DNB CET REVIEW 2ND EDITION ERRATA

CORRECTIONS IN RED

PAGE 27, 137

(284) Peak HCG levels are seen by what intrauterine age?

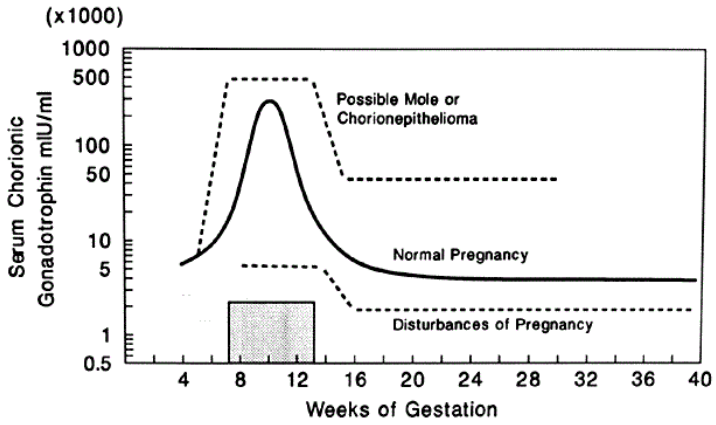
(A) 8-10 weeks
(B) 11-13 weeks
(C) 20 weeks
(D) 25 weeks

ANSWER: (A) 8-10 weeks

REF: Dutta 6th ed p-58.59, Current OB/GYN 10th ed chapter 8, William's 22nd ed chapter 3

HUMAN CHORIONIC GONADOTROPHIN:

- hCG is a glycoprotein composed of 2 subunits, **alpha and beta**. The alpha subunit is common to all glycoproteins, and the beta subunit confers unique specificity to the hormone. Typically, neither subunit is active by itself; only the intact molecule exerts hormonal effects.
- Synthsized by **syncytiotrophoblasts** of the placenta
- Half life is **32-37 hours**, in contrast to that of most protein and steroid hormones, which have half-lives measured in minutes.
- In early pregnancy doubling time is 2 days
  - Can be detected as early as 9 days after the midcycle LH peak, which occurs 8 days after ovulation and only 1 day after implantation.
- The hCG level above which one should identify an embryo by transvaginal ultrasonography (TVU) is now 1,000 to 2,000 mIU/mL, as determined by the second international standard.
- Levels progressively rise and reach maximum by **8-10 weeks/60-80 days** / 1st trimester
- Falls until 18-20 weeks and remains low until term.
- Disappears from circulation by 2 weeks



PAGE 43

(23)Maximum filling of ventricles is seen in?

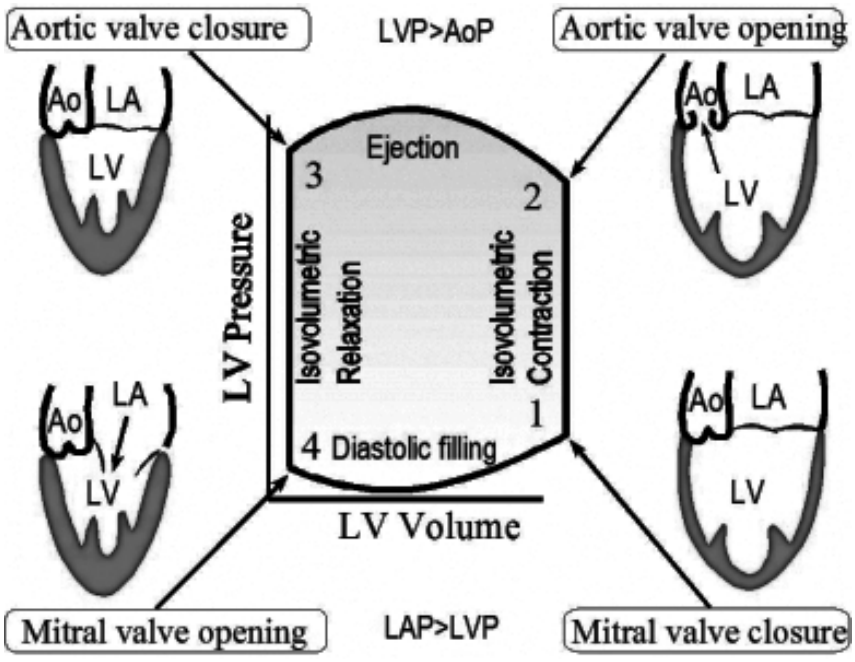
(A) Protodiastole
(B) Isovolumetric relaxation
(C) Ventricular phase of diastole
(D) Atrial contraction

ANSWER: (C) Ventricular phase of diastole

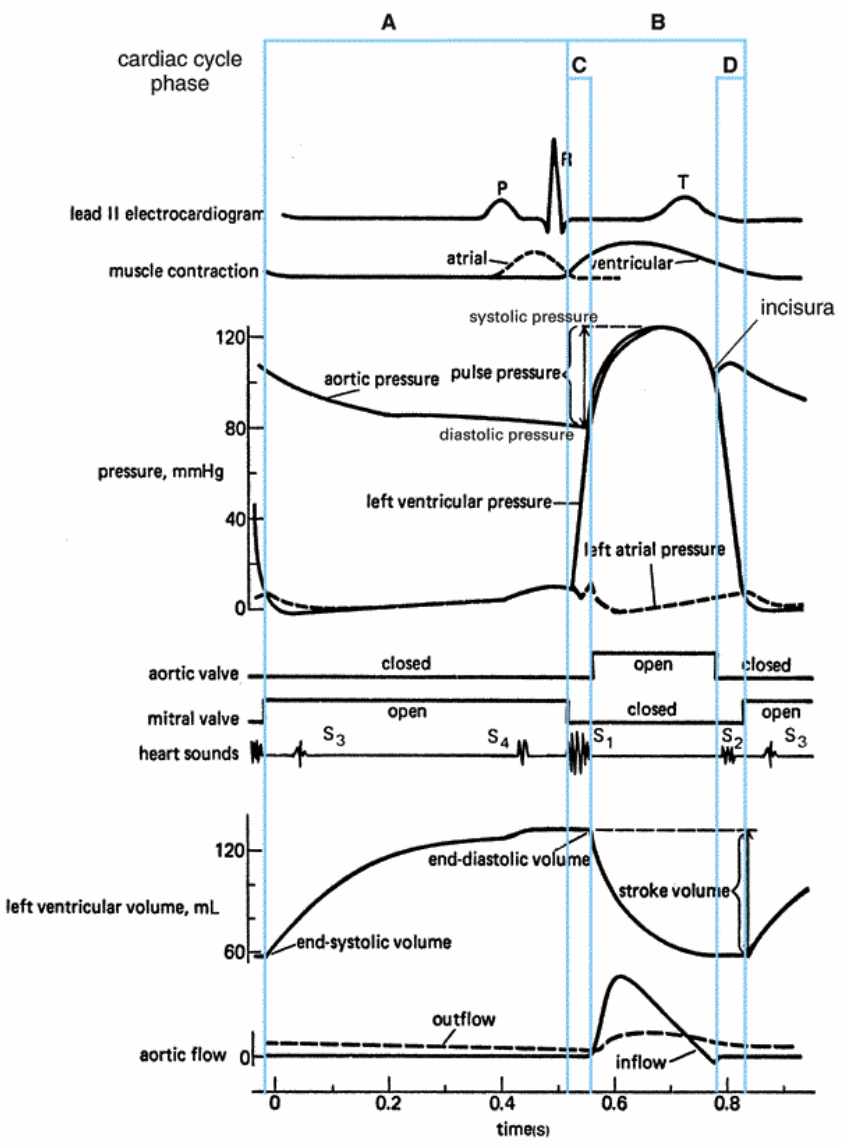
REF: Ganong's 22nd ed chapter 3 & 29, Color atlas of physiology 6th edition by Stefan Silbernagl, Agamemnon Despopoulos Page 192

Since the ventricles are 80% full by the first quarter of diastole, this is referred to as rapid ventricular filling

Valvular events	Cardiac events	ECG	JVP
Opening of semilunar valves	End of isovolumetric relaxation phase	End of T wave	V-Y descent
Closure of semilunar valves	End of diastole or beginning of isovolumetric contraction	Later half of 'R' wave	End of 'x' descent
Opening of AV valves	End of isovolumetric contraction	S-T segment	Peak of 'c' wave
Closure of AV valves	Beginning of isovolumetric relaxation , beginning of diastole	Later half of 't' wave	



Cardiac cycle phases: A , diastole; B , systole; C , isovolumetric contraction; and D , isovolumetric relaxation.



Note:

1. Maximum left ventricular volume – At the end of Isovolumetric contraction
2. Minimum left ventricular volume – At the end of isovolumetric relaxation
3. Maximum filling of ventricles – During ventricular phase of diastole

PAGE 51

(47) Cofactor involve in sulphur containing amino acid metabolism is?

(A) Folic acid
(B) Biotin

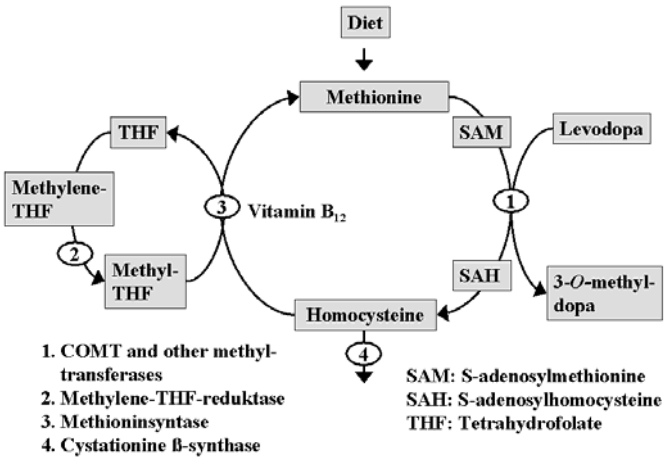
(C) Vitamin B 1
(D) Vitamin B 12

ANSWER: (D) Vitamin B 12

REF: Lehninger Principles of Biochemistry 4th edition page 674

Methionine, cysteine, homocysteine, and taurine are the 4 common sulfur-containing amino acids, but only the first 2 are incorporated into protein.

Vitamin B 12 is a co-factor for methionine synthetase; it helps to convert homocysteine to methionine



Note:

- While both folic acid and vitamin B12 are involved in sulphur containing amino acid metabolism, vitamin B12 acts as cofactor and folate acts as substrate.
- Thiamine and biotin anre two sulphur containing vitamins

PAGE 6 & 47

(36) All are correct about stomach EXCEPT:

(A) Pylorus has more acid secreting cells
(B) Lots of mucous secreting cells in pylorus
(C) Chief cells secrete pepsinogen
(D) Parietal cells secrete intrinsic factor

ANSWER: (A) Pylorus has more acid secreting cells

REF: Gray's anatomy 39th ed p- 1192

"Pyloric glands are mostly populated with mucus-secreting cells, parietal cells are few and chief cells scarce"
Note: There are other cells that secrete mucus (as in the foveolar cells of the stomach), but they are not usually called "goblet cells" because they do not have this distinctive shape.

GASTRIC GLANDS:
They can be divided into three groups-the cardiac, principal (in the body and fundus) and pyloric glands

I. **Principal glands:**
- Located in body and fundus
- In the walls of the gland are at least five distinct cell types: chief, parietal, mucous neck, stem and neuroendocrine
- **Chief cells:** source of pepsinogen, rennin and lipase. Contain zymogens , contain abundant RNA and hence intensely basophilic
- **Parietal (oxyntic) cells:** are the source of gastric acid and of intrinsic factor
- **Neuroendocrine cells:** These cells synthesize a number of biogenic amines and polypeptides important in the control of motility and glandular secretion. In the stomach they include cells designated as G cells secreting gastrin, D cells (somatostatin), and ECL (enterochromaffin-like) cells (histamine).

II. **Pyloric glands:**
Pyloric glands are mostly populated with mucus-secreting cells, parietal cells are few and chief cells scarce. In contrast, neuroendocrine cells are numerous, especially G cells, which secrete gastrin when activated by appropriate mechanical stimulation (causing increased gastric motility and secretion of gastric juices).

III. **Cardiac glands:**
Mucus-secreting cells predominate and parietal and chief cells, although present, are few

PAGE 23

(236) All of the following drugs are used in treatment of Hirsutism EXCEPT:

(A) Cyproterone acetate
(B) Spironolactone
(C) Flutamide
(D) Mefipristone

ANSWER: (D) Mefipristone

REF: Harrison's 18th ed chapter 49

DRUGS USED TO TREAT HIRSUTISM:
- **Spironolactone:** Antialdosterone antiandrogenic compound.
- **Cyproterone acetate:** A progestin that also has strong antiandrogenic action. In addition to single form, it is also available in some formulations of combined oral contraceptives.

- **Finasteride:** 5 alpha reductase inhibitor that inhibits conversion of testosterone to more active 5 alpha hydroxy testosterone.
- **Metformin:** Antihyperglycemic drug used for diabetes mellitus. However, it is also effective in treatment of hirsutism associated with insulin resistance (e.g. polycystic ovary syndrome)
- **Eflornithine:** Blocks putrescine that is necessary for the growth of hair follicles.
- **Flutamide:** Androgen receptor antagonist.

PAGE 23

(237) True about atrial myxoma is?

(A) Most common in left atrium
(B) Reoccurs after excision
(C) Distant metastases are seen
(D) More common in males

ANSWER: (A) More common in left atrium

REF: Harrison's 17th ed p- 1495

CARDIAC MYXOMA:

- Most common primary cardiac tumors
- occur in all ages without sex preference
- Most are sporadic , some are familial
- Most common site: left atrium
- Myxomas are benign and therefore distant metastases are not seen

Sporadic myxomas	Familial myxomas
• Solitary • Located in atria , most commonly left • Unlikely to have post op recurrence • Occurs in younger individual	• Multiple • More likely to have post op recurrence

PAGE 53

(56) Gonorrhea can be identified by?

(A) Growth on MacConkey medium
(B) Growth at 37^{O} C
(C) By the fermentation of glucose
(D) Growth in 45%/60% bile

ANSWER: (C) By the fermentation of glucose

REF: Anantnarayan 8th e p- 230

This question is based upon biochemical identification of neisseria species.

All the medically significant species of *Neisseria* are positive for both catalase and oxidase.

Different *Neisseria* species can be identified by the sets of sugars from which they will produce acid. For example, ***N. gonorrhea*** **makes acid from only glucose; however** ***N. meningitis*** **produces acid from both glucose and maltose.**
(**Mnemonic: G**-onnococci→**G**-lucose**, M**-enin **G**-ococci→ **M**-altose & **G**-lucose)

Other features:

- Meningococcus possess antiphagocytic polysaccharide capsule while Gonococcus doesn't.
- Gonococcus is Kidney shaped/coffee bean shaped while meningococcus is lens shaped
- Gonococcus has plasmid, meningococcus rarely has
- *N. gonorrhoeae* infections have a high prevalence and low mortality, whereas *N. meningitidis* infections have a low prevalence and high mortality.

PAGE 8 & 55

(1) Atypical pneumonia is caused by all EXCEPT:

(A) Mycoplasma
(B) Adeno virus
(C) Chlamydia
(D) Hemophilus

ANSWER: (D) Hemophilus

REF: Jawetz's Medical Microbiology, 24th Edition Section VII. Diagnostic Medical Microbiology & Clinical Correlation > Chapter 48,
http://emedicine.medscape.com/article/234240-overview ,
http://en.wikipedia.org/wiki/Community-acquired_pneumonia

TYPICAL COMMUNITY ACQUIRED PNEUMONIA:

- **Typical bacterial pathogens that cause CAP include Streptococcus pneumoniae (both penicillin-sensitive and -resistant strains), H influenzae (both ampicillin-sensitive and -resistant strains), and Moraxella catarrhalis (all strains penicillin-resistant). These 3 pathogens account for approximately 85% of CAP cases.**
- S pneumoniae remains the most common agent responsible for CAP
- **In selected patients;** S aureus may cause CAP in individuals with influenza (eg, human seasonal influenza and H1N1 [swine] influenza). K pneumoniae CAP occurs primarily in individuals with chronic alcoholism. P aeruginosa is a cause of CAP in patients with bronchiectasis or cystic fibrosis.

ATYPICAL COMMUNITY-ACQUIRED PNEUMONIA PATHOGENS: Atypical pneumonias can be divided into zoonotic and Nonzoonotic atypical pathogens.

- Zoonotic atypical CAP pathogens include Chlamydophilia (Chlamydia) psittaci (psittacosis), Francisella tularensis (tularemia), and Coxiella burnetii (Q fever).
- Nonzoonotic atypical CAP pathogens are caused by Legionella species, Mycoplasma pneumonia (in young age), or Chlamydophilia (Chlamydia) pneumonia, viruses (RSV, Adenovirus, Influenza virus, Parainfluenza virus, SARS)
- Respiratory viruses are the single most important cause of community-acquired pneumonia in pediatric age group**.**

Organism	**Clinical Setting**	**Gram-Stained Smears of Sputum**	**Laboratory Studies**	**Preferred Antimicrobial Therapy**
Streptococcus pneumoniae	Chronic cardiopulmonary disease; follows upper respiratory tract infections	Gram-positive diplococci	Gram-staining smear of sputum; culture of blood, pleural fluid; urinary antigen	Penicillin G (or V, oral); fluoroquinolones or vancomycin for highly penicillin resistant
Hemophilus influenzae	Chronic cardiopulmonary disease; follows upper respiratory tract infections	Small gram-negative coccobacilli	Culture of sputum, blood, pleural fluid	Ampicillin (or amoxicillin) if β-lactamase-negatie; cefotaxime or ceftriaxone
Staphylococcus aureus	Influenza epidemic; nosocomial	Gram-positive cocci in clumps	Culture of sputum,blood, pleural fluid	Nafcillin
Klebsiella pneumoniae	Alcohol abuse,diabetes mellitus; nosocomial	Gram-negative encapsulated rods	Culture of sputum, blood, pleural fluid	A cephalosporin; for severe infection, add gentamicin or tobramycin
Escherichia coli	Nosocomial; rarely, community acquired	Gram-negative rods	Culture of sputum, blood, pleural fluid	A third-generation cephalosporin
Pseudomonas aeruginosa	Nosocomial; cystic fibrosis	Gram-negative rods	Culture of sputum, blood	Antipseudomonal cephalosporin or carbapenem or -lactam/β-lactamae inhibitor plus an aminogycoside
Anaerobes	Aspiration, periodontitis	Mixed flora	Culture of pleural fluid or of materil obtained by transthoracic aspiration; bronchoscopy with proected specimen brush	Clindamycin
Mycoplasma pneumoniae	Young adults; summer and fall	PMNs and monocytes; no bacterial pathogens	Complement fixation titre, cold agglutinin serum titres are not helpful as they lack sensitivity and specificity; PCR	Erythromycin, azithromycin, or clarithromycin; doxycycline, fluoroquinolones

Legionella species	Summer and fall; exposure to contaminated construction site, water source, air conditioner; community acquired or nosocomial	Few PMNs; no bacteria	Direct immunofluorescent examination of sputum or tissue; immunofluorescent antibody titre; culture of sputum or tissue; Legionella urinary antigen (*L pneumophila* serogroup 1 only); PCR	Erythromycin, azithromycin, or clarithromycin, with or without rifampin; fluoroquinolones
Chlamydophilia pneumoniae	Clinically similar to *M pneumoniae* pneumonia, but prodromal symptoms last longer (up to 2 weeks); sore throat with hoarseness common; mild pneumonia in teenagers and young adults	Nonspecific	Isolation very difficult; microimmunofluorescence with TWAR antigens is the recommended assay	Doxycycline, erythromycin, clarithromycin; fluoroquinolones
Moraxella catarrhalis	Pre-existing lung disease; elderly; corticosteroid or immunosuppressive therapy	Gram-negative diplococci	Gram stain and culture of sputum or bronchial aspiration	Trimethoprim-sulfamethoxazole or amoxicillin-clavulanic acid or second or third generation cephalosporin
Pneumocystis jiroveci	AIDS, immunosuppressive therapy	Not helpful in diagnosis	Cysts and trophozoites of *P jiroveci* on methenamine silver or Giemsa stains of sputum or bronchoalveolar lavage fluid; direct immunofluorescent antibody on BAL fluid	Trimethoprim-sulfamethoxazole, pentamidine isethionate

PAGE 28 & 140

(293) True about Turner's syndrome is? (OMIT ALL EXCEPT)

(A) Normal breast
(B) Normal gonads
(C) Normal intelligence
(D) Long stature

ANSWER: (C) Normal intelligence

REF: Harrison's 18th ed chapter 349, Robbins 7th edition page 179,

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

Repeat from December 2010, June 2009 (not December 11)

Turner's syndrome	45,X or 45,X/46,XX	Streak gonad or immature ovary	External genitalia-Female	Internal genitalia Hypoplastic female	Breast Immature female
	Clinical Features				
	Infancy: lymphedema, web neck, shield chest, low-set hairline, cardiac defects and coarctation of the aorta, urinary tract malformations and horseshoe kidney				
	Childhood: short stature, cubitus valgus, short neck, short 4th metacarpals, hypoplastic nails, micrognathia, scoliosis, otitis media and sensorineural hearing loss, ptosis and amblyopia, multiple nevi and keloid formation, autoimmune thyroid disease, visuospatial learning difficulties				
	Adulthood: pubertal failure and primary amenorrhea, hypertension, obesity, dyslipidemia, impaired glucose tolerance and insulin resistance, autoimmune thyroid disease, cardiovascular disease, aortic root dilation, osteoporosis, inflammatory bowel disease, chronic hepatic dysfunction, increased risk of colon cancer, hearing loss				

PAGE 46

(33) Time duration required to generate an action potential is?

(A) Threshold
(B) Rheobase
(C) Chronaxie
(D) Refractory period

ANSWER: (C) Chronaxie

REF: Electrotherapy Simplified by Nanda page 276, Clinical neurophysiology by Jasper R Daube page 864

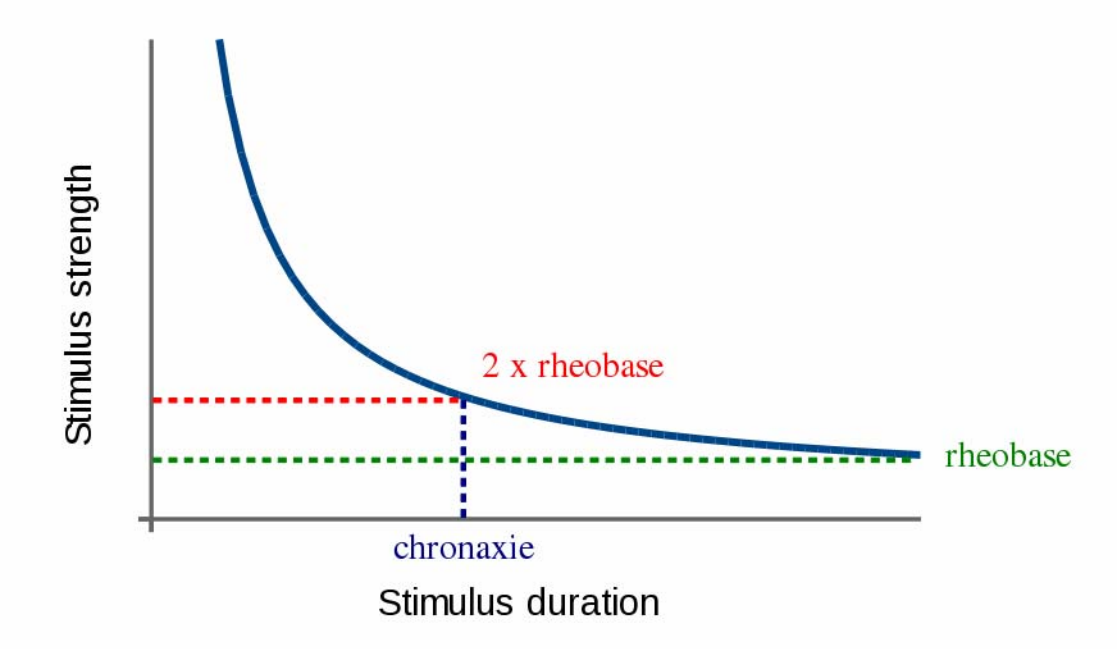
STRENGTH DURATION CURVE:
Graphic representation of the relationship between intensity (Y axis) and various durations (X axis) of the threshold electric stimulus of a nerve or muscle

The terms "chronaxie" and "rheobase" were coined in 1909 by the French physiologist Louis Lapicque. Rheobase is a measure of current amplitude and chronaxie is a measure of time (duration)

Rheobase is the intensity of electric current of infinite duration necessary to produce minimum action potential.

Chronaxie (or chronaxy) is the time required for an electric current double the strength of the rheobase to elicite first visible action potential

Each action potential is followed by a refractory period, which can be divided into an *absolute refractory period*, during which it is impossible to evoke another action potential, and then a *relative refractory period*, during which a stronger-than-usual stimulus is required



PAGE 66 & 493

(91) & 63) Asteroid bodies are seen in?

(A) Sarcoidosis
(B) Syphilis
(C) Chromoblastomycosis
(D) Sporotrichosis

ANSWER: (A) Sarcoidosis > (D) Sporotrichosis

REF: Robbins' 7th edition, page 734, 8th ed p- 738, Emergency Dermatology by Ronni Wolf, Batya B. Davidovici, Jennifer L. Parish, Lawrence Charles Paris page 133, Infectious Diseases of the Skin by Dirk M. Elston page 50, American Journal of Dermatopathology: June 1998 - Volume 20 - Issue 3 - pp 246-249

Repeat from June 2010

See APPENDIX-25 for list of "FEW IMPORTANT BODIES IN MEDICAL SCIENCE"

"Giant cells may contain **asteroid bodies** which are typical of sarcoid granulomas although **not pathognomonic**. Asteroid bodies are stellate inclusion bodies found in the multinucleated giant cells of **60 % of Sarcoidosis cases"**

"The asteroid bodies are observed in **40% of the rare cases of sporotrichosis.** They can be seen in other granulomatous reactions however extracellular structuresmade of spiculae of eosinophelic materialinvolved by a center containing yeasts (**Spendore-Hoeppli phenomenon**) are specific of asteroid bodies of sporotrichosis"

"**Extracellular asteroid bodies** comprised of eosinophelic spicule surrounding a central yeat form are thought to be distinguished from asteroid bodies of other granulomatous reactions that are typically intracellular" (Rodriguez & Barrera, 1997)

Asteroid bodies were first observed by splendore (1908) in Brazil in human sporotrichosis. Thay have been reported by many authors since and today their presence is generally accepted as presumptive (not diagnostic) evidence of sporotrichosis.

Sporotrichotic asteroid body must not be confused with the intracellular asteroid bodies seen in giant cells of granulomatous reactions, which are filamentous and myelin figures that contain lipid.

PAGE 85

(146) Endemic cretinism is seen when Iodine uptake is less than?

(A) 5 micro gram/day
(B) 20 micro gram/day
(C) 50 micro gram/day
(D) 75 micro gram/day

ANSWER: (B) 20 micro gram

REF: Park 20th edition page 540, WHO model formulary 2008 Page 499, http://www.unsystem.org/SCN/archives/npp03/ch06.htm

There are two schools of thoughts in this matter. While most of the text books are in favour of less than 25 micro grams WHO says its less than 20 micrograms. Luckily bith the options were not provided.

Endemic cretinism occurs in regions where goitre is widespread and severe and is associated with an iodine intake of less than 20 micrograms per day.

It is now known that endemic cretinism is associated with high rates of goitre and with severe iodine deficiency; for example, with dietary iodine intakes of about or **below 20 mcg** (micrograms) per day compared with a normal daily intake of 80-150 mcg; while goitre alone is seen at intake levels below 50 mcg iodine per day.

By WHO standards, a population is iodine deficient when its average UIE (urine iodine excretion) falls below 50 micrograms

The recommended intake of iodine is: (WHO)

Age group	**RDA (daily)**
Adults	150 micrograms
Pregnant and lactating women	200 micrograms
Infants	50 micrograms

Children (2-6 years)	90 micrograms
Children (7-12 years)	120 micrograms

PAGE 96

(183) After a leisure trip, a patient comes with gritty pain in eye, and joint pain. What is the most probable diagnosis?

(A) Reiter's syndrome
(B) Bachet's syndrome
(C) Sarcoidosis
(D) SLE

ANSWER: (A) Reiter's syndrome

REF: Harrison's 18th ed chapter 325

This is a case of reiter's syndrome or reactive arthritis with a **classical triad of arthritis, conjunctivitis (gritty feeling in eyes) & urethritis.**

Important points about reactive arthritis:

- *Reactive arthritis* (ReA) refers to acute nonpurulent arthritis complicating an infection elsewhere in the body. In recent years, the term has been used primarily to refer to Seronegative arthritis following enteric or urogenital infections
- *S. flexneri* has most often been implicated in cases of ReA
- Other bacteria identified definitively as triggers of ReA include several *Salmonella* spp., Yersinia enterocolitica, Y. pseudotuberculosis, Campylobacter jejuni, and Chlamydia trachomatis
- Characteristic skin lesions : circinate balanitis , keratoderma blennohemmorhagicum

About bechet's disease remember

Diagnostic Criteria of Behchet's Disease
Recurrent oral ulceration plus two of the following: Recurrent genital ulceration Eye lesions Skin lesions Pathergy test

PAGE 106

(205) In severe MS all are true EXCEPT:

(A) Pulsatile liver
(B) Atrial fibrillation
(C) Opening snap delayed from S2
(D) Length of murmur is increased

ANSWER: (C) Opening snap delayed from S2

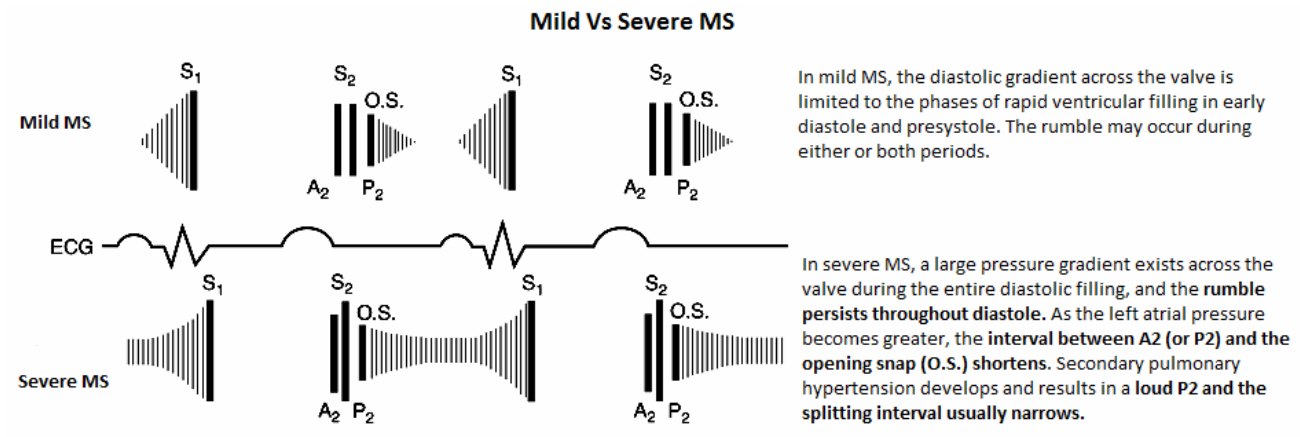
REF: Harrison's 18th ed chapter 237, Cardiovascular Pathophysiology by F. M. Kusumoto page 160

"Time interval between aortic valve closure (A2) and opening snap (OS) varies inversely with the severity of the MS, that is as the stenosis worsens, left atrial pressure increases and the gradient between left atrial and left ventricular pressures increases forcing the mitral valve open sooner (shorter S2 – OS interval)"

The timing of the opening snap is important as it indicates the severity of the lesion. In the early stage the timing is about 0,09 to 0,13 sec after the aortic component of S2. As left atrial pressure increases, in severe cases this interval may be as short as 0.04 to 0.05 seconds.

Severe TR often gives rise to a pulsatile liver edge. Note however that both severe MR and MS can give rise to TR

Severity of mitral stenosis		
Degree of mitral stenosis	**Mean gradient**	**Mitral valve area**
Mild mitral stenosis	<5 mmHg	>1.5 cm^2
Moderate mitral stenosis	5 - 10 mmHg	1.0 - 1.5 cm^2
Severe mitral stenosis	> 10 mmHg	< 1.0 cm^2



MITRAL STENOSIS:

Etiology:

- Rheumatic fever
- Congenital
- Severe mitral annular calcification
- SLE, RA

Signs of Severe mitral stenosis:

- Mitral valve area< 1 cm square
- End diastolic pressure gradient > 10 mm Hg
- Decreased interval between A2 and Opening snap.

- Increased duration of mid diastolic murmur
- Normal area of mitral valve : 4-6 cm square
- Left atrial pressure is increased
- Pulmonary pressure is increased
- Right ventricular hypertrophy occurs
- S1 accentuated , normally split S2 with accentuated P2
- Low pitched rumbling mid-diastolic murmur best heard at apex in left lateral position

Chest X ray features:

- Straightening of left cardiac border
- Prominent main pulmonary artery
- Dilation of upper lobe pulmonary veins
- Kerley B lines
- Backward displacement of esophagus

PAGE 124

(247)Treatment of $T_4 N_0 M_0$ stage of head and neck carcinoma is?

(A) Surgery alone
(B) Radiotherapy alone
(C) Chemoradiation
(D) Surgery and Radiotherapy

ANSWER: (D) Surgery and Radiotherapy

REF: Mastery of surgery 5th ed volume 1 page 308, Schwartz's Principles of Surgery 9th Chapter 18 Disorders of the Head and Neck table 18-3, Bailey & Love's 25th edition page 740, Harrison's 18th ed chapter 88

Repeat in December 2009

Current treatment guidelines for head and neck squamous cell carcinoma have been published by the National Comprehensive Cancer Network (NCCN).

- **Single modality therapy is adequate for T1 & T2 (stage I and II) lesions. Surgery and radiotherapy are equally effective.**
- **"For T3 and T4 (with or without N1, M1) surgery is the principal modality followed by post operative Radiotherapy for lesions situated primarily in the oral cavity. In contrast, for T3 &T4 oropharyngeal malignancies are often treated initially with chemoradiation.**

As in the question the specific site is not mentioned we have to choose intelligently

Mastery of surgery describes surgery followed by radiotherapy for all T3-T4 lesions other than oropharynx (lip, tongue, retromolar trigone, oral cavity, buccal mucosa and hard palate)

Bailey says "There is an increasing move to manage extensive disease of the oropharynx with chemoradiotherapy, provided that patients are medically fit to tolerate the toxicity.

For T3 & T4 oropharyngeal Harrison says “Such patients can also be treated with curative intent, but not with surgery or radiation therapy alone. Combined modality therapy including surgery, radiation therapy, and chemotherapy is most successful. It can be administered as induction chemotherapy (chemotherapy before surgery and/or radiotherapy) or as concomitant (simultaneous) chemotherapy and radiation therapy. The latter is currently most commonly used and best evidence–supported”

Head and neck cancer staging AJCC:

The system is uniform for all head and neck sites except for the nasopharynx.

AJCC/ TNM Staging for Head & Neck Cancer			
Primary tumor			
TX	Unable to assess primary tumor		
T0	No evidence of primary tumor		
Tis	Carcinoma in situ		
T1	Tumor is <2 cm in greatest dimension		
T2	Tumor >2 cm and <4 cm in greatest dimension		
T3	Tumor >4 cm in greatest dimension		
T4 (lip)	Primary tumor invading cortical bone, inferior alveolar nerve, floor of mouth, or skin of face (e.g., nose or chin)		
T4a (oral)	Tumor invades adjacent structures (e.g., cortical bone, into deep tongue musculature, maxillary sinus) or skin of face		
T4b (oral)	Tumor invades masticator space, pterygoid plates, or skull base and/or encases the internal carotid artery		
Regional lymphadenopathy			
NX	Unable to assess regional lymph nodes		
N0	No evidence of regional metastasis		
N1	Metastasis in a single ipsilateral lymph node, 3 cm or less in greatest dimension		
N2a	Metastasis in single ipsilateral lymph node, >3 cm and <6 cm		
N2b	Metastasis in multiple ipsilateral lymph nodes, all nodes <6 cm		
N2c	Metastasis in bilateral or contralateral lymph nodes, all nodes <6 cm		
N3	Metastasis in a lymph node >6 cm in greatest dimension		
Distant metastases			
MX	Unable to assess for distant metastases		
M0	No distant metastases		
M1	Distant metastases		
TNM staging			
Stage 0	Tis	N0	M0
Stage I	T1	N0	M0
Stage II	T2	N0	M0

Stage III	T3	N0	M0
	T1–3	N1	M0
Stage IVa	T4a	N0	M0
	T4a	N1	M0
	T1–4a	N2	M0
Stage IVb	Any T	N3	M0
	T4b	Any N	M0
Stage IVc	Any T	Any N	M1

PAGE 141

(296) All are true about Wilm's tumor EXCEPT:

(A) Present at 5 years of age
(B) Hematuria is the presenting symptom
(C) Presents as abdominal mass
(D) Most commonly metastasize to lung

ANSWER: (A) Presents at 5 years of age

REF: OP Ghai 7th edition page 592, Nelson 17th ed page 1711

WILM'S TUMOR:

- Wilms tumor, also designated **nephroblastoma,** is a complex mixed embryonal neoplasm of the kidney composed of three elements: blastema, epithelia, and stroma.
- The incidence is approximately 8 cases/million children younger than 15 yr of age.
- It usually occurs in children between 2–5 yr of age (2-3 years in O P Ghai), although it has also been encountered in neonates, adolescents, and adults.
- Most tumors are sporadic, but familial predisposition may be autosomal dominant
- One Wilms tumor gene, **WT1**, located at **11p13**, has been isolated. *WT1* encodes a zinc finger transcription factor that is critical for normal kidney development.

Syndromes Associated with Wilms Tumor and Their Clinical and Genetic Characteristics		
Syndrome	**Clinical Characteristics**	**Chromosome or other abnormalities**
WAGR	Aniridia, genitourinary abnormalities, mental retardation	Del 11p13 (*WT1* & *PAX6* loci)
Denys-Drash	Early-onset renal failure with renal mesangial sclerosis, male pseudohermaphrodism, increase risk of Wilms tumor	*WT1* mutations
Beckwith-Wiedemann	Organomegaly (liver, kidney, adrenal, pancreas), macroglossia omphalocele, hemihypertrophy	Uniparental paternal disomy, duplication 11p15.5, loss of imprinting, mutation of p57KIP57 have been described. Del 11p15.5 (*WT2* locus)

- **Presentation**

- ✓ Asymptomatic abdominal mass (most common presentation, can be bilateral)
- ✓ Abdominal pain
- ✓ Hematuria
- ✓ Hypertension
- ✓ Fever
- ✓ Anorexia

- **Poor prognostic factors:**
  - ✓ Unfavourable histology
  - ✓ Hyperploidy
  - ✓ Large tumor
  - ✓ Advanced stage (II , IV)
- **Metastasis:**
  - ✓ Usually to lung (mc) and then to liver
  - ✓ Non hematogenous
  - ✓ Bone metastasis are rare

Staging System Developed by the Third National Wilms Tumor Study Group	
Stage I	Tumor limited to kidney and is completely excised. Capsular surface intact; no tumor rupture; no residual tumor apparent beyond margins of excision
Stage II	Tumor extends beyond kidney but is completely excised. Regional extension of tumor; vessel infiltration; tumor biopsied or local spillage of tumor confined to the flank. No residual tumor apparent at or beyond margins of excision
Stage III	Residual nonhematogenous tumor confined to the abdomen. Lymph node involvement of hilus, periaortic chains, or beyond; diffuse peritoneal contamination by tumor spillage; peritoneal implants of tumor; tumor extends beyond surgical margins microscopically or macroscopically; tumor not completely removable because of local infiltration into vital structures
Stage IV	Deposits beyond stage III (e.g., lung, liver, bone, brain)
Stage V	Bilateral renal involvement at diagnosis

PAGE 176

(130) Confounding can be removed by?

(A) Assign confounders to both cases and controls

(B) Stratification

(C) Matching

(D) All of the above

ANSWER (D) All of the above

REF: Park 20th edition page 68, http://en.wikipedia.org/wiki/Confounding

There are various ways to modify a study design to actively exclude or control confounding variables

1. In Case-control studies assign confounders to both groups, cases and controls, equally.
2. In Cohort studies matching is often done by only admitting certain age groups or a certain sex into the study population, and thus all cohorts are comparable in regard to the possible confounding variable
3. Stratification:
4. Controlling for confounding by measuring the known confounders and including them as covariates in multivariate analyses

PAGE 202

(30) Most abundant extracellular buffer is?

(A) Hemoglobin
(B) Plasma proteins
(C) Bicarbonate
(D) Phosphate

ANSWER: (C) Bicarbonate

REF: Ganong 22nd edition chapter 39, Fluid Electrolyte & Acid Base by Jack L. Keyes page 80 table 5-1

Repeat from June 2008

COMPARTMENT WISE BUFFER SYSTEMS ARE:

Compartment			**Major buffer**
ECF	Blood	Plasma	Plasma proteins
		RBC	Hemoglobin
	Interstitial fluid		Bicarbonate
ICF			Phosphate> Proteins

NOTE:

- Bicarbonate is the predominant buffer of ECF as a whole
- Hemoglobin is the predominant buffer of blood as a whole because hemoglobin is present in large amounts the **hemoglobin in blood has six times the buffering capacity of the plasma proteins**
- Although hemoglobin is intracellular (inside RBC), it is often considered ECF buffer because RBC is a cellular component of ECF and because of the cellular permeability of RBC membrane.

PAGE 211

(59) Chlamydia escapes killing by?

(A) Inhibit phagolysosome fusion

(B) Causes cell membrane perforation

(C) Produces factors that camouflage it

(D) Molecular mimicry

ANSWER: (A) Inhibit phagolysosome fusion

REF: Chlamydia pneumoniae: infection and disease by Herman Friedman, Yoshimasa Yamamoto, Mauro Bendinelli Page 103, Textbook of bacteriology by Kenneth Todar table 2 (http://textbookofbacteriology.net/antiphago.html)

Microorganisms invading tissues are first and foremost exposed to phagocytes. Bacteria that readily attract phagocytes and that are easily ingested and killed are generally unsuccessful as pathogens. In contrast, most bacteria that are successful as pathogens interfere to some extent with the activities of phagocytes or in some way avoid their attention.

A summary of bacterial mechanisms for interference with phagocytes is given in the table below.

BACTERIAL INTERFERENCE WITH PHAGOCYTES		
BACTERIUM	**TYPE OF INTERFERENCE**	**MECHANISM**
Streptococcus pyogenes	Kill phagocyte	Streptolysin induces lysosomal discharge into cell cytoplasm
	Inhibit neutrophil chemotaxis	Streptolysin is chemotactic repellent
	Resist engulfment (unless Ab is present)	M Protein on fimbriae
	Avoid detection by phagocytes	Hyaluronic acid capsule
Staphylococcus aureus	Kill phagocyte	Leukocidin lyses phagocytes and induces lysosomal discharge into cytoplasm
	Inhibit opsonized phagocytosis	Protein A blocks Fc portion of Ab; polysaccharide capsule in some strains
	Resist killing	Carotenoids, catalase, superoxide dismutase detoxify toxic oxygen radicals produced in phagocytes

	Inhibit engulfment	Cell-bound coagulase hides ligands for phagocytic contact
Bacillus anthracis	Kill phagocytes or undermine phagocytic activity	Anthrax toxin EF
	Resist engulfment and killing	Capsular poly-D-glutamate
Streptococcus pneumoniae	Resist engulfment (unless Ab is present)	Capsular polysaccharide
Klebsiella pneumoniae	Resist engulfment	Polysaccharide capsule
Haemophilus influenzae	Resist engulfment	Polysaccharide capsule
Pseudomonas aeruginosa	Kill phagocyte	Exotoxin A kills macrophages; Cell-bound leukocidin
	Resist engulfment	Alginate slime and biofilm polymers
Salmonella typhi	Resist engulfment and killing	Vi (K) antigen (microcapsule)
Salmonella enterica (typhimurium)	Survival inside phagocytes	Bacteria develop resistance to low pH, reactive forms of oxygen, and host "defensins" (cationic proteins)
Listeria monocytogenes	Escape from phagosome	Listeriolysin, phospholipase C lyse phagosome membrane
Clostridium perfringens	Inhibit phagocyte chemotaxis	ø toxin
	Inhibit engulfment	Capsule
Yersinia pestis	Resist engulfment and/or killing	Protein capsule on cell surface
Yersinia enterocolitica	Kill phagocytes	Yop proteins injected directly into neutrophils
Mycobacteria	Resist killing and digestion	Cell wall components prevent permeation of cells; soluble substances detoxify of toxic oxygen radicals and prevent acidification of phagolysosome
Mycobacterium tuberculosis	Inhibit lysosomal fusion	Mycobacterial sulfatides modify lysosomes
Legionella pneumophila	Inhibit phagosome-lysosomal fusion	Unknown
Neisseria gonorrhoeae	Inhibit phagolysosome formation; possibly reduce respiratory burst	Involves outer membrane protein (porin) P.I
Rickettsia	Escape from phagosome	Phospholipase A
Chlamydia	Inhibit lysosomal fusion	Bacterial substance modifies phagosome
Brucella abortus	Resist killing	Cell wall substance (LPS?)
Treponema pallidum	Resist engulfment	Polysaccharide capsule material
Escherichia coli	Resist engulfment	O antigen (smooth strains); K antigen (acid polysaccharide)
	Resist engulfment and possibly killing	K antigen

The inflammatory and phagocytic responses of the host to invading bacteria are immediate and nonspecific. A second, specific immune response is soon encountered by invasive bacteria.

PATHOGEN STRATEGIES TO DEFEND AGAINST THE SPECIFIC IMMUNE DEFENSES

I. Immunological Tolerance to a Bacterial Antigen:

Tolerance is a property of the host in which there is an immunologically-specific reduction in the immune response to a given antigen (Ag). Tolerance to an Ag can arise in a number of ways, but three are possibly relevant to bacterial infections.

(A) **Fetal exposure to Ag**. If a fetus is infected at certain stages of immunological development, the microbial Ag may be seen as "self", thus inducing tolerance.

(B) **High persistent doses of circulating Ag**. Tolerance to a bacterium or one of its products might arise when large amounts of bacterial antigens are circulating in the blood. The immunological system becomes overwhelmed.

(C) **Molecular mimicry**. If a bacterial Ag is very similar to normal host "antigens", the immune responses to this Ag may be weak giving a degree of tolerance. Resemblance between bacterial Ag and host Ag is referred to as molecular mimicry. Some bacterial capsules are composed of polysaccharides (hyaluronic acid, sialic acid) so similar to host tissue polysaccharides that they are not immunogenic.

II. Antigenic Disguises:

Some pathogens can hide their unique antigens from opsonizing antibodies or complement. Bacteria may be able to coat themselves with host proteins such as fibrin, fibronectin, or even immunolobulin molecules. In this way they are able to hide their own antigenic surface components from the immunological system.

- ***S. aureus*** produces cell-bound coagulase and clumping factor that cause fibrin to clot and to deposit on the cell surface.
- **Protein A** produced by *S. aureus*, and the analogous **Protein G** produced by*Streptococcus pyogenes*, bind the Fc portion of immunoglobulins, thus coating the bacteria with antibodies and canceling their opsonizing capacity by the disorientation.

- The fibronectin coat of ***Treponema pallidum*** provides an immunological disguise for the spirochete.
- ***E. coli* K1**, that causes meningitis in newborns, has a capsule composed predominantly of sialic acid providing an antigenic disguise, as does the hyaluronic acid capsule of ***Streptococcus pyogenes***.

III. Immunosuppression:

Some pathogens (mainly viruses and protozoa, rarely bacteria) cause immunosuppression in their infected host. Suppressed immune responses are occasionally observed during chronic bacterial infections such as **leprosy** and **tuberculosis**.

IV. Persistence of a Pathogen at Bodily Sites Inaccessible to Specific Immune Response:

Intracellular pathogens can evade host immunological responses as long as they stay inside of infected cells and they do not allow microbial Ag to form on the cell surface. This is seen in macrophages infected with ***Brucella***, ***Listeria*** or ***M. leprae***.

V. Induction of Ineffective Antibody:

Antibodies tend to range in their capacity to react with Ag (the ability of specific Ab to bind to an Ag is called **avidity**). If Abs formed against a bacterial Ag are of low avidity, or if they are directed against unimportant antigenic determinants, they may have only weak antibacterial action.

In the case of **Neisseria gonorrhoeae** the presence of antibody to an outer membrane protein called rmp interferes with the serum bactericidal reaction and in some way compromises the surface defenses of the female urogenital tract.

VI. Antibodies Absorbed by Soluble Bacterial Antigens:

Some bacteria can liberate antigenic surface components in a soluble form into the tissue fluids. These soluble antigens are able to combine with and "neutralize" antibodies before they reach the bacterial cells

- **Streptococcus pneumoniae** and **Neisseria meningitidis** are known to release capsular polysaccharides during growth in tissues.

- Protein A, produced by ***S. aureus*** may remain bound to the staphylococcal cell surface or it may be released in a soluble form. Protein A will bind to the Fc region of IgG.

VII. Local Interference with Antibody Activity:

Some pathogens produce enzymes that destroy antibodies.

- *Neisseria gonorrhoeae*, *N. meningitidis*, *Haemophilus influenzae*, *Streptococcus pneumoniae* and *Streptococcus mutans*, which can grow on the surfaces of the body, produce **IgA proteases** that inactivate secretory IgA by cleaving the molecule at the hinge region, detaching the Fc region of the immunoglobulin.
- Soluble forms of **Protein A** produced *S. aureus* agglutinate immunoglobulin molecules and partially inactivate IgG.

VIII. Antigenic Variation

One way bacteria can trick forces of the immunological response is to periodically change antigens, i.e., to undergo antigenic variation.

- ***Neisseria gonorrhoeae*** can change fimbrial antigens during the course of an infection.
- The "relapses" of relapsing fever caused by the spirochete, ***Borrelia recurrentis***, are a result of antigenic variation by the organism.

PAGE 212

(62) Interleukin responsible for pyrexia is?

(A) IL1

(B) IL6

(C) INF gamma

(D) IFN alpha

ANSWER: (A) IL1

REF: Harrison's 17th ed chapter 308, Oxford Journals Medicine Clinical Infectious Diseases Volume 31, Issue Supplement 5 Pp. S178-S184

See APPENDIX-19 for list of "Cytokines" and there physiological role

"IL-1 is the most potent endogenous pyrogen (EP)"

In the classical model of pathogenesis, induction of fever is mediated by the release of pyrogenic cytokines such as tumor necrosis factor (TNF), interleukin (IL)-1, IL-6. Given that IL-6 expression is under the control of TNF and IL-1, it has been proposed that IL-6 is a downstream mediator of fever from IL-1 and TNF.

ENDOGENPOUS PYROGENS (EP)		
Cytokine	**EP activity**	**Comment**
IL-1	+++	Most potent EP in humans, both IL-1α and IL-1β are EP
TNF-α	++	Possible role for both soluble & membrane bound form
IL-6	++	IL-6 acts distsally of TNF and IL-1 in cytokine cascade
INF	+ or ++	INFα > INFβ > INFγ

PAGE 346-347

(1) Which is not associated with community acquired pneumonia?

(A) Legionella
(B) Klebsiella
(C) Chlamydia
(D) Pneumococcus

ANSWER: (B) Klebsiella

REF: Jawetz's Medical Microbiology, 24th Edition Section VII. Diagnostic Medical Microbiology & Clinical Correlation > Chapter 48,
http://emedicine.medscape.com/article/234240-overview ,
http://en.wikipedia.org/wiki/Community-acquired_pneumonia

TYPICAL COMMUNITY ACQUIRED PNEUMONIA:

- **Typical bacterial pathogens that cause CAP include Streptococcus pneumoniae (both penicillin-sensitive and -resistant strains), H influenzae (both ampicillin-sensitive and -resistant strains), and Moraxella catarrhalis (all strains penicillin-resistant). These 3 pathogens account for approximately 85% of CAP cases.**
- S pneumoniae remains the most common agent responsible for CAP
- **In selected patients;** S aureus may cause CAP in individuals with influenza (eg, human seasonal influenza and H1N1 [swine] influenza). K pneumoniae CAP occurs primarily in individuals with chronic alcoholism. P aeruginosa is a cause of CAP in patients with bronchiectasis or cystic fibrosis.

ATYPICAL COMMUNITY-ACQUIRED PNEUMONIA PATHOGENS: Atypical pneumonias can be divided into zoonotic and Nonzoonotic atypical pathogens.

- Zoonotic atypical CAP pathogens include Chlamydophilia (Chlamydia) psittaci (psittacosis), Francisella tularensis (tularemia), and Coxiella burnetii (Q fever).
- Nonzoonotic atypical CAP pathogens are caused by Legionella species, Mycoplasma pneumonia (in young age), or Chlamydophilia (Chlamydia) pneumonia, viruses (RSV, Adenovirus, Influenza virus, Parainfluenza virus, SARS)
- Respiratory viruses are the single most important cause of community-acquired pneumonia in pediatric age group**.**

Organism	**Clinical Setting**	**Gram-Stained Smears of Sputum**	**Laboratory Studies**	**Preferred Antimicrobial Therapy**
Streptococcus pneumoniae	Chronic cardiopulmonary disease; follows upper respiratory tract infections	Gram-positive diplococci	Gram-staining smear of sputum; culture of blood, pleural fluid; urinary antigen	Penicillin G (or V, oral); fluoroquinolones or vancomycin for highly penicillin resistant
Hemophilus influenzae	Chronic cardiopulmonary disease; follows upper respiratory tract infections	Small gram-negative coccobacilli	Culture of sputum, blood, pleural fluid	Ampicillin (or amoxicillin) if β-lactamase-negatie; cefotaxime or ceftriaxone
Staphylococcus aureus	Influenza epidemic; nosocomial	Gram-positive cocci in clumps	Culture of sputum,blood, pleural fluid	Nafcillin
Klebsiella pneumoniae	Alcohol abuse,diabetes mellitus; nosocomial	Gram-negative encapsulated rods	Culture of sputum, blood, pleural fluid	A cephalosporin; for severe infection, add gentamicin or tobramycin
Escherichia coli	Nosocomial; rarely, community acquired	Gram-negative rods	Culture of sputum, blood, pleural fluid	A third-generation cephalosporin
Pseudomonas aeruginosa	Nosocomial; cystic fibrosis	Gram-negative rods	Culture of sputum, blood	Antipseudomonal cephalosporin or carbapenem or -lactam/β-lactamae inhibitor plus an aminogycoside
Anaerobes	Aspiration, periodontitis	Mixed flora	Culture of pleural fluid or of materil obtained by transthoracic aspiration; bronchoscopy with proected specimen brush	Clindamycin

Mycoplasma pneumoniae	Young adults; summer and fall	PMNs and monocytes; no bacterial pathogens	Complement fixation titre, cold agglutinin serum titres are not helpful as they lack sensitivity and specificity; PCR	Erythromycin, azithromycin, or clarithromycin; doxycycline, fluoroquinolones
Legionella species	Summer and fall; exposure to contaminated construction site, water source, air conditioner; community acquired or nosocomial	Few PMNs; no bacteria	Direct immunofluorescent examination of sputum or tissue; immunofluorescent antibody titre; culture of sputum or tissue; Legionella urinary antigen (*L pneumophila* serogroup 1 only); PCR	Erythromycin, azithromycin, or clarithromycin, with or without rifampin; fluoroquinolones
Chlamydophilia pneumoniae	Clinically similar to *M pneumoniae* pneumonia, but prodromal symptoms last longer (up to 2 weeks); sore throat with hoarseness common; mild pneumonia in teenagers and young adults	Nonspecific	Isolation very difficult; microimmunofluorescence with TWAR antigens is the recommended assay	Doxycycline, erythromycin, clarithromycin; fluoroquinolones
Moraxella catarrhalis	Pre-existing lung disease; elderly; corticosteroid or immunosuppressive therapy	Gram-negative diplococci	Gram stain and culture of sputum or bronchial aspiration	Trimethoprim-sulfamethoxazole or amoxicillin-clavulanic acid or second or third generation cephalosporin
Pneumocystis jiroveci	AIDS, immunosuppressive therapy	Not helpful in diagnosis	Cysts and trophozoites of *P jiroveci* on methenamine silver or Giemsa stains of sputum or bronchoalveolar lavage fluid; direct immunofluorescent antibody on BAL fluid	Trimethoprim-sulfamethoxazole, pentamidine isethionate

PAGE 374

(133) Acute Gouty arthritis is seen early in treatment following?

(A) Probenecid
(B) Allopurinol

(C) Colchicine
(D) Rasburicase

ANSWER: (B) Allopurinol > (A) Probenecid

REF: Goodman Gillman manual of pharmacology and therapeutics 2008 edition page 458, Katzung 9th edition page 599, Lippincott pharmacology 6th edition page 443, Gout: Diagnosis and Management of Gouty Arthritis and Hyperuricemia by Robert Terkeltaub, M.D., N. Lawrence Edwards, M.D. 2nd ed page 189

Although the treatment of the hyperuricemia of gout depends upon lowering blood uric acid levels, most physicians caution against employing drugs such as *allopurinol, probenecid,* or *sulfinpyrazone* during an acute attack, since the therapy itself, at least during the initial stages, may exacerbate the condition.

"The incidence of acute attacks of gouty arthritis may increase during the early months of **allopurinol** therapy as a consequence of mobilization of tissue stores of uric acid. **Co-administration of colchicine helps suppress such acute attacks.** After reduction of excess tissue stores of uric acid, the incidence of acute attacks decreases and colchicine can be discontinued"

"Concomitant colchicine or NSAIDs are indicated early in the course of therapy to avoid precipitating an attack of gout, which may occur in up to 20% of gouty patients treated with probenecid alone"

Now if we read the following text it will become very clear that the **percentage of acute flare with allopurinol even when used along with colchicine (44.4%) is higher than percentage of acute flare with probenecid even when used alone (20%).**

In a RCT of gout patients initiated with allopurinol, the percentage of flares was lower for canakinumab (monoclonal antibody to IL1β) groups (25 mg 27.3%, 15 mg 16.7%, 100 mg 14.8%, 200 mg 18.5%, 300 mg 15.1%) than for the colchicine group (44.4%)

PAGE 401

(209) Horner tranta's spot is seen in?

(A) Trachoma
(B) Phlectenular Keratoconjunctivitis
(C) Vernal Keratoconjunctivitis
(D) Giant Papillary conjunctivitis

ANSWER: (C) Vernal Keratoconjunctivitis

REF: Khurana 4th ed p-451, Yanoff & Ducker- ophthalmology 2nd edition page 407

A characteristic manifestation of limbal **vernal conjunctivitis** is the presence of **Horner-Trantas dots**, which are white, chalk-like dots composed of eosinophils and epithelial debris.

PAGE 407

(229) An athlete presented with red coloured urine after 2 days of history of severe exertion. The most probable cause is?
(A) Hemoglobinuria
(B) Hemosiderinuria
(C) Hematuria
(D) Myoglobinuria

ANSWER: (D) Myoglobinuria

REF: Harrison's Internal Medicine 17th edition chapter 382, Physiology and pathology of the urine by john dixon p-49, Wintrobe's Clinical Hematology, Volume 12th ed page 1031

Red color urine in an athlete may be due to hemoglobin or myoglobin. Henoglobinuria develops immediately after the sternous exercise and resolves in hours while myoglobinurea develops after 24-48 hours.

"A heme-positive dipstick test in long-distance runners is often due to myoglobinuria, or occasionally to hemoglobinuria (March hemoglobinuria), rather than hematuria" (http://www.nephrologyrounds.org/crus/nephus_0504.pdf; Nephrology Rounds May 2004 Volume 2 Issue 5)

March hemoglobinuria:
March hemoglobinuria is an unusual hemolytic disorder characterized by hemoglobinuria, increased plasma hemoglobin, and decreased plasma haptoglobin in susceptible individuals after strenuous exercise that involves forceful contact of the body with a hard surface. Hemoglobinuria is precipitated by prolonged marches or competitive running, but the syndrome has also been noted in conga drum players and people participating in karate exercises.

Passage of red or dark urine after physical exertion is often the only complaint. Occasionally, symptoms include nausea; vague abdominal, back, or thigh pain; and a burning feeling in the soles of the feet.

Hemoglobinuria characteristically occurs **immediately after exercise and lasts for only a few hours**. March hemoglobinuria most commonly affects athletes at the beginning of a running career or on resumption of road training

Myoglobinuria:
Myoglobinuria may mimic the presence of hematuria. Myoglobin may appear in the urine of athletes secondary to the breakdown of muscle fibers. *Myoglobinuria* indicates the breakdown of muscle fibers during excessive exercise; it usually appears 24-48 *hours* after exercise. Urine dipstick can not differentiate between myoglobin and hemoglobin.

	Hematuria	Hemoglobinuria	Myoglobinuria
Mechanism	RBCs lyse on contact with the reagent pad, causing a	Free Hb filtered into urine as a result of	Free Mb filtered into urine as a result of

	positive reaction (speckled pattern may result if low-grade)	hemoglobinemia (usually detectable as red plasma)	myoglobinemia (not visually detectable in plasma).
Clinical	Bleeding into urinary space (can occur at any level of the urinary or reproductive tract). Commonly due to inflammation, trauma, neoplasia, hemostatic disorders.	Intravascular hemolysis of any cause (immune-mediated, toxic, mechanical, infectious, etc).	Myocyte injury allowing release of myoglobin which reaches bloodstream and is readily filtered at the glomeruli.
Urine examination	RBC will be present in urine sediment examination	Red supernatant, No RBC in urine sediment	Red supernatant, No RBC in urine sediment

PAGE 426

(283) Investigation of choice in post-menopausal bleeding is?

(A) Fractional curettage
(B) D & C
(C) Colposcopy guided endometrial biopsy
(D) PAP smear

ANSWER: (A) Fractional curettage

REF: Novak's gynecology 13th edition page 453, Dutta 4th ed page 331, Te Linde 9th ed page 1379

Post menopausal bleeding most commonly occurs due to endometrial atrophy but can also occurs due to endometrial cancer and cervical cancer.

"A case of postmenopausal bleeding is considered to be due to endometrial carcinoma unless proved otherwise"

However in a case of postmenopausal bleeding ruling out both endometrial and cervical cancer is always a priority and since in fractional curettage samples of both endometrial and cervical tissue is taken, it is the investigation of choice. If fractional curettage is not performed then endocervical curettage (ECC) should be performed in addition to evaluate endocervix.

CAUSES OF POSTMENOPAUSAL BLEEDING	
Cause	**Percentage**
Exogenous estrogen	30
Atrophic endometritis/vaginitis	30
Endometrial cancer	15
Endometrial or cervical polyp	10
Endometrial hyperplasia	5
Miscellaneous (cervical cancer, uterine sarcoma, trauma)	10

CAUSES OF POSTMENOPAUSAL UTERINE BLEEDING	
Cause	**Percentage**
Endometrial atrophy	60-80
Exogenous estrogen (HRT)	15-25
Endometrial cancer	10
Endometrial polyps	2-12
Endometrial hyperplasia	5-10

Fractional curettage:
The endocervical canal is curetted before cervical dilatation and the tissue is placed in a specifically labeled container. The uterus is then sounded, cervix is dilated and the endometrium is curetted. The endometrial tissue is placed in separate container.

NOTE:

- TVS has a good sensitivity and could be recommended as the first step in the investigation of postmenopausal bleeding
- Office endometrial aspiration biopsy is the accepted first step in evaluating a patient with abnormal uterine bleeding or suspected endometrial pathology

PAGE 428, 757

(293) & (249) All are the effects of gestational diabetes on fetus EXCEPT:

(A) Macrosomia
(B) Hypoglycemia
(C) Congenital malformations
(D) Increased perinatal mortality

ANSWERS: (C) Congenital malformations

REF: Current Diagnosis & Treatment Obstetrics & Gynecology, 10th edition chapter 18, Willians 22nd edition Table 52–2 & 52-8, http://en.wikipedia.org/wiki/Gestational_diabetes

Diabetes is the most common medical complication of pregnancy.
Gestational diabetes mellitus is a type of diabetes mellitus. Gestational diabetes is defined as carbohydrate intolerance of variable severity with onset or first recognition during pregnancy. This definition applies whether or not insulin is used for treatment. Undoubtedly, some women with gestational diabetes have previously unrecognized overt diabetes

CLASSIFICATION OF DIABETES COMPLICATING PREGNANCY:

Class	Onset	Fasting Plasma Glucose	2-hour Postprandial Glucose	Therapy
A_1	Gestational	< 105 mg/dL	< 120 mg/dL	Diet
A_2	Gestational	> 105 mg/dL	> 120 mg/dL	Insulin

Class	Age of Onset (yr)	Duration (yr)	Vascular Disease	Therapy
B	Over 20	< 10	None	Insulin

C	10 to 19	10 to 19	None	Insulin
D	Before 10	> 20	Benign retinopathy	Insulin
F	Any	Any	Nephropathy[a]	Insulin
R	Any	Any	Proliferative retinopathy	Insulin
H	Any	Any	Heart	Insulin

Women in classes B to H, corresponding to the White classification (1978), have overt diabetes antedating pregnancy.

MATERNAL AND FETAL EFFECTS OF GDM:

There has been an important shift in focus concerning adverse fetal consequences of gestational diabetes. Importantly, unlike in women with overt diabetes, **fetal anomalies are not increased** (Sheffield and colleagues, 2002). Similarly, whereas pregnancies in women with overt diabetes are at greater risk for fetal death, this danger is not apparent for those who have only postprandial hyperglycemia (namely, class A_1 gestational diabetes) (Lucas and co-workers, 1993; Sheffield and colleagues, 2002).

I. **Fetal effects**
   1. Fetal demise (Increased perinatal mortality)
   2. Macrosomia
   3. Increased risk of low blood glucose (hypoglycemia), jaundice, high red blood cell mass (polycythemia) and low blood calcium (hypocalcemia) and magnesium (hypomagnesemia)

II. **Maternal effects**
   1. Increased frequency of hypertension
   2. Increased frequency of cesarean delivery
   3. Risk for cardiovascular complications associated with abnormal serum lipids, hypertension, and abdominal obesity—the *metabolic syndrome*

MATERNAL & FETAL EFFECTS OF OVERT DIABETES:

I. **Fetal effects**
   1. First trimester abortion
   2. Preterm delivery
   3. Congenital malformations
   4. Hydramnios
   5. Macrosomia
   6. Fetal demise

II. **Neonatal effects**
   1. Respiratory distress
   2. Hypoglycemia
   3. Hypocalcemia
   4. Hyperbilirubinemia
   5. Cardiac hypertrophy
   6. Low risk of developing Type 1 diabetes

III. **Maternal effects**
   1. 10 fold increase in maternal death
   2. Ketoacidosis, hypertension, preeclampsia, and pyelonephritis
   3. With the possible exception of diabetic retinopathy, however, the long-term course of diabetes is not affected by pregnancy.

CONGENITAL MALFORMATIONS IN INFANTS OF WOMEN WITH OVERT DIABETES

Anomaly		Ratios of Incidence[a]
Caudal regression		252
Situs inversus		84
Spina bifida, hydrocephaly, or other central nervous system defect		2
Anencephaly		3
Heart anomalies	Atrial septal defects Ventricular septal defects Transposition of the great vessels Coarctation of the aorta Tetralogy of Fallot Truncus arteriosus Dextrocardia Cardiomegaly	4
Anal/rectal atresia		3
Renal anomalies		5
Agenesis		4
Cystic kidney		4
Duplex ureter		23

[a]Ratio of incidence is in comparision with the general population.

Note:

- Chromosomal aberrations are not seen in diabetes mellitus complicating pregnancy but congenital malformations are seen
- Both chromosomal aberrations and congenital malformations are not seen in gestational diabetes

PAGE 440

(323) All are seen in PTSD; post-traumatic stress disorder EXCEPT:

(A) Emotional numbing
(B) Hallucination
(C) Hyper arousal
(D) Vivid dreams

ANSWER: (B) Hallucination

REF: Kaplan & Sadock's Synopsis of Psychiatry: 10th Edition, page 615, Posttraumatic Stress Disorder in Litigation: Guidelines for Forensic Assessment by Robert I. Simon page 48, Psychiatry at a Glance by Cornelius Katona, Claudia Cooper, Mary Robertson page 27

The characteristic features of PTSD involve:

1. **Persistent intrusive thinking or re-experiencing**. Blank (1985) has identified four types of intrusive recall in PTSD
    I. Vivid dreams and nightmares of traumatic events

II. Remaining under the influence of vivid dreams after awakenin, with difficulty in making contact with reality
III. Conscious flashbacks experienced as intrusive, vivid hallucinations (any or all of the sences, with preserved insight)
IV. Unconscious flashbacks felt as sudden, discrete experiences leading to actions that repeat or recreate a traumatic event.

2. **Avoidance** of reminders of events
3. **Emotional numbning**, detachment and estrangement, loss of interest and sense of foreshortened future
4. **Hyperarousal with autonomisc symptoms**, hypervigillance, sleep disturbance, irritability, poor concentration

Most of the books have described all the provided options as features of PTSD. However hallucinations are reclassified as pseudohallucination, psychotic hallucination, and dissociative hallucination. By getting into the topic i found that the hallucinations in PTSD are actually Pseudo hallucinations (with intact insight).
Read following lines from **"A Guide to Psychiatric Examination by Carmelo Aquilina, James Warner page 84"**

"Pseudohallucinations have the vividness of true perception but the patient knows that they are an internal event, ie insight is retained. Flashbacks in PTSD and the so called Widow's Hallucination have these qualities"

PAGE 512

(118) Regarding poliovirus responsible for poliomyelitis all are true EXCEPT:

(A) Type 3 is most common in India
(B) Type 1 is most common in India
(C) Type 1 is responsible for most epidemics
(D) Type 2 is eradicated worldwide

ANSWER: (B) Type 1 is most common in India

REF: Park's textbook 20th edition page- 176-183, Neurological practice: an Indian perspective by Wadia- Page 113

- In south east asia region, India is the only country reporting polio cases with most of the cases reported from Bihar and Uttar Pradesh.
- **There is marked chance in the ratio of total number of wild polio virus 1 isolate to wild polio virus 3 isolate from approximately 7:1 to 1:13**. (due to pulse polio programme and vaccine)
- No wild poliovirus 2 has been detected anywhere in the world since 1999
- Wild polio virus 1 is responsible for most of the epidemics.

PAGE 541

(197) Minimum hCG level that a urine pregnancy test can detect is?

(A) 5 m IU/ ml

(B) 10-20 m IU/ ml
(C) 20-30 m IU/ ml
(D) 35 m IU/ ml

ANSWER: (A) 5 m IU/ ml

REF: Danforth's Obstetrics and Gynecology, 10th Edition page 4, Current OB/GYN > Chapter 9. Normal Pregnancy & Prenatal Care > Normal Pregnancy >

URINE PREGNANCY TEST:

Sensitive, early pregnancy test measure changes in levels of hCG. There is less cross-reaction with luteinizing hormone (LH), follicle stimulating hormone (FSH), and thyrotropin, which all share common α subunit with hCG, when the β subunit of hCG is measured. hCG is produced by the syncytiotrophoblast 8 days after fertilization and may be detected in the maternal serum after implantation occurs, 8–11 days after conception. hCG levels peak at approximately 8-10 weeks of gestation. Levels gradually decrease in the second and third trimesters and increase slightly after 34 weeks. The half-life of hCG is 2 days. After termination of pregnancy levels drop exponentially. Normally, serum and urine hCG levels return to nonpregnant values (< 5 m U/mL) 21–24 days after delivery.

- hCG is measured in milli-international units per milliliter (m IU/ml)
- The detection of greater than 35 m IU of human chorionic gonadotropin (hCG) in the first morning void has a very high specificity for pregnancy

β Hcg in m IU/ml	Result
Under 5 m IU/ml	Negative- Not pregnant
Between 5-25 m IU/ml:	"Equivocal"- Maybe pregnant may not be- Repeat test
Over 25 m IU/ml	Positive- Pregnant

PAGE 570 & 606

(98) Reference weight of Indian men and women is?

(A) 60 and 55 kg
(B) 60 and 50 kg
(C) 55 and 50 kg
(D) 50 and 45 kg

ANSWER: (A) 60 and 55 kg

REF: Park 20th edition page 547, Park 21st ed page 584

Note: The 20th edition of park textbook have older data related to reference Indian man and woman which was changed by ICMR in the year 2011.

CRITERIA FOR INDIAN REFERENCE MAN AND WOMAN:

Particulars	Indian reference man	Indian reference woman
Age	18-29 years	18-29 years
Height	1.73 meters	1.61m
BMI	20.3	21.2
Weight	60 kg	55 kg

Daily activities	8 hours of moderate occupation 8 hours in bed 4-6 hours sitting and moving around 2 hours walking and recreation	8 hours of household work 8 hours in bed 4-6 hours sitting and moving around 2 hours walking and recreation
Energy requirement	Light work= 2320 kcal/day Moderate work= 2730 kcal/day Heavy work= 3490 kcal/ day	Light work= 1900 kcal/day Moderate work= 2230 kcal/day Heavy work= 2850 kcal/ day
Protein allowance	1 gm/ day/ kg	1 gm/ day/ kg
Fat intake	25-40 gm/day	20-30 gm/day

PAGE 580 & 651

(209) Which amongst the following have longest half-life?

(A) Radon
(B) Radium
(C) Plutonium
(D) Iridium

ANSWER: (C) Plutonium

REF: http://www.nrc.gov/reading-rm/doc-collections/fact-sheets/plutonium.html,

Also see APPENDIX-67 for "ISOTOPES USED IN RADIOTHERAPY"

A half-life is the time in which one half of the atoms of a radioactive substance disintegrates into another nuclear form, hence, the time to halve its radioactive strength.

If the details of isotopes are not provided then it is assumed that the examiner is talking about the most stable isotope

- The most stable isotope of Radon is ^{222}Rn which has a half life of 3.8 days
- The most stable isotope of Radium is ^{226}Ra which has a half life of 1622 years
- The most stable isotope of Iridium is ^{192}Ir which has a half life of 74 days
- The most stable isotope of Plutonium is ^{244}Pu which has a half life of 80 million years

PAGE 587

(16) Goblet cells are present in all EXCEPT:

(A) Small intestine
(B) Large intestine
(C) Esophagus
(D) Stomach

ANSWER: (D) Stomach

REF: http://en.wikipedia.org/wiki/Goblet_cell

Repeat from December 2008

Goblet cells are glandular simple columnar epithelial cells whose sole function is to secrete mucin, which dissolves in water to form mucus. They use both apocrine and merocrine methods for secretion.

Locations of Goblet cells:

They are found scattered among the epithelial lining of organs, such as the intestinal and respiratory tracts. They are found inside the trachea, bronchus, and larger bronchioles in respiratory tract, small intestines, the colon, and conjunctiva in the upper eyelid.
They may be an indication of metaplasia, such as in Barrett's esophagus.

Note: There are other cells that secrete mucus (as in the foveolar cells of the stomach), but they are not usually called "goblet cells" because they do not have this distinctive shape.

PAGE 638

(170) Treatment of $T_4 N_0 M_0$ stage of head and neck carcinoma is?

(A) Surgery alone
(B) Radiotherapy alone
(C) Chemoradiation
(D) Surgery and Radiotherapy

ANSWER: (D) Surgery and Radiotherapy

REF: Mastery of surgery 5th ed volume 1 page 308, Schwartz's Principles of Surgery 9th Chapter 18 Disorders of the Head and Neck table 18-3, Bailey & Love's 25th edition page 740, Harrison's 18th ed chapter 88

Repeat in December 2011

Current treatment guidelines for head and neck squamous cell carcinoma have been published by the National Comprehensive Cancer Network (NCCN).

- **Single modality therapy is adequate for T1 & T2 (stage I and II) lesions. Surgery and radiotherapy are equally effective.**
- **"For T3 and T4 (with or without N1, M1) surgery is the principal modality followed by post operative Radiotherapy for lesions situated primarily in the oral cavity. In contrast, for T3 &T4 oropharyngeal malignancies are often treated initially with chemoradiation.**

As in the question the specific site is not mentioned we have to choose intelligently

Mastery of surgery describes surgery followed by radiotherapy for all T3-T4 lesions other than oropharynx (lip, tongue, retromolar trigone, oral cavity, buccal mucosa and hard palate)

Bailey says "There is an increasing move to manage extensive disease of the oropharynx with chemoradiotherapy, provided that patients are medically fit to tolerate the toxicity.

For T3 & T4 oropharyngeal Harrison says “Such patients can also be treated with curative intent, but not with surgery or radiation therapy alone. Combined modality therapy including surgery, radiation therapy, and chemotherapy is most successful. It can be administered as induction chemotherapy (chemotherapy before surgery and/or radiotherapy) or as concomitant (simultaneous) chemotherapy and radiation therapy. The latter is currently most commonly used and best evidence–supported”

Head and neck cancer staging AJCC:

The system is uniform for all head and neck sites except for the nasopharynx.

AJCC/ TNM Staging for Head & Neck Cancer			
Primary tumor			
TX	Unable to assess primary tumor		
T0	No evidence of primary tumor		
Tis	Carcinoma in situ		
T1	Tumor is <2 cm in greatest dimension		
T2	Tumor >2 cm and <4 cm in greatest dimension		
T3	Tumor >4 cm in greatest dimension		
T4 (lip)	Primary tumor invading cortical bone, inferior alveolar nerve, floor of mouth, or skin of face (e.g., nose or chin)		
T4a (oral)	Tumor invades adjacent structures (e.g., cortical bone, into deep tongue musculature, maxillary sinus) or skin of face		
T4b (oral)	Tumor invades masticator space, pterygoid plates, or skull base and/or encases the internal carotid artery		
Regional lymphadenopathy			
NX	Unable to assess regional lymph nodes		
N0	No evidence of regional metastasis		
N1	Metastasis in a single ipsilateral lymph node, 3 cm or less in greatest dimension		
N2a	Metastasis in single ipsilateral lymph node, >3 cm and <6 cm		
N2b	Metastasis in multiple ipsilateral lymph nodes, all nodes <6 cm		
N2c	Metastasis in bilateral or contralateral lymph nodes, all nodes <6 cm		
N3	Metastasis in a lymph node >6 cm in greatest dimension		
Distant metastases			
MX	Unable to assess for distant metastases		
M0	No distant metastases		
M1	Distant metastases		
TNM staging			
Stage 0	Tis	N0	M0
Stage I	T1	N0	M0
Stage II	T2	N0	M0

Stage III	T3	N0	M0
	T1–3	N1	M0
Stage IVa	T4a	N0	M0
	T4a	N1	M0
	T1–4a	N2	M0
Stage IVb	Any T	N3	M0
	T4b	Any N	M0
Stage IVc	Any T	Any N	M1

PAGE 687

(1) Winging of scapula is due to damage to the nerve supply of?

(A) Serratus anterior

(B) Latissimus dorsi

(C) Trapezius

(D) Deltoid

ANSWER: (A) Serratus anterior > (C) Trapezius

REF: Gray's anatomy 39th edition page 558, Textbook of Orthopaedics and Trauma by GS Kulkarni page 2600, Operative Techniques in Shoulder and Elbow Surgery by Gerald R. Williams, Matthew L. Ramsey, Sam W. Wiese page 267

Also see APPENDIX-7 for "BRACHIAL PLEXUS LESIONS"

Winging of scapula is a deformity in which the vertebral border and the inferior angle of scapula become unduly prominent.

Gray's anatomy has described winging of scapula in both nerve injury to serratus anterior and trapezius muscle. So we need to look down the detailed list of causes of winging of scapula.

CAUSES OF WINGING OF SCAPULA: winging of scapula can be primary, secondary or voluntary

I. Primary:

(A) Neurological disorders:

(1) Long thoracic nerve palsy (Serratus anterior weakness)

(2) Spinal accessory nerve palsy (Trapezius weakness)

(3) Dorsal scapular nerve palsy (Rhomboid weakness)

(B) Bony abnormalities:

(1) Osteochondroma of scapula

(2) Fracture malunion

(C) Soft tissue disorders:

(1) Soft tissue contractures

(2) Fascioscalulohumeral dystrophy

(3) Congenital abscence of parascapular muscles

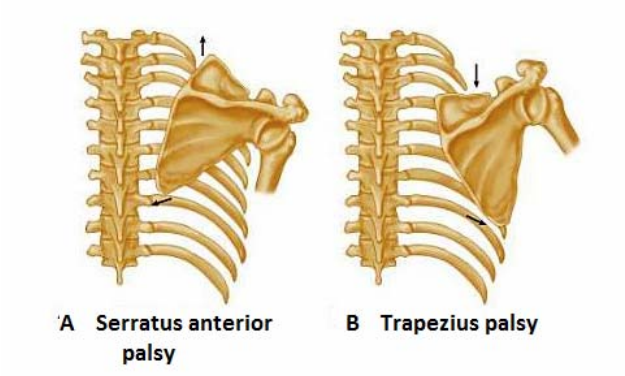
(4) Traumatic repture of parascapular muscles

II. **Secondary:** Disorders of glenohumeral joint

III. **Voluntary:** Psychiatric patients or for secondary gain

Note:

- Long thoracic nerve injury (nerve to serratus anterior) is the most common cause of winging of scapula
- Serratus anterior winging can be distinguished from trapezius winging by the direction of scapular laxity (fig)



PAGE 695

(24) hCG is secreted by?

(A) Syncytiotrophoblast

(B) Chorionic membrane

(C) Yolk sac

(D) Liver

ANSWER: (A) Syncytiotrophoblast

REF: Danforth's Obstetrics and Gynecology, 10th Edition page 4, Current OB/GYN > Chapter 9

hCG is produced by the **syncytiotrophoblast** 8 days after fertilization and may be detected in the maternal serum after implantation occurs, 8–11 days after conception. hCG levels peak at approximately 8-10 weeks of gestation. Levels gradually decrease in the second and third trimesters and increase slightly after 34 weeks. The half-life of hCG is 2 days. After termination of pregnancy levels drop exponentially. Normally, serum and urine hCG levels return to nonpregnant values (< 5 m U/mL) 21–24 days after delivery.

- hCG is measured in milli-international units per milliliter (m IU/ml)
- The detection of greater than 35 m IU of human chorionic gonadotropin (hCG) in the first morning void has a very high specificity for pregnancy

β Hcg in m IU/ml	Result
Under 5 m IU/ml	Negative- Not pregnant
Between 5-25 m IU/ml:	"Equivocal"- Maybe pregnant may not be- Repeat test
Over 25 m IU/ml	Positive- Pregnant

PAGE 755

(245) Additional protien and calorie requirement in pregnancy is?

(A) 50 kcal/ day calorie, 10 g/day protein

(B) 100 kcal/ day calorie, 20 g/day protein

(C) 300 kcal/ day calorie, 30 g/day protein

(D) 500 kcal/ day calorie, 50 g/day protein

ANSWER: (C) 300 kcal/ day calorie, 30 g/day protein

REF: William's 22nd edition chapter 8, COGT 10th edition table 9–1

Recommended Daily Dietary Allowances for Nonpregnant, Pregnant, and Lactating Women:

	Nonpregnant Women (years)				Pregnant Women	Lactating Women
	15–18	19–24	25–50	50+		
Energy (kcal)	2100	2100	2100	2000	+300	+500

	Nonpregnant Women (years)				Pregnant Women	Lactating Women
	15–18	19–24	25–50	50+		
Protein (g)	48	46	46	46	+30	+20
Fat-soluble vitamins						
Vitamin A (RE)/(IU)	800	800	800	800	800	1300
Vitamin D (IU)	400	400	200	200	400	400
Vitamin E (IU)	8	8	8	8	10	12
Water-soluble vitamins						
Vitamin C (mg)	60	60	60	60	70	95
Folate (µg)	180	180	180	180	400	280
Niacin (mg)	15	15	15	13	17	20
Riboflavin (mg)	1.3	1.3	1.3	1.2	1.6	1.8
Thiamine (mg)	1.1	1.1	1.1	1.0	1.5	1.6
Vitamin B_6 (mg)	1.5	1.6	1.6	1.6	2.2	2.1
Vitamin B_{12} (µg)	2	2	2	2	2.2	2.6
Minerals						
Calcium (mg)	1300	1000	1000	1200	1000	1000
Iodine (µg)	150	150	150	150	175	200
Iron (mg)	15	15	15	10	30	15
Magnesium (mg)	300	280	280	280	300	355
Phosphorus (mg)	1200	800	800	800	1200	1200
Zinc (mg)	12	12	12	12	15	19

PAGE 778

APPENDIX-6

BRACHIAL ARCHES

There are six pharyngeal arches, but in humans the fifth arch only exists transiently during embryologic growth and development. Since no human structures result from the fifth arch, the arches in humans are I, II, III, IV, and VI. The first three contribute to structures above the larynx, while the last two contribute to the larynx and trachea

PHARYNGEAL ARCH	MUSCULAR CONTRIBUTIONS	SKELETAL CONTRIBUTIONS	NERVE	ARTERY	CORROSPONDING POUCH STRUCTURES
1st (mandibular	Muscles of mastication,	Maxilla, mandible (only as a model	Trigeminal nerve (V2 and	Maxillary artery,	Eustachian tube, middle ear,

arch)	Anterior belly of the digastric, Mylohyoid, Tensor tympani, Tensor veli palatini	for mandible not actual formation of mandible), Incus and Malleus, Meckel's cartilage, Ant. ligament of malleus, Sphenomandibular ligament	V3)	external carotid artery	mastoid antrum, and inner layer of the tympanic membrane.
2nd (hyoid arch)	Muscles of facial expression, Buccinator, Platysma, Stapedius, Stylohyoid, Posterior belly of the digastric	Stapes, Styloid process, hyoid (lesser horn and upper part of body), Reichert's cartilage, Stylohyoid ligament	Facial nerve (VII)	Stapedial Artery	middle ear, palatine tonsils
3rd	Stylopharyngeus	Hyoid (greater horn and lower part of body), (delete thymus from here)	Glossopharyngeal nerve (IX)	Common carotid/Internal carotid	Inferior parathyroid, Thymus
4th	Cricothyroid muscle, all intrinsic muscles of soft palate including levator veli palatini	Thyroid cartilage, epiglottic cartilage	Vagus nerve (X) Superior laryngeal nerve	Right 4th aortic arch: subclavian artery Left 4th aortic arch: aortic arch	Superior parathyroid, ultimobranchial body (which forms the Para follicular C-Cells of thyroid gland)
6th	All intrinsic muscles of larynx except the cricothyroid muscle	Cricoid cartilage, arytenoid cartilages, corniculate cartilage	Vagus nerve (X) Recurrent laryngeal nerve	Right 6th aortic arch: pulmonary artery Left 6th aortic arch: Pulmonary artery and ductus arteriosus	Rudimentary structure, becomes part of the fourth pouch contributing to thyroid C-cells.

PAGE 784

APPENDIX- 10 (NEW AND IMPROVED, replace with older one)

SOME IMPORTANT EPITHELIUM LININGS:

System	Tissue	Epithelium
Circulatory	Blood vessels, Lymph vessels	Simple squamous
Digestive	Ducts of submandibular glands	Stratified columnar
Digestive	Gingiva, Dorsum of tongue, Hard palate,	Stratified squamous, keratinized
Digestive	Oesophagus	Stratified squamous, non-keratinized
Digestive	Stomach, Small intestine, Large intestine, Rectum, Gall bladder	Simple columnar, non-ciliated
Digestive	Anus	Stratified squamous, non-keratinized superior to Hilton's white line Stratified squamous, keratinized inferior to Hilton's white line
Endocrine	Thyroid follicles	Simple cuboidal
Nervous	Ependyma	Simple cuboidal
Integumentary	Skin - superficial layer	Stratified squamous, keratinized
Integumentary	Sweat gland ducts	Stratified cuboidal
Integumentary	Mesothelium of body cavities	Simple squamous
Reproductive - female	Ovaries	Simple cuboidal
Reproductive - female	Fallopian tubes, Endometrium (uterus)	Simple columnar, ciliated
Reproductive - female	cervix (endocervix)	Simple columnar
Reproductive - female	cervix (ectocervix), Vagina	Stratified squamous, non-keratinized
Reproductive - female	Labia majora	Stratified squamous, keratinized
Reproductive - male	Rete testis	Simple cuboidal
Reproductive - male	Ductuli efferentes, Vas deferens, Seminal vesicle	Pseudostratified columnar
Reproductive - male	Epididymis	Pseudostratified columnar, with stereocilia
Reproductive - male	Ejaculatory duct	Simple columnar
Respiratory	oropharynx, Lingual epiglottis	Stratified squamous, non-keratinized
Respiratory	Larynx, Laryngeal epiglottis, Trachea	Pseudostratified columnar, ciliated
Respiratory	Larynx - True vocal cords	Stratified squamous, non-keratinized
Respiratory	Respiratory bronchioles	Simple cuboidal
Sensory	Cornea	Stratified squamous, non-keratinized
Sensory	Nose	Pseudostratified columnar

System	Tissue	Epithelium
Urinary	Kidney – PCT	Simple cuboidal, with microvilli
Urinary	Kidney - Ascending thin limb	Simple squamous
Urinary	Kidney – DCT	Simple cuboidal, without microvilli
Urinary	Kidney - Collecting duct	Simple cuboidal
Urinary	Renal pelvis, Ureter, Urinary bladder, Prostatic urethra,	Transitional
Urinary	Membranous urethra, Penile urethra	Pseudostratified columnar, non-ciliated
Urinary	External urethral orifice	Stratified squamous

APPENDIX-19

CYTOKINES:

NAME	MAJOR CELLULAR SOURCE	SELECTED BIOLOGIC EFFECTS
IFN -α, β	Macrophages (IFNα) fibroblasts (IFNβ)	Antiviral
IFN- 'Ɣ	$CD4^+$ T cells, NK cells	Activates macrophages, TH1 differentiation
TNF-α	Macrophages, T cells	Cell activation, Fever, cachexia, antitumor
TNF-β, LT (lymphotoxin)	T cells	Activates PMNs
IL-1	Macrophages	Cell activation, Fever (Pro inflammatory)
IL-2	T cells	T cell growth and activation
IL-3	T cells	Hematopoiesis
IL-4	T cells, mast cells	B cell proliferation and switching to IgE, TH2 differentiation
IL-5	T cells	Differentiation of eosinophil, activates B cells
IL-6	T cells, Macrophages	Both pro inflammatory and anti inflammatory, mediator of fever & acute phase response
IL-7	Bone marrow stroma cells	T cell progenitor differentiation
IL-8	Macrophages, T cells	Chemotactic for neutrophils
IL-10	Macrophages, T cells	Inhibits activated macrophages and dendritic cells, Anti-inflammatory
IL-12	Macrophages	Differentiation of T cells, activation of NK cells
GM-CSF	T cells, macrophages, monocytes	Differentiation of myeloid progenitor cells
M-CSF	Macrophages, monocytes, fibroblasts	Differentiation of monocytes and macrophages
G-CSF	Fibroblasts, monocytes, macrophages	Stimulates neutrophil production in bone marrow

APPENDIX-21

HYPERSENSITIVITY REACTIONS

TYPE 1: Allergy/Atopy/ Anaphylaxis	**True Anaphylaxis** Free Ag →fixed IgE (on mast cell		Wheal & Flare reaction, Casoni's test, anaphylaxis, prusnitz kunster reaction, Theobald smith phenomenon, Schultz dale phenomenon, Atopic dermatitis,
	Pseudoanaphylaxis (Anaphylactoid reaction) Free Ag→direct degranulation of mast cell (not IgE mediated)		Iodine-containing radio contrast (mc), aspirin and muscle relaxants, morphine
TYPE 2: Antibody dependent cytotoxic hypersensitivity	Free Ab→fixed Ag	IgM IgG/ Complement mediated	Autoimmune hemolytic anemia Hemolytic disease of the newborn (erythroblastosis fetalis) Autoimmune thrombocytopenic purpura Goodpasture's syndrome Pemphigus vulgaris Bullous pemphigoid Acute rheumatic fever Pernicious anemia Myasthenia gravis Graves' disease
		ADCC- antibody-dependent cell-mediated cytotoxicity	Acute & chronic transplant rejection
TYPE 3: Immune complex	Free Ag+ Free Ab→**Immune complex**		Serum sickness Arthus reaction Systemic lupus erythematosus (SLE) Immunoglobulin therapy Hyperacute graft rejection Acute necrotizing vasculitis Polyarteritis nodosa Post streptococcal glomerulonephritis Shick's test
TYPE 4: Delayed hypersensitivity, cell-mediated, antibody-independent	Ag +T cell mediated (CD4/CD8)	**Delayed:** CD4 mediated [contact & tuberculin types with in 72 hours, Granulomatous type; 21-28	Contact dermatitis Mantoux (tuberculin) test Lepromin test Acute transplant rejection Multiple sclerosis Phlyctenular keratoconjunctivitis Jones mote reaction (cutaneous basophilic hypersensitivity)

		days]	
		<u>Direct cell toxicity</u>: CD8 mediated	Perforin dependent killing Fas-FasL dependent killing

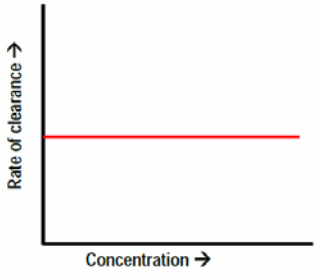
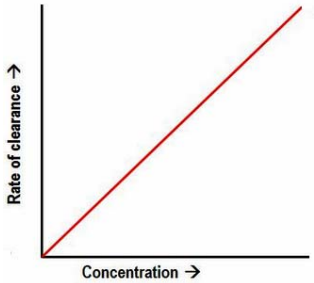
Also know: <u>Graft rejection</u> is type 4 hypersensitivity unless type is provided. (Most of the MCQ guides have quoted this falsely)

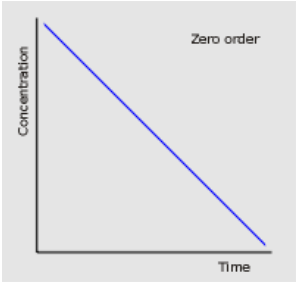
Hyperacute	Type 3
Acute	Type 4>2 (both if provided) Acute cellular rejection = type 4 (better prognosis) Acute vascular rejection = type 2
Chronic	Type 2

PAGE 816

<u>APPENDIX-33</u>

<u>CHEMICAL KINETICS OF DRUGS</u>

	Zero order kinetics	**First order kinetics**	**Second order kinetics**
Rate of reaction	Rate of reaction is independent of the concentration of the reactant(s) Constant amount of drug elimination per unit time	Rate of reaction is proportional to the concentration of only one reactant Constant fraction of drug elimination per unit time	Rate of reaction is proportional to the concentrations of one second-order reactant, or two first-order reactants.
Rate of elimination / Clearance	Independent of plasma concentration (i.e. Constant with concentration) 	Proportional to plasma concentration (i.e. Less at low concentration, More at high concentration) 	Proportional to the concentration of the square of a single reactant or the product of the concentration of two reactants
Half life	**T ½ = $[A_0]$/2K** Less at low concentration More at high concentration	**T ½ = 0.693/K** I.e. T ½ is Constant, t ½ depends on K only t ½ is independent of initial concentration	**T ½ = 1/K $[A_0]$** Each successive half-life is double the preceding one. t ½ depends on both K and initial concentration
Example	*<u>Mnemonic= Zero WATT</u>*	Most of the drugs follows	Gas -phase decomposition

	Power • **Zero** order kinetics by • **W** = warfarin • **A** = Alcohol, Aspirin • **T** = Theophylline • **T** = Tolbutamide • **Power** = Phenytoin	first order kinetics	of NO_2 ($2\ NO_2 \rightarrow 2\ NO + O_2$)
Graph	Linear relationship between time from peak concentration and plasma concentration 		

PAGE 852

APPENDIX-57

TYPES OF NERVE INJURY

Sunderland classification of injuries describes three types: neuropraxia, axonotmesis, and neurotmesis.

		Neuropraxia	Axonotmesis	Neurotmesis
Severity		Least severe	Severe	Most severe
Pathology	myelin	physiologic block of impulse conduction without anatomic destruction	Lost	Lost
	Axon		Lost	Lost
	Endoneurium		Intact	Lost
	Perineurium		Intact	Lost
	Epineurium		Intact	Occasionally intact
Electrical phenomena	EMG	Normal	Fibrillations	Fibrillations
	Distal conduction	Preserved	Absent	Absent
	Motor unit action potential	Absent	Absent	Absent
Wallerian degeneration		Absent	Present	Present
Neuroma		Absent	Neuroma in continuity	End-neuroma
Losses distal to injury		Motor>sensory,	All sensory motor &	All sensory motor &

		Autonomic intact	autonomic	autonomic
Example		Pressure Ischemia	Crash injury	Transection, stretch, laceration, LA toxicity
Recovery	Repair	Not required	Not necessary	Necessary
	Rate	Hours to weeks, spontaneous	1-2 mm/day after repair	1-2 mm/day after repair
	Motor march	Absent (no order)	As per order of innervation	As per order of innervation
	Quality	Perfect	Perfect	Always imperfect/ incomplete

PAGE 866

APPENDIX-68

ALPHABETICAL LIST OF EPONYMOUS FRACTURES

Fracture	Description	Mechanism of injury
Aviator's fracture	Fracture neck of talus	Dorsiflexion
Bumper fracture	compression fracture of lateral condyle of tibial (always intra articular)	Forced valgus of knee when struck from side by car bumper
Boxer's fracture	Fracture of distal 5th metacarpal	Punching solid object
Bosworth fracture	Fracture of distal fibula with posterior dislocation of the proximal fibula behind the tibia	Severe external rotation of the foot
Bennett's fracture	Intra-articular fracture of base of first metacarpal	Axial load along metacarpal in a partially flexed thumb (Abductor pollicis longus pull)
Barton's fracture	Intra articular distal radius fracture involving the articular surface with dislocation of the radio carpal joint	Fall on outstretched hand
Bankart's fracture	Fracture of anterior glenoid associated with anterior shoulder dislocation	External rotation and abduction of shoulder
Colles' fracture	Distal radius fracture with dorsal angulation, impaction and radial drift	Fall on outstretched hand
Cotton's fracture	Trimalleolar fracture of ankle	
Clay shoveller's fracture	Stress avulsion fracture of Spinous process of C6, C7 or T1	Forced hyper flexion of neck
Chopart's fracture-dislocation	Foot dislocation through talonavicular and calcaneocuboid joints with associated fractures, usually after ankle twisting. Treated in a non-weight bearing cast for 6-8 weeks	
Chauffeur's fracture	Intra-articular fracture of radial styloid	Forced ulnar deviation of the wrist causing avulsion of the radial styloid
Chance fracture	Horizontal fracture of vertebral body	Hyper flexion of spine, seen in car accidents when lap belts were used

Duverney fracture	Isolated fracture of the iliac wing	Direct trauma
Essex-Lopresti fracture	Comminuted radial head fracture with interosseous membrane disruption and distal radioulnar joint subluxation	Fall from height
Gosselin fracture	V-shaped distal tibia fracture extending into the tibial plafond	
Galeazzi fracture	Radius shaft fracture with dislocation of distal radioulnar joint	Blow to forearm
Holdsworth fracture	Unstable spinal fracture-dislocation at the thoracolumbar junction	
Hume fracture	Olecranon fracture with anterior dislocation of radial head	
Hill-Sachs fracture	Impacted posterior humeral head fracture occurring during anterior shoulder dislocation	
Hangman's fracture	Fracture of both pedicles of C2	Distraction and extension of neck (judicial hanging)
Jones fracture	Fracture of base of 5th metatarsal extending into intermetatarsal joint	Inversion of ankle (pronator brevis pull)
Jefferson fracture	Burst fracture of 1st cervical vertebra	Compression of neck
Lisfranc fracture	Fracture dislocation of midfoot	Forced plantar flexion of foot or dropping heavy weight on foot
Le Fort's fracture of the ankle	Vertical fracture of distal fibula with avulsion of medial malleolus	
Le Fort fractures	Series of facial fractures	Direct trauma to face
Moore's fracture	Distal radius fracture with ulnar dislocation and entrapment of styloid process under annular ligament	
Monteggia fracture	Proximal ulna fracture with dislocation of radial head	Blow to forearm
March fracture	Stress fracture of 2nd /3rd metatarsal shaft	Heavy or unaccustomed exercise
Malgaigne's fracture	Vertical pelvic fracture through both pubic rami and the ilium or sacroiliac joint with vertical displacement	High energy impact to pelvis (front to back)
Maisonneuve fracture	Spiral fracture of proximal fibula	External rotation of ankle
Pipkin fracture-dislocation	Posterior dislocation of hip with avulsion fracture of fragment of femoral head by the ligamentum teres	Impact to the knee with the hip flexed (dashboard injury)
Pilon (Hammer) fracture	Intra-articular fracture of tibial plafond. Usually but not always with fibular fracture. Usually but not always with fibular fracture	High velocity injuries
Pott's fracture	Bimalleolar fracture of the ankle	Eversion of ankle
Rolando fracture	Intra articular T or Y shaped Comminuted fracture of base of 1st metacarpal	Axial load along the metacarpal causing splitting of the proximal articular surface
Runner's fracture	Stress fracture of distal fibula 3-8cm above the lateral malleolus	Repeated axial stress on fibula
Stieda fracture	Avulsion fracture of the medial femoral condyle at the origin of the medial collateral ligament	
Smith's fracture	Distal radius fracture with volar displacement	Fall on outstretched hand with wrist in flexed position
Shepherd's fracture	Fracture of the lateral tubercle of the posterior process of the talus	
Segond fracture	Lateral tibial plateau avulsion fracture with anterior	Internal rotation of the knee

	cruciate ligament tear	
Salter-Harris fractures	Fractures involving a growth plate	various
Tillaux fracture	Salter-Harris III fracture of the tibia	Forced lateral rotation of foot
Toddler's fracture	Undisplaced spiral fracture of distal tibia in children under 8 years old	Low-energy trauma, often rotational

PAGE 871

APPENDIX-70

HEALTH RELATED DAYS:

World leprosy day	30 January
World cancer day	4th February
World disabled day	15th March
World TB day	24th March
World Health day	7th April
World Malaria day	25th April
Red cross day	8th May
World no tobacco day	31st May
Anti Filaria day	5th June
World blood donation day	14th June
International day against drug abuse	26th June
World diabetes day	27th June
World Zoonosis day	6th July
World population day	11th July
World breast feeding week	1-7 August
Suicide prevention day	10th September
Alzheimer's day	21st September
World Rabies day	28th September
World Heart day	29th September
International day for elderly	1st October
Mental health day	10 October
Ether day/ Anesthesia day	16th October
World diabetes day	14th November
World RTA day	16th November
World COPD day	19th November
AIDS day	1st December
Hepatitis day	4th December

Note:

- On 20 December 2006, the United Nations General Assembly passed Resolution 61/225. It designates 14 November, the current World Diabetes Day.

- Earlier WHO announced 14th novenber as world diabetes day in the year 1998.
- From 1991 to 1998 WHO celebrated world diabetes day on 27th June.
- In India the world diabetes day is still celebrated on 27th of June every year.