1) Trendelenberg test is positive due to…..?
2) Notochord exists as….?
3) Neuroglia….?
4) Hyaline Arteriosclerosis seen in …..?
5) Which Ig crosses through placenta …..?
6) M.C cardiac lesion seen in pregnancy ……?
7) M.C valve disease due to M.I ? (Sorry, I forgot the question. It was asked in similarly) Herd Immunity?
9) Koplik's spots seen in ……?
10) A ? patient with hypertension & suffering from Thyroiditis, DOC ……?
11) Hypocalcemia is seen with the following?
12) Most common position of Uterus?
13) Shortest ø of pelvis is……?
14) Right base of the heart is formed by …..?
15) Right side Mid calvicle the lung ends at which rib?
16) Water supply in hilly areas?
17) Prophylaxis DOC for Meningitis?
1 Anaemia is seen with all except?
19) Dose of Folic acid during pregnancy? (prophylaxis dose)
20) Vitamin "A" dosage is given in….? (?. of doses.)
21) What is Apoptosis?
22) Extrinsic factor in blood coagulation? (PT/PTT)
23) Curschmann's spirals are due to?
24) Bell's palsy?
25) In Dialysis which toxicity is seen commonly?
26) TOC for Gastric ulcer?
27) Squamous non-keratinizing is seen in….? a) Tongue b) Trachea c) Oesophagus d) Vagina
2 Tumour marker CA-125 is related to pancreatic Ca &…….?
29) Bilaterally kidneys are shrunken in …….?
30) Poisoning due to _____preserved are hair, Etc..etc….?
31) The hormone helps in milk secretion?
32) Most abundant ICF is …….?
33) Features like-Hypogonadism, Loss of Hair, Pigmentation of skin Etc..etc Deficiency due to?
34) Most common features of alcohol withdrawl?
35) Negri bodies are characteristic of ?
36) M.C opportunistic Infection in immuno compromised patient?
37) Mallory-weiss syndrome? (Mallory bodies seen with….?)
3 Sickness benefit under ESI Act is given for the following illness?
39) Glucose is reabsorbed at?
40) M.C pemphigus seen in India?

41) India ia at which stage of Demographic?
42) Urinary incontinence in Older people is due to……?
43) Breast cancer is due to all, Except?

44) Black & white colour vision is due to ……?
45) Grey colour……? (Extremely sorry I forgot what was asked ,{Ophthalmology Q} if any one can remember please ADD)
46) Kussmaul breathing is due to or seen in ……?
47) Cellulitis is caused by…….?
4 Auer rods are seen in ……?
49) Gynaecomastia is due to drugs……..?
50) In a new born Jaundice occurs on 3-5days;its not due to …….?
51) Transmitted by faeco-orally, Except ?
52) ß-Thalassemia inherited as ……..?
53) Foreign body inhaled usually lodges in which lung?
54) Change in blood viscosity causes ……?
55) * Question regarding Dentition? “Eruption”
56) Food poisoning 4-6 hrs organism responsible ……..?
57) Dreaming is common in which type of sleep?
5 Second heart sound is due to ……..?
59) “SAFE” ; ‘S’-stands for ……..?
60) Long term status of blood sugar explained by …….? (Ans: HbA1c ; The way the question was formed is different, any way the matter is most Important.)
61) Hyperglycemic drugs reduces weight ……..?
62) Surfactant is formed from which type of cells?
63) Spleenectomy is helpful in ……..?
64) Incineration done for which of the following?
65) Anti-gliadin antibodies are seen in?

) The following are example of Apootosis Except-
a) Graft versus host disease
b) Menstrual cycle
c) Pathological atrophy following duct obstruction
d) Tumour necrosis

2) The normal tensile strength of tissue at the site of wound is gained after:
a) 1 week of wound healing
b) 2 weeks of wound healing
c) 2 months of wound healing
d) 2 years of wound healing

Ths best test for BEST Disrase

Best disease is characterized by a striking accumulation of lipofuscin-like material in the macula that often results in an "egg-yolk-like" appearance. In addition, patients affected with Best's disease display an abnormal electrophysiologic sign known as a depressed Arden ratio. The electro-oculogram (EOG) is a measurement of the electric potential that

normally exists across the retinal pigment epithelium. This potential normally doubles in response to bright light. However, in Best's disease, this increase does not exist. This test can be used to diagnose patients without classic macular lesions, as well as identifying patients that are unlikely to have the disease.

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qs of sept 2008 paper

21 trisomy asso. with
ALL
CLL
AML
CML

2) esophagus length
40
25
15
30

3)pre malignant cond. of esophagus
barrets

4)length of external aud. canal cartilagenous part
8
16
24
12

5)max. Na absorption at
PCT
DCT
LOH
CT

6)SUNRAY apearance on x ray
osteoclastoma
osteoblastoma
osteosarcoma
chondroblastoma

7)m\c tumor in spine
sec.
ewings sarcoma

oteosarcoma
m. myeloma

punched out lesion in skull
ewings sarcoma
m.myeloma
sec.
oteosarcoma
9)m\c reason for bradycardia in MI
septal MI
right vent. MI
left ventricular MI

10) S1 split seen in
RBBB
?
?
?

11)snow flake cat. (from prev.papers)

12)after injury to one eye other aslo worsen
glucoma
cat.
sym. opthalmia
?

13)qs from placenta abroptia ...bleeding per vagina tender and hard

very few qs from Obs

14) 60 year old man with left hydroceal + ???

ans. nephroma

15)lateral epicondyal fac.
non union
tardy ulnar nerve palsy
?
all

16)m\c parasitic infection in AIDS
strongiloids

17)toxin responsibel for TSS in femals
exo toxin

! in stap. aures food poisoning diarrhea occur due 2
endotoxin
vagus
exotoxin
?

19)pheochromocytoma diagnosis
24 hours urine metabolites VMA+ CA
MIBG
CT scan
surgery

20) what we use for thyroid scan
I 131

Q)DRUG WHICH CAUSES REVERSIBLE GYNECOMASTIA
-CIMETEDINE
-OMEPRAZOLE
Q)DOC FOR PROPHYLAXIS OF MENINGOCOCCAL MENINGITIS--RIFAMPIN
Q)A PT WITH THROMBOCYTOPENIA.1ST IOC
*-BLEEDING TIME
-PLATELET COUNT
-PROTHROMBIN TIME
Q)MC CAUSE OF SOLITARY THYROID NODULE
-FOLLICULAR ADENOMA
Q)TUMOR MARKER4BOTH PANCREATIC &COLON CA
-CA125
-CA19
Q)A PT VID AN INFERIOR WALL MI IN SHOCK.REASON?
ANS-RIGHT VENTRICULAR INFARCTION
Q)ALL R FEATURES OF ATRIAL MYXOMA EXCEPT
-FEVER
-CLUBBING
-EMBOLI
*HYPERTENSION
Q)DOC IN SVT--ADENOSINE
Q)A FEMALE PT HAS CHEST PAIN (NON EXERTIONAL)
AUSCULTATION--MULTIPLE NON EJECTION ? ..IOC?
*ECHO
Q)A PT PESENTS WITH MI.EARLEST MARKER?
CK-MB
TROP-T
MYOGLOBIN
Q)STAPH AUREUS FOOD POISONING-CAUSE OF NAUSEA?

ANS--DIRECT VAGAL STIMULATION
Q)A Q ON WEGENERS GRANULOMATOSIS
Q)A PT ON TPN.WHAT COULD BE THE CAUSE OF MORTALITY IN THAT PT?
OPTIONS CANT RECALL,BUT I MARKED INFECTIONS DUE2CENTRAL LINE
Q)A Q ON APGAR SCORE
Q)A Q ON ARDS--DIAGNOSTIC CRITERIA

some more recalls!

Q)A PT WITH BULBAR URETRAL RUPTURE.UR 1ST MN: WUD BE
--SUPRAPUBIC CYSTOSTOMY
--FOLEY'S
--CONSERVATIVE MN
--REFER2UROLOGIST

Q)MJ MUSCLE FOR EYE INTORSION?

Q)ERYTHRODERMA IS ASSOCIATED WID A/E
--LEPROMATOUS LEPROSY
--AIR BORNE DERMATITIS

Q)DERMATITIS HERPETIFORMIS IS A/W
*ULCERATIVE COLITIS

Q)MC TYPE OF PEMPHIGUS IN INDIA?
*P.VULGARIS

Q)LA SAFE IN RF?
GALLAMINE

Q)A PT WID MYASTHENIA GRAVIS IS RESISTANT2
--*DEPOLARISING MR
--NON DEPOLARISING MR
--BOTH
--NONE

Q)A PSM Q 2FIND OUT RELATIVE RISK

Q)ANOTHER PSM Q ON PANEL DISCUSSION..Q WAS AS2VAT WAS IT ABOUT

Q)A Q ON SARCOIDOSIS(PATHO)

Q)WHICH ANTIBODY HAS BEST4 CELIAC DISEASE(SENSITIVE &SPECIFIC)?
*ANTI ENDOMYSIAL AB

Q)BEST PROGNOSTIC FACTOR 4 A/C PANCREATITIS
-*S.LIPASE

Q)A Q ON ZES--WHICH IS NOT TRUE
ONE OF THE OPTION READ--REDUCED BAO:MAO WHICH S D ANS

Q)AN OHA WHICH IS USED 2TREAT OBESITY
ANS WAS SUM BIGUANIDE GIVEN IN THE OPTIONS

Q)ANOTHER Q ON DIAB DRUG
WHICH ONE DOES'NT CAUSE HYPOGLYCEMIA
(SORRY,CANT RECALL OPTIONS)

BUT FRIENDS,,NEXT TIME DO READ A BIT ABOUT ORAL HYPOGLYCEMICS AS WELL AS SOME IMP BITS ABOUT ANTI-OBESITY DRUGS
(SEEMS NAT BOARD HAS LOST IT'S LONG LASTED LOVE 4 MALARIA,TB,N OTHER PARASITIC INFECTIONS!NOT A SINGLE Q WAS ASKED!!))

Q)DRUG USED4TREMORS IN HYPERTHYROIDISM?
--PROPRANOLOL

Q)AN EASY Q--WHICH CAUSES HYPOCALCEMIA?
ANS WAS CALCITONIN

Q)A PT ON TPN FOR A WEEK DEVELOPS FEATURES((WHICH WAS SUGGESTIVE OF ZN DEFICIENCY))..DEFICIENCY OF VAT?

Q)A Q ON NEPHROTIC SYNDROME IN CHILDREN....

Q)HYALINE ARTERIOSCLEROSIS IS SEEN IN
*BENIGN HTN

some more questions frm FMGE sept 2008

1-ring shaped ulcers seen in?
2-neurotrophic keratitis-nerve involved
3-question on sympathetic ophthalmia
4-extra capsular cataract surgery-parts of lens which are excised?

5- SAFE strategy for trachoma-S stands for?
6-rubro iridis is not seen in?
7-questions on cost benefit and cost accounting in PSM
8-central tendancy seen in-(refer biostats PSM)
9-question on odds ratio
10-complication of measles virus -encephalomelyitis
some more-
NNN Media used for- Ans-leshmania donavani
Treatment of first degree testicular cancer
Fourniers (sorry for d spelling,I don remember exactly!) gangrene seen in- Ans scrotum
Treatment Regimen for hodkgins lymphoma
Shape of tracheal cartilage-ans-horse shoe shape
Comonest congenital anomaly of trachea
Erythoderma is not seen in-?
Commonest lupus in india-
Fate of notochord-?
Mysanthia gravis is not inhibited by-depolarizing agents,non depolarizing agents,both,?
Rarest form of opportunistic fungal infection seen in AIDS-
White line of frenkel seen in-ans Scurvy
Xray sign of rickets
Splenomegaly not seen in-?
The first clinical presentation of acoustic neuroma-ans-facial nerve involvement
Stones r seen most commonly in submandibular salivary gland
The calories required by one year old child
4-5 questions on Tracheostomy
Sunray sign on xray seen in-?osteosarcoma
Tardy ulnar palsy seen in-?
Rigor mortis is due to-?
Vitreous hemorrhage on autopsy seen in poisoning of-?
One question on [bleep]-Refer Forensic Medicin
Most specific method of diagnosis of kalazar

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questions

Q.a patient with tb had decrease SERUM level of Na and k..... and has hyperpigmentation of crease of hand??...
ans.. secondary hyperaldeosteroism.
Q.MC. CAUSE OF DEATH IN INDIA?
ANS. CORONAY HEART DISEASE.
Q.CXR AP VIEWON RIGHT SIDE OF CARDIAC SILHOUTE U CAN SEE ALL EXCEPT?
ANS. SUPERIOR VENA CAVA
Q.NORMAL TO INCREASE SIZE OF KIDNEY IS SEEN ALL EXCEPT?

ANS. *CHRONOC GN
DIABETIC NEPHROPATHY
AIDS REALTED NEPHROPATHY
PKD
Q.SPLEEN IS SUPPORT BY WHICH LIGAMNET FROM UPPER SIDE?
ANS. GASTROSPLENIC LIGAMENT.
Q.STAIN FOR AMLYDOSIS
ANS. CONGO RED
Q.AUER RODS ARE SEEN IN?
AN. AML
Q.WHICH TUMOR IS MC IN DOWN SYNDROME?\
ANS MAY BE RETINOBLASTOMA (PLZ MAKE SURE FROM BOOK)

1.Trendelenburg's test

Also known as:
Brodie-Trendelenburg test
Trendelenburg-Brodie test

Associated persons:
Sir Benjamin Collins Brodie
Friedrich Trendelenburg

Description:

Test for varicose veins. Patient lies on his back and raises his leg to empty the veins. A tourniquet is applied just below the saphenous opening. The patient is then stood up and the tourniquet removed in 60 seconds. Normally the vein should fill from below within 35 seconds with the tourniquet in situ. Earlier filling indicates incompetence of a communicating vein. If on release the veins fill rapidly from above it is due to incompetent sapheno-femoral valves.

2. Trendelenburg's test

Trendelenburg's test is a test of the saphenous and other veins.[1]
It is named for Friedrich Trendelenburg.[2][3]
It should not be confused with Trendelenburg's sign, which involves the muscles of the hip.

Trendelenburg's sign:

Trendelenburg's sign is found in people with weak abductor muscles of the hip. It is named after the German surgeon Friedrich Trendelenburg.
The Trendelenburg sign is said to be positive if, when standing on one leg, the pelvis drops on the side opposite to the stance leg. The weakness is present on the side of the stance leg. The body is not able to maintain the center of gravity on the side of the stance leg. Normally, the body shifts the weight to the stance leg, allowing the shift of the center of gravity and consequently stabilizing or balancing the body. However, in this scenario, when the patient/person lifts the opposing leg, the shift is not created and the patient/person cannot maintain balance leading to instability.

Essentially, Trendelenburg sign is caused by paralysis of the gluteus medius and minimus muscles.

Paralysis may arise due to nerve damage, namely, the superior gluteal nerve
---

1.
Trendelenburg test is a standard clinical assessment of hip stability.

A positive test indicates gluteus medius weaknessand is observed when weight is supported by the affected limband the pelvis on the healthy side falls instead of rises.

This test was used in this study to compare the two approaches. Incases of minimal abductor weakness there may be a delayedpositive test. It is for this reason that an element of musclefatigue was taken into account by considering the pelvicposition at 30 seconds of single leg stance. Since gait analysisallows subtle differences to be detected compared to clinicalanalysis, it was hoped this test would be an effective measureto compare the two approaches

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2.

The notochord exists transiently during the life of most vertebrates.

A notochord is the defining characteristic of members of the phylum Chordata, a large and diverse biological group which includes all animals with spines, or backbones, along with more primitive chordates. The notochord has very large cells which are densely arranged within a protective sheath. Chordates are divided into three subphyla, depending on what form their notochords take and when they appear. At any developmental stage, a notochord acts as a form of support for the animal that possesses it, giving animals the ability to do things such as walking upright.

The most primitive group of chordates, urochordates, also called tunicates, only have a notochord in the larval stages of development. The animals in this group are pelagic,

meaning that they are found in the world's oceans, and there are a number of representative species, most of whom are only known to biologists. These animals do provide an insight into the development of chordates, however, showing the notochord at an early stage of development.

The next group of chordates, cephalochordates, also called lancelets, possess a notochord into adulthood, and also live in the ocean. The notochord runs all the way along the body, even up into the head, and the animals lack a protective layer of bone such as a spine. The notochord acts as an axial support, providing a strong core for the animal. The notochord is highly flexible, but not compressible, allowing the animal to move freely without damaging the notochord.

In the highest class of chordates, the vertebrates, the notochord only exists when the animal is in an embryonic form. As the vertebrate develops, the notochord is first ensheathed in and then replaced by spinal vertebrae, protective cases of bone which cover the delicate spinal cord. The spinal column is able to support a much larger and more complex organism, and is much stronger than the notochord. This allowed early vertebrates to make the leap to the land and develop into well known species such as humans.

In all chordates, the notochord exists in some form when the animal is in a larval or embryonic stage[/size]. The development of the notochord from there helps shape what sort of animal it will turn into, whether it be a sac like tunicate or a pure bred Norwegian Fjord horse. The development of the notochord represents a major advance in evolution, as it allowed animals to get much larger and more complex, a drastic departure from simpler orders of animals which existed previously.

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3. NeuroGLIA

Glial cells, commonly called neuroglia or simply glia (Greek for "glue"), are non-neuronal cells that provide support and nutrition, maintain homeostasis, form myelin, and participate in signal transmission in the nervous system. In the human brain, glia are estimated to outnumber neurons by about 10 to 1.

Glial cells provide support and protection for neurons, the other main type of cell in the nervous system. They are thus known as the "glue" of the nervous system. The four main functions of glial cells are to surround neurons and hold them in place, to supply nutrients and oxygen to neurons, to insulate one neuron from another, and to destroy pathogens and remove dead neurons. They also modulate neurotransmission.

The supportive tissue of the nervous system, including the network of branched cells in the central nervous system (astrocytes, microglia, and oligodendrocytes) and the

supporting cells of the peripheral nervous system (Schwann cells and satellite cells). Also called glia, reticulum.

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4. Hyaline arteriolosclerosis:

Hyaline arteriolosclerosis can be seen in patients with diabetes mellitus and with hypertension. Hyaline arteriolosclerosis are more common in diabetic sufferers.

5. M.C cardiac lesion seen in pregnancy -->

Mitral stenosis is the most common rheumatic valvular lesion seen in pregnancy due to its prevalence in young women.

6. M.C valve disease due to ----

Mitral valve prolapse (MVP) : is the most common forms of valve disease, affecting 6 percent of all women. In this condition one or both of the valve leaflets is enlarged or floppy, preventing the valve from closing evenly. When the valve shuts, the leaflets bulge into the left atrium. You may have heard mitral valve prolapse referred to as click-murmur syndrome from the sound the valve makes when it closes.

7. Herd Immunity:

In immunology, herd immunity refers to a situation in which a high percentage of a population is immune to a disease, essentially stopping the disease in its tracks because it cannot find new hosts. You may also hear this concept referred to as “community immunity.” The threshold for herd immunity varies, depending on the disease, with more virulent agents requiring vaccination of a higher percentage of the population to crate the desired herd immunity. In addition to being used in disease prevention, community immunity is also utilized to fight ongoing outbreaks.

Most vaccination policies are focused on creating herd immunity. Many countries require vaccinations in childhood, for example, protecting children from common diseases and ensuring that when these diseases enter the population, they cannot prey on children or adults, who have been previously vaccinated or exposed to the disease. The creation of herd immunity is especially important in crowded environments which facilitate the spread of disease, like schools.

Immunologists try to prevent the outbreak of diseases by creating herd immunity, but they are not always successful. Sometimes a disease mutates or is entirely new, or a batch of vaccinations is faulty, or a large percentage of the population fails to get vaccinated, creating a situation in which an outbreak can occur, because much of the population is vulnerable. In the event of an outbreak of a major disease, agencies like the World Health

Organization can dispatch teams within days to figure out the cause of the outbreak and develop a vaccine, in the hopes of creating herd immunity to halt the outbreak in its tracks.

For some diseases, herd immunity thresholds are as low as 50%, especially when combined with good hygiene. In other instances, up to 90% of the population may need to be vaccinated to create the desired herd immunity. It is also extremely important to receive regular boosters, as some vaccines lose their efficacy over time, leaving people vulnerable to an outbreak. Herd immunity led to the eradication of smallpox, and it explains why diseases such as polio and diphtheria are rare in developed nations with established vaccination policies.

The concept of herd immunity is often used to encourage reluctant parents to vaccinate their children. In addition to ensuring that their children are protected from fully preventable diseases, childhood vaccinations also benefit society at large by creating herd immunity. Likewise, adults may be reminded to receive boosters to help protect their communities.

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9. Koplik’s spots seen in ---

Also known as: Filatov’s spots, Flindt’s spots Maculae Koplik.

Associated persons:
Nil Feodorovich Filatov
Nikolaj Flindt
Henry Koplik

Description:
Small, grain-of-sand sized, irregular, bright red spots with blue-white centres, occurring on the inside of the cheek (buccal mucosa). Seen only on measles (rubeolae) they are, by themselves, a diagnostic sign in measles. The spots usually occur briefly after the fever begins and a couple of days before the generalized rash appears. Not infrequently, the spots disappear as the eruption develops.

Koplik's spots are bluish-white spots seen on the mucous membranes of the mouth and are pathognomonic of measles.

They often appear a few days before the rash arrives and can be a useful sign to look for in children known to be exposed to the measles virus.

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10. A ? patient with hypertension & suffering from Thyroiditis, DOC --

L-Thyroxine has long been the treatment drug of choice

Goals of
therapy (Rx) To bring free T4 (L-thyroxine) and TSH levels into normal range and improve symptoms. 1st choice therapy L-thyroxine.

11. Hypocalcemia is seen with the following-->

Hypocalcemia can be seen in chronic renal insufficiency.

Hypocalcemia may be seen in a number of disorders affecting the synthesis or action of PTH or vitamin D or following sequestration of calcium into a functionally inaccessible compartment.

Many of these represent chronic illnesses where hypocalcemic symptoms develop insidiously or where the complication of hypocalcemia is anticipated early and appropriate treatment initiated prior to acute decompensation.

Hypocalcemia occurs in the setting of acute systemic illness (eg, toxic shock syndrome), a finding that has been linked to elevated free fatty acids levels in this setting. It has also been associated with specific drugs, including antineoplastic agents such as doxorubicin and cytarabine and other agents such as ketoconazole, pentamidine, and foscarnet.

12.Most common position of Uterus?
The most common position is for the long axis of the uterus to lie in approximately the same axis as the pelvis, that is, with the upper part tilted forward and the lower end, the cervix, inserted into the top of the vagina. There is therefore an angle between the uterus and the vagina, the long axis of which corresponds to the lower part of the pelvis.

13. Shortest ø of pelvis is-->
The interspinous diameter is the shortest in the pelvis.

Obstetrical Pelvic Diameters
The shape and size of the bony pelvis are important factors determining the outcome of labour. The pelvic inlet is the entrance to the true pelvis. The plane of the pelvic inlet is almost circular in a normal female or gynaecoid pelvis with a slightly greater transverse than anterior-posterior diameter. The true diameter of the pelvic inlet is difficult to assess clinically. An estimate can be made from the diagonal conjugate diameter which is measured between the midpoint of the sacral promontory and the nearest point on the posterior surface of the pubic symphysis. This measure is about 1.5cm greater than the obstetrical conjugate diameter. In practice it is rarely possible to reach the promontory on clinical examination. The pelvic outlet is narrower than the pelvic inlet. The anterior-

posterior diameter of the pelvic outlet is measured from the inferior border of the pubic symphysis to the sacrococcygeal joint, and is usually about 12.5cm. The transverse diameter of the pelvic outlet is measured between the ischial tuberosities and is usually about 10cm. The plane of least pelvic dimensions represents the narrowest part of the birth canal. The anteroposterior diameter extends from the inferior margin of the pubic symphysis posteriorly through a line which connects the ischial spines, the transverse diameter. Both diameters can be assessed clinically, and the interspinous diameter is the shortest in the pelvis.

14. Right base of the heart is formed by --->

The base of the heart is opposite to that of the apex is mainly formed by the left atria, but getting some contribution from the right atrium.

15. Right side Mid calvicle the lung ends at which rib --10 Rib?

The border of the right lung lies immediately inside the pleural margin from the cupola down to about the 6th costal cartilages.

It then lies about two spaces above the pleural margin:
it crosses the 6th rib in the midclavicular line and the 8th rib in the midaxillary line, and reaches the vertebral column at the level of the 10th rib.

16. Water supply in hilly areas---?

Developing springs as a source for rural water supply is ideally suited for the following situations: (a). The springs are perennial (at least discharge at the rate of 20 lpcd during peak summer) and of good water quality (to be tested for chemical and bacteriological) contamination); (b) Chance of polluting the catchment is minimum; (c) The beneficiary population lives within close proximity of the springs preferably in the down stream areas of the springs.

In the era of decentralized planning, community mobilization, resource mobilization, cost sharing, community management, cost recovery and operation and maintenance plays a significant role in the water supply schemes. In 1989, the Pazhakulam Social Service Society (PASSS) has taken the initiative for the development of natural springs as alternative safe drinking water to the communities in the remote hilly areas of Southern Kerala. The main objective of the spring development programme was to demonstrate to the Government, Non-Governmental Organizations and the people of Kerala, that it is possible to design and develop alternate delivery of drinking water through private sector initiatives. The concept of rejuvenation of springs is an alternative mechanism to increase the coverage and accessibility of safe water supply in the remote areas. PASSS has taken the leadership in demonstrating the programme since 1991 and more than 1150 springs have already been developed with the active participation of the community. The salient features of the programme is as follows:

Established water committees as the nucleus of community organization, participation and management of the programme; Capacity building and empowerment; Capital cost sharing mechanism has become mandatory;
Full O & M by water committees/community; Low cost participatory design; Panchayat institutions as a partner; Integration of water, sanitation and hygiene; Water quality monitoring; Community monitoring and follow up.

17. Prophylaxis DOC for Meningitis -->

Meningitis is an inflammation of the leptomeninges and underlying subarachnoid cerebrospinal fluid

Cephalexin/Cefazolin

PO/IV
Stable vs Staph penicillinase
Spectrum: MSSA, PSSP, most E. coli, and some Klebs
Can be dose thrice weekly in HD pts
[1.5 grams IV TIW]
DoC: surgical prophylaxis, bacterial peritonitis in CAPD pts [1 gm in the dwell bag]

Ampicillin/Amoxicillin

Amp (IV, PO) Amox (PO)
Spectrum: PenG + H. flu and some E. coli
DoC: Listeria monocytogenes and
Enterococcus [Amp 2g IV q4h]

Dental Prophylaxis
Amox 1 gram PO x 1 prior to appt.
Integral in H. pylori regimens
ADRs
Non-allergic rashes (9%) – esp. when associated with a viral illness (mononucleosis - EBV)
Amox better tolerated PO and better absorbed (Amp must be taken on empty stomach)
---

more qns from FMGE sept 08

who discovered xrays?
roentgen

which disease is X linked recessive?
Hemophlia

most common cause of death in people older than 70 yrs?
cardiovascular disease, cancer,respiratory disease?

in retinal detachment?? options were red colour, blue colour, green colour? i cant remember exactly.

in which poisoning shud the vitrous humor be preserved?

malignant pustule?
anthrax

a qn from varicocele frm surgery, i cant remember..

frnds, there were over 20 questions frm Ophthalmology and Biochemistry and many from parasitology in Microbiology ..
few questions were simple and thats all we can recollect! there were many confusing questions too...
all da best for 2009....

Prophylaxis DOC for Meningitis?

PRESCRIPTIONS FOR PROPHYLAXIS OF MENINGITIS :
PROCEDURES FOR GGHB COMMUNITY PHARMACISTS

· Identify a prescription for meningitis prophylaxis:

Rifampicin 600mg (or 10mg/kg for children) twice daily for 2 days

or

Ciprofloxacin 500mg as a single dose (although not yet licensed for this purpose).

Establish the patient’s eligibility for exemption from prescription charge.

If not exempt via NHS, reassure the patient that a special GGHB exemption will apply.

Dispense the prescription.

Please do not collect the prescription charge.

Please submit an invoice for the prescription charge on headed notepaper to myself at GGHB Headquarters, noting patient name and date.

Submit the prescription in the non-exempt category to the PPD.

PRESCRIPTIONS FOR PROPHYLAXIS OF MENINGOCOCCAL SEPTICAEMIA

You will be aware of the increased incidence of meningitis in recent weeks. All cases are notified to the GGHB Public Health Department. Treatment of the individual case in an acute hospital is accompanied by management of the public health implications in primary care. Typically, specialists in Communicable Disease will identify close family and friends of the patient who may require prophylactic treatment. The treatment options currently are:

Rifampicin 600mg (or 10mg/kg for children) twice daily for 2 days

or

Ciprofloxacin 500mg as a single dose (although not yet licensed for this purpose)

The options for supply of these prescriptions are as follows:

community administration programme (e.g. school, church)

hospital supply

supply through the local GEMS centre

prescription on HBP forms by the Public Health Consultant on call

prescription on GP10

The last of these is frequently the preferred option. Treatment needs to be started quickly but not urgently and both drugs need to be used cautiously in combination with other therapies. Community pharmacists can reinforce these points.

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1 Anaemia is seen with all except?

Anemia
Definition
If you have anemia, people may say you have tired blood. That's because anemia — a condition in which there aren't enough healthy red blood cells to carry adequate oxygen to your tissues — can make you feel tired.

There are many forms of anemia, each with its own cause. Anemia can be temporary or long term, and it can range from mild to severe.

Anemia is a common blood disorder. Women and people with chronic diseases are at increased risk of the condition.

If you suspect you have anemia, see your doctor. Anemia can be a sign of serious illnesses. Treatments for anemia range from taking supplements to undergoing medical procedures. You may be able to prevent some types of anemia by eating a healthy, varied diet.

Symptoms
The main symptom of most types of anemia is fatigue. Other anemia symptoms include:

•Weakness
•Pale skin
•A fast or irregular heartbeat
•Shortness of breath
•Chest pain
•Dizziness
•Cognitive problems
•Numbness or coldness in your extremities
•Headache
Initially, anemia can be so mild it goes unnoticed. But signs and symptoms increase as the condition worsens.

Causes
Blood consists of both a liquid called plasma and cells. Floating within the plasma are three types of blood cells:

•White blood cells. These blood cells fight infection.
•Platelets. These blood cells help your blood clot after a cut.
•Red blood cells (erythrocytes). These blood cells carry oxygen from your lungs, via your bloodstream, to your brain and the other organs and tissues. Your body needs a supply of oxygenated blood to function. Oxygenated blood helps give your body its energy and your skin a healthy glow.
Red blood cells contain hemoglobin — a red, iron-rich protein that gives blood its red color. Hemoglobin enables red blood cells to carry oxygen from your lungs to all parts of

your body, and to carry carbon dioxide from other parts of the body to the lungs so that it can be exhaled.

Most blood cells, including red blood cells, are produced regularly in your bone marrow — a red, spongy material found within the cavities of many of your large bones. To produce hemoglobin and red blood cells, your body needs iron, protein and vitamins from the foods you eat.

Anemia is a state in which the number of red blood cells or the hemoglobin in them is below normal. When you're anemic, your body produces too few healthy red blood cells, loses too many of them or destroys them faster than they can be replaced. As a result, your blood is low on red blood cells to carry oxygen to your tissues — leaving you fatigued. Common types of anemia and their causes include:

•Iron deficiency anemia. This most common form of anemia affects about one in five women, half of pregnant women and 3 percent of men in the United States. The cause is a shortage of the element iron in your body. Your bone marrow needs iron to make hemoglobin. Without adequate iron, your body can't produce enough hemoglobin for red blood cells. The result is iron deficiency anemia.

One way your body gets needed iron is when blood cells die — the iron in them is recycled and used to produce new blood cells. So, if you lose blood, you lose iron. Women with heavy periods who lose a lot of blood each month during menstruation are at risk of iron deficiency anemia. Slow, chronic blood loss from a source within the body — such as an ulcer, a colon polyp or even colon cancer — also can lead to iron loss and iron deficiency anemia.

Your body also gets iron from the foods you eat. An iron-poor diet can lead to this anemia. In pregnant women, a growing fetus can deplete the mother's store of iron, leading to iron deficiency anemia.

•Vitamin deficiency anemias. In addition to iron, your body needs folate and vitamin B-12 to produce sufficient numbers of healthy red blood cells. A diet lacking in these and other key nutrients can cause decreased red blood cell production. People who have an intestinal disorder that affects the absorption of nutrients are prone to this type of anemia. Some people are unable to absorb vitamin B-12 for a variety of reasons and develop vitamin B-12 deficiency anemia, which is sometimes called pernicious anemia. Vitamin deficiency anemias fall into a group of anemias called megaloblastic anemias, in which the bone marrow produces large, abnormal red blood cells.
•Anemia of chronic disease. Certain chronic diseases — such as cancer, rheumatoid arthritis, Crohn's disease and other chronic inflammatory diseases — can interfere with the production of red blood cells, resulting in chronic anemia. Kidney failure also can be a cause of anemia. The kidneys produce a hormone called erythropoietin, which stimulates your bone marrow to produce red blood cells. A shortage of erythropoietin, which can result from kidney failure or be a side effect of chemotherapy, can result in a shortage of red blood cells.

•Aplastic anemia. This is a life-threatening anemia caused by a decrease in the bone marrow's ability to produce all three types of blood cells — red blood cells, white blood cells and platelets. Many times, the cause of aplastic anemia is unknown, but it's believed to often be an autoimmune disease. Some factors that can be responsible for this type of anemia include chemotherapy, radiation therapy, environmental toxins, pregnancy and lupus.
•Anemias associated with bone marrow disease. A variety of diseases, such as leukemia and myelodysplasia, a pre-leukemic condition, can cause anemia by affecting blood production in the bone marrow. The effects of these types of cancer and cancer-like disorders vary from a mild alteration in blood production to a complete, life-threatening shutdown of the blood-making process. Additionally, other cancers of the blood or bone marrow, such as multiple myeloma, myeloproliferative disorders and lymphoma, can cause anemia.
•Hemolytic anemias. This group of anemias develops when red blood cells are destroyed faster than bone marrow can replace them. Certain blood diseases can cause increased red blood cell destruction. Autoimmune disorders can cause your body to produce antibodies to red blood cells, destroying them prematurely. Certain medications, such as some antibiotics used to treat infections, also can break down red blood cells. Hemolytic anemias may cause yellowing of the skin (jaundice) and an enlarged spleen.
•Sickle cell anemia. This inherited and sometimes serious anemia, which affects mainly people of African and Arabic descent, is caused by a defective form of hemoglobin that forces red blood cells to assume an abnormal crescent (sickle) shape. These irregular-shaped red blood cells die prematurely, resulting in a chronic shortage of red blood cells. Sickle-shaped red blood cells can also block blood flow through small blood vessels in the body, producing other, often painful, symptoms.
•Other anemias. There are several other, rarer forms of anemia, such as thalassemia and anemias caused by defective hemoglobin.
Sometimes, no cause of anemia can be identified.

Risk factors
These factors place you at increased risk of anemia:

•Poor diet. Anyone — young or old — whose diet is consistently low in iron and vitamins, especially folate, is at risk of anemia. Your body needs iron, protein and vitamins to produce sufficient numbers of red blood cells.
•Intestinal disorders. Having an intestinal disorder that affects the absorption of nutrients in the small intestine — such as Crohn's disease and celiac disease — puts you at risk of anemia. Surgical removal of or surgery to the parts of the small intestine where nutrients are absorbed can lead to nutrient deficiencies and anemia.
•Menstruation. In general, women are at greater risk of iron deficiency anemia than are men. That's because women lose blood — and with it, iron — each month during menstruation.
•Pregnancy. Pregnant women are at an increased risk of iron deficiency anemia because their iron stores have to serve the increased blood volume of the mother as well as be a source of hemoglobin for the growing fetus.

•Chronic conditions. For example, if you have cancer, kidney or liver failure, or another chronic condition, you may be at risk of what's called anemia of chronic disease. These conditions can lead to a shortage of red blood cells. Slow, chronic blood loss from an ulcer or other source within the body can deplete your body's store of iron, leading to iron deficiency anemia.
•Family history. If your family has a history of an inherited anemia, you also may be at increased risk of the condition.
Certain infections, blood diseases and autoimmune disorders, exposure to toxic chemicals, and the use of some medications can affect red blood cell production and lead to anemia. Other people at risk of anemia are people with diabetes, people who are dependent on alcohol (alcohol interferes with the absorption of folic acid) and people who adhere to a strict vegetarian diet, who may not get enough iron or vitamin B-12 in their diet.

When to seek medical advice
See your doctor if you're feeling fatigued for unexplained reasons, especially if you're at risk of anemia. Some anemias, such as iron deficiency anemia, are common. But don't assume that if you're tired, you must be anemic. Fatigue has many causes besides anemia.

Some people learn that their hemoglobin is low, which indicates anemia, when they go to donate blood. Low hemoglobin may be a temporary problem remedied by eating more iron-rich foods or taking a multivitamin containing iron. However, it may also be a warning sign of blood loss in your body that may be causing you to be deficient in iron. If you're told that you can't donate blood because of low hemoglobin, ask your doctor if you should be concerned.

If you have a family history of an inherited anemia, such as sickle cell anemia, talk to your doctor and possibly a genetic counselor about your risk and what risks you may pass on to your children.

Tests and diagnosis
Doctors diagnose anemia with the help of a medical history, a physical exam and blood tests, including a complete blood count (CBC). This blood test measures levels of red blood cells and hemoglobin in your blood. Some of your blood may also be examined under a microscope to study the size, shape and color of your red blood cells, which may indicate a diagnosis. For example, in iron deficiency anemia, red blood cells are smaller and paler in color than normal. In vitamin deficiency anemias, red blood cells are enlarged and fewer in number.

If you receive a diagnosis of anemia, your doctor may order additional tests to determine the underlying cause. For example, iron deficiency anemia can result from chronic bleeding of known or unknown ulcers, benign polyps in the colon, colon cancer, tumors, or kidney failure. Your doctor may test for these and other conditions that may underlie the anemia.

Occasionally, it may be necessary to study a sample of your bone marrow to diagnose anemia.

Complications
When anemia is severe enough, it may interfere with your ability to do everyday tasks. You may be too exhausted to work or play. Although anemia is often treatable, it may take several weeks to months for red blood cell levels to return to normal after treatment. Ask your doctor what to expect from treatment.

If you've been diagnosed with anemia — it's often detected during routine blood tests — ask your doctor what treatment is necessary. Then be sure to follow through on treatment, even if you quickly start to feel better. Left unchecked, anemia can lead to a rapid or irregular heartbeat — an arrhythmia. Your heart must pump more blood to compensate for the lack of oxygen in the blood when you're anemic. This can even lead to congestive heart failure. Untreated pernicious anemia can lead to nerve damage and decreased mental function, as vitamin B-12 is important not only for healthy red blood cells but also for optimal nerve and brain function.

Some inherited anemias, such as sickle cell anemia, can be serious and lead to life-threatening complications. Losing a lot of blood quickly results in acute, severe anemia and can be fatal.

Treatments and drugs
Anemia treatment depends on the cause:

•Iron deficiency anemia. This form of anemia is treated with iron supplements, which you may need to take for several months or longer. If the underlying cause of iron deficiency is loss of blood — other than from menstruation — the source of the bleeding must be located and stopped. This may involve surgery.
•Vitamin deficiency anemias. Pernicious anemia is treated with injections — often lifetime injections — of vitamin B-12. Folic acid deficiency anemia is treated with folic acid supplements.
•Anemia of chronic disease. There's no specific treatment for this type of anemia. Doctors focus on treating the underlying disease. Iron supplements and vitamins generally don't help this type of anemia. However, if symptoms become severe, a blood transfusion or injections of synthetic erythropoietin, a hormone normally produced by the kidneys, may help stimulate red blood cell production and ease fatigue.
•Aplastic anemia. Treatment for this serious anemia may include blood transfusions to boost levels of red blood cells. You may need a bone marrow transplant if your bone marrow is diseased and can't make healthy blood cells. You may need immune-suppressing medications to lessen your immune system's response and give the transplanted bone marrow a chance to start functioning again.
•Anemias associated with bone marrow disease. Treatment of these various diseases can range from simple medication to chemotherapy to bone marrow transplantation.
Treatment of these types of anemia usually involves a consultation from a blood specialist (hematologist).

•Hemolytic anemias. Managing hemolytic anemias includes avoiding suspect medications, treating related infections and taking drugs that suppress your immune system, which may be attacking your red blood cells. Short courses of treatment with steroids or gamma globulin can help suppress your immune system's attack on your red blood cells. If the condition has caused an enlarged spleen, you may need to have your spleen removed. The spleen — a small organ below your rib cage on the left side — filters out and stores defective red blood cells. Certain hemolytic anemias can cause the spleen to become enlarged with damaged red blood cells.
•Sickle cell anemia. Treatment for this incurable anemia may include the administration of oxygen, pain-relieving drugs, and oral and intravenous fluids to reduce pain and prevent complications. Doctors also commonly use blood transfusions, folic acid supplements and antibiotics. A bone marrow transplant may be an effective treatment in some circumstances. A cancer drug called hydroxyurea (Droxia, Hydrea) also is used to treat sickle cell anemia in adults.

Prevention

Many types of anemia can't be prevented. However, you can help avoid iron deficiency anemia and vitamin deficiency anemias by eating a healthy, varied diet that includes foods rich in iron, folate and vitamin B-12.

The best sources of iron are beef and other meats. Other foods rich in iron include beans, lentils, iron-fortified cereals, dark green leafy vegetables, dried fruit, nuts and seeds. Folate, and its synthetic form, folic acid, can be found in citrus juices and fruits, dark green leafy vegetables, legumes and fortified breakfast cereals. Vitamin B-12 is plentiful in meat and dairy products. Foods containing vitamin C, such as citrus fruits, help increase iron absorption.

Eating plenty of iron-containing foods is particularly important for people who have high iron requirements, such as children — iron is needed during growth spurts — and pregnant and menstruating women. Adequate iron intake is also crucial for infants, strict vegetarians and long-distance runners.

Doctors may prescribe iron supplements or multivitamins containing iron for people with high iron requirements. But iron supplements are appropriate only when you need more iron than a balanced diet can provide. Don't assume that if you're tired that you simply need to take iron supplements. Overloading your body with iron can be dangerous.

Anaemia is a condition in which the haemoglobin concentration in the blood is below a defined level, resulting in a reduced oxygen-carrying capacity of red blood cells.

About half of all cases of anaemia can be attributed to iron deficiency; other common causes include infections, such as malaria and schistosomiasis, and genetic factors, which result in thalassaemias and sickle-cell disease.

In its severe form, anaemia is associated with fatigue, weakness, dizziness and drowsiness. Pregnant women and children are particularly vulnerable.

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19) Dose of Folic acid during pregnancy?

Taking 400 micrograms of synthetic folic acid daily from fortified foods and/or supplements has been suggested.

The Recommended Dietary Allowance (RDA) for folate equivalents for pregnant women is 600-800 micrograms, twice the normal RDA of 400 micrograms for women who are not pregnant.

Prophylaxis vs Neural Tube Defects (NTD):

The Society of Obstetricians and Gynaecologists of Canada , in its 1993 Policy Statement, recommended that all women of child bearing potential, whether planning pregnancy or not, should consider maintaining a folic acid intake of at least 0.4 mg daily, either in the diet or as a supplement.

Pregnant women with no previous history of fetal NTD and no other predisposing factors are advised to maintain an intake of at least 0.4 mg daily until 10 to 12 weeks after last menstrual period.

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20) Vitamin “A” dosage is given in….? (?. of doses.)

The World Health Organization recommends single-large-dose vitamin A supplementation for postpartum women in areas of prevalent vitamin A deficiency; neonatal dosing is under consideration.

It is safe to give fertile women, independentof their vitamin A status, as much as 10,000IU (3000 µg RE) daily at any time duringpregnancy.

21) What is Apoptosis?

Apoptosis, by contrast, is a process in which cells play an active role in their own death (which is why apoptosis is often referred to as cell suicide).

Apoptosis, or programmed cell death, is a normal component of the development and health of multicellular organisms. Cells die in response to a variety of stimuli and during apoptosis they do so in a controlled, regulated fashion.

This makes apoptosis distinct from another form of cell death called necrosis in which uncontrolled cell death leads to lysis of cells, inflammatory responses and, potentially, to serious health problems.

22) Extrinsic factor in blood coagulation? (PT/PTT)

PT, PTT, D-DIMER

This panel of tests is used to evaluate the extrinsic coagulation system. They may also aid in screening for congenital deficiencies of factors II, V, VII, X as well as deficiencies of prothrombin dysfibrinogenemia, and afibrinogenemia. Levels of PT, PTT and D-dimmer, can determine heparin effect, warfarin anticoagulant therapy, liver failure, disseminated intravascular coagulation (DIC), vitamin K deficiency. This test includes the following: prothrombin time (PT) and partial thromboplastin time (PTT), and D-dimmer.

Prothrombin time (PT)

This test is used to evaluate the adequacy of the extrinsic system and common pathway in the clotting mechanism. Prothrombin time (PT) test provides a control for long-term anticoagulant therapy that usually involves the use of a coumarin derivative (eg, Coumadin®).This therapy attempts to impede thrombus formation without the threat of mortality from hemorrhage.

Partial Thromboplastin Time

This test is used to evaluate the intrinsic coagulation system. It is also used to monitor heparin therapy, to aid in detecting classical hemophilia A, Christmas disease, and detection of congenital deficiencies of factors II, V, VIII, IX, X, XI, and XII. PTT is used to screen for the presence of dysfibrinogenemia, disseminated intravascular coagulation, liver failure, congenital hypofibrinogenemia, vitamin K deficiency, congenital deficiency of Fitzgerald factor, congenital deficiency of prekallikrein, high molecular weight kininogen, and circulatory anticoagulant.

D-DIMER

This test is a very specific confirmatory test for disseminated intravascular coagulation (DIC). This test is also used for the detection of deep vein thrombosis (DVT) and to detect acute myocardial infarction and unstable angina. The Fragment D-dimmer assess both thrombin and plasmin activity.

23) Curschmann’s spirals are due to?

Curschmann's spirals have been observed in the sputum of patients affected by lung cancer, asthma, chronic bronchitis, or in asymptomatic smokers.

Spirally twisted masses of mucus occurring in the sputum in bronchial asthma.

Curschmann's spirals:

coiled, basophilic plugs of mucus formed in the lower airways and found in sputum and tracheal washings; indicate chronic obstruction.

Curschmann's Spirals refer to parts of the desquamated epithelium seen in biopsies from asthmatic patients. They are named after German physician Heinrich Curschmann (1846-1910). They are often seen in association with eosinophilic infiltration and Charcot-Leyden crystals.

However, to date, their clinical significance and pathogenesis have not been completely explained.

24) Bell’s palsy?
WHAT IS BELL'S PALSY?
Bells palsy is a condition that causes the facial muscles to weaken or become paralyzed. It's caused by trauma to the 7th cranial nerve, and is not permanent.

WHY IS IT CALLED BELL'S PALSY?
The condition is named for Sir Charles Bell, a Scottish surgeon who studied the nerve and its innervation of the facial muscles 200 years ago.

HOW COMMON IS BELL'S PALSY?
Bells palsy is not as uncommon as is generally believed. Worldwide statistics set the frequency at approximately .02% of the population (with geographical variations). In human terms this is 1 of every 5000 people, and 40,000 Americans every year.

IS BELL'S PALSY ALWAYS ON THE SAME SIDE?
The percentage of left or right side cases is approximately equal, and remains equal for recurrences.

IS THERE ANY DIFFERENCE BECAUSE OF GENDER OR RACE?
The incidence of Bells palsy in males and females, as well as in the various races is also approximately equal. The chances of the condition being mild or severe, and the rate of recovery is also equal.

WHAT CONDITIONS CAN INCREASE THE CHANCE OF HAVING BELL'S PALSY?
Older people are more likely to be afflicted, but children are not immune to it. Children tend to recover well. Diabetics are more than 4 times more likely to develop Bells palsy than the general population. The last trimester of pregnancy is considered to be a time of increased risk for Bell's palsy. Conditions that compromise the immune system such as HIV or sarcoidosis increase the odds of facial paralysis occurring and recurring.

CAN BELL'S PALSY AFFECT BOTH SIDES OF THE FACE?
It is possible to have bilateral Bells palsy, but it's rare, accounting for less than 1% of cases. With bilateral facial palsy, it's important to rule out all other possible diagnoses with thorough diagnostic tests.

CAN BELL'S PALSY AFFECT OTHER PARTS OF THE BODY?
Bells palsy should not cause any other part of the body to become paralyzed, weak or numb. If any other areas are affected Bell's palsy is not the cause of the symptoms, and further testing must be done.

HOW DO THE SYMPTOMS OF BELL'S PALSY PROGRESS?
Very quickly. Most people either wake up to find they have Bells palsy, or have symptoms such as a dry eye or tingling around their lips that progress to classic Bell's palsy during that same day. Occasionally symptoms may take a few days to be recognizable as Bells palsy. The degree of paralysis should peak within several days of onset - never in longer than 2 weeks (3 weeks maximum for Ramsey Hunt syndrome). A warning sign may be neck pain, or pain in or behind the ear prior to palsy, but it is not usually recognized in first-time cases.

IS BELL'S PALSY CONTAGIOUS?
No, it is not contagious. People with Bells palsy can return to work and resume normal activity as soon as they feel up to it.

WHAT ABOUT RECOVERY FROM BELL'S PALSY?
Approximately 50% of Bells palsy patients will have essentially complete recoveries in a short time. Another 35% will have good recoveries in less than a year.

Regardless of the trigger, Bell's palsy is best described as an event - trauma to the nerve. As with any other injury, healing follows. The quality and duration of recovery is dependent on the severity of the initial injury. If the nerve has suffered nothing more than a mild trauma, recovery can be very fast, taking several days to several weeks. An "average" recovery is likely to take between a few weeks and a few months. The nerve regenerates at a rate of approximately 1-2 millimeters per day, and can continue to regenerate for 18 months, probably even longer. Improvement of appearance can continue beyond that time frame.

IS MUSCLE ATROPHY A CONCERN?

Not as a rule. It takes longer for the muscles to start to atrophy than it takes for most people to fully recover.

IS BELL'S PALSY LIKELY TO HAPPEN AGAIN?
The possibility of recurrence had been thought to be as high as 10 - 20%. These figures have been lowered as more has been learned about conditions that are now diagnosed as other types of facial palsies. Estimates of the rate of recurrence still vary widely, from around 4 - 14%. Most recent reports hover at 5 - 9%. The average timespan between recurrences is 10 years.

25) In Dialysis which toxicity is seen commonly?

Aluminum toxicity

Aluminum toxicity, prevalent among individuals with chronic renal failure, is associated with disabling osteomalacia, encephalopathy, and anemia.

The control of aluminum intake has included standards to limit the amount of aluminum in the dialysis fluid in addition to the use of nonaluminum containing phosphate binders.

Deferoxamine mesylate, a heavy metal chelating agent, is used to remove aluminum from the tissues of dialysis patients. Chelation therapy has resulted in improvements of clinical symptoms and bone histology. Ocular, auditory, and infectious adverse effects have occurred with the use of deferoxamine.

26) TOC for Gastric ulcer?

For people with Helicobacter pylori infection, the main goal is to get rid of the bacteria that causes the infection.

H2 receptor antagonist such as famotidine (Pepcid) or nizatidine (Axid) or a proton pump inhibitor such as omeprazole (Prilosec) or esomeprazole (Nexium) to suppress acid, combined with two antibiotics.

Those who do not have an H. pylori infection may be prescribed ulcer-healing medications such as antacids, H2 receptor antagonists, or proton pump inhibitors. Long-term treatment may be needed.

If the ulcer bleeds, endoscopy can control bleeding in most cases.

Surgery may be recommended for persons who do not respond to medicines or endoscopy. Surgical procedures for gastric ulcers include:

Vagotomy -- cuts the vagus nerve, which controls the stomach's production of gastric acid
Partial gastrectomy -- removes part of the stomach

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27) Squamous non-keratinizing is seen in….?

Squamous nonkeratinizing

Function: barrier, protection.
Location: wet surfaces: oral cavity, esophagus, and vagina.

Origin: ectoderm
• cells of basal layer (stratum germinatinum, stratum basale)

• cells of stratum spinosum

• squamous cells

Stratified squamous keratinizing (epidermis)

Function: barrier, protection.

Location: dry surfaces: skin.

Origin: ectoderm
• keratinocytes of stratum germinatinum (stratum basale)

• keratinocytes of stratum spinosum

• keratinocytes of stratum granulosum

• keratinocytes of stratum lucidum

• squames of keratin of stratum corneum

• melanocytes

Stratified cuboidal & columnar

Function: barrier, conduit.

Location: sweat gland, ducts of exocrine glands, anorectal junction.

Origin: ectoderm

Transitional

Function: barrier, distensible property.

Location: renal calyces, ureters, bladder, urethra.