resolution especially in chronic dacryocystitis

Vitreous Hemorrhage

History- Age; Trauma; Chronic illness; Diabetes; Hypertension; Glaucoma or its treatment; Similar episode; Diminution of vision/redness/pain

Clinical Examination - Vision; Rule out Trauma – Corneal/scleral laceration, angle recession, iris tear, foreign body, lens dislocation etc. ; Evidence of inflammation – Flare/Cells in aqueous, Hypopyon, Posterior Synechiae, Pigment on the lens surface, Keratic precipitates. ; Iris/Angle neovascularisation; Intraocular pressure; If fundus is visible – Renital Detachment (RD), disc neovascularisation, neovascularisation elsewhere, peripheral retinal tears, Macular neovascularisation, evidence of vessel occlusion, foreign body

Investigations - *General*- Blood Hb, TLC, DLC, Erythroytic sedimentation Rate; Urine Routine; Blood Sugar – Fasting and Post-prandial; X-ray Chest PA View; Montoux Test. *Ophtalmologic* - X-Ray Orbit – AP and lateral view; Ultrasound, if media is hazy – to see for posterior vitreous detachment/ RD/Tumour/foreign body; CAT Scan, if a foreign body is suspected to be in the coats of the eye; Culture of forinces/aqueous/vitreous, if there is a suspicion of infection

Differential Diagnosis - Hazy Viterous due to Posterior Uveitis; Asteroid Hyalosis; Chronic Endophthalmitis

Non-Surgical Management - Treat inflammation with topical and oral steroids; Treat infection if any ; Reduce Intraocular pressure, if raised

Surgical Managemet - Do a USG and if there is RD or element of traction on the surface of retina then do an early vitrectomy and definitive RD surgery; Otherwise wait for at least 10-12 weeks for age. to resolve; Tie an encircling band if there is evidence of traction; Endolaser in Diabetics/ Retinal Vasculitis/Vein Occlusions

Fuch's Endothelial Corneal Dystrophy (FECD)

History- A patient may complain have less than satisfactory 6/6 vision-Early morning vision may be reported as misty. As the day progresses, the mist clears. An observant patient may make this complaint, Mistiness may remain much longer than merely in the morning. It may persist the whole day. In the early stages, it is improved by use of hypertonic drops and ointment; Patients may have difficulty performing visual tasks, which require attention to fine letters or figures; Patients may see halos around the sources of light; Patients may feel a gritty or foreign body sensation during part of or during the whole day; Progressive fall in the corrected visual acuity occurs over previous months or years; Attacks of redness, pain, and watering, lasting for hours or days occurs; Constant redness, pain, watering, and poor vision may be present; Rapid onset of symptoms of fading vision and irritation after an intraocular operation, especially for cataract, may occur.

Family History - Important as may be autosomal dominant

Past history - Of medication used eg., topical sodium chloride 5%; antiglaucoma medication; A slow and poor recovery of vision may occur after a cataract operation; Nd Y AG laser surgery for secondary cataract - increasing visual deterioration may develop, sometimes weeks or months.

Demography - Elderly women

Clinical Examination Bilateral ; Asymmetric disease of central cornea; Vision - Snellen's chart; Retinoscopy - Red reflex reveals diffuse mottled appearance of descemets membrane

Lids - Lids are normal in early cases. -They may appear red and congested in advanced cases.

Conjunctiva - Conjunctiva is normal in early cases; It may be highly congested, especially around the limbus, when epithelial erosion, bullae formation, or infected ulceration is present. *Corneal epithelium* **-** The corneal epithelium is normal and transparent in early cases; Bedewing of the epithelium occurs because of epithelial edema; Epithelial bullae may be present.; Pannus formation occurs; Ulceration with or without infection may be present; The corneal epithelium may be thick and opaque; Clouding of anterior cornea – technique-Indirect illumination, Sclerotic scatter ; Late Sub epithelial scarring and end stage cicatrisation (diffuse sheet of scar) *Bowmann's scarring. Corneal stroma* - The corneal stroma has a normal transparency in early cases; Appearance of striae in the deeper layers is observed due to folds in the Descemet membrane; Edema of the corneal stroma occurs, first posteriorly and later anteriorly; Thickening of the corneal stroma develops; Vascularization is present; Late A diffuse ground - glass like stromal haze of central cornea. *Descemet's membrane* – Thickened; Has a beaten-metal appearance. *Corneal endothelaium* - Corneal guttae multiple, central guttae associated with a fine stippling of pigment on the posterior corneal surface.

Method of examination - Direct illumination - Appearance of guttae Golden, refractile mounds on the posterior corneal surface. Specular reflection - Black holes in the endothelial mosaic; Beaten metal appearance may be seen in specular reflection. A similar appearance may be visible at the edge of the central corneal on retroillumination.

(Please note Guttae are excrescences of Desceme'ts membrane produced by abnormal endothelial cells)

Anterior chamber -is normal unless it is involved in some complication of the cornea. *Pupillary reaction* - direct; consensual should be checked as associated glaucoma + optic atrophy (GOA) may go exist -Iris, lens, vitreous, and retina are not involved in the process; Lens changes should be noted (cataract) (for surgical management).

Investigation Corneal sensation is usually diminished, IOP Intraocular pressure (Digital tension, Schiotz, Goldmann) - Intraocular pressure (IOP) is within the reference range; IOP may be raised independently of the disease. *Pachymetry (indicates relative endothelial function)* - To measure central corneal thickness (CCT); Morning and evening values if difference greater than 10% chance

for decompensation after cataract surgery; Pachymetry is a good way of gauging the increase in corneal edema. The thickness can be compared with the new readings on subsequent visits. Increasing thickness of the cornea means increasing corneal endothelial decompensation. Presence of Descemet folds, epithelial bedewing, and corneal thickness of greater than 0.62 mm indicates potential decompensation. *Specular microscopy* - Endothelial cell density, hexagonality, and polymegethism may be recorded. The following 5 stages may be seen - Stage 1 The guttate excrescences are in the form of dark structures with sharply defined single bright spots at their center. The structures are considerably smaller in size than a single endothelial cell. Such an excrescence does not lie near the boundary wall of the cell. Stage 2 The excrescence is almost the size of the endothelial cell. The surrounding cells have a stretched appearance.Stage 3 The excrescence is considerably larger, and many cells are involved in one lesion. The dark structure is 5-10 times the size of an endothelial cell. The adjacent cells are abnormal and have missing boundaries. Many lesions are seen close to each other, but they do not coalesce. The excrescences are of 2 types, a smooth round shape or a rough excrescence. Stage 4 The individual excrescences have coalesced. The net result is multilobed, rather than a round outline. The dark areas have many bright spots. The multilobulated structures cover considerable area. The cells between the excrescence masses tend to become abnormal. Coalesced areas contain both the smooth and the rough variety of excrescences. Stage 5 An organized mosaic of endothelial cells is difficult to see. Many stages may be observed in the different areas of the same eye. Please Note <1000/mm2 may decompensate after cataract or other intraocular surgery; polymegathism (variation in size) ; polymorphism (variation in shape); Increase in endothelial cell size is proportional to increased corneal hydration

Transmission Electron microscopy shows degeneration of endothelial cells - Vacuolization; Cell membrane disruption; Fibroblast like changes

Differential Diagnosis - guttae producing conditions - Aging; Interstitial keratitis; pseudo guttae (transient edema of endothelial cells- trauma, Intraocular inflammation, Contact lens wear, Toxins, Infections, thermokeratoplasty. These disappear with resolution of the condition; Macular and posterior polymorphous dystrophies; Chanders syndrome (ICE) ; Iridocorneal endothelial syndrome has baten bronge endothelial pattern with overlying corneal edema. *Edema producing conditions* - Aphakic or pseudophakic bullous keratopathy of numerous causes *(Note The posterior collagenous layer differs in structure and collagen content)* ; Congenital hereditary endothelial dystrophy; Non-guttate corneal endothelial degeneration.

Non – Surgical Management Aim To decrease edema and pain- Topical hyperosmotic agents to treat corneal edema - 5% sodium chloride eye drops during the day (especially early morning) ; 5% sodium chloride ointment at night time (to reduce early morning edema) ; Glycerin can also be used but it produces lot of stinging. Dehydrating the cornea by a hair dryer on low or no heat setting held at arms length to reduce edema by increasing evaporation. Antiglaucoma medications or oral carbonic anhydrase inhibitors may help in decreasing corneal oedema of patients by reducing intraocular pressure.Contact lens (varying effects) - A high water content soft contact lens - permits evaporation from lens surface and may improve visual acuity by reducing irregular astigmatism; Hard lens corrects irregular astigmatism but worsens edema and vision as it reduces evaporation from corneal surface; Bandage soft contact lens to reduce discomfort in a patient with bullae (advance and recurrent corneal erosions) corneal edema, avoid long term use as risk of infection). Supportive treatment for ruptured bullae - Soft contact lenses can be useful in cases of bullae formation; Cycloplegics, local antibiotics, and pad and bandage treat the eye for a couple of days; Retrobulbar injection of absolute alcohol is useful to patients with painful, totally blind eyes.

Please note If folds are present in descemet's membrane a contact lens has no benefit in terms of vision.

Surgical Management Failing vision in the presence of epithelial edema and stromal haze, which cannot be treated by the instillation of 5% sodium chloride drops and ointment, necessitates recourse to surgery. A selection has to be made between the following 2 options (1) keratoplasty alone, when

no cataract formation is present, or (2) if cataract is present and adds significantly to visual disability and specular endothelial microscopy results suggest the need for a corneal graft, then a combined corneal transplant, cataract extraction, and lens implant procedure should be performed. Conditions that increase occlusion and decrease tear evaporation eg., ptosis, should be corrected. Total conjunctival flap for patients with limited usual potential due to other causes than corneal edema eg., ARMD (This is suffer for long term than a bandage contact lens). Penetrating keratoplasty (PKP) for vision restoration in patients whose normal day to day activities are affected, has a good prognosis (early surgery in young age, poor vision in fellow eye, high visual requirement). *Anesthesia* - Local anesthesia with 50/50 mixture of 0.75 % bupivacaine and 2% lidocaine and 150 U of hyaluronidase; Anesthesia may be retrobulbar or peribulbar. Good hypotony should be obtained with a mechanical presure device (eg, Honan balloon, Super Pinky ball, mercury bag); After thoroughly ascertaining cardiovascular status, general anesthesia may be used in selected cases; Combined PKP + cataract extraction + IOL triple procedure depends on CCT + specular <1000 cells /mm2 of 800 + clinical and symptomatic visual fluctuation especially morning blue and > 10% variation in corneal thickness between morning and evening central corneal thickness and stromal edema on slit lamp. Severity of cataract

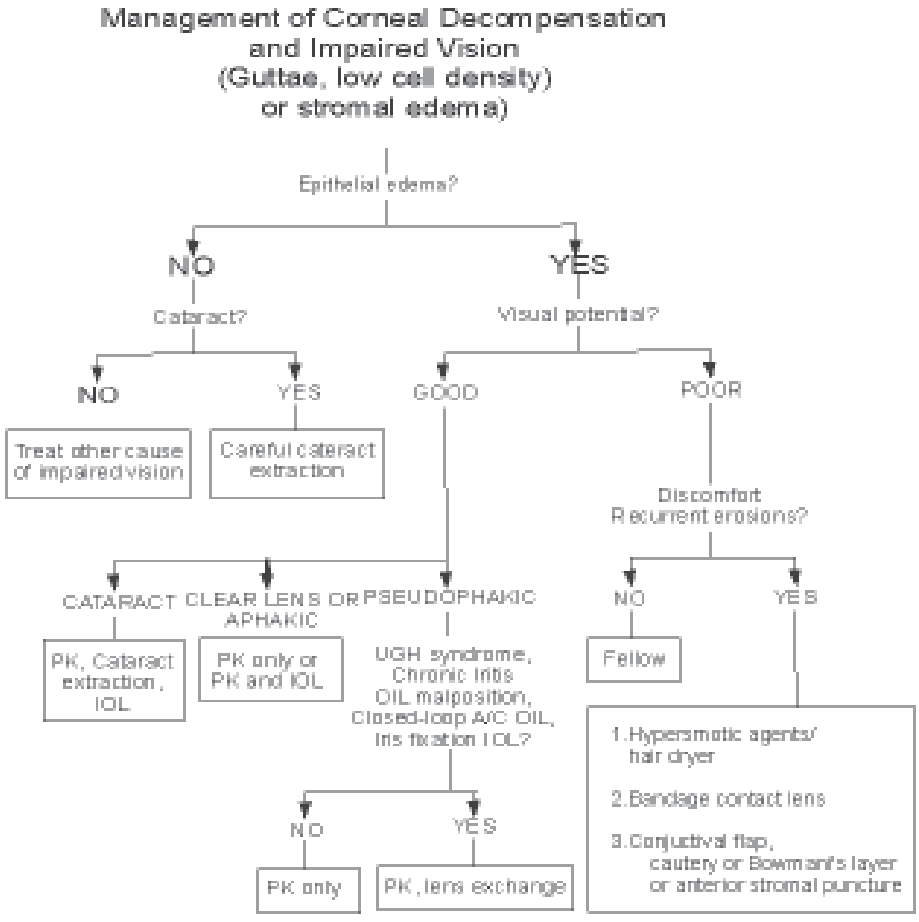


Figure 18-8
Management of corneal decompensation and impaired vision

Disadvantages for individual Procedures - Increased cost and rehabilitation time; Corneal graft more likely to fail; Poor visibility during the second procedure; Difficult to calculate IOL Dower.
Others – Histology **-** In early stages, the focal thickening of the Descemet membrane is similar to those seen in the Hassall-Henle warts of the peripheral cornea. The corneal endothelium appears stretched and thinned over the dome of the excrescences; In advanced cases, a generalized thickening of Descemet membrane is observed. This thickening appears to "bury" the cornea guttata that formed in the earlier stages; In normal corneas, histologic preparations show lamellar separation as an artifact. In the cases of corneal edema, the artifactitious lamellar separation of the lamellae is reduced. Subepithelial bullae fomlation is seen at the anterior corneal surface. In the periphery of the cornea, subepithelial fibrous tissue usually is seen. Intraepithelial cysts filled with cellular debris are seen. Intraepithelial basement membrane formation may occur due to the misdirection of the epithelial cells. Bowman membrane is normal, unless it has been involved in ulcer formation and keratitis, after the rupture of a bulla.

Post Chemical Burns
History - of contact with chemical agent, type and nature of agent, quantity involved, whether eye wash / irrigation was done, associated face injuries, unilateral/bilateral. Details of treatment received whether medical / surgical. Any other significant history.
Clinical Examination - Visual eiuty, Injuries over the face / eylids, Upper lids/both lid involvement, Loufmctival involvement – Limber ischaemia , Modified Rooparhll classification, Presence or Absence of Necrosis, Corneal Clarify / effinthetal defects/ vascularisation of corma, presence or absence of particulate matter, Any symbepharma in old case / harmic burns AE to lens status
Investigations - Fluotoscime staining to see the efithehal defects shirmer test / drug eye / prot rose Bengal staining
Medical Management - Acute Cases (Irrigation; Removal of partials of chemical; Debribem cut; Tear Substitutes; Antibiotics; Antiglaucoma Medication; Cycloplegics; role of cortieosterouds; Ascorbate Citrate
Surgical - Tissue Adhesives; Glass Rod for prevention of rymblopharaous; Bandage contact lens; Luibal stem care transplantation; AMT; Symplepharas Release; Penetrating keralohlasty ; Conjunctival slapping

Diabetic Retinopathy
History - Duration of diabetes, Age of onset of diabetes, Control of Blood Sugar, History suggestive of Neuropathy, History suggestive of Nephropathy, Associated H/O Hypertension, Pregnancy, Heredity
Clinical Examination - Venous dilation; Microaneurysms; Intra retinal haemorrhages- dot and blot haemmorrhages; Hard exudates; Macular oedema - Focal /Diffuse; Soft exudates; Venous beading and loops; Neovascularisation of disc or periphery; Fibrovascular bands; Tractional retinal detachment; Preretinal membrane formation; Vitreous haemorrhage; Posterior vitreous detachment; Iris Neovascularisation.
Investigations - Blood sugar; Kianey function tests . Lipid profile; USG in opaque media to see vitreous haemorrhage(diffuse!organized) & retinal detachment .
Fluoresceine Angiography - Areas of capillary nonperfusion; Distortion of foveal avascular zone; Intra retinal neovascularization; Intra retinal micro vascular abnormalities; Macular oedema- focal! diffuse; CSME; Areas of leaks - Disc! macula/elsewhere.
Differential Diagnosis - BRVO,CRVO; Hypertensive retinopathy; Renal retinopathy; Eales, disease; Sickle cell disease SLE; Radiation retinopathy
Non Surgical Management General Management - Diabetic control and its effect on retinopathy(Diabetic control and complications trial study; Primary and secondary prevention of retinopathy), Antiplatelet Therapy(Use ofNSAID as cyclo-oxygenase inhibitors), Use of Hypo Lipidemic Agents like Lovastatin and Prevastatin and their effect on retinopathy.

Photocoagulation - PRP - Indications (High risk characteristics (HRC)) ; Focal treatment / Macular grid- Indications; Criteria for CSME according to ETDRS; ETDRS Guidelines for follow up treatment after initial PRP; Factors for additional photocoagulation; Technique of additional laser photocoagulation; Size and duration of spots in photocoagulation; Areas to be avoided during photocoagulation; Complications of laser
Cryoablation - Indications; Method and areas where cryo spots are to be applied.
Surgical Management - Vltrectomy - Indications of Vitrectomy in diabetic eye disease; Surgical objectives and techniques; Risk of removal of lens with vitrectomy - Progression of Rubeosis Iridis; How to stop bleeding while doing vitrectomy(Raising bottle height, Bipolar diathermy, Endolaser; Fluid gas exchange; Silicone oil temponade its indications, technique, complications, removal methodology; Indications for fluid gas exchange.

Ptosis
History - H/O Diabetics, ENT Problem, Fever, Trauma, Parotid surgery Etc.
Clinical Examination - Upper Motor Neurone/L.M.N Difference; Bells Pheromone +/-, 6th Nerve involvement
Investigations - ENT check up/CT/Rule out diabetes
Differential Diagnosis - UMN/LMN involvement to be ruled out.
Non – Surgical Management - Ocular Lubricants/ oral steroids.
Surgical Management - Tarsoraphy /Jascial N Decompresssion
Any Other - Prognosis – invariably bad.

MEDICINE
Cerebro-vascular disease
History - Detailed history relating to the event onset, progress, neurological deficit(s); Assessment of risk factors for CVA; If young patient, to evaluate for 'Stroke in young'; Medication/Treatment history
Clinical examination - Vital Signs-pulse, BP, RR, Temp., Eval. Of carotids; Detailed Neurological exam including (Cranial N Plasies – Speech;Pupillary Signs;Motor System Examination); Exam of Heart/CNS; To evaluate for causes of stroke in young;Should be able to identify the Vascular Territory involved Fundus Exam.
Investigations-All invest but specifically-ECG;NCCT(Head);Role of CECT(Head);MRI(Brain);Carotid Doppler; Role of ECHO.
Differential diagnosis-CVA (Conbouc, Haemorrhagic,Thrombotic); In young patients (Aneurysm,AVM); Other Causes (Vasculitis;SOL; Causes of stroke in young)
Management - Immediate M/M; Supportive Care; Specific — Role of Thrombolysis in Thrombotic Events/Infarcts— When , How; M/M of HT in setting of Stroke—How 10w to bring down BP, Any other - Should discuss causes/Risk factors for CVA in elderly patients; Should discuss causes/Risk factors for stroke in young patients; Should be able to identify Vascular territory; Discussion on posterior circulation stroke should be there

Multi-valvular heart disease
History-Detailed history of symptoms– Palpitations, Dyspnoes, PND, Orthopndea, EDEMA, Hospitalizations, Embousations; History suggestive of RHD, other connective tissue disease, IE; Should be able to identify-RT V/s LT sided Valve Lesions, Stenotic V/s Regurgitant Lesions
Clinical examination- Detailed ESP, GPE-Pulse(especially for)- BP, Signs of IE, Evidence of RF, If suspected AR-Look for features of MARFAN'S, Syndrome,JVP, EDEMA; CVS – Detailed, thorough exam of CVS-all areas; Abdomen; Fundus Exam; CNS
Investigations– ECG, Discuss Findings; CXR—Discuss Findings; ECHO—What all can be seen If IE – Blood c/s— How many/when; RF – ASLO/CRP, other anti strept Ab
Differential diagnosis- To give diagnosis as Which all valves are involved list in order of severity; Etiology (? Rheumatic ? MARFANS etc.); Presence/Absence of-Pulmonary A hypertension, Congestive cardiac failure, Arrythmias/Normal Sinus Rhythm, Rheumatic Aaivety, Infective Endo Carditis.

Management - Discuss M/m in relation of; Valve involvement- Conservative, Surgical, Others of BMV; CHF; IE; RF; Emborisation; AF; Special Simulation eg. In Pregnancy discuss prognosis and outcome
Any other – Discuss-Prophylaxis for RF; IE Prophylaxis; Anticoagulation; Digoxcin-Role and Toxicity; M/m of Embolisation in setting of IE; Fungal Endocarditis

Cerebellar disease
History- Detailed History of Onsent, Progression of complaints; Family History – should trace involvement in family for inherited forms of cerebellar disease; Drugs/Toxics – History especially of ; Other Neoplasms – Paraneoplastic involvement
Clinical examination - Detailed neurological exam Especially of – CNS and also spine; Other systems- To be able to identify cause of cerebellar involvement.
Investigations - Role of MR/CT; Discuss findings
Differential diagnosis-Acuteonset-Chroniconset;Symmetrical - Symmetrical;Asymmetrical – Asymmetrical.To discuss D/D according to individual situations.
Management -To identify the involvement as; Degenerative; Inherited; Drug/Toxin related; Infective; Vascular involvement; Paraneoplastic; M/m of individual situation
Any other -To discuss D/D appropriate IE clinical situation eg. Age.

Congenital Heart Disease
History - Onset of Symptoms – childhood, adolescence, adults; Discuss the symptoms; Cynosis – if yes cyanotic spells feeding; Growth and milestones in children; Respiratory infection
Clinical examination-Any compiler eg. Stroke, etc.,Detailed Cardiovascular- Exam, GPE – Sxanosis, Cuilbbing, JVP, EDEMA; Evaluate for other inherited/congenital malformation/disorders.
Investigations – ECG; CXR; ECHO; Polycythemia/Hci; ABG
Differential diagnosis - To reach diagnosis as congenital HD. Cyanotic, Acynotic and then further discuss the individual differential thesis according to the case in hand; Eisenmenger – to discuss in detail.
Management - ISSUEO regarding; M/m of cyanotic spells(in children); M/m of CHF; M/m relating to; Operability; Surgery; Prognosis and Outcome

Short Case
Myopathy - Disease of Muscle/Nerve
History-History of symptoms,weakness especially, Onset, Progress, Prox V/s Distae, Severity, Fasciculations, Atrosphy, MSI Fatigue; To identify cause if possible on history; Paraneoplastic involvement
Clinical examination - Complete physical exam including; Detailed neurological examination; Focus on demonstration of (focus on LMN signs); Refrences-Planter response, Atrophy of MSIS; Skin Exam; Spine Exam
Investigations – Discuss + CPK– total = MSL Enzymes; LDH; NCN; EMG - if NM jn – Discuss tests for sis; MSI Biopsy
Differential diagnosis - Should be able to give D/D of LMN involvement- N,MSI and how to differentiate, NMJ; D/D of individual disorders eg. If muscle involvement –Myopathy,MSI Dystrophy and then give elistology, Myositis
Management-Depends upon clinical situation

Fibro-cavitory lung disease
History- Detailed history of Symptoms –Cough, dyspnoea/breathlessness, expectoration, fever, edema; Past history of TB; Family history of TB
Clinical examination-GPE – Especially - Trachee Position, Gyanosis, Dubbing, Cympladenspalthy, Edema, Detailed respiratory examination
Investigations- CXR; ECG; Role of CI
Differential diagnosis - Discuss complications

Management - Discuss ATT; DOTS DOTS-Plus; MDR TB; ATT induces hepatitis

Pleural disease
History - Detailed history of symptoms; History to EUCIT cause/etiology; History of TB/Past TT with ATT
Clinical examination - Detailed respiratory exam
Investigations - CXR(Pa); Pleural Tap/Thoracentesis; And analysis and interpretation.
Differential diagnosis - To discuss D/D of Pl. Effusion; Discuss the interpretation of PI fluid analysis; Role of serum-pl. fluid gradient; Discuss etiology of pl. effusion
Management - Role of Tule Thoracostomy-,Indications, Procedure, Contraindications,Complications; Role of Pleurodesis-Indications,Procedure

Pulmonary mass / consolidation
History- History/Exam - Detailed History; Physical Examination; Evaluate especially for-Lymph nodes, EVC obstruction, Pupils – Horner's syndrome; Staging if suspected mass lesion
Clinical examination - CXR(Pa) – Discuss radin findings – Lat view; Role of CT(Thorax); Broncho Scopy
Discuss - Etiology of lung masses; Classification – Grading, Staging; Complications of Ca Lung-Pulmonary, Extra – especially paraneoplastic manifestations, SVCobstructionCranial Nerve Palsies

Ascites
History - Symptoms due to oscites due to underlying disease; Detailed history
Clinical examination - GPE – Cymphadenopathy-Edema, Facial Puffiness, Vital Signs; P/a – eliciting signs of oscites
Investigations - Especially – ascitie fluid analysis; SAAG – Wgh SAAG-10No; Causes of Ascites – investigation depend upon etiology
Management - Depends upon cause

Case - Nephrotic Syndrome
History- Swelling of face, hands and legs; Frothy urine; Diabetes mellitus; History towards Syhilis, Malaria, Tuberculosis and HIV; History of Cancers- Breast cancer, Lung cancer and Hodgekin's lymphoma; Collagen vascular diseases(Lupus)- skin lesions, oral ulcers, joint pains, prolonged fever; Blood transfusion- Hepaitis B Hepatitis C and HIV; Drug history-NSAIDs, D Pencillamine, heroin, Alternative and complimentary medicines (Heavy metals); Personal history- STD- Syphilis, HIV and Hepatitis; Family History- Renal diseases
Clinical Examination - General Exam-Anasarca, Malar Rash; Swelling of tissues around the eyes (periobital edema 0); welling of feet and ankles; Scrotal edema; Dysnoea (fluid overload- pulmonary edema) ; Dry Skin; CVS- Pericardial effusion; RS- Pleural effusion ; ABD- Ascites, genital edema; CNS- Diabetic retinopathy and neuropathy
Investigations - Baseline; Blood sugar; Urinanalysis- protein cast, lipid cast; 24 hour urinary protein ->3.5gm/day; LFT- Serum albumin< 3.0 gm/dl; RFT; Lipid Profile(increased cholesterol and TGL) ; Low Iron and Vitamin-D levels; Special-ANA,Anti dsDNA, Hbsag, HCV, Serum protein electrophoresis; ASO titre; USG-Enlarged kidneys- Dimensions; Renal vein thrombosis; Renal biopsy
Complications - Infections- Peritonitis, Cellulitis and Sepis; Hypercoagubality- DVT, Pulmonary Embolism and Renal Vein Thrombosis; Hyperlipedemia; Chronic Kidney disease; Growth delay in children
Differential Diagnosis - Nephritic syndrome; Cardiac Failure; Hepatic Failure; Hypothyroidism; Cushings syndrome; In children Fluid overload mistaken as lung allergic conditions
Treatment - If systemic causes of nephtric syndrome is present therapy should be instituted for the systemic disease; Diet; Low Protein; Low Salt-1-2 gms; Resticted fluid; Corticosteroids to reduce

the proteinurea and edema; Salt free Albumin- to restore the blood volume; Diuretics- to maintain fluid balance and caution to avoid hypovolemia; Cyclophosphamide when bot responding to steroids; ACE inhibitors, ARB reduce the rate of progression of the renal disease
Complications- Edema- Diuretics; Dyslipedemia- statins; Thromboembolic phenomena- If renal vein thrombosis- Heparin; Vitamin-D supplementation

Extra Pyrammodal Disorder
Extra Pyramidal disorders are Associated with abnormalities in basal ganglia constituted by five paired nuclei namely candate nucleus, putamen , globus pallidus , sub-thalmic nuclei and substantia nigra.
The common movement disorders are- Parkinsonism; Tremors; Chorea; Athetosis; Dystonia; Hemi ballismus
History - H/O of Rheumatic fever, past H/o encephalitis (viral), H/o epilepsy. ; H/o drug intake (methyldopa, phenothiazines, O.C. pills etc.) ; Family history of similar complaints; Psychological history; H/o exposure to toxics like carbon monoxide, manganese etc. ; Duration and progress of the disease. ; H/o associated Jaundice or any evidence of Chronic Liver disorder (for Wilson's disease). ; H/o vasculitis, arthralgia, low grade fever(for S.I.E.) ; Any aggravating or relieving factors.
Clinical Examinations - Most important in a case of Parkinsonism, look for the following features- Lack of facial expression(Mask-like) ; On the face; Note tremors, absence of blinking, dribbling of salvia, glabellar- tap, ocular movements , for supra nuclear gaze palasy, feel for a greasy or sweaty blow (due to autonomic dysfunction) ; Speech— Monotonus, soft , poorly articulated and faint. ; Ask the patient to write(Look for micrographia) ; Paucity of movements; Ask the patient to walk, turn quickly and stop and restart. (Note the difficulty in starting, stuffing ,freezing and festination) ; Look for propulsion and retropulsion (with care) ; Resting tremors with the arms relaxed ("pill rolling" movement)- on finger- nose testing, the resting tremors disappears. ; Test for wrist tone , for cog-wheel or lead- pipe rigidity.
Examination of a patient with with other extra pyramidal movement disorders - Higher mental functions; Chorea- gremacing of face, jerking of head. ; Examination of the patient's arms for chorei from movement, Athetosis And hemi –bellimus and tremors (description of the movements of the arms is very important. ; Shake hands with the patient "Milk-maid grip"- lack of sustained grip. ; Ask the patient to hold hands out and look for the classical choreic posture. (Finger and thumb hyper restricted and wrist flexed due to hypotonia) ; Reflexes; Conjuctival injection (ataxia telengec tasia) ; K.F. ring (wilson's disease) ; Examination of cardio-vascular system
Investigations - Complete Haemogram; L.F.T. ; Detailed Opthalmic examination; ASLO titres; Work up for SLE; Thyroid function tests if autoimmune thyroidities is suspected; Serum copper and ceruloplasmin levels. ; L.P. ; CT Scan Brain; MRI S can of Brain; EEG if required; Estimation of drug levels(if required) ; ECHO
Treatment – Parkinsonism - Symptomatic, supportive and palliative; Physical therapy and psycho-therapy; Requirement of lifelong medications; Treatment programme to be personalized; Medical treatment for compensated phase and decompensated phase.; Medications under evaluation; Role of surgery
Treatment of other Movement Disorderso-Drug treatment of Tremors; treatment of Dystonia; Role of surgery; Use of botulinum toxin

Rheumatological Diseases
The diagnosis and assessment of articular disease are based on the clinical processes of history-taking and examination. Blood tests and radiological investigations are often useful, and may be essential , but they cannot replace careful history- taking and examination of the patient. Unnecessary investigations are expensive , may cause anxiety to patients, and if taken out of clinical context, lead to over-diagnosisand over-treatment. The initial history and examination may lead immediately to the diagnosis of a specific disorder, such as rheumatoid arthritis, or may indicate a pathological process such as vas ulitisor synovitis, or may suggest that the condition is self-limiting or non-

pathological. Appropriate assessing the patient and the results of the investigations, the physician should be able to offer the patient an explanation of his symptoms in terms the patients can understand and formulate with the patient a plan for treatment.

History - The patient should be asked first about the nature of the main problem and it's impact on their daily life. Factor's such as the patient's age, sex, race and employment are often relevant. The mode of the onset of the symptoms and any precipitating factors such as trauma should be asked about. The development of symptoms, their evolution over time, and pattern of remission and relapse are also important. The effect of previous therapies, the patient's compliance with them and any adverse reaction are important in planning future treatment.

Pain - Pain is a cardinal symptom of rheumatic disease and should be enquired about in detail.

Site - It is important, and sometimes surprisingly difficult, to establish whether the pain arises from a joint, muscle, bone or other tissue, in general, pain arising in a joint is worse when the joint is moved, whereas pain arising elsewhere will not be affected by joint movement. Associated symptoms such as joint swelling suggesting inflammatory joint disease or paraesthesiae and weakness, suggesting a neurological cause for the pain, may also help. It is helpful to ask patients to demonstrate on their own bodies the site where pain is felt.

Quality or character - Pain is notoriously difficult to describe in words, but some features are very useful. Pain, which is worse at night, disturbs sleep and is unrelenting and unaffected by position, is strongly suggestive of serious disease such as malignancy and requires urgent investigation. The pain of malignancy is usually less acutely severe than the pain of an acute inflammatory condition such as gout. The severe shooting pain of nerve root entrapment, which travels down a limb or around the trunk, is often characteristic enough to be diagnostically useful. Relieving and exacerbating factors- Joint pain due to mechanical problems without inflammation is typically worsened by movement and rapidly relived by rest. Inflamed joints are often painful at rest and somewhat better after a few minutes of use. Patients with active inflammatory joint disease are often troubled by severe night pain and stiffness.

Stiffness and restriction of movement - Stiffness is highly subjective and variable sensation of tightness and resistance to movement. Patients may equate stiffness with fatigue, pain, weakness, loss of range of movement or swelling. Most patients with joint pain experience an initial, shot-lived sensation of stiffness after immobility. This needs to be differentiated from the severe stiffness experienced with patients with inflamed joints when they begin to move after sleep or rest. This stiffness typically " wears off"after minutes or hours, and patient can often quantify this time quite accurately. The duration of "early morning stiffness" on first arising after the night's sleep may be used to assess the changing severity of inflammatory disease. A duration of more than 30 minutes of morning stiffness remains one of the American Rheumatism Association criteria for rheumatoid arthritis.

Swelling - Patients may notice swelling, but it is unwise to assume that what the patient is describing is synovial swelling or joint infection unless the description is very clear, or there is evidence of synovitis or effusion on examination. Stiffness , paraesthesiae from nerve entrapment, malallignment, discolouration or pain itself may lead the patient to a perception of swelling which is not confirmed by examination. If swelling or deformity is present, it is necessary to determine whether it is due to fluid, soft-tissue or bone.

Family History - The family history may be useful; a history of similar problems in other family members may give a clue to HLA B27- related arthropathies, psoriatic arthritis, gout or some autoimmune rheumatic diseases.

Systemic symptoms - Symptoms of systemic illness such as fever, weight loss and malaise may be due to active inflammatory joint disease, but should also alert the doctor to the possibility of an underlying infection, malignancy or tuberculosis, may be relevant. Non- articular symptoms associated with joint disease such as ankylosing spondylitis should be specifically asked for ; patients will seldom associate a history of a painful red eye; skin rash or urethral discharge with their painful swollen knee. Tact, and appropriate explanation, are needed when enquiring about sexually transmitted diseaseand genital symptoms, but failure to ask the relevant questions may result in misdiagnosis.

Essential points in the history - * Pain -Onset; site and radiation; referred pain character effect of movement and rest night pain and unremitting pain * Stiffness morning- duration—, Immobility stiffness and gelling, Swelling and deformity, Disability and handicap, Systemic illness, extra-articular features, sleep and depression, Social and family history.

Patients should be asked specifically about sleep disturbance depression, which are commonly features of chronic painful conditions. Fatigue is a characteristic symptoms of autoimmune disease such as reheumatoid arthritis and systemic lupus erythematosus.

Examination of the locomotor system

Swelling - Swelling around a joint may be cause by fluid, as in an intra-articular effusion or effusion into an inflamed bursa, by soft tissues such as synovium or extra-articular fat pads, or by bony enlargement as in the osteoarthritis. Subluxation, as the metacarpophalangeal joint, may also give an impression of swelling. Intra-articular fluid produces a swelling defined by the margins of the joint capsule. The synovial lining layer is normally too thin to be palapable but the thickened inflamed synovium in chronic synovitis such as rheumatoid arthritis may have a “boggy” consistency and be easily felt. Effusion without synovial thickening, where the joint line can be clearly felt , is usually due to trauma or osteoarthritis. Fluid can often be shifted from one area of the joint to another by compressing one side of the swollen joint.

Tenderness, redness and warmth - Inflammed joints are tender along the joint line. Paplation may also localize tenderness to the sites of attachment of tendons or ligaments, bursae, muscles or fat pads . Inflammed joints are usually warmer than the surrounding tissues. Redness over the joint is a sign of intense inflammation, usually due to gout, pseudogout or infection.

Limitation of movement, deformity and instability - Limitation of a movement is a common symptom of joint disease. Older people have less mobile joints than the young, women are generally more flexible than men and joint mobilityis greater in some races than others. It is important to develop a feeling for the normal for a particular race, age and sex so that generalized hypermobility, as well as restriction of movement at specific joints may be detected. Limitation of movement of a joint may be due to swelling, soft-tissue contracture, tendon rupture, muscle weakness, joint subluxation or dislocation as well as pain , the commonest cause. The extent of loss of function resulting from limitation of movement should be assessed. The pattern of loss of movement may indicate whether it is due to inflammation of the joint itself or to another cause such as rupture of tendon. There may be a greater range of passive than active movement. This is commonly due to pain but may be due to muscular weakness or tendon rupture. Deformity of a joint may result from contracture of the capsule or surrounding soft tissue , subluxation, ankylosis in an abnormal position or bony or soft tissue swelling. Deformity usually results in some loss of function and placing of the abnormal stresses on the joint. A joint is said to be unstable if a greater than normal range of passive movement is possible. If there is partial loss of congruity of the joint surfaces the joint is subluxed; if there is complete loss of cartilage-to-cartilage contact the jont is located.

Look - Position in which the joint is held; Swelling; Deformity; Associated tissues-skin changes, muscle wasting nails. Feel – Warmth; Tenderness; swelling; bone, soft-tissue(synovium or other 0 or fluid? ; Crepitus; soft-tissue(synovial) or bony? Move - ACTIVE MOVEMENT; assess the range, rhythm and ease of movement the patient can achieve. PASSIVE movement; Compare the range of movement when you move the joint Assess the stability of the joint. Function - Assess the degree (or loss) of useful function eg; of the hand or arm.

Extra-articular manifestations of common rheumatic diseases -

Skin -Nodules - Rehematic fever, rheumatoid arthritis, gout, Hyperlipidaemias;Rash - Systemic lupus erythematosus, rheumatic fever, Still’s, Disease, psoriasis, Reiter’s dermatomyositis, cutaneous Vasculitides, drugs Lyme arthritis, viral infection, Kawasaki’s disease, HIV – related arthritis; Erythema nodosum - TB, drugs, streptococcoal sore throat, Behcets, sarcoidosis, fungal infections, idiopathic, inflammatory bowel disease, leprosy (erythema nodosum leprosum); Other panniculitis - Inflammatory bowel disease, SLE, Weber-Christian, Disease, malignancy;Raynaud’s - Progressive systemic scelrosis, polymyositis – dermatomyositis, SLE , rheumatoid arthritis, vasculitis; Sclerodactyly -

Progressive systemic sclerosis, CREST, overlap. Syndromes; Leg ulcers - Fetly's syndrome, behcet's systemic vasculitis, inflammatory bowel disease;Livedo reticularis - SLE, polyarteritis nodosa;Hairloss - SLE, drugs (cytotoxic) hupothyroidism; Ski pustules - Gonococcaemia, Behcet's; Heberden's nodes - Primary osteoarthritis; Bouchard's nodes - Primary osteoarthritis

Oral cavity-Aptheous ulcers - Behcet's inflammatory bowel disease; Superficial painless; Ulcers - Reactive arthritis; Dry mouth - Sjogren's syndrome – primary, secondary

Ocular - Conjunctivitis - reactive arthritis, relapsing polychondritis; Scleritis- episcleritis — Rheumatoid arthritis, relapsing polychondritis; Anterior uveitis — Juvenile chronic arthritis, seronegative spondyloarthritis; Iritis - Spondylarthritis, Behcet's; Dry eyes — Sjogren's syndrome

Summary of the Screening Examination of the Joints -Gait - Watch the patient as he stands, walks and sits. Pain, stiffnessor deformity of the lower limb joints or back may lead to a limp. An abnormal gait, a limp, or abnormal posture may indicate disease in the spine, hip, knees or feet. With the patient sitting, examine neck- range of movement and lymph nodes, elbow- range of movement And swelling, wrist-range of movement and swelling, hands- for skin, nails, joint swelling, deformity, pinch and power grips.With the patient lying, examine hip- rotation, knees- for effusion, range of movement, feet- for deformity and MTPJ squeeze pain.With the patient lying, examine Spine- for abnormal contour and range of lumbar movement.

Investigations- Nonspecific Tests-The tests included are completed blood counts and ESR, routine urinalysis, total proteins with albumin and globulin levels, C-reactive protein (CRP) and a host of acute-phase rectant (APR). The time profile of each APR is different, e.g. CRP values can change within 24 hours while ESR takes a few days. This difference allows judicious use of the tests. Their serial estimation helps to monitor disease activity between clinical activity and ESR. ESR should be measured by the Westergren method. High ESR is a feature of inflammatory rheumatic disorders. Very high ESR(>100mm in 1st hour) is commonly seen in infections (TB), rhematic diseases (SLE, Still's disease) and malignancies (myeloma, leukaemias, lymphomas). CRP estimation is not required routinely. In normal healthy persons the levels are complement pathway is activated. Normal C_4 and lowered C_3 levels indicate activation of the alternate pathway. Genetically determined low levels of individual complement components are sometimes seen. These individuals may develop SLE or other rheumatic diseases.

Immunoglobulins - Rise in immunoglobulin levels is a nonspecific findings and hence is the routine estimation is not essential. Multiple myeloma and agammaglobulinaemia are the two main indications fro estimation of individual immunoglobulins. The same can be said of routine protein electrophoresis. Other immunological tests - Circulating immune complexes are of research interest. However, in routine practice their detection has not been found to be of great help. Further, no single method has found universal acceptance. Cryoglobulins, PEG precipitation, Clq binding and Raji cell assay are some of the better known.

Non-Surgical Management –Non-steoridal anti-inflammatory drugs;Arthoplasty; Anti-cytokine agents; Immunosuppressive therapy

Surgical management-Open on arthroscopic synovectomy; Reconstructive hand surgery; Arthroplasty; Total Joint Replacement; Reconstructive hand surgery;

Thyrotoxicosis

What is the difference between the terms thyrotoxicosis and hyperthyroidism?

History - Male- Female; Age; Stress? ; Smoking; Pregnancy; Hyperactivity/ irritability; Heat intolerance/ Sweating; Palpitations; Weight loss with increased appetite; Diarrhoea; Polyuria; Loss of libido

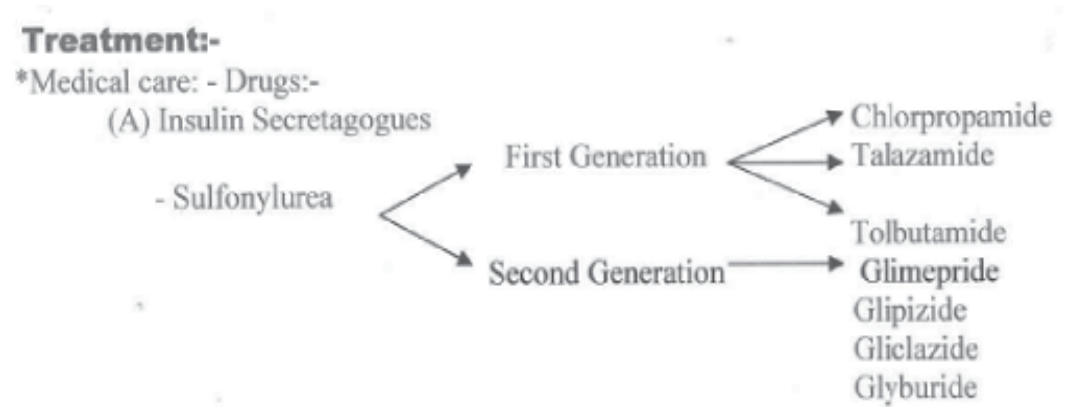
Clinical examination - Tachycardia, AF; Tremor; Goiter; Muscles wasting, proximal myopathy without fasciculation,chorea; HPP; Eye signs; Gynaecomastia; Skin 'changes'/Thyroid dermopathy; Scoring of orbital changes; NO SPECS scheme

Investigations-TSH, uncombined TSH, uncombined T3; TPO antibiotics; TBll or TSI measurement; Radionuclide scar; Liver function tests; S. Ferritin levels; Haemogram/ PBS for microcytic anaemia & thrombocytopenia

Differential Diagnosis-Nodular thyroid disease; Destructive thyroiditis; Ectopic thyroid tissue; Factitious thyrotoxicosis; TSH-secreting pituitary tumor ; Panic attacks, mania; Phaeochromocytoma
Management-Antithyroid drugs; Surgery-subtotal thyroidectomy —Thyrotoxic crisis, Complications of surgery.

Diabetes Mellitus

Definition- The national diabetes data group &WHO have issued diagnostic Criteria for D.M. - Symptoms of Diabetes plus random blood glucose concentration ≥ 200mg/dL or Fasting plasma or glucose≥126mg/dL or Two hour plasma Glucose≥ 200mg/dL during an oral glucose tolerance test.
History - Polyuria, Polydipsia, Polyphagia, weight loss; Blurred vision, lower extremity paresthesias, yeast infection, particularly Voluovaginitis in women, balanitis in men; Obese patients; Patient first degree relative with type 2 diabetes mellitus; Patients with hypertension; Patients with high triglyceride or HDL- Cholesterol <35mg/dL. ; Polycystic ovary disease
Examination - Obesity; Anthropometric data — Height, Weight, Waist-hip ratio, Fat fold thickness; Skin Changes; Hair loss; Prominent veins; Callus formation; Crackers and fissures; Ulcers in the foot and deformities; Peripheral pulses; Blood pressure recording (both recumbent and standing) ; Auscultations for bruits over carotid and femoral arteries; Neurological examination — cranial nerves, Deep tendon reflexes, Autonomic function
Differentials - Diabetes incipidus; Insulin resistance; Obesity; Maturity onset diabetes mellitus; Latent autoimmune diabetes of adults(LADA) ; Stein- Leventhal syndrome
Investigation - Fasting plasma glucose (fpg) greater than or equal to 126MG/dL or random glucose greater than or equal to 200mg/dL and classic symptoms polyuria, polydipisia, polyphagia, weight loss; Hemoglobin Alc (HbAlc or Alc) or glycosylated hemoglobin (GHb) ; Screening urine microralbumin measurements is recommended; Measuring insulin or C-Peptide Concentrations; Antibodies to insulin, islet cells, or glutmatic acid decarboxylase (GDA)



- Non- Sulfonylurea
- Repaglinide
- Nateglinide

(B) Biguanides – Metformin; (C) L.glucosidace inhibitor –Acarose, - Meglitol; (D) Thiazolidinedione – Rosiglilatazone, Pioglitazone
Ii1sulin— (A) Short Acting (Lispro, Insulin Aspart, Regular); B) Intermediate acting (NPH, Lente); (C) Long Acting (Ultralente, Glargine) ; Complication monitoring; SMBG; Surgery— - Whole pancreas transplantation

Diet-Weight reduction (in obese); Hypocaloric Diet; Total calories between 1000-1200 kcal/ day; For obese- 20 kcal/kg ideal weight; For normal adults (sedentary)- 30 kcal/kg ideal body weight; for normal adults(manual worker) and growing children 40kcal /kg ideal; body weight; Carbohydrate- 50-60 % of total calories; Fibers - 25 gm of fibres per 1000 kcal; Proteins- 25-30% of total calories; Fats - 25-30% of total calories

Exercise - Isotonic exercise like brisk walking, swimming or cycling are recommended; Aerobic exercise for 30-45 minutes/ day, 5 times per week should be advocated; Exercise regimen should start with warm up stretching for 10 minutes aerobic exercise for 30- 45 mintues, and cool down streching for 5-10 minutes.

Hypoglycemia

History- Tremor; Sweating; Anxiety; Pallor -Nausea; Shivering; Palpitation; Impaired concentration; Confusion; Inappropriate behavior;Difficulty in speaking; Aggression or non- cooperation; Focal/ generalized seizure; Hunger; Weakness; pigastric discomfort; Blurred Vision.

Examination-Tachycardia;Increased Pulse pressure; Focal neurological deficit including transient nemiplegia; Drowsiness progressing to coma; Permanent neurological change, if prolonged hypoglycemia.

Grading- - Clinically, hypoglycemia may be usefully graded as follows

Grade 1— Biochemical hypoglycemia in the absence of symptoms; Grade 2— Mild symptomatic- treated successfully by the patient; Grade 3— Severe- assistance required from another person; Grade 4— Very severe- causing coma or convulsion.

Differential - * Nonketotic Hypersomolar State; * Diabetic Ketoacidosis; * Metabolic encephalopathy

Invetigation - *Random blood sugar; *ABG; * Chest x rays

Management - In type 1 OM

Grade 1-2 Hypoglycemia— 2-4 Dextrose tablets; 2 tsfSugar (10 gm), honey or jam (ideally in water); A small glass of carbonated sugar- containing soft drink.

Grade 3-4 Hypoglycemia— Buccal glucose gel—Proprietary thick glucose gel (e.g. Hypostop) Or honey, can be smeared on the buccal mucosa (variable efficacy) ; Intravenous glucose— Administer 25ml of 50% glucose (or 10Oml Of 20%Dextrose) into a large vein, ideally after cannulation; Glucagon- - 1 mg can be given I. V , SIC or I.M

In type 2 DM - Intravenous dextrose — Mandatory continuous infusion of 5% or 10% may be required for several days; Diaz oxide; Hydrocortisone; Glucagon; Mannitol — to reduce cerebral odema; Octriotide- - for prevention of sulphonylurea - induced hypoglycemia. However, clinical experience is very limited.

Diabetic Ketoacidosis

History – Nausea; Vomiting; Thirst, Polyurea; Abdominal Pain; Shortness of breath; Confusion, Drowsiness, Coma; Acute weight Loss; Generalized muscular weakness; Visual Disturbances; Muscular Cramps.

Physical Examination-Tachycardia; Dry Mucous Membranes, Reduced Skin turgor; Dehydration, Hypotension; Tachypnoea, Kussmaul Respiration/Respiratory distress; Abdominal Tenderness; Lethargy, Obtundation, Cerebral Odema, Coma.

Differential Diagnosis-Alcoholic Ketoacidosis; Acute Appendicitis; Hyperosmolar Hyperglycemic Nonketotic coma; Salicylate Toxicity; Hyponatremia; Hypothermia; Lactic Acidosis; Metablic Acidosis.

Investigation-Random Blood Sugar; Urine for Ketones; ABG; S. Electrolytes (K+, Na+, Mg++, Cl-, Bicarbonate, Phosphate) ; Acid-base status-PH, Hco3-, Pco2, â-Hydroxybutyrate; Renal Function test; Blood Culture; Chest X-Rays; ECG

Management-Fluids and Electrolytes - Volumes- - lL/h X 3, thereafter adjust according to requirements; Fluids- Normal Saline (150mmol/L) is routine, ypotonic (“Half- normal”) Saline (75 mmol/L), if serum

Sodium exceeds 150mmol/L, 5% Dextrose 1 L 4-6 hrly when blood glucose has fallen to 15mmol/L; Potassium-No potassium in first 1L unless initial plasma potassium <3.5 mo1/L. Thereafter, add 40mmol/L KCL; 3.5-5.5 mmo1/L, add 20mmol KCL; > 5.5 mmo1/L, 40mmol KCL; severe hypo kalemia may require more aggressive KCL replacement; Insulin—1. By continuous intravenous infusion- - regular insulin is administrated as I.V(0.1 U/Kg) or 1.M (0.4U/Kg), then 0.1 U/Kg/hr continuous I. V infusion; increase 2-10 fold if no response by 2-4hour; Administer intennediate or long acting insulin as soon as patient is eating. Allow for over lap in insulin infusion and subcutaneous insulin injection.

Other Points - Search for and treat precipitating cause (e.g. infection, MI) ; Hypotension usually responds to adequate fluid replacement; CVP monitoring in elderly patients or if cardiac disease present; NG tube, if conscious level impaired to avoid aspiration of gastric content; Urinary Catheter; Continuous ECG monitoring may warn of hyper or hypokalemia; ARDS mechanical ventilation (100% 02; IPPV) avoid fluid overload; Mannitol (up to 1 gm /kg I. V) if cerebral odema suspected; Meticulously updated clinical & biochemical record using a purpose designed flow chart.

Hyperosmalar Coma

History- Elderly with known history of type 2 DM; Polyuria; Weight loss; Diminished oral intake that culminates in mental confusion, lethargy, and coma; Prior hospitalization for hyperglycemia; Increasing thirst with polyuria, polydipsia, and weight loss; Drowsiness and lethary—Delirium, Coma, Seizuers, Visual/Disturbance; Clues to underlying DM- Needle pricks or calluses on finger tips, Obesity, Aonthosis nigricans; Diabetic dermopathy; Necrobiosis on the pretibial surface; lower extremety infections,(e.g. cellulites , carbuncles) ; Balanities; Valvovaginitis; Thrush; Gingivitis; Tachycardia; Hypotension; Seizures, Hemiparesis; A positive Babinski sign; Myoclonic jerks; Change in muscle tone; Nystagmus, diplopia and altered mental status

Differentials-Diabetes insipidus; Diabetic Ketoacidosis; Myocardial Infarction; Aphasia; Pulmonary Embolism

Investigation-Plasma glucose; Arterial blood gases (pH,Pco2,Hco3-,K+,Na+); Plasma ketones; Serum osmolality and calculated serum osmolality; Urinalysis; Plasma Electrolytes; Calculated Anion Gap; Creatinine and BUN; Complete blood count and differential; Creatine Kinase (Rhabdomyolysis) ; Chest radiograph; CT scan of the head; Electrocardiogram

Treatment-Intravenous fluid hydration and electrolyte hemoeostasis; Corrections of hyperglycemia; Treatment of underlying diseases; Cardiopulmonary monitoring; Neurological Monitoring

Diabetic Neuropathy

History-Sensory Symptoms— Negative or Positive, Diffuse or Focal; Negative Sensory Symptoms are feelings of numbness or deadness; Loss of Balance; Especially with eyes closed;Painless injuries;

Positive Sensory Symptoms may be describe as Burning—Pricking pain, Tingling Sensation,Pins & Needles Feeling, Aching, tightness, Hypersensitivity to touch; Motor Prombles— Distal, proximal, More focal weakness, Fine hand coordination and difficulty with tasks, Foot slapping and toe scuffing or frequent tripping, Weakness, Limb weakness, Difficulty climbing up the stairs and getting up from a seated or supine position, falls; Autonomic Symptoms— Dry skin due to lack of sweating or excess defined areas, Poor dark adaptation, sensitivity to bright light, Cardiovascular postural hypotension lightheadedness, fainting,Urinary (Urgency, incontinence, dribbling),Gastrointestinal (nocturnal diarrhea, constipation, nausea, or vomiting), -Sexual (Erectile impotence and ejaculatory ability to reach sexual climax in woman)

Examination - Symmetrical or Asymmetric neuropathies involved, * Symmetric polyneuropathies- - multiple nerves diffusely and symmetrically involved; Distal symmetric polyneuropathy, Small Fiber neuropathy -Diabetic autonomic neuropathy, Diabetic neturonathic cachexia; Asymmetric neuropathy- - Single or multiple cranial mononeuropathies—Cranial Mononeuropathy, Somatic Mononeuropathy, Diabetic Polyradiculopathy, Diabetic radiculoplexopathy, Chronic inflammatory dymyelinating polyneuropathy.

Physical - Loss of ankle jerks or loss of vibratory sensation over the great toes, Weakness of small foot muscles.
Differentials-Alcohol(Ethanol) related neuropathy; Chronic Inflammatory Dymyelinating Polyradiculoneuropathy - Nutritional Neuropathy; Sarcoidosis and Neuropathy; Thyroid Disease; Toxic Neuropathy; Uremia Neuropathy; Vasculitic Neuropathy
Investigation -Complete blood count (CBC); Complete metabolic panel (Electrolytes and liver function panel) ; Vitamin B-12 and folate levels; Thyroid-stimulating hormone and thyroxine; Erythrocyte sedimentation rate; Serum protein electrophoresis with immunofixation electrophoresis; Antinuclear antibody; Rheumatoid factor; Paraneplastic antibodies; Elevated hemoglobin Alc levels>8%; MRI of the cervical, thoracic, and lumbar regions; Electro physiologic studies(Electromyography and nerve conduction studies).
Treatment-Medical care - General aspects of treatment; Education on foot care; regular foot examinations; Current treatments for pain-Tight and stable glycemic control is probably the most important, Gabapentin, Pregabalin, Topical lidocaine, and duloxetine, Phenytoin, lamotrigine, and opioids, Topical therapy with capsaicin;Treatments for autonomic dysfunction - For Erectile impotence, Papaverine, Sildenafil; Glycopyrrolate; Aldose reductase inhibitors (e.g., Alrestatin, sorbinil, tolrestat) ; Alpha-lipoic acid; Gamma- Linolenic acid; Nerve growth factor(NGF)

Diabetic Nephropathy
History-History of Diabetes; Passing of foamy urine; Otherwise unexplained proteinuria in a patient with diabetes
Examination -Foot edema secondary to hypoalbumineia; other associated disorders such as peripheral vascular disease; Hypertension; Evidence of diabetic retinopathy after funduscopy or fluorescein angiography; Peripheral vascular occlusive disease * Evidence for diabetic neuropathy; Evidence for fourth heart sound during cardiac auscultation * Nonhealing skin ulcers/osteomyelitis
Differentials - Multiple Myeloma; Nephritis, interstitial; Nephrosclerosis; Nephrotic Syndrome; Renal artery stenosis; Renal Vein Thrombosis; Renovascular Hypertension
Investigations- Urinalysis; Microalbuminuria; 24- hour urinalysis for urea; Microscopic urinalysis; Renal ultrasound- Kidney size, Obstruction, Echogenicity studies; Serum and urinary electrophoresis; Renal biopsy
Treatment-Medical care— Glycemic control; Antihypertensive treatment; RAS inhibition -ACE..I & ARB; Specific therapies- - includes modification &/or to treatment of risk factors, Peritoneal dialysis, Hemdialysis, CAPD, Continuous Renal Replacement therapy ((A) Continuous arteriovenous hemodiafittration with or without dialysis, (B) Continuous veno-venous hemodaifiltration with or without dialysis)) ; Surgical —Renal replacement therapies, Kidney pancreas transplantation, A. V fistula

Diet - ADA suggests diets of various energy intake (caloric values) ; With advancing renal diseases, protein restriction of as much as 0.8- 1 gm/kg/day may retard the progression of nephropathy.
Activity - No restriction in activity, unless associated complication of diabetes like coronary artery disease or peripheral vascular disease.

Ophthalmologic Complications
History - Transient disturbance of refraction; Gradual loss of vision - suggestive of maculopathy or cataract; Sudden painless loss of vision-vitreous hemorrhage. Retinal arterial & venous thrombosis may also occur in diabetic patients; Appearance of 'floaters'- possible small/recurrent vitreous hemorrhage; Chronic pair & redness- rubeosis & secondary glaucoma; Field defects and impaired night vision.
Examination-Visual acuity, maculopathy, cataract, glaucoma; Fundus Examination — Background retinopathy, Pre-proliferative retinopathy, Proliferative retinopathy, Advanced diabetic eye disease, Maculopathy

Differential Diagnosis- Branch retinal vein occlusion; Central retinal vein occlusion; Ocular ischemia syndrome; Retinopathy, haemoglobinopathy; Sickle cell disease.
Investigation - Blood sugar; Fundus, Slit -Lamp Examination; Ultrasound Eye, Flurescein angiography (for macular edema)
Management-Background retinopathy – Explanation, Search for other complication, Review of glycemia control, Proliferative retinopathy , Laser Photocoagulation (pan-retinal photocoagulation) ; Advanced diabetic eye ds (Retinal detachment owing to fibrin traction, Rubeosis iridis (new vessels on the iris), Pan- retinal photocoagulation, Surgical vasectomy – Enucleation; Maculopathy, Photocoagulation,Control of hypertension

Diabetic Foot Disease & Perepheral Vascular Disease
History-Intermittent claudication; Rest pain; Leriche Syndrome (buttock & leg claudatcation, erectile impotence as a result of major stenosis of the aortofemoral vessels);Foot ulceration- past or present; Smoking habits; Family history of atherosclerotic disease; Other manifestations of atherosclerosis- i.e of MI, TIA, stroke and lipid status.
Examination-Palpation of peripheral pulses; Auscultation for bruits; Trophic changes in skin; Limb temperature (Limb is pale & cold in the presence of significant ischemia but may appear red with critical impairment of blood flow ('Sunset Foot'). ; Buerger's Sign; Examination of ulcer- Base, Edge, painful or Painless; Dry and warm; Callus formation; Gangrene
Differential Diagnosis -Cellulites; Deep skin and soft tissue infections-Gangrene; Acute Osteomyditis; Chronic Osteomyelites.
Investigation- Complete Haemogram; Blood Sugar; Pus culture & Sensitivity; X-rays feet & joints; Doppler Studies; Duplex scanning; Oxygen tension; Angiography; Nuclear medicine bone scans; MRI of foot, Bone biopsy Culture
Management – Peripheral Vascular ds Aspirin; Foot care— Inspect feet daily, Check foot wear for forigen objects before wearing, Have feet measured carefully when purchasing shoes, Keep feet away from heaters, Fires and hot water bottles, Check feet temperature of bathwater before bathing, Avoid walking barefoot especially outdoors, Avoid unaccustomed lengthy walks when on holiday; Vasodilators; Surgical Sympatheotomy- Lumbar sympathecto; Reconstructive surgery; Angioplasty; Amputation; Rehabitation Diabetic Foot— High risk patients should be identified during routine foot examination performed on all patients with DM; Patient Education— Carefull selection of foot wears, Daily inspection of feet to detect early sign of poor fitting foot wears/ minors trauma, Daily foot hygine to keep the skin clean and moist. Avoidance of self treatment of foot abnormalities and high risk behavior (e.g. Walking barefoot), Prompt consultation with a health care provider if an abnormalities arise; Risk factor modification— Orthotic shoes and devices, Callus management, Nail Care, Proplylactic measures to reduce increased skin pressure from abnormal bony architecturesmoking, dyslipidemia, hypertension, Antibiotic (IV & oral)— According to culture sentivity report Wound debridement,Osteomyelitis is best treated by a combination of prolonged antibiotic (IV & Oral) and Possible debridement of infected bone, A recent consensus statement from ADA identified six interventions with demonstrated efficacy in diabetic foot wound— Off- loading, Debridement, Wound dressings, Appropriate Use of antibiotics, Revascularization, Limited amputation; Hyperbaric oxygen.

Erectile Dysfunction
History-Through sexual medical and psychosocial history; Difficulty obtaining erection; Rapid (premature) ejaculation; Obtain information about current medications and prior surgeries; *Any h/o of pelvic surgery, trauma, prior prostate surgery, or radiation to the prostate; Tobacco use, alcohol intake, caffeine intake, and illicit drug; Stress factors and tension at work and at home; Indication of depression; loss of libido; Problems and tension in the sexual relationship lethargy, moodiness; Stress from work or other sources.

Examination-Penile plaques; Small tsetse; Evidence of possible prostate cancer; Prostatitis, vascular disorder; Benign prostatic hyperplasia; Status of the genitalia and prostate; Size and texture of the testes; Abnormalities of the penis such as hypospadias and peyronie plagues.
Differentials-Abdominal trauma; Atherosclerosis; Cirrhosis liver; Depression; Haemo chromatosis; Hypertension; Hyperthyroidism; Hypopituitarism (Panhypopituitarisum) ; Hypothyroidism; Non bacterial prostatitis; Peripheral arterial occlusive disease; Peyronie Disease; Priapism; Prostate Cancer; Prostatitis; Endovascular Hypertension; Sclerodema; Sickle Cell Anemia; Antidepressant medication; Antipsychotic; Antihypertensive; Hyperlipidemia medications
Investigation- Evaluation of the patient's hormone status; Measuring morning serum testosterone level, total and free; Measurement of luteinizing hormone, prolactin; Evaluating the patient for diabetes with a hemoglobin Alc measurement; Lipid profile, and prostate specific antigen; Investigate the hypothalamic-pituitary- gonadal axis by evaluating testosterone level; Serum thyroid- stimulating hormone evaluation; Urinalysis looking for RBCs, WBCs, protein and glucose; Nocturnal penile tumescence testing, testing for penile blood flow studies; Angiography; Duplex ultraSonography; Ultrasonography of testes
Treatment-Medical care— Use of oral PDE-5 inhibitor - most common practice, Combination therapy with one of the PDE-% Inhibitors plus Yohimbine, MUSE or intracavemosal injection, in selected cases; * Drugs are PDE-5 inhibitors — Sildenafil (Viagra), Vardenafil (Levitra), Tadalafil (Clalis), Vasodilators (nitroglycerine), Pentoxifylline (trental) ; Yohimbine; Apomorphine (Uprima) ; Phentolamine (Vasomax) ; * Androgens - Alprostaladill, POE 1 - small supossity that can be introduced in to the uretha; Intraurethral therapy (MUSE) ; Hormonal (testosterone) therapy - Hypogonadotrophic hyponganadism- parenteral testosterone 200mg I.M
Surgical-Penile implants - Semirigid or malleable rod implants, Fully inflatable implants, Self- contained inflatable unitary implants, Vascular Reconstructive surgery, Microvascular arterial bypass surgery Others - Psychological care; Vaccume devices- to draw blood in to penis; Penile injection therapy
Dermatologic Manifestations-Diabetic dermopathy-(pigmented pretibial papules)- erythematous area and evolves in to an area of circular hyperpigmentation, more common in elderly men with DM.
Nerobiosis lipoidica diabeticorum-- young women with type IDM. Usually begins in the pretibial region as an erthemations plaque or Papules that gradually enlarge darkeen and develop irregular margins, with atrophic centers and ulceration. *Acanthosis nigricans*- (hyper pigmented velvety plaques seen on the neck, axilla or extrensor surfaces)- features of severe insulin resistance and accompanying diabetes.
Gramuloma annulare- erthematous plaques on the extremities or trunk. *Sclerdema*- areas of skin thickening on the back or neck at the site of , previous superficial infections. *Lipoatrophy* and Lipohypertrophy at insulin injection sities but are unusual with use of human insulin. *Xerosis* & pruritus.
Infections - Pneumonia; Urinary Tract infection; Skin and soft tissue infection – Furuncles, Carbuncles, Cellulitis, Gas gangrene; Emphysematous pyelonephrities ; Emphysematous cystitis; Superficial and deep candidial infection;Vulvovaginitis, Balanitis; Post operative wound infection; Rhino cerebral mucormycosis; Emphysematous infection of gallbladder; "Malignant" or invasive otitis extema, osteomyelitis and meningitis Pulmonary tuberculosis

Macrovascular Complications
Coronary artery disease- (Silent ischemia, myocardial infarction)
History - Chest pain! chest discomfort—Retrostemal, Radiating to back left arm, neck, jaw, Heavy, squeezing, Crushing, Stabbing or burning; Epigastric discomfort; Weakness; Fatigue; Breathlessness; Nausea, Vomiting; Anxiety; Sweating; Sudden loss of consciousness; Confusional state; Palpitation; Risk factor (Hypertension, cigarette smoking, alcohol, family history etc).
Examination - Anxious; Restless; Pallor; Perspiration; Coolness of extremities; Tachycardia! Bradycardia, Arrhythmia; Hypertension! hypotension; Precordium quiet; Apical impulse difficult to

palpate; Fourth heart sound and third heart sound; Decrease intensity of first heart sound; Paradoxical splitting of second heart sound; Transient midsystolic or late systolic apical systolic murmur; Pericardial friction rub; Carotid pulse; Elevated temperature (up to 38°c) ; Arterial pulse
Differential Diagnosis- Gastro-Esophageal reflux disease; Pneumonitis, Asthma; Mediastinitis; Dissection of aorta; Pericarditis; Myocarditis; Oesophageal rupture; Cafe coronary; Pulmonary embolism
Investigations – ECG, Cardiac Biomarkers – Myoglobin, Creatine phosphokinase (ck) – CKMB, Cardiac specific troponin- T and I, Lactate Detrydrogenase, AST (SOOT), Leukocytosis, ESR; Cardiac Imaging - Two- dimensional echocardiography, Radionuclide imaging techniques- Myocardial perfusion imaging with 201 TI or 99m, Tc- sestamibi, Radionuclide ventriculography; Angiography
Management-Medical; Oxygen; Aspirin; Clopedogerel, Heparin/low molecular weight Heparin (LMMH); Morphine; Nitroglycerine; Beta blockers; ACE Inhibitor; Calcium Channel Blocker; Statins; Thrombolysis (streptokinase, Urokinase, tenecteplase and reteplase) ; GP nb/liJ,a inhibitor; Glycemia control (insulin)
Surgical - CABG (Coronary artery bypass Grafting)
Others - Diet; Exercise/activity; Bowel (constipation) ; Sedation;

Macrovascular Complications
Cerebrovascular accidents - (Occlusive stroke and transient ischemic attacks)
History - Sudden onset of loss of sensation (one side of body); Sudden onset weakness (one side of body) ; Change in vision; Gait disturbance; Inability to speak/understand; Sudden, severe headache; Seizure; Fever/sepsis; Risk factors (hypertension, cigarette, smoking, alcohol, family history etc)
Examination - High motor function; Cranial nerves; Motor examination; Sensory tests; Gait; Coordination; Carotid bruit
Differential Diagnosis-Intracranial hemorrhage; Subarachnoid hemorrhage; Migrane; Meningitis; Metabolic Encephalopathy; Cerebal Venous thrombosis; Subdural Hematoma, Neoplasm; Head Injury; Todd's paralysis; Multiple Scletosis; Vestibular disorder; Hysteria
Investigation - Complete haemogram with platlet count; ESR; Bleeding time, clotting time and prothrombin time; Sickle cell test; Anticardioplipin antibodies; C-reactive protein; Lipid profile; Uric acid and electrolytes; Serum protein C and S level; Homocystein level in blood; Chest x Rays; ECG, Hotler monitoring; 2D Echocardiography; CSF; VDRL of blood and CSF; Fluorescent treponemal antibody absorption test (FTA- ABS) ; HIV; Computed tomography; Carotid Doppler; MRI of Brain; MRA or digital subtraction angiography; SPECT of brain
Management-Medical; Maintenance of vitals ;Temperature; Pulse; Respiration (ventilation); Blood pressure;Fluid and electrolytes;Prevention of complications like pulmonary aspiration, seizures, thromboplebitis and bedsores; Glycemic control; Mannitol, Dexamethasone; Aspirin; Clopidogreal; Heparin / low molecular weight heparin (LMWH); Thrombolytic therapy (RT -P A- Recombinant tissue plasminogen activator); Neuroprotective agents
Surgical - Carotid endarterectomy; Extracranial to intracranial bypass surgery; Angioplasty and stenting
Others - Diet; Exercise! activity; Stroke prevention (modification of risk factors)

Jaundice
History-Alcohol intake (quantity and duration) ; Length of history of liver disease (P/H Hepatitis, Jaundice including contact, H/o during addiction (i/V), Tatoos, transfusions. H/o drug intake, overseas travel, H/o fever) ;H/o D.M, Cardiac failure, arthropathy; Treatment history; History suggestive of complication, (e.g.) any P/H encephalopathy, G.I. bleeding, abdominal pain, distention of abdomen secondary to ascites; P/H any operations; H/o travel to endemic areas (for malaria, leptospirosisLook for signs of)
Examination- Note the patient's racial origin (for Hepatitis B and Hepatitis c) ; chronic liver disease(Spider Neave, Gynaecomasia, Palmar erythema, petichae, etc.) ; Look for signs of Liver failure,

flapping , breath, confusion , stupor; Small of alcohol; Anaemia/ WT loss/Tatoos/ body piercing, needle pricks/scratch marks/oedema xanthelesma/ K.F. rings/ Duputren's contractures/ lymphode lubbing; *Examination of the Abdomen*; Exclude severe Rt. Heart failure, Tricuspid Regurgitation, Constrictive Pericarditis;Rectal Examination.

Investigations-The following list covers all the causes for Jaundice. One could narrow it down to the selected one's depending on the provisional diagnosis made — Full Haemogram including peripherial smear and reticulocyte count; L.F.T. including P.T/INR; Auto immune workup; Ascitic Tap; Abdomino-Pelvic U/S Scan; Hepatitis-B and Hepatitis-C; Abdominal Doppler Studies- Arterial; CT scan Abdomen; Barium Series; Endoscopy; Alpha Feto Protein; FNAC and Liver biopsy if indicated; ERCP/MRCP (Diagnostic and Thearapetic) ; Serum Ammonia levels and copper levels ; Serum Electrolyte

Differential Diagnosis (Few Of them) - Haemolytic Disease; Hepatitis/ Cirrhosis of Liver; G.I . Malignancy with Metastastasis; Primary biliary cirohosis; Wilson's disease; Obstructive Jaundice; Hemolysis due to infection

Non Surgical Management-Depends on the diagnosis made-Diuretics; Salt restriction;Removal of precipitating causes; Treatment of infections;Treatment of Haemolysis;Treatment of chronic Hepato cellular failure; Treatment of G.I. Bleeding;Steroids in specific auto immune hepatitis;i/v Octreoatide, Beta Blockers

Surgical Treatment -Sclerotherapy; Esophageal banding (Superior to Sclerotherapy); Peritoneal shunts; Various specific surgical procedures for obstructive jaundice; Splenectomy.

Diabetes Mellitus

Definition-The national diabetes data group &WHO have issued diagnostic Criteria for D.M. Symptoms of Diabetes plus random blood glucose concentration $\geq$ 126mg/dL during on oral glucose tolerance test.

History-Polyuria, Polydipsia, Polyphagia, weight loss; Blurred vision, lower extremity paresthesias, yeast infection, particularly Voluovaginitis in women, balanitis in men; Obese patients; Patient first degree relative with type 2 diabetes mellitus; Patients with hypertension; Patients with high triglyceride or HDL- Cholesterol <35mg/dL; Polycystic ovary disease

Examination-Obesity; Anthropometric data- Height, Weight, Waist-hip ratio, Fat fold thickness; Skin Changes; Hair loss; Prominent veins; Callus formation; Crackers and fissures; Ulcers in the foot and deformities; Peripheral pulses; Blood pressure recording (both recumbent and standing) ; Auscultations for bruits over carotid and femoral arteries; Neurological examination - cranial nerves; Deep tendon reflexes; Autonomic function

Differentials-Diabetes incipidus; Insulin resistance; Obesity; Maturity onset diabetes mellitus; Latent autoimmune diabetes of adults(LADA); Stein- Leventhal syndrome

Investigation-Fasting plasma glucose (fpg) greater than or equal to 126MG/dL or random glucose greater than or equal to 200mg/dL and classic symptoms polyuria, polydipisia, polyphagia, weight loss

Hypothyroidism

History - Age - Simple Goitre- is commonly seen in girls approaching pubertal age. Hashimoto's thyroiditis- middle aged human. Autoimmune Hypothyroidism 4/1000 women- 1/1000men. Sex-majority of thyroid disorder common in females>men. Place- endemic area for goiter. Any history of swelling in neck - When was the swelling 1st sighted?, Has the size of swelling remained same/ gradually increasing?, Any pain at the sight of swelling?(In Hasimoto's thyroiditis there will be some discomfort in the neck) ; Any history of painful enlargement of thyroid gland with fever? Thyroiditis)-Initially there will be thyrotoxic state-> Hypothyroid state ;History of symptoms S/o Hypothyroidism - History of tiredness, weakness, History of loss of appetite with weight gain, History of cold intolerance, Recent excessive loss of hair, History of any change in voice(hoarseness of voice), History of parasthesia suggested by tingling numbness in limb, any loss of sensations suggested by inability to

feel foot wear (this complication/symptom is very rare/late), Any history of dysponea on exertion and effort angina, History s/o constipation

Personal History - Decrease in appetite, History of constipation, History of loss of libido

Menstrual history/ Obstetric history-Complete obstetric history- GPL status, Ask specifically for history of any congenital Hypothyroidism in baby. Suggested by - Prolonged jaundice, Feeding problem, Flaccidity, Macroglossia, Delayed bone maturation, Umbilical hernia, Permanent neurological deficits in the child.

Menstrual history - History of increase flow (menorrhagia) followed by decrease flow, (oligomenorrhea) and then amenorrhoea; History of recurrent abortions.

Drug/ Treatment history - H/o recent thyroid surgery or treatment with radio iodine in recent past. B] iodine excess {eg- contrast imaging}. C} Drug history-Amiodarone, lithium, Antithyroid drugs, Para amino salicyclic acid, Interferon alfa

Family History - H/o similar illness in the family; Past history of snake bite (causing pan hypopitutariam) pituitary disease or surgery, Excessive post partum hemorrhage.

Clinical Examination - General examination- Build and nourishment-Patient is obese and overweight. Patient appears lethargic & tired. Facies- Dull expressionless facies, periorbital puffiness with boggy eyelid - Coarse hair, patchy alopecia; Dry and rough skin; Facial pallor.Pulse- Sinus bradycardia. Blood pressure- Diastolic pressure may be high due to hypercholesterolemia & artherosclerosis. Pallor- Moderate to severe. Usually a normocytic normochromic anemia; Sclera may be lemon yellow tinged s/o carotenemia. Edema of feet- Non pitting edema. Puffiness of face, supraclavicular fossa, neck. [due to deposition of mucinuous material, Mucopolysacharides hyaluronic acid & chondroitin sulphate]

Systemic Examination-Cardiovascular system-Pulse- Sinus bradycardia. [mention rate, volume, character, peripherial; pulses condition of vessel wall);Blood pressure - Mention in which limb& which position; Diastolic hyperten; Heart sounds, may be muffled , features due to pericardial effusion. (30%); Central nervous system-Higher mental function- Shows memory impairment & mental slowing;Depression; Myxedema coma; Slow and sluggish speech [bradylalia] with hoarse voice;Carnial nerve-8thnerve deafness. There may be conductive deafness due to fluid accumulation in middle ear cavity. [serious cavity] ; Tone - Hypotonia; Power - usually normally; Reflexes - Sluggish;Sensory- Entrapment syndromes. Eg- carpal tunnel syndrome- tingling numbness in the distribution of the median nerve; Myopathy - Painful muscles cramps [Hoffman's syndrome}; Calf muscle hypertrophy; Cerebellar ataxia; GIT - Macroglossia- Decreased bowel sounds, Presence of free fluid; Respiratory system- Vocal cord edema- hoarseness of voice- Pleural effusion, Respiratory muscle fatigue; Examination of thyroid - Inspection-Pizzilo's method- Ask patient to keep patients behind head and to press head against clasped hand. Ask patient to swallow-thyroid swelling moves with degulgition. Look for any dilated veins over swelling. Look for any scar over the anterior part of neck s/o any previous thyroid surgery; Palpation-Always palpate with patient's head flexed, Gland is palpated from behind by four fingers with thumb at nape, Palpate for any swelling. If present- note for Local/entire gland involved, position, size, shape, extent consistency, mobile/fixed, Palpate individual lobe-lahey's method, Place thumb on the thyroid &ask patient to swallow [rile's method] To palpate whether swelling moves with deglutition; Other associate conditions- autoimmune conditions like; Vitiligo; Pernicious anaemia; Addison's disease; Type 1 DM; Alopecia areata

Differential Diagnosis-Differential diagnosis will depend on what symptom is presenting with and accordingly the discussion will go on pertaining to the evaluation of that particular symptom.

Investigations-Complete hemogram-Low Hb- Anaemia usually normocytic anaemia [Anaemia may be due to menorrhagia], Serum electrolytes- Hyponatremia; Serum cholesterol- Total cholesterol

usually> 250mg- High triglycerides; ECG- Sinus Bradycardia-Low voltage complexes [height of R waves in limb leads<5mm& that of precordial leads<10mm]m, Non specific ST-T wave changes; Thyroid function test-TSH usually>20mu/1

Clincal Suspicion of Hypothyroidism Determine TSH & Free T4

FreeT4 &TSH	Free T4low	FreeT4low	FreeT4High
Normal	TSH High	TSH normal or low	TSH high
Euthyroid	Primary	Secondary	Thyroidhormone
Hypothyroidism	Hypothyroidism	resistance [defect InTHBR]	

NB- During early stages of hypothyroidism T$ &FT4 lie just below the normal range. T3 is normal & TSH is barely elevated. This is sub clinical hypothyroidism or failing thyroid syndrome.

	Normal	hyperthyroid	Hyperthyroid
T$[ug/dl]	4.5-12.5	>12.5	<4.5
FT4[ng/dl	0.9-2.0	>2.0	<0.9
Ts[ng/dl]	80-220	>220	<80
TSH[uU/mml]	0.3-6.0	<0.3	>6.0

Demonstration of autoantibodies-Antimicrosomal antibody for which Thyroid Peroxidase antigen is positive in 80%cases of Hashimoto's thyroiditis. Antithyroglobuin antibodies positive in 60% cases of Hashimoto's thyroiditis. Radio Iodine uptake scan- Decrease uptake of radio iodine. FNAC and usage of thyroid- Evidence of inflammatory infiltrate &in Hashimoto helps to differentiate a thyroid mass suspected of Hashimoto's& e/o Hurthle cells. FNAC helps to differentiate a thyroid mass suspected of Hashimoto's from lymphoma.

Others-Increase in creatine kinase; Increase in LDH; Increase in AST

Chest X-Ray &ECHO Increase in cardiac silhouette s/o pericardial effusion.

investigations for other associated endocrine disorders lide addison's disease etc.

Treatment - If there is no thyroid residual thyroid function the daily replacement dose of levothyroxine is 1.6 ug/kg/body wt[100-150 ug/day] ; If there is underlying autonomous function [eg- developing hypothyroidism or after treatment of Grave's disease] the dose will be 75-125ug/day

Special consideration- In elderly patient> 60 years esp with CAD starting dose is 12,5-25 ug/day [angina may develop] ; Adults under 60years without e/o CAD starting dose is 50-100ug/day; In the pregnancy the dose may be increased >50% during pregnancy & returned to previous levels after delivery; There are no universally accepted guidelines for the management of mild or subclinical hypothyroidism which is defined as biochemical evidence of thyroid hormone deficiency in patients without any clinical features/o hypothyroidism. In patients with subclinical or mild hypothyroidism esp. if TSH> 6mu/L & TPO antibodies are increased the starting dose will be 25-50 ug/day; NB-The tablets to be taken in the morning in empty stomach with water; Patient who miss doses can be advised to take up 3 doses of the skipped doses at once because T4 has along life [7days]

Follow-Up- TSH response is gradual & should be measured about 2 months after instituting the therapy. Adjustment of the dose is made in 12.5 or 25 ug increments if TSH is high every 2-3 weeks. NB- In patients with pituitary hypothyroidism replacement should be made only after replacement of hydrocortisone has been initiated. [as it may result in adrenal crisis]

Myxoedema Coma - Levothyroxine- 500ug iv bolus followed by50-100 ug/day. [It can be given through NGT if iv preparation not available]; An alternative is to give Liothyronine [T3] in the dose of 10-15ug iv Q8thor Q12th OR Combination of L-thyroxine [200ug] & Liothyronine [25ug] single iv bolus followed by daily treatment with levothyroxine 50-100ug/day & T3 10 ug Q8th; Inj. Hydrocortisone 500mg iv 6th or 8th hrly as there is concomitant impairment of adrenal reserve.

Supportive Measures-External warming with space blankets if temprature,30dC; Precipitating factors should be corrected;Hypertonic saline for hyponatremia; iv glucose for hypoglycemia; Sedatives should be avoided if possible or sused in reduced doses; Ventilatory support with regular ABG may be needed in the initial 48 hrs; Medication should be continued lifelong; If patients with a dose of> 200ug/ day & still elevated TSH, other causes must be excluded. Eg. Malabsorption, estrogen therapy & drugs that interfere with T4 absorption or clearance such as cholestyramine, ferrous

sulphate, calcium supplements, lovastain, aluminium hydroxide, rifampicin, amiodarone, carbamazepine phenyotin.
Surgical Treatment - Not indicated unless there is tracheo esophageal compression or for cosmetic purpose. [Goitre]
Neonatal screening and prevention - Neonatal screening by measurement of TSH or T4 levels in heel prick blood sample . when diagnosis is confirmed thyroid supplements are given. Iodine supplements to prevent iodine deficiency.

PEDIATRICS

Congenital Heart Disease
Acyanotic Congenital Heart Disease
History- Onset of cong heart disease in infancy or if later then slow and insidious onset; CHF- Suck rest suck cycle and feeding diaphoresis in infancy in shunts, respiratory distress, edema feet rare in infants,periorbital oedema; Adequacy of feeding e.g. adequate urine, sleeps well after feed, number of sucking movements with each breath; Exertional dyspnoea in older children; H/O LRTI i.e. cough, fever,respiratory distress; H/O IE –Sudden worsening over previous status, fever,petichae, increasing pallor or worsening CCF;Treatment history e.g. digoxin, diuretics, antibiotics; Course in hospital- F/H , SE history for treatment options.
Examination-Cyanosis circumoral, tongue, fingers, toes, baseline or intermittent on crying; Clubbing; Pallor; Abnormal facies e.g. Downs in VSD,Elfins in AS; Pulse- Collapsing in PDA, all pulses important for Co and takayasu, rate fast in CCF, volume poor in AS; BP in all limbs- Co, takayasu, AR; CCF- tachycardia, tachypnoea, hepatomegaly , JVP is difficult to see in infants due to short neck and oedema feet rare in infants; Anthropometry for FTT;If fever then look for Signs of IE e.g. splinter haemmorhages,spleen tip, Roth spots; CVS-precordial buldge or pulsations e.g. suprasternal of xiphisternal, visible apex; Palpate for sounds and thrills , parasternal heave and apex; Percussion of borders and second space; Auscultate all sounds e.g. S1 S2 S3 S4; S1 in mitral area loud or faint in pansystolic murmers, S2 in pulmonary or aortic area for intensity, splitting; S3 and S4 at mitral area; Listen carefully whether pansystolic e.g. VSD or ejection systolic murmur e.g. ASD, AS,PS or continuous murmur e.g. PDA; Radiation; Grade systolic murmurs; Liver spleen
Discussion-Type of CHD; Single defect or more than one e.g. ASD and VSD, VSD and AR; Know the common associations and look for features; Size of defect in VSD, PDA OR ASD; Signs of Eisenmenger in shunts; Severity of valvular stenosis in AS,PS; Severity of coarctation; Any IE; X ray changes; ECG changes; ECHO interpretation; Need for surgery /medial management e.g. in VSD small or large, chances of spontaneous closure,, ASD if very small no need to intervene, PDA always needs closure, AS if mild to be followed up otherwise balloon dilatation and if unfit for balloon then surgery, PS if significant then balloon, CO balloon or surgery; Time for surgery; Tell complete diagnosis e.g. type of heart disease, rhythm, IE, CHF, reversal of shunt

Cyanotic Congenital Heart Disease
History - Spells e.g. excessive crying , getting more blue, and respiratory distress; squatting; Dyspnoea on exertion; FTT; H/O palliative surgery e.g. BT shunt and whether it is functioning.
Examination - Cyanosis ,Clubbing,Pallor; S2 single or split or loud P2; Usually an ejection systolic murmur; Features of CHF suggest increased pulmonary blood flow
Discussion - Type of CHD-Generally groups diagnosed e.g. TOF physiology covers TOF,DORV, TA, Pulmonary atresia; Increased pulmonary blood flow or decreased; Decreased pulmonary blood flow with S2 single ejection systolic murmurs - TOF physiology; Increased pulmonary blood flow-Features of CHF with cyanosis and S2 not single, look for pulse volume e.g. high volume pulse in truncus, LA enlargement for Ebstiens anomaly, X ray and ECG almost essential for diagnosis; X ray changes; ECG changes; ECHO interpretation; Need for palliative/ total correction surgery; Time for surgery; prognosis

RHD

History - Age of onset later than 3-4 yrs as it is acquired disease; Fever, joint pains, , chest pain, palpitations, palpable nodules for activity; H/o abnormal choriform movements; Exertional dyspnoea in MR, PND in MS; Oedema feet, ascitis dyspnoea for CHF; Treatment history e.g. digoxin, diuretics, penidura; Course in hospital; F/H; SE history of over crowding; P/H of sore throat or CHF.

Examination - Cyanosis , Pallor, ,JVP, hepatomegaly For CHF; Clubbing, . splinter haemmorhages,spleen tip, Roth spots for IE; Pulse for arrhythmias and pulse deficit for AF if irregularly irregular pulse; Pulse volume increased in AR and MR; Subcutaneous nodules at bony prominences; Peripheral signs of AR (must check BP of upper and lower limbs and piston shots along with others) CVS - Precordial buldge or pulsations e.g. suprasternal of xiphisternal, visible apex; Palpate for sounds and thrills , parasternal heave and apex (site and type); Percussion of heart borders and in second space; Auscultate all sounds e.g. S1 S2 S3 S4; S1 in mitral area loud or faint in pansystolic murmers, S2 in pulmonary or aortic area for intensity, splitting; S3 and S4 at mitral area; S1 loud in PS and soft in MR;

S2 - loud P2 in PAH; Systolic murmur at apex in MR, Diastolic at apex in MS , at aortic area in AR; Cardiomegaly

Discussion - Activity or not; Arrhythmia or IE or CHF; Type of leison and severity; Predominant leison in mixed leisons; Medical treatment and prophylaxis; Need for surgery or intervention by cardiologist; Discuss ECG, x ray and ECHO findings.

RAP (Recurrent Abdominal Pain)

Mostly psychological

History - Criteria to label as RAP i.e more than 3 episodes over 3 months; Red flag signs e.g. Night pain, age <3 yrs, site away from midline; Find likely causes e.g. constipation because of spasm or dilatation of proximal bowel, burning micturition,passage of worms; Ask H/O of TB; food allergy so any food item; heavy metal poisoning egg pb,As,Hg; Psychological causes e.g. role modeling, school phobia, forced feeding, new sibling; Pain chart; Treatment history*Examination* - Generally normal but do thoroughly to gain confidence and not to miss anything as minimal investigations; Liver spleen for TB, kidney lump, any other abdominal lump egg wilms, lymphoma, neuroblastoma, bezoars, ovarian tumour, collagen vascular egg JRA,SLE, arrhythmias e.g. SVT*Discussion* - Likely cause in a patient Minimal investigations unless pointers i.e. counts, stool for worms, giardia, urine for pyelonephritis,, ultrasound abdomen for hydronephrosis, kocks, abdominal mass , ovarian tumour, , montoux etc. ; Placebo treatment and psychological intervention; occasionally H pylori and pyloric biopsy; esophagitis endoscopy retrosternal pain; chronic pancreatitis abdominal calcification on x ray; hyperthyroidism, hematocolpos; mesenteric cysts; porphyria; choledocal cyst, mass RU quadrant; inflammatory bowel so barium if chronic diarrhea; sickle cell disease in affected populations due to asoocclusive episodes; rarely abdominal migraine – by excluding others

Hemiplegia*History*-Etiology-Infections; viral exanthems mumps, chickenpox, HSV; Meningitis- acute fever, altered sensorium, seizures; Ear discharge, sinusitis, mastoiditis, for brain abcess and sinus venosis thrombosisDiarrhea dehydration in saggital sinus thrombosis; TBM; Trauma for head or SC injury, fat embolism, hematoma; Hematological disorders- anemia , H/O bleeding disorders ITP,TTP; Pallor, bone pains , wt loss, pallor for leukemia; H/O pain in hand and foot in vascular occlusion episodes in sickle cell anemia.Polycythemia in cyanotic heart disease; Cardiac lesions-IE,Cyanotic HD,RHD,Cardiac surgery; VasculitisFever rash and joint pains; Claudication in Takayasu; Skin lesions and muscle pain in Dermatomyositis; Inflammatory bowel disease as diarrhea; Drug abuseSystemic vascular disease- Recurrent TIAs in Moyamoya's disease; Homocystieneurea –developmental delay, lens dislocation; MELAS syndrome vomiting, headache, seizures, cortical blindness, acidosis; diabetes; Prothrombotic statesOnset - Acute Subacute Chronic; Progressive or static; Involvement of UL and LL equal or not; Cranial nerve involvement; Sensory involvement; Bladder bowel involvement; Seizures, speech, cognitive impairment; Gait; Involuntary movements; Cerebellar signs, root pains; Visual disturbance

Examination-Pallor,petichae,cyanosis,clubbing; Dysmorphic face; Rash, arthritis's, nodules; Lens dislocation; BP in UL and LL; Pulses; Carotid bruit; Ear discharge; Lymphadenopathy, hepatosplenomegqaly; Fundus for Roth spots; CVS for any cardiac disease; CNS examination higher function abnormalities, altered sensorium or speech or visual defects; Cranial nerves- Ipsilateral hemiplegia- lesion above brain stem and Crossed hemiplegia lesion below brain stem; Motor examination upper motor neuron signs in both upper and lower limb; Sensory system for cortical sensations for cortical involvement; Peripheral sensations if ipsilateral loss of dorsal column sensation and contralateral loss of pain and temperature then hemisection of spinal cord in upper cervical lesion; Cerebellar signs; Meningeal signs; Involuntary movements*Discussion*-Site of leison; Upper motor neuron lesion above mid cervical cord; Cortex - language, cortical sensations, cognitive and visual apraxia, seizures; Internal capsule posterior limb- dense hemiplegia, pure motor, similar in cerebral peduncle and upper Pons; Brain stem -cranial nerve involvement; High cervical cord ipsilateral loss of dorsal column sensation and contralateral loss of pain and temperature. Absence of cranial nerve involvementAcuteness and likely etiology - Acute- vascular, abcess; Subacute (days to weeks) Subdural hematoma, bacterial abcess, fungal granuloma, meningitis, AIDSChronic(months) -Neoplasm, chronic subdural hematoma, degenerative disease

Paraplegia
History-Like hemiplegia + - H/O birth asphyxia,backache, bowel bladder abnormalities, spinal abnormality , trauma; H/O Tb ,increasing head size (hydrocephalous) ;
Examination - Spinal deformities, gibbus, spina bifida (tuft of hair),head size; Complete CNS examination
Discussion - UMN or LMN type; UMN type; Cortical signs- If present then ACA ischemia, superior saggital or cortical venous thrombosis, acute hydrocephalous; No cortical signs-spinal lesion below cervical cord, bowel bladder involvement, sensory level,LMN type- Cauda acquina lesion, Anterior horn cells, Gullen Barre syndrome, Myopathy Neuropathy,Polio

Quadriplegia(cord leison,cerebral palsy)
History-Examination similar to hemiplegia
Discussion-UMN lesion- anoxia, hypotension trauma,upper cervical cord (bowel bladder involvement+sensory loss) ; Mixed- lower cervical cord (LMN in upper limb and UMN in lower limb)LMN type distal weakness-Peripheral nerve -symmetric,distal weakness, numbness, less severe weakness GBS, neuropathies; Anterior horn cell symmetrical, no numbness Werding Hoffman's disease (Proximal type of weakness), fasciculations; Muscle disease symmetrical disease myopathies, AHC-asymmetric polio-Neuromuscular junction fluctuating, symmetrical botulinism, ,myasthenia gravis, periodic paralysis

Monoplegia*History* - Examination similar to hemiplegia
Discussion – Rare cortical lesion localized disease UMN type mass effectLMN type – nerve injury e.g. sciatic nerve injury ,polio, erbs palsy, median nerve or ulnar nerve injury.

Floppy Infant
History-Onset of weakness, Birth asphyxia, Fluctuating in myasthenia with ptosis,weak cry,difficulty in crying, H/O mgso4 in mother, phenpbarb, cocaine, warfarin in mother, Botulinism, Repeated aspirations and pneumonias, Features of increased ICT (SOL) ; Forceps delivery in neonate; Inj at buttock causing sciatic nerve injury; Polio or polio vaccine; Lower spinal cord injury; pseudoparalysis
Examination-Sepsis, congenital intrauterine infections, Ptosos, arthrogryposis,resp difficulty talipes, maternal hydroamnios in myotonic dystrophy, mothers handshake clinches the diagnosis,Thin extremities, ptosis, jt contactures at birth cong muscular dystrophy kernicturus early stage, Syndrome eg downs, pradder willi obesity not common in infancy, achondroplasia, marfans,trisomy 13, cat cry.examine in ventral suspension, pull to sitm head control, stroke the soles, reaching out, support wt on legs, reflexes absent , standing sitting.*Discussion*-HIE causing atonic diplegia IEMS,

hypothyroidism, ehlers danlos, benign congenital hypotonea by exclusion, werding hoffman increasing severity with age,congenitalmyopathies

Hepatosplenomegaly

Liver bigger or spleen *History*-Causes- Address kalazaar belt or malaria, fever or not infective pathology, BT for hemolytic anemias, Age e.g. neonatal cholestasis in young infants ,CLD after 3-6 yrs of age, Jaundice, Abdominal distension for ascitis cirrhosis, TB,Multiple swellings in neck, axilla or inguinal region TB malignancy e.g. lymphoma, PHT features e.g. hemetemesis, malena, Effects of liver disease e.g. fat soluble vitamin deficiency e.g. rickets, vitamin A deficiency or ecchymosis due to vitamin K or coagulation defect, Features of liver cell failure e.g. spider naevi, palmer ereythema, testicular atrophy, loss of pubic and axillary hair, Features of heart disease causing liver disease; Fever for liver abcess, TB; HIV –ecurrent infections,FTT; Teeth falling with fever anemia in histiocytosis; Mile stones for storage disorders and seizures or incoordination; P/H or F/H of TB*Examination* - Pallor, jaundice, clubbing , Lymph nodes, Ascitis, Liver size, consistency, surface margin size and span, If like CLD then KF ring, dystonia, Rickets, ecchymosis bitot spots or corneal xerosis, Features of liver cell failure e.g. spider naevi, palmer erythema, testicular atrophy, loss of pubic and axillary hair, flapping tremor. If spleen big then features of hypersplenism e.g. anemia, petichae, Dysmorphic features in MPS, Iridocyclitis in JRA, CHF*Discussion* - Liver or spleen as primary disease or part of systemic disease; Severity of disease e.g. in liver; Enlarged liver specially left lobe in CLD; Shrunken liver in cirrhosis; Large spleen in kalazaar, chronic malaria, tropical splenomegaly.CML; Moderate in PHT,TB, hemolytic anemias; Small in typhoid, CHF,leukemias; Hypersplenism in splenic enlargement; Investigations for CLD , hypersplenism ; Treatment of CLD medically, intervention for PHT e.g scelerotherapy or shunt.

Anemia

Nutritional, hemolytic, aplastic, chronic inflammation, bone marrow replacement

History - Age of onset e.g newborn as ABO, Rh, G6PD deficiency , haemmorrhage,thalassemia by 6 month to 2 yr, erythroblastemia of infancy by 4 month, nutritional by late infancy ie 6 months to 2 yr ,Fanconis anemia y 4 -6 yr ; Community e.g thalessemia in Punjabis,S indhis,Gujratis,G6PD in Parsis, Sindhis,HbE in eastern India, HbD in Punjabis, sikle cell in tribals; Sex e.g G6PD and PK in males mostly; History of pica in iron deficiency anemia; Dietary history; history of goat milk intake; H/O anemia in mother; Birt history of preterm or twin delivery; Drug intake e.g aplastic anemia in chloromycetin, sulphas. G6PD hemolysis with sulphas nitrourantion, antimalarials; H/O chronic bleeding e.g hematemesis hematurea; Chronic liver disease or heart disease; Bone marrow suppression e.g. viral infection, kalazaar, sepsis, TB , falciparum malaria, hepatitis virus; F/H of Blood transfusion (BT) in thallessemia, TB, jaundice or splenectomy; H/O consanguineous marriage; Other cell lines involvement e.g. petichae in aplastic anemia or marrow replacement; H/O chronic inflammatory disease e.g. TB, JDA; H/O renal diseaseH/O BT ; Chronic diarrhea or malabsorption; Anatomical defects e.g. cleft palate, TEF , jejenostomy etc affecting proper feeding; Newborn signs of intrauterine sepsis,*Examination* - Degree of pallor; CHF in severe anemia; Petichae or eccymosis in aplastic anemia or malignancies; Joint swelling; Liver spleen in TB, leukemia/lymphoma, hemolytic anemias, malaria, kalazaar, storage disorders; Associated chronic disease e.g. TB, JRA; Anatomical defects of GIT; Hemolytic facies, hypothyroid; Eyes microcornea in Fanconis anemia,conjunctival vessel tortuosity or retinal haemorrhage or retinal aneurysms in sickle cell anemia; Nail changes eg Koilonechia in iron deficiency anemia, dyskeratotic nails in dyskeratosis congenita; PEM/FTT; Nutritional deficiencies eg angular stomatitis or bitot spots etc. ; Skeletal anomalies eg absent radius, absent /bifid /triphalangeal thumb, polydactyly,syndactyly in Fanconis anemia; Skin changes rg jaundice in hemolytic anemia, hyperpigmentation in Fanconis anemia,non healing ulcers in sickle cell disease; Lymphadenopathy in TB leulemia/lymphoma, infectious mononucleosis.

Discussion - Severity of anemia; Complications of anemia eg CHF; Associated problems eg nutritional deficiencies; Cause of nutritional deficiencies eg dietry,anatomical defect of GIT, malabsorption etc. ; If hemolytic anemia then requirement of blood transfusion, complications in growth development,

puberty, hypersplenism, iron deposition in variuos tissues eg liver, heart, endocrine system, etc, chelation therapy details; Treatment by chelation; Genetic councelling regarding antenatal diagnosis,premarital screening of carriers, masss popultion screening and related programmes; Treatment and prognosis in aplastic anemia, bone marrow transplantation, ATG, stem cell transplantation; If malingnancy then discuss about treatment and prognosis of malingnancy.

Developmental Delay/Cp/Microcephaly

History - Etiology-Antenatal history (fever with rash,maternal bleed, hypertension in mother),perinatal history, (cord prolapse, asphyxia, twins, prolonged labour) and neonatal history (kernicturus, seizures, hypoglucemia, meningitis) ; Seizures; History of cranial N palsies; Detailed developmental history regarding onset and progression of developmental delay.All 4 fields of dev to be tested and compaired for any discrepancy in dev in diferent fields; Associated problems eg feeding, speech, vision , hearing etc; Family history of dev delay, deaths in siblings, syndromes etc.

Examination - Head size for microcephaly; Developmental assesment by various manouvers in supine or prone suspenaion, pull to sit etc; Primitive reflexes; CNS examination in detail; Find if hemiplegia, diplegia, quadriplegia etc. chorioathetoid movements etc. ; Neurocutaneous stigmata for tuberous sclerosis, sturge weber etc. ; Fundus examination for chorioretinitis or spots.

Discussion - Type of neurological deficit; Site of leison; Likely Cause of dev delay; Associated deficts in vision, hearing, spreech etc. ; Intervention required eg occupationa;l therapy, drugs, orthopedic , speech therapist, hearing aid etc. ; Functional deficit and prognosis; Education in school or special institution.

TBM

History - Fever duration; Altered sensorium; Seizures; Ophistonic posture; Past history of pul TB; Family history of TB very imp; History of cranial N palsies; History of cough, diarrhoea,etc for pul or abdomoinal TB

Examination - Anthropometry for malnutrition; Head size for hydrocephalous; AF for buldge; Detailed CNS examination to look for deficits; Chest examination; Hepatosplenomegaly.

Discussion - Site of leison usually diffuse; DD of encephalitis , meningitis , vascular leison to be ruled out; Investigations to prove diagnosis of TB; Treatment.DOTS.

Large Head

History - Since when noticed; Any records of head circumference available; Seizures; Paraplegia or incontinance; Spine swelling; Dev history; Past history of meningitis, head trauma as they can cause hydrocephalous

Examination - Head circumference; AF open or not and if open whether buldging; Dev examination; Sunsetting sign; CNS examination –particularly look for paraplegia, sphintor tone, 3rd 6th cr nerves for raised ICT.

Short Stature

History - When growth slowing noticed; Other family members short or not; Chronic inflamatiry disease or organ failure; Diarrhoea or malabsorbtion; F/H of celiac disease; If pubertal age then onset of puberty

Examination - Height; Upper lower segment; Mid parental ht; Wt for age and wt for ht; Abnormal facies; SMR if relevant

Discussion - Failure to thrive or short stature; Relation ogf chronological age, bone age and ht age; Possible cause eg familial or genetic or disease or others; Role of checking TFT; Tests for growth harmomne assay; Role and use of GH therapy.

Rash

History- H/o Repeated infections-unusual infections; Immunization status; Medication
Exposure to Domestic pets, other animals; Arthopod bites, animals bites; Exposure to sick individual; Exposure to allergens, contaminated water & animal urine; Site of onset, direction & rate of spread; Associated with fever, period between appearance of rash and fever; Rash with fever, Irritability, Convulsions, Headache and Neck Stiffness; Rash with Joint pains, (Polyarthritis migratory) ; Associated with Joint paints, Pain abdomen, Red Colour Urine, Mylagia; Rash come/go on different parts of the body, with itching; Associated with bleeding points; Site of injury (Bleeding diathesis – Lichen planus) ; H/o Sorethroat; Aggravating factors.
Clinical Examination - Type Location, Distribution, Colour Desquamation, other findings.
Type - Petchiae, Purpura, Blanchable, Nonblanchable Purpura, Macule, Papule, Pustules, Plaque, Vesicle, Nodule, Confluent Desquamating rash.
Location - Ear-Measles; Face; Scalp – Atopic; Extensor Surfaces; Buttocks – HSP; Site of Trauma; Truncal – Measles, Rebella, Erthema infections; Peripheral (Acral) ; Diaper Area. ; Distribution ; Progression - Sun Exposed Areas; Site of Exposure; Hands Soles. ; Colour-Erythematous; Thickening.
Gen Physical Examination- Eyes – Congestion; Exudate; Photophobia; Hemorrhage
Pharynx – Congestion ; Exudate
Regional Lympladenopthy
Lips – Fissuring of lips (Kawasaki)
Tongue – Central Coating Strawberry
Hand, Feet – Desuamation
Ear – Discharge
Joint – S/o Inflammation
Pallor
Vitals – Temp, PR, HR, BP, CFT
Systemic Physical Examination -
GIT – Hepato Splenomegaly
Other Organomergaly
Tenderness
CVS – New Murmurs
CNS – Photophobia
Investigations
CBP, ESR
Urine Analysis
Platelet Count
Coagulation Profile
Allergic skin test
Microscopic examination of a sample of the affected skin
Skin biopsy
Other specific tests – for various disease
Differetial Diagnosis - Centrally distributed maculopapular rash; Measles; Rubella; Erythema Infectiosum; Examthem subitum; Infectious Mononuclesous; Drug induced eruption; Typhus; Leptospirois; Lymes disease; Typhoid fever; Dengue fever; Ery thema marginatum; SLE; Stills disease; Peripheral Eruption; Secondary syphills; Erythema muliforme; ; Bact endocarditis; Confluent desquamative rash; Scarlet fever (second dis) ; Kawasaki Disease; TSS (Toxicshock Sydnormes) ; SSSS(Staphylococcal Scaled Skin Syndrome) ; Vesiculiobulious ; Varicella; Disseminated herpes virus infection; Ecthyma gangrenosum; Urtricarial; Serum Sickness; Erythermomdosum ; Acute febrile neutrophilic dermatosis; Purpuric; Infective endocarditis; Dengue fever; Typhus fever; HUS; HSP; Viral haemorhagic fever

Non Surgical Management-Isolation ; Oral hygiene; Nutritious Diet; Bed Rest; Injection Vit 'A'; Stop – offering Drug; Antibiotics; Antihistarmines; Specific Treatment

Vomiting

Definition - All retrograde ejection of gastrointestinal contents from the mouth.

History - Characteristics -Frequency ; Volume ; Colour; Smell; Contents; Association with nausea; Triggering and relieving factors ; Cyclical

Associations –Fever-Gastroenteritis, Otitis Media, Systemic Infecitons; Loose Stools-Gastroenteritis, Food Poisoning; Projectile Nature with- Abnormal Neurological Examination(CNS infection, ICSOL) ; Signs of Intestinal Obstruction(Intussusceptions, Pyloric Stenosis, Adhesions, Appendicitis, Hernia) ; Poor growth / weight loss-Renal Metabolic Disease; Headache / Nausea-Migraine, Psychogenic; Cough-Whooping Cough, Bronchial Asthma; Drug Ingestion

Examination - Nature of Vomitus- Colour, Contents, Amount, Smell; CNS Examination- Head Circumference; Eye Examination for Loss of disc cupping, Absent venous pulsations, Raised disk margins, Papilledema; Macewen's / Carackpot sign; Cranial nerve deficits; Focal Neurolgocial deficits; Investigations – MRI / CT; P/A Examination- Visible peristaltic waves, Distension, Tenderness, Palpable Mass, Emptiness in any quadrant, Bowel sounds, Investigations – X-ray / USG / Endoscopy

Differential Diagnosis – Neonatal — Atresia / Stenosis - Abdominal Distension, Failure to pass Meconium; Malrotation - Distension, meconium not passed; Volvulus- Distension, meconium not passed; Necrotizing Enterocolitis -Distension, Bilious vomiting, Blood / Mucus PR; Meconium Plug - Distension, III-defined mass palpable; GE Reflux - Effect of posture and cough; Inborn errors of metabolism, - Adrenogenital syndrome etc. ; Birth Asphysia - Irregular, slow gasping breathing with bradycardia / normal HR; Hyprocephalus - HC > 97th percentile for GA— Bulging ; Faulty Feeding Technique

Infancy - Congenital Hypertrophic Pyloric stenosis-Vomiting starting in the 2nd / 3rd week of life, Becoming increasingly projectile, Visible gastric peristaltic waves, Palpable pyloric mass; Volvulus-Abdominal distension; Intussusception-Paroxysmal pain causing loud outcries, Red-currant jelly stools, Elongated, sausage – shaped in right upper quadrant; GE Reflux; Overfeeding; Gastroenteritis-Fever, Loose Stools, Pain abdomen; CNS Infections-,Bulging fontanelle, Headache, Convulsions; Peritonitis-Fever, pain abdomen; Inborn errors of metabolism

Childhood - Gastroenteritis- Fever, loose stools, pain abdomen; Medications; Toxins; Hepatitis - Fever, jaundice, anorexia, pain abdomen; Pneumonia -Fever, Cough, expectoration, Dull percussion note, Crepitations audible, Consolidation on X-ray; ICSOL- Headache, vomiting, papilloedema, focal signs; Appendicitis- Fever, vomiting, pain abdomen; Peritonitis; Intestinal Obstruction-Hernia / intussusception / peritonitis/foreign bodies/ rumors. ; Intussusceptions; Psychogenic

Management - NonSurgical—Sips of cold, clear fluids; ORT; Drugs-> Metoclopramide, Domperidone, Phenothiazines; Antibiotics for Infecitons. Surgical -CHPS – Ramstedt's operation(Pyloromyotomy) ; GERD – Nissen's Fundopication Prosthesis; Appendicitis–Appendicectomy; Hydrocephalus – Shunts; Intussusceptions – Hydrostatic pressure with Enema Exploration; Intestinal Obstruction – Exploration

Hemiplegia

History - Most importance should be given for a proper and accurate history taking, since it gives the clue for a correct diagnosis, in CNS cases, unlike other systems where a proper diagnosis may be made even by clinical findings alone. Following points must be stressed in the presenting complaints- Was the onset acute, sub acute or gradual? Was it associated or preceded with fever and/ or a seizure? Any severe headache, vomiting or change in consciousness, preceding the onset of hemiplegia. If so, sudden or gradual? Is the condition deteriorating, static or improving?

Past history- Any history suggestive of vasculitis, rheumatic fever, trauma or thrombosis or bleeding disorders. Review of developmental history, antenatal, perinatal, and post natal should always be recorded. Family history and consanguinity must be noted.

Clinical Examination - A detailed general examination, including temperature, pulse, BP and anthropometric measurements should be noted. Any facial dysmorphism, cutaneous lesions like café au lait spots, angiomas or depigmentation and condition of teeth have to be noted. Look for any mid line defect. Examination of chest, abdomen and palpation of femoral pulse must always be a part of general physical examination. Note for unusual body order. Palpation of anterior fontanel, and auscultation for bruits done in erect posture-over both globes, temporal fossae, and mastoid region (six sites).

CNS examination - Higher function assessment according to the age of the patient.

Cranial nerves-Olfactory-rarely assessed, Optic nerve - Elicit the blink reflex, (in a child>3 mon.old) Fundi examination- optic disc, retinal hemorrhage, chorioretinitis, Look for visual acuity, and field of vision depending on the age, and sensorium of child. III, IV, VI Cranial nerves- Enophthalmos ptosis, meosis, lack of sweating, on the ipsilateral side of face.. See the papillary reflex, If ptosis is present, look for 'Marcys Gunn sign,' Test the movements of all extraocular muscles. If the child is unconscious see the 'doll's eye movement. Examine for internuclear ophthalmoplegia by looking for defective adduction of the medial rectus and nystagmus of the abducting eye. See for nystagmus, and if present stage it (stage 1,2,3). V Nerve- Look for deviation of jaw.. corneal and conjunctival reflexes. Test sensation of face. VII Nerve- Is the facial nerve affected. If so, ipsilateral or contralateral to the side of hemiplegia..Look for'emotional 'facial weakness. Confirm whether UMN or LMN type of involvement.. Look at the naso labial fold and wrinkling of fore head. Is the taste salivation and/ or tear production affected? Look for the strength of orbicularis occuli. Elicit 'Mc Carthy reflex' VIII (Cochlear and Vestibular). Rinne's test for defective hearing. Test for vertigo

IX and X - Any nasal regurgitation or change in voice? Movement of palate. XI Nerve- Test the sterno mastoid and trapezius muscles XII Nerve- Look for deviation of tongue when protruded, fasciculations when the tongue is inside the mouth, and/ or atrophy.

Motor system- Look for abnormal movements. Muscle power (0-5- MRC scale). Tone, pronator sign for upper extremities and Barre sign. for lower limbs, depending on the age of the child.. Look for coordination by applying specific tests for cerebellar function- Finger nose test,' tapping in a circle (one cm.diameter) test', look for rebound phenomenon.

Sensory system in older child - Temperature, light touch, crude touch, based on a dermatomal distribution. Examine for position and vibration sense, and elicit Roberg's sign.

Reflexs- Elicit superficial and major tendon reflexes, knowing its root value, and cutaneous nerve involved..Elicit plantar reflex and ankle clonus

Infants - Posture and muscle tone, primitive reflexes- Moro reflex, Tonic neck reflex, Righting-reflex, palmar and plantar grasp reflexes, Vertical suspension, and Landau reflex

Investigations-Besides, routine CBC, Mantoux and X Ray chest, the following investigations should be done- CSF examination. Electrolyte estimation (Na, K, Cl2) ; PT, APTT.

Antiphospholipid antibodies (in SLE), ANA ; Estimation of antithrombin III level, protein C, S, and factor V Leiden. ECG, ECHO, EEG, NCV, and CT brain; Cerebral angiogram, or MR angiography (MRA)/ Functional MRI Evoked potentials VERs/BAERs and, SSEPs) ; Radiography of Spine and Myelography

Diffusion-Weighted Magnetic Resonance Imaging in acute stroke conditions; Magnetic Resonance Spectroscopy (MRS), when cerebral metabolites are of significance ; Positron-emission tomography (PET), esp. in children for surgical programmes.. Single – photon – emission computerized tomography (SPECT) for conditions where regional blood flow study is important.

Differential Diagnosis-The following conditions should be kept in mind to arrive at a provisional diagnosis. Of the following, only 3 most probable conditions judged by history and clinical signs must be discussed in differential diagnosis- Hemiplegic cerebral palsy, Infantile hemiplegia (to be considered only in a child up to 6 years),Vascular causes, including IEM (haemarrahage, thrombosis, vasculitis, trauma, hemiplegic migraine),Infective causes- bacterial, viral, fungal (Meningitis, encephalitis, TBM),Demyelintng- Acute Desseminated Encephalo Myelitis (ADEM), Todd's paralysis also may be considered, if hemiplegia follows a prolonged seizure, Space occupying lesions

Non-surgical management - Depends on etiology. Measures to reduce cerebral edema, correction of fluid and electrolytes imbalance, if any has to be done. Long term management like physiotherapy, speech therapy, and rehabilitative measures may be required in some cases. Referral to a child development center (CDC) is advisable for selective patients.
Surgical management - Surgical management will be required in space occupying lesions and selective cases of spastic cerebral palsy.
Any other - Site of the lesion, (Localisation) must be discussed before considering the differential diagnosis..

Cerebral Palsy
History - The candidates should be very clear about the duration of the illness – present illness; A patient with recurrent episodes of almost identical illnesses with symptom free intervals – the last episode to be described in full and others included under the heading of past illness; All events (including treatment) should be described in the chronological order - as the disease develops Describing each symptom separately is not desirable; Illnesses which have origins in the neonatal period to be described from birth e.g. birth asphyxia with seizures leading to Cerebral palsy; Describe in detail only relevant history. Discredit for spending too much time on irrelevant history; All aspects of history to be discussed and documented. At postgraduate level they must know which is relevant and which is irrelevant; At the end of history the candidate should be able to draw some conclusions regarding the problem e.g. Degenerative diseases of brain -Intracranial space occupying lesion, Chronic liver disease
Physical Examination - General physical examination - Observation about general appearance, Diagnostic clues – positive and negative; Growth - Detailed measurements only in growth problems; Development - Details only in cases of developmental delay; System involved - Give all details; Other systems - Positive findings only.
Summary - The candidates should summarize the case in 4 or 5 sentences – only relevant history, positive findings and important negative findings
Diagnosis - Complete diagnosis to be given; Substantiate the diagnosis based on history, findings and course of the disease – both positive and negative; Differential diagnosis only if there are valid reasons; Candidates should state why the DD is suggested
Investigations - Relevant investigations only; The candidates should know why he is suggesting a particular investigation.
Treatment - Essential treatment only; Rationale of treatment should be explained.

Coma/Altered Sensorium
History- When? How? Under what circumstances? How long? Who were there? Witness should be questioned. History of fever - CNS infection, febrile encephalopathy(cerebral malaria, Dengue encephalopathy, leptospirosis with aseptic meningitis etc) ; History of Headache, vomiting – increase in ICP; History of seizures; If known seizure disorder – whether omission/over dosage of AED; History of fall/head trauma – concussion, intracranial injury; History of drug intake – suspicion is very misleading – family members on drugs; History of Pica – Lead poisoning; Similar episodes before – lethargy, vomiting, Coma – Inborn error of metabolism; Underlying chronic medical conditions – CRF, hepatic encephalopathy etc
Clinical Examination-ABC; Glasgow Coma Scale; Pulse – bradycardia - '! ICT; Temp. – Febrile seizures, CNS infection, febrile encephalopathy; Respiration – effortless tachypnea-metabolic acidosis, DKA -slow breathing – respiratory depression, ataxic breathing – cerebellar herniatino; BP – HT, hypertensive encephalopathy, increased ICP; Pupils – asymmetry – unilateral dilated fixed pupil – Tentorial herniation-Pinpoint – OPC, barbiturates, Dilated, fixed – postictal, atropine etc.
Differential Diagnosis- Metabolic; Trauma, Tumour, Toxins; CNS infection; Vascular; Demyelination; Postictal; HIE; Persistent vegetative state

Management -Non Surgical - ABC; Draw blood fro biochemistry/drug/toxin; 25% dextrose if hypoglycemia; IV mannitol if increase in ICP; IV antibniotics if suspicion of meningitis; IV anticonvulsants, seixures; Sublingual nifedipine if hypertensive; Antidotes for poisoning; Investigate the cause and manage accordingly
Surgical - Extradural/subdural tap; Cerebral abscess – drainage; Tumous with obstructive hydrocephalus – VP shunt
Any other - Water & electrolyte balance – look for SIADH/central diabetes insipidus; Nutrition; Care of back/bladder/bowel/eye; Passive physiotherapy; Parent counseling & support; Dolls eye movement – Full – brainstem intact; Fundi – retinal hemorrhages – ICH accidental or non accidental-Palilloedema – increased ICP; Skin rash – Meningococcal meningitis- Hepatis encephalopathy; Icterus – Hepatic encephalopathy; Bruises, subconjuctival haemorrhage, black eye, Battle's sign, bleeding from ENT – ICH following head trauma; Focal deficit – opposite hemispherical lesion; Meningeal signs – CNS infection, subarachnoid hemorrhage
Investigations - Blood sugar – hypo/hyperglycemia; Serum electrolytes, ABG; Blood urea, serum creatinine; LFT, serum ammonia – Reye's syndrome, IEM; Serum lactate pyruvate, toxic screening, serum AED levels; If fever – QBC, MSAT, Dengue serology, WIDAL(fever workup), NEC, CSF, peripheral smear; CT brain P & C – Trauma, tumor, vascular, CNS infection, HIE, ADEM; LP & CSF analysis; EEG – triphasic waves in metabolic encephalopathy; PLEDS in hepes encephalitis; Spikes and waves in non convulsive status eiplepticus; MRI brain – ADEM, HSV encephalitis

Bleeding Disorder
History- Localized Vs Generalized bleeding; Markers of serious bleed; First presentation Vs recurrent bleeding; Differences in presentation of a bleeding and clotting disorder in relation to -Age of presentation, Gender, Site of bleeding, Relation with injury (early/late bleed), Family history(and pedigree chart) ; Drugs related with thrombocytopenia/thrombasthenia/pancytopenia; Bleeding disorders due to chronic liver disease, chronic renal disease, autoimmune disorders with vasculitis; Clinical pointers towards HIV in a multi transfused case; Previous history of bleeds with reference to site, amount, need for hospitalization a d transfusion.
Examination - Significance of Pallor out of proportion to bleed, and lymophadenopathy; Identification of lesion like petich, purpura, echymosis, nodules and hematoma; Distribution; Hess's test
Detailed examination of joints and bones; Significance of organomegaly; Markers of vasculitis and autoimmune disease
Differential Diagnosis -Bleeding due to supectd thrombocytopenia; Bleeding due to thrombocytopenia with other series also affected (Pacnytopenia) ; Clotting disorder; Vasculitis Syndromes
Management -Acute bleed; Specific underlying cause

Paraplegia
Paralysis or weakness of both lower limbs
Site of lesion - Cerebrum - Trauma – Parasagittal, Tumor – Parasagittal meningioma, Thrombosis of a) Unpaired anterior cerebral A b) Sagittal sinsus; Spinal Cord; Spinal roots; Peripheral nerve; Muscle
Clinical features and History taking Onset – mode; Acute; Subacute; Chronic; Intermittent(Very rare – T.B.);Progression-Static,Progressive,Improving;Cause-Trauma,TBcontact,Primary tumor, Vaccine/ Dogbite, Family Similar illness (Hereditary spastic paraplegia), Drug intake(Causing neuritis/ myopathy)
Complications- CNS; PNS; Automatic; Other systems

Sypmptoms and Signs - Spinal paraplegia -clumsiness of gait, refusal to stand or walk, loss of bower/bladder control, loss of sensation

Signs - Scoliosis
Note- Presence of scoliosis in females before puberty and males of all ages should strongly suggest neuro muscular disorder of spinal cord pathology -Sking abnormalities over spine, tuft of hari,

pigmentation, sinus, mass, Pes cavus, Tropic ulcers, Posture. Hip flexion corresponds to L1, L2 so is affected in high lesions. Thus in thoracic lesions – assume a from – legged posture; Preserved L3 allows some knee flexion and extnsion; Lower lumbar lesion results in preserved hip/knee movements and ankle dorsiflexion; Hip extension corresponds to L5, S1, S2 and so is affected in all but lower sacral lesions; Lesions at S3 or below completely spare lower limb sensory and motor function, but cause paralysis of bladder and anal sphincters and saddle anesthesia - Muscle bulk/joint deformity/ contractures, Head size/shape/fontalnelles, Scar on abdomen(VP shunt), Tender spine, Cutaneous markers of T.B., Herpetic vesicles(Herpes-myceletis), Cafa au lait spots(Neuro fibromatosis)

Q. How to differentiate UMN from LMN causes

A. Cortial sensory loss; Seizures; CNS dysfunction(e.g.) altered senorium, mental retardation

Q. How to differentiate between Automatic Autonomous bladder

A. Automatic	Autonomous
Site of lesion-UMN	LMN
Symptoms-Frequency/Urgency	Overflow incontinence
Bladder size-Decreased	Increased
Pyramedal sign +	_

Q. What questions do you ask about bladder function-Sensation of bladder filling;Starting micturition; Sustaining;Stopping;Satisfaction of emptying

Q. What is Paraplegia in flexion

A. Involvement of pyramidal tract + posterior column

Q. What is Paraplegia in extension

A. Involvement of only pyramidal tract

Q. Salient features of Transverse Myectis

A. Motor loss of below the level; iSensory loss below the level(all modalities) ; Bladder involvement; Most common site – Mid-thoracic region; Evolves over a period of several days to weeks

Q. How will you mention the diagnosis

A. 5 levels-Motor level; Sensory level; Reflex level; Vertebral level; Autonomic involvement

Spinal Paraplegia

Etiolgoy- Congenital malformation - Arachnoid cyst; AV malformation; Atlanto aial dislocation; Arnold chiari malformation; Myclomeningocele;Familial spastic paraplegia - Autosomal Dominant (A.D) ; Autosomal Revessive (A.R.) ; X linked Recessive (X.R.); Infections - T.B. ; Hopkins paxalysis(Post Mycoplasma) ; Diskitis; Epidural abscess; Herpes Zoster; Metabolic - Adrenomyelo neuropathy; Argininemia; Krabbes disease; Transverse Myelities - Devic's disease; Encephalomyelitis; Idiopathic ; Trauma - Consussion; Epidural hematoma; Dislocaton/fracture; Tumors - Astrocytoma; Ependymoma; Neuroblastoma; Ewings sarcoma

Investigations– MRI- Single most important investigation but clinical findings should guide this investigation; CT-Important role in identifying extent of bony defect in spina bifida, Also investgation of choice in Atlantoaxial anomalies or instability; Plain X-Ray – Spina bifida Atlantoaxial anomalies; Lumbar Puncture(L.P)—Tuberculosis-Total Count(T.C) – usually > 500, lymphocyte predominant; Protein – increased; Glucose – decreased; AFB +/- (Acid Fast Bacillus) ; PCR +ve (>95%)

Note- LP is contra indicated in acute stage, since it aggravates parapareis Herpes zoster myelitis— TC- Pleocytosis, Protein- mild evelation, Glucose –normal or decreased;Tumours (e.g) (Astrocytoma) (Secondaries –Acute myeloid leukemia(AML), Acute lymphocytic leukemia(ALL)

Electromyography(EMG)- When suspecting metabolic myopathies (e.g.) Severe Congenital Autosomal Recessive Muscular Dystrophy (SCARMD) ; To delineate segmented involvement to patchy lesions

Nerve Conduction Velocity (NCV)-When features are suggestive of peripheral neuropathy
Arteriography- A.V. malformation
ANA/Anti ds DNA—SLE – Lupres myciopathy

Monoplegia

History - Establish if the case is a monoplegia (or) part of hemiplegia, quadriplegia or paraplegia. Then the following history is a must.

Age of onset -Infant-Brachial plexitis(ERB's Palsy); 10-15 yrs-Monomelic spinal Muscular; Atrophy; any age-Injury to plexus or Nerve – Infection; Sex - Male-Monomelic spinal muscular atrophy; Family History - Hereditary brachial plexopathy; Homocystinuria; Birth History -Birth injury-C.P; ERB's Palsy-Trauma-Extended arm during prolonged surgery; Fall & Injury to plexus; Trivical-Hereditary brachial plexopathy

Infection- Poliomyclitis; Osteomyelitis leadin to neuropathy; Osteomyelitis humerus upper end plexitis – neuritis;Immunisation-OPV; Tetanus Toxiod precipitating hereditary Brachial plexopathy; Asthma(Hopkins syndrome-Asthmatic Amyotrophy; Site of Involvement-Promixal muscles; Distal Muscles; Pain- At rest; Increased by movement; Inability to use Limb - Ex. Trauma/others; Pain + Inability to use Limb - Plexitis; Trauma; Sensation - Present/absent/extend of loss of sensation; H/o. Bleeding Diathesis; Duration-Acute; Chronic; Progressive; Recurrent eg. Hereditary Brachial Plexopathy—Improving or not; H/o. ICT- Headache, Vomiting and seizures; Contractures; Foot drop – peroneal nerve palsy;

Clinical Features - General appearance- (Facies-Storage disease – mucolipidosis, Causes compression at the carpal tunnel in children; Eyes- Cataract, Storage disease subluxation lens – homocystinuria; Lymph adenopathy – Neuroblastoma etc.; Anemia – bleeding disorder; Skin Purpunt Spot;System examination-Higher Functio; CNS- Conscious/unconscious; Group according to Glasgow coma scale; Cranial Nerves— Cr. Nerve- Visual impairment – subluxation lens – homocystinciria, FUNDUS- Bleeding/Chroid tubercle or secondaries; Cranial Nerve- Fascial palsy a part of ERB's Palsy; Other Cranial nerves - Particularly lower cranial nerves at the atlanto accipital region; motor system- Inspection-; Nutrition-; Wasting – Poliomyclitis; Wrist Drop – Peroneal Nerve Palsy/Contractures; Faciculation; Trauma; Power-; Tone-; Hypertonia/Hypojonig – Cerebral Palsy; Hypotonia - Poliomyelitis/Plexitis; Reflexes-DTR, Brish & Exagerrated – CP(Hypotonic); DTR Plexitis/neuropathy; Superficial reflexes ; Plantar extensor – C.P ; Neonatal Replexes- (Ex.) Moro present or absent; spine-Local tenderness/swelling; Abnormality eg. Short neck; Gait-Assessing Recovery- Proximal to distal. This is plotted by Tinel Sign(Tingling in the distal part of a limb caused by tapping over the regenerating segment of a nerve);Resp System-Wheeze – Asthma; Paradoxical breathing – Diaph paralysis. As part of ERB's (T1 Involvement);Cardiovascular System-Myxoma of the heart - recurrent Monoplagia; Cynotic heart disease eg. Fallots producing Thromoembolism; Abdomen- Hepato splenomegaly – storage disease

Differential Diagnosis - Hypotonic Cerebral Palsy-Tone but DTR exaggerated. Plantar extensor/H/o of birth injury; Plexopathy/Neuropathy-Involvement of proximal muscles with or without sensory loss. There is gradual improvement over the years Eg. Brachial Palsy; Aneterior Horn Cells-Poliomyelitis; Tumours-Primary – malignanat schwannoma secondary – neuroblastoma; Trauma-Neonatal brachial Neuropathy ; To plexus;To bone Osteomyelitis - neuritis; In born error of Metabolis-Homocystinuria,Storage disorder – Carpal tunnel syndrome; Spinal abnormality- Cervical & lumbar; Degenerative disorder (Monoplegia + fasciculation, Tremar, Blindness + Consanguinity)—monomelic spinal muscular atrophy. This is a rare disorder Fasciculation common, SCHINDLER's Disease Acute, Sudden on set Monoplegia/Hemiplegia + Blindness

Investigations -Hemogram – Hb; PCV – increased in cyanotic heart disease; TC; DC; Smear abnormal cells/Blast cells or B.T.C.T. ; Blood culture & Antibiogram – Oseteomycitis – Neuritis; Nerve Conduction –(eg) Normal in spinal muscular atrophy. But EMG is consistent with denervation; CT/MRI/Sonogram – For spinal tumours; Electromyogram(EMG) - To distinguish between plexus injury and nerve injury; Screening test - For Amino Aciduria- (eg) Homocystinuria; Muscle Biopsy-

Monomelic spinal muscular atrophy; CSF- (Eg.) Normal except for a mild elevation of protein in lumbar plexitis
Treatment- Non Surgical-Antibiotics if necessary; Physiotherapy; Electrical Stimulation; Others. Surgical-Removal o tumour; Eg. Malg. Schwannoma; Restoration of injured nerve
Prevention-Prevention of EB's Palsy by careful delivery of baby; Takin precaution to avoid trauma, infection etc. in Precipitating Heridatory Bracial Plexopathy; Avoiding prolonged extension of upper limb/arm during Surgery – to avoid trauma to plexus

Gasrointestinal Bleeds
History- Age of baby; Nature and site of bleeding-epistaxis/hematemasis/malena/hematochezia; Onset-Acute/Chronic; Amount of bleeding; History of ingestion of spurious compounds eg. Bismuth, Iron preparation etc; Any other bleeding manifestation-Petechiae, Purpura; Urine output
Clinical Examination- General Survey- Consciousness; Anemia/Jaundice/Cyanosis/Neck glands/Neck veins/ Jaundice/Cold peripheri/BP/Pulse/Respiration
Systemic Examination- Inspection -Abdominal distention/Prominent superficial veins; Local Examination of Anal fissure. Palpation - Free fluids; Flow of veins; Organomegaly; Palpable mass; PR examination
Percussion - Shifting dullness. Auscultation - Peristalsis sound
Investigation - APT test; Hb%, TC, DC, ESR, CRP, Platelet count, BT, CT, Grouping, Cross-matching ; Blood Urea/Creatinine; LFT; PT, PTT; Blood C/S; Urine C/S; Stool fro Ocult blood; USG Abdomen; Barium Swallow; Endoscopy; Colonoscopy; Detection of H Pylori; Radio nuclide study to determine site of bleeding of GIT where endoscopy and barium study have failed; Angiography in selective cases and to detect vascular malformation
Differential Diagnosis - Varicose vein; Exraphepatic Portal Hypertension; Cirrhosis; Meckel's diverticulum; Polyp; H Pylori
*Managemen*t - Non-Surgical - O_2 Administration; IV drip 10-20 ml/kg of Ringer Lactate or NS rapidly, repeat SOS till urine output, pulse & BP improves and cutaneous perfusion normalized; Hematocrit to maintain at 30^o; Blood transfusion if needed accordingly; NG tube suction; Bleeding site to be determined-mucosal lesion/varicela bleed; Treatment of Mucosal lesion- Antacid/Ranitidine/ Omeprazole, Treatment of causes-ITP etc, Treatment of variceal bleed, Vasopression/Nitroglycerin, Glypression/Somatostatin, Octreotide , Beta blockers fro recurrent variceal bleed, Ballon tamponade , After patient is stabilized - Endoscopic sclerotherapy-every week for 3-6 wks, Observe for ulceration/ stricture/disphagia, If bleeding continues emergency surgery, Shunt surgery or, Portal decompressive surgery. Factory indicating poor prognosis-[Massive hematemesis; Initial hematocrit<20o; HB<7gm%; Transfusion exceeding 85ml/kg; Failure to identify source of bleeding; Coexisting coagulation disorder; Associated liver disease of other systemic disease]

Fever and Rash
Fever is one of the common presentations of children admitted to the hospital. Very often fever is of viral origin and lasts fro a short duration. Quite often fever becomes a diagnostic enigma. However, when fever presents with rash or rash associated with fever, the diagnosis becomes relatively easier because rash acts as a clue to the diagnosis of the disease. The skin rash with fever may reflect the effect of toxin(scarlet fever), bacteria(meningococcemia), virus(dengue), inflammation(drug rash) or vasculitis(henoch scholein purpura). The onset of rash relative to the course of underlying illness if any should be ascertained. The temporal relationship of fever with rash should be seeked. Associated features should be looked for e.g. diarrhoea(enteroviral) and severe illness(meningococcal). Lcocatino of rash would also give a clue about the aetiology. History of similar illness in the neighborhood or siblings, and the use of drugs must be noted. A systematic history, clinical examination further aided by investigations would assist in the diagnosis of the disorder. A pertinent differential diagnosis can at least be deduced, thus making the management easier. The following key issues need to be addressed while discussing/presenting a case of fever with rash.

History- Age of the child; Temporal relationship of fever with rash; Prodromal symptoms; Site of onset of rash and its distribution; direction and rate of spread of rash; Morphology of rash(macular, popular, pustular) ; Progression/Course of rash; +/- Pruritis photosensitivity(waxing & waning pattern in erythema infectiousness) ; Associated systemic symptoms-Cough, coryza, conjunctivitis, arthritis, LN pathy, diarrhoea, abdominal pain, chest pain, bleeding from any other site, carditis; Did the patient become toxin at any point int h4 course of illness; Immunization history(rules out vaccine preventable exanthems); H/o contact with individuals with similar illness(infectious aetiology); Is the individual immunocompromised; H/o intake of drugs, food allergy(drug rash); Travel history to endemic areas vital(viral hemorrhagic fevers); H/o animal or arthropod bites(body lics, rat fleamite, rat bite); Similar illness in the nighbourbood or siblings; H/o joint pains; H/o Pica

Examination - Fully expose the area preferably in natural light.

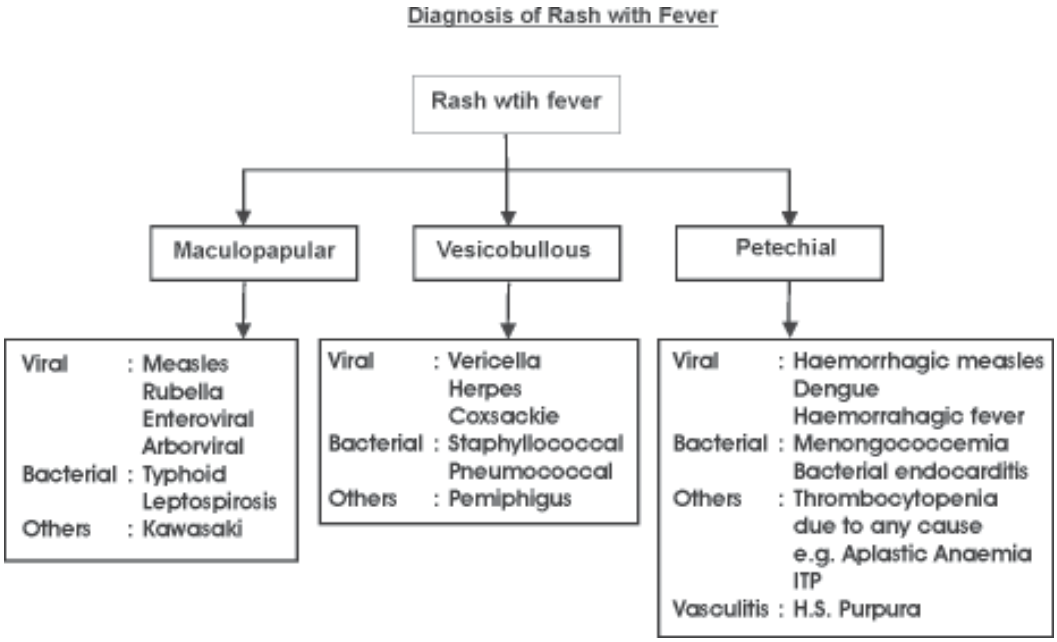
Rash–Morphology-Colour,size,consistency,margins,surfacecharacteristics;Distribution-Symmetrical/ asymmetrical, centrifugal/centripetal; Flexor/Extensor configurations - Nummular/discoid, annular, circiant4e, arcirate, gyrati/serpiginous, linecir, grouped, reticulate; Are onlythe exposed areas affected ; Are the genitals/mucous membrane also involved; Nikolskyi sign(positive in SSSS, TEN) ; Vitals;Signs and symptoms of sepsis.

Shock – Strep Toxic Shock Syndrome(TSS), staph TSS, ecthyma gangrenosum, purpura fulminans, dengue hemorrhagic fever, meningococcemia.

Oral Examination; Koplik spots – Measles; Forchheimer spots – palatal petichiae – German measles, Infectious mononucleosis, scarlet fever. Pharyngitis – Rh fever, IMN; Strawberry tongue – Scarlet fever, Kawasaki disease;

L.N. - Post auricular and sub-occipital group-German measles

Joints - Arthritis in German measles; Meningitis; Enteroviruses, meningococcemia, aseptic meningitis. Hepatosplenomegaly – infectious mononucleosis, typhoid, TORCH infectious(neonates) ; Heart – Murmur(Rh. Fever, SABE)



	Measles	Rubella	Exanthema Subitum	Fifth Disease	Varicella
Incubation period	8-12 days	2-3 weeks	5-15 days	4-14 days	10-21 days
Etiological agent	Pramyxovirus	RNA-Toga virus	HHV-	Parvoirus- B-19	Varicels-zostor virus
Prodrome	Upper respiratory syndrome, Conjunctivitis, Fever, Koplik Spots	Mild fever and coryza	Sudden onset of high fever	Low grade fever	Brief illness headache and fever
Rash characteristics and Location	Maculopapular starts from face behind the ears goes down till feet in next 2 days	Macular confluent begins on face and spreads	Rah 3-4 dyas after fever begins on trunk and spread oil	Slapped check appearance later spreading to trunk and limbs more prompt- extensive surface	Papular rash later changing to clear vesicles. Appears in crops. Starts from trunk and then spreads
Post rash staining	Fades in some order	Fades quickly and nil	Maculopapular fades within 3 days-nil	Lasts fro 1-3 weeks. Lacy reticular pattern on fading-may was and wane	Scrab formation after 4-7 days hypopigmented lesion
Fever and associated symptoms	Fever at peak when rash appears and remains high grade x 2 days after rash	Mild fever, Post occipital and retroauricular lymphadenopely	Sudden onset of high fever. Fever falls along with appearance of rash	Fever is mild	Fever rises with each fresh crop of rash
Complications	Pneumonia, Encephalitis, Diarrhoea, Malnutrition	Embryopathy	Febrile seizures	Arthropathy and myalgia. Aplastic crises in patients with hemolytic anaemia	Pneumonia, encephalitis in Immunocom-promised. Severe illness in adolescent and adults

Investigations - Hb, TLC, DLC, ESR, platelet count; Chest X-ray; Blood culture(bacterial infection) ; Tourniquet test; Viral serology; TORCH screen; LE cell, rheumatoid factor; ECHO(cardiac involvement) ; ECG(rheumatic fever) ; Urine analysis(Bacterial endocarditis) ; Lumbar puncture
Management - Maintenance of vitals (oxygenation, BP, blood sugar, temperature, airway, etc.) ; Temperature control; Intravenous fluids; Antibiotics fro bacterial infections (rational antibiotic therapy) ; Blood/blood product transfusion as per requirement; Specific therapy for specific disorders

Anemia

History- Present — Acute or Chronic - Lethargy; Fatigue; Scholastic backwardness; Frequent infections; H/o Exertional dyspnoea breathlessness; H/o Bleeding – any site e.g. PR. Past— H/o Pallor; H/o bleeding disorders; H/o Malaria; H/o Blood transfusions; H/o Worsening of pallor with infections; H/o Chronic infections – bronchicetasis, osteomylitis, dactylitis; H/o Connective tissue disorders – Rhumatoid arthritis, SLE; Chronic renal Diseas; Chronic diarrhea; H/o Surgery – involving stomach/terminal clieum(B12 def). Personal History — H/o Chewing pencil(lead) ; H/o Pica-mud, ice cube, etc. ; Hook work – H/o walking barefoot – open defecation. Family History — Anemia; Blood transfusions; Splenectomy; Bleeding disorders. Birth History — Preterm birth; H/o bleeding in the newborn period. Dietetic History — Strict vegan (B12 def.) ; Iron def. Diet; Duration of breast feeding; Time of cow's milk introduction; Milk protein introlerance; H/o goat's milk intake(folate def.). Drug History - Phenytoin; Methotrexate; Pyrimethamine
Clinical Examinaton –General -Stunted growth; Thin built, marasmic/obese(rare) ; Pallor-nails-bulbar conjunctiva-tongue, Blue line of gums; Eyes-blue sclera; Tongue-papillae are atrophied red painful tongue(pernicious anemia) ; Nails-longitudinal-ridges; Koilonychia; Thins, brittle nails; Bony abnormalities; Triphalangeal thumb(Diamond Blackfan anemia)CVS - Tachycardia; Cardiomegaly; Murmurs; Venous hum;Abd - Splenomegaly; (Mother's Splenomegaly);CNS - Alaxia; Hyporeflexia; Clonus; Babinski positive
Investigations - Blood —Haemoglobin – low; Red cell indices – MCV, MCH, RDW; Retic count – Decrease in Diamond Blackfan, Increased in hemolytic anemia; TC, DC; Neutropaenia; Hypersegmented(>5 segments) nucleated neutrophils>5% - folic acid def. ; Platelet increased in iron def. Anemia; Blood smear – study morphology of RBC, nucleated RBC, basophilic stippling(lead poisoning) ; Decrease in c/c infections; Iron binding capacity – increased in iron def., decrease in c/c injections; Serum ferritin – decrease in iron deficiency; Adenosine deaminase activity in RBC, increased in Diamond Blackfan; Lactate dehydrogenase increased in hemolysis; Serum folate level; Bore Marrow – Aspiration — Morphology of cells; Erythroid hyperplasia(iron def.) ; Meglablastic precurcessor; Absent harmodiderin slain; Ring sideroblasts; Culture – reduced no. of erythrocyte colony forming units; Biopsy if indicated; Urine - Methyy malonic acid excretion orotic acid crystal – orotic aciduria; Schelling test - to confirm absence o intrinsic factor in pernicious an anemia
Approach - Anemia alone or other marrow elements involved. In aplastic anemia only anemia is seen; Is there reticulocytosis? Seen in hemolysis and bleeding; Peripheral smear-Spherocytosis-hereditary spherocytosis, Wilson disese, Autoimmune hemolytic anemia, Sickle cells-sickle cell anemia, Target cells, Nucleated RBC, Microangiopathy, Bite cells-G6PD deficiency—RBC size-,Microcytic(lead poisoning, thalassemia, irondefeciency),Macrocytic anemia(folate deficiency, B122 deficiency, Pearson syndrome , Normocytic-Chronic disease, renal failure, hypothyroidism
Differential Diagnosis-Infancy - Diamond Blackfan; Transient erythro blastopaenia of childhood; Physiological anaemia of infancy; Red cell hypoplasia; Drug induces – eg; ChloramphenicolD/d of Microcytic an anemia - Iron def anemia; Sideroblastic **anemia; Lead poisoning; A trans ferinanimia;D/d** of Megaloblastic anemia - Folic acid deficiency; Abnormalities of folate metabolism; Drug induced eg; Methotrexate, Pyrimethamine; Vitamin B12 deficiency ; Orotic aciduria; Thiamnie responsive anemia;Haemolytic anemias - Membrane defects(spherocytosis, elliptocytosis) ; Enzyme deficiencies, G6PD deficiency; Auto immune haemolytic anemias; Hyper splenism; Hemoglobinopathies(Sickle cell anemia) ; Thalassemias;Pancytopenias - Constitutional/acquired

Management-Iron def. Oral ferrous iron.(sulphate, gluconate, fumarate) ; Parenteral Iron-only in cases of malabsorption; Imrove diet-decrease milk ot 500ml/day, Iron rich foods, combination foods with Vitamin C; Severe anaemia – blood transfusion packed cells; Frank CCF-Exchange transfusion with fresh packed cells
Special situation - Iron chelating therapy; Steroids in Diamond Blackfan; Vitamin B12 supplementaion, Folinic acid supplementation; Folate supplementation in haemolytic anemia; Recombinant human erythropoietin eg- renal failure; Bone marrow transplantation Pancytopenias; Gene therapy
Surgical management; Splenetomy

Quadriplegia
History-History of epidemics; History of Recent Viral Infection; Adequate Immunization; History of cousanguinivity; Fetal movement antenataly; March of evens of paralysis; History of Trauma to spinal cord History suggestive of TB; History of convulsions/altered sensorium; History suggestive of cranial nerve palsies; History of respiratory difficulty; Bladder/bowel disturbances
Clinical Examination-Assessment of higher functions, Cranial nerves, S/o Meningitis, Whether UMN for LMN type of Paralysis, Tone, Involuntary movement, Fundoscopy
Investigations - Apart from routine investigations, NCV studies, Muscle biopsy, CT/MRI of skull and spine, EEG
Differential Diagnosis-Poliomyelitis, G B Syndrome, Spinal dystrophies, Cong. Myopathies, Cerebral palcies hypo and hypertonic, Cord injuries
Non surgical management - Role IV IG, Role of rest, Role of physiotherapy, Nutritional management, Care of bladder and bowl, Prevention of bed sores,. Role of ATT
Surgical Management - Cord decompression, Release of contractures, Selective neurecting

Seizure Disorder
History – Aims- to differentiate Febrile seizure from afebrile seizure and age related childhood seizure disorder like infantile spasm; To differentiate provoked seizure from unprovoked seizure; To differentiate Seizures from Pseudo seizure- Situation of episode, Any triggering factorm, Detailed eye witness account (eye witness should be asked to imitate the actions which he has seen), Onset , Level of consciousness, Presence of aura,Recovery, Speech, Bowel/bladder incontinence, Any injury sustained, Duration of seizure activity, Presence of fever (whether followed the seizure or preceded the seizure), Drug treatment history Any antiepileptic drugs, their dosage and history of discontinuation; Potentially epileptogenic drugs like ciprofloxacin, antimalarials; Drugs with important interactions; History of provoked seizure e.g. drug intoxication - Meningitis / encephalitis, Head Injury, Intracranial hemorrhage, Interruption in treatment, Metabolic events like hypoglycemia, hypocalcaemia, hypomagnesaemia, dyselectrolytemia, hypoxia etc.;Special attention should be given to identify — Absence seizure, Myoclonic seizure,Simple partial , Complex partial seizure; History of heart disease, depression, anxiety, head injury, past h/o febrile seizure ;Family History - H/o of febrile seizure; H/O afebrile seizure; H/O sudden unexpected death; H/O syncope;Social History - Education; Employment; Driving Status; Home situations; Sports; Use of alcoholic drinks
Clinical Examination-Look for episodes of seizure /Pseudo seizure by hyperventilation;Neurocutaneous markers;Dysmorphic features, micro/ macrocephaly;Asymmetry in body size; Cerebral bruit; Tremor; Hair loss; Weight gain; Gum hypertrophy, Hirsutism; Acne, ataxia; Neonate with neurometabolic disorder, failure to thrive, rash, vomiting etc Absent reflexes, visual field defects; Neurodevelopmental delay, cerebral palsy, other neurologic disorders;Long tract signs, focal neurological deficits, raised intracranial pressure; Mental retardation, hyperactivity etc.CVS exam if syncope is considered
*Investigation*s -Hemogram to diagnose evidence of infection,Electrolyte estimation,Metabolic parameters like calcium, magnesium, sugar,EEG, Video EEG, Neuroimaging- MRI is better than CT. Show differentiating features of neurocysticercosis and tuberculoma,Newer modalities like EEG telemetry. MEG. MRI advances. SISCOM

Diffential Diagnosis -Diagnosis of seizure- absence, myoclonic, febrile etc; Movement disorder;Syncopal attack; Migraine

Non Surgical Management - Treatment of acute attack - Emphasis on ABC; Use of Lorazepam, Midazolam, Diazepam, their doses and method of administration; Use of Phenytoin, and then Phenobarb; Frequency of repeating the drugs to abort an attack;Treatment of neonatal seizure - Use of 10% Dextrose bolus and continuous IV, Use of Calcium, Sodium, Magnesium salt, Use of Phenobarb first and then Phenytoin; Treatment of solitary seizure - Whom to treat? First seizure if symptomatic due to etiology like atrophy, inflammatory granuloma, infarct, migration defect;Type of seizure- Choice of drugs depends upon -type of seizure, age, Economic factors, Specific epileptic syndromes;Treatment of febrile seizure-Use of Lorazepam, Midazolam, and Diazepam-IV and per rectal;Method of giving per rectal and doses. Give demonstration;Use of antipyretics and diazepam during fever episode, if recurrent seizure;Treatment of childhood epileptic syndromes — These are age related. Some may have progressive natural course. Some like Benign neonatal convulsion, benign rolandic epilepsy may not require any treatment. Many syndromes evolve with passage of time and diagnosis may have to be revised. West Syndrome- ACTH/Steroids;
SodVallproate;Clonazepa;Vigabatrin; Pyriodoxin;Lennox Gestaut Syndrome- Lamotrigine, Sod alproate, Vigabatrin; Landau Kleffner Syndrome- Sod Valproate, Steroids;Juvenile Myoclonic epilepsy- Sod Valproate
Treatment of inflammatory granuloma, Neurocysticercosis, tuberculoma. Treat seizure disorder with appropriate AED. Treat Neurocysticercosis and tuberculoma as per protocol. Treatment of other seizure disorder; For generalized tonic clonic seizure- Phenyotin,Sod Valproate, Carbamazepine; Partial Seizure- Phenobarbitone, CarbamazepinePhenytoin ; Discuss cost of drugs, their availability, efficacy, side effects before taking final decision. Carbamazepine is a good first choice and superior to Sod Valproate for partial seizure Discuss drug interactions and adjustment of drugs doses. Duration of treatment-Generalized epilepsy-2 years;Partial seizures due to granuloma- 18-24 months;Absence attacks -18-24 months; Symptomatic epilepsy due to underlying disease process like mental retardation,atrophic lesion, post traumatic causes- longer duration. Relapse rate has been found t be varying from 20-30 %. The longer the seizure free duration the greater is the chance of having achieved a cure and lesser the chance for relapse. Tapering is done over a period of 6-12 weeks. Monotherapy / Poly therapy- Follow up- every 3-6 months. Basically to identify side effects of drugs and adjustments of their doses, if required.

Surgical Management - Indication-failure of medical therapy, Intractable epilepsy, Surgical procedure likely to give more benefit in control, Patient is significantly disabled due to seizure; Contraindication- Generalised interictal discharges, Epileptogenic focus inaccessible to surgery, Multiple seizure patterns; Types of surgery- Ant temporal lobectomy for mesial temp sclerosis, Corpus callosotomy for drop attacks, Lesionectomy for tumors, hemartoma, Hemispherectomy

Any Other - Ketogenic diets- Diets rich in fats and with low protein and carbohydrates will produce ketosis like situations, which is beneficial in seizure disorder by reducing its incidence. He has been useful in mentally retarded and handicapped children where compliance can be ensured. Due to high fat children refuse t take such diets.

Hemiplegia

History - Most importance should be given for a proper and accurate history taking, since it gives the clue for a correct diagnosis, in CNS cases, unlike other systems where a proper diagnosis may be made even by clinical findings alone. Following points must be stressed in the presenting complaints- Was the onset acute, sub acute or gradual? Was it associated or preceded with fever and lor a seizure? Any severe headache , vomiting or change in consciousness, preceding the onset of hemiplegia. .If so ,sudden or gradual? Is the condition deteriorating, static or improving? Past history - Any history suggestive of vasculitis, rheumatic fever, trauma or thrombosis or bleeding disorders. Review of developmental history, antenatal, perinatal, and post natal should always be recorded. Family history and consanguinity must be noted.

Clinical Examination - A detailed general examination, including temperature,pulse,BP and anthropometric measurements should be noted. Any facial dysmorphism, cutaneous lesions like cafe au lait spots, angiomas or depigmentation and condition of teeth have to be noted.
Look for any mid line defect. Examination of chest, abdomen and palpation of femoral pulse must always be a part of general physical examination .Note for unusual body order. Palpation of anterior fontanel, and auscultation for bruits done in erect posture- over both globes, temporal fossae, and mastoid region (six sites) CNS examination- Higher function assessment according to the age of the patient.
Cranial nerves. Olfactory(I) rarely assessed, Optic nerve(II)-Elicit he blink reflex,(in a child> 3 mon.old) Fundi examination- optic disc,retinal hemorrhage ,chorioretinitis,
Look for visual acuity, and field of vision depending on the age ,and sensorium of the child.
III,IV, VI Cranial nerves- Enophthalmos ,ptosis ,meosis ,lack of sweating, on the ipsilateral side.offace.. See the pupillary reflex, If ptosis is present, look for 'Marcus Gunn sign,' Test the movements of all extraocular muscles. If the child is unconscious see the 'doll's eye movement. Examine for internuclear ophthalmoplegia by looking for defective adduction of the medial rectus and nystagmus of the abducting eye. See for nystagmus, and if present stage it (stage 1,2,3) V Nerve- Look for deviation of jaw. .corneal and conjunctival reflexes. Test sensation of face. VII Nerve- Is the facial nerve affected. If so ,ipsilateral or contralateral to the side of hemiplegia.. Look for 'emotional 'facial weakness. Confirm whether UMN or LMN type of involvement.. Look at the naso labial fold and wrinkling of fore head. Is the taste ,salivation and lor tear production affected? Look for the strength of orbicularis occuli. Elicit 'Mc Carthy reflex' VIII (Cochlear and Vestibular) Rinne's test for defective hearing. Test for vertigo IX and X - Any nasal regurgitaion or change in voice? Movement of palate. XI Nerve- Test the sterno mastoid .and trapezius muscles XII Nerve-Look for deviation of tongue when protruded, fasciculations when the tongue is inside the mouth ,and lor atrophy. Motor system- Look for abnormal movements. Muscle power (0-5- MRC scale). Tone, pronator sign for upper extremities. and Barre sign. for lower limbs, depending on the age of the child.. Look for coordination by applying specific tests for cerebellar function- Finger nose test,' tapping in a circle (one cm.diameter) test' ,look for rebound phenomenon. Sensory system in older child. Temperature,light touch, crude touch ,based on a dermatomal distribution. Examine for position and vibration sense, and elicit Romberg's sign. Reflexes- Elicit superficial and major tendon reflexes, knowing its root value, and cutaneous nerve involved. Elicit plantar reflex and ankle clonus. Infants- Posture and muscle tone, primitive reflexes- Moro reflex, Tonic neck reflex,Righting - reflex, palmar and plantar grasp reflexes, Vertical suspension, and Landau reflex
Investigations - Besides, routine CBC, Mantoux and X Ray chest ,the following investigations should be done- CSF examination, Electrolyte estimation (Na + ,K+ ,Cl -), PT, APTT, Antiphospholipid antibodies(in SLE) 1 A. NA , Estimation of antithrombin III level, protein C , S,and factor V Leiden. ECG, ECHO, EEG, EMG ,NCV, and CT brain, Cerebral angiogram,or MR angiography(MRA) / Functional MRI , Evoked potentials VERs /BAERs and ,SSEPs), Radiography of Spine and Myelography , Diffusion -Weighted Magnetic Resonance Imaging in acute stroke conditions Magnetic Resonance Spectroscopy (MRS), when cerebral metabolites are of significance. Positron -emission tomography (PET),esp. in children for surgical programmes.. Single -photon -emission computerized tomography (SPECT) for conditions where regional blood flow study is important.
Differential Diagnosis - The following conditions should be kept in mind to arrive at a provisional diagnosis. Of the following, only 3 most probable conditions judged by history and clinical signs must be discussed in differential diagnosis-Hemiplegic cerebral palsy,Infantile hemiplegia (to be considered only in a child up to 6 years), Vascular causes, including IEM (haemarrahage,thrombosis, vasculitis ,trauma, hemiplegic migraine),Infective causes- bacterial, viral,fungal (Meningitis, encephalitis, TBM),Demyelintng- Acute Desseminated Encephalo Myelitis(ADEM),Todd's paralysis also may be considered,ifhemiplegia follows a prolonged seizure, Space occupying lesions
Non-surgical management - Depends on etiology. Measures to reduce cerebral edema, correction of fluid and electrlytes imbalance, if any has to be done. Long term management like physiotherapy,

speech therapy, and rehabilitative measures may be required in some cases. Referral to a child development center (CDC) is advisable for selective .patients.
Surgical management - Surgical management will be required in space occupying lesions .and selective cases of spastic cerebral palsy.
Any other - Site of the lesion,(Localisation) must be discussed before considering the differential diagnosis..

Ear Discharge
History - Name, Age, Sex, Address, Occupation, Brought by, Reliability , Presenting Complaints- Ear - Ear discharge- duration, Insidious,sudden, Progressive,stable, Intermittent,continuous, Color of discharge, Amount – copious, scanty, Smell - foul smell ,not, Aggravating,relieving factor, H/o FB ears; b. Earache-Site, duration, Sudden,insidious,character, Association with otorrhoea,vomiting,giddiness, Radiation, Periodicity, Aggravating, relieving factors; Hearing Loss – Side,duration, Insidious,sudden, trauma, exposure to sound,H/o drug, ingestion (Ototoxic) , Progressive,stable, Associated giddiness;itching in ear- -Side / duration, Association with discharge, HOH, Aggraveting relieving factor; Vertigo- Duration, Periodicity, Associated with nausea,vomiting, Frequency of attack , Aggravated by change in posture, Associated with discharge' HOH;Tinnitus— Side, High pitched,Low pitched;Fever-Associated with headache & chill, Periodicity, With nausea,vomiting;
Hyperacusis, Aautophony, Dip!acusis;Diplopia;Facial weakness; Swelling behind ear; H/o trauma;H/o swimming, diving;Nose-Nasal discharge, Side - duration (Intermittent,continuous, Stable,progressive), Nature – watery, mucoid, purulent,blood stained, FouL smelling / or not , Aggravating, relieving factor; Nasal obstruction- Side, duration, Birth, acquired, Intermittent, persistent, paradoxical- Associated with sneezing, Progressive or not - Aggravating, relieving factors, Mouth breathing,snoring; Headache-Side, duration(Time of onset, Periodicity), Blurring of vision, vomiting (Character – throbbing, piercing, cutting), Aggravating, relieving factor, Associated with ear discharge,bleeding;Anosmia- - Side, duration, Intermittent, Permanent, Associated with sneezing or loss of taste. Sneezing- Duration, With mouth breathing, With rhinorrhea, nasal block, anosmia; Post nasal drip- -Duration, Amount; Allergy- Specific,seasonal ;Throat -Sore Throat-Duration, Recurrent ,not , With fever and dysphagia;Past History - Any prolonged medication for TB, Ototoxic Drugs, Prolonged use of ear drops, nasal drops, Hlo HIV, HIO recurrent URT infection, Hlo measles, whopping cough, Surgical history- Tonsillectomy, adenoidectomy, DNS surgery, myringotomy, myringoplasty, Medical History- Use of antibiotics, analgesics,eardrops, antihistamines, H/o native treatment;Family History - Similar Complaints, Deafness, Overcrowding;Contact History - H/o Contact with known case of TB ;Immunization History - DPT, OPV, BCG, Measles, HIB, Pneumococcal, conjugated, polysaccharide;Dietic history - Total calories expected, Calories deficit, Protein deficit
Examination - General Examination - General Condition WT, HT, Head Circumference, Anemia, BP ,Pulse, Temperature, Nutritional status, Febrile, Cyanosis, Generalized lymph adenopathy, Clubbing, Systemic Examination
Ear - Pinna, Pre auricular and post auricular region, External auditory canal (without speculum, with speculum) , Tympanic membrane (Position, Color, Surface, Mobility) , Middle ear, Mastoid, 3-finger test, Eustation tube, Tragus sign, Fistula test , Tuning fork test (rinne test , weber test, absolute bone conduction) , Ear examination under microscope
Nose - External nose, Vestibule, Anterior rhinoscopy(nasal passage, septum, floor, roof, lateral wall), Posterior rhinoscopy (Choana, Posterior end of turbinate, Discharge in middle meatus, Adenoids, ET opening, tubal tonsils, fossa of rossenmuller), Examination of paranasal sinus, Maxillary sinus (Frontal sinus, Ethmoidal sinus, Sphenoid sinus)

Oral - Lips, Buccal mucosa, teeth, tongue , Hard I soft palate, Tonsils, anterior and posterior, pillar, Posterior pharyngeal wall, Indirect laryngoscopy (IDL Mirror) , CVS,RS,ABDOMEN, CNS examination , Sensorium, Neck rigidity, Kerning's sign, Brudzinski sign, CN palsy
Investigation - Cufture swab and sensitivity, gram stain of ear discharge, Blood culture, TC, DC, Hb, Bleeding time, clotting time, blood grouping, X-ray - mastoid, paranasal sinuses, chest, CT - scan - sinuses, skull (to detect fractures of base of skull) , Audiogram, bera (in children), otoacoustic emission (OAE) , Mantoux test, Sinus endoscopy
Differential Diagnosis - Otitis extema - Severe pain, Tragus sign positive, Serous discharge;Acute suppurative otitis media - Sudden onset of discharge, No previous h/o of discharge, Initially blood tinged but later mucopurulent ;Chronic Supparative otitis media- Safe type - Mucoid discharge, Profuse discharge, Central perforation Unsafe type - Purulent I foul smelling discharge, Scanty amount, Attic or marginal perforation;Tauma - H/o of trauma, Sudden onset, Associated with bleeding;Neoplasia - Glomus jugular, Frank bleeding
Treatment - Otitis externa - I.G. Packing, Aural toilet, Eardrops, Systemic antibiotics and analgesics, Incision and drainage if abscess formation occurs (Furuncle)
Acute Suppurative otitis media - Aural toilet, Eardrops, Systemic antibiotics and analgesics, Surgical - myringotomy
Chronic otitis media - Safe type -Aural toilet , Eardrops, Systemic antibiotics, Treatment of the contributory cause (eg adenoids, tonsils) , Surgical, Aural polyp, snare technique, Myringoplasty with or without ossicle reconstruction. Unsafe type - Surgical - Canal wall down procedure(Atticotomy , Modified radical mastoidectomy, Radical mastoidectomy) ; Canal wall up procedure (Intact canal wall mastoidectomy, Posterior tympanotomy) ; Reconstructive (Myringoplasty, Tympanoplasty); Conservative treatment (Aural toilet)

Respiratory Case (Cough)

History - Onset of cough- Sudden- Foreign body/ Aspiration, Gradual- URI/LRI / Asthma; Nature of cough- Croupy cough, Throat clearing cough- postnasal drip, Dry brassy cough; Cough Timing- during sleep - Tropical Eosinophillia post nasal drip, Bronchospasm, Psychogenic cough disappears with sleep. On awakening - Productive cough, LRI, Cystic Fibrosis; Cough with Feeding- Gastro esophageal Reflux disease, Aspiration, Discoordinated swallow; Seasonal cough- Allergic Rhinitis. Bronchospasm / Asthma; Associated factors- Aggravating factors- Activity, Food intake; Relleving factors- sleep, posture; Associated illness- Cong heart disease, CNS-convulsions , Renal Uremia, Immunological diseases-OI
Clinical Examination - General Examination Anthropometry - Height, Weight, Head and chest Circumference, Clubbing Cyanosis, Pallor, examination of sinusitis Allergic Rhinitis
Examination of Chest - Inspection—Respiratory rate, Chest wall movements, retraction Flaring of nares, Respatory grunt, Stridor, Inter coastal muscle. Palpation- Chest measurements, Chest expansion, VF, VR
Percussion- Dullness, Hyper resonance, Tidal Percussion. Auscultation- Air entry, Breath Sounds and its character , adventitious sounds-rhonchi,rales,crackles
Tan. illumination of chest
Investigations - Radiology- Chest X-Ray/ Fluoroscopy, CT,MRI; Bronchogram, Pulmonary Arteriogram, Aortogram ; Hematological - CBC, ESR ; Microbiological- Sputum, gastric or bronchial lavage, Culture a Sensitivity ; Purified Protein Derivative (PPD) ; PuImonary Function Testing; Blood Gas analysis; Endoscopic studies- Laryngoscope, Bronchoscope, Thoracoscopy; Thoracocentesis ; Lung BiopsyI Pleural biopsy; Sweat test

Differential Diagnosis- Asthma, Gastro esophageal reflux, Post-viral airway hyper-responsiveness, Post-nasal drip, Congenital malformation, Tuberculosis, Foreign body, Bronchiectasis, Environmental pollution, Psychogenic cough, Cystic fibrosis
Non-Surgical Management - Supportive therapy - Oxygen, bronchial lavage, Medical- Bronchodllators, Anti microbials, Antitussives
Surgical Management - Bronchoscoplc aspiration or removal of foreign body or mass, Lobectemy / Pneumenectomy,
Any Other - Chest Physiotherapy, Prophylaxis -Immunization, Chemoprophylaxis ,Patient education

Cerebral Palsy
History- The candidates should be very clear about the duration of the illness - present illness. A patient with recurrent episodes of ahnost identical illnesses with symptomfree intervals - the last episode to be described in full and others included under the heading of past illness. All events (including treabnent) should be described in the chronological order - as the disease develops Describing each symptom separately is not desirable.Illnesses which has origins in the neonatal period to be described from birth e.g. birth asphyxia with seizures leading to Cerebral palsy. Describe in detail only relevant history. Discredit for spending too much time on irrelevant history. All aspects of history to be discussed and documented. At postgraduate level they must know which is relevant and which is irrelevant.At the end of history the candidate should be able to draw some conclusions regarding the problem e.g. Degenerative diseases of brain Intracranial space occupying lesion Chronic liver disease
Physical Examination - General physical examination (Observation about general appearance, Diagnostic clues - positive and negative);Growth - Detailed measurements only in growth problems. Development - Details only in cases of developmental delay. System involved - Give all details. 5.Other systems - Positive findings only.
Summary - The candidates should summarize the case in 4 or 5 sentences - only relevant history I positive findings and important negative findings
Diagnosis - Complete diagnosis to be given, Substantiate the diagnosis based on history, findings and course of the disease - both positive and negative, Differential diagnosis only if there are valid reasons, Candidates should state why the DD is suggested,
Investigations - Relevant investigations only, The candidates should know why he is suggesting a particular investigation.
Treatment - Essential treatment only, Rationale of treatment should be explained

Feedback form for participants

Speciality_____Date for workshop_____Venue_____

You are requested to give your views on the various aspects of this workshop'. Please put a tick mark [ ] on one of the three [3] choices in each question given below which indicates your opinion. Your reaction will be of great value to us in terms of improving the workshops in future. Therefore, you are requested to give honest response [You need not write your name. Your answers will be kept confidential].

1. How do you rate the relevance of the objectives?

   [1] Irrelevant [2] Fairly relevant [3] Quite relevant

2. How far have the objectives been achieved?

   [1] Achieved meagerly [2] Achieved to some extent [3] Achieved fully

3. Name the sessions that you found most appropriate.

   [1]

   [2]

   [3]

4. To what extent were you given opportunity of active involvement in the workshop?

   [1] Very little [2] Fair [3] Considerable

5. Name the sessions that you liked the best and the least. Give reasons.

   [1] Sessions liked the bestand reasons
   [2] Sessions liked the least and reasons

6. How do you rate the quality of handouts and background materials?

[1] Poor [2] Satisfactory [3] Excellent

Give your suggestions to improve the same.

7. How far will the workshop help you?

[1] Meagerly [2] To some extent [3] Considerably

8. Mention the sessions that you feel should be omitted in the future workshops.

[1]

[2]

[3]

9. Mention the sessions that you feel should be added in the future workshops.

[1]

[2]

[3]

10. Was the programme duration adequate?

[1] Inadequate [2] Somewhat adequate [3] Quite adequate

11. If the workshop is repeated again would you like to- [Yes/No. Reasons]

[1] Suggest it to your colleagues
[2] Nominate subordinates

12. Please give some suggestions for the improvement of content and methodology of the workshop of same duration.

[1]

[2]

[3]

Any other comments or suggestion?

[1]

[2]

[3]

Letter to myself

To

Dear Dr.

I have attended the CME programme , once I go back to my hospital/ institution, I would do the followings activities to enhance my competencies

1. I will form DNB club with my colleagues for regular study.
2. I will form a plan for regular self study.
3. I will maintain a diary for recording new things I learn during my clinical posting.
4. I will clear my DNB examination in FIRST ATTEMPT
5. I will aim to GET GOLD MEDAL in the specialty

Yours sincerely,

( )
(Name of the candidate)