WBUHS (2008-2020)

3rd Prof. M.B.B.S, Part - II

Paper – I I

Answers of Surgery Question Papers for M.B.B.S Students

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Subject: Surgery **Time: 2 1/2 hrs.**

Paper: II **Marks: 60**

Attempt all questions

Group -A

1. **Write down the effect of prostatic hypertrophy on urethra and urinary bladder. Mention the medical and surgical treatment of benign prostatic hypertrophy. 5+5+5**

Group - B

2. **Answer any one of the following:**

a) **A 30 year old lady presents with 3cm solitary nodule on right thyroid lobe. Give the differential diagnosis. How will you manage such patient? 5+10**

b) **A middle aged bus conductor presents with non healing ulcer and pigmentation in left lower leg near medial malleolus. How will you examine, investigate and manage this patient? 5+5+5**

Group - C

3. **Write short notes on any three of the following: 3x5**
   a) **Branchial fistula.**
   b) **Regional anaesthesia.**
   c) **Endotracheal intubation.**
   d) **Undescended testis.**
   e) **Complications of chemotherapy.**

Group - D

4. **Write short notes on any three of the following: 3x5**
   a) **I.V.U**
   b) **Lucid interval.**
   c) **Cleft palate.**
   d) **Warthin's tumour.**
   e) **Varicocele.**

Answers.

1. **Effect of prostatic hypertrophy on urethra and urinary bladder:**

BPH is the most common cause of lower urinary tract symptoms (LUTS), which are divided into storage, voiding, and symptoms which occur after urination.

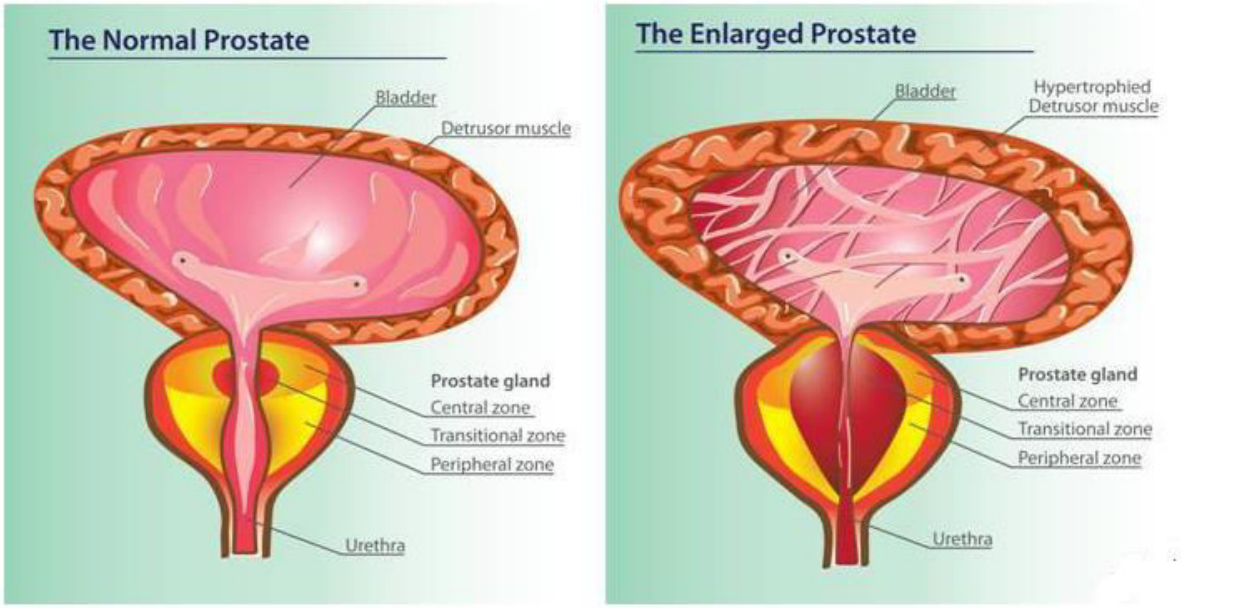
Storage symptoms include the need to urinate frequently, waking at night to urinate, urgency (compelling need to void that cannot be deferred), involuntary urination, including involuntary urination at night, or urge incontinence (urine leak following a strong sudden need to urinate).

Voiding symptoms include urinary hesitancy (a delay between trying to urinate and the flow actually beginning), intermittency (not continuous), involuntary interruption of voiding, weak urinary stream, straining to void, a sensation of incomplete emptying, and terminal dribbling (uncontrollable leaking after the end of urination, also called post-micturition dribbling).These symptoms may be accompanied by bladder pain or pain while urinating, called dysuria.

Bladder outlet obstruction (BOO) can be caused by BPH. Symptoms are abdominal pain, a continuous feeling of a full bladder, frequent urination, acute urinary retention (inability to urinate), pain during urination (dysuria), problems starting urination (urinary hesitancy), slow urine flow, starting and stopping (urinary intermittency), and nocturia.

BPH can be a progressive disease, especially if left untreated. Incomplete voiding results in residual urine or urinary stasis, which can lead to an increased risk of urinary tract infection.

BPH most often occurs during this second growth phase. As the prostate enlarges, it presses against the urethra. The bladder wall becomes thicker. One day, the bladder may weaken and lose the ability to empty fully, leaving some urine in the bladder.



Diagrammatic representation of BPH with the enlarged prostate transition zone causing obstruction of the prostatic urethra and the secondry changes in the bladder leading to hypertrophy of the detrusor muscle

Treatment:

Medical therapy: as follows

Alpha-blockers **These medications relax the smooth**	**5-alpha-reductase inhibitors** **These medications block the**	**Other drugs** **Phosphodiesterase 5**

muscles of the prostate and bladder neck to improve urine flow and reduce bladder blockage.	**production of DHT, which accumulates in the prostate and may cause prostate growth**	**inhibitors**
Nonselective • **Phenoxybenzamine 10 mg twice a day** **Alpha-1, short-acting** • **Prazosin 2 mg twice a day** **Alpha-1, long-acting** • **Terazosin 5 or 10 mg daily** • **Doxazosin 4 or 8 mg daily** **Alpha-1a selective** • **Tamsulosin 0.4 or 0.8 mg daily** • **Alfuzosin 10 mg daily** • **Silodosin 4 or 8 mg daily**	**Finasteride 5 mg daily** **Dutasteride 0.5 mg daily** **Subcutaneous implant Yearly** **Triptorelin pamoate 3.75 mg every month**	**Tadalafil**

Surgical indications include:

- **Refractory urinary retention (failing at least one attempt at catheter removal),**
- **Recurrent urinary tract infection from BPH,**
- **Recurrent gross hematuria from BPH,**
- **Bladder stones from BPH, renal insufficiency from BPH, or**
- **Large bladder diverticula.**

Conventional Surgical treatment:

- **Transurethral resection of the prostate: best option.**
  - **Risks of TURP: include retrograde ejaculation (75%), impotence (5–10%), and incontinence (<1%). Complications include bleeding, urethral stricture or bladder neck contracture, perforation of the prostate capsule with extravasation, and if severe, TUR syndrome resulting from a hypervolemic,hyponatremic state due to absorption of the hypotonic irrigating solution.**
  - **Clinical manifestations of the TUR syndrome include nausea, vomiting, confusion, hypertension, bradycardia, and visual disturbances. The risk of the TUR syndrome increases with resectiontimes >90 minutes.**
  - **Treatment includes diuresis and, in severe cases, hypertonic saline administration.**

- **Transurethral incision of the prostate—Men with moderate to severe symptoms and a small prostate often have posterior commissure hyperplasia (elevated bladder neck). These patients will often benefit from an incision of the prostate. This procedure is more rapid and less morbid than TURP.**
- **Open simple prostatectomy—When the prostate is too large to be removed endoscopically, an open enucleation is necessary.Glands >100 g are usually considered for open enucleation.**
  - **A simple suprapubic prostatectomy is performed transvesically and is the operation of choice in dealing with concomitant bladder pathology.**
  - **In a simple retropubic prostatectomy, the bladder is not entered.**

Minimally Invasive Therapy: Laser therapy—Many different techniques of laser surgery for the prostate have been described.

Two main energy sources of lasers have been utilized—Nd:YAG and holmium:YAG.

2. a)

Differential diagnosis of apparent solitary thyroid nodules:

<table>
<tr><th colspan="2">1) Benign thyroid neoplasms</th><th>2) Malignant thyroid neoplasms</th><th>3) Other thyroid abnormalities</th><th>4) Nonthyroid lesions</th></tr>
<tr><td rowspan="4">a) Follicular adenoma
i) Colloid
ii) Simple
iii) Foetal
iv) Embryonal
v) Hurthle cell</td><td>b) Papillary adenoma</td><td rowspan="4">a) Papillary carcinoma
b) Follicular Carcinoma
c) Medullary carcinoma
d) Anaplastic carcinoma
e) Metastatic cancer
f) Sarcoma
g) Lymphoma</td><td rowspan="4">a) Thyroiditis
b) Thyroid cyst
c) Infections
d) Granulomatous disease (e.g., sarcoidosis)</td><td rowspan="4">a) Lymphadenopathy
b) Thyroglossal duct cyst
c) Parathyroid adenoma
d) Laryngocele</td></tr>
<tr><td>c) Teratoma</td></tr>
<tr><td>d) Lipoma</td></tr>
<tr><td>e) Dermoid cyst</td></tr>
</table>

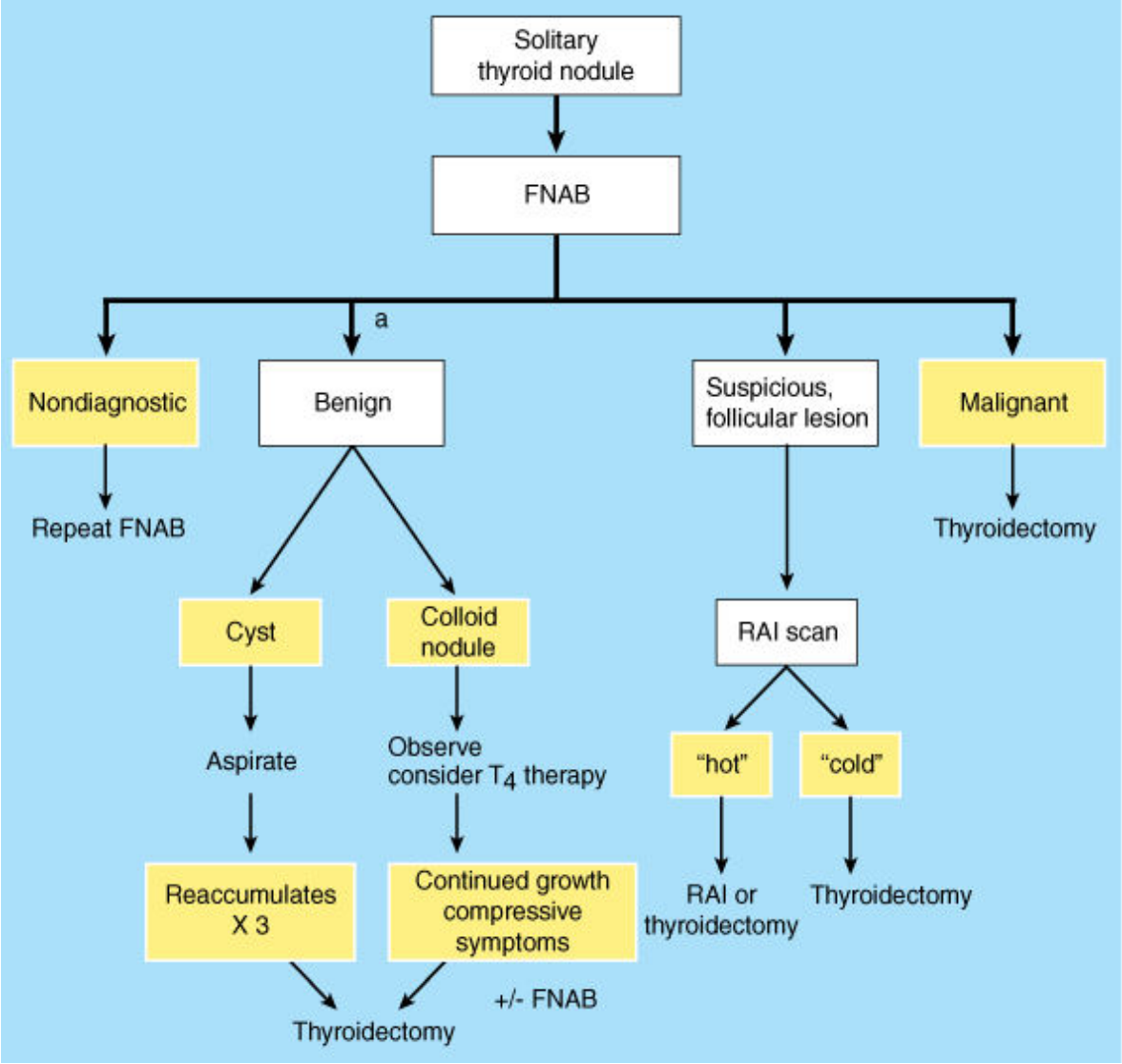
Diagnostic Tools of Solitary Thyroid Nodule:

<table>
<tr><td>Clinical examination</td><td rowspan="2">Radiological studies:
• Neck ultrasonography
• Isotope scanning of the thyroid
• CT scan</td><td rowspan="2">Histopathological studies:
• Fine needle aspiration cytology
• Frozen section .
• Final histopathological examination.</td></tr>
<tr><td>Laboratory studies:
T3,T4,TSH</td></tr>
</table>

Treatment of the Solitary Cold Thyroid Nodule:

Non-Surgical:	Surgical:
-No treatment, just follow-up by FNAC -Hormone suppressive therapy -Aspiration of a cyst -Ethanol injection -Recently, Laser photocoagulation	-Isthmo-lobectomy -Near total thyroidectomy -Total thyroidectomy

Workup of a solitary thyroid nodule:



2. b) Diagnosis of this case is venous ulcer.

Examination of the leg should for this patient:

- **Palpation of pulses**
- **Signs of venous disease**
  - **Brawny skin**
  - **Haemosiderin pigmentation**
  - **Varicose eczema**
  - **Atrophie blanche (patchy areas of ischemia)**
  - **Lipodermatosclerosis.**
- **Signs of arterial disease**
  - **Shiny, hairless, pale and cool skin**
- **Ulcer examination - describe where possible for every ulcer:**
  - **Position**
  - **Colour**

    - **Tenderness**
    - **Temperature**
    - **Shape**
    - **Size**
    - **Specific to the ulcer:**
        - **Base**
        - **Edge**
        - **Depth**
        - **Discharge**
        - **Relationship to other structures**
        - **Lymph nodes**
    - **State of local tissues, including pulses - if cannot feel pulses then use Doppler**
- **Surrounding region**
    - **For pain, oedema, erythema, warmth, induration, discoloration, maceration, dryness, scarring from previous wounds, hair pattern, gangrenous digits, clubbing, cyanosis, capillary refill, and varicose veins.**

Investigations: The evaluation of patients with venous ulceration primarily includes noninvasive methods to elucidate the distribution and extent of pathology. Duplex ultrasound is the first line of investigation, as it provides assessment of both reflux and obstruction conditions. In patients with iliofemoral pathology, axial imaging with computed tomography scan or magnetic resonance imaging should be performed. If the treatment of iliofemoral vein obstruction is warranted, then invasive assessment using venography and/or intravascular ultrasound should be used to guide the interventional procedure. Venous valve reflux can be identified and accurately characterized by duplex ultrasound, whereas the ultrasound assessment of functional abnormality associated with obstruction is less reliable. In patients with ulceration, the evaluation for and treatment of proximal venous obstruction has resulted in improved ulcer healing.

A venous leg ulcer can be susceptible to bacterial infection. Symptoms of an infected leg ulcer can include:

- **Worsening pain**
- **A green or unpleasant discharge coming from the ulcer**
- **Redness and swelling of the skin around the ulcer**
- **A high temperature (fever)**

Nonsurgical Treatment

- **Infected ulcers**

- **Necessitate treatment of the infection first.**
- **Staphylococcus aureus, Streptococcus pyogenes, and Pseudomonas species are responsible for most infections.**
- **Usually treated with local wound care, wet-to-dry dressings, and oral antibiotics.**
- **Topical antiseptics should be avoided.**
- **Severe infections require intravenous antibiotics.**

❖ **Leg elevation**

Leg elevation can temporarily decrease edema and should be instituted when swelling occurs. This should be done before a patient is fitted for stockings or boots.

❖ **Compression therapy**

- **Compression therapy is the primary treatment for CVI.**

- **Elastic compression stockings**
  - **Fitted to provide a compression gradient from 30 to 40 mm Hg, with the greatest compression at the ankle.**
  - **Donned on arising from bed and removed at bedtime.**
  - **Effective in healing ulcers but can take months to obtain good results.**
  - **Stockings do not correct the abnormal venous hemodynamics and must be worn after the ulcer has healed to prevent recurrence.**
  - **Principal drawback is patient compliance.**
- **Unna boots**
  - **Paste gauze compression dressings that contain zinc oxide, calamine, and glycerin.**
  - **Used to help prevent further skin breakdown.**
  - **Provide nonelastic compression therapy.**
  - **Changed once or twice a week.**
  - **Healing time for ulcers is less than that of elastic compression alone.**
- **Pneumatic compression devices**
  - **Provide dynamic sequential compression.**
  - **Used primarily in the prevention of deep vein thrombi in hospitalized patients.**
  - **Also used successfully to treat venous insufficiency.**

❖ **Topical medications**

- **Largely ineffective as a stand-alone therapy for venous stasis ulcers.**
- **Topical therapy is directed at absorbing wound drainage and avoiding desiccation of the wound.**
- **Antiseptics can be counterproductive. Hydrogen peroxide, povidone-iodine, acetic acid, and sodium hypochlorite are toxic to cultured fibroblasts and should be used for the shortest duration necessary to control ulcer infection.**

❖ **Surgical Therapy: Skin grafting.**

3. a) Branchial fistula

Branchial cleft fistulas (BCF) originate from the 1st to 3rd branchial apparatus during embryogenesis of the head and neck.

- **Anomalies of the 2nd branchial cleft are by far the most commonly found.**
- **They can be a cyst, a sinus tract or fistulas.**
- **Fistulas (or sinus tract if they end blindly) display themselves as small cutaneous opening along the anterior lower third border of the sternocleidomastoid muscle, communicates proximally with the tonsillar fossae, and can drain saliva or a mucoid secretion.**

- **Management consists of excision since inefficient drainage may lead to infection.**

❖ **1st BCF are uncommon, located at the angle of the mandible, and communicating with the external auditory canal. They have a close association with the fascial nerve.**
❖ **3rd BCF are very rare, run into the piriform sinus and may be a cause of acute thyroiditis or recurrent neck infections.**

b) Regional anaesthesia

Introduction:

- **Local anesthetic applied around a peripheral nerve at any point along the length of the nerve (from spinal cord up to, but not including, the nerve endings) for the purposes of reducing or preventing impulse transmission**
- **No CNS depression (unless overdose (OD) of local anesthetic); patient conscious**
- **Regional anesthetic techniques categorized as follows:**
  - **Epidural and spinal anesthesia**
  - **Peripheral nerve blockades**
  - **IV regional anesthesia**

Physiology:

- **Physiologic response to central blockade is determined by the effects of interrupting the afferent and efferent innervation of somatic (sensory and motor innervation) and visceral (autonomic nervous system).**
- **Somatic blockade:**
  Prevention of pain.
  Skeletal muscle relaxation.

Nerve fiber classification.

Class	Action	Myelin	Size	C_m
Aα	Motor	Yes	++++	++++
Aβ	Light touch, pressure, pain	Yes	+++	+++
Aγ	Muscle spindles, (proprioception)	Yes	+++	++
Aδ	Pain, temperature	Yes	++	+
B	Preganglionic sympathetic fibers	Yes	++	+
C	Pain, pressure	No	+	+++

- **Fibers blocked more easily small and myleinated.**
- **Less easily – large – unmylinated.**

Indications	Contraindications	Complications
❑ Avoidance of some of the dangers	❑ Allergy to local anesthetic	❑ Failure of technique

of general anesthesia (e.g. known difficult intubation, severe respiratory failure, etc.) ❑ Patient specifically requests regional anesthesia ❑ For high quality post-op pain relief ❑ General anesthesia not available	❑ Patient refusal, lack of cooperation ❑ Lack of resuscitation equipment ❑ Lack of IV access ❑Coagulopathy ❑ Certain types of preexisting neurological dysfunction ❑ Local infection at block site	❑ Systemic drug toxicity due to overdose or intravascular injection ❑ Peripheral neuropathy due to intraneural injection ❑ Pain or hematoma at injection site

Spinal vs epidural anesthesia:

SPINAL	EPIDURAL
• Easier to perform • Smaller dose of LA required (usually < toxic IV dose) • Rapid blockade (onset in 2-5 minutes) • Very effective blockade Hyperbaric LA solution - position of patient important	• Technically more difficult; greater failure rate • Larger volume/doses of LA (usually > toxic IV dose) • Significant blockade requires 10-15 minutes • Effectiveness of blockade can be variable • Use of catheter allows for continuous infusion or repeat injections • Slower onset of side effects • Position of patient not as important • SG of LA solution not as important

- **Peripheral nerve blockade:**
  - **The use of ultrasound for regional anesthesia is relatively new.**
  - **Ultrasound guided regional anesthesia have largely focused on brachial plexus blockade in the interscalene, supraclavicular, infraclavicular and axillary regions.**
  - **Recent studies examining the efficacy of ultrasound guidance for femoral, sciatic, psoas compartment, celiac plexus and stellate ganglion blocks are promising, while ultrasound visualization of the epidural space can facilitate neuraxial blockade in children, adults and parturients.**

3. c) Endotracheal intubation: Endotracheal intubation is the placement of a tube into the trachea, either orally or nasally for airway management. Endotracheal tube forms an open passage in the upper airways. To be able to ventilate the lungs, the air must be free to enter and exit the lungs. The patient is connected to the mechanical ventilator to provide continuous respiration with an endotracheal tube.

Endotracheal intubation can be performed under emergency conditions in the following circumstances:

- **Apnea, respiratory failure.**
- **Airway obstruction: variable-level obstruction in the upper and lower airways.**
- **Inadequate oxygenation (hypoxia), inadequate ventilation (hypercarbia).**

- **Disruption of the airway reflex.**
- **In case the patient is hemodynamically unstable.**
- **The consciousness changes as far as being unable to protect airway (GCS <8).**
- **Cardiopulmonary resuscitation.**
- **Flail chest/pulmonary contusion, in case the breathing effort puts the patient's life in danger. In case the treatment of patient is not successful without intubation.**

In urgent conditions, nasal, oral, awake, fiber optic, and rigid intubation and, if necessary, intubation through the laryngeal tube can be technically applied, and the choice of method is decided according to the patient's clinical condition.

Comparison of intubation performed under emergency conditions and intubation performed under elective conditions.

Emergency	Elective
Difficult intubation may not be predictable.	**Predict airway difficulty.**
There may not be enough time for preparation.	**Prepare equipment and assistants for intubation.**
It can be difficult to reach experienced personnel.	**Confirm availability of help in an emergency.**
There may be a risk of full stomach/aspiration.	**Safely perform the intubation.**
Patient status may not be stable.	**Patient is more stable than emergency situations.**

Intubation risks:

There are some risks related to intubation, such as:

- **Injury to teeth or dental work**
- **Injury to the throat or trachea**
- **A buildup of too much fluid in organs or tissues**
- **Bleeding**
- **Lung complications or injury**
- **Aspiration (stomach contents and acids that end up in the lungs)**

3. d) Undescended testis;

Key facts:

- **Testicular descent from the fetal abdominal site into the scrotum is normally complete by birth.**
- **Absence of a scrotal testis (cryptorchidism) may be due to agenesis (rare), intraabdominal arrest, incomplete descent (intracanalicular), or ectopic descent (inguinal, perineal, crural, penile).**
- **Incidence: 2-4% of newborn boys falling to 1.5% at 6 months.**
- **Commoner on the right side.**

Clinical features:

- **Undescended testis can be noted at the postnatal check, by parents, or by the GP.**
- **Rarely presents acutely as torsion (tender mass in inguinal region).**
- **A retractile testis is one that can be brought down into the scrotum with gentle manipulation but retracts into the superficial inguinal pouch either spontaneously or with minor pressure.**

Diagnosis and investigations:

- **No investigations are required in palpable undescended testis.**
- **Chromosomal studies and HCG stimulation test may be requested in bilateral impalpable testes.**
- **Ultrasound may help locate an impalpable testis.**
- **Diagnostic laparoscopy is definitive and allows further management.**

Treatment:

- **Testis should be brought to the scrotum at 1-2 years of age to avoid secondary damage due to trauma, torsion, and increased ambient temperature.**
- **Hormone manipulation is ineffective in true undescended testis.**
- **Intracanalicular or ectopic testis should be managed by one-stage orchidopexy.**
- **Intraabdominal testis can be brought down by one- or two-stage orchidopexy (50-90% success).**
- **Laparoscopy for bilateral impalpable testes.**
- **Scrotal position facilitates self-examination to detect signs of neoplastic change (~4 x normal in an abdominal testis).**

Complications:

- **Postoperative atrophy of the testis (< 2%) unless intraabdominal position (10-50%).**
- **Retraction.**

Indications for orchidopexy:

- **Maximize sperm production**
- **Prevent testicular torsion**
- **Repair of associated inguinal hernia**
- **Cosmesis**
- **Reduce chance of malignancy development and improve self-examination success.**

Complications of undescended testis:

- **Infertility:**
  - **Fertility is lower in individuals with UDT than those with normally descended testis and paternity rates are especially lower in those with bilateral UDT.**
  - **In the latter, even when orchidopexy is done early, paternity rates of only 23-50% are noted.**

    {The causes of impaired fertility in these patients are:-
    - **Testicular atrophy of UDT with increasing age (38% of testes are smaller than their counterparts by Tanner Stage 5 puberty).**
    - **Histopathological changes in UDT i.e. lack of normal enlargement of seminiferous tubules, decrease in ratio of spermatogonia per tubule, delay in transformation of gonocytes to spermatogonia and lack of proliferation of Leydig cells and their atrophy, Sertoli cell abnormalities and interstitial fibrosis.**
    - **Congenital anomalies of ductal system frequently seen in patients with UDT e.g. epididymal abnormalities.}**
- **Malignancy:**
  - **About 10% of all germ cell testicular tumors occur in cryptorchid testis.**
  - **The risk of developing tumor in intraabdominal testis is 6 folds higher than in cryptorchid testis at other locations.**
  - **The tumor develops in 20% of the contralateral descended testis in a cryptorchid patient.**
  - **There is a 25% chance of developing tumor in opposite cryptorchid testis if one cryptorchid testis develops tumor.**
  - **Cancers arising in uncorrected abdominal testes are seminomatous, while those originating following successful orchidopexy, regardless of their original location,are non seminomatous germ cells tumor.**
  - **Also, orchidopexy does not decrease the risk of testicular cancer.**
  - **The presentation of testicular tumors occurs mainly in the third decade.**

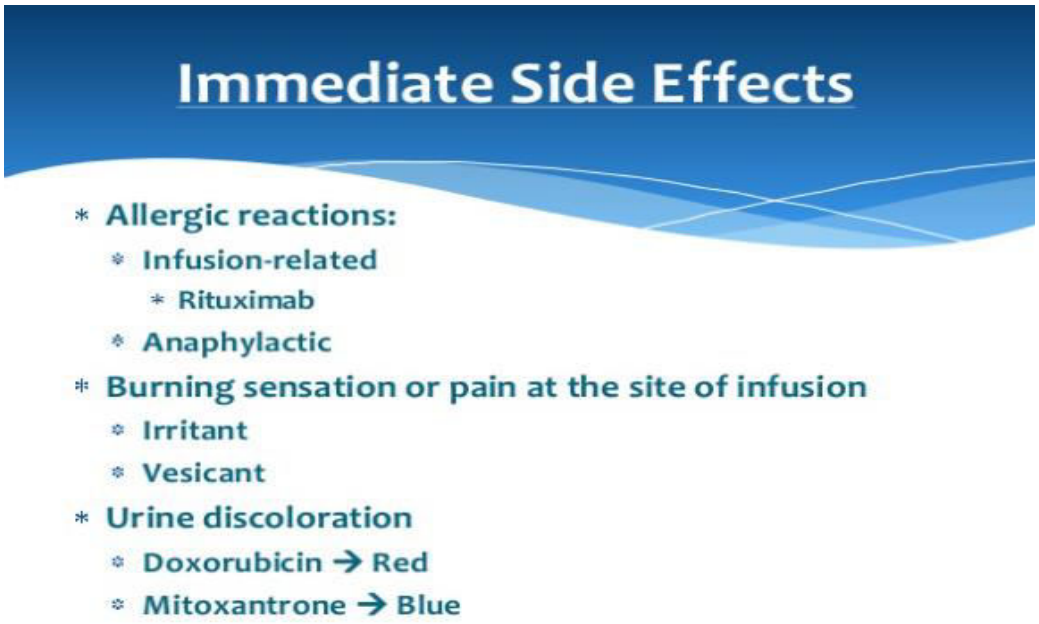
  - **Cause for development for malignancy:**
  - **Progressive degeneration of germ cells with secondary dysplasia seen in cryptorchid testis due to their extra scrotal position that remains at a higher temperature than the inguinal/ abdominal testis.**
  - **Intrinsic abnormality of testis rather than secondary dysplasia(theory of dysgenetic germplasm)**
  - **Altered hormonal milieu during intra-embryonic life.**

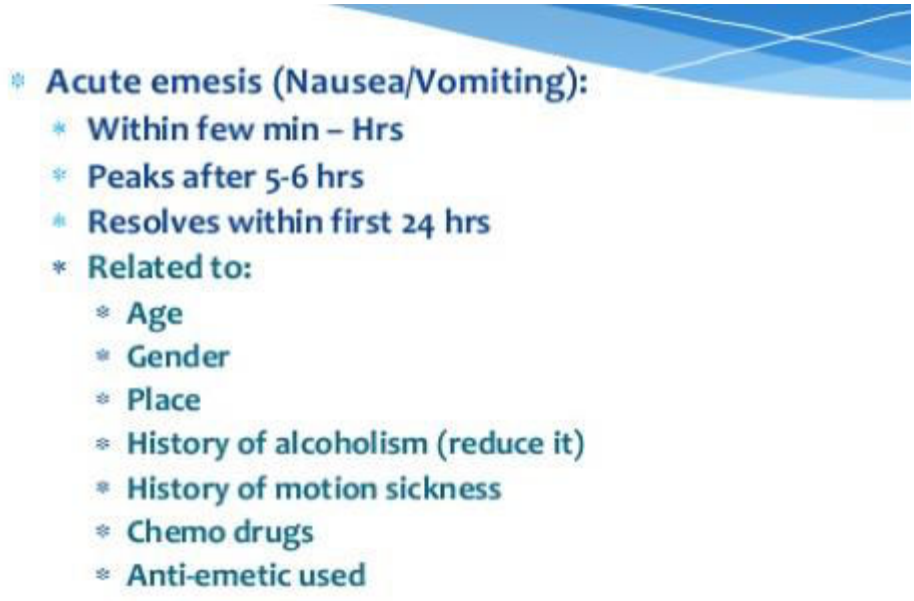
  - **Carcinoma- in situ (CIS) and malignant transformation:**
    - ✓ **Carcinoma-in-situ is a premalignant condition and has been found 2-3% of testicular biopsies performed in adult patient who underwent orchidopexy when young.**
    - ✓ **The occurrence of CIS is associated with an increased risk of carcinoma; especially if contralatarel testis already harbours tumor.**

- **Torsion of testis: The anatomic abnormalities I,e. high investment of investing tunica vaginalis and epididymal abnormalities makes testis mobile and predisposes it for testicular torsion.**

- **Trauma: Inguinal testes are especially at a greater risk of direct trauma. This is most commonly seen in children with cerebral palsy using wheelchair braces.**
- **Psychological aspects: These are related to an empty scrotum. Peer ridicule, impairment of body image and fear of sterlity are important negative responses that cause worry to the patient and his parents. These aspects become all more important near puberty.**
- **Inguinal hernia: Patent processus vaginalis is commonly associated with UDT; in more than 65% patients. The incidence is slightly lower for ectopic undescended testes. Repair of associated hernia is important to prevent future complications and orchidopexy is done simultaneously.**

3. e) Complications of Chemotherapy:



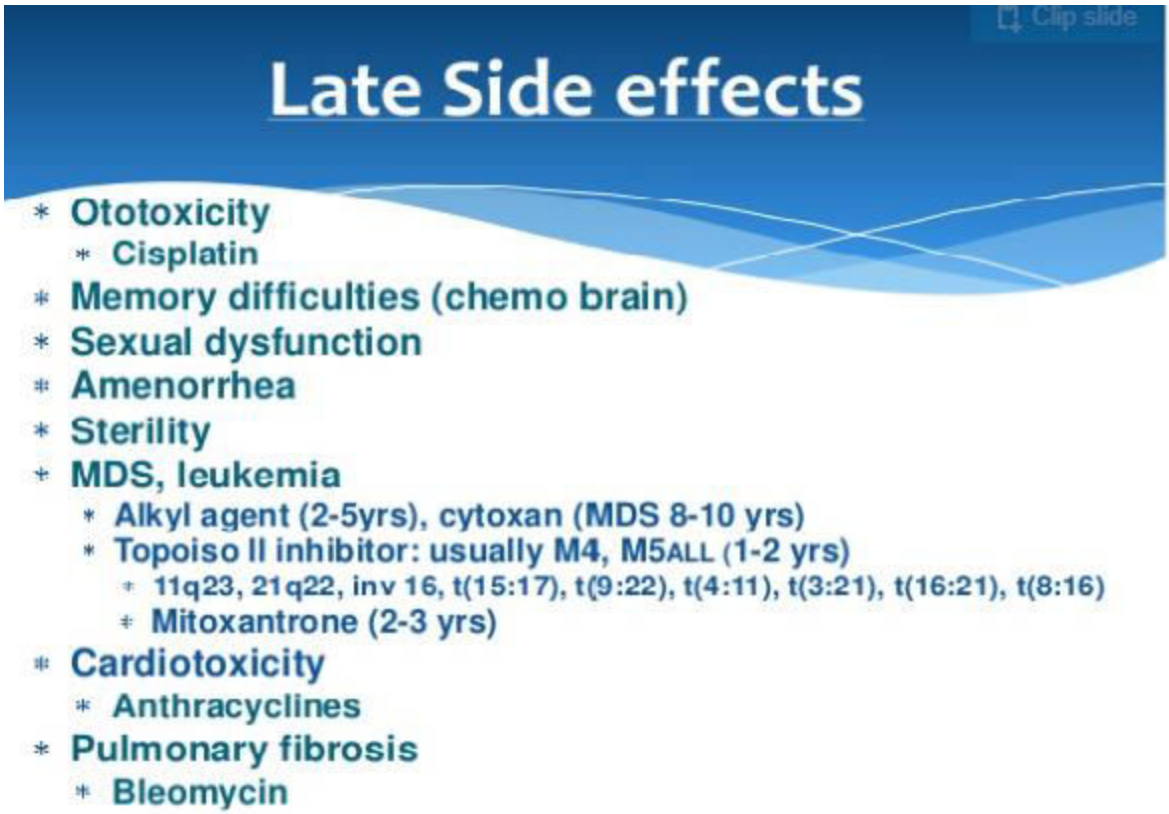


Within days

- Delayed-onset emesis:
  - > 24 hrs after chemo – 7 days
  - Related to types of chemo drugs (Platinum, Cytoxan, Doxo)
- Fatigue
- Myelosuppression:
  - During the nadir of chemo
  - Mucositis
  - Neuropenic fever +/- infection
- Diarrhea or Constipation
- Reduced appetite
- Metallic taste

Within weeks

- Hair loss (Alopecia)
  - Taxanes, Cisplatin, Doxo
- Peripheral neuropathy
  - Pacltaxel, Oxalipatin, Cisplatin
- Dry skin or pigmentaion
- Nail changes
- Fluid retention
  - Docetaxel



4. a) IVU - also referred as intravenous pyelography (IVP) or excretory urography (EU), is a radiographic study of the renal parenchyma, pelvicalyceal system, ureters and the urinary bladder. This exam has been largely replaced by CT urography.

Terminology

Some prefer the term "urogram" to refer to visualization of the kidney parenchyma, calyces, and pelvis after intravenous injection of contrast, and reserve the term "pyelogram" to retrograde studies involving the collecting system. In practice, both terms are often used interchangeably.

Procedure

Indications

- **check for normal function of kidneys**
- **check for anatomical variants or congenital anomalies (e.g.horse-shoe kidney)**
- **check the course of the ureters**
- **detect and localise a ureteric obstruction (urolithiasis)**
- **assess for synchronous upper tract disease in those with bladder transitional cell carcinoma (TCC)**

Patient preparation

- **overnight fasting prior to the date of examination; a laxative would help to achieve a good preparation**
- **on the day of procedure take a scout/pilot film to check patient preparation and also for radio-opaque calculi**
- **check serum creatinine level to be within normal range (as per hospital guidelines)**
- **take a history of the patient for any known drug allergies followed by a written informed consent for the procedure**

Technique

Exposures are generally in the 65-75 kV range, mA of 600-1000, with exposure of <0.1 sec. Higher kV ranges reduce contrast of the renal parenchyma.

- **IV access is required for administration of a water soluble contrast**
  - **nonionic contrast is preferred**
- **dose will vary as per the weight of the patient; generally up to 1.5 ml/kg body weight is well tolerated by patient**
- **the contrast dose is usually instilled at a fast (bolus) rate**
- **the calyces are usually visualized in <2 minutes following contrast administration - this is the nephrogram**
- **serial images are taken at 5-20 minutes for visualisation of the pelvicalyceal systems and ureters when required and with operator preference**
- **additional views taken are prone and obliques for ureters**
- **the full length 10-15 minute film is performed with a compression band applied to the patient**
  - **compression should not be applied if ureteral calculi, ureteral obstruction, recent surgery, nephrostomy, or abdominal aortic aneurysm is suspected**
- **lastly take a full bladder and post void film**

There is a wide variation in protocols. One protocol is suggested below, but additional images should usually be obtained to answer the clinical question:

- **Scout images**
- **(1-2 minutes) Nephrogram**
- **(>3 minutes) Early and late images of the upper collecting system (abdominal compression then applied)**
- **Tomography may be obtained, if desired**
- **(10-15 minutes) Supine, after release of compression, images of the upper collecting system and proximal ureters**
- **(20 minute) Supine image**
- **(20 minute) Prone image**

Emergency medications and emergency equipment must always be available in case the patient has a reaction to contrast.

4. b) In emergency medicine, a lucid interval is a temporary improvement in a patient's condition after a traumatic brain injury, after which the condition deteriorates.

- **A lucid interval is especially indicative of an epidural hematoma. An estimated 20 to 50% of patients with epidural hematoma experience such a lucid interval.**
- **The lucid interval occurs after the initial concussive force of the trauma, then lapses into unconsciousness again after recovery when bleeding causes the hematoma to expand past the extent for which the body can compensate.**
- **After the injury, the patient is momentarily dazed or knocked out, and then becomes relatively lucid for a period of time which can last minutes or hours. Thereafter there is rapid decline as the blood collects within the skull, causing a rise in intracranial pressure, which damages brain tissue.**
- **In addition, some patients may develop "pseudoaneurysms" after trauma which can eventually burst and bleed, a factor which might account for the delay in loss of consciousness.**
- **Because a patient may have a lucid interval, any significant head trauma is regarded as a medical emergency and receives emergency medical treatment even if the patient is conscious.**
- **Delayed cerebral edema, a very serious and potentially fatal condition in which the brain swells dramatically, may follow a lucid interval that occurs after a minor head trauma.**
- **Lucid intervals may also occur in conditions other than traumatic brain injury, such as heat stroke and the postictal phase after a seizure in epileptic patients.**

4.c) Cleft palate is types of clefting congenital deformity caused by abnormal facial development during gestation.

A cleft is a fissure or opening gap.
It is the non-fusion of the body's natural structures that form before birth.
Clefts can also affect other parts of the face, such as the eyes, ears, nose, cheeks and forehead.
Anatomy:

Hard Palate	Soft Palate
– **Bones: Maxilla(Palatine Processes) + Palatine Bones(Horizontal Lamina)** – **Blood Supply: Greater Palatine Artery** – **Nerve Supply: Anterior Palatine Nerve**	– **Fibromuscular shelf attached like a shelf to posterior portion of hard palate** – **Tenses, elevates, contracts Passavant's Ridge** – **Muscles: Tensor Veli Palatini(CNV), Levator Veli Palatini(Primary Elevator), Musculus Uvulae, Palatoglossus, Palatopharyngeus(CN IX and X)**

Embryology:

Primary Palate- Triangular area of hard palate anterior to incisive foramen to point just lateral to lateral incisor teeth- Includes that portion of alveolar ridge and four incisor teeth.

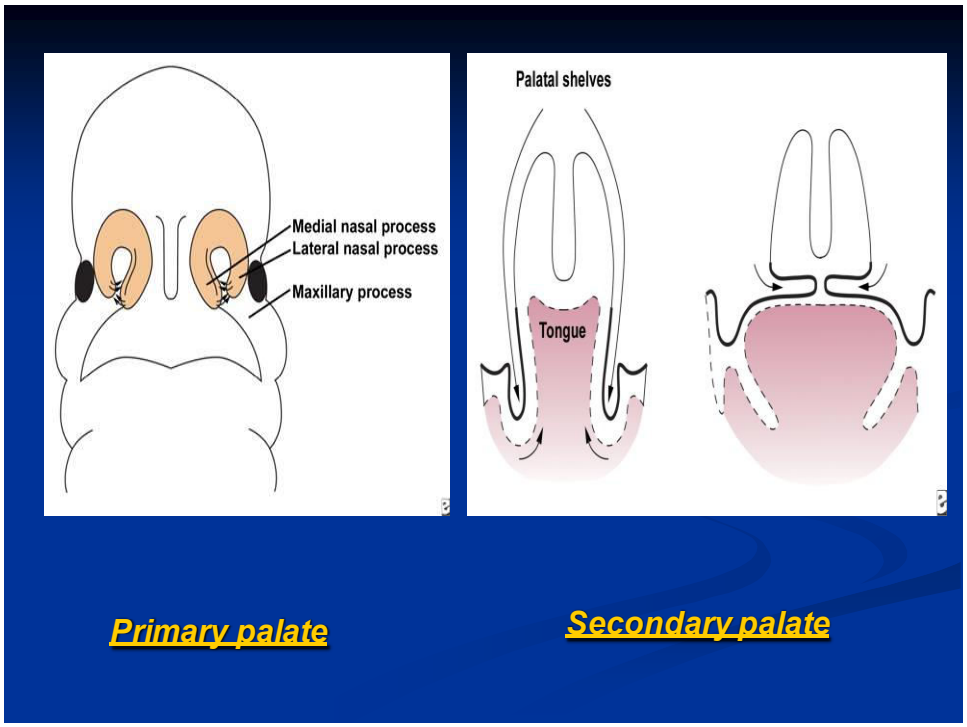
Secondary Palate- Remaining hard palate and all of soft palate

Primary Palate
- **Forms during 4th to 7th week of Gestation**
- **Two maxillary swellings merge**
- **Two medial nasal swelling fuse**
- **Intermaxillary Segment Forms: Labial Component(Philtrum)**

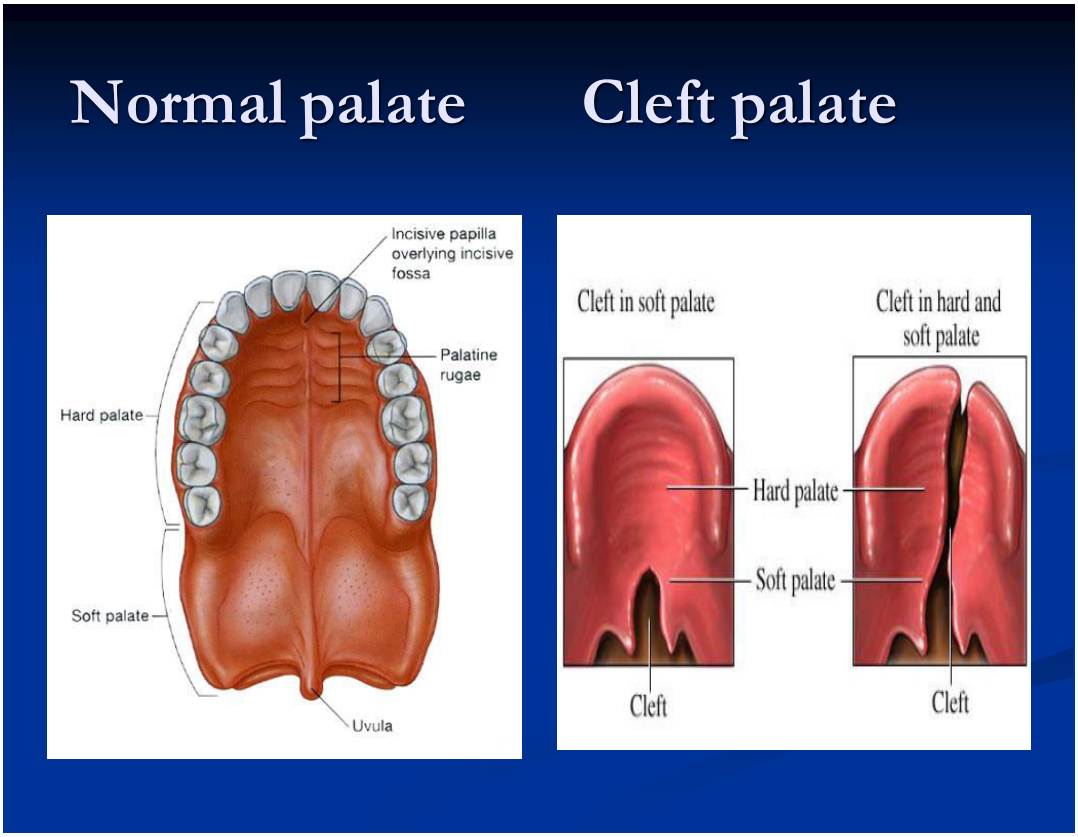
- **Maxilla Component(Alveolus + 4 Incisors) Palatal Component(Triangular Primary Palate)**

Secondary Palate

- **Forms in 6th to 9th weeks of gestation**
- **Palatal shelves change from vertical to horizontal position and fuse**
- **Tongue must migrate antero-inferiorly.**



- **Clefts of the primary palate : result from a failure of mesoderm to penetrate into the grooves between the medial nasal and maxillary processes, which prohibits their from fusion with each another**
- **Clefts of the secondary palate: result from a failure of the palatine shelves to fuse with one another. The cause for this is failure of the tongue to descend into the oral cavity.**



Classification:

- **Group I (A):-**

Defects of the soft palate alone

- **Group II (B)**

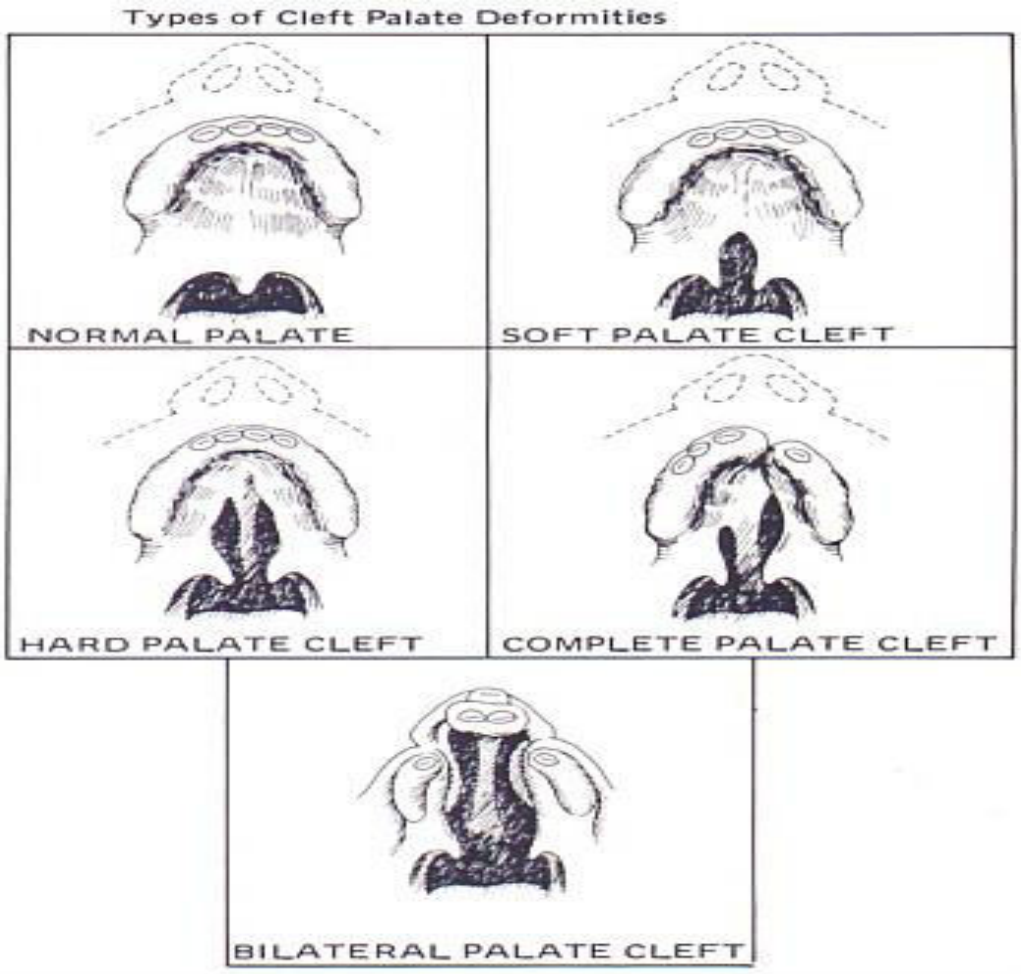
Defects involving the hard and soft palates (not extending anterior to the alveolus)

- **Group III (C)**

Defects involving the palate through to the alveolus

- **Group IV (D)**

Complete bi-lateral clefts.



Complications:

- **Feeding difficulties:**

One of the most immediate concerns after birth is feeding as cleft palate make sucking difficult or cause gagging or nasal regurgitation
To overcome this problem by using a special bottle nipple or a small artificial palate (obturator) that fits into the roof of the mouth .
The upright sitting position allows gravity to help the baby swallow the milk more easily

- **Ear infections and hearing loss:**

Babies with cleft palate are especially susceptible to *middle ear infections*. Ear infections are often due to a dysfunction of the tube that connects the middle ear and the throat.
Over time, repeated ear infections can *damage hearing*, but hearing loss may resolve with treatment.
It's important for children with cleft palate to be *evaluated regularly* by an audiologist.
Most children with clefts have tubes inserted in their ears to drain fluids and help prevent infections.

- **Dental problems:**

If the cleft extends through the *upper gum*, tooth development will likely be affected.
A pediatric dentist should monitor tooth development and oral health from an early age.

- **Speech difficulties:**

Because both the lip and palate are used in forming sounds, the development of normal speech can be affected. A speech pathologist can evaluate your child and provide speech therapy.

- **Psychological challenges:**

Children with clefts may face social, emotional and behavioral problems due to differences in appearance and the stress of intensive medical care.

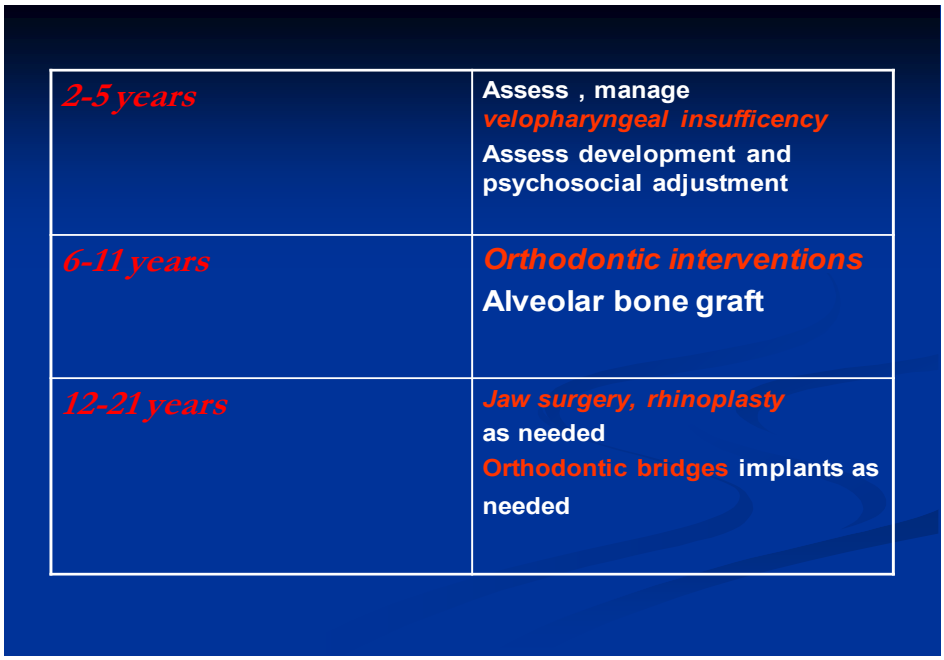
Diagnosis:

- **Prenatal ultrasounds can detect a cleft palate prior a child's birth.**
- **By detecting the cleft abnormality during a pregnancy, the expecting parents can have a prenatal consultation with a plastic surgeon.**
- **The symptoms of these abnormalities are visible during the first examination after birth.**

Treatment:

- **Children born with a cleft palate may need the skills of several medical professionals to correct the problems associated with the cleft**
- **Treatment usually requires a complex, lengthy treatment plan lasting until adulthood (see table below).**

Age	*Intervention*
Prenatal *Birth-1 month*	Referred to cleft lip and palate team Diagnosis and genetic counseling Address *psychosocial issues* Provide *feeding instructions* Make feeding plan
1-15 months	Check feeding and growth *Repair cleft lip* Check ears and hearing
16-24 months	Assess *ears and hearing* Assess *speech and language* Check development



Aims of repair:

- **Separate the oral and nasal cavities, this separation involves the formation of a valve that is necessary for normal speech.**

Also the muscles at the back of the palate need to be put in their proper place across the
Adjunct tools:

- **Cell blocks**
- **Histochemistry**
- **Immunohistochemistry**
- **Electron microscopy**
- **Flow cytometry**
- **Immuno electron microscopy**
- **Molecular pathology -In situ hybridization, PCR etc**

Future directions:

- **Aspirating non palpable lesions using MRI**
- **Molecular pathology eg In Situ Hybridization**
- **Replacing diagnostic surgical pathology?**
- **Combined with MRI - replacing autopsy?**
- **Cleft so the child can learn to speak normally.**

Time of operation: General agreement exists that surgical correction of a cleft palate should be accomplished when patients are younger than 1 year, before significant speech development occurs.

4. d) Warthin tumors, also known as lymphomatous papillary cystadenomas, are benign, sharply demarcated tumors of the salivary gland. They are of lymphoid origin and most commonly arise from parotid gland tail. They may be bilateral or multifocal in up to 20% of cases and are the most common neoplastic cause of multiple solid parotid masses.

Epidemiology

- **Warthin tumors are the 2nd most common benign parotid tumor (after pleomorphic adenoma) and represent up to 10% of all parotid tumors.**
- **They are the commonest bilateral or multifocal benign parotid tumor.**
- **They typically occur in the elderly (6th decade), and twice as common in men (2:1).**
- **Patients typically present with painless parotid swelling.**

Morphology

They are often multicentric (20%) and are usually small (1-4 cm). They have a typically heterogeneous appearance on all modalities, often with cystic components (30%).

Location

Tends to favor the parotid tail region at the level of the mandibular angle.
In <10% cases, they are found elsewhere, including the submandibular glands, cervical lymph nodes, and ectopic nests of salivary tissue, e.g. larynx, maxillary antrum, oral cavity (e.g. lower lip, buccal mucosa).

Associations

- **Smoking**
- **Irradiation**

Radiographic features

Has a greater tendency to undergo cystic change (~30%) than any other salivary gland tumor.

Ultrasound

Tumors that are large (e.g. >5 cm) tend to have a higher proportion of cystic content than smaller lesions had and in some cases can be composed almost entirely of cystic material. They are often hypervascular.

CT

- **Classic appearance is a well-defined heterogeneous solid cystic lesion within the superficial lobe of parotid/parotid tail**
- **Well defined**
- **No calcification**
- **Cystic changes appear as intralesional lower attenuation**
- **Moderate enhancement**
- **Presence of mural nodule is strongly suggestive of warthin tumor**
- **Can be often seen bilaterally**

MRI

Well defined and can be bilateral.

Nuclear medicine

Often shows uptake with Tc^{99}-pertechnetate, thallium, and FDG-PET.

Treatment and prognosis

They are benign with an extremely low incidence of malignant transformation (~1%). Some advocate surgical excision while others favor conservative management with follow-up imaging.

The commonest surgery is a superficial parotidectomy and recurrence rate is low.

4. e) Definition: A varicocele is an abnormal enlargement of the pampiniform venous plexus in the scrotum. This plexus of veins drains the testicles.

Pathogenesis: The testicular blood vessels originate in the abdomen and course down through the inguinal canal as part of the spermatic cord on their way to the testis. Upward flow of blood in the veins is ensured by small one-way valves that prevent backflow. Defective valves, or compression of the vein by a nearby structure, can cause dilatation of the testicular veins near the testis, leading to the formation of a varicocele.

Causative factors:

- **8-10 cm longer left testicular Vv. → increased hydrostatic pressure in upright position**
- **Entry of left testicular Vv into renal vein at 90^0**
- **"Nutcraker phenomenon" due to passage of left testicular vein between SMA & Aorta**
- **Congenital absence of valve in left vein in 40%**
- **Intrinsic ectasia of plexus due to cremaster atrophy**
- **Loaded left colon**
- **Left sided RCC.**

Clinical features:

- **A varicocele often produces no signs or symptoms.**
- **Rarely, it may cause pain. The pain may:**
  - **Vary from sharp to dull discomfort**
  - **Increase with standing or physical exertion, especially over long periods**
  - **Worsen over the course of a day**
  - **Be relieved when lying on back**
- **Visible or palpable (able to be felt) enlarged vein.**
- **Feeling of heaviness in the testicle(s)**
- **Atrophy (shrinking) of the testicle(s)**
- **Alteration of testosterone levels**
- **Benign prostatic hyperplasia (BPH) and related urinary problems.**

Clinical examinations:

- **Increases on standing.**
- **"Bag of worm" feel**

- **Grades of varicocele:**
  Grade I – Palpable only during valsulva maneuver
  Grade II – Palpable without Valsulva in standing upright position
  Grade III – Visible through scrotal skin

  Subclinical – detected during USG

Investigations:

- **Doppler stethoscope (5.3 MHz probe) -audible rush of blood on valsulva**
- **Colour Doppler –detects Sub Clinical Varicocele also**
- **Ultra sound of abdomen.**
- **Semen examination**

Treatment:

- **Expectant treatment – in adolescent males who are asymptomatic with normal size of testis**

Indication of intervention:

- **Asymptomatic varicocele with >20% volume loss of Testis (>2ml)**
- **Symptomatic varicocele**
  - **Impaired sperm quality**
  - **Pain**
  - **Cosmetic reasons**
- **Medically unfit.**

The West Bengal University of Health Sciences

M.B.B.S. 3rd Professional Part – II Examination, 2018

Subject: Surgery **Time: 2 1/2 hrs.**

Paper: II **Marks: 60**

Group –A

1. Enumerate the causes of anuria. How would you differentiate between prerenal and renal anuria? Give the management of calculus anuria(principles only) 5+5+5

Group – B

2. a) What are the anatomical and pathophysiological changes that lead to the development of the primary varicose veins of the lower limbs? How would you test clinically the competence of the valves of the sapheno-femoral, sapheno-popliteal junction and leg perforators? Give the management of a patient with primary varicose vein with sapheno-femoral incompetence. 6+4+5

Or

b) Define thyrotoxicosis. Enumerate the grade-wise presentation of the eye signs in thyrotoxicosis. Give the brief outline of the diagnosis and options of management of Graves Disease. 2+3+5+5

Group – C

3. Write short notes on any three of the following: 3x5

a) Magnetic resonance cholangio pancreatography (MRCP)

b) Epidural Anaesthesia.

c) Split – thickness skin graft

d) Choledochal cyst

e) Complication of External beam radiation therapy

Group – D

4. Write short notes on any three of the foloowing: 3x5

a) Epulis

b) Ultrasound Wave Therapy

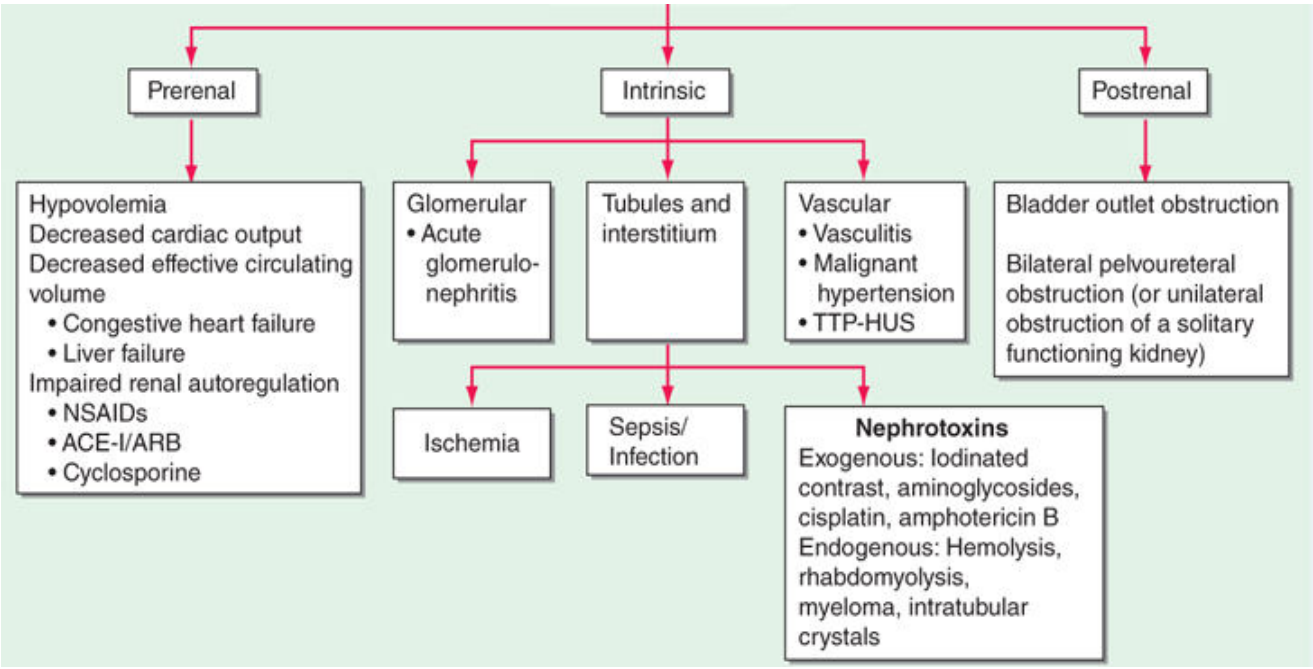
c) Flail chest with paradoxical respiration

d) Secondary Brain Injury

e) Diabetic foot.

1. Answer.

Causes of anuria:



Differentiation between pre renal and renal anuria:

Indices	Prerenal	Renal
Urine sediment	Hyaline casts	Abnormal
Specific gravity	>1.020	~1.010
Urine osmolality ($mOsm/kg\ H_2O$)	>500	<350
U_{Na} (mmol/L)	<20	>40
Fractional excretion Sodium (%) Urea (%)	 <1 <35	 >2 >35

Management of calculus anuria:

- **Calculus anuria occurs in patients with ureteric obstruction in a solitary kidney, obstruction in the only functioning kidney or in bilateral obstructed kidneys. The mechanism of renal damage in obstruction is not well understood. However possible mechanism is high intrapelvic pressure and decrease renal blood flow. Reversibility of renal function depends upon the duration and degree of obstruction.**

- **Return of renal function depends upon many factors other than the duration and degree of obstruction, such as absence of infection, presence of intra-renal or extra renal pelvis in obstructed kidneys.**
- **Calculus anuria is a urological emergency. Management in form of urinary diversion and definite surgical treatment can save the patient from developing chronic renal failure.**
- **USG of abdomen is more sensitive in detecting the hydronephrosis but it is less sensitive for detection of ureteric calculi.**
- **Early intervention with DJ stenting / PCN is the initial treatment of choice. Bilateral URSL can be done as an emergency procedure in selected cases.**
- **The factors influencing the reversibility of renal failure include age, associated comorbdities (D.M, H.T.N.), duration of obstruction, associated sepsis, cortical thickness, diuresis after relieving obstruction. Initial creatinine or level of obstruction had no influence on the reversibility of renal failure.**
- **Majority of the patients with calculous anuria can be managed with endourological procedures.**

2. a) Pathophysiology:

- **The pathophysiology behind their formation is complicated and involves the concept of ambulatory venous hypertension.**
- **In healthy veins, the flow of venous blood is through the superficial system into the deep system and up the leg and toward the heart. One-way venous valves are found in both systems and the perforating veins.**
- **Incompetence in any of these valves can lead to a disruption in the unidirectional flow of blood toward the heart and result in ambulatory venous hypertension (AVH).**
- **Incompetence in the superficial venous system alone usually results from failure at valves located at the SFJ and SPJ.**
- **The gravitational weight of the column of blood along the length of the vein creates hydrostatic pressure, which is worse at the more distal aspect of the length of vein.**
- **Reflux at or near the SFJ does not always come through the terminal valve of the GSV, nor does it always involve the entire trunk of the GSV.**
- **Reflux can enter the GSV below the sub terminal valve or even immediately below the junction, passing through a failed sub terminal valve to mimic true SFJ incompetence.**
- **Reflux can also pass directly into any of the other veins that join the GSV at that level, or it may pass a few centimeters along the GSV and then abandon the GSV for another branch vessel.**
- **Incompetence of the perforating veins leads to hydrodynamic pressure. The calf pump mechanism helps to empty the deep venous system, but if perforating vein valves fail, then the pressure generated in the deep venous system by the calf pump mechanism are transmitted into the superficial system via the incompetent perforating veins.**
- **Once venous hypertension is present, the venous dysfunction continues to worsen through a vicious circle. Pooled blood and venous hypertension leads to venous dilatation, which then causes greater valvular insufficiency.**
- **Over time, with more local dilatation, other adjacent valves sequentially fail, and after a series of valves has failed, the entire superficial venous system is incompetent. This can then cause subsequent perforator and deep venous valvular dysfunction.**

- **The clinical findings of varicose veins, reticular veins, and telangiectasias are due to the hypertension in the superficial venous system that spreads to collateral veins and tributary veins, causing dilated tortuous structures.**
- **In contrast to the superficial veins, the deep veins do not become excessively distended. They can withstand the increased pressure because of their construction and the confining fascia.**

The Trendelenburg Test or Brodie-Trendelenburg test is a test which can be carried out as part of a physical examination to determine the competency of the valves in the superficial and deep veins of the legs in patients with varicose veins.

Procedure: With the patient in the supine position, the leg is flexed at the hip and raised above heart level. The veins will empty due to gravity or with the assistance of the examiner's hand squeezing blood towards the heart.

A tourniquet is then applied around the upper thigh to compress the superficial veins but not too tight as to occlude the deeper veins. The leg is then lowered by asking the patient to stand.

Normally the superficial saphenous vein will fill from below within 30–35 seconds as blood from the capillary beds reaches the veins; if the superficial veins fill more rapidly with the tourniquet in place there is valvular incompetence below the level of the tourniquet in the "deep" or "communicating" veins. After 20 seconds, if there has been no rapid filling, the tourniquet is released. If there is sudden filling at this point, it indicates that the deep and communicating veins are competent but the superficial veins are incompetence. The test is reported in two parts, the initial standing up of the patient (positive or negative based on rapid filling) and the second phase once the tourniquet is removed (positive or negative based upon rapid filling).

For example, a possible outcome of the test would be negative-positive meaning that the initial phase of the test was negative indicating competence in the deep and communicating veins and the second phase of the test was positive meaning that there is superficial vein incompetence.

The test can be repeated with the tourniquet at different levels to further pinpoint the level of valvular incompetence:

- **Above the knee - to assess the mid-thigh perforators**
- **Below the knee - to assess incompetence between the short saphenous vein and the popliteal vein.**

Superficial veins of the leg normally empty into deep veins, however retrograde filling occurs when valves are incompetent, leading to varicose veins.

Multiple tourniquette test is done to detect perforator incompetence.

- **Hand-held Doppler (HHD). Listen over SFJ and SPJ and apply calf compression with other hand and listen for reflux lasting 1-2sec. Most accurate outpatient method of diagnosis and localization of primary venous reflux disease.**
- **Colour duplex. Gold standard investigation in defining anatomy and incompetence.**

Treatments available:

- **Conservative management**

- **Surgery**
- **Endovenous Laser Therapy**
- **Foam Sclerotherapy**
- **Radiofrequency ablation**

❖ **Compression therapy**
  - **Compression therapy is the primary treatment for CVI.**

- **Elastic compression stockings**
  - **Fitted to provide a compression gradient from 30 to 40 mm Hg, with the greatest compression at the ankle.**
- **Pneumatic compression devices**
  - **Provide dynamic sequential compression.**
  - **Used primarily in the prevention of deep vein thrombi in hospitalized patients.**
  - **Also used successfully to treat venous insufficiency.**

❖ **Endovenous ablation is an image-guided, minimally invasive treatment. It uses radiofrequency or laser energy to cauterize (burn) and close the abnormal veins that lead to varicose veins.**

❖ **Foam sclerotherapy is a minimally invasive technique for men and women to eliminate unsightly varicose veins and spider veins. The procedure involves injecting a foam sclerosant in a blood vessel to close it. The blood reroutes itself through healthy veins, restoring more normal blood flow.**

❖ **Radiofrequency ablation is a minimally invasive treatment for varicose veins.**

❖ **Surgery:**

Varicose vein surgery is one of the most common forms of surgery. Varicose veins can be surgically removed or closed off using a number of different techniques. This will not harm the blood supply in the legs, because the blood will re-direct into other healthy veins. Surgery can be done on both superficial and deep veins. In varicose vein surgery, the varicose veins are removed to relieve the symptoms.

The most common ways to surgically remove varicose veins are:

- **Vein stripping: Two incisions (cuts) are made: One at the top of the leg just below the groin and one behind the knee joint or at the ankle. The vein is then tied or clamped off at the top incision using a technique called vein ligation. A long wire is sent through the lower incision up through the vein, and at the lower end a button-like cap is attached to the wire. This allows the entire vein to be pulled out through the incision near the groin.**

- **Phlebectomy: Very small incisions of just a few millimeters are made along the affected vein. A small hook is used to pull the vein out as far as possible through these incisions and then it is cut and removed in several pieces. This technique is used mainly for smaller veins that branch off of the main veins. This procedure uses the small incisions to avoid the scarring caused when larger cuts are made.**

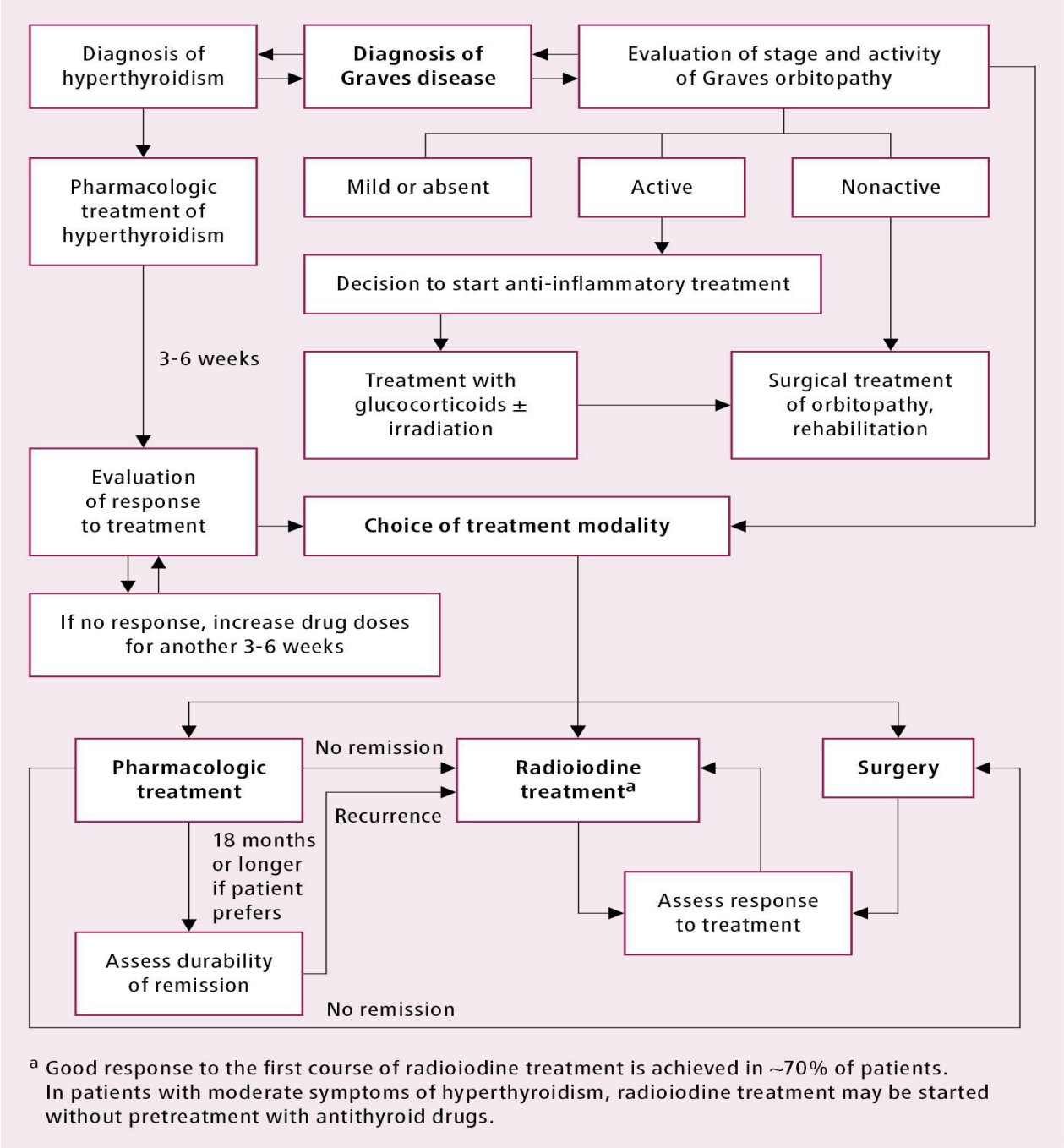
2. b)

Hyperthyroidism is the condition that occurs due to excessive production of thyroid hormone by the thyroid gland. Thyrotoxicosis is the condition that occurs due to excessive thyroid hormone of any cause and therefore includes hyperthyroidism.

Eye signs of thyrotoxicosis according to grade:

Class	Description
Class 0	No signs or symptoms
Class 1	Only signs (limited to upper lid retraction and stare, with or without lid lag)
Class 2	Soft tissue involvement (oedema of conjunctivae and lids, conjunctival injection, etc.)
Class 3	Proptosis
Class 4	Extraocular muscle involvement (usually with diplopia)
Class 5	Corneal involvement (primarily due to lagophthalmos)
Class 6	Sight loss (due to optic nerve involvement)

Algorithm of treatment of Graves disease



[a] Good response to the first course of radioiodine treatment is achieved in ~70% of patients. In patients with moderate symptoms of hyperthyroidism, radioiodine treatment may be started without pretreatment with antithyroid drugs.

3. a) M.R.C.P

Answer.

It is a non-invasive imaging technique to visualize intra and extrahepatic biliary tree and pancreatic ductal system.

It can provide the diagnostic range equivalent to the ERCP and so it can replace the ERCP in high risk patient to avoid significant morbidity.

Indications: MRCP can be used to evaluate various conditions of pancreatobiliary ductal system, some of them are:

- **Identification of congenital anomalies of the cystic and hepatic ducts**
- **Post-surgical biliary anatomy and complications**
- **Pancreas divisum**
- **Anomalous pancreaticobiliary junction**
- **Choledocholithiasis**
- **Biliary strictures**
- **Chronic pancreatitis**
- **Pancreatic cystic lesions**
- **Trauma to biliary system**

Physics

The technique exploits the fluid which is present in the biliary and pancreatic ducts as a contrast agent by acquiring the images using heavily T2 weighted sequences. Since the fluid-filled structures in the abdomen have a long T2 relaxation time as compared to the surrounding soft tissue, these structures appear hyperintense than the surrounding on a heavily T2 weighted sequence and can easily be distinguished.

Technique and protocols

No contrast is administered within the body.

Fasting for 4 hours prior to the examination is required to reduce gastroduodenal secretions, reduce motility to eliminate motion artifacts and to promote distension of gall bladder. MRCP is performed on a 1.5-T or superior MRI system, using a phased-array body coil.

All protocols obtain heavily T2 weighted sequences. Most commonly obtained sequences are:

- **RARE: rapid acquisition and relaxation enhancement**
- **FRFSE: fast-recovery fast spin-echo coronal oblique 3D respiratory triggered**
- **HASTE: half-Fourier acquisition single shot turbo spin echo- Axial 2D breath hold sequence which provide superior images and can be performed in single breath hold (<20 sec) and a fat suppressed sequence**
- **additional sequence which can be acquired to evaluate duct wall is fat suppressed T1 GRE sequence**

For optimum visualization of ducts, acquired images are reformatted in different planes using multiplanar reconstruction (MPR) and maximum intensity projection (MIP).

The advantage of FRFSE, as a 3D technique, is the ability to perform multiplanar reconstructions. However, despite respiratory triggering, this sequence is often prone to motion artifact.

Technical modifications: With the evolution of MRCP, modified techniques came into existence. Commonly applied modified MRCP techniques are:

- **Secretin stimulated MRCP**
- **Functional MRCP**
- **Negative oral contrast to 'null' the duodenum**
  - **commercially available agents**
  - **natural products such as pineapple juice which is rich in manganese and shortens T2 relaxation time**

Practical points: Artifacts related to technique and reconstruction, motion or susceptibility artifacts due to metal clips and gas may give rise to poor spatial resolution as a result of that misinterpretation.

3. b) Epidural Anaesthesia

Answer.

Introduction: Epidural anesthesia is regional anesthesia that blocks pain in a particular region of the body. The goal of an epidural is to provide analgesia, or pain relief, rather than anesthesia which leads to total lack of feeling. Epidurals block the nerve impulses from the lower spinal segments.

{Difference from spinal anesthesia: Spinal anaesthesia is a technique whereby a local anaesthetic drug is injected into the cerebrospinal fluid. This technique has some similarity to epidural anaesthesia, and lay people often confuse the two techniques. Important differences include:

- **To achieve epidural analgesia or anaesthesia, a larger dose of drug is typically necessary than with spinal analgesia or anaesthesia.**
- **Onset of analgesia is slower with epidural analgesia or anaesthesia than with spinal analgesia or anaesthesia.**
- **An epidural injection may be performed anywhere along the vertebral column (cervical, thoracic, lumbar, or sacral), while spinal injections are typically performed below the second lumbar vertebral body to avoid piercing and consequently damaging the spinal cord.**
- **It is easier to achieve segmental analgesia or anaesthesia using the epidural route than using the spinal route.**
- **An indwelling catheter is more commonly placed in the setting of epidural analgesia or anaesthesia than with spinal analgesia or anaesthesia.}**

Indications: Injecting medication into the epidural space is primarily performed for analgesia.

For analgesia alone, where Surgery is not contemplated. An epidural injection or infusion for pain relief (e.g. in child birth) is less likely to cause loss of muscle power, but has to be augmented to be sufficient for surgery.

- **As an adjunct to general anaesthesia. This may reduce the patient's requirement for opioid analgesics. This is suitable for a wide variety of surgery, for example gynaecological surgery (e.g. hysterectomy), orthopaedic surgery (e.g. hip replacement), general surgery (e.g.laparotomy) and vascular surgery (e.g. open aortic aneurysm repair).**
- **As a sole technique for surgical anaesthesia. Some operations, most frequently Caesarean section, may be performed using an epidural anaesthetic as the sole technique. This can allow the patient to remain awake during the operation.**
- **For post-operative analgesia, after an operation where the epidural technique was used as either the sole anaesthetic, or was used in combination with general anaesthesia. Analgesics are given into the epidural space typically for a few days after surgery, provided a catheter has been inserted. Through the use of a patient-controlled epidural analgesia (PCEA) infusion pump, a person has the ability to give themselves an occasional dose of pain medication through an epidural catheter.**
- **Treatment of back pain. Injection of analgesics and steroids into the epidural space may improve some forms of back pain.**
- **Treatment of chronic pain or palliation of symptoms in terminal care, usually in the short- or medium-term.**

Epidural analgesia during childbirth:

Advantages	Disadvantages
• Better pain relief than other pain medication • Fewer babies needing naloxone to counter opiate use by the mother • Decreased maternal hyperventilation and increased oxygen supply to baby • Decreased circulating adrenocorticotropic hormone and decreased fetal distress [	• More use of instruments to assist with the birth • Increased risk of Caesarean section for fetal distress • Longer delivery (second stage of labour) • Increased need for oxytocin to stimulate uterine contractions • Increased risk of very low blood pressure • Increased risk of muscular weakness for a period of time after the birth • Increased risk of fluid retention • Increased risk of fever

Epidural analgesia has been demonstrated to have several benefits after surgery, including:

- **Effective analgesia without the need for systemic opioids.**
- **The incidence of postoperative respiratory problems and chest infections is reduced.**
- **The incidence of postoperative myocardial infarction ("heart attack") is reduced.**

- **The stress response to surgery is reduced.**
- **Motility of the intestines is improved by blockade of the sympathetic nervous system.**
- **Use of epidural analgesia during surgery reduces blood transfusion requirements.**

Complications:

- **Failure to achieve analgesia or anaesthesia occurs in about 5% of cases, while another 15% experience only partial analgesia or anaesthesia**
- **The following factors are associated with failure to achieve epidural analgesia/anaesthesia**
  - **Obesity**
  - **Multiparity**
  - **History of a previous failure of epidural anaesthesia**
  - **History of regular opiate use**
  - **Cervical dilation of more than 7 cm at insertion**
  - **The use of air to find the epidural space while inserting the epidural instead of alternatives such as saline or lidocaine.**
  - **Accidental dural puncture with headache. This may cause cerebrospinal fluid (CSF) to leak out into the epidural space, which may in turn cause a post dural puncture headache (PDPH). Most cases resolve spontaneously with time.**
  - **Delayed onset of breastfeeding and shorter duration of breastfeeding.**
  - **Bloody tap.**
  - **Catheter misplaced into a vein.**
  - **High block, as described above.**
  - **Catheter misplaced into the subarachnoid space. This may result in a high block, or, more rarely, a *total spinal*, where anaesthetic is delivered directly to the brainstem, causing unconsciousness and sometimes seizures.**
  - **Neurological injury lasting less than 1 year.**
  - **Epidural abscess formation.**
  - **Epidural haematoma formation.**
  - **Neurological injury lasting longer than 1 year.**
  - **Paraplegia.**
  - **Arachnoiditis.**
  - **Death (extremely rare, less than 1 in 100,000).**

3. c) Split thickness skin graft.

Answer. Introduction:

- **Split-thickness skin grafts (of varying thickness). These are sometimes called Thiersch grafts.**
- **They are used to cover all sizes of wound, are of limited durability and will contract.**
- **They may be used to provide valuable temporary wound closure before better cosmetic secondary correction after rehabilitation.**

Donor site: The split thickness skin graft leaves behind adnexal remnants such as hair follicles and sweat glands, foci from which epidermal cells can repopulate and resurface the

donor site. It is usually harvested with either a special blade or dermatome that can be set to a desired thickness.

Recipient site: Split thickness grafts are usually used to resurface larger defects. Depending on how much of the dermis is included, STSGs undergo secondary contraction as they heal.

Mechanism of graft survival	Mechanism of graft failure
i. Plasmatic imbibition — Initially, the skin grafts passively absorbs the nutrients in the wound bed by diffusion. ii. Inosculation — By day 3, the cut ends of the vessels on the underside of the dermis begin to form connections with those of the wound bed iii. Angiogenesis — By day 5, new blood vessels grow into the graft and the graft becomes vascularized	i. Poor wound bed — Because skin grafts rely on the underlying vascularity of the bed, wounds that are poorly vascularized with bare tendons or bone, or because of radiation, will not support a skin graft. ii. Sheer — Sheer forces separate the graft from the bed and prevent the contact necessary for revascularization and subsequent "take". iii. Hematoma/seroma — Hematomas and seromas prevent contact of the graft to the bed and inhibit revascularization. They must be drained by day 3 to ensure "take". iv. Infection — Bacteria have proteolytic enzymes that lyse the protein bonds needed for revascularization.

Also read: Full thickness — Full thickness skin grafts (FTSGs) consist of the entire epidermis and dermis.

Donor site - the full thickness skin graft leaves behind no epidermal elements in the donor site from which resurfacing can take place. Thus, the donor site of a FTSG must be closed. It must be taken from an area that has skin redundancy. It is usually harvested with a knife between the dermis and the subcutaneous fat.

Recipient site - Full thickness skin grafts are usually used to resurface smaller defects because they are limited in size. It is commonly used to resurface defects of the face. It provides a better color consistency, texture, and undergoes less secondary contraction.

Mechanism of graft survival and failure are same as STSG.

3. d) Choledochal cyst.

Answer.

Choledochal cysts represent congenital cystic dilatations of the biliary tree. Diagnosis relies on the exclusion of other conditions (e.g. tumour,gallstone, inflammation) as a cause of biliary duct dilatation.

Epidemiology: Choledochal cysts are rare, with an incidence of 1:100,000-150,000. Although they may be discovered at any age, 60% are diagnosed before the age of 10 years. There is a strong female predilection with M:F ratio of 1:4. There is a greater prevalence in East Asia.

Clinical presentation: Classically presentation includes the triad of:

- **Abdominal pain**
- **Jaundice**
- **Abdominal mass**

This triad is however only present in ~40% (range 19-60%) of cases, with palpable mass being the least common manifestation.

Pathology: Their aetiology is uncertain, but a close association with the anomalous formation of the pancreaticobiliary ductal junction is reported in some subtypes. Due to this anomaly, there is a large common channel draining pancreatic and bile duct. Thus the pancreatic juices cause cholangitis and bile duct wall destruction, which together with distal stenosis due to scarring result in the formation of a choledochal cyst.

Associations

A number of associations are recognised, including:

- **biliary atresia**
- **hepatic fibrosis**
    - **associated with type V (Caroli disease)**

Classification: Commonly accepted classification currently is one devised by Todani et al. There are five main types, with several subtypes some of which can be pathologically unrelated:

- **type I: most common, accounting for 80-90% (this type can present *in utero*)**
    - **Ia: dilatation of extrahepatic bile duct (entire)**
    - **Ib: dilatation of extrahepatic bile duct (focal segment)**
    - **Ic: dilatation of the common bile duct portion of extrahepatic bile duct**
- **type II: true diverticulum from extrahepatic bile duct**
- **type III: dilatation of extrahepatic bile duct within the duodenal wall (choledochocoele)**
- **type IV: next most common**
    - **IVa: cysts involving both intra and extrahepatic ducts**
    - **IVb: multiple dilatations/cysts of extrahepatic ducts only**
- **type V: multiple dilatations/cysts of intrahepatic ducts only (Caroli disease)**

The Komi classification classifies choledochal cyst into 3 types based on the anomalous union of the pancreatic-bile duct (AUPBD).

Radiographic features: Imaging of the biliary tree can be achieved with ultrasound, CT, direct contrast studies (ERCP, PTC) or MRI.

Ultrasound: The key to the diagnosis is a dilated cystic lesion which communicates with the bile duct and is separate from the gall bladder. A careful search for other causes needs to also be undertaken (see differential below), remaining cognizant that both stone formation and malignancy are associated with choledochal cysts.

CT and MRCP: Findings are similar to ultrasound, with a greater ability to demonstrate intrahepatic disease. It is important to remember that with CT IVC the contrast mixing in the cyst may be inhomogeneous. It does, however, have the advantage that it conclusively demonstrates communication with the biliary tree.

Treatment and prognosis: The only feasible treatment is surgical excision, with reconstruction. A number of possible approaches exist, depending on cyst location and other factors. Typically a Roux-en-Y hepaticojejunostomy is performed.

Complications: The two most frequent complications of choledochal cysts are stone formation and malignancy. Complications include:

- **Stone formation: most common**
- **Malignancy**
  - **Cholangiocarcinoma**
  - **Lifetime incidence 10-15%**
- **The cyst may rupture leading to bile peritonitis**
  - **Most frequently seen in neonates**
- **Pancreatitis.**

3. e) Complications of external beam radiation therapy:

Answer.

Some side effects depend on the type and location of radiation therapy.

Head and neck:

- **Dry mouth**
- **Mouth and gum sores**
- **Difficulty in swallowing**
- **Stiffness in the jaw**
- **Nausea**
- **Hair loss**
- **A type of swelling called lymphedema**
- **Tooth decay**

Chest:

- **Shortness of breath**
- **Breast or nipple soreness**
- **Shoulder stiffness**
- **Cough, fever, and fullness of the chest, known as radiation pneumonitis. This happens between 2 weeks and 6 months after radiation therapy.**
- **Radiation fibrosis, which causes permanent lung scars from untreated radiation pneumonitis.**

Stomach and abdomen:

- **Nausea and vomiting**
- **Diarrhea**

Pelvis:

- **Diarrhea**
- **Rectal bleeding**
- **Incontinence**
- **Cystitis**

Additionally, men and woman may have different symptoms.

Potential side effects for men include:

- **Sexual problems, such as erectile dysfunction, which is the inability to get or maintain an erection**

- **Lowered sperm counts and reduced sperm activity. This can occur from radiation therapy to the testes or prostate. And it may affect a man's ability to father a child. Learn about ways to preserve your fertility.**

For women:

- **Changes in menstruation, such as having menstruation stop**

- **Symptoms of menopause, such as vaginal itching, burning, and dryness**

- **Infertility. This may occur if both ovaries receive radiation therapy.**

4. a) Epulis

Answer.

Introduction:

- **Epulis is any benign tumor (i.e. lump) situated on the gingival or alveolar mucosa.**
- **Most of them are granulomas associated with chronic gingivitis. A few are true neoplasms.**
- **There are three types: fibromatous, ossifying and acanthomatous.**
- **Epulis appears as a single or multiple fold of tissue that grows in excess around the alveolar vestibule.**
- **Usually, the edge of the denture rests in between two of the folds. The excess tissue is firm and fibrous, and ulcerations may be present.**
- **The great majority of cases are seen beneath ill-fitting dentures of long use and in persons who do not take their dentures out overnight.**

- **The lesion seems to result from a combination of chronic, mild trauma and low-grade infection by bacteria or candida yeast.**
- **It is occasionally seen in patients without dentures but with high palatal vaults or with the habit of breathing through their mouths.**

Signs and symptoms:

- **The lesion is usually painless.**
- **The usual appearance is of two excess tissue folds in alveolar vestibule/buccal sulcus, with the flange of the denture fitting in between the two folds.**
- **It may occur in either the maxillary or mandibular sulci, although the latter is more usual.**
- **Anterior locations are more common than posterior.**
- **Less commonly there may be a single fold, and the lesion may appear on the lingual surface of the mandibular alveolar ridge.**
- **The swelling is firm and fibrous, with a smooth, pink surface. The surface may also show ulceration or erythema.**
- **The size of the lesion varies from less than 1 cm to involving the entire length of the sulcus.**

Causes:

- **The cause is usually pressure from the flange of a denture which causes chronic irritation and a hyperplastic response in the soft tissues.**
- **Women during pregnancy can also present with an epulis, which will resolve after birth.**
- **Fibroepithelial polyps, pedunculated lesions of the palate beneath an upper denture, are associated with this condition.**
- **A cobble-stone appearance similar to an epulis fissuratum in a patient without dentures can be diagnostic of Crohn's disease.**
- **Epulis fissuratum can also appear around dental implants.**

Diagnosis:

- **The diagnosis is made clinically, and usually this is clear cut if the lesion is associated with the flange of a denture.**
- **Tissue biopsy is not usually indicated before removal of the lesion, since the excises surgical specimen is usually sent for histopathologic examination and the diagnosis is confirmed retrospectively.**
- **Rarely, incisional biopsy may be indicated to rule out neoplasia, e.g. in the presence of suspicious ulceration. The appearance may also be confused with pyogenic granuloma.**
- **The excessive tissue is composed of cellular, inflamed fibrous connective tissue. The appearance of an epulis fissuratum microscopically is an overgrowth of cells from the fibrous connective tissue. The epithelial cells are usually hyperkeratotic and irregular, hyperplastic rete ridges are often seen.**

Treatment:

- **Treatment is by surgical excision (complete removal) of the fibrous tissue overgrowth and addressing the causative factor to prevent recurrence of the lesion.**
- **Common techniques for removal of the excess tissue include traditional removal with a surgical scalpel, electrical scalpel, or laser excision with a laser scalpel, e.g. a carbon dioxide laser, erbium:YAG laser, Neodymium-YAG laser, or diode laser.**
- **The poorly fitting denture can be adapted to fit better (a "reline") or a new denture constructed.**
- **Alternatively, the section of flange that is sharp/over-extended can be smoothed and reduced with a drill.**

4. b) Ultrasonic wave therapy.

Answer. Therapeutic ultrasound is extensively used by physiotherapists and is popular with patients.

- **Ultrasound is a form of MECHANICAL energy**
- **Mechanical vibration at increasing frequencies is known as sound energy.**
- **Below 16Hz, these vibrations are not recognizable as sound**
- **The normal human sound range is from 16Hz to something approaching 15-20,000 Hz**
- **Beyond this upper limit, the mechanical vibration is known as ULTRASOUND.**
- **The frequencies used in therapy are typically between 1.0 and 3.0 MHz**
- **1MHz = 1 million cycles per second**
- **As the energy within the sound wave is passed to the material, it will cause oscillation of the particles of that material.**
- **Any increase in the molecular vibration in the tissue can result in heat generation, and ultrasound (US) can be used to produce thermal changes in the tissues**
- **All sound is produced by the vibration of a membrane**
- **Ultrasound (> 20,000 Hz) is produced by the vibration of synthetic crystals**
- **The crystal contracts and expands when exposed to alternating electric current**
- **These oscillations of the crystal produce pressure waves = ultrasound waves.**

- **Ultrasound is applied using a transducer or applicator that is in direct contact with the patient's skin. Gel is used on all surfaces of the head to reduce**
-
- **friction and assist transmission of the ultrasonic waves. Therapeutic ultrasound in physical therapy is alternating** compression **and** rarefaction **of sound waves with a** frequency **of >20,000 cycles/second. Therapeutic ultrasound frequency used is 0.7 to 3.3** MHz**. Maximum energy absorption in** soft tissue **occurs from 2 to 5 cm. Intensity decreases as the waves penetrate deeper. They are absorbed primarily by** connective tissue**:** ligaments**,** tendons**, and**fascia **(and also by** scar tissue**).**
- **Conditions for which ultrasound may be used for treatment include the follow examples: Ligament** Sprains**, Muscle** Strains**,**Tendonitis**, Joint Inflammation,** Plantar fasciitis**,** Metatarsalgia**, Facet Irritation,** Impingement syndrome**,** Bursitis**,**Rheumatoid arthritis**,** Osteoarthritis**, and Scar Tissue Adhesion.**

Indications

- **Benefits:**
  - **Decrease pain and muscle spasm**
  - **Increase extensibility of collagen**
  - **Increase blood flow**
  - **Increase metabolic rate**
  - **Increase tissue healing**
  - **Organization of healing tissues**
- **Contraindications:**
  - **Over the heart**
  - **Over the cordal spine**
  - **Over epiphyseal area of growing bones**
  - **Infected areas**
  - **Bleeding areas**
  - **Neoplasms**
  - **Thrombophlebitis**
- **Precautions:**
  - **Over analgesic areas**
  - **Myositis ossificans**
  - **Fibrotic myopathy**
  - **Plastic and metal implants**
  - **Over the carotid sinus**
  - **Over a pregnant uterus**

4. c) Answer.

Flail chest with paradoxical respiration:

Flail chest is a life-threatening medical condition that occurs when a segment of the rib cage breaks due to trauma and becomes detached from the rest of the chest wall. Two of the symptoms of flail chest are chest pain and shortness of breath.

It occurs when multiple adjacent ribs are broken in multiple places, separating a segment, so a part of the chest wall moves independently. The number of ribs that must be broken varies by differing definitions: some sources say at least two adjacent ribs are broken in at least two places, some require three or more ribs in two or more places. The flail segment moves in the opposite direction to the rest of the chest wall: because of the ambient pressure in

comparison to the pressure inside the lungs, it goes in while the rest of the chest is moving out, and vice versa. This so-called "paradoxical breathing"is painful and increases the work involved in breathing.

Signs and symptoms:

Two of the symptoms of flail chest are chest pain and shortness of breath.

The characteristic paradoxical motion of the flail segment occurs due to pressure changes associated with respiration that the rib cage normally resists:

- **During normal inspiration, the diaphragm contracts and intercostal muscles pull the rib cage out. Pressure in the thorax decreases below atmospheric pressure, and air rushes in through the trachea. The flail segment will be pulled in with the decrease in pressure while the rest of the rib cage expands.**
- **During normal expiration, the diaphragm and intercostal muscles relax increasing internal pressure, allowing the abdominal organs to push air upwards and out of the thorax. However, a flail segment will also be pushed out while the rest of the rib cage contracts.**

Paradoxical motion is a late sign of flail segment; therefore, an absence of paradoxical motion does not mean the patient does not have a flail segment.

The constant motion of the ribs in the flail segment at the site of the fracture is extremely painful, and, untreated, the sharp broken edges of the ribs are likely to eventually puncture the pleural sac and lung, possibly causing a pneumothorax. The concern about "mediastinal flutter" (the shift of the mediastinum with paradoxical diaphragm movement) does not appear to be merited.[8] Pulmonary contusions are commonly associated with flail chest and that can lead to respiratory failure. This is due to the paradoxical motions of the chest wall from the fragments interrupting normal breathing and chest movement. Typical paradoxical motion is associated with stiff lungs, which requires extra work for normal breathing, and increased lung resistance, which makes air flow difficult.[9] The respiratory failure from the flail chest requires mechanical ventilation and a longer stay in an intensive care unit. It is the damage to the lungs from the flail segment that is life-threatening.

Causes:

The most common causes of flail chest injuries are vehicle collisions, which account for 76% of flail chest injuries.

Another main cause of flail chest injuries is falling. This mainly occurs in the elderly, who are more impacted by the falls as a result of their weak and frail bones, unlike their younger counterparts who can fall without being impacted as severely.

Falls account for 14% of flail chest injuries.

In children, the majority of flail chest injuries result from common blunt force traumas or metabolic bone diseases, including a group of genetic disorders known as osteogenesis imperfecta.

Diagnosis:

Diagnosis:

A chest radiograph of a flail chest associated with right sided pulmonary contusion and subcutaneous emphysema

Diagnosis is by medical imaging with either plain X ray or CT scan.

Treatment:

Treatment of the flail chest initially follows the principles of advanced trauma life support. Further treatment includes:

- **Good pain management includes intercostal blocks and avoiding opioid pain medication as much as possible. This allows much better ventilation, with improved tidal volume, and increased blood oxygenation.**
- **Positive pressure ventilation, meticulously adjusting the ventilator settings to avoid pulmonary barotrauma.**
- **Chest tubes as required.**
- **Adjustment of position to make the person most comfortable and provide relief of pain.**
- **Aggressive pulmonary toilet**

Surgical fixation can help in significantly reducing the duration of ventilatory support and in conserving the pulmonary function.

4. d) Secondary brain injury:

Answer.

- **Secondary brain injury refers to the changes that evolve over a period of time (from hours to days) after the primary brain injury. It includes an entire cascade of cellular, chemical, tissue, or blood vessel changes in the brain that contribute to further destruction of brain tissue.**
- **It occurs after a variety of brain injury including subarachnoid hemorrhage, stroke, and traumatic brain injury and involves metabolic cascades. Secondary injury can result from complications of the injury. ... Ischemia is one of the leading causes of secondary brain damage after head trauma.**
- **Signs of a possible secondary brain injury include: Severe, frequent headaches. Vision problems or loss of visual stability. Memory loss or difficulty with short-term memory.**
- **Important elements of therapy are: head position, normoglycemia, osmotherapy, normal body temperature, optimal blood pressure, adequate oxygenation. barbiturate therapy. Neutral head and neck position is recommended to prevent intracranial hypertension.**
- **Primary Brain injury is the damage sustained as a direct result of the impact on the skull and intracranial contents. Secondary brain injury refers to the changes that evolve over a period of time (from hours to days) after the primary brain injury.**

4. e) Diabetic foot:

Answer.

Pathophysiology:

Diabetic foot disease involves a complex sequence of changes that lead to the destruction of the foot's function and structure. Peripheral neuropathy, arthropathy, arterial insufficiency, and infection are the major derangements underlying diabetic foot disease. The final common pathway is often one of ulceration, gangrene, and limb loss.

Peripheral Neuropathy

Peripheral neuropathy is common among diabetics and is the most difficult component of diabetic foot disease to treat.Its severity ranges from minimal somatosensory changes to total anesthesia with the complete absence of sweating. Diabetic neuropathy affects the motor, sensory, and autonomic nervous systems, which leads to multiple pathologic conditions.

Destruction of myelinated motor fibers produces a motor polyneuropathy that tends to be bilateral and symmetric. Attenuation of the Achilles reflex is an early sign of motor neuropathy. Progression leads to loss of function and atrophy of the lumbricals and intrinsic muscles of the foot. Unopposed, the extrinsic muscles distort the foot, leading to depression of the metatarsal heads, digital contractures, "clawing" of the toes, and a varus malformation. These alterations allow pressure points and ulcers to develop over the proximal interphalangeal joints and beneath the metatarsal heads.

Sensory neuropathy involving type A myelinated fibers leads to loss of proprioception, light tough, pressure, and vibration and is noted to develop early. Compromise of these nerves leads to an ataxic gate and pressure ulceration. Loss of type C sensory fibers limits a patient's ability to detect painful or noxious stimuli, which contributes to neuroarthropathy, injuries, and the formation of ulcers. C fibers also participate in nociceptive or neuroinflammatory responses that blunt the signs of trauma and infection in diabetic feet. Sensory neuropathy can also paradoxically produce pain and paresthesias. Neuropathic pain can be severe and quite difficult to differentiate from other foot problems. Diabetic paresthesias are characterized by hyperesthesia, burning, or tingling sensations and tend to resolve over time.

Autonomic neuropathy produces abnormal vasomotor responses. Autosympathectomy increases arteriovenous shunting, which leads to osteopenia. Hypohidrosis results in dry, cracked skin, which compromises the skin barrier and increases the susceptibility to infections.

Neuroarthropathy

Neuroarthropathy or Charcot's foot is a condition in which extensive destruction of the joints occurs within the foot consequent to a loss in muscular stability, osteopenia, and insensitivity to pain. It is present in many disorders of peripheral nerves but is most common in diabetics. Sensory neuropathy permits mechanical insults to go unrecognized by the patient, which produce severe stresses on osteopenic bone. Bony dissolution and loss of structural integrity follow, leading to collapse of the midfoot arch.

Minor foot trauma (e.g., minor fracture, sprain, and contusion) often catalyzes neuropathic skeletal changes. Localized warmth and swelling at the site occur initially; this is usually

nontender and out of proportion to the injury. The warmth and swelling of this inflammatory stage of neuropathic osteoarthropathy can be difficult to distinguish from infection. Chronically, skeletal encroachment occurs in the tarsometatarsal and metatarsophalangeal joints followed by collapse of the arch. Subluxation or even lateral dislocation of the forefoot occurs, producing a bowed, "rocker-bottom" foot, which is extremely susceptible to ulceration.

Arterial Insufficiency

Altered glucose metabolism in diabetics leads to changes in response to endothelial injury, dyslipidemia, enhanced platelet activity, and increased blood viscosity. These factors contribute to an increased incidence of atherosclerotic occlusive disease and hypercoagulability in diabetic patients. Arterial insufficiency contributes to the development of neuropathy, limits a patient's ability to heal ulcers and traumatic injuries, and in the worst case, produces ischemic ulcers. The distribution of atherosclerotic disease in diabetics typically involves the femoropopliteal and tibial vessels with relative sparing of aortoiliac and pedal vessels. Consequently, femoral to distal tibial or pedal bypasses are feasible for limb salvage in many diabetics with critical ischemia.

Infection

Diabetic foot infections range from superficial cellulitis to wet gangrene and abscess formation. Peripheral neuropathy and neglected skin lesions or ulcers often initiate deep-space infections. Fungal infections contribute to ulceration and secondary bacterial infection either by direct microscopic breaks in the skin or through toenail loss. Loss of proprioception facilitates traumatic injury. Arterial insufficiency and the diminished inflammatory response allow for rapid progression of an infection. The hallmark of diabetic foot infections is a polymicrobial infection. Gram-positive species, including streptococci, staphylococci, enterococci, and gram-negative anaerobes are present.

Assessment:

Assessment of the patient with diabetic foot disease begins with a thorough history with attention given to the presence of cardiac disease, renal disease, peripheral vascular disease, smoking history, and habits of foot care. Laboratory studies include a basic chemistry, a complete blood cell count, a hemoglobin A_{1c} concentration, and a urinalysis. A comprehensive physical examination with emphasis on the neurologic and vascular function of the lower extremities is essential.

The feet should be carefully examined for capillary refill, nail thickening, ulcers or ischemic lesions, signs of infection, intrinsic muscle wasting, bony prominences, and evidence of tenderness. Ischemic ulcers tend to occur in the toes, whereas neuropathic ulcers present over bony prominences and the heel in patients who are bed bound. Gentle probing of ulcers to determine whether there is palpable bone at the base is encouraged as a highly specific and accurate test for the detection of osteomyelitis. Superficial wound cultures are of little utility. Pulse assessment of the femoral, popliteal, dorsalis pedis, and posterior tibial

arteries should be performed. In the absence of palpable pulses, a continuous-wave Doppler probe should be used to assess for the presence of signals.

Noninvasive laboratory studies are often helpful in assessing the degree of vascular insufficiency and predicting healing potential. Studies may include segmental limb pressures with Doppler waveforms, calculation of ankle-brachial indices, and toe pressures. Any gradient greater than 30 mm Hg between any level indicates the presence of a high-grade stenosis or occlusion. Calcification within the arterial wall often renders diabetic vessels incompressible and waveform interpretation must be used to identify disease. In diabetics, toe or partial foot amputations require ankle pressures of at least 55 mm Hg and toe pressures of at least 30 mm Hg to heal. Transcutaneous oxygen measurements may also predict healing if there is at least a pressure of 30 mm Hg.

Imaging

- **Plain radiographs are useful in the assessment of the diabetic foot. They can identify soft tissue calcifications, gas from bacterial infections, the presence of foreign bodies, and fractures. Plain radiographs can detect osteomyelitis but are most sensitive in the later stages when bony destruction has occurred. Three-phase bone scans are highly sensitive for osteomyelitis except in the presence of severe vascular disease.**
- **Arteriography is required when foot perfusion appears inadequate and arterial reconstruction is planned. The location and degree of occlusive disease are readily assessed with arteriography. Assessment of the outflow vessels and, in particular, communication of the pedal vessels with the pedal arch is also important. Magnetic resonance angiography (MRA) is highly accurate in the detection of large-vessel disease and is useful in patients who are intolerant of contrast dyes.**
- **Magnetic resonance imaging (MRI) of the diabetic foot provides unparalleled imaging of the soft tissues. Contrast-enhanced sequences can be used to determine the extent of osseous and soft tissue infection and can delineate areas of ischemia or necrosis**

Management: Optimal treatment of diabetic patients requires a comprehensive program involving control of hyperglycemia, foot protection, and the timely treatment of ulcers, osteoarthropathy, and infections. Diabetics with high-risk foot conditions benefit from education regarding risk factors and their appropriate management.

Prevention

Prevention of ulceration is imperative to prevent limb loss. Every diabetic patient should have an annual foot examination by a physician and, if the patient has neuropathy, at every visit with a health care professional. Self-examinations should occur daily. Hyperglycemia should be controlled to slow the development of neuropathy. The risk of ulceration is increased in diabetic patients with longstanding diabetes, male gender, poor glucose control, neuropathy, or the presence of vascular, renal, or cardiac disease complications of diabetes. Patients with signs of increased plantar pressure such as erythema, warmth, or callus formation should use protective footwear that cushions and redistributes weight appropriately. Nonambulatory or bed-bound patients should also have their feet protected; the Rooke boot is a sheepskin and foam boot that is ideal for this purpose (Osbourne Medical, Rochester, Minn.). Calluses should be débrided by a specialist. Dryness and fungal

infections should be treated appropriately with petroleum-based products (e.g., Aquaphor ointment) and topical antifungals, respectively.

Neuropathic Ulcers

Diabetic foot ulcerations fall into two main categories, ischemic and neuropathic, with some overlap between. In about 30% of patients with plantar neuropathic ulcerations, ischemia will prolong healing times. Vascular assessment is always required to ascertain the presence of coexisting occlusive disease. However, even with adequate arterial inflow, neuropathic ulcers may remain unchanged for months secondary to impaired inflammatory and healing responses. These ulcers are subject to the constant risk of infection, and prolonged ulcer healing lengthens the exposure to this risk. Infection requires prompt management when it arises.

The extent and anatomic site of neuropathic ulcers direct their management. They are most commonly located beneath one of the metatarsal heads or the interphalangeal joint of the hallux. Aggressive débridement is essential in establishing the depth of the ulcer. This requires removal of excessive callus and all necrotic debris. Daily wound care minimizes the risk of infection.

Protecting the weightbearing surface of the foot and offloading mechanical stress is paramount in the treatment of neuropathic ulcers not associated with ischemia. Nonweightbearing for 6 to 12 weeks is an ideal, though poorly tolerated, method to promote healing. An alternative treatment is total contact casting (TCC), which uses a protective short leg cast to mechanically offload the foot at the ulcer site and control edema while allowing some degree of ambulation. TCC results in healing of 70% to 100% of neuropathic plantar ulcers within 5 to 8 weeks. Neuropathic ulcers 3 cm or less in diameter can be treated with TCC unless severe ischemia, deep ulceration, infection, or significant edema is present. TCC is generally well tolerated and complications infrequently occur from inadequate padding of bony prominences, cast friction, or skin irritation. Specialized shoes with arch bars or formed orthotic insoles are another method to redistribute pressure evenly across the plantar foot.

Once the ulcer is healed, the foot must be continually protected in appropriate footwear. In the diabetic foot, all calluses should be considered preulcerative and should not be mistaken as "benign." Calluses signify areas of repetitive trauma over bony prominences due to vertical, shear, and compressive forces. Calluses should be reduced, bony prominences padded or relieved, and skin lotion used after bathing the feet once or twice daily, drying well between the toes. Antibiotics are used in neuropathic and ischemic foot ulcers only in the presence of cellulitis, abscess, osteomyelitis, or pyarthrosis.

Ischemic Ulcers and Gangrene

Gangrene and ischemic ulcers are common complications with which surgeons are confronted.

- **Aggressive arterial reconstruction has been paramount in reducing amputation rates over the past several decades.**

- **With current techniques, 90% of diabetics presenting with ischemic ulceration or gangrene are found to have surgically correctable occlusive disease.**
- **Vascular reconstruction in diabetic patients typically involves bypass from the femoral or popliteal arteries to tibial, peroneal, or pedal vessels.**
- **In the absence of continuous tibial vessels, a bypass to a patent pedal vessel or a peroneal artery with good ankle collaterals is a reasonable option.**
- **Autologous vein should be used whenever possible because synthetic grafting to infrageniculate vessels has poor long-term patency.**
- **Primary patency rates of 80% at 1 year and limb-salvage rates of 92% at 3 years are achievable.**
- **Although endovascular treatment of flow-limiting arterial stenoses and occlusions has not achieved the long-term patency of distal bypass with autologous vein grafts, this modality is effective in short-term improvements in circulation. Endovascular therapy may be preferable in patients unable or unwilling to undergo traditional bypass.**

Invasive Foot Infection:

Infection often originates in areas of recent trauma or chronic ulceration and may progress rapidly to acute foot sepsis.The initial management is directed at resuscitation and control of the foot infection.

- **Drainage and débridement of necrotic tissues are the primary goals in invasive diabetic foot infections with revascularization delayed until the infection has been controlled.**
- **Diabetic foot infections are often much more extensive than assumed given the propensity of infection to track along the tendon and fascial sheaths.**
- **Wound and sinus tracts should be opened to permit free drainage; involved bone or joints should be resected.**
- **Subsequent procedures may be necessary to ensure adequate drainage.**
- **Deep cultures of the wound should be obtained.**
- **Initial broad-spectrum antibiotics should be administered and refined based on culture and sensitivity data.**
- **Partial foot amputations may be warranted to control sepsis.**
- **These amputations are left open, and are designed to remove large areas of devitalized tissue, destroyed bones, and septic joints.**
- **Careful consideration must be given to preserve as much functional tissue as possible.**
- **Guillotine amputation at or above the ankle is required when infection extends proximal to the midfoot and is associated with hindfoot ischemia.**
- **Definitive amputation follows when infection has been controlled and metabolic derangements improve.**

Amputation

- **Amputations are indicated for removal of gangrenous or necrotic tissue.**
- **The level of amputation depends on the proximal extent of the disease and the degree of vascular insufficiency.**
- **With normal circulation, amputation can usually be performed just proximal to the extent of the necrotic tissue.**
- **Distal toe ulcers will often slough once revascularization has occurred and should be followed for several months before amputation is offered.**

- **Although some allow dry gangrenous digits to autoamputate, button or ray amputations are preferable to spare the discomfort and distress autoamputations can cause patients.**
- **Resection of the distal metatarsal head and removal of all cartilage and sesamoid bones are essential maneuvers in the performance of ray amputations.**
- **A transmetatarsal amputation is considered if multiple toes or the distal forefoot is involved. A transmetatarsal amputation is inadequate in the presence of the midplantar foot ischemia, especially if the medial two or lateral three rays must be sacrificed. In this scenario, midfoot or hindfoot amputations (e.g., Chopart's) along with the appropriate tenotomies may be considered.**
- **Even if successful, however, hindfoot amputations leave patients with a "peg leg," which may not be as functional as a below-knee amputation.If ischemic disease or infection involves the proximal foot, a below-knee amputation is indicated in the ambulatory patient.**
- **Furthermore, in the presence of extensive neuropathic osteoarthropathy involving the tarsal bones with total collapse of the midfoot, a below-knee amputation may be preferable to extensive attempts at reconstruction.**
- **Above-knee amputations may be appropriate in chronically nonambulatory patients, patients with fixed knee contractures, or patients with marginal, unreconstructible, distal circulation.**

Adjunctive Therapies

Despite good wound care, adequate offloading, and treatment of infection, diabetic foot ulcers will occasionally be recalcitrant to standard care. Several therapies exist to improve wound healing, including hyperbaric oxygen therapy, platelet-derived growth factors, bioengineered skin and dermal substitutes, granulocyte colony-stimulating factor, and vacuum-assisted dressings. Vacuum-assisted dressings (VAC; Kinetic Concepts, Inc., San Antonio, Texas) have been particularly useful in stimulating reepithelialization and granulation tissue formation and are well tolerated by patients.

The West Bengal University of Health Sciences

M.B.B.S. 3rd Professional Part – II Examination, 2018

Subject: Surgery **Time: 2 1/2 hrs.**

Paper: II **Marks: 60**

Group –A

1. **What are the different forms of renal calculi? Discuss the clinical presentation and the management of a stone in the renal pelvis. 5+10**

Group – B

2. **a) What is ANDI to classify benign lesions of breast? Discuss the management of discharge from nipple. 7+8**

Or

b) Classify thyroid neoplasm. Discuss the management of solitary thyroid nodule 3cm of size of a 30 year old female. 5+10

Group – C

3. **Answer in brief on any three of the following: 3x5**
   a) **Lucid interval**
   b) **Thyroid storm**
   c) **Ranula**
   d) **ERCP**
   e) **Testicular torsion**

Group – D

4. **Write short notes (any three): 3x5**
   a) **PSA**
   b) **Brachytherapy**
   c) **Biomarkers**
   d) **Triage**
   e) **Regional anaesthesia**

1. Types of renal stone:

Radio-opaque:

- **Calcium phosphate stones are the most radiodense stones, being almost as dense as bone. A phosphate calculus (calcium phosphate often with ammonium magnesium phosphate (struvite)) is smooth and dirty white. Magnesium ammonium phosphate (struvite) stones are less radiodense than calcium containing stones. It grows in alkaline urine, especially when urea-splitting Proteus organisms are present.The calculus may enlarge to fill mostof the collecting system, forming a stag-horn calculus.**

- **Calcium oxalate stones are slightly less radiodense. Oxalate stones are irregular with sharp projections. A calcium oxalate monohydrate stone is hard and radiodense.**

Relatively radiolucent:

- **Cystine stones are relatively radiodense because they contain sulphur.**

An uncommon congenital error of metabolism leads to cystinuria.
Cystine stones are often multiple and may grow to form a cast of the collecting system.
They are resistant to ESWL.

Completely radiolucent:

- **Uric acid: These are hard, smooth and often multiple and multifaceted. Pure uric acid stones are radiolucent.**
- **Triamterene:**
- **Xanthine: Occurs in patients with xanthine oxidase deficiency.**
- **Indinavir: found in AIDS patients.**

Oxalate stone.

- **Oxalate is a normal waste product of metabolism and is relatively insoluble. Normally, approximately 10–15% of oxalate found in the urine originates from the diet; the vast majority is a metabolic by-product.**
- **Most of the oxalate that enters the large bowel is consumed by bacterial decomposition.**
- **Diet, however, can have an impact on the amount of oxalate found in the urine. Once absorbed from the small bowel, oxalate is not metabolized and is excreted almost exclusively by the proximal tubule.**
- **The presence of calcium within the bowel lumen is an important factor influencing the amount of oxalate that is absorbed. The control of oxalate in the urine plays a pivotal role in the formation of calcium oxalate calculi.**
- **Small changes in oxalate levels in the urine can have a dramatic impact on the supersaturation of calcium oxalate.**
- **The principal precursors of oxalate are glycine and ascorbic acid; however, the impact of ingested vitamin C (<2 g/day) is negligible.**
- **Hyperoxaluria may develop in patients with bowel disorders, particularly inflammatory bowel disease, small bowel resection, and bowel bypass. Renal calculi develop in 5–10% of patients with these conditions.**
- **Chronic diarrhea with fatty stools results in a saponification process. Intraluminal calcium binds to the fat, thereby becoming unavailable to bind to oxalate. The unbound oxalate is readily absorbed.**
- **Excessive oxalate may occur secondary to the accidental or deliberate ingestion of ethylene glycol (partial oxidation to oxalate).**
- **This may result in diffuse and massive deposition of calcium oxalate crystals and may occasionally lead to renal failure.**
- **Formed in acidic urine.**
- **Calcium oxalate crystals in urine appear as 'envelopes' microscopically. They may also form 'dumbbells.**

Clinical features:

- **Silent calculus: Renal failure may be the first indication of bilateral silent calculi, although secondary infection usually produces symptoms first.**
- **Pain: Pain occurs in 75 per cent of people with urinary stones. Fixed renal pain occurs in the renal angle,the hypochondrium, or in both. It may be worse on movement.**

- **Pain resulting from renal stones rarely lasts more than 8 hours in the absence of infection.**
- **There is no pyrexia, although the pulse rate rises because of the severe pain.**
- **Haematuria (microscopic or occasionally macroscopic).**
- **Struvite staghorn calculi classically present with recurrent UTIs.**
- **Malaise, weakness, and loss of appetite can also occur.**
- **Less commonly, struvite stones present with infective complications (pyonephrosis, perinephric abscess, septicaemia, xanthogranulomatous pyelonephritis).**

Diagnostic tests:

- **Plain abdominal radiography: calculi that contain calcium are radiodense. Sulphur-containing stones (cystine) are relatively radiolucent on plain radiography.**
- **Completely radiolucent stones (e.g. uric acid, triamterene, indinavir) are usually suspected on the basis of the patient's history and/or urine pH (pH <6 gout; drug history-triamterene, indinavir), and the diagnosis may be confirmed by ultrasound, CTU, or MRU.**
- **Renal ultrasound: its sensitivity for detecting renal calculi is ~95%. A combination of plain abdominal radiography and renal ultrasonography is a useful screeing test for renal calculi.**
- **IVU: increasingly being replaced by CTU. Useful for patients with suspected indinavir stones (which are not visible on CT).**
- **CTU: a very accurate method of diagnosing all but indinavir stones. Allows accurate determination of stone size and location and good definition of pelvicalyceal anatomy.**
- **MRU: cannot visualize stones, but is able to demonstrate the presence of hydronephrosis.**

Treatment:
Acute presentations (renal colic)

- **Analgesia, e.g. diclofenac; IV fluids.**
- **Small stones (<0.5cm) may be managed expectantly as most will pass spontaneously.**
- **Emergency treatment with percutaneous nephrostomy and/or ureteric stent insertion is necessary if either pain or obstruction is persistent.**
- **The traditional indications for intervention are pain, infection, and obstruction.**

Elective presentations:

- **Extra-corporeal shock-wave lithotripsy (ESWL):**
  - **Focused, externally generated electrohydraulic or ultrasonic shock-waves.**
  - **Targeted on to the calculus using ultrasound, X-ray, or a combination.**
  - **Causes stone disintegration and the fragments are then voided - effective for stones upto 1cm.**
- **Percutaneous nephrolithotomy (PCNL):**
  - **For stones in the renal pelvis or calyces.**
  - **Percutaneous track into the renal pelvis using fluoroscopic guidance.**
  - **Nephroscope is inserted and the calculus visualized.**
  - **Removed either in total or, if large, following fragmentation.**
- **Open nephrolithotomy/ureterolithotomy:**
  - **For large staghorn calculi or complex stones.**

Prevention of recurrence:

- **Increase oral fluid intake and reduce calcium intake.**
- **Correct metabolic abnormalities.**
- **Treat infection promptly.**
- **Urinary alkalization, e.g. sodium (mainly for cystine and urate stones).**
- **Thiazide diuretics (for idiopathic hypercalciuria).**

Answers.

2. a) ANDI: Aberrations of Normal Development and Involution

The basic principles underlying the aberrations of normal development and involution (ANDI) classification of benign breast conditions are:

- **Benign breast disorders and diseases are related to the normal processes of reproductive life and to involution;**
- **There is a spectrum of breast conditions that ranges from normal to disorder to disease; and**
- **The ANDI classification encompasses all aspects of the breast condition, including pathogenesis and the degree of abnormality.**

ANDI Classification of Benign Breast Disorders			
	Normal	**Disorder**	**Disease**
Early reproductive years (age 15–25)	**Lobular development**	**Fibroadenoma**	**Giant fibroadenoma**
	Stromal development	**Adolescent hypertrophy**	**Gigantomastia**
	Nipple eversion	**Nipple inversion**	**Subareolar abscess**
			Mammary duct fistula
Later reproductive years (age 25–40)	**Cyclical changes of menstruation**	**Cyclical mastalgia**	**Incapacitating mastalgia**
	Nodularity		
	Epithelial hyperplasia	**Bloody nipple**	

	of pregnancy	**discharge**	
Involution (age 35–55)	**Lobular involution**	**Macrocysts**	
		Sclerosing lesions	
	Duct involution		
	–Dilatation	**Duct ectasia**	**Periductal mastitis**
	–Sclerosis	**Nipple retraction**	
	Epithelial turnover	**Epithelial hyperplasia**	**Epithelial hyperplasia with atypia**

ANDI = Aberrations of normal development and involution.

Discharges from the nipple:

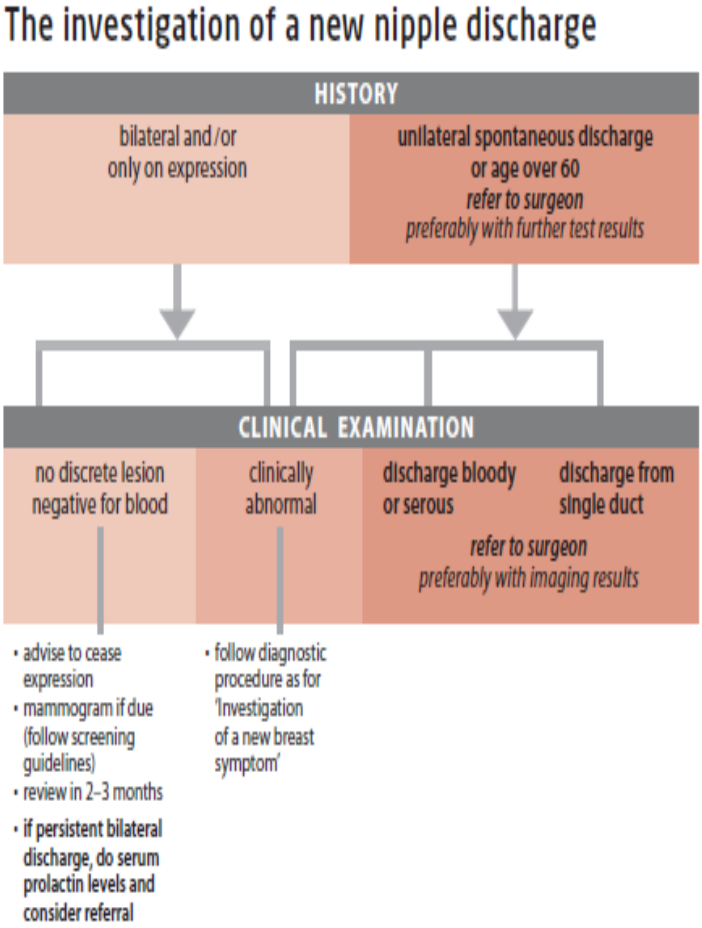
- **Discharge from the surface**
  - **Paget's disease**
  - **Skin diseases (eczema, psoriasis)**
  - **Rare causes (e.g. chancre)**
- **Discharge from a single duct**
  - **Blood-stained**
    - **Intraduct papilloma**
    - **Intraduct carcinoma**
    - **Duct ectasia**
  - **Serous (any colour)**
    - **Fibrocystic disease**
    - **Duct ectasia**
    - **Carcinoma**
- **Discharge from more than one duct**
  - **Blood-stained**
    - **Carcinoma**
    - **Ectasia**
    - **Fibrocystic disease**
  - **Black or green**
    - **Duct ectasia**
  - **Purulent**
    - **Infection**
  - **Serous**
    - **Fibrocystic disease**
    - **Duct ectasia**
    - **Carcinoma**
  - **Milk:**

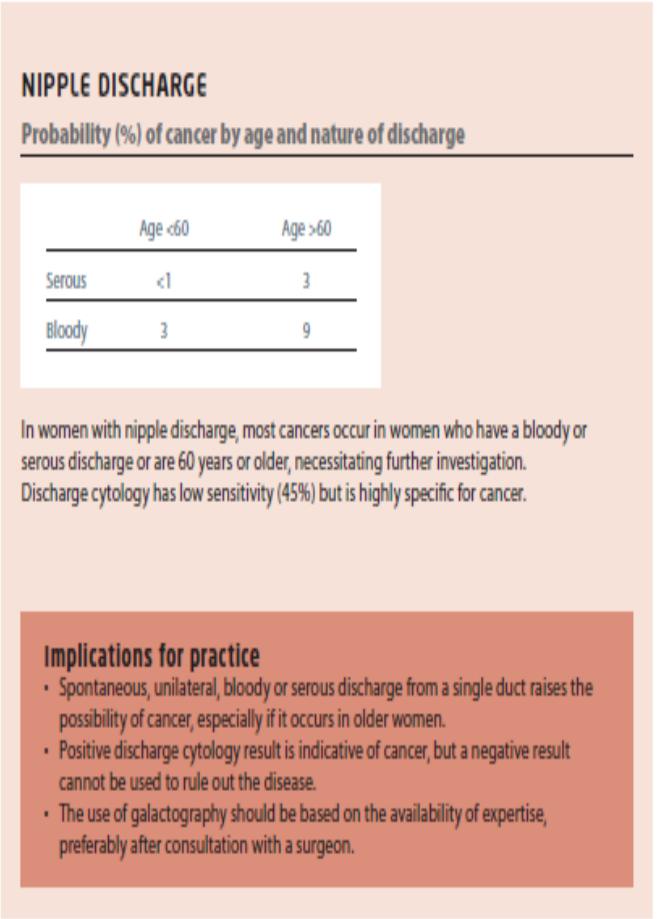
- **Lactation**
- **Rare causes (hypothyroidism, pituitary tumour)**

- **Discharge can occur from one or more lactiferous ducts.**
- **Management depends on the presence of a lump (which should always be given priority in diagnosis and treatment) and the presence of blood in the discharge or discharge from a single duct.**
- **Mammography is rarely useful except to exclude an underlying impalpable mass. Cytology may reveal malignant cells but a negative result does not exclude a carcinoma or in situ disease.**
- **A clear, serous discharge may be 'physiological' in a parous woman or may be associated with a duct papilloma or mammary dysplasia.**
- **Multiduct, multicoloured discharge is physiological and the patient may be reassured.**
- **A blood-stained discharge may be caused by duct ectasia, a duct papilloma or carcinoma. A duct papilloma is usually single and situated in one of the larger lactiferous ducts; it is sometimes associated with a cystic swelling beneath the areola.**
- **A black or green discharge is usually the result of duct ectasia and its complications.**

Investigations:

i. **FNAC or Core Needle biopsy: to know the cytology / histopathology.**
ii. **Digital mammography: to detect any suspicious lesion.**
iii. **USG of breast: Ultrasonography is not typically used unless the nipple discharge is accompanied by a palpable mass or a positive mammographic finding. Ultrasonography may be useful in presurgical localization if galactography reveals a dilated duct larger than a few millimeters in width. Modern, high-resolution ultrasonographic techniques are becoming more sensitive for the visualization of intraductal changes. Tiny, solitary papillomas can sometimes be visualized by using this sophisticated technology (see the images below).**
iv. **MRI and stereotactic biopsy for non palpable breast lesions.**
v. **Fiber-ductoscopy is an experimental technique that may eventually play a role in the evaluation of nipple discharge.**





Treatment:

- **Microdochectomy.**
- **Cone excision of the major ducts (after Hadfield)-(subareolar resection)**
- **Simple and modified radical mastectomy.**

2. b)

Classification of thyroid neoplasms

Benign - Follicular adenoma

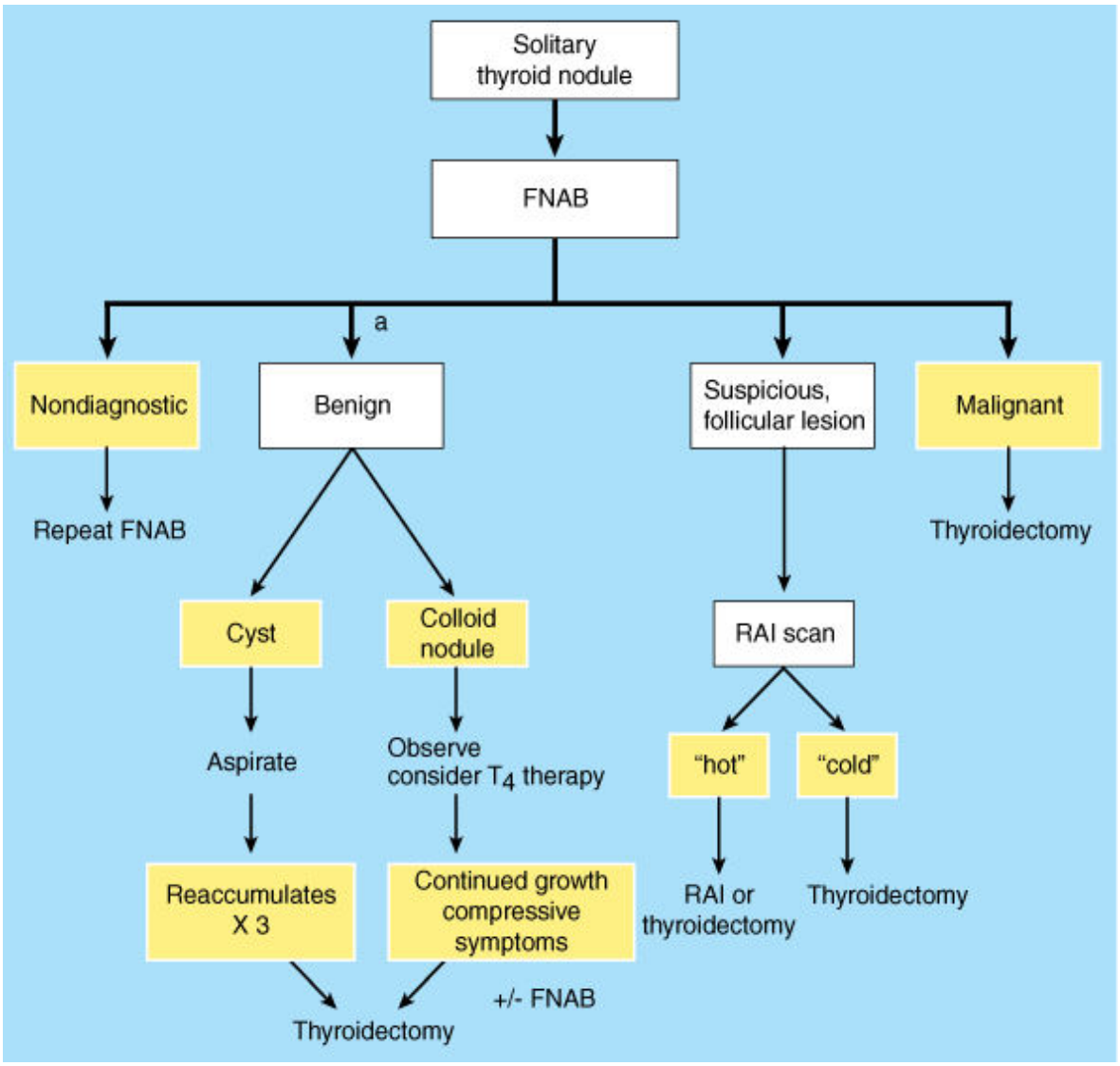
Malignant - Primary

- **Follicular epithelium –differentiated**
  - **Follicular**
  - **Papillary**
- **Follicular epithelium –undifferentiated**
  - **Anaplastic**
- **Parafollicular cells**
  - **Medullary**

- **Lymphoid cells**
  Lymphoma
  Secondary
- **Metastatic**

Local infiltration

Management of solitary thyroid nodule 3cm of size of a 30 year old female



3. a) Lucid interval: In emergency medicine, a lucid interval is a temporary improvement in a patient's condition after a traumatic brain injury, after which the condition deteriorates.

- **A lucid interval is especially indicative of an epidural hematoma. An estimated 20 to 50% of patients with epidural hematoma experience such a lucid interval.**

- **The lucid interval occurs after the initial concussive force of the trauma, then lapses into unconsciousness again after recovery when bleeding causes the hematoma to expand past the extent for which the body can compensate.**
- **After the injury, the patient is momentarily dazed or knocked out, and then becomes relatively lucid for a period of time which can last minutes or hours. Thereafter there is rapid decline as the blood collects within the skull, causing a rise in intracranial pressure, which damages brain tissue.**
- **In addition, some patients may develop "pseudoaneurysms" after trauma which can eventually burst and bleed, a factor which might account for the delay in loss of consciousness.**
- **Because a patient may have a lucid interval, any significant head trauma is regarded as a medical emergency and receives emergency medical treatment even if the patient is conscious.**
- **Delayed cerebral edema, a very serious and potentially fatal condition in which the brain swells dramatically, may follow a lucid interval that occurs after a minor head trauma.**
- **Lucid intervals may also occur in conditions other than traumatic brain injury, such as heat stroke and the postictal phase after a seizure in epileptic patients.**

3. **b) Thyroid storm:**

   Answer. Thyroid storm or thyrotoxic crisis is a rare but severe and potentially life-threatening complication of hyperthyroidism (overactivity of the thyroid gland). It is characterized by a high fever (temperatures often above 40 °C/104 °F), fast and often irregular heart beat, vomiting, diarrhea, and agitation. Hypertension with a wide pulse pressure occurs in early to mid crisis, with hypotension accompanying shock occurring in the late stage. Heart failure and heart attack may occur. Death may occur despite treatment. Most episodes occur either in those with known hyperthyroidism whose treatment has been stopped or become ineffective, or in those with untreated mild hyperthyroidism who have developed an intercurrent illness (such as an infection)

Thyroid storm is characterized by an acute onset of symptoms of hyperthyroidism (fast heart rate, restlessness, agitation) accompanied by other features such as fever (temperatures often above 40 °C/104 °F), hypertension, mental status changes, diarrhea, and vomiting.

Individuals can exhibit varying signs of organ dysfunction. Patients may experience liver dysfunction, and yellow discoloration of the skin is considered a poor prognostic sign. Heart (cardiac) symptoms include abnormal heart rhythms, decreased blood flow to the heart and heart attacks, and congestive heart failure, which may lead to cardiovascular collapse. Mortality can be as high as 20-30%.

In some situations, individuals may not experience the classic signs of restlessness and agitation, but instead present with apathetic signs of weakness and confusion

Causes: The transition from hyperthyroidism to thyroid storm is typically triggered by a non-thyroidal insult including, but not limited to fever, sepsis, dehydration, myocardial infarction, and psychiatric diseases. Individuals are at higher risk of thyroid storm if their hyperthyroidism is incompletely treated or if their anti-thyroid drugs are discontinued. Many of these individuals have underlying primary causes of hyperthyroidism (Graves

disease, toxic multi-nodular goiter, solitary toxic adenoma). However, thyroid storm can occur in individuals with unrecognized thyrotoxicosis experiencing non-thyroid surgery, labor, infection, or exposure to certain medications and radiocontrast dyes.

Precipitating factors for thyroid storm

Severe infection

Diabetic ketoacidosis

Hypoglycemia

Thyroid surgery

Non-thyroid surgery

Parturition

Struma ovarii

Molar pregnancy

Trauma (i.e. hip fracture)

Burns

Myocardial infarction

Pulmonary embolism

Stroke

Heart failure

Radioactive iodine treatment

Medication side effect (anesthetics, salicylate, pseudoephedrine, amiodarone)

Exposure to iodinated contrast

Withdrawal of antithyroid treatment

Emotional stress

Intense exercise

Laboratory findings

As with hyperthyroidism, TSH is suppressed. Both free and serum (or total) T3 and T4 are elevated. An elevation in thyroid hormone levels is suggestive of thyroid storm when accompanied by signs of severe hyperthyroidism but is not diagnostic as it may also correlate with uncomplicated hyperthyroidism. Moreover, serum T3 may be normal in critically ill patients due to decreased conversion of T4 to T3. Other potential abnormalities include the following:

- **Hyperglycemia likely due to catecholamine-mediated effects on insulin release and metabolism as well as increased glycogenolysis, evolving into hypoglycemia when glycogen stores are depleted**
- **Elevated aspartate aminotransferase (AST), bilirubin and lactate dehydrogenase (LDH)**
- **Hypercalcemia and elevated alkaline phosphatase due to increased bone resorption**
- **Elevated white blood cell count**

Management:

The main strategies for the management of thyroid storm are reducing production and release of thyroid hormone, reducing the effects of thyroid hormone on tissues, replacing fluid losses, and controlling temperature.[3] Thyroid storm requires prompt treatment and hospitalization. Often, admission to the intensive care unit is needed.

Iodine

Aministration of inorganic iodide (potassium iodide or Lugol's iodine to reduce the synthesis and release of thyroid hormone. Iodine reduces the synthesis of thyroid hormone via the Wolf-Chaikoff effect. Antithyroid Medications

Antithyroid drugs (propylthiouracil or methimazole) are used to reduce the synthesis and release of thyroid hormone. Propylthiouracil is preferred over methimazole due to its additional effects on reducing peripheral conversion of T4 to T3, however both are commonly used.

Beta Blockers

The administration of beta-1-selective beta blockers (e.g. metoprolol) is recommended to reduce the effect of circulating thyroid hormone on end organs.In addition, propanolol at high doses also reduces peripheral conversion of T4 to T3, which is the more active form of thyroid hormone. Although previously unselective beta blockers (e.g., propranolol) have been suggested to be beneficial due to their inhibitory effects on peripheral deiodinases recent research suggests them to be associated with increased mortality. Therefore, cardioselective beta blockers may be favourable.

Corticosteroids

High levels of thyroid hormone result in a hypermetabolic state, which can result in increased breakdown of cortisol, a hormone produced by the adrenal gland. This results in a

state of relative adrenal insufficiency, in which the amount of cortisol is not sufficient. Guidelines recommend that corticosteroids (hydrocortisone and dexamethasone are preferred over prednisolone or methylprednisolone) be administered to all patients with thyroid storm.

Supportive Measures

In high fever, temperature control is achieved with fever reducers such as paracetamol/acetaminophen and external cooling measures (cool blankets, ice packs). Dehydration, which occurs due to fluid loss from sweating, diarrhea, and vomiting, is treated with frequent fluid replacement. In severe cases, mechanical ventilation may be necessary. Any suspected underlying cause is also addressed.

3. c) Ranula:

- **Ranula refers to a collection of extraglandular and extraductal saliva in the floor of the mouth originating from the sublingual salivary gland. It may rarely originate from injury to the submandibular gland (SMG) duct. It is a pseudocyst, as it does not contain an epithelial lining.**
- **The term ranula originates from the Latin word for frog (rana) as the cyst is said to look like the underbelly of a frog.**
- **A plunging ranula extends into the submandibular triangle of the neck through a defect in the mylohyoid muscle, or less commonly, by passing behind the posterior edge of the muscle.**

Clinical features:

- **A ranula usually presents as a translucent blue, dome-shaped, fluctuant swelling in the tissues of the floor of the mouth.**
- **If the lesion is deeper, then there is a greater thickness of tissue separating from the oral cavity and the blue translucent appearance may not be a feature.**
- **A ranula can develop into a large lesion many centimeters in diameter, with resultant elevation of the tongue and possibly interfering with swallowing (dysphagia).**
- **The swelling is not fixed, may not show blanching and is non-painful unless it becomes secondarily infected.**
- **The usual location is usually is lateral to the midline, which may be used to help distinguish it from a midline dermoid cyst.**
- **A cervical ranula presents as a swelling in the neck, with or without a swelling in the mouth. In common with other mucoceles, ranulas may rupture and then cause recurrent swelling.**
- **Ranulas may be asymptomatic, although they can fluctuate rapidly in size, shrinking and swelling, making them hard to detect.**

Diagnostic criteria:

- **Mostly seen in young children and adolescents, both sexes are equally affected. Swelling in floor of mouth, which may be painful. Mostly unilateral, on one side of frenulum.**
- **Shape is spherical**
- **Size varies from 1 - 5 cm in diameter**
- **Color is pale blue with characteristics semi transparent appearance.**

- **Surface is smooth and mucous membrane is mobile over the swelling.**
- **Tenderness is absent**
- **Fluctuation test is positive**
- **Transillumination test is positive**
- **Cervical lymph nodes are not enlarged.**
- **May or may not have prolongation in the neck.**

Treatment:

- **Treatment of ranulas usually involves removal of the sublingual gland. Surgery may not be required if the ranula is small and asymptomatic.**
- **Marsupialization may sometimes be used, where the intra-oral lesion is opened to the oral cavity with the aim of allowing the sublingual gland to re-establish connection with the oral cavity, but it is often unsuccessful.**
- **Excision of sublingual sailavary gland is often needed.**

3. d) ERCP:

Answer.

- **ERCP is a diagnostic procedure designed to examine diseases of the liver, bile ducts and pancreas.**
- **ERCP is performed under intravenous sedation, usually without general anesthesia.**
- **ERCP is an uncomfortable but not painful procedure. There is a low incidence of complications.**
- **ERCP can provide important information that cannot be obtained by other diagnostic examinations, for example, abdominal ultrasound, CT scan, or MRI.**
- **Frequently, therapeutic measures can be performed at the time of ERCP to remove stones in the bile ducts or to relieve obstruction of the bile ducts.**

Endoscopic retrograde cholangio-pancreatography (ERCP) is a diagnostic test to examine:

- **the duodenum (the first portion of the small intestine),**
- **the papilla of Vater (a small nipple-like structure with openings leading to the bile ducts and the pancreatic duct),**
- **the bile ducts, and**
- **the gallbladder and the pancreatic duct.**

The procedure is performed by using a long, flexible, viewing instrument (a duodenoscope) about the diameter of a pen. The duodenoscope can be directed and moved around the many bends of the stomach and duodenum. The modern duodenoscope uses a thin fiber-optic bundle to transmit light to the tip of the endoscope, and a thin wire with a chip also at the tip of the endoscope to transmit digital video images to a TV screen. The duodenoscope is inserted through the mouth, through the back of the throat, down the food pipe (esophagus), through the stomach and into the duodenum. Once the papilla of Vater is identified, a small plastic catheter (cannula) is passed through an open channel of the endoscope into the opening of the papilla, and into the bile ducts and/or the pancreatic duct. Contrast material (dye) is then injected and X-rays are taken of the bile ducts and the pancreatic duct. Another open channel in the endoscope also allows other instruments to be passed through it in

order to perform biopsies, to insert plastic or metal stents or tubing to relieve obstruction of the bile ducts or pancreatic duct caused by cancer or scarring, and to perform incisions by using electrocautery (electric heat).

The liver is a large solid organ located beneath the right diaphragm. The liver produces bile, which is stored in the gallbladder (a small sac located beneath the liver). After meals, the gallbladder contracts and empties the bile through the cystic duct, into the bile ducts, through the papilla of Vater, and into the intestine to help with digestion. The pancreas is located behind the stomach. It produces a digestive juice that drains through the pancreatic duct--which usually joins the bile duct within the papilla,--and then enters the intestine.

An important procedure related to ERCP is endoscopic ultrasonography which uses a similar endoscope that, in addition to the camera, has an ultrasound probe on its tip to examine the bile ducts, gallbladder, pancreatic duct, and pancreas ultrasonographically. Ultrasonographically-directed needle biopsies of the pancreas can be taken through a channel in the endoscope.

A second, newer procedure related to ERCP is the use of miniature endoscopes that are passed through the operating channel of a duodenoscope and can be inserted directly into the bile and pancreatic ducts. The inside of the ducts can be visualized, and directed biopsies can be taken. Other therapeutic interventions also are possible.

The liver, bile ducts, gallbladder, pancreas and the papilla of Vater can be involved in numerous diseases, causing myriad of symptoms. ERCP is used in diagnosing and treating the following conditions:

- **Blockage of the bile duct by gallstones, cancer, strictures (scarring) or compression from adjacent organs or tumors**
- **Jaundice (yellow coloring of the skin) due to obstruction of the bile duct, also causing darkening of the urine and light colored stool.**
- **Persistent or recurrent upper abdominal pain which cannot be diagnosed by other tests**
- **Unexplained loss of appetite and weight**
- **Confirming the diagnosis of cancer of the pancreas or the bile duct, so that surgery or other treatment can be tailored to the disease**
- **When there is suspicion that the Sphincter of Oddi within the Papilla of Vater, that controls the flow of bile and pancreatic juice, is not working normally (Sphincter of Oddi dysfunction)**

ERCP is a highly specialized procedure which requires a lot of experience and skill. The procedure is quite safe and is associated with a very low risk when it is performed by experienced physicians. The success rate in performing this procedure varies from 70% to 95% depending on the experience of the physician. Complications can occur in approximately one to five percent depending on the skill of the physician and the underlying disorder. The most common complication is pancreatitis which is due to irritation of the pancreas from the dye used to take pictures and can occur even with very experienced physicians. This "injection" pancreatitis usually is treated in the hospital for one to two days.

Another possible complication is infection. Other serious risks including perforation of the intestine, drug reactions, bleeding, depressed breathing. Irregular heart beat or heart attack are extremely rare and is mainly due to the sedation. In case of complications, patients usually need to be hospitalized, but surgery rarely is required.

3. **e) Testicular torsion:**

Pathophysiology: Testicular torsion is a condition whereby the testicle twists in such a way that its blood supply becomes compromised. If left untreated, the blood flow to the testicle ceases and the testicle dies. The earlier the surgery to untwist the testis can be undertaken the better the results, with a testicular salvage rate of 100 per cent if the testicle can be untwisted within 6 hours of the torsion taking place.

For torsion to occur, one of several abnormalities must be present:
• Inversion of the testis is the most common predisposing cause.The testis is rotated so that it lies transversely or upside down.
• High investment of the tunica vaginalis causes the testis to hang within the tunica like a clapper in a bell.
• Separation of the epididymis from the body of the testis permits torsion of the testis on the pedicle that connects the testis with the epididymis.

Cause: Normally, when there is a contraction of the abdominal muscles, the cremaster contracts as well. In the presence of one of the abnormalities described above, the spiral attachment of the cremaster favours rotation of the testis around the vertical axis. Straining on stool, lifting of a heavy weight and coitus are all possible precipitating factors. Alternatively, torsion may develop spontaneously during sleep.

Clinical features:

- **Testicular torsion is most common between 10 and 25 years of age, although a few cases occur in infancy.**
- **Redness of the skin and a mild pyrexia.**
- **Typically, there is sudden agonising pain in the groin and the lower abdomen.**
- **The patient feels nauseated and may vomit.**
- **Torsion of a fully descended testis is usually easily recognised. The testis seems high and the tender twisted cord can be palpated above it.**
- **The cremasteric reflex is lost.**

Differential diagnosis:

- **Epididymo-orchitis in the olderpatient; however, in epididymo-orchitis there will usually be dysuria associated with the accompanying urinary infection. Elevation of the testis reduces the pain in epididymo-orchitis and makes it worse in torsion.**
- **Torsion of a testicular appendage.The most common structure to twist is the appendix of the testis (the pedunculated hydatid of Morgagni).**
- **In mumps orchitis the cord is not particularly thickened and the condition is often bilateral.**
- **Idiopathic scrotal oedema.**

Management:

- **If there is any doubt as to the diagnosis, then urgent scrotal exploration is indicated.**
- **Doppler ultrasound scanning can confirm the absence of the blood supply to the affected testis.**
- **Exploration for torsion should be performed through a scrotal incision. If the testis is viable when the cord is untwisted it should be prevented from twisting again by fixation with nonabsorbable sutures between the tunica vaginalis and the tunica albuginea. The other testis should also be fixed because the anatomical predisposition is likely to be bilateral.**
- **An infarcted testis should be removed – the patient can be counselled later about a prosthetic replacement.**

4. b) Introduction:

- **Brachytherapy (brak-e-THER-uh-pee) is a procedure that involves placing radioactive material inside the body.**
- **Brachytherapy is one type of radiation therapy that's used to treat cancer.**
- **Brachytherapy is sometimes called internal radiation.**

Types: Different types of brachytherapy can be defined according to

- **The placement of the radiation sources in the target treatment area,**
- **The rate or 'intensity' of the irradiation dose delivered to the tumour, and**
- **The duration of dose deliver.**

Source placement: The two main types of brachytherapy treatment in terms of the placement of the radioactive source are interstitial and contact.

- **In the case of interstitial brachytherapy, the sources are placed directly in the target tissue of the affected site, such as the prostate or breast.**
- **Contact brachytherapy involves placement of the radiation source in a space next to the target tissue. This space may be a body cavity (intracavitary brachytherapy) such as the cervix, uterus or vagina; a body lumen (intraluminal brachytherapy) such as the trachea or oesophagus; or externally (surface brachytherapy) such as the skin. A radiation source can also be placed in blood vessels (intravascular brachytherapy) for the treatment of coronary in-stent restenosis.**

Clinical applications:

- **Brachytherapy is commonly used to treat cancers of the cervix, prostate,breast, and skin.**
- **Brachytherapy can also be used in the treatment of tumours of the brain, eye, head and neck region (lip, floor of mouth, tongue, nasopharynx andoropharynx), respiratory tract (trachea and bronchi), digestive tract (oesophagus, gall bladder, bile ducts,rectum, anus), urinary tract (bladder,urethra, penis), female reproductive tract (uterus, vagina, vulva), and soft tissues.**

Commonly used radiation sources (radionuclides) for brachytherapy

Radionuclide	Type
Cesium-137 (^{137}Cs)	β^-- particles
Cobalt-60 (^{60}Co)	β^-- particles
Iridium-192 (^{192}Ir)	γ-rays
Iodine-125 (^{125}I)	Electron Capture, ε
Palladium-103 (^{103}Pd)	Electron Capture, ε
Ruthenium-106 (^{106}Ru)	β^-- particles
Radium-226 (^{226}Ra)	β^-- particles

Side effects: Side effects of brachytherapy are specific to the area being treated. Because brachytherapy focuses radiation in a small treatment area, only that area is affected.

4. **c) Biomarkers: A biomarker, or biological marker, generally refers to a measurable indicator of some biological state or condition. The term is also occasionally used to refer to a substance whose detection indicates the presence of a living organism.**

   Biomarkers are often measured and evaluated to examine normal biological processes, pathogenic processes, or pharmacologic responses to a therapeutic intervention. Biomarkers are used in many scientific fields.

- **In medicine, a biomarker can be a traceable substance that is introduced into an organism as a means to examine organ function or other aspects of health. For example, rubidium chloride is used as a radioactive isotope to evaluate perfusion of heart muscle.**

- **It can also be a substance whose detection indicates a particular disease state, for example, the presence of an antibody may indicate an infection. More specifically, a biomarker indicates a change in expression or state of a protein that correlates with the risk or progression of a disease, or with the susceptibility of the disease to a given treatment.**

Biological marker (biomarker)
A characteristic that is objectively measured and evaluated as an indicator of normal biological processes, pathogenic processes, or pharmacological responses to a therapeutic intervention.

Clinical endpoint
A characteristic or variable that reflects how a patient feels or functions, or how long a patient survives.

Surrogate endpoint
A biomarker intended to substitute for a clinical endpoint. A clinical investigator uses epidemiological, therapeutic, pathophysiological, or other scientific evidence to select a surrogate endpoint that is expected to predict clinical benefit, harm, or lack of benefit or harm.

Advantages and Disadvantages of Biomarkers

Advantages	**Disadvantages**
Objective assessment	**Timing is critical**
Precision of measurement	**Expensive (costs for analyses)**
Reliable; validity can be established	**Storage (longevity of samples)**
Less biased than questionnaires	**Laboratory errors**
Disease mechanisms often studied	**Normal range difficult to establish**
Homogeneity of risk or disease	**Ethical responsibility**

DISEASE	BIOMARKER
DIABETES MELLITUS	RBS,FBS, HbA1c, Retinal assessments
HYPERTENSION	BP,HR, Plasma Renin, Angiotensin I,II, Aldosterone
HEART FAILURE	PRO-BNP,
ASTHMA/COPD	PFTs, Leukotrienes
CARDIAC ISCHEMIA	Troponins, Myoglobins
CANCER MARKERS	PSA, HER-2/Neu, EGFR
OXIDATIVE BIOMARKERS	MDA, Hydrogen Peroxide, α1-Antiproteinase
ANTIOXIDANT BIOMARKERS	SOD, Glutathione, Catalase

Safety biomarkers:

- Safety testing can be classified as follows:

1) Liver safety tests: AST , ALT , ALP, GGT, Bilirubin
2) Renal safety tests: BUN, Sr Creatinine, GFR
3) Hematology safety biomarkers: Complete blood count
4) Bone safety biomarkers: Calcium, Inorganic phosphates
5) Basic metabolic safety biomarkers: Glucose , Cholesterol, Uric acid

Surrogate biomarkers:

Disease	Surrogate Endpoints	Clinical Endpoints
Hypertension	Blood pressure	Stroke
Dyslipidemia	Cholesterol, LDL	Coronary artery disease
Diabetes	Glycosylated hemoglobin (HbA1c)	Retinopathy, nephropathy, neuropathy, heart disease
Glaucoma	Intraocular pressure	Loss of vision
Cancer	Biomarkers Tumor shrinkage, Response rate	Progression-Free Survival Overall Survival

EXAMPLES OF PREDICTIVE BIOMARKERS

DRUG	INDICATION	BIOMAKER
Imatinib	CML	BCR-ABL (PCR), c-KIT
Erlotinib	NSCLC, pancreatic	EGFR and KRAS mutation
Gefitinib	NSCLC	EGFR and KRAS mutation
Trastuzumab	Breast cancer	HER2

PHARMACODYNAMIC (PD) BIOMARKERS

- These are the biomarkers which demonstrate that a drug hits its target and impacts its biochemical pathway.
- Such types of biomarkers are necessary to demonstrate proof of the drug's mechanism of action.
- This class of biomarkers:

✓ Constitute the majority of biomarkers in early phases of drug discovery (preclinical, phase I, and phase II).

✓ Can help to determine effective dose and dose schedule.

- The non-imaging biomarkers include proteins, cytokines, and enzyme activity in serum, CSF, or tissue lysates, proteins by immunohistochemistry (IHC), and DNA and RNA gene expression. **Ex: Ki67 in Ca Prostate**

PROGNOSTIC BIOMARKERS

- Prognostic biomarkers can predict the risk or outcome of a disease in patient population without the involvement of therapy.
- In addition to its predictive power, prognostic biomarkers may help enrich a clinical trial by choosing people more likely to respond to treatment.
- Examples include **prostatic specific antigen** to predict survival in prostatic cancer patients and **CRP** as a risk factor in cardiovascular events.

LIMITATIONS OF BIOMARKERS

- Expensive (cost for analyses)
- Storage (longevity of samples)
- Laboratory errors
- Normal range is difficult to establish
- The following are the major pitfalls in the translation from biomarker discovery to clinical utility:

1. Lack of making different selections before initiating the discovery phase.
2. Lack in biomarker characterisation/validation strategies.
3. Robustness of analysis techniques used in clinical trials.

4. d) Triage:

Triage is the process of determining the priority of patients' treatments based on the severity of their condition. This rations patient treatment efficiently when resources are insufficient for all to be treated immediately. The term comes from the French verb *trier*, meaning to separate, sift or select. Triage may result in determining the order and priority of emergency treatment, the order and priority of emergency transport, or the transport destination for the patient.

Triage may also be used for patients arriving at the emergency department, or telephoning medical advice systems, among others. This article deals with the concept of triage as it occurs in medical emergencies, including the prehospital setting, disasters, and emergency department treatment.

The term *triage* may have originated during the Napoleonic Wars from the work of Dominique Jean Larrey. The term was used further during World War I by French doctors treating the battlefield wounded at the aid stations behind the front. Those responsible for the removal of the wounded from a battlefield or their care afterwards would divide the victims into three categories:

- **Those who are likely to live, regardless of what care they receive;**
- **Those who are unlikely to live, regardless of what care they receive;**
- **Those for whom immediate care might make a positive difference in outcome.**

For many emergency medical services (EMS) systems, a similar model may sometimes still be applied. In the earliest stages of an incident, such as when one or two paramedics exist to twenty or more patients, practicality demands that the above, more "primitive" model will be used. However once a full response has occurred and many hands are available, paramedics will usually use the model included in their service policy and standing orders.

As medical technology has advanced, so have modern approaches to triage, which are increasingly based on scientific models. The categorizations of the victims are frequently the result of triage scores based on specific physiological assessment findings. Some models, such as the START model may be algorithm-based. As triage concepts become more sophisticated, triage guidance is also evolving into both software and hardware decision support products for use by caregivers in both hospitals and the field.

Types:

- **Simple triage: Simple triage is usually used in a scene of an accident or "mass-casualty incident" (MCI), in order to sort patients into those who need critical attention and immediate transport to the hospital and those with less serious injuries. This step can be started before transportation becomes available.**

Upon completion of the initial assessment by physicians, nurses or paramedical personnel, each patient may be labelled which may identify the patient, display assessment findings, and identify the priority of the patient's need for medical treatment and transport from the emergency scene. At its most primitive, patients may be simply marked with coloured

flagging tape or with marker pens. Pre-printed cards for this purpose are known as a triage tags.

Many triage systems use triage tags with specific formats

A triage tag is a prefabricated label placed on each patient that serves to accomplish several objectives:

- **Identify the patient.**
- **Bear record of assessment findings.**
- **Identify the priority of the patient's need for medical treatment and transport from the emergency scene.**
- **Track the patients' progress through the triage process.**
- **Identify additional hazards such as contamination.**

Triage tags may take a variety of forms. Some countries use a nationally standardized triage tag, while in other countries commercially available triage tags are used, and these will vary by jurisdictional choice.

More advanced tagging systems incorporate special markers to indicate whether or not patients have been contaminated by hazardous materials, and also tear off strips for tracking the movement of patients through the process. Some of these tracking systems are beginning to incorporate the use of handheld computers, and in some cases, bar code scanners.

- **Advanced triage:**

  - **In advanced triage, doctors and specially trained nurses may decide that some seriously injured people should not receive advanced care because they are unlikely to survive. It is used to divert scarce resources away from patients with little chance of survival in order to increase the chances for others with higher likelihoods.**
  - **The use of advanced triage may become necessary when medical professionals decide that the medical resources available are not sufficient to treat all the people who need help. The treatment being prioritized can include the time spent on medical care, or drugs or other limited resources. This has happened in disasters such as terrorist attacks, mass shootings, volcanic eruptions, earthquakes, tornadoes, thunderstorms, and rail accidents. In these cases some percentage of patients will die regardless of medical care because of the severity of their injuries. Others would live if given immediate medical care, but would die without it.**
  - **In these extreme situations, any medical care given to people who will die anyway can be considered to be care withdrawn from others who might have survived (or perhaps suffered less severe disability from their injuries) had they been treated instead. It becomes the task of the disaster medical authorities to set aside some victims as hopeless, to avoid trying to save one life at the expense of several others.**
  - **If immediate treatment is successful, the patient may improve (although this may be temporary) and this improvement may allow the patient to be categorized to a lower priority in the short term. Triage should be a continuous process and categories should be checked regularly to ensure that the priority remains correct given the patient's condition. A trauma score is invariably taken when the victim first comes into hospital**

and subsequent trauma scores are taken to account for any changes in the victim's physiological parameters. If a record is maintained, the receiving hospital doctor can see a trauma score time series from the start of the incident, which may allow definitive treatment earlier.

- **Continuous integrated triage:**

Continuous integrated triage is an approach to triage in mass casualty situations which is both efficient and sensitive to psychosocial and disaster behavioral health issues that affect the number of patients seeking care (surge), the manner in which a hospital or healthcare facility deals with that surge (surge capacity) and the overarching medical needs of the event.

Continuous integrated triage combines three forms of triage with progressive specificity to most rapidly identify those patients in greatest need of care while balancing the needs of the individual patients against the available resources and the needs of other patients.

- **Reverse triage: Usually, triage refers to prioritising admission. A similar process can be applied to discharging patients early when the medical system is stressed. This process has been called "reverse triage".**

- **Undertriage and overtriage: Undertriage is the underestimating the severity of an illness or injury. An example of this would be categorizing a Priority 1 (Immediate) patient as a Priority 2 (Delayed) or Priority 3 (Minimal). Overtriage is the overestimating of the severity of an illness or injury. An example of this would be categorizing a Priority 3 (Minimal) patient as a Priority 2 (Delayed) or Priority 1 (Immediate). Acceptable overtriage rates have been typically up to 50% in an effort to avoid undertriage.**

- **Palliative care:**

For those patients that have a poor prognosis and are expected to die regardless of the medical treatment available, palliative care such as painkillers may be given to ease suffering before they die.

Evacuation

In the field, triage sets priorities for evacuation or relocation to other care facilities.

Alternative care facilities

Alternative care facilities are places that are set up for the care of large numbers of patients, or are places that could be so set up. Examples include schools, sports stadiums, and large camps that can be prepared and used for the care, feeding, and holding of large numbers of victims of a mass casualty or other type of event. Such improvised facilities are generally developed in cooperation with the local hospital, which sees them as a strategy for creating surge capacity. While hospitals remain the preferred destination for all patients, during a mass casualty event such improvised facilities may be required in order to divert low-acuity patients away from hospitals in order to prevent the hospitals becoming overwhelmed.

Secondary (in-hospital) triage:

- **In advanced triage systems, secondary triage is typically implemented by emergency nurses, skilled paramedics, or battlefield medical personnel within the emergency departments of hospitals during disasters, injured people are sorted into five categories.**
- **Some crippling injuries, even if not life-threatening, may be elevated in priority based on the available capabilities. During peacetime, most amputation injuries may be triaged "Red" because surgical reattachment must take place within minutes, even though in all probability the person will not die without a thumb or hand.**

4. e) Regional anaesthesia:

Introduction:

- **Local anesthetic applied around a peripheral nerve at any point along the length of the nerve (from spinal cord up to, but not including, the nerve endings) for the purposes of reducing or preventing impulse transmission**
- **No CNS depression (unless overdose (OD) of local anesthetic); patient conscious**
- **Regional anesthetic techniques categorized as follows:**
  - **Epidural and spinal anesthesia**
  - **Peripheral nerve blockades**
  - **IV regional anesthesia**

Physiology:

- **Physiologic response to central blockade is determined by the effects of interrupting the afferent and efferent innervation of somatic (sensory and motor innervation) and visceral (autonomic nervous system).**
- **Somatic blockade:**
  Prevention of pain.
  Skeletal muscle relaxation.

Nerve fiber classification.

Class	Action	Myelin	Size	C_m
Aα	Motor	Yes	++++	++++
Aβ	Light touch, pressure, pain	Yes	+++	+++
Aγ	Muscle spindles, (proprioception)	Yes	+++	++
Aδ	Pain, temperature	Yes	++	+
B	Preganglionic sympathetic fibers	Yes	++	+
C	Pain, pressure	No	+	+++

- **Fibers blocked more easily small and myleinated.**
- **Less easily – large – unmylinated.**

Indications	Contraindications	Complications
❑ Avoidance of some of the dangers of general anesthesia (e.g. known difficult intubation, severe respiratory failure, etc.) ❑ Patient specifically requests regional anesthesia ❑ For high quality post-op pain relief ❑ General anesthesia not available	❑ Allergy to local anesthetic ❑ Patient refusal, lack of cooperation ❑ Lack of resuscitation equipment ❑ Lack of IV access ❑ Coagulopathy ❑ Certain types of preexisting neurological dysfunction ❑ Local infection at block site	❑ Failure of technique ❑ Systemic drug toxicity due to overdose or intravascular injection ❑ Peripheral neuropathy due to intraneural injection ❑ Pain or hematoma at injection site

{Go through
Epidural and spinal anaesthesia :

- **Anatomy of Spinal/Epidural Area**
  - **The spinal cord lies within the spinal canal.**
  - **Surround by meanings, dura mater sub archnoid space, then piamatter, end by hoarse tail (Couda equina)**
  - **The spinal cord receives blood supply from ant spinal artery, and posterior spinal artery.**
  - **Spinal cord extends to L2, dural sac to S2**
  - **Nerve roots (cauda equina) from L2 to S2**

- **Needle inserted below L2 should not encounter cord, thus L3-L4, L4-L5 interspace commonly used**

Structures penetrated:

- **Skin, subcutaneous fat**
- **Supraspinous ligament**
- **Interspinous ligament**
- **Ligamentum flavum (last layer before epidural space)**
- **Dura + arachnoid for spinal anesthesia**
- **Spinal anaesthesia:**

❑ Relatively small LA dose injected into subarachnoid space in the dural sac surrounding the spinal cord + nerve roots
❑ LA solution may be made hyperbaric (of greater specific gravity (SG) than the cerebrospinal fluid (CSF) by mixing with 10% dextrose, thus increasing spread of LA to the dependent (low) areas of the subarachnoid space
Epidural anaesthesia:
❑ LA deposited in epidural space (potential space between ligamentum flavum and dura)
❑ Solutions injected here spread in all directions of the potential space; SG of solution does not affect spread
❑ Initial blockade is at the spinal roots followed by some degree of spinal cord anesthesia as LA diffuses into the subarachnoid space through the dura
❑ Larger dose of LA used}

Spinal vs epidural anesthesia:

SPINAL	EPIDURAL
• Easier to perform • Smaller dose of LA required (usually < toxic IV dose) • Rapid blockade (onset in 2-5 minutes) • Very effective blockade Hyperbaric LA solution - position of patient important	• Technically more difficult; greater failure rate • Larger volume/doses of LA (usually > toxic IV dose) • Significant blockade requires 10-15 minutes • Effectiveness of blockade can be variable • Use of catheter allows for continuous infusion or repeat injections • Slower onset of side effects • Position of patient not as important • SG of LA solution not as important

Complications:

Spinal Anaesthesia	Epidural Anaesthesia
– Failure of technique – Hypotension, bradycardia if block reaches T2-4 (sympathetic nervous system (SNS) block) – Post-spinal headache – Extensive spread of anesthetic ("high spinal") – Persistent paresthesias (usually transient)	– Failure of technique – Hypotension - common – Bradycardia if cardiac sympathetics blocked (only if T2-4 block) – Systemic toxicity of LA (accidental intravenous) – Accidental subarachnoid injection can lead to total spinal anesthesia – Catheter complications (shearing,

– Epidural or subarachnoid hematoma – Spinal cord trauma, infection	kinking, vascular or subarachnoid placement) – Epidural or subarachnoid hematoma

- **Peripheral nerve blockade:**
  - **The use of ultrasound for regional anesthesia is relatively new.**
  - **Ultrasound guided regional anesthesia have largely focused on brachial plexus blockade in the interscalene, supraclavicular, infraclavicular and axillary regions.**
  - **Recent studies examining the efficacy of ultrasound guidance for femoral, sciatic, psoas compartment, celiac plexus and stellate ganglion blocks are promising, while ultrasound visualization of the epidural space can facilitate neuraxial blockade in children, adults and parturients.**

4. a) PSA is a 34KD glycoprotein enzyme produced by the columnar acinar and ductal prostatic epithelial cells.

- **PSA is present in both benign and malignant cells, although the expression of PSA tends to be reduced in malignant cells and may be absent in poorly differentiated tumours. Large amounts are secreted into the semen, and small quantities are found in the urine and blood.**
- **The function of serum PSA is unclear, although it is known to liberate the insulin-like growth factor type 1 from one of its binding proteins.**
- **75% of circulating PSA is bound to plasma proteins (complexed PSA) and metabolized in the liver, while 25% is free and excreted in the urine.**
- **Complexed PSA is stable, bound to alpha-1 antichymotrypsin and alpha-2 macroglobulin.**
- **Free PSA is unstable**
- **The normal range for the serum PSA assay in men is <4.0ng/ml, though this varies with age.**

The age-adjusted normal range for PSA

Normal PSA range (ng/ml)	
Age range	**Normal PSA range (ng/ml)**
All ages	**<4.0**
40 - 49	**<2.5**
50 - 59	**<3.5**
60 - 69	**<4.5**
>70	**<6.5**

- **In the absence of prostate cancer, serum PSA concentrations also vary physiologically, according to race and prostate volume.**

Indications for checking serum PSA:

- **Patient request, following counselling**
- **Lower urinary tract symptoms**

- **Abnormal digital rectal examination**
- **Progressive bone pain, especially back pain**
- **Unexplained anaemia, anorexia, or weight loss**
- **Spontaneous thrombo-embolism or unilateral leg swelling**
- **Monitoring of prostate cancer patients**

Increased level of serum PSA:

- **Increased levels of PSA may suggest the presence of prostate cancer.**
  - **Obesity has been reported to reduce serum PSA levels.**
  - **PSA levels can be also increased by prostatitis, irritation, benign prostatic hyperplasia (BPH), and recent ejaculation, producing a false positive result.**
  - **Digital rectal examination (DRE) has been shown in several studies to produce an increase in PSA.**

PSA velocity: Despite earlier findings recent research suggests that the rate of increase of PSA (e.g. >0.35 ng/mL/yr, the 'PSA velocity) is not a more specific marker for prostate cancer than the serum level of PSA.

The West Bengal University of Health Sciences

M.B.B.S. 3rd Professional Part – II Examination, 2017

Subject: Surgery **Time: 2 1/2 hrs.**

Paper: II **Marks: 60**

Group –A

Group – B

a) Discuss the clinical features, investigations and management of Pheochromocytoma. 4+5+6

Group – C

1. Answer in brief on (any three): 3x5 = 15

a) Myocutaneous flap.
b) Autotransfusion.
c) PCNL.
d) Subdural hemorrhage.
e) Complications of spinal anaesthesia.

Group – D

2. Write briefly on (any three): 5x3 = 15

a) Tongue ulcers.
b) Pulmonary embolism.
c) DVT.
d) ABPI.
e) Ludwig's Angina.

2. b) Answer: Pheochromocytoma is said to follow the rule of ten:

- **10% are multifocal;**
- **10% are bilateral;**
- **10% are extra-adrenal;**
- **10& are malignant;**
- **10% occur in children.**

Clinical features: Symptoms and signs are caused by catecholamine excess and are typically intermittent.

- **Excess catecholamine secretion leads to characteristic episodes of;**

Headache;	Sweating;	Palpitations

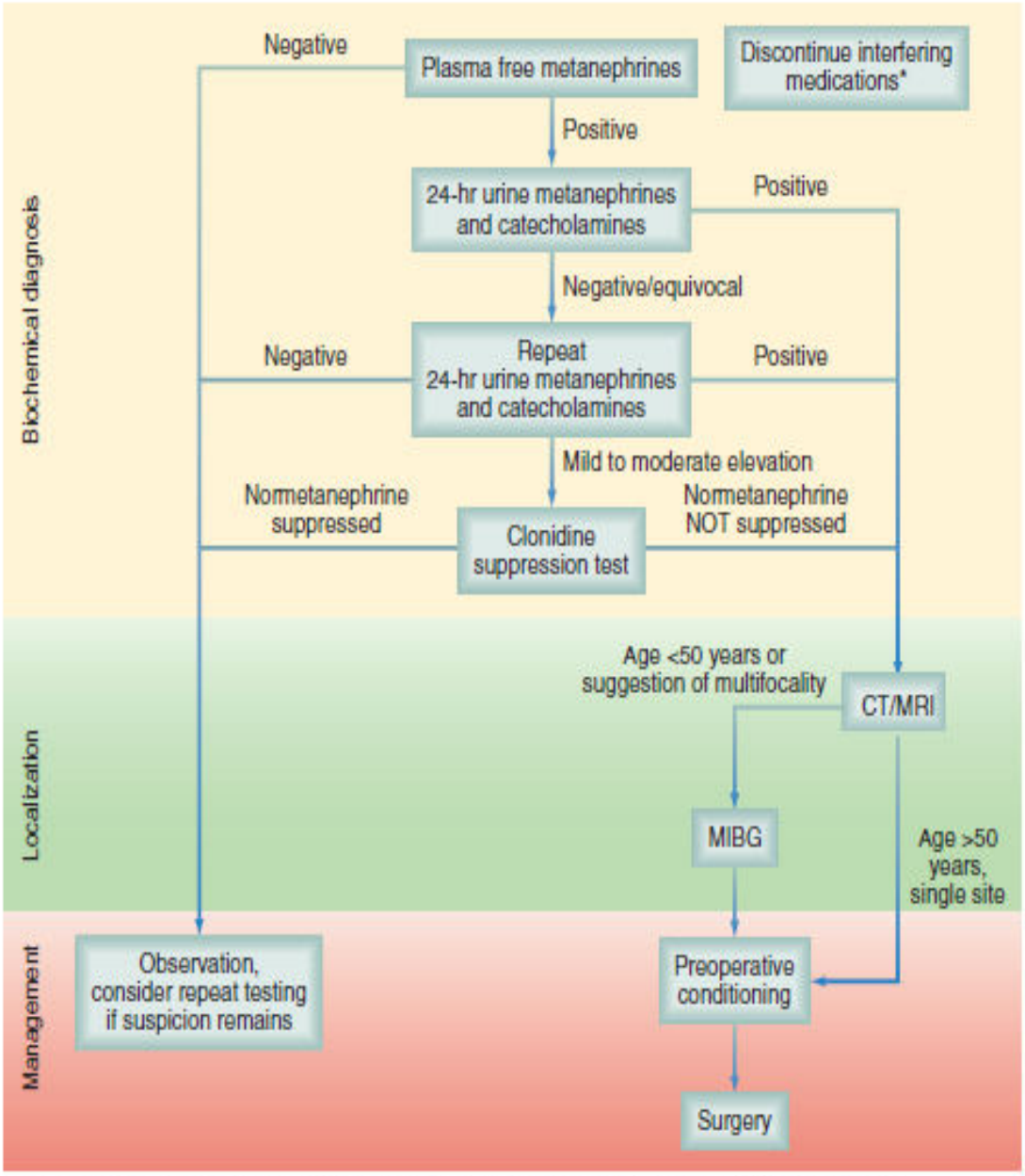
  - **Paroxysmal hypertension, tachydysrhythmias, and a feeling of impending doom or death may also occur.**

- **Attacks can be triggered by activities causing mechanical pressure on the tumour (e.g. physical exercise, defecation, intercourse), by ingestion of alcohol, labour, general anaesthesia, and surgical procedures.**
- **Only 50% of patients have persistent hypertension. The other 50% have normal blood pressure or are hypotensive between the acute episodes.**

Clinical signs of phaeochromocytoma:

• **Hypertension** ○ **Paroxysmal** ○ **Continuous**	• **Headache**	• **Palpitation**	• **Weight loss**	• **Nausea**
	• **Sweating**	• **Pallor**	• **Hyperglycaemia**	• **Psychological effects**

Algorithm for the diagnosis, localization, and management of pheochromocytoma:



*Including sympathomimetics, phenoxybenzamine, acetaminophen, many psychotropic drugs.

Diagnosis and investigations: Consider the diagnosis in patients with characteristic paroxysmal episodes, in those with unusually labile or intermitted hypertension, in those with a family history of phaeochromocytoma or related conditions (see MEN syndromes), and in hypertensive children.

- **24h urine collection and assessment for VMA and NorAd is most accurate for diagnosis.**

- **Clonidine suppression test (failure of urine levels to fall after clonidine dose) confirms the diagnosis where urine levels are border line.**
- **Provocative testing (e.g. stimulation with bolus IV glucagons) is rarely necessary and risky.**

Localizing studies:

- **Thoraco-abdominal CT or MRI scanning: first-line test especially for adrenal and sympathetic chain tumours.**
- **MIBG (meta-iodo-benzyl-guanidine) scanning localizes extra-adrenal sites not seen on CT or MRI.**

Treatment:

Medical treatment:

- **It is imperative to control the blood pressure prior to any surgery**
- **Alpha-blockers are given(e.g. phenoxybenzamine 10mg bd/tds up to the maximum dose tolerated) until hypertension controlled.**
- **Beta-blockade (e.g. propranolol) can be added after hypertension is controlled to control the beta-adrenergic effects (tachycardia).**
- **Alternative treatments with doxazosin (alpha/beta blocker) or calcium channel blockers have been described but are not widely used.**

Surgical treatment:

- **The principle of surgery is complete resection of the tumour (with clear negative margins if suspected of malignancy).**
- **Laparoscopic adrenalectomy is the treatment of choice for smaller adrenal tumours (< 8-10cm); open adrenalectomy for larger tumours.**
- **Local or radical excision are appropriate for extra-adrenal tumours.**

Postoperative:

- **Patients should be observed for 24 hours in the intensive care or high dependency unit as hypovolaemia and hypoglycaemia may occur.**
- **Lifelong yearly biochemical tests should be performed to identify recurrent, metastatic or metachronous phaeochromocytoma.**

3. b) Autotransfusion – A process of collecting a patients own shed blood , cleaning it, and giving back the washed red blood cells.

Autologous Blood – Blood donated and received by the same person.

Benefits of Autotransfusion:

- **Lower risk of clerical error**
- **Significant reduction in blood-borne infection**
- **Fresh red blood cells**
- **Better oxygen transfer**
- **More cost-effective medicine**

Mechanism of autotransfusion: As bleeding starts to occur, blood is collected through a suction line and collected in a sterile reservoir. The collected blood is then washed with a specialized instrument to remove contaminants & debris. This washed blood can now be safely returned to the patient, thus decreasing the risk and need for banked blood.

By collecting all of the patients' blood that was lost, the patient is able to avoid complications associated with blood bank transfusions and can get on the right track to a healthy recovery!

Indications: Autotransfusion can be utilized in many other surgical procedures including:

- **Cardiothoracic Surgery**
- **Vascular Surgery**
- **Orthopedic Surgery**
- **General Surgery**
- **Gynecological Surgery**
- **Urological Procedures**
- **Trauma**
- **Organ transplantation procedures**
- **Selected neurosurgical procedures**

3. d) Subdural haematoma.

Answer. Introduction: A subdural hematoma or subdural haematoma, also known as a subdural hemorrhage (SDH), is a type of hematoma, a form of traumatic brain injury. Blood gathers within the outermost meningeal layer, between the dura mater, which adheres to the skull, and the arachnoid mater, which envelops the brain.

Usually resulting from tears in bridging veins which cross the subdural space, subdural hemorrhages may cause an increase in intracranial pressure (ICP), which can cause compression of and damage to delicate brain tissue.

Subdural hematomas are often life-threatening when acute.

Chronic subdural hematomas, however, have better prognosis if properly managed.

Classification: Subdural hematomas are divided into acute, sub-acute, and chronic, depending on their speed of onset.

Acute subdural hematomas that are due to trauma are the most lethal of all head injuries and have a high mortality rate if they are not rapidly treated with surgical decompression.

Clinical features: Symptoms of subdural hemorrhage have a slower onset than those of epidural hemorrhages because the lower pressure veins bleed more slowly than arteries.

Therefore, signs and symptoms may show up in minutes, if not immediately but can be delayed as much as 2 weeks.

If the bleeds are large enough to put pressure on the brain, signs of increased ICP or damage to part of the brain will be present.

Other signs and symptoms of subdural hematoma can include any combination of the following:

A history of recent head injury	**Numbness**	**Weakness or lethargy**	**Ataxia, or difficulty walking**
Loss of consciousness or fluctuating levels of consciousness	**Headache (either constant or fluctuating)**	**Nausea or vomiting**	**Altered breathing patterns**
Irritability	**Dizziness**	**Loss of appetite**	**Hearing loss or hearing ringing (tinnitus)**
Seizures	**Disorientation**	**Personality changes**	**Blurred Vision**
Pain	**Amnesia**	**Inability to speak or slurred speech**	**Deviated gaze, or abnormal movement of the eyes**

Pathophysiology: Collected blood from the subdural bleed may draw in water due to osmosis, causing it to expand, which may compress brain tissue and cause new bleeds by tearing other blood vessels. The collected blood may even develop its own membrane.

Substances that cause vasoconstriction may be released from the collected material in a subdural hematoma, causing further ischemia under the site by restricting blood flow to the brain. When the brain is denied adequate blood flow, a biochemical cascade known as the ischemic cascade is unleashed, and may ultimately lead to brain cell death.

The body gradually reabsorbs the clot and replaces it with granulation tissue.

Diagnosis:

Subdural hematomas occur most often around the tops and sides of the frontal and parietal lobes.They also occur in the posterior cranial fossa, and near the falxcerebri and tentorium cerebelli.Unlike epidural hematomas, which cannot

expand past the sutures of the skull, subdural hematomas can expand along the inside of the skull, creating a concave shape that follows the curve of the brain, stopping only at thedural reflections like the tentorium cerebelli and falxcerebri.

On a CT scan, subdural hematomas are classically crescent-shaped, with a concave surface away from the skull. However, they can have a convex appearance, especially in the early stage of bleeding.

Treatment: It is important that a patient receive medical assessment, including a complete neurological examination, after any head trauma. A CT scan or MRI scan will usually detect significant subdural hematomas.

Treatment of a subdural hematoma depends on its size and rate of growth.

Some small subdural hematomas can be managed by careful monitoring until the body heals itself.

Other small subdural hematomas can be managed by inserting a temporary small catheter through a hole drilled through the skull and sucking out the hematoma; this procedure can be done at the bedside.

Large or symptomatic hematomas require a craniotomy, the surgical opening of the skull.

3. e) Complications of spinal anaesthesia.

Complications are:

- **Pain - 25% of patients still experience pain despite spinal anaesthesia.**
- **Post-dural headache from cerebrospinal fluid (CSF) leak.**
- **Hypotension and bradycardia through blockade of the sympathetic nervous system.**
- **Limb damage from sensory and motor block.**
- **Epidural or intrathecal bleed.**
- **Respiratory failure if block is 'too high'.**
- **Direct nerve damage.**
- **Hypothermia.**
- **Damage to the spinal cord - may be transient or permanent.**
- **Spinal infection.**
- **Aseptic meningitis.**
- **Haematoma of the spinal cord - enhanced by use of LMWH pre-operatively.**
- **Anaphylaxis.**
- **Urinary retention.**
- **Spinal cord infarction.**
- **Anaesthetic intoxication.**

4. a) Tongue ulcers:

Tongue ulcers are open sores or cuts on the tongue. Tongue ulcers can be painful and raw and can be irritated by eating and drinking. One of the most common

types of tongue ulcers is the canker sore, which may arise for an unknown reason or be linked to a number of different irritants.

Causes of Tongue Ulcer

- **Canker sores: the most common cause of ulcers on the tongue is canker sores. These white or yellow blisters typically develop from a red patch or cut in the mouth which fills with pus as the sore grows. Canker sores come in a variety of sizes and typically have an irregular shape. Cuts in the mouth, hormonal changes, the end of the menstrual cycle, stress or a lack of iron, B-12 and folic acid in the diet can trigger the development of a canker sore. These sores are not dangerous, though they can be uncomfortable.**
- **Poor oral care: tongue ulcers can occur due to poor oral care.**
- **Infections in the mouth can manifest themselves as sores on the tongue. These sores will often appear in other parts of the mouth in addition to those which appear on the tongue itself. Gingivostomatitis, caused by viral or bacterial infection, infection of the herpes simplex virus, oral thrush, an oral yeast infection, or oral lichen planus, an immune system disorder can lead to sores developing throughout the mouth, most notably on the tongue. Those who develop sores on the tongue often may suffer from leukoplakia, a condition that causes chronic irritation of the tongue.**
- **Oral cancer: if the sores on the tongue are severe, do not fade after a few days or become discolored there may be a more serious issue causing them. Sores that grow very quickly or appear to become hard may be a sign that you are developing oral cancer.**
- **Patients suffering from ulcers on the tongue may also develop a general feeling of discomfort or uneasiness throughout the body. If the infection has started to spread, the lymph nodes may become swollen or you may develop a slight fever.**

Treatments for Tongue Ulcer:

- **Most ulcers on the tongue can be managed with home remedies. The ulcers will typically heal on their own, but remedies can be used to limit the pain and quicken the time it takes to rid the sore.**
- **Rinsing the mouth with salt water is a common recommendation for those suffering from ulcers in the mouth.**
- **Food remedies such as applying butter to the ulcer can minimize the pain. Canker sores can be treated with medication that will dull the pain and help the ulcer heal.**
- **Keeping the mouth clean is essential.**
- **Eliminate items like hot foods, foods which are spicy or acid.**
- **Treat the definitive cause.**

4. b) Pulmonary embolism.

Answer.

Aetiology:

- **95% of PE follows DVT in leg.**
- **The source of embolus in the remaining 5% is from right ventricle, pelvic, renal or hepatic veins.**
- **Embolisms of foreign bodies (e.g. bullets) or septic material are clinical curiosities.**

Clinical Features: Depend on the size of the emboli. A high index of suspicion is required for diagnosing this condition. A large majority of patients may be completely asymptomatic.
Amongst the symptomatic patients, the following symptoms may be noted.
Symptoms:The common symptoms, in decreasing order of frequency are,

- **Dyspnoea**
- **Pleuritic chest pain**
- **Cough**
- **Hemoptysis**

Signs: Are usually non-specific. These include

- **Tachypnoea (Respiratory rate > 20/min)**
- **Localized crepitations (rales)**
- **Loud P2 (second heart sound)**
- **Tachycardia**
- **Fever**
- **Evidence of DVT**

Features of massive pulmonary embolism;
These include syncope, disorientation or altered sensorium, central chest pain, central cyanosis, raised JVP, and acute cor pulmonale.
Investigations: Routine investigations include ECG, chest X ray, and arterial blood gas (ABG) analysis. Specific investigations include a ventilation/perfusion scan, angiography or spiral CT scan. Duplex scanning of leg veins is added to confirm the source of thromboembolism.
Chest X Ray The initial chest radiograph is rarely diagnostic, and often normal. Several abnormalities may be noted. These include:

- **Elevation of one dome of diaphragm**
- **Parenchymal infiltrates/infarction**
- **Oligemia of affected lungfield (→estermark's sign)**
- **Pleural effusion**

ECG is useful to exclude other causes of chest pain, notably myocardial infarction. It may show the following:

- **Sinus tachycardia**
- **T wave inversion (Leads V1-V4)and non-specific ST changes**
- **Right bundle branch block**
- **S1Q3T3 pattern with right axis deviation and RBBB (right bundle branch block) is diagnostic, but found in less than 20% of cases.**

ABG (arterial blood-gas analysis) may show low PaO_2, with a normal or low $PaCO_2$ and acidosis. However a normal PaO_2 does not exclude PE.

Ventilation/Perfusion lung scan (V/Q scan): This is the mainstay of diagnosis in patients who are not acutely ill.
Ventilation–perfusion mismatch (normal perfusion but no ventilation) is classically seen in a localized area of the lung.
Traditionally the perfusion scan is performed first, and if a perfusion defect is noted, the ventilation scan is done. Failure of a segment of lung to show perfusion in the presence of adequate ventilation is diagnostic of PE.

Pulmonary angiography is the most specific and accurate investigation in the diagnosis of PE.
It is usually done in two settings: when the diagnosis is in doubt, or when massive PE is suspected where a decision regarding surgical embolectomy or thrombolysis has to be made urgently.

Spiral (Helical) CT scan. Contrast-enhances spiral CT is replacing V/Q scan for diagnosis of PE in stable cases. It is said to be more reliable than the V/Q scan.

Treatment:

- **General supportive measures include oxygen therapy by mask or nasal prongs, pain relief by intravenous morphine (3-5 mg), correction of acidosis, fluid therapy (maintaining a CVP of about 12 mm Hg), and ionotropic support with dobutamine or isoprenaline, if indicated.**
- **Heparin remains the drug of choice for PE causing no or minimal haemodynamic disturbances. Heparin is changed to oral anticoagulants after a few days, and these should be continued for at least six months. An INR (international normalized ratio) of 2-3.5 should be maintained.**
- **Thrombolytic therapy. Thrombolytic therapy is an attractive alternative, especially in submassive and massive PE. It may also be used in patients of PE who do not respond adequately to heparin therapy. It is also useful in patients with underlying cardio-pulmonary disease, who have a prohibitive surgical risk (of dying from surgical embolectomy).**
- **Tissue plasminogen activator is probably better with fewer side effects and a better clot lysis rate.**
- **Pulmonary Embolectomy: This approach is still being practiced, especially in centers where facilities for cardiopulmonary bypass are not available, even though the mortality approaches 50%. An alternative (and better) surgical approach to pulmonary artery is by median sternotomy with cardiopulmonary bypass. Some form of pulmonary embolectomy (surgical, catheter aspiration) is indicated in massive PE. In a patient with sudden collapse and no right-sided cardiac output, emergency open surgical embolectomy can be life saving.**

- **Catheter Embolectomy An embolectomy catheter with a suction-cup at its tip is introduced via the jugular vein or femoral vein, and negotiated into the pulmonary artery. The thrombus is sucked into the catheter and pulled back to the phlebotomy incision, maintaining he suction on the cup. It is then delivered out of the phlebotomy incision.**
- **Inferior Vena Cava Filters These are an alternative to IVC (inferior vena cava) ligation or plication in patients with repeated PE.**

- **Indications for IVC Filter placement in Pulmonary Embolism:**

Absolute Indications	Relative indications
• Anticoagulation (AC) contraindicated • Recurrent PE despite anticoagulation • Bleeding forcing discontinuation of AC • After pulmonary embolectomy • Failure of IVC interruption	• Large (>5 cm) free-floating iliac thrombus • Propagating thrombus despite AC • Chronic PE with cor pulmonale • High-risk patient * • Septic PE

A high risk patient is one with significant COPD (chronic obstructive pulmonary disease) with > 50% decrease of pulmonary bed who would not be able to tolerate even minor PE from DVT

The West Bengal University of Health Sciences

M.B.B.S. 3rd Professional Part – II Examination, 2016

Subject: Surgery **Time: 2 1/2 hrs.**

Paper: II **Marks: 60**

Group –A

1. **Enumerate the causes of painless haematuria. Discuss the investigation and treatment in a patient of 65 years presented with painless haematuria. 5+5+5 = 15**
   Answer. Causes of painless haematuria:

Causes	Diagnosis	Treatment
Renal Tumours: **The commonest primary renal tumour is renal cell carcinoma, an adenocarcinoma of collecting tubule origin. It commonly presents with haematuria.**	**By ultrasound scanning. Diagnosis is made by CT scanning**	**Treatment is by surgical excision**
Transitional Cell carcinoma of the renal collecting system usually gives haematuria.	**Diagnosis may be difficult, requiring retrograde imaging and ureteroscopy.**	**Treatment is by either local excision or, for high grade or larger lesions, nephro-ureterectomy. Immunotherapy is used for metastases with limited success; radiotherapy has little place except for palliation of bone metastases.**
Angiomyolipoma is a hamartomatous lesion, which may grow to great size and be associated with major haemorrhage;.	**CECT.**	**Treatment is by radiological emobolisation or surgery, conserving normal renal tissue where possible**
Stone disease:	**IVU**	**ESWL** **PCNL** **Open surgery**
Glomerulonephritis		

Pyelonephritis (ascending urinary tract infection)		
Papillary Necrosis: This condition occurs in diabetics and in patients with deficiencies of oxygenation, particularly sickle cell disease.		
Ureteric stones	**IVU**	**ESWL** **Ureterscopic retrieval**
Cystitis: Typically cystitis is painful and in men is commonly associated with bladder outflow obstruction. Schistosomiasis, interstitial cystitis and drug related cystitis are rarer causes of bladder inflammation causing bleeding.	**Diagnosis is by urine microscopy and culture, assisted by cystoscopy. Bladder biopsies may be necessary.**	
Bladder tumour	**Biopsy** **CECT** **MRI**	**TURBT** **Intravesical chemotherapy** **Chemotherapy** **Cystectomy**
BHP **Postatic adenocarcinoma**	**USG** **TRUS – Biopsy** **PSA**	**TURP** **Surgery** **Hormone therapy**
Rare causes of haematuria **Arteriovenous malformations,** **Tuberculosis,** **Arteritis,** **Patients on anticoagulant therapy**		

Investigations of Haematuria: A general physical examination (blood pressure, the prostate in a male and the gynaecological organs in a female),

- **First investigation in a patient with reported haematuria is urinary examination.**

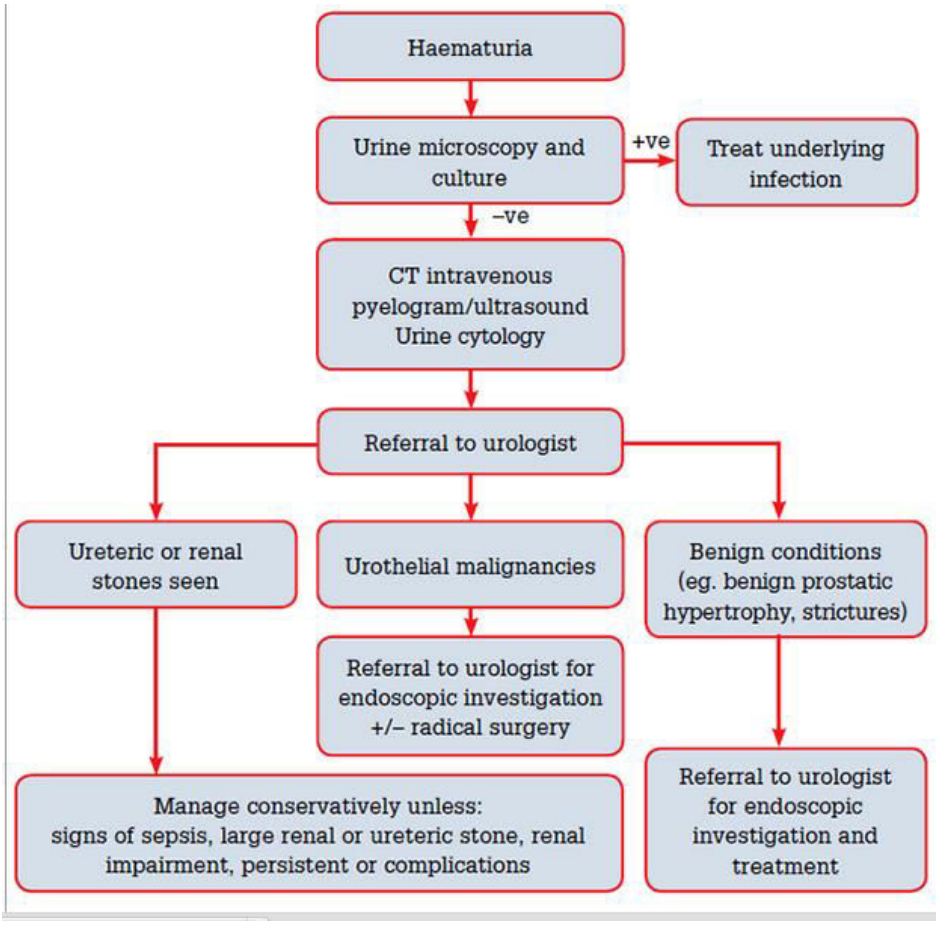
This must include microscopy for red and white blood cells and bacteria.
The presence of any crystals, ova or parasites should be noted and culture of a mid stream specimen carried out.
If schistosomiasis or tuberculosis is suspected a first void urine sample is usually needed.
The level of protein in the urine must be assessed, but in heavy haematuria it may be difficult to be sure if light proteinuria is due to the haemoglobin present.
If no red blood cells are found in the urine but haemoglobin is present the patient should be investigated for causes of haemoglobinuria

- **All patients should have a full blood count with an erythrocyte sedimentation rate.**
- **Serum urea, creatinine and electrolytes should be measured, along with albumin, calcium and liver function tests if the patient is unwell or in renal failure.**
- **The presence or absence of proteinuria may guide initial investigation, since the combination of these favour a glomerular problem. Here C reactive protein and24 hour urine protein excretion will be informative, as may serum electrophoresis and autoantibodies.**
- **Ultrasound will show renal cortical thickness and density.**
- **In the majority of cases a renal biopsy with immunoglobulin histochemistry will be necessary to make a definitive diagnosis.**
- **In cases of microscopic haematuria without proteinuria, and all macroscopic cases, a "surgical" investigation plan can be followed.**
- **Other imaging may be done either by intravenous urography or a combination of plain abdominopelvic radiography and ultrasound of the urinary tract.**
- **If no abnormality is found then a flexible cystoscopy under local anaesthetic may be performed,**
- **Bladder lesion will require a transurethral biopsy and examination under anaesthetic for both treatment and diagnosis.**

Management:

Approach to haematuria:

- **Thorough history including urinary symptoms**
- **Recent history (trauma/muscle injury/causes of factitious haematuria/exexrcise/foreign travel)**
- **Systemic features (fever, weight loss) other symptoms (bleeding, bruising)**
- **Co-morbidity**
- **Drug history**
- **Occupation**
- **Family history**



Conventional urological investigation involves urine culture (where, on the basis of associated cystitis, symptoms urinary infection is suspected), urine cytology, cystoscopy, renal ultrasonography, and IVU.

Diagnostic cystoscopy:

Nowadays this is carried out using a flexible, fibreoptic cystoscope, unless radiological investigation demonstrates a bladder cancer, in which case one may forego the flexible cystoscopy and proceed immediately to rigid cystoscopy and biopsy under anaesthetic (transurethral resection of bladder tumourTURBT).

Treatment:

General measures:

- **Haemodynamically stabilise the patient.**
- **Assess for anaemia: if required blood transfusion after admission.**
- **If gross haematuria: foley's catheterisation.**
- **Antibiotics.**

- **Urinary tract infection should be ruled out before any further investigations for haematuria are undertaken.**
- **Ureteric and renal stones can cause episodes of haematuria, however it is important to consider other causes if this does not settle or if there are risk factors for urinary tract malignancy.**
- **Initial investigations for haematuria should include CT-IVP, urine cytology, full blood examination, renal function, and PSA in men.**
- **A urological referral is recommended in patients presenting with macroscopic haematuria, persistent microscopic haematuria, abnormal urine cytology, irritative lower urinary tract symptoms or recurrent urinary tract infections.**
- **Find out the cause.**
- **Treat the cause.**

Group – B

2. Answer any of the following questions:

a) Classify thyroid cancer. Discuss the management of F.N.A.C proved follicular neoplasm of (R) lobe of thyroid in a lady of 45 years. 5+5+5 = 15

Answer. Classification of thyroid neoplasms

***Benign* - Follicular adenoma**

***Malignant* - Primary**

- **Follicular epithelium –differentiated**
  - **Follicular**
  - **Papillary**
- **Follicular epithelium –undifferentiated**
  - **Anaplastic**
- **Parafollicular cells**
  - **Medullary**
- **Lymphoid cells**

Lymphoma

Secondary

- **Metastatic**
- **Local infiltration**

The work up includes:

History - short history (not necessary in WDTC) and recent rapid increase in size.

Symptoms of local involvement viz change n voice, respiratory difficulty, dysphagia.

Physical examination- hard consistency, local limitation of mobility, involvement of strap muscles, and obliteration of carotid pulse all suggests malignancy. Pizello's method, Lahey's method and Berry's sign are all contributory.

Evidence of metastases in bones, lungs and liver besides cervical lymph nodes.

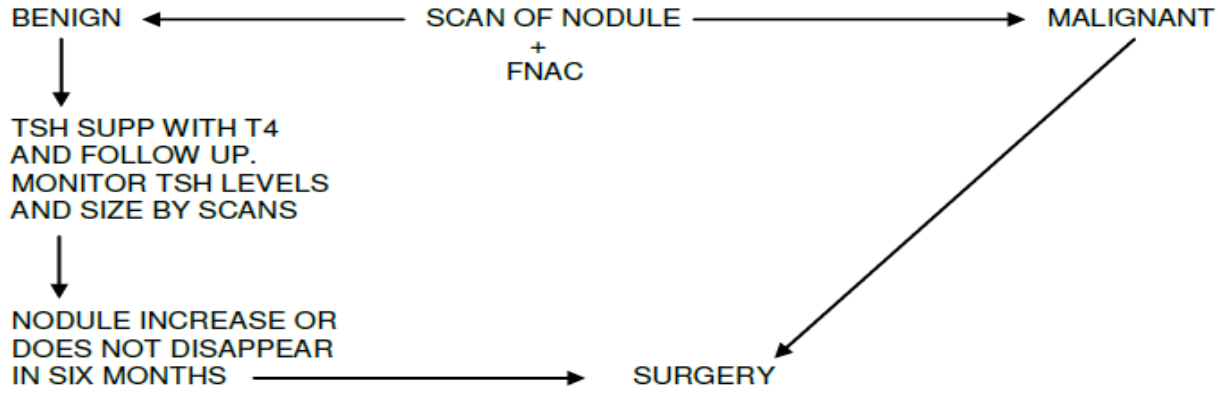
Dysfunction of Thyroid, Functioning tumours are known to produce clinical hyperthyroidism in 5% cases.

Investigations:

High Resolution Ultrasound: Halo sign, commet tail sign, and type 3 intranodular vascularity of malignancy are helpful. Involvement of trachea, vessels, strap muscles can be made out.

Fine Needle Aspiration Cytology: Cellular diagnosis of DTC is now feasible; ultrastructural study and histo/histocytochemistry are a great help.
Trucut Biopsy:Selected indication is in inoperable cases to plan out management. Also to differentiate lymphoma from anaplastic carcinoma.
Monoclonal antibodies for lymphoma: This can differentiate anaplastic carcinoma from lymphoma.
Imprint smear: Better interpretation than FNAC/ Frozen section.
CT/MRI: Specific indication is to know extent of tumour spread and local tissue plane infiltration.
Scintiscan: Presently limited use. Cold nodule classically in malignancy. Still helpful in solitary nodule thyroid.
Tumor markers: Very significant is TGB which if done before and after management helps in follow up and can indicate recurrence/ mets. CEA, CD-34, MVD, p 53 protein have also correlation.
X-ray Chest and X-ray of bone met site: Demonstrates local secondary deposit.
Ultrasound Liver: Done to detect liver metastases wherever suspected.
Skeletal survey: Done by radionuclide scan (Gamma camera) wherever indicated can alter the
clinical staging of the disease.

Flow chart:



One point to note that F.N.A.C cannot distinguish between follicular adenoma and carcinoma. So we have to go for right subtotal thyroidectomy and send the specimen for HP examination to find out the final diagnosis and treat accordingly.

Surgery for Follicular Carcinoma:

- **From clinicopathological stand point these carcinomas are divided into low risk and high risk.**
- **Three important risk factors viz vascular invasion, metastases and age greater than 45 years.**
- **The result of cytological and frozen section histology in this tumour is high in accuracy of both in interpretation.**

- **In low risk patient with intrathyroidal (non invasive) lesion less than 2.5 cm in size (determined on scan) a hemithyroidectomy with isthumusectomy suffices.**
- **Completion thyroidectomy is indicated if histology reveals a more invasive form of carcinoma postoperatively.**
- **In all other follicular carcinoma, a total or near total thyroidectomy is indicated.**

This is recommended in presence of even metastases (the incidence of which is fairly high with this carcinoma) as it facilitates adjuvant treatment with I -131.

- **If nodes are involved, ipsilateral functional block dissection with central compartment clearance is to be undertaken.**

Hürthle Cell Tumors:

- **Hürthle cell, though modified follicular cell is now considered a separate tumor altogether, contrary to earlier belief, majority HCN are benign.**
- **Capsular and vascular invasion on histology and metastases clinically characterize malignant HCN.**
- **There is an indeterminate form also. The potential incidence of indeterminate forms creates controversy regarding extent of surgery.**
- **The undisputed benign forms are best treated by hemithyroidectomy.**
- **For ―aggressive‖ benign type, indeterminate type and frankly malignant HCN, a total or near thyroidectomy is the standard.**
- **If malignancy is found on post operative histology in a pre operative diagnosed benign lesion, a completion thyroidectomy is then indicated.**
- **The approach to lymph nodes is same as in follicular carcinoma; if nodes are involved, ipsilateral functional block dissection with central compartment clearance is to be undertaken.**

Treatment: Low-Versus High-Risk Criteria in Papillary Thyroid Cancer

Low risk	High risk
1. Women < 50 years 2. Men < 40 years 3. Well-or moderately differentiated tumors 4. Tumor < 4 cm in diameter 5. Tumor confined to the thyroid gland 6. No distant metastases	1. Women ≥ 50 yr 2. Men ≥ 40 yr 3. Poorly differentiated tumors, tall-cell, columnar cell, or oxyphilic variants. 4. Tumor ≥ 4 cm in diameter 5. Local invasion 6. Distant metastases

Surgical treatment:

- **A lobectomy and isthmusectomy is satisfactory for papillary cancers less than 1.0 cm in size without lymphatic or systemic metastases.**
- **Lobectomy with isthmusectomy - for patients with papillary cancers that are greater than 1.0 cm when patients are categorized as low risk for recurrence or mortality.**
- **Total thyroidectomy is uniformly accepted for treatment of high-risk papillary carcinoma.**
- **In addition, patients with a history of head and neck irradiation should undergo total thyroidectomy due to the high incidence of carcinoma at sites other than the nodule and their increased lifetime risk of developing thyroid cancer in the remaining thyroid gland.**

Role of post operative remnant ablation:

Indications:

• Stage III and IV disease

• All patients with stage II disease younger than age 45 years

• Most patients with stage II disease 45 years or older
• Selected patients with stage I disease - especially those with multifocal disease, nodal metastases, extra-thyroidal or vascular invasion, and/or more aggressive histologies.

Protocol of thyroid hormone withdrawal for remnant ablation
Aim: Achieve TSH of more than 30 mU/L

- **Single-dose exogenous rTSH results in TSH levels between 51–82 mU/L**
- **Endogenous TSH elevation can be achieved by**
  - **Stopping levothyroxine (LT4) and switching to levo-triiodothyronine (LT3) for 2–4 weeks followed by withdrawal of triiodothyronine (T3) for 2 weeks**
  - **Discontinuation of LT4 for 3 weeks without use of T3**
  - **rTSH stimulation can be used for remnant ablation.**

Role of post operative diagnostic RAIU scan:
• Indication:
– When the extent of thyroid remnant cannot be assessed from the surgical report/ USG
– If the result is likely to alter the subsequent management
– If the result is likely to alter the dose of RAI treatment
• If used, low dose I 131 (1-3mCi)/ I123 scan recommended
– Low iodine diet (50microgram/day for 1-2 weeks) is recommended prior to scan
• Post therapy scan:

- **Detects additional foci in 10-26% cases, alters the stage in 10%**
- **Done 1 week post radio-iodine therapy.**

Role of TSH suppression therapy:
Mechanism of action:

- **Decreases the expression of thyroid specific proteins**
- **Decreases the rate of cell growth**

- **High risk pts:**
  - **Keep TSH < 0.1mIU/L**
  - **After remission continue suppressive therapy x 3-5 yrs**
- **Low risk patients**
  - **TSH= 0.1-0.5 mIU/L**
  - **After remission keep it between 0.5-1mIU/mL**

There is no role for routine adjunctive chemotherapy.

b) What are the aetiologies of pancreatitis? How will you investigate and treat a case of acute pancreatitis? 5+5+5 = 15

Answer. See the Question.1 of Group – A of Supplementary Paper – I of 2011

Aetiologies of acute pancreatitis:

Metabolic	Mechanical	Vascular	Infection
○ Alcohol ○ Hyperlipoproteinemi	○ Cholelithiasis ○ Postoperative	○ Postoperative (cardiopulmonar	○ Mumps ○ Coxsackie B

a ○ Hypercalcemia ○ Drugs ○ Genetics ○ Scorpion venom	○ Pancreas divisum ○ Post-traumatic ○ Retrograde pancreatography ○ Pancreatic duct obstruction: pancreatic tumor, ascaris infestation ○ Pancreatic ductal bleeding ○ Duodenal obstruction	y bypass) ○ Periarteritis nodosa ○ Atheroembolism	○ Cytomegalovirus ○ Cryptococcus

Aetiologies of chronic pancreatitis:

- **Intraductal plugging and obstruction - Eg, ethanol (ETOH) abuse, stones, tumors**
- **Direct toxins and toxic metabolites - These act on the pancreatic acinar cell to stimulate the release of cytokines, which stimulate the stellate cell to produce collagen and to establish fibrosis; cytokines also act to stimulate inflammation by neutrophils, macrophages, and lymphocytes (eg, ETOH, tropical sprue)**
- **Oxidative stress - Eg, idiopathic pancreatitis**
- **Necrosis-fibrosis - Recurrent acute pancreatitis that heals with fibrosis**
- **Ischemia - From obstruction and fibrosis; important in exacerbating or perpetuating disease rather than in initiating disease**
- **Autoimmune disorders - Chronic pancreatitis has been found in association with other autoimmune diseases, such as Sjögren syndrome, primary biliary cirrhosis, and renal tubular acidosis.**
- **Secondary forms of autoimmune chronic pancreatitis are associated with primary biliary cirrhosis, primary sclerosing cholangitis, and Sjögren syndrome.**
- **While alcohol greatly influences the understanding of its pathophysiology because it is the most common etiology (60-70%), approximately 20-30% of cases are idiopathic and 10% of cases are due to rare diseases.**

Investigations and diagnosis:

- **Blood Investigations - Full blood count, Renal function tests, Liver Function, serum calcium, serum amylase and lipase, Arterial blood gas.**
- **Imaging - Chest Xray (for exclusion of perforated viscus), Abdominal Xrays (for detection of "sentinel loop" dilated duodenum sign, and gallstones which are radioopaque in 10%) and CT abdomen**
- **Amylase and lipase:Elevated serum amylase and lipase levels, in combination with severe abdominal pain, often trigger the initial diagnosis of acute pancreatitis.**
- **Serum lipase rises 4 to 8 hours from the onset of symptoms and normalizes within 7 to 14 days after treatment.**
- **Serum amylase may be normal (in 10% of cases) for cases of acute or chronic pancreatitis (depleted acinar cell mass) and hypertriglyceridemia.**
- **Reasons for false positive elevated serum amylase include salivary gland disease (elevated salivary amylase) and macroamylasemia.**

- **If the lipase level is about 2.5 to 3 times that of amylase, it is an indication of pancreatitis due to alcohol.**

Computed tomography

Regarding the need for computed tomography, practice guidelines state:

"Many patients with acute pancreatitis do not require a CT scan at admission or at any time during the hospitalization. For example, a CT scan is usually not essential in patients with recurrent mild pancreatitis caused by alcohol. A reasonable indication for a CT scan at admission (but not necessarily a CT with IV contrast) is to distinguish acute pancreatitis from another serious intra-abdominal condition, such as a perforated ulcer."

"Patients with persisting organ failure, signs of sepsis, or deterioration in clinical status 6–10 days after admission will require CT (recommendation grade B)."

CT abdomen should not be performed before the 1st 48 hours of onset of symptoms as early CT (<48 h) may result in equivocal or normal findings.

CT Findings can be classified into the following categories for easy recall :

Intrapancreatic - diffuse or segmental enlargement, edema, gas bubbles, pancreatic pseudocysts and phlegmons/abscesses (which present 4 to 6 wks after initial onset)

Peripancreatic / extrapancreatic - irregular pancreatic outline, obliterated peripancreatic fat, retroperitoneal edema, fluid in the lessar sac, fluid in the left anterior pararenal space

Locoregional - Gerota's fascia sign (thickening of inflamed Gerota's fascia, which becomes visible), pancreatic ascites, pleural effusion (seen on basal cuts of the pleural cavity), adynamic ileus, etc.

- **Magnetic resonance imaging:**

While computed tomography is considered the gold standard in diagnostic imaging for acute pancreatitis, magnetic resonance imaging (MRI) has become increasingly valuable as a tool for the visualization of the pancreas, particularly of pancreatic fluid collections and necrotized debris.Additional utility of MRI includes its indication for imaging of patients with an allergy to CT's contrast material, and an overall greater sensitivity to hemorrhage, vascular complications, pseudoaneurysms, and venous thrombosis.

Another advantage of MRI is its utilization of magnetic resonance cholangiopancreatography (MRCP) sequences. MRCP provides useful information regarding the etiology of acute pancreatitis, i.e., the presence of tiny biliary stones (choledocholithiasis or cholelithiasis) and duct anomalies. Clinical trials indicate that MRCP can be as effective a diagnostic tool for acute pancreatitis with biliary etiology as endoscopic retrograde cholangiopancreatography, but with the benefits of being less invasive and causing fewer complications.

Classification by severity

Progression of pathophysiology

Acute pancreatitis can be further divided into mild and severe pancreatitis. About 20% of the acute pancreatitis are severe with a mortality of about 20%. This is an important classification as severe pancreatitis will need intensive care therapy whereas mild pancreatitis can be treated on the common ward.

Necrosis will be followed by a systemic inflammatory response syndrome (SIRS) and will determine the immediate clinical course. The further clinical course is then determined by bacterial infection. SIRS is the cause of bacterial (Gram negative) translocation from the patient's colon.

There are several ways to help distinguish between these two forms. One is the above mentioned Ranson Score.

Prognostic indices

In predicting the prognosis, there are several scoring indices that have been used as predictors of survival. Two such scoring systems are the Ranson criteria and APACHE II (Acute Physiology, Age and Chronic Health Evaluation) indices. Most, but not all studies report that the Apache score may be more accurate.

Pracactice guidelines state:

"The two tests that are most helpful at admission in distinguishing mild from severe acute pancreatitis are APACHE-II score and serum hematocrit. It is recommended that APACHE-II scores be generated during the first 3 days of hospitalization and thereafter as needed to help in this distinction. It is also recommended that serum hematocrit be obtained at admission, 12 h after admission, and 24 h after admission to help gauge adequacy of fluid resuscitation."

"Immediate assessment should include clinical evaluation, particularly of any cardiovascular, respiratory, and renal compromise, body mass index, chest x ray, and APACHE II score".

Ranson criteria is a clinical prediction rule for predicting the severity of acute pancreatitis.

At admission
Age in years > 55 years

White blood cell count > 16000 cells/mm3

Blood glucose > 10 mmol/L (> 200 mg/dL)

Serum AST > 250 IU/L

Serum LDH > 700 IU/L

At 48 hours
Calcium (serum calcium < 2.0 mmol/L (< 8.0 mg/dL)

Hematocrit fall > 10%

Oxygen (hypoxemia PO2 < 60 mmHg)

BUN increased by 1.8 or more mmol/L (5 or more mg/dL) after IV fluid hydration

Base deficit (negative base excess) > 4 mEq/L

Sequestration of fluids > 6 L

The criteria for point assignment is that a certain breakpoint be met at anytime during that 48 hour period, so that in some situations it can be calculated shortly after admission. It is applicable to both gallstone and alcoholic pancreatitis.

Alternatively, pancreatitis can be diagnosed by meeting any of the following:

APACHE II score

Apache score of ≥ 8 Organ failure Substantial pancreatic necrosis (at least 30% glandular necrosis according to contrast-enhanced CT)

Interpretation If the score ≥ 3, severe pancreatitis likely. If the score < 3, severe pancreatitis is unlikely Or

Score 0 to 2 : 2% mortality Score 3 to 4 : 15% mortality Score 5 to 6 : 40% mortality Score 7 to 8 : 100% mortality

"Acute Physiology And Chronic Health Evaluation" (APACHE II) score > 8 points predicts 11% to 18% mortality.

Balthazar scoring: Computed Tomography Severity Index (CTSI) is a grading system used to determine the severity of acute pancreatitis. The numerical CTSI has a maximum of ten points, and is the sum of the Balthazar grade points and pancreatic necrosis grade points:

CT SEVERITY INDEX AND MODIFIED CTSI

CT SEVERITY INDEX (BALTHAZAR, 1990)		MODIFIED CT SEVERITY INDEX (MORTELE, 2004)	
PROGNOSTIC INDICATOR	POINTS	PROGNOSTIC INDICATOR	POINTS
PANCREATIC INFLAMMATION		PANCREATIC INFLAMMATION	
NORMAL PANCREAS	0	NORMAL PANCREAS	0
ENLARGED PANCREAS	1	PANCREATIC ABN +/- PERIPANCREATIC INFLAMMATION	2
PANCREATIC ABNORMALITIES WITH PERIPANCREATIC INFLAMMATION	2	PANCREATIC OR PERIPANCREATIC FLUID COLLECTION/ FAT NECROSIS	4
SINGLE FLUID COLLECTION	3		
2/MORE COLLECTION OR GAS	4		

CT SEVERITY INDEX (BALTHAZAR,1990)		MODIFIED CT SEVERITY INDEX (MORTELE,2004)	
PROGNOSTIC INDICATOR	POINTS	PROGNOSTIC INDICATOR	POINTS
PANCREATIC NECROSIS		PANCREATIC NECROSIS	
NONE	0	NONE	0
<30%	2	<30%	2
30-50%	4	>30%	4
>50%	6	EXTRAPANCREATIC COMPLICATIONS	2

CTSI's staging of acute pancreatitis severity has been shown by a number of studies to provide more accurate assessment than APACHE II, Ranson, and C-reactive protein (CRP) level. However, a few studies indicate that CTSI is not significantly associated with the prognosis of hospitalization in patients with pancreatic necrosis, nor is it an accurate predictor of AP severity.

Glasgow Imrie criteria

3 or more positive criteria within 48h of admission = severe attack

Age > 55y

WBC > 15 000

Glucose > 7mmol/L

Blood urea > 7mmol/L

Albumin < 35g/L

Corrected calcium < 2mmol/L

PaO_2 < 10kPa

Marshall Scoring System

Most sensitive for evaluation of AP patients.

- 50% of the patients with necrotising acute pancreatitis develop organ failure with severe acute pancreatitis.
- 15% of edematous acute pancreatitis develop organ failure.

Score > 3 is associated with

- Severe course,
- Systemic complications and
- Significant correlation with fatal outcome (P = 0.007) .

Criteria for organ failure based on Marshall scoring system:

Organ system	Score				
	0	1	2	3	4
Respiratory (PaO2/FiO2)	>400	301-400	201-300	101-200	<100
Renal (Serum Creatinine md/dl)	≤1.5	>1.5 - ≤ 1.9	>1.9 - ≤ 3.5	>3.5 - ≤ 5.0	> 5.0
Cardiovascular (systolic blood pressure, mm Hg)	>90	<90, fluid responsive	<90, fluid unresponsive	<90, pH<7.3	<90, pH<7.2

BISAP: Bedside index for severity in Acute Pancreatitis

1. **BUN > 25mg/dl.**
2. **Impaired mental status (Glasgow Coma Score < 13)**
3. **SIRS**
4. **Age > 60 years**
5. **Pleural effusion detected on imaging.**

- **Incremental increases in the BISAP score (3 or more) have been shown to correlate with an increased risk of organ failure pancreatic necrosis and mortality**

One point is assigned for each variable within 24 hours of presentation and added for a composite score of 0-5.

SOFA SCORE

	SOFA Score				
Variables	**0**	**1**	**2**	**3**	**4**
Respiratory (PaO2/FiO2)	**> 400**	**≤ 400**	**≤ 300**	**≤ 200 (with respiratory support)**	**≤ 100 (with respiratory support)**
Coagulation (Platelets x $10^3/\mu L$)	**> 150**	**≤ 150**	**≤ 100**	**≤ 50**	**≤ 20**
Liver (Bilirubin: mg/dl)	**< 1.2**	**1.2 – 1.9**	**2.0 – 5.9**	**6.0 – 11.9**	**> 12.0**
Cardiovascular (Hypotension)	**No hypotension**	**Mean Arterial Pressure < 70 mm of Hg**	**Dopamine ≤ 5 (microgram/kg/min) or Dobutamine (any dose) Adrenergic agents administered for atleast one hour**	**Dopamine > 5 (microgram/kg/min), Epinephrine ≤ 0.1 (microgram/kg/min), or Norepinephrine ≤ 0.0 Adrenergic agents administered for atleast one hour**	**Dopamine > 15 (microgram/kg/min), Epinephrine > 0.1 (microgram/kg/min), or Norepinephrine > 0.0 Adrenergic agents administered for atleast one hour**
Central nervous system (Glasgow coma scale)	**15**	**13-14**	**10-12**	**6-9**	**<6**
Renal (Creatinine – mg/dl or Urine output ml/day	**<1.2**	**1.2-1.9**	**2.0-3.4**	**3.5-4.9 or < 500**	**> 5.0 or < 200**

Treatment

- **Pain control**
- **Bowel rest**

In the management of acute pancreatitis, the treatment is to stop feeding the patient, giving him or her nothing by mouth, giving intravenous fluids to prevent dehydration, and sufficient pain control. As the pancreas is stimulated to secrete enzymes by the presence of food in the stomach, having no food pass through the system allows the pancreas to rest.

- **Nutritional support**

Recently, there has been a shift in the management paradigm from TPN (total parenteral nutrition) to early, post-pyloric enteral feeding (in which a feeding tube is endoscopically or radiographically introduced to the third portion of the duodenum). The advantage of enteral feeding is that it is more physiological, prevents gut mucosal atrophy, and is free from the side effects of TPN (such as fungemia). The additional advantages of post-pyloric feeding are the inverse relationship of pancreatic exocrine secretions and distance of nutrient delivery from the pylorus, as well as reduced risk of aspiration.

Disadvantages of a naso-enteric feeding tube include increased risk of sinusitis (especially if the tube remains in place greater than two weeks) and a still-present risk of accidentally intubating the bronchus even in intubated patients.

- **Antibiotics:**

Prophylactic Antibiotic Therapy

- Avoid prophylactic antibiotic doses – use ONLY for DEFINED INFECTIONS

  (**INFECTIVE NECROSIS or EXTRAPANCREATIC inf)**
- Infection : Source : Gut flora

  Organisms : ***Escherichia coli*** , ***Klebsiella pneumonia*** , ***Enterococcus sp.***

INDICATIONS : 1. Infective necrosis
2. Sterile necrosis > 50%
3. Extrapancreatic infections

- **Preferred antibiotics** : 1. Carbapenem (Imipenem+cilastatin)
  2. Quinolones
  3. Metronidazole
  4. 3rd generation cephalosporines

- **ERCP**

Early ERCP (endoscopic retrograde cholangiopancreatography), performed within 24 to 72 hours of presentation, is known to reduce morbidity and mortality.

The indications for early ERCP are as follows :

- **Concomitant cholangitis**
- **Significant persistent biliary obstruction (bilirubin > 5 mg/ dl)**
- **ERCP in severe biliary pancreatitis without biliary sepsis or obstruction**

The disadvantages of ERCP are as follows :

ERCP precipitates pancreatitis, and can introduce infection to sterile pancreatitis

The inherent risks of ERCP i.e. bleeding

It is worth noting that ERCP itself can be a cause of pancreatitis.

Surgery:

Surgery is indicated for

(i) Infected pancreatic necrosis and

(ii) Diagnostic uncertainty and

(iii) Complications.

The most common cause of death in acute pancreatitis is secondary infection.

Infection is diagnosed based on 2 criteria

Gas bubbles on CT scan (present in 20 to 50% of infected necrosis)

Positive bacterial culture on FNA (fine needle aspiration, usually CT or US guided) of the pancreas.

Surgical options for infected necrosis include:

- **Minimally invasive management - necrosectomy through small incision in skin (left flank) or stomach**
- **Conventional management - necrosectomy with simple drainage**
- **Closed management - necrosectomy with closed continuous postoperative lavage**
- **Open management - necrosectomy with planned staged reoperations at definite intervals (up to 20+ reoperations in some cases)**

- **Other measures: Pancreatic enzyme inhibitors are not proven to work.**
- **The use of octreotide has not been shown to improve outcome.**

Group – C

3. Write short notes on (any three): 3x5 = 15

a) Epidural anaesthesia.
b) Venous ulcer lower leg.
c) Spina bifida.
d) MEN syndrome.
e) Principle of skin grafting.

Answer.

a) Introduction: Epidural anesthesia is regional anesthesia that blocks pain in a particular region of the body. The goal of an epidural is to provide analgesia, or pain relief, rather than anesthesia which leads to total lack of feeling. Epidurals block the nerve impulses from the lower spinal segments.

Difference from spinal anesthesia: Spinal anaesthesia is a technique whereby a local anaesthetic drug is injected into the cerebrospinal fluid. This technique has some similarity to epidural anaesthesia, and lay people often confuse the two techniques. Important differences include:

- **To achieve epidural analgesia or anaesthesia, a larger dose of drug is typically necessary than with spinal analgesia or anaesthesia.**
- **Onset of analgesia is slower with epidural analgesia or anaesthesia than with spinal analgesia or anaesthesia.**
- **An epidural injection may be performed anywhere along the vertebral column (cervical, thoracic, lumbar, or sacral), while spinal injections are typically performed below the second lumbar vertebral body to avoid piercing and consequently damaging the spinal cord.**
- **It is easier to achieve segmental analgesia or anaesthesia using the epidural route than using the spinal route.**
- **An indwelling catheter is more commonly placed in the setting of epidural analgesia or anaesthesia than with spinal analgesia or anaesthesia.**

Indications: Injecting medication into the epidural space is primarily performed for analgesia.

For analgesia alone, where Surgery is not contemplated. An epidural injection or infusion for pain relief (e.g. in child birth) is less likely to cause loss of muscle power, but has to be augmented to be sufficient for surgery.

- **As an adjunct to general anaesthesia. This may reduce the patient's requirement for opioid analgesics. This is suitable for a wide variety of surgery, for example gynaecological surgery (e.g. hysterectomy), orthopaedic surgery (e.g. hip replacement),**

general surgery (e.g.laparotomy) and vascular surgery (e.g. open aortic aneurysm repair).

- **As a sole technique for surgical anaesthesia. Some operations, most frequently Caesarean section, may be performed using an epidural anaesthetic as the sole technique. This can allow the patient to remain awake during the operation.**
- **For post-operative analgesia, after an operation where the epidural technique was used as either the sole anaesthetic, or was used in combination with general anaesthesia. Analgesics are given into the epidural space typically for a few days after surgery, provided a catheter has been inserted. Through the use of a patient-controlled epidural analgesia (PCEA) infusion pump, a person has the ability to give themselves an occasional dose of pain medication through an epidural catheter.**
- **Treatment of back pain. Injection of analgesics and steroids into the epidural space may improve some forms of back pain.**
- **Treatment of chronic pain or palliation of symptoms in terminal care, usually in the short- or medium-term.**

Epidural analgesia during childbirth:

Advantages	Disadvantages
• Better pain relief than other pain medication • Fewer babies needing naloxone to counter opiate use by the mother • Decreased maternal hyperventilation and increased oxygen supply to baby • Decreased circulating adrenocorticotropic hormone and decreased fetal distress [	• More use of instruments to assist with the birth • Increased risk of Caesarean section for fetal distress • Longer delivery (second stage of labour) • Increased need for oxytocin to stimulate uterine contractions • Increased risk of very low blood pressure • Increased risk of muscular weakness for a period of time after the birth • Increased risk of fluid retention • Increased risk of fever

Epidural analgesia has been demonstrated to have several benefits after surgery, including:

- **Effective analgesia without the need for systemic opioids.**
- **The incidence of postoperative respiratory problems and chest infections is reduced.**
- **The incidence of postoperative myocardial infarction ("heart attack") is reduced.**
- **The stress response to surgery is reduced.**
- **Motility of the intestines is improved by blockade of the sympathetic nervous system.**
- **Use of epidural analgesia during surgery reduces blood transfusion requirements.**

Complications:

- **Failure to achieve analgesia or anaesthesia occurs in about 5% of cases, while another 15% experience only partial analgesia or anaesthesia**
- **The following factors are associated with failure to achieve epidural analgesia/anaesthesia**
  - **Obesity**
  - **Multiparity**
  - **History of a previous failure of epidural anaesthesia**
  - **History of regular opiate use**
  - **Cervical dilation of more than 7 cm at insertion**
  - **The use of air to find the epidural space while inserting the epidural instead of alternatives such as saline or lidocaine**

- **Accidental dural puncture with headache. This may cause cerebrospinal fluid (CSF) to leak out into the epidural space, which may in turn cause a post dural puncture headache (PDPH). This can be severe and last several days, and in some rare cases weeks or months. If severe it may be successfully treated with an epidural blood patch (a small amount of the subject's own blood given into the epidural space via another epidural needle which clots and seals the leak). Most cases resolve spontaneously with time.**
- **Delayed onset of breastfeeding and shorter duration of breastfeeding.**
- **Bloody tap.**
- **Catheter misplaced into a vein.**
- **High block, as described above.**
- **Catheter misplaced into the subarachnoid space. If the catheter is accidentally misplaced into the subarachnoid space (e.g. after an unrecognised accidental dural puncture), normally cerebrospinal fluid can be freely aspirated from the catheter (which would usually prompt the anaesthetist to withdraw the catheter and resite it elsewhere). If, however, this is not recognised, large doses of anaesthetic may be delivered directly into the cerebrospinal fluid. This may result in a high block, or, more rarely, a *total spinal*, where anaesthetic is delivered directly to the brainstem, causing unconsciousness and sometimes seizures.**
- **Neurological injury lasting less than 1 year.**
- **Epidural abscess formation.**
- **Epidural haematoma formation.**
- **Neurological injury lasting longer than 1 year.**
- **Paraplegia.**
- **Arachnoiditis.**
- **Death (extremely rare, less than 1 in 100,000).**

a) Venous ulcer lower leg.

Venous ulcer:

Introduction:

- **Chronic venous disease, including chronic venous insufficiency and chronic venous ulceration, is a common and important medical problem that causes significant morbidity. Venous ulcers are expensive to treat and adversely impact patient's quality of life.**
- **Venous ulcers occur more commonly in the elderly, the peak prevalence occurring between ages 60 and 80 years.**

- **A venous leg ulcer can develop after a minor injury if there is a problem with the circulation of blood in your leg veins. If this happens, the pressure inside the veins increases.**

Risk factors:

- **Obesity – this increases the risk of high pressure in the leg veins**
- **Not being able to move for a long period of time – this can weaken the calf muscles, which can affect circulation in the leg veins**
- **Having previously had deep vein thrombosis (DVT)– blood clots that develop in the leg, which can damage valves**
- **Varicose veins – swollen and enlarged veins caused by malfunctioning valves**
- **Previous injury to the leg, such as a broken or fractured bone, which may cause DVT**
- **Previous surgery to the leg, such as a hip replacement or knee replacement, which can prevent you from moving about**
- **Increasing age – as people generally find it harder to move about as they get older**

{Pathophysiology: (go through):

- **Venous hypertension: Deep vein thrombosis, perforator insufficiency, superficial and deep vein insufficiencies, arteriovenous fistulas and calf muscle pump insufficiencies lead to increased pressure in the distal veins of the leg and finally venous hypertension.**
- **Fibrin cuff theory: Fibrin gets excessively deposited around capillary beds leading to elevated intravascular pressure. This causes enlargement of endothelial pores resulting in further increased fibrinogen deposition in the interstitium. The "fibrin cuff" which surrounds the capillaries in the dermis decreases oxygen permeability 20-fold. This permeability barrier inhibits diffusion of oxygen and other nutrients, leading to tissue hypoxia causing impaired wound healing.**
- **Inflammatory trap theory: Various growth factors and inflammatory cells, which get trapped in the fibrin cuff promote severe uncontrolled inflammation in surrounding tissue preventing proper regeneration of wounds. Leukocytes get trapped in capillaries, releasing proteolytic enzymes and reactive oxygen metabolites, which cause endothelial damage. These injured capillaries become increasingly permeable to various macromolecules, accentuating fibrin deposition. Occlusion by leukocytes also causes local ischemia thereby increasing tissue hypoxia and reperfusion damage.**
- **Dysregulation of various cytokines.**
- **Dysregulation of various pro-inflammatory cytokines and growth factors like tumor necrosis factor-α (TNF-α), TGF-β and matrix metalloproteinases lead to chronicity of the ulcers.**
- **Miscellaneous: Thrombophilic conditions like factor V Leiden mutation, prothrombin mutations, deficiency of antithrombin, presence of antiphospholipid antibodies, protein C and S deficiencies and hyperhomocysteinemia are also implicated.}**

Clinical features:
Venous leg ulcers are open, often painful, sores in the skin that take more than four to six weeks to heal. They most often develop on the inside of the leg, just above the ankle.
Symptoms:

- **Swollen ankles (oedema)**
- **Discolouration and darkening of the skin around the ulcer**

- **Hardened skin around the ulcer, which may make your leg feel hard and resemble the shape of an upside-down champagne bottle**
- **A heavy feeling in your legs**
- **Aching or swelling in your legs**
- **Red, flaky, scaly and itchy skin on your legs (varicose eczema)**
- **Swollen and enlarged veins on your legs (varicose veins)**
- **An unpleasant and foul-smelling discharge from the ulcer**

Signs of an infection:

A venous leg ulcer can be susceptible to bacterial infection. Symptoms of an infected leg ulcer can include:

- **Worsening pain**
- **A green or unpleasant discharge coming from the ulcer**
- **Redness and swelling of the skin around the ulcer**
- **A high temperature (fever)**

Nonsurgical Treatment

❖ **Infected ulcers**

- **Necessitate treatment of the infection first.**
- **Staphylococcus aureus, Streptococcus pyogenes, and Pseudomonas species are responsible for most infections.**
- **Usually treated with local wound care, wet-to-dry dressings, and oral antibiotics.**
- **Topical antiseptics should be avoided.**
- **Severe infections require intravenous antibiotics.**

❖ **Leg elevation**

Leg elevation can temporarily decrease edema and should be instituted when swelling occurs. This should be done before a patient is fitted for stockings or boots.

❖ **Compression therapy**

- **Compression therapy is the primary treatment for CVI.**

- **Elastic compression stockings**
  - **Fitted to provide a compression gradient from 30 to 40 mm Hg, with the greatest compression at the ankle.**
  - **Donned on arising from bed and removed at bedtime.**
  - **Effective in healing ulcers but can take months to obtain good results.**
  - **Stockings do not correct the abnormal venous hemodynamics and must be worn after the ulcer has healed to prevent recurrence.**
  - **Principal drawback is patient compliance.**
- **Unna boots**
  - **Paste gauze compression dressings that contain zinc oxide, calamine, and glycerin.**
  - **Used to help prevent further skin breakdown.**
  - **Provide nonelastic compression therapy.**

- Changed once or twice a week.
- Healing time for ulcers is less than that of elastic compression alone.

- **Pneumatic compression devices**
  - Provide dynamic sequential compression.
  - Used primarily in the prevention of deep vein thrombi in hospitalized patients.
  - Also used successfully to treat venous insufficiency.

❖ **Topical medications**

- **Largely ineffective as a stand-alone therapy for venous stasis ulcers.**
- **Topical therapy is directed at absorbing wound drainage and avoiding desiccation of the wound.**
- **Antiseptics can be counterproductive. Hydrogen peroxide, povidone-iodine, acetic acid, and sodium hypochlorite are toxic to cultured fibroblasts and should be used for the shortest duration necessary to control ulcer infection.**

❖ **Surgical Therapy: Skin grafting.**

c) Spina bifida.

Definition: It is a congenital abnormality with developmental defect in the spinal column with incomplete closure of vertebral canal due to failure in fusion of vertebral arches ± protrusion and dysplasia of the spinal cord or its membranes.

- ✓ **It arises in the first few weeks of gestation, with unknown cause**
- ✓ **It arises when one vertebrae does not close normally and leave the spinal cord exposed**
- ✓ **It is a midline defect of the: Skin, bone spinal column and spinal cord.**
- ✓ **3 in 1,000 births**

Types:

Occulta :

- **Mildest form of spina bifida**
- **The outer part of some of the vertebrae are not completely**
- **The split in the vertebrae is so small that the spinal cord does not protrude**
- **The condition is asymptomatic**
- **Bony abnormality seen by X – ray**
- **Nerves may be involved when associated with hairy patch or other skin changes**

Cystica:

1- Meningeocele:

- **Vertebral arches are unfused & with herniation of the meninges part of the cord or nerves root may present in the sac but they conduct impulses normally.**
- **Meningeocele is covered by the outer layer of skin and the inner layer of meninges which communicate with meninges lining the spinal subarachnoid space2- Myelomeningeocele:**
- **Sac contains neural elements that protrude through the spinal defects**
- **The overlying skin is thin & leaks of spinal fluid**
- **Secondary infection is common , Neurological and Orthopaedic problem are present.**
- **Hydrocephalus(arnold- chiari malformation)**
- **Clinical Picture: It will differ according to the level of lesion (most common site is lumbosacral region)**
- **Flaccid paralysis, muscle weakness, wasting, decreased or absent tendon reflexes**
- **Decreased or absent extroceptive & proprioceptive sensation**
- **Rectal & bladder incontinence, Hydrocephalus, Sever vasomotor changes**
- **Paralytic or congenital deformities as in club foot , pes cavus.**
- **Pressure ulceration due to poor sensation, Osteoporosis , soft tissue contracture**
- **Physical emotion& mental delay**
- **Prognosis:**
- **With successful closure of simple meningeocele prognosis is good**
- **Myelomeningeocele - die from infection, if survive after proper closure - stationary disability**

Proposed Aetiologies for Spina Bifida:

Multi-factorial inheritance.	**Vitamin deficiencies/folate.**	**Zinc deficiency.**	**Viral infection.**	**Mineral deficiency.**
Potato blight.	**Maternal fever**	**High sound intensity.**	**Alcohol.**	**Medication – Phenytoin, Epilim, etc.**

Screening and diagnosis:

Prenatal Tests	**Evaluation**	**Imaging Studies**	**Gait Analysis**
AFP	**Analysis of individual medial history**	**X-rays**	
Ultrasound	**Physical examination**	**Ultrasound**	

Testing of Amniotic fluid	**Evaluation of critical body systems**	**CT scan** **MRI**	

Treatment:

- **No cure**
- **Regular check-ups with physician**
- **Surgery (24 hours after birth)**
- **Medication**
- **Physiotherapy**
- **Bowel, bladder training.**

Surgery:

- **Usually performed within 24 hours after birth.**
- **They remove the infected area and replace it with muscle tissue and skin.**
- **Helps protect against hydrocephalus.**

d) MEN syndrome.

Introduction:

- **Multiple Endocrine Neoplasia (MEN) syndromes are familial conditions characterized by the occurrence of tumors involving two or more endocrine glands in a patient and family members.**
- **There are two major forms of MEN, namely MEN-type 1 (MEN1, →Wermer's syndrome) and MEN-type 2(MEN2, Sipple's syndrome); each form is characterized by the development of tumors of specific endocrine glands.**
- **The MEN syndromes are uncommon, but because they are inherited as autosomal dominant disorders, the finding of MEN in a patient has important implications for other family members. First degree relatives of a patient with known MEN have about a 50% risk of developing the disease.**
- **Occasionally, the MEN syndromes may arise sporadically (*i.e.* without a family history).**

MEN 1:

- **Autosomal dominant.**
- **The MEN1 gene was originally mapped to chromosome 11q13 by a combination of genetic linkage studies and tumor deletion mapping. It encodes a protein merlin.**
- **The MEN1 gene consists of 10 exons spanning 9 kb of genomic DNA and encodes a 610–amino acid protein product termed menin.**

Features of MEN 1

Major disease components	Associated tumors
• **Primary hyperparathyroidism** • **Pancreatic endocrine tumors (PET)** • **Anterior pituitary tumor**	• **Facial angiofibroma** • **Collagenoma** • **Adrenal cortical tumor** • **Lipoma** • **Foregut carcinoid**

Components of MEN-1 with estimated penetrance (in parentheses) at age 40 year:

Endocrine features	Non-endocrine features
PHPT- Parathyroid hyperplasia (90%)	Lipomas (30%)
Entero-pancreatic tumor 1. **Gastrinoma** (40%) 2. Insulinoma (10%) 3. Non-functioning (**NF**) including **pancreatic polypeptide** (20%) 4. Other rare types: (2%) **glucagonoma** **VIPoma** **somatostatinoma** *etc.*	Facial angiofibromas (85%) Collagenomas (70%) Ependymoma (1%)
Foregut carcinoid **Thymic carcinoid** NF (2%) **Bronchial carcinoid** NF (2%)	
Gastric entero-chromaffin-like tumor NF (10%)	
Anterior pituitary tumor Prolactinoma (20%) Other: GH + PRL, GH, NF (each 5%) ACTH (2%), TSH (rare)	
Adrenal cortex NF (25%)	
Pheochromocytoma (<1%)	

- **Parathyroid gland tumours. By age 40, 95% of patients have hypercalcaemia which is the commonest manifestation.**
- **Pancreatic islet cell tumours.**
  - **Prevalence of 30-75%.**
  - **Usually multicentric, slow-growing.**
  - **Secrete multiple polypeptides (insulin and gastrin commonest).**

    - **Gastrinoma leads to Zollinger-Ellison syndrome (recurrent and multiple peptic ulcers, severe reflux oesophagitis, and diarrhoea).**
    - **Rarer tumours are VIPoma, glucagonoma, somatostatinoma.**
- **Anterior pituitary tumours.**
    - **Detected in 15-40%.**
    - **Commonest is prolactinoma.**
    - **Rarer are GH- (causes acromegaly) or ACTH- (causes Cushing's disease) secreting tumours.**

Carcinoid tumours (thymus, lungs, foregut), adrenal tumours, lipomas, and pinealomas have also been reported to appear in MEN-1 patients.

MEN 2A	MEN2B
Medullary thyroid carcinoma	Medullary thyroid carcinoma
Pheochromocytoma	Pheochromocytoma
Hyperparathyroidism	Marfanoid body habitus
Lichen planus amyloidosis	Mucosal neuromas
Hirschsprung's disease	Ganglioneuromatosis of the gastrointestinal tract

MEN 2:
Clinical Features of Sporadic MTC, MEN 2A, MEN 2B, and FMTC:

CLINICAL SETTING	FEATURES OF MTC	INHERITANCE PATTERN	ASSOCIATED ABNORMALITIES	GENETIC DEFECT
Sporadic MTC	Unifocal	None	None	Somatic *RET* mutations in >20% of tumors
MEN 2A	Multifocal, bilateral	Autosomal dominant	Pheochromocytomas, hyperparathyroidism	Germline missense mutations in extracellular cysteine codons of *RET*
MEN 2B	Multifocal, bilateral	Autosomal dominant	Pheochromocytomas, mucosal neuromas, megacolon, skeletal abnormalities	Germline missense mutation in tyrosine kinase domain of *RET*
FMTC	Multifocal, bilateral	Autosomal dominant	None	Germline missense mutations in extracellular or intracellular cysteine codons of *RET*

Treatment:
Surgical treatment:
MEN-1

- **Parathyroidectomy.**
- **Pancreatic tumours: enucleation of individual tumours in the head of the pancreas and distal pancreatectomy for tumours in the tail/body.**
- **Hypophysectomy and external beam irradiation are considered for pituitary tumours.**

MEN-2

- **Total thyroidectomy (TT) indicated in patients identified by genetic screening. Symptomatic patients need TT and cervical nodal dissection for the lymph nodes on the involved side.**
- **Laparoscopic adrenalectomy for phaeochromocytoma.**
- **Parathyroidectomy for MTC in patients belonging to families in which hyperparathyroidism is frequently associated.**

Medical treatment
MEN-1 Prolactinomas can be treated with dopamine agonists (bromocriptine/cabergoline).

e) Principle of skin grafting.

Answer.

- **The skin is a large, indispensable, complex organ that develops both from the ectoderm (epidermis) and the mesoderm (the dermis).**
- **Most of the epidermal layers survive without blood supply except at their junction with the dermis. The basal layer of the epidermis also contains melanocytes.**
- **The dermis contains within it a substrate called collagen, interspersed with elastic tissue, sebaceous glands attached to hair follicles, sweat glands, blood vessels, nerves as well as lymphatics.**
- **Together with the epidermis the skin as a whole is a barrier against environmental assault and also is the principal site for communication with its surroundings.**
- **It serves a thermostatic function by judicious use of secretion of sweat controlled by the nervous system. The sebaceous glands play a smaller role in this regard and are not influenced by the nervous system. The two together also lubricate the skin and some of these secretions can be called as excretory in nature.**
- **The skin because of its size is a huge storehouse for blood which can be diverted to other crucial areas at the time of adversity, for example in shock. A large rapidly occurring burn means depletion of a fair volume of blood as also loss of fluids due to passive evaporation of water as well as the inflammatory exudate.**
- **The skin with its rich reticulo-endothelial system protects the body from invasion by bacteria and viruses. Because of its multilayered structure it is also resistant to mechanical, thermal and radiation injury up to a limit.**
- **The hair follicles and sebaceous glands as well as sweat glands, which all lie in the dermis, have openings on the surface of the epidermis. The epidermal layer invaginates throughout these tubular structures and lines them from within and therefore the regeneration of the epidermis can occur from these invaginated layers as long as some of these structures are preserved in the dermis after an injury or after a skin graft is harvested. Sridhar from Chennai adds "When split skin graft is taken the donor site heals**

by epithelialisation from cut ends of sebaceous glands/ducts/sweat gland ducts and hair follicles. That is why the healing is fast and donor site has mottled appearance."

- **Skin grafting means detaching (cutting) a piece of skin from one site and placing it on a bed which revascularises it.**
- **The procedure of skin grafting is actually a transplantation of a complex organ which serves many vital functions.**
- **A split skin graft is classified by its thickness. Purely epidermal grafts are rarely ever done at the present time. Depending upon how much dermis is taken the graft is called a thin, intermediate or a thick split skin graft. A thick graft leaves behind much less regenerative capacity in the donor area than a thin graft and consequently the donor site will take longer to heal and will be left with poorer quality skin.**
- **When a full thickness graft is cut, the regenerative power to heal in the donor area is nil and therefore the donor area needs to be closed primarily (small areas) or requires to be covered with another split skin graft.**
- **The thickness of the graft determines the value and quantity of the constituents that are transferred.**
- **The thicker the graft the greater is the biological requirement for the graft to "take".**
- **When a graft is placed on a recipient bed, the process of "take" begins. For some hours it is bathed and nourished by plasmatic circulation or serum imbibition. Simultaneously fortuitous and accidental apposition of the vessels in the bed and those in the graft allows blood to be sucked into the graft (inosculation). Soon afterwards active penetration of the graft by blood vessels from the bed begins and is well established by the fifth day. This is when the graft regains its colour which was lost when it was harvested because of severe vasoconstriction as a result of trauma.**
- **A graft that has 'taken' (to its bed) remodels itself over a period of several weeks or months. This process is called consolidation in which collagen gets fragmented and is re-laid, elastic fibers degenerate and form anew. No graft remains identical to what it was when it was harvested and there is always some loss of its collagen content and its elasticity. This loss is highest in thin grafts and the least in full thickness grafts. When a graft is harvested it tends to coil on itself because of its elasticity. The thicker the graft the greater the recoil. A thin graft can be spread easily and remains in that state. Those grafts which coil up on harvesting contract the least after they 'take'. The reverse is true of thinner grafts.**
- **In nature all wounds contract by the laying down of fibrous tissue through fibroblasts unless interfered with by local or systemic disease. A large granulating wound of some duration therefore has already started the process of contraction while a freshly created surgical wound which gets primarily closed shows little or no contraction. Therefore, a surgically created wound which is covered by a full thickness graft will show the minimum contraction while at the other end of the scale a large chronic, granulating wound covered by a split thickness graft, particularly if it is thin, will show maximum contraction.**
- **When contraction is complete, the pathological state left behind is called a contracture (e.g. a burn contracture across the elbow or the ankle).**
- **A graft fails to "take" when it cannot be revascularised or when the process of revascularization is harmed by a shearing force due to improper immobilization of the graft and the bed.**
- **Failure of revascularization can occur when the bed on which the graft is placed has poor vascularity or the revascularization is prevented by a blood clot between the graft and the bed.**

- **Though all granulating wounds can be considered as harbouring infection, a well prepared granulating wound will 'take' grafts. Suppuration is another matter. Pus and multiplying organisms have proteolytic enzymes, prevent capillary formation and an overt presence of pus is the poorest environment for 'take' of a graft.**
- **Debilitating conditions, such as uncontrolled diabetes, hypoproteinemia or local irradiation usually have adverse implications for the recipient beds.**
- **Completely normal reinnervation of grafts is extremely rare. Reinnervation depends on the bed on which the graft is placed rather than the graft. The greater the number of nerve endings in the bed, the better is the reinnervation. Reinnervation occurs by penetration of nerves into the graft but also by insinuation of hollow myelin tubes which are remnants of the remodeling process in the graft.**

Group – D

4. Write briefly on (any three): 5x3 = 15

a) Bleeding from gum.
b) Hydrocephalus.
c) Post burn contracture.
d) Ionizing radiation.
e) Hamartoma.

Answer.
a) Bleeding from gum.

There are many possible causes of gingival bleeding. The main cause of gingival bleeding is the formation and accumulation of plaque at the gum line due to improper brushing and flossing of teeth. The hardened form of plaque is called tartar. An advanced form of gingivitis as a result of formation of plaque is periodontitis.

Other causes that can exacerbate gingival bleeding include:

- **Placement of new dentures**
- **Tooth or gum infection**
- **Diabetes mellitus**
- **Idiopathic thrombocytopenic purpura**
- **Leukemia**
- **Malnutrition**
- **Use of aspirin and anticoagulants(blood thinners) such as warfarin and heparin[5]**
- **Hormonal imbalances during puberty and pregnancy**
- **Iron overload**

Other less common causes are:

- **Vitamin C deficiency (scurvy) and vitamin K deficiency**
- **Dengue fever**

Diagnosis:

- **An examination by the dentist or dental hygienist should be sufficient to rule out the issues such as malnutrition and puberty. Additional corresponding diagnosis tests to certain potential disease may be required. This includes oral glucose tolerance test for diabetes mellitus, blood studies, human gonadotrophin levels for pregnancy, and X-rays for teeth and jaw bones.**
- **In order to determine the periodontal health of a patient, the dentist or dental hygienist records the sulcular depths of the gingiva and observes any bleeding on probing. This is often accomplished with the use of a periodontal probe. Alternatively, dental floss may also be used to assess the Gingival bleeding index. It is used as an initial evaluation on patient's periodontal health especially to measure gingivitis. The number of bleeding sites is used to calculate the gingival bleeding score.**
- **Bleeding on probing is a poor positive predictor of periodontal disease, but conversely lack of bleeding is a very strong negative predictor. The clinical interpretation of this research is that while BOP presence may not indicate periodontal disease, continued absence of BOP is a strong predictor (approximately 98%) of continued periodontal health.**

Treatment

- **Corresponding treatments for diagnosed diseases should be taken as first priority.**
- **Dentist or hygienists should be visited once every three months for plaque removal.**
- **Soft-bristle toothbrush is recommended for brushing your teeth. Hard-bristled toothbrushes may be softened by leaving under hot running water (very hot) before brushing every time, followed by gentle brushing.**
- **Flossing twice a day can prevent the building up of plaques.**
- **Tobacco should be avoided as tobacco can aggravate the bleeding gums.**
- **A balanced healthy diet should also be taken into account.**
- **Physiotherapy programme using over-the-counter toothpaste with triclosan should be used with home care.**
- **If there is persistent continuation of inflammation and bleeding, a prescription of antiplaque rinse would be useful.**

b) Introduction: Hydrocephalus, also known as "water on the brain", is a medical condition in which there is an abnormal accumulation of cerebrospinal fluid (CSF) in the ventricles, or cavities, of the brain. This may cause increased intracranial pressure inside the skull and progressive enlargement of the head, convulsion, and mental disability. Hydrocephalus can also cause death.

Signs and Symptoms:

Symptoms of increased intracranial pressure:

- **Headaches, vomiting, nausea, papilledema, sleepiness, or coma.**
- **Elevated intracranial pressure may result in uncal and/or cerebellar tonsill herniation, with resulting life threatening brain stem compression.**
- **The triad (Hakim triad) of gait instability, urinary incontinence and dementia is a relatively typical manifestation of the distinct entity normal pressure**

hydrocephalus (NPH). Focal neurological deficits may also occur, such as abducens nerve palsy and vertical gaze palsy (Parinaud syndrome due to compression of the quadrigeminal plate, where the neural centers coordinating the conjugated vertical eye movement are located).

Normal pressure hydrocephalus: The symptoms depend on the cause of the blockage, the person's age, and how much brain tissue has been damaged by the swelling.

In infants with hydrocephalus, CSF fluid builds up in the central nervous system, causing the fontanelle (soft spot) to bulge and the head to be larger than expected.

Early symptoms may also include:	**Symptoms that may occur in older children can include:**	
• **Eyes that appear to gaze downward** • **Irritability** • **Seizures** • **Separated sutures** • **Sleepiness** • **Vomiting**	• **Brief, shrill, high-pitched cry** • **Changes in personality, memory, or the ability to reason or think** • **Changes in facial appearance and eye spacing** • **Crossed eyes or uncontrolled eye movements** • **Difficulty feeding** • **Excessive sleepiness** • **Headache** • **Irritability, poor temper control**	• **Loss of bladder control (urinary incontinence)** • **Loss of coordination and trouble walking** • **Muscle spasticity (spasm)** • **Slow growth (child 0-5 years)** • **Slow or restricted movement** • **Vomiting.**

Pathology:

Hydrocephalus is usually due to blockage of cerebrospinal fluid (CSF) outflow in the ventricles or in the subarachnoid space over the brain. In a person without hydrocephalus, CSF continuously circulates through the brain, its ventricles and the spinal cord and is continuously drained away into the circulatory system. Alternatively, the condition may result from an overproduction of the CSF fluid, from a congenital malformation blocking normal drainage of the fluid, or from complications of head injuries or infections.

Clsssification: Hydrocephalus can be caused by impaired cerebrospinal fluid (CSF) flow, reabsorption, or excessive CSF production.

The most common cause of hydrocephalus is CSF flow obstruction, hindering the free passage of cerebrospinal fluid through the ventricular system and subarachnoid space (e.g.,stenosis of the cerebral aqueduct or obstruction of the interventricular foramina -

foramina of Monro secondary to tumors, hemorrhages, infections or congenital malformations).

Hydrocephalus can also be caused by overproduction of cerebrospinal fluid (relative obstruction) (e.g., papilloma of choroid plexus).

Based on its underlying mechanisms, hydrocephalus can be classified into communicating and non-communicating (obstructive). Both forms can be either congenital or acquired.

Communicating:

Communicating hydrocephalus, also known as non-obstructive hydrocephalus, is caused by impaired cerebrospinal fluid resorption in the absence of any CSF-flow obstruction between the ventricles and subarachnoid space.

Various neurologic conditions may result in communicating hydrocephalus, including subarachnoid/intraventricular hemorrhage, meningitis, Chiari malformation, and congenital absence of arachnoidal granulations (Pacchioni's granulations).

Scarring and fibrosis of thesubarachnoid space following infectious, inflammatory, or hemorrhagic events can also prevent resorption of CSF, causing diffuse ventricular dilatation.

- **Normal pressure hydrocephalus (NPH) is a particular form of communicating hydrocephalus, characterized by enlarged cerebral ventricles, with only intermittently elevated cerebrospinal fluid pressure.**
- **Hydrocephalus ex vacuo also refers to an enlargement of cerebral ventricles and subarachnoid spaces, and is usually due to brain atrophy (as it occurs in dementias), post-traumatic brain injuries and even in some psychiatric disorders, such as schizophrenia. As opposed to hydrocephalus, this is a compensatory enlargement of the CSF-spaces in response to brain parenchyma loss - it is not the result of increased CSF pressure.**

Non-communicating: Non-communicating hydrocephalus, or obstructive hydrocephalus, is caused by a CSF-flow obstruction ultimately preventing CSF from flowing into the subarachnoid space (either due to external compression or intraventricular mass lesions).

- **Foramen of Monro obstruction may lead to dilation of one or, if large enough (e.g., in Colloid cyst), both lateral ventricles.**
- **The aqueduct of Sylvius, normally narrow to begin with, may be obstructed by a number of genetically or acquired lesions (e.g., atresia, ependymitis, hemorrhage, tumor) and lead to dilation of both lateral ventricles as well as the third ventricle.**
- **Fourth ventricle obstruction will lead to dilatation of the aqueduct as well as the lateral and third ventricles.**
- **The foramina of Luschka and foramen of Magendie may be obstructed due to congenital failure of opening (e.g., Dandy-Walker malformation).**

Congenital

The cranial bones fuse by the end of the third year of life. For head enlargement to occur, hydrocephalus must occur before then. The causes are usually genetic but can also be acquired and usually occur within the first few months of life, which include

- **Intraventricular matrix hemorrhages in premature infants,**
- **Infections,**
- **Type ii arnold-chiari malformation,**
- **Aqueduct atresia and stenosis, and**
- **Dandy-Walker malformation.**

In newborns and toddlers with hydrocephalus, the head circumference is enlarged rapidly and soon surpasses the 97th percentile. Since the skull bones have not yet firmly joined together, bulging, firm anterior and posterior fontanelles may be present even when the patient is in an upright position.

The infant exhibits fretfulness, poor feeding, and frequent vomiting.

As the hydrocephalus progresses, torpor sets in, and the infant shows lack of interest in his surroundings. Later on, the upper eyelids become retracted and the eyes are turned downwards (due to hydrocephalic pressure on the mesencephalic tegmentum and paralysis of upward gaze). Movements become weak and the arms may become tremulous.

Papilledema is absent but there may be reduction of vision. The head becomes so enlarged that the child may eventually be bedridden.

About 80-90% of fetuses or newborn infants with spina bifida—often associated with meningocele or myelomeningocele—develop hydrocephalus.

Acquired: This condition is acquired as a consequence of CNS infections, meningitis, brain tumors, head trauma, intracranial hemorrhage (subarachnoid or intraparenchymal) and is usually extremely painful.

Treatment:

Hydrocephalus treatment is surgical. It involves the placement of a ventricular catheter (a tube made of silastic), into the cerebral ventricles to bypass the flow obstruction/malfunctioning arachnoidal granulations and drain the excess fluid into other body cavities, from where it can be resorbed.

Most shunts drain the fluid into the peritoneal cavity (ventriculo-peritoneal shunt), but alternative sites include the right atrium (ventriculo-atrial shunt), pleural cavity (ventriculo-pleural shunt), and gallbladder

An alternative treatment for obstructive hydrocephalus in selected patients is theendoscopic third ventriculostomy (ETV), whereby a surgically created opening in the floor of the third ventricle allows the CSF to flow directly to the basal cisterns, thereby shortcutting any obstruction, as in aqueductal stenosis.

c) Post burn contracture:

Post-burn scars: Post-burn scars are inevitable even with the best of treatment because they depend upon the depth of burn injury. Except for the superficial dermal burns, all deeper burns (2nd degree deep dermal and full thickness) heal by scarring.This scarring can only be minimised by various physical therapy measures and plastic surgical procedures but not eliminated completely.

Post-burn scar contractures: A burn patient who receives the best of treatment is expected to heal without any contractures. The incidence of post-burn contractures is extremely high in our country. Quite often, they are not only multiple in a given patient but also very severe and diffuse.

- **The deeper tissues may be affected either due to their involvement in the initial burn injury (e.g., electrical burns) or secondary to the presence of a skin contracture over a prolonged period of many years, which leads to shortening of musculo-tendinous units and neurovascular structures.**
- **The joints may be subluxated or dislocated, with joint capsule and ligaments becoming tight in the direction of the contracture.**
- **The bones may be deformed, especially in growing children, e.g., mandibular deformity in cases of post-burn contractures of the neck.**
- **Presence of one or more of above along with a contracture may alter the physical therapy and/or surgical treatment of a contracture. For example, an unstable scar or chronic non-healing ulcer(s) will not heal without surgical release of the contracture.**
- **Physical therapy prior to surgery may not be possible in these cases.**
- **Massive raw areas need wound closure with skin grafting before contracture can be subjected to physical therapy.**
- **Wide excision of Marjolin's ulcer has to be combined with release of contracting bands.**
- **A post-burn contracture associated with a hypertrophic or an atrophic scar or a depigmented area may all need excision–release to achieve best results not only functionally but also aesthetically.**

Prevention and treatment:

- **The most important and effective method of controlling the wound contraction is to close the wound at the earliest using split-skin grafts in deep dermal and full thickness burns.**
- **Contraction can be inhibited by applying grafts to fresh wounds (as in early excision) or over healthy granulating areas (after eschar separation). Although full thickness skin grafts inhibit contraction almost completely, it is not possible in a clinical setting.**
- **The split-skin grafts may also need expansion with meshing in extensive burns. Although this leads to complete healing of the wound, the latter is largely covered with epithelium in the interstices of the meshed graft.**
- **It is widely believed that thicker the graft, greater will be the inhibition of contraction. This holds good only if the grafts are harvested from a given site.**

Timing of surgery in post-burn contractures:

As a general "rule", surgical intervention for post-burn contractures should not be undertaken during the active phase of healing and scarring, i.e., as long as the scar is immature and highly vascular. This usually takes 1 year or so.

With the passage of time, some mild contractures may improve with a better final result than if they had been surgically managed.

There are several exceptions to this general rule of scar maturation before doing surgical intervention.

- **Ectropion of the eyelids, especially the upper eyelid with constant danger of keratoconjunctivitis, corneal ulceration, scarring or perforation with loss of vision**
- **Incapacitating contracture of the neck with inability to look forwards**
- **Severe microstomia causing interference with adequate nutrition and maintenance of orodental hygiene.**

Surgical intervention:

Release of contracture

Complete release of contracture should be done, avoiding damage to any important underlying structure, e.g., arteries, nerves, tendons, etc.

In general, a contracture should be released by incision rather than by excision.

Excision may, however, be required in certain circumstances, e.g., (a) small adjoining depigmented or hypertrophic areas, excision of which will add to the final aesthetic result (b) atrophic/unstable scars/chronic non-healing ulcer(s)/ discharging sinuses should be excised along with release of contracture to obtain healthy bed for split-skin graft "take" (c) scars may also be excised so as to apply the graft/flap in accordance with principles of aesthetic units. Partial excision of hypertrophic scars may sometimes be done, e.g., in a case of post-burn contracture of neck, the scars may extend from chin, neck onto the chest and even abdomen.

In general, the contracture should be released completely on the table in one go. However, in severe long-standing contractures, there is considerable shortening of musculotendinous units and neurovascular structures. Hence, it may not be possible to achieve complete release.

- **Crippling contractures of hand, especially dorsal contracture with metacarpophalangeal joints going in hyperextension leading to permanent damage to extensor mechanism with various deformities .**
- **Contractures of both the knees, which force the patient to be on "all the fours" and endangers the very dignity of being an upright human being**
- **Post-burn contractures with associated adjoining chronic raw areas needing skin cover**
- **Contractures with infected hypertrophic scars and abscesses, which need excision/drainage for their recovery**
- **Any severe, incapacitating contracture unlikely to improve at all with physical therapy measures**

After the complete release of a post-burn contracture, the recreated defect has to be covered using skin grafts or a skin flap. Most commonly, the raw areas resulting after release of post-burn contractures are covered with skin grafts. Flap covers are used in special situations.

d) Ionizing radiation.

Introduction: Ionizing radiation is any type of particle or electromagnetic wave that carries enough energy to ionize or remove electrons from an atom. There are two types of electromagnetic waves that can ionize atoms: X-rays and gamma-rays, and sometimes they have the same energy.

- **Forms of Ionizing Radiation:**
  - **ALPHA**
    - **Travel only a short distance in air (~ 4" in air)**
    - **Stopped by dead skin, film of water, sheet of paper**
    - **Very hazardous when taken into the body**
    - **Avoid inhalation or ingestion**
  - **BETA**
    - **Penetrate human body to depth of 0.1-o.5"**
    - **Can penetrate wood to about 1.5"**
    - **Stopped by 0.5" aluminum or Plexiglas**
  - **GAMMA**
    - **Deep penetrating**
    - **Need steel, lead, etc. to shield**
  - **X –Radiation**
    - **Commonly thought of as electromagnetic radiation produced by an x-ray machine**
    - **Penetration depends on wavelength and material being irradiated.**
    - **Often use concrete to shield**

Occupations are at risk:

- **Healthcare/Medicine**
  - **Oncology**
  - **Radiation therapy**
  - **Dentistry**
- **Researchers**
- **Miners**
  - **Uranium, phosphate, etc.**
- **Nuclear power plant employees**

Symptoms of exposure:

- **High level doses of radiation (generally doses of more than 100 rads), if received all at once, cause short-term effects that appear within hours, days, or weeks. Known as acute radiation syndrome**
  - **Initial symptoms: nausea, vomiting, and malaise.**
  - **After latent period: infections, fever, hemorrhage, loss of hair, diarrhea, loss of body fluid, CNS effects**
  - **>600 rads leads to death**
- **Low level doses – risk is proportional to dose, but disagreement or uncertainty about exact responses.**

Adverse effects:

- **Cancer**
- **Birth defects**
- **Cataracts**
- **Shortening of lifespan**
- **If reproductive organs irradiated:**
  - **Genetic mutations may occur in sperm or egg cells**

There are 3 main uses of ionising radiation in medicine:

- **Treatment**
- **Diagnosis**

- **Sterilisation**

Radiation therapy:

Radiation therapy uses ionising radiation to treat cancer i.e. to destroy cancerous cells.

There are two techniques in radiation therapy that are used to treat cancer using ionising radiation:

- **External beam Radiotherapy**
- **Intra-operative Radiotherapy: used in carcinoma breast**
- **Radio isotope therapy: used in carcinoma thyroid**
- **Deep inspiration breath hold:**
- **Brachytherapy : Brachytherapy is used to treat the following cancers:**
  - **Uterus**
  - **Cervix**
  - **Prostate**
  - **Intraocular**
  - **Skin**
  - **Thyroid**
  - **Bone**

e) Hamartoma.

Introduction: A hamartoma (from Greek *hamartia* "fault, defect" and *-oma,* denoting a tumor or neoplasm) is a benign (noncancerous) tumorlike malformation made up of an abnormal mixture of cells and tissues found in areas of the body where growth occurs. It is considered

a developmental error and can occur at a number of sites. A nonneoplastic mass can also arise in this way; therefore, misdiagnosis is possible, as is subsequent overtreatment with its added morbidity and mortality. Developmental remnants may be considered hamartomatous if they form discrete tumorlike masses.

The literature describes several examples of hamartomas, including the following:

- **Hemangioma and other vascular tumors that are not true neoplasms**
- **Peutz-Jeghers polyp of the bowel, juvenile or retention**
- **Polyp of the large bowel**
- **Bronchial hamartoma**
- **Melanocytic nevi**
- **Neurofibromatosis in von Recklinghausen disease**
- **Neuroepithelial cells in tuberous sclerosis**
- **Hamartomas of the hypothalamus and tuber cinereum**
- **A variety of bony hamartomas**

A hamartoma resembles a neoplasm, but in most cases, it does not show any tendency to evolve into one. However, cases of neoplastic evolution have occurred with these lesions. Examples are chondrosarcomas arising in osteochondromas and neurofibrosarcomas arising in patients with von Recklinghausen disease.

In addition, neoplasms can be associated with hamartomas without directly arising from them. Examples are fibromas of the ovary and malignant ovarian tumors arising in patients with Peutz-Jeghers syndrome. Hamartomas should be distinguished from choristomas; the former are composed of tissues that are normally present at a given site, whereas the latter are malformations of tissues that are not normally found at that site.

Hamartomas may not cause any problems and are usually identified incidentally. Uncomplicated hamartomas have no tendency to grow, except as determined by the normal growth controls of the body. However, this does not mean that hamartomas are harmless. Morbidity can arise by means of a variety of mechanisms, including the following:

- **Obstruction**
- **Pressure, direct or indirect**
- **Infection**
- **Infarction**
- **Hemorrhage and iron deficiency anemia**
- **Fracture**
- **Misdiagnosis of neoplasm**
- **Neoplastic transformation**

Classification

For ease of description, the hamartomatous bone tumors are classified into the following four groups:

- **Bone-forming tumors**
- **Cartilage-forming tumors**

- **Fiber-forming tumors**
- **Benign non–matrix-forming tumors**

Lesion Class	Tumors
Bone-forming	• **Bone island** • **Osteopoikilosis** • **Melorheostosis** • **Osteopathia striata**
Cartilage-forming	• **Osteochondroma** • **Multiple osteochondroma** • **Epiphyseal osteochondroma** • **Enchondromatosis**
Fiber-forming	• **Nonossifying fibroma** • **Fibrous dysplasia**
Benign non–matrix-forming	• **Hemangioma of bone** • **Skeletal hemangiomatosis**

Laboratory Studies

Laboratory studies are not required unless patients are symptomatic. If needed, laboratory tests may include the following preoperative investigations:

- **Complete blood count (CBC)**
- **Measurement of urea, electrolyte, calcium, phosphate, and alkaline phosphatase levels**
- **Liver function tests if metastases are suspected**

Treatment

Because most hamartomas are found incidentally, their treatment depends on the patient's signs and symptoms at the time of presentation. Pain is a common finding and can be treated conservatively with analgesics. If symptoms are not controlled, further investigation is usually required and surgical management is considered.

Most patients may not require any intervention. However, for those who do require intervention, procedures may include biopsy, curettage, and fracture fixation. If bony involvement is extensive, amputation of digits or limbs may result.

Indications for surgical intervention

Regardless of the lesion or mass present, clinicians must adhere to certain basic principles. Whenever possible, benign tumors should be treated conservatively. Surgical intervention may be indicated in the following situations:

- **If the diagnosis is in any doubt and if biopsy is required to distinguish between benign and malignant lesions**
- **If pathologic fractures occur due to bone weakness**
- **If the patient becomes symptomatic, with bony masses compressing the surrounding structures or interfering with muscle and joint movement so that function is impaired or that prominent bony protuberances cause cosmetic deformities**

For patients undergoing surgery, the preoperative workup and management are similar for most hamartomatous lesions (see Laboratory Studies, Plain Radiography, and Other Imaging Studies). Specific intraoperative details are beyond the scope of this article; however, indications for surgical treatment of various hamartomas are briefly summarized below.

Specific postoperative details are also beyond the scope of this article. Follow-up care can range from no further intervention and simple reassurance to 6-month or annual visits, depending on the type of hamartoma and on the risk of malignant change.

The West Bengal University of Health Sciences

M.B.B.S. 3rd Professional Part – II Examination, 2015

Subject: Surgery **Time: $2^{1}/_{2}$ hrs.**

Paper: II **Marks: 60**

Group –A

1. **Classify goiter. How will you investigate and treat a 50 years old man with clinically discrete nodule of 3 cm diameter in right lobe of thyroid.** **5+5+5 = 15**

Answer.

Classification of thyroid swellings

Simple goitre (euthyroid)	Diffuse hyperplastic	Physiological Pubertal Pregnancy
	Multinodular goitre	
Toxic	Diffuse (Graves' disease)	
	Multinodular	
	Toxic adenoma	
Neoplastic	Benign	
	Malignant	
Inflammatory	Autoimmune	Chronic lymphocytic thyroiditis
		Hashimoto's disease
	Granulomatous	De Quervain's thyroiditis
	Fibrosing	Riedel's thyroiditis
	Infective	Acute (bacterial thyroiditis, viral thyroiditis, 'subacute thyroiditis')
		Chronic (tuberculous, syphilitic)
	Other	Amyloid

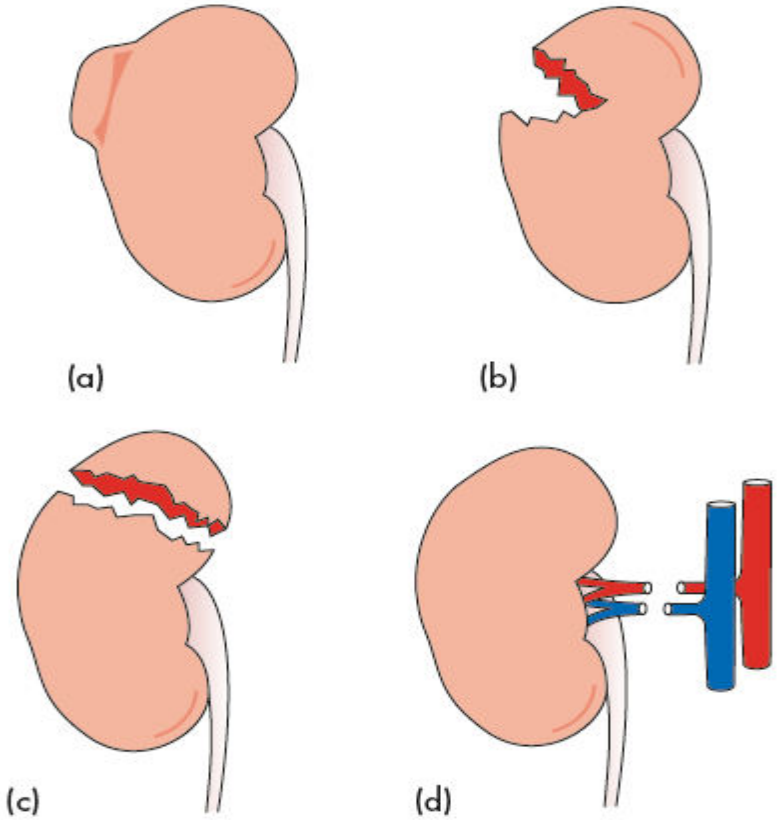
For the next part of the question **See the Question 1.a of Group – A of Paper –II of 2011**

Group – B

2. a) Classify renal injury. Discuss clinical features and management of a patient having injury to left kidney following blunt trauma in left loin. 4+5+6 = 15

Answer.

Types of closed renal trauma: (a) subscapular haematoma; (b) laceration; (c) avulsion of one pole; (d) avulsion of the renal pedicle.



<u>Classified according to the Organ Injury Scaling (OIS) Committee Scale:</u>

Minor	**I**	Contusion	Microscopic or gross haematuria, Urological studies normal
		Haematoma	Subcapsular, nonexapnding without parenchymal laceration.
	II	Haematoma	Nonexapnding perirenal haematoma confined to renal retroperitoneum.
		Laceration	<1cm parenchymal depth of renal cortex without urinary extravasation.

Major	**III**	Laceration	>1cm depth of renal cortex, without collecting system rupture or urinary extravasation
	IV	Laceration	Parenchymal laceration extending through the renal cortex, medulla and collecting system.
		Vascular	Main renal artery or vein injury with contained haemorrhage.
	V	Laceration	Completely shattered kidney.
		Vascular	Avulsion of renal hilum which devascularizes kidney.

Clinical features of closed renal trauma:

- There is local pain and tenderness, sometimes with superficial soft-tissue bruising.
- Haematuria: Haematuria may not appear until days after the injury. Profuse bleeding may cause clot colic.
- Severe delayed haematuria: Sudden haematuria between the third day and third week after trauma in a recovering patient is caused by a clot becoming dislodged.
- Meteorism: Abdominal distension 24–48 hours after renal injury is probably a result of retroperitoneal haematoma implicating splanchnic nerves.

Management and treatment:

- Watchful treatment of closed renal trauma is often successful. Consider the possibility of injury to other organs at an early stage.
- Cross-match blood and secure intravenous access if there, is any evidence of hypovolaemic shock or continuing haemorrhage.
- Advise bed-rest while there is macroscopic haematuria and restrict activity for a week after the urine clears.
- Administer appropriate analgesia.
- Keep hourly observations.
- Antibiotics should be given to prevent infection of the haematoma.
- Check the urine passed for haematuria and chart the result.
- Urgent intravenous urography (IVU) or contrast-enhanced.
- CT will clarify the extent of renal damage and show that the other kidney is normal.
- Blood should be sent for grouping and serum saved for crossmatching in all cases.

Surgical treatment in closed renal trauma:

- Exploration of the kidney may be associated with massive blood loss as the haematoma is opened
- Check that the contralateral kidney is functioning because nephrectomy is a possibility

Complications:

- Heavy haematuria may lead to clot retention requiring bladder washout.
- Pararenal pseudohydronephrosis may occur weeks later from a combination of complete cortical tear and ureteric obstruction caused by scarring.
- Hypertension, resistant to drugs, resulting from renal fibrosis, may occur long after injury. Nephrectomy may be necessary.
- Post-traumatic aneurysm of the renal is rare. There is loin pain and a non-tender swelling may be felt if the aneurysm is large.
- Congestion of the parenchyma leads to intermittent haematuria.

- Aortography is diagnostic. Excision or nephrectomy is indicated to prevent fatal rupture of the aneurysm.

Or

b) Discuss the clinical features, complications and management of undescended testis. 5+5+5 = 15

Answer.

Key facts:

- Testicular descent from the fetal abdominal site into the scrotum is normally complete by birth.
- Absence of a scrotal testis (cryptorchidism) may be due to agenesis (rare), intraabdominal arrest, incomplete descent (intracanalicular), or ectopic descent (inguinal, perineal, crural, penile).
- Incidence: 2-4% of newborn boys falling to 1.5% at 6 months.
- Commoner on the right side.

Clinical features:

- Undescended testis can be noted at the postnatal check, by parents, or by the GP.
- Rarely presents acutely as torsion (tender mass in inguinal region).
- A retractile testis is one that can be brought down into the scrotum with gentle manipulation but retracts into the superficial inguinal pouch either spontaneously or with minor pressure.

Diagnosis and investigations:

- No investigations are required in palpable undescended testis.
- Chromosomal studies and HCG stimulation test may be requested in bilateral impalpable testes.
- Ultrasound may help locate an impalpable testis.
- Diagnostic laparoscopy is definitive and allows further management.

Treatment:

- Testis should be brought to the scrotum at 1-2 years of age to avoid secondary damage due to trauma, torsion, and increased ambient temperature.
- Hormone manipulation is ineffective in true undescended testis.
- Intracanalicular or ectopic testis should be managed by one-stage orchidopexy.
- Intraabdominal testis can be brought down by one- or two-stage orchidopexy (50-90% success).
- Laparoscopy for bilateral impalpable testes.
- Scrotal position facilitates self-examination to detect signs of neoplastic change (~4 x normal in an abdominal testis).

Complications:

- Postoperative atrophy of the testis (< 2%) unless intraabdominal position (10-50%).
- Retraction.

Indications for orchidopexy:

- Maximize sperm production
- Prevent testicular torsion
- Repair of associated inguinal hernia
- Cosmesis
- Reduce chance of malignancy development and improve self-examination success.

Complications of undescended testis:

- **Testicular cancer.** Testicular cancer usually begins in the cells in the testicle that produce immature sperm. What causes these cells to develop into cancer is unknown. Men who've had an undescended testicle have an increased risk of testicular cancer. The risk is greater for undescended testicles located in the abdomen than in the groin. Surgically correcting an undescended testicle might decrease, but not eliminate, the risk of future testicular cancer.
- **Fertility problems.** Low sperm counts, poor sperm quality and decreased fertility are more likely to occur among men who've had an undescended testicle. A decrease in cells in the testicle that produce sperm has been found as early as 1 year old.
- **Testicular torsion.** Testicular torsion is the twisting of the spermatic cord, which contains blood vessels, nerves and the tube that carries semen from the testicle to the penis. This painful condition cuts off blood to the testicle. If not treated promptly, it might result in the loss of the testicle. Testicular torsion occurs 10 times more often in undescended testicles than in normal testicles.
- **Trauma.** If a testicle is located in the groin, it might be damaged from pressure against the pubic bone.
- **Inguinal hernia.** If the opening between the abdomen and the inguinal canal is too loose, a portion of the intestines can push into the groin.

Group – C

3. Write short notes on (any three) **3x5 = 15**

a) **Breast abscess.**
b) **Meconium ileus.**
c) **Basal cell carcinoma.**
d) **Premalignant conditions of penile carcinoma.**
e) **Acute pancreatitis.**

Answer.

a) **Breast abscess. See the Question 2.c of Group – B of Paper – II of 2009**
b) **Meconium ileus.**

Meconium ileus refers to a newborn bowel obstruction of the distal ileum due to abnormally thick impacted meconium. Unlike in the meconium plug syndrome, the meconium is abnormal in consistency.

Meconium found in the intestine of a newborn, consisting of succus entericus (bile salts, bile acids, and debris from the intestinal mucosa) and is normally evacuated within 6 hours of birth or earlier.

Meconium ileus occurs when meconium becomes inspissated and obstructs the distal ileum and is usually a manifestation of cystic fibrosis. Approximately 20% of infants with cystic fibrosis present with meconium ileus at birth. It is more common in white populations and affects both sexes almost equally.

Although it is usually understood as synonymous with cystic fibrosis until proven otherwise, it may also be seen with pancreatic atresia or stenosis of the pancreatic duct.

Only rarely does it occur without cystic fibrosis or pancreatic abnormality, and is thought to be related to gut immaturity (more favorable outcome).

Radiographic features:

Radiograph:

Non specific and may show dilated bowel loops proximal to the impaction. Classically, there is a paucity or absence of air-fluid levels and a "bubbly" appearance of the distended intestinal loops on radiographs. Occassionally, has a mottled appearance on radiographs during the first 2 days of life.

Fluoroscopy: contrast enema

Will show a microcolon involving the entire large bowel and may show impacted meconium pellets particularly in the right colon or in the distal ileum.

Ultrasound:

Prenatal ultrasound findings associated with meconium ileus include

- Echogenic bowel which can be dilated and thick walled
- Polyhydramnios
- Fetal ascites
- Peritoneal wall calcifications
- Intra-abdominal cysts

Treatment and prognosis:

Water soluble contrast enema usually clears the impacted meconium

Complications include:

- Ileal atresia or stenosis
- Ileal perforation resulting in meconium peritonitis
- Volvulus with or without pseudocyst formation

Differential diagnosis:

General imaging differential considerations include

- Total colonic Hirschsprung disease
- Meconium plug syndrome

c) Basal cell carcinoma. See the Question 3.b of Group – C of Paper – I of 2009

d) Premalignant conditions of penile carcinoma.

Precancerous conditions of the penis have the potential to develop into penile cancer.

The most common precancerous conditions of the penis are:

- **Penile intraepithelial neoplasia (pein):** Penile intraepithelial neoplasia (PeIN) is the most common precancerous condition of the penis. PeIN is a general term used to describe precancerous conditions of the penis that may develop into invasive squamous cell carcinoma (SCC) if not treated.
- **Balanitis xerotica obliterans (BXO):** Balanitis xerotica obliterans (BXO) is an inflammatory condition of skin that affects the foreskin and the glans (glans penis or head) of the penis. It may also be called penile lichen sclerosus.
- **Buschke-Lowenstein tumour:** Buschke-Lowenstein tumour is most commonly found on the glans (glans penis or head) of the penis. It has been suggested that Buschke-Lowenstein tumour is a low-grade cancer related to verrucous carcinoma of the penis. It is a slow-growing tumour that may grow as big as 15 cm.
- **Bowenoid papulosis:** Bowenoid papulosis has an extremely low chance of developing into cancer. It affects younger men more often than older men.
  The following risk factor may increase a man's chance of developing bowenoid papulosis:
  - HPV infection
- **Leukoplakia:** Leukoplakia has the potential to become squamous cell carcinoma (SCC).

e) Acute pancreatitis. See the Question 2.a of Group – B of Supplementary Paper – I of 2011.

Group – D

4) Answer briefly on (any three): 3x5 = 15

a) Glasgow Coma Scale.
b) Radiofrequency ablation of tumours.
c) Tension pneumothorax.
d) Epulis.
e) Complications of spinal anaesthesia.

Answer.

a) Glasgow Coma Scale. See the Question 4.d of Group – D of Supplementary Paper –II of 2010.
b) Radiofrequency ablation of tumours.

Definition: Radiofrequency ablation for tumour is a minimally invasive procedure that uses electrical energy and heat to destroy cancer cells.

During radiofrequency ablation for cancer, imaging tests are used to guide a thin needle through the skin or through an incision and into the cancer tissue. High-frequency energy passes through the needle and causes the surrounding tissue to heat up, killing the nearby cells.

Radiofrequency ablation is sometimes used to treat cancers in the:

- Bone
- Kidney
- Liver
- Lung
- Prostate

RFA is done under local or general anaesthetic, depending on individual circumstances. The doctor uses an ultrasound or a CT scan to guide the procedure. A probe called an electrode applies electrical current (radiofrequency) to a tumour. The current heats the cancer cells to a high temperature which destroys (ablates) them.

The risk of complications with RFA is low.

Lesser side effects may include:

- Some pain or discomfort
- Slight fever
- Generally feeling unwell.

c) Tension pneumothorax. See the Question 4.e of Group – D of Supplementary Paper – II of 2013.

d) Epulis. See the Question 4.b of Group – D of Paper – II of 2011.

e) Complications of spinal anaesthesia.

Complications are:

- Pain - 25% of patients still experience pain despite spinal anaesthesia.
- Post-dural headache from cerebrospinal fluid (CSF) leak.
- Hypotension and bradycardia through blockade of the sympathetic nervous system.
- Limb damage from sensory and motor block.
- Epidural or intrathecal bleed.
- Respiratory failure if block is 'too high'.
- Direct nerve damage.
- Hypothermia.
- Damage to the spinal cord - may be transient or permanent.
- Spinal infection.
- Aseptic meningitis.
- Haematoma of the spinal cord - enhanced by use of LMWH pre-operatively.
- Anaphylaxis.
- Urinary retention.
- Spinal cord infarction.

- Anaesthetic intoxication

The West Bengal University of Health Sciences

M.B.B.S. 3rd Professional Part – II Examination, 2014

Subject: Surgery **Time: 2 1/2 hrs.**

Paper: II **Marks: 60**

Group –A

1. **Discuss the clinical features, investigations and treatment of Thyrotoxicosis. 5+5+5 = 15**
   [The West Bengal University of Health Sciences, Paper – II of 2014]

Answer:

For reading

- **Causes and pathological features**
- TSH secreting pituitary adenoma.
- Autoimmune stimulation (Graves's disease).
  - Thyroid stimulating antibodies (IgG) bind to TSH receptors and stimulate the thyroid cells to produce and secrete excessive amounts of thyroid hormones.
  - Thyroid gland hypertrophies and becomes diffusely enlarged.
  - The autoimmune process leads to mucopolysaccharide infiltration of the extra-ocular muscles and may lead to exophthalmus.
- T_3, T_4 secreting site in the thyroid.
  - Nodule in a multinodular goitre (Plummer's syndrome).
  - Adenoma or (very rarely) carcinoma.
- Thyroiditis (large amount of preformed hormones are released after the destruction of follicles, with transient thyrotoxicosis).
- Exogenous intake of thyroid hormones (factitious thyrotoxicosis).

Clinical features:

- **Weight loss, heat intolerance, sweating (due to stimulated metabolism and heat production).**
- **Tremor, nervousness, irritability, emotional disturbance, tiredness, and lethargy (due to CNS overactivity).**
- **Cardiac features are caused by beta-adrenergic sympathetic activity:**
  - **palpitations, tachycardia, and arrhythmias.**

- **Eye signs can be:**
  - **minimal/mild (soft tissue oedema, chemosis);**
  - **very prominent (severe exophthalmus, corneal ulcers, diplopia);**
  - **ophthalmopathy is usually bilateral but may only involve one eye.**
- **Pretibial myxoedema, thyroid acropachy, vitiligo, and alopecia are rare.**

Primary thyrotoxicosis	Secondary thyrotoxicosis
Eye signs:exopthalmos – more prominent. **CNS signs: tremor – more prominent.** **Cardiac signs - tachycardia**	**CNS signs: tremor – less prominent.** **Cardiac signs – tachycardia** **Cardiomegaly.CCF** **Cardiac signs are more prominent.**

Thyroid storm (thyrotoxic crisis):

- **Rare presentation of extreme signs of thyrotoxicosis and severe metabolic disturbances.**
- **Precipitated by non-thyroid surgery, major trauma, infection, imaging studies with iodinated contrast medium in patients with unrecognized thyrotoxicosis.**
- **Features are insomnia, anorexia, vomiting, diarrhoea, marked sweating, fever, marked tachycardia.**
- **Early clinical diagnosis of the condition and immediate treatment decrease the risk of fatal outcome.**

Investigations:

- **Thyroid function test: TSH level; free T_4 and free T_3 (in all causes but pituitary) - T3 thyrotoxicosis is diagnosed by estimating the free T3. It should be suspected if the clinical picture is suggestive but routine tests of thyroid function reveal a normal T4 but suppressed TSH.**
- **Positive serology for thyroid autoantibodies.**
- **Radioactive iodine scan (or technetium scan): helpful in distinguishing the diagnosis of Graves's disease, thyroiditis, toxic nodule (unilateral uptake with negative scan on the contralateral side), or toxic mutinodular goitre.**

<u>Note: Thyrotoxicosis should always be considered in:</u>

- **Children with a growth spurt, behaviour problems or myopathy**
- **Tachycardia or arrhythmia in the elderly**
- **Unexplained diarrhoea**
- **Loss of weight.**

Treatment:
Principles of treatment of thyrotoxicosis: Non-specific measures are rest and sedation and in established thyrotoxicosis should be used only in conjunction with specific measures.

- **Medical treatment:**

- **Antithyroid drugs block hormone synthesis.**
  - **Carbimazole 20mg bd, then reducing dose**
  - **Propylthiouracil 200mg bd: blocks the peripheral conversion of T_4 to T_3.**

- **Beta-blockers (propranolol 40-120mg/day, carvedilol) are used to control tachycardia and tremor.**

- **Radioactive iodine (I^{131})-**

  - **Contraindicated in severe eye disease (could worsen after I^{131} treatment),**
  - **Young women (risk of teratogenicity in pregnancy),**
  - **Patients who are main carers of small children.**

Surgical treatment

- **Total thyroidectomy (for Graves's disease). Indicated in patients who are not candidates for I^{131} therapy. It is the treatment of choice in those with eye disease and patients where control of symptoms has been difficult on medication.**
  - **Slightly higher risk of RLN injury and hypoparathyroidism (due to increased vascularity of the gland and the local fibrosis).**
- **Thyroid lobectomy: for isolated nodules or adenomas.**

Treatment options	Advantage	Disadvantage
Antithyroid drugs	No surgery and no use of radioactive materials.	○ Treatment is prolonged and the failure rate is at least 50 per cent. The duration of treatment may be tailored to the severity of the toxicity with milder cases being treated for only six months and severe for two years before stopping therapy.
Surgery	The goitre is removed, the cure is rapid, and the cure rate is high if surgery has been adequate.	○ Recurrence of thyrotoxicosis occurs in approximately 5 per cent of cases when Subtotal Thyroidectomy is carried out. ○ There is a risk of permanent hypoparathyroidism and nerve injury. ○ Young women tend to have a poorer cosmetic result from the scar.

Group – B

2) a) Describe the pathophysiology of BHP. Mention the medical and surgical management of BHP.
6+4+5 = 15

Answer.

Note:

- **BPH develops in the transition zone.**
- **It is truly a hyperplastic process resulting from an increase\in cell number.**
- **Microscopic evaluation reveals a nodular,growth pattern that is composed of varying amounts of stroma and epithelium. Stroma is composed of varying amounts of collagen and smooth muscle.**
- **The differential representation of the histologic components of BPH explains, in part, the potential responsiveness to medical therapy.**
- **As BPH nodules in the transition zone enlarge, they compress the outer zones of the prostate, resulting in the formation of a so-called surgical capsule. This boundary separates the transition zone from the peripheral zone and serves as a cleavage plane for open enucleation of the prostate during open simple prostatectomies performed for BPH.**

Pathophysiology:

- **The symptoms of BPH to either the obstructive component of the prostate or the secondary response of the bladder to the outlet resistance.**
- **The obstructive component can be subdivided into the mechanical and the dynamic obstruction.**
- **As prostatic enlargement occurs, mechanical obstruction may result from intrusion into the urethral lumen or bladder neck, leading to a higher bladder outlet resistance.**
- **Prior to the zonal classification of the prostate, urologists often referred to the "3 lobes" of the prostate, namely, the median and the 2 lateral lobes.**
- **Prostatic size on digital rectal examination (DRE) correlates poorly with symptoms, in part, because the median lobe is not readily palpable.**
- **The dynamic component of prostatic obstruction explains the variable nature of the symptoms experienced by patients.**
- **The prostatic stroma, composed of smooth muscle and collagen, is rich in adrenergic nerve supply.**
- **The level of autonomic stimulation thus sets a tone to the prostatic urethra. Use of alpha-blocker therapy decreases this tone, resulting in a decrease in outlet resistance.**

Treatment: watchful waiting is the appropriate management of men with mild symptom scores.

- **Medical treatment:**

Classification of Medical Therapy and Recommended Dosage in BPH.

Classification	Oral Dosage
Alpha-blockers	
Nonselective	
Phenoxybenzamine	10 mg twice a day
Alpha-1, short-acting	
Prazosin	2 mg twice a day
Alpha-1, long-acting	
Terazosin	5 or 10 mg daily
Doxazosin	4 or 8 mg daily
Alpha-1a selective	
Tamsulosin	0.4 or 0.8 mg daily
Alfuzosin	10 mg daily
5-alpha-reductase inhibitors	
Finasteride	5 mg daily
Dutasteride	0.5 mg daily
Subcutaneous implant	Yearly
Triptorelin pamoate	3.75 mg every month

Conventional Surgical treatment:

- **Transurethral resection of the prostate: best option.**
  - **Risks of TURP: include retrograde ejaculation (75%), impotence (5–10%), and incontinence (<1%). Complications include bleeding, urethral stricture or bladder neck contracture, perforation of the prostate capsule with extravasation, and if severe, TUR syndrome resulting from a hypervolemic,hyponatremic state due to absorption of the hypotonic irrigating solution.**
  - **Clinical manifestations of the TUR syndrome include nausea, vomiting, confusion, hypertension, bradycardia, and visual disturbances. The risk of the TUR syndrome increases with resectiontimes >90 minutes.**
  - **Treatment includes diuresis and, in severe cases, hypertonic saline administration.**

- **Transurethral incision of the prostate—Men with moderate to severe symptoms and a small prostate often have posterior commissure hyperplasia (elevated bladder neck). These patients will often benefit from an incision of the prostate. This procedure is more rapid and less morbid than TURP.**
- **Open simple prostatectomy—When the prostate is too large to be removed endoscopically, an open enucleation is necessary.Glands >100 g are usually considered for open enucleation.**
  - **A simple suprapubic prostatectomy is performed transvesically and is the operation of choice in dealing with concomitant bladder pathology.**
  - **In a simple retropubic prostatectomy, the bladder is not entered.**

Minimally Invasive Therapy

- **Laser therapy—Many different techniques of laser surgery for the prostate have been described. Two main energy sources of lasers have been utilized—Nd:YAG and holmium:YAG**

.

<u>Techniques:</u>

- **Visual contact ablative techniques are more time-consuming procedures.**
- **Interstitial laser therapy places fibers directly into the prostate, usually under cystoscopic control.**

- **Transurethral electrovaporization of the prostate.**
- **Hyperthermia—Microwave hyperthermia is most commonly delivered with a transurethral catheter.**
- **Transurethral needle ablation of the prostate.**
- **High-intensity focused ultrasound.**
- **Intraurethral stents.**

Or

Group – C

3) Write short notes on (any three) 3x5 = 15

a) Paget's disease of nipple.
b) Electric burns.
c) Dentigerous cyst.
d) Lucid interval.
e) Fistula in ano.

Answers.

a) Paget's disease of nipple:

Introduction: Paget Disease of the Nipple is characterized by eczematoid changes of the nipple, which may involve the surrounding areola.

Clinical features:

- **Burning, pruritus, and hypersensitivity may be prominent symptoms.**
- **Almost always accompanied by an underlying malignancy, either invasive ductal carcinoma or DCIS.**
- **Palpable masses are present in approximately 60% of patients.**

Investigations:

- **Mammography should be performed to identify other areas of involvement.**
- **If clinical suspicion is high, a pathologic diagnosis should be obtained by wedge biopsy of the nipple and underlying breast tissue.**

Treatment: is mastectomy or BCT with excision of the nipple-areolar complex (sometimes called a central lumpectomy), followed by radiation therapy.

Prognosis: The prognosis is related to tumor stage.

b) Electric burns.

Introduction: Factors influencing severity include the voltage (high is >1,000 V), resistance, type of current, current pathway through the body, and duration of contact with an electrical source. Severity of injury frequently is underestimated when only the entrance and exit wounds are considered.

Electrical injuries:

- **Low voltage (< 1000V). Domestic electrical supply. Causes local contact wounds but no deep injury. May cause cardiac arrest.**
- **High voltage (> 1000V). High tension cables, power stations, lightning. Causes cutaneous and deep tissue damage with entry and exit wounds.**
- **ECG on admission for all injuries; continuous cardiac monitoring for 24h for significant injuries.**
- **In high voltage injury, muscle damage may require fasciotomy.**
- **Myoglobinuria can cause renal failure: urine output > 75-100mL/h.**

Complications:

• **Cardiopulmonary arrest (more common with alternating current).**	• **Associated fractures related to fall or severe muscle contraction.**	• **Cataracts.**
• **Thrombosis.**	• **Spinal cord injury,**	• **Rhabdomyolysis may occur and result in myoglobin release from injured cells of deep tissues. Precipitation of protein in the renal tubules can cause acute renal failure.**

d) Venous ulcer:

Introduction:

- **Chronic venous disease, including chronic venous insufficiency and chronic venous ulceration, is a common and important medical problem that causes significant morbidity. Venous ulcers are expensive to treat and adversely impact patient's quality of life.**
- **Venous ulcers occur more commonly in the elderly, the peak prevalence occurring between ages 60 and 80 years.**
- **A venous leg ulcer can develop after a minor injury if there is a problem with the circulation of blood in your leg veins. If this happens, the pressure inside the veins increases.**

Risk factors:

- **Obesity – this increases the risk of high pressure in the leg veins**

- **Not being able to move for a long period of time – this can weaken the calf muscles, which can affect circulation in the leg veins**
- **Having previously had deep vein thrombosis (DVT)– blood clots that develop in the leg, which can damage valves**
- **Varicose veins – swollen and enlarged veins caused by malfunctioning valves**
- **Previous injury to the leg, such as a broken or fractured bone, which may cause DVT**
- **Previous surgery to the leg, such as a hip replacement or knee replacement, which can prevent you from moving about**
- **Increasing age – as people generally find it harder to move about as they get older**

{Pathophysiology: (go through):

- **Venous hypertension: Deep vein thrombosis, perforator insufficiency, superficial and deep vein insufficiencies, arteriovenous fistulas and calf muscle pump insufficiencies lead to increased pressure in the distal veins of the leg and finally venous hypertension.**
- **Fibrin cuff theory: Fibrin gets excessively deposited around capillary beds leading to elevated intravascular pressure. This causes enlargement of endothelial pores resulting in further increased fibrinogen deposition in the interstitium. The "fibrin cuff" which surrounds the capillaries in the dermis decreases oxygen permeability 20-fold. This permeability barrier inhibits diffusion of oxygen and other nutrients, leading to tissue hypoxia causing impaired wound healing.**
- **Inflammatory trap theory: Various growth factors and inflammatory cells, which get trapped in the fibrin cuff promote severe uncontrolled inflammation in surrounding tissue preventing proper regeneration of wounds. Leukocytes get trapped in capillaries, releasing proteolytic enzymes and reactive oxygen metabolites, which cause endothelial damage. These injured capillaries become increasingly permeable to various macromolecules, accentuating fibrin deposition. Occlusion by leukocytes also causes local ischemia thereby increasing tissue hypoxia and reperfusion damage.**
- **Dysregulation of various cytokines.**
- **Dysregulation of various pro-inflammatory cytokines and growth factors like tumor necrosis factor-α (TNF-α), TGF-β and matrix metalloproteinases lead to chronicity of the ulcers.**
- **Miscellaneous: Thrombophilic conditions like factor V Leiden mutation, prothrombin mutations, deficiency of antithrombin, presence of antiphospholipid antibodies, protein C and S deficiencies and hyperhomocysteinemia are also implicated.}**

Clinical features:
Venous leg ulcers are open, often painful, sores in the skin that take more than four to six weeks to heal. They most often develop on the inside of the leg, just above the ankle.
Symptoms:

- **Swollen ankles (oedema)**
- **Discolouration and darkening of the skin around the ulcer**
- **Hardened skin around the ulcer, which may make your leg feel hard and resemble the shape of an upside-down champagne bottle**
- **A heavy feeling in your legs**
- **Aching or swelling in your legs**
- **Red, flaky, scaly and itchy skin on your legs (varicose eczema)**
- **Swollen and enlarged veins on your legs (varicose veins)**
- **An unpleasant and foul-smelling discharge from the ulcer**

Signs of an infection:

A venous leg ulcer can be susceptible to bacterial infection. Symptoms of an infected leg ulcer can include:

- **Worsening pain**
- **A green or unpleasant discharge coming from the ulcer**
- **Redness and swelling of the skin around the ulcer**
- **A high temperature (fever)**

Nonsurgical Treatment

❖ **Infected ulcers**

- **Necessitate treatment of the infection first.**
- **Staphylococcus aureus, Streptococcus pyogenes, and Pseudomonas species are responsible for most infections.**
- **Usually treated with local wound care, wet-to-dry dressings, and oral antibiotics.**
- **Topical antiseptics should be avoided.**
- **Severe infections require intravenous antibiotics.**

❖ **Leg elevation**

Leg elevation can temporarily decrease edema and should be instituted when swelling occurs. This should be done before a patient is fitted for stockings or boots.

❖ **Compression therapy**

- **Compression therapy is the primary treatment for CVI.**

- **Elastic compression stockings**
  - **Fitted to provide a compression gradient from 30 to 40 mm Hg, with the greatest compression at the ankle.**
  - **Donned on arising from bed and removed at bedtime.**
  - **Effective in healing ulcers but can take months to obtain good results.**
  - **Stockings do not correct the abnormal venous hemodynamics and must be worn after the ulcer has healed to prevent recurrence.**
  - **Principal drawback is patient compliance.**
- **Unna boots**
  - **Paste gauze compression dressings that contain zinc oxide, calamine, and glycerin.**
  - **Used to help prevent further skin breakdown.**
  - **Provide nonelastic compression therapy.**
  - **Changed once or twice a week.**
  - **Healing time for ulcers is less than that of elastic compression alone.**
- **Pneumatic compression devices**
  - **Provide dynamic sequential compression.**
  - **Used primarily in the prevention of deep vein thrombi in hospitalized patients.**
  - **Also used successfully to treat venous insufficiency.**

❖ **Topical medications**

- **Largely ineffective as a stand-alone therapy for venous stasis ulcers.**
- **Topical therapy is directed at absorbing wound drainage and avoiding desiccation of the wound.**

- **Antiseptics can be counterproductive. Hydrogen peroxide, povidone-iodine, acetic acid, and sodium hypochlorite are toxic to cultured fibroblasts and should be used for the shortest duration necessary to control ulcer infection.**

❖ **Surgical Therapy: Skin grafting.**

Group – D

4) Answer briefly on (any three): 3x5 = 15

a) Penile carcinoma.
b) Muscle relaxant.
c) Flail chest.
d) Epidural anaesthesia.
e) Compartment syndrome.

Answers.

a) Go through the following table:

Premalignant cutaneous lesions:

- **Cutaneous horn.**
- **Pseudoepitheliomatous micaceous and keratotic balanitis.**
- **Balanitis xerotica obliterans.**
- **Leukoplakia.**
- **Erythroplasia of Queyrat.**
- **Bowen's disease.**
- **Buschke Lowenstein tumour: also known as verrucous carcinoma or giant condyloma acuminatum**

Classification of penile cancer:

- **Squamous cell carcinoma (SCC) is the most common penile cancer**
- **Kaposi's sarcoma**
- **Basal cell carcinoma,**
- **Melanoma, sarcoma,**
- **Paget's disease.**

- **Metastases are occasionally seen from bladder, prostate, rectum, and other primary sites.**

Incidence and aetiology of SCC:

Penile cancer is rare, representing 1% of male cancers.
The incidence appears to be decreasing, most occurring in elderly men

Risk factors for SCC:

- **Age: penile cancer incidence rises during the 6th decade and peaks in the 8th decade. It is unusual <40 years, but has been reported in children.**
- **Premalignant lesions: 42% of patients with penile SCC are reported to have had a pre-existing penile lesion.**
- **A prepuce (foreskin): penile cancer is rare in men circumcised at a young age. It is virtually non-existent in Israel. It is thought that chronic irritation with smegma and inflammation (balanitis) is contributory.**
- **Human papilloma virus (HPV) wart infection, especially with types 16, 18, and 21.**
- **Smoking and tobacco products.**

Pathology and staging of penile SCC:

- **SCC starts as a slow-growing papillary, flat or ulcerative lesion on the glans (48%), prepuce (21%), glans and prepuce (9%), coronal sulcus (6%), or shaft (2%) preceded by carcinoma in situ.**
- **The remainder are indeterminate.**
- **It grows locally beneath the foreskin before invading the corpora cavernosa, urethra, and, eventually, the perineum, pelvis, and prostate.**
- **Metastasis is initially to the superficial then deep inguinal and, subsequently, iliac and obturator lymph nodes.**
- **Skin necrosis, ulceration, and infection of the inguinal lymph nodes may lead to sepsis or haemorrhage from the femoral vessels.**
- **Blood-borne metastasis to lungs and liver is rare.**
- **Histologically, SCC exhibits keratinization, epithelial pearl formation, and mitoses.**
- **Grading is low (75%), intermediate (15%), or high (10%); grading correlates with prognosis, as does the presence of vascular invasion.**

Presentation:

- **A hard, painless lump on the glans penis is the most common presentation.**
- **A bloody discharge may be confused with haematuria.**
- **Rarely, a groin mass or urinary retention are presenting symptoms.**
- **Examination reveals a solid non-tender mass or ulcer beneath or involving the foreskin.**
- **There is usually evidence of local infection.**
- **In more advanced disease, prepuce, glans, shaft, scrotum, and even perineum are replaced by tumour.**
- **The inguinal lymph nodes are examined. They may be enlarged, fixed, or even ulcerate overlying skin.**

Investigations:

• **A biopsy is indicated**	• **Chest radiology**	**Pelvic CT scan**	**Serum calcium**	**Liver function tests**

Treatment:

The primary tumour:

- **The first-line treatment of penile cancer, regardless of the inguinal node status, is surgery.**
- **Circumcision is appropriate for preputial lesions, but local recurrence observed in.**
- **Penis-preserving wide excision of glanular lesions with skin graft glanular reconstruction may be suitable for smaller tumours, giving good cosmetic and functional results.**
- **Alternatives to surgery include laser or cryoablation, radiotherapy or brachytherapy, photodynamic therapy, or topical 5-fluorouracil.**
- **For more advanced tumours, partial or total penile amputation is required, depending on the extent of the tumour. Partial amputation is preferable, provided a 2cm margin of palpably normal shaft can be obtained.**
- **Local recurrence occurs in 10%, if the excision margin is positive.**

Total amputation involves excision of the scrotum and its contents, with formation of a perineal urethrostomy. The most common complication is urethral meatal stenosis.

Radiotherapy remains an alternative, but disadvantages include radio-resistance, leading to reported recurrence rates of 30-60%; tissue necrosis and damage leading to urethral stricture, fistula, and pain.

Lymphadenopathy:

- **Six weeks of broad-spectrum antimicrobials are given after the primary tumour has been removed. Nodes become clinically insignificant in 50% of patients, who may then be followed-up.**
- **For those with persistent inguinal lymphadenopathy, in the absence of demonstrable pelvic or metastatic disease, bilateral inguinal lymphadenectomy should be considered.**

Radiotherapy and chemotherapy are alternative or adjuvant treatments for metastatic nodal disease in unfit, elderly, or inoperable patients.

- **Prophylactic lymphadenectomy is currently practised for tumours exhibiting vascular invasion, high grade, or stages T2-4.**
- **Distant metastatic disease is treated using single-agent systemic chemotherapy: cisplatin, bleomycin, or methotrexate.**
- **Experience with combination chemotherapy is increasing.**

b) Muscle relaxants.

Introduction:

- **A muscle relaxant is a drug which affects skeletal muscle function and decreases the muscle tone. It may be used to alleviate symptoms such as muscle spasms, pain, and hyperreflexia.**
- **The term "muscle relaxant" is used to refer to two major therapeutic groups: neuromuscular blockers and spasmolytics.**
- **Neuromuscular blockers act by interfering with transmission at the neuromuscular end plate and have no central nervous system (CNS) activity. They are often used during surgical procedures and in intensive care and emergency medicine to cause temporary paralysis.**

- **Spasmolytics, also known as "centrally acting" muscle relaxants, are used to alleviate musculoskeletal pain and spasms and to reducespasticity in a variety of neurological conditions.**
  While both neuromuscular blockers and spasmolytics are often grouped together as muscle relaxants, the term is commonly used to refer to spasmolytics only.

Clinical use:

- **Spasmolytics such as carisoprodol, cyclobenzaprine, metaxalone, and methocarbamol are commonly prescribed for low back pain or neck pain, fibromyalgia, tension headaches and myofascial pain syndrome. However, they are not recommended as first-line agents; in acute low back pain, they are not more effective than paracetamol or nonsteroidal anti-inflammatory drugs (NSAIDs), and in fibromyalgia they are not more effective than antidepressants.**
- **Muscle relaxants (according to one study) were not advised for orthopedic conditions, but rather for neurological conditions such as spasticity in cerebral palsy and multiple sclerosis. Dantrolene, although thought of primarily as a peripherally acting agent, is associated with CNS effects, whereas baclofen activity is strictly associated with the CNS.**
- **Muscle relaxants are thought to be useful in painful disorders based on the theory that pain induces spasm and spasm causes pain.**
- **In general, muscle relaxants are not approved by FDA for long-term use.**
- **Muscle relaxants such as tizanidine are prescribed in the treatment of tension headaches.**

Side effects:

- **Muscle relaxants are very powerful drugs which may produce negative effects, including heart failure and paralysis. Patients most commonly report sedation as the main adverse effect of muscle relaxants.**
- **Cyclobenzaprine produces confusion and lethargy, as well as anticholinergic side effects. When taken in excess or in combination with other substances, it may also be toxic.**
- **Patient can experience dry mouth, fatigue, lightheadedness, constipation or blurred vision.**
- **Some serious but unlikely side effects may be experienced, including mental or mood changes, possible confusion and hallucinations, and difficulty urinating.**
- **In a very few cases, very serious but rare side effects may be experienced: irregular heartbeat, yellowing of eyes or skin,fainting, abdominal pain including stomachache, nausea or vomiting, lack of appetite, seizures, dark urine, or loss of coordination.**

d) Epidural anaesthesia

Introduction: Epidural anesthesia is regional anesthesia that blocks pain in a particular region of the body. The goal of an epidural is to provide analgesia, or pain relief, rather than anesthesia which leads to total lack of feeling. Epidurals block the nerve impulses from the lower spinal segments.

Difference from spinal anesthesia: Spinal anaesthesia is a technique whereby a local anaesthetic drug is injected into the cerebrospinal fluid. This technique has some similarity to epidural anaesthesia, and lay people often confuse the two techniques. Important differences include:

- **To achieve epidural analgesia or anaesthesia, a larger dose of drug is typically necessary than with spinal analgesia or anaesthesia.**
- **Onset of analgesia is slower with epidural analgesia or anaesthesia than with spinal analgesia or anaesthesia.**
- **An epidural injection may be performed anywhere along the vertebral column (cervical, thoracic, lumbar, or sacral), while spinal injections are typically performed below the second lumbar vertebral body to avoid piercing and consequently damaging the spinal cord.**
- **It is easier to achieve segmental analgesia or anaesthesia using the epidural route than using the spinal route.**
- **An indwelling catheter is more commonly placed in the setting of epidural analgesia or anaesthesia than with spinal analgesia or anaesthesia.**

Indications: Injecting medication into the epidural space is primarily performed for analgesia.

For analgesia alone, where Surgery is not contemplated. An epidural injection or infusion for pain relief (e.g. in child birth) is less likely to cause loss of muscle power, but has to be augmented to be sufficient for surgery.

- **As an adjunct to general anaesthesia. This may reduce the patient's requirement for opioid analgesics. This is suitable for a wide variety of surgery, for example gynaecological surgery (e.g. hysterectomy), orthopaedic surgery (e.g. hip replacement), general surgery (e.g.laparotomy) and vascular surgery (e.g. open aortic aneurysm repair).**
- **As a sole technique for surgical anaesthesia. Some operations, most frequently Caesarean section, may be performed using an epidural anaesthetic as the sole technique. This can allow the patient to remain awake during the operation.**
- **For post-operative analgesia, after an operation where the epidural technique was used as either the sole anaesthetic, or was used in combination with general anaesthesia. Analgesics are given into the epidural space typically for a few days after surgery, provided a catheter has been inserted. Through the use of a patient-controlled epidural analgesia (PCEA) infusion pump, a person has the ability to give themselves an occasional dose of pain medication through an epidural catheter.**
- **Treatment of back pain. Injection of analgesics and steroids into the epidural space may improve some forms of back pain.**
- **Treatment of chronic pain or palliation of symptoms in terminal care, usually in the short- or medium-term.**

<u>Epidural analgesia during childbirth:</u>

Advantages	Disadvantages
• Better pain relief than other pain medication • Fewer babies needing naloxone to counter opiate use by the mother • Decreased maternal hyperventilation and increased oxygen supply to baby • Decreased circulating adrenocorticotropic hormone and decreased fetal distress [	• More use of instruments to assist with the birth • Increased risk of Caesarean section for fetal distress • Longer delivery (second stage of labour) • Increased need for oxytocin to stimulate uterine contractions • Increased risk of very low blood pressure • Increased risk of muscular weakness for a period of time after the birth

	• **Increased risk of fluid retention** • **Increased risk of fever**

Epidural analgesia has been demonstrated to have several benefits after surgery, including:

- **Effective analgesia without the need for systemic opioids.**
- **The incidence of postoperative respiratory problems and chest infections is reduced.**
- **The incidence of postoperative myocardial infarction ("heart attack") is reduced.**
- **The stress response to surgery is reduced.**
- **Motility of the intestines is improved by blockade of the sympathetic nervous system.**
- **Use of epidural analgesia during surgery reduces blood transfusion requirements.**

Complications:

  - **Failure to achieve analgesia or anaesthesia occurs in about 5% of cases, while another 15% experience only partial analgesia or anaesthesia**
  - **The following factors are associated with failure to achieve epidural analgesia/anaesthesia**
    - **Obesity**
    - **Multiparity**
    - **History of a previous failure of epidural anaesthesia**
    - **History of regular opiate use**
    - **Cervical dilation of more than 7 cm at insertion**
    - **The use of air to find the epidural space while inserting the epidural instead of alternatives such as saline or lidocaine**
- **Accidental dural puncture with headache. This may cause cerebrospinal fluid (CSF) to leak out into the epidural space, which may in turn cause a post dural puncture headache (PDPH). This can be severe and last several days, and in some rare cases weeks or months. If severe it may be successfully treated with an epidural blood patch (a small amount of the subject's own blood given into the epidural space via another epidural needle which clots and seals the leak). Most cases resolve spontaneously with time.**
- **Delayed onset of breastfeeding and shorter duration of breastfeeding.**
- **Bloody tap.**
- **Catheter misplaced into a vein.**
- **High block, as described above.**
- **Catheter misplaced into the subarachnoid space. If the catheter is accidentally misplaced into the subarachnoid space (e.g. after an unrecognised accidental dural puncture), normally cerebrospinal fluid can be freely aspirated from the catheter (which would usually prompt the anaesthetist to withdraw the catheter and resite it elsewhere). If, however, this is not recognised, large doses of anaesthetic may be delivered directly into the cerebrospinal fluid. This may result in a high block, or, more rarely, a *total spinal*, where anaesthetic is delivered directly to the brainstem, causing unconsciousness and sometimes seizures.**
- **Neurological injury lasting less than 1 year.**
- **Epidural abscess formation.**
- **Epidural haematoma formation.**
- **Neurological injury lasting longer than 1 year.**
- **Paraplegia.**

- **Arachnoiditis.**
- **Death (extremely rare, less than 1 in 100,000).**

e) Compartment syndrome

Compartment syndrome is increased pressure within one of the body's compartments which contains muscles and nerves.

Compartment syndrome most commonly occurs in compartments in the leg or arm.

There are two main types of compartment syndrome: acute and chronic.

Acute compartment syndrome occurs after a traumatic injury such as a car crash. The trauma causes a severe high pressure in the compartment which results in insufficient blood supply to muscles and nerves. Acute compartment syndrome is a medical emergency that requires surgery to correct. If untreated, the lack of blood supply leads to permanent muscle and nerve damage and can result in the loss of function of the limb.

Chronic compartment syndrome: When compartment syndrome is caused by repetitive use of the muscles, as in a cyclist, it is known as chronic compartment syndrome

Chronic exertional compartment syndrome is an exercise-induced condition in which the pressure in the muscles increases to extreme levels during exercise. The pressure creates a decrease in blood flow to the affected area which leads to a deprivation of oxygen to the muscles. The symptoms are a sensation of extreme tightness in the affected muscles followed by a burning sensation if exercise is continued. Chronic exertional compartment syndrome usually occurs in athletes who participate in repetitive impact sports such as running.

- ❖ **Acute compartment syndrome can also occur after injuries without bone fractures, including:**

- **Crush injuries**
- **Burns**
- **Overly tight bandaging**
- **Prolonged compression of a limb during a period of unconsciousness**
- **Surgery to blood vessels of an arm or leg**
- **A blood clot in a blood vessel in an arm or leg**
- **Extremely vigorous exercise, especially eccentric movements (extension under pressure)**
- **Taking anabolic steroids can also contribute to developing compartment syndrome.**

- ❖ **Chronic compartment syndrome develops over days or weeks.**

Also called exertional compartment syndrome, it may be caused by regular, vigorous exercise. The lower leg, buttock, or thigh is usually involved.

- ➢ **Acute compartment syndrome usually develops over a few hours after a serious injury to an arm or leg. Some symptoms of acute compartment syndrome include:**

- **A new and persistent deep ache in an arm or leg**
- **Pain that seems greater than expected for the severity of the injury**
- **Numbness, pins-and-needles, or electricity-like pain in the limb**
- **Swelling, tightness and bruising**

- ➢ **Symptoms of chronic compartment syndrome (exertional compartment syndrome) include worsening aching or cramping in the affected muscle (buttock, thigh, or lower leg) within a half-hour of starting exercise. Symptoms usually go away with rest, and muscle function remains normal. Exertional compartment syndrome can feel like shin splints and be confused with that condition.**

Diagnosis:

Compartment syndrome is a clinical diagnosis made by a physician. It can be tested for by gauging the pressure within the muscle compartments. If the pressure is sufficiently high, a fasciotomy will be required to relieve the pressure.

Most commonly compartment syndrome is diagnosed through a diagnosis of its underlying cause and not the condition itself.

Treatment:

- **Most people with acute compartment syndrome require immediate surgery to reduce the compartment pressure. A surgeon makes long incisions through the skin and the fascia layer underneath (fasciotomy), releasing excessive pressure.**

Other supportive treatments include:

- **Keeping the body part below the level of the heart (to improve blood flow into the compartment)**
- **Giving oxygen through the nose or mouth**
- **Giving fluids intravenously**
- **Taking pain medications**

- **Chronic compartment syndrome can first be treated by avoiding the activity that caused it and with stretching and physical therapy exercises. Surgery is not as urgent in chronic or exertional compartment syndrome, but it may be required to relieve pressure.**

The West Bengal University of Health Sciences

M.B.B.S. 3rd Professional Part – II Examination, 2013

Subject: Surgery **Time: $2^{1}/_{2}$ hrs.**

Paper: II **Marks: 60**

Group –A

1) Define hydronephrosis. Discuss the causes and management of unilateral hydronephrosis. 2+5+8 = 15

Answer. Hydronephrosis is an aseptic dilatation of the kidney caused by obstruction.

Causes of Unilateral hydronephrosis

Causes of unilateral ureteric obstruction

Extramural obstruction

- Tumour from adjacent structures, e.g. cervix, prostate, rectum, colon or caecum
- Idiopathic retroperitoneal fibrosis
- Retrocaval ureter

Intramural obstruction

- Congenital stenosis, physiological narrowing of the pelviureteric junction leading to pelviureteric junction obstruction
- Ureterocele and congenital small ureteric orifice
- Inflammatory stricture following removal of ureteric calculus, repair of a damaged ureter or tuberculous infection
- Neoplasm of the ureter or bladder cancer involving the ureteric orifice

Intraluminal obstruction

- Calculus in the pelvis or ureter
- Sloughed papilla in papillary necrosis (especially in diabetics, analgesic abusers and sickle cell disease)

Management of unilateral hydronephrosis:

Imaging:

- Ultrasound scanning is the least invasive means of detecting hydronephrosis and is regularly used to diagnose pelviureteric junction obstruction in utero.
- IVU helps only if there is significant function in the obstructed kidney. The extrarenal pelvis is dilated and the minor calyces lose their normal cupping and become 'clubbed'. Contrast slowly diffuses to fill the obstructed system down to the blockage.
- Isotope renography is the best test to confirm obstructive dilatation of the collecting system. A substance (usually diethylenetriaminepenta-acetic acid (DTPA) or MAG-3) is injected intravenously. The DTPA is labelled with technetium-99m, a gamma-ray emitter, so that the passage of 99mTc-labelled DTPA through the kidneys can be tracked using a gamma camera.

99mTc-DTPA is cleared from a normal kidney but stays in the renal pelvis on the obstructed side and is retained even if urine flow is increased by administering frusemide.

- Whitaker test is indicated: A percutaneous puncture of the kidney is made and fluid is infused at a constant rate with monitoring of intrapelvic pressure. An abnormal rise in pressure confirms obstruction.
- Retrograde pyelography is rarely indicated but will confirm the site of obstruction immediately before corrective surgery

Treatment:

- The indications for operation are bouts of renal pain, increasing hydronephrosis, evidence of parenchymal damage and infection.
- Nephrectomy should be considered only when the kidney has been largely destroyed.
- Mild cases should be followed by serial ultrasound scans and operated upon if dilatation is increasing.

Types of operation:

- Pyeloplasty: In the Anderson–Hynes operation, the upper third of the ureter and the renal pelvis are mobilised. A renal vein overlying the distended pelvis can be divided, but an artery in this situation should be preserved to avoid infarction of the territory that it supplies. The anastomosis is made in front of such an artery. A nephrostomy tube or a ureteric stent protects the anastomosis.
  Laparoscopic pyeloplasty is becoming increasingly popular.

- Endoscopic pyelolysis: Disruption of the pelviureteric junction by a balloon passed up the ureter and distended under radiographic control has been used to treat idiopathic pelviureteric junction obstruction.

The long-term efficacy of this and various forms of endoscopic pyelotomy has still to be proved.

Group – B

2) a) Describe lymphatic drainage of breast. Mention the risk factors of breast carcinoma. How to manage a 52 years old female patient with locally advanced breast carcinoma?

4+3+8 = 15

Answer. Lymphatic drainage of breast

The boundaries for lymph drainage of the axilla are not well demarcated, and there is considerable variation in the position of the axillary lymph nodes.

The 5 axillary lymph node groups are:

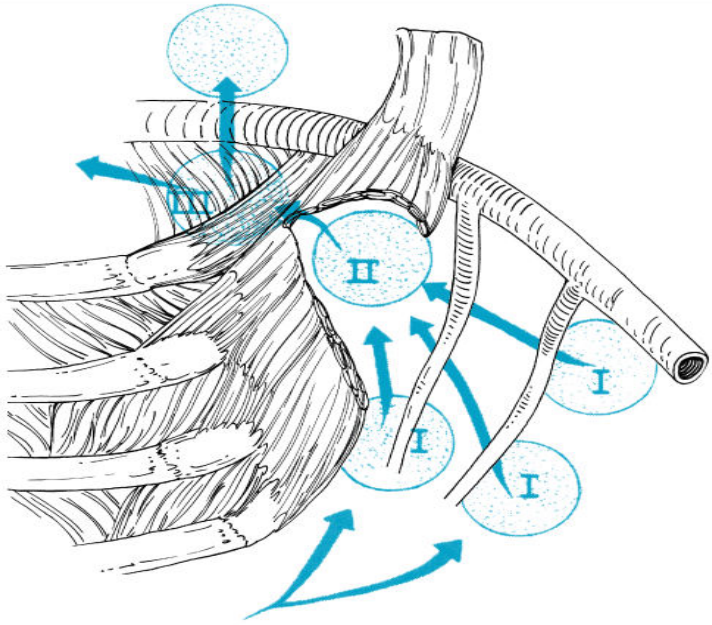
- The axillary vein group (lateral) that consists of 4 to 6 lymph nodes, which lie medial or posterior to the vein.

- The external mammary group (anterior or pectoral group) that consists of 5 or 6 lymph nodes, which lie along the lower border of the pectoralis minor muscle contiguous with the lateral thoracic vessels and receive most of the lymph drainage from the lateral aspect of the breast.
- The scapular group (posterior or subscapular) that consists of 5 to 7 lymph nodes, which lie along the posterior wall of the axilla at the lateral border of the scapula contiguous with the subscapular vessels.
- Tthe central group that consists of 3 or 4 sets of lymph nodes, which are embedded in the fat of the axilla lying immediately posterior to the pectoralis minor muscle and receive lymph drainage both from the axillary vein, external mammary, and scapular groups of lymph nodes and directly from the breast;
- The subclavicular group (apical) that consists of 6 to 12 sets of lymph nodes, which lie posterior and superior to the upper border of the pectoralis minor muscle and receive lymph drainage from all of the other groups of axillary lymph nodes.

  And

- The interpectoral group (Rotter's) that consists of 1 to 4 lymph nodes, which are interposed between the pectoralis major and pectoralis minor muscles and receive lymph drainage directly from the breast.

The lymph fluid that passes through the interpectoral group of lymph nodes passes directly into the central and subclavicular groups.



Axillary lymph node groups: Level I includes lymph nodes located lateral to the pectoralis minor muscle (PM); level II includes lymph nodes located deep to the PM; and level III includes lymph nodes located medial to the PM. Arrows indicate the direction of lymph flow.

Risk factors of breast carcinoma;

Factors Important in Populations	Factors Important in Individual Patients	Histologic Risk Factors
Age at menarche and menopause	Gender (female >> male)	Proliferative breast disease
Parity	Age (steady increase with age)	Atypical ductal hyperplasia (ADH)
Age at first birth	Family history (mothers, sisters, daughters)	Atypical lobular hyperplasia (ALH)
Breast-feeding	History of previous breast cancer (noninvasive or invasive, ipsilateral or contralateral)	Lobular carcinoma in situ (LCIS)
Exogenous hormone use or exposure		
Alcohol consumption		

Management of 52 year old female presented with locally advanced breast carcinoma

Diagnosis

Tissue Diagnosis:

- Establishing a tissue diagnosis is the initial priority on presentation of LABC. In many patients, core biopsy of the tumor is diagnostic.
- Core needle is preferred over fine needle aspiration, as cytology is insufficient to confirm lymphovascular invasion.
- Additionally, multiple cores should be extracted both to confirm invasive cancer and to evaluate hormone receptor status and HER2/neu expression.

Bilateral mammography:

- Prompt bilateral mammography in this setting is essential (except in known contraindications of BCT e.g. inflammatory breast cancer, ulcerative lesions).
- Diffuse, suspicious microcalcifications or multiple lesions in different quadrants indicate multicentric disease, and are a contraindication to breast conservation therapy (BCT).

Breast and axillary ultrasound:

- Breast and axillary ultrasound frequently yield valuable information regarding the extent of disease.
- In particular, axillary ultrasound can be used for image-guided FNA ultrasound detection of apical axillary/infraclavicular nodal metastases has been shown to provide important prognostic information.

Metastatic Work up:

- A baseline bone scan, abdominal, pelvic and chest CT scans are recommended for detection of metastatic disease.
- The most common sites of metastasis is bones followed by lung, liver and brain. The metastasis to bones may be osteoblastic or osteoclastic.

Therapeutic management:

Neoadjuvant chemotherapy:

- Currently, optimal control is achieved with preoperative chemotherapy followed by surgery and radiation.
- Since patients with LABC benefit from the tumor downstaging and improved resectability that can be achieved with neoadjuvant chemotherapy, this sequence has become the preferred approach for patients with bulky, locally advanced disease at time of diagnosis.
- Neoadjuvant chemotherapy offers several advantages compared with traditional postoperative regimens.

Neoadjuvant chemotherapy regimen:

- Currently, doxorubicin-based chemotherapy is the most widely-studied induction Regimen-
  three neoadjuvant treatment arms:
  (1) doxorubicin and cyclophosphamide alone;
  (2) doxorubicin, cyclophosphamide, and docetaxel; or
  (3) preoperative doxorubicin and cyclophosphamide followed by postoperative docetaxel.

Other neoadjuvant regimens currently being evaluated include trastuzumab, Navelbine, capecitabine, and gemcitabine.

There has been many combinations and schedule for chemotherapy a few recommended are mentioned as follows.
1. **FAC**: 5-FU + Doxorubicin + Cyclophosphamide
2. **AC:** Doxorubicin + Cyclophosphamide
3. **AC followed by paclitaxel:** Doxorubicin + Cyclophosphamide 1 Cycled every 21 days for 4 cycles followed by paclitaxel

Neoadjuvant endocrine therapy:

- Neoadjuvant endocrine therapy for estrogen receptor-positive LABC also holds great Promise- letrozole are more effective than tamoxifen. Presently it is always combined with neoadjuvant chemotherapy

Monitoring response to neoadjuvant chemotherapy:

- A significant response to the primary CT regimen is observed in about 80% of cases.
- Conventional modalities for assessing chemotherapy response, including clinical examination, mammogram, and breast ultrasound, are incorrect in identifying patient receiving CR patients in nearly half of cases.
- Breast MRI, PET ,and nuclear medicine sestamibi uptake scans are showing encouraging results but not yet routinely recommended.

Surgical: Breast conservation therapy and mastectomy
Treatment of the primary tumor is surgical.

Treatment of the remainder of the breast tissue for control of occult disease can be accomplished by either surgery or irradiation.

Criteria for BCT in post-neoadjuvant LABC:

- Patient desire for breast preservation
- Absence of multicentric disease (tumors in different quadrants of the breast)
- Absence of diffuse microcalcifications on mammogram
- Absence of skin involvement consistent with inflammatory breast cancer
- Residual tumor mass amenable to a margin-negative lumpectomy resection

Contraindications to breast-conservation treatment include

- Presence of two or more primary tumors in separate areas of the breast,
- Diffuse malignant-appearing microcalcifications,
- A history of prior therapeutic irradiation to the breast that precludes full-breast irradiation for the present condition,
- Active collagen vascular disease.

For women who are not well treated by breast-conserving therapy, mastectomy (MRM) is recommended.

Management of Regional Lymph Nodes

- Axillary lymph node metastases = regional manifestation of metastatic breast cancer.
- When mobile axillary lymph nodes contain tumor, an axillary dissection provides excellent local control of this axillary disease.
- If the axillary lymph nodes are involved, their removal accomplishes both goals: defining prognosis and diminishing the risk of subsequent axillary recurrence.
- Presently, Axillary dissection is preferred modality for treatment of axillary lymph node in LABC.

Postoperative systemic therapy

- Patients with hormone receptor-positive breast cancer should receive at least 5 years of either tamoxifen or an aromatase inhibitor.
  - Aromatase inhibitors should be given only to postmenopausal women, as these drugs do not block estrogen production from functioning ovaries.
  - Any woman of unknown menstrual status can have ovarian function assessed by measurement of serum follicle-stimulating hormone (FSH), luteinizing hormone (LH), and estradiol levels.
  - The role of ovarian ablation/suppression for premenopausal, hormonereceptor-positive breast cancer patients is not yet defined.
  - Tumors over-expressing HER2/neu also require treatment with adjuvant trastuzamab.

Or

b) A 50 year old gentleman presented with painless haematuria. What may be the possible causes? How would you investigate the case? Give an outline of the management.

5+5+5 = 15

Answer. Causes of painless haematuria:

Causes	Diagnosis	Treatment
Renal Tumours: The commonest primary renal tumour is **renal cell carcinoma, an adenocarcinoma** of collecting tubule origin. It commonly presents with haematuria.	By ultrasound scanning. Diagnosis is made by CT scanning	Treatment is by surgical excision
Transitional Cell carcinoma of the renal collecting system usually gives haematuria.	Diagnosis may be difficult, requiring retrograde imaging and ureteroscopy.	Treatment is by either local excision or, for high grade or larger lesions, nephro-ureterectomy. Immunotherapy is used for metastases with limited success; radiotherapy has little place except for palliation of bone metastases.
Angiomyolipoma is a hamartomatous lesion, which may grow to great size and be associated with major haemorrhage;.	CECT.	Treatment is by radiological emobolisation or surgery, conserving normal renal tissue where possible
Stone disease:	IVU	ESWL PCNL Open surgery
Glomerulonephritis		

Pyelonephritis (ascending urinary tract infection)		
Papillary Necrosis: This condition occurs in diabetics and in patients with deficiencies of oxygenation, particularly sickle cell disease.		
Ureteric stones	IVU	ESWL Ureterscopic retrieval
Cystitis: Typically cystitis is painful and in men is commonly associated with bladder outflow obstruction. Schistosomiasis, interstitial cystitis and drug related cystitis are rarer causes of bladder inflammation causing bleeding.	Diagnosis is by urine microscopy and culture, assisted by cystoscopy. Bladder biopsies may be necessary.	
Bladder tumour	Biopsy CECT MRI	TURBT Intravesical chemotherapy Chemotherapy Cystectomy
BHP **Postatic adenocarcinoma**	USG TRUS – Biopsy PSA	TURP Surgery Hormone therapy
<u>Rare causes of haematuria</u> **Arteriovenous malformations,** **Tuberculosis,** **Arteritis,** **Patients on anticoagulant therapy**		

Investigations of Haematuria: A general physical examination (blood pressure, the prostate in a male and the gynaecological organs in a female),

- First investigation in a patient with reported haematuria is urinary examination.

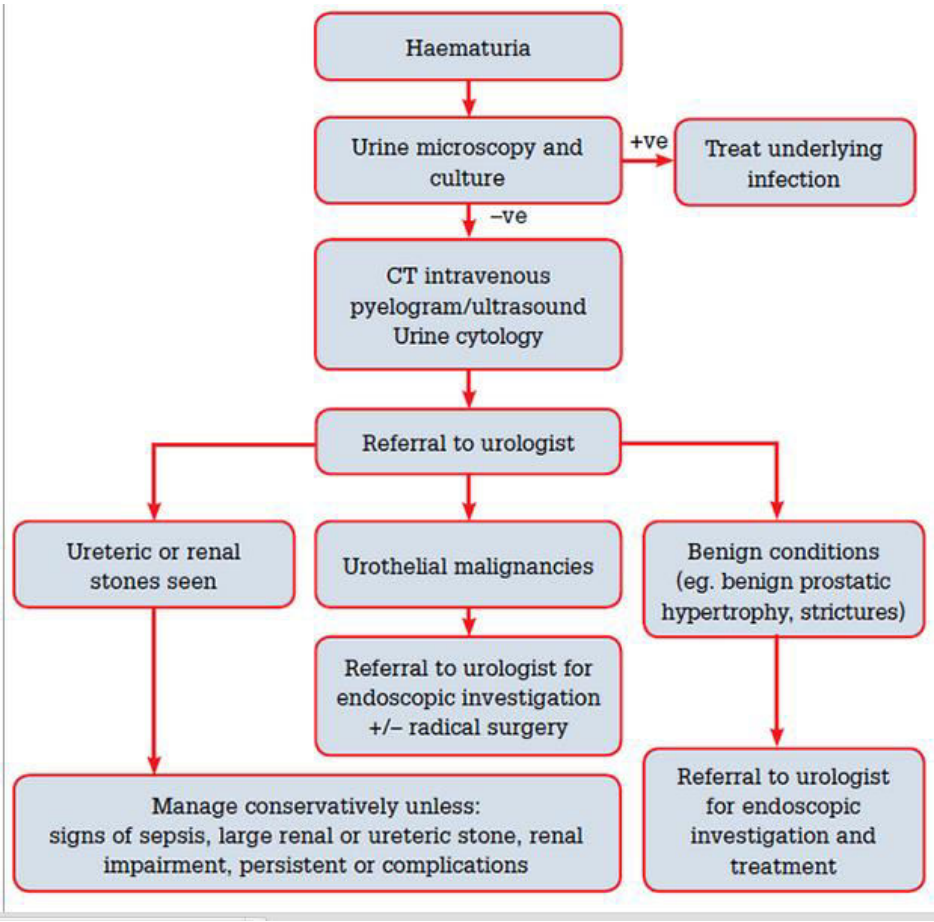
This must include microscopy for red and white blood cells and bacteria.
The presence of any crystals, ova or parasites should be noted and culture of a mid stream specimen carried out.
If schistosomiasis or tuberculosis is suspected a first void urine sample is usually needed.
The level of protein in the urine must be assessed, but in heavy haematuria it may be difficult to be sure if light proteinuria is due to the haemoglobin present.
If no red blood cells are found in the urine but haemoglobin is present the patient should be investigated for causes of haemoglobinuria

- All patients should have a full blood count with an erythrocyte sedimentation rate.
- Serum urea, creatinine and electrolytes should be measured, along with albumin, calcium and liver function tests if the patient is unwell or in renal failure.
- The presence or absence of proteinuria may guide initial investigation, since the combination of these favour a glomerular problem. Here C reactive protein and24 hour urine protein excretion will be informative, as may serum electrophoresis and autoantibodies.
- Ultrasound will show renal cortical thickness and density.
- In the majority of cases a renal biopsy with immunoglobulin histochemistry will be necessary to make a definitive diagnosis.
- In cases of microscopic haematuria without proteinuria, and all macroscopic cases, a "surgical" investigation plan can be followed.
- Other imaging may be done either by intravenous urography or a combination of plain abdominopelvic radiography and ultrasound of the urinary tract.
- If no abnormality is found then a flexible cystoscopy under local anaesthetic may be performed,
- Bladder lesion will require a transurethral biopsy and examination under anaesthetic for both treatment and diagnosis.

Management:

Approach to haematuria:

- Thorough history including urinary symptoms
- Recent history (trauma/muscle injury/causes of factitious haematuria/exexrcise/foreign travel)
- Systemic features (fever, weight loss) other symptoms (bleeding, bruising)
- Co-morbidity
- Drug history
- Occupation
- Family history



Conventional urological investigation involves urine culture (where, on the basis of associated cystitis, symptoms urinary infection is suspected), urine cytology, cystoscopy, renal ultrasonography, and IVU.

Diagnostic cystoscopy:

Nowadays this is carried out using a flexible, fibreoptic cystoscope, unless radiological investigation demonstrates a bladder cancer, in which case one may forego the flexible cystoscopy and proceed immediately to rigid cystoscopy and biopsy under anaesthetic (transurethral resection of bladder tumourTURBT).

Treatment:

General measures:

- Haemodynamically stabilise the patient.

- Assess for anaemia: if required blood transfusion after admission.
- If gross haematuria: foley's catheterisation.
- Antibiotics.
- Urinary tract infection should be ruled out before any further investigations for haematuria are undertaken.
- Ureteric and renal stones can cause episodes of haematuria, however it is important to consider other causes if this does not settle or if there are risk factors for urinary tract malignancy.
- Initial investigations for haematuria should include CT-IVP, urine cytology, full blood examination, renal function, and PSA in men.
- A urological referral is recommended in patients presenting with macroscopic haematuria, persistent microscopic haematuria, abnormal urine cytology, irritative lower urinary tract symptoms or recurrent urinary tract infections.
- Find out the cause.
- Treat the cause.

Group – C

3. Write short notes on (any three) **3x5 = 15**

a) **Cleft lip.**
b) **Thyroglossal cyst.**
c) **Spinal anaesthesia.**
d) **Types of skin graft.**
e) **Role of ERCP in obstructive jaundice.**

Answers

Before answering the question of cleft lip please go through the following.

EMBRYOLOGY

Facial development takes place between 4^{th} -8^{th} weeks of intrauterine life. The face has a clearly human appearance by the 10^{th} week. The boundaries of the primitive mouth (stomodeum) are the paired mandibular prominences inferiorly, paired maxillary prominences (MxPs) laterally and the frontonasal prominence (FNP) cranially. These five facial prominences in a human embryo form the adult facial features. Bilateral ectodermal thickenings called nasal placodes develop on the inferolateral aspect of the FNP by the 4^{th} week. Elevation of the margins of the FNP leads to development of median nasal prominence (MNP) and the lateral nasal prominence (LNP) on each side. The depressed central region of the nasal placode is called the nasal pit which forms the external nares.

Development of Upper lip: The MxPs migrate medially and fuse first with the LNP and then with the MNP. The LNP and the MNP also fuse with each other, thus the external nares get cut off from the stomodeum. Merging of the two MNP's with each other results in the development of the globular process which forms the philtrum & the Cupid's bow region of the upper lip, premaxilla, primary palate, nasal tip and the nasal septum. The LNP's form the nasal alae. The lateral portions of the upper lip are formed from the maxillary prominences from each side.

Cleft Lip: A unilateral CL results from failure of fusion of the MNP and the MxP on one side. A bilateral CL occurs due to failure of fusion of the MxP on either side with the globular process (merged MNP's). The globular process which normally contributes to development of philtrum forms a wide short disc called prolabium. The MNP's are thus not restrained by the lateral attachment to the MxPs. This manifests as complete bilateral CL with anterior overprojection of premaxilla & prolabium. The clefts and the projection vary in degree from case to case. Failure of fusion of MNP's in the midline results in median CL. A variation in the normal sequence has been proposed to account for the development of incomplete cleft lips. The initial MxP-MNP fusion remains intact during the early stages of cleft formation, but the MxP becomes somewhat disconnected with MNP during its later forward growth resulting in an incomplete cleft. The Simonart's band (see below) may represent the persistence of initial MxP-MNP fusion.

Development of the Palate: From each MxP, a plate like lateral palatine process grows medially. Simultaneously, a median palatine process forms from the posterior aspect of the merged MNP's. All three elements are initially widely separated due to the vertical orientation of the lateral palatine processes which are located on either side of the tongue. During the 8th week of IUL, the orientation of the lateral palatine processes alters from vertical to horizontal to initiate their fusion. The medial edge epithelium of the palatal shelves degenerates in a process called 'programmed cell death' permitting mesenchymal coalescence of the palatal shelves in the midline. Fusion also occurs between the lateral palatine processes and the posterior margin of the median palatine process. The fusion of these three elements begins at the junction of the lateral palatine processes with the median palatine process in the midline (from the point of the incisive foramen) and proceeds both posteriorly towards uvula and anteriorly towards alveolus. The medial edge of the lateral palatine processes also fuses with the free lower edge of the nasal septum, thus separating the two nasal cavities from each other and from the mouth.

The portion derived from the median palatine process forms the premaxilla which carries the four incisors, and is called the primary palate; the lateral palatine processes form the secondary palate. Ossification occurs in the primary palate and the anterior portion of the secondary palate to form the hard palate. The posterior portion of the secondary palate does not undergo ossification and forms the soft palate. A palatine raphe in the adult palate indicates the line of fusion of the lateral palatine processes.

Cleft palate: Clefts of the primary palate occur anterior to the incisive foramen and result from the failure of the mesenchymal masses in the lateral palatine process(es) to fuse with those in the median palatine process. Clefts of the secondary palate occur posterior to the incisive foramen and result from the failure of the mesenchymal masses in the lateral palatine processes to fuse with each other and with the nasal septum. Clefting of either primary or secondary palate can be either unilateral or bilateral, complete or incomplete (depending on the degree of fusion that occurred during the embryonic development). In addition to overt clefts of the palate, anomalies of the mesenchymal merging of the palatal

shelves can result in submucous cleft palate (SMCP). There occurs imperfect muscle union (muscular diastasis) across the soft palate with an intact mucosal surface (zona pellucida), bifid uvula and a notched posterior hard palate.

a) Cleft lip: Clefts of the lip (CL) and alveolus with or without cleft of palate (CL/P) are the most common congenital craniofacial defects and the fourth most frequent birth defects in the world.

Clefts of the lip may be

- Unilateral or bilateral,
- Complete or incomplete,
- Right or left sided and
- May occur with or without cleft palate.

They frequently occur as isolated anomalies but may be associated with syndromes such as

• Treacher Collins syndrome, • Down syndrome, • Apert syndrome,	• Goldenhar syndrome, • Pierre Robin sequence, • Van der Woude syndrome

Risk factors:

- **Family history:** Parents with a family history of cleft lip or cleft palate face a higher risk of having a baby with a cleft.
- **Race**: Common in Native Americans and least common in African-Americans.
- **Sex:** Males are twice as likely to have a cleft lip with or without cleft palate. Cleft palate without cleft lip is more common in females.
- **Exposure to certain substances during pregnancy:** Cleft lip and cleft palate may be more likely to occur in pregnant women who smoke cigarettes, drink alcohol or take certain medications.
- **Having diabetes:** Women diagnosed with diabetes before pregnancy may have an increased risk of having a baby with a cleft lip with or without a cleft palate.
- **Being obese during pregnancy.** There is some evidence that babies born to obese women may have increased risk of cleft lip and palate.

Classification:

Group I: Cleft lip only (Pre-alveolar clefts)
Group Ia: Cleft of lip and alveolus
Group II: Cleft palate only (Post alveolar clefts)
Group III: Cleft of lip, alveolus and palate (Alveolar clefts)
Group I and III clefts are subdivided into unilateral / bilateral / median, and Group II is subdivided into clefts of hard and soft palates.

Complications:

- Difficulty with feedings
- Difficulty swallowing, with potential for liquids or foods to come out the nose

- Nasal speaking voice

Diagnosis:

Traditionally, the diagnosis is made at the time of birth by physical examination. Recent advances in prenatal diagnosishave allowed obstetricians to diagnose facial clefts in utero.

Treatment:

- Within the first 2–3 months after birth, surgery is performed to close the cleft lip.
- While surgery to repair a cleft lip can be performed soon after birth, often the preferred age is at approximately 10 weeks of age, following the "rule of 10s" coined by surgeons
- If the cleft is bilateral and extensive, two surgeries may be required to close the cleft, one side first, and the second side a few weeks later. The most common procedure to repair a cleft lip is the **Millard procedure.**

b) Thyroglossal cyst

Definition: Thyroglossal cyst is a fluid-filled sac resulting from incomplete closure of the thyroglossal duct.

Anatomy: The thyroglossal duct arises embryologically between the first and second pharyngeal pouches. It runs as a hollow tube from the foramen caecum on the dorsal surface of the tongue, becoming a solid cord of cells migrating through the tongue and into the midline of the neck. The tract usually passes in front of the hyoid bone and then loops up behind it before descending in the midline of the neck where the cells divide to form the two lobes of the thyroid gland either side of the midline. The duct normally atrophies in the sixth week of gestation.

Clinical features:

• Usually presents in children or young adults.	• 75% appear in front of the hyoid bone and the majority of the rest at the level of the thyroid or cricoid cartilage of the larynx.	• 5% become infected presenting as a painful, red neck swelling.
• 90% present as a painless midline cyst.	• The cyst is mobile and moves up on swallowing and protrusion of tongue.	• 15% have a fistula to the skin (due to infection or incomplete excision).
• 10% appear on one side of the midline, usually the left.	• If large enough it will transilluminate.	• Papillary carcinoma of the thyroglossal ductal cells is rare. Treatment is by excision.

Diagnosis and investigations:

- CT scan - often reveal a well circumscribed cyst related to the midline of the hyoid bone.
- Fine-needle aspiration may reveal a cloudy infected fluid or a straw-coloured fluid.

Treatment:

Infected thyroglossal cyst:

- Majority respond to antibiotics.
- Surgical drainage if abscess formed or failure to respond to antibiotics.
- Elective excision of the cyst once acute infection has resolved.

Surgery

- Excision is recommended for most cysts.
- Remove through a transverse midline incision in a skin crease.
- Divide the platysma muscle and dissect the cyst out bluntly.
- On the deep surface it will be found to be attached to the hyoid bone: excise approximately 1cm of the bone, removing any underlying thyroglossal duct epithelium.
- Close the wound in layers with a suction drain.
- If there is a fistula or sinus in the neck excise it through a transverse elliptical incision. Again use blunt dissection and remove the middle part of the hyoid bone (Sistruck procedure).

c) Spinal anaesthesia

Spinal anaesthesia (or **spinal anesthesia**), also called **spinal** analgesia,**spinal block** or subarachnoid **block** (SAB), is a form of regional **anaesthesia** involving injection of a local **anaesthetic** into the subarachnoid space, generally through a fine needle.

Mechanism:

Regardless of the anaesthetic agent (drug) used, the desired effect is to block the transmission of afferent nerve signals from peripheral nociceptors. Sensory signals from the site are blocked, thereby eliminating pain. The degree of neuronal blockade depends on the amount and concentration of local anaesthetic used and the properties of the axon. Thin unmyelinated C-fibres associated with pain are blocked first, while thick, heavily myelinated A-alpha motor neurons are blocked moderately. Heavily myelinated, small preganglionic sympathetic fibers are blocked first. The desired result is total numbness of the area. A pressure sensation is permissible and often occurs due to incomplete blockade of the thicker A-beta mechanoreceptors. This

allows surgical procedures to be performed with no painful sensation to the person undergoing the procedure.

Indications	Contraindications	Complications
• Orthopaedic surgery on the pelvis, femur, tibia and the ankle • Hip replacement • Knee replacement • Hip fracture surgery • Spinal opioids e.g. Diamorphine 500-1000mcg along with general anaesthesia for post operative analgesia in laparoscopic bowel surgery • Lower limb vascular surgery • Endovascular aortic aneurysm repair • Hernia (inguinal or epigastric) • Haemorrhoidectomy (Piles), fistulae and fissures • Nephrectomy and Cystectomy in combination with general anaesthesia • Transurethral resection of the prostate and transurethral resection of bladder tumours • Abdominal and vaginal hysterectomies • Laparoscopy assisted vaginal hysterectomies combined with general anaesthesia • Caesarean sections	• Non-availability of patient's consent • Local infection or sepsis at the site of lumbar puncture • Bleeding disorders, thrombocytopaenia, or systemic anticoagulation (secondary to an increased risk of a spinal epidural hematoma) • Space occupying lesions of the brain • Anatomical disorders of the spine • Hypovolaemia e.g. following massive haemorrhage, including in obstetric patients	• Hypotension (Spinal shock) - Due to sympathetic nervous system blockade. Common but usually easily treated with intravenous fluid and sympathomimmetic drugs such as Ephedrine, Phenylephrine or Metaraminol • Post dural puncture head ache (PDPH) or post spinal head ache - Associated with the size and type of spinal needle used • Cauda equina injury - very rare, due to the insertion site being too high • Cardiac arrest - very rare. • Spinal canal haematoma, with or without subsequent neurological sequelae due to compression of the spinal nerves • Epidural abscess - May present as meningitis or and abscess with back pain, fever, lower limb neurological impairment and loss of bladder/bowel function.

d) Types of skin graft

Graft taxonomy:

- **Autologous**: The donor skin is taken from a different site on the same individual's body (also known as an autograft).
- **Isogeneic**: The donor and recipient individuals are genetically identical (e.g., monozygotic twins, animals of a single inbred strain; isograft or syngraft).

- **Allogeneic**: The donor and recipient are of the same species (human→human, dog→dog; allograft).
- **Xenogeneic**: The donor and recipient are of different species (e.g., bovine cartilage; xenograft or heterograft).
- **Prosthetic**: Lost tissue is replaced with synthetic materials such as metal, plastic, or ceramic (prosthetic implants).

Graft classification:

- **Split-thickness:** A split-thickness skin graft (STSG) is a skin graft including the epidermis and part of the dermis. Its thickness depends on the donor site and the needs of the patient. It can be processed through a skin mesher which makes apertures onto the graft, allowing it to expand up to nine times its size. Split-thickness grafts are frequently used as they can cover large areas and the rate of autorejection is low. The same site can be harvested again after six weeks. The donor site heals by re-epithelialisation from the dermis and surrounding skin and requires dressings.
- **Full-thickness:** A full-thickness skin graft consists of the epidermis and the entire thickness of the dermis. The donor site is either sutured closed directly or covered by a split-thickness skin graft.
- **Composite graft:** A composite graft is a small graft containing skin and underlying cartilage or other tissue. Donor sites include, for example, ear skin and cartilage to reconstruct nasal alar rim defects.

e) Role of ERCP in obstructive jaundice.

Causes of obstructive jaundice:

- Intraluminal abnormalities of bile ducts:
  - Gallstones;
  - Blood clot;
  - Parasites (e.g. flukes).
- Mural abnormalities of bile ducts:
  - Cholangiocarcinoma;
  - Congenital atresia;
  - Choledochal cyst.
  - Sclerosing cholangitis;
  - Biliary cirrhosis (primary (autoimmune) or secondary to sepsis);
  - Traumatic/post surgical stricture.
- Extrinsic compression of bile ducts:
  - Pancreatitis;
  - Tumours, e.g. head of pancreas, ampulla of Vater;
  - Lymphadenopathy of porta hepatis nodes.

ERCP can be done to know the cause of obstructive jaundice as well as to treat the cause in some cases.

Role of ERCP in obstructive jaundice:

Disease	Diagnostic	Thearapeutic
Choledocholithiasis	o CBD stone can be detected. o If stones are not found, bile can be collected to test for microlithiasis if clinically appropriate.	Endoscopic sphincterotomy and stone extraction.
Choledochal cyst	o Endoscopic retrograde cholangiography (ERC) is typically performed on adults and larger children. o Cholangiography can demonstrate areas of cystic dilatation, the presence of stones, and excludes complete obstruction of the bile duct. It is also effective in demonstrating the presence of pancreaticobiliary maljunction.	
Biliary stricture	o Can delineate the level of stricture	Stenting
Cholangiocarcinoma	o Distal tumors are assessed by endoscopic retrograde cholangiopancreatography (ERCP) o Endoscopic brush biopsy can be taken.	Palliative treatment – by stenting.
Acute Pancreatitis	o Can detect acute biliary pancreatitis with biliary obstruction or cholangitis.	ERCP and papillotomy
Chronic pancreatitis	o ERCP has been considered the gold standard for the morphologic diagnosis of the chronic pancreatitis.	
Carcinoma head of pancreas Periampullary carcinoma	o ERCP sometimes is required to solidify the diagnosis of pancreatic cancer. The classic findings of a long, irregular stricture in a pancreatic duct with distal dilation or a "double duct sign" in which there is cutoff of both the pancreatic duct and distal bile duct at the level of the genu of the pancreatic duct are pathognomonic	Palliative stenting.

Group – D

4) Answer briefly on (any three): 3x5 = 15

a) Oral submucosal fibrosis.
b) Wax bath.
c) Subdural haematoma.
d) Intussusceptions.
e) Marjolin's ulcer.

Answers

a) Oral submucosal fibrosis.

Introduction: Oral submucous fibrosis is a progressive disease in which fibrous bands form beneath the oral mucosa. It is a precancerous lesion.
Scarring produces con tracture, resulting in limited mouth opening and restricted tongue movement.

Aetiology: The condition is almost entirely confined to the Asian population .

Pathology: It is characterised pathologically by epithelial fibrosis with associated atrophy and hyperplasia of the overlying epithelium.
The epithelium also shows changes of epithelial dysplasia.

Causes: Dried products such as paan masala and gutkha have higher concentrations of areca nut and appear to cause the disease.

Other causes include:

- Immunological diseases
- Extreme climatic conditions
- Prolonged deficiency to iron and vitamins in the diet

Clinical features: In the initial phase of the disease, the mucosa feels leathery with palpable fibrotic bands. In the advanced stage the oral mucosa loses its resiliency and becomes blanched and stiff. The disease is believed to begin in the posterior part of the oral cavity and gradually spread outward.

Other features of the disease include:

Xerostomia	Pain in the ear or deafness	Restriction of the movement of the soft palate	Thinning and stiffening of the lips	Dryness of the mouth and burning sensation
Recurrent ulceration	Nasal intonation of voice	A budlike shrunken uvula	Pigmentation of the oral mucosa	Decreased mouth opening and tongue protrusion

Treatment includes:

- Abstention from chewing areca nut (also known as betel nut) and tobacco

- Minimizing consumption of spicy foods, including chiles
- Maintaining proper oral hygiene
- Supplementing the diet with foods rich in vitamins A, B complex, and C and iron
- Forgoing hot fluids like tea, coffee
- Forgoing alcohol
- Employing a dental surgeon to round off sharp teeth and extract third molars
- Restricted mouth opening can be treated with either intralesional steroids or surgical excision and skin grafts.

Treatment also includes following:

• Chewable pellets of hydrocortisone.	• Surgical treatment is recommended in cases of progressive fibrosis when interincisor distance becomes less than 2 centimetres .
• Intralesional injection Hyaluronidase.	• Pentoxifylline, IFN-gamma, Colchicine tablets, Lycopene.
• Submucosal injections of hydrocortisone.	• Recently scientists have proven that intralesional injection of autologous bone marrow stem cells is a safe and effective treatment modality in oral sub mucosal fibrosis.

b) Wax bath.

Introduction: A wax bath is a treatment that involves the submersion of hands, feet, or elbows into a container with melted paraffin wax to relieve pain, treat or prevent muscle injury, or deep clean and moisturize the skin.

Method of application: Hot wax bath treatment involves specially formulated wax that is heated in a container. A regulator maintains a safe temperature for the skin. Once the wax is melted, a hand, foot, or elbow is dipped in the wax, removed, and allowed to air-dry for two minutes, with the procedure repeated for five to ten minutes. After there are enough wax layers, the hand, foot, or elbow is wrapped in plastic to protect clothing and furniture and left to stand for 15 minutes. Once hardened, the wax is ready to peel off.

Application:

- Commonly used to relieve pain in arthritis, muscle injury, and rheumatism,
- Wax bath therapy can also relieve conditions such as inflammation, fibromyalgia, eczema, bursitis, psoriasis, and tendinitis.

c) Subdural haematoma.

Introduction: A subdural hematoma or subdural haematoma, also known as a subdural hemorrhage (SDH), is a type of hematoma, a form of traumatic brain injury. Blood gathers within the outermost meningeal layer, between the dura mater, which adheres to the skull, and the arachnoid mater, which envelops the brain.

Usually resulting from tears in bridging veins which cross the subdural space, subdural hemorrhages may cause an increase in intracranial pressure (ICP), which can cause compression of and damage to delicate brain tissue.

Subdural hematomas are often life-threatening when acute.

Chronic subdural hematomas, however, have better prognosis if properly managed.

Classification: Subdural hematomas are divided into acute, sub-acute, and chronic, depending on their speed of onset.

Acute subdural hematomas that are due to trauma are the most lethal of all head injuries and have a high mortality rate if they are not rapidly treated with surgical decompression.

Clinical features: Symptoms of subdural hemorrhage have a slower onset than those of epidural hemorrhages because the lower pressure veins bleed more slowly than arteries.

Therefore, signs and symptoms may show up in minutes, if not immediately but can be delayed as much as 2 weeks.

If the bleeds are large enough to put pressure on the brain, signs of increased ICP or damage to part of the brain will be present.

Other signs and symptoms of subdural hematoma can include any combination of the following:

A history of recent head injury	Numbness	Weakness or lethargy	Ataxia, or difficulty walking
Loss of consciousness or fluctuating levels of consciousness	Headache (either constant or fluctuating)	Nausea or vomiting	Altered breathing patterns
Irritability	Dizziness	Loss of appetite	Hearing loss or hearing ringing (tinnitus)
Seizures	Disorientation	Personality changes	Blurred Vision
Pain	Amnesia	Inability to speak or slurred speech	Deviated gaze, or abnormal movement of the eyes

Pathophysiology: Collected blood from the subdural bleed may draw in water due to osmosis, causing it to expand, which may compress brain tissue and cause new bleeds by tearing other blood vessels. The collected blood may even develop its own membrane.

Substances that cause vasoconstriction may be released from the collected material in a subdural hematoma, causing further ischemia under the site by restricting blood flow to the brain. When the brain is denied adequate blood flow, a biochemical cascade known as the ischemic cascade is unleashed, and may ultimately lead to brain cell death.

The body gradually reabsorbs the clot and replaces it with granulation tissue.

Diagnosis:

Subdural hematomas occur most often around the tops and sides of the frontal and parietal lobes.They also occur in the posterior cranial fossa, and near the falx cerebri and tentorium cerebelli. Unlike epidural hematomas, which cannot expand past the sutures of the skull, subdural hematomas can expand along the inside of the skull, creating a concave shape that follows the curve of the brain, stopping only at thedural reflections like the tentorium cerebelli and falx cerebri.

On a CT scan, subdural hematomas are classically crescent-shaped, with a concave surface away from the skull. However, they can have a convex appearance, especially in the early stage of bleeding.

Treatment: It is important that a patient receive medical assessment, including a complete neurological examination, after any head trauma. A CT scan or MRI scan will usually detect significant subdural hematomas.

Treatment of a subdural hematoma depends on its size and rate of growth.

Some small subdural hematomas can be managed by careful monitoring until the body heals itself.

Other small subdural hematomas can be managed by inserting a temporary small catheter through a hole drilled through the skull and sucking out the hematoma; this procedure can be done at the bedside.

Large or symptomatic hematomas require a craniotomy, the surgical opening of the skull.

READ THE TABLE BELOW:

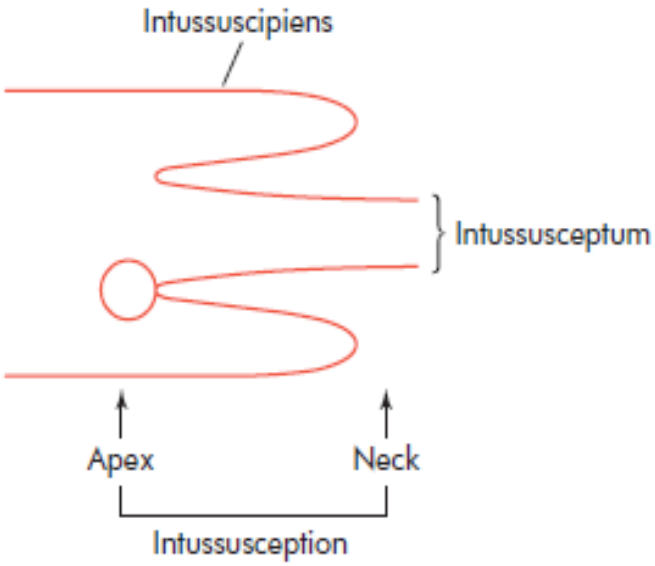
Hematoma type	Epidural	Subdural
Location	Between the skull and the dura	Between the dura and the arachnoid

Involved vessel	Temperoparietal locus (most likely) - Middle meningeal artery Frontal locus - anterior ethmoidal artery Occipital locus - transverse or sigmoid sinuses Vertex locus - superior sagittal sinus	Bridging veins
Symptoms	Lucid interval followed by unconsciousness	Gradually increasing headache and confusion
Appearance on CT	Biconvex lens	Crescent-shaped

d) Intussusceptions.

Introduction:

- Incidence approximately 2 per 1000 live births.
- Peak age of presentation at 3-10 months.
- M:F = 2:1.
- Fewer than 10% have a clear focal pathological cause that starts the intussusception (Apex: older children are more likely to have an apex).



Types:

○ Ileoileal	○ Ileocolic – most common.	○ Ileoileocolic	○ Colocolic	○ Multiple	○ Retrograde	○ Others

Clinicopathological features:

- Invagination/telescoping of the proximal bowel (called the intussusceptum, e.g. terminal ileum/ileo-caecal valve) into the distal bowel (called the intussuscepiens, e.g. caecum/ascending colon).
- May be due to enlargement of lymphatic patches of Peyer (idiopathic)
- Pathology at the apex may be:
    - Meckel's diverticulum;
    - polyp;
    - lymphoma.

Clinical features:

- Classic triad of features is:
    - abdominal pain (associated with pallor, screaming, and restlessness);
    - palpable sausage-shaped mass (mid-abdominal or right upper quadrant);
    - passage of red-currant jelly stool. (Rectal examination may reveal bloody mucus and the lead point may rarely be palpable.)
- Typically the infant is relatively settled between bouts of pain.
- Signs of shock (lethargy, poor feeding, hypotonia) require urgent fluid resuscitation.
- Features of obstruction (distension and vomiting) may occur.

Diagnosis and investigations:

- Ultrasound (diagnostic text of choice): intussusception in cross-section (doughnut or target sign).
- Plain X-ray: may show soft tissue mass, small bowel obstruction, free air indicating perforation.
- Air (or rarely gastrograffin) contrast enema is diagnostic and may be therapeutic.

Treatment:

- Immediate IV fluid resuscitation to correct fluid losses and to restore fluid, electrolyte, and acid/base balance.
- Maintenance fluid replacement and replacement of continued losses (vomiting or nasogastric losses). Reduction only attempted once fluid balance restored.

- Analgesia and sedation (morphine 0.2mg/kg) will aid process of reduction.

Methods of reduction:

Radiological reduction

- Air enema therapeutic in 75% of cases.
- Usually performed in radiology department under screening control.
- Surgeon should be present.
- Evidence of irreducible obstruction or perforation mandates immediate halt.
- Partial or incomplete reduction may warrant repeat attempt after 4-6 hours.
- Informed consent includes risk of peforation.

Surgical reduction:

- Laparotomy indicated without enema if evidence of peritonitis or perforation.
- Manual reduction by retrograde squeezing and gentle proximal traction.
- Resection and anastomosis if bowel viability is in doubt (~10% require resection).
- Post-reduction septic shock may occur with release of bacterial products from viable but damaged bowel segment.
- Most recover rapidly with resumption of oral feeding in 24-48h and discharge home in 4-5 days.

Complications:

- Recurrence rate is 5-7% in non-operative cases, and about 3% for operative reduction.
- Morbidity is low but delayed diagnosis, inadequate resuscitation, and failure to recognize ischaemic or perforated bowel account for 1% mortality.

e) Marjolin's ulcer.

Introduction: Marjolin's ulcer refers to an aggressive ulcerating squamous cell carcinomapresenting in an area of previously traumatized, chronically inflamed, or scarred skin. They are commonly present in the context of chronic wounds including burn injuries, venous ulcers, ulcers from osteomyelitis, and post radiotherapy scars.

Appearance: Slow growth, painlessness (as the ulcer is usually not associated with nerve tissue), and absence of lymphatic spread due to local destruction of lymphatic channels.

Characteristic: Histologically, the tumour is a well-differentiated squamous cell carcinoma. This carcinoma is aggressive in nature, spreads locally and is associated with a poor prognosis. 40% occur on the lower limb and the malignant change is usually painless.

This malignant change of the wound happens a long time after initial trauma, usually 10–25 years later. Its edge is everted and not always raised.

Diagnosis: Wedge biopsy is the favored method of diagnosis. Tissue specimens obtained should be taken from both the centre and margin of lesion, as the central ulcerated deposits may be necrotic.

Treatment: Treatment is usually surgical, with a wide excision of the lesion; typically a 1 cm margin all around is required. Radiation therapy is also a good alternative in most cases.

The West Bengal University of Health Sciences

M.B.B.S. 3rd Professional Part – II Examination, 2012

Subject: Surgery **Time: $2^{1}/_{2}$ hrs.**

Paper: II **Marks: 60**

Group –A

1) a) Classify Thyroid Neoplasms. Write clinical features, investigations and management of papillary carcinoma of the thyroid gland (a lady of 25 years old). 3+4+3+5=15

Answer. Classification of thyroid neoplasms

Benign - Follicular adenoma

Malignant - Primary

- Follicular epithelium –differentiated
  - Follicular
  - Papillary
- Follicular epithelium –undifferentiated
  - Anaplastic
- Parafollicular cells
  - Medullary
- Lymphoid cells

Lymphoma

Secondary

- Metastatic
- Local infiltration

Clinical features of papillary carcinoma thyroid:

i. Papillary thyroid carcinoma (PTC) represents 85% of thyroid carcinomas. PTC is often multifocal.
ii. The most common presenting symptom is a thyroid swelling.
iii. Papillary cancer tends to spread via the lymphatics with a known propensity for involvement of the mid- and lower-anterior cervical lymph nodes.
iv. Enlarged cervical lymph nodes may be the presentation of papillary carcinoma.
v. Invasion of adjacent structures and distant metastases at the time of presentation are uncommon.
vi. Recurrent laryngeal nerve paralysis is very suggestive of locally advanced disease.

Diagnosis: Essentials of Diagnosis

- History of irradiation to the neck in some patients.
- Painless or enlarging nodule, dysphagia, or hoarseness.
- Firm or hard, fixed thyroid nodule; ipsilateral cervical lymphadenopathy.

- Normal thyroid function; nodule stippled with microcalcifications and solid (ultrasound), cold (radioiodine scan); positive or suspicious cytology.
- Family history of thyroid cancer.

Investigations:

- Thyroid function test
- USG guided FNAC: Ultrasonography and radionuclide thyroid scans cannot distinguish benign from malignant nodules and therefore are not essential in the workup of a thyroid nodule. Ultrasonography is commonly used to direct FNA biopsy and is a sensitive method for determining whether a lesion is solid or cystic.
- CT/USG of neck.
- Indirect laryngoscopy.

Pathology:

- Microscopically, it is composed of papillary projections of columnar epithelium.
- Psammoma bodies are present in about 60% of cases.
- Mixed papillary-follicular, follicular variants of papillary carcinoma, and poorly differentiated cancers including tall cell and columnar cell papillary thyroid cancers are sometimes found.
- The rate of growth may be stimulated by TSH.
- A *BRAF* mutation is the most common mutation in papillary thyroid cancer and is associated with lymph node metastases and a higher recurrence rate.

Treatment: Low-Versus High-Risk Criteria in Papillary Thyroid Cancer

Low risk	High risk
1. Women < 50 years 2. Men < 40 years 3. Well-or moderately differentiated tumors 4. Tumor < 4 cm in diameter 5. Tumor confined to the thyroid gland 6. No distant metastases	1. Women ≥ 50 yr 2. Men ≥ 40 yr 3. Poorly differentiated tumors, tall-cell, columnar cell, or oxyphilic variants. 4. Tumor ≥ 4 cm in diameter 5. Local invasion 6. Distant metastases

Surgical treatment:

- A lobectomy and isthmusectomy is satisfactory for papillary cancers less than 1.0 cm in size without lymphatic or systemic metastases.
- Lobectomy with isthmusectomy - for patients with papillary cancers that are greater than 1.0 cm when patients are categorized as low risk for recurrence or mortality.
- Total thyroidectomy is uniformly accepted for treatment of high-risk papillary carcinoma.
- In addition, patients with a history of head and neck irradiation should undergo total thyroidectomy due to the high incidence of carcinoma at sites other than the nodule and their increased lifetime risk of developing thyroid cancer in the remaining thyroid gland.

Role of post operative remnant ablation:

Indications:

• Stage III and IV disease
• All patients with stage II disease younger than age 45 years
• Most patients with stage II disease 45 years or older
• Selected patients with stage I disease - especially those with multifocal disease, nodal metastases, extra-thyroidal or vascular invasion, and/or more aggressive histologies.

Protocol of thyroid hormone withdrawal for remnant ablation

Aim: Achieve TSH of more than 30 mU/L

- Single-dose exogenous rTSH results in TSH levels between 51–82 mU/L
- Endogenous TSH elevation can be achieved by
  - Stopping levothyroxine (LT4) and switching to levo-triiodothyronine (LT3) for 2–4 weeks followed by withdrawal of triiodothyronine (T3) for 2 weeks
  - Discontinuation of LT4 for 3 weeks without use of T3
  - rTSH stimulation can be used for remnant ablation.

Role of post operative diagnostic RAIU scan:

• Indication:
– When the extent of thyroid remnant cannot be assessed from the surgical report/ USG
– If the result is likely to alter the subsequent management
– If the result is likely to alter the dose of RAI treatment
• If used, low dose I 131 (1-3mCi)/ I123 scan recommended
– Low iodine diet (50microgram/day for 1-2 weeks) is recommended prior to scan
• Post therapy scan:
  - Detects additional foci in 10-26% cases, alters the stage in 10%
  - Done 1 week post radio-iodine therapy.

Role of TSH suppression therapy:

Mechanism of action:

  - Decreases the expression of thyroid specific proteins
  - Decreases the rate of cell growth

- High risk pts:
  - Keep TSH < 0.1mIU/L
  - After remission continue suppressive therapy x 3-5 yrs
- Low risk patients
  - TSH= 0.1-0.5 mIU/L
  - After remission keep it between 0.5-1mIU/mL

There is no role for routine adjunctive chemotherapy.

Or

b) A 40 years old gentleman presented with bilateral knobby renal lump in the abdomen. How do you investigate and treat such a patient (operation details not required)? 7+8=15

Answer. The differential diagnosis are:

- Bilateral hydronephrosis: **See the Question.1 of Group –A of Paper –II of 2013**
- Bilateral renal cell carcinoma: **See the Question 2.b of Group - B of Supplementary Paper - II of 2013**
- Adult polycystic kidney disease.

Special points for treatment of bilateral hydronephrosis:

Disorders associated with bilateral hydronephrosis include:

Acute bilateral obstructive uropathy	Neurogenic bladder	Retroperitoneal fibrosis
Bladder outlet obstruction	Posterior urethral valves	Uteropelvic junction obstruction
Chronic bilateral obstructive uropathy	Prune belly syndrome	Vesicoureteric reflux

The following tests can show bilateral hydronephrosis:

- CT scan of the abdomen or kidneys
- IVP
- Pregnancy (fetal) ultrasound
- Renal scan
- Ultrasound of the abdomen or kidneys

Treatment:

Placing a tube into the bladder (Foley catheter) may open the blockage.

Other treatments include:

- Draining the bladder
- Relieving pressure by placing tubes in the kidney through the skin
- Placing a tube (stent) through the ureter to allow urine to flow from the kidney to bladder

The underlying cause of the blockage needs to be found and treated once the buildup of urine is relieved.

Special points regarding management of bilateral RCC:

- The approach to the patient with bilateral RCC differs from the standard approach of radical nephrectomy.
- Bilateral RCC occurs with a frequency as high as 3%.
- Radical nephrectomy in these patients obviously commits patients to long-term dialysis or renal transplantation and the morbidities of these conditions.
- Staging these patients is essentially the same as previously outlined, with the notable exception that either MR or CT angiography is often used to assess the extent of tumor within the kidney and the renal artery anatomy.
- Surgical alternatives to radical nephrectomy include open or laparoscopic partial nephrectomy, ex vivo partial nephrectomy (bench surgery followed by autotransplantation
- Given the lack of effective adjuvant therapy and the risk of inadequate excision and subsequent recurrence from various renal sparing approaches, partial nephrectomy with an adequate parenchymal margin remains the preferred treatment.

<u>Adult polycystic kidney disease:</u>

Imaging:

- There are multiple cysts in both kidneys and sometimes cysts in the liver and other organs.
- Blood and debris in the cysts may mimic the heterogeneity of a cystic adenocarcinoma.\
- Cytologic examination of cyst fluid obtained by fine-needle aspiration will rule out cancer.
- The urogram is typical: the renal shadows are enlarged in all directions; the renal pelvis is compressed and elongated; and the calyces are stretched over the cysts.

Treatment:

- As kidney failure develops, a low-protein diet postpones the inevitability of renal replacement therapy.
- Infection, anaemia, hypertension and disturbances of calcium metabolism also need treatment.
- Surgery to uncap the cysts (Rovsing's operation) is rarely indicated.

Group – B

2) Write short notes on (any three): 3x5=15

a) **Breast biopsies.**
b) **Causes of haematuria.**
c) **Antegrade Pyelography.**
d) **Stress gastritis.**
e) **P.S.A.**

Answers

a) Breast biopsies:

Breast Biopsy techniques:

Fine needle aspiration Biopsy (FNAB):

The primary advantage of the FNAB is the simplicity of the technique, readily available and it is relatively atraumatic for the patient. Limitation of the FNAB is availability of a experienced cytopathologist for interpretation of the results. The major limitation of FNAB is the inability of the cytology to distinguish invasive cancer from in situ disease.

Core Needle Biopsy (CNB):
The Core needle Biospy (CNB) is performed by using automated gun attached to 14 / 18 gauge cutting needle. Tissue cores can be processed for complete pathologic assessment. Sensitivity rate for CNB are almost 100% for diagnosing the breast lesions.

FNAC / core biopsy:

- In a clinically and mammographically suspicious mass, sensitivity and specificity of FNAC approaches 100%.
- FNAC requires an experienced cytopathologist for accurate interpretation Histopathologic type and grade of malignancy, ER / PR receptor status, HER2/neu status can also be reported on cytopathology by an experienced cytopathologist using a cell block.
- False negative rate for core needle biopsy is very low. However, a tissue specimen that does not show breast cancer cannot conclusively rule out malignancy as sampling error can occur.
- Histologic type and grade of malignancy, receptor status and HER2/neu status can be easily made out on core needle biopsy.
- Fewer "suspicious for malignancy" report with core-needle biopsy. However, more painful,and leaves a parenchymal scar.
- Any patient scheduled for neoadjuvant chemotherapy should have the tumour pathology type and grade, receptor and HER2/neu status documented either on FNAC or core needle biopsy before starting chemotherapy, for in patients with complete response to chemotherapy, there will be no tumour tissue in the surgical specimen.

Incisional Biopsy:
There are few indications for open surgical biopsy to incise a suspicious breast mass.

- It is done in a fungating breast mass. The edge biopsy with some normal skin margin is taken in fungating breast mass.
- Some cases of inflammatory breast cancer may also be suitable for incisional biopsy if core biopsy is not diagnostic.

Excisional biopsy:

Any suspicious breast lesion in which histologic diagnosis was not possible by one of the needle biopsy techniques because of either technical considerations, then an excisional biopsy is indicated.
Grossly, an attempt should be made to excise to excise an approx. 1 cm thickness of normal appearing tissue surrounding the index lesion.

b) Causes of haematuria: see the Question. 2b of Group – B of Paper II of 2013

c) Antegrade pyelography: An antegrade pyelogram is a type of X-ray used to diagnose an obstruction of the upper urinary tract. During the procedure, a contrast dye is injected into a portion of the ureter (narrow tube that carries urine from the kidney to the bladder) closest to the kidneys called the renal pelvis. The flow of the contrast dye can then be observed with X-ra images as it moves from the kidneys into the ureters and urinary bladder.

Purpose:

- To determine the site of a known or suspected ureteral obstruction caused by a stricture, stone, or tumor
- To aid in the placement of a nephrostomy tube, a catheter that is surgically positioned in the kidney for drainage
- To assess the function of the upper collecting system of the kidney after surgery.

Special concerns:

- People who have an allergy to shellfish or iodine may experience an allergic reaction to the contrast dye.
- Pregnant women should not undergo this test because exposure to ionizing radiation may harm the fetus.
- This test may not be safe for people with bleeding disorder.

d) Stress gastritis:

Introduction:

Stress-induced gastritis—also referred to as stress-related erosive syndrome, stress ulcer syndrome, and stress-related mucosal disease—can cause mucosal erosions and superficial hemorrhages in patients who are critically ill or in those who are under extreme physiologic stress, resulting in minimal-to-severe gastrointestinal (GI) blood loss and leading to blood transfusion if not addressed.

Causes:

- Severe trauma
- Massive burns
- Hypotension
- Sepsis with positive blood culture results
- CNS injury with raised intracranial pressure
- Mechanical ventilation
- Multiorgan failure.

Clinical features:

- Coffee ground vomitus
- Melena
- Hematemesis (in extreme cases)
- Orthostasis (unusual)

Diagnosis and investigations: A high degree of clinical awareness is the key to early diagnosis. The presence of any of the previously discussed clinical features should alert the clinician to the presence of stress gastritis.
Useful investigations and diagnostic tools include the following:

- Hematocrit
- Coagulation profile

Procedures:

- Nasogastric tube and lavage: Useful test to confirm the presence of blood in the upper GI tract and to quantify the amount of blood if found (roughly assessed by the amount of normal saline needed before the aspirate becomes clear)
- Endoscopy: Useful only in the diagnosis of stress-induced gastritis

Management:

Prophylaxis of stress gastritis is the goal of management. Monitor the pH of the gastric contents (target: pH >4.0). If the pH level is below the target pH, consider doubling the dose of the agent used if the patient was previously on prophylaxis therapy.

Pharmacotherapy:

The following medications are used in the management of stress-induced gastritis:

- Sucralfate: Primary agent for prophylaxis
- Histamine 2 (H2) receptor blockers (eg, ranitidine, famotidine, cimetidine, nizatidine)
- Proton pump inhibitors (eg, esomeprazole, pantoprazole)

e) P.S.A.

- PSA is a 34KD glycoprotein enzyme produced by the columnar acinar and ductal prostatic epithelial cells.
- PSA is present in both benign and malignant cells, although the expression of PSA tends to be reduced in malignant cells and may be absent in poorly differentiated tumours. Large amounts are secreted into the semen, and small quantities are found in the urine and blood.
- The function of serum PSA is unclear, although it is known to liberate the insulin-like growth factor type 1 from one of its binding proteins.
- 75% of circulating PSA is bound to plasma proteins (complexed PSA) and metabolized in the liver, while 25% is free and excreted in the urine.
- Complexed PSA is stable, bound to alpha-1 antichymotrypsin and alpha-2 macroglobulin.
- Free PSA is unstable
- The normal range for the serum PSA assay in men is <4.0ng/ml, though this varies with age.

The age-adjusted normal range for PSA

Normal PSA range (ng/ml)	
Age range	**Normal PSA range (ng/ml)**
All ages	<4.0
40 - 49	<2.5
50 - 59	<3.5
60 - 69	<4.5
>70	<6.5

- In the absence of prostate cancer, serum PSA concentrations also vary physiologically, according to race and prostate volume.

Indications for checking serum PSA:

- Patient request, following counselling
- Lower urinary tract symptoms
- Abnormal digital rectal examination
- Progressive bone pain, especially back pain
- Unexplained anaemia, anorexia, or weight loss
- Spontaneous thrombo-embolism or unilateral leg swelling
- Monitoring of prostate cancer patients

Increased level of serum PSA:

- Increased levels of PSA may suggest the presence of prostate cancer.
- Obesity has been reported to reduce serum PSA levels.
- PSA levels can be also increased by prostatitis, irritation, benign prostatic hyperplasia (BPH), and recent ejaculation, producing a false positive result.
- Digital rectal examination (DRE) has been shown in several studies to produce an increase in PSA.

  PSA velocity:

  Despite earlier findings recent research suggests that the rate of increase of PSA (e.g. >0.35 ng/mL/yr, the 'PSA velocity) is not a more specific marker for prostate cancer than the serum level of PSA.

Group – C

Write short notes on (any three) **3X5 = 15**

a. **Paraphimosis.**
b. **Lucid interval.**
c. **Chest drain.**

d. **Torsion of Testes.**
e. **Tissue expansion.**

Answers

a) Paraphimosis.

Introduction: Paraphimosis is an uncommon medical condition where the foreskin becomes trapped behind the glans penis, and cannot be reduced). If this condition persists for several hours or there is any sign of a lack of blood flow, paraphimosis should be treated as a medical emergency, as it can result in gangrene.

Causes:

Paraphimosis is usually caused by well-meaning medical professionals or parents who handle the foreskin improperly: The foreskin may be retracted during penile examination, penile cleaning, urethral catheterization, or cystoscopy; if the foreskin is left retracted for a long period, some of the foreskin tissue may become edematous (swollen with fluid), which makes subsequent reduction of the foreskin difficult.

Treatment:

- Phimosis (both pathologic and normal childhood physiologic forms) is a risk factor for paraphimosis.
- Physiologic phimosis resolves naturally as a child matures, but it may be advisable to treat pathologic phimosis via long-term stretching or elective surgical techniques (such as preputioplasty to loosen the pruptial orifice or circumcision to amputate the foreskin tissue partially or completely
- Paraphimosis can often be effectively treated by manual manipulation of the swollen foreskin tissue. This involves compressing the glans and moving the foreskin back to its normal position, perhaps with the aid of a lubricant, cold compression, and local anesthesia as necessary.
- If this fails, the tight edematous band of tissue can be relieved surgically with a dorsal slit or circumcision.
- An alternative method, the Dundee technique, entails placing multiple punctures in the swollen foreskin with a fine needle, and then expressing the edema fluid by manual pressure.
- Some recommend delaying elective circumcision until after paraphimosis has been resolved.

b) Lucid interval: See the Question 3.d of Paper – II of 2014.

c) Chest drain

Introduction:

- **Also known as chest drain.**

- **A chest drain is a tube inserted through the chest wall between the ribs and into the pleural cavity to allow drainage of air (pneumothorax), blood (haemothorax), fluid (pleural effusion) or pus (empyema) out of the chest.**

Characteristics:

Chest tubes are commonly made from clear plastics like PVC and soft silicone. Chest tubes are made in a range of sizes measured by their external diameter from 6 Fr to 40 Fr. Chest tubes, like most catheters, a measured in French catheter scale. For adults, 20 Fr to 40 Fr (6.7 to 13.3mm external diameter) are commonly used, and 6 Fr to 26 Fr for children. Conventional chest tubes feature multiple drainage fenestrations in the section of the tube which resides inside the patient, as well as distance markers along the length of the tube, and a radiopaque stripe which outlines the first drainage hole. Chest tubes are also provided in right angle, trocar, flared, and tapered configurations for different drainage needs. As well, some chest tubes are coated with heparin to help prevent thrombus formation, though the effect of this is disputed.

Channel style chest drains, also called Blake drains, are so-called silastic drains made of silicone and feature open flutes that reside inside the patient. Drainage is thought to be achieved by capillary action, allowing the fluids to travel through the open grooves into a closed cross section, which contains the fluid and allows it to be suctioned through the tube. Though these chest tubes are more expensive than conventional ones, they are theoretically less painful, and drainage with these chest tubes has been proven to be clinically adequate for cardiac surgery drainage.

Indications:

- **Pneumothorax: accumulation of air or gas in the pleural space.**
- **Pleural effusion: accumulation of fluid in the pleural space**
    - **Chylothorax: a collection of lymphatic fluid in the pleural space**
    - **Empyema: a pyogenic infection of the pleural space**
    - **Hemothorax: accumulation of blood in the pleural space**
    - **Hydrothorax: accumulation of serous fluid in the pleural space**
    - **Postoperative: for example, thoracotomy, oesophagectomy, cardiac surgery.**

Contraindications:

- **Contraindications to chest tube placement include refractory coagulopathy**
- **Presence of a diaphragmatic hernia.**
- **Hepatic hydrothorax.**
- **Additional contraindications include scarring in the pleural space (adhesions).**

Complications:

- **Major insertion complications include hemorrhage, infection, and reexpansion pulmonary edema.**
- **Injury to the liver, spleen or diaphragm is possible if the tube is placed inferior to the pleural cavity. Injuries to the thoracic aorta and heart can also occur.**
- **Minor complications include a subcutaneous hematoma or seroma, anxiety, shortness of breath (dyspnea), and cough (after removing large volume of fluid).**
- **Chronic pain related to chest tube induced scarring of the intercostal space is not uncommon.**

Subcutaneous emphysema indicates backpressure created by a clogged drain or insufficient negative pressure.

d) Pathophysiology: Testicular torsion is a condition whereby the testicle twists in such a way that its blood supply becomes compromised. If left untreated, the blood flow to the testicle ceases and the testicle dies. The earlier the surgery to untwist the testis can be undertaken the better the results, with a testicular salvage rate of 100 per cent if the testicle can be untwisted within 6 hours of the torsion taking place.

For torsion to occur, one of several abnormalities must be present:
• Inversion of the testis is the most common predisposing cause.The testis is rotated so that it lies transversely or upside down.
• High investment of the tunica vaginalis causes the testis to hang within the tunica like a clapper in a bell.
• Separation of the epididymis from the body of the testis permits torsion of the testis on the pedicle that connects the testis with the epididymis.

Cause: Normally, when there is a contraction of the abdominal muscles, the cremaster contracts as well. In the presence of one of the abnormalities described above, the spiral attachment of the cremaster favours rotation of the testis around the vertical axis. Straining on stool, lifting of a heavy weight and coitus are all possible precipitating factors. Alternatively, torsion may develop spontaneously during sleep.

Clinical features:

- **Testicular torsion is most common between 10 and 25 years of age, although a few cases occur in infancy.**
- **Redness of the skin and a mild pyrexia.**
- **Typically, there is sudden agonising pain in the groin and the lower abdomen.**
- **The patient feels nauseated and may vomit.**
- **Torsion of a fully descended testis is usually easily recognised. The testis seems high and the tender twisted cord can be palpated above it.**
- **The cremasteric reflex is lost.**

Differential diagnosis:

- **Epididymo-orchitis in the olderpatient; however, in epididymo-orchitis there will usually be dysuria associated with the accompanying urinary infection. Elevation of the testis reduces the pain in epididymo-orchitis and makes it worse in torsion.**
- **Torsion of a testicular appendage.The most common structure to twist is the appendix of the testis (the pedunculated hydatid of Morgagni).**
- **In mumps orchitis the cord is not particularly thickened and the condition is often bilateral.**
- **Idiopathic scrotal oedema.**

Management:

- **If there is any doubt as to the diagnosis, then urgent scrotal exploration is indicated.**
- **Doppler ultrasound scanning can confirm the absence of the blood supply to the affected testis.**
- **Exploration for torsion should be performed through a scrotal incision. If the testis is viable when the cord is untwisted it should be prevented from twisting again by fixation with nonabsorbable sutures between the tunica vaginalis and the tunica albuginea. The other testis should also be fixed because the anatomical predisposition is likely to be bilateral.**
- **An infarcted testis should be removed – the patient can be counselled later about a prosthetic replacement.**

Group – D

4) Answer briefly on (any three): 3x5 = 15

a) Anaesthetic monitoring devices.
b) Radiotherapy in treatment of Carcinoma breast.
c) Ameloblastoma.
d) Transluminal U.S.G
e) Short Wave diathermy.

Answers.

a) Anaesthetic monitoring devices.

Classification of methods of monitoring depth of anaesthesia:

A. Clinical techniques and conventional monitoring
   - I. Clinical sign
   - II. Skin conductance
   - III. Isolated forearm technique
   - IV. Spontaneous surface electromyogram (SEMG)
   - V. Lower oesophageal contractility
   - VI. Heart rate variability

B. Brain electrical activity monitoring
   1. Spontaneous EEG activity monitors.
      - (i) EEG
      - (ii) Compressed spectral analysis
      - (iii) EEG with compressed spectral analysis
      - (iv) Cerebral function monitor (CFM)
      - (v) Cerebral function analysis monitor (CFAM)
      - (vi) Bispectral index
      - (vii) Entropy
      - (viii) Narcotrend®
      - (ix) Patient state analyzer
      - (x) SNAP index
      - (xi) Cerebral state monitor/Cerebral state index
   2. Evoked brain electrical activity monitors.
      - (i) Somatosensory evoked potential (SSEP)
      - (ii) Visual evoked potential (VEP)
      - (iii) Auditory evoked potential (AEP)

b) Radiotherapy in treatment of Carcinoma breast.

Indications of Loco-Regional Radiotherapy In Breast Cancer

1.Indication after Breast Conservative Surgery (BCS)

Contra-indication of BCS

Absolute :	Relative:
Prior RT to breast/ chest wall • Pregnancy • Diffuse, suspicious micro calcification • Wide spread disease where negative margin cannot be obtained • Positive surgical margins	• Active connective tissue disease. • Large tumor in relative small breast • Poor compliance anticipated

.

Indication of Radiotherapy

No of positive node	RT to breast only
1-3 positive nodes	• RT to breast • RT to supraclavicular nodal area may be considered
4+ positive nodes	• RT to breast • RT to supraclavicular nodal region • RT to Internal Mammary nodes may be considered

2. Indications of radiotherapy post-mastectomy

- Tumor size < 5 cm & clear margins > 1 cm & negative axilla - RT not indicated
- Size > 5cms & margins < 1cm & negative axilla - RT to chest wall only
- Negative axilla, tumor more than 5 cms or positive margins:

o RT to chest wall with scar boost
o RT to supraclavicular nodal area
o RT to Internal Mammary chain is a matter of debate

- 1-3 axillary node positive:
    - RT to chest wall with scar boost
    - RT to supraclavicular nodal area
    - Some prefer to treat internal mammary nodes as well

- 4 or more nodes positive:
    - RT to chest wall with scar boost
    - RT to supraclavicular nodal area
    - Internal mammary nodes may be treated

Advantage of Intensity Modulated Radiotherapy (IMRT) over conventional RT in breast Cancer:

o Better cosmesis
o Reduced incidence of radiation induced malignancy in contra-lateral breast
o Reduced incidence of coronary artery disease (when left breast is treated)
o Reduced pulmonary complication

Role of radiotherapy does not influence presence or absence of marker.

Role of Radiotherapy in Post Operative LABC:

- Postoperative radiotherapy can be given immediately after surgery or sandwitch radiotherapy following three cycles of chemotherapy and subsequently again three cycle of chemotherapy, or six cycle of chemotherapy followed by radiotherapy.
- Timing of radiotherapy will depend on if residual lesion or positive margin; after six cycle of chemotherapy in case no lesion is there, or three cycle of chemotherapy followed by radiotherapy followed by another three cycle of chemotherapy in case a lesion or positive margin is there.
- The dose of radiation is 50 Gy / 5 wks including chest wall, internal mammary, supraclavicular infraclavicular and axillary portals. Radiation can be given by telecobalt, low or high energy accelerators and photon beam.

c) Ameloblastoma

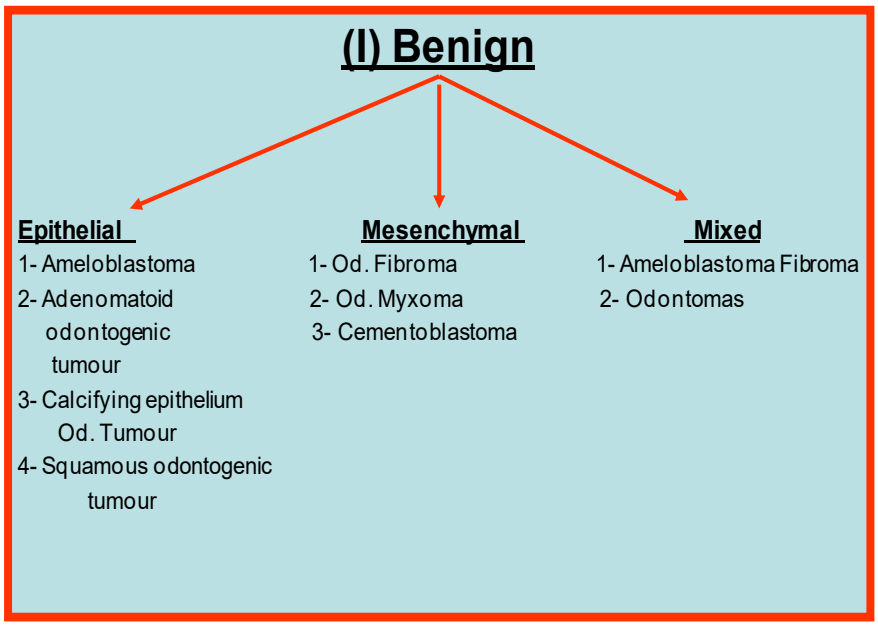
Go through:

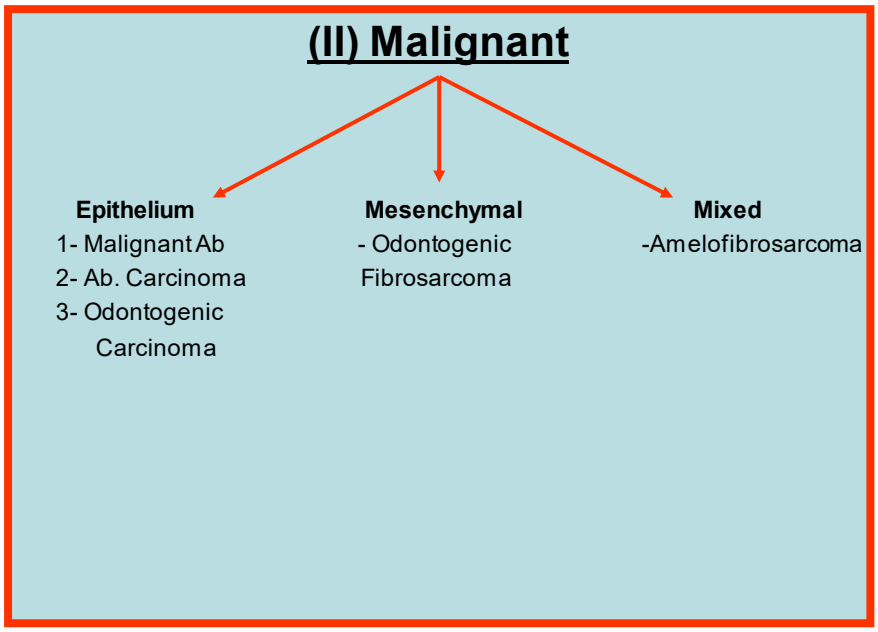
Odontogenic Tumours

Important causes of swelling of the jaws:

1) Cysts especially odontogenic ones.
2) Odontogenic tumours (most common neoplasms).
3) Giant cell lesions.
4) Fibro-osseous lesions.
5) Primary (non-odontogenic) neoplasms of bone.
6) Metastatic neoplasms.
7) Chronic osteomyelitis.

Classification of odontogenic tumours according to the behaviour of the lesions





Ameloblastoma:

- It is the most common neoplasm of the jaws.

Clinical features:

- Age: Between 3rd to 5th decades.

- Sex: No sex predilection
- Site: Mandible more than maxilla 80% of cases were in mandible . In the mandible 70% of cases were in molar ramous region.

Characters :

- Asymptomatic
- Slowly growing
- Discovered by X – ray

Radiographic Appearances :

1) Multilocular radiolucent area.(Honey comb pattern) & expanded lingually

2) Unilocular radiolucent area usually associated with impacted tooth (as in dentigerous cyst).

Macroscopic appearances :

- Solid or cystic or both together.
- Traversed by bony ridges.

Microscopic appearances:

-Different histological patterns are seen under microscope.

-The epithelium forming this neoplasm resembles that epithelium which forms the enamel organ.

- The fibrous stroma surrounding this epithelium vary in both quantity & cellularity (not neoplastic one).

- In most cases the stroma is collagenous with few cells while in other cases , the stroma may be abundantly cellular .

Two main histological patterns are commonly seen in ameloblastoma.

1- Follicular

2- Plexiform.

Diagnosis:

1- biopsy for histological examination.

2- X- Ray .

3- Magnetic resonance (solid or cystic types).

Treatment :

1- Surgical removal with safety margins.

2- Large tumours may require bony resection or even the whole jaw.

3- Ab id radioresistent tumour so it is not treated by radiotherapy.

Prognosis :

1- Solid & multicystic have a higher tendency to recur when treated conservatively (i.e. without safety margin)

2- Maxillary ameloblastoma when allowed to reach a large size may give rise to serious complication .

3- Adequate treatment gives good prognosis.

d) Transluminal USG. See Question 4.d of Group-D of Supplementary Paper - II of 2014.

e) Short wave diathermy:

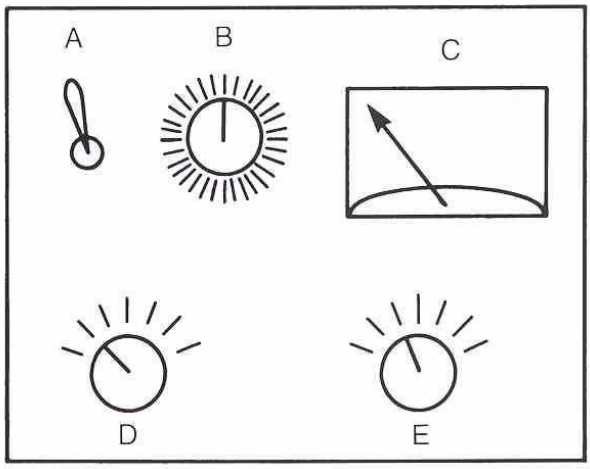
Introduction: Diathermy is a therapeutic treatment most commonly prescribed for joint conditions such as rheumatoid arthritis and osteoarthritis. In diathermy, a high-frequency electric current is delivered via shortwave, microwave, or ultrasound to generate deep heat in body tissues.

Short wave diathermy

- Radio Transmitter With FCC Assigned Frequencies
  - 27.12 MHz at 11 M
  - 13.56 MHz at 22 M
  - 40.68 MHz at 7.5 M

Shortwave Diathermy Unit:

- Power Supply Powers Radio Frequency Oscillator (RFO)
- RFO Provides Stable Drift-Free Oscillations at Given Frequency
- Power Amplifier Generate Power To Drive Electrodes
- Output Resonant Tank Tunes In The Patient for Maximum Power Transfer



  - A=Power Switch
  - B=Timer
  - C=Power Meter(monitors current from power supply not current entering patient-volume control)
  - D=Output Intensity(%max power to patient)
  - E=Tuning Control(tunes output from RFO)
- Power Output Should Provide Energy To Raise Tissue Temp To Therapeutic Range (40-45 deg C) (80-120 watts)
- Should Exceed SAR-Specific Absorption Rate (rate of energy absorbed /unit area of tissue mass)
- Generates Both an Electrical and a Magnetic Field
- Ratio Depends on Characteristics of Both The Generator and the Electrodes
  - SWD Units at 13.56 MHz= Stronger Magnetic Field
  - SWD Units at27.12 MHz = Stronger Electrical Field

The West Bengal University of Health Sciences

M.B.B.S. 3rd Professional Part – II Examination, 2011

Subject: Surgery **Time: $2^{1}/_{2}$ hrs.**

Paper: II **Marks: 60**

Group –A

1) a) Classify carcinoma of breast. How will you manage a case of early carcinoma breast in a 40 yr old lady. 4+5+6=15

Answer.

Classification of breast cancer:

Noninvasive Epithelial Cancers
LCIS
DCIS or intraductal carcinoma
- Papillary, cribriform, solid, and comedo types

Invasive Epithelial Cancers (percentage of total)
Invasive lobular carcinoma (10%)
Invasive ductal carcinoma
- Invasive ductal carcinoma, NOS (50%-70%)
- Tubular carcinoma (2%-3%)
- Mucinous or colloid carcinoma (2%-3%)
- Medullary carcinoma (5%)
- Invasive cribriform carcinoma (1%-3%)
- Invasive papillary carcinoma (1%-2%)
- Adenoid cystic carcinoma (1%)
- Metaplastic carcinoma (1%)

Mixed Connective and Epithelial Tumors
Phyllodes tumors, benign and malignant
Carcinosarcoma
Angiosarcoma
Adenocarcinoma

The lady presented with stage I carcinoma breast.

Investigations:

- Complete blood cell count, complete metabolic panel, and chest x-ray.
- A bone scan if the alkaline phosphatase or calcium level is elevated.
- CT scan of the liver if liver function panel is abnormal.
- CT scan of the thorax to exclude presence of lung secondaries.

Surgical options:

- Mastectomy with or without reconstruction.
- Modified radical mastectomy (MRM) involves total (simple) mastectomy and axillary lymph node dissection. It is indicated for patients with clinically positive lymph nodes or a positive axillary node based on previous SLNB or FNAB.

- Total (simple) mastectomy with SLNB is for patients with a clinically negative axilla. A skin-sparing mastectomy (preserves skin envelope and inframammary ridge) may be performed with immediate reconstruction, resulting in improved cosmesis: The nipple-areolar complex, a rim of periareolar breast skin, and any previous excisional biopsy or partial mastectomy scars are excised.
- Immediate reconstruction at the time of mastectomy should be offered to eligible patients. Options include latissimus dorsi myocutaneous flaps, transverse rectus abdominis myocutaneous flaps, and inflatable tissue expanders followed by exchange for saline or silicone implants. Immediate reconstruction has been shown not to affect patient outcome adversely. The detection of recurrence is not delayed, and the onset of chemotherapy is not changed.
- Follow-up after mastectomy: physical examination every 3 to 6 months for 3 years, then every 6 to 12 months for the next 2 years, and then annually. Mammography of the contralateral breast should continue yearly. Regular gynecologic follow-up is recommended for all women (tamoxifen increases risk of endometrial cancer).

- **Breast conservation therapy (BCT):** partial mastectomy and SLNB (or axillary lymph node dissection) followed by breast irradiation.

Contraindications to BCT:

- Pregnancy
- Multicentric disease
- Diffuse indeterminate micro-calcification
- Previous RT
- Large tumour/ breast ratio
- Collagen vascular disease
- Large breast size
- Central tumour

For patients with large tumors who desire BCT, neoadjuvant chemotherapy or neoadjuvant hormonal therapy may be offered to attempt to reduce the size of the tumor to make BCT attempt possible.

- **Management of the axilla:** Approximately 30% of patients with clinically negative exams will have positive lymph nodes in an axillary lymph node dissection (ALND) specimen. The presence and number of lymph nodes involved affect staging and thus prognosis.
  - ALND. Patients with clinically positive lymph nodes or with positive SLN should undergo ALND for local control.

ALND involves the following:

- Removal of level I and level II nodes and, if grossly involved, possibly level III nodes. Motor and sensory nerves are preserved unless there is direct tumor involvement.
- An ALND should remove 10 or more nodes. The number of nodes identified is often pathologist dependent.

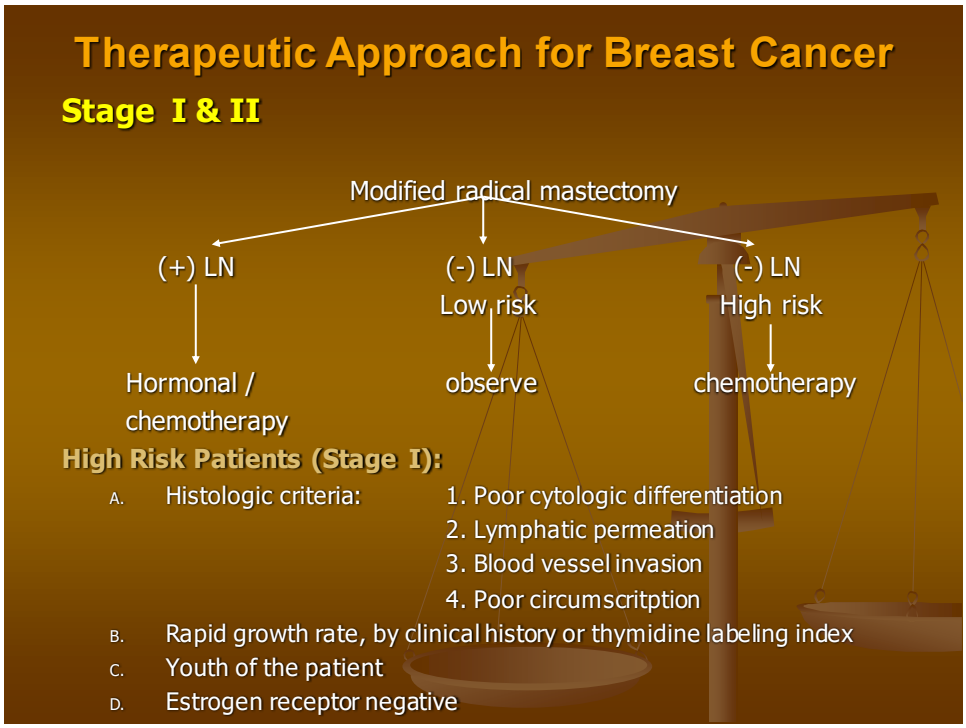
- Patients with 4 or more positive lymph nodes should undergo adjuvant radiation to the axilla. Selective patients with 1 to 3 positive nodes may also benefit from radiation therapy to the axilla.
- Intraoperative complications: potential injury to the axillary vessels and neuropathy secondary to injury to the motor nerves of the axilla (the long thoracic, thoracodorsal, and medial pectoral nerves).

Adjuvant chemotherapy is given in appropriate patients after completion of surgery.

- All node-positive patients should receive adjuvant chemotherapy.
  - Regimens are guided by the tumor biomarkers. Typical regimens comprise four to eight cycles of a combination of cyclophosphamide and an anthracycline, followed by a taxane administered every 2 to 3 weeks.
  - Patients with ER-positive tumors receive adjuvant hormonal therapy for 5 years. Tamoxifen is given to premenopausal women, and aromatase inhibitors are given to postmenopausal women (aromatase inhibitors are not used in premenopausal women).
  - **(In postmenopausal women older than 70 years, chemotherapy is performed less frequently. In postmenopausal women with tumors with ER or PR positivity, tamoxifen or an aromatase inhibitor is frequently the sole adjuvant medical therapy.)**
  - In patients with Her2/neu-positive tumors, polychemotherapy is combined with biological therapy targeting the Her2/neu protein: Trastuzumab is a recombinant monoclonal antibody that binds to Her2/neu receptor to prevent cell proliferation. Adding trastuzumab to a chemotherapy regiment of doxorubicin, cyclophosphamide, and paclitaxel was associated with an increase in the disease-free survival by 12% and a 33% reduction in the risk of death at 3 year.

- Node-negative patients may have increased disease-free survival from adjuvant chemotherapy and/or hormonal therapy.

  - Up to 30% of node-negative women die of breast cancer within 10 years if treated with surgery alone.
  - Node-negative patients who are at high risk and benefit the most from adjuvant chemotherapy include those with tumors greater than 1 cm, higher tumor grade, Her2/neu expression, aneuploidy, Ki-67 expression, increased percentage in S phase, lymphovascular invasion, and ER/PR-negative tumors.
  - Polychemotherapy in combination with tamoxifen was superior to tamoxifen alone in increasing disease-free and overall survival, especially in ER-negative patients, regardless of tumor size.
- Adjuvant whole-breast radiation after BCT decreases the breast cancer recurrence rate from 30% to less than 7% at 5 years.



Or

b) Classify renal neoplasms. How will you diagnose and manage a case renal cell carcinoma? 4+5+6=15

Answer. See the answer of Question 2.a of Group –B of Supplementary Paper- II of 2013

Group – B

2) Write short notes on (any three): 3x5=15

a) Flail chest.
b) Post operative pyrexia.
c) Brain death.
d) Split thickness skin graft.
e) Omphalocele.

Answers

a) Flail chest: See the Question 4.c of Group – D of Supplementary Paper - II of 2014.

b) Post operative pyrexia:

Postoperative fever is a common condition challenging doctors to find the right diagnosis, because it can be a hallmark of serious underlying conditions. One third of patients develop fever after surgery depending on type of surgery but only a small percentage turn out to be due to infection.

Definition: Postoperative fever is defined as a temperature >38°C (100°F) on 2 consecutive postoperative days, or >39°C (102.2°F) on any 1 postoperative day.

Causes:

Category	Day	Description
Wind	POD1-2	the lungs, i.e. pneumonia, aspiration, and pulmonary embolism. Once attributed to atelectasis, but a recent review suggests that is not supported by existing clinical evidence.

Water	POD3-5	urinary tract infection, related to indwelling catheter (during surgery or currently i.e.Foley catheter)
Walking (or VEINS, which then sounds like "Weins")	POD4-6	deep vein thrombosis or pulmonary embolism
Wound	POD5-7	surgical site infection, which in obstetrics or gynaecology, may refer to the uterus.
Wonder drugs or “What did we do?”	POD7+	drug fever, infections related to intravenous lines or reaction to blood products

Pathophysiology: Fever is a manifestation of cytokine release in response to a variety of stimuli. Fever-associated cytokines, including interleukin (IL)-1, IL-6, tumor necrosis factor (TNF)-alpha, and interferon (IFN)-gamma, are produced by a variety of tissues and cells. There is some evidence that IL-6 is the cytokine most closely correlated with postoperative fever.

Causes of post operative fever

Infectious	Noninfectious
Abscess	Acute hepatic necrosis
Acalculous cholecystitis	Adrenal insufficiency
Bacteremia	Allergic reaction
Decubitus ulcers	Atelectasis
Device-related infections	Dehydration
Empyema	Drug reaction
Endocarditis	Head injury
Fungal sepsis	Hepatoma
Hepatitis	Hyperthyroidism
Meningitis	Lymphoma
Osteomyelitis	Myocardial infarction
Pseudomembranous colitis	Pancreatitis
Parotitis	Pheochromocytoma
Perineal infections	Pulmonary embolus
Peritonitis	Retroperitoneal hematoma
Pharyngitis	Solid organ hematoma
Pneumonia	Subarachnoid hemorrhage
Retained foreign body	Systemic inflammatory response syndrome
Sinusitis	Thrombophlebitis
Soft tissue infection	Transfusion reaction
Tracheobronchitis	Withdrawal syndromes
Urinary tract infection	Wound infection

Important Factors to Remember When Evaluating Postoperative Fever

- Age
- General Health
- Length of Surgery
- Type of anesthesia
- Surgical trauma
- Time since surgery
- Drug therapy
- Laboratory results
- Status of Patient
  - Pain
  - Signs of lnfection
  - Urinary retention
  - Constipation

Treatment:

- Treatment is best aimed at the associated pathological process instigating it.
- The antipyretic agents are not given until the cause of the fever has been determined or at least until a maljr pathologic condition has been ruled out.
- Mild fevers require no pharmaceutical therapy unless that patient is uncomfortable as a result of marked debilitation.
- Once the cause of the fever is identified, antipyretic agents can be utilized in cases where the temperature elevation is marked or persistent. Drugs of choice are aspirin and acetaminophen. When fevers are resistant to salicylates, steroid preparations may be utilized.
- Each fever should be carefully evaluated and the more dangerous etiologic causes ruled out.
- Treat the actual cause.

c) Brain death.

Brain death is the complete and irreversible loss of brain function (including involuntary activity necessary to sustain life).
Some causes of brain death include (but are not limited to):

- Trauma to the brain (i.e. severe head injury caused by a motor vehicle crash, gunshot wound, fall or blow to the head)
- Cerebrovascular injury
- Anoxia
- Brain tumour

Clinical testing for brainstem death:

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Absence of cranial nerve reflexes	Absence of motor response	Absence of spontaneous respiration
• Pupillary reflex	The absence of a motor response to painful	After preventilation with 100% O2 for at least

• Corneal reflex • Pharyngeal (gag) and tracheal (cough) reflex • Oculovestibular (caloric) reflex	stimuli applied to the head/face and the absence of a motor response within the cranial nerve distribution to adequate stimulation of any somatic area is an indicator of brainstem death. The presence of spinal reflexes does not preclude brainstem death	5 minutes, the patient is disconnected from the ventilator for 10 minutes to confirm absence of respiratory effort, during which time the arterial PCO2 level should be >8 kPa (60 mmHg) to ensure adequate respiratory stimulation. To prevent hypoxia during the apnoeic period, O2 (6 L/min) is delivered via an endotracheal catheter.

d) Split thickness skin graft.

Introduction:

- Split-thickness skin grafts (of varying thickness). These are sometimes called Thiersch grafts.
- They are used to cover all sizes of wound, are of limited durability and will contract.
- They may be used to provide valuable temporary wound closure before better cosmetic secondary correction after rehabilitation.

Donor site: The split thickness skin graft leaves behind adnexal remnants such as hair follicles and sweat glands, foci from which epidermal cells can repopulate and resurface the donor site. It is usually harvested with either a special blade or dermatome that can be set to a desired thickness.

Recipient site: Split thickness grafts are usually used to resurface larger defects. Depending on how much of the dermis is included, STSGs undergo secondary contraction as they heal.

Mechanism of graft survival	Mechanism of graft failure
i. Plasmatic imbibition — Initially, the skin grafts passively absorbs the nutrients in the wound bed by diffusion. ii. Inosculation — By day 3, the cut ends of the vessels on the underside of the dermis begin to form connections with those of the wound bed iii. Angiogenesis — By day 5, new blood vessels grow into the graft and the graft becomes vascularized	i. Poor wound bed — Because skin grafts rely on the underlying vascularity of the bed, wounds that are poorly vascularized with bare tendons or bone, or because of radiation, will not support a skin graft. ii. Sheer — Sheer forces separate the graft from the bed and prevent the contact necessary for revascularization and subsequent “take”. iii. Hematoma/seroma — Hematomas and seromas prevent contact of the graft to the bed and inhibit revascularization. They must be drained by day 3 to ensure “take”. iv. Infection — Bacteria have proteolytic enzymes that lyse the protein bonds needed for revascularization.

Also read: Full thickness — Full thickness skin grafts (FTSGs) consist of the entire epidermis and dermis.

Donor site — The full thickness skin graft leaves behind no epidermal elements in the donor site from which resurfacing can take place. Thus, the donor site of a FTSG must be closed. It must be taken from an area that has skin redundancy. It is usually harvested with a knife between the dermis and the subcutaneous fat.

Recipient site - Full thickness skin grafts are usually used to resurface smaller defects because they are limited in size. It is commonly used to resurface defects of the face. It provides a better color consistency, texture, and undergoes less secondary contraction.

Mechanism of graft survival and failure are same as STSG.

e) Omphalocele.

Introduction:

- An omphalocele is a type of abdominal wall defect in which the intestines, liver, and occasionally other organs remain outside of the abdomen in a sac because of a defect in the development of the muscles of the abdominal wall (exomphalos).
- Omphalocele occurs in 2.5/10,000 births and is associated with a high rate of mortality (25%) and severe malformations, such as cardiac anomalies (50%) and neural tube defect (40%).
- Approximately 15% of live-born infants with omphalocele have chromosomal abnormalities.
- Associated with foreshortened bowel and malrotation.
- Small abdominal cavity and pulmonary hypoplasia.
- May be associated with maternal use of valproic acid

Embryology:

- Normally, midgut returns to the abdomen by 10^{th} week of gestation
- Somatic layers of cephalic, caudal, and lateral folds join to close abdominal wall
- With omphalocele, folds fail to close

Associated syndromes and anomalies:

• Gastrointestinal	• Trisomy 13	• Beckwith-Wiedemann	• Cleft palate
• Cardiac	• OEIS complex (omphalocele	• Pentalogy of Cantrell	Pulmonary hypoplasia

Diagnosis:

- AFP synthesized in fetal liver and excreted by fetal kidneys and crosses placenta by 12 weeks
- Elevated maternal MSAFP in neural tube defects, abdominal wall defects, duodenal or esophageal atresia
- 40% false positive rate
- Fetal ultrasound after 14 weeks gestation
- Amniocentesis and fetal echocardiography.

Treatment:

- NGT to low intermittent suction
- Use of bowel bags, saran wrap
- Conservation of body heat and fluid losses
- Antibiotics
- Careful positioning to avoid kinking of mesenteric vessels
- 1.5 times maintenance fluids with isotonic fluids.

Surgical Management:

- Operative repair within 2-4 hours of birth
- Primary closure for smaller defects
- Delayed primary closure for large defects
  - Avoid compromised ventilation and abdominal compartment syndrome
  - Use of silo with sequential reduction of abdominal contents
  - Later fascial closure

Group – C

3) Write short notes on (any three) 3x5 = 15

a) **Retrosternal goiter.**
b) **Parotid abscess.**
c) **Alvarado score of acute appendicitis.**
d) **T.U.R.P.**
e) **Oxalate stone.**

Answers

a) Retrosternal goiter.

Introduction:

A retrosternal goitre occurs when the thyroid enlarges downwards into the chest.Although the great majority of retrosternal goitres are extensions from the neck, pure intrathoracic goitres do occur. Retrosternal goitres are more likely to be left sided.

Classification:

Primary (0.2-3%)	Intrathoracic goiters arising from ectopic mediastinal thyroid tissue and having blood supply from intrathoracic vessels.
Secondary	Intrathoracic goiters descended from neck and having blood supply from inferior thyroid artery.

Higgins sub classified intrathoracic goiters by extent-

Completely intrathoracic	More than 80 % intrathoracic with barely detectable or no cervical component.
Partially intrathoracic	More than 50 % intrathoracic
Substernal	Both cervical and mediastinal component, more than 50% in neck

Shahian classified substernal goiters as follows-

Type	**Location**	**Anatomy**	**Prevalence**	**Approach, remarks**
I	Anterior mediastinum	Anterior to great vessels, trachea, RLN	85%	Transcervical(sternotomy only if intrathoracic goiter diameter > thoracic inlet diameter)
II	Posterior mediastinum	Posterior to great vessels, trachea, RLN	15%	As above, sternotomy / right posterolateral Thoracotomy if type IIb
IIA	Ipsilateral extension			
IIB Contralateral extension B1-posterior to trachea and esophagus B2-between trachea and esophagus				
III	Isolated mediastinal goiter	No connection to orthotopic gland, mediastinal blood supply	<1%	Transcervical/ sternotomy

Clinical manifestations;

- They are related to compression or displacement of aero digestive tract and mediastinal great vessels.
- Most present with palpable neck mass with lower border not reachable even on deglutition. Rarely, goiter — "Plongeont" is present in which neck mass disappears into thoracic cavity and appears again on swallowing or coughing.
- Early tracheal compression may manifest as nocturnal choking, cough, dyspnea, asthma or obstructive pulmonary disease.

- Acute airway obstruction may occur due to hemorrhage within tumor which may even need emergency tracheostomy.
- There is higher incidence of vocal cord palsy due to stretching and ischemia of recurrent laryngeal nerve. Extrinsic compression of esophagus may result in dysphagia.
- *Pemberton's sign* is described as development of head and neck venous engorgement with facial congestion, plethora and venous distension when both arms are raised over the head.
- Obstruction to superior vena cava or subclavian vein may result in development of collateral venous drainage leading to facial flushing/edema and dilated neck and upper thoracic veins.
- The downhill esophageal varices secondary to superior vena cava obstruction may lead to gastrointestinal tract bleeding in absence of other signs of portal hypertension.
- Cervical cutaneous nerves may be pressed resulting in pain in head, neck chest and shoulders.
- Compression of sympathetic plexus may lead to Horner's syndrome.
- Thoracic duct occlusion may lead to chylothorax. In some cases symptoms may be positional and occurring when patient turns neck to the side of goiter or raises arms.
- Majority of Retrosternal goiters are euthyroid with 20 % developing thyrotoxicosis in long standing goiters.

Imaging:

Chest x-ray	Soft tissue shadow, calcification, tracheal deviation/ compression
CECT Scan	Look for continuity of cervical goiter, extent of goiter, calcification(punctate/coarse/ring like), relation of goiter to adjoining structures- trachea, esophagus, larynx, pharynx, major vessels, lymphadenopathy

Treatment: Surgery is the treatment of choice for retrosternal goiters. The various reasons for surgery include compressive symptoms, ineffectiveness of suppressive therapy, or risk of malignancy.

b) Parotid abscess. See the Question 3.a of Group – C of Supplementary Paper - II of 2014.

c) Alvarado score of acute appendicitis.

Introduction:

The **Alvarado score** is a clinical scoring system used in the diagnosis of appendicitis. The score has 6 clinical items and 2 laboratory measurements with a total 10 points. It was introduced in 1986 and although meant for pregnant females, it has been extensively validated in the non-pregnant pregnant population. The modified Alvarado score is at present in use.

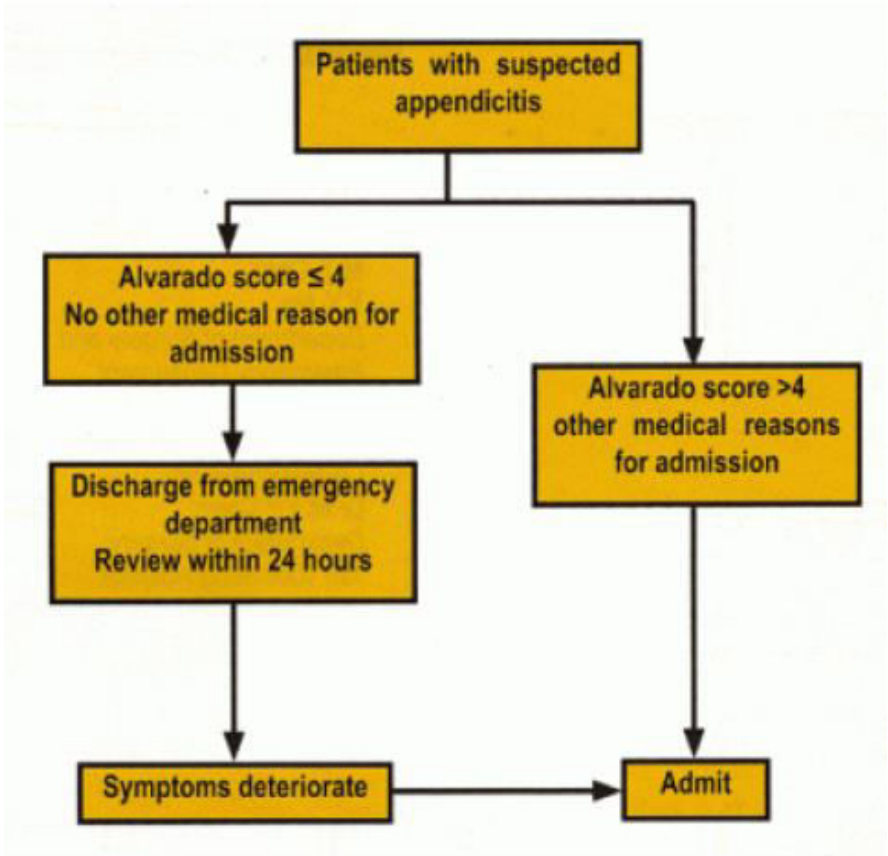
Alvarado score for appendicitis

Symptoms	Score
Migratory right iliac fossa pain	1
Nausea / Vomiting	1
Anorexia	1
Signs	
Tenderness in right iliac fossa	2
Rebound tenderness in right iliac fossa	1
Elevated temperature	1
Laboratory findings	
Leucocytosis	2
Shift to the left of neutrophils	1
Total	**10**

5-6 → Possible

7-8 → Probable

> 9 → Very probable



d) TURP:

Introduction:

- A Transurethral Resection of Prostate (TURP) is done by removing the inner portion of the prostate that is pressing on the urethra.
- Transurethral resection of the prostate (TURP) is performed by inserting a resectoscope through the urethra and resecting prostatic tissue with an electrically powered cutting-coagulating metal loop.

Indications and contraindications:

- The most frequent indication (50–60%) for TURP is LUTS refractory to medical therapy.
- The following BPE/BPO complications are considered strong indications for TURP
  (1) Recurrent urinary retention,
  (2)BPH- or BPE-related macro-haematuria refractory to medical therapy with 5a-reductase inhibitors (5- ARI),
  (3) Renal insufficiency or upper urinary tract dilatation,
  (4) Bladder stones, and
  (5) Recurrent urinary tract infection (UTI).

The only contraindications for TURP are untreated UTI and bleeding disorders.

Solutions used for irrigation:

- Glycine (1.5%)
- Glucose (5%)
- Normal saline (0.9%)

Risks:

- Retrograde ejaculation
- Impotence
- Incontinence

Complications:

- Bleeding,
- Urethral stricture or bladder neck contracture,
- Perforation of the prostate capsule with extravasation,
- TUR syndrome resulting from a hypervolemic, hyponatremic state due to absorption of the hypotonic irrigating solution.

TURP syndrome:

Signs/symptoms

Cardiovascular and Respiratory	CNS	Metabolic	Other
Hypertension	Agitation/confusion	Hyponatremia	Hypo-osmolality
Brady/tachyarrhythmias	Seizures	Hyperglycinemia	Hemolysis
Congestive heart failure	Coma	Hyperammonemia	
Pulmonary edema and hypoxemia	Visual disturbances (blindness)		
Myocardial infarction			
Hypertension			

e) Oxalate stone.

- Oxalate is a normal waste product of metabolism and is relatively insoluble. Normally, approximately 10–15% of oxalate found in the urine originates from the diet; the vast majority is a metabolic by-product.
- Most of the oxalate that enters the large bowel is consumed by bacterial decomposition.
- Diet, however, can have an impact on the amount of oxalate found in the urine. Once absorbed from the small bowel, oxalate is not metabolized and is excreted almost exclusively by the proximal tubule.
- The presence of calcium within the bowel lumen is an important factor influencing the amount of oxalate that is absorbed. The control of oxalate in the urine plays a pivotal role in the formation of calcium oxalate calculi.
- Small changes in oxalate levels in the urine can have a dramatic impact on the supersaturation of calcium oxalate.
- The principal precursors of oxalate are glycine and ascorbic acid; however, the impact of ingested vitamin C (<2 g/day) is negligible.
- Hyperoxaluria may develop in patients with bowel disorders, particularly inflammatory bowel disease, small bowel resection, and bowel bypass. Renal calculi develop in 5–10% of patients with these conditions.
- Chronic diarrhea with fatty stools results in a saponification process. Intraluminal calcium binds to the fat, thereby becoming unavailable to bind to oxalate. The unbound oxalate is readily absorbed.
- Excessive oxalate may occur secondary to the accidental or deliberate ingestion of ethylene glycol (partial oxidation to oxalate).
- This may result in diffuse and massive deposition of calcium oxalate crystals and may occasionally lead to renal failure.
- Formed in acidic urine.
- Calcium oxalate crystals in urine appear as 'envelopes' microscopically. They may also form 'dumbbells.

Group – D

4) Answer briefly on (any three): 3x5 = 15

a) Wax bath.
b) Epulis.
c) M.R.I scan in Surgery.
d) Radiation Dermatitis.
e) Spinal Anaesthesia.

Answers

a) Wax bath. See the Question 4.b of Group – D of Paper –II of 2013

b) Epulis

Introduction:

- Epulis is any benign tumor (i.e. lump) situated on the gingival or alveolar mucosa.
- Most of them are granulomas associated with chronic gingivitis. A few are true neoplasms.
- There are three types: fibromatous, ossifying and acanthomatous.
- Epulis appears as a single or multiple fold of tissue that grows in excess around the alveolar vestibule.
- Usually, the edge of the denture rests in between two of the folds. The excess tissue is firm and fibrous, and ulcerations may be present.
- The great majority of cases are seen beneath ill-fitting dentures of long use and in persons who do not take their dentures out overnight.
- The lesion seems to result from a combination of chronic, mild trauma and low-grade infection by bacteria or candida yeast.
- It is occasionally seen in patients without dentures but with high palatal vaults or with the habit of breathing through their mouths.

Signs and symptoms:

- The lesion is usually painless.
- The usual appearance is of two excess tissue folds in alveolar vestibule/buccal sulcus, with the flange of the denture fitting in between the two folds.
- .It may occur in either the maxillary or mandibular sulci, although the latter is more usual.
- Anterior locations are more common than posterior.
- Less commonly there may be a single fold, and the lesion may appear on the lingual surface of the mandibular alveolar ridge.
- The swelling is firm and fibrous, with a smooth, pink surface. The surface may also show ulceration or erythema.
- The size of the lesion varies from less than 1 cm to involving the entire length of the sulcus.

Causes:

- The cause is usually pressure from the flange of a denture which causes chronic irritation and a hyperplastic response in the soft tissues.
- Women during pregnancy can also present with an epulis, which will resolve after birth.
- Fibroepithelial polyps, pedunculated lesions of the palate beneath an upper denture, are associated with this condition.
- A cobble-stone appearance similar to an epulis fissuratum in a patient without dentures can be diagnostic of Crohn's disease.
- Epulis fissuratum can also appear around dental implants.

Diagnosis:

- The diagnosis is made clinically, and usually this is clear cut if the lesion is associated with the flange of a denture.
- Tissue biopsy is not usually indicated before removal of the lesion, since the excises surgical specimen is usually sent for histopathologic examination and the diagnosis is confirmed retrospectively.
- Rarely, incisional biopsy may be indicated to rule out neoplasia, e.g. in the presence of suspicious ulceration. The appearance may also be confused with pyogenic granuloma.
- The excessive tissue is composed of cellular, inflamed fibrous connective tissue. The appearance of an epulis fissuratum microscopically is an overgrowth of cells from the fibrous connective tissue. The epithelial cells are usually hyperkeratotic and irregular, hyperplastic rete ridges are often seen.

Treatment:

- Treatment is by surgical excision (complete removal) of the fibrous tissue overgrowth and addressing the causative factor to prevent recurrence of the lesion.
- Common techniques for removal of the excess tissue include traditional removal with a surgical scalpel, electrical scalpel, or laser excision with a laser scalpel, e.g. a carbon dioxide laser, erbium:YAG laser, Neodymium-YAG laser, or diode laser.
- The poorly fitting denture can be adapted to fit better (a "reline") or a new denture constructed.
- Alternatively, the section of flange that is sharp/over-extended can be smoothed and reduced with a drill.

c) M.R.I Scan in Surgery.

- MRI allows the clinician to see high quality images of the inside of the body:
- MRI can be used for preoperative evaluation of the patients.
- It can be used intra-operatively also.

- The most prevalent intra-operative application for MRI is neurosurgery, especially for the removal of brain tumors. The system is also used for interventional neurovascular procedures.

Magnetic resonance imaging

Strengths

- No ionising radiation
- Excellent soft-tissue contrast
- Best imaging technique for
  - Intracranial lesions
  - Spine
  - Bone marrow and joint lesions

Evolving use

- Staging
- MRCP
- MR angiography
- Breast malignancy
- Pelvic malignancy
- Cardiac imaging

Weaknesses

- Absolute contraindications
  - Ocular metallic foreign bodies
  - Pacemakers
  - Cochlear implants
  - Cranial aneurysm clips
- Relative contraindications
  - First trimester of pregnancy
  - Claustrophobia

Long scan times so patients may not be able to keep still, especially if in pain

Limited availability

Expensive

d) Radiation dermatitis.

Introduction:

Radiation dermatitis (also known as **radiodermatitis**) is a skin disease associated with prolonged exposure to ionizing radiation.

Radiation dermatitis occurs to some degree in most patients receiving radiation therapy, with or without chemotherapy.

Types: There are three specific types of radiodermatitis: acute radiodermatitis, chronic radiodermatitis, and eosinophilic, polymorphic, and pruritic eruption associated with radiotherapy.

- **Acute radiodermatitis** occurs when an "erythema dose" of ionizing radiation is given to the skin, after which visible erythema appears up to 24 hours after. Radiation dermatitis generally manifests within a few weeks after the start of radiotherapy.Acute radiodermatitis, while presenting as red patches, may sometimes also present with desquamationor blistering. Erythema may occur at a dose of 2 Gy radiation or greater.

The National Cancer Institute (USA) has developed a 4 stage criteria for the classification of acute radiation dermatitis:

  - Grade 1 – Faint erythema or desquamation.
  - Grade 2 – Moderate to brisk erythema or patchy, moist desquamation confined to skin folds and creases. Moderate swelling.
  - Grade 3 – Confluent, moist desquamation greater than 1.5 cm diameter, which is not confined to the skin folds. Pitting oedema (severe swelling).
  - Grade 4 – Skin necrosis or ulceration of full thickness dermis (middle layer of skin).
- **Chronic radiodermatitis** occurs with chronic exposure to "sub-erythema" doses of ionizing radiation over a prolonged period, producing varying degrees of damage to the skin and its underlying parts after a variable latent period of several months to several decades.

Chronic radiation-induced changes in the skin are characterised by:

  - Disappearance of follicular structures (pores)
  - Increase in collagen and damage to elastic fibres in the dermis
  - Fragile surface skin (epidermis)
  - Telangiectasia (prominent blood vessels).
- **Other: Eosinophilic, polymorphic, and pruritic eruption associated with radiotherapy** is a skin condition that occurs most often in women receiving cobalt radiotherapy for internal cancer.

Risk factors for radiation dermatitis

Radiation-induced dermatitis is more likely to occur in patients with certain risk factors:

- Poor nutrition
- Pre-existing skin disease
- Application of skin creams to exposed area immediately before treatment

- Overlapping skin folds
- Obesity
- Prolonged or multiple procedures requiring radiation exposure
- Total radiation doses of greater than 55 Gy, or large individual doses per fraction (greater than 3–4 Gy per dose)
- Concurrent cetuximab therapy, in patients receiving radiation for head and neck cancer.

General management of radiation dermatitis:

Patients with acute radiation dermatitis should be carefully assessed.

- Check that the radiation dose and distribution are correct
- Consider discontinuing concomitant medication that may have contributed to the reaction.
- Consider alternative explanations for the skin changes, such as contact dermatitis or infection.

- Patients may wash the affected skin with a gentle non-soap cleanser and dry it with a soft, clean towel before each irradiation session. Emollients, moisturisers, gels, emulsions and dressings applied after treatment may reduce discomfort.
- Topical corticosteroids may be prescribed for radiation dermatitis for 2 to 4 weeks. It is uncertain whether these are of benefit.

Patients receiving radiation therapy should be advised to avoid:

- Sun exposure by covering the treated area with protective clothing or SPF 50+ broad-spectrum sunscreen.
- Topical skin irritants, such as perfumes, deodorants and alcohol-based lotions
- Scratching of the skin in the affected area.

e) Spinal Anesthesia. See the Question 3.c of Group – C of Paper –II of 2013

The West Bengal University of Health Sciences

M.B.B.S. 3rd Professional Part – II Examination, 2010

Subject: Surgery **Time: $2^{1}/_{2}$ hrs.**

Paper: II **Marks: 60**

Group –A

1)a) What are the functions of parathormone? Write in detail about clinical, feature, investigations and management of hyperparathyroidism. 3+4+4+4 = 15

Answer. See the Question no 2.b of Group – B of Supplementary Paper – II of 2014 for primary hyperparathyroidism.

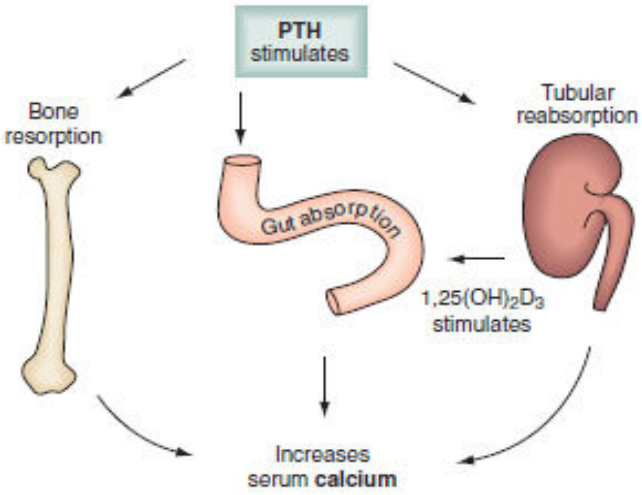
Functions of parathormone:

- Calcium regulation:

Actions of Major Calcium-Regulating Hormones:

HORMONE	BONE	KIDNEY	INTESTINE
Parathyroid hormone	Stimulates resorption of calcium and phosphate	Stimulates resorption of calcium and conversion of $25(OH)D_3$; inhibits resorption of phosphate and bicarbonate	No direct effects
Vitamin D	Stimulates transport of calcium	Inhibits resorption of calcium	Stimulates calcium and phosphate absorption
Calcitonin	Inhibits resorption of calcium and phosphate	Inhibits resorption of calcium and phosphate	No direct effects

Calcium homeostasis and PTH:



Secondary Hyperparathyroidism in Renal Failure

- Renal osteodystrophy- osteitis fibrosa cystica, osteomalacia, and adynamic bone disease.
- It is associated with osteopenia, bone cysts, brown tumors, and decreased bone strength resulting in long bone fractures because of dystrophic bone formation.
- Osteomalacia is characterized by lower bone turnover, mineralization deficiency, and accumulation of unmineralized osteoid.
- Adynamic bone disease is characterized by hypocellular bone surfaces with little or no evidence of remodeling, and it is common in patients with normal or low PTH or severe diabetes and aluminum intoxication. It has been associated with long-term peritoneal dialysis. It can cause fractures and microfractures leading to bone pain.

Treatment:

- Treat renal failure.
- Surgery: Subtotal parathyroidectomy seems to be the preferred surgical approach in most, but not all patients.
- The residual parathyroid tissue in the neck or forearm will grow and cause recurrent disease if survival is prolonged and patients do not receive a renal transplant.
- Nodular proliferation in glands seems to predispose to recurrence more often than homogeneous gland hyperplasia does.
- Cryopreservation of excised tissue (if available) is a good strategy when total parathyroidectomy with autotransplantation is planned in the event that the autograft is nonfunctional.

Tertiary HPT occurs in two settings

- The first is in a subset of patients with secondary HPT in which the parathyroid glands become autonomous and hypercalcemia develops.
- The second was first recognized by St. Goar, who described how secondary HPT can persist even after patients underwent renal transplantation; he postulated that the parathyroids became autonomous.
    - Theoretically, reversal of parathyroid hyperplasia should be expected after successful renal transplantation.
    - Nevertheless, studies show that hypercalcemia can persist in 8.5% to 53% of transplant recipients.
    - Of these, less than 1% require parathyroidectomy for tertiary HPT.
    - Transplant patients may have additional factors that can contribute to persistent tertiary HPT; glucocorticoids, cyclosporine, thiazide diuretics, and alterations in the glomerular filtration rate as a result of tubular injury or rejection episodes can influence parathyroid function and bone response.
    - Accordingly, patients with severe secondary HPT should not undergo renal transplantation until their secondary HPT has been treated.
    - Surgical treatment of tertiary HPT after renal transplantation is not common and is reserved for patients without resolution of symptoms, patients with hormonal and chemical abnormalities such as elevated or increasing iPTH levels and an increase in serum calcium to greater than 12.0 mg/dL that persists more than 1 year after

transplantation, and patients with acute hypercalcemia (calcium level >12.5 mg/dL) in the immediate post-transplant period.

Or

b) Give differential diagnosis of scrotal swelling. Write in detail about management of testicular tumour. 5+10 = 15

Answer.

Differential diagnosis of scrotal swelling: **See the answer of Question 4.a of Group – D of Supplementary Paper –II of 2013.**

Management of testicular tumour: **See the answer of Question 2.b of Group – B of Paper –II of 2014.**

Group – B

2. Write short notes on (any three): 3x5 = 15

a) Ectopia vesicae.
b) Neurofibromatosis.
c) Paget's disease of nipple.
d) Fistula in ano.
e) Varicocele.

Answer.

a) Ectopia vesicae: Exstrophy of the bladder, the medical term for which is ectopia vesicae is a congenital malformation of the urinary bladder.

A part of the wall of the bladder in front is absentand the inner part of the bladder is exposed. It is a rare condition affecting one in 50,000 people.

Types of ectopia vesicae:

- Complete exstrophy - In this case, the ends of the pubic bones in front of the pelvis are widely separated (normally they fuse before birth). The abdominal muscles in front also separate, the umbilicus may be absent, and there may be an umbilical or inguinal hernia associated with it. In males, the urethral opening at the tip of the penis is placed on the upper part of the shaft of the penis and the penis curves upwards; this is known as epispadias and almost always accompanies this condition.

- Incomplete exstrophy- Here, the pubic bones are fused while the external genital organs may be normal or there may be a minor defect where the urethra opens at the tip of the penis.

Causes of ectopia vesicae:

1. The cause is congenital due to failure of development of the front wall of the urinary bladder and along with it failure of the pubic bones to fuse. The abdominal muscles in front also fail to develop causing a defect in the abdominal wall.
2. It is seen more often in males.

Symptoms and signs of ectopia vesicae:

- The sides of the bladder are attached to the skin of the abdomen in front.
- The inner lining of the bladder has a tendency to bleed easily and get infected as it is exposed to the air.
- Urine which enters the bladder from the kidneys via the ureters will leak outside the body causing further infection and unhygienic conditions.
- The umbilicus may be absent, and there may an umbilical hernia (some tissue protruding through the umbilical opening)or there may be an inguinal hernia (tissue protruding through a defect or weakness in the lower abdominal wall near the groin.
- Epispadias of the penis is seen. The urethral opening which is normally at the tip of the penis is somewhere on the upper side of the shaft of the penis. This causes difficulty in urinating. The penis is usually shorter and broader than normal.
- The testes may be in another area, not in the normal position in the scrotum (ectopic testes)and the scrotum may not be completely developed.
- In females, the clitoris may be cleft (as if divided into two)and the two labia minora are separated. The uterus and vagina may be abnormal.
- The anal opening is loose or lax.

Complications of ectopia vesicae:

- Hydronephrosis - The kidneys may enlarge and have multiple bubbles in it called hydronephrosis; this happens when there is an obstruction to the passage of urine in the ureters.
- The inner lining of the urinary bladder may change causing cancer.
- Infection in the bladder spreads upwards to the ureters and kidneys. Recurrent kidney infection may occur.
- The person always smells of urine, and it is socially embarrassing.
- The child goes into renal failure which may be fatal.

Diagnosis of ectopia vesicae:

The diagnosis happens at birth if not earlier.

Treatment of ectopia vesicae:

Surgery is the only way to treat exstrophy of the bladder. The operation is usually done when the child is between four and six years of age. In the first step the urine has to be diverted by creating an opening in the lower part of the intestine. If this is successful, the surgery to repair the bladder wall, repair other defects in the abdominal muscles or hernia is done simultaneously.

This procedure is not very successful as diverting the urine to the intestine leads to narrowing of the opening or infection time and again.

Nowadays, surgery is attempted when the child turns one. The bladder is reconstructed, and the anal opening is tightened to prevent incontinence.

b. Neurofibromatosis:

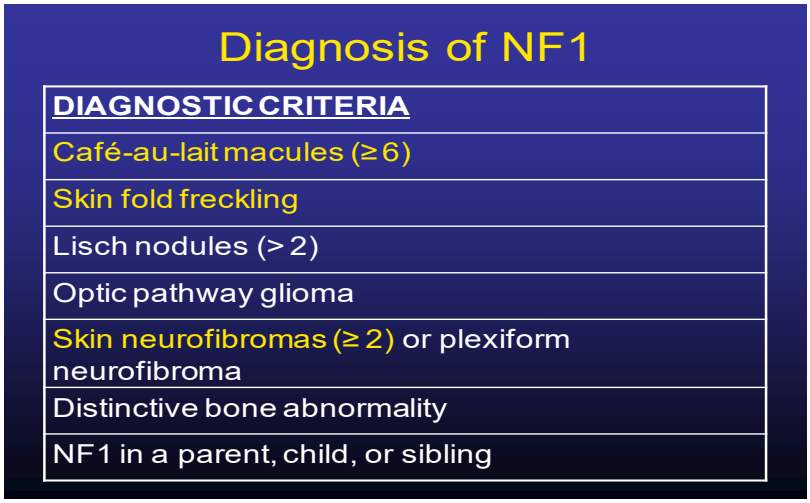
Introduction: Neurofibromatosis is a genetic disorder that disturbs cell growth in your nervous system, causing tumors to form on nerve tissue. These tumors may develop anywhere in your nervous system, including in your brain, spinal cord and nerves. Neurofibromatosis is usually diagnosed in childhood or early adulthood.

Types: Three distinct types of neurofibromatosis exist, each with different signs and symptoms.

- **Neurofibromatosis 1 (NF1):**

Neurofibromatosis 1 (NF1) usually appears in childhood. Signs and symptoms include:

- Flat, light brown spots on the skin
- Freckling in the armpits or groin area.
- Soft bumps on or under the skin (neurofibromas).
- Tiny bumps on the iris of your eye (Lisch nodules).
- Bone deformities.
- Learning disabilities.
- Larger than average head size
- Short stature.



- **Neurofibromatosis 2 (NF2):**

Neurofibromatosis 2 (NF2) is much less common than NF1. Signs and symptoms of NF2 usually result from the development of vestibular schwannomas (also known as acoustic neuromas) in both ears.

These benign tumors grow on the nerve that carries sound and balance information from the inner ear to the brain (the eighth cranial nerve). Resulting signs and symptoms generally appear in the late teen and early adult years and may include:

- Gradual hearing loss
- Ringing in the ears
- Poor balance

In some cases, NF2 can lead to growth of schwannomas in other nerves of the body, including the cranial, spinal, visual (optic) and peripheral nerves. Associated signs and symptoms may include:

- Facial drop
- Numbness and weakness in the arms or legs
- Pain
- Balance difficulties

In addition, NF2 may result in vision problems due to abnormal growth on the retina (mostly in children) or due to the development of cataracts.

- **Schwannomatosis:**

Schwannomatosis is a rare form of neurofibromatosis only recently recognized. It rarely affects people before their 20s or 30s. Schwannomatosis causes painful tumors called schwannomas to develop on cranial, spinal and peripheral nerves, but not on the nerve that carries sound and balance information from the inner ear to the brain (the eighth cranial nerve).

Because tumors don't grow on this nerve, schwannomatosis doesn't cause hearing loss, making it different from NF2. As with NF2, though, schwannomatosis doesn't cause cognitive impairment. Schwannomatosis mainly causes chronic pain, which can occur anywhere in the body.

Causes:

Neurofibromatosis 1 (NF1): The NF1 gene is located on chromosome 17. Normally, this gene produces a protein called neurofibromin, which is abundant in nervous system tissue and helps regulate cell growth. A mutation of the NF1 gene causes a loss of neurofibromin, which allows cells to grow uncontrolled.

Neurofibromatosis 2 (NF2): A similar problem occurs with NF2. The NF2 gene is located on chromosome 22, which produces a protein called merlin. A mutation of the NF2 gene causes loss of merlin, which also leads to uncontrolled cell growth.

Schwannomatosis: Schwannomatosis may be associated with a mutation of the SMARCB1 gene located on chromosome 22. Other gene mutations may be involved in schwannomatosis. The occurrence of schwannomatosis may be inherited or may be sporadic (spontaneous), but these are not known yet.

Investigations:

- Physical examination and medical history. Diagnosis of neurofibromatosis 1 (NF1) based on a physical examination, checking for the characteristics of NF1.
- Eye exam. Examination by an eye doctor (ophthalmologist) can detect tiny bumps on the iris of your eye (Lisch nodules) and cataracts.

- Ear exam. Hearing and balance tests such as audiometry, electronystagmography and brainstem auditory evoked response can help determine the level of hearing and balance function in a person with NF2.
- Imaging tests. Imaging tests, such as X-rays, CT scans and MRIs, aren't always required, but they can help identify bone abnormalities, deep tumors in the brain or spinal cord, and very small tumors. An MRI can also help identify optic pathway gliomas in your eye.Imaging tests may also be particularly helpful in monitoring NF2 and schwannomatosis.
- Genetic tests.

Treatment: Neurofibromatosis can't be cured, but one should monitor for complications and treat the symptoms.

c) Paget's disease of nipple.

Introduction:

Paget disease of the nipple is a rare type of cancer involving the skin of the nipple and the areola. Most people with Paget disease of the breast also have one or more tumors inside the same breast. These breast tumors are either ductal carcinoma in situ or invasive breast cancer.

Malignant cells known as Paget cells are a telltale sign of Paget disease of the breast. These cells are found in the epidermis (surface layer) of the skin of the nipple and the areola. Paget cells often have a large, round appearance under a microscope; they may be found as single cells or as small groups of cells within the epidermis.

Aetiology:

- Paget disease of the breast occurs in both women and men, but most cases occur in women.
- Approximately 1 to 4 percent of all cases of breast cancer also involve Paget disease of the breast.
- The average age at diagnosis is 57 years, but the disease has been found in adolescents and in people in their late 80s.

Cause: The most widely accepted theory is that cancer cells from a tumor inside the breast travel through the milk ducts to the nipple and areola. This would explain why Paget disease of the breast and tumors inside the same breast are almost always found together.

Symptoms:

- Itching, tingling, or redness in the nipple and/or areola
- Flaking, crusty, or thickened skin on or around the nipple
- A flattened nipple
- Discharge from the nipple that may be yellowish or bloody

Because the early symptoms of Paget disease of the breast may suggest a benign skin condition, and because the disease is rare, it may be misdiagnosed at first. People with Paget disease of the breast have often had symptoms for several months before being correctly diagnosed.

Diagnosis:

Nipple biopsy:

- Surface biopsy: A glass slide or other tool is used to gently scrape cells from the surface of the skin.
- Shave biopsy: A razor-like tool is used to remove the top layer of skin.
- Punch biopsy: A circular cutting tool, called a punch, is used to remove a disk-shaped piece of tissue.
- Wedge biopsy: A scalpel is used to remove a small wedge of tissue.

Additional diagnostic tests:

Diagnostic mammogram, an ultrasound exam, or a magnetic resonance imaging scan to look for possible tumors.

Treatment:

- Mastectomy, with or without axillary lymph node dissection.
- Studies have shown, however, that breast-conserving surgery that includes removal of the nipple and areola, followed by whole-breast radiation therapy, is a safe option for people with Paget disease of the breast who do not have a palpable lump in their breast and whose mammograms do not reveal a tumor.
- People with Paget disease of the breast who have a breast tumor and are having a mastectomy should be offered sentinel lymph node biopsy to see whether the cancer has spread to the axillary lymph nodes. If cancer cells are found in the sentinel lymph node(s), more extensive axillary lymph node surgery may be needed .
- Depending on the stage and other features of the underlying breast tumor (for example, the presence or absence of lymph node involvement, estrogen and progesterone receptors in the tumor cells, and HER2 protein overexpression in the tumor cells), adjuvant therapy, consisting of chemotherapy and/or hormonal therapy, may also be recommended.

d) Fistula – in – ano. See the Question 3.e of Group – C of Paper –II of 2014.

e) Varicocele

Definition: A varicocele is an abnormal enlargement of the pampiniform venous plexus in the scrotum. This plexus of veins drains the testicles.

Pathogenesis: The testicular blood vessels originate in the abdomen and course down through the inguinal canal as part of the spermatic cord on their way to the testis. Upward flow of blood in the veins is ensured by small one-way valves that prevent backflow. Defective valves, or compression of the vein by a nearby structure, can cause dilatation of the testicular veins near the testis, leading to the formation of a varicocele.

Causative factors:

- 8-10 cm longer left testicular Vv. → increased hydrostatic pressure in upright position
- Entry of left testicular Vv into renal vein at 90^0
- "Nutcraker phenomenon" due to passage of left testicular vein between SMA & Aorta

- Congenital absence of valve in left vein in 40%
- Intrinsic ectasia of plexus due to cremaster atrophy
- Loaded left colon
- Left sided RCC.

Clinical features:

- A varicocele often produces no signs or symptoms.
- Rarely, it may cause pain. The pain may:
  - Vary from sharp to dull discomfort
  - Increase with standing or physical exertion, especially over long periods
  - Worsen over the course of a day
  - Be relieved when lying on back
- Visible or palpable (able to be felt) enlarged vein.
- Feeling of heaviness in the testicle(s)
- Atrophy (shrinking) of the testicle(s)
- Alteration of testosterone levels
- Benign prostatic hyperplasia (BPH) and related urinary problems.

Clinical examinations:

- Increases on standing.
- "Bag of worm" feel
- Grades of varicocele:
  Grade I – Palpable only during valsulva maneuver
  Grade II – Palpable without Valsulva in standing upright position
  Grade III – Visible through scrotal skin

  Subclinical – detected during USG

Investigations:

- Doppler stethoscope (5.3 MHz probe) -audible rush of blood on valsulva
- Colour Doppler –detects Sub Clinical Varicocele also
- Ultra sound of abdomen.
- Semen examination

Treatment:

- Expectant treatment – in adolescent males who are asymptomatic with normal size of testis

Indication of intervention:

- Asymptomatic varicocele with >20% volume loss of Testis (>2ml)
- Symptomatic varicocele
  - Impaired sperm quality
  - Pain
  - Cosmetic reasons

- Medically unfit.

Group – C

3. Write short notes on (any three) **3x5 = 15**

a) Subdural haematoma.
b) Muscle relaxants.
c) I^{131} scan.
d) Congenital hypertrophic pyloric stenosis.
e) Lumbar puncture.

Answers.

a) Subdural haematoma. See the answer of question no. 4.c of Group – D of paper –II of 2013.

b) Muscle relaxants. See the answer of question no. 4.b of Group – D of paper –II of 2014.

c) I^{131} scan. It is a form of application of nuclear medicine.

Role of I^{131} scan:

- Determine if the thyroid gland is working properly
- Help diagnose problems with the thyroid gland, such as an overactive thyroid gland, a condition called hyperthyroidism, cancer or other growths
- Assess the nature of a nodule discovered in the thyroid gland
- Detect areas of abnormality, such as lumps (nodules) or inflammation
- Determine whether thyroid cancer has spread beyond the thyroid gland
- Evaluate changes in the gland following medication use, surgery, radiotherapy or chemotherapy.

Benefits:

- Provides unique information—including details on both function and anatomic structure of the body that is often unattainable using other imaging procedures.
- It yields the most useful information needed to make a diagnosis or to determine appropriate treatment, if any.
- It is less expensive and may yield more precise information than exploratory surgery.

Risks:

- The radiation risk is very low compared with the potential benefits.
- Low risk of development of cancer.
- Injection of the radiotracer may cause slight pain and redness which should rapidly resolve.

Limitations:

- Pregnant women.
- Lactating/breastfeeding women.

d) Congenital hypertrophic pyloric stenosis.

- This is the most common surgical cause of non-bilious vomiting in infants.
- It occurs in 1 of 400 live births.
- The male-to-female ratio is 4:1.

Diagnosis:

- History and physical examination
  - Occurs generally in neonates who are 2 to 5 weeks of age.
  - Vomiting
    - Characteristically forceful or projectile and occurs 30 to 60 minutes after feeding.
    - Formula intolerance initially suspected but does not resolve with change of feeds.
  - Dehydration
    - Lethargy.
    - Absence of tears.
    - Sunken anterior fontanelle.
    - Dry mucous membranes.
    - Decreased urine output.
  - The "olive" mass
    - Mass palpated to the right and above the umbilicus.
    - Approximately 2 cm in diameter, firm, and mobile.
  - **Abdominal ultrasonography:**
    - Pyloric diameter greater than 14 mm, muscular thickness greater than 4 mm, and pyloric length greater than 16 mm are diagnostic of pyloric stenosis with 91% to 100% sensitivity and 100% specificity (J Pediatr Surg 1987; 22:950).
  - **Upper GI contrast study:**
    - Enlarged stomach.
    - Poor gastric emptying.
    - Elongated, narrow pyloric channel or "string sign."
- **Management:**
  - Preoperative fluid resuscitation
    - 20-mL/kg bolus.
    - 5% dextrose in normal saline to achieve urine output of 2 mL/kg/hour.
    - Addition of potassium and changing to 5% dextrose in 0.45% normal saline occurs when urine output is adequate.
  - Correction of the hypochloremic hypokalemic metabolic alkalosis.
  - **Operative intervention**
  - Indicated only after adequate resuscitation and correction of metabolic alkalosis.
  - Pyloromyotomy
    - Division of the hypertrophied pyloric muscle, leaving the mucosa intact.
    - Open or laparoscopic technique.

- Postoperative feeding
  - Begin electrolyte solution by mouth 6 hours after pyloromyotomy.
  - Over the next 12 hours, formula or pumped breast milk can be started and should reach goal within 24 hours.
  - Parents should be advised that vomiting may occur postoperatively as a result of swelling at the pyloromyotomy, but this problem is self-limited.
  - If the pyloric mucosa is perforated and repaired during surgery, nasogastric drainage is recommended for 24 hours.

e) Lumbar puncture.

Introduction:

A lumbar puncture is adiagnostic and at times therapeutic medical procedure. Diagnostically it is used to collect cerebrospinal fluid (CSF) to confirm or exclude conditions such as meningitis and subarachnoid hemorrhage and it may be used in diagnosis of other conditions. Therapeutically it may be used to reduce increased intracranial pressure. Under some circumstances, lumbar puncture cannot be performed safely (e.g. a severe bleeding tendency). It is regarded as a safe procedure, but post-dural-puncture headache is common.

Indication:

- Suspicion of meningitis
- Suspicion of subarachnoid hemorrhage (SAH)
- Suspicion of central nervous system (CNS) diseases such as Guillain-Barré syndrome and carcinomatous meningitis
- Therapeutic relief of pseudotumor cerebri

Contraindication:

Absolute contraindications for lumbar puncture are the presence of infected skin over the needle entry site and the presence of unequal pressures between the supratentorial and infratentorial compartments.

The latter is usually inferred from the following characteristic findings on computed tomography (CT) of the brain:

- Midline shift
- Loss of suprachiasmatic and basilar cisterns
- Posterior fossa mass
- Loss of the superior cerebellar cistern
- Loss of the quadrigeminal plate cistern

Relative contraindications for lumbar puncture include the following:

- Increased intracranial pressure (ICP)
- Coagulopathy

- Brain abscess.

Side effects:

- Bach-ache.
- Headache.
- Swelling and bruising.

Group – D

4. Answer briefly on (any three): 3x5 = 15

a) Ludwig's angina.
b) Meningo-myelocele.
c) Empyema thoracis.
d) Referred pain.
e) Patent ductus arteriosus.

Answer.

a) Ludwig's angina.

Introduction: Ludwig's angina, otherwise known as Angina Ludovici, is a serious, potentially life-threatening cellulitis, or connective tissue infection, of the floor of the mouth, usually occurring in adults with concomitant dental infections and if left untreated, may obstruct the airways, necessitating tracheotomy.

Aetiology:

- Dental caries, recent dental treatment, poor dental hygiene (accounts for 75-90% of cases)
- Mixed infections, due to both aerobes and anaerobes, are of the cellulitis associated with Ludwig's angina. Typically, these include alpha-hemolytic streptococci, staphylococci and bacteroides groups.
- Trauma: mandibular fracture, facial trauma, tongue piercing, frenuloplasty
- Infections of oral malignancy
- Submandibular sialadenitis
- Systemic compromise such as AIDS, glomerulonephritis, diabetes mellitus, aplastic anemia, transplant recipients, chemotherapy; IVDA.

Clinical features:

- Bilateral 'wood like' swelling in the submandibular, sublingual and submental spaces
- Double chin appearance
- Skin is tense and tends to pit and blanch on pressure
- Rapidly spreading edema

- Edema and congestion of floor of the mouth
- Elevation and protrusion of tongue
- Elevation of the tongue is associated with dysphagia, odynophagia, dysphonia and cyanosis.
- Dysphagia and drooling of saliva
- Dyspnea in supine position →impending laryngeal edema
- Septicemia: High grade fever, Malaise, Body aches, Leukocytosis.

Investigations:

- Laboratory tests – hemogram, blood glucose, etc.
- Panoramic x-ray – to identify possible odontogenic sources
- Cervical, profile and posterior-anterior radiographs – to observe the volume increasing in the soft tissues and any deviation of the trachea
- Ultra sound has been recommended to differentiate between cellulitis, abscess and adenopathy in head and neck infection
- USG has a sensitivity of 95% and specificity of 75%
- CT scan:
    - CT scan is most widely used modality
    - Readily available, can localize abscesses in the head and neck
    - Not as effective as ultrasound in determining abscess from cellulitis.

Treatment:

- Airway management.
- Medical treatment;
    - Intravenous access, fluid resuscitation, and administration of IV antibiotics
    - Antibiotic therapy should be administered empirically and tailored to culture and sensitivity results.
    - Antibiotic therapy should be administered empirically and tailored to culture and sensitivity results
- Surgical treatment:
    - Incision and drainage: Bilateral submandibular incisions as well as a midline submental incision.Incision approximately 3 to 4 cm below the angle of the mandible and below the inferior extent of swelling roughly parallel to the inferior border of mandible.

b) Meningo-myelocele. Meningomyelocele is a congenital malformation that arises due to a failure of the neural tube to close. The spinal cord and the surrounding dura and arachnoid mater

herniates through a defect in the vertebral column. The herniation damages the nerves that exit the vertebral column below the location of the swelling, causing paralysis.

Aetiopathogenesis:

- Myelomeningocele, the most common type of spina bifida aperta
- Has an average incidence of 1/1000 live births.
- In this disorder, there is protrusion of a varying amount of spinal neural tissue outside the spinal canal confines.
- It has been associated with folate deficiency in the mother; intake of folate during pregnancy has reduced the incidence considerably.
- There is a deficiency of the skin, muscle, and bony elements, with the open neural placode exposed anywhere from the thoracic to sacral levels.

Clinical features:

- Varying degrees of motor and sensory deficits with autonomic (bladder and bowel) dysfunction accompany this defect.
- The degree of the deficit is directly related to the level of the defect, which often determines the child's capability to ambulate in the future. Hence, thoracic defects have the highest incidence of weakness and sacral defects often have only bladder involvement.
- Hydrocephalus is also present in 80% of patients and sometimes manifests after surgical closure of the defect.
- The other significant association is the Chiari II malformation.
- Associated brain anomalies include corpus callosal anomalies, fused tectal plates, and thalamic fusion.

Diagnosis: Serum and amniotic fluid α-fetoprotein screening and prenatal ultrasound have been significantly helpful in diagnosing open neural tube defects in the prenatal period.

Treatment:

- Surgical closure of the myelomeningocele is undertaken within 24 to 48 hours of birth to avoid CNS infection (e.g., meningitis, ventriculitis).
- Ventricular shunts, if indicated, are placed concurrently with myelomeningocele closure or at a later date.

c) Empyema thoracis. See the Question 3.c of Group – C of Supplementary Paper –II of 2013.

d) Referred pain. Referred pain, also called reflective pain, is pain perceived at a location other than the site of the painful stimulus. An example is the case of ischemia brought on by a myocardial infarction (heart attack), where pain is often felt in the neck, shoulders, and back rather than in the chest, the site of the injury.

Characteristics:

- The size of referred pain is related to the intensity and duration of ongoing/evoked pain.
- Temporal summation is a potent mechanism for generation of referred muscle pain.
- Central hyperexcitability is important for the extent of referred pain.

- Patients with chronic musculoskeletal pains have enlarged referred pain areas to experimental stimuli. The proximal spread of referred muscle pain is seen in patients with chronic musculoskeletal pain and very seldom is it seen in healthy individuals.
- Modality-specific somatosensory changes occur in referred areas, which emphasize the importance of using a multimodal sensory test regime for assessment.
- Referred pain is often experienced on the same side of the body as the source, but not always.

Examples:

Location	Description
Upper chest/left limb	Myocardial ischaemia (the loss of blood flow to a part of the heart muscle tissue) is possibly the best known example of referred pain; the sensation can occur in the upper chest as a restricted feeling, or as an ache in the left shoulder, arm or even hand.
Head	“Ice-cream headache” or “brain freeze” is another example of referred pain, in which the vagus nerve or thetrigeminal nerve in the throat and the palate, respectively, transmit pain signals, because of the rapid cooling and rewarming of the capillaries in the sinuses.
General	Phantom limb pain, a type of referred pain, is the sensation of pain from a limb that has been lost or from which a person no longer receives physical signals. It is an experience almost universally reported by amputees and quadriplegics.
Right tip ofscapula.	Liver, gallbladder.
Left shoulder	Thoracic diaphragm, Spleen (Kehr's sign), lung
Back	Pancreas
Palm of Hand	Palmaris longus. A problem originating in the forearm might be felt in the palm, and not in the forearm. Appearances can be deceiving.

e) Patent ductus arteriosus. See the Question 3.b of Group – C of Supplementary Paper –II of 2014.

The West Bengal University of Health Sciences

M.B.B.S. 3rd Professional Part – II Examination, 2009

Subject: Surgery **Time: $2^{1}/_{2}$ hrs.**

Paper: II **Marks: 60**

Group –A

1) a) A 35 year old lady presents with a Solitary nodule in right lobe. How would you come to a diagnosis and manage such a patient? 8+7 = 15

Answer. The solitary thyroid nodule is defined as a discrete palpable swelling in an otherwise impalpable gland.

It is a clinical diagnosis. Many of these cases prove to be multinodular but presenting as a single thyroid nodule.

Differential diagnosis of apparent solitary thyroid nodules:

<table>
<tr><th colspan="2">1) Benign thyroid neoplasms</th><th>2) Malignant thyroid neoplasms</th><th>3) Other thyroid abnormalities</th><th>4) Nonthyroid lesions</th></tr>
<tr><td rowspan="5">a) Follicular adenoma
i) Colloid
ii) Simple
iii) Foetal
iv) Embryonal
v) Hurthle cell</td><td>b) Papillary adenoma</td><td rowspan="5">a) Papillary carcinoma
b) Follicular Carcinoma
c) Medullary carcinoma
d) Anaplastic carcinoma
e) Metastatic cancer
f) Sarcoma
g) Lymphoma</td><td rowspan="5">a) Thyroiditis
b) Thyroid cyst
c) Infections
d) Granulomatous disease (e.g., sarcoidosis)</td><td rowspan="5">a) Lymphadenopathy
b) Thyroglossal duct cyst
c) Parathyroid adenoma
d) Laryngocele</td></tr>
<tr><td>c) Teratoma</td></tr>
<tr><td>d) Lipoma</td></tr>
<tr><td>e) Dermoid cyst</td></tr>
</table>

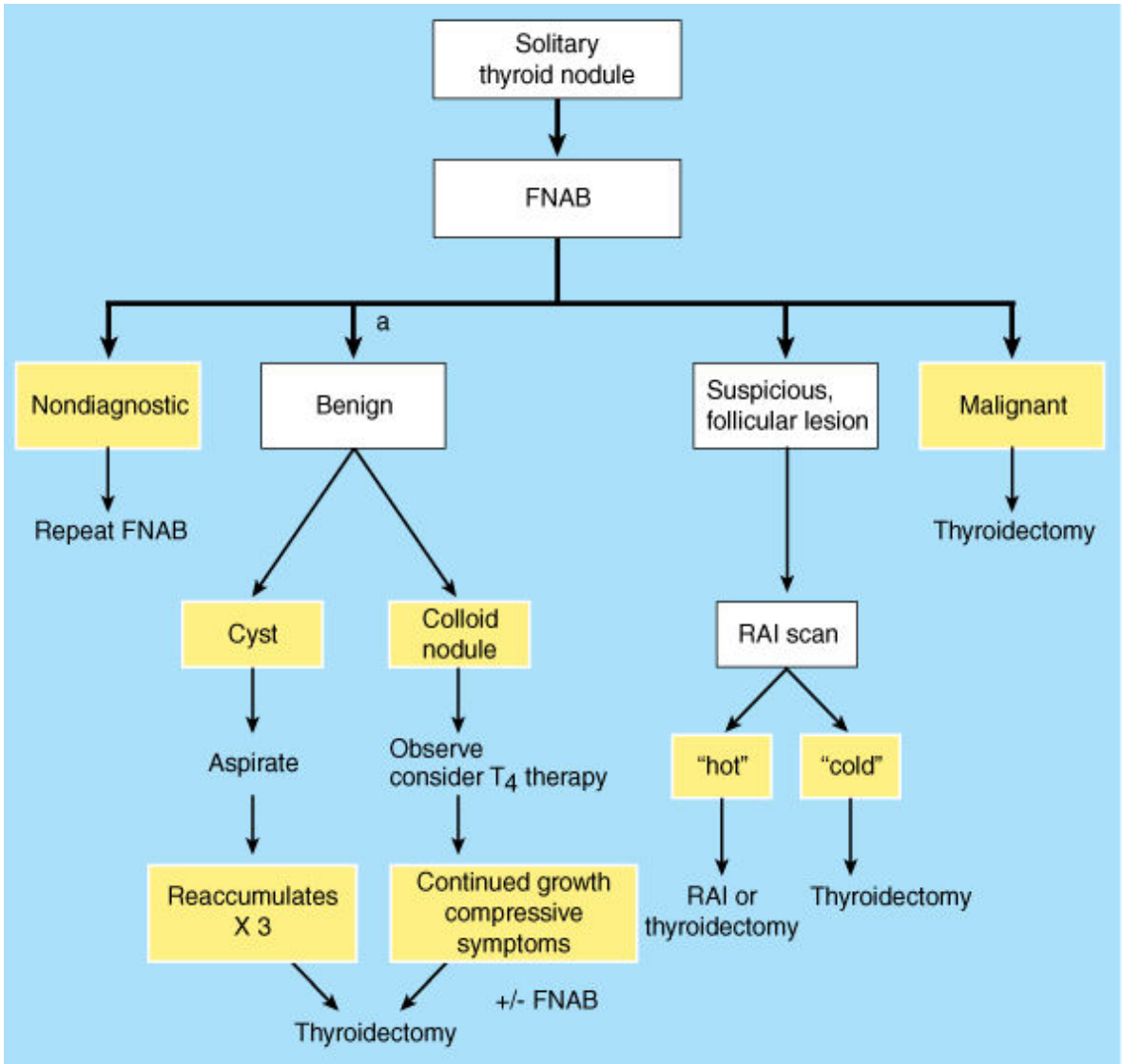
Diagnostic Tools of Solitary Thyroid Nodule:

<table>
<tr><td>Clinical examination</td><td rowspan="2">Radiological studies:
• Neck ultrasonography
• Isotope scanning of the thyroid
• CT scan</td><td rowspan="2">Histopathological studies:
• Fine needle aspiration cytology
• Frozen section .
• Final histopathological examination.</td></tr>
<tr><td>Laboratory studies: T3,T4,TSH</td></tr>
</table>

Treatment of the Solitary Cold Thyroid Nodule:

Non-Surgical:	Surgical:
-No treatment, just follow-up by FNAC -Hormone suppressive therapy -Aspiration of a cyst -Ethanol injection -Recently, Laser photocoagulation	-Isthmo-lobectomy -Near total thyroidectomy -Total thyroidectomy

Workup of a solitary thyroid nodule:



Or

b) Classify kidney tumours. Mention different modes of presentation of Renal Adeno Carcinoma. Outline the management of such a patient. 3+5+7 =15

Answer. See the Question. 2.a of Group – B of Supplementary Paper –II of 2013.

Group – B

2) Write short notes on (any three): 3x5 = 15

a) Salivary calculi.
b) Fournier's gangrene.
c) Breast abscess.
d) M.E.N syndrome.
e) Complications of undescended testis.

Answer.

a) Salivary calculi.

Introduction:

- Salivary gland calculi occur most commonly within the submandibular gland (80%) and to a lesser extent the parotid.
- Composed of calcium phosphate and carbonate and may be related to sialadenitis (inflammation of a salivary gland).
- Most common in adults.

Clinical features:

- Pain and swelling of the affected gland on eating and drinking.
- If there is partial obstruction of the duct the swelling can last minutes or several hours.
- Complete obstruction leads to persistent swelling and infection.
- The patient may also experience colicky pain in the duct when eating.

Points in the examination of the submandibular gland:

- Examine the gland from behind and feel the swelling by running the finger backwards under the jaw. If you cannot feel a lump ask the patient to suck a sour sweet and re-examine them
- Examine the duct orifice from the front. Ask the patient to open their mouth wide and point their tongue upwards. The ducts lie near the midline at the root of the tongue. Look for redness, pus and impacted stone
- Examine the gland bimanually from the front. Wear gloves and place the finger of one hand over the gland. The index of the other hand is placed in the mucosal surface of the mandible and the gland palpated between the two

Diagnosis and investigations:

- Radiographs of the submandibular gland and duct and parotid gland and duct are helpful, although many calculi are not radio-opaque.
  - Stone in the parotid gland itself may be seen if a tangential view of the cheek is taken.
  - Lower occlusal X-ray of the teeth will show a stone in the submandibular duct.
  - A lateral oblique X-ray of the mandible will show a calculus in the submandibular gland.
- Submandibular duct radiography (sialography) is technically difficult and rarely gives more information than plain radiographs, although the flushing effect of the radio-opaque dye may give a therapeutic benefit.
- Parotid sialography may show a filling defect even when a calculus is invisible on plain X-ray. May provide considerable therapeutic benefit due to the flushing effect.

Treatment:

- Stones in the intra-oral part of the ducts can be removed under general anaesthesia.
- Stones in the submandibular gland – treatment is removal of the submandibular gland.
- Removal of a calculus from the parotid gland is a rare operation
- Most parotid gland disease is treated conservatively with sialogogues and intermittent massage of the gland towards the duct.

b) Fornier's gangrene.

Definition: Named after French venereologist Jean Alfred Fournier (1883).

Fournier gangrene is defined as a polymicrobial necrotizing fasciitis of the perineal, perianal, or genital areas.

Aetiology and risk factors:

- Initially described as idiopathic
- Now in more than 75% cases inciting cause in known
- Necrotizing process commonly originates from infection in anorectum, urogenital tract or skin of genitalia.

Aetiology:

Ano-rectal causes	Uro-genital causes
• Infection in the perineal glands • Manifestation of colorectal injury, • Malignancy or diverticulitis	• Infection in the bulbourethral glands • Urethral injury • Iatrogenic injury • Lower urinary tract infections

Pathogenesis:

- Bacteria act synergistically causing obliterative endarteritis & production of various enzymes causing destruction
- There is imbalance between host immunity & virulence of organism.
- Bacteriology: The main are Staphylococcus aureus, β-hemolytic Streptococcus, Pseudomonas sp., E. coli and Klebsiella sp. Uncommon: Clostridia, Bacteroides and Cornybacteria.

Mechanism of spread

Entry of bacteria (act through synergism)
↓
Fibrinoid coagulation of nutrient vessels
↓
Decreased locally blood supply to skin
↓
Decreased tissue oxygen tension
↓
Growth of anaerobes & microaerophilic organisms
↓
Production of enzyme (Collagenase, Lecithinase, Hyaluronidase)
↓
Digestion of fascial barrier
↓
Rapid spread of infection

Incidence:

- Age – 30 – 60 years
- Sex – 10 times more common in males
- Social habits – More common in male homosexuals (more prone for Rectal injury)

Clinical features:

- Begins with insidious onset of pruritus and discomfort of external genitalia
- Prodromal symptoms of fever and lethargy.
- The hallmark of Fournier gangrene is out of proportion pain and tenderness in the genitalia.
- Increasing genital pain and tenderness with progressive erythema of the overlying skin
- Dusky appearance of the overlying skin; subcutaneous crepitation; feculent odor
- Obvious gangrene of a portion of the genitalia; purulent discharge from wounds
- As gangrene develops, pain subsides (Nerve necrosis)

Investigations:

Laboratory	Imaging
• (CBC) Complete blood count • Electrolytes • BUN / Serum creatinine • Blood Sugar • ABG • Blood and urine culture with sensitivity • Coagulation profile for DIC	• Conventional radiography • Ultrasonography • C.T. Scanning • MRI

Treatment:

Medical	Surgical
1. Restoration of normal organ perfusion 2. Reduction of systemic toxicity 3. Broad spectrum antibiotics to cover anaerobes as well (ciprofloxacin+clindamycin+metronidazole) 4. Vancomycin for MRSA 5. Tetanus prophylaxis 6. Irrigation with super oxidised water 7. Hyperbaric oxygen therapy 8. IV immunoglobulins to neutralize super antigen as streptotoxin A & B (as adjuvant) 9. Antifungal – if required	• Repeated aggressive debridement • Shameful exposure of testis • Preservation of testes (subcutaneous pocket from desiccation) • Reconstruction after infection is over • Fecal diversion • Urinary diversion • Vacuum assisted closure (VAC)

Complications:

• ARF	• MSOF
• ARDS	• Tetanus
• Septicemia and gram negative shock	• Death

c) Breast abscess.

A. **Lactational mastitis:** Lactational mastitis may occur either sporadically or in epidemics.

- The most common causative organism is Staphylococcus aureus.
- It presents as a swollen, erythematous, and tender breast; purulent discharge from the nipple is uncommon.

- In the early cellulitic phase, the treatment is antibiotics. The frequency of nursing or pumping should be increased. Approximately 25% progress to abscess formation.
- Breast abscesses occur in the later stages and are often not fluctuant.
- The diagnosis is made by failure to improve on antibiotics, abscess cavity seen on ultrasound, or aspiration of pus.
- Treatment is cessation of nursing and surgical drainage.

B. **Nonpuerperal abscesses**
Nonpuerperal abscesses result from duct ectasia with periductal mastitis, infected cysts, infected hematoma, or hematogenous spread from another source.

- They usually are located in the peri/retroareolar area.
- Anaerobes are the most common causative agent, although antibiotics should cover both anaerobic and aerobic organisms.
- Treatment is surgical drainage.
- Unresolved or recurring infection requires biopsy to exclude cancer. These patients often have a chronic relapsing course with multiple infections requiring surgical drainage.
- Repeated infections can result in a chronically draining periareolar lesion or a mammary fistula lined with squamous epithelium. Treatment is excision of the central duct along with the fistula once the acute infection resolves. The fistula can recur even after surgery.

d) M.E.N syndrome.

Introduction:

- Multiple Endocrine Neoplasia (MEN) syndromes are familial conditions characterized by the occurrence of tumors involving two or more endocrine glands in a patient and family members.
- There are two major forms of MEN, namely MEN-type 1 (MEN1, →Wermer‘s syndrome) and MEN-type 2(MEN2, Sipple‘s syndrome); each form is characterized by the development of tumors of specific endocrine glands.
- The MEN syndromes are uncommon, but because they are inherited as autosomal dominant disorders, the finding of MEN in a patient has important implications for other family members. First degree relatives of a patient with known MEN have about a 50% risk of developing the disease.
- Occasionally, the MEN syndromes may arise sporadically (*i.e.* without a family history).

MEN 1:

- Autosomal dominant.
- The MEN1 gene was originally mapped to chromosome 11q13 by a combination of genetic linkage studies and tumor deletion mapping. It encodes a protein merlin.
- The MEN1 gene consists of 10 exons spanning 9 kb of genomic DNA and encodes a 610–amino acid protein product termed menin.

Features of MEN 1

Major disease components	Associated tumors
• Primary hyperparathyroidism • Pancreatic endocrine tumors (PET) • Anterior pituitary tumor	• Facial angiofibroma • Collagenoma • Adrenal cortical tumor • Lipoma • Foregut carcinoid

Components of MEN-1 with estimated penetrance (in parentheses) at age 40 year:

Endocrine features	Non-endocrine features
PHPT- Parathyroid hyperplasia (90%)	Lipomas (30%)
Entero-pancreatic tumor 1. **Gastrinoma** (40%) 2. Insulinoma (10%) 3. Non-functioning (**NF**) including **pancreatic polypeptide** (20%) 4. Other rare types: (2%) **glucagonoma** **VIPoma** **somatostatinoma** *etc.*	Facial angiofibromas (85%) Collagenomas (70%) Ependymoma (1%)
Foregut carcinoid **Thymic carcinoid** NF (2%) **Bronchial carcinoid** NF (2%)	
Gastric entero-chromaffin-like tumor NF (10%)	
Anterior pituitary tumor Prolactinoma (20%) Other: GH + PRL, GH, NF (each 5%) ACTH (2%), TSH (rare)	
Adrenal cortex NF (25%)	
Pheochromocytoma (<1%)	

- Parathyroid gland tumours. By age 40, 95% of patients have hypercalcaemia which is the commonest manifestation.
- Pancreatic islet cell tumours.
    - Prevalence of 30-75%.
    - Usually multicentric, slow-growing.
    - Secrete multiple polypeptides (insulin and gastrin commonest).

    - Gastrinoma leads to Zollinger-Ellison syndrome (recurrent and multiple peptic ulcers, severe reflux oesophagitis, and diarrhoea).
    - Rarer tumours are VIPoma, glucagonoma, somatostatinoma.
- Anterior pituitary tumours.
    - Detected in 15-40%.
    - Commonest is prolactinoma.
    - Rarer are GH- (causes acromegaly) or ACTH- (causes Cushing's disease) secreting tumours.

Carcinoid tumours (thymus, lungs, foregut), adrenal tumours, lipomas, and pinealomas have also been reported to appear in MEN-1 patients.

MEN 2A	**MEN2B**
Medullary thyroid carcinoma	Medullary thyroid carcinoma
Pheochromocytoma	Pheochromocytoma
Hyperparathyroidism	Marfanoid body habitus
Lichen planus amyloidosis	Mucosal neuromas
Hirschsprung's disease	Ganglioneuromatosis of the gastrointestinal tract

MEN 2:
Clinical Features of Sporadic MTC, MEN 2A, MEN 2B, and FMTC:

CLINICAL SETTING	**FEATURES OF MTC**	**INHERITANCE PATTERN**	**ASSOCIATED ABNORMALITIES**	**GENETIC DEFECT**
Sporadic MTC	Unifocal	None	None	Somatic *RET* mutations in >20% of tumors
MEN 2A	Multifocal, bilateral	Autosomal dominant	Pheochromocytomas, hyperparathyroidism	Germline missense mutations in extracellular cysteine codons of *RET*
MEN 2B	Multifocal, bilateral	Autosomal dominant	Pheochromocytomas, mucosal neuromas, megacolon, skeletal abnormalities	Germline missense mutation in tyrosine kinase domain of *RET*
FMTC	Multifocal, bilateral	Autosomal dominant	None	Germline missense mutations in extracellular or intracellular cysteine codons of *RET*

Treatment:
Surgical treatment:
MEN-1

- Parathyroidectomy.
- Pancreatic tumours: enucleation of individual tumours in the head of the pancreas and distal pancreatectomy for tumours in the tail/body.
- Hypophysectomy and external beam irradiation are considered for pituitary tumours.

MEN-2

- Total thyroidectomy (TT) indicated in patients identified by genetic screening. Symptomatic patients need TT and cervical nodal dissection for the lymph nodes on the involved side.
- Laparoscopic adrenalectomy for phaeochromocytoma.
- Parathyroidectomy for MTC in patients belonging to families in which hyperparathyroidism is frequently associated.

Medical treatment
MEN-1 Prolactinomas can be treated with dopamine agonists (bromocriptine/cabergoline).

e) Complications of undescended testis:

- **Infertility:**
  - Fertility is lower in individuals with UDT than those with normally descended testis and paternity rates are especially lower in those with bilateral UDT.
  - In the latter, even when orchidopexy is done early, paternity rates of only 23-50% are noted.
  - The causes of impaired fertility in these patients are:-
    - Testicular atrophy of UDT with increasing age (38% of testes are smaller than their counterparts by Tanner Stage 5 puberty)
    - Histopathological changes in UDT i.e. lack of normal enlargement of seminiferous tubules, decrease in ratio of spermatogonia per tubule, delay in transformation of gonocytes to spermatogonia and lack of proliferation of Leydig cells and their atrophy, Sertoli cell abnormalities and interstitial fibrosis.
    - Congenital anomalies of ductal system frequently seen in patients with UDT e.g. epididymal abnormalities.
- **Malignancy:**
  - About 10% of all germ cell testicular tumors occur in cryptorchid testis.
  - The risk of developing tumor in intraabdominal testis is 6 folds higher than in cryptorchid testis at other locations.
  - The tumor develops in 20% of the contralateral descended testis in a cryptorchid patient.
  - There is a 25% chance of developing tumor in opposite cryptorchid testis if one cryptorchid testis develops tumor.
  - Cancers arising in uncorrected abdominal testes are seminomatous, while those originating following successful orchidopexy, regardless of their original location,are non seminomatous germ cells tumor.
  - Also, orchidopexy does not decrease the risk of testicular cancer.
  - The presentation of testicular tumors occurs mainly in the third decade.

Cause for development for malignancy:

- Progressive degeneration of germ cells with secondary dysplasia seen in cryptorchid testis due to their extra scrotal position that remains at a higher temperature than the inguinal/ abdominal testis.
- Intrinsic abnormality of testis rather than secondary dysplasia(theory of dysgenetic germplasm)
- Altered hormonal milieu during intra-embryonic life.

Carcinoma- in situ (CIS) and malignant transformation:

Carcinoma-in-situ is a premalignant condition and has been found 2-3% of testicular biopsies performed in adult patient who underwent orchidopexy when young.

The occurrence of CIS is associated with an increased risk of carcinoma; especially if contralatarel testis already harbours tumor.

c) **Torsion of testis:** The anatomic abnormalities I,e. high investment of investing tunica vaginalis and epididymal abnormalities makes testis mobile and predisposes it for testicular torsion.

d) **Trauma:** Inguinal testes are especially at a greater risk of direct trauma. This is most commonly seen in children with cerebral palsy using wheelchair braces.

e) **Psychological aspects:** These are related to an empty scrotum. Peer ridicule, impairment of body image and fear of sterlity are important negative responses that cause worry to the patient and his parents. These aspects become all more important near puberty.

f) **Inguinal hernia:** Patent processus vaginalis is commonly associated with UDT; in more than 65% patients. The incidence is slightly lower for ectopic undescended testes. Repair of associated hernia is important to prevent future complications and orchidopexy is done simultaneously.

Group – C

3) Write short notes on (any three) **3x5 = 15**

a) **Anorectal malformations.**
b) **Extradural haematoma.**
c) **Cardiopulmonary resuscitation (CPR).**
d) **MRI.**
e) **Complications of Radiotherapy.**

Answer.

a) Anorectal malformations.

Anorectal Embryology: By 6 weeks of gestation, the urorectal septum moves caudally to divide the cloaca into the anterior urogenital sinus and posterior anorectal canal. Failure of this septum to form results in a fistula between the bowel and urinary tract (in boys) or the vagina (in girls). Complete or partial failure of the anal membrane to resorb results in an anal membrane or stenosis. The perineum also contributes to development of the external anal opening and genitalia by formation of cloacal folds, which extend from the anterior genital tubercle to the anus. The perineal body is formed by fusion of the cloacal folds between the anal and urogenital membranes. Breakdown of the cloacal membrane anywhere along its course results in the external anal opening being anterior to the external sphincter (i.e., anteriorly displaced anus).

Aetiology:

- Unknown INCIDENCE 1 in 5000
- 1% chance of second child being affected SEX 60% male

Wingspread anatomical classification of Anorectal malformations (1984)

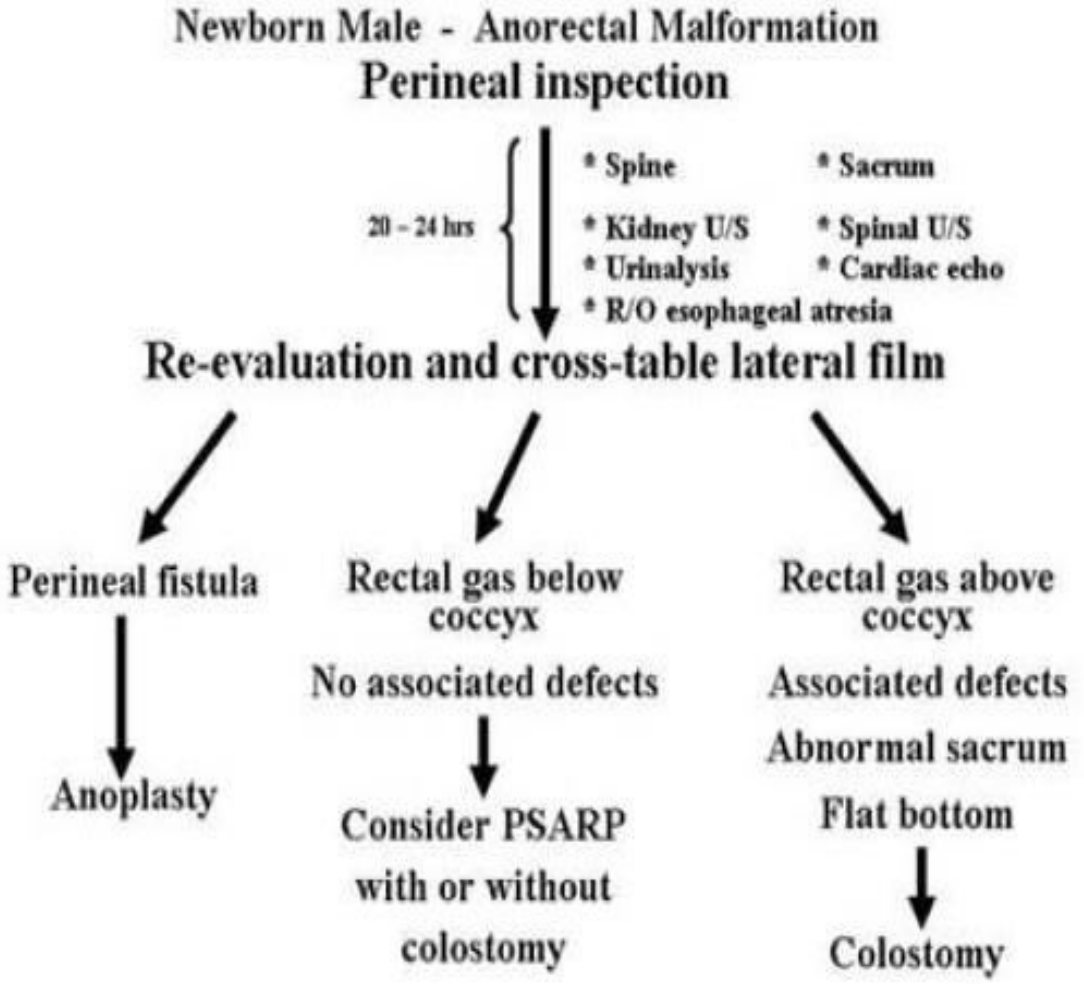
FEMALE	MALE
High Anorectal agenesis With rectovaginal fistula Without fistula Rectal atresia	**High** Anorectal agenesis With rectoprostatic urethral fistula Without fistula Rectal atresia
Intermediate Rectovestibular fistula Rectovaginal fistula Anal agenesis without fistula	**Intermediate** Rectobulbar urethral fistula Anal agenesis without fistula
Low Anovestibular fistula Anocutaneous fistula Anal stenosis	**Low** Anocutaneous fistula Anal stenosis
Cloacal malformations	
Rare malformations	Rare malformations

(Therapeutic and Prognostic)
Males
Cutaneous (perineal) fistula Rectourethral fistula Bulbar Prostatic Recto-bladder neck fistula Imperforate anus without fistula Rectal atresia
Females
Cutaneous (perineal fistula) Vestibular fistula Imperforate anus without fistula Rectal atresia Cloaca Complex malformations

Associated Anomalies:

- **Sacrum and Spine**
  Sacral deformities appear to be the most frequently associated defect.
- **Genitourinary Defects**
  The frequency of associated genitourinary defects varies from 20% to 54%.
- Anal atresia may occur as a part of the VACTERL group of anomalies
  V **V**ertebral body segmentation defect
  A **A**nal atresia
  C **C**ardiovascular (PDA, VSD)
  TE **T**racheo esophagial fistula
  R unilateral **R**enal agenesis
  L **L**imb anomaly (radial ray hypoplasia)
- **CVS**: Tetrology of Fallot or VSD
- **GI**: Tracheo-esophageal abnormality , Duodenal atresia , Hirschsprung's .

Algorithm for management of ARM in newborn male

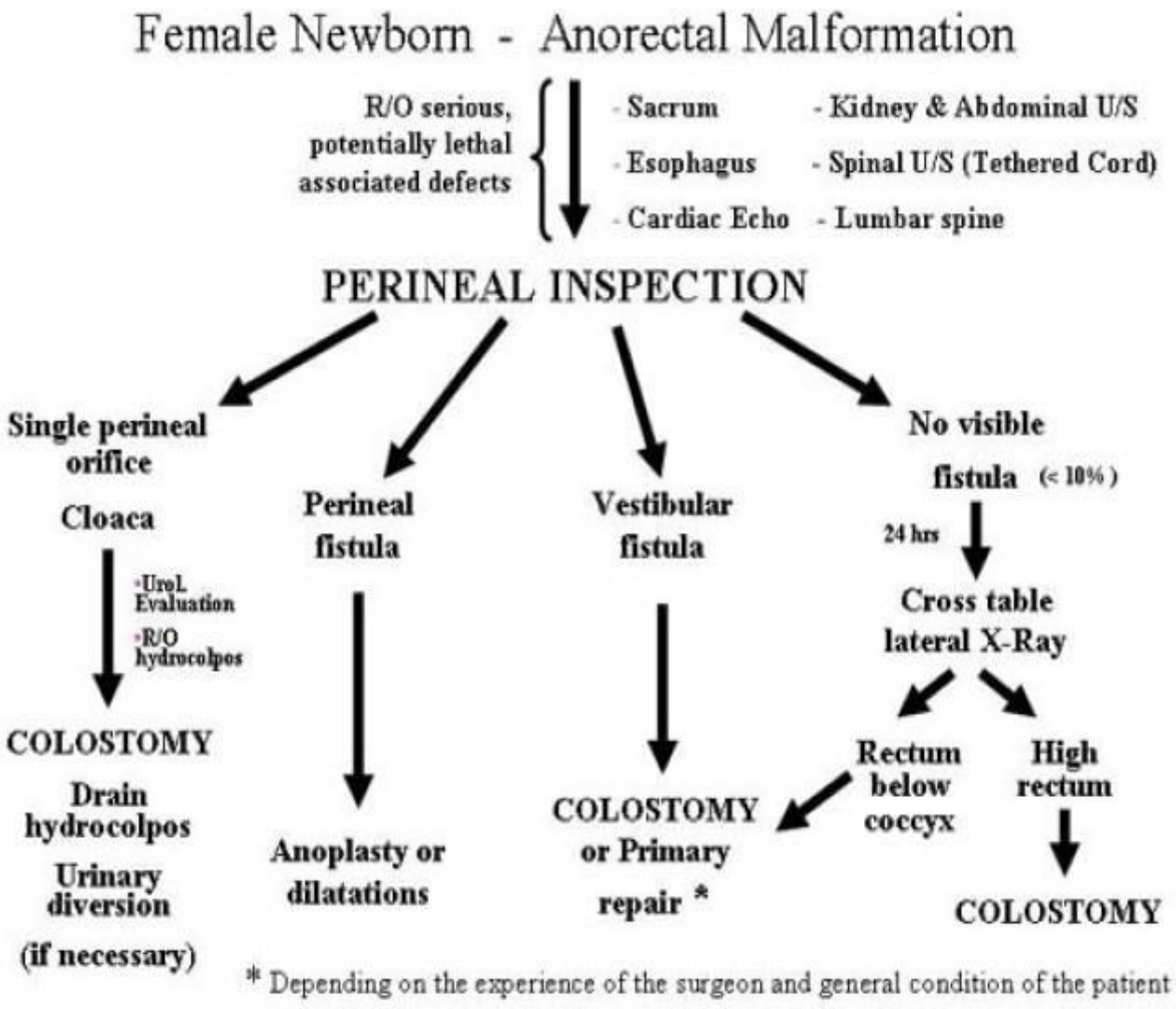


Posterior Sagittal Ano-RectoPlasty – PSARP.

PSARP involves:

- Stimulation of muscles to demonstrate the midline and sphincter
- Posterior sagittal incision - length depends on severity of abnormality and required extent of dissection
- Rectum identified. Abdominal approach may be required in addition in 10% of males and 40% of cloacae Rectum dissected.
- Separation from genitourinary tract - often the most difficult part
- Repositioning the neoanus within the sphincteric mechanism

Algorithm for management of ARM in newborn female



b) Extradural haematoma. See the Question 2.c of Group – B of Supplementary Paper –II of 2012.

c) Cardiopulmonary resuscitation (CPR).

Cardiopulmonary resuscitation (CPR) is a combination of techniques, including chest compressions, designed to pump the heart to get blood circulating and deliver oxygen to the brain until definitive treatment can stimulate the heart to start working again in a person who is in cardiac arrest.

According to the International Liaison Committee on Resuscitation guidelines, CPR involves chest compressions at least 5 cm (2 in) deep and at a rate of at least 100 per minute in an effort to create artificial circulation by manually pumping blood through the heart and thus the body. The rescuer may also provide breaths by either exhaling into the subject's mouth or nose or using a device that pushes air into the subject's lungs. This process of externally providing ventilation is termed artificial respiration. Current recommendations place emphasis on high-quality chest compressions over artificial respiration; a simplified CPR method involving chest compressions only is recommended for untrained rescuers.

Medical use:

- CPR is indicated for any person unresponsive with no breathing or breathing only in occasional agonal gasps, as it is most likely that they are in cardiac arrest.
- If a person still has a pulse but is not breathing (respiratory arrest) artificial respirations may be more appropriate, but, due to the difficulty people have in accurately assessing the presence or absence of a pulse, CPR guidelines recommend that lay persons should not be instructed to check the pulse, while giving healthcare professionals the option to check a pulse. In those with cardiac arrest due to trauma CPR is considered futile but still recommended. Correcting the underlying cause such as pneumothorax or pericardial tamponade may help.

Complications:

- While CPR is a last resort intervention, without which a patient without a pulse will all but certainly die, the physical nature of how CPR is performed does lead to complications that may need to be rectified.
- Common complications due to CPR are rib fractures, sternal fractures, bleeding in the anterior mediastinum, heart contusion, hemopericardium, upper airway complications, damage to the abdominal viscus - lacerations of the liver and spleen, fat emboli, pulmonarycomplications - pneumothorax, hemothorax, lung contusions.

d) MRI. Magnetic resonance imaging (MRI) is a noninvasive medical test that helps physicians diagnose and treat medical conditions. MRI uses a powerful magnetic field, radio frequency pulses and a computer to produce detailed pictures of organs, soft tissues, bone and virtually all other internal body structures.

Magnetic resonance imaging
Strengths
_ No ionising radiation
_ Excellent soft-tissue contrast
_ Best imaging technique for
  - Intracranial lesions
  - Spine
  - Bone marrow and joint lesions

Evolving use
_ Staging
_ MRCP
_ MR angiography
_ Breast malignancy
_ Pelvic malignancy
_ Cardiac imaging

Weaknesses

- Absolute contraindications
  - Ocular metallic foreign bodies
  - Pacemakers
  - Cochlear implants
  - Cranial aneurysm clips
- **Relative contraindications**

- First trimester of pregnancy
- Claustrophobia

- Long scan times so patients may not be able to keep still, especially if in pain
- Limited availability
- Expensive.

e) Complications of radiotherapy.

Common side effects of radiation therapy:

Early and short term:

- Skin problems, such as dryness, itching, blistering, or peeling. These issues usually resolve a few weeks after treatment has finished.
- Fatigue (a persistent sense of tiredness or exhaustion). Fatigue associated with cancer treatment is different from fatigue from lack of sleep; it is a feeling of exhaustion that does not improve with rest.

Late and long term:

- Lymphoedema.
- Development of second cancer.

Rare side effects:

- Rib fracture occurs when the radiation weakens the rib cage near the treatment area.
- Heart problems can develop when radiation therapy is given to the left side of the chest. If they happen, heart problems arise years after radiation therapy is finished.
- Radiation pneumonitis.
- Brachial plexopathy can happen when radiation damages nerves in the upper chest. It may cause tingling, pain and weakness in the affected hand and arm that is usually permanent.

Site-specific side effects: In addition to the general side effects described above, some side effects of radiation therapy depend on where the radiation is given.

- *Head and neck*: Side effects of radiation therapy to the head and neck may include dry mouth,difficulty swallowing, mouth and gum sores, stiffness in the jaw, nausea, and lymphedema. In addition, tooth decay may occur.
- *Chest*: Side effects from radiation therapy to the chest may include difficulty in swallowing, shortness of breath, breast or nipple soreness, and shoulder stiffness. Some people may develop a cough, fever, and fullness of the chest that is diagnosed as radiation pneumonitis.If left untreated, radiation pneumonitis can cause radiation fibrosis (permanent scarring of the lungs from radiation), which can lead to more serious heart and lung problems.
- *Stomach and abdomen*: Side effects from radiation therapy to the stomach and abdomen may include nausea, vomiting, or diarrhea.
- *Pelvis:* Side effects from radiation to the pelvic area may include diarrhea, rectal bleeding,incontinence, bladder irritation, and sexual problems in both men and women. For instance, radiation to the prostate can result in impotence (the inability to maintain an erection).
  Radiation therapy to the pelvis can also affect the reproductive system.
  Some women receiving high doses of radiation therapy may stop menstruating and experience symptoms of menopause, such as vaginal itching, burning, and dryness.
  Permanent infertility (the inability to conceive a child or maintain a pregnancy) can occur, but generally only if both ovaries receive radiation.

Men receiving radiation therapy to the testes or to nearby organs, such as the prostate, will have lowered sperm counts and reduced sperm activity, which affects fertility (the ability to father a child).

Group – D

4) Answer briefly on (any three): 3x5 = 15

a) **Fat embolism.**
b) **Odontomes.**
c) **Short wave diathermy.**
d) **Tension pneumothorax**
e) **Hypokalemia.**

Answers

a) Fat embolism.

Etiology:

- 95% of PE follows DVT in leg.
- The source of embolus in the remaining 5% is from right ventricle, pelvic, renal or hepatic veins.
- Embolisms of foreign bodies (e.g. bullets) or septic material are clinical curiosities.

Clinical Features: Depend on the size of the emboli. A high index of suspicion is required for diagnosing this condition. A large majority of patients may be completely asymptomatic.
Amongst the symptomatic patients, the following symptoms may be noted.
Symptoms:The common symptoms, in decreasing order of frequency are,
 Dyspnoea
 Pleuritic chest pain
 Cough
 Hemoptysis
Signs: Are usually non-specific. These include
 Tachypnoea (Respiratory rate > 20/min)
 Localized crepitations (rales)
 Loud P2 (second heart sound)
 Tachycardia
 Fever
 Evidence of DVT

Features of massive pulmonary embolism;
These include syncope, disorientation or altered sensorium, central chest pain, central cyanosis, raised JVP, and acute cor pulmonale.
Investigations: Routine investigations include ECG, chest X ray, and arterial blood gas (ABG) analysis. Specific investigations include a ventilation/perfusion scan, angiography or spiral CT scan. Duplex scanning of leg veins is added to confirm the source of thromboembolism.

Chest X Ray The initial chest radiograph is rarely diagnostic, and often normal. Several abnormalities may be noted. These include:
 Elevation of one dome of diaphragm
 Parenchymal infiltrates/infarction
 Oligemia of affected lungfield (→estermark‘s sign)
 Pleural effusion
ECG is useful to exclude other causes of chest pain, notably myocardial infarction. It may show the following:
 Sinus tachycardia
 T wave inversion (Leads V1-V4)and non-specific ST changes
 Right bundle branch block
 S1Q3T3 pattern with right axis deviation and RBBB (right bundle branch block) is diagnostic, but found in less than 20% of cases.

ABG (arterial blood-gas analysis) may show low PaO2, with a normal or low PaCO2 and acidosis. However a normal PaO2 does not exclude PE.

Ventilation/Perfusion lung scan (V/Q scan): This is the mainstay of diagnosis in patients who are not acutely ill. Ventilation–perfusion mismatch (normal perfusion but no ventilation) is classically seen in a localized area of the lung.
Traditionally the perfusion scan is performed first, and if a perfusion defect is noted, the ventilation scan is done. Failure of a segment of lung to show perfusion in the presence of adequate ventilation is diagnostic of PE.

Pulmonary angiography is the most specific and accurate investigation in the diagnosis of PE.
It is usually done in two settings: when the diagnosis is in doubt, or when massive PE is suspected where a decision regarding surgical embolectomy or thrombolysis has to be made urgently.

Spiral (Helical) CT scan**.** Contrast-enhances spiral CT is replacing V/Q scan for diagnosis of PE in stable cases. It is said to be more reliable than the V/Q scan.

Treatment:

- General supportive measures include oxygen therapy by mask or nasal prongs, pain relief by intravenous morphine (3-5 mg), correction of acidosis, fluid therapy (maintaining a CVP of about 12 mm Hg), and ionotropic support with dobutamine or isoprenaline, if indicated.
- Heparin remains the drug of choice for PE causing no or minimal haemodynamic disturbances. A loading dose of 150-200 mg/kg is given, followed by continuous infusion, maintaining the APTT to 1.5-2.5 times the normal.
- Heparin is changed to oral anticoagulants after a few days, and these should be continued for at least six months. An INR (international normalized ratio) of 2-3.5 should be maintained.
- Thrombolytic therapy**.** Thrombolytic therapy is an attractive alternative, especially in submassive and massive PE. It may also be used in patients of PE who do not respond adequately to heparin therapy. It is also useful in patients with underlying cardio-pulmonary disease, who have a prohibitive surgical risk (of dying from surgical embolectomy).
- Tissue plasminogen activator is probably better with fewer side effects and a better clot lysis rate.
- Pulmonary Embolectomy: This approach is still being practiced, especially in centers where facilities for cardiopulmonary bypass are not available, even though the mortality approaches 50%. An alternative (and better) surgical approach to pulmonary artery is by median sternotomy with cardiopulmonary bypass. Some

form of pulmonary embolectomy (surgical, catheter aspiration) is indicated in massive PE. In a patient with sudden collapse and no right-sided cardiac output, emergency open surgical embolectomy can be life saving.

- Catheter Embolectomy An embolectomy catheter with a suction-cup at its tip is introduced via the jugular vein or femoral vein, and negotiated into the pulmonary artery. The thrombus is sucked into the catheter and pulled back to the phlebotomy incision, maintaining he suction on the cup. It is then delivered out of the phlebotomy incision.
- **Inferior Vena C**ava Filters These are an alternative to IVC (inferior vena cava) ligation or plication in patients with repeated PE.

- Indications for IVC Filter placement in Pulmonary Embolism:

Absolute Indications	**Relative indications**
• Anticoagulation (AC) contraindicated • Recurrent PE despite anticoagulation • Bleeding forcing discontinuation of AC • After pulmonary embolectomy • Failure of IVC interruption	• Large (>5 cm) free-floating iliac thrombus • Propagating thrombus despite AC • Chronic PE with cor pulmonale • High-risk patient * • Septic PE

A high risk patient is one with significant COPD (chronic obstructive pulmonary disease) with > 50% decrease of pulmonary bed who would not be able to tolerate even minor PE from DVT.

Conclusion:
DVT is common in clinical practice. Awareness regarding the condition is lacking in the country, even amongst physicians. Prophylaxis against DVT should be given where indicated, because this decreases the incidence of pulmonary embolism. DVT should be treated appropriately and aggressively. Low molecular weight heparins have made the treatment of DVT simple, because this does not entail any laboratory monitoring.

b) Odontomas.

Definition: Odontomas are composed of all mature components of dental hard and soft tissue: enamel, dentin, and pulp tissue. Because of their limited slow growth and well-differentiation, they are generally considered to represent hamartomas rather than true neoplasm.

Types:

- Compound odontoma,
- Complex odontomas,
- Odontogenic hamartoma.

Clinical features:

- They are the most common odontogenic tumors and they interfere with eruption of permanent teeth.
- They begin to develop as normal dentition start to develop and cease when the teeth development ends.
- There is no sex predilection.

- They are usually asymptomatic and are discovered during routine radiographic examination when there is delayed eruption of permanent tooth.

Location: Somewhat more common in the maxilla.The compound type is more often in the anterior maxilla while the complex type occurs more often in the posterior regions of either jaw.

Compound odontoma	Complex odontoma	Odontogenic hamartoma
It is a collection of small radiopaque masses, some or all may be tooth-like structures "denticles". It tends to occur in 62% in the anterior region of the maxilla and usually associated with the crown of an unerupted canine. It is formed by exuberant growth of the dental lamina or into a number of small enamel organs by proliferation of the enamel organ.	It is composed of haphazardly arranged dental hard and soft tissue. It has no resemblance to a normal tooth. It tends to occur in 70% in the posterior region of the mandible. There might be a missing tooth if it arises from a normal tooth follice.	The odontogenic hamartoma is a developmental malformation of the dental tissues.

Radiological features:

- Early lesions are radiolucent with smooth, well-defined contours.
- Later a well-defined radiopaque appearance develops.
- Most odontomas are small and do not exceed the size of a normal tooth in the region.
- However, large ones do occur and these may cause expansion of the jaw.
- Most odontomas are asymptomatic and as a result are discovered upon routine radiographic examination.

Treatment:

✖ Odontomas are treated by simple local excision and the prognosis is excellent

✖ They don't recur and are not invasive.

c) Short wave diathermy. See the Question 4.e of Group – D of Paper –II of 2012.

d) Tension pneumothorax. See the Question 4.e of Group – D of Supplementary Paper –II of 2013.

e) Physiology of K^+:

- K^+ is the major intracellular cation, with only 2% of total body K^+ located in the extracellular space.
- The normal serum concentration is 3.3 to 4.9 mmol/L (12.9 to19.1 mg/dL).
- Approximately 50 to 100 mmol (195 to 390 mg/dL) K^+ is ingested and absorbed daily.

- Ninety percent of K^+ is renally excreted, with the remainder eliminated in stools.

Hypokalemia

Causes: K^+ depletion from inadequate intake alone is rare.

Common causes of K^+ depletion in the surgical patient include:

- GI losses (e.g., diarrhea, persistent vomiting, nasogastric suctioning),
- Renal losses (e.g., diuretics, fluid mobilization, amphotericin B), and
- Cutaneous losses (e.g., burns).

Other causes of hypokalemia include:

- Acute intracellular K^+ uptake (associated with insulin excess, metabolic alkalosis, myocardial infarction, delirium tremens, hypothermia, and theophylline toxicity).
- Hypokalemia may also occur in the malnourished patient after initiation of total parenteral nutrition (refeeding syndrome), caused by the incorporation of K^+ into rapidly dividing cells.

Clinical manifestations:

- Mild hypokalemia [K^+ >3 mmol/L is generally asymptomatic.
- The symptoms present with severe K^+ deficiency [K^+ <3 mmol/L (11.7 mg/dL)] and are primarily cardiovascular.
- Early electroencephalogram (ECG) manifestations include ectopy, T-wave depression, and prominent U waves.
- Severe depletion increases susceptibility to reentrant arrhythmias.

Treatment:

- In mild hypokalemia, oral replacement is suitable. Typical daily therapy for the treatment of mild hypokalemia in the patient with intact renal function is 40 to 100 mmol KCl orally in single or divided doses.
- Parenteral therapy is indicated in the presence of severe depletion, significant symptoms, or oral intolerance. K^+ concentrations (administered as chloride, acetate, or phosphate) in peripherally administered intravenous fluids should not exceed 40 mmol/), and the rate of administration should not exceed 20 mmol (78 mg)/hour.
- However, higher K^+ concentrations [60 to 80 mmol/L (234 to 312 mg/dL)] administered more rapidly (with cardiac monitoring) are indicated in cases of severe hypokalemia, for cardiac arrhythmias, and in the management of diabetic ketoacidosis.
- Administration of high K^+ concentrations via subclavian, jugular, or right atrial catheters should be avoided because local K^+ concentrations may be cardiotoxic.
- Hypomagnesemia frequently accompanies hypokalemia and generally must be corrected to successfully replenish K^+.

The West Bengal University of Health Sciences

M.B.B.S. 3rd Professional Part – II Examination, 2008

Subject: Surgery **Time: 2½ hrs.**

Paper: II **Marks: 60**

Group –A

1) a) Enumerate the causes of haematuria. How will you confirm the diagnosis? What will you do for a patient diagnosed to have carcinoma of the urinary bladder? 5+5+5 = 15

Answer. See the Question 1 of Group – A of Supplementary Paper – II of 2014

Transurethral resection of bladder tumour (TURBT) - usually provides definitive histological diagnosis.This is usually undertaken under general or spinal anaesthesia; bimanual examination is mandatory before and after bladder tumour resection, to assess size, position, and mobility. The pathologist should report on the tumour type, grade, and stage; in particular, the presence or absence of muscularis propria should be noted, since its absence will preclude reliable T staging.

- Staging investigations: are usually reserved for patients with biopsy-proven muscle-invasive bladder cancer unless clinically indicated, since superficial TCC and CIS disease are rarely associated with metastases.

- Pelvic CT or MRI may demonstrate extra-vesical tumour extension or iliac lymphadenopathy, reported if >8mm in maximal diameter.
- Chest X-ray
- Isotope bone scan is obtained in cases being considered for radical treatment.
- Staging lymphadenectomy (open or laparoscopic) may be indicated in the presence of CT-detected pelvic lymphadenopathy if radical treatment is under consideration

TNM staging of bladder carcinoma

Tx Primary tumour cannot be assessed
T0 No evidence of primary tumour
Ta Non-invasive papillary carcinoma
Tis Carcinoma *in situ*
T1 Tumour invades subepithelial connective tissue
T2 Tumour invades muscularis propria (detrusor): T2a inner half; T2b outer half
T3 Tumour invades beyond muscularis propria into perivesical fat: T3a = microscopic; T3b = macroscopic
T4aTumour invades any of: prostate, uterus, vagina, bowel
T4bTumour invades pelvic or abdominal wall
Nx Regional (iliac and para-aortic) lymph nodes cannot be assessed
N0 No regional lymph node metastasis
N1 Metastasis in a single lymph node <2cm in greatest dimension
N2 Metastasis in a single lymph node 2â€“5cm or multiple nodes <5cm
N3 Metastasis in a single lymph node or multiple nodes >5cm in greatest dimension
Mx Distant metastasis cannot be assessed
M0 No distant metastasis
M1 Distant metastasis present

A summary of the management of bladder cancer:

Histology	Further treatment	Urological follow-up
G1/2, Ta/1 TCC	Single-dose intravesical chemotherapy	Review cystoscopies, commencing 3 months
Persistent multifocal recurrent G1/2, Ta/1	Intravesical chemotherapy—6 weekly doses	Review cystoscopies, commencing 3 months
G3, T1 TCC	Intravesical BCG—6 weekly doses	Review cystoscopies, commencing 6-12 weeks
CIS (carcinoma *in situ*, severe intraepithelial dysplasia)	Intravesical BCG—6 weekly doses Â± maintenance	Cystoscopies + biopsy and cytology, commencing 3 months
Pt2/3, N0, M0 TCC, SCC, or adenocarcinoma	Radical cystectomy, radiotherapy, or palliative TURBT (unfit)	Cystoscopies if bladder is preserved; urethral washings for cytology
T4 or metastatic TCC, SCC, or adenocarcinoma	Systemic chemotherapy; multidisciplinary team symptom palliation	Palliative treatment for local bladder symptoms

Or

B) Discuss the pathogenesis of Multinodular goiter. Mention the complications of such a Goitre. How do you manage such a patient? 5+5+5 =15

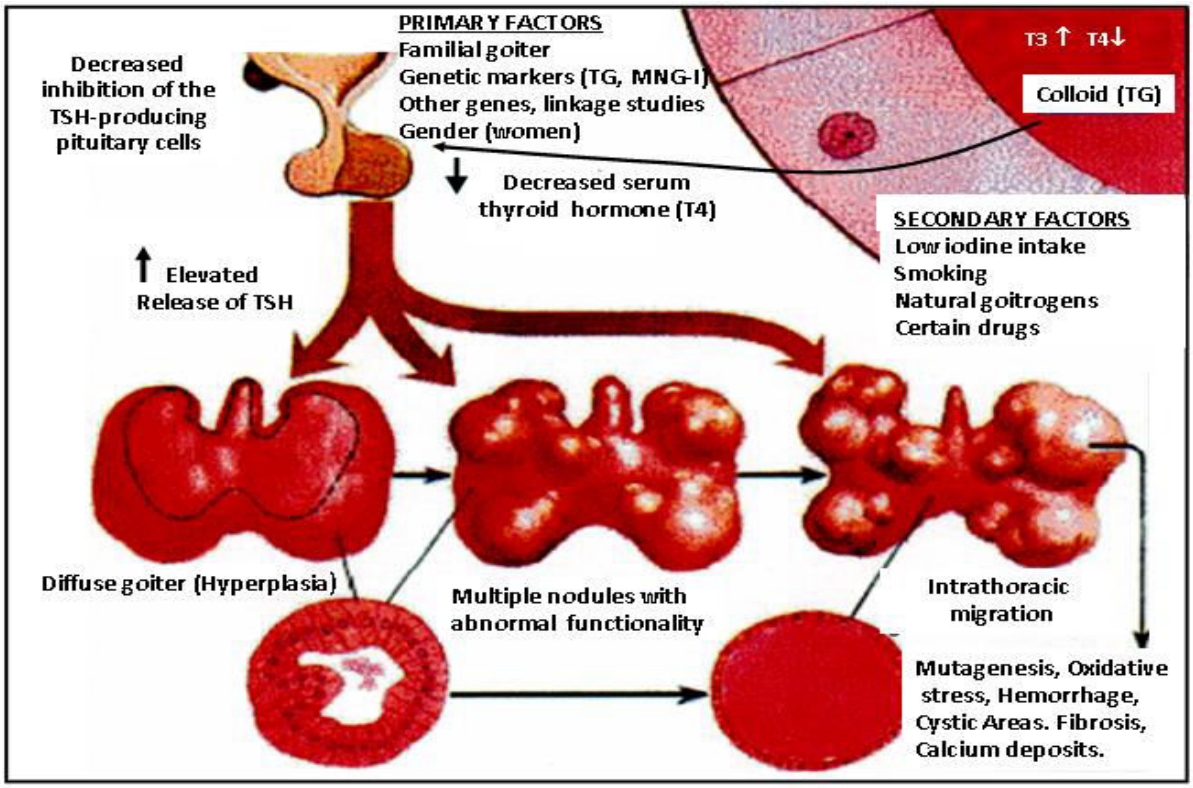
Answer. Factors That May Be Involved in the Evolution of Muitinodular Goiter.

Primary factors
1. Functional heterogeneity of normal follicular cells, cause unknown, possibly genetic and
Acquisition of new inheritable qualities by replicating epithelial cells
2. Subsequent functional and structural abnormalities in growing goiters
Secondary factors (Stimuli to New Follicle Generation)
1. TSH (induced by, e.g., iodine deficiency, goitrogens, inborn errors of thyroid hormone synthesis)
2. Other thyroid-stimulating factors

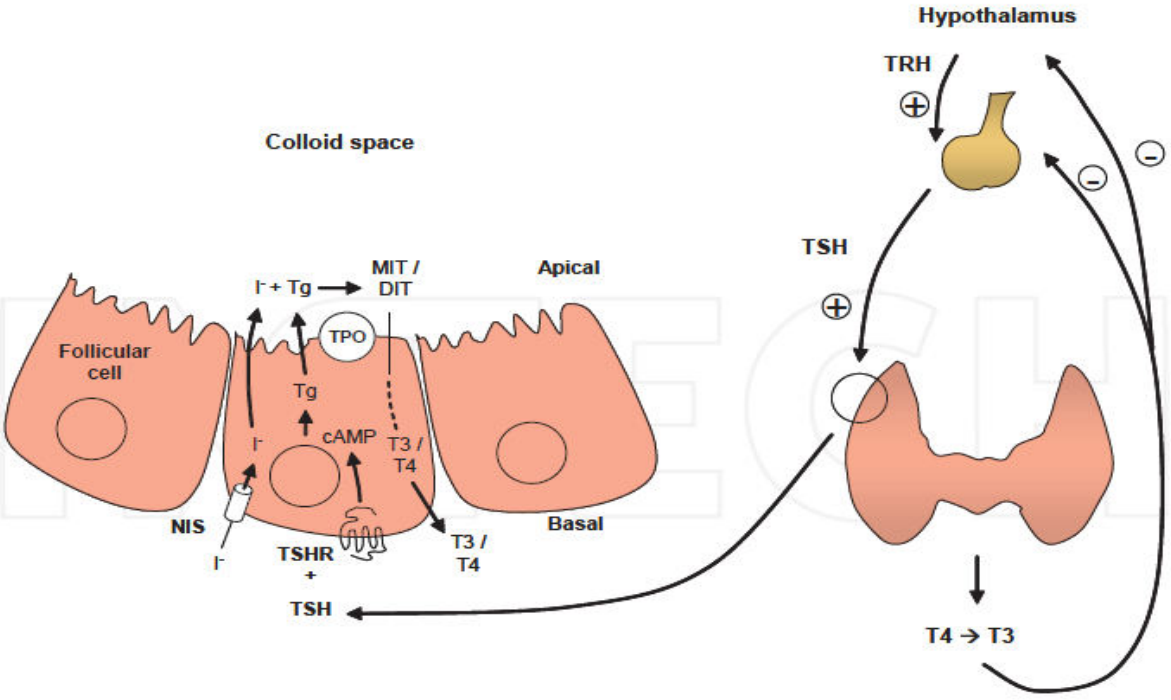
Pathogenesis:

- Reflects impaired synthesis of thyroid hormone most often caused by iodine deficiency
- Impairment leads to compensatory ↑ in TSH levels → hypertrophy and hyperplasia of follicular cells → gross enlargement of gland
- Euthyroid metabolic state
- Degree of enlargement is proportional to level and duration**.**
- The pathogenesis of MNG encompasses processes of diffuse follicular hyperplasia, focal nodular proliferation and eventual acquisition of functional automaticity. The development of MNG is a result of long-term exposure of the thyroid gland to proliferative stimuli, such as iodine deficiency, goitrogens and

inborn error of thyroid hormone synthesis. All of the above results in insufficient thyroid hormone production and stimulate pituitary secretion of thyroid stimulating hormone (TSH).



Go through: Regulation of thyroid hormone production



The pathogenesis of MNG encompasses processes of diffuse follicular hyperplasia, focal nodular proliferation and eventual acquisition of functional automaticity. The development of MNG is a result of long-term exposure of the thyroid gland to proliferative stimuli, such as iodine deficiency, goitrogens and inborn error of thyroid hormone synthesis.
All of the above results in insufficient thyroid hormone production and stimulate pituitary secretion of thyroid Stimulating hormone (TSH). TSH is a glycoprotein with stimulatory effect on the trophic and iodine metabolism pathway in the thyroid follicular cells. TSH binding to the cell membrane G protein-coupled receptor activates the camp and phospholipase C signalling pathways, which in turn upregulates the
Process of iodine uptake and organification, thyroglobulin synthesis, iodotyrosine coupling,and iodothyronine (T3, T4) secretion, leading to a short-term response in thyroid hormone production. In the long-term, TSH also stimulates proliferation of follicular cells to increase the functional mass of thyroid gland. Clinically, TSH stimulation results in enlargement of thyroid gland, increased radio-iodine uptake and increased T4 and T3 levels.
Nodule formation is postulated to be the result of both an inherent and acquired heterogeneity
In proliferative and functional upregulation of the follicular cells. The thyroid follicular cells are inherently heterogeneous with regard to thyroid hormone production and proliferation in response to TSH stimulation, such that under intermediate level of stimulation, a subpopulation of follicular cells outgrows other cells and expand into macroscopic nodules. On the other hand, follicular cells acquiring activating somatic mutations in the cell proliferation pathways can expand clonally to form a nodule. About 60 – 70% of nodules form by the later mechanism and are monoclonal in origin. Somatic mutations leading to constitutive activation of TSH receptors are found in about 60% of autonomously functioning nodules. The remaining,40% of functioning nodules are TSH receptor mutation negative with poorly understood,genetic mechanism behind.

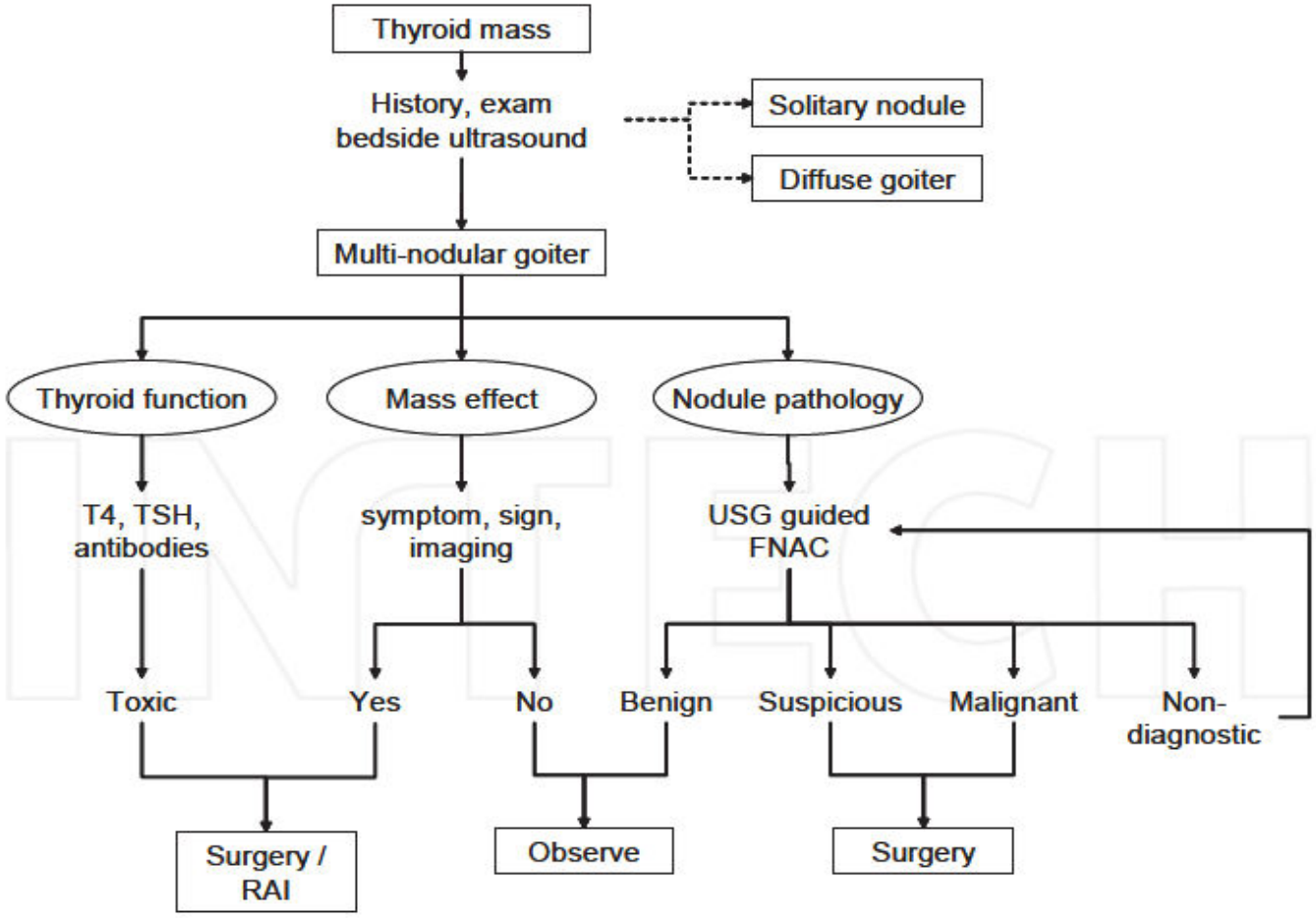
An adenoma with reduced iodine uptake is scintigraphically detected as a “cold nodule”. A defective iodine transport (membrane expression of sodium / iodine symporter protein) and iodine organification is implicated in the hypofunctionality. However, the molecular event accounting for the proliferative advantage is yet to be identified.
Unlike thyroid carcinomas, which also manifest as cold nodules in scintigraphy, *BRAF* and *ras* mutation are uncommon in benign cold adenoma.Recently, oestrogen was shown to stimulate growth of thyroid progenitor cells while simultaneously inhibiting the expression of sodium / iodine symporter mrna, providing a possible explanation of growth / function dissociation in cold thyroid adenomas. Although the majority of thyroid nodules are benign, about 5-10% of nodules may harbour a cancerous focus. In addition, it is known that about 6% of goiter patients eventually develop thyroid cancer, especially from MNG to thyroid cancer, suggesting a possible progression from benign thyroid lesions to malignant disease. [20] More recently, one group has identified a germline missense mutation (1016C>T) in TITF-1/NKX2. 1 encoding a mutant TTF-1 protein (A339V), which may contribute to predisposition for MNG patients to papillary thyroid carcinoma (PTC), further highlighting a causal link between these two diseases. Ectopic expression of the A339V mutant protein in a normal rat thyroid PCCL3 cell line results in a significant up-regulation of cellular proliferation of these thyroid cells and promotes their TSH-independent growth, which was in part attributed to activated Stat3 survival signal in these cells.

Complications of MNG:

- Neck pain or dragging sensation,
- Cosmetic problems,
- Pressure symptoms like dysphagia / dyspnea
- Hypo or hyperthyroidism.
- Symptoms of toxicity.

- High risk of thyroid cancer in patients with multinodular goiter

Management of multinodular goiter:



Investigations:
The investigative workup of a patient with MNG includes
 T4 and TSH levels to evaluate thyroid function

 High resolution USG and FNA should be used for suspicious or dominant nodules where malignancy is suspected. Predominantly solid nodules especially when they are hypoechoic or display a sonoluscent rim surrounding the lesion(—halo sign‖) should be evaluated by FNAC.

 X ray Neck AP and Lateral views to assess tracheal position, retro tracheal extension or incipient compression.
 Chest X ray to evaluate for any features of retrosternal extension.
 CT/MRI are useful in select cases where a retrosternal extension is suspected. It provides accurate delineation of the depth of goiter extension into the chest and its relation to the trachea, esophagus and great veins.
 Thyroid scintigraphy is another option to confirm the extent and functional status of the gland but it is not needed routinely. It does not provide as good anatomical detail as a CT/MRI. It is especially useful in the

hyperthyroid patient with a dominant nodule as it defines the area of hyperactivity thereby allowing a proper choice of therapeutic modality.

The indications for treatment are as following

Strong

 Compressive symptoms
 Hyperthyroidism
 Suspected malignancy

Relative

 Cosmesis
 Potential for Tracheo-esophageal compression

The various therapeutic options include

· Suppressive levothyroxine therapy
· Surgical
· Radioactive iodine therapy.

Suppressive Levothyroxine Therapy

The results of T4 suppressive therapy are inconsistent and marginal. This therapy has a limited role in management of patients with MNG.The aim of such therapy is to consistently suppress TSH levels to <0.5 mu/l. The problem of this therapy is that a significant number of patients become hyperthyroid with time. The overall poor response to T4 supression therapy is because large amounts of thyroid tissue is likely to be hormone insensitive. Another factor for inconsistent response is the variable TSH dependency of thyrocytes. Only small goiter would respond and that too partially. Goitres that respond do so within a period of 6 months

Radioactive Iodine Therapy

It is of limited value and is useful in only two classes of patients.
A) Those with small goiters may benefit.
B) For patients with substantially increased perioperative risk and reasonable thyroid gland function.
RAI therapy is of no value in large multinodular goiters with poorly functioning nodules as the efficacy of RAI therapy depends on the presence of reasonable gland activity all over the thyroid.

Surgery

Surgical treatment is the modality of choice in the management of patients with multinodular goitre. The results of surgery are immediate and tissue is available for histological confirmation of the diagnosis and evaluation for any malignant change. Surgery is the only treatment option in those with compressive symptoms or those with suspected malignancy. Surgical treatment options are between Subtotal and Total thyroidectomy.

Group – B

2) Write short notes on (any three): 3x5 = 15

a) Venous ulcer.
b) Epididymal cyst.
c) Tetany.
d) Thyroglossal cyst.
e) Dermoid cyst.

Answer.

a) Venous ulcer. See the Question 4.c of Group –D of Supplementary Paper – II of 2014.

b) Epididymal cyst. See the Question 3.b of Group – C of Supplementary Paper – II of 2012

c) Tetany.

Introduction: Tetany is a medical sign consisting of the involuntary contraction of muscles, which may be caused by disease or other conditions that increase the action potential frequency of muscle cells or the nerves that innervate them. Muscle cramps which are caused by the disease tetanus are not classified as tetany; rather, they are due to a lack of inhibition to the neurons that supply muscles.

Pathophysiology:

Hypocalcemia: It is the primary cause of tetany. Low ionized calcium levels in the extracellular fluid increase the permeability of neuronal membranes to sodium ions, causing a progressive depolarization, which increases the possibility of action potentials. This occurs because calcium ions interact with the exterior surface of sodium channels in the plasma membrane of nerve cells. When calcium ions are absent the voltage level required to open voltage gated sodium channels is significantly altered (less excitation is required). If the plasma Ca^{2+} decreases to less than 50% of the normal value of 9.4 mg/dl, action potentials may be spontaneously generated, causing contraction of peripheral skeletal muscles. Hypocalcemia is not a term for tetany but is rather a cause of tetany.

Causes:

- Diarrhea
- Kidney disease
- Thyroid or pancreas problems
- Pregnancy and breast feeding
- Malnutrition
- Vitamin D deficiency
- Some medications
- Calcium deficiency

Clinical features:

Symptoms:

Severe	Non severe
• Abdominal pain • Chronic diarrhea • Muscle pain • Tingling in hands or feet • Twitching fingers	• Loss of muscle coordination • Loss/change in vision • Paralysis • Seizures • Slurred speech • Sudden difficulty with memory, thinking, talking, writing or reading • Sudden weakness or numbness on one side

Diagnosis:

- Trousseau sign: occlusion of the brachial artery by squeezing triggers cramps in the fingers.

- Chvostek sign: tetany can be demonstrated by tapping anterior to the ear, at the emergence of the facial nerve. A resultant twitch of the nose or lips suggests low calcium levels.
- Tetany is characterized by contraction of distal muscles of the hands (carpal spasm with extension of interphalangeal joints and adduction and flexion of the metacarpophalangeal joints) and feet (pedal spasm) and is associated with tingling around the mouth and distally in the limbs.
- EMG studies reveal single or often grouped motor unit discharges at low discharge frequency during tetany episodes.

Treatment:

Detection of low calcium levels is important. Tetany can be fatal if it involves laryngeal or pharyngeal muscles; which blocks the airway.

IV Treatment:

- Calcium carbonate is used in the initial stage of Hypocalcemia.
- Calcium gluconate is used for muscle cramps or nerve weakness.
- Calcium chloride is for patients in serious condition.

d) Thyroglossal cyst. See the Question 3.b of Group –C of Paper – II of 2013.

E) Dermoid cyst:

Introduction: Cyst lined by squamous epithelium containing desquamated cells.
Contents: mixture of sweat, sebum, desquamated epithelial cells, hair.

Clinical types:

- Congenital / sequestration dermoid.
  - Site: along lines of embryonic fusion (midline of body or face)
  - Formation: dermal cells sequestrated in subcutaneous plane > proliferate & liquify > cyst > grows & indents mesoderm(future bone) > bony defects

Location:

- Medial Nasal Dermoid Cyst (root of nose at fusion lines of frontal process),
- External And Internal Angular Dermoid (fusion line of frontonasal and maxillary processes)
- Sublingual Dermoid
- Pre –Auricular Dermoid
- Post Auricular Dermoid

Clinical features:

- Manifests in childhood or adolescence
- Typically a painless slow growing swelling
- Soft, cystic, fluctuant, yield to pressure of finger and will not slip away
- Transillumination negative
- Putty in consistency
- No impulse on coughing
- Underlying bony defect – clue to diagnosis
- Location along line of fusion

Other types:

- Implantation dermoid: in women, tailors, agriculturists who sustain repeated minor injuries > sharp injury- epidermal cells implanted in subcutaneous plane- dermoid cyst > fingers, palm, sole of foot > hard in consistency (skin is thick).

- Teratomatous dermoid: arise from totipotent cells > ectodermal, mesodermal, endodermal elements > ovary, testis,retroperitoneum, mediastinum

- Tubulo-embryonic dermoid > from ectodermal tubes > thyroglossal cyst, post- anal dermoid.

Investigations:

- Blood – TC, DC,Hb,ESR
- Urine Examination
- FNAC-
- X ray- subjacent bone eroded by dermoid
- Ultrasonography- mass cystic/ solid
- CT scan- size , shape , local spread

Treatment: Excision of the cyst.

Group – C

3) Write short notes on (any three) **3x5 = 15**

a) Exomphalos.
b) Skin grafting.
c) Spinal anaesthesia.
d) Double contrast enema.
e) Brachytherapy.

Answer.

a) Exomphalos. Same as omphalocele. See the Question 2.e of Group – B of Paper – II of 2011.
b) Skin grafting.

Skin grafts:

Definition
A skin graft is a piece of dermis and epidermis that is completely removed from its original bodily attachment (the donor site). It is fixed to a recipient site and develops a new blood supply from the underlying tissue.

- Autograft: transfer from one part of a person's body to another part
- Isograft: transfer between genetically identical individuals

- Allograft: transfer between individuals of the same species
- Xenograft: transfer between individuals of different species

Full thickness skin grafts (Wolfe grafts)

- Contain epidermis plus the entire thickness of dermis.
- Adnexal structures, e.g. Hair, are included.
- Harvested by elliptical excision from sites of skin laxity, e.g. Postauricular skin crease, supraclavicular, pre-auricular, groin, or medial upper arm skin.
- Graft secured with a tie-over dressing, e.g. Proflavine-soaked cotton wool, and inspected after a week.
- Donor site sutured closed.

Split thickness skin grafts (Thiersch grafts)

- Consist of epidermis plus a variable thickness of dermis.
- Harvested by shaving off a layer of skin with a skin graft knife or dermatome. Can be taken from any area of the body (thigh skin most often usedâ€”plentiful and easy to access).
- Graft is often fenestrated (to stop blood or serous fluid collecting under it) or meshed (to expand the graft).
- Graft secured with glue, sutures, or staples, then a non-adherent, compressive dressing. Inspected after 5 days.
- Defect heals by re-epithlialization from skin appendages.

Graft healing:
Stages of graft take

- Adherence (immediate): fibrin bond between graft and recipient bed.
- Serum imbibition (days 2-4): graft absorbs fluid and nutrients from bed.
- Revascularization (after day 4): blood enters the graft, either by flowing directly into the graft vessels (inoculation) or by new vessel ingrowth.

Reasons for graft failure:

- Shearing: revascularization cannot occur if the graft is mobile.
- Infection: either of the bed or the graft tissue.
- Separation of graft from its bedâ€”by haematoma or seroma.
- Inadequate bed, e.g. Bare cortical bone; tendon without paratenon.
- Damage to the graft, e.g. Poor surgical technique, excessive dressing pressure.

Split thickness grafts versus full thickness grafts

Points	**Split skin graft**	**Full thickness skin graft**
Cosmesis	Thin, often hypertrophic skin	Good cosmesis
Contracture	Frequent	Less frequent
Availability	Plentiful; can re-harvest after 14 days	Limited by skin laxity
Take	Good, low metabolic needs	Needs optimal bed

Donor scar	Minimalcolour change only	Linear scar
Contraindications	Inadequate bed, e.g. Exposed bone, tendon, cartilage (in which case flap needed) Infected bed Areas where cosmesis is paramount	Large area to be covered Inadequate bed

c) Spinal anaesthesia. See the Question 3.d of Group – C of Supplementary Paper – II of 2013

d) Double contrast enema.

A double-contrast barium enema is a procedure in which x-rays of the colon and rectum are taken after a liquid containing barium is put into the rectum. Barium is a silver-white metallic compound that outlines the colon and rectum on an x-ray and helps show abnormalities. Air is put into the rectum and colon to further enhance the x-ray.

The double contrast barium enema is rapidly being replaced by CT colongraphy, but remains in some centres for:

- The detection of polyps and colorectal cancer
- Follow up screening for postoperative colorectal cancer
- Evaluation of diverticular disease
- Failed colonoscopy
- Investigation of non-specific abdominal pain

The barium enema procedure is contraindicated in certain conditions:

- Toxic megacolon
- Pseudomembranous colitis
- Imminent rectal biopsy within 7 days of procedure or within 7 days after the rectal biopsy.

Single contrast barium enema is not suitable in these situations because it is less sensitive at detecting small polyps and early changes of inflammatory bowel disease. The 'double contrast' refers to the use of positive and negative contrast agents to increase the sensitivity of the examination.

The double contrast study is sensitive to visualize mucosal irregularities.

- Positive contrast: barium or barium-like agent, e.g. Gastrografin
- Negative contrast: air or CO_2

e) Brachytherapy. See the Question 4.c of Group – D of Supplementary Paper – II of 2012

Group – D

4) Answer briefly on (any three): 3x5 = 15

a) Dental cyst.
b) Flail chest.
c) Glasgow coma scale.
d) Therapeutic use of ultrasound.
e) Patent ductus arteriosus.

Answer.

Dental cyst.

- **The periapical cyst (also termed radicular cyst, and to a lesser extentdental cyst) is the most common odontogenic cyst.**
- **It is caused by pulpal necrosis secondary to dental caries or trauma.**
- **It arises from epithelial residues in periodontal ligament as a result of inflammation. The inflammation usually follows death of dental pulp. Radicular cysts are found at root apices of involved teeth. These cysts may persists even after extraction of offending tooth; such cysts are called residual cysts.**
- **The cyst lining is derived from the cell rests of Malassez.**
- **Usually, the periapical cyst is asymptomatic, but a secondary infection can cause pain.**
- **On radiographs, it appears a radiolucency (dark area) around the apex of a tooth's root.**

Signs and symptoms:

- **Dental cysts can cause several problems but some remain undetected for months or even years. Many cause problems when they become infected (causing pain and/or swelling).**
- **They slowly replace some bony tissue (they can weaken the jaws),**
- **They press against other teeth and structures.**
- **They prevent the normal function of the teeth and mouth tissues.**

Treatment:

- **The type of treatment patients will receive usually depends on the type of cyst they have. Usually, an emergency dentist drains the pus from an infected cyst to relieve pain and pressure for those patients with an abscess.**
- **In emergency dentist will clean the space where the abscess was previously located to remove any remnants of infection or other debris. '**
- **The treatment for a periapical abscess – which is formed due to an infection inside the pulp of the tooth – is even more complex than the other forms, with an initial X-ray required to identify its exact location. A root canal procedure will then be carried out after the cyst is identified.**

a) Flail chest. See the question 2.a of Group – B of Paper – II of 2011
b) Glasgow coma scale. See the Question 4.d of Group – D of Supplementary Paper –II of 2010
c) Therapeutic use of ultrasound.

Therapeutic ultrasound refers generally to any type of ultrasonic procedure that uses ultrasound for therapeutic benefit. This includes HIFU, lithotripsy, targeted ultrasound drug delivery, trans-dermal ultrasound drug delivery, ultrasound hemostasis, cancer therapy, and ultrasound assisted thrombolysis.

It may use focused ultrasound (FUS) or unfocused ultrasound.

Ultrasound is a method of stimulating the tisue beneath the skin's surface using very high frequency sound waves, between 800,000 Hz and 2,000,000 Hz, which cannot be heard by humans.

Indications for use:

1. Soft tissue injuries 8. Myositis ossificans 2. Chronic connective tissue and joint dysfunction 9. Nerve entrapments 3. Osteoarthritis 10. Plantar warts 4. Periarthritis (non septic) 11. Ganglia 5. Bursitis 12. Chronic sprains / strains 6. Tensosynovitis 13. Muscle spasm 7. Tendonitis, bursitis, capsulitis

Contraindications and precautions:

Contraindications 1. Cancerous lesions 6. Metal implants 2. Pregnant uterus 7. Eyes, heart, reproductive organs 3. Over the spinal column / brain 8. Deep vein thrombosis 4. Fractures 9. Tissue under therapy with radiation 5. Growing epiphyseal junction

Precautions

1. Bony prominences 2. Decreased sensitivity 3. Decreased circulation

d) Patent ductus arteriosus. See the Question 3.b of Group – C of Supplementary Paper – II.