Fanconi anemia - a type of idiopathic refractory anemia characterized by pancytopenia, hypoplasia of the bone marrow, and congenital anomalies, occurring in members of the same family (an autosomal recessive trait); the anemia is normocytic or slightly macrocytic, macrocytes and target cells may be found in the circulating blood, and the leukopenia usually is due to neutropenia. Congenital anomalies include short stature; microcephaly; hypogenitalism; strabismus; anomalies of the thumbs, radii, and kidneys and urinary tract; mental retardation; and microphthalmia.

Fanconi syndrome is a disease of the proximal renal tubules of the kidney in which glucose, amino acids, uric acid, phosphate and bicarbonate are passed into the urine, instead of being reabsorbed. The loss of bicarbonate results in Type 2 or proximal renal tubular acidosis. The loss of phosphate results in the bone disease rickets (even with adequate vitamin D and calcium), because phosphate is necessary for bone development.

Bilirubin occurs in plasma in four forms (Nelson 18/e Chapter-352):

- free or unbound bilirubin (the form responsible for kernicterus, because it can cross cell membranes)
- unconjugated bilirubin tightly bound to albumin
- conjugated bilirubin (the only fraction to appear in urine). In the liver it is conjugated with glucuronic acid by the enzyme Glucuronyltransferase, making it soluble in water.
  - δ fraction - the fraction of bilirubin covalently bound to albumin* in conventional methods it is measured as part of conjugated bilirubin. Because of its covalent bond during the recovery phase of hepatocellular jaundice, it may persist in the blood for a week or more after urine clears.

Although the terms direct and indirect bilirubin are used equivalently with conjugated and unconjugated bilirubin, this is not quantitatively correct, because the direct fraction includes both conjugated bilirubin and δ bilirubin.

Hepatorenal syndrome (HRS) is defined as functional renal failure in patients with end-stage liver disease. The pathophysiology of HRS is poorly defined, but the hallmark is intense renal vasoconstriction (mediated by hemodynamic, humoral, or neurogenic mechanisms) with coexistent systemic vasodilation. The diagnosis is supported by the findings of oliguria (<1 mL/kg/day), a characteristic pattern of urine electrolyte abnormalities (urine sodium of <10 mEq/L, fractional excretion of sodium of <1%, urine : plasma creatinine ratio <10, and normal urinary sediment), absence of hypovolemia, and exclusion of other kidney pathology. The best treatment of HRS is timely liver transplantation, as complete renal recovery can be expected.

Elevations in serum AP, 5′ nucleotidase (5′NT), and γ-glutamyl transpeptidase (GGT) levels are also sensitive indicators of obstruction or inflammation of the biliary tract.

Pancoast syndrome is a/w squamous cell ca (not adeno)

Herpes simplex encephalitis (HSE)-

- m/c viral encephalitis
- *In children older than 3 months and in adults, herpes simplex encephalitis (HSE) is usually localized to the temporal and frontal lobes and is caused by herpes simplex virus type 1 (HSV-1).
  - In neonates, however, brain involvement is generalized, and the usual cause is herpes simplex virus type 2 (HSV-2), which is acquired at the time of delivery.
○ HSE must be distinguished from herpes simplex meningitis, which is more commonly caused by HSV-2 and which often occurs in association with a concurrent herpetic genital infection.

○ CT- low density lesions in temporal lobes. Hemorrhage is highly suggestive and seen in later disease course.

○ MRI- temporal lobe hyperintense

- Metavir Scoring is done for- Chronic heaptits
- Natalizumab in multiple sclerosis n Crohn's as an antibody against alpha 4 integrin inhibiting T lymphocyte adhesion n migration to lesions in these diseases.
- NEW classification liver cancer -->OKUDA, CLIP primary cause of community acquired pneumonia ----> MRSA
- albright's osteodystrophy is NORMAL calcium, not decreased calcium

- Plummer vinson's syndrome- Combination of symptomatic hypopharyngeal webs and Iron deficiency anemia in middle aged female. Presence of web is in postcricoid* region. Hemoglobin is low and there is hypochromic microcytic anemia. All cases have a low serum iron and high iron binding capacity. Iron shold be given orally to correct Hb levels.

- Atrial fibrillation- Enlarged left atrium and *increased left atrial pressure.

- Procollagen suicide phenomenon seen in- OI (Osteogenesis Imperfecta)

The etiologic agent in over 90% cases of post-transfusion hepatitis is hepatitis C. It is also associated with incidence of chronic active hepatitis upto 16% and an *8-10% increase of cirrhosis or hepatoma or both. CMV infection is not a significant problem in immunocompetent recipients and hence blood is not routinely tested for CMV. (Q-178 GT-87)

- Diabetes insipidus(DI) is a condition characterized by excessive thirst and excretion of large amounts of severely diluted urine, with reduction of fluid intake having no effect on the latter. There are several different types of DI, each with a different cause.
  ○ The most common type in humans is central DI, caused by a deficiency of arginine vasopressin(AVP), also known as antidiuretic hormone(ADH). Vasopressin acts at the distal tubule and collecting ducts to increase permeability to water not a/w electrolytes i.e. free water. Hence central DI is a/w *free waterclearence. Under normal circumstances, this would result in the movement of free water from renal tubule to capillary. Under conditions of vasopressin deficiency, as in nephrogenic DI water remains in the tubules, resulting in excretion of dilute urine.
  ○ The second common type of DI is nephrogenic diabetes insipidus, which is caused by an insensitivity of the kidneys to ADH. Symptoms are similar to neurogenic DI except plasma levels of vasopressin are increased.

- Hyperuricemia- A purine rich diet can cause hyperuricemia. Foods high in the purines adenine and hypoxanthine may be more potent in exacerbating hyperuricemia. Hyperuricemia is a commonly reported trigger of acute gout. This is believed to be due to temperature dependent precipitation of uric acid crystals in tissues at below normal temperature. Though asymptomatic hyperuricemia does increase the risk of acute gouty arthritis prophylaxis is not recommended.

- HBeAg negative means-
  ○ Chronic carrier low infectivity
  ○ Precore mutant (infective but still HBeAg negative)
  ○ both can be differentiated by VIRAL LOAD , ALT LEVEL

- Fanconi anemia- a type of idiopathic refractory anemia characterized by pancytopenia, hypoplasia of the bone marrow, and congenital anomalies. The anemia is normocytic or slightly macrocytic, macrocytes and target cells may be found in the circulating blood, and the leukopenia usually is due to neutropenia. Congenital anomalies include short stature; microcephaly; hypogenitalism; strabismus; anomalies of the thumbs, radii, and kidneys and urinary tract; mental retardation; and microphthalmia

- Congenital hypoplastic anemia- hypoproliferative macrocytic anemia. A macrocytic anemia resulting from congenital hypoplasia of the bone marrow, which is grossly deficient in erythroid precursors while other elements are normal; anemia is progressive and severe, but leukocyte and platelet counts are normal or slightly reduced; survival of transfused erythrocytes is normal; minor congenital anomalies are found in some patients. (Syn: congenital nonregenerative anemia, Diamond-Blackfan anemia, familial hypoplastic anemia, pure red cell anemia, erythrogenesis imperfecta, Diamond-Blackfan syndrome.)
Q- Which of the following is not an indication of Digoxin-specific Fab antibodies in children-
1. Supraventricular tachycardia------ans
2. Overdose > 4 mg
3. Serum digoxin levels > 10ng/ml
4. Progressive bradycardia

Explanation-

Treatment of Digoxin toxicity-
- Supraventricular tachycardia- KCl
- Frequent Ventricular premature beats- Lidocaine/Phenytoin
- Ventricular tachycardia/Fibrillation- Cardioversion/Digibind
- Second or third degree heart block- Atropine/Catheter/Pacing/Digibind

Indications of digibind are-
1. Accidental overdose >4mg
2. Serum digoxin level > 10 ng/ml
3. Life threatening rhythm disturbance→ Ventricular arrhythmias/Progressive bradycardia(2nd or 3rd degree heart block)

Q. A 40 yr old male alcoholic presents with a 6 days history of binge drinking. Serum chemistry tests reveal the following:
Electrolytes- Na+ 145, K+ 5, Cl- 105, HCO3- 15
Creatinine- 1.5 mg/dl
Glucose- 172
The nitroprusside agent gives a minimally positive result. Optimal therapy to ameliorate the patient’s acid-base disorder would include 5% dextrose in-
1. Water
2. NS-----------------------------------------ans
3. NS, insulin, and sodium bicarbonate
4. 1/2 NS and insulin

Discussion- H/17 P-291/292
Metabolic acidosis d/t alcohol (alcoholic ketoacidosis). Patients suffering from alcoholic ketoacidosis do well on glucose and saline. Neither insulin nor alkali is required unless the acidosis is extreme (bicarbonate <6 to 8)

Q. All are tests for stoppage of circulation EXCEPT:
1. Magnus test
2. Winslow’s test--------ans
3. Icard’s test
4. Diaphanous test

Discussion-
Winslow’s test- No movement of reflection of light shown on mirror, or surface of water in bowl, kept on the chest. Its a Test of respiration.

• The sequence of events in AIP(2nd M/C porphyria) attacks usually is-
  - Abdominal pain
  - Psychiatric symptoms, such as hysteria
  - Peripheral neuropathies, mainly motor neuropathies
  - Most patients are completely free of symptoms between attacks
  - AIP displays neurovisceral symptoms but no skin manifestations.

• The S4 heart sound is associated with any process that increases the stiffness of the ventricle including:
  - Hypertrophy of the ventricle
  - Long-standing hypertension (causes ventricular hypertrophy)
  - Aortic stenosis (causes ventricular hypertrophy)
  - Overloading of the ventricle (causes ventricular hypertrophy)
- Fibrosis of the ventricle (eg. post-MI)
- Congestive Heart Failure

- Lymphoma of the brain (usually diffuse large cell) is increasingly common as a sporadic tumor and occurs frequently in immunosuppressed patients, specially those with AIDS. It's clinical sensitivity to glucocorticoids can mistakenly suggest a diagnosis of multiple sclerosis, and its complete disappearance or dramatic improvement on CT after steroid therapy is baffling. Radiosensitivity is a well known feature of most primary CNS lymphomas, which almost always are of "B-Cell origin."

- Fibromyalgia- A history of sleep disturbances and widespread musculoskeletal pain associated with trigger points* (localised area of tenderness on palpation).

- Alimentary glycosuria: Glycosuria developing after the ingestion of a moderate amount of sugar or starch, which normally is metabolized without appearing in the urine. Seen in hyperthyroidism.

- In 1-alpha-hydroxylase deficiency deficiency of Vit. D cannot be corrected by supplementation.

- Auerbach's regime used in treatment of---> Malignant pheochromocytoma

- Sarcoidosis- mostly incidious but presents acutely as-
  - Heerford Waldenstrom syndrome*- fever, parotid enlargement, anterior uveitis, and facial nerve palsy
  - Lofgren's syndrome- erythema nodosum, arthralgias, and bilateral hilar lymphadenopathy

- A normal MCV within 2 days after acute bleeding---> because BM has not got enough time to release reticulocytes(which are larger than mature RBCs and could potentially increase the MCV)

### Gonococcal urethritis vs Non-specific urethritis

<table>
<thead>
<tr>
<th>Gonococcal urethritis</th>
<th>Non-specific urethritis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gonococcal urethritis presents <em>within the first week of sexual exposure.</em> Gonococcal urethritis has a thick creamy exudate</td>
<td>Non-specific urethritis is most commonly caused by Chlamydia trachomatis. The IP is 2-3wks following sexual exposure. Purulent exudate in non-specific urethritis is thin and watery</td>
</tr>
</tbody>
</table>

Patient complain of dysuria and increased frequency of urination in both types of urethritis. Gram stain of exudate in both types has numerous nutrophils, but in gonoccocal urethritis, gram negative diplococci are visible in the cytoplasm. Chlamydia is not visible on routine gram stains

- Allelic heterogeneity is the phenomenon in which different mutations at the same locus(or gene) cause the same disorder. Eg. Beta-thalassemia

- Wilson's d/e---> wrong statement--> Ceruloplasmin<100
  - Defective copper incorporation into apoceruloplasmin leads to excess catabolism and low blood levels of ceruloplasmin.

- Few important points-
  - Chronic fundal gastritis a/w pernicious anemia
  - Chronic antral gastritis a/w H.pylori infection
  - Barretts esophagus l/t adenocarcinoma
  - Achalasia cardia l/t squamous cell carcinoma

### Q. A lady on rifampicin & warfarin develop pulmonary embolism. The best management is--

1) Replace warfarin by long term jeparin therapy
2) Give LMWH
3) Replace warfarin 4 acetacumarin
4) Replace rifampicin 4 ethambutol-----ans given

Discussion-

1) Since the lady is on rifampicin she must b suffering frm tb or leprosy...As rifampicin is cidal drug and most imp drug in treatment regimen of both tb and leprosy, it cannot be replaced by ethambutol, which is a satic drug. So i
think warfarin should be stopped and replaced by LMWH.
2) Yea, u r right. But we have no such option in the question. The second option says that "Give LMWH", not "replace warfarin with LMWH". Rifampin do induce the metabolism of Warfarin in liver and affects INR. The induction of metabolism is unpredictable. Long term heparin therapy as such carries a lot of risk of complications. Acenocumarin also interact with Rifampin. So, the 4th Option is the best option among all of these. Isoniazid is also a -Cidal drug, so shd be there in therapy too.

Q- Digitalis act in atrial fibrillation by-
1)increasing AV node refactororiness  
2)decreasing atrial contractility  
3)inhibiting Na+K+ATPase  
4)inhibiting Na+H+ATPase  
Discussion- Ans is -1)
Basic mechanism to Na+K+ATPase inhibition h, then why the ans. Is 1)... Digitalis also acts on the Vagus nerve through the same channel, and increases its discharge (i.e., it increases vagus tone). Increased vagus tone in turn inhibits AV node and make it refractory. So, it prevents the impulses generated in atria to reach Ventricles, control ventricular rate and improves hemodynamic stability in Atrial fibrillation.

Q. Damage to categorial hemisphere usually leads to-
 a. normal speech  
b. increased speech  
c. decreased speech answer  
d. senseless fluent speech  
Discussion- ans given for this in aa is {d} 
also causes Dyslexia which is a broad term applied to impaired ability to read, due to an inherited abnormality.
Causes of Dyslexia: 
Reduced ability to recall speech sounds, so there is trouble translating them mentally into sound units (phonemes).
There is a defect in the magnocellular portion of the visual system that slows processing and also leads to phonemic deficit. There is decreased blood flow in angular gyrus in categorical hemisphere in both cases.

Q. Most sensitive diagnostic test 4 dengue is-
 a. IgM elisa  
b. complement fixation test  
c. neutralization test answer... routinely done at airports in china n taiwan  
d. electron microscopy  
Discussion- Famous by name nsst...... igm elisa was usedd to b... now it is nsst 95% sp n 99% sn...

• Primary Immunodeficiency disorders:
  ◦ Primary B-cell diseases include panhypogammaglobulinemia (Bruton disease), an X-linked deficiency of all three major classes of immunoglobulins, as well as other selective deficiencies of the immunoglobulins or their subgroups. This condition presents after 3 months of age (after maternal antibodies wane) with recurrent and often simultaneous bouts of otitis media, pneumonia, diarrhea, and sinusitis.
  ◦ T-cell: Among the T-cell diseases is DiGeorge anomaly, in which defective embryologic development of the third and fourth pharyngeal pouches results in hypoplasia of both thymus and parathyroid glands. Associated findings with DiGeorge anomaly include CATCH: C for cardiac, A for abnormal faces, T for thymic hypoplasia, C for cleft palate, and H for hypocalcemia.
  ◦ Combined B and T-cell diseases:
- X-linked recessive Wiskott-Aldrich syndrome of mild T-cell dysfunction, diminished serum IgM, marked elevation of IgA and IgE, eczema, recurrent middle-ear infections, lymphopenia, and thrombocytopenia.
- Severe combined immunodeficiency disease (SCID), have deficient T- and B-cells. Consequently, they are both marked lymphopenia and agammaglobulinemia, as well as hypoplasia of the thymus.
- Ataxia telangiectasia and chronic mucocutaneous candidiasis.

- Complication of malaria (CHAPLIN):
  - Cerebral malaria/ Coma
  - Hypoglycemia
  - Anaemia
  - Pulmonary edema
  - Lactic acidosis
  - Infections
  - Necrois of renal tubules (ATN)

- wind swept deformity of hands------rheumatoid arthritis
  - wind swept deformity of knee------rickets

- most common cause of pyometra in india---carcinoma cervix
  in western(developed)countries-----senile endometritis

when nothing mentioned „we should choose what's commoner in india and hence choose cancer cervix as the answer.

- In COPD:
  - Bronchitis has B... so they are called "Blue bloaters". Cyanosis is characteristic finding.
  - emPhysema has P...so they are called "Pink Puffers".

- Behcet's syndrome-
  1. Recurrent genital ulcers
  2. Skin lesions
  3. Eye lesions
  4. Pathergy test

- **Supraventricular tachycardia**: treatment ABCDE:
  - Adenosine
  - Beta-blocker
  - Calcium channel antagonist
  - Digoxin
  - Excitation (vagal stimulation)

- **Ventricular tachycardia**: treatment LAMB:
  - Lidocaine
Neurofibromatosis: diagnostic criteria (type-1) CAFE SPOT:
- Cafe-au-lait spots
- Axillary-Inguinal
- Freckling Fibroma
- Eye: lisch nodules
- Skeletal (bowing leg, etc)
- Pedigree/ Positive family history
- Optic Tumor (glioma)

Q. Which of the following is true regarding Asthma-
A) Reduced FRC, Reduced Residual Volume
B) Reduced FRC, Increased Residual Volume
C) Increased FRC, Reduced Residual Volume
D) Increased FRC, Increased Residual Volume

- Necrobiosis lipoidica diabeticorum --> found over anterior surface of legs
- Persons with PV usually have diminished cerebral blood flow and are particularly at risk for developing thrombotic complications. Functional platelet abnormalities may cause both thrombotic and bleeding problems (gi tract is common site of bleeding), and affected persons are frequently iron-deficient.
- T wave inversion of the anterior leads especially V1-V4 seen in --> pulmonary embolism. Geneva score is used in pul. embolism

<table>
<thead>
<tr>
<th>Anti mitochondrial antibody</th>
<th>Primary biliary cirrhosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anti smooth muscle antibody</td>
<td>Autoimmune hepatitis</td>
</tr>
</tbody>
</table>

- Coma vigil --> akinetic mutism --> a state in which patient lies with eyes open, yet unresponsive to the outside world --> seen in typhoid
- Mycoplasma pneumoniae --> diagnosis is confirmed by Cold agglutinins. As in other atypical pneumonias, radiographic abnormalities may be more prominent than would be predicted by auscultation of chest (normal or nearly normal)
- Lissencephaly --> smooth brain (lack development of gyri and sulci)

Q- Breath sounds are decreased in all except
1. Lobar pneumonia -------- ans
2. Pleural effusion
3. Pneumothorax
4. Atelectasis

Explanation- Due to consolidation in lobar pneumonia, high pitched bronchial breadth sound and whispering pectoriloquy are present

Q. Which of the following is true regarding Rheumatoid arthritis - (AI 94)
a) Typically involves small and large joints symmetrically but spares the cervical spine
b) Causes pleural effusion with low sugar
c) Pulmonary nodules are absent
d) Enthesopathy prominent
**Q. Hyperbaric oxygen is useful in the treatment of all, except:**

1. CO poisoning
2. Gas gangrene
3. Atelectasis-----------------ans
4. Cyanide poisoning

**Explanation:** In atelectasis alveolar collapse leads to shunting of the desaturated blood at the alveolar capillary level and thus the desaturated blood is not available for oxygenation.

- Plasma levels of many polypeptide hormones (parathyroid hormone, insulin, glucagons, LH and prolactin) rise with renal failure. Uremic patients are also resistant to the action of insulin. The glucose intolerance of uremia results mainly from this peripheral resistance to the action of insulin.

- **Fanconi Syndrome** is characterised by glycosuria, aminoaciduria, and phosphaturia (mnemonic **GAP**). Aminoaciduria is generalised and other associated abnormalities may be bicarbonaturia, hyperkaluria, uricosuria, and sodium wasting. Clinical features are linear growth failure and vitamin D deficiency rickets. Symptoms typically appear in 1st six months of life and consists of vomiting, polydipsia, polyuria and constipation.

- **Mycosis fungoides**---> Cutaneous T-Cell lymphoma (NHL)---> name mycosis fungoides is somewhat misleading--it loosely means "mushroom-like fungal disease"

- During immediate 30 min postictal period serum prolactin level rises. This does not happen in pseudoseizure--> hence used to differentiate the two

- Asthma may be aggravated by Chlamydia or mycoplasma pneumoniae. Treatment with macrolide antibiotics benefits patients with BA. In asthma airway obstruction is reversible,

- Multiple sclerosis also causes facial palsy but taste is not lost unlike in bell’s palsy

- A lesion to the left prefrontal area produces depression and uncontrollable crying. In contrast a comparable lesion to the right prefrontal area may produce laughter, euphoria and a tendency to joke and make puns.

- Y decent and S3 are produced in the rapid filling phase of cardiac cycle. That’s why S3 is also known as filling sound.

- **Mineral deficiency**-
  - **Zinc deficiency** is characterised by alopecia, a maculopapular rash around the mouth and eyes, taste dysguesia and smell abnormalities and problems with healing of wounds.
  - **Essential fatty acid deficiency** is characterised by an eczematous rash and thrombocytopenia.
  - **Magnesium deficiency** is associated with resistance to activity of parathormone with subsequent hypocalcemia and tetany. Muscle weakness, irritability, delirium and convulsions are also noted. Alcoholism is the most common cause of hypomagnesemia
  - **Copper deficiency** is associated with iron deficiency anemia, dissecting aortic aneurysm and kinky hair syndrome

- Because helper T cells are integral to normal cellular immunity (type IV hypersensitivity) tests that evaluate cellular immunity are impaired in HIV. This lack of immune response is called **anarchy**. In vitro stimulation of T-cell response to phytohemagglutinin, a potent T-cell mitogen is also impaired.

- **DIC-** **Antithrombin III** levels may be low due to the combination of increased consumption and decreased synthesis. Other findings- Hypofibrinogenemia, Thrombocytopenia, Fibrin degradation products and prolonged prothrombin time

- **Lhermitte’s sign:** also known as **barber chair sign** is an electrical shocklike sensation (induced by flexion or other movements of the neck) that radiates down the back into the legs. Rarely it radiates into the arms. Found in **MS**, Cervical spondylosis & posterior column lesion.

- **DVT-Risk factors:**
  - PTH(=mnemonic)-
    - PNH, Polycythemia
- Thrombophilia--> activated protein C resistance, protein C and S deficiency
- Hematological, Hyperviscosity syndrome, Homocysteinuria
  - Autoimmune-Antiphospholipid syndrome
  - Drugs- Antipsychotics(recent evidence)
    - OCP: 3rd G > 2nd G

- **Pierre Robin Syndrome**- isolated cleft palate, retrognathia, and a posteriorly displaced tongue(glossitis) and is associated with early respiratory and feeding difficulties
- **J-curve phenomenon**- point beyond which blood pressure reduction in hypertensive subjects is no longer beneficial and possibly even deleterious. Clinical trials have found no evidence of ‘j-curve phenomenon’ at blood pressure reductions achieved in clinical practice.
- Channelopathy of Sodium, potassium and calcium are autosomal dominant
- **Clinical Relapse**- In type A hepatitis once the patient is fully cured he may again develop symptoms of fever, malaise, anorexia and vomiting without jaundice. The LFT is normal.

Q. A 40 yrs old lady underwent cholecystectomy for gall stones. 24 hrs after the surgery she became confused and developed tachycardia. On examination lungs were clear. Her arterial blood gas analysis showed pO2 = 50, pCO2= 28, pH=7.49. The most important next investigation to establish the cause of her deteriorating condition would be-
  1. Chest roentgenogram
  2. Blood culture
  3. ECG
  4. Lung scan---------ans

Discussion- Development of respiratory distress, hypoxia and confusion following surgery; without any obvious reason should make one to suspect PTE. Other risk factors for PTE are Obesity, Pregnancy, OCPs etc. Lung scanning is the principal imaging test for diagnosis of PTE, which detects perfusion defect indicating decreased or absent pulmonary blood flow. Chest X-Ray is normal or near normal in most cases of PTE and a normal or near normal chest X-Ray in a dyspnoic patient suggests PE. Well established abnormalities include focal oligemia(Westernmark’s sign), a peripheral wedged shaped opacity above the diaphragm(*Hompton’s hump), or an enlarged right descending pulmonary artery(Palla’s sign). The ECG is often abnormal in PE, but findings are not sensitive, not specific. ECG changes include sinus tachycardia*, new onset atrial fibrillation or flutter and S1Q3T3 (it is a sign of acute cor pulmonale). Any cause of acute cor pulmonale (PE, PTX, bronchospasm, etc) can result in the S1Q3T3 finding on the electrocardiogram. Anterior T wave inversions suggest the diagnosis of massive or sub-massive PE. Overall the greatest utility of the ECG in the patient with suspected PE is ruling out other potential life-threatening diagnoses such as MI.

- Pericarditis in renal failure(acute or chronic) is an indication to initiate haemodialysis, because untreated uremic pericarditis may progress to pericardial tamponade. Other indications for haemodialysis include encephalopathy, volume overload, and intractable hyperkalemia. Also BM depression, mainly due to reduced erythropoietin combined with mildly reduced red cell half-life, causes hematocrit to fall almost universally in renal failure(acute and chronic). This does not determine need for dialysis.

  ⇒ Causes of wide pulse pressure:
    - AR
    - Hyperthyroidism
    - Pregnancy
    - Severe anemia
    - Paget’s disease
    - Beri-beri

These are also found in (are causes of): Water hammer pulse / Systolic HT

<table>
<thead>
<tr>
<th></th>
<th>Dementia(MMSE &lt;24)</th>
<th>Delirium</th>
</tr>
</thead>
<tbody>
<tr>
<td>Onset</td>
<td>Insidious</td>
<td>Acute</td>
</tr>
<tr>
<td>Course</td>
<td>Usually protracted</td>
<td>Usually recover in 1 week</td>
</tr>
<tr>
<td>Consciousness</td>
<td>N</td>
<td>Clouded</td>
</tr>
<tr>
<td>Orientation</td>
<td>N</td>
<td>Disturbed</td>
</tr>
<tr>
<td>-------------</td>
<td>---</td>
<td>-----------</td>
</tr>
<tr>
<td>Memory</td>
<td>Immediate retention and recall normal Immediate retention and recall disturbed* Recent memory disturbed only in late stages Recent memory disturbed</td>
<td></td>
</tr>
<tr>
<td>Comprehension</td>
<td>Impaired only in late stages</td>
<td>Impaired</td>
</tr>
<tr>
<td>Sleep-wake cycle</td>
<td>N</td>
<td>Disturbed</td>
</tr>
<tr>
<td>Attention and concentration</td>
<td>N</td>
<td>Disturbed</td>
</tr>
<tr>
<td>Diurnal variation</td>
<td>-nt</td>
<td>Marked, Sundowning +nt--&gt; Onset or exacerbation of delirium during the evening or night</td>
</tr>
<tr>
<td>Perception</td>
<td>Hallucinations may occur</td>
<td>Visual illusions and hallucinations very common</td>
</tr>
</tbody>
</table>

- First sign seen in hypoglycemia--> loss of fine motor skills
- **Wernicke's encephalopathy**- classic triad(GOA)-
  - Global confusion
  - Ophthalmoplegia
  - Ataxia
When the diagnosis is suspected thiamine should be administered before glucose since glucose can precipitate worsening of the disease. Many of the patients who recover from the acute encephalopathy will be left with a profound defect in memory and learning k/a **Korsakoff’s psychosis.**

- Drugs that can be cleared with-
  - Hemodialysis- **BLAST**
    - Barbiturate
    - Lithium
    - Alcohol(includes--> methanol, ethylene glycol)
    - Salicylates
    - Theophylline(charcoal hemoperfusion preferred)
  - Acid diuresis- **3 Queens PACT**
    - Quinine
    - Quinidine
    - ChloroQuine
    - Phencyclidene
    - Amphetamine
    - Cocaine
    - TCA, Tocainide
  - Alkaline diuresis- **MSC PDF**
    - Mtx
    - Salicylate, Sulfonamide
    - Chlorpropamide
    - Phenobarbitone
    - Diflunisal
    - Fluorine
  - Saline diuresis- **ABC FILM**
    - Alcohol
    - Br
    - Ca
    - Fluorine
    - INH
    - Li
    - Meprobamate
  - Hemoperfusion- **BEG CPMT**
    - Barbiturates
    - Ethchlorvynol
    - Glutethimide
- Chloramphenicol
- Phenytoin/Procaainamide
- Meprobamate/Methaqualone
- Theophylline

- **Drugs which **can-not **be cleared with hemodialysis**- **ABCDs**
  - Anti-cholinergic (TCA & Organophosphates)
  - Benzodiazepines, Beta blockers
  - Compound like kerosene oil
  - Dextropropoxyphene (Co-proxamol)
  - Digoxin

**The psychogenic stupor** can be differentiated from organic stupor by-
  - The doll’s head eye phenomenon or oculocephalic reflex - **absent** in psychogenic stupor
  - Oculovestibular reflex - **present**
  - Protective reflexes - **present**
  - Resistance to eye opening - **present**

**Bruit is found in hepatoma** - **highly vascular tumor**
- **Relative polycythemia (due to relative decrease in plasma volume)** - **seen in dengue hemorrhagic fever**
- Vit. A causes rupture of lysosomal membranes (but carotenoids do not cause toxicity except a reversible yellow discoloration of skin)

**CARDIOLOGY**

**Hypertension**

1. Mr. Sharma 46 years old has BP of 158/102 he has-
   1. Normal BP
   2. Pre hypertensive
   3. Stage I HT
   4. Stage II HT--------ans

**Discussion:** New classification of Hypertension (JNC VII):

<table>
<thead>
<tr>
<th>Classification</th>
<th>Systolic</th>
<th>Diastolic</th>
</tr>
</thead>
<tbody>
<tr>
<td>NORMAL</td>
<td>&lt;120</td>
<td>and</td>
</tr>
<tr>
<td>Pre-hypertension</td>
<td>120-139</td>
<td>and/or</td>
</tr>
<tr>
<td>Stage 1</td>
<td>140-159</td>
<td>and/or</td>
</tr>
<tr>
<td>Stage 2</td>
<td>≥ 160</td>
<td>and/or</td>
</tr>
<tr>
<td>Isolated systolic HT</td>
<td>≥ 140</td>
<td>and</td>
</tr>
</tbody>
</table>

- **Cuff** width should be >2/3* of arm circumference
- **Diastolic pressure:** disappearance of sounds (Korotkoff V*) - AIPGE 2008
- **Pseudohypertension:** Pseudohypertension is when blood pressure measurements are elevated but the blood pressure is actually normal. Seen in elderly d/t arteriolosclerosis (not atherosclerosis)

2. Goldblatt hypertension is seen in-
   1. Renovascular disease----------ans
   2. Drug induced
   3. Adrenal medulla tumor
   4. Children

**Discussion**-

Renovascular disease (Renal artery stenosis) - **Gold Blatt kidney**:
- Most frequently **atheromatous** (elderly, cigarette smokers with peripheral vascular disease) or **fibro muscular dysphasia*** in young ladies
- The kidney with RAS becomes **small and shrunken**. The decrease in bloodflow to the kidney with RAS (Goldblatt's kidney) causes hyperplasia of JG apparatus and increased renin production
which lead to retention of sodium and also produces HT but escapes the effects of HT d/t stenosis. The other kidney however shows microscopic changes of benign nephrosclerosis (hyaline arteriolosclerosis) d/t effects of HT.

- RAS, can occur in Takayasu disease but it does not occur in PAN

**Note:** Best screening test for RAS is Gadolium enhanced MRI angiography (N.Q) Previously it used to be HRCT. Most confirmatory test for RAS is conventional angiography (FAQ)

From copy-

Increased renin-

1. R. Renal Artery
2. R. Renal Vein
3. L. Renal Artery
4. L. Renal Vein

3. All are causes of hypertension with hypokalemia except-

1. Bilateral RAS
2. CRF
3. Conn syndrome
4. Crushing syndrome

**Discussion**-

HT also occurs in ARF/Ac. Glomerulonephritis
M/C cause of sec. HT --> Renal

**Q. BP in pheochromocytoma**-

1. Sustained HT
2. Sustained HT with post. hypo
3. Episodic HT
4. Episodic HT with post. hypo = ans (post. --> Postural Ck)

**Discussion**- (P=2269 H/17)

FNAC not done in pheochromocytoma because --> Highly vascular

Pheochromocytoma --> Sx --> BP decreases --> give NS

**Hypertension Management**:

- **BP = Cardiac output (CO) x Peripheral resistance (PR)**
- **CO = Heart rate (HR) x Stroke volume (SV)**
- **So, BP = HR x SV x PR**

A. **Drugs which reduce HR**- Beta blockers

B. **Drugs which reduce stroke volume**-

1. **Beta blockers:** beta blockers have negative inotropic & negative chronotropic effects (so beta blocker should be used with caution or should be avoided in CHF* with HT)
2. **Diuretics:** They reduce blood volume so they reduce the preload*
3. **Nitrate:** They primarily dilate the venules thereby cause peripheral pooling of blood. So they reduce the preload.

C. **Drugs which reduce the peripheral resistance**-

1. **Alpha blockers**
2. **Calcium channel blockers**
3. **ACE-I**
4. **Direct vasodilators** [Hydralazine, alpha-Methyl Dopa, Sodium Nitroprusside(A+V), indapamide]
5. Bosentan (New Q) --> Endothelial receptor blocking drug (Pri. Pul. HT, Raynaud's d/e)

New drugs for HT- Aliskiren, Fendolpam

*Never use sublingual nifedipine* to reduce BP (big drop in BP and increase stroke risk ) (N/Q)

Injection frusemide should not be used in severe HT But can be used in severe HT in LVF. (N.Q)

\[ BP = CO \times PR \]
1. Systolic
2. Diastolic
3. MAP-------------ans
4. PP

- End diastolic volume--> 120 ml
- End systolic volume--> 40 ml
- Stroke volume--> **80 ml**
- EF--> 2/3 (66%)
- NEF--> 65-72% (Ck)
- Single best test to calculate EF--> Echocardiography

Diuretic **not** used in which HT-
- Pheochromocytoma (already dehydrated)
- also Polycythemia(H/17 P-362)

**CHF**

4. Earliest sign of LVF-
   1. S3
   2. Basal crepitation
   3. Tachycardia-------------ans
   4. Raise JVP

**Discussion**-(H/17 P-1443)

LVF-
Ix of choice--> EF

1. Resting **tachycardia** (Earliest feature of LVF--> Tachycardia)
2. **Pulsus alternans**
3. Auscultation: **S3 gallop** (Best feature of LVF but heard normally in Children, Pregnancy)

**Signs of RVF: Raised JVP, Edema Ascites, Hepatomegaly**

15. **Pulsus alternans is seen in**-
   1. Aortic regurgitation
   2. Hypertrophic cardiomyopathy
   3. A-V block
   4. Severe LVF-------------ans

**DISCUSSIONS**-
- **Bisferiens pulse** (Two systolic peaks)- AR*, AR with AS*, HOCM* (Bisferiens pulse is best assessed in *branchial/radial artery*)
- **Dicrotic pulse** (Two waves: One is systole and one is diastole)- *Dilated cardiomyopathy*.
- **Pulsus alternans**(regular alteration of amplitude with regular rhythm)- Severe LVF*. (S-3 is found with Pulsus alternans)
- **Pulsus paradoxus** (decrease in systolic arterial pressure during inspiration)- Pericardial tamponade*, Severe COPD, Bronchial Asthma*, SVC obstruction

5. Which is **not a major Framingham’s criteria in CHF**-
   1. Cardiomegaly
   2. Paroxysmal nocturnal dyspnea
   3. S-3 gallop
   4. Hepatomegaly-------------ans

**Discussion**-
Framingham criteria for diagnosis of CHF:

**Major criteria**-
1. Paroxysmal nocturnal dyspnea
2. Raised JVP
3. Crepitations
4. Cardiomegaly
5. Acute pulmonary edema
6. S-3
7. Increased venous pressure (>16 cm H2O)
8. Positive hepatojugular reflux

Minor criteria-
1. Extremely edema
2. Orthopnea (night cough)
3. Dyspnea on excretion
4. Hepatomegaly
5. Pleural effusion
6. Vital capacity reduced by 1/3 from normal
7. Tachycardia (> 120/m) --> Earliest criteria even though a minor criteria

At least one major and two minor criteria are required*

6. Which of the following is not seen in CHF-
1. Increase serum sodium-------------------ans(AIPG 2007)
2. Increase catecolamines
3. Increase blood urea
4. Pedal edema

Discussion-
Compensatory mechanism-
1. Increase epinephrine: It leads to tachycardia
2. Increase nor-epinephrine: It leads to increase peripheral resistance, there-by blood supply to muscles and skin is reduced but blood supply to heart and brain is normal
3. Reduce renal blood flow: It lead to prerenal azotemia and hyperaldosteronism
4. Increase aldosterone: It leads to more Na and water retention but primary Na+ gain is exceeded by secondary water gain....(See MK Vol-1 P-290 for more)

- Percentage of blood flow to Kidney- 20%
- CHF: (Cold extremities--> Less blood to skeletal muscle and skin)
- CHF: decreased bodd flow to kidney--> hyperaldosteronism but dilutional hyponatremia
- Hyponatremia without edema: SIADH
- New drugs for CHF-
  - Epleremone--> Aldosterone receptor blocker
  - Nesiritide

7. Which of the following agents does not reduce mortality in patients with congestive heart failure-
1. Digitalis-----------------ans(KDT P=501)
2. Furosemide
3. Enalapril
4. Beta blocker

Discussion-
Basic principle of drug therapy in CHF(H/17 P-1449)-
1. Drugs to reduce preload = NO3, diuretic
2. Drug to increase pumping of myocardium = digoxin
3. Drug to reduce after load = ACE-I

(Extra Question- Controversial question)
Q. Which of the following agents is contraindicated in patients with CHF-
1. Digitalis
2. Furosemide
3. Enalapril
4. Beta blocker--------------------------ans(See explanation to Q-3)
**Cardiomyopathy** --> P-1481 H/17

8. Cardiomyopathy is seen in all **except**-
   1. Pompe’s disease
   2. Friedich’s ataxia
   3. Lowe syndrome------------------------ans (AIIMS Nov 2007)
   4. Duchenne Muscular dystrophy

**Discussion**-

**Dilated cardiomyopathy:** Pre-disposing conditions:
1. **Alcohol**
2. Drugs doxorubicin(causes irreversible dose dependent cardiomyopathy), Cyclophosphamide*, Cocaine(also vasoconstriction--> MI), Imatinib,trastuzumab
3. Autoimmune
4. Peri- or postpartum*
5. Selenium deficiency*
6. Duchenne myopathy*
7. Freidrich’s ataxia
8. Glycogen storage disease(Pompe’s disease)

9. **SAM is seen in**-
   1. HOCM---------------------ans
   2. Constrictive pericarditis
   3. DCM
   4. Restrictive CM

**Discussion**- SAM: Systolic Ant. Movement of Mitral valve

**Hypertrophic cardiomyopathy(HOCM):**
1. **Autosomal dominant** inheritance*
2. 70% have mutations in genes encoding beta-myosin, alpha-tropomyosin, and troponin-T
3. Family history of sudden death may be there

**Symptoms & signs:**
1. Sudden death(Also in AS,MI,MVP)
2. Jerky carotid pulsation*(FAQ)
3. ‘a’ wave in JVP
4. Double apex beat*(FAQ)
5. S4 heart sound
6. Harsh ejection systolic murmur
7. The intensity of murmur **increases** on standing and Valsalva*(most commonly asked question in world)---> also see Q-17

**Echo:** SAM--> Systolic anterior movement of mitral value*.

**Rx:** Septal myomotomy--> surgical or chemical with **alcohol**(absolute alcohol)

**Drugs contraindicated:** Digoxin(absolutely), Nitrates, Beta agonist, Diuretics

**Pericardial diseases**

11. **Pulsus paradoxus is seen in**-
    1. Cardiac tamponade------------------------ans
    2. Pulmonary oedema
    3. S-3
    4. CP

**Discussion**-

**Cardiac tamponade**-

**Signs:** pulsus paradoxus*, **Ewart sign** is positive*, S3 is absent*, Y descent is never prominent*
**Diagnosis:** Beck’s triad-
1. Falling BP
2. Rising JVP
3. Small, quiet heart

**ECG:** Electrical alternans*, low voltage ECG*
**Echo is diagnostic:** echo-free zone around the heart

10. **What is not seen in CP (Constrictive Pericarditis)-**
   1. Nephrotic syndrome
   2. Protein losing enteropathy
   3. S-3 -----------------------------ans
   4. Kussmaul sign

**Discussion:**
Constrictive pericarditis does not occur in rheumatic pericarditis(FAQ)

**Clinical Features:** These are mainly of right heart failure with raised JVP; **Kussmaul’s sign** is positive (JVP rising paradoxically with inspiration). **Prominent Y descent**, S-3 absent, **diastolic pericardial knock**, hepatosplenomegaly, ascites and oedema. The apical pulse is reduced and may retract in systole (**Broadbent’s sign**)

**NB:** Diastolic pericardial knock is also heard in early diastole. S-3 is also heard early diastole but Harrison 17/e Table 232-2, Page No. 1491 mentions that S-3 is absent in CP.

**Complications:**
1. Nephrotic syndrome*
2. Protein loosing enteropathy

---

**Q. A 35 yrs old male C/O substernal chest pain aggravated by inspiration and relieved by sitting up. He has a H/O TB. Lung fields are clear to auscultation, and heart sound are somewhat distant. CXR shows an enlarged cardiac silhouette. The next step in evaluation is-**

1. Right lateral decubitus film
2. Cardiac catheterisation
3. Echocardiogram----------ans
4. Serial ECGs

**Explanation:** GT-87 Q285; H/17 P-1489
The patient's pleuritic chest pain that is relieved by sitting up is most likely d/t pericarditis. A pericardial friction rub may initially be present, then disappear, with the heart sounds becoming fainter as an effusion develops. Lungsounds are typically clear. An enlarged cardiac silhouette without clear chest x-ray findings of heart failure suggests pericardial effusion. Echocardiography is the most sensitive, specific way of determining whether pericardial fluid is present. The effusion appears as an echo-free space between the moving epicardium and stationary pericardium. It is unnecessary to perform cardiac catheterisation for the purpose of evaluating pericardial effusion. Radionuclide scanning is not a preferred method for demonstrating pericardial fluid.

---

**RHD and Endocarditis**

16. **Carey comb murmur is seen in-**
   1. Severe pulmonary HT
   2. Ac. Rheumatic carditis---------ans
   3. Mitral stenosis
   4. Pure aortic regurgitation

17. **Which is not seen in mitral value prolapse-**
   1. Pansystolic murmur----------------ans
   2. Mid or late systolic click
   3. Cerebral embolism
   4. Ventricular premature contractions

**DISCUSSION:** Systolic but not pansystolic. Pansystolic murmur is seen in TR.
Mitral valve prolapse
- It is the commonest valvular lesion in the world. It is seen in *5-7% of the young girls*. Occurs alone or with: ASD, PDA, Cardiomyopathy, Turner’s syndrome, Marfan’s syndrome*, Osteogenesis imperfecta, Pseudoxanthoma elasticum, WPW syndrome

**Signs:**
- a. Non ejection Mid-Systolic click*
- b. A late systolic murmur*. *(Intensity of the murmur increases on standing and Valsalva*)

18. **Left ventricular hypertrophy is not seen in**-
   1. MS-------------------------ans
   2. AS
   3. Aortic incompetence
   4. Essential hypertension

**DISCUSSION- (FAQ)**
MS(H/17 P-1465)-
- **Presentation:** Dyspnoea* on exertion (main symptom); hemoptysis*, Hoarseness of voice (Auntner syndrome)
- **On auscultation:** Loud S-1*; Opening snap*(pliable valve*); rumbling mid-diastolic murmur*, Loud P2. Graham steel murur of PR and Carvello sign (murmur is loud during inspiration) also found.
- **Severity is indicated by**- Longer the diastolic murmur, the closer the opening snap to A-2
- **Echocardiography diagnostic**. Reduced EF slope is characteristic*

| LVH and S3 not found in MS. |

19. **Mitral regurgitation may occur in**-
   1. Mitral valve prolapse
   2. Rupture of papillary muscle
   3. Rupture of chordae tendineae
   4. All-----------------------------ans

**DISCUSSION-**
Mitral regurgitation(H/17 P-1469)-
**Causes:**
- a. Functional(LV dilatation)
- b. Annular calcification(elderly)
- c. Rheumatic fever
- d. Infective endocarditis
- e. Mitral valve prolapse
- f. Rupture chordae tendinea
- g. Papillary muscle dysfunction/rupture
- h. Connective tissue disorder/(Ehlers-Danlos,Marian’s)
- i. Congenital(may be associated with other defects.eg ASD,AV canal)

**Symptoms:** Easy fatigability* (commonest symptom)

20. **Dancing carotids is also known as**-
   1. Hill’s sign
   2. Quincke’s sign
   3. Corrigan’s sign------------------------ans
   4. Traube’s sign

**DISCUSSION-(AIIMS May 2005)**
**AR-Peripheral signs***-
- 1. Austin Flint murmur* may be heard in severe AR
- 2. Collapsing (water hammer) pulse*
- 3. Corrigan’s sign* (carotid pulsation)
- 4. de Musset’s sign* (head nodding)
- 5. Duroziez's sign* (**femoral** diastolic murmur as blood flows backwards in diastole)
6. Hill Sign - BP difference in upper and lower limb --> (UL<LL)
7. Quincke’s sign* (capillary pulsations in nail beds)
8. Traube’s sign* (piston sound over femoral arteries)
9. Wide pulse pressure*

21. Hepatomegaly with liver pulsations indicates-
   1. TR------------------------ans
   2. MR
   3. Pulmonary hypertension
   4. MS

DISCUSSION- (AIPG 2009)

Tricuspid regurgitation:

Causes-
   1. Functional (Commonest cause of TR) seen in cor-pulmonale*
   2. Pulmonary hypertension

Signs: Giant \textit{v} waves*, Prominent \textit{y} descent* in JVP, \textit{Pansystolic} murmur*, Carvallo’s sign, Pulsatile hepatomegaly*, jaundice, ascites

22. HACEK does not include-
   1. Haemophilus influenze----------------ans
   2. Actinobacillus
   3. Cardiobacterium
   4. Eikenella

DISCUSSION- (AIIMS May 2007)
   • HACEK
      ○ Haemophilus species (\textit{Haemophilus parainfluenzae}, \textit{Haemophilus aphrophilus}, \textit{Haemophilus paraphrophilus})
      ○ \textit{Actinobacillus actinomycetemcomitans}
      ○ \textit{Cardiobacterium hominis}
      ○ \textit{Eikenella corrodens}
      ○ Kingella

Commonest cause of native endocarditis--> S.aureus(H/17)

ECG

23. Prolong QT interval is seen in (MTR)-
   1. CRF---------------- ans
   2. Cirrhosis
   3. COPD
   4. None

DISCUSSION- (See Q-32)

Normal QT: 0.36-0.44 sec*

Prolonged QT interval*: Electrolyte imbalance (Hypokalemia*, Hypocalcemia*, Hypomagnesemia*), Class 1A anti arrhythmic drugs (\textit{quinidine*}) bradycardia, head injury, hypothermia, \textit{sotalol*}, antihistamines, \textit{macrolides*}(eg \textit{erythromycin}), \textit{amiodarone*}, \textit{Phenothiazine*}, \textit{Tricyclic*}, \textit{Torse De Pointes}(caused d/t hypokalemia)

Short QT interval: Hyperkalemia, Hypermagnesemia, Class 1B anti arrhythmic drugs, Digoxin, Acute MI

24. A 73 year old patient develops acute renal failure secondary to bladder obstruction. His serum creatinine is 8 mg% with potassium of 6.5 meq/L. The patient had an electrocardiogram, which revealed peaked T waves as the only abnormality. If the hyperkalemia were not corrected, what would be the expected next electrocardiographic abnormality-
   1. T-wave inversion
   2. PR prolongation and P-wave flattening--------ans
   3. Prolongation of the QRS interval
   4. Widening of the QRS interval
DISCUSSION-(AIPG 2008) FAQ

Hyperkaliemia: Tall, tented T wave*, Prolong PR, P-Wave disappear, Wide QRS (*sine wave* appearance)

Hypokaliemia: T wave become smaller and then disappear, prominent U waves*, prolong PR, ST segment sagging.

25. ECG changes in Mobitz type I is-
   1. Progressive PR interval prolongation with a P wave followed by dropped QRS-------ans
   2. QRS wide with ST and T directed opposite to QRS
   3. PR interval is >0.62 sec
   4. R and T phenomenon

DISCUSSION-(Confusing question)
Progressive PR interval prolongation with a P wave followed by dropped QRS

26. First ECG change in acute MI is-
   1. ST segment elevation
   2. Loss of R wave
   3. Inverted T wave
   4. Tall peaked T wave-------------------ans

DISCUSSION- (Confusing question) PGI Dec 2006

MI:
   • Within minutes: the T wave may become peaked* (Earliest features)*
   • With 2-3 hrs: ST segments may begin to rise(Pardees sign)*
   • Within 8-12 hrs: the T wave inversion*
   • Within 24-48hrs: pathological Q waves* begin to form. Q waves usually persist in old MI*

Tall T is the earliest manifestation of acute MI.(FAQ)

CAD

27. A 40 year old man, smoker, complains of epigastric pain since an hour. On electrocardiographic examination he is found to have ST elevations suggesting an inferior wall infraction. Next step in the management would be-
   1. Aspirin---------------------------ans
   2. Thrombolytic therapy
   3. Pantoprazole
   4. Beta-blockers

DISCUSSION- (Confusing question) AIIMS Nov 2007

• Best method to diagnose angina--> History
• Add beta-blocker in all cases of angina if no C/I exists
• See next question

28. Initial treatment of choice for a patient of anterior wall MI with cardiogenic shock is-
   1. PTCA--------------------------------------------ans
   2. Intra aortic balloonpumping(IABP)
   3. Streptokinase
   4. Dopamine drip

DISCUSSION- AIPG 2008 (Confusing question)--->H/17 P-1514

Basic concept of management of MI (Concept of Reperfusion therapy and concept of myocardium salvage)

Management of acute coronary syndrome (ACS)-
   1. Aspirin
   2. Thrombolysis (if no contraindication) or primary angioplasty
   3. Beta-blocker
   4. ACE-inhibitor: Role of ACEI in acute MI: To control BP, To control LVEF, To help in the remodelling of infarct tissue

Rolazapine-It is new angina drug useful for refectory angina (New Drug)
Extra Q (AIIMS May 2008). Drug used to perform stress ECHO-

a. Thallium
b. Dobutamine-----------------ans
c. Dopamine
d. Adenosine

29. Most important prognostic factor in an infarct case is-
   1. Ejection fraction-----------------ans
   2. CPK
   3. Ventricular tachycardia
   4. Left main coronary artery obstruction

Discussion - Killip's Classification for assessing the prognosis of a case of MI. It is based on degree of LV dysfuction-

<table>
<thead>
<tr>
<th>Feature</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I- No signs of pulmonary or venous congestion</td>
<td>5-10%</td>
</tr>
<tr>
<td>Class II- Crepts at lung bases, S3, tachypnea</td>
<td>0-25%</td>
</tr>
<tr>
<td>Class III- Severe heart failure, Pulmonary edema, systolic BP &gt; 90</td>
<td>50-60%</td>
</tr>
<tr>
<td>Class IV- Cardiogenic shock, systolic BP &lt; 90 mental confusion, cyanosis</td>
<td>80-90%</td>
</tr>
</tbody>
</table>

30. Which drug is used to prevent restenosis in stent-
   1. Sirolimus-----------------ans
   2. Cyclosporin
   3. Prednisolone
   4. Erythromycin

Discussion - (New Question)

Percutaneous transluminal coronary angioplasty (PTCA) involves balloon dilatation of the stenotic vessel(s). Stenting reduces restenosis rates. Drug-coated stents reduce restenosis. Drug use is rapamycin*, sirolimus, paclitaxel(MCQ)

Predisposing factors for ventricular free-wall rupture after MI include advanced age as well as the first MI, probably due to lack of coronary collaterals. Most commonly seen b/n 1-4 days after MI. Lateral and anterior walls are most often involved and it is typically seen with large MI involving >20% of the ventricle. Rupture typically occurs at the junction of infarct with the normal tissue. Patients are diagnosed by ECG and treatment involves operative treatment if possible.

Arrhythmias

31. A 48 year man is admitted to CCU with an acute inferior MI. Two hours after admission, his BP is 86/52 mgHg; his pulse is 40/mt with sinus rhythm. Which of the following would be the most appropriate initial therapy-
   1. Immediate insertion of a temporary transvenous pacemaker
   2. I/V administration of atropine sulfate, 0.6 mg-----------------ans
   3. Administration of normal saline, 300ml over 15 min
   4. I/V administration of dobutamine, 0.35 mg/min

Discussion - (AIIMS Nov 2008)

Atropine--> unsuccessful --> Saline

32. Torse-de-pointes is caused by-
   1. Hypermagnesemia
   2. Hypokalemia-----------------ans
   3. Hyperkalemia
   4. Hypercalcaemia

Discussion -
• Hypokalemia (commonest cause)
• Prolonged QTc interval*
• ECG oscillates along baseline→

Causes- Common causes for torsades de pointes include diarrhea, hypomagnesemia and hypokalemia. Drug interactions such as erythromycin or moxifloxacin, taken concomitantly with inhibitors like nitroimidazole, dietary supplements, and various medications like methadone, lithium, tricyclic antidepressants or phenothiazines may also contribute.

Treatment- Magnesium sulfate

Cardiac Neoplasm

33. True statements regarding cardiac neoplasms include-
1. Lymphoma is the most common malignant neoplasm that primarily involves the heart
2. The most common site for a myoma is the right atrium
3. Myxomas may arise as part of a familial syndrome that also includes pigmented skin lesions and endocrine abnormalities----------------ans(
4. A midsystolic “plop” typically indicates the presence of a cardiac myxoma

**DISCUSSION:** (H/17 P-1495)- Cardiac myxoma is a rare benign* cardiac tumour in *left atrium usually sporadic*, may be familial* (autosomal-dominant)*. It may mimic infection endocarditis (fever, weight loss, clubbing, raised ESR), or mitral stenosis* (left atrial obstruction, systemic emboli, Af). A ‘tumour plop’ may be heard, and signs may changed according to posture. Histologically they are composed of stellate cells in a loose myxoid background.

In contrast Rhabdomyomas are the most common primary cardiac tumors in *infants and children* and often occur in a/w tuberous sclerosis. Histologically the so-called *spider cells* may be seen. Papillary fibroelastosis usually are incidental lesions found at the time of autopsy and are probably hamartomas rather than true neoplasms.

<table>
<thead>
<tr>
<th><strong>Carney complex</strong> comprises-</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. myxomas (cardiac*, skin, and/or breast),</td>
</tr>
<tr>
<td>2. lentigines and/or *pigmented nevi, and</td>
</tr>
<tr>
<td>3. endocrine overactivity (primary nodular adrenal cortical disease with or without Cushing’s syndrome, testicular tumors, and/or pituitary adenomas with gigantism or acromegaly).</td>
</tr>
</tbody>
</table>

Certain constellations of findings have been referred to as the NAME syndrome (nevi, atrial myxoma, myxoid neurofibroma, and ephelides) or the LAMB syndrome (lentigines, atrial myxoma, and blue nevi), although these likely represent subsets of the Carney complex.

Tests: Enchocardiography
Treatment: Excision

Rheumatology

34. TNF- alpha is involved in the pathogenesis of which of the following disorder-
1. RA-------------------ans
2. SLE
3. Psoriasis
4. None

**DISCUSSION**-(Maheshwari P-244) TNF-alpha is involved in the pathogenesis of RA

**Diagnostic criteria of RA- Four of seven** criteria are required*
1. *Morning stiffness*- lasting 1 hour before maximal improvement
2. *Arthritis of 3 or more joint areas*-14 possible joint areas are right or left PIP, MCP, Wrist, Elbow, Knee, Ankle, and MTP joints
3. Arthritis of hand joints
4. Symmetrical arthritis
5. Rheumatoid nodules*
6. Positive Serum rheumatoid factor*
7. Radiographic changes- **erosions**(Hallmark) or unequivocal bone decalcification localized in or most marked adjacent to the involved joint.

**Backache never a symptom of RA.**

35. False positive Rheumatoid factor can be associated with all except-
   1. Inflammatory bowel disease-----------ans(See Q-37)
   2. HbsAg
   3. VDRL
   4. Coombs test--> diagnose SLE

**DISCUSSION**-
Laboratory findings in RA- RA factor(IgM) is positive in 75% patients of RA though it is found in 5% of healthy persons. **Anti CCP antibody** are also seen.

- The presence of Rheumatoid factor in RA correlates with extraarticular manifestation of the disease

**Other conditions in which RA factor is positive are**- SLE, Sjogren’s syndrome, Interstitial pulm fibrosis, Hepatitis B, Leprosy, Subac. bacterial endocarditis, Chronic liver disease, Sarcoidosis, IMN, Tuberculosis, **Syphilis**, Visceral leishmaniasis, Malaria

**Treatment of RA(KDT P-202)**-
   1. NSAIDS- To control the symptoms and signs of local inflammatory process. These drugs have minimal effect on the progression of the disease.
   2. Disease modifying antirheumatic drugs- These drugs alter the course of RA. These drugs have minimal effect on the inflammation. So are not useful as analgesics. These drugs should be used early* in the course of the disease as they slow the progression of disease. The most commonly used DMARD is Methotrexate.
      1. **Immunosuppressants**- The immunosuppressive drugs used are Methotrexate, Azathioprine, *leflunomide*, cyclosporine and cyclophosphamide
      2. Other drugs- d-penicillamine, and sulfasalazine, Gold , Hydroxychloroquine
   3. Biological response modifiers-
      1. **TNF alpha neutralizing agents**-
         1. **Monoclonal antibody** to TNF alpha (Infliximab)
         2. TNF-alpha type –II receptor antagonist (Etanercept)(New Drug)
         3. **Adalimumab** (Human antibody to TNF)(New Drug)
      2. **IL-1 Receptor antagonist** = ANAKINRA Q (New Drug)
   4. **Adjuvent drugs**- Corticosteroids
   5. **Gene therapy**- Genes which intercept the pathway of inflammatory cascade reaction are used
   6. **Surgery**- Synovectomy, tenosynovectomy, arthroplasty and total joint replacements

36. Rituximab (Anti-CD 20 antibody) is used in all except-
   1. SLE
   2. RA
   3. PNH-----------ans
   4. NHL

**DISCUSSION**- (AIIMS Nov 2008) **Rituximab(anti CD20 antibody)**--> CLL also

37. Seronegative arthritis include-
   1. AS
   2. Reiter’s arthritis
   3. Psoriatic arthritis
   4. all------------------------ans

**DISCUSSION**-
Spondylo-Arthrosis:

It is a group of disease which include-
1. Ankylosing spondylitis
2. Reiter’s disease
3. Reactive Arthritis(Yersinia, Salmonella, Gonococcus)
4. Psoriatics arthritis
5. Juvenile rheumatoid arthritis(JRA) [not adult rheumatoid arthritis which is DW4/DR4]

Features:
1. Seronegativity
2. HLA B27 associated
3. Axial arthritis: Pathology in spine (Spondylo-) and sacroilias (SI) joints (Backache is the presenting symptom)
4. Asymmetrical large-joint oligoarthritis (i.e. <5 joints) or monoarthritis
5. Enthesitis: Inflammation of the site of insertion of tendon or ligament into bone
6. Extra-articular manifestations eg. anterior uveitis, aortic regurgitation, Crohn’s or UC

REITER’S DISEASE (Imp): It is characterised by a triad of-
1. Seronegative oligoarthritis*
2. Conjunctivitis
3. Nonspecific urethritis, 1-3 weeks following bacterial dysentery (epidemic form) or exposure to sexually transmitted disease

Arthritis occurring alone following sexual exposure or enteric infection is known as reactive arthritis. Reiter’s disease can occur in epidemic* form (FAQ) AIIMS Nov 2008 Exam

Etiology- Enteropathogenic Bacteria in Reactive Arthritis Salmonella, Shigella, Campylobacter, Yersinia Chlamydia

Clinical Features-
1. It presents with monoarthritis of a knee or an asymmetrical inflammatory arthritis of interphalangeal joints
2. Patient can have heel pain, Achilles tendinitis or plantar fasciitis with presence of circinate balanitis. (Presence of rash of keratoderma blennorrhagia (AIIMS Nov 2008) is diagnostic of Reiter’s disease in the absence of classical triad.) --> Ck printed matter
3. Ocular involvement (mild bilateral conjunctivitis)

Extra question-
Q. Circinate balanitis is seen in (AIPG 2007)-
1. RA
2. SLE
3. Reiter’s disease--------ans
4. Gout

Q. What is not seen in Reiter’s syndrome (AIIMS Nov 2008)
1. Subcutaneous nodules-----------------ans
2. Keratoderma blennorrhagicum
3. Circinate balanitis
4. Oral ulcers

38. Schirmer test is positive in-
1. Sjogren’s syndrome----------------ans
2. Behcer’s syndrome
3. SLE
4. Whipple’s disease

Discussion-

SJOGREN’S SYNDROME-

Definition- Chronic, slowly progressive autoimmune disease characterized by lymphocytic infiltration of
the exocrine glands resulting in xerostomia and dry eyes.

Causes of secondary Sjogren's syndrome-
1. Rheumatoid Arthritis (most common cause)
2. SLE
3. Scleroderma
4. Mixed connective tissue disease
5. Primary biliary cirrhosis
6. Vasculitis
7. Chronic active hepatitis
8. Polymyositis
9. Hashimoto's thyroiditis
10. Interstitial pulmonary fibrosis

Antibodies to Ro/SS-A and La/SS-B antigens; Antibodies to alpha-fodrin --- a salivary gland specific protein

NHL common in Sjogren ans also in HIV.......

Clinical Manifestations (Triad)-
1. Dry eye
2. Dry mouth
3. B/L Parotid enlargement

39. Sudden renal failure occurs in-
1. SLE
2. PSS------------------------ans
3. RA
4. AS

DISCUSSION-

SYSTEMIC SCLEROSIS-

Definition- This is a generalised disorder of connective tissue characterised by fibrosis and degenerative changes in the skin (scleroderma) and many internal organs

Diagnosis Antibodies seen are-
1. Antinuclear antibodies (ANA)
2. Antibodies to single stranded RNA
3. Anti-Scl-70

Part of Crest Syndrome (Anticentromere Ab)

40. Which radiological feature is most important to help differentiate rheumatoid arthritis with SLE-
1. Erosion------------------------ans
2. Juxta articular osteoporosis
3. Subluxation of MCP joint
4. Swelling of PIP joint

DISCUSSION-

Laboratory findings in a cause of SLE Antibodies. The antibodies seen in SLE are-
1. Antinuclear antibodies (ANA)- Most sensitive test
2. Anti ds DNA- most specific* for SLE. High titres are associated with nephritis and disease activity.
3. Anti Sm – Also specific for SLE
4. Anti-Ro (SS-A) – Associated with subacute cutaneous lupus and ANA negative lupus
5. Anti-La (SS-B) – Risk for nephritis is low if present
6. Anti-histone- More frequent in drug induced LE.
7. U1RNP ab- Seen in mix connective tissue disorder. Which include SLE, PSS and polymyositis

41. Most specific diagnostic finding in gout is-
1. Raised serum uric acid
2. Uric acid crystals in urine
3. Presence of monosodium urate crystals in synovial fluid under polarized microscopy
4. Presence of calcium pyrophosphate crystals in synovial fluid under polarized microscopy

**DISCUSSION**

**Gout**
1. Males > females. Great toe, Grade 4 tenderness
2. Presence of monosodium urate crystals in synovial fluid under polarized microscopy (Serum uric acid level may be normal)
3. For acute pain – colchicine, indomethacin (But aspirin is contraindicated)
4. For prophylaxis- Allopurinol
5. Uricosuric drugs- Probenecid, Sulphinpyrazone

42. C-ANCA Ab are seen in-
   1. Wegener's granulomatosis
   2. PAN
   3. Churg-Strauss
   4. SLE

**DISCUSSION** (AIIMS May 2007)
- C-ANCA antibodies are seen in Wegener’s granulomatosis these are directed against proteinase-3
- P-ANCA antibodies are seen in Microscopic polyangitis, Churg-Strauss syndrome, Crescentic glomerulonephritis, Goodpasture’s Syndrome

43. RAS is seen in all except-
   1. PAN
   2. Takayasu
   3. Atherosclerosis
   4. Fibromuscular dysplasia

44. True about giant cell arteritis are all except-
   1. High dose steroid is drug of choice
   2. ESR is usually elevated
   3. Internal carotid artery is particularly susceptible
   4. Mainly affect people below age of 40 years

**DISCUSSION**

Temporal arthritis (Polymyalgia rheumatica frequently occur with Temporal arteritis)-
- Mainly effect people above age of 60 years
- Headache
- Internal carotid artery is particularly susceptible
- Visual disturbance can occur (affects retinal A.--> branch of Int. Carotid)
- ESR is usually elevated
- High dose steroid is drug of choice

45. All of the following are true about Kawasaki disease except-
   1. Rash
   2. Lymphadenopathy
   3. Extremities edema
   4. Purulent conjunctival congestion

**DISCUSSION** (AIPG) H/17 P-2130
1. Rash
2. Lymphadenopathy (does not respond to antibiotics)
3. Extremities edema
4. Conjunctival congestion it is non-purulent
Rx: IVIg

46. Anti Jo-1 antibody & Anti synthetase antibody are seen in-
1. Systemic lupus erythematosus
2. Systemic sclerosis
3. Dermatopolymyositis------------------ans
4. Dermatitis herpetiformis

**DISCUSSION**
In polymyositis eye muscle are not involved and biopsy is confirmatory.

47. Malignant RA occur if there is severe involvement of-
   1. Eye
   2. Skin------------------ans
   3. Nerve
   4. Joint

**Discussion** - It is a very simple and easy question!!!!

**NEUROLOGY**

Refresh your knowledge of neuroanatomy to understand Neurology much better....

**Cerebral Hemisphere**

48. A 30 year, male c/o headache for last 1 year & gradually progressive paraparesis for last 1 month. Most likely cause-
   1. ACA infarct
   2. Meningioma----------------ans
   3. TB spine
   4. MCA infarct

*(Extra Question). MCA infarct leads to contralateral-
   1. Leg paralysis
   2. Arm paralysis
   3. Both------------------ans
   4. None

49. Which statement regarding Gerstmann’s syndrome* is true-
   1. Affected patients have difficulty distinguishing right from left----------------ans
   2. It results from a lesion of the nondominant parietal lobe
   3. Motor deficit is a prominent feature
   4. Apraxia is common

**Discussion** - (DPG 2006)

**Parietal lobe**

**A. Dominant parietal lobe**

**Disease:** Gartsman syndrome*

**Features:**
1. Acalculia
2. Agraphia
3. Finger anomia(can not name finger)
4. Difficulty in **right and left** differentiation

**Extra points:**
- **Frontal lobe Disease:** Foster Kennedy syndrome*, Ipsilateral optic atrophy and contralateral papilledema. It results from the simultaneous presence of raised intracranial pressure and optic nerve compression secondary to tumor – classically due to a meningioma of the olfactory groove or more commonly due to a meningioma of the sphenoid wing.
- **Occipital lobe disease:** Cortical blindness*
• **Temporal* lobe disease**: Kluver Bucy syndrome*(Bilateral temporal lobe involvement)

50. If Wernicke’s area is damaged in the dominant hemisphere, it will result in-
   1. Irrelevant and rapid speech------------------------------------ans
   2. Senseless speech and difficulty in outflow of words
   3. Speech with difficulty in outflow of words
   4. Speech with difficulty in naming objects

**Discussion**

**Speech**

**Anatomy & Physiology**

- **Broca’s area** = Lies in **dominant inferior** frontal gyrus*. It is motor speech area(Speech outflow is disturbed in injury)
- **Wernicke’s area** = Lies in dominant temporal lobe*. It is sensory speech area
- **Arcuate fibres** = They **connect** the two speech areas

**Physiology of speech**: Spoken words --> Go to ears --> Go to auditory area in temporal lobe via 8\(^{\text{th}}\) nerve--> Go to Wernike’s area where speech is **understood**--> Message go to Broca’s area via Arcuate fiber--> Broca’s area is a motor speech which gives command to vocal cord and lip area in the motor cortex--> speech is spoken

**Diseases**:

- Broca’s area involvement--> Speech out flow and fluency is lost
- Wernicke’s area involvement--> Speech understanding is lost. Patient speaks senselessly
- **Nominal aphasia**(Anomic aphasia) is seen in **metabolic encephalopathy**. It is an early feature of **Alzheimer’s disease***

**Urinary bladder**

![Diagram of Nerve supply to the bladder and sphincters.](image-url)
Anatomy

- Nerve supply-
  - Sympathetic: T11-L2 (Hypogastric N) --> Supply Trigone
  - Parasympathetic: S2, S3, S4 (Pelvic N)* --> Supply Detrusor
  - Somatic: S2, S3, S4 (Pudendal nerve)* --> Supply Ext. Sphincter

Physiology: Afferent by all three nerves i.e. Pelvic, Hypogastric, Pudendal nerve. Efferent mainly by pelvic nerve. Detrusor muscle is unique---> does not follow starling’s law. Area of social behaviour and personality lies in frontal lobe.

Types of urinary bladder disturbance:
1. Automatic/Hypertonic bladder/Complete spastic*: Lesion in the spiral segment above S2,S3,S4. Small capacity bladder: Frequent voiding at interval (Bladder start behaving like a newborn bladder)
2. Autonomic/Hypotonic bladder- *Lesion at S2, S3, S4, Large capacity bladder, Distention with overflow
3. Cortical/Uninhibited Bladder- Frontal -Inappropriate micturition, loss of social inhibition control

52. Painless burn in hand is a characteristic feature of-
1. Syringomyelia------------------ans
2. Thalamic syndrome
3. Cord compression
4. SLE

Discussion- (H/17 P-2594; 2589 Figure 372-1)

53. Which is not a feature of extramedullary tumor-
1. Early corticospinal signs and descending paralysis
2. Root pain or Midline Back-Pain
3. CSF Abnormal
4. Sacral sparing------------------ans

Discussion- (AIPG 2008)

Comparison of intramedullary and extramedullary comparison-

<table>
<thead>
<tr>
<th>Symptoms</th>
<th>Intramedullary</th>
<th>Extramedullary*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Root pains</td>
<td>Uncommon</td>
<td>Common</td>
</tr>
<tr>
<td>2. Vertebral pain</td>
<td>Uncommon</td>
<td>Common</td>
</tr>
<tr>
<td>3. Motor Weakness</td>
<td>Late</td>
<td>Early</td>
</tr>
<tr>
<td>4. Sacral Involvement</td>
<td>Late</td>
<td>Early---&gt; Commonly involved</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Signs</th>
<th>Intramedullary</th>
<th>Extramedullary*</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Brown Sequard</td>
<td>Uncommon</td>
<td>Common</td>
</tr>
<tr>
<td>2. Dissociative sensory loss</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td>3. Bladder/bowel dysfunction</td>
<td>Early</td>
<td>Late</td>
</tr>
</tbody>
</table>

Investigation
• **Froin syndrome** - alteration in the cerebrospinal fluid due to block, which is yellowish and coagulates spontaneously in a few seconds after withdrawal, owing to its greatly increased protein (albumin and globulin) content; noted in loculated portions of the subarachnoid space isolated from spinal fluid circulation by an inflammatory or neoplastic obstruction. Syn: loculation syndrome.

54. Which findings are found in Brown Sequard syndrome-

<table>
<thead>
<tr>
<th>Ipsilateral loss</th>
<th>Contralateral loss</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Pain and temperature</td>
<td>Vibration and motor</td>
</tr>
<tr>
<td>2. Vibration and motor</td>
<td>Pain and temperature</td>
</tr>
<tr>
<td>3. Vibration and Pain</td>
<td>Motor &amp; temperature</td>
</tr>
<tr>
<td>4. Pain &amp; vibration</td>
<td>Temp. and motor</td>
</tr>
</tbody>
</table>

**Answer** - 2

• **Beevor’s sign** - Lesion at T9-T10* paralyse the lower but not the upper abdominal muscles, resulting in upward movement of umbilicus when abdominal wall contracts (beevor’s sign)

Q. An 80 yrs old male with mid alzheimer’s disease has been started on donepezil 5 mg after he continued to have difficulty in financial matters and keeping track of the day of the week and time. After 3 months the family feels that there has been no improvement. There are no complaints of nausea, dizziness or hypotension. The patients wife feels the medicaton is unnecessary. The best advice is-

1. Discontinue the donepezil
2. Increase the donepezil dose to 10 mg
3. Continue donepezil to prevent further plaque formation
4. Continue donepezil for 3 to 6 months and reevaluate mental status

**Explanation** - GT-87 Q-174
The best course would be to continue the donepezil and see if it slows progression of cognitive function loss based on MMSE or family assessment. The success of the intervention needs to be evaluated over a longer time period realising that success may mean maintaining baseline function. The anticholinesterase inhibitors do not prevent plaque formation. Increasing dose is rarely helpful and often causes side effects. There is no data to suggest that one cholinesterase inhibitor works better than another.

**Cranial nerves & Brain stem**

56. All are true about Benedict’s syndrome except-

1. Contralateral choreoathetosis
2. Dense hemiplegia------------------ans
3. Lesion is in mid brain
4. Ipsilateral 3rd nerve palsy

**Discussion** - (AIPG 2007) H/17 P-2525; MK-I P-302

**Site**--> **Midbrain**: PCA Occlusion-

<table>
<thead>
<tr>
<th>Weber’s synd</th>
<th>C/L hemiparesis + ipsilateral 3rd nerve palsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nothnagel syndrome</td>
<td>C/L cerebellar signs + ipsilateral 3rd nerve palsy</td>
</tr>
<tr>
<td>Benedikt's syndrome</td>
<td>C/L cerebellar signs + ipsilateral 3rd nerve palsy</td>
</tr>
</tbody>
</table>

**Claude syndrome = Benedikt's + Nothnagel syndrome**

**NB** - Nothnagel syndrome--> Same presentation as Benedikt’s
Bilateral infarction in distal PCA produces cortical blindness (blindness with preserved pupillary light reaction. The patient is often unaware of the blindness or may even deny it (Anton’s Syndrome)

57. **Millard Gubler Syndrome** consists of the following EXCEPT-
   1. Ipsilateral 5th Cranial Nerve Palsy
   2. Ipsilateral 6th Cranial nerve Palsy
   3. Ipsilateral 7th Cranial Nerve Palsy
   4. Contralateral hemiplegia

**Discussion**-

- **Site:** Pons
  - Foville's syndrome (dorsal pontine injury) = lat. gaze palsy, ipsilateral VI, VII, palsy, C/L hemiparesis
  - **Millard-Gubler synd** (ventral pontine injury) = ipsilateral VI, VII palsy C/L hemiparesis

58. A 30 year old male patient presented with h/o dizziness, vertigo, dysphagia, along with Horner syndrome on the same side. Loss of pain and temperature sensations on the left side was noted. The artery most likely to be involved in the condition described above is-
   1. Anterior inferior cerebella
   2. Posterior inferior cerebella
   3. Middle cerebral
   4. Superior cerebellar

**Discussion**- (AIIMS May 2006)-- Wallenberg's Syndrome--> PICA involved--> Classic findings

59. A 45 year old man presents with a daily headache. He has two attacks per day over the past 3 weeks, associated with tearing and reddening of his right eye as well as nasal stiffness. The neurologic examination is non focal. The most likely diagnosis is-
   1. Maigraine headache
   2. Cluster headache
   3. Tension headache
   4. Brain headache

**Discussion**-
Precipitated by alcohol
Tx: **Oxy**gen
Prophylaxis: Li, Valproate, Amitriptaline (Propranolol not effective)

**CVA** (H/17- 2513)

60. Most common cause of subarachnoid haemorrhage is-
   1. Hypertension
   2. A.V. Malformation
   3. Berry aneurysm
   4. None

**Discussion**- H/17 P-1726: **Trauma** is the m/c cause. Among options--> 3

61. The site of cerebral hemorrhage in hypertension are all except-
   1. Pons
   2. Internal capsule
   3. Putamen
   4. Thalamus

**Discussion**- Site of hypertensive bleed are- Cerebellum, Pons, Putamen, Thalamus

**UMN & LMN Lesions**

62. UMN lesion is characterized by-
   1. Weakness & spasticity
   2. Fasciculations
   3. Rigidity
   4. Localized muscle atrophy
Diseases causing dementia

63. Alzheimer’s disease typically affects-
   1. Nucleus basalis of Meynert-----------------ans
   2. Amygdala
   3. Parietal cortex
   4. Basal ganglion

Discussion- (AIIMS Nov 07) Controversial Question

Alzheimer’s Disease- (P-406 ROAMS)

- Pathology: At autopsy, the most severe pathology is usually found in the hippocampus, temporal cortex, and nucleus basalis of Meynert.(lateral septum)
- Microscopically- neuritic senile plaques and cytoplasmic neurofibrillary tangles. The neuritic plaque contain A-beta amyloid, proteoglycans, Apo E, Alpha-1 antichymotrypsin
- Macroscopically- Diffuse atrophy of cerebral cortex with secondary enlargement of ventricular system
- Biochemically- Decrease in cerebral cortical levels of Acetylcholine, choline acetyltransferase and nicotinic cholinergic receptors. Reduction in norepinephrine levels in brainstem nuclei.

- Treatment:
  - Tacrine
  - Donepezil
  - Newer drugs:
    - Rivastigmine
    - Galantamine
    - Memantine

- Drugs which reduce progression of AD-
  - Estrogen replacement therapy*
  - Ginkgo
  - NSAID*
  - Selegiline
  - Vitamine E

64. Triad of normal–pressure hydrocephalus includes-
   1. Tremor, aphasia dementia
   2. Ataxia, aphasia, gait disorder
   3. Gait disorder, urinary incontinence, dementia---------------------ans
   4. Gait disorder, urinary incontinence, lower cranial nerve palsy

Discussion- (H/17 -2546)

NORMAL PRESSURE HYDROCEPHALUS-

- It is a triad of abnormal gait (Ataxic gait), dementia and urinary incontinence
- A type of communicating hydrocephalous with patent aqueduct of sylvius
- NPH is caused by obstruction to normal flow of CSF over the cerebral convexity and delayed absorption into the venous system
- H/O conditions producing scarring of basilar meninges such as meningitis, SAH and head trauma
- Enlarged lateral ventricles (Hydrocephalus) with little or no cortical atrophy.
- CSF- Pressure is in high normal range. TLC, DLC, Protein, Sugar are normal
- Treatment- Ventricular peritoneal shunt

Extra question(AIPG 2007)- Which of the following disease doesn’t have autosomal dominant mode of inheritance-
   1. Marfan’s syndrome
   2. Polycystic kidney disease
   3. Fabry’s disease------------------ans
4. Huntington’s disease

**Discussion:** Apha-galactosidase A deficiency. X-linked recessive lysosomal storage disease
**Rx:** Enzyme replacement therapy

Ghent's criteria is used for the diagnosis of Marfan's syndrome

---

**Parkinsonism** *(H/17 P-2549)*

65. True about Rotigotine-
   1. It is a non ergot alkanoid-----------------ans
   2. It is a MAO – B inhibitor
   3. It is an anti-oxidant
   4. none

**Discussion:**

**Newer drugs**-
   - **Ergot alkaloid:** Bromocriptine, Cabergoline, Pergolide
   - **Non-ergot alkaloid:** Pramipaxol, Ropinirol, Rotigotine

**Mx of Tremors:** Antimuscarinics--> Procyclidine, Benztropine, Benzhexol

Read whole drug clssification from P-415 KDT

**Neuroprotective therapy**-
   1. Chronic use of NSAIDS or the use of estrogen replacement in postmenopausal women may delay or prevent the onset for PD(also Alzheimer’s)
   2. **Selegiline** monotherapy delayed the need for levodopa therapy
   3. **Coenzyme Q10,** an antioxidant and a cofactor of complex I of the mitochondrial oxidative chain, has been shown to have neuroprotective effects

**Demyelinating disorders**

66. The most common presenting finding of **multiple sclerosis** is-
   1. Internuclear ophthalmoplegia
   2. Transverse myelitis
   3. Cerebellar ataxia
   4. Optic neuritis------------------ans

**Discussion:** H/17 P-2611

- **Exercise induced weakness** is a characteristic symptom of MS
- **Heat sensitivity**-
  - Appearance of new symptoms or the worsening of pre-existing symptoms on exposure to heat
  - Conduction block which may occur in response to increase temperature or metabolic derangements
- **Lhermitte's symptom**- Generation of *ectopic impulse* giving rise to Lhermitte’s Sympt, paroxysmal symptoms or paresthesias

**Investigations**-
   1. **MRI--> Dawson’s finger sign:** Lesion appear to extent from the ventricular surface, corresponding to a pattern of perivenous demyelination
   2. **Evoked responses**- To detect slowed or absent conduction in visual, auditory, somatosensory or motor pathways
   3. **CSF**-
      - **Mononuclear pleocytosis,** TLC usually <20
      - **Increase IgG**
• Oligoclonal banding*

**Treatment of Acute Attack**

1. Glucocorticoid treatment
2. Plasma exchange
3. **Methotrexate**
4. IV Ig
5. Pulse cyclophosphamide, Azathioprine
6. Pulse Methyl Prednisolone

**New Drugs for prophylaxis:**

• Glatiramer acetate
• Mitoxantrone
• IFN-beta-1a/1b
• Natalizumab

**Epilepsy & Syncope**

**67. Which of the following would help exclude the diagnosis of seizure in a patient with sudden loss of consciousness?**

1. A brief period of tonic – clonic movements at the time of falling
2. An aura of a strange odor before falling
3. Sudden return to normal mental function upon awakening, though with a feeling of physical weakness
4. Urinary incontinence

**Discussion** (AIPG 06) Pt. is usually confused following seizure in **complex** partial seizure

**EPILEPSY** (H-17 P-2498)-

Seizure- Paroxysmal event due to abnormal, excessive and hypersynchronous discharges from an aggregate of CNS neurons. Manifestation of a seizure can include impairment or loss of consciousness and sensory, motor or behavioural abnormalities. The term **epilepsy** describes a **syndrome characterised by recurrent seizures**.

**CLASSIFICATION**-

1. Partial seizures:
   i. Simple partial---> no alteration in consciousness
   ii. Complex partial---> transient inability to maintain normal contact with environment.
   iii. Partial seizures with secondary generalization

2. Primary generalised seizures:
   i. Absence (Petit mal)
   ii. Tonic-clonic (Grand mal)
   iii. Tonic
   iv. Atonic
   v. Myoclonic

3. Unclassified:
   i. Neonatal Seizure
   ii. Infantile Spasm

4. Status epilepticus

**PARTIAL SEIZURES**- Seizure activity is restricted to discrete areas of cerebral cortex. Partial seizures are often associated with structural abnormalities of the brain. In such case CT/MRI of brain is **mandatory**.

1. **Simple partial seizure**- The consciousness is fully preserved during the seizure simple partial seizure cause motor, sensory autonomic or psychic symptoms.
i. **Jacksonian march**- Movements begin in a very restricted region and gradually progress to include a large area (not whole area).

ii. **Todd's paralysis**- Localised paresis in the involvement region--> followed by complete revival

iii. Seizure partialis **continua**- Seizure activity continue for hours to days.

2. **Complex partial seizure**- Begins with **aura**. Focal seizure activity accompanied by a transient impairment of patient’s ability to maintain normal contact with the environment

3. **Partial seizures with secondary generalization**- Partial seizure can spread to involve both hemisphere and produce generalised seizure. It is frequently observed following **simple partial seizure**.

**GENERALISED SEIZURES**- Arise from both cerebra simultaneously. Result from cellular, biochemical or structural abnormalities

**Phases of a tonic-clonic seizure**: Prodromal phase, Tonic phase, Clonic phase, Post-ictal phase

**69. Drug of choice in absence seizure**-
   1. Valproate----------ans
   2. Phenytoin
   3. Carbamazepine
   4. Topiramate

**Discussion**- (AIIMS 08)--> Drug table H/17 Chapter-363(Table 363-8) 
Remember--> Saare dard ki ek dawa hai Valproate

**Meningitis & Encephalitis**

**70. The least common complication of meningitis is**-
   1. Hypernatremia----------ans(Hypnatremia--> d/t SIADH)
   2. Hydrocephalus
   3. Brain abscess
   4. Death

**Discussion**- H/17 P-2621

**Causes**-
- S. Pneumoniae > N. Meningitidis
- Children- S. Aureus (P-2622)

**Complications of Meningitis**- Raised ICP, Cranial nerve palsy, Vasculitis, Focal deficit, Seizure, Hydrocephalus, SIADS, Brain abscess, Mental retardation, Death, Subdural effusion (very common in children with H. Influenza infection)

**71. Which is not true about progressive multi focal leucoencephalopathy**-
   1. Degeneration of cortico cerebellar system----------ans(Ck 2 in copy)
   2. Difficulty in speech
   3. Indolent course over few years
   4. Brain biopsy is definitive diagnosis

**Discussion**- H/17 P-2634

**Progressive Multifocal Leukoencephalopathy**-
- Progressive multifocal leukoencephalopathy(PML) is a rare demyelinating CNS disorder caused by the reactivation of **JC virus** (JCV--> a polyomavirus)
- The virus stays latent in the kidneys and lymphoid organs until reactivation
- PML mainly occurs in adults with impaired cell-mediated immunity, especially AIDS patients but also in those with lymphoproliferative and myeloproliferative disorders
- JCV causes lytic infection of oligodendrocytes in the **white matter**
- Symptoms include altered mental status, aphasia, ataxia, hemiparesis or hemiplegia and visual field disturbances. Seizures occur in about 18%.
- CSF--> Normal
- Rx: Risperidone(P-425 KDT) and mirtazapine(P-439 KDT)
72. True about Amoebic meningoencephalitis include-
   1. Acute form of meningoencephalitis
   2. Common in tropical countries
   3. Feco-oral mode of transmission
   4. Trophozoites in cerebrospinal fluid is diagnostic

Discussion- H/17 P-1279
Infection caused by the ameba Naegleria fowleri can also cause acute meningoencephalitis (primary ambic meningoencephalitis), whereas that caused by Acanthamoeba and Balamuthia more typically produces subacute or chronic granulomatous amebic meningoencephalitis. Naegleria thrive in warm, iron-rich pools of water, including those found in drains, canals, and both natural and human-made outdoor pools. Infection has typically occurred in immunocompetent children with a history of swimming in potentially infected water. The CSF, in contrast to the typical profile seen in viral encephalitis, often resembles that of bacterial meningitis with a neutrophilic pleocytosis and hypoglycorrhachia. Motile trophozoites can be seen in a wet mount of warm, fresh CSF. No effective treatment has been identified, and mortality approaches 100%.

MND

73. Motor neuron disease is characterized by-
   1. Sensory loss
   2. Rigidity
   3. Fasciculation
   4. Focal seizure

Discussion- Riluzole (New drug)---> H/17 P-2572---> read about it

Myasthenia gravis

74. Not a characteristic feature of myasthenia gravis-
   1. Sometimes spontaneous regression
   2. Proximal muscle involvement
   3. Absent deep reflexes
   4. Fatigue with exertion

Discussion- H/17 P-2672

In MG, the fundamental defect is a decrease in the number of available AChRs at the postsynaptic muscle membrane.

Laboratory testing-
   1. Edrophonium chloride(Tensilon)---> Screening test
   2. Anti-AChR radioimmunoassay: ~85% positive in generalised MG; 50% in ocular MG. Definite diagnosis if positive but negative result does not exclude MG. ~40% of AChR antibody-negative patients with generalised MG have anti-MuSK antibodies
   3. Repetitive nerve stimulation: decrement of >15% at 3Hz: highly probable
   4. Single-fibre electromyography: confirmatory, but not specific

Reflexes Normal---> MG, Parkinson's disease

75. In myasthenia gravis, face appearance is-
   1. Snarling
   2. Mouse like
   3. Mask like
   4. None

Discussion: Mouse like face---> CRF

RESPIRATORY SYSTEM
Obstructive/Restrictive Diseases

77. FEV1/FVC% is reduced in all except-
   1. Chronic bronchitis
   2. Bronchial asthma
   3. Interstitial lung disease---------ans
   4. Emphysema

Discussion-

<table>
<thead>
<tr>
<th>Obstructive</th>
<th>Restrictive</th>
</tr>
</thead>
<tbody>
<tr>
<td>FEV1</td>
<td>Decrease</td>
</tr>
<tr>
<td>FVC</td>
<td>decrease</td>
</tr>
<tr>
<td>Ratio</td>
<td>Decrease</td>
</tr>
<tr>
<td></td>
<td>Normal/Increase</td>
</tr>
</tbody>
</table>

decrease--> small decrease
Decrease--> Big Decrease

78. In an emphysematous patient with bullous lesion which is the best investigation to measure lung volume-
   1. Body plethysmography---------ans
   2. Helium dilution
   3. Trans diaphragmatic pressure
   4. DLCO

Discussion- (AIIMS Nov 08)

• RV, FRC, and TLC- Cannot be measured by spirometry because they include the volume of gas present in the lungs
• Two techniques of commonly used to measure these volumes: helium dilution and plethysmography
• The helium dilution method may underestimate the volume of gas in the lungs if they are slowly communicating airspaces, such as bullae. In this situation, lung volumes can be measured more accurately with a body plethysmograph, a sealed box in which the patient sits while panting against a closed mouth piece

79. First to be seen in acute severe asthma is-
   1. Pulsus paradoxus--------ans
   2. Hypercapnia
   3. Acidosis
   4. Cyanosis

Discussion- (AIIMS May 08) H/17 P-1596

Features of Severe attack:
   1. Inability to speak complete sentence in one breathe*
   2. Pulse >120 per min.
   3. Respiratory rate >25/min
   4. Pulsus paradoxus of >10 mmHg
   5. PEF between 33 to 50% of predicted

Features of Life-threatening attack-
   1. Silent Chest*
   2. Cyanosis*
   3. Bradycardia*
   4. Confusion* (altered sensorium)
   5. PEF < 33% of predicted*

80. False regarding chronic bronchitis is-
   1. Hemoptysis
   2. >2 consecutive years
   3. Production cough
4. Cough for 3 months in a year---------------ans(AIPG '06)--> 3 consective months

Discussion- H/17 P-1639

81. SIRS includes all except-
1. Hypothermia
2. Leukopenia
3. Tachycardia
4. Hypotension---------------ans

Discussion-

<table>
<thead>
<tr>
<th>Definitions used to describe the condition of septic patients</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Bacteremia</strong></td>
</tr>
<tr>
<td><strong>Septicemia</strong></td>
</tr>
<tr>
<td><strong>Systemic inflammatory response syndrome (SIRS)</strong> (Only this is Imp.)</td>
</tr>
<tr>
<td>1. <strong>Fever</strong> (oral temperature &gt;38°C) or <strong>hypothermia</strong> (&lt;36°C)</td>
</tr>
<tr>
<td>2. <strong>Leukocytosis</strong> (&gt;12,000/L), <strong>leukopenia</strong> (&lt;4,000/L), or &gt;10% <strong>bands</strong>; may have a <strong>non-infectious etiology</strong></td>
</tr>
<tr>
<td>3. <strong>Tachypnea</strong> (&gt;24 breaths/min)</td>
</tr>
<tr>
<td>4. <strong>Tachycardia</strong>* (heart rate &gt;90 beats/min)</td>
</tr>
<tr>
<td><strong>Sepsis</strong></td>
</tr>
<tr>
<td><strong>Severe sepsis (similar to &quot;sepsis syndrome&quot;)</strong></td>
</tr>
<tr>
<td>1. <strong>Cardiovascular</strong>: Arterial systolic blood pressure ≤90 mmHg or mean arterial pressure 70 mmHg that responds to administration of intravenous fluid</td>
</tr>
<tr>
<td>2. Renal: Urine output &lt;0.5 mL/kg per hour for 1 h despite adequate fluid resuscitation</td>
</tr>
<tr>
<td>3. Respiratory: PaO₂/FIO₂ ≤ 250 or, if the lung is the only dysfunctional organ, ≤ 200</td>
</tr>
<tr>
<td>4. Hematologic: Platelet count &lt;80,000/L or 50% decrease in platelet count from highest value recorded over previous 3 days</td>
</tr>
<tr>
<td>5. Unexplained metabolic acidosis: A pH ≤ 7.30 or a base deficit ≥ 5.0 mEq/L and a plasma lactate level &gt; 1.5 times upper limit of normal for reporting lab</td>
</tr>
<tr>
<td>6. Adequate fluid resuscitation: Pulmonary artery wedge pressure ≥ 12 mmHg or central venous pressure ≥ 8 mmHg</td>
</tr>
<tr>
<td><strong>Septic shock</strong></td>
</tr>
<tr>
<td><strong>Refractory septic shock</strong></td>
</tr>
<tr>
<td><strong>Multiple-organ dysfunction syndrome (MODS)</strong></td>
</tr>
</tbody>
</table>

**Cor Pulmonale**

82. Cardinal sign for the diagnosis of chronic corpulmonale is-
1. Raised jugular venous pressure
2. Right ventricular failure
3. Left ventricular hypertrophy
4. Right ventricular dilatation------------------ans
Cor pulmonale is right ventricle enlargement* with or without failure* secondary to lung parenchyma, pleura or chest wall disease.

A 29 year old unmarried female presents with dyspnea, her chest x-ray is normal, FVC-92%, FEV1/FVC-89% DLCO-59%. On exercise her oxygen saturation drops from 92% to 86%. What is the diagnosis-
1. Alveolar hypoventilation
2. Primary pulmonary hypertension------------ans(Bosenta--> New drug)
3. Interstitial lung disease
4. Anxiety

**Pneumonia & Sepsis**

Multiple cysts in lungs are common in infection with-
1. Staphylococcus------------ans(May AIIMS 2008)
2. Streptococcus
3. Esch. Coli
4. H.Influenzae

S. pneumoniae is the most common cause of CAP
In ventilator associated pneumonia M/C non-MDR pathogen- S.Pneumoniae and MDR pathogen- P. aeruginosa

'CURB-65' score indicate severity for-
1. COPD
2. Asthma
3. Pneumonia------------ans
4. Ca Lung

Severity Core adverse features: 'CURB-65' score:
1. Confusion
2. Urea > 7mmol/L (7 x 2.8 = 19.6 mg %)
3. Respiratory rate >30/min
4. BP < 90 systolic and/or < 60 mm Hg diastolic
5. Age > 65

Score-
0-1 = Home treatment possible
2 = Hospital Therapy
3 = Indicates severe pneumonia

**Fungal Infection**

Rx of refractory histiocytosis-
1. Cladarabine------------ans
2. High dose MTX
3. High dose cytosine arabinoside
4. Fludarabine

**CLASSIFICATION AND PATHOLOGY - Histiocytosis**-
1. Class I (Histiocytosis X/Langerhans cell histiocytosis)-
   - Eosinophilic granuloma
   - Hand Shullar Christian disease
   - Letterer-Siwe disease
2. Class II
3. Class III

Three classes of childhood histiocytosis are recognised, based on histopathologic findings. The most well-known childhood histiocytosis, previously known as histiocytosis X, constitutes class I and includes the clinical entities of Eosinophilic granuloma, Hand-Schuller-Christian disease (classic triad of signs consists of diabetes insipidus, exophthalmos, and bony lesions composed of histiocytes), and Letterer-Siwe disease. The name Langerhan’s cell histiocytosis (LCH) has been applied to the class I histiocytes.

87. A 54 year old smoker man comes with fever, hemoptysis, weight loss and oligoarthritis. Serial akiagram shows fleeting opacities. What is the diagnosis?

1. ABPA
2. Ca lung
3. TB
4. Wegener’s granulomatosis

Discussion - (AIIMS Nov 08)

Allergic bronchopulmonary aspergilosis (ABPA): This results from a Type I and III* hypersensitivity reaction to Aspergillus fumigatus. Early on, the allergic response causes bronchoconstriction, but as the inflammation persists, permanent damage occurs, causing bronchiectasis*. The bronchial asthma of ABPA likely involves an IgE-mediated hypersensitivity, whereas the bronchiectasis associated with this disorder is thought to result from a deposition of immune complexes in proximal airways.

Symptoms: Wheeze, cough, sputum (plugs of mucus containing fungal hyphae), dyspnoea and recurrent pneumonia*

Investigations: CXR (transient segmental collapse or consolidation, bronchiectasis). The chest roentgenogram may show transient, recurrent infiltrates or may suggest the presence of proximal bronchiectasis. High resolution chest CT is a sensitive, noninvasive technique for the recognition of proximal bronchiectasis. Aspergillus in sputum; positive aspergillus skin test and/or aspergillus-specific IgE RAST (radioallergosorbent test); positive serum precipitins; eosinophilia; raised serum IgE

Treatment:
1. Prednisolone
2. Bronchodilators

TB

88. Which of the following is a new drug for ATT?

1. Linezolid
2. Thiacetazone
3. Capreomycin
4. PAS

Discussion - H/17 P-1006

<table>
<thead>
<tr>
<th>Second line drugs</th>
<th>Newer drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thiacetazone</td>
<td>Rifabutin</td>
</tr>
<tr>
<td>Capreomycin</td>
<td>Rifapentene</td>
</tr>
<tr>
<td>PAS</td>
<td>Linezolid</td>
</tr>
<tr>
<td>Ethionamide</td>
<td>Amox/clav combination</td>
</tr>
<tr>
<td>Kanamycin</td>
<td>Ofloxacin</td>
</tr>
<tr>
<td>Ciprofloxacin</td>
<td>Clarithromycin</td>
</tr>
<tr>
<td>Cycloserine</td>
<td>Azithromycin</td>
</tr>
<tr>
<td>Amikacin</td>
<td>Clofazimine</td>
</tr>
</tbody>
</table>

New technique (New question)- P-1015
- IF-gamma release assay (PQ)
- BACTEC radiometric method: 14-C labelled palmitic acid. It can detect growth in 5-8 days. Only method to detect live bacilli
89. True about sarcoidosis-
   1. Hypercalcemia
   2. Raise ACE
   3. Mantoux negative
   4. All-----------------------------ans(Kvein’s test--> Like mantoux)

90. Disseminated tuberculosis is characterized by all except-
   1. Fever
   2. Weight loss
   3. Mantoux test always positive------------------------ans
   4. Meninges and adrenals may be involved

91. Mrs. Gupta is in mid trimester pregnancy. She has developed TB. Best treatment is-
   1. HR
   2. HRE----------------ans(Safety against pyrazinamide is less documented)
   3. HRZ
   4. HZE

**Pulmonary Embolism**

92. Most common feature of pulmonary embolism is-
   1. Sweating
   2. Tachypnea----------------ans
   3. Tachycardia
   4. Pain chest
   Discussion- (H/17, P = 1651)
   Dyspnea is the most frequent symptom of PE, and tachpnea is it's most frequent sign.

93. A 55 year old lady posted for hip replacement surgery. All of the following are known causes of DVT except-
   1. PNH
   2. OCP
   3. Infections------------------------ans
   4. Surgery for 1 hour
**Pneumoconiosis**

94. 'Calcification of diaphragmatic pleura' is characteristic of pneumoconiosis due to-

1. Asbestosis------------------ans
2. Berylliosis
3. Silicosis
4. Anthracosis

**Discussion**

- Asymptomatic pleural plaque

95. In which condition, paradoxical respiration is seen-

1. Severe asthma
2. Bulbar polio
3. Diaphragmatic palsy-------------------ans
4. Single rib fracture

**Discussion**

- Flail chest > Diaphragmatic palsy.

Paradoxical respirations are seen as movement of the chest wall opposite of what one would expect to see, i.e. inflation of the lung during expiration. Paradoxic respirations are seen and deflation of the lung during inspiration. This occurs usually after blunt trauma to the chest, resulting in flail chest (loss of stability of the thoracic cage due to multiple rib fractures, and/or separation of the ribs from the sternum) or with paralysis of the diaphragm.

**Lung Cancer**

96. Commonest symptoms of primary lung cancer-

1. Cough------------------ans(FAQ)
2. Hemoptysis
3. Dyspnea
4. Pain chest

Discussion- (H/17 P=554)
- Lung cancer:
  - NSCLC (80%)
    - SqCC
    - Adenocarcinoma
    - Bronchioalveolar carcinoma
  - SCLC (20%)
    - Oat cell
    - Intermediate
    - Combined cell type

97. Which carcinoma of lung responds best to chemotherapy-
1. Squamous cell type
2. Adeno carcinoma
3. Anaplastic
4. Small cell type----------------ans

Discussion-
Treatment:
- Non-small cell tumors: Excision is the treatment of choice. Curative radiotherapy is an alternative if respiratory reserve is poor for Sx. Chemotherapy + radiotherapy for more advanced disease. Chemotherapy Paclitaxel + cisplatin
- Small cell tumors are nearly always disseminated at presentation. They may respond to chemotherapy etoposide + cisplatin or etoposide + cisplatin + paclitaxel or Cyclophosphamide + doxorubicin + vincristine or Cisplatin + radiotherapy (if limited disease). Pleural drainage/pleurodesis for symptomatic pleural effusions

Solitary Pulmonary Nodule with "Ground Glass" Opacity: At present, only two radiographic criteria are reliable predictors of the benign nature of an SPN: lack of growth over a period >2 years and certain characteristic patterns of calcification. Calcification alone does not exclude malignancy. However, a dense central nidus, multiple punctate foci, and "bull's eye" (granuloma) and "popcorn ball" (hamartoma) calcifications are all highly suggestive of a benign lesion.

100. Most common lung cancer in INDIA-
1. Squamous
2. Adeno-ca
3. SCCL
4. None----------------ans(Secondaries)

Superior vena cava syndrome- Superior vena cava obstruction is most commonly caused by bronchogenic carcinoma. Increased venous pressure produces edema of the upper body, cyanosis, dilated subcutaneous collateral vessels in the chest, and headache. Cervical lymphadenopathy may also be present. When carcinoma is the cause of superior vena cava syndrome, the treatment is usually palliative and consists of diuretics and radiation.

Pleural effusion

98. *Low sugar in pleural effusion is a characteristic feature of-
1. CCF
2. Pancreatitis
3. TB
4. RA----------------ans

99. Commonest causes of pleural effusion in India & world respectively are-
1. Viral/Viral----------------ans
2. TB/HIV
3. TB/CHF
4. TB/Nephrotic syndrome due diabetic

Since answer key was not given answer below are based on explanations that was provided. Verify them in case of doubt....

ENDOCRINOLOGY

1. Acute fulminant diabetes is related to-
   1. DKA
   2. Viral infection----------------ans
   3. MI
   4. Pregnancy

Discussion-
Fulminant diabetes mellitus (New Question): It is acute onset diabetes, can occur in any age, occurs after viral infection. Serum insulin level are reduced but there are no antibodies against insulin or beta cell.

Diabetic cheiroarthropathy- Arthropathy of the joints of the hands and fingers is a complication of Juvenile onset DM.

2. Which is not a feature of type II DM-
   1. Obesity
   2. Positive family history
   3. Low serum insulin----------------ans(PGI May 06)
   4. Insulin Resistance

Discussion-
Type 1 DM Type 2 DM
Age at onset Usually young Adults
Body weight Lean & thin* Obese
Family h/o Less common than in type 2 Strongly +ve
Insulin requirement Absolute (alpha-glucosidase inhibitors can be given) OHA, later insulin
Serum insulin level Low High(initially)Normally or low (late stage). Insulin resistance is characteristic.

- MODY-
  ○ AD (Strong familial association)
  ○ Younger age group
  ○ No Ab
  ○ *Impaired insulin secretion (Can be treated by drugs)
  ○ HT, Hyperlipidaemia not seen

- LADA- Late onset autoimmune diabetes of Adults. GAD antibodies: Persons with LADA usually test positive for GAD antibodies, whereas in type 1 diabetes these antibodies are more commonly seen in adults rather than in children.

3. Not a feature of DKA is
   1. Tachypnea
   2. Bradycardia----------------ans
   3. Abdominal pain
   4. Dehydration

Discussion-
Diabetic Ketoacidosis (VIP Topic--> H/17 P-2283)-

Clinical features-
Symptoms-
• Nausea
• Vomiting
• Abdominal pain* (PGI June 06)
• Altered mental function
• Shortness of breath

Signs-
• Tachycardia (AIIMS Nov 07)
• Dry mucous membrane
• Dehydration
• Hypotension
• Kussmaul respiration* --> d/t metabolic acidosis
• Fruity odour in breadth --> d/t acetone
• Tachypnea
• Abdominal tenderness --> may resemble acute pancreatitis or ruptured viscus
• Fever may be there (indicates infection)

Investigations-
1. Hyperglycemia- Blood sugar 400-600 mg%
2. TLC- Leucocytosis*. It is a feature of DKA. It does not indicate infection (Presence of fever indicate infection* --> DPG 2004)
3. Hypertriglyceridemia- Hyperlipoproteinemia (dyslipidemia found earliest in DM)
4. BUN/Creatinine – increase (d/t intravascular fluid depletion)
5. Pseudohypoglycemia** (PGI June 2007) --> A low serum sodium concentration resulting from volume displacement by massive hyperlipidemia or hyperproteinemia, or by hyperglycemia.
6. Hyperkalemia(DPG)- shifting of K+ from intracellular to extracellular compartment due to decrease in insulin
7. Metabolic acidosis(Kussmaul's respiration)- Low HCO₃ with increase anion gap. (AIIMS May 2007)
8. Plasma ketones- Positive
9. S. Osmolality increased; 300-320 mosm/kg
   ➢ (Na+K) x 2 + BUN/2.8 + RBS/18
   ➢ Na contributes 270 of normal 290 serum osmolality
   ➢ RBS--> to convert from m.mol divide by 18. Here RBS increases and hence S. Osmolality also increases.
10. Hyperamylasemia

MCQ: Blood pH is the single best test to know the prognosis of a case of DKA

Complications of DKA-
• Cerebral edema (most dangerous complication) --> AIPG 2006. Most frequently seen in children
• Venous thrombosis--> S/C Heparin given as a *preventive measure
• Adult respiratory distress syndrome
• MI
• Acute gastric dilatation

Treatment-
1. Fluids --> 0.9% saline
2. Insulin --> Regular Insulin I/V (Insulin is given I/V in DKA) --> AIPG 2006. Regular insulin is the only insulin which is given I/V other long acting --> S/C. Regular insulin is a short acting insulin.
3. Treatment precipitating events like non-compliance; infection (by antibiotics)
4. K+ replacement -->Initially when patient comes initially he is hyperkalemic, later on when patient is treated with insulin, serum potassium level goes down and may require potassium replacement
5. Injection HCO₃ I/V if pH < 7
4. Mr. Ram Lal, 80 years, a known case of diabetes, was brought to emergency ward in unconscious state. His blood sugar was 900 mg%, dry tongue. BP 80/50. Urine ketones negative. Which of the following fluid you will give to treat his marked dehydration:

1. Injection RL
2. Injection N/2 saline
3. Give plain water by Ryles tube
4. Injection N saline

Discussion: (AIIMS Nov 2006) H/17 P-2285

**Treatment:**

1. Fluid -->Total fluid deficit(9-10L) should be reversed over 1-2 day. Initially give normal saline to stabilize the patients hemodynamically. Then Give 0.45% saline
2. Regular Insulin to be given IV.
3. Subcutaneous heparin because these patients are prone to venous thrombosis

5. Which of the following is not included in intensive management of diabetes mellitus:

1. Pregnancy
2. Postural hypotension due to autonomic neurotherapy
3. DM with acute MI
4. Post kidney transplant

Discussion:

**Diabetic neuropathy and diabetic foot care:** (VIP Topic)

Occurs due to ischaemia (absent dorsalis pedis pulses) and peripheral neuropathy (injury or infection over pressure points, eg the metatarsal heads)

**Signs:**

Neuropathy: Loss of Sensation(DPG): (vibration is the first to be lost) in gloves & ‘stocking’ distribution, absent ankle jerks

**MCQ:** Callus formation is an one of the early manifestation of diabetic foot

6. Which electrolyte imbalance can occur in a diabetic patient with normal blood urea & serum creatinine:

1. Hyperkalemia
2. Hypokalemia
3. Hypernatremia
4. Hypocalcemia

7. Epalrestat is a-

1. Lipid lowering agent
2. Anti hypertensive drug
3. Anti diabetic drug--------ans
4. None
Discussion- (New Question)

**Pathophysiology of diabetic complication**-
- In diabetes glucose is converted into **sorbitol** which is a tissue toxin*
- Enzyme required in this step is **aldose reductase** *(Glucose ---- aldolase reductase----- > Sorbitol)*
- Recently a new drug Eparrestat (NQ) has been launched which is a **aldose reductase inhibitor** (NQ) which is supposed to reduce the complication of diabetes.

8. Which of the following is a food of highest glycemic index-
1. Butter
2. Potato
3. Rice
4. Rasgulla--------ans
Discussion- (AIPG 2007)
Avoid food of high glycemic index (i.e. any food like sugar, glucose powder etc. which are absorbed immediately in GIT and raise blood sugar very fast).

**NB: Cornflake is a food of very high glycemic index***

Besides glucose and water, alcohol is absorbed immediately in GIT. But, when alcohol + fatty food--> Nasha der se aayega.

9. True about Samagyi phenomena

3am 8am
1. Hypoglycemia  Hypoglycemia
2. Hypoglycemia  Hyperglycemia
3. Hyperglycemia  Hyperglycemia
4. Hyperglycemia  Hypoglycemia
Discussion- Answer-2 (AIIMS Nov 2005)

**Somogyi phenomena**- It is the morning (8 am) hyperglycemia which follows hypoglycemia at late night (2 am). This occurs due to release of counter regulatory hormones (*glucagon, 1st to be released, followed by adrenaline--> epinephrine not other Catecholamine, steroids, growth hormones thyroid*). Treatment is to reduce the night dose of insulin.

10. Detemir is a-
1. Sulphonylurea
2. Insulin--------ans
3. Thiopglitazone
4. Alpha glucosidase inhibitor
Discussion-

**Pharmacology of Insulin**-
1. Ultra short acting: Lispro, Apert*, Glulisine*
2. Short acting: Regular insulin
3. Intermediate acting: Lente, NPH
4. Long acting: Ultra Lente, PZI, Insulin Glargine, **Detemir (New question)**
5. Inhalation insulin: Exubera (New drug)--> Withdrawn (Lung Ca increases)

MCQ: Glulisine, Aspart, Glargine, Detemir, and Exubera are new insulin

11. Which of the following is a GLP-1 agonist
1. Sitagliptin
2. Glulisine
3. Exenatide--------ans
4. None
Discussion-

**New concepts and new oral drugs in diabetes:**
1. Amylin effect (Physiology):
   1. Normally when we eat food, intestine secretes amylin (released by beta-cells of pancreas with insulin--> both deficient in diabetes
   2. Amylin reduces glucagon secretion and slows down the gastric emptying
   3. Pharmacology: New anti diabetic drugs which increases amylin secretion(Amylin agonist)--> Pramlintide

2. Incretin effect (Physiology):
   1. Normally when anybody eats food it causes increased insulin secretion
   2. In a new study it is seen that normal people who take oral glucose tend to have higher serum insulin level as compared to those normal people to whom glucose was given intravenous although blood sugar was same in both the groups of people. It is because oral glucose causes release of some polypeptide from the intestine which raise serum insulin level (Insulinotropic polypeptide).
   3. These polypeptide include GLP-1, GIP(Secreted by L-Cells of intestine)
   4. These two polypeptide are degraded by DPP-IV Enzyme
   5. Pharmacology:
      1. New anti diabetic drugs which raised GLP-1 and GIP(injection)-
         1. Exenatide*
         2. Liraglutide
      2. New antidiabetic drugs which raised GLP1 and GIP level by inhibiting DPP-IV(DPP-IV inhibitors)---> Oral and hence very popular
         1. Sitagliptin
         2. Vildagliptin

Note: Anti-diabetic drugs which do not cause hypoglycemia-
   1. Metformin
   2. Alpha glucosidase inhibitor
   3. Thioglitazone
   4. DPP-IV inhibitor

12. As per JNC VII criteria target goal of BP control in diabetes patients is-
   1. <130/80-----------------ans
   2. <125/80
   3. <120/80
   4. <125/75

Discussion- BP < 130/ <80 mmHg (<125 / <75 is associated with renal disease)

13. A 70 years old patient was brought to emergency ward in a comatose state. His ABG was done and findings are as follows-

   Na = 135 meq/lit, K = 5 meq/lit
   HCO3 = 10 meq/lit
   Blood glucose = 20 mmol/lit
   BUN = 4 mmol/lit (Normal value of BUN = 2.86 -7.14 mmol/L or 8 to 20 mg/dL)

What is your diagnosis-
   1. Non ketonic hyperosmolar diabetic coma
   2. DKA
   3. Hypoglycemic coma
   4. Lactic acidosis

Discussion- Confusing question(AIPG 2007)

In this case patient has metabolic acidosis because his serum bicarbonate is 10meq/lit. So possibility of choice A is ruled out. His blood sugar is 20 mmol/lit i.e. 360 mg% (20 x18=360) so choice C is ruled out Both choice 2 and 4 are possible in this case but patient’s age is 70 yrs so better ans is 4.....Dr Bhatia...If age was 17 then--> DKA.........
I think the question was about the student's ability to differentiate b/n DKA and HHS.......and clearly answer is DKA...also bicarbonate level is normal to slightly decreased in HHS...which also supports the answer.......(H/17 P-2282)

14. Pretibial myxedema is seen in-
   1. Hyperthyroid
   2. Hypothyroid
   3. Grave disease----------------ans
   4. Hashimoto thyroiditis
Discussion- H/17 P-2233 (Table 335-6)
Graves-
   • Hyperthyroidism
   • Dermatopathy--> Tibial myxedema
   • Ophthalnopathy

Extra question: Af is seen in-
   1. Hypertheroidism----------------ans
   2. Hypotheroidism
   3. Grave disease
   4. Hashimoto thyroiditis

15. A 40 year old woman present with a firm nodular midline neck mass. Blood tests reveal the presence of antibodies to thyroglobulin. Likely cause is-
   1. Graves’ disease
   2. Thyroid carcinoma
   3. Toxic multinodular goitre
   4. Hashimoto’s thyroiditis----------------ans
Discussion-

Hashimoto’s thyroiditis-
   • Most common auto-immune cause of goitrous hypothyroidism
   • 20-60 yrs old women
   • Anti thyroglobulin antibodies and Thyroid peroxidase antibodies are present in the serum
   • Antinuclear factor (ANF) may be positive
   • Thyroxine therapy

16. All are true about de Quervain’s thyroiditis except-
   1. Raised ESR
   2. Subsides spontaneously
   3. Autoimmune etiology----------------ans
   4. Pain and swelling of the thyroid occurs
Discussion- Viral infection (B+L 25/e P-800)

17. Pituitary apoplexy is not associated with which of the following medical conditions-
   1. DM
   2. Hyperthyroidism----------------ans
   3. Sickle cell anaemia
   4. Hypertension
Discussion- (AIPG 08) Ref. H/17 P-2198

Extra question(AIIMS May 07): First hormone to decrease after Pituitary radiation is-
   1. GH-----------ans
   2. TSH
   3. ACTH
   4. FSH
Discussion- Though the pattern of hormone loss is variable, after cranial irradiation, GH deficiency is most common, followed by gonadotropin TSH and ACTH deficiency
19. Secondary hyperparathyroidism is seen in all except-
   1. Chronic renal failure
   2. Cushing’s syndrome------------------------ans
   3. Malabsorption of calcium from gut
   4. Rickets

Discussion- (AI 06)

<table>
<thead>
<tr>
<th>PTH</th>
<th>Ca++</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary</td>
<td>Increase</td>
</tr>
<tr>
<td>Secondary</td>
<td>Increase</td>
</tr>
<tr>
<td>Tertiary</td>
<td>Increase</td>
</tr>
</tbody>
</table>

I. Primary hyperparathyroidism-
   • **Causes:** The most common cause of primary hyperparathyroidism is *parathyroid adenoma*.
   • **Important point:** Adenomas are most often located in the *inferior parathyroid gland* (MCQ). Chief cells are predominant in both hyperplasia and adenoma

II. Secondary hyperparathyroidism- *Ca²⁺ decrease*, PTH Increase (MCQ)
   • **Causes:** Low vitamin D intake, CRF, Rickets, Osteomalacia, Malabsorption

III. Tertiary hyperparathyroidism- *Ca²⁺ Increase*, PTH Increase (FAQ)
   • **Causes:** Occurs after prolonged secondary hyperparathyroidism, causing glands to act *autonomously* having undergone hyperplastic or adenomatous change. This *causes increase in Ca²⁺ from secretion of PTH unlimited by feedback control*. Seen in chronic renal failure.

IV. Malignant hyperparathyroidism-
   • **Causes:** *Parathyroid-related protein* (PTHrp) is produced by *some squamous cell lung cancers, breast* and *renal cell carcinomas*. (AIIMS May 2007)
   • **Note:** Hypercalcemia in malignant occur due to *increase level of PTHrp*. In this PTH level are *not* raised (LQ)

18. Hypercalcemia is seen in all except-
   1. Prolonged immobilization
   2. Multiple myeloma
   3. Sarcoidosis
   4. Acute renal failure------------------------ans

Discussion- (AIIMS Nov 07)
   1. **Osteoblast**: These are the bone forming cell. Whenever there is increase activity of osteoblast it causes raised level of *serum alkaline phosphatase* (MCQ). That is why level of serum alkaline phosphatase is raised in *children and pregnancy* (MCQ)
   2. **Osteocytes**: They are the bone *maintaining* cells. They *regulate bone activity*.
   3. **Osteoclast**: They cause *bone resorption* (MCQ). In Multiple myeloma there is increase activity of osteoclast that is why there are *lytic lesion and Hypercalcemia* in MM(MCQ)

**Note:** In MM there is *no increase* in activity of osteoblast. That’s why serum alkaline phosphatase level are *normal* in MM and bone scan is also *normal*.

**Other causes of Hypercalcemia:**
   1. Lithium, Thiazide (*Furosemide*--> Ca loss)
   2. Ca of breast, Lung, kidney, MM, lymphoma
   3. Vit D intoxication, Vit A intoxication, Aluminium intoxication
   4. Hyperparathyroidism, *Hyperthyroid*
   5. Sarcoidosis
   6. Prolong immobilization
   7. Milk alkali syndrome

20. Which is not true of hypocalcemia-
1. Can occur in tumor lysis syndrome
2. Inverse relation with Mg++ levels-----------------ans
3. Prolonged QT interval
4. Latent tetany is seen

Discussion- Option 2 --> Inverse relation is only found in CRF and Gietelman's syndrome

Latent tetany-
- Chvostek sign
- Trousseau sign

Hypocalcemia---> Causes:
1. Idiopathic (Autoimmune)- associated with other autoimmune disorders. Lab tests of idiopathic hypoparathyroid – Ca2+ (decrease), PO4 (Increase)
2. Infantile hypoparathyroidism: It is associated with thymic aplasia (Di-George syndrome)
3. Post operative: Surgery (thyroidectomy)---> Leads to transient hypocalcemia
4. Pseudohypoparathyroidism(Ca decrease, PTH Increase) is a group of disorders characterized by hypocalcemia due to renal resistance to PTH. There are several subtypes caused by different mutations involving the PTH receptor or it's G protein or adenyly cyclase. PTH levels are high and the PTH receptors in bone are typically involved, such that bony changes of hyperparathyroidism may be evident. Various phenotypic abnormalities may be associated – classically, short stature, round face, obesity, short fourth metacarpals, ectopic bone formation, and mental retardation

21. Cushing's disease is characterized by-
1. Increased urinary catecholamines
2. Increased serum ACTH and serum cortisol-------------------ans
3. Increased serum ADH
4. Decreased serum ACTH and increased serum cortisol

Discussion- Traditionally, only an individual who has an ACTH-producing pituitary tumor is defined as having Cushing's disease, whereas Cushing's syndrome refers to all causes of excess cortisol: exogenous ACTH tumor, adrenal tumor, pituitary ACTH-secreting tumor, or excessive glucocorticoid treatment.

Cushing Syndrome-
A) ACTH-dependent causes: (Increase ACTH)
1. Cushing's disease: Bilateral adrenal hyperplasia due to an ACTH secreting pituitary adenoma
2. Ectopic ACTH production: Especially smell cell lung cancer and carcinoid tumours(MCQ), Pancreatic Ca, bronchial adenoma
B) ACTH-independent causes: (decrease ACTH due to –ve feedback)
1. Iatrogenic: Pharmacological doses of steroids(common)
2. Adrenal adenoma or carcinoma: may be associated with abdominal pain and virilization in women
3. Adrenal nodular hyperplasia

<table>
<thead>
<tr>
<th>Causes of Cushing Syndrome</th>
<th>S. ACTH</th>
<th>Diagnosis</th>
<th>Rx</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iatrogenic (drug--&gt;Steroid)</td>
<td>Reduced</td>
<td>History</td>
<td>Reduce dose</td>
</tr>
<tr>
<td>Pituitary adenoma (Cushing’s d/e)</td>
<td>Raised</td>
<td>CT(Head)</td>
<td>Surgery</td>
</tr>
<tr>
<td>Ectopic ACTH secreting tumor</td>
<td>Raised</td>
<td>CT(Chest)</td>
<td>Surgery</td>
</tr>
<tr>
<td>Adrenal adenoma/Ca</td>
<td>Reduced</td>
<td>CT(abdomen)</td>
<td>Surgery</td>
</tr>
</tbody>
</table>

Features of Cushing's syndrome:
1. Symptoms due to excess of glucocorticoid
   1. The loss of proteins from the muscle in particular causes severe weakness of muscles (AIIMS May 07)
   2. The protein collagen fibres in the subcutaneous tissue are diminished so that the subcutaneous tissue tear easily resulting in development of large purplish striae where they have torn apart.
   3. Elevated blood glucose concentration
4. Mobilization of fat from the lower part of the body with concomitant extra deposition of fat in the thoracic and upper abdominal regions giving rise to a buffalo torso. The excess secretion of steroid also leads to an edematous appearance of the face ("moon facies").

2. Symptoms due to excess of mineralocorticoid
   1. Salt and water retention leads to **hypertension**
   2. Significant K+ depletion (**hypokalemia**)

3. Symptoms due to excess of androgen
   1. Hirsutism
   2. Facial acne
   3. Oligomenorrhea
   4. Amenorrhea

22. All of the following will cause myopathy except-
   1. Cushing syndrome
   2. X-linked hypophosphatemic rickets-----------------ans (AIIMS May 07)
   3. Nutritional osteomalacia
   4. Oncogenic osteomalacia

23. All are features of primary hyperaldosteronism except-
   1. Hypokalemia
   2. Hypertension
   3. Metabolic acidosis-----------------ans
   4. Low plasma rennin

Discussion-

Conn’s Syndrome-
- Primary hyperaldosteronism d/t aldosterone secreting adrenal adenoma
- Edema is not a feature of Primary aldosteronism but a feature of Secondary aldosteronism
- **Epleronone**- Aldosterone receptor antagonist

24. Addison’s disesase is characterized by-
   1. Hypokalemia
   2. Peptic ulceration
   3. Hypoglycemia------------------ans
   4. Increase aldosterone

Discussion- (AIIMS 07)

**ADDISON’S DISEASE** (Hypofunctioning of adrenal cortex)-
- Increase ACTH--> Bronze Pigmentation of skin
- Mineralocorticoid deficiency-
  - Hyponatremia with contraction of ECF Volume
  - Hyperkalemia
  - Acidosis
- Glucocorticoid deficiency--> Hypoglycemia

25. Frequency of tuning fork used to test vibration sense is
   1. 128----------------ans
   2. 256
   3. 512
   4. 1024

Discussion- For checking posterior column involvement --> do Rhomberg’s test

Pluripotent stem cells-
   1. L
     1. B--> CLL
     2. T--> Mycosis fungoides
2. M
   1. RBCs --> PV
   2. Platelets --> ET
   3. N/B/E/M --> CML--> Myeloproliferative disorders

NEPHROLOGY

53. Osmotic demyelination syndrome occur in treatment of-
   1. Acute GN
   2. SIADH-----------------ans
   3. CRF
   4. UTI

Discussion- (FAQ)
- Hyponatremia (sodium<135mEq/L)
- Low serum osmolality
- BUN and serum uric acid tends to fall because of plasma dilution
- Serum potassium and Bicarbonate levels are normal in SIADH

T/t of SIADH:

Acute SIADH
   1. Standard first line therapy is water restriction, then
   2. Infusion of hypertonic saline (3%)

A rapid correction will produce central pontine myelinolysis (Osmotic demyelinating syndrome) which is an acute potentially fatal neurological syndrome characterized by quadriparesis, ataxia and abnormal extraocular movements.

Chronic SIADH: Hyponatremia can be corrected with -> Demeclocycline or fludrocortisone

54. Pseudohyponatremia occurs in which of the following conditions-
   1. SIADH
   2. CHF
   3. Hyperlipidemia-----------------ans
   4. Severe dehydration

Discussion: An erroneously low serum sodium concentration d/t volume displacement by massive hyperlipidemia/hyperproteinemia/hyperglycemia

55. All occurs in malignant hyperthermia except-
   1. Hyperkalemia
   2. Bradycardia-----------------ans
   3. Hypertension
   4. DIVC

Discussion- (AIIMS May 2007)
Malignant Hyperthermia is clinical syndrome observed during general anaesthesia associated with rapidly increasing temperature as great as 1*C/5min. It has mutations in the ryanodine receptor. Clinical features of which has been described as:
- Hyperthermia (temperature may rise to more than 105*F). The heat production is due to increased muscle metabolism(both aerobic and anaerobic), glycolysis and hydrolysis of high energy phosphates involved in the process of contraction/relaxation
- EtCO₂ may rise more than 100mm Hg (normal 32 to 42 mm Hg). This is the most sensitive early sign of malignant hyperthermia
- TACHYCARDIA (and not BRADYCARDIA). Hypertension and Cardiac arrhythmias can also occur
- Severe metabolic acidosis (pH < 7.0)
- Hyperkalemia, muscle rigidity, increased rigidity, increased creatine phosphokinase, increased myoglobin
Renal failure, DIC, pulmonary and cerebral edema
Death

- Acidosis- Hyperkalemia
- Alkalosis- Hypokalemia

56. Interpret the following data

HCO₃⁻ = 8 meq/lit
pH = 7.40
PCO₂ = 20 mmHg
PO₂ = 90 mmHg
Na = 136 meq/lit.
K = 4 meq/lit

What is your diagnosis-
1. Uncompensated metabolic acidosis
2. Uncompensated Respiratory acidosis
3. Compensated Respiratory acidosis
4. Compensated metabolic acidosis-----------------ans

Discussion- Since all options given are acidosis means pH should have been lower than 7.35 but it is 7.4 here means it is compensated, and acidosis is d/t decrease in bicarbonate level as it is here, so metabolic acidosis component is primary, which has been compensated by resp. alkalosis as decrease in CO₂ suggests; to bring pH to normal.

Basic fundamental
A) Acidosis occur by two mechanism
   1. Gain of acid
   2. Loss of alkali
B) Alkalosis occur by two mechanism
   1. Loss of acid
   2. Gain of alkali

Note:
   a) CO₂ is an acid, being controlled by lungs. HCO₃⁻ is an alkali, being controlled by kidneys.
   b) Any disturbance of CO₂ leads to respiratory acidosis/alkalosis
   c) Any disturbance of HCO₃⁻ leads to metabolic acidosis/alkalosis

57. Interpret the following data

HCO₃⁻ = 8meq/lit
pH = 7.20
PCO₂ = 80 mm Hg
PO₂ = 90 mmHg

1. Mix metabolic acidosis & respiratory acidosis------------------ans
2. Mix metabolic acidosis & respiratory acidosis
3. Mix metabolic acidosis & respiratory alkalosis
4. Mix metabolic alkalosis & respiratory alkalosis

Discussion-

<table>
<thead>
<tr>
<th>Disturbance</th>
<th>Primary alteration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory acidosis</td>
<td>Increased pCO₂</td>
</tr>
<tr>
<td>Respiratory alkalosis</td>
<td>Decreased pCO₂</td>
</tr>
<tr>
<td>Metabolic acidosis</td>
<td>Decreased plasma HCO₃</td>
</tr>
<tr>
<td>Metabolic alkalosis</td>
<td>Increased plasma HCO₃</td>
</tr>
</tbody>
</table>

58. All of the following can cause high anion gap acidosis EXCEPT-
1. Ethylene glycol
2. Starvation
3. Glue-sniffing-------------------------ans
4. Diabetic ketoacidosis
Discussion- (AIIMS May 2008) H/17 P-290

METABOLIC ACIDOSIS-

1. Normal anion gap metabolic acidosis- Glue sniffing/RTA
2. Increased anion gap metabolic acidosis-
   a) Increased unmeasured anions*
      1. Lactic acidosis
      2. Ketoacidosis--> Alcoholic/Diabetic/Starvation
   b) Poisoning
      1. Salicylates
      2. Methanol
      3. Ethylene glycol
   c) Accumulation of endogenous acid* --> ARF and CRF

ANION GAP: \[\text{Na} - (\text{Cl} + \text{HCO}_3^-)\]
Unmeasured anions in the body are anionic proteins, phosphates, sulphate and organic anions.
Unmeasured cations in the body include calcium, magnesium and potassium (not sodium)
In equilibrium state: Normal plasma anion gap = 14 ± 4 (MCQ)

59. In which type of Renal tubular acidosis, hyperkalemia occurs-
1. Type IV----------------------ans
2. Proximal RTA
3. Distal RTA
4. Seen in all above cases

Discussion-
Renal tubular disease (All questions come from this chart only)

<table>
<thead>
<tr>
<th>Type</th>
<th>Increased K</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type IV</td>
<td></td>
</tr>
<tr>
<td>Type II (Proximal)</td>
<td>Decreased K, <strong>Fanconi syndrome</strong></td>
</tr>
<tr>
<td>Type I</td>
<td>Decreased K, Urine pH &gt; 5.5, Renal stone</td>
</tr>
</tbody>
</table>

60. Fusion of foot process occurs in-
1. MPGN
2. Membranous GN
3. F & SG----------------------ans
4. None

Discussion- Chart Review ROAMS P-198

61. Ramlal, a 65 year male, a known case of Ca colon complains of generalized swelling in his body. His urine examination shows proteinuria of 4 gm/24 hrs. Which type of Glomerulonephritis is most likely to be caused in this-
1. Minimal charge
2. Membranous-------------------ans
3. Focal & segmental
4. Membranous proliferative

62. Complement level are reduced in all except-
1. Membranoproliferative GN
2. SLE
3. Post streptococcal GN-------------------ans
4. Cryoglobulinemia

Discussion- (Controversial Question: LQ-Latest Question)
Seen in all but returns to normal in 3(Ck in question if it should be increased in place of reduced from AIIMS paper)
64. Rani, a 24 year lady, gave birth to a healthy child, 4 days later, she complaints of inability to pass urine, she was diagnosed as having ARF & was treated by repeated dialysis. Even 1 year after that also she could not regain her normal renal function. What could had been her problem-

1. Membranoproliferative GN
2. Mesangioproliferative GN
3. Acute cortical necrosis--
4. Focal & segmental glomerulosclerosis

Discussion- Renal cortical necrosis---> emedicine(classical presentation) supported by one more source...others fairly ruled out(1and 2 are same thing; 4 presents as nephrotic syndrome)

65. Which is not a feature of Barter syndrome-

1. HT------------------------ans
2. Decreased K+
3. Metabolic alkalosis
4. All

Discussion- BGLG

<table>
<thead>
<tr>
<th></th>
<th>K</th>
<th>Metabolic</th>
<th>BP</th>
<th>S. Aldosterone</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bartter*</td>
<td>Decreased</td>
<td>Alkalosis</td>
<td>N</td>
<td>Increased</td>
</tr>
<tr>
<td>Gitelman</td>
<td>Decreased</td>
<td>Alkalosis</td>
<td>N</td>
<td>Increased</td>
</tr>
<tr>
<td>Liddle</td>
<td>Decreased</td>
<td>Alkalosis</td>
<td>High</td>
<td>Decreased</td>
</tr>
<tr>
<td>Gordon</td>
<td>Increased*</td>
<td>Acidosis*</td>
<td>High</td>
<td>Decreased</td>
</tr>
</tbody>
</table>

- Massive bleeding: >1000 ml blood loss
- Give blood for blood (Saline won't do.....)

66. Features of Gordon's syndrome (Pseudo hypo aldosteronism):

1. HT
2. Increased K+
3. Metabolic acidosis
4. All-----------------------ans

67. Acute tubular necrosis (ATN) is characterized by-

1. Hyperkalemia
2. Dilutional hyponatremia
3. Metabolic acidosis
4. All-----------------------ans

Discussion- ATN presents with acute renal failure (ARF) and is one of the most common causes of ARF. The presence of "muddy brown casts" of epithelial cells found in the urine during urinalysis is pathognomonic for ATN.

69. All improves after dialysis except-

1. Pericarditis
2. Peripheral neuropathy-------ans
3. Metabolic acidosis
4. Seizure

Discussion- Ref. Wiki--> Dialysis (confirm again)

71. Emphysematous pyelonephritis is caused by-

1. E.coli----------------apra
2. Proteus
3. Pseudomonas
4. Klebsiella

Discussion- a- H/17- 1826

Occurs in diabetic patients, often in concert with urinary obstruction and chronic infection. Emphysematous pyelonephritis is usually characterized by a rapidly progressive clinical course, with high fever, leukocytosis, renal parenchymal necrosis, and accumulation of fermentative gases in the kidney and perinephric tissues. Most patients also have pyuria and glucosuria. E. coli causes most cases, but occasionally other Enterobacteriaceae are isolated.
73. Chronic tubulo interstitial disease is caused by-
   1. Lithium
   2. Cyclosporin
   3. Hyper uricemia
   4. All------------------------ans

Discussion- H/17 for detailed list

Causes of Tubulointerstitial Disease-

A. Toxins
   1. Exogenous toxins-
      1. Analgesic nephropathy
      2. Lead nephropathy
      3. Miscellaneous nephrotoxins (antibiotics, closporine, radiographic contrast media, heavy metals)
   2. Metabolic toxins
      1. Acute uric acid nephropathy
      2. Gouty nephropathy
      3. Hyperkalemic nephropathy
      4. Hypokalemic nephropathy
      5. Miscellaneous (hyperoxaluria, cystinosis)

B. Neoplasia
   1. Lymphoma
   2. Leukemia
   3. Multiple myeloma

74. Which of the following statements about adult polycystic kidney disease is true-
   1. Autosomal recessive disorder
   2. Low erythropoietin level
   3. Hematuria can occur----------------------ans
   4. Berry aneurysm don’t have association with polycystic kidney disease

Discussion-

Autosomal dominant polycystic kidney disease(ADPKD)-

Genes on chromosomes 16 (PKD1) and 4 (PKD2)

Common Clinical Features-
   1. Flank pain and vague abdominal discomfort
   2. Acute loin pain or renal colic due to hemorrhage into the cysts
   3. Hypertension may appear after age 20 years
   4. Nocturia, hematuria and urinary infection appear in the third and fourth decade
   5. Uraemia
   6. High erythropoietin level(read from google books and multiple sites on internet)
   7. Nephrolithiasis commonly due to calcium oxalate may be there
   8. Cysts may be there in liver, pancreas, and spleen.Cysts are not present in lungs(extra-abdominal)
   9. Intracranial (berry) aneurysms also may be present which can cause SAH
   10. Azotemia is usually progressive
   11. Commonest extra renal manifestation is colonic diverticulosis
   12. Diagnosis is by IVP or ultrasound

77. Oliguria denotes urinary output less than-
   1. 400ml
   2. 100ml
   3. 200ml
   4. 500ml------------------------ans

Discussion- Anuria => < 100 ml

ARF-
1. Oliguric < 500 ml/day (Anuric is called when < 100)
2. Non-oliguric > 500 ml/day (Here urine is of poor quality i.e. contains little waste because blood is not filtered well, despite the fact that an adequate amount of urine is excreted (>500 ml)

78. Normal urine contains protein secreted by tubules, which is that-
   1. IgA
   2. Tom-Horsfall
   3. Urokinase
   4. All------------------------ans

Discussion- Commercially urokinase comes from urine only

**GIT+LIVER**

80. An 18yrs old male presents with haematemesis, he has history of marked fever for the past 14 days for which he was managed with drugs, moderate splenomegaly is present; most likely diagnosis-
   1. Duodenal ulcer
   2. Drug induced gastritis--------ans
   3. Esophageal varices
   4. None of the above

Discussion- Factor against diagnosis of portal hypertension--> no ascites, age of presentation (ref-harrison, P=1977, chap-302)

83. Initial Treatment of choice for Zollinger Ellison syndrome is-
   1. Octreotide
   2. Gastrectomy
   3. H2 blocker
   4. Proton pump inhibitor-----------------ans

Discussion-

Zollinger-Ellison syndrome-
- A non-beta islet cell tumor that produces gastrin and is associated with gastric acid hypersecretion and peptic ulcer disease
- The tumors are biologically malignant in 60% of cases
- Tumor size varies from 2 mm to 20 cm

Tumor distribution-
- Majority of gastrinomas occurred within the pancreas

Clinical Manifestations:
  A) Peptic ulcer
  B) Diarrhoea
  C) Presence of MEN I (P-429 ROAMS)

Diagnosis:
1. **Fasting gastrin level.** Fasting gastrin levels are usually <150pg/ml. Virtually all gastrinoma patients have a gastrin level > 150-200pg/ml
2. A BAO/MAO ratio >0.6 is highly suggestive of ZES, but a ratio <0.6 does not exclude the diagnosis
   - BAO- Basal Acid Output
   - MAO- Mean Acid Output
3. **Gastrin provocative tests**
   - Secretin injection test- A paradoxical (H/17 P-1869) increase in gastrin of > 200 pg within 15 min of secretin injection has a sensitivity and specificity of >90% for ZES
   - Calcium infusion test- less sensitive and specific than secretin test
4. **Tumor Localization**
   - Endoscopic ultrasound (EUS) permits imaging of the pancreas with a high degree of resolution (<5mm)
• Somatostatin analogue 111-In-pentetreotide (Octreoscan) with sensitivity and specificity rate of > 75%.
• For metastases: Abdominal CT scan, MRI, or Octreoscan

Rx:
• PPIs (Omeprazole) are the treatment of choice
• Surgery---> resection of tumor in pancreas (earlier total gastrectomy was primary operation)-->
  GT-87 Q-69

Therapy of metastatic endocrine:
  a. Streptozotocin
  b. 5-fluorouracil
  c. Doxorubicin
  d. IFN-alpha
  e. Hepatic artery embolization
  f. 111-In-pentetreotide

**Secretin**- It is produced in the S cells of the duodenum in the crypts of Lieberkühn. It stimulates the secretion of bile from the liver. It also increases watery bicarbonate solution from pancreatic duct epithelium. Secretin increases water and bicarbonate secretion from duodenal Brunner's glands in order to buffer the incoming protons of the acidic chyme. It also enhances the effects of cholecystokinin to induce the secretion of digestive enzymes and bile from pancreas and gallbladder, respectively. It counteracts blood glucose concentration spikes by triggering increased insulin release from pancreas, following oral glucose intake. It also reduces acid secretion from the stomach by **inhibiting gastrin** release from G cells. In addition, secretin stimulates pepsin secretion, which can help break down proteins in food digestion.

84. A 40 year old obese lady comes to you with jaundice. Her LFT report is as follows-

Bilirubin: 10 mg%
Direct: 6 mg%
SGOT: 12 mg%
SGPT: 14 mg%
Alkaline phosphatase: 14
Serum Protein: 6 gm%
Serum Albumin: 2 gm%
Serum Globulin: 4 gm%

**Most likely diagnosis**-
1. Acute hepatitis
2. Chronic liver disease-------------------ans(was written in classroom but ck from other sources)
3. Gall stone
4. Dubin Johnson syndrome

**Discussion**- Reversal of A:G ratio

88. Rx of hepatitis B-
1. Lamivudine
2. IF
3. Both ------------------ans
4. None

**Discussion**-

**Drugs used for treatment of hepatitis B are:**
1. Interferon alpha
2. Pergylated Interferon
3. Lamivudine
4. Adefovir dipivoxil
5. Entecavir (an oral guanosine analogue polymerase inhibitor)
Newer drugs for Hepatitis B
1. Telbivudine
2. Emtricitabine
3. Tenofovir
4. Clevudine

Treatment or hepatitis C --> Interferon alpha + Ribavirin (oral guanosine nucleoside)

92. In spider neavi which of following hormone is responsible for dialation of arteries-
1. Estrogen-----------------ans
2. DHEAS
3. Testosterone
4. Hepato Toxin

Discussion- (AIIMS Nov 08)

Treatment-
1. **Pentoxiphylline** in alcoholic hepatitis : In severe alcoholic hepatitis, oral pentoxifylline reduces mortality particularly from hepatorenal failure (Pentoxiphylline is an analogue of theophylline it is used to treat **intermittent claudication**)
2. **Glucocorticoids** is helpful in pts with severe alcoholic hepatitis. But no role in established cirrhosis

93. Hepatic-encephalopathy may be precipitated by all of the following except-
1. GI bleed
2. Barbiturates
3. Hyper kalemia----------------ans
4. Surgery

Discussion- (AIPG 07) **Acidosis good--> Hyperkalemia good**

Precipitating factors:

I. Increased nitrogen load:
   - **GI bleeding** (most common precipitating factor)
   - Excess dietary protein
   - Azotemia
   - Constipation

II. Electrolyte and metabolic abnormality: **Hypokalemia, Alkalosis, Hypovolumia, Hyponatremia**

III. Drugs: Sedatives, **Excess diuretics**

IV. Stress: Infection, Surgery

94. Which is not a feature of non-cirrhotic portal HT-
1. Splenomegaly
2. Hematemesis
3. Ascites--------------------------ans
4. All are seen

Discussion- (FAQ)

**NON CIRRHOTIC PORTAL FIBROSIS**: It is also called **Idiopathic portal hypertension**. Its incidence is very high in **India** accounts for 15% to 18% of all patients with portal hypertension

Clinical features:
- **Massive hematemesis**
- **Splenomegaly is present**
- Rarely patient develop encephalopathy
Ascites is uncommon
Jaundice and signs of liver cell failure are uncommon

Investigations:
- Normal liver function test
- Normal PT
- S. Proteins normal
- Portography-
  - Weeping willow appearance – sudden cut off peripheral portal vein branches
  - Tree in winter appearance – filling of a large number of collaterals with gross distortion of intrahepatic pattern
  - Splenic vein is dilated and tortuous

Treatment- In the lines of usual variceal bleed

96. False about hemochromatosis -
1. Hypogonadism
2. Hepatoma can occur
3. Pseudo Gout can occur
4. Desferrioxamine is treatment of choice-------------ans(Venesection)

Discussion- (AIPGE 2008) Hemochromatosis causes bronze diabetes.

Management- Main treatment consists of weekly venesection of 500 ml blood. The chelating agent deferoxamine (I/V or S/C) is indicated for patients with hemochromatosis or in those with secondary iron overload due to thalassemia who cannot tolerate phlebotomies. An oral chelator--> Deferasirox is new drug

97. Which of the following is implicated in pathogenesis of Crohn’s disease-
1. Mycobacterium avium paratuberculosis---------------ans
2. Mycoplasma
3. Rota virus
4. Streptococci

Discussion- (AIIMS May 08)

CD Infectious etiologies-

1. Bacterial
   - Salmonella
   - Shigella
   - Toxigenic Escherichia Coli
   - Campylobacter
   - Yersinia
   - Clostridium difficile
   - Gonorrhea
   - Chlamydia trachomatis

2. Mycobacterial--> M. avium

3. Parasitic-
   - Amebiasis
   - Isospora
   - Trichuris trichiura
   - Hookworm
   - Strongyloides

4. Viral
   - Cytomegalovirus
   - Herpes simplex
   - HIV

5. Fungal
   - Histoplasmosis
   - Candida
Atherosclerosis--> Chlamydia

Q. A 40 yr old cigarette smoker complains of epigastric pain, well localised non radiating and described as burning. The pain is partially relieved by eating. There is no weight loss. He has not used NSAID. The pain has gradually worsened over several months. The most sensitive way to make a specific diagnosis is-

1. Barium x-ray
2. Endoscopy
3. ECG
4. Serum gastrin

Discussion- (GT-87 Q-166)
Localised epigastric burning pain relieved by eating rerquires evaluation of peptic ulcer disease. Upper GI endoscopy provides the best sensitivity and specificity; barium swallow is less expensive, but is less accurate in defining mucosal disease. Patients with refractory or recurrent disease should have serum gastrin levels measured to rule out gastrinoma. A positive antibody test for H.pylori would only indicate previous exposure.

Q-22. The earliest manifestations of serious gram –ve infection may consist of a triad of signs that include-

1. Tachypnoea, hypotension, and an altered sensorium
2. Tachypnoea, hypotension and lactic acidosis
3. Thrombocytopenia, hypotension and lactic acidosis
4. Mild hyperventilation, respiratory alkalosis and an altered sensorium

Answer-4 (ck with medicine)

SIRS may have non infectious etiology

1. Temperature <36 or >38 degree centigrade
2. HR > 80 bpm
3. TLC >12000 or <4000
4. RR >20, PaCO2 >32
5. Sepsis: SIRS + Documented blood infection

Septicemia(Sepsis Syndrome ck differs from Bhatia notes): SEPSIS + One or more organ dysfunction

- Classical triad: Hyperventilation, respiratory alkalosis and an altered sensorium
  - For improved prognosis in Septicemia:
    - Tight glycemic controls(Imp: Since AIIMS has published this study)
    - Transferrin
    - Recombinant therapy: Drecotogin-alpha or Xigris or associated protein antibody