 # Anaesthesia

@which is the anaesthetic of choice for hypotension during surgery for aortic stenosis?
a)dopamine
b)ephedrine
c)phenylephrine
d)dobutamine
Ans B

Intraoperative hypotension is usually defined as a mean arterial pressure less than 25% of the patient's usual value. Untreated, it may result in inadequate organ perfusion. Hypotension may be due to a low cardiac output, low systemic vascular resistance or a combination of both. Risk factors include chronic hypertension and advanced age. The most common causes of intraoperative hypotension are anaesthetic drugs (including regional anaesthesia) and hypovolaemia. Initial management usually involves fluid bolus to optimise preload, and use of drugs to increase contractility and provide vasoconstriction

ephedrine and metaraminol are commonly used as first line treatment

hypothermia during anaesthesia true is?
a) beneficial to patient
b) can be prevented by giving warm fluids
c) cooled by conduction
d) normal
Ans A
Although hypothermia is generally regarded as deleterious, it can be beneficial in some situations. **Hypothermia decreases the overall metabolic rate by 8% per °C to about half the normal rate at 28°C.**[3] Oxygen demand drops and those tissues that have high oxygen consumption normally, such as brain and heart, have a proportionally greater reduction of oxygen use. This allows aerobic metabolism to continue through greater periods of compromised oxygen supply, thereby reducing the production of anaerobic byproducts such as superoxide radicals and lactate. Additional protection can be attributed to decreased release of excitatory neurotransmitters, reduced synthesis and release of kinases and proinflammatory cytokines, and decreased apoptosis.[12] In addition, hypothermia lowers intracranial pressures and cerebral perfusion pressure.[3]

Substantial protection against cerebral ischemia and hypoxia can be gained by providing a 1°C to 3°C reduction in core temperature. Therapeutic hypothermia is used in many neurosurgery cases and in other procedures such as coronary artery bypass surgery in which tissue ischemia can be anticipated. Therapeutic hypothermia has also been shown to improve outcome during recovery from cardiac arrest and recovery.

Radiation is the major source of heat loss in most surgical patients.

Conduction refers to loss of kinetic energy from molecular motion in skin tissues to surrounding air. Water absorbs far more conducted heat than air, and this accounts for more rapid hypothermia during accidental drowning, as well as the efficacy of water baths to cool hyperthermic patients. For this to be effective, warmed air or water must be moved away from the skin surface by currents, a process called convection. This accounts for the cooling effect of wind and laminar air flow in many surgical suites. Conduction and convection account for ~15% of body heat loss.

Roughly 22% of heat loss occurs by evaporation, as energy in the form of heat is consumed during the vaporization of water. Water evaporates from the body even when not sweating, but mechanisms that enhance sweating increase evaporation. As long as skin temperature is greater than its surroundings, radiation and conduction provide heat loss. At very high environmental temperatures, these processes cannot work, and evaporation is the only manner in which heat can be dissipated.

@ shelf life of suxamethonium:
a. 6 months
b. 1 yr
c. 2 yr
d. 3 yr
Ans C

SUXAMETHONIUM CHLORIDE

Suxamethonium chloride is the dicholine ester of acetylcholine. It is presented as a clear colourless aqueous solution of pH 3.0–5.0 with a shelf-life of 2 years, and stored at 4°C. Spontaneous hydrolysis occurs in warm or alkaline conditions.

@ Which one is contraindication in epileptic patient:
a. propofol
b. thiopentone
c. ketamine
d. midazolam
Ans C
the main anaesthetic drugs to avoid in epileptic patients are alfentanil, remifentanil and sevoflurane, although their contraindication are only relative,
ketamine, at usual doses, has significant epileptogenic potential and should be, therefore, avoid- ed in epileptic patients

Propofol: in spite of the conflicting results in the literature, there is clinical evidence that propofol has anticonvulsant effect and is considered a safe drug for sedation, induc- tion and maintenance of general anesthesia in children and adults [4,8,33,34,42.] In patients with epilepsy, the occurrence of seizures of epileptic origin is extremely rare with the use of propofol, and occurs frequently in the recovery from anesthesia

@ in a surgery being done for intracranial space occupying lesion, which one is used:
a. sevoflurane
b. isoflurane
c. desflurane
d. halothane
Ans B

Inhalational anesthetics:Isoflurane has less effect on CBF and ICP than halothane has. **Because isoflurane depresses cerebral metabolism, it may have a cerebral protective effect when the ischemic insult is not severe**. Data favor the use of isoflurane over either halothane or enflurane. Isoflurane in concentrations of >1 minimum alveolar concentration should be avoided, however, because it can cause substantial increases in ICP.Sevoflurane. Clinical studies have demonstrated, however, that sevoflurane's effect on cerebral hemodynamics is either similar to or milder than that of isoflurane. The disadvantage of sevoflurane is that its biodegraded metabolite may be toxic in high concentrations. There is no evidence of an adverse effect at clinically used concentrations, however, unless sevoflurane is administered in a low-flow circuit for prolonged periods. Rapid emergence from anesthesia

with sevoflurane may be an advantage because it facilitates early postoperative neurologic evaluation.

@ true about celiac block ?

a) Block at L3 level
b) done usually b/l using alchol,phenol
c) Most common side effect is diarrhoea and hypotension
d) Done for lower abdominal malignancies

ans: C

common indication: CA PANCREAS
supplies Upper GIT lower esophagus,liver pancreas etc

The injection consists of a local anesthetic. On occasion, epinephrine, clonidine or a steroid medication may be added to prolong the effects of the celiac plexus block.

ndications

Indicated to control pain of the epigastric viscera, especially due to primary or metastatic upper abdominal cancers.
The most frequent pathology associated with use of this block is pancreatic cancer and associated metastasis.
Not frequently used for chronic pancreatitis or to provide anesthesia for intraabdominal surgery.

Drugs utilized
For a sensory block:
0.25% Bupivacaine with or without 1:200,000 epi.

For a neurolytic block:
50% - 100% Alcohol diluted with sterile water or local anesthetic.
Total volume to be no more than 15-20 ml for each injection.
Overfilling the space may cause the alcohol to leak and spread posteriorly, resulting in alcohol neuritis.

Anatomy
The celiac plexus is situated **retroperitoneally in the upper abdomen.**
It is at the level of the **T12 and L1 vertebrae**, anterior to the crura of the diaphragm.
It encases the **anterolateral surface of the abdominal aorta and the celiac and superior mesenteric arterie**s.
It continues inferiorly as the superior mesenteric plexus and then as the inferior mesenteric plexus.
The vena cava lies anteriorly on the right, the aorta anteriorly on the left.

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Pathology

@
micropuncture of cell membrane with a needle..repair occurs by
a)hydrophobic interactions
b)linear movement of proteins
c)active process requiring ATP
Ans C

Cell wounding is an important driver of the innate immune response of ventilator-injured lungs. We had previously shown that the majority of wounded alveolus resident cells repair and survive deformation induced insults. This is important insofar as wounded and repaired cells may contribute to injurious deformation responses commonly referred to as biotrauma. The central hypothesis of this communication states that extracellular adenosine-5′ triphosphate (ATP) promotes the repair of wounded alveolus resident cells by a P2Y2-Receptor dependent mechanism. Using primary type 1 alveolar epithelial rat cell models subjected to micropuncture injury and/or deforming stress we show that 1) stretch causes a dose dependent increase in cell injury and ATP media concentrations; 2) enzymatic depletion of extracellular ATP reduces the probability of stretch induced wound repair; 3) enriching extracellular ATP concentrations facilitates wound repair; 4) purinergic effects on cell repair are mediated by ATP and not by one of its metabolites; and 5) ATP mediated cell salvage depends at least in part on P2Y2-R activation. While rescuing cells from wounding induced death may seem appealing, it is possible that survivors of membrane wounding become governors of a sustained pro-inflammatory state and thereby perpetuate and worsen organ function in the early stages of lung injury syndromes. Means to uncouple P2Y2-R mediated cytoprotection from P2Y2-R mediated inflammation and to test the preclinical efficacy of such an undertaking deserve to be explored.

@ tumor marker of rhabdomyosarcoma
a) desmin
b) synaptiphysin

c) cytokeratin
d) vimentin

Desmin and muscle actin are positive.MyoD1 and myogenin (myf4) are also useful markers.
desmin is an excellent **marker** for **rhabdomyosarcoma**

@ Which of the following is not seen in apoptosis
a) Chromatin condensation
b) Dna fragmentation
c) Inflammation
d) Cell membrane shrinkage

Ans C
Apoptosis is the process of programmed cell death (PCD) that may occur in multicellular organisms.
Biochemical events lead to characteristic cell changes (morphology) and death. These changes include blebbing, cell shrinkage, nuclear fragmentation, chromatin condensation, and chromosomal DNA fragmentation. (See also apoptotic DNA fragmentation.)
In contrast to necrosis, which is a form of traumatic cell death that results from acute cellular injury, apoptosis generally confers advantages during an organism's life cycle. For example, the differentiation of fingers and toes in a developing human embryo occurs because cells between the fingers apoptose; the result is that the digits are separate. Unlike necrosis, apoptosis produces cell fragments called apoptotic bodies that phagocytic cells are able to engulf and quickly remove before the contents of the cell can spill out onto surrounding cells and cause damage.
Removal of dead cells [edit]
The removal of dead cells by neighboring phagocytic cells has been termed efferocytosis
Dying cells that undergo the final stages of apoptosis display phagocytotic molecules, such as phosphatidylserine, on their cell surface.
Phosphatidylserine is normally found on the cytosolic surface of the plasma membrane, but is redistributed during apoptosis to the extracellular surface by a protein known as scramblase. These molecules mark the cell for phagocytosis by cells possessing the appropriate receptors, such as macrophages
Upon recognition, the phagocyte reorganizes its cytoskeleton for engulfment of the cell. The removal of dying cells by phagocytes occurs in an orderly manner without eliciting an inflammatory response.

@ In HIV which part of the brain does not show atrophy?
A. Anterior cingulate gyrus
B. Globus pallidus
C. Caudate nucleus

D. Lower white matter
Ans B
Compared to controls, **HIV** patients had an enlargement of bilateral **globus pallidus** and a global brain **atrophy**
HIV encephalopathy is pathologically characterized by diffuse white matter pallor and rarefaction, as well as astrocytic cell death. The blood–brain barrier breaks down and HIV-induced cytokines and neurotoxins are produced, causing dendritic simplification and neuronal loss. Regionally, **central white matter and deep gray matter structures – such as the basal ganglia, thalamus, and brainstem** – are particularly vulnerable to atrophy
Surface-based anatomical maps also revealed **regional atrophy in the caudate** and as well as **corpus callosum thinning and ventricular expansion**
Brain regions affected

HIV is associated with pathological changes in mainly subcortical and fronto-striatal areas of the brain, including the basal ganglia, deep white matter, and hippocampal regions. Neuroimaging studies of HIV patients indicate that **significant volume reductions are apparent in the frontal white matter**, whereas subcortically, **hypertrophy is apparent in the basal ganglia, especially the putamen.**
@if a chromosome divides in an axis perpendicular to its usual axis of division it is going to form

a:ring chromosome
b:isochromosome
c: acrocentric
d:subtelocentric
Ans B
Isochromosomes
When a chromosome divides along the axis perpendicular to its usual axis of division. Results in 2 copies of one arm and none of the other.
If on autosome --> Lethal.
If on the X chromosome --> Turner syndrome

@ crumpled tissue paper appearance of cells is seen in
a) gauchers disease
b) glucocerebrosides
Ans a
Gauchers disease
The disease is caused by a defect in housekeeping gene lysosomal glucocerebrosidase (also known as beta-glucosidase, EC 3.2.1.45, PDB 1OGS) on the first chromosome (1q21). The enzyme is a 55.6 KD, 497 amino acids long protein that catalyses the breakdown of

glucosylceramide, a cell membrane constituent of red and white blood cells. The macrophages that clear these cells are unable to eliminate the waste product, which accumulates in fibrils, and turn into **Gaucher cells, which appear on light microscopy to resemble crumpled-up paper**.

@ Stain most commonly used for fungal elements

a) masson trichome

b) silver methamine stain

Ans B

Methenamine silver (GMs) and periodic acid-schiff (pas) are the two most common stains used to look for fungi in tissues and in cytology specimens in the daily practice of pathology. the presence of fungus in the tissue sections provides an indisputable evidence of invasive infection

Surgery

@ Most common cause of death in pelvic fracture

a) Hypovolemic shock
b) Bladder injury
c) Nuerogenic shock

Ans A

Arterial hemorrhage is one of the **most** serious problems associated with **pelvic fractures**, and it remains the **leading cause of death** attributable to **pelvic fracture**.

@ In sentinel lymph node biopsy for carcinoma breast, nerve commonly injured is:

a. nerve to latissimus dorsi
b. nerve to serratus anterior
c. intercostobrachial nerve
d. lateral pectoral nerve

Ans C

Complications of sentinel lymph node biopsy and axillary node sampling include hematoma and wound infection. Seroma formation occurs in less than 5% of patients after axillary node sampling. The majority of these situations do not require aspiration. Seromas are also more common in elderly patients. Lymphedema rarely, if ever, occurs after either axillary node sampling or sentinel lymph node biopsy, but is seen in about 5% to 10% of patients if postoperative radiotherapy is given after these procedures. Damage to the intercostobrachial nerve is much less common in axillary node sampling and sentinel lymph node biopsy than during more extensive axillary dissections. With careful attention to technique, damage to the nerve can usually be avoided in axillary surgery.

@ Due to popularity of refrigeration reducing the need to preserve food, which cancer's incidence has dramatically declined:
a. esophagus
b. stomach
c. colon
d. nasopharynx
Ans B
there is clear evidence of the dietary pattern and risk of stomach cancer. The advent of refrigeration has dramatically reduced stomach cancer incidence as it has revolutionised food preservation. Consumption of large amounts of red chillies, food at very high temperatures and alcohol consumption are the main risk factors for stomach cancer in India. Consumption of a tobacco extract 'Tuibur' has been linked to the high rates of Stomach cancer in Mizoram.

Primary prevention is the best strategy for prevention of stomach cancer.

@ Left scrotal swelling in smoker smoking 35 pack years presenting with microscopic hematuria is attributed to

a) Epididymitis
b) Seminoma
c) Renal cell carcinoma

Ans C

The mechanism of metastasis to the spermatic cord from RCC is not well understood but is most likely retrograde spermatic vein flow from the renal vein

@ Damage control surgery

a) minimum intervention to stabilize and can be operated later
b) Avoid damage so that useful for later management

Ans A

Damage control surgery (**DCS**) is a form of surgery utilized in severe unstable injuries typically by trauma surgeons. This form of surgery puts more emphasis on preventing the trauma triad of death, rather than correcting the anatomy.

major component of the surgery is early recognition of a person who could benefit from it, which often means bypassing the emergency department except for attempts of immediate stabilization techniques, such as gaining airway access.[4][6] Typically the operating room is heated higher than normal to help deal with the associated hypothermia.[5]

The procedure comprises three different steps that are needed for full effect. In the first procedure a laparotomy is performed to control hemorrhage.[7] Generally this procedure will last no longer than one hour.[5] After immediate life threats have been surgically managed, the area is then covered temporarily and the person sent to an intensive care unit for the second phase.[7][8]

In the second phase the patient is given a combination of various medications and treatments to help restore a physiologic balance, especially with regards to their temperature, oxygenation, and pH level.[3] An important element of treatment at this stage is passive rewarming, as generally it will reverse most of the ill effects of the trauma triad.[2] This phase generally lasts no longer than two days but is dependent on the persons condition.[7] When the person does not have their condition improved within the first 24 hours it could mean there was missed hemorrhage which could require immediate surgery, regardless of the reversal of the trauma triad.[9]

In the third phase, the person is operated on again and more definitive procedures are performed

@ A neonate with meningomyelocele awaiting surgery.Solution to cover meningomyelocele is sterile gauze soaked in

a) methylene blue
b) normal saline

c) tincture benzioc
Ans B

@ lithium potentiates the action of neuro muscular blockers. hence it should be stopped how many days before surgery:
a. 1 day
b. 2 days
c. 3 days
d. 4 days
Ans A

- oral anticoagulants should be stopped or its effects reversed depending on indication for surgery
- aspirin may need to be stopped up to 7 days before surgery to minimise perioperative blood loss
- oestrogen containing oral contraceptives should be stopped at least 4 weeks before surgery to reduce the risk of deep vein thrombosis

- lithium should be stopped 24 h prior to surgery
- hypoglycaemic agents need to be omitted on the day of surgery

@Salivary gland tumor hot spot on tc99 scan is ?
A)Adenolymphoma
B) Pleomorphic adenoma

Warthin's tumor or adenolymphoma is the benign tumor of the salivary gland. It is often referred as tumor where diagnosis can be established without biopsy, by Tc99 pertechnetate scan. All tumor give a "cold spot" in Tc99 pertechnetate scan except, adenolymphoma which gives a "HOT SPOT."
@ Mortality in emergency abdominal aorta aneurysm repair surgery
A) 10%
B)20%
C)40%
D)>50%
Ans B

Thirty-day mortality after repair of non-ruptured aneurysms was **0.2 percent in good-risk** and **2.2 percent in high-risk patients**

Overall 30-day mortality after EVAR in 1037 patients was 21.2%

@ A 5yr child burnt with boiling water. method used to calculate burnt area

A) lund - browder chart

B) rule of 9

C) palm method

Ans A

For children and infants, the Lund-Browder chart is used to assess the burned body surface area. Different percentages are used because the ratio of the combined surface area of the head and neck to the surface area of the limbs is typically larger in children than that of an adult

Assessment of burn area

Assessment of burn area tends to be done badly, even by those who are expert at it. There are three commonly used methods of estimating burn area, and each has a role in different scenarios. When calculating burn area, erythema should not be included. This may take a few hours to fade, so some overestimation is inevitable if the burn is estimated acutely.

Palmar surface—The surface area of a patient's palm (including fingers) is roughly 0.8% of total body surface area. Palmar surface are can be used to estimate relatively small burns (< 15% of total surface area) or very large burns (> 85%, when unburnt skin is counted). For medium sized burns, it is inaccurate.

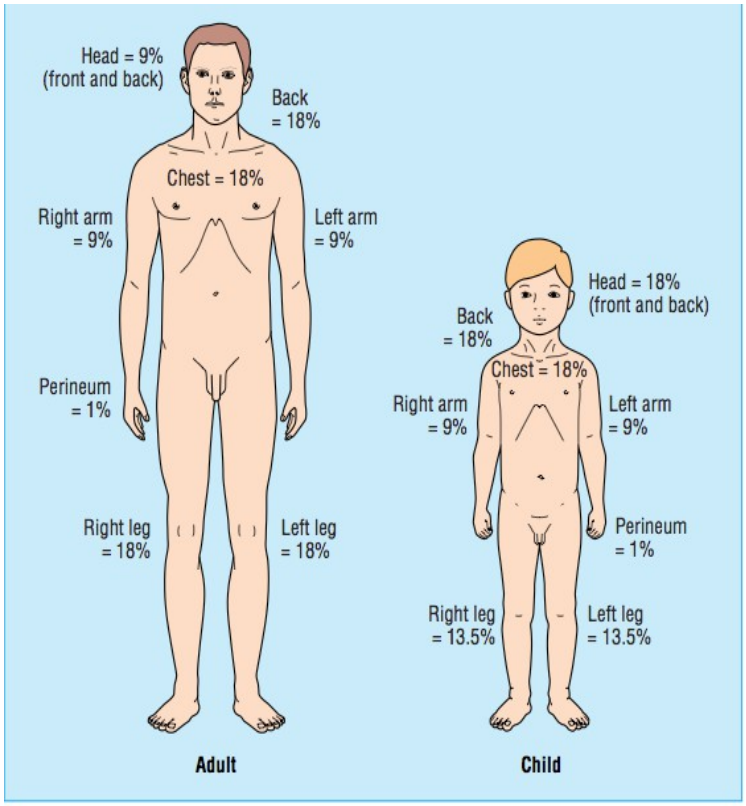
Wallace rule of nines—This is a good, quick way of estimating medium to large burns in adults. The body is divided into areas of 9%, and the total burn area can be calculated. It is not accurate in children.

Lund and Browder chart—This chart, if used correctly, is the most accurate method. It compensates for the variation in body shape with age and therefore can give an accurate assessment of burns area in children.

@ A child had circumferential burn of thighs, buttock,face and scalp. Percentage of burn is

a) 45%
b) 28%

Ans A



@ A driver wearing seat belt applied brake suddenly, what is the most common structure injured:
a. liver
b. spleen
c. mesentery
d. abdominal aorta
Ans C

Mesenteric injury in blunt trauma arises from a shearing force .In experimental animals the injury can be reproduced by compression between two opposing surfaces such as the abdominal wall and spine. In this animal model the site of injury cannot be related to the intraluminal pressure, the fixation of the bowel at the ligament of Treitz, or the presence or absence of air and fluid within the intestine. Such shearing forces can be generated not only by the traumatic incident itself but also **by protective devices such as car safety belts during a sudden deceleratio**n, especially if these are worn across the abdomen rather than across the bony pelvis. Experimentally, more severe intra-abdominal injuries are associated with higher abdominal compression loads.

Anatomy
@ pleural tap pierces all except
a) skin
b) interostal muscle
c) endo thoracic fascia
d) pulmonary pleura
Ans D

Pleural Tap, Thoracocentesis
in 8th or 9th intercostal space at MAL higher at area of maximum dullness with a lot of fluid and chest tube inserted: needle passes skin, ***superficial fascia, external intercostal muscle, internal intercostal muscle, innermost intercostal membrane/muscle, endothoracic fascia, parietal pleura***

@ anorectal ring is formed by all except:
a. external anal sphincter
b. puborectalis
c. anococcygeal raphe
d. internal anal sphincter
Ans C
What is '**anorectal ring**'?
Ans. It is a muscular ring at anorectal junction **formed by** fusion of puborectalis, deep external and internal sphincter

@ kluver bucy syndrome ,all are true except
1 visual agnosia
2 hypersexuality
3 hypermetamorphosis
4 seizure
Ans D
Klüver–Bucy syndrome is a syndrome resulting from bilateral lesions of the amygdala. Klüver–Bucy syndrome may present with **hyperphagia, hypersexuality, hyperorality, and docility.**
Generally included are the following:
Docility. Characterized by exhibiting diminished fear responses or reacting with unusually low aggression. This has also been termed "placidity" or "tameness".
Dietary changes and/or **Hyperphagia.**
Characterized by **eating inappropriate objects (pica) and/or overeating.**
Hyperorality. This was described by Ozawa et al. as "an oral tendency, or compulsion to examine objects by mouth".[2][3][4]
Hypersexuality. Characterized by a heightened sex drive or a tendency to seek sexual stimulation from unusual or inappropriate objects.
Visual agnosia. Characterized by an inability to recognize familiar objects or people

@ structure not present at floor of third ventricle-
a) optic chiasma
b) third nerve
c) infundibulum
d) mamillary body

Ans b
the floor of the third ventricle is formed by the following structures: the optic chiasm, mammillary bodies, tuber cinereum, hypothalamus, subthalamus, infundibulum, posterior perforated substance, and the upper part of the midbrain tegmentum.
@ loss of lacrimation occurs due to injury to:
a. nasociliary nerve
b. greater petrosal nerve
c.
d.
Ans B
The greater (superficial) petrosal nerve carries gustatory (taste) and parasympathetic fibres. Postganglionic parasympathetic fibres from pterygopalatine ganglion **supply lacrimal gland and the mucosal glands of the nose, palate, and pharynx**.
The gustatory fibres do not relay in the ganglion and are distributed to the palate.
GOB
@A Pregnant lady has H/O Juvenile myoclonic epilepsy ,To prevent seizures in children , which drug is used

a) Levitiracitam
b) Carbamazepine
c) valproic acid

Ans a
The most effective anti-epileptic medication for JME is valproic acid However many physicians may not start with valproic acid due to the risk of adverse effects especially in young women. A higher incidence of cleft lip/palate has been reported in pregnant woman on valproic acid. Lamotrigine, levetiracetam, topiramate, zonisamide are often used first. Carbamazepine may aggravate primary generalized seizure disorders such as JME. Treatment is lifelong. Patients should be warned to avoid sleep deprivation.

Dermatology
@ Painful ulcers in thoracic dermatomal distribution are seen in

a) varicella zoster
b) herpes simplex

Ans A
Varicella zoster
Small tightly grouped vesicles
• On erythematous base along dermatome
• Prodromal pain
• Adults and Children
• Diagnosis usually clinically
• Bedside Tzanck smear can be supportive but cannot distinguish between herpes zoster and herpes simplex
• Viral culture is gold standard for diagnosis
@ . A male patient with recurrent oral ulcers, epdidydimitis, blurred

vision

a) Behcet’s syndrome

Behcet’s syndrome

The manifestations of the syndrome can be broadly divided into the following:

Mucocutaneous: (i) oral aphthous ulceration, present in 98% of patients with similar genital (scrotal/labial) ulceration in 80%; and (ii) skin lesions (acneiform folliculitis, erythema nodosum and occasionally ulceration) and pathergy reaction [8].

Arthritis/arthralgia: in up to 50% of patients, usually non-destructive.

Ophthalmic involvement: panuveitis and retinal vasculitis in 50% of patients.

Systemic vasculitis: thrombophlebitis, arteritis and aneurysm formation.

Additionally, gastrointestinal ulceration, epididymitis, central nervous system lesions and pulmonary lesions may all occur on the basis of the underlying vasculitis

@ Fine grouped pinhead papules over dorsum of hand ,forearm and shaft of penis in a children?

A.scabies

B.molluscum contagiosum

C.Lichen planus

D. Lichen nitidus

ANS-D. Lichen nitidus

Lichen nitidus most commonly presents as an incidental finding on physical examination or after the patient notices an insidious onset of the lesions.[1] It is characteristically asymptomatic.[2] Physical examination reveals pinpoint- to pinhead-sized skin-colored papules that may be scaly or have a central depression.[3] They usually are found on the forearms, trunk, and the glans and shaft of the penis.

Lichen nitidus can be discrete or generalized. In the discrete form, papules typically do not coalesce; however, they may form or group at sites of trauma or skin pressure (the isomorphic or “Koebner” phenomenon).[2] In the generalized (confluent) form, papules coalesce into red-yellow to brown plaques, especially in joint flexures, wrist and forearm ventral surfaces, or inframammary areas, making the clinical diagnosis more challenging.[3] Biopsy may be helpful because lichen nitidus has a characteristic histologic appearance.[3]

The etiology of lichen nitidus is unknown, and no laboratory abnormalities or associations with systemic disease have been established. Because lichen nitidus is rare, definitive establishment of the epidemiology is difficult. One study[2] of 43 cases demonstrated

a male-to-female ratio of almost 4:1, although the generalized (confluent) form may be more common in women.[3] No racial predisposition or known genetic inheritance pattern has been noted.[1,2] Lichen nitidus primarily affects children and young adults, with a median age of seven years in males and 13 years in females.

@ A 7 year old boy with boggy swelling of the scalp with multiple discharge sinuses with cervical lymphadenopathy with easily pluckable hair. What would be done for diagnosis?

A. Pus for culture
B. KOH mount
C. Biopsy
Ans b

Boggy swelling in the scalp with easily pluckable hair points towards inflammatory tinea capitis (a variant of tinea capitis).

To make diagnosis of tinea capitis KOH mount is simple, inexpensive, quick, and sensitive test.

Tinea Capitis

- is dermatophyte infection of scalp.
- predominantly a disease of childhood.

Three patterns are recognized viz. Non inflammatory, inflammatory, and favus.

Non inflammatory	Inflammatory (Kerion)	Favus
• Caused by anthrophilic organism e.g. (T. verrucosum) • Broken hairs may appear as grey black dots (black dot tinea) • Lymphadenopathy (occipital) invariable	• Caused by zoophilic dermatophytes e.g. M. canis • Boggy swelling with pulsation • Hair easily pluckable	• Caused by T. schoenleinii • Often results in cicatrial alopecia.

Diagnosis :

- KOH mount simple, inexpensive, quick and sensitive test.
- Culture for identification of species. (SDA is media of choice).
- Woods lamp – tinea capitis is visible as green fluorescence.

Treatment :

- Griseofulvin is drug of choice.
- Given for 4 weeks.

@ Painful blisters with erythematous base are diagnosed by

a) direct immunoflourescence
b) indirect immunoflourescence
c) cytopathology
d) histopathology

.

Herpes simplex	Grouped blisters on erythematous base, rapidly become pustular, painful, history of recurrences

The diagnosis of HSV-1 infection is usually made by the appearance of the lesions and the patient's history. However, if the pattern of the lesions is not specific to HSV, its diagnosis can be made by viral culture, PCR, serology, direct fluorescent antibody testing, or Tzanck test. Viral culture should be obtained from vesicles when possible. The vesicle should be unroofed with a scalpel or sterile needle, and a swab should be used to soak up the fluid and to scrape the base. The swab should be sent in special viral transport media directly to the laboratory (or placed on ice if transport will be delayed). Vesicles contain the highest titers of virus within the first 24 to 48 hours of their appearance (89 percent positive).[9] In general, viral culture for all types of HSV has a sensitivity of approximately 50 percent.[6] Viral isolates usually grow in tissue culture by five days.

PCR is a more sensitive method in the laboratory diagnosis of HSV infection.[4] It is useful for the detection of asymptomatic viral shedding. Direct fluorescent antibody testing may be performed from air-dried specimens, and can detect 80 percent of true HSV-positive cases compared with culture results.[10] Immunoglobulin G antibodies that are type-specific to HSV develop the first several weeks after infection and persist indefinitely. A Tzanck test is difficult to perform correctly without specific training in its use, but it may be done in the office setting by scraping the floor of the herpetic vesicle, staining the specimen, and looking for multinucleated giant cells. Its results do not specify the type of HSV infection, but if done correctly, its sensitivity is 40 to 77 percent for acute herpetic gingivostomatitis

Medicine

@ hypertensive pt presents with headache vomiting neck stiffness without any neurologic deficit . Diagnosis is

a) SAH
b) ishaemic stroke
c) intracranial hemorrhage
d) Extradural hemorrhage

Ans A

@ Fever ,neck stiffness , rash , shock are caused by

a) Nisseria menigitidis

Ans A

@ Limb weakness with decreasing CPK levels with increasing age is due to

a) Hereditary myopathy

b) Dystrophin deficiency
c) Congenital myopathy

Ans B

Although nonspecific, the creatine kinase (CK) level usually is normal in the electrolyte and endocrine myopathies (notable exceptions are thyroid and potassium disorder myopathies). However, the CK level may be highly elevated (10 to 100 times normal) in the inflammatory myopathies and can be moderately to highly elevated in the muscular dystrophies.[16,23,25] Other conditions that can be associated with elevated CK levels include sarcoidosis, infections, alcoholism, and adverse reactions to medications. Metabolic (storage) myopathies tend to be associated with only mild to moderate elevations in CK levels.

Dystrophin deficiency

Elevated CPK (creatine phosphokinase) levels in blood: Elevated CPK levels are more common at younger ages and decreases later in life, perhaps because muscle degeneration occurs more rapidly at younger ages, when there is also more muscle mass to deteriorate

@ Chronic alcohol patient with 2 days h/o free alcohol
Presented with seizure

a) Valproate
b) Diazepam
c) Midazolam
d) Thiopentone

Ans b

Benzodiazepines

Benzodiazepines, such as diazepam or lorazepam, are the most commonly used drug for the treatment of alcohol withdrawal and are generally safe and effective in suppressing alcohol withdrawal signs. Chlordiazepoxide and diazepam are the benzodiazepines most commonly used in alcohol detoxification.[28] Benzodiazepines can be life saving, particularly if delirium tremens appears during alcohol withdrawal. Benzodiazepines should only be used short term in alcoholics who aren't already dependent on benzodiazepines as benzodiazepines share cross tolerance with ethanol and there is a risk of replacing the addiction with a benzodiazepine dependence or worse still adding an additional addiction. Furthermore, disrupted GABA benzodiazepine receptor function is part of alcohol dependence and chronic benzodiazepines may prevent full recovery from alcohol induced mental effects. Benzodiazepines have the problem of increasing cravings for alcohol in problem alcohol consumers and they also increase the volume of alcohol consumed by problem drinkers.[32] The combination of benzodiazepines and alcohol can amplify the adverse psychological effects of each other causing enhanced depressive effects on mood and increase suicidal actions and are generally contraindicated except for alcohol withdrawal

@ Most common Slow growing vascular tumor of brain, spinal cord ?

a) cavernous hemangioma
b) hemangioblastomas

Ans B

Vascular Tumors

These rare, noncancerous tumors arise from the blood vessels of the brain and spinal cord. The most common vascular tumor is the hemangioblastoma, which is linked in a small number of people to a genetic disorder called von Hippel-Lindau disease

@ Not a major criteria in acute rheumatic fever-
a.polyarthralgia
b.subcutaneous nodules
c.chorea
d. carditis
ans –A

Major criteria

- Polyarthritis: A temporary migrating inflammation of the large joints, usually starting in the legs and migrating upwards.
- Carditis: Inflammation of the heart muscle (myocarditis) which can manifest as congestive heart failure with shortness of breath, pericarditis with a rub, or a new heart murmur.
- Subcutaneous nodules: Painless, firm collections of collagen fibers over bones or tendons. They commonly appear on the back of the wrist, the outside elbow, and the front of the knees.
- Erythema marginatum: A long-lasting reddish rash that begins on the trunk or arms as macules, which spread outward and clear in the middle to form rings, which continue to spread and coalesce with other rings, ultimately taking on a snake-like appearance. This rash typically spares the face and is made worse with heat.
- Sydenham's chorea (St. Vitus' dance): A characteristic series of rapid movements without purpose of the face and arms. This can occur very late in the disease for at least three months from onset of infection.

Minor criteria

- **Fever of** 38.2–**3**8.9 °C (101–102 °F)
- Arthralgia: Joint pain without swelling (Cannot be included if polyarthritis is present as a major symptom)
- Raised erythrocyte sedimentation rate or C reactive protein
- Leukocytosis
- ECG showing features of heart block, such as a prolonged PR interval (Cannot be included if carditis is present as a major symptom)
- Previous episode of rheumatic fever or inactive heart disease

Other signs and symptoms [edit]

- A**bdominal pain**
- **Nose bleeds**
- Prece**d**ing streptococcal infection: recent scarlet fever, raised antistreptolysin O or other streptococcal antibody titre, or positive throat culture

@ alzheimer's disease is associated with leision in which area

a) parietal and temporal
b) frontal and occipital
c) temporal and occipital
d) parietal and occipital

Ans A

Alzheimer's disease is characterised by loss of neurons and synapses in the cerebral cortex and certain subcortical regions. This loss results in gross atrophy of the affected regions, including degeneration in the **temporal lobe and parietal lobe,** and parts of the frontal cortex and cingulate gyrus.[36] Degeneration is also present in brainstem nuclei like the locus coeruleus.[57] Studies using MRI and PET have documented reductions in the size of specific brain regions in people with AD as they progressed from mild cognitive impairment to Alzheimer's disease, and in comparison with similar images from healthy older adults

In addition to NFTs, the anatomic pathology of AD includes senile plaques (SPs; also known as beta-amyloid plaques) at the microscopic level and cerebrocortical atrophy at the macroscopic level

The **hippocampus and medial temporal lobe** are the initial sites of tangle deposition and atrophy. .This can be seen on brain magnetic resonance imaging early in AD and helps support a clinical diagnosis

@ In Mental Status examination , which of the following is a measure of awareness response

a) **perception**
b) Judgement
c) Insight
d) Cognition

Ans D

the MSE is not to be confused with the mini-mental state examination (MMSE), which is a brief neuro-psychological screening test for dementia. The **mental status examination** or **mental state examination**, abbreviated **MSE**, is an important part of the clinical assessment process in psychiatric practice. It is a structured way of observing and describing a patient's current state of mind, under the domains of

- **Appearance**
- **Attitude**
- **Behavior**
- **mood and affect**
- **speech**
- **Thought process**

- **thought content**
- **perception**
- **cognition**
- **insight**
- **judgment.**

Cognition

- This section of the MSE covers the patient's level of alertness, orientation, attention, memory, visuospatial functioning, language functions and executive functions. Unlike other sections of the MSE, use is made of structured tests in addition to unstructured observation. Alertness is a global observation of level of consciousness i.e. **awareness of, and responsiveness to the environment**, and this might be described as alert, clouded, drowsy, or stuporose. Orientation is assessed by asking the patient where he or she is (for example what building, town and state) and what time it is (time, day, date). Attention and concentration are assessed by the serial sevens test (or alternatively by spelling a five-letter word backwards), and by testing digit span. Memory is assessed in terms of immediate registration (repeating a set of words), short-term memory (recalling the set of words after an interval, or recalling a short paragraph), and long-term memory (recollection of well known historical or geographical facts). Visuospatial functioning can be assessed by the ability to copy a diagram, draw a clock face, or draw a map of the consulting room. Language is assessed through the ability to name objects, repeat phrases, and by observing the individual's spontaneous speech and response to instructions. Executive functioning can be screened for by asking the "similarities" questions ("what do x and y have in common?") and by means of a verbal fluency task (e.g. "list as many words as you can starting with the letter F, in one minute"). The mini-mental state examination is a simple structured cognitive assessment which is in widespread use as a component of the MSE.

Category	Possible points	Description
Orientation to time	5	From broadest to most narrow. Orientation to time has been correlated with future decline.[7]
Orientation to place	5	From broadest to most narrow. This is sometimes narrowed down to streets,[8] and sometimes to floor.[9]
Registration	3	Repeating named prompts
Attention and calculation	5	Serial sevens, or spelling "world" backwards[10] It has been suggested that serial sevens may be more appropriate in a population where English is not the first language.[11]
Recall	3	Registration recall
Language	2	Naming a pencil and a watch
Repetition	1	Speaking back a phrase
Complex commands	6	Varies. Can involve drawing figure shown.

@ priapism caused by-

a) Sea snake
b) Rattle Snake
c) Spanish fly
d) Scorpion

Ans C

Priapism may be associated with haematological disorders, especially sickle-cell disease, sickle-cell trait, and other conditions such as leukemia, thalassemia, and

Fabry's disease, and neurologic disorders such as spinal cord lesions and spinal cord trauma (priapism has been reported in hanging victims; see death erection). Priapism may also be associated with glucose-6-phosphate dehydrogenase deficiency, which leads to decreased NADPH levels. NADPH is a co-factor involved in the formation of nitric oxide, which may result in priapism.[4] Raised levels of adenosine may also contribute to the condition by causing blood vessels to dilate, thus influencing blood flow into the penis
Sickle cell disease often presents special treatment obstacles. Hyperbaric oxygen therapy has also been used with success in some patients.[6] Priapism is also found to occur in extreme cases of rabies. Priapism can also be caused by reactions to medications. The most common medications that cause priapism are **intra-cavernous injections for treatment of erectile dysfunction (papaverine, alprostadil). Other groups reported are antihypertensives, antipsychotics (e.g., chlorpromazine, clozapine), antidepressants (most notably trazodone), anticoagulants, cantharides (Spanish Fly) and recreational drugs (alcohol, heroin and cocaine).** Priapism has also been linked to achalasia. [citation needed] Priapism is also known to occur from bites of the Brazilian wandering spider and the black widow spider.[citation needed] PDE-5 inhibitors have been evaluated as preventive treatment for recurrent priapism

@a pt.has renal failure and bone pain.serum M spike.35% plasma cells on Bone marrow biopsy.diagnosis-
a)multiple myeloma
b)monoclonal gammopathy with unknown significance,
c)smouldering myeloma
d)plasma cell leukaemia

Ans A

Diagnostic criteria [edit]

In 2003, the International Myeloma Working Group[2] agreed on diagnostic criteria for symptomatic myeloma, asymptomatic myeloma and MGUS (monoclonal gammopathy of undetermined significance), which was subsequently updated in 2009:

- **Symptomatic myeloma:**
  1. Clonal plasma cells >10% on bone marrow biopsy or (in any quantity) in a biopsy from other tissues (plasmacytoma)
  2. A monoclonal protein (paraprotein) in either serum or urine (except in cases of true non-secretory myeloma)
  3. Evidence of end-organ damage felt related to the plasma cell disorder (*related organ or tissue impairment*, ROTI, commonly referred to by the acronym "CRAB"):
     - 3.1 Hyper**C**alcemia (corrected calcium >2.75 mmol/L)
     - 3.2 **R**enal insufficiency attributable to myeloma
     - 3.3 **A**nemia (hemoglobin <10 g/dL)
     - **B**one lesions (lytic lesions or osteoporosis with compression fractures)

Asymptomatic (smoldering) myeloma:

- Serum paraprotein >30 g/L AND/OR
- Clonal plasma cells >10% on bone marrow biopsy AND

- NO myeloma-related organ or tissue impairment

Monoclonal gammopathy of undetermined significance (MGUS):

- Serum paraprotein <30 g/L AND
- Clonal plasma cells <10% on bone marrow biopsy AND
- NO myeloma-related organ or tissue impairment

@ Bilateral babinski sign Positive is seen in ?

a) Cerebral leision
b) Brainstem leision
c) thalamic leision
d) Cerebellar leision

Ans B

Stroke. Babinski's reflex varies with the site of the stroke. If it involves the cerebrum, it produces unilateral Babinski's reflex accompanied by hemiplegia or hemiparesis, unilateral hyperactive DTRs, hemianopsia, and aphasia. If it involves the brain stem, it produces bilateral Babinski's reflex accompanied by bilateral weakness or paralysis, bilateral hyperactive DTRs, cranial nerve dysfunction, incoordination, and an unsteady gait. Generalized signs and symptoms of stroke include headache, vomiting, fever, disorientation, nuchal rigidity, seizures, and coma.

@ False regarding iodine treatment ?

a) inhibits release of thyroid hormone
b) contraindicated for treatment of hyperthyroidism
c) it will result in iodism
d) it also inhibits ???

Ans B

Iodine has several effects on thyroid function. In hyperthyroid patients, iodine acutely inhibits hormonal secretion within hours , but the responsible mechanisms are uncertain. This is the most acute effect of iodine on thyroid status, occurring within one to two days of the start of therapy.

A second effect involves inhibition of thyroid hormone synthesis. In normal subjects, the administration of pharmacologic amounts of iodine leads to temporary inhibition of iodine organification in the thyroid gland, thereby diminishing thyroid hormone biosynthesis, a phenomenon called the Wolff-Chaikoff effect [2]. However, within two to four weeks of continued exposure to excess iodine, organification and thyroid hormone biosynthesis resume in a normal fashion. This is called escape from the Wolff-Chaikoff effect.

@ TEE better than TTE because?

a) convinient
b) better visualisation of atheromatous plaque of asc aorta
c) better to visualise left atrial appendage thrombi

Ans c

The left atrial (LA) appendage is a common source of cardiac thrombus formation associated with systemic embolism. Transesophageal echocardiography allows a detailed evaluation of the structure and function of the appendage by two-dimensional imaging and Doppler interrogation of appendage flow. Specific flow patterns, reflecting appendage function, have been characterized for normal sinus rhythm and various abnormal cardiac rhythms. Appendage dysfunction has been associated with LA appendage spontaneous echocardiographic contrast, thrombus formation and thromboembolism. These associations have been studied extensively in patients with atrial fibrillation or atrial flutter, in patients undergoing cardioversion of atrial arrhythmias and in patients with mitral valve disease.

@ Right Left confusion is seen in

a) Milard gublard syndrome
b) gerstmann syndrome
c) Antons syndrome

Ans B

Gerstmann syndrome is characterized by four primary symptoms:

- Dysgraphia/agraphia: deficiency in the ability to write
- Dyscalculia/acalculia: difficulty in learning or comprehending mathematics
- Finger agnosia: inability to distinguish the fingers on the hand
- Left-right disorientation

This disorder is often associated with brain lesions in the dominant (usually left) hemisphere including the angular and supramarginal gyri near the temporal and parietal lobe junction.

@Not found in Iron deficiency anemia

a) increased RDW
b) Decreasd TIBC
c) decreased serum iron
d) Low serum ferritin

Likely lab test results in people with iron deficiency

- A complete blood count would likely reveal microcytic anemia
- Low serum ferritin

- *Low* serum iron
- High TIBC (total iron binding capacity)

It is possible that the fecal occult blood test might be positive, if iron deficiency is the result of gastrointestinal bleeding.

@ In nephrotic syndrome patient with steroid toxicity, alternative treatment includes -

a) levamisole
b) cyclophosphamide
c) mycophenolate

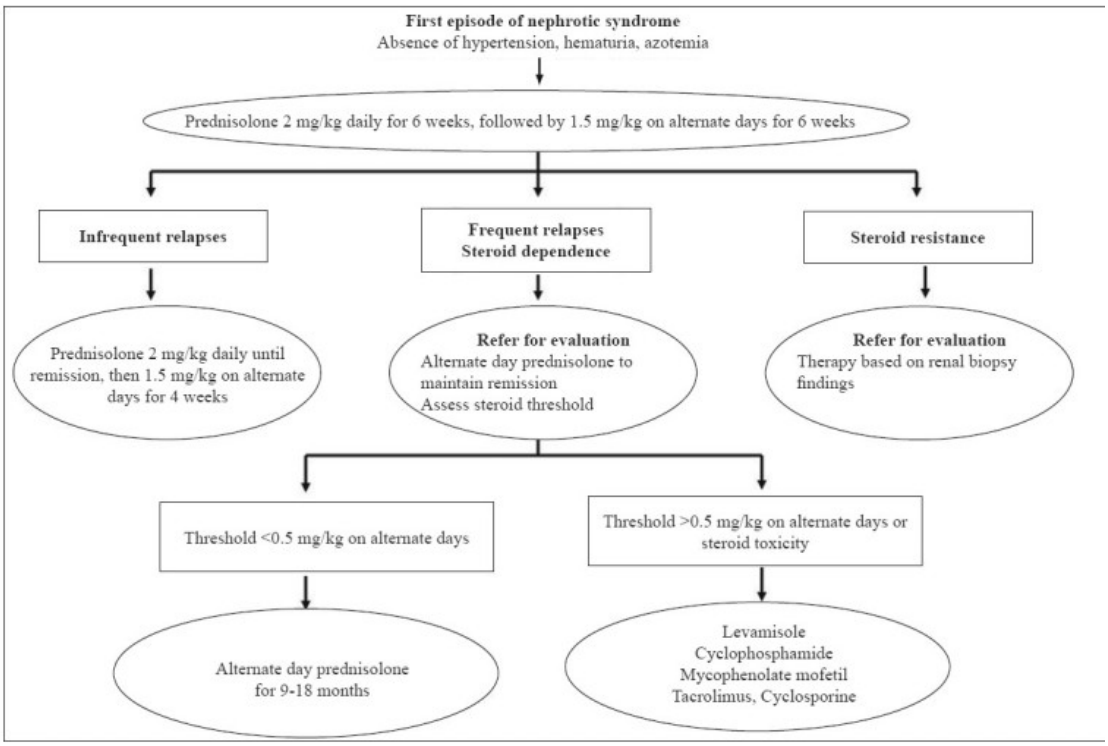
Ans B

The advantages of using these drugs should be balanced against their potential toxicity. There are few studies comparing one agent with another, but evidence for efficacy is strongest for cyclophosphamide and CsA. Levamisole has a modest steroid sparing effect and is a satisfactory initial choice for patients with frequent relapses or steroid dependence.

Treatment with ***cyclophosphamide is preferred*** in patients showing: (i) **significant steroid toxicity**, (ii) severe relapses with episodes of hypovolemia or thrombosis, and (iii) **poor compliance or difficult follow-up, where 12 weeks therapy** might be possible to ensure than long-term compliance.

Treatment with CsA or tacrolimus is recommended for patients who continue to show steroid dependence or frequent relapses despite treatment with the above medications.12 Either of these agents is effective in maintaining remission in most patients with steroid-sensitive nephrotic syndrome. The chief concern with their use is nephrotoxicity, but with careful assessment of renal function, minimizing the maintenance dose and utilizing renal biopsies in those receiving prolonged therapy, this risk can be minimized. Recent case series14 and a controlled trial15 support the use of MMF as a steroid sparing agent. The lack of renal, hemodynamic and metabolic toxicity with this agent makes it an attractive alternative to calcineurin inhibitors.

In some patients receiving therapy with levamisole, MMF and calcineurin inhibitors, treatment with prednisolone might be tapered and discontinued after 6-12 months. Some patients who respond to therapy with levamisole, MMF, and calcineurin inhibitors may relapse once these medications are discontinued. Relapses during or following therapy with these agents are treated with prednisolone as described above.



@ LBBB is seen in

a) recent mi
b) ashman focus
c) hyperkalemia
d) hypokalemia

Ans A

Among the causes of LBBB are:

- Aortic stenosis
- Dilated cardiomyopathy
- Acute myocardial infarction
- Extensive coronary artery disease
- Primary disease of the cardiac electrical conduction system
- Long standing hypertension leading to aortic root dilatation and subsequent aortic regurgitation

Long-Short Rule (Ashman Phenomenon): The ***earlier in the cycle a PAC occurs and the longer the preceding cycle***, the more likely **the PAC will be conducted with aberration**

This is because the refractory period of the ventricular conduction system is proportional to cycle length or heart rate; the longer the cycle length or slower the heart rate, the longer the recovery time of the conduction system. In most individuals the **right bundle normally recovers more slowly than the left bundle,** and a ***critically timed PAC is therefore more likely to conduct with RBBB than with LBBB***. In diseased hearts, however, LBBB aberrancy is also seen. Dr. Richard Ashman and colleagues first described this in 1947 in patients with atrial fibrillation. He noted that the **QRS complexes ending a short RR interval were often of a RBBB pattern if the preceding RR interval was long.**

@Bilateral ptosis. but no diplopia Emg myopathic pattern.. Ach Receptor antibosies -ve , Slight limb weakness , male patient Dx is

a) CPEO
b) Ocular Myasthenia gravis
c) Generalised Myasthenia gravis
d) If AchR Ab negative , exclude other diagnosis

Ans A
Chronic progressive external ophthalmoplegia (CPEO), also known as progressive external ophthalmoplegia (PEO), is a type of eye disorder characterized by slowly progressive inability to move the eyes and eyebrows.[1] It is often the only feature of mitochondrial disease, in which case the term CPEO may be given as the diagnosis. In other people suffering from mitochondrial disease, CPEO occurs as part of a syndrome involving more than one part of the body, such as Kearns-Sayre syndrome.
CPEO is a **slowly progressing disease.** It may begin at any age and progresses over a period of 5–15 years.[1] The first presenting symptom of ptosis is often unnoticed by the patient until the lids droop to the point of producing a visual field defect. Often, patients will tilt the head backwards to adjust for the slowly progressing ptosis of the lids. In addition, as the ptosis becomes complete, the patients will use the frontalis (forehead) muscle to help elevate the lids. The **ptosis is typically bilateral**, but may be unilateral for a period of months to years before the fellow lid becomes involved. Ophthalmoplegia or the inability/difficulty to move the eye is usually symmetrical. As such, **double vision is not often a complaint** of these patients. In fact, the progressive ophthalmoplegia is often unnoticed till decreased ocular motility limits peripheral vision. Often someone else will point out the ocular disturbance to the patient. Patients will move their heads to adjust for the lost of peripheral vision caused by inability to abduct or adduct the eye. All directions of gaze are affected, however, downward gaze appears to be best spared. This is in contrast to Progressive Supranuclear Palsy (PSP) which typically affects vertical gaze and spares horizontal gaze.
Elevated acetylcholine receptor antibody level which is typically seen in myasthenia gravis has been seen in certain patients of mitochondrial associated ophthalmoplegia
. Facial muscles may be involved which lead to atrophy of facial muscle groups producing a thin, expressionless face with some having difficulty with chewing. **Neck, shoulder and extremity weakness with atrophy** may affect some patients and ***can be mild or severe***.
@pco2-30,po2-115,pH-7.45.pt has compensated
a.respiratory alkalosis
b.metabolic alkalosis
c. respiratory.acidosis
d. metabolic.acidosis
Ans A
Four-Step Guide to ABG Analysis

- Is the **pH normal, acidotic or alkalotic?**
- Are the **pCO2 or HCO3 abnormal**? Which one appears to influence the pH?
- If both the pCO2 and HCO3 are abnormal, the **one which deviates most from the norm** is most likely causing an abnormal pH.
- Check the pO2. Is the **patient hypoxic**?

	pH	$PaCO_2$	HCO_3
Respiratory Acidosis			
Acute	< 7.35	> 45	Normal
Partly Compensated	< 7.35	> 45	> 26
Compensated	Normal	> 45	> 26
Respiratory Alkalosis			
Acute	> 7.45	< 35	Normal
Partly Compensated	> 7.45	< 35	< 22
Compensated	Normal	< 35	< 22
Metabolic Acidosis			
Acute	< 7.35	Normal	< 22
Partly Compensated	< 7.35	< 35	< 22
Compensated	Normal	< 35	< 22
Metabolic Alkalosis			
Acute	> 7.45	Normal	> 26
Partly Compensated	> 7.45	> 45	> 26
Compensated	Normal	> 45	> 26

@H/o fever,headache followed by GTCS , 2 episodes,CSF cell-300 WBCs)/µL,protein-70 mg/dL,sugar-50 **mg/dL**

a) herpes encephalitis
b) cerebral malaria
c) Pyogenic meningitis

Ans A

Acutely, a typical “viral profile” is identified. Red blood cells (RBCs) and xanthochromia may be seen. Patients typically have mononuclear pleocytosis of 10-500 white blood cells (WBCs)/µL (average, 100 WBCs/µL). As a result of the hemorrhagic nature of the underlying pathologic process, the RBC count may be elevated (10-500/µL).

Protein levels are elevated to the range of 60-700 mg/dL (average, 100 mg/dL). Glucose v**alues may be normal or mildly decreased (30-40 mg/dL).**

@ difference between seizure and syncope-

a) urine incontinence
b) clear consciousness with weakness
c) post ictal confusion/sensorium in epilepsy

Ans C

Comparison of clinical features in cardiogenic syncope versus seizure disorders

Clinical features	Cardiogenic syncope	Seizure disorders
Loss of consciousness	Typical	Common
Episode duration	Seconds	Minutes
Involuntary movements	Common	Typical
Amnesia	Yes	Yes
Arrhythmia	Common	Rare*
Electroencephalogram	Slow waves Flattening	Focal or general spike activity
Responsive to AEDs	No	Often
Short term mortality†	High	Low

*Except sinus tachycardia.

Features that Distinguish Generalized Tonic-Clonic Seizure from Syncope

Features	Seizure	Syncope
Immediate precipitating factors	Usually none	Emotional stress, Valsalva, orthostatic hypotension, cardiac etiologies
Premonitory symptoms	None or aura (e.g., odd odor)	Tiredness, nausea, diaphoresis, tunneling of vision
Posture at onset	Variable	Usually erect
Transition to unconsciousness	Often immediate	Gradual over seconds[a]
Duration of unconsciousness	Minutes	Seconds
Duration of tonic or clonic movements	30–60 s	Never more than 15 s
Facial appearance during event	Cyanosis, frothing at mouth	Pallor
Disorientation and sleepiness after event	Many minutes to hours	<5 min
Aching of muscles after event	Often	Sometimes
Biting of tongue	Sometimes	Rarely
Incontinence	Sometimes	Sometimes
Headache	Sometimes	Rarely

@ after failure of medical therapy,what is the best option for epilepsy patients

a) deep brain stimulation
b) vagus stimulation
c) epilepsy surgery

deep brain stimulation is used in cases of parkinsonism not responding to medicines
Epilepsy: The procedure has been tested for effectiveness in patients with severe epilepsy.[38] A review of 189 studies covering DBS as well

as vagus nerve stimulation and closed-loop stimulation (responsive neurostimulator [RNS]) indicate that neurostimulation provides "another tool with which to treat the complex disease of medically refractory epilepsy

Vagus nerve stimulation (**VNS**) is an adjunctive treatment for certain types of intractable epilepsy and treatment-resistant depression.

@ .genetic defect in MODY

a) hnf 1a
b) HEPATOCYTE NUCLEAR FACTOR 4A

Ans A

Mutations in the HNF1A **gene** encoding **hepatocyte nuclear factor**-1 alpha are the most common cause of **MODY** in most adult populations

@ Embryonal Hb contains

a) **Zeta and epsilon**
b) **Alpha and Gamma**
c) **Alpha and Beta**

Ans A

- **Embryonic hemoglobin** is a tetramer produced in the blood islands in the embryonic yolk sac during the mesoblastic stage (first week of pregnancy until the end of the pregnancy). The protein is commonly referred to as **Hemoglobin** ϵ or **HbE**.
- Chromosomal abnormalities can lead to a delay in switching from embryonic hemoglobin.[1]
- Subtypes include **Gower 1, Gower 2, and Portland 2**

The epsilon globin gene (HBE) is normally expressed in the embryonic yolk sac: **two epsilon chains** together **with two zeta chains (an alpha-like globin)** constitute the ***embryonic hemoglobin Hb Gower I***; **two epsilon chains together with two alpha chains f**orm the ***embryonic Hb Gower II.*** Both of these embryonic hemoglobins are normally supplanted by fetal, and later, adult hemoglobin

In the embryo:

- Gower 1 (ζ2ε2)
- Gower 2 (α2ε2) (PDB 1A9W)
- Hemoglobin Portland (ζ2γ2).

In the fetus:

- Hemoglobin F (α2γ2) (PDB 1FDH).
- In adults:
- Hemoglobin A (α2β2) (PDB 1BZ0) - The most common with a normal amount over 95%
- Hemoglobin A2 (α2δ2) - δ chain synthesis begins late in the third trimester and in adults, it has a normal range of 1.5-3.5%
- Hemoglobin F (α2γ2) - In adults Hemoglobin F is restricted to a limited population of red cells called F-cells. However, the level of Hb F can be elevated in persons with sickle-cell disease and beta-thalassemia.

Variant forms that cause disease:

- Hemoglobin H (β4) - A variant form of hemoglobin, formed by a tetramer of β chains, which may be present in variants of α thalassemia.
- Hemoglobin Barts (γ4) - A variant form of hemoglobin, formed by a tetramer of γ chains, which may be present in variants of α thalassemia.
- Hemoglobin S (α2βS2) - A variant form of hemoglobin found in people with sickle cell disease. There is a variation in the β-chain gene, causing a change in the properties of hemoglobin, which results in sickling of red blood cells.
- Hemoglobin C (α2βC2) - Another variant due to a variation in the β-chain gene. This variant causes a mild chronic hemolytic anemia.
- Hemoglobin E (α2βE2) - Another variant due to a variation in the β-chain gene. This variant causes a mild chronic hemolytic anemia.
- Hemoglobin AS - A heterozygous form causing Sickle cell trait with one adult gene and one sickle cell disease gene
- Hemoglobin SC disease - A compound heterozygous form with one sickle gene and another encoding Hemoglobin C.