NBME FORM 7 BLOCK 1 ANSWERS

1. E. Rosuvastatin
- Statin decreases LDL
- Niacin increases HDL
- Fibrates decreases triaglycerides

HMG-coA reductase inhibitors are more effective for bringing the LDL to normal level....
They inhibit cholestrol precursor,Mevalonate
S/E...Rhabdomyolisis
reversible high level liver function test

Lovastatin,pravastatin,simvastatin,Atorvastatin,Ro sovastatin.....astatin

2. A. Bronchoconstriction

Zafirlucast,Montelucast.....Leukoterine receptor blockers prevent bronchoconstriction

3. B. “Do you know what kinds of foods are high in sodium?”

4. E. Primordial germ cell migration:

the tumor shows a haphazard arrangement of somatic tissues representing derivatives of ecto...,meso.. and endoderm.....it is a characteristic of germ cells.

Teratoma is derivative of primitive streak which involves process of germ cell migration.
- For number 4...This is a case of Sacrococcygeal teratoma..)....
- Sacrococcygeal teratoma is one of the extragonadal germ cell tumors ...
- These tumors can be found anywhere on the midline, particularly the retroperitoneum, the anterior mediastinum, the sacrococcyx, and the pineal gland. Other less common sites include the orbit, suprasellar area, palate, thyroid, submandibular region, anterior abdominal wall, stomach, liver, vagina, and prostate. The classic theory suggests that germ cell tumors (GCTs) in these areas are derived from local transformation of primordial germ cells misplaced during embryogenesis...which means defect in primordial germ cell migration

http://emedicine.medscape.com/article/278174-overview.....

My answer Is EE

F ... is for sacral agenesis... “Somite development”
D..is for spina bifida ... “neural tube closure”
C..is for neurocristopathies, which include conditions such as frontonasal dysplasia, Waardenburg-Shah syndrome, and DiGeorge syndrome, ...etc...”neural crest formation”
B..is clear...Hematopoiesis
A...is for cranial abnormalities.....”head mesenchyme proliferation”

5. A. Amitriptyline

Tricyclic antidepressant overdose....Tricyclics have a narrow therapeutic index, i.e., the therapeutic dose is close to the toxic dose.

The peripheral autonomic nervous system, central nervous system and the heart are the main systems that are affected following overdose.

Initial or mild symptoms typically develop within 2 hours and include tachycardia, drowsiness, a dry mouth, nausea and vomiting, urinary retention, confusion, agitation, and headache.
More severe complications include hypotension, cardiac rhythm disturbances, hallucinations, and seizures. Electrocardiogram (ECG) abnormalities are frequent and a wide variety of cardiac dysrhythmias can occur, the most common being sinus tachycardia and intraventricular conduction delay resulting in prolongation of the QRS complex and the PR/QT intervals.
Seizures, cardiac dysrhythmias, and apnea are the most important life threatening complications.[

6. B. Class II MHC molecule peptide loading

Professional APCs are very efficient at internalizing antigen, either by phagocytosis. The acidic compartments of macrophages are also responsible for the degradation of ingested micro-organisms) or by receptor-mediated endocytosis, and then displaying a fragment of the antigen, bound to a class II MHC molecule, on their membrane. The T cell recognizes and interacts with the antigen-class II MHC molecule complex on the membrane of the antigen-presenting cell.

7. D. Pyruvate carboxylase
- the problem is that she is hypoglycemia and then with an infusion w/ glucose, it helps.
- So the problem, is making glucose…hence, you need pyruvate carboxylase enzyme for gluconeogenesis.

A deficiency of pyruvate carboxylase can cause lactic acidosis as a result of lactate build up. Normally, excess pyruvate is shunted into gluconeogenesis via conversion of pyruvate into oxaloacetate, but because of the enzyme deficiency, excess pyruvate is converted into lactate instead.

As a key role of gluconeogenesis is in the maintenance of blood sugar, deficiency of pyruvate carboxylase can also lead to hypoglycemia

Pyruvate carboxylase is a mitochondria enzyme requiring biotin. It is activated by acetylCoA(from beta- oxidation. The product oxaloacetate(OAA), a citric acid cycle intermediate, cannot leave the mitochondria but is reduced to malate that can leave via a Malate shuffle. In the cytoplasm malate is reduced to (OAA).

Pyruvate carboxylase deficiency is an inherited disorder that causes lactic acid and other potentially toxic compounds to accumulate in the blood. High levels of these substances can damage the body's organs and tissues, particularly in the nervous system. Pyruvate carboxylase deficiency is a rare condition, with an estimated incidence of 1 in 250,000 births worldwide. This disorder appears to be much more common in some Algonkian Indian tribes in eastern Canada.
http://en.wikipedia.org/wiki/Pyruvat...ase_deficiency

8. A. Golgi complex

I-cell disease is an autosomal recessive disorder caused by a deficiency of GlcNAc phosphotransferase, which phosphorylates mannose residues to mannose-6-phosphate on N-linked glycoproteins in the Golgi apparatus within the cell.

Without mannose-6-phosphate to target them to the lysosomes, the enzymes are transported from the Golgi to the extracellular space, resulting in large intracellular inclusions of molecules requiring lysosomal degradation in patients with the disease .

The Golgi apparatus is unable to target the lysosomal protein (which is normal) to the lysosome. Without proper functioning of N-acetylglucosamine-1-phosphotransferase, a build up of substances occurs when enzymes are unable to travel inside of the lysosome

I- Cell Disease: (Inclusion cell disease). Inherited lysosomal storage disorder; failure of addition to mannose 6 phosphate to lysosome proteins(enzyme are secreted outside the cell instead of being targeted to the lysosme. Result in coarse facial future, clouded corneas, restricted join movement, and high plama levels of lysosomes. Often fatal childhood.
http://en.wikipedia.org/wiki/I-cell_disease

9. B. "I understand you concern. Let's talk more about your specific worries."

10. E. Neuromuscular junction

Myasthenia gravis is a chronic autoimmune neuromuscular disease characterized by varying degrees of weakness of the skeletal (voluntary) muscles of the body.

The hallmark of myasthenia gravis is muscle weakness that increases during periods of activity and improves after periods of rest. Certain muscles such as those that control eye and eyelid movement, facial expression, chewing, talking, and swallowing are often, but not always, involved in the disorder.

In myasthenia gravis, antibodies block, alter, or destroy the receptors for acetylcholine at the neuromuscular junction which prevents the muscle contraction from occurring

11. A. Decrease

Leuprolide: antagonist: pulsatile: treats prostate..meaning decrease LH/FSH.

http://en.wikipedia.org/wiki/Gonadot...easing_hormone

12. B. Centromere
a. Raynaud's phenomenon…CREST syndrome…anti-centromere antibody

Crest Syndrome: Calcifications, Raynaud's phenomenon, Esophageal dysmotility, Sclerodactyly, and Telangiectasia. Limited skin involvement, often confined to fingers and face. More benign clinical course. Associated with Anticentromere Antibody.

13. A. Acute inflammation

Acute pancreatitis

14. C. D. Parvovirus B19

Two of the sources say that the answer is D. Parvovirus B 19 (maybe because it has to do with sickle cell anemia hence the hemoglobin & hematocrit is low but then wouldn't you see sickle cell shape instead). Plus, parvovirus affect RBC precursors (reticulocytes), so, isn't the ret ct kinda normal (since this is the case, you know that the bone marrow is working and it is compensation. E. Pneumocystis jiroveci (formerly P.carinii) is usually seen in adults.

AIDS
Parvovirus B19 is a cause of chronic anemia in individuals who have AIDS. It is frequently overlooked. Treatment with erythropoetin or intravenous immunoglobulin have been helpful in some patients. The parvovirus infection may trigger an inflammatory reaction in AIDS patients who have just begun antiretroviral therapy.[14].

http://en.wikipedia.org/wiki/Parvovirus_B19

15. G. Parathorone-related protein

When associated with the lung, it often causes ectopic production of parathyroid hormone-related protein (PTHrP), resulting in hypercalcemia.

Squamous-cell carcinoma of the lung is more common in men than in women. It is closely correlated with a history of tobacco smoking, more so than most other types of lung carcinoma. It most often arises centrally in larger bronchi, and while it often metastasizes to locoregional lymph nodes (particularly the hilar nodes) early in its course, it generally disseminates outside the thorax somewhat later than other major types. Large tumors may undergo central necrosis, resulting in cavitation. A squamous-cell carcinoma is often preceded for years by squamous-cell metaplasia or dysplasia in the respiratory epithelium of the bronchi, which later transforms to carcinoma in situ. In carcinoma in situ, atypical cells may be identified by cytologic smear test of sputum, bronchoalveolar lavage or samples from endobronchial brushings. However, squamous-cell carcinoma in situ is asymptomatic and undetectable on X-ray radiographs. Eventually, it becomes symptomatic, usually when the tumor mass begins to obstruct the lumen of a major bronchus, often producing distal atelectasis and infection. Simultaneously, the lesion invades into the surrounding pulmonary substance. On histopathology, these tumors range from well-differentiated, showing keratin pearls and cell junctions, to anaplastic with only minimal residual squamous-cell features.
http://en.wikipedia.org/wiki/Squamous-cell_carcinoma

16. A. Alkaline phosphatase activity

Paget's disease may be diagnosed using one or more of the following tests:

Pagetic bone has a characteristic appearance on X-rays. A skeletal survey is therefore indicated.

http://upload.wikimedia.org/wikipedi...gets_skull.jpg

An elevated level of alkaline phosphatase in the blood in combination with normal calcium, phosphate, and aminotransferase levels in an elderly patient are suggestive of Paget's disease.
Bone scans are useful in determining the extent and activity of the condition. If a bone scan suggests Paget's disease, the affected bone(s) should be X-rayed to confirm the diagnosis

The pathogenesis of Paget's disease is described in 3 stages, which are:
i. Osteoclastic activity
ii. Mixed osteoclastic-osteoblastic activity
iii. Exhaustive (burnt out) stage

Initially, there is a marked increase in the rate of bone resorption at localized areas caused by large and numerous osteoclasts. These localized areas of osteolysis are seen radiologically as an advancing lytic wedge in long bones or osteoporosis circumscripta in the skull. The osteolysis is followed by a compensatory increase in bone formation induced by osteoblasts recruited to the area. This is associated with accelerated deposition of lamellar bone in a disorganized fashion. This intense cellular activity produces a chaotic picture of trabecular bone ("mosaic" pattern), rather than the normal linear lamellar pattern. The resorbed bone is replaced and the marrow spaces are filled by an excess of fibrous connective tissue with a marked increase in blood vessels, causing the bone to become hypervascular. The bone hypercellularity may then diminish, leaving a dense "pagetic bone," also known as burned-out Paget's disease.

17. A. Discuss comfort care with the patient's wife

all choices except A are artificial procedures that prolong his life.

18. D. Schistocytes

Normal lactate dehydrogenase is 25-90 U/L...this patient has 1000 U/L..definitely TTP

TTP: Thrombotic Thrombocytopenia Purpura: is a rare disorder of the blood-coagulation system, causing extensive microscopic thromboses to form in small blood vessels throughout the body (thrombotic microangiopathy). Most cases of TTP arise from inhibition of the enzyme ADAMTS13, a metalloprotease responsible for cleaving large multimers of von Willebrand factor (vWF) into smaller units. A rarer form of TTP, called Upshaw-Schülman syndrome, is genetically inherited as a dysfunction of ADAMTS13. If large vWF multimers persist there is tendency for increased coagulation.

http://en.wikipedia.org/wiki/Thrombo...openic_purpura

TPP: FA page 349) Symptoms: pentada of neurologic and renal symptoms, fever, thrombocytopenia, and microangiopathic hemolytic anemia. Lab: Schistocytes, increase LDH.

19. B. GM2-ganglioside

Tay sach disease: AR, Progresive neurodegeneration, developmental delay, cherry red spot on macula, lysosomal with onion skin , NO HEPATOMEGALY. Def. enzime: Hexosaminidase A; Accumulated substrate: G2 ganglioside. (FA page 111).

20. B. Smell

Olfatory CN I is the only CN without thalamic relay to cortex. Function: Smell (FA 413)

21. F. Increased volume of distribution

22. D. Pneumothorax

Pneumothorax is defined as the presence of air or gas in the pleural cavity, that is, in the potential space between the visceral and parietal pleura of the lung. The result is collapse of the lung on the affected side. Air can enter the intrapleural space through a communication from the chest wall (ie, trauma) or through the lung parenchyma across the visceral pleura.

Primary spontaneous pneumothorax (PSP) occurs in people without underlying lung disease and in the absence of an inciting event . In other words, air is present in the intrapleural space without preceding trauma and without underlying clinical or radiologic evidence of lung disease.

Risks factors for primary spontaneous pneumothorax (PSP) include the following:

•Smoking
•Tall, thin stature in a healthy person
•Marfan syndrome
•Pregnancy
•Familial pneumothorax
Blebs and bullae (sometimes called emphysematouslike changes or ELCs) are related to the occurrence of primary spontaneous pneumothorax.

Secondary spontaneous pneumothorax (SSP) occurs in people with a wide variety of parenchymal lung diseases, that is, these individuals have underlying pulmonary structural pathology (see the image below). Air enters the pleural space via distended, damaged, or compromised alveoli. Patients may present with more serious clinical symptoms and sequelae due to comorbid conditions.

Diseases and conditions associated with secondary spontaneous pneumothorax include the following:

•Chronic obstructive lung disease (COPD) or emphysema: Increased pulmonary pressure due to coughing with a bronchial plug of mucus or phlegm bronchial plug may play a role.
•Asthma
•Human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS) with PCP infection
•Necrotizing pneumonia
•Tuberculosis
•Sarcoidosis
•Cystic fibrosis
•Bronchogenic carcinoma or metastatic malignancy
•Idiopathic pulmonary fibrosis
•Inhalational and intravenous drug use (eg, marijuana, cocaine)[8]
•Interstitial lung diseases associated with connective tissue diseases
•Lymphangioleiomyomatosis
•Langerhans cell histiocytosis
•Severe acute respiratory syndrome (SARS): A reported 1.7% of SARS patients developed spontaneous pneumothorax.
•Thoracic endometriosis and catamenial pneumothorax
•Collagen vascular disease, including Marfan syndrome

23. C. Ganciclovir

Ganciclovir is an antiviral medication used to treat or prevent cytomegalovirus (CMV) infections
It is first phosphorylated to a deoxyguanosine triphosphate (dGTP) analogue. This competitively inhibits the incorporation of dGTP by viral DNA polymerase, resulting in the termination of elongation of viral DNA.

Adverse effects:
Ganciclovir is commonly associated with a range of serious haematological adverse effects. granulocytopenia, neutropenia, anaemia,

thrombocytopenia, fever, nausea, vomiting, dyspepsia, diarrhoea, increased serum creatinine and blood urea concentrations

Toxicity:

Ganciclovir is considered a potential human carcinogen, teratogen, and mutagen. It is also considered likely to cause inhibition of spermatogenesis. Thus, it is used judiciously and handled as a cytotoxic drug in the clinical setting

24. E. Mitral regurgitation

Holosystolic murmur is best heard at the apex in the left lateral decubitus position....Mitral Regurgitation .

Murmur:

Blowing holosystolic murmur
Heard best at the apex
Radiation to the axilla and inferior edge of left scapula.
Possible associated findings:

S2: wide physiologic splitting
S3

http://depts.washington.edu/physdx/heart/tech.html

25. D. Rest (systolic < diastolic) vs. Moderate exercise (systolic < diastolic)
- the cardiac cycle where by coronary arteries deliver oxygen to the myocardium is during diastole.....whether it is systolic or diastolic.....this is my assumption.
- no matter if the person is at rest or doing exercise, the coronary blood flow is always more during diastole. although the duration of flow is greatest during rest as oppose to exercise when the heart rate is faster and thus diastole is of less duration.

Coronary blood flow occurs mostly during diastole, because during systole the blood vessels within the myocardium are compressed. Increased heart rates, which reduce the time for diastole filling, can reduce the myocardial blood supply and cause ischemia.

Dynamic exercise increases coronary blood flow in proportion to the heart rate, with peak values during maximal exercise typically three to five times the resting level .

26. F. Surfactant

Severely premature infants may have underdeveloped lungs, because they are not yet producing their own surfactant. This can lead directly to respiratory distress syndrome, also called hyaline membrane disease, in the neonate.

To try to reduce the risk of this outcome, pregnant mothers with threatened premature delivery prior to 34 weeks are often administered at least one course of glucocorticoids, a steroid that crosses the placental barrier and stimulates the production of surfactant in the lungs of the fetus.

27. E. Increase B-hydroxybutyrate; increase osmality; increase anion gap

- Case of diabetic ketoacidosis...high glucose ...high ketones
- Betahydroxy butyrate and acetoacetate both increase.....
- Osmolarity =2 Serum Na+serum glucose /18+BUN/2.8 ...so if u increase glucose u increase
plasma osmolarity
- Anion Gap = Serum Na - (serum Chloride + Serum HCO3)...in the case of diabetic Ketoacidosis
- anions of acid (acetoacetate and beta hydroxybutyrate) replace HCO3 ...hence we only subtract Chloride from the Serum Na ...that makes the anion gap to increase....

n DKA
*beta-Hydroxybutyric acid is a ketone body....inc
*Due to loss of water.....inc. serum osmolality.
*The anion gap is the difference in the measured cations and the measured anions in serum, plasma, or urine. The magnitude of this difference (i.e. "gap") in the serum is often calculated in medicine when attempting to identify the cause of metabolic acidosis.

only Na+, Cl− and HCO3− (+/- K) are used when calculating the anion gap... healthy adults is 8-12 mEq/L

([Na+]) − ([Cl−]+[HCO3−])

a useful mnemonic to remember the high anion gap is MUDPILES (methanol, uremia, diabetic ketoacidosis, propylene glycol, isoniazid, lactic acidosis, ethylene glycol, salicylates).

28. D. Pseudomonas aeruginosa

It is a Gram-negative, aerobic, rod-shaped bacterium with unipolar motility and oxidase positive

P. aeruginosa secretes a variety of pigments, including pyocyanin (blue-green),
Because it thrives on most surfaces, this bacterium is also found on and in medical equipment, including catheters, causing cross-infections in hospitals and clinics. It is implicated in hot-tub rash.

29. E. Mitral valve prolapse

Marfan syndrome can lead to mitral valve prolapse (MVP)

30. A. Increase hemoglobin F; increase Hemoglobin A2: hemoglobin A ratio; increase reticulocyte count
- Patient has beta thalassemia
o Normal adult hemoglobin is alpha 2, beta 2, and alpha 2, gamma 2
o Increase HbA2 (>3.5%)
▪·You compare the ratio of Adult 2 with Adult 1
▪·Adult 2 is normal, hence, adult 2 has a higher normal adult hemoglobin than Adult 1
o Both major & minor = increase HbF (alpha 2; gamma 2)
▪·Adult 1 normally has alpha 2, beta 2
▪·Adult 2 normally has alpha 2, delta 2
▪·In beta thalassemia, you change beta 2 in adult 1 with HbF (alpha 2, gamma 2)
▪·So, now, the adult 1 has alpha 2, HbF…aka alpha 2, gamma 2
▪·Hence, you see an increase in HbF
o Reticulytes is normal/to increase because there is nothing wrong with the bone marrow so therefore, it doesn't the reticulocytes.
o embryonic hemoglobin: 2z, 2e
o transition hemoglobin: 2z, 2y
o fetal hemoglobin: 2alpha, 2y
o adult hemoglobin HbA1: 2 alpha, 2 beta
o adult 2: 2 alpha, 2 delta

Beta thalasemia: prevalent in mediterranea population.
Defect: point mutation in splicing sites and promoter sequences
Beta thalasemia minor (heterozygote)
1. beta chain in unproduced
2. usually asyntomptomatic
3. Dx confirmed by increase HbA2 (> 3.5%)
Beta thalasemia major: (Homozygote)
1. beta chain is absent- severe anemia requiring blood transfusion (2º hemochromatosis)
2. Marrow expansion (crew cut on skull x-ray)- skeletal defromities, chipmunk facies
Both major and minor - increase Hb F (FA page 343).

31. G. B2 adrenergic agonist

Beta 2 adrenergic agonist: Bronchodilation (relax bronchial smooth mucles)

32. C. Thyroid-stimulating hormone

TSH: Thyroid-stimulating hormone (also known as TSH or thyrotropin) is a peptide hormone synthesized and secreted by thyrotrope cells in the anterior pituitary gland, which regulates the endocrine function of the thyroid gland. TSH is the 1st step in evaluation of thyroid function.

33. B. Adenocarcinoma of the endometrium

Adenocarcinoma of the endometrium:
Carcinoma endometrial is the Most common gynecologic malignancy. peak occurrence at 55-65 year of age. Clinically presents with vaginal bleeding. Typically proceeded by endometrial hyperplasia. Risk factors include prolonged use of estrogen without progestins, obesity, diabetes, hypertension, nulliparity and late menopause. Increase myometrial invasion, decrease prognosis. (FA page 487)

34. C. Globus pallidus

Parkinson disease
Surgery and deep brain stimulation
Placement of an electrode into the brain. The head is stabilized in a frame for stereotactic surgery. Treating motor symptoms with surgery was once a common practice, but since the discovery of levodopa, the number of operations declined. Studies in the past few decades have led to great improvements in surgical techniques, so that surgery is again being used in people with advanced PD for whom drug therapy is no longer sufficient.[35] Surgery for PD can be divided in two main groups: lesional and deep brain stimulation (DBS). Target areas for DBS or lesions include the thalamus, the globus pallidus or the subthalamic nucleus.[35] Deep brain stimulation (DBS) is the most commonly used surgical

treatment. It involves the implantation of a medical device called a brain pacemaker, which sends electrical impulses to specific parts of the brain. DBS is recommended for people who have PD who suffer from motor fluctuations and tremor inadequately controlled by medication, or to those who are intolerant to medication, as long as they do not have severe neuropsychiatric problems.[29] Other, less common, surgical therapies involve the formation of lesions in specific subcortical areas (a technique known as pallidotomy in the case of the lesion being produced in the globus pallidus).[35]

35. A. Deep fibular (peroneal)

PED: peroneal; eversion; dorsiflexion

Deep Fibular nerve: Weakness inversion, Loss extension of the digits, loss dorsiflexion (foot drop), sensory loss on antero lateral leg and dorso of the foot, first web space. (Kaplan anatomy page 295 and 295)

36. B. Placenta accrete

Placeta accreta: An invasion of the myometrium which does not penetrate the entire thickness of the muscle. This form of the condition accounts for around 75% of all cases.

Placenta perccreta: The worst form of the condition is when the placenta penetrates the entire myometrium to the uterine serosa (invades through entire uterine wall). This variant can lead to the placenta attaching to other organs such as the rectum or bladder[1

37. C. Ileum

Crohn Disease: Also known as regional enteritis, is an inflammatory disease of the intestines that may affect any part of the gastrointestinal tract from mouth to anus, causing a wide variety of symptoms. It primarily causes abdominal pain, diarrhea (which may be bloody if inflammation is at its worst), vomiting, or weight loss,[1][2][3] but may also cause complications outside the gastrointestinal tract such as skin rashes, arthritis, inflammation of the eye, tiredness, and lack of concentration.[1]

Crohn's disease is thought to be an autoimmune disease, in which the body's immune system attacks the gastrointestinal tract, causing inflammation; it is classified as a type of inflammatory bowel disease. There is evidence of a genetic link to Crohn's disease, putting individuals with siblings afflicted with the disease at higher risk.[4] It is thought to have a large environmental component as evidenced by a higher incidence in western industrialized nations compared to other parts of the world. Males and females are equally affected. Smokers are two times more likely to develop Crohn's disease than.

http://en.wikipedia.org/wiki/Crohn's_disease.
Gross morphology: transmural inflamation, Cobblestone mucosa, creeping fat, bowell wall thickning, (string sign on barium swallow x- ray) linear, ulcers, fissures, fistulas.
Microscopic morphology: Noncaseating granulomasand lymphoid

38. B. 0.19

positive predictive value Positive (PPV): TP/ (TP+FP)
PPV= 70/(70+300)= 0.18 in this case aprox. 0.19

39. C. Membranous nephropathy

Membranous Glomerulonephritis: LM -diffuse capillary and GBM thickening. EM " spike and dome" appearance with subepithelial deposits. IF - granular.

Caused by drugs (penicillamine, Gold, NSAID). Most common cause of adult nephrotic syndrome.
The closely related terms membranous nephropathy[1] and membranous glomerulopathy[2] both refer to a similar constellation but without the assumption of inflammation.

Some patients may present as nephrotic syndrome with proteinuria, edema with or without renal failure. Others may be asymptomatic and may be picked up on screening or urinalysis as having proteinuria. A definitive diagnosis of membranous nephropathy requires a kidney biopsy.

40. A. Esctasy (3,4-methylenedioxymethamphetamine)

Intoxication for Amphetamine: Psychomotor agitation , impaired judgment, pupillary dilation, hypertension, tachycardia, euphoria, prolonged weakfulness sand attention, cardiac arrythmia, delusions, hallucinations, fever. (FA 448)

Amphetamine: Blocks of neurotransmiter uptake at the presynaptic nerve terminal

41. F. Tronchanteric bursa

- Trochanteric bursitis is characterized by painful inflammation of the bursa located just superficial to the greater trochanter of the femur. Patients typically complain of lateral hip pain, although the hip joint itself is not involved. The pain may radiate down the lateral aspect of the thigh.
- The term greater trochanteric pain syndrome (GTPS) is now being commonly substituted for trochanteric bursitis, because the inflammatory etiology of the pain is being refuted by current research, using ultrasonographic, magnetic resonance imaging (MRI) – based, and histologic evidence.
- Flexion or anteversion (140°): iliopsoas (with psoas major from vertebral column); tensor fascia latae, pectineus, adductor longus, adductor brevis, and gracilis. Thigh muscles acting as hip flexors: rectus femoris and sartorius.

42. E. X-linked recessive

Duchenne muscular dystrophy (DMD) is a recessive X-linked. The disorder is caused by a mutation in the dystrophin gene, located in humans on the X chromosome (Xp21). The dystrophin gene codes for the protein dystrophin, an important structural component within muscle tissue.

43. C. Medial collateral ligament

Medial collateral ligament" known as the tibial collateral ligament too.MCL strains and tears are fairly common in American football. Mostly the center and the guards are ones who get this injury, due to the grip trend on their cleats.

An MCL injury can be very painful and is caused by a valgus stress to a slightly bent knee, often when landing, bending or on high impact. Depending on the grade of the injury, the lowest grade (grade 1) can take between 2 and 10 weeks for the injury to fully heal. Recovery times for grades 2 and 3 are difficult to predict because of the amount of damage done can take weeks to several months. It is difficult to apply pressure on the injured leg for at least a few days.

44. A. They attributed the difference in success rates to chance alone

In order for it to be stagistical significance, you need the P value to be 0.05 aka 95%

But since the P value here is 0.3 aka 70%..it is only due to chance.

The P value for a study should be less than 0.05.

This value means that when we perform a study, the 95% of the time the observations are rite & 5 % of the time the observation are due to chance alone.

In this question the value of P is 0.3 which means that the probability that these observations are due to chance alone is 30%, which is not a statistically acceptable number.

45. E. St. John's wort

St John's wort has been shown to cause multiple drug interactions through induction of the cytochrome P450 enzyme CYP3A4, but also CYP2C9. This results in the increased metabolism of those drugs, resulting in decreased concentration and clinical effect.
Examples of drugs causing clinically-significant interactions with St John's wort
Class Drugs
antiretrovirals non-nucleoside reverse transcriptase inhibitors, protease inhibitors
benzodiazepines alprazolam, midazolam
hormonal contraception combined oral contraceptives
immunosuppressants calcineurin inhibitors, ciclosporin, tacrolimus
others, digoxin, methadone, omeprazole, phenobarbitol, theophylline, warfarin, levodopa, suboxone, Irinotecan

46. D. Increased osmolarity in the tubular lumen (urine)
o the boy has DM type 1
o in type 1 you have high glucose, no insulin
o high glucose will reabsorb all of the water into the cells, this pull water away from the tubule (urine) and you get hypertonic urine

http://en.wikipedia.org/wiki/Diabetic_ketoacidosis

Osmotic diuresis is increased urination caused by the presence of certain substances in the small tubes of the kidneys.[1] The excretion occurs when substances such as glucose enter the kidney tubules and cannot be reabsorbed (due to a pathological state or the normal nature of the substance). The substances cause an increase in the osmotic pressure within the tubule, causing retention of water within the lumen, and thus reduces the reabsorption of water, increasing urine output (ie. diuresis). The same effect can be seen in therapeutics such as mannitol, which is used to increase urine output and decrease extracellular fluid volume.
Substances in the circulation can also increase the amount of circulating fluid by increasing the osmolarity of the blood. This has the effect of pulling water from the interstitial space, making more water available in the blood and causing the kidney to compensate by removing it as urine. In hypotension, often colloids are used intravenously to increase circulating volume in themselves, but as they exert a certain amount of osmotic pressure, water is therefore also moved, further increasing circulating volume. As blood pressure increases, the kidney removes the excess fluid as urine.
Sodium, chloride, potassium are excreted in Osmotic diuresis, originating from Diabetes Mellitus (DM). Osmotic diuresis results in dehydration from polyuria and the classic polydipsia (excessive thirst) associated with DM.
http://en.wikipedia.org/wiki/Osmotic_diuresis

47. A. 312
- CPM ...stands for count per minute ..which is a measure of radioactivity. It is the number of atoms in a given quantity of radioactive material that are detected to have decayed in one minute.
- Mixed Lymphocyte Reaction... a test used to check class II compatibilty testing ...in this test lymphocytes from one individual being tested(donor) are irradiated so that they cant proliferate.. but will act as a stimulator cells for the presentation of MHC antigens ...the other individual cells (Recipient) are added to the culture ,and uptake of tritiated thymidine is used as an indicator of cell proliferation....if the MHC class II antigens are different ,proliferation will occur ..if they are the same, no proliferation will occurr....
- Proliferation is directly proportional to CPM
- high CPM .....high proliferation....MHC II different ...not compatible
- low CPM ...less proliferation...MHC II similar...less risk of rejection....compatible

MLR lymphocytes from the donor and recipient are mixed and allowed to react. If they are compatible there will not be stimulation and the lymphocyte may not proliferate, some lymphocytes will degenerate and the count will reduce, if they are not compatible the donor lymphocte will be stimulated and will start to proliferate with a increased count. You can see the only count that is smaller thean the host is 312.

if they are not compatible and the lymphocytes are activated and start proliferating, the donor lymphocytes will be outnumbered... the point is that you don't want the donor lymphocytes to proliferate at a higher rate than the guy's own...

http://books.google.com/books?id=EJXyPS-TGPUC&pg=RA1-PA56&lpg=RA1-PA56&dq=thymidine+incorporation+kidney+transplantation&source=bl&ots=nELLEsr3xz&sig=z-ZHxqqt0ufGTSrKW_fHRUzsAFg&hl=en&ei=Za2_Ta7iJI64hAfG04WuBQ&sa=X&oi=book_result&ct=result&resnum=6&sqi=2&ved=0CEYQ6AEwBQ#v=onepage&q=thymidine%20incorporation%20kidney%20transplantation&f=false

In this test, "stimulator" lymphocytes from a potential donor are first killed by irradiation and then mixed with live "responder" lymphocytes from the recipient; the mixture is incubated in cell culture to permit DNA synthesis, which is measured by incorporation of tritiated thymidine. The greater the amount of DNA synthesis in the responder cells, the more foreign are the class II MHC proteins of the donor cells. A large amount of DNA synthesis indicates an unsatisfactory "match"; i.e., donor and recipient class II (HLA-D) MHC proteins are not similar, and the graft is likely to be rejected. The best donor is, therefore, the person whose cells stimulated the incorporation of the least amount

48. B. Acidification causing increased ammonium ion excretion

Ammonium is basic, hence, to excrete it, you need to acidifiy it.

Lactulose inhibits bacterial ammonia production by acidifying the content of the bowel. It promotes growth of colonic flora. The growing biomass uses ammonia and nitrogen from amino acids to synthesise bacterial protein, which in turn inhibits protein degradation to NH3. Lactulose leads to less ammonia by inhibiting bacterial urea degradation and reduces colonic transit time, thus reducing the time available for ammonia production and expediting ammonia elimination.

49. D. Metaplasia of the esophageal epithelium
This is a case of GERD...with esophagitis for the last 8 months...
Gastroesophageal reflux disease (GERD) occurs when the amount of gastric juice that refluxes into the esophagus exceeds the normal limit, causing symptoms with or without associated esophageal mucosal injury (ie, esophagitis).
Pathogenesis of GERD...Transient relaxation of LES and Ineffective esophageal clearance of reflux material (bile acid and gastric juice)...hence the amount of acid that is secreted by the stomach doesnt have any significant effect ...also whether there is H pylori or not has no significance ...as well as whether the stomach is atrophic or not ...

Hence I go for metaplasia of the esophageal epithelium (given the 8 month history)

Barrett's esophagus is a condition in which the normal squamous epithelium of the esophagus has been replaced by an abnormal red columnar epithelium called specialized intestinal metaplasia. Specialized intestinal metaplasia is red, like normal stomach tissue, but does not look like stomach tissue under the microscope.

50. E. urolithiasis

- acidic, low protein in the fluid meaning that the (normal protein is 1.02); blood 4+ (lots of hemolysis...so think RBC cast); glucose is negative is it is not bacteria bc bacteria takes up glucose; bilirubin is negative so it is not the liver problem; leukocyte esterase negative so it can't be E. Coli;
o B. Nodular prostatic hyperplasia is not the answer because he is too you and he doesn't alkaline phosphtase increase
o C. RCC is not it because RCC is chronic...this guy has acute
o D. Schistomoasis...is a worm..so you should see a present of eosophil...and plus this patient glucose is negative. F

Urolithiasis is the condition where urinary calculi are formed in the urinary tract.

Most common kidney stons...Calcium Oxalate,Calcium phosphate,or both...Radiopaque
Oxalate crystal..result fromEthylen glycol

Secound most common....Ammonium Magnesium phosphate..Struvite....caused by infecction with Urease-positive bugs.....can form staghorn calculi

Uric acid....associated with hyperuricemia.....readiolucent

Cysteine...secondary to Cystinuria..Hexogonal shape.....faintly radiopaque...treat with alkalization

In some cases, kidney stones pass out of the body without producing any symptoms. Most kidney stones, however, do cause some symptoms. The most common symptom from kidney stones is pain. The pain from kidney stones is a result of the stone getting stuck on its way out.

If fever and chills accompany any of these symptoms, an infection may be present and medical attention is needed sooner rather than late.

Laboratory Testing:
Laboratory testing includes urinalysis to detect the presence of blood (hematuria) and bacteria (bacteriuria) in the urine.
Other tests include blood tests for creatinine (to evaluate kidney function), BUN and electrolytes to detect dehydration, calcium to detect hyperparathyroidism, and a complete blood count to detect infection.

X-rays: A standard x-ray of the kidney, ureter, and bladder, may be adequate as a first step for identifying many stones since most are visible on x-ray.

Ultrasound: This test uses high frequency sound waves to produce pictures. Ultrasounds can detect a dilated kidney and ureter caused by a stone lodged in the ureter. Ultrasound, however, cannot reliably detect all stones especially stones located outside the kidney. It is the preferred imaging method for kidney stone patients who are pregnant.

IVP (Intravenous Pyelogram): For an IVP, a special dye is injected into the patient's veins. The dye collects in the urinary system and produces a white shadow when an x-ray is taken. The dye allows to precisely locate the stone and to determine the condition of the kidneys and ureters. Most kidney stones can be precisely located using this procedure.

CT Scan (Computerized Tomography): This test uses a scanner and a computer to create images of the urinary system. A CT scan done to look for kidney stones does not use contrast material. It is the most common imaging test used today to evaluate a possible kidney stone attack.

Block 2

1. A. Continuous proliferation

Caspase-9 is an initiator caspase, encoded by the CASP9 gene.

The aspartic acid specific protease caspase-9 has been linked to the mitochondrial death pathway. It is activated during programmed cell death (apoptosis). Induction of stress signaling pathways JNK/SAPK causes release of cytochrome c from mitochondria and activation of apaf-1 (apoptosome), which in turn cleaves the pro-enzyme of caspase-9 into the active form.

2. A. Cryptosporidium parvum

Primary symptoms of C. parvum infection are acute, watery, and non-bloody diarrhoea. C. parvum infection is of particular concern in immunocompromised patients, where diarrhea can reach 10–15L per day. Other symptoms may include anorexia, nausea/vomiting and abdominal pain. Infection is caused by ingestion of sporulated oocysts transmitted by the fecal-oral route

Entamoeba histolytica is an anaerobic parasitic protozoan. The active (trophozoite) stage exists only in the host and in fresh loose feces; cysts survive outside the host in water, in soils, and on foods, especially under moist conditions on the latter.

http://upload.wikimedia.org/wikipedia/commons/c/cf/Trophozoites_of_Entamoeba_...rocytes.JPG

http://en.wikipedia.org/wiki/File:En...e_cycle-en.svg

Giardia lamblia is a flagellated protozoan parasite that colonizes and reproduces in the small intestine, causing giardiasis. The giardia parasite attaches to the epithelium by a ventral adhesive disc, and reproduces via binary fission. Giardiasis does not spread via the bloodstream, nor does it spread to other parts of the gastro-intestinal tract, but remains confined to the lumen of the small intestine.

http://upload.wikimedia.org/wikipedia/commons/0/08/Giardia_lamblia_SEM_869...8_lores.jpg

Giardia infection can occur through ingestion of dormant cysts in contaminated water, food, or by the faecal-oral route (through poor hygiene practices). The Giardia cyst can survive for weeks to months in cold water, and therefore can be present in contaminated wells and water systems, especially stagnant water sources such as naturally occurring ponds, storm water storage systems, and even clean-looking mountain streams.

http://en.wikipedia.org/wiki/File:Gi...e_cycle_en.svg

Strongyloides stercoralis is a nematode that can parasitize humans. The adult parasitic stage lives in tunnels in the mucosa of the small intestine.

Strongyloides stercoralis is a nematode that can parasitize humans. The adult parasitic stage lives in tunnels in the mucosa of the small intestine.

Many people infected are usually asymptomatic at first. Symptoms include dermatitis: swelling, itching, larva currens, and mild hemorrhage at the site where the skin has been penetrated. If the parasite reaches the lungs, the chest may feel as if it is burning, and wheezing and coughing may result, along with pneumonia-like symptoms (Löffler's syndrome). Eventually, the intestines could be invaded, leading to burning pain, tissue damage, sepsis, and ulcers. In severe cases, edema may result in obstruction of the intestinal tract as well as loss of peristaltic contractions.

Strongyloidiasis in immunocompetent individuals is usually an indolent disease. However, in immunocompromised individuals, strongyloidiasis can cause a hyperinfective syndrome (also called disseminated strongyloidiasis) due to the reproductive capacity of the parasite inside the host. This hyperinfective syndrome has a mortality rate of close to 90%.

http://en.wikipedia.org/wiki/File:St...aliz_larva.jpg

3. B. Cytotoxic T-lymphocytes

- B2 microglobublin is located on MHCI..and MHCI is associated with CD8 T cells
- β2 microglobulin is a component of MHC class I molecules, which are present on all nucleated cells (excludes red blood cells).

Mice models deficient for the β2 microglobulin gene have been engineered. These mice demonstrate that β2 microglobulin is necessary for cell surface expression of MHC class I and stability of the peptide binding groove.
In fact, in the absence of β2 microglobulin, very limited amounts of MHC class I (classical and non-classical) molecules can be detected on the surface. In the absence of MHC class I, CD8 T cells cannot develop. (CD8 T cells are a subset of T cells involved in the development of acquired immunity.)Low levels of β2 microglobulin can indicate non-progression of HIV.

Levels of beta-2 microglobulin can be elevated in multiple myeloma and lymphoma, though in these cases primary amyloidosis (amyloid light chain) and secondary amyloidosis (Amyloid associated protein) are more common

4. B. 1, 25-dihydroxycholecalciferol

Calcitriol , also called 1,25-dihydroxycholecalciferol or 1,25-dihydroxyvitamin D3, is the hormonally active form of vitamin D with three hydroxyl groups.
It increases the level of calcium (Ca^{2+}) in the blood by
(1) increasing the uptake of calcium from the gut into the blood,
(2) decreasing the transfer of calcium from blood to the urine by the kidney, and
(3) increasing the release of calcium into the blood from bone.

Calcitriol is produced in the cells of the proximal tubule of the nephron in the kidneys by the action of 25-hydroxyvitamin D3 1-alpha-hydroxylase

This is a case of secondary hyperparathyroidism: decrease serum calcium, increase serum phosphate, increase PHT, decrease Vitamin D

5. E. Diagnostic test: CSF Polymerase chain reaction; Virus: herpes simplex

consistent use of CSF-PCR for HSV serology established a diagnosis in the majority of acute aseptic meningitis patients.

6. C. Left gastric
- FA pg. 300

Esophageal varices are extremely dilated sub-mucosal veins in the lower esophagus. They are most often a consequence of portal hypertension, commonly due to cirrhosis; patients with esophageal varices have a strong tendency to develop bleeding.

The majority of blood from the esophagus is drained via the esophageal veins, which carry deoxygenated blood from the esophagus to the azygos vein, which in turn drains directly into the superior vena cava. These veins have no part in the development of esophageal varices.

The remaining blood from the esophagus is drained into the superficial veins lining the esophageal mucosa, which drain into the coronary vein (left gastric vein), which in turn drains directly into the portal vein. These superficial veins (normally only approximately 1mm in diameter) become distended up to 1–2 cm in diameter in association with portal hypertension.

http://upload.wikimedia.org/wikipedia/commons/b/b6/Esophageal_varices..._-_wale.jpg

7. E. Increased plasmin generation

- Increase plasmin to increase fibrinolysis (to degrade fibrin = bleeding)
- normal coagulation is disrupted and abnormal bleeding occurs from the skin (e.g. from sites where blood samples were taken), the gastrointestinal tract, the respiratory tract and surgical wounds. The small clots also disrupt normal blood flow to organs (such as the kidneys), which may malfunction as a result.

The activation of the coagulation cascade yields thrombin that converts fibrinogen to fibrin; the stable fibrin clot being the final product of

hemostasis. The fibrinolytic system then functions to break down fibrinogen and fibrin. Activation of the fibrinolytic system generates plasmin (in the presence of thrombin), which is responsible for the lysis of fibrin clots. The breakdown of fibrinogen and fibrin results in polypeptides called fibrin degradation products (FDPs) or fibrin split products (FSPs). In a state of homeostasis, the presence of plasmin is critical, as it is the central proteolytic enzyme of coagulation and is also necessary for the breakdown of clots, or fibrinolysis.

8. B. p53

p53 is a tumor suppressor protein that in humans is encoded by the TP53 gene. p53 is important in multicellular organisms, where it regulates the cell cycle and, thus, functions as a tumor suppressor that is involved in preventing cancer

p53 has many mechanisms of anticancer function, and plays a role in apoptosis, genomic stability, and inhibition of angiogenesis. In its anti-cancer role, p53 works through several mechanisms:

It can activate DNA repair proteins when DNA has sustained damage.
It can induce growth arrest by holding the cell cycle at the G1/S regulation point on DNA damage recognition (if it holds the cell here for long enough, the DNA repair proteins will have time to fix the damage and the cell will be allowed to continue the cell cycle).
It can initiate apoptosis, the programmed cell death, if DNA damage proves to be irreparable.

If the TP53 gene is damaged, tumor suppression is severely reduced. People who inherit only one functional copy of the TP53 gene will most likely develop tumors in early adulthood, a disease known as Li-Fraumeni syndrome. The TP53 gene can also be damaged in cells by mutagens (chemicals, radiation, or viruses), increasing the likelihood that the cell will begin decontrolled division. More than 50 percent of human tumors contain a mutation or deletion of the TP53 gene.

The question has nothing to do with the stem itself, so forget about MRI, biopsy, etc.
The question is: Which one of the answer choices is different from the others?
All except p53 are proto-oncogenes.
So their inhibition would be beneficial and it wouldn't be expected in a tumor.
Inhibiton of P53, on the other hand, is consistent with the tumor scenario.
Therefore, the answer should be choice B. (As a side node, MDM2 gene mutations, which codes a protein inhibitory of P54), is common in astrocytomas
"impaired activity" of either P53 or Rb leads the cell cycle not regulated...so u get cancer
so(B)

Mutation of the p53 tumor suppressive peptide is the most usual suspect underlying almost any malignancy. In particular, it is evident in almost 2/3 of pt's suffering from astrocytoma.

9. A. Angiotensn II

The renin-angiotensin system (RAS) or the renin-angiotensin-aldosterone system (RAAS) is a hormone system that regulates blood pressure and water (fluid) balance.

When blood volume is low, juxtaglomerular cells in the kidneys secrete renin. Renin stimulates the production of angiotensin I, which is then converted to angiotensin II. Angiotensin II causes blood vessels to constrict, resulting in increased blood pressure. Angiotensin II also stimulates the secretion of the hormone aldosterone from the adrenal cortex. Aldosterone causes the tubules of the kidneys to increase the reabsorption of sodium and water into the blood. This increases the volume of fluid in the body, which also increases blood pressure.
If the renin-angiotensin-aldosterone system is too active, blood pressure will be too high. There are many drugs that interrupt different steps in this system to lower blood pressure. These drugs are one of the main ways to control high blood pressure (hypertension), heart failure, kidney failure, and harmful effects of diabetes.[2][3
Activation

The system can be activated when there is a loss of blood volume or a drop in blood pressure (such as in hemorrhage). Alternatively, a decrease in plasma NaCl concentration will stimulate the macula densa to release renin.
1. If the perfusion of the juxtaglomerular apparatus in the kidney's macula densa decreases, then the juxtaglomerular cells release the enzyme renin.
2. Renin cleaves a zymogen, an inactive peptide, called angiotensinogen, converting it into angiotensin I.
3. Angiotensin I is then converted to angiotensin II by angiotensin-converting enzyme (ACE)[4] which was thought to be found mainly in lung capillaries. However new evidence suggests the ACE is found in all blood vessel endothelial cells.[5]
4. Angiotensin II is the major bioactive product of the renin-angiotensin system, binding to receptors on intraglomerular mesangial cells, causing these cells to contract along with the blood vessels surrounding them and causing the release of aldosterone from the zona glomerulosa in the adrenal cortex. Angiotensin II acts as an endocrine, autocrine/paracrine, and intracrine hormone.

http://en.wikipedia.org/wiki/Renin-angiotensin_system

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so when blood volum is become higher by receiving 1 liter fluid.....>dec. renin-angiotensin aldosteron system

10. E. Osteoclasts

Osteopetrosis,, also known as marble bone disease is an extremely rare inherited disorder whereby the bones harden, becoming denser,

Normal bone growth is achieved by a balance between bone formation by osteoblasts and bone resorption (break down of bone matrix) by osteoclasts.

In osteopetrosis, the number of osteoclasts may be reduced, normal, or increased. Most importantly, osteoclast dysfunction mediates the pathogenesis of this disease.

deficiency of carbonic anhydrase in osteoclasts is noted. The absence of this enzyme causes defective hydrogen ion pumping by osteoclasts and this in turn causes defective bone resorption by osteoclasts, as an acidic environment is needed for dissociation of calcium hydroxyapatite from bone matrix. Hence, bone resorption fails while its formation persists. Excessive bone is formed.

Osteopetrosis :
Calcium....unaffected
Phosphate...unaffected
Alkalin phosphatase....elevated
Parathyroid Hormone....unaffected

Symptoms:

Pain
Frequent fractures, especially of the long bones, which often do not heal
Nerve compression, leading to headache, blindness, deafness
Hematological difficulties, including anemic thrombocytopenia, leukopenia
Enlarged spleen
Osteomyelitis
Frontal bossing of the skull
Unusual dentition, including malformed and unerrupted teeth
Infection
Bleeding
Stroke

The probable diagnosis is osteopetrosis...frontal bossing is not the major factor to diagnosis rickets.h/o frequent fractures more common in osteopetrosis.and long bones always have broadened metaphysis.also have increased bone density and thick bone cortex.
we cant find increase bone density and broadened metaphysis n diaphysis of long bones in rickets.
so...decreased osteoclastic function is the best ans..

11. D. Y and W

Respiratory Acidosis: Ph ↓, PO2 ↓, PCO2 ↑, represented by the letters Y – W

Barbiturate overdose
Poisoning by barbiturates
Classification and external resources
ICD-10 T42.3
eMedicine med/207
=
Phenobarbital causes a "depression" of the body's systems, mainly the central and peripheral nervous systems; thus, the main characteristic of phenobarbital overdose is a "slowing" of bodily functions, including decreased consciousness (even coma), bradycardia, bradypnea, hypothermia, and hypotension (in massive overdoses). Overdose may also lead to pulmonary edema and acute renal failure as a result of shock.

The electroencephalogram of a person with phenobarbital overdose may show a marked decrease in electrical activity, to the point of mimicking brain death. This is due to profound depression of the central nervous system, and is usually reversible.[24]

http://en.wikipedia.org/wiki/Phenobarbital

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decreased respiration (hypoventilation) causes increased blood carbon dioxide and decreased o2 and pH (a condition generally called acidosis).

12. D. Major depressive disorders

13. D. Negotiate a plan to give the tea when the child's gastrointestinal tract will allow administration of liquids.

14. B. Ear

Gout: Asymetric joint distribution. Joint is swollen, red, and painful. classic manifestation is painfull MTP joint of the big toe (Podagra). Tophus formation often on external ear, oleocranon bursa, or achiles tendon. Acute attack tends to occur after a large meal or alcohol consumption (alcohol metabolism compete for same excretion sites in the kidney as uric acid, causing decrease uric acid secretion and subsequent buildup in blood.
Finding: Precipitation of monosodic ureate crystals into joints due to hyperuricemia, which can be caused by Lesch Nyhan Syndrome, PRPP excess....
Crystals are needle shaped birefringent= yellow under parallel light
TTO: Colchicine, NSAIDs, probenecid, allopurinol. FA page 380

15. A. Gain of stabilizing hydrophobic interactions in the deoxygenated form of hemoglobin S

1-Hemoglobin is an assembly of four globular protein subunits(polypeptide chains)....
2- Each subunit is composed of a protein chain tightly associated with a non-protein heme group.
3-Each protein chain arranges into a set of alpha-helix structural segments connected together in a globin fold arrangement,
4.Adult hemoglobin is made of 2 alpha and 2 beta chains
5-These four polypeptide chains are bound to each other and stabilized by
a- salt bridges...which is a noncovalent bonding...due to interaction between anionic carboxylate (RCOO-) and cationic ammonium (RNH3+) in the amino acids
b-hydrogen bonds,
c- hydrophobic interactions....oil and water do not combine because of hydrophobic interaction ...and hydrophobic interaction is a property of nonpolar molecules and this interaction is also used in the case of protein folding where by most folded proteins have a hydrophobic core in which side chain packing stabilizes the folded state, and charged or polar side chains on the solvent-exposed surface where they interact with surrounding water molecules.
6-the hydrophobic effect is important to understand the structure of proteins that have hydrophobic amino acids, such as alanine, valine, leucine, isoleucine, phenylalanine, and methionine grouped together with the protein...and hydrophobic interaction is between individual aminoacids
7-Ordinarily, the hemoglobin molecules exist as single, isolated units in the red cell, whether they have oxygen bound or not...
8-Sickle hemoglobin exists as isolated units in the red cells when they have oxygen bound.
9-When sickle hemoglobin releases oxygen in the peripheral tissues, however, the molecules tend to stick together and form long chains or polymers

Let us see what happens in sickle cell anemia...glutamic acid (hydrophillic) is replaced by hydrophobic valine (which increases the number of hydrophobic amino acids ...which in turn increases hydrophobic interactions) ..and these hydrophobic interactions stabilize the polymerized sickle hemoglobin....

16. D. Optic tract/visual cortex

significant functional aspects of the occipital lobe is that it contains the primary visual cortex and is the part of the brain where dreams come from.

A. Caudate nucleus
B. Primary motor cortex area
C. Thalamus
E. Posterior Limb

Homonymous hemianopsia can be congenital, but is usually caused by brain injury such as from stroke, trauma, tumors, infection, or following surgery.
Vascular and neoplastic (malignant or benign tumours) lesions from the optic tract, to visual cortex can cause a contralateral homonymous hemianopsia. Injury to the right side of the brain will affect the left visual fields of each eye. The more posterior the cerebral lesion, the more symmetric (congruous) the homonymous hemianopsia will be. For example, a person who has a lesion of the right optic tract will no longer see objects on his left side. Similarly, a person who has a stroke to the right occipital lobe will have the same visual field defect, usually more congruent between the two eyes, and there may be macular sparing. A stroke on the right side of the brain (especially parietal lobe), in addition to producing a homonymous hemianopsia, may also lead to the syndrome of hemispatial neglect.

17. E. Schizoid

Schizoid personality disorder (SPD) is a personality disorder characterized by a lack of interest in social relationships, sometimes sexually apathetic, a tendency towards a solitary lifestyle, secretiveness, and emotional coldness. SPD is not the same as schizophrenia, although they share some similar characteristics such as detachment or blunted affect and there is increased prevalence of the disorder in families with schizophrenia.

18. C. Gardner syndrome

Gardner syndrome, also known as familial colorectal polyposis, is an autosomal dominant form of polyposis characterized by the presence of multiple polyps in the colon together with tumors outside the colon. The extracolonic tumors may include osteomas of the skull, thyroid cancer, epidermoid cysts, fibromas and sebaceous cysts, as well as the occurrence of desmoid tumors in approximately 15% of affected individuals. The countless polyps in the colon predispose to the development of colon cancer; if the colon is not removed, the chance of colon cancer is considered to be very significant. Polyps may also grow in the stomach, duodenum, spleen, kidneys, liver, mesentery and small bowel. In a small number of cases, polyps have also appeared in the cerebellum. Cancers related to GS commonly appear in the thyroid, liver and kidneys.
At this time, there is no cure, and in its more advanced forms, it is considered a terminal diagnosis with a life expectancy of 35–45 years; treatments are surgery and palliative care, although some chemotherapy has been tried with limited success.

Gardner syndrome is now known to be caused by mutation in the APC gene located in chromosome 5q21 (band q21 on chromosome 5). This is the same gene as is mutant in familial adenomatous polyposis (FAP), a more common disease that also predisposes to colon cancer.

19. E. Uterine artery

The uterine artery usually arises from the anterior division of the internal iliac artery. It travels to the uterus, crossing the ureter anteriorly, reaching the uterus by traveling in the cardinal ligament.
It travels through the parametrium of the inferior broad ligament of the uterus.
It commonly anastomoses (connects with) the ovarian artery.
The uterine artery is the major blood supply to the uterus and enlarges significantly during pregnancy.

http://en.wikipedia.org/wiki/File:Gray589.png

20. D. Papillary transitional cell carcinoma with invasion into detrusor muscle

Transitional cell carcinoma (TCC, also urothelial cell carcinoma or UCC) is a type of cancer that typically occurs in the urinary system: the kidney, urinary bladder, and accessory organs. It is the most common type of bladder cancer and cancer of the ureter, urethra, and urachus; it is the second most common type of kidney cancer.
TCC arises from the transitional epithelium, a tissue lining the inner surface of these hollow organs.
TCCs are often multifocal, with 30-40% of patients having more than one tumour at diagnosis. The pattern of growth of TCCs can be papillary, sessile (flat) or carcinoma-in-situ (CIS).
The most common site of TCC metastasis outside the pelvis is bone (35%); of these bone metastases, 40% are in the spine.
When the term "urothelial" is used, it specifically refers to a carcinoma of the urothelium, meaning a TCC of the urinary system.
http://en.wikipedia.org/wiki/File:Bladder_urothelial_carcinoma_(1)_pT1.JPG
http://en.wikipedia.org/wiki/File:Blasentumor.jpg

21. B. Measurement of serum C-peptide concentration

Factitious Hypoglycemia (self injection of insulin).
Glucose .. dec.
insulin .. Inc
C peptide ... dec
No ketoacidosis.

C. P. serves as an important linker between the A- and the B- chains of insulin and facilitates the efficient assembly, folding, and processing of insulin in the endoplasmic reticulum. Equimolar amounts of C-peptide and insulin are then stored in secretory granules of the pancreatic beta cells and both are eventually released to the portal circulation. Initially, the sole interest in C-peptide was as a marker of insulin secretion and has as such been of great value in furthering the understanding of the pathophysiology of type 1 and type 2 diabetes. During the past decade, however, C-peptide has been found to be a bioactive peptide in its own right, with effects on microvascular blood flow and tissue health.

C-peptide should not be confused with c-reactive protein or Protein C.

Newly diagnosed diabetes patients often get their C-peptide levels measured as a means of distinguishing type 1 diabetes and type 2 diabetes. C-peptide levels are measured instead of insulin levels because insulin concentration in the portal vein ranges from two to ten times higher than in the peripheral circulation. The liver extracts about half the insulin reaching it in the plasma, but this varies with the nutritional state. The pancreas of patients with type 1 diabetes is unable to produce insulin and therefore they will usually have a decreased level of C-peptide, whereas C-peptide levels in type 2 patients are normal or higher than normal. Measuring C-peptide in patients injecting synthetic insulin can help to determine how much of their own natural insulin these patients are still producing, of if they produce any at all.

C-peptide is also used for determining the possibility of gastrinomas associated with Multiple Endocrine Neoplasm syndromes (MEN 1). Since a significant number of gastrinomas are associated with MEN involving other hormone producing organs (pancreas, parathyroids, and pituitary), higher levels of C-peptide together with the presence of a gastrinoma suggest that organs besides the stomach may harbor neoplasms.
C-peptide levels are checked in women with Polycystic Ovarian Syndrome (PCOS) to determine degree of insulin resistance.

Both excess body weight and a high plasma concentration of C-peptide predispose men with a subsequent diagnosis of prostate cancer to an increased likelihood of dying of the disease, according to the results of a long-term survival analysis reported in the October 6, 2008

22. B. Gastrointestinal
....the patient is exposed to high levels of radiation and this causes acute radiation syndrome

Stages of Acute radiation Syndrome
1) prodrome...nausea, vomiting, anorexia, fatigue, diarrhea, abdominal cramping, and dehydration which are GIT Symptom
2) clinical latency,
3) manifest illness, and
4) recovery or death

my answer is GIT

here is the link http://emedicine.medscape.com/article/834015-overview

23. D. Prolactin

The pituitary stalk (also known as the infundibular stalk or simply the infundibulum) is the connection between the hypothalamus and the posterior pituitary.

It carries axons from the magnocellular neurosecretory cells of the hypothalamus down to the posterior pituitary where they release their hormones into the blood.

This connection is called the hypothalamohypophyseal tract, and is responsible for the release of oxytocin and antidiuretic hormone.....

All choices except Oxytocin are hormones of anterior pituitary

24. D. Mean number of procedures performed: 13; Standard deviation: 5.1

In the question we are asked about heterogeneity(variability)

-The distances between the scores and the mean (Xi-M) are called deviations
-The greater the variety or heterogeneity of the scores, the greater the deviations
-If the scores were clustered around the mean, the deviations would be small, but they would increase as the scores became more spread out or more varied.

-And one way to measure variability (heterogenous population in this case) is by calculating the value of the Standard Deviation ..

-Generally SD - is an index of variability: the SD increases in value as the distribution becomes more variable. The less the variability in the distribution, the lower the value of the SD.

In short when we get high SD that means high variabilty (high heterogenous population in this case)....low SD means low variabilty (less hetrogenous population)...

So to check variabilty(hterogeneity) ...compare the standard deviation values not the mean of the experiment...

25. B. Midbrain

Light in either retina sends a signal via CNII to pretectal nuclei in MIDBRAIN that activate bilateral EDINGER-WESTPHAL nuclei;pupil contract bilateraly

Cranial nerve nuclei:

CN III,IV.....Midbrain
....V,VI,VII,VIII...Pons
....IX,X,XI,XII....Medulla

26. A. Coxackievirus

Coxsackievirus is a virus that belongs to a family of non enveloped linear positive-sense ssRNA viruses, Picornaviridae and the genus Enterovirus, which also includes poliovirus and echovirus.

Coxsackieviruses are divided into group A and group B viruses

Group A coxsackieviruses tend to infect the skin and mucous membranes, causing herpangina, acute hemorrhagic conjunctivitis (AHC), and hand, foot and mouth (HFM) disease.

Both group A and group B coxsackieviruses can cause nonspecific febrile illnesses, rashes, upper respiratory tract disease, and aseptic meningitis.

Group B coxsackieviruses tend to infect the heart, pleura, pancreas, and liver, causing pleurodynia, myocarditis, pericarditis, and hepatitis (inflammation of the liver not related to the hepatotropic viruses).

Coxsackie B infection of the heart can lead to pericardial effusion. Muffled heart sounds and pulsus paradoxus are signs of this.

27. D. Molluscum contagiosum

Molluscum contagiosum (MC) is a viral infection of the skin or occasionally of the mucous membranes. It is caused by a DNA poxvirus called the molluscum contagiosum virus (MCV). MCV has no animal reservoir, infecting only humans.

The virus commonly spreads through skin-to-skin contact. This includes sexual contact or touching or scratching the bumps and then touching the skin. Handling objects that have the virus on them (fomites), such as a towel, can also result in infection. Molluscum contagiosum is contagious until the bumps are gone-which, if untreated, may be up to 6 months or longer.

Molluscum contagiosum lesions are flesh-colored, dome-shaped, and pearly in appearance. They are often 1–5 millimeters in diameter, with a dimpled center. They are generally not painful, but they may itch or become irritated. Picking or scratching the bumps may lead to further infection or scarring.

28. E. Germline inactivation of the BRCA-1gene

BRCA1 is a human tumor suppressor gene that produces a protein called breast cancer type 1 susceptibility protein.
BRCA1 is expressed in the cells of breast and other tissue, where it helps repair damaged DNA, or destroy cells if DNA cannot be repaired. If BRCA1 itself is damaged, damaged DNA is not repaired properly and this increases risks for cancers.
Certain variations of the BRCA1 gene lead to an increased risk for breast cancer.

Women with an abnormal BRCA1 or BRCA2 gene have up to an 60% risk of developing breast cancer by age 90; increased risk of developing ovarian cancer is about 55% for women with BRCA1 mutations and about 25% for women with BRCA2 mutations.

29. D. Hearing loss

Vestibulocochlear nerve....VIII....hearing and balance

30. C. Rapid emptying of hyperosmolar chime into the small bowel

The dumping syndrome is Most people are unable to tolerate certain foods after gastric bypass, especially foods with high sugar or fat content. Eating these foods can cause the “dumping syndrome,” which may cause nausea and vomiting, diarrhea, a bloated feeling, dizziness and sweating.

31. D. Porphobilinogen

Acute intermittent porphyria (AIP) is a rare autosomal dominant.
Under normal circumstances, heme synthesis begins in the mitochondrion, proceeds into the cytoplasm, and finishes back in the mitochondrion. However, without porphobilinogen deaminase, a necessary cytoplasmic enzyme, heme synthesis cannot finish, and the metabolite porphobilinogen accumulates in the cytoplasm.
Additional factors must also be present such as hormones, drugs, and dietary changes that trigger the appearance of symptoms. Symptoms of AIP may include abdominal pain, constipation, and muscle weakness.
Patients with AIP are commonly misdiagnosed with psychiatric diseases. Subsequent treatment with anti-psychotics increases the accumulation of porphrobiliogen, thus aggravating the disease enough that it may prove fatal.

32. D. Release of acetylcholine into the primary synaptic cleft

Botulinum toxin is a protein produced by the bacterium Clostridium botulinum, and is extremely neurotoxic
The heavy chain of the toxin is particularly important for targeting the toxin to specific types of axon terminals. The toxin must get inside the axon terminals in order to cause paralysis. Following the attachment of the toxin heavy chain to proteins on the surface of axon terminals, the toxin can be taken into neurons by endocytosis. The light chain is able to cleave endocytotic vesicles and reach the cytoplasm. The light chain of the toxin has protease activity. The type A toxin proteolytically degrades the SNAP-25 protein, a type of SNARE protein. The SNAP-25 protein is required for vesicle fusion that releases neurotransmitters from the axon endings (in particular Acetylcholine).[58] Botulinum toxin specifically cleaves these SNAREs, and so prevents neuro-secretory vesicles from docking/fusing with the nerve synapse plasma membrane and releasing their neurotransmitters.

33. E

The spinothalamic tract is a sensory pathway originating in the spinal cord. It transmits information to the thalamus about pain, temperature, itch and crude touch. The pathway decussates at the level of the spinal cord, rather than in the brainstem like the posterior column-medial lemniscus pathway and corticospinal tract.
The cell bodies of neurons that make up the spinothalamic tract are located in the spinal ganglia. These neurons receive input from sensory fibers that innervate the skin and internal organs.
A. Left Dorsal columns (pressure, vibration, touch and proprioception)
C. Left Lateral Corticospinal tract (voluntary motor)
E. Left Spinothalamic tract: pain & temperature

34. E. Ret

Multiple endocrine neoplasia type 2 (also known as "Pheochromocytoma and amyloid producing medullary thyroid carcinoma" "PTC syndrome," and "Sipple syndrome" is a group of medical disorders associated with tumors of the endocrine system. The tumors may be benign or malignant (cancer). They generally occur in endocrine organs (e.g. thyroid, parathyroid, and adrenals), but may also occur in endocrine tissues of organs not classically thought of as endocrine.
Most cases of MEN2 derive from a variation in the RET proto-oncogene, and are specific for cells of neural crest origin.

The protein produced by the RET gene plays an important role in the TGF-beta (transforming growth factor beta) signaling system. Because the TGF-beta system operates in numerous tissues throughout the body, variations in the RET gene can have effects in numerous tissues throughout the body.
MEN2 generally results from a gain-of-function variant of a RET gene

35. E. T-lymphocyte mediated immunity

Sensitized T lymphocytes encounter antigen and then release lymphokines (leads to macrophage activation. no antibody involved.
http://en.wikipedia.org/wiki/File:T_cell_activation.png

legionella is a facultative intracellular microbe

intracellular microbes need t cell response

36. E. Suspensory (Cooper) ligaments

The suspensory ligaments of Cooper play an important role in the change in appearance of the breast that often accompanies the development of inflammatory carcinoma of the breast in which blockage of the local lymphatic ducts causes swelling of the breast. Because the skin remains tethered by the suspensory ligaments of Cooper, it takes on a dimpled appearance reminiscent of the peel of an orange (peau d'orange). Carcinomas can also decrease the length of Cooper's ligaments leading to a dimpling.

37. E. Transcytosis

Transcytosis is the process by which various macromolecules are transported across the interior of a cell. Vesicles are employed to intake the macromolecules on one side of the cell, draw them across the cell, and eject them on the other side. While transcytosis is most commonly observed in cells of an epithelium, the process is also present elsewhere. Blood capillaries are a well-known site for transcytosis, though it occurs in other cells, including neurons, osteoclasts and intestinal cells.
Eg : Insulin and Antibodies.

38. D. Vitamin B6 (pyridoxine)

Peripheral neuropathy and CNS effects are associated with the use of isoniazid and are due to pyridoxine (vitamin B6) depletion, but are uncommon at doses of 5 mg/kg. Persons with conditions in which neuropathy is common (e.g., diabetes, uremia, alcoholism, malnutrition, HIV-infection), as well as pregnant women and persons with a seizure disorder, may be given pyridoxine (vitamin B6) (10–50 mg/day) with isoniazid.

39. A. Increase in Filtration fraction
- FF=GFR/RPF.....Angiotrensine II constricts the efferent a. ....>.inc GFR....>inc FF

40. A. PR interval

The normal PR interval is from 120 ms to 200 ms in length.

The drugs that most commonly cause first-degree heart block are those that increase the refractory time of the AV node, thereby slowing AV conduction.
These include calcium channel blockers, beta-blockers, cardiac glycosides, and anything that increases cholinergic activity such as cholinesterase inhibitors.

CCB
The calcium channel blockers known as
1. Non-dihydropyridines decrease the force of contraction of the myocardium
such as verapamil or diltiazem, may be avoided (or used with caution) in individuals with cardiomyopathy.
2. Dihydropyridine calcium channel blockers are often used to reduce systemic vascular resistance and arterial pressure, but are not used to treat angina...such as Nefidipin

Many calcium channel blockers also slow down the conduction of electrical activity within the heart, by blocking the calcium channel during the plateau phase of the action potential of the heart.
This results in a negative chronotropic effect, or a lowering of heart rate. This can increase the potential for heart block.This results in a negative chronotropic effect, or a lowering of heart rate. This can increase the potential for heart block.

41. B. Digoxin

Anti-dig Fab fragments is an antidote for Cardiac glycosides toxicity

this drug is one of the rare drug in which you can you antibodies to eliminate it or use antibodies as antitode. Remember, anti-Fab antibodies can help clear this drug

42. D. Increased vascular hydrostatic pressure

Changes in the variables in Starling's equation can contribute to the formation of edema either by an increase in hydrostatic pressure within the blood vessel, a decrease in the oncotic pressure within the blood vessel or an increase in vessel wall permeability. The latter has two effects. It allows water to flow more freely and it reduces the oncotic pressure difference by allowing protein to leave the vessel more easily.

43. C. P pili

P pili mediate the binding of uropathogenic E. coli to a digalactoside receptor determinant present in the urinary tract epithelium.

44. D. Normal histologic findings

No visible change by light microscopy in first 2-4 hours
contraction bands visible after 1-2 hours ..early coagulative necrosis after 4 hours. Release of contents of necrotic cells into blood stream and the beginning of neutrophil emigration.

45. B. Inferior vena cava

Normal portal pressure is generally defined between 5 and 10 mm Hg. However, once the portal pressure rises to 12 mm Hg or greater, complications can arise, such as varices and ascites.

Treatment is

1.by diuretic drugs
2.by surgery to join the portal vein to the inferior vena cava (bypassing the liver)
3.implanting a stent within the liver to join portal tract veins to a hepatic vein tributary (TIPSS - transcutaneous intrahepatic porto-systemic shunt)

46. C. Cohort study

Cohort study
It is observational and prospective.....compares between a group with a given risk factors. (Bus driveres.....driving in seated position during 20 years) to a group without to assess (Bus condactors ...walking...)whether the risk factor increase the likelihood(MI) of disease.

47. D

An esophageal motility study (EMS) or esophageal manometry is a test to assess motor function of the Upper Esophageal Sphincter (UES), Esophageal body and Lower Esophageal Sphincter (LES).
Indications: An EMS is typically done to evaluate suspected disorders of motility or peristalsis of the esophagus. These include achalasia, diffuse esophageal spasm, nutcracker esophagus and hypertensive lower esophageal sphincter. These disorders typically present with dysphagia, or difficulty swallowing, usually to both solids and liquids even initially. Other patients with spasm disorders may have the test done to diagnose chest pain thought not to be of cardiac cause. The test is not useful for anatomical disorders of the esophagus (that is, disorders that distort the anatomy of the esophagus), such as peptic strictures and esophageal cancer.
http://en.wikipedia.org/wiki/Esophageal_motility_study

48. C

Chronic Granulomatosa Disease: Lack of NADPH oxidase - decrease reactive oxygen species(superoxide) and absent of respiratory burts in neutrophils. Increase susceptibility to catalase + organisms (S. Aureus, E. Coli, Aspergillus). Negative Nitroblue tetrazolium dye reduction test. (FA page 212)
http://en.wikipedia.org/wiki/Chronic...matous_disease

49. B. Ideas of reference

50. B. Alpha adrenergic antagonist

Alpha-blockers help treat benign prostatic hyperplasia (BPH) by relaxing smooth muscle tissue found in the prostate and the bladder neck. This allows urine to flow out of the bladder more easily.

Block 3

1. A. Decreases the risk of bias

If there is no other bias in the study, statistical significance guarantees that there is a 95 percent chance that the study's result reflects what is happening in the population at large.

2. C. mu receptors

Opioid receptors are a group of G protein-coupled receptors with opioids as ligands.
Morphine is shown to bind to mu receptors.

3. D. Jugular foramen

The jugular foramen may be subdivided into three compartments, each with their own contents.

The anterior compartment transmits the inferior petrosal sinus.
The intermediate transmits the glossopharyngeal, vagus, and accessory nerves (aka cranial nerves number IX, X, and XI respectively).
The posterior transmits the sigmoid sinus (becoming the internal jugular vein) and some meningeal branches from the occipital and ascending pharyngeal arteries.

In this case the Sternocleidomastoid m. and Trapezoid are inervated by Accessory n.....XI

4. E. Production of extended spectrum B-lactamse

The antimicrobial resistance to all mentioned antibiotics ....Beta-lactam antibiotics (Penicillin and its generation) is caused by production of extended-spectrum beta lactamase

5. C. 2:1

X-linked recessive inheritance is a mode of inheritance in which a mutation in a gene on the X chromosome causes the phenotype to be expressed (1) in males (who are necessarily hemizygous for the gene mutation because they have only one X chromosome) and
(2) in females who are homozygous for the gene mutation (i.e., they have a copy of the gene mutation on each of their two X chromosomes)....so it is 2:1

this Q don't have all the information requiered to give the answer, don't says nothing about her husband; but CARRIER female has half of the son affected and half of the daughters carrier then 50/50 (1.1)

The Q is about the expected ratio of carrier mother viable female to male children for X-linked recessive disease...the female child must get another affected X from daddy..to become sick so she needs to receive two genes,but male child just needs one X that it comes from her carrier mother to get the disease...so the ratio of getting affected X between female and male children is 2:1

6. A. Inferior mesenteric

A had it's origin than the renal artery, no Superior mesenteric bc it's origin is superior to the top of the kidneys

The Inferior Mesenteric Artery (IMA) branches off the anterior surface of the abdominal aorta below the renal artery branch points, and approximately midway between these and the aortic bifurcation (into the common iliac arteries). Supplies the large intestine from the left colic (or splenic) flexure to the upper part of the rectum, which includes the descending colon, the sigmoid colon, and part of the rectum. Proximally, its territory of distribution overlaps (forms a watershed) with the middle colic artery, and therefore the superior mesenteric artery. The SMA and IMA anastomose via the marginal artery of the colon (artery of Drummond). The territory of distribution of the IMA is more or less equivalent to the embryonic hindgut.
http://en.wikipedia.org/wiki/Inferior_mesenteric_artery

7. E. Dyskinease of the cilia

Kartagener syndrome, sinusitis, bronchiectasis, infertility, is Autosomal R, there is abnormal ciliary motion and impaired mucociliary clearence, reason for the clinical signs

8. B. Gastrin

Autoimmune Metaplastic Atrophic Gastritis (AMAG) is an inherited form of atrophic gastritis characterized by an immune response directed toward parietal cells and intrinsic factor.[1] The presence of serum antibodies to parietal cells and to intrinsic factor are characteristic findings. The autoimmune response subsequently leads to the destruction of parietal cells, which leads to profound hypochlorhydria (and elevated gastrin levels). The inadequate production of intrinsic factor also leads to vitamin B12 malabsorption and pernicious anemia. AMAG is typically confined to the gastric body and fundus.

Since there is a decrease in the hypochlorhydria → it will secret gastrin to increase Hcl.

9. D. C8

Complement component 8 is a protein involved in the complement system. A hereditary deficiency of C8 can result in increased susceptibility to Neisseria infections, such as meningitis and gonorrhea.
Deficiency of C5-C8 leads to Neisseria bacteremia.

10. C

The Urachus is the part of the allantoids duct between the bladder and the umbilicus.
The median umbilical ligament is a structure in human anatomy. It is a shriveled piece of tissue that represents the remnant of the embryonic urachus.

It extends from the apex of the bladder to the umbilicus, on the deep surface of the anterior abdominal wall. It is unpaired.

It is covered by the median umbilical fold
Lateral to this structure are the medial umbilical ligament (which is a different structure, not to be confused) and the lateral umbilical ligament.

11. A. Autoimmune

Addison Disease: Chronic adrenal insufficiency due to adrenal atrophy or destruction by disease (Autoimmmune, TB, metastasis). 1ºdeficiency of aldosterone and cortisol causing hypotension, and skin hyperpigmentation. (FA page 291)

Because primary hypocortisolism is manifested as a deficiency in glucocorticoid release from the adrenal cortex, increased ACTH will be released by the pituitary in order to trigger release of the absent glucocorticoid; it is because of this overstimulation of ACTH that bronzing of the skin occurs. In secondary or tertiary hypocortisolism, there is a deficiency of either CRH or ACTH release by the hypothalamus or pituitary gland, respectively. The former will manifest as no ACTH release while the latter will manifest as physiologic (normal) ACTH release; neither will cause an overproduction of ACTH. On examination, the following may be noticed:[2]
•Low blood pressure that falls further when standing (orthostatic hypotension)
•In long-standing Addison's Disease, the pinna of the ear may become calcified
•Most people with primary Addison's have darkening (hyperpigmentation) of the skin, including areas not exposed to the sun; characteristic sites are skin creases (e.g. of the hands), nipple, and the inside of the cheek (buccal mucosa), also old scars may darken. This occurs because melanocyte-stimulating hormone (MSH) and adrenocorticotropic hormone (ACTH) share the same precursor molecule, Pro-opiomelanocortin (POMC). After production in anterior pituitary gland, POMC gets cleaved into Gamma-MSH, ACTH and Beta-lipotropin. The subunit ACTH undergoes further cleavage to produce Alpha-MSH, the most important MSH for skin pigmentation. In secondary and tertiary forms of Addison's, skin darkening does not occur.
•Medical conditions such as type I diabetes, autoimmune thyroid disease (Hashimoto's thyroiditis and goiter) and vitiligo often occur together with Addison's (often in the setting of Autoimmune polyendocrine syndrome). Hence, symptoms and signs of any of the former conditions may also be present in the individual with Addison's.

http://en.wikipedia.org/wiki/Addison's_disease

12. C. Membrane

Ethanol (ethyl alcohol) and isopropanol (isopropyl alcohol) are alcohols that kill bacteria. Alcohols kill bacteria by first making the lipids that are part of the outer protective cell membrane of each bacterium cell more soluble in water so that the cell membrane begins to lose its structural integrity and fall apart. As the cell membrane disintegrates, alcohol can then enter the cell and denature proteins within each bacterium.

13. C. Fallopian tube

Neisseria are fastidious Gram-negative cocci that require nutrient supplementation to grow in laboratory cultures. Specifically, they grow on chocolate agar with carbon dioxide

Infection of the genitals in females with N. gonorrhoeae can result in pelvic inflammatory disease if left untreated, which can result in infertility. Pelvic inflammatory disease results if N. gonorrhoeae travels into the pelvic peritoneum (via the cervix, endometrium and fallopian tubes). Infertility is caused by inflammation and scarring of the fallopian tube. Infertility is a risk to 10 to 20% of the females infected with N. gonorrhoeae.

Normal fertilization occurs in the ampulla.

14. B. A childhood illness

Varicella zoster virus (VZV) is one of eight herpes viruses known to infect humans (and other vertebrates). It commonly causes chicken-pox in children and Herpes zoster (shingles) in adults and rarely in children. Primary VZV infection results in chickenpox (varicella), which may rarely result in complications including encephalitis or pneumonia. Even when clinical symptoms of chickenpox have resolved, VZV remains dormant in the nervous system of the infected person (virus latency), in the trigeminal and dorsal root ganglia.[1] In about 10–20% of cases, VZV reactivates later in life producing a disease known as shingles. Serious complications of shingles include postherpetic neuralgia, zoster multiplex, myelitis, herpes ophthalmicus, or zoster sine herpete.

Herpes zoster (or simply zoster), commonly known as shingles and also known as zona, is a viral disease characterized by a painful skin rash with blisters in a limited area on one side of the body, often in a stripe. The initial infection with varicella zoster virus (VZV) causes the acute (short-lived) illness chickenpox which generally occurs in children and young people. Once an episode of chickenpox has resolved, the virus is not eliminated from the body but can go on to cause shingles—an illness with very different symptoms—often many years after the initial infection.
http://en.wikipedia.org/wiki/Herpes_zoster

15. B. Cardiac tampoande

Cardiac tamponade is caused by a large or uncontrolled pericardial effusion, i.e. the buildup of fluid inside the pericardium.[2] This commonly occurs as a result of chest trauma (both blunt and penetrating),[3] but can also be caused by myocardial rupture, cancer, uraemia, pericarditis, or cardiac surgery,[2] and rarely occurs during retrograde aortic dissection.
http://en.wikipedia.org/wiki/Cardiac_tamponade

16. A. Anticholinesterase drug only

An acetylcholinesterase inhibitor (often abbreviated AChEI) or anti-cholinesterase is a chemical that inhibits the cholinesterase enzyme from breaking down acetylcholine, increasing both the level and duration of action of the neurotransmitter acetylcholine.
Some major effects of cholinesterase inhibitors:
Actions on the autonomic nervous system, that is parasympathetic nervous system will cause bradycardia, hypotension, hypersecretion, bronchoconstriction, GI tract hypermotility, and decrease intraocular pressure.
SLUDGE syndrome.
Actions on the neuromuscular junction will result in prolonged muscle contraction.
http://en.wikipedia.org/wiki/Acetylc...rase_inhibitor

17. E. T lymphocytes

IL 2: Secreted by Th cells. Stimulates growth of helper and cytotoxic T cells (FA page 205)
is an interleukin, a type of cytokine immune system signaling molecule, which is a leukocytotrophic hormone that is instrumental in the body's natural response to microbial infection and in discriminating between foreign (non-self) and self. IL-2 mediates its effects by binding to IL-2 receptors, which are expressed by lymphocytes, the cells that are responsible for immunity.
http://en.wikipedia.org/wiki/Interleukin_2

18. D. Peripheral vascular disease

Peripheral vascular disease (PVD), commonly referred to as peripheral arterial disease (PAD) or peripheral artery occlusive disease (PAOD), refers to the obstruction of large arteries not within the coronary, aortic arch vasculature, or brain. PVD can result from atherosclerosis, inflammatory processes leading to stenosis, an embolism, or thrombus formation. It causes either acute or chronic ischemia (lack of blood supply). Often PAD is a term used to refer to atherosclerotic blockages found in the lower extremity.[1]
PVD also includes a subset of diseases classified as microvascular diseases resulting from episodal narrowing of the arteries (Raynaud's phenomenon), or widening thereof (erythromelalgia), i.e. vascular spasms.
Peripheral artery occlusive disease is commonly divided in the Fontaine stages, introduced by René Fontaine in 1954 for ischemia:[2][3]
1. Mild pain when walking (claudication), incomplete blood vessel obstruction;
2. Severe pain when walking relatively short distances (intermittent claudication), pain triggered by walking "after a distance of >150 m in stage IIa and after

19. B. Necrosis of epithelial cells in proximal convoluted tubules

ATN, It may be classified as either toxic or ischemic. Toxic ATN occurs when the tubular cells are exposed to a toxic substance (nephrotoxic ATN). Ischemic ATN occurs when the tubular cells do not get enough oxygen, a condition that they are highly sensitive and susceptible to, due to their very high metabolism.
Acute tubular necrosis is classified as a "renal" (i.e. not pre-renal or post-renal) cause of Acute renal failure. Diagnosis is made by a FeNA (fractional excretion of sodium) > 3% and presence of muddy casts in urinalysis. On histopathology, there is usually tubulorrhexis, that is, localized necrosis of the epithelial lining in renal tubules, with focal rupture or loss of basement membrane. Proximal tubule cells can shed with variable viability and not be purely "necrotic".

20. B. Leishmani major

Leishmania is a genus of Trypanosomatid protozoa, and is the parasite responsible for the disease leishmaniasis. It is spread through sandflies of the genus Phlebotomus in the Old World, and of the genus Lutzomyia in the New World. Their primary hosts are vertebrates; Leishmania commonly infects hyraxes, canids, rodents, and humans.

Cutaneous leishmaniasis (localized and diffuse) infections appear as obvious skin reactions.
The most common is the Oriental Sore (caused by Old World species L. major, L. tropica, and L. aethiopica). In the New World, the most common culprits is L. mexicana.
Cutaneous infections are most common in Afghanistan, Brazil, Iran, Peru, Saudi Arabia and Syria.
http://upload.wikimedia.org/wikipedia/commons/e/e0/Leishmaniasis_life_cycle_d...ram_en.svgn

21. D. Glomerular filtration

-Serum BUN/cr ratio is greater than 15 and this is prerenal azotemia....which is caused by a decrease in cardiac output(evidenced by the increase in heart rate and decreased BP in the question stem)....the decreased cardiac output causes hypoperfusion of the kidneys ...and these causes A decrease in GFR ...this in turn causes An increase in BUN and Cr

22. B. Left subthalamic nucleus

Hemiballismus is usually characterized by involuntary flinging motions of the extremities. The movements are often violent and have wide amplitudes of motion. They are continuous and random and can involve proximal and/or distal muscles on one side of the body. Some cases even include the facial muscles.
It is common for arms and legs to move together. The more a patient is active, the more the movements increase. With relaxation comes a decrease in movements.

The subthalamic nucleus essentially provides the excitement needed to drive the globus pallidus. Injury to this area or its efferent or afferent connections can induce this disorder. The structure itself is a regulator of motor function and is also involved in associative and limbic functions.

It was traditionally thought that the disorder was only caused by injury to the subthalamic nucleus, but new studies are showing that damage to other areas of the brain can also be responsible for causing this disorder. Hemiballismus caused by lesions in the subthalamic nucleus is more severe than other forms of the disorder.

23. A. Do not proceed with treatment and determine if her parents are on their way.

for q 23...Legally, a patient is incompetent if unable to do the following:

-- (1) respond knowingly and intelligently to questions about recommended treatment;

--(2) participate in treatment decisions by means of rational thought processes; and

-- (3) understand the items of minimum basic medical treatment information with respect to that treatment

--hence the paranoid schizophrenic patient is legally competent and able to decide and refuse treatment....

---here is more explanation

it could be said that even a patient whose 'delusions are plainly irrational 'may be competent to make a treatment decision if he or she fulfills the following criteria

1-comprehending and retaining information relating to the decision

2-believing the information provided by the treating physician

3-weighing it in the balance when making a choice....

24. A. Allopurinol

Azathioprine is an immunosuppressive agent. It is first metabolised to 6-mercaptopurine, which in turn is converted to inactive products by xanthine oxidase. Allopurinol inhibits the second step of metabolism, and higher 6-mercaptopurine plasma levels result, with associated toxic effects on the bone marrow and other tissues. The resulting blood dyscrasias, leucopenia, thrombocytopenia or pancytopenia, can be life threatening.

25. D. Staphyloccus aureus

Staphylococcus aureus :
is a facultative anaerobic, Gram-positive coccus, and is the most common cause of staph infections. It is frequently part of the skin flora found in the nose and on skin. About 20% of the human population are long-term carriers of S. aureus.

S. aureus can cause a range of illnesses from minor skin infections, such as pimples, impetigo, boils (furuncles), cellulitis folliculitis, carbuncles, scalded skin syndrome, and abscesses, to life-threatening diseases such as pneumonia, meningitis, osteomyelitis, endocarditis, toxic shock syndrome (TSS), chest pain, bacteremia, and sepsis.

26. E. Talk to her about a nonbarrier method of contraception

q 26--When the patient came to the clinic to be fitted for the diaphragm...she is gonna be given informed consent before the procedure....and one part of the informed consent is ...informing the patient about alternative treatment benefits and disadvantages....

--simply put...before any procedure ...informed consent....part of that is explaining alternatives...

Evaluate her for a sexually transmitted disease
There is no reason to assume that she has sex with anyone but her husband.
A. *Fit her with a diaphragm as requested*
A diaphragm requires forethought and planning. Her history of getting drunk and having unprotected sex with her husband, underscored by the elective abortion, shows that she lacks the forethought that a barrier method like condoms or a diaphragm requires to be effective.
B. *Help her to deal with her feelings about the abortion*
Well, secondarily - but your first concern is the reason for her visit today. This is not the best answer, even though it's not inappropriate.
C. *Refer her to Alcoholics Anonymous*
You do not have enough information to diagnose her with alcoholism. You may wish to investigate or address her binge drinking at some point, but this is not the reason for her visit, which is contraception.
D. *Talk to her about a nonbarrier method of contraception*
This is the best answer. As long as her abortion was uncomplicated (no endometritis post-abortion), she is an excellent candidate for an IUD. If an IUD is contraindicated, then injectable or implantable hormonal contraception would be good options. Even an OCP would have better compliance than a diaphragm in this patient.
27. B.

when the products accumulate in one direction .....reactions in that direction dont proceed and
the reaction proceeds from the substrate 2 to downard direction in this pathway

28. A. Bone infarct

Sickle-cell disease may lead to various acute and chronic complications, several of which are potentially lethal.
Sickle cell crisis

Sickle cell disease results in anaemia and crisis that could be of many types including the vaso-occlusive crisis, aplastic crisis, sequestration crisis, hyper haemolytic crisis and others. Most episodes of sickle cell crises last between five and seven days.

Vaso-occlusive crisis
Pain crisis, or sickle crisis - when the flow of blood is blocked to an area because the sickled cells have become stuck in the blood vessel. These are also called "vasoocclusive crises." The pain can occur anywhere, but most often occurs in the chest, arms and legs. Painful swelling of the fingers and toes, called dactylitis, can occur in infants and children under 3 years of age. Priapism is a painful sickling that occurs in the penis. Any interruption in blood flow to the body can result in pain, swelling and possible death of the surrounding tissue not receiving adequate blood and oxygen.

29. C. Neuroleptic malignant syndrome

Neuroleptic malignant syndrome (NMS) is a life- threatening neurological disorder most often caused by an adverse reaction to neuroleptic or antipsychotic drugs. It generally presents with muscle rigidity, fever, autonomic instability and cognitive changes such as delirium, and is associated with elevated creatine phosphokinase (CPK).

Incidence of the disease has declined since its discovery (due to changes in prescription habits), but it is still a potential danger to patients being treated with antipsychotics. Because of its unpredictability, there is no one set course of action to treat the syndrome, but generally, removal of the antipsychotic drug treatment, along with supportive medical management, lead to a positive outcome.

30. A. Ferritin

Ferritin serves to store iron in a non-toxic form, to deposit it in a safe form, and to transport it to areas where it is required. The function and structure of the expressed ferritin protein varies in different cell types. This is controlled primarily by how much mRNA is translated, and how stable the mRNA is. mRNA concentration is further tweaked by changes to how it is stored and how efficiently it is transcribed.The presence of iron itself is a major trigger for the production of ferritin, with some exceptions (such as the yolk ferritin of the gastropod Lymnaea, which lacks an iron-responsive unit).

Free iron is toxic to cells as it acts as a catalyst in the formation of free radicals from reactive oxygen species via the Fenton Reaction. Hence vertebrates use an elaborate set of protective mechanisms to bind iron in various tissue compartments. Within cells, iron is stored in a protein complex as ferritin or hemosiderin. Apoferritin binds to free ferrous iron and stores it in the ferric state. As ferritin accumulates within cells of the reticuloendothelial system, protein aggregates are formed as hemosiderin. Iron in ferritin or hemosiderin can be extracted for release by the RE cells although hemosiderin is less readily available. Under steady state conditions, the serum ferritin level correlates with total body iron stores; thus, the serum ferritin FR5RI is the most convenient laboratory test to estimate iron stores.

31. E. Neuroblastoma

Neuroblastoma...Most common tumor of the adrenal medulla in children.
can occur anywhere along the sympathetic chain
HVA...itis abreakdown product of Dopamine in urine
N-MYC...oncogene
less likely to develop HTN

32. C. Liver

Most malignant (cancerous) liver tumors arise when cancer spreads (metastasizes) from another part of the body to the liver, most commonly from the colon.

33. B. Loss of function of muscles innervated by the radial nerve

The muscular branches of the radial nerve supply the Triceps brachii, Anconæus, Brachioradialis, Extensor carpi radialis longus, and Brachialis, and are grouped as medial, posterior, and lateral.
http://en.wikipedia.org/wiki/File:Hu...l_fracture.png
http://upload.wikimedia.org/wikipedi...e2/Gray818.png

34. A. Choriocarcinoma

A complete mole is caused by a single sperm combining with an egg which has lost its DNA (the sperm then redupplicates forming a "complete" 46 chromosome set) The genotype is typically 46,XX (diploid) due to subsequent mitosis of the fertilizing sperm, but can also be 46,XY (diploid).
In contrast, a partial mole occurs when an egg is fertilized by two sperm or by one sperm which reduplicates itself yielding the genotypes of 69,XXY (triploid) or 92,XXXY (quadraploid). Complete hydatidiform moles have a higher risk of developing into choriocarcinoma -- a malignant tumor of trophoblast cells -- than do partial moles.

Choriocarcinoma is a malignant, trophoblastic and aggressive cancer, usually of the placenta. It is characterized by early hematogenous spread to the lungs. It belongs to the far end of the spectrum of gestational trophoblastic disease (GTD), a subset of germ cell tumors.

35. D. Pyelonephritis

Pyelonephritis is an ascending urinary tract infection that has reached the pyelum (pelvis) of the kidney. If the infection is severe, the term "urosepsis" is used interchangeably (sepsis being a systemic inflammatory response syndrome due to infection). It requires antibiotics as therapy, and treatment of any underlying causes to prevent recurrence. It is a form of nephritis. It can also be called pyelitis.

Severe cases of pyelonephritis lead to sepsis, a systemic response to infection characterized by fever, a raised heart rate, rapid breathing and decreased blood pressure (occasionally leading to septic shock). When pyelonephritis or other urinary tract infections lead to sepsis, it is termed urosepsis.

Most cases of "community-acquired" pyelonephritis are due to bowel organisms that enter the urinary tract. Common organisms are E. coli (70-80%) and Enterococcus faecalis. Hospital-acquired infections may be due to coliforms and enterococci, as well as other organisms uncommon in the community (e.g. Klebsiella spp., Pseudomonas aeruginosa). Most cases of pyelonephritis start off as lower urinary tract infections, mainly cystitis and prostatitis.

Acute pyelonephritis is a potentially organ- and/or life-threatening infection that characteristically causes some scarring of the kidney with each infection and may lead to significant damage to the kidney (any given episode), kidney failure, abscess formation (eg, nephric, perinephric), sepsis, or sepsis syndrome/shock/multiorgan system failure.

Most cases of "community-acquired" pyelonephritis are due to bowel organisms that enter the urinary tract. Common organisms are E. coli (70-80%) and Enterococcus faecalis.

Antibiotics are the mainstay of treatment. Mild cases may be treated with oral therapy, but generally intravenous antibiotics are required for the initial stages of treatment. The type of antibiotic depends on local practice, and may include fluoroquinolones (e.g. ciprofloxacin), beta-lactam antibiotics (e.g. amoxicillin or a cephalosporin), trimethoprim (alone or in combination with sulfamethoxazole). Aminoglycosides are generally avoided due to their toxicity, but may be added for a short duration.

36. D. Posterior inferior cerebellar

The posterior inferior cerebellar artery (PICA), the largest branch of the vertebral artery, is one of the three main arterial blood supplies for the cerebellum.

It winds backward around the upper part of the medulla oblongata, passing between the origins of the vagus and accessory nerves, over the inferior cerebellar peduncle to the undersurface of the cerebellum, where it divides into two branches.
The medial branch continues backward to the notch between the two hemispheres of the cerebellum; while the lateral supplies the under surface of the cerebellum, as far as its lateral border, where it anastomoses with the anterior inferior cerebellar and the superior cerebellar branches of the basilar artery.
Branches from this artery supply the choroid plexus of the fourth ventricle.
Diseases

Infarction of this artery due to thrombosis or a stroke leads to lateral medullary syndrome, also known as PICA syndrome or Wallenberg syndrome. Severe occlusion of this or vertebral arteries could lead to Horner's Syndrome as well.
http://upload.wikimedia.org/wikipedi..._Willis_en.svg

37. A. Botulinium

Lambert-Eaton myasthenic syndrome (LEMS) main causal cancer small-cell lung cancer
In LEMS, antibodies against VGCC, particularly the P/Q-type VGCC, decrease the amount of calcium that can enter the nerve ending, hence less acetylcholine can be mobilized to the neuromuscular junction. Apart from skeletal muscle, the autonomic nervous system also requires acetylcholine neurotransmission; this explains the occurrence of autonomic symptoms in LEMS. P/Q voltage-gated calcium channels are also found in the cerebellum, explaining why some experience problems with coordination. Antibodies may also bind other VGCCs. Many people with LEMS, both with and without VGCC antibodies, have detectable antibodies against the M1 subtype of the acetylcholine receptor; it is thought that their presence participates in a lack of compensation for the slow calcium influx.

38. C. Glucose-6-phophatase

Glycogen storage disease type I (GSD I) or von Gierke's disease, is the most common of the glycogen storage diseases. This genetic disease results from deficiency of the enzyme glucose-6-phosphatase. This deficiency impairs the ability of the liver to produce free glucose from glycogen and from gluconeogenesis. Since these are the two principal metabolic mechanisms by which the liver supplies glucose to the rest of the body during periods of fasting, it causes severe hypoglycemia. Reduced glycogen breakdown results in increased glycogen storage in liver and kidneys, causing enlargement of both. Both organs function normally in childhood but are susceptible to a variety of problems in the adult years. Other metabolic derangements include lactic acidosis and hyperlipidemia. Frequent or continuous feedings of cornstarch or other carbohydrates are the principal treatment. Other therapeutic measures may be needed for associated problems.

39. A. Efferent glomerular arterioles

ACE inhibitors reduce the progress of diabetic nephropathy independently from their blood pressure-lowering effect.This action of ACE inhibitors is used in the prevention of diabetic renal failure.
ACE inhibitors block the conversion of angiotensin I to angiotensin II.
Under normal conditions, angiotensin II will constrict the efferent arterioles of the kidney leads to increased perfusion pressure in the glomeruli....>inc GFR.

40. E. Thromboxane A2

Thromboxane A2 (TXA2), produced by activated platelets, has prothrombotic properties, stimulating activation of new platelets as well as increasing platelet aggregation.

41. C. Magnesium trisilicate

ADEQUATE DOSES OF MAGNESIUM TRISILICATE MAY CAUSE DIARRHEA DUE TO THE ACTION OF SOLUBLE MAGNESIUM SALTS IN THE ENTERIC TRACT.

Magnesium trisilicate is an inorganic compound that is used as a food additive.
It can also be used in oral pharmaceutical formulations and food products as a glidant. It is also used therapeutically as an antacid, and also for the treatment of ciprofloxacin overdose or toxic

42. A. Capsular polysaccharide

Neisseria meningitidis, the meningococcus, is a Gram-negative, oxidase-positive diplococcus.
Identical in its staining and morphological characteristics to Neisseria gonorrhoeae.

However, at an ultrastructural level, N. meningitidis has a prominent polysaccharide capsule not seen in the gonococcus.

The capsule is antiphagocytic and is an important virulence factor in meningococcal disease. N. meningitidis strains are grouped on the basis of their capsular polysaccharides, into 12 serogroups, some of which are subdivided according to the presence of outer membrane protein and lipopolysaccharide antigens.

Neisseria meningitidis is a heterotrophic gram-negative diplococcal bacterium best known for its role in meningitis and other forms of meningococcal disease such as meningococcemia.

Lipooligosaccharide (LOS) is a component of the outer membrane of N. meningitidis which acts as an endotoxin which is responsible for fever, septic shock, hemorrhage due to the destructions of red blood cells.[9] Other virulence factors include a polysaccharide capsule which prevents host phagocytosis and aids in evasion of the host immune response; and fimbriae which mediate attachment of the bacterium to the epithelial cells of the nasopharynx

43. E. Uncompensated respiratory acidosis

Compensated Respiratory acidosis.....low PH, high Bicarbonate ,and high Pco2 while
Uncompensated Respiratory acidosis .....low PH, low Bicarbonate and high Pco2

here is how to ID Acid Base: 3 rules to follow
1. look at the pH: Acid, basic respective
2. look at CO2 and HCO3--> which one of these is the one that changes the most from their normal value
3. Find out if Acid-Base is compensate or Not-- by looking at the lower of #2--> that is does it increase slightly or decrease slightly?
So, if HCO3 and CO2 goes the same direction-- either both high or both low-- no compensation occur
now call me if you understand or need more info
1. ph is 6.8 so it is acid
2. PCO2 is the one that changes most
3. pCO2 of this patient is 80
4. normal pCO2 is 33-45 mmHG
35
5. HC03 of this patient is 12
normal HC03 is 22-28
a difference of 10
6. PCO2 changes the most from their normal value
respiratory
respiratory (#2) acidosis (#1)

- Metabolic acidosis
o PC02 predicted = (1.5)(HC03-) + 8
o PC02 Measured (given in the equation) > PC02 predicted...Co-Existing metabolic acidosis & respiratory acidosis
o PC02 Measured (given in the equation) < PC02 predicted...Co-Existing metabolic acidosis & respiratory alkalosis

- Metabolic alkalosis
o PC02 predicted = (40) + [(0.7)(HC03 given in the equation – 24)
o PC02 Measured (+/5 given in the equation) > PC02 predicted…Co-Existing metabolic alkalosis & respiratory acidosis
o PC02 Measured (+/5 given in the equation) < PC02 predicted…Co-Existing metabolic alkalosis & respiratory alkalosis
- Respiratory acidosis
o Acute problem:
▪·Look to see if HC03 is compensated or not
▪·Normal PCO2 is 40; normal HC03 is 24
▪·1. (increase 1 mEq/L for HC03)/(increase 10 mmHg PCO2)…then it is compensated
▪·2. If PC02 is compensated, but, HC03 is > 24…Co-Existing respiratory acidosis & metabolic alkalosis
▪·3. If PC02 is compensated, but, HC03 is < 24…Co-Existing respiratory acidosis & metabolic acidosis
- Respiratory alkalosis
o Acute problem
▪·Look to see if PC02 is compensated or not
▪·Normal PC02 is 40; normal HC03 is 24
▪·1. (Decrease 2 mEq/L for HC03)/(decrease 10 mmHg PCO2)…then it is compensated
▪·2. If PC02 is compensated, but, HC03 is > 24…Co-Existing respiratory alkalosis & metabolic alkalosis
▪·3. If PC02 is compensated, but, HC03 is < 24…Co-Existing respiratory alkalosis & metabolic acidosi

44. A. Airway compression

In normal patients, after a small amount of gas has been exhaled, the flow is limited by airway compression and determined by the elastic recoil of the lung and resistance upstream of that point.

In restrictive diseases, the maximum flow rate is reduced, as is the total volume expired. The flow is abnormally high in the latter part of expiration because of increased recoil.

In obstructive diseases, the flow rate is very low in relation to lung volume, and a scooped out appearance is often seen following the point of maximal flow.

http://www.frca.co.uk/article.aspx?articleid=100023

45. B. Distention of air spaces distal to terminal bronchi

Empysema...permanent enlargement of all part of the respiratory unit...respiratory bronchioles, alveolar, alveoli
Causes:
cigarette smoking
alpha1 antitrypsin deficiency

There are 2 types....

1. Centriacinar...is characterized by trapping of air in the respiratory bronchiole...elastic fibers of the distal TB are destroyed, causing obstruction to airflow...this causes the trapped air to distend the RBs, whose elastic tissue support is destroyed.

2.panacinar...is characterized by trapping of air in the entire respiratory unite behind the collapsed TB

http://www.google.com/imgres?imgurl=http://www.meddean.luc.edu/lumen/meded/Ra...0CBsQ9QEwAg

46. D. Severe combined immunodeficiency

Severe combined immunodeficiency (SCID), (also known as "Alymphocytosis," "Glanzmann–Riniker syndrome," "Severe mixed immunodeficiency syndrome," and "Thymic alymphoplasia") is a genetic disorder in which both "arms" (B cells and T cells) of the adaptive immune system are impaired due to a defect in one of several possible genes.

SCID is a severe form of heritable immunodeficiency. It is also known as the "bubble boy" disease because its victims are extremely vulnerable to infectious diseases and some of them, such as David Vetter, become famous for living in a sterile environment.

SCID is the effect of a highly compromised, so much it is almost considered absent, immune system. The gene mutations that cause SCID are not just for the disorder. The buildup of dATP, which induces the cell to make cytochrome c, destroys and signals for apoptosis in all rapidly proliferating cells. This includes cells in the GI tract, immune system lymphocytes, and sperm cells.

Chronic diarrhea, ear infections, recurrent Pneumocystis jirovecii pneumonia, and profuse oral candidiasis commonly occur. These babies, if untreated, usually die within 1 year due to severe, recurrent infections unless they have undergone successful Hematopoietic stem cell transplantation.

47. E. “Let’s review how the inhaled corticosteroid will help in the control of your symptoms.”

48. D. Period prevalence

...The study design is Case Control and in case control studies u can only determine the prevalence of the disease...In this case the duration is one

year and it is period prevalence rather than point prevalence

49. A. Cytoplasm

The sarcoplasmic reticulum serves as a repository for Ca++.
1. In rested muscle, Ca++ is found in high concentration in the cisternae at the triad.

2. In recently active muscle, the calcium is found in the narrowed, longitudinal portion from which it moves to the triad as time passes.

3. During contraction, Ca++ is found in high concentration outside the sarcoplasmic reticulum among the myofilaments

50. E. Toxoid

Diphtheria toxoid the formaldehyde-inactivated toxin of Corynebacterium diphtheriae, used as an active immunizing agent against diphtheria, usually in mixtures with tetanus toxoid and pertussis vaccine (DTP or DTaP) or with tetanus toxoid alone (DT for pediatric use and Td, which contains less diphtheria toxoid, for adult use)

Nbme form 7 block 4 answers

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1. A. Cisplatin

A- Cisplatin Dose-related and cumulative renal insufficiency, is the major dose-limiting toxicity of Cisplatin. Renal toxicity has been noted in 28% to 36% of patients treated with a single dose of 50 mg/m2. It is first noted during the second week after a dose and is manifested by elevations in BUN and creatinine, serum uric acid and/or a decrease in creatinine clearance. Renal toxicity becomes more prolonged and severe with repeated courses of the drug. Renal function must return to normal before another dose of Cisplatin can be given. Elderly patients may be more susceptible to nephrotoxicity.
The same side effects (nephrotoxicity & ototoxicity) had the aminoglycosides and the loop diuretics

2. A. Capsular polysaccharide

the patient had a cryptococcal infection, this fungus is heavily encapsulated yest, found in soil, pingeon -droppings. Culture on Subouraud agar. Sain with India Ink and with the latex agglutination test detects polysaccharide capsular antigen.

3. A. Diabetic glomerulosclerosis

A Diabetic nephropathy in light Micro Kimmelstiel-Wilson lesions "wire loop" the basement membrane is thick and in the pic you can see nodules in the periphery of the glomerulos classic imagine of diabetic GS, the nodules had the name of Kimmrdiel-Wilson

4. D. Prevents endometrial hyperplasia

the risk of endometrial cancer increased if you used estrogen and don't have a balance with the progesterone hormone, but remember the risk of HRT after five years, to get breast cancer is around 30-40%

5. A. Basal cells

At days 3 and 7, the mitotic rates of basal cells of regenerating epithelium were 3 times higher than that of controls.
Processes that are involved in active wound healing can lead to an increased risk for basal cell carcinoma in the skin.

A) Basal cells: the layer that do the regeneration for skin damage; closer to the blood vessels
C) Langerhans cells is the macrophages of the skin.

6. B. Degradation of p53 tumor suppressor protein

p53 (also known as protein 53 or tumor protein 53), is a tumor suppressor protein that in humans is encoded by the TP53 gene. p53 is important in multicellular organisms, where it regulates the cell cycle and, thus, functions as a tumor suppressor that is involved in preventing cancer.

If the TP53 gene is damaged, tumor suppression is severely reduced. People who inherit only one functional copy of the TP53 gene will most likely develop tumors in early adulthood, a disease known as Li-Fraumeni syndrome. The TP53 gene can also be damaged in cells by mutagens (chemicals, radiation, or viruses), increasing the likelihood that the cell will begin decontrolled division. More than 50 percent of human tumors contain a mutation or deletion of the TP53 gene.

7. E. Zenker diveriticulum

Zenker diverticulum often causes clinical manifestations such as dysphagia (difficulty swallowing), and sense of a lump in the neck; moreover, it may fill up with food, causing regurgitation (reappearance of ingested food in the mouth), cough (as some food may be regurgitated into the airways), halitosis (smelly breath, as stagnant food is digested by microrganisms) and involuntary gurgling noises when swallowing. It rarely, if ever, causes any pain.
“undigested food”. There is a pouch on the upper esophagus and holding all the undigested food.

wikipedia.org/wiki/Zenker's_diverticulum

8. B. “You do have a very serious muscle disease called ALS. This cannot be cured, but I will be here to help you with everything I can. Have you heard of ALS?”

make diagnosis and offer to educate the patient

9. D. Tissue plasminogen activator

acute MI, tPA catalyzes the conversion of plasminogen into plasmin.
To be most effective in ischemic stroke, tPA must be administered as early as possible after the onset of symptoms. Protocol guidelines require its use intravenously within the first three hours of the event, after which its detriments may outweigh its benefits.

Warfarin is used for chronic anticoagulation vs. TPA is for acute MI.

10. C. Decreased diffusing capacity for carbon monoxide (DLco)

pulmonary edema increases the thickness of the alveolo-capillary space, increasing the distance the oxygen must diffuse to reach blood. This impairs gas exchange leading to hypoxia, increases the work of breathing, and eventually induces FIBROSIS OF THE AIRSPACE.

After a patient is treated for ARDS, the membrane will be thickened = hyaline membrane, hence, the CO can’t be diffused. So we get a decreased diffusing capacity for carbon monoxide (DLco)

11. A. “I recommend that he undergo a hearing screening as soon as possible.”

- He should at least have words at 20 months (the "2 rule" is 200 words and 2-word sentences at 2 y/o, right?), so B is wrong. It is possible that he is just late in speaking, but correctable problems like hearing disorders have to be ruled out.
- He is active and shows no neurological Sx except for his lack of speech development. Hydrocephalus is not a good differential, so C is wrong.
- D is a behavioral intervention intended to engage the child and help him overcome a reluctance to adopt speech. This is a friendly and engaged child already.
- Friendly + good eye contact = no autism, so E is wrong

A set of hearing tests (A) would be the best next step in diagnosis.

12. A. Dopamine agonist

The treatment of choice for prolactinomas is dopamine agonist administration, which results in tumor shrinkage, normalization of prolactin, and restoration of gonadal function in the majority of patients.
- bromocriptine: Its use is limited by a high incidence of side effects, a short duration of action, and a lack of effectiveness in some patients.
- cabergoline: a long-acting oral dopamine agonist specific for the D2 receptor; the most interesting feature of cabergoline in terms of patient compliance is its extremely long half-life. Most patients can be treated with a single weekly dose, is in contrast to the 1-3 times daily administration required for brornocriptine.

13. B. Ligand gated ion channels

Valproate
- blocks the voltage-gated sodium channels and T-type calcium channels.
- Inhibition of the transamination of GABA (by inhibiting GABA transaminase, then GABA would increase in concentration)

Carbamazepine
- stabilizes the inactivated state of sodium channels, meaning that fewer of these channels are available to subsequently open, making brain cells less excitable (less likely to fire).
- potentiates GABA receptors

Phenytoin
- reducing electrical conductance among brain cells by stabilizing the inactive state of voltage-gated sodium channels.

14. A. competitive antagonist
A competitive antagonist cause a parallel shift to the right in the dose response curve for the agonist

15. E. GTP

[IMG]file:///Users/Guess/Library/Caches/TemporaryItems/msoclip/0/clip_image002.png[/IMG]

GTP is essential to signal transduction, particularly with G-proteins, in second-messenger mechanisms where it is converted to GDP (guanosine diphosphate) through the action of GTPases.

Adenylate cyclase (EC 4.6.1.1, also known as adenylyl cyclase, adenyl cyclase or AC) is a lyase enzyme. It is a part of the cAMP-dependent pathway

Adenylate cyclase can be activated or inhibited by G proteins, which are coupled to membrane receptors and thus can respond to hormonal or other stimuli. Following activation of adenylate cyclase, the resulting cAMP acts as a second messenger by interacting with and regulating other proteins such as protein kinase A and cyclic nucleotide-gated ion channels.

G proteins (guanine nucleotide-binding proteins) are a family of proteins involved in transmitting chemical signals outside the cell, and causing changes inside the cell. They communicate signals from many hormones, neurotransmitters, and other signaling factors. [1]

G protein-coupled receptors are transmembrane receptors. Signal molecules bind to a domain located outside the cell. An intracellular domain activates a G protein. The G protein activates a cascade of further compounds, and finally causes a change downstream in the cell.

G proteins function as molecular switches. When they bind guanosine triphosphate (GTP), they are 'on', and when they bind guanosine diphosphate (GDP), they are 'off'.

G proteins regulate metabolic enzymes, ion channels, transporters, and other parts of the cell machinery, controlling transcription, motility, contractility, and secretion, which in turn regulate systemic functions such as embryonic development, learning and memory, and homeostasis.[2]

http://en.wikipedia.org/wiki/Adenylate_cyclase

http://bioweb.wku.edu/courses/biol566/Images/Gprot2.jpg

16. C. Left ventricular stroke volume: Decreased; Left atrial pressure: increased; Peripheral vascular pressure: increased.
Mitral Regurgitation: Loudest at apex and radiates toward the axilla. Enhanced by maneuvers that ↑ TPR (eg. Squatting, hand grip), or LA return (eg. expiration). MR is often due to ischemic heart disease , mitral valve prolapsed, or LV dilation. Stroke Volume affected by contractility, afterload and preload. Stroke Volume ↑when: ↑ Preload, ↓ Afterload, (FA page 251, 255).

S1, S2 are normal means that the valves are normal.

S3 is abnormal means that person has volume overload = too much fluid = ejection problem
Left ventricular stroke volume decrease because you're not pumping blood out therefore, you have a decrease in stroke volume.

Left atrial pressure has to increased because there are so much blood in the left ventricular and it will back up from ventricle to atrium…

Peripheral vascular resistance is increase bc you push all the blood from you lower extremity to your heart and hence you have increase preload.

If it is S4 problem…the patient heart is small/constricted/blood goes in and it has to pushes out…so therefore, you have an increase left ventricular stroke volume; decrease left atrial pressure, decrease peripheral vascular resistance.

If you have a decrease peripheral vascular resistance, you won't have a heart problem, but you will have an edema problem because you have a decrease in preload
17. C. lipopolysaccharide stimulating production of tumor necrosis factor.

Lipopolysaccharides (LPS), also known as lipoglycans, are large molecules consisting of a lipid and a polysaccharide joined by a covalent bond; they are found in the outer membrane of Gram-negative bacteria, act as endotoxins and elicit strong immune responses in animals. Most cases of septic shock (approximately 70%) are caused by endotoxin-producing Gram-negative bacilli. Endotoxins are bacterial wall lipopolysaccharides (LPS) consisting of a toxic fatty acid (lipid A) core common to all Gram-negative bacteria, and a complex polysaccharide coat (including O antigen) unique for each species. Analogous molecules in the walls of Gram-positive bacteria and fungi can also elicit septic shock. Free LPS attaches to a circulating LPS-binding protein, and the complex then binds to a specific receptor (CD14) on monocytes, macrophages, and neutrophils. Engagement of CD14 (even at doses as minute as 10 pg/mL) results in intracellular signaling via an associated "Toll-like receptor" protein 4 (TLR-4), resulting in profound activation of mononuclear cells and production of potent effector cytokines such as IL-1 and TNF-α.
Immune response

LPS function has been under experimental research for several years due to its role in activating many transcription factors. LPS challenge also produces many types of mediators involved in septic shock. Humans are much more sensitive to LPS than other animals (e.g., mice). A dose of 1 μg/kg induces shock in humans, but mice will tolerate a dose up to a thousand times higher.[13] This may relate to differences in the level of circulating natural antibodies between the two species.[14][15] Said et al. showed that LPS causes an IL-10-dependent inhibition of CD4 T-cell expansion and function by up-regulating PD-1 levels on monocytes which leads to IL-10 production by monocytes after binding of PD-1 by PD-L.

18. F. Vagus nerve

Innervation: Both right and left vagus nerves ascend to the brain in the carotid sheath, lateral to the carotid artery.

The right vagus nerve gives rise to the right recurrent laryngeal nerve, which hooks around the right subclavian artery and ascends into the neck between the trachea and esophagus. The right vagus then crosses anteriorly to the right subclavian artery and runs posterior to the superior vena cava and descends posterior to the right main bronchus and contributes to cardiac, pulmonary, and esophageal plexuses. It forms the posterior vagal trunk at the lower part of the esophagus and enters the diaphragm through the esophageal hiatus.

The left vagus nerve enters the thorax between left common carotid artery and left subclavian artery and descends on the aortic arch. It gives rise to the left recurrent laryngeal nerve, which hooks around the aortic arch to the left of the ligamentum arteriosum and ascends between the trachea and esophagus. The left vagus further gives off thoracic cardiac branches, breaks up into pulmonary plexus, continues into the esophageal plexus, and enters the abdomen as the anterior vagal trunk in the esophageal hiatus of the diaphragm. Both the vagal nerves have their cell bodies contained in the two nodose ganglia.

19. A. Alendronate

Alendronic acid (INN) or alendronate sodium (USAN, sold as Fosamax by Merck) is a bisphosphonate drug used for osteoporosis and several other bone diseases. It is marketed alone as well as in combination with vitamin D (2,800 U and 5600 U, under the name Fosamax+D). Merck's U.S. patent on alendronate expired in 2008 and Merck lost a series of appeals to block a generic version of the drug from being certified by the U.S. Food and Drug Administration (FDA).

Side-effects
• Gastrointestinal tract: ulceration of the esophagus; this may require hospitalition and intensive treatment. Gastric and duodenal ulceration may also occur. December 31, 2008, the FDA reported alendronate and related drugs may carry an increased risk for esophageal cancer.[3]

• General: infrequent cases of skin rash, rarely manifesting as Stevens-Johnson syndrome and toxic epidermal necrolysis, eye problems (uveitis, scleritis) and generalized muscle, joint, and bone pain [4] (rarely severe) have been seen. In laboratory tests decreased calcium and phosphate values may be obtained but reflect action of the drug and are harmless.
• Osteonecrosis of the Jaw - Deterioration of the Temporomandibular Joint (TMJ) may occur while on this drug, if dental work of any kind is carried out.[5] Although osteonecrosis is uncommon, it occurs primarily in patients being administered intravenous biphosphonates, with most cases being reported in cancer patients.[citation needed]
• Neurological: Rare instances of auditory hallucinations and visual disturbances have been associated with alendronate and other bisphosphonates.[6]
• Bone: Alendronate has been linked in long-term users to the development of low-impact femoral fractures.[7] Further, studies suggest that users of alendronate have an increase in the numbers of osteoclasts and develop giant, more multinucleated osteoclasts; the significance of this development is unclear.[8] People who have taken Fosamax has been linked to a rare type of leg fracture that cuts straight across the upper thigh bone after little or no trauma. (Subtrochanteric fractures) [9] This is because Fosamax makes the thigh bone more brittle and stops the cells in the body that remodel the bone. Studies are showing that people who have taken Fosamax for more than five years are at risk for developing these kind of fractures. In some cases, patients have reported that, after weeks or months of unexplained aching, their thigh bones simply snapped while they were walking or standing. One doctor reports that a 59-year old previously healthy woman visiting New York City was riding a subway train one morning when the train jolted. She shifted all her weight to one leg, felt a bone snap, and fell to the floor of the train. An x-ray in a local emergency room revealed a comminuted spiral fracture involving the upper half of the right femur. She had been taking Fosamax for 7 years. [10] On Oct. 13, 2010 the Food and Drug Administration issued a warning about these fractures.

http://en.wikipedia.org/wiki/Alendronic_acid

20. A. Dislocation

Gluteal fold is sign of CHD (congenital hip dysplasia).
If patient was in their teens (13-16) then it would be a slipped epiphyseal disc

A dislocated hip is a condition that can be congenital or acquired. Congenital hip dislocations are much more common in girls than in boys.

The pelvis and femur are the two main bones that form the hip joint. There is an articulation of the head of the femur and the acetabulum of the pelvis. Together, they make the hip joint an enarthrodial joint. There are two pelvic bones (right and left), each consisting of the Ilium, ischium, and the pubis. They connect to form the symphysis pubis on the anterior side, while the posterior side connects with the sacrum and coccyx to form sacroiliac joints.[2]

These bones are joined with help of strong ligaments, making them slightly, movable joints. There are five strong and dense ligaments that help to reinforce the hip joint. They include the iliofemoral ligament, the teres ligament, the pubofemoral ligament, the ischiofemoral ligament, and the zona orbicularis ligament. The iliofemoral ligament helps to prevent hip

hyperextension, as it is one of the strongest ligaments in the body. The teres ligament slightly limits hip adduction, while the pubofemoral ligament limits excessive extension and abduction. The ischiofemoral ligament limits internal rotation of the hip, while the zona orbicularis ligament helps maintain contact in the joint.

Congenital hip dislocation must be detected early when it can be easily treated by a few weeks of traction. If it is not detected, the child's hip may develop incorrectly seen when the child begins to walk. If one hip is affected the child will have a limp and lurch and with bilateral dislocation there will be a waddling gait. On physical exam, with the baby in the supine position, the examiner flexes the hips and knees both to 90 degrees, and, holding the knees, pushes gently downward, which may induce a posterior dislocation or subluxation. Keeping the baby in this 90 degree flexed position, the examiner then externally rotates the thighs. A normal infant will demonstrate no evidence of dislocation. It can also be detected with the Galeazzi test. Congenital hip dislocation is much more common in girls than boys.

Acquired hip dislocations are extremely painful and commonly occur during car accidents. They may be treated by surgical realignment and traction.

http://en.wikipedia.org/wiki/Dislocation_of_hip

21. E. Urinary tract infection.

BPH
Common in men > 50 y/o. Hyperplasia (No hypertrophy) of the prostate gland. May be due to an age- related ↑ in estradiol with possible sensitization of the prostate to growth-promoting effects of DHT. Characterized by a nodular enlargement of the periurethral (lateral and middle) lobes, which compress the urethra into a vertical slit. Often present with ↑ frequency of urination, nocturia, difficulty starting and stopping the stream of urine, and dysuria. May lead to distention and hypertrophy of the bladder, hydronephrosis, and UTIs. Not considered a premalignant lesion. ↑PSA. Tx : α1 antagonists, (Terazosin, Tamsulosin), which cause relaxation o

22. G. 810/900 = 90%

Specificity: TN / (TN+ FP)
Specificity= 810/ (90+ 810) = 0.9 (90%)

23. B. Cell-mediated

It has been 40 days since since his bone marrow transplantation..hence, it has to be a delayted T-cell mediated hypersensitivity.

Hyperacute Rejection: antibody mediated type II due to presence of presence of performed antidonor antibodies in the transplant recipient. Occurs within minutes after transplantation.

Acute Rejection: Cell mediated due to cytotoxic T lymphocytes reacting against foreign MHCs. Occurs weeks after transplantation. Reversible with immunosuppressant such as cyclosporine and OKT3. (FA page 213)

Chronic rejection: T cell- antibody mediated vascular damage (obliterative vascular fibrosis), occurs months to years after transplantation. Irreversible.

GRAF -VERSUS-HOST DISEASE. GRAFTED IMMUNOCOMPETENT T CELLS PROLIFERATE IN THE RADIATED IMMUNOCOMPROMISED HOST AND REJECT CELLS WITH " FOREINGN" PROTEINS, RESULTING IN SEVERE ORGAN DYSFUNCTION. MAJOR SYMPTOMS INCLUDE A MACULARPAPULAR RASH, JAUNDICE. hepatomegal and diarrhea. (FA page 213)

24. B. Bipolar disorder

manic episode

Mania = elevated mood, which can take the form of euphoria; increase in energy and a decreased need for sleep, with many often getting as little as 3 or 4 hours of sleep per night, while others can go days without sleeping; pressured speech, with thoughts experienced as racing; Attention span is low, and a person in a manic state may be easily distracted. Judgment may become impaired; engage in behavior that is quite abnormal for them. They may indulge in substance abuse, particularly alcohol or other depressants, cocaine or other stimulants. Their behavior may become aggressive, intolerant, or intrusive. People may feel out of control or unstoppable, or as if they have been "chosen" and are "on a special mission" or have other grandiose or delusional ideas. Sexual drive may increase. At more extreme phases of bipolar I, a person in a manic state can begin to experience psychosis, or a break with reality, where thinking is affected along with mood.

25. A. Osteoblast activity at the fracture
= increased osteoblastic activity in response to the stress fracture.

26. D. Increase in erythrocyte 2,3-bisphoglycerate concentration

As the blood circulates to body tissues in which the partial pressure of oxygen is less, the hemoglobin releases the oxygen into the tissue because the hemoglobin cannot maintain its full bound capacity of oxygen in the presence of lower oxygen partial pressures.

A rightward shift, by definition, causes a decrease in the affinity of hemoglobin for oxygen. This makes it harder for the hemoglobin to bind to oxygen (requiring a higher partial pressure to achieve the same oxygen saturation), but it makes it easier for the hemoglobin to release bound oxygen.

Right shift = CADET face right:
- CO2
- acid and altitude
- 2,3 DPG
- exercise
- temperature

2,3-DPG, is an organophosphate, which are created in erythrocytes during glycolysis. The production of 2,3-DPG is likely an important adaptive mechanism, because the production increases for several conditions in the presence of diminished peripheral tissue O2 availability, such as hypoxemia, chronic lung disease, anemia, and congestive heart failure, among others. High levels of 2,3-DPG shift the curve to the right, while low levels of 2,3-DPG cause a leftward shift, seen in states such as septic shock and hypophosphatemia.

27. D. The greater diversity of epitopes recognized by the polyclonal antisera permits identification of strains not recognized by the monoclonal antibody

Polyclonal antibodies (or antisera) are antibodies that are obtained from different B cell resources. They are a combination of immunoglobulin molecules secreted against a specific antigen, each identifying a different epitope.
These antibodies are typically produced by immunization of a suitable mammal, such as a mouse, rabbit or goat. Larger mammals are often preferred as the amount of serum that can be collected is greater. An antigen is injected into the mammal. This induces the B-lymphocytes to produce IgG immunoglobulins specific for the antigen. This polyclonal IgG is purified from the mammal's serum.

By contrast, monoclonal antibodies are derived from a single cell line.
http://en.wikipedia.org/wiki/Polyclonal_antibodies

Monoclonal is from IgG vs. Polyclonal comes from variety of immunologin therefore it can recognizes a lot of things.

28. B. Filgrastim

Filgrastim is a granulocyte colony-stimulating factor (G-CSF) analog used to stimulate the proliferation and differentiation of granulocytes.[1] It is produced by recombinant DNA technology. The gene for human granulocyte colony-stimulating factor is inserted into the genetic material of Escherichia coli. The G-CSF then produced by E. coli is only slightly different from G-CSF naturally made in humans.
Therapeutic uses
Filgrastim is used to treat neutropenia[2] (a low number of neutrophils), stimulating the bone marrow to increase production of neutrophils. Causes of neutropenia include chemotherapy and bone marrow transplantation.
Filgrastim is also used to increase the number of hematopoietic stem cells in the blood before collection by leukapheresis for use in hematopoietic stem cell transplantation. It is produced by many companies worldwide.

29. C. Pleomorphic epithelial cells forming duct-line structures
this is adenocarcinoma of prostate gland... prostate gland cancer metastasizes to vertebrae via the venous plexus around it.. . B is keratin pearls of squamous cell cancer so cant be it, D is basophil u can never find a basophil in cancer, E is uniform cells, cancer is never uniform cells, they are anaplastic cells, A is agregates of plasma cell and plasma blasts this feature is of multiple myeloma
30. C. Increase sodium in the urine
Addison disease...

Metabolic acidosis (increased blood acidity), due to loss of the hormone aldosterone because sodium reabsorption in the distal tubule is linked with acid/hydrogen ion (H+) secretion. Low levels of aldosterone stimulation of the renal distal tubule leads to sodium wasting in the urine and H+ retention in the serum.
31. A. Capsule
Haemophilus influenzae type B....

The Hib conjugate vaccine is an inactivated vaccine. It is made by chemically bonding a polysaccharide (sugar) to a protein. This long chain of sugar molecules makes up the surface capsule of the bacterium.
32. E. Synchronized discharge of thalamocortical neurons

Absence seizures or petit mal epilepsy

There is a correlation between SLOW WAVE SLEEP (stage 3 and 4 aka delta sleep) and absence seizures ....one of the correlation is Slow-wave sleep as well as generalized absence seizures are characterized by the occurrence of synchronized oscillations in thalamocortical systems that spontaneously appear and disappear.

What are this synchronized oscillations ?

- Oscillations =Remember from high school physics ...if u put a load to a spring it oscillates ....the same thing neurons oscillate (another term is repetitive variation) ...which is called neural oscillation

Neural oscillation is rhythmic or repetitive neural activity in the central nervous system .And this can be due to rhythmic increases and decreases in action potential activity, which then produce rhythmic activation of synapses in target neurons.

This oscillations can be physiological or pathological.

Pathological oscillations = Specific types of neural oscillations may also appear in pathological situations, such as Parkinson's disease or epilepsy. Interestingly, these pathological oscillations often consist of an aberrant version of a normal oscillation. For example, one of the best known types is the spike and wave oscillation (synchronized oscillation) , which is typical of generalized or absence epileptic seizures, and which resembles normal sleep spindle oscillations.
33. C. Direct branch from the aorta

Gonadal vessels come from the abdominal aorta

Blood supply to the testis primarily originates from the testicular artery, which arises from the aorta. Other sources of blood supply include the deferential artery, which supplies the epididymis and the vas deferens and the cremasteric artery supplies the peritesticular tissues.
The testicular artery (the male gonadal artery, also called the internal spermatic arteries in older texts) is a branch of the abdominal aorta that supplies blood to the testis. It is a paired artery, with one for each of the testes.

It is the male equivalent of the ovarian artery.

http://en.wikipedia.org/wiki/File:Gray531.png
34. F. 60%

There are 15 CAG gene number of CAG repeats..hence, it is at the 60% disease risk

I go with 60 % ...for the following reasons

1-Autosomal dominant diseases occurrence is affected by many things ...among those is the number of trinucleotide repeats in the proband...also variable penetrance ...and etc.....
Which means AD diseases are not always shown up 100 %

2-Clearly the question gives us the trinucleotide repeat expansion peneterance figures

and for 15 Trinucleotide Repeat Expansion the risk is 60 % ....

Huntington's Disease is an AD disease which means that an affected individual TYPICALLY inherits a defective gene from an AFFECTED parent. If the parent has a trinucleotide repeat count that is normal (40 (full penetrance).

Back to the question. it can be any trinuc expansion disorder. But used Hunt. disease to illustrate some points. First, risk doesn't necessarily reflect that the parent has the disorder but the parent can be an unaffected carrier with trinuc expansions that will not result in full penetrance. Second, the graph is very important. It shows that individuals inheriting a gene with a trinuc expan. of 20 have a 100% risk of the disease (full penetrance), as well as those with CAG repeats of 9 or less have 0% risk. The curve represents reduced penetrance (where some individuals fail to exhibit the trait even though they carry the abnormal allele. As the number of CAG repeats increase there is an increase in the percentage of individuals at risk of the disease. This implies that penetrance is increasing up to point where it becomes full or complete (all individuals who have the abnormal allele will manifest (signs/symptoms) the disease. The answer that best fits the curve is 60%

35. C. Incubation under strict anaerobic conditions

Clostridium perfringens is a Gram-positive, rod-shaped, anaerobic, spore-forming bacterium of the genus Clostridium. C. perfringens is ever present in nature and can be found as a

normal component of decaying vegetation, marine sediment, the intestinal tract of humans and other vertebrates, insects, and soil.

Clostridium perfringens is the most common bacterial agent for gas gangrene, which is necrosis, putrefaction of tissues, and gas production. It is caused primarily by Clostridium perfringens alpha toxin. The gases form bubbles in muscle (crepitus) and the characteristic smell in decomposing tissue

In the United Kingdom and United States, C. perfringens bacteria are the third-most-common cause of food-borne illness, with poorly prepared meat and poultry the main culprits in harboring the bacterium. The clostridium perfringens enterotoxin (CPE) mediating the disease is heat-labile (inactivated at 74 °C) and can be detected in contaminated food, if not heated properly, and feces .

36. D.

graph A: LH
graph B: FSH
graph C: progesterone

37. C. Plasma volume

This patient is taking exogenously Na+, then, you drink lots of water, then you will dilute the plasma and increase in plasma volume.

High sodium intake increases body weight , plasma volume, cardiac index , and stroke volume index.

In the body, sodium is processed by the kidneys. However, when a person eats too much sodium, the kidneys cannot process all of it. The excess sodium ends up in the bloodstream. Because the mineral retains water, the volume of blood in the body increases. As a result, the circulatory system has to work harder to pump the blood. Over time, this added strain on the system can result in heart disease and kidney failure

38. D. Increase K+ excretion
Patient had CHF, "LVF", always if pulmonary edema LHF the main symptom the SOB
Can't get blood out of the heart b/c the LV fails, increased the EDV because all the blood can not get out, then the pressure and volume will go back to the left atrium, back into the pulmonary vessels, increased the hydrostatic pressure and then PULMONARY EDEMA

What happen in the CHF?: Decreased the cardia output regulated by RAA System like the Blood pressure, the renal perfusion is decreased then the renin increases---- renin convert angiotensinogen into angiotensin I. ACE found mainly in endotelial cells of pulmonary vessels, converts angiotensin I into angiotensin II. Angiotensin II has a potent effects to stimulate secretion of aldosterone and to cause arteriolar vasoconstriction. stimulates reabsorption of Na+ and ALSO Cause increased renal excretion of potassium affecting the plasma K_ concentration
39. D. Inhibition of prostacyclin (PGI2) formation without inhibition of thromboxane A2 in platelets
http://www.medicalnewstoday.com/releases/38786.php

Prostacyclin (PGI2) chiefly prevents formation of the platelet plug involved in primary hemostasis (a part of blood clot formation). It does this by inhibiting platelet activation.[4] It is also an effective vasodilator. Prostacyclin's interactions in contrast to thromboxane (TXA2), another eicosanoid, strongly suggest a mechanism of cardiovascular homeostasis between the two hormones in relation to vascular damage
40. D. Medial longitudinal fasciculus

The MLF carries information about the direction that the eyes should move.

It yokes the cranial nerve nuclei III (Oculomotor nerve), IV (Trochlear nerve) and VI (Abducens nerve) together, and integrates movements directed by the gaze centers (frontal eye field) and information about head movement (from cranial nerve VIII, Vestibulocochlear nerve). It is an integral component of saccadic eye movements as well as vestibulo-ocular and optokinetic reflexes.

It also carries the descending tectospinal tract and medial vestibulospinal tracts into the cervical spinal cord, and innervates some muscles of the neck and upper limbs

Lesions of the MLF produce internuclear ophthalmoplegia and can be a presenting symptom of multiple sclerosis where it presents as nystagmus and occasionally diplopia.These lesions cause damage to the ipsilateral (same side) eye, but nystagmus on the contralateral (opposite side) eye.
Horizontal gaze to the right results from activation of the right abducens nucleus and the left oculomotor nucleus by fibers in the MLF. Lesions in the MLF results in an internuclear opthalmoplegia, like above explained by maryam, then there is an inability to adduct one eye on attempted gaze to the opposite side. For example a lesion in the right MLF results in an inability to adduct the right eye on attempted gaze to the left (see figure IV-5-10 Voluntary Horizontal Conjugate gaze) in Kaplan
41. B. Enlarging subcarina lymph node
- Explanations?

42. B. Lingual thyroid
Lingual thyroid is a RARE condition, with an incidence of 1:100,000. This infrequent congenital anomaly is often asymptomatic until a pathologic stress such as systemic disease or physiologic stress such as puberty causes enlargement of the ectopic tissue, leading to dysphagia, dysphonia, and dyspnea. The work-up should include routine blood work including thyroid function tests thyrotropin, thyroxine, and thyroid hormone binding ratio; iodine thyroid scintigraphy; and computerized tomography or magnetic resonance imaging. The majority of patients require surgical excision of the symptomatic mass and, in case of absence of orthotopic thyroid tissue, long-term thyroid hormone replacement.

http://onlinelibrary.wiley.com/doi/10.1097/MLG.0b013e31816f6922/abstract?syst...maintenance
43. A. Ask the patient if he is concerned about the appearance of his skin

Acne vulgaris (or acne) is a common human skin disease, characterized by areas of skin with seborrhea (scaly red skin), comedones (blackheads and whiteheads), papules (pinheads), pustules (pimples), nodules (large papules) and possibly scarring.[1] Acne affects mostly skin with the densest population of sebaceous follicles; these areas include the face, the upper part of the chest, and the back. Severe acne is inflammatory, but acne can also manifest in noninflammatory forms.[2] The lesions are caused by changes in pilosebaceous units, skin structures consisting of a hair follicle and its associated sebaceous gland, changes that require androgen stimulation.

http://en.wikipedia.org/wiki/Acne_vulgaris

44. B. DNA synthesis

Folate defficiency: megaloblastic anemia (PMN nucleus more that 5 lobes as is seen in the fig)
THF is formed from the vitamin Folate through 2 reductions catalyzed by DHF reductase. It picks up 1- carbon unit from a variety of donors and enters the active 1- carbon pool. Important pathways requiring forms of THF from this pool include the synthesis of all purines and thymidine, which in turn are used for DNA and RNA synthesis during cell growth and division. (Kaplan Bioch.book page 268).

45. B. Cholelithiasia

Gemfibrozil (fibrate): ↑HDL, ↓LDL, ↓ TG. Mech of action: Upregulate LPL, ↑TG clearance.
Side effects: Myositis, Hepatotoxicity (↑ LFTs), Cholesterol Gallstones (FA page 278)

46. C. Generation of reactive oxygen specieis
Reactive Oxygen Species (ROS): When molecular O2 is partially reduced, unstable products, called (ROS) are formed. These react rapidly, with lipids to cause, peroxidation, with proteins, and with other substrates, resulting in denaturation and precipitation in tissues.
ROS include: Superoxide, Hydrogen peroxide, Hydroxyl radical.
The rate of ROS production can increase dramatically under certain conditions, such as reperfusion injury in a tissue that has been temporally deprived of oxygen. ATP levels will be low and NADH levels high in a tissue deprived of O2 (as in an MI).

47. C. Musculocutaneous

48. A. Coartation of the aorta
Coartation of the aorta: Infantil type (preductal) aortic stenosis proximal to insertion of ductus arteriosus.
Adult type: (postductal) stenosis is distal to ductus arteriosus.
Associated with notching ribs (due to collateral circulation), hypertension in upper extremities, weak pulses in lower extremities. Associated with Turner syndrome.

Symptoms may be absent with mild narrowings (coarctation). When present, they include: difficulty breathing, poor appetite or trouble feeding, failure to thrive. Later on, children may develop symptoms related to problems with blood flow and an enlarged heart. They may experience dizziness or shortness of breath, faint or near-fainting episodes, chest pain, abnormal tiredness or fatigue, headaches, or nosebleeds. They may have cold legs and feet or have pain in their legs with exercise (intermittent claudication) Wikipedia,
to add ;Clinical sings proximal to the constriction---- increased upper ext BP, dilatation of aorta and aorta valve ring (regurgitation) increased risk for developing an aorta dissection, increased cerebral blood flow (increased risk for berry aneurysm.
Clinical signs distal to the constriction; ----dicreased blood pressure in the lower extremity, leg claudication(pain in calf or bottocks when walking, dicreased renal blod flow with activates RAA system causing the phisiophathology of the hepertension

49. D. Herniation of the uncus
the patient had and epidural hematoma, first lost the consciusness and then get back in ER, dilated pupil in the right secondary to the compression of the uncus and squeeze the III nerve oculomotor affecting the parasympatetic input to the right eye, cause of the dilatation.
to add: Epidural hematoma (EDH) is a rapidly accumulating hematoma between the dura mater and the cranium. These patients have a history of head trauma with loss of consciousness, then a lucid period, followed by loss of consciousness. Clinical onset occurs over minutes to hours. Many of these injuries are associated with lacerations of the middle meningeal artery. A "lenticular", or convex, lens-shaped extracerebral hemorrhage will likely be visible on a CT scan of the head. Although death is a potential complication, the prognosis is good when this injury is recognized and treated. http://www.reference.com/browse/uncal
to add:In uncal herniation, a common subtype of transtentorial herniation, the innermost part of the temporal lobe, the uncus, can be squeezed so much that it goes by the tentorium and puts pressure on the brainstem, most notably the midbrain.[5] The tentorium is a structure within the skull formed by the meningeal layer of the dura mater. Tissue may be stripped from the cerebral cortex in a process called decortication.[6]

The uncus can squeeze the third cranial nerve, which may affect the parasympathetic input to the eye on the side of the affected nerve, causing the pupil of the affected eye to dilate and fail to constrict in response to light as it should. Pupillary dilation often precedes the somatic motor effects of cranial nerve III compression, which present as deviation of the eye to a "down and out" position due to loss of innervation to all ocular motility muscles except for the lateral rectus (innervated by cranial nerve VI) and the superior oblique (innervated by cranial nerve IV). The symptoms occur in this order because the parasympathetic fibers surround the motor fibers of CNIII and are hence compressed first.

Compression of the ipsilateral posterior cerebral artery will result in ischemia of the ipsilateral primary visual cortex and contralateral visual field deficits in both eyes (contralateral homonymous hemianopsia (Wikiped
In uncal herniation, a common subtype of transtentorial herniation, the innermost part of the temporal lobe, the uncus, can be squeezed so much that it goes by the tentorium and puts pressure on the brainstem, most notably the midbrain.[5] The tentorium is a structure within the skull formed by the meningeal layer of the dura mater. Tissue may be stripped from the cerebral cortex in a process called decortication.[6]

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Compression of the ipsilateral posterior cerebral artery will result in ischemia of the ipsilateral primary visual cortex and contralateral visual field deficits in both eyes (contralateral homonymous hemianopsia).
Another important finding is a false localizing sign, the so called Kernohan's notch, which results from compression of the contralateral cerebral crus containing descending corticospinal and some corticobulbar tract fibers. This leads to ipsilateral (same side as herniation) hemiparesis. Since the corticospinal tract predominately innervates flexor muscles, extension of the leg may also be seen. With increasing pressure and progression of the hernia there will be distortion of the brainstem leading to Duret hemorrhages (tearing of small vessels in the parenchyma) in the median and paramedian zones of the mesencephalon and pons. The rupture of these vessels leads to linear or flamed shaped hemorrhages. The disrupted brainstem can lead to decorticate posture, respiratory center depression and death. Other possibilities resulting from brain stem distortion include lethargy, slow heart rate, and pupil dilation.[6] Uncal herniation may advance to central herniation.[4]

A complication of an uncal herniation is a Duret hemorrhage. This results in the midbrain and pons being compressed, possibly causing damage to the reticular formation. If untreated, death will ensue.
http://en.wikipedia.org/wiki/Brain_herniation
50. C. Δ G: -4.7; Direction: 1

Δ G < 0 Thermodynamically spontaneous (energy released often irreversible)
Δ G > 0 Thermodynamically nonspontaneous (energy required)
$\Delta G^{\circ} = 0$ energy involved under standarizer conditions
(Kaplan Bioch. Book page 122)

A →← B + C Δ G = -11.1 kcal/mol spontaneous/exothermic
C + D →← E Δ G = +6.4 kcal/mol nonsponatenous/

Calculating the change in G for the third reaction: -11.1 + 6.4 = -4.7

Interpreting the change in G = -4.7 (the reaction is spontaneous and the reaction of the reaction is from left to right…from reactants to products….hence…1)